



# Attachment 21

# THE COST OF INACTION

**A socioeconomic analysis of  
environmental and health  
impacts linked to exposure  
to PFAS**





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*Gretta Goldenman, Meena Fernandes, Michael Holland, Tugce Tugran, Amanda Nordin, Cindy Schoumacher and Alicia McNeill*

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and Alicia McNeill

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# Preface

## Purpose of this report

There is a growing demand for monetary values within chemical policy. The purpose of this report is to estimate the costs for society related to the negative effects and impacts on human health and environment due to the exposure to PFAS and C<sub>4-14</sub> non-polymer fluoro-surfactants in particular. The purpose is also to highlight the economic case for taking effective and timely action to manage the risks of negative impacts.

The information in this report is intended to be used to raise awareness on the costs and long-term problems that the use of PFAS may cause for the environment and human health. The use of monetary values provides an additional important basis for strategic decisions within chemical agencies both at the national level as well as on the EEA level.

## Disposition

The report is divided into three main parts. The first part provides a regulatory baseline and outlines the methodology to assess the socioeconomic costs related to the negative impacts on the environment and human health. The second part presents five case studies chosen for this study. They are aimed at illustrating the key pathways for impacts from PFAS and to gather information on actual costs incurred by society in reducing exposure to PFAS. The third part presents the estimates of health and environment-related costs of inaction linked to exposure to PFAS as well as the aggregated costs of inaction.

## Scope and limitations

This study focuses on the C<sub>4-14</sub> non-polymer fluoro-surfactants with the aim of providing a monetised estimate of total damage to health and the environment associated with PFAS exposures in the European Economic Area (EEA). The report therefore focuses on costs of inaction in the EEA countries. It uses data specific to Nordic countries when available, but also draws cost data from other European countries, the USA and Australia, where relevant. Not all costs can be quantified and monetised; some costs are therefore assessed qualitatively.

The study considers only the socioeconomic costs incurred by society due to impacts from PFAS exposures. It does not include or monetise costs for business such as for example substitution costs.

## Financing and workforce

The study was carried out by Milieu Consulting in Brussels. Authors include Gretta Goldenman, Meena Fernandes, Tugce Tugran, Amanda Nordin, Cindy Schoumacher and Alicia McNeill from Milieu and Michael Holland from EMRC. The health economic models and calculations were developed and described by Meena Fernandes. The methodological framework for estimating costs for society, as well as the environmental economic models and calculations, were developed and described by Michael Holland.

The Nordic Chemical Group (NKG) has been the project principal. The steering group under NKG comprised members from Sweden, Denmark, Norway and Iceland, including Toke Winther and Lars Fock (The Danish Environmental Protection Agency), Audun Heggelund (Norwegian Environment Agency), Signe Krarup (The Danish Ministry of Environment and Food) and Åsa Thors (The Swedish Chemicals Agency). Experts supporting the steering group for parts of the project included Jenny Jans, Inger Cederberg, Mattias Carlsson Feng, Daniel Borg (The Swedish Chemicals Agency) and Ísak Sigurjón Bragason (The Environment Agency of Iceland).

## Acknowledgments

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### *Background and case study information*

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# Summary

This study investigates the socioeconomic costs that may result from impacts on human health and the environment from the use of PFAS (per and polyfluoroalkyl substances). Better awareness of the costs and long-term problems associated with PFAS exposure will assist authorities, policy-makers and the general public to consider more effective and efficient risk management.

The production of PFAS, manufacture and use of PFAS-containing products, and end-of-life disposal of PFAS have resulted in widespread environmental contamination and human exposure. PFAS have been found in the environment all around the world and almost everyone living in a developed country has one or more PFAS in his/her body.

Because of the extreme persistence of PFAS in the environment, this contamination will remain on the planet for hundreds if not thousands of years. Human and environmental exposure will continue, and efforts to mitigate this exposure will lead to significant socioeconomic costs – costs largely shouldered by public authorities and ultimately taxpayers.

The focus of this study is on the costs of inaction with respect to regulation of PFAS in the countries comprising the European Economic Area (EEA). Costs of inaction are defined as the costs that society will have to pay in the future if action is not taken to limit emissions of PFAS today. The PFAS covered in this study are the C<sub>4</sub>-14 non-polymer fluorosurfactants.

The goal for the study has been two-fold:

1. to establish a framework for estimating costs for society related to negative impacts on health and the environment associated with PFAS exposure; and
2. to provide monetary values for those societal costs, documented by case studies.

## Conclusions

The work of estimating the health and environment-related costs to society related to PFAS exposure has relied on the development of assumption-based scenarios. This reflects the limited data available in the academic literature, government documents and press reports. Whilst the uncertainties of the analysis need to be acknowledged, it is also important to recognise that, for several issues, there is little or no uncertainty:

1. PFAS are ubiquitous in the environment, and almost all people have PFAS in their bodies today. Monitoring in both Sweden and the USA concludes that around 3% of the population are currently exposed above proposed limit values, primarily through contamination of drinking water but also via other sources;
2. Many sources of PFAS exposure exist, linked to specialist applications (e.g. AFFFs for firefighting at airports and some industrial locations) and non-specialist uses (e.g. use in consumer goods such as pizza boxes, clothing and cosmetics);
3. Non-fluorinated alternatives for many of these uses are already on the market, and therefore certain uses of PFAS can be reduced;
4. The costs for remediating some cases of contamination run to many millions of EUR. Total costs at the European level are expected to be in the hundreds of millions of EUR as a minimum;
5. A large and growing number of health effects have been linked to PFAS exposure and evidence is mounting that effects occur even at background level exposures.

Current and proposed limit values for drinking water may be further reduced in recognition of growing information on, health and environmental risks. This would increase the costs of environmental remediation estimated here.

As explained throughout the study, the calculations rest on a number of assumptions, though these have been checked against e.g. data on costs incurred to ensure that they are linked to real-world experience. As more information becomes available, calculations will become more precise. Moreover, these findings are conservative. The figures are likely to get larger, in that the numbers of PFAS on the market and the volumes produced keep increasing. Further inaction will lead to more sources of contamination, more people exposed, and higher costs for remediation. The longer that PFAS contamination remains in the environment without remediation, the wider it will spread and the greater the quantity of soil or groundwater that will need to be decontaminated.

## Methodology

Two methodologies have been developed, one for estimating health-related costs, the other for estimating costs of environmental remediation. Both methodologies are based on cases concerning exposure to PFAS. Data from the Nordic countries have been used when available, but the estimates also draw on cost data from other European countries, the USA and Australia, where relevant.

## Impact pathways (the case studies)

Five case studies following the life-cycle of PFAS, from their production and use in product manufacturing, to the product's use and end-of-life disposal are used to illustrate how exposures to humans and the environment occur. Other instances of PFAS contamination provide additional data on direct costs incurred.

Case Study 1 considers exposures due to the production of PFAS in Europe. It reviews pollution linked to the Chemour factories in Dordrecht, Netherlands, the Miteni facility in the Veneto region of Italy, and the 3M plant near Antwerp, Belgium. The study estimates that up to 20 facilities actively produce fluorochemicals in Europe, that these facilities are significant sources of PFAS released to the environment, and that the exposure of workers at these plants is high.

The impacts from the manufacture and commercial use of PFAS-containing products are the focus of Case Study 2. Industrial activities with the potential to release PFAS to the environment include textile and leather manufacturing; metal plating, including chromium plating; paper and paper product manufacturing; paints and varnishes; cleaning products; plastics, resins and rubbers; and car wash establishments. The study assumes that a range of 3% to 10% of these facilities use PFAS. The study did not identify any fluorochemical production facilities in the Nordic countries. However, Eurostat statistics indicate that other industrial activities with the potential to release PFAS to the environment do take place in the region, such as metal plating and manufacture of paper products.

Case Studies 3 and 4 consider the use phase of PFAS-containing products. Case Study 3 examines exposure to PFAS-containing aqueous film-forming foams (AFFFs) used in firefighting drills and to extinguish petroleum-based fires. The AFFFs have contributed to groundwater contamination, especially around airports and military bases. Nearby communities have been affected by elevated levels of PFAS in their drinking water. Case Study 4 looks at PFAS-treated carpets, PFAS-treated food contact materials, and cosmetics as examples of how a product's use is likely to lead to direct human exposure through ingestion and dermal absorption. The use of products also result in releases to the environment when the product is washed off or laundered, entering sewers and treatment plants, and eventually waterways.

Case Study 5 looks at end-of-life impacts of PFAS-treated products. Municipal waste incineration may destroy PFAS in products if 1000 °C operating temperatures are reached. If landfilled, the PFAS will remain even after the product's core materials break down. The compounds will eventually migrate into liquids in the landfill, then into leachate collection systems or directly into the natural environment. They may then contaminate drinking water supplies, be taken up by edible plants and bioaccumulate in the food chain.

## Health-related costs to society

To calculate health-related costs to society, the researchers looked for consensus regarding health endpoints affected by exposure to PFAS. Reviews of the scientific evidence have reached contradictory conclusions about the relevant health endpoints of human exposure to PFAS. However, some consensus has emerged concerning liver damage, increased serum cholesterol levels (related to hypertension), decreased immune response (higher risk of infection), increased risk of thyroid disease, decreased fertility, pregnancy-induced hypertension, pre-eclampsia, lower birth weight, and testicular and kidney cancer.

The methodology draws upon risk relationships developed in the course of specific epidemiological studies for populations exposed to PFAS at different levels. Workers exposed to PFAS in the workplace were used to exemplify a high level of exposure. Communities affected by PFAS, e.g. because of proximity to manufacturing sites or sites where fluorinated AFFFs were used, were assumed to have been exposed at a medium level; this level of exposure was assumed to have been experienced by 3% of the European population. The general population was considered to have experienced exposure at low (background) levels.

Table 1 provides an overview of the estimated annual costs for just a few health endpoints where risk ratios were available for affected populations. For example, the annual health-related costs for the elevated risk of kidney cancer due to occupational exposure to PFAS was estimated to be on the order of EUR 12.7 to EUR 41.4 million in the EEA countries. The estimated costs were substantially higher for elevated and background levels of exposure due to the greater number of persons affected. The total annual health-related costs, for the three different levels of exposure, was found to be at least EUR 2.8 to EUR 4.6 billion in the Nordic countries and EUR 52 to EUR 84 billion in the EEA countries.<sup>1</sup> Despite the high level of uncertainty and the assumptions underlying the calculations, the findings suggest that the health-related costs of exposure to PFAS are substantial.

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<sup>1</sup> The health-related costs due to occupational exposure to PFAS in the Nordic countries was not estimated due to an absence of information about the number and location of chemical production plants or manufacturing sites.

**Table 1: Estimates of annual health impact-related costs (of exposure to PFAS)**

Exposure level	"Exposed" population and source	Health endpoint	Nordic countries		All EEA countries	
			Population at risk	Annual costs	Population at risk	Annual costs
Occupational (high)	Workers at chemical production plants or manufacturing sites	Kidney cancer	n.a.	n.a.	84,000–273,000	EUR 12.7–41.4 million
Elevated (medium)	Communities near chemical plants, etc. with PFAS in drinking water	All-cause mortality	621,000	EUR 2.1–2.4 billion	12.5 million	EUR 41–49 billion
		Low birth weight	8,843 births	136 births of low weight	156,344 births	3,354 births of low weight
		Infection	45,000 children	84,000 additional days of fever	785,000 children	1,500,000 additional days of fever
Background (low)	Adults in general population (exposed via consumer products, background levels)	Hypertension	10.3 million	EUR 0.7–2.2 billion	207.8 million	EUR 10.7–35 billion
<i>Totals</i>			<i>Nordic countries</i>	<i>EUR 2.8–4.6 billion</i>	<i>All EEA countries</i>	<i>EUR 52–84 billion</i>

Some overlap occurs in the figures above, because workers and affected communities are also exposed to background levels of PFAS. At the same time, these costs are likely to be underestimates due to the lack of epidemiological-based risk relationships for calculating other health endpoints and related costs.

## Non-health (environment-related) costs to society

The second methodology compiled information on direct costs incurred by communities taking measures to reduce PFAS exposure through remediation of drinking water. Based on these direct costs, ranges of costs per persons affected or per case were developed. These unit costs then became the foundation for aggregating the costs of remediation when environmental contamination, e.g., PFAS concentrations in drinking water, reach certain levels. It should be noted that the ranges are broad, even when normalized against population.

The approach to derive ranges for the mean is dependent on the amount of data available. For the costs of water treatment, for example, several estimates were available, and in such cases it is unlikely that the true mean will be at either extreme of the range from the studies. Therefore, it is reasonable to truncate the observed range, for example by removing estimates that are sufficiently removed from other data as to be considered outliers. For some costs, however, very few estimates are available, each of which may be equally valid for representation of the average: in such a case the observed range in values is adopted as the range of plausible mean values.

Where no range is available from the studied literature, a range has been estimated. For example, the range of +/-90% is used for establishing a health assessment regime (here considered as a non-health cost as it deals with management of the problem, rather

than impacts on the health of society). In this example, the range is extremely broad for two reasons, first because of the lack of data available and second because of the potential for variation in the implementation of a health assessment programme.

As with the health-based estimates, the study assumes that 3% of the European population is exposed to drinking water with PFAS concentrations over regulatory action levels, such that the water treatment works serving them will require upgrading and maintenance over the next 20 years. The assumption of 20 years reflects potential for remediation to resolve problems perhaps through decontamination or the use of alternative supplies, or the potential for remedial action to persist for many years. Recognising the uncertainties that exist in the analysis and the available data, costs of remediation have been quantified using a scenario-based approach. For each scenario a number of parameters are specified, relating for example to the size of the affected population and the duration of maintenance works.

Table 2 shows the range of costs for the various categories of actions related to environmental remediation.

**Table 2: Summary of estimates of mean cost data for non-health expenditures, 20 years**

Action taken when PFAS found	Unit	Best estimate	Range from studies	Adopted range
Monitoring – checks for contamination due to industrial or AFFF use	Cost per water sample tested	EUR 340	EUR 278–402	EUR 278–402
	Cost/case of contamination	EUR 50,000	EUR 5,200–5.8 million	EUR 25,000–500,000
Health assessment (including biomonitoring)	Cost/person	EUR 50	No range	EUR 5–95 (+/-90%)
	Total biomonitoring and health assessment per case where considered appropriate	EUR 3.4 million	EUR 2.5 million–4.3 million	EUR 1 million–5 million
Provision of temporary uncontaminated supply	Cost/person	No relevant data		
Provision of a new pipeline	Cost/person	EUR 800	EUR 37–5,000	EUR 100–1,500
Upgrading water treatment works (capital)	Cost/person	EUR 300	EUR 8–2,200	EUR 18–600
Upgrading water treatment works (maintenance)	Cost/person	EUR 19	EUR 8–30	EUR 8–30
Excavation and treatment of soils – contamination from industrial or AFFF use	Cost/kg PFAS	EUR 280,000	EUR 100,000–4.3 million	EUR 100,000–1 million
	Cost/case	EUR 5 million	EUR 100,000–3 billion	EUR 300,000–50 million

In Table 3 the range of costs for the various categories of actions related to environmental remediation for the five Nordic countries are shown. The overall range of costs is EUR 46 million – 11 billion.

**Table 3: Detailed breakdown of ranges for non-health costs to the Nordic countries, assuming that 1 to 5% (best estimate 3%) of the population is exposed above a statutory limit and that water treatment is required over a 20 year period**

	N people affected (3%)	Screening and monitoring	Health assessment	Upgrade treatment works and maintenance	Soil remediation	Total
Denmark	170,000	EUR 70,000–8.3 million	EUR 280,000–27 million	EUR 7.4 million–274 million	EUR 0–798 million	EUR 8 million–1.1 billion
Finland	160,000	EUR 250,000–22 million	EUR 270,000–26 million	EUR 7.2 million–265 million	EUR 2.2 million–2.1 billion	EUR 10 million–2.4 billion
Iceland	10,000	EUR 10,000–900,000	EUR 20,000–1.6 million	EUR 400,000–1.6 million	EUR 100,000–86 million	EUR 1 million–105 million
Norway	160,000	EUR 170,000–20 million	EUR 260,000–25 million	EUR 6.8 million–250 million	EUR 1.6 million–1.9 billion	EUR 9 million–2.2 billion
Sweden	290,000	EUR 480,000–47 million	EUR 490,000–46 million	EUR 13 million–472 million	EUR 4.3 million–4.5 billion	EUR 18 million–5.1 billion
<i>Nordic total</i>	<i>790,000</i>					<i>EUR 46 million–11 billion</i>

The cost estimates provided in the table are likely to be more robust at the aggregate, European level than at the national level.

Table 4 provides aggregated costs covering environmental screening, monitoring (where contamination is found), water treatment, soil remediation and health assessment for the five Nordic countries and for the other EEA countries and Switzerland.

**Table 4: Aggregated costs covering environmental screening, monitoring where contamination is found, water treatment, soil remediation and health assessment**

	Best estimate	Low	High
Denmark	EUR 145 million	EUR 8 million	EUR 1.1 billion
Finland	EUR 214 million	EUR 10 million	EUR 2.4 billion
Iceland	EUR 12 million	EUR 1 million	EUR 105 million
Norway	EUR 194 million	EUR 9 million	EUR 2.2 billion
Sweden	EUR 423 million	EUR 18 million	EUR 5.1 billion
Other EEA+CH	EUR 15.9 billion	EUR 776 million	EUR 159.9 billion
Total	EUR 16.9 billion	EUR 821 million	EUR 170.8 billion

Parallel calculations for all 31 EEA Member Countries and Switzerland arrive at a range of costs for environmental remediation totalling EUR 821 million to EUR 170 billion. The

lower and upper bounds should be considered illustrative because of the limited information available. However, based on the literature review, there is a firm basis for concluding that the lower bound estimates would be exceeded. A best estimate in the order of EUR 10–20 billion is certainly plausible. The potential for higher costs is also possible: An estimate of the costs for one case identified in the course of the research, concerning the town of Rastatt in Baden-Wurttemberg in Germany is in the range of EUR 1 to 3 billion, with the estimated extent of the problem being seen to increase over time. The source of contamination in this case is understood to be contaminated waste paper materials that were spread on agricultural land, demonstrating that serious problems are not always linked to airfields and PFAS manufacture.

A number of other costs related to PFAS contamination are outside the scope of the quantification carried out in this report. These include loss of property value, reputational damage to a polluting company, ecological damage and the costs incurred by public authorities in responding to affected communities – including public outreach, surveys of contamination and remedial measures.

## Abbreviations used

<i>6:2 FTS</i>	6:2 Fluorotelomer sulfonate
<i>AFFF</i>	Aqueous film-forming foam (also aqueous firefighting foam)
<i>ATSDR</i>	US Agency for Toxic Substances and Disease Register
<i>BB/CC</i>	Beauty (or Blemish) Balm / Colour Corrector
<i>CA DTSC</i>	California Department of Toxic Substances Control
<i>CAS</i>	Chemical Abstracts Service
<i>CLH</i>	Harmonised classification and labelling
<i>CLP</i>	Classification, labelling and packaging or Regulation (EC) No 1272/2008 on the classification, labelling and packaging of substances and mixtures
<i>CMR</i>	Carcinogenic, mutagenic and toxic for reproduction
<i>C8</i>	Alternative name for PFOA (due to its eight carbon atoms)
<i>D4/D5</i>	Octamethylcyclotetrasiloxane (D4); decamethylcyclopentasiloxane (D5)
<i>DALY</i>	Disability-adjusted life year
<i>DW</i>	Drinking water
<i>ECHA</i>	European Chemicals Agency
<i>EDC</i>	Endocrine disrupting chemical/s
<i>EEA</i>	European Economic Area countries
<i>EFSA</i>	European Food Safety Authority
<i>EFTA</i>	European Free Trade Agreement
<i>E-PRTR</i>	European Pollutant Release and Transfer Registry
<i>EU</i>	European Union
<i>EUR</i>	Official currency for 19 of the 28 members of the European Union (EU)
<i>FCM</i>	Food contact material
<i>GAC</i>	Granular activated carbon
<i>GenX</i>	Replacement for PFOA
<i>GHS</i>	Globally Harmonized System of Classification and Labelling of Chemicals
<i>HFC</i>	Highly Fluorinated Chemical
<i>KEMI</i>	Swedish Chemicals Agency
<i>MCL</i>	Maximum contaminant level
<i>MS</i>	Member State
<i>NATO</i>	North Atlantic Treaty Organization
<i>NGO</i>	Non-governmental organization
<i>NHANES</i>	National Health and Nutrition Examination Survey (US)
<i>NOx</i>	Nitrogen oxide

<i>NO<sub>2</sub></i>	Nitrogen dioxide
<i>OECD</i>	Organisation for Economic Co-operation and Development
<i>PBT</i>	Persistent, bioaccumulative and toxic
<i>PFAS or PFASs</i>	Per- and polyfluoroalkyl substances
<i>PFBA</i>	Perfluorobutanoic acid
<i>PFBS</i>	Perfluorobutane sulfonic acid
<i>PFCAs</i>	Perfluorinated carboxylic acids
<i>PFCs</i>	Perfluorinated compounds
<i>PFDA</i>	Perfluorodecanoic acid
<i>PFDeA</i>	Perfluorodecanoic acid
<i>PFDoDA</i>	Perfluorododecanoic acid
<i>PFNA</i>	Perfluorononanoic acid
<i>PFHpA</i>	Perfluoroheptanoic acid
<i>PFHpS</i>	Perfluoroheptane sulfonic acid
<i>PFHxA</i>	Perfluorohexanoic acid
<i>PFHxS</i>	Perfluorohexane sulfonic acid
<i>PFHxSF</i>	Perfluorohexane sulfonyl fluoride
<i>PFOA</i>	Perfluorooctanoic acid
<i>PFOS</i>	Perfluorooctane sulfonic acid
<i>PFPE</i>	Perfluoropolyether
<i>PFPeA</i>	Perfluoropentanoic acid
<i>PFSA</i> s	Perfluoroalkane sulfonates
<i>PFTDA</i>	Perfluorotetradecanoic acid
<i>PFTTrDA</i>	Perfluorotridecanoic acid
<i>PFUnDA</i>	Perfluoroundecanoic acid
<i>PM</i>	Particulate matter
<i>POPs</i>	Persistent Organic Pollutants
<i>POSF</i>	Perfluorooctane sulfonyl fluoride
<i>PPP</i>	Purchasing power parity
<i>PTFE</i>	Polytetrafluoroethylene (Teflon)
<i>PVDF</i>	Polyvinylidene fluoride
<i>RAC</i>	Risk Assessment Committee (under REACH)
<i>REACH</i>	Regulation (EC) No 1907/2006 concerning the Registration, Evaluation, Authorisation and Restriction of Chemicals
<i>RIVM</i>	Dutch National Institute for Public Health and the Environment
<i>RME</i>	Risk management evaluation
<i>SEAC</i>	Socio-Economic Assessment Committee (under REACH)
<i>SEK</i>	Swedish krona
<i>SMEs</i>	Small and Medium Enterprises
<i>SMR</i>	Standardized mortality ratio
<i>SO<sub>x</sub></i>	Sulphur oxide
<i>SO<sub>2</sub></i>	Sulphur dioxide
<i>SVHC</i>	Substances of very high concern
<i>TDI</i>	Tolerable daily intake

<i>TOF</i>	Total organic fluorine
<i>UBA</i>	German Federal Environmental Agency (Umweltbundesamt)
<i>UNEP</i>	United Nations Environment Programme
<i>USD</i>	United States Dollar
<i>USEPA</i>	US Environmental Protection Agency
<i>USFAA</i>	US Federal Aviation Agency
<i>USFDA</i>	US Food and Drug Administration
<i>VAT</i>	Value-added tax
<i>VOCs</i>	Volatile organic compounds
<i>vP</i>	Very persistent
<i>vPvB</i>	Very persistent, very bio-accumulative
<i>WHO</i>	World Health Organization
<i>WTP</i>	Willingness to pay
<i>WWTP</i>	Wastewater treatment plant



# 1. Introduction

Per- and polyfluoroalkyl substances (PFAS) are a large group of chemical compounds that have been used in a wide range of commercial products since the 1950s. They are now found in the environment all around the world. Most people in industrialised countries have one or more PFAS in their blood.

PFAS are highly persistent. Though some PFAS may partially degrade under environmental conditions, they will all eventually transform into highly stable end products that will remain in the environment for hundreds or thousands of years<sup>2</sup>, such that human and environmental exposure will continue long into the future. Human epidemiological studies have found associations between exposure to PFAS and hepatocellular damage affecting liver function in adults, obesogenic effects in females, kidney cancer, low birthweight, reduced length of gestation, and reduced immune response to routine childhood immunizations.<sup>3</sup>

Because of their persistence, PFAS can travel long distances and have been found even in remote regions such as the high Himalayas and the Arctic where no direct sources of PFAS are known. The compound PFOA, for example, has been found in top predators such as polar bears.<sup>4</sup> Moreover, the PFAS tend to be highly mobile and to move readily into ground and surface waters once released to the environment.

In the 1950s, when highly fluorinated compounds were first commercialised, the focus was on long-chain PFAS – the so-called C8 substances used in the manufacture of Teflon-coated cookware, water- and stain-resistant textiles, and fire-fighting foams. Evidence emerged in the 1980s and 1990s of the toxicity and bio-accumulability of the long-chain PFAS, such as PFOS and PFOA. These long-chain surfactants have been well-studied and are now regulated in different parts of the world to varying extent, leading to complete or partial phase-outs in the EU and the USA. However, PFOA and its derivatives continue to be manufactured in China, India and Russia and as of 2017, China was reported to be the only known manufacturer of PFOS and its derivatives.<sup>5</sup> Despite being heavily restricted, these substances are still detected in some consumer products (see section 4.4.3 of this report concerning cosmetics), and other long-chain PFAS continue to be manufactured and used. Some producers have replaced the C8s with short-chain homologues – the C6s and C4s; they claim that the short-chain PFAS are “safer” in

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<sup>2</sup> Wang Z *et al.* (2017) A never-ending story of per- and polyfluoroalkyl substances (PFASs)? *Environmental Science & Technology*, Mar 7;51 (5). pp 2508–2518.

<sup>3</sup> Grandjean P *et al.* (2014). Changing interpretation of human health risks from perfluorinated compounds. *Public health reports*, vol. 129: (6). pp. 482–485.

<sup>4</sup> Vierke L *et al.* (2012). Perfluorooctanic acid (PFOA) – main concerns and regulatory developments in Europe from an environmental point of view. *Environmental Sciences Europe*. v 24: (16).

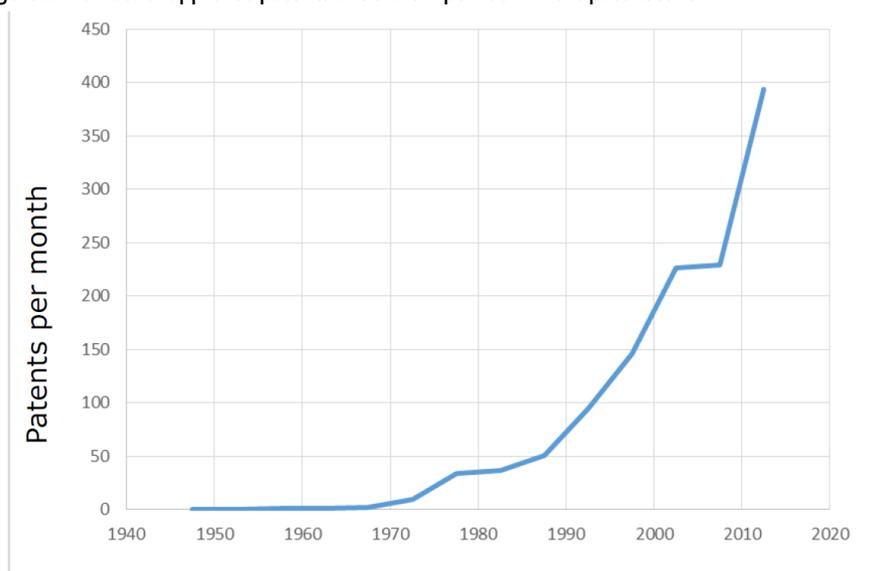
<sup>5</sup> Interstate Technology Regulatory Council (2017). History and use of per- and polyfluoroalkyl substances.

that they are not as bioaccumulative as the long-chain PFAS. In the meantime, evidence is emerging that the short-chain alternatives pose similar risks to human health.<sup>6</sup>

Moreover, the number of different PFAS on the global market keeps growing. A 2015 study reported more than 3,000 PFAS were on the global market for commercial use.<sup>7</sup> This number was updated in 2018 by a search carried out for the OECD which found over 4,700 different CAS numbers for perfluorinated compounds.<sup>8</sup> Other compounds may also be under production, but their identities are protected for confidential business reasons.

The number of possible applications of PFAS are also growing rapidly. Figure 1 shows an increasing trend in the number of patents with “perfluor” in the patent text that are approved in the USA each month.<sup>9</sup>

Figure 1: Number of approved patents in US with “perfluor” in the patent text



Source Fischer, S., 2017. “Known uses of PFAS”, presentation at Nordic workshop on joint strategies for PFAS, 5.04.2017.

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<sup>6</sup> Kotthoff M *et al.* (2015). Perfluoroalkyl and polyfluoroalkyl substances in consumer products. *Environmental Science and Pollution Research International*. 22(19): 14546–14559.

<sup>7</sup> Swedish Chemicals Agency (2015). Occurrence and use of highly fluorinated substances and alternatives: Report from a government assignment.

<sup>8</sup> For a list of 4,730 PFAS-related CAS numbers compiled from publicly accessible sources of information, see OECD (2018). *Toward a new comprehensive global database of per and polyfluoroalkyl substances (PFASs): summary report on updating the OECD 2007 list of per and polyfluoroalkyl substances (PFASs)*.

<sup>9</sup> Swedish Chemicals Agency (2015). Occurrence and use of highly fluorinated substances and alternatives: Report from a government assignment. Report 7/15.

A large proportion of these compounds are polymers and therefore exempted from registration requirements under the EU REACH Regulation<sup>10</sup>; of the others only a few are registered. Very little information is available on quantities produced and for half of all PFAS, almost no information can be found concerning their uses.

The quantities of PFAS produced globally also keeps growing. Fluorotelomers used primarily in aqueous firefighting foams (AFFFs), in textiles to provide stain resistance and surface finishing, and as surfactants are a major component of the market. A recent market research report estimated that production of fluorotelomers globally will grow from approximately 21,030,000 kg in 2013 to 47,800,000 kg by 2020, for a 2020 value of USD 539.3 million (EUR 466 million).<sup>11</sup> The main drivers of growth are an increased demand from the textile sector (34.8% of total demand in 2013) and government norms leading to use of AFFFs in firefighting systems.

Today, PFAS are found in cosmetics, food contact materials, inks, medical devices, mobile phones, pharmaceuticals and textiles. They are used in pesticide formulations, metal production, oil production and mining. They are capable of long-range transport, are highly mobile, and constitute a severe threat to clean water supplies around the globe.

The long-term socioeconomic costs of the PFAS already in products or released to the environment are poorly understood. PFAS released over the course of a product's lifecycle will remain in the natural and man-made environments for an indefinite time. One of the concerns is that the contamination may be poorly reversible or even irreversible, and may reach levels that could render natural resources such as soil and water unusable far into the future. This could result in continuous exposure and unavoidable harmful health effects, particularly for vulnerable populations, such as children. For example, PFOS in firefighting foams applied during the 2005 Buncefield explosion contaminated an aquifer that is an important public drinking water source for the Greater London area, so that it is no longer available as a water supply.<sup>12</sup>

Consensus statements from leading scientists studying PFAS, i.e., the Helsingør Statement<sup>13</sup>, the Madrid Statement<sup>14</sup>, and the Zurich Statement<sup>15</sup> highlight the health and environmental risks posed by the highly fluorinated chemicals as a group. The statements emphasize the extreme persistence of the carbon-fluorine bond in nature and call for regulatory as well as non-regulatory actions to address the risks associated with all highly fluorinated chemicals, including the short-chain PFAS.

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<sup>10</sup> Commission Regulation (EU) No 1907/2006 concerning the Registration, Evaluation, Authorisation and Restriction of Chemicals (REACH), establishing a European Chemicals Agency, amending Directive 1999/45/EC and repealing Council Regulation (EEC) No 793/93 and Commission Regulation (EC) No 1488/94 as well as Council Directive 76/769/EEC and Commission Directives 91/155/EEC, 93/67/EEC, 93/105/EC and 2000/21/EC. ("REACH Regulation"), O.J. 396, 30.12.2006, p. 1.

<sup>11</sup> Press release, Fluorotelomers Market to Reach USD 539.3 Million Worldwide by 2020, Digital Journal. Accessed 10.11.2018.

<sup>12</sup> Matt Gable, UK Environment Agency, as cited in IPEN (2018). Fluorine-free firefighting foams (3f) viable alternatives to fluorinated aqueous film-forming foams (AFFF).

<sup>13</sup> Scheringer M *et al.* (2014). Helsingør Statement on poly- and perfluorinated alkyl substances (PFASs)', *Chemosphere*, vol. 114, pp. 337–339.

<sup>14</sup> Blum A *et al.* (2015). The Madrid Statement on Poly- and Perfluoroalkyl Substances (PFASs). *Environmental health perspectives*. Vol. 123, no. 5. pp. A107–11.

<sup>15</sup> Ritscher A *et al.* (2018). Zürich Statement on Future Actions on Per- and Polyfluoroalkyl Substances (PFASs), *Environmental Health Perspectives*, vol. 126, no 8.

This study looks at how the production of PFAS, manufacture and use of PFAS-containing products, and end-of-life disposal of PFAS has resulted in widespread environmental contamination and human exposure, resulting in significant socio-economic costs. It sets forth a methodological framework for estimating costs for society related to negative impacts on the environment and human health, including health-related costs and costs for remediation, and uses case studies to illustrate the main impact pathways from PFAS releases and to gather information on direct costs incurred by society to date to reduce exposure to PFAS.

The focus of the study is on the costs of inaction in the countries comprising the European Economic Area (EEA). It uses data specific to Nordic countries when available, but also draws cost data from other European countries, the USA and Australia, where relevant. The scope is C<sub>4</sub>-14 non-polymer fluorosurfactants.

It is important to remember that the burden of PFAS-related costs such as health-related and remediation costs is largely shouldered by governments and the citizens who pay taxes, while the pollution partly is caused by private operators. By compiling information on societal costs related to PFAS, it is hoped that this study will bring about more effective and cost-efficient management of the risks posed by PFAS.

## 2. The regulatory framework as baseline in relation to PFAS

For the purposes of this study, we are defining the “cost of inaction” as the costs to society from existing and future exposures to PFAS if no further measures to curb such exposures are taken. The term “further measures” could refer to additional policy measures as well as better enforcement and implementation of existing policies and regulations.<sup>16</sup> The case studies and other information collected for this study are intended to provide an overview of the baseline with respect to PFAS exposure.

The aim is dual: (1) to establish a framework for estimating costs for society due to negative impacts on human health and the environment related to PFAS exposure; and (2) to provide monetary values for the costs borne by society, by using costs derived from actual cases involving health impacts or where remedial measures were taken to address PFAS contamination. The overall intention is to highlight the economic case for taking effective and timely action to manage the risks of negative impacts from PFAS exposure.

Costs of inaction may refer to different things. One type of cost is related to staying within regulatory guidelines for drinking water (see the subsection below). For example, cases where drinking water supplies were contaminated have led to costs ranging from replacement of water supplies (bottled water, drilling of new wells) to removal of the PFAS contamination from the drinking water by further treatment (reverse osmosis, activated charcoal filters) before delivery to consumers.

Another type of cost is the health-related expenses incurred by people exposed to PFAS and suffering from negative health effects as a result. Cases where human populations have been exposed to PFAS over time have been linked to a number of adverse health effects, leading to greater health care costs, loss of production due to absence from work or lower productivity, and a lower quality of life.

Less tangible costs might be the loss of use of a natural resource such as groundwater or the loss of property value for homeowners in affected areas. The extreme persistence and mobility in the environment of PFAS is also a consideration, since PFAS contamination tends to continue to spread and costs of clean up through remediation of soil or water will increase if actions are delayed.

In recent years, other studies have aimed to estimate costs of inaction related to chemicals exposure. A 2013 UNEP study on costs of inaction on the sound management of chemicals.<sup>17</sup> looked at available literature concerning environmental and health costs

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<sup>16</sup> The OECD defines inaction as the lack of development of “no new policies beyond those which currently exist”. See OECD. (2008). Environmental Outlook to 2030. Chapter 18: “Chemicals”.

<sup>17</sup> UNEP (2013). Cost of inaction on the sound management of chemicals. Report Number: DTI/1551/GE.

linked to a wide range of chemical effects, including heavy metals (mercury, lead), outdoor pollutants (NO<sub>x</sub>, NO<sub>2</sub>, PM, SO<sub>x</sub>, SO<sub>2</sub>, VOCs), pharmaceuticals and pesticides. Based on data available it is estimated that accumulated health costs related to pesticide poisonings in Sub-Saharan Africa will reach around USD 97 billion by 2020.

The 2014 study for the Nordic Council on costs linked to effects of endocrine disrupting substances on male reproductive health is more focused. It reviewed the strength of the evidence regarding negative effects of chemicals considered endocrine disruptors and estimated numbers of incidences of negative effects as well as related costs to society.<sup>18</sup> It equated costs of illness with the economic value of reducing risks of exposure to endocrine disruptors. A theme in both studies is the lack of data concerning numbers of chemical exposures and related costs.

## 2.1 Guideline values for protection of health related to PFAS exposure

For the purpose of estimating costs of inaction, it is important to note when levels of contamination require remedial action. Among the tools used by regulatory authorities to control pollutants in environmental media such as groundwater and soil or in water or food for human consumption are limit or guideline values. Such values are important for determining when contamination is at levels that pose unacceptable risks to human health or the environment so that (1) action to remediate the resource is required; and (2) restriction of a certain use or substance is needed to prevent further contamination.

Guideline values for acceptable concentrations of PFAS in drinking water are currently in flux. Recent analyses of epidemiological evidence, including of immunotoxicological impairment at background levels of exposure to PFAS<sup>19</sup>, have led to several regulatory authorities issuing opinions suggesting recommended concentration levels be lower than levels set previously.

Most limit values or guidelines to date are for individual long-chain PFAS (PFOS, PFOA, PFHxS, due to their known toxicity and bioaccumulability, e.g., the 2015 World Health Organization recommendation of 0.4 µg/l (400 ng/l) for PFOS and 4 µg/l (400 ng/l) for PFOA in drinking water.

More recent guidelines recognise the potential for harmful impacts from groupings of PFAS, including the short-chain PFAS. This is reflected in the group parameter for PFAS proposed in February 2018 for revision of Council Directive 98/83/EC on the quality of water intended for human consumption (Drinking Water Directive).<sup>20</sup> The Commission proposal suggests regulating the whole class of PFAS, i.e., values of

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<sup>18</sup> Nordic Council of Ministers (2014). The Cost of Inaction – a socioeconomic analysis of costs linked to effects of endocrine disrupting substances on male reproductive health. TemaNord 2014:557.

<sup>19</sup> Grandjean P (2018). Delayed discovery, dissemination, and decisions on intervention in environmental health: a case study on immunotoxicity of perfluorinated alkylate substances. *Environmental Health* (2018) 17:62.

<sup>20</sup> Proposal for a Directive of the European Parliament and of the Council on the quality of water intended for human consumption COM/2017/0753 final - 2017/0332 (COD).

0.1 µg/l (100 ng/l) for individual PFAS and 0.5 µg/l (500 ng/l) for PFAS as a group.<sup>21</sup> This is an approach already used for pesticides in drinking water.

The limit values for PFAS in drinking water set by Sweden and Denmark are also parameters for groups of PFAS. The Swedish National Food Agency has set a group limit value for PFAS at 90 ng/l.<sup>22</sup> This also serves as an action level. If the sum of 11 PFAS in drinking water exceeds that level, action is to be taken as soon as possible to reduce the PFAS to concentrations as low as practically possible below that action level. Denmark applies a limit value of 100 ng/l for the sum of 12 PFAS in drinking water (the parameter for PFAS in soil is 0.4 mg/kg TS).<sup>23</sup>

Germany's Federal Umweltbundesamt (UBA) first published recommended values in 2006 based on a request by the Hochsauerla Valley (see Case Study 3.5.2.2). Since then, new data has led to further revisions and the nd Public Health Department prompted by the PFAS contamination incident in Moehne current UBA guidelines set the lifelong precautionary value at 100 ng/l per se for PFOA and PFOS and 300 when both are present.<sup>24</sup>

In December 2018, European Food Safety Authority (EFSA) published a scientific opinion on health risks related to PFOS and PFOA in the food chain.<sup>25</sup> A previous opinion issued in 2008 set values for tolerable daily intake (TDI) of PFOS at 150 ng/kg bw/day and for PFOA at 1500 ng/kg bw/day. This has been calculated as equivalent to limit values of 70 ng/l for PFOS and 700 ng/l for PFOA.

The most recent EFSA opinion sets tolerable daily intake (TDI) for PFOS in food at 13 ng/kg bw/week and for PFOA at 6 ng/kg bw/week.<sup>26</sup> This has been calculated as equivalent to limit values of 6.5 ng/l for PFOS and 3 ng/l for PFOA<sup>27</sup> which enables the values to be compared to those set for drinking water.

In the USA, guideline values are also undergoing revision. In 2016 the US Environmental Protection Agency issued a lifetime drinking water health advisory that set limit values for PFOA at 70 ng/l and for PFOS also at 70 ng/l.<sup>28</sup> The advisory notes that when these two chemicals co-occur in a drinking water source, a conservative and health-protective approach would be to set the sum of the concentrations ([PFOA] + [PFOS]) at 70 ng/l.

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<sup>21</sup> The Commission's explanatory document points out that these values exceed those referred to in Sweden or the USA and therefore compliance should be feasible.

<sup>22</sup> Swedish National Food Agency (2017). Riskhantering - PFASs i dricksvatten och fisk.

<sup>23</sup> Danish Environmental Protection Agency (2015). Perfluorerede alkylsyreforbindelser (PFAS-forbindelser) incl. PFOA, PFOS og PFOSA.

<sup>24</sup> German Environment Agency (2017). Fortschreibung der vorläufigen Bewertung von per- und polyfluorierten Chemikalien (PFC) im Trinkwasser and German Environment Agency (2011). Grenzwerte, Leitwerte, Orientierungswerte, Maßnahmenwerte Aktuelle Definitionen und Höchstwerte.

<sup>25</sup> EFSA Panel on Contaminants in the Food Chain, Knutsen HK *et al.*, 2018. Scientific opinion on the risk to human health related to the presence of perfluorooctane sulfonic acid and perfluorooctanoic acid in food. EFSA Journal 16(12):5194. DOI: <https://doi.org/10.2903/j.efsa.2018.5194>

<sup>26</sup> From the 2018 EFSA draft abstract on human epidemiological studies. The panel noted that *for both compounds exposure of a considerable proportion of the population exceeds the proposed TWIs.*

<sup>27</sup> Grandjean P (2018). Delayed discovery, dissemination, and decisions on intervention in environmental health: a case study on immunotoxicity of perfluorinated alkylate substances. Environmental Health (2018) 17:62.

<sup>28</sup> Environment Protection Agency (2016). Fact Sheet PFOA& PFOS Drinking Water Health Advisories.

In 2018, the US Agency for Toxic Substances & Disease Register (ATSDR) issued a draft toxicological profile for perfluoroalkyls.<sup>29</sup> The draft profile suggested provisional minimal risk levels (MRLs) of 7 ng/l for PFOS and 11 ng/l for PFOA – parameters that are seven to ten times lower than the lifetime advisory levels set by USEPA.

**Table 5: Regulatory parameters for PFAS in drinking water (DW) (ng/l)**

Standard	PFOS	PFOA	PFNA	PFAS (single)	PFAS (group)
WHO guidelines for drinking water (2015)	40	400			
Sweden NFA action level (sum of 11 PFAS, 2014)					90
Denmark (sum of 12 PFAS, 2015)					100 <sup>1</sup>
Germany (2017)	100	100	60		300 <sup>2</sup> ; 7,000 <sup>3</sup>
EU proposed level single PFAS in DW (2018)				100	
EU proposed level total PFAS in DW (2018)					500
EFSA TDI in food (2008)	70	700			
Draft EFSA TDI in food (2018) <sup>4</sup>	6.5	3			
US EPA lifetime DW health advisory (2016)	70	70			70
US ATSDR draft finding (2018)	7	11			
State of New Jersey (2018)	13	14	13 (binding)		70
State of Minnesota (2017)	27	35			

Note: 1) Sum of 12 PFAS.  
2) PFOS+PFOA.  
3) PFAS except PFOS and PFOA.

Source: Estimated. Grandjean P (2018). Delayed discovery, dissemination, and decisions on intervention in environmental health: a case study on immunotoxicity of perfluorinated alkylate substances. *Environmental Health* (2018) 17:62.

Several individual US states are setting parameters for PFAS in drinking water at even more stringent levels. In July 2018, the US state of New Jersey adopted a maximum contaminant level (MCL) for perfluorononanoic acid (PFNA) of 0.013 µg/l (13 ng/l).<sup>30</sup> It is considering the recommendation of the New Jersey Drinking Water Quality Institute to set an MCL for PFOS at 0.014 µg/l (14 ng/l). Likewise, the state of Minnesota decided in 2017 to update their health values basing them on the vulnerability of foetuses and infants who are exposed via their mothers, rendering the values significantly lower than those set by the federal USEPA (see Table 5).<sup>31</sup>

The lowering of mandatory and advisory levels for PFAS in drinking water indicate a growing awareness that exposure to PFAS even at low levels can have negative impacts on human health. In particular, studies have found impaired immunological responses to vaccines at levels of exposure as low as 1 ng/l in serum – levels that are exceeded in most humans.<sup>32</sup>

<sup>29</sup> Agency for Toxic Substances and Disease Registry (2018). Draft toxicological profile for perfluoroalkyls.

<sup>30</sup> New Jersey Register, Adopted Amendments: N.J.A.C. 7:9E-2.1; 7:10-5.2, 5.3, and 12.30; and 7:18-6.4.

<sup>31</sup> Minnesota Department of Health Perfluoroalkyl Substances (PFAS). Accessed 09.10. 2018.

<sup>32</sup> Grandjean P (2018). Delayed discovery, dissemination, and decisions on intervention in environmental health: a case study on immunotoxicity of perfluorinated alkylate substances. *Environmental Health* (2018) 17:62.

As the proposal for revision of the EU Drinking Water Directive notes, these substances do not belong in the environment. The proposal points out that Directive 2008/105/EC on environmental quality standards in the field of water policy sets a limit value of 0.65 ng/l for PFOS and suggests a precautionary approach as the way forward.

Given that these regulatory parameters are currently a moving target, this study proposes to use Sweden's action level of 90 ng/l as the point of comparison in considering when a resource is considered contaminated by PFAS, such that remedial action should be taken.

## 2.2 Other regulatory actions underway

Other regulatory efforts underway are aimed at controlling PFAS on the market, because of evidence of their negative impacts. Within the European Economic Area (EEA), member countries are subject to the provisions of the EU REACH Regulation, as well as to the regulation implementing the Stockholm Convention on persistent organic pollutants.

PFOS has been restricted under the Stockholm Convention since 2009. During the fall of 2014, Norway and Germany joined in submitting a proposal for the EU to restrict PFOA, its salts and related substances.<sup>33</sup> This led to the adoption of Commission Regulation (EU) 2017/1000 of 13 June 2017 amending Annex XVII to REACH, as regards perfluorooctanoic acid (PFOA), its salts and PFOA-related substances.

In March 2017, Sweden and Germany proposed to consider PFHxS a substance of very high concern.<sup>34</sup> This was adopted by the European Chemicals Agency (ECHA) later the same year, and the substance is now on the Candidate List. Norway has registered an intention to submit a restriction proposal for PFHxS under REACH.

Sweden and Germany also jointly proposed in 2017 to restrict the manufacturing and placing on the market of six PFAS (PFNA, PFDA, PFUnDA, PFDoDA, PFTTrDA and PFTTeDA), as well as their salts and precursors.<sup>35</sup> The aim in restricting these long-chain (C9-C14) PFAS is to prevent industry from switching to them once the restriction of PFOA goes into effect in 2020. Both the RAC (Risk Assessment Committee) and the SEAC (Committee for Socio-economic Analysis) have agreed to the restriction proposal; public consultation on the SEAC opinion closed on 19 November 2018.

The Stockholm Convention on persistent organic pollutants (POPs) continues to consider measures related to PFAS additional to the 2009 listing of PFOA for global restriction (Annex B). In September 2018, the POPs Review Committee agreed to recommend to the Parties to the Convention that PFOA be phased out, because its PBT qualities, the occurrence of PFOA in environmental compartments, and the evidence of long-range environmental transport supported the conclusion that it is likely to lead to significant adverse effects such that global action is warranted. It also evaluated the

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<sup>33</sup> ECHA (2014). Germany and Norway propose a restriction on Perfluorooctanoic acid (PFOA), its salts and PFOA related substances.

<sup>34</sup> ECHA (2017a). Inclusion of substances of very high concern in the Candidate List for eventual inclusion in Annex XIV.

<sup>35</sup> ECHA (2017b). Public consultation. Germany, in collaboration with Sweden, proposed a restriction on C9-C14 perfluorocarboxylic acids (PFCAS), their salts and related substances (precursors).

exempted uses of PFOS based on the availability of alternatives and recommended most of them for removal or to be made time-restricted. The POPs Review Committee also adopted the risk profile for PFHxS, thereby moving it to the next stage of a risk management evaluation (part of the process for considering whether to list a chemical in the Convention).<sup>36</sup> The next meeting of the Parties takes place in April 2019 when the decisions will be taken on the table concerning the listing of PFOA for global phase-out and for removing exemptions for uses of PFOS.

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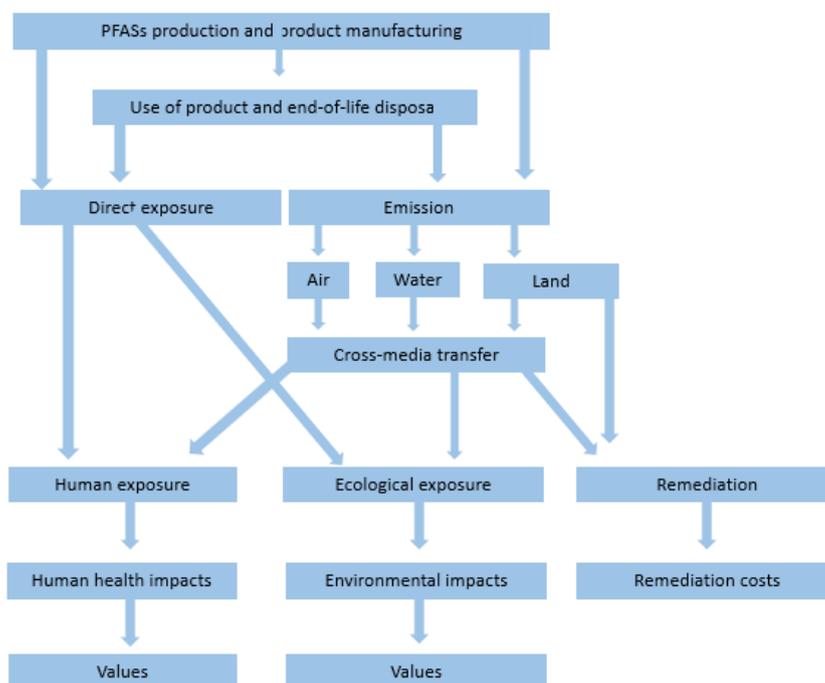
<sup>36</sup> Stockholm Convention (Website) Report of the POPs Review Committee at the work of its fourteenth meeting, UNEP/POPS/POPRC.14/6.

### 3. Methodology to assess environment and health-related costs

This chapter describes the methodology for building an integrated socioeconomic model to assess the environmental and health-related costs of PFAS exposure in European countries. No such methodology had been developed at the time this study was conducted. In a stocktake of socioeconomic assessments for PFOA and its salts carried out for the OECD<sup>37</sup>, the need for a method to draw together information on the long-term environmental and health costs related to PFAS exposure was expressed.

The impact pathway shown in Figure 2 provides an overall framework for the socio-economic analysis.

Figure 2: Generic impact pathways for linking substances to possible impacts



<sup>37</sup> Gabbert, S. (2018). Economic assessments and valuations of environmental and health impacts caused by Perfluorooctanoic acid (PFOA) and its salts. OECD Environment Working Paper No 128.

The figure links production and use of PFAS to impacts and their economic valuation. It provides a template for assessment of each source, enabling the analyst to consider which impacts are relevant in a case.

Following on from Figure 2, data needs for the assessment are identified in Table 6, which presents an overview of the socioeconomic assessment method that guided the analysis. The method defines six stages for the assessment of both health and environmental impacts. For each stage, it defines a set of input parameters, data sources and key assumptions by stage of the assessment process. The evidence available on the extent of PFAS exposure and its impact is often restricted to a case of contamination in a specific geographic area. One of the key assumptions noted for several stages is therefore the transferability of scientific findings from one specific context to another.

**Table 6: Assessing the socio-economic impacts of PFAS exposure**

Stage	Input parameters	Data sources	Key assumptions
Defining sources of PFAS exposure	Uses of PFAS, e.g., production, product manufacture, product use	Scientific and grey literature, on-line research	Future applications
	Number of activities involving PFAS (by use)	Case studies	Future use, alternatives
	Identification of impact pathways (by use)	Case studies	Assumptions of future conformance
Identification of impacts	Health impacts Listing of impacts linked to PFAS, e.g. cancers Environmental impacts Contamination of resources such as drinking water	Case studies and scientific literature	Causality for PFAS in general and then for specific PFAS
Quantification of impacts	For each effect identified above: Size of population or receiving body at risk Prevalence of disease Response function	National (etc.) statistics, scientific literature, including reviews	Transferability
Valuation	Health Unit values e.g. EUR/death	Documents submitted to ECHA, OECD, etc. Further review of the literature	Transferability
	Environment Willingness to pay to avoid loss of ecosystem services Cost of damage to commercial fisheries, agriculture, etc. Cost of environmental remediation	Documents submitted to ECHA, OECD, etc. (eg. D4/D5 dossier) Further review of the literature. Market prices Published case study materials	Transferability
	Value transfer	Factors including exchange rates, size of population affected and income levels to improve applicability of values to the target population	Valuation literature, exchange rate databases
	Discount rates	Standard European Commission practice (constant 4%) + alternatives of 0% and 2%	Validity of constant rates over extended timescales
Aggregation	Contextual data permitting quantification of effects beyond the case study materials that are available	National (etc.) statistics, scientific literature, including reviews	Transferability

The specificities of the methodologies for assessing health impacts can be found in Section 5.1 while those for the environmental impacts can be found in Section 5.2.

The lack of systematic and standardised evidence in addition to the underlying uncertainties with regards to the extent of impact and their consequences presented challenges for the process of developing quantitative estimates. The robustness of each quantitative finding is explored through sensitivity analysis. The sensitivity analysis allowed for the construction of lower and upper bounds for each quantitative estimate to reflect the underlying uncertainties. Other costs that could not be quantified, but for which strong evidence was identified, are assessed in a qualitative manner.

### 3.1 Health-related costs

This section describes the specific methodology for assessing the health-related costs of PFAS exposure. A growing body of scientific literature suggests that PFAS exposure can lead to a wide range of adverse health impacts at different levels of exposure. To date, a monetisation of these health-related costs has not been developed due to the lack of a global consensus on the specific health impacts linked to PFAS exposure and a complete understanding of the level of exposure needed to trigger a health impact. This cost of inaction assessment therefore presents a first attempt to monetise the impacts for several of the identified health endpoints of PFAS exposure.

The methodology takes findings from various epidemiological studies showing relative risks due to exposure. It considers “what-if” scenarios, where scenarios assume the transferability of epidemiological studies from one context to another in some cases, and the subsequent impacts. It then extrapolates those findings to the “exposed” population in the Nordic countries and the EU. The design of these scenarios attempts to reflect the underlying uncertainties with respect to the level of exposure and the epidemiological evidence. The methodology for assessing the health-related costs follows four basic steps: (1) identification of endpoints, (2) responses to levels of exposure, (3) quantifying impacts and (4) aggregation. Each step is described below.

#### 3.1.1 Identification of endpoints

In assessing the potential health impacts of exposure to PFAS, toxicologists and epidemiologists need information on both human health endpoints (which are defined as conditions or diseases that reflect poorer health and an increased risk of mortality) and substance exposure. Data on human health endpoints may be obtained from for example from public health records or from surveys of exposed individuals. The level of human exposure to PFAS can be inferred from e.g. data collected from monitoring PFAS contamination in drinking water or other local sources, or it can be investigated more closely through the analysis of blood samples.

Identifying health impacts related to exposure to PFAS is challenging for several reasons. Health impacts are typically identified through studies that compare a relatively “exposed” group and a relatively “unexposed group” while controlling for other factors

related to the health endpoint. From a causal perspective the ideal setting would be a randomized control study (experiment), in which individuals would be assigned at random into treatment and control groups (exposed/unexposed groups). This would ensure random assignment of background characteristics in each group, i.e the background characteristics in each group would on average be the same. However, in settings with environmental pollution or toxic substances, this is neither ethical nor economically feasible.

A potential solution to this methodological problem is to look for a “natural experiment” where the exposed group has similar background characteristics as the unexposed group, for example two neighborhoods in close geographic proximity where one is more exposed than the other. However, given the persistence of PFAS and their ability to travel long distances, it is unlikely that a group in close proximity to the contaminated area would not have an elevated level of exposure. In fact, considering that PFAS have been found in some of the most remote places in the world, it may not be possible to find a truly unexposed group among humans.

To further complicate inference, contamination often consists of more than one PFAS compound, making it difficult to attribute the exposure to a single compound. The lack of regular biomonitoring of PFAS in humans (through collection and analysis of blood samples) in many countries also presents a severe limitation in the data available on the health impacts of PFAS at different levels of exposure and in different contexts. In addition, the sample sizes for most epidemiological investigations are quite small, which limits the extent to which health endpoints can be identified with a reasonable level of confidence. This is especially challenging for health endpoints that are relatively uncommon in the general population such as testicular and kidney cancer. In conclusion, the above mentioned reasons may explain why some epidemiological studies find statistically significant effects while others do not.

The first part of Annex 2 presents an overview of epidemiological studies that have linked PFAS exposure with a range of different health endpoints. The sample size, the population studied, and the time period are indicated for each study.

Several regulatory bodies and expert panels around the globe have carried out reviews of the scientific evidence in order to reach conclusions about the relevant health endpoints of human exposure to PFAS. These reviews usually seek a certain level of consensus across different scientific studies in order to conclude that PFAS have an adverse impact on that health endpoint. In drawing conclusions about specific health endpoints, these reviews utilise a “strength of the evidence” approach. The scope of the evidence considered as well as the evaluation of the strength of the evidence varies, however, across the available reviews. This leads to different conclusions in terms of the recognised health endpoints of PFAS (see Table 7).

For example, an Expert Health Panel convened by the Australian Department of Health presented a review of recent literature reviews of the potential health impacts of PFAS in May 2018.<sup>38</sup> The study did not find conclusive evidence for any of the health

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<sup>38</sup> Australia Government Department of Health (2018). Expert Health Panel for Per- and Poly-Fluoroalkyl Substances (PFAS).

endpoints identified in other expert reviews. However, the Panel came to the conclusion that because current evidence is primarily based on weak study designs and is inconsistent in many respects, some degree of important health effects for individuals exposed to PFAS could not be ruled out based on the existing evidence.

The provisional report from the European Food Safety Authority (EFSA) concludes that PFAS exposure can lead to metabolic disease, immunotoxicity, and developmental toxicity, but finds the evidence with respect to cancer and endocrine disruption not sufficiently robust. The draft toxicological profile from the US Agency for Toxic Substances and Disease Registry (ATSDR) on the other hand suggests that the evidence demonstrates a relationship with several health endpoints such as asthma, pregnancy-induced hypertension and an increased risk of thyroid disease. This assessment applies not only for PFOA and PFOS, but also several other PFAS compounds such as PFHxS and PFDeA.

**Table 7: Reviews of health endpoints linked to PFAS exposure**

Category	Health endpoint	EFSA	ATSDR	C8 Health Project	US EPA	OECD
Metabolic disease	Liver damage	✓ PFOA)	✓ (PFOA, PFOS, PFHxS)		✓ (PFOA)	✓
	Ulcerative colitis			✓		
	Increased serum cholesterol levels	✓ (PFOS, PFOA)	✓ (PFOA, PFOS, PFNA, PFDeA)	✓	✓ (PFOA, PFOS)	
Immuno- toxicity	Decreased immune response (e.g. antibody response to vaccines)	✓ (PFOS)	✓ (PFOA, PFOS, PFHxS, PFDeA)		✓ (PFOA)	✓
	Increased risk of asthma diagnosis		✓(PFOA)			
Endocrine disruption	Increased risk of thyroid disease (elevated hormones)		✓ (PFOA, PFOS)	✓	✓ (PFOA)	✓
	Elevated sex hormones					✓
	Decreased fertility		✓ (PFOA, PFOS)		✓ (PFOS)	
	Pregnancy-induced hypertension/pre-eclampsia		✓ (PFOA, PFOS)	✓	✓ (PFOA)	
	Delayed menstruation and earlier menopause				✓ (PFOA)	
Developmental outcomes	Lower birth weight	✓ (PFOS, PFOA)	✓(PFOA, PFOS)		✓ (PFOS)	✓
Carcinogenicity	Testicular and kidney cancer			✓	✓ (PFOA)	✓

Another key assessment was made by the C8 Health Project, which was established as part of a class action legal settlement made by a chemical manufacturer in West Virginia.<sup>39</sup> The C8 Health Project was led by a Science Panel of three epidemiologists who carried out a series of scientific studies using biomonitoring data gathered from the site.

<sup>39</sup> The chemical manufacturer was Dupont. In 2017, Dupont merged with the Dow Chemical Company. Dupont is now a subsidiary of the Dow Chemical Company.

The Science Panel concluded that the exposure had a probable link with seven health conditions and diseases: High cholesterol (hypercholesteremia), ulcerative colitis, thyroid function, testicular cancer, kidney cancer, preeclampsia, as well as elevated blood pressure during pregnancy (pregnancy-induced hypertension). These endpoints roughly reflect the health impacts identified in a 2018 review conducted for the OECD of epidemiological research studies.<sup>40</sup> Lastly, the US EPA issued two reports in 2016 for the health effects for PFOA and PFOS.<sup>41</sup> It recognised almost all of the health endpoints identified by other reviews and panels.

Some epidemiological studies were considered as conclusive evidence in support of a certain health endpoint by one assessment, but not as conclusive by another. For example, the EFSA opinion prominently cites a study conducted as part of the C8 Health Project<sup>42</sup> as providing strong evidence that low birthweight is a relevant health endpoint.<sup>43</sup> Yet the C8 Science Panel did not find the evidence sufficiently strong for low birthweight as an endpoint. The lack of consistency across these assessments and the differential weighting of specific epidemiological studies reflects the challenges at present to reach a global consensus on the health endpoints of PFAS contamination.

As per Table 7, the body of scientific evidence and research has grown over time, collectively suggesting that PFAS do have adverse health impacts on humans. A July 2018 study<sup>44</sup> notes that

“[a]ccumulated evidence from studies of experimental animal models and of humans from highly exposed populations supports the conclusion that PFOA and PFOS, along with other carboxylate and sulfonate PFAS, are multi-system toxicants. In other words, exposure to PFAS is associated with toxicological findings in many types of tissues and systems.”

The International Agency for Research on Cancer (IARC) provides additional insights about the carcinogenicity endpoint. The agency published a monograph in June 2018 concluding that there is limited evidence in humans for the carcinogenicity of PFOA. However, a positive association between PFAS exposure and cancers of the testis and the kidney was observed, rendering the overall evaluation that PFOA is possibly carcinogenic to humans.<sup>45</sup>

This cost of inaction assessment of PFAS is built on the assumption that exposure to PFAS is linked to the health endpoints indicated in Table 7. In addition, the assessment assumes that different short- and long-chain PFAS compounds will lead to similar adverse health impacts.

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<sup>40</sup> Gabbert S (2018). Economic assessments and valuations of environmental and health impacts caused by Perfluorooctanoic acid (PFOA) and its salts. OECD Environment Working Paper No 128.

<sup>41</sup> EPA (2016). Health effects support document for perfluorooctanoic acid (PFOA). Document no: EPA822R16003 and Health effects support document for perfluorooctane sulphate (PFOS). Document No: EPA822R16002.

<sup>42</sup> Stein C R *et al.* (2009). Serum levels of perfluorooctanoic acid and perfluorooctane sulfonate and pregnancy outcome. *American journal of epidemiology*, 170(7), pp.837–846.

<sup>43</sup> The main reason for its prominence is its large sample size. The authors did not consider other studies investigating this endpoint to be sufficiently powered.

<sup>44</sup> Hopkins *et al.* (2018). Recently Detected Drinking Water Contaminants: GenX and Other Per- and Polyfluoroalkyl Ether Acids. *American Water Works Association Journal* 110:7 p.13–28.

<sup>45</sup> IARC (2018). Perfluorooctanoic acid (PFOA).

### 3.1.2 Responses to levels of exposure

In the absence of data showing clear dose-response relationships, this study has instead considered health impacts found by epidemiological studies of populations at three levels of exposure: (1) background (low) PFAS exposure levels; (2) elevated (medium) PFAS exposure levels and (3) occupational (high) exposure.

Individuals with a higher level of exposure to PFAS can be expected to have a higher concentration of the contaminant in their blood, resulting in elevated health risks. The Expert Health Panel from Australia noted that individuals in highly exposed communities typically have a blood serum concentration about ten times higher than the general population while workers can have a blood serum concentration one thousand times higher than the general population.<sup>46</sup> In other words, it is assumed that the blood serum concentration serves as a rough proxy of PFAS exposure.

Blood samples from population studies such as the National Health and Nutrition Examination Survey (NHANES) in the United States were assumed to provide information on background (low) PFAS exposure. Medium and high PFAS exposure levels were drawn from epidemiological studies based on populations affected by known incidents of PFAS contamination. Communities in close proximity to PFAS production or with PFAS-contaminated drinking water were considered to have been exposed at medium levels while occupationally exposed individuals, e.g., at chemical production plants, were considered to have high levels of exposure.

Some adverse health impacts appear to materialise at high PFAS exposure levels while for other conditions background exposure generates an elevated risk of disease. Carcinogenicity is most clearly linked with a high level of exposure to PFAS. One study<sup>47</sup> found the risk of kidney cancer among residents in close proximity to PFAS contamination was elevated only among those with an above-average level of exposure. Factors that may lead some residents in the same community to have a higher level of exposure than others include the person's age, the number of years lived in the community, and the level of contamination in his or her household's drinking water. The IARC (2018) assessment also concludes that the evidence regarding the elevated risk of kidney cancer is most convincing for the case of occupational exposure, the highest category of exposure.<sup>48</sup>

At the same time, it is not possible to rule out that other residents in the same community with a below-average exposure to PFAS do not have an elevated risk of kidney cancer. The lack of statistical significance may be due to the low risk of kidney cancer in the general population.<sup>49</sup>

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<sup>46</sup> Australia Government Department of Health (2018). Expert Health Panel for Per- and Poly-Fluoroalkyl Substances (PFAS).

<sup>47</sup> Vieira V M *et al.* (2013). Perfluorooctanoic acid exposure and cancer outcomes in a contaminated community: a geographic analysis. *Environmental health perspectives*, 121(3).

<sup>48</sup> IARC (2018). Perfluorooctanoic acid (PFOA).

<sup>49</sup> National Cancer Institute [NCI] Surveillance, Epidemiology, and End Results data for 2005–2011. The kidney cancer endpoint is also difficult to assess as the survival rate is high at 73%. Testicular cancer, which is another possible endpoint of PFAS exposure, has an even higher survival rate of 95%. Studies with a large sample over a long time frame have a greater likelihood of detecting a differential risk of developing these types of cancer.

Metabolic disease is linked with all levels of PFAS exposure with less severe endpoints such as elevated cholesterol and ulceritis associated with background (low) exposure and more critical endpoints such as liver damage associated with elevated (medium) exposure. The link between the level of PFAS exposure and health endpoints appears to be similar for endocrine disruption and developmental toxicity. Studies suggest that background (low) levels of PFAS contamination can lead to immunotoxicity, especially for infants and children.

### 3.1.3 *Quantifying impacts*

A quantitative valuation or monetisation was then undertaken for a selection of the identified health endpoints for which there is a reasonable level of global consensus. Given the limited epidemiological evidence from the specific contexts, the assessment considers “what-if” scenarios. These scenarios assume the transferability of quantitative findings from epidemiological studies from one context to another in some cases, and the subsequent impacts.

The quantitative findings from epidemiological studies are typically in the form of a standardised mortality ratio, an odds-ratio or a relative risk. These statistical terms reflect the elevated risk in an “exposed” population compared with a “less exposed” population after controlling for other factors.<sup>50</sup> The assessment selected risks that were estimated with a high level of confidence. In statistical terms, this implies that the risk was estimated with a margin of error of 5% or less.

The what-if scenarios constructed define linkages between PFAS exposure, the health endpoint and mortality related to the endpoint. Additional studies that investigated the relationship between the health endpoint in question and mortality were also reviewed to characterise the last linkage in the chain of the what-if scenario. The estimated number of health impacts such as deaths through the what-if scenario was then monetised using the “value-of-statistical-life” approach.

The quantitative analysis applies the lower bound of the range recommended by ECHA of EUR 3.5 to EUR 5 million per life lost.<sup>51</sup> The ECHA value of life estimates are commonly used by regulatory agencies in Europe. The lower bound value is also comparable to the value of a statistical life in the EU reported by the OECD, which was EUR 3 million in 2012.<sup>52</sup> When adjusted for inflation, the OECD value approaches the minimum ECHA value.

The monetised figures presented in this study should be understood as the minimum health-related costs of inaction in that only a few health endpoints and a few impacts of these endpoints could be investigated. More data on exposure-response relationships is

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<sup>50</sup> Epidemiological models may control for a wide range of individual and environmental factors such as age, gender, educational background and occupation.

<sup>51</sup> ECHA (2016). Valuing selected health impacts of chemicals: Summary of the results and a critical review of the ECHA study.

<sup>52</sup> OECD (2012). Mortality risk valuation in environment, health and transport policies.

needed for estimating the impacts with respect to other endpoints. The monetised figures can provide an indication of the potential costs of inaction, and where more research is needed to develop more robust what-if scenarios and estimations.

#### 3.1.4 Aggregation

Estimates of impacts or costs were then aggregated across the Nordic countries and the EEA when possible. A major challenge in carrying out the aggregations was the determination of the size of the relevant exposed population. For example, the scenarios for occupational (high) and elevated (medium) levels of exposure required assumptions regarding the number of chemical plants or other sources of PFAS contamination and the average number of persons exposed in each location.

To the extent possible, information to use as a basis for these assumptions were gathered through the case studies. A range of plausible values was considered in the absence of conclusive information. The scenarios for background (low) exposure assume that all individuals living in the Nordic countries or the EEA are affected. The estimations for background (low) exposure could then draw on country-level population statistics available from Eurostat.

The annual number of deaths or cases of a health conditions were generated for the exposed population based on the level of exposure and the epidemiological evidence. The additional level of mortality and disease can therefore be understood as the annual costs due to PFAS exposure for the relevant geographic area – the Nordic countries or the EEA – under the specific scenario. These costs would not be incurred if the PFAS exposure, as defined by the scenario, did not occur. Therefore these estimated costs can be understood as potential costs of inaction.

The analysis generated a point estimate, a lower bound and an upper bound for each scenario. The generation of these three different values stem from the findings of the epidemiological studies, which provided the parameter relating to the level of exposure to PFAS and the elevated health risk. The point estimate was calculated using the main finding from the epidemiological study. The 95% confidence interval for the finding from the epidemiological study was used to generate the lower and upper bound estimates.

The findings from the estimations can be found in Section 5.1. The calculations underlying these estimations including the parameter values can be found in Annex 2.

## 3.2 Environment-related costs

Two distinct types of environment-related cost are associated with the present research:

- environmental remediation; and
- loss of ecosystem services.

### 3.2.1 Approaches to environmental remediation

The most technically and economically efficient techniques for reducing contamination of the environment with PFAS arise at manufacture and application prior to sale to end-users, and are as follows:

- using alternatives;
- improved containment at industrial sites manufacturing PFAS;
- increased use efficiency of PFAS materials, for example by re-cycling unused solution at manufacturing sites. Recycling of most applications post-consumer is generally not feasible. A possible exception may be AFFFs that have gone past their use-by date; and
- improved containment at industrial sites using PFAS, for example using controlled application processes and controlled disposal of residues.

Using these techniques, PFAS will either not be present at all, in cases where non-PFAS alternatives are used<sup>53</sup>, or where they are used, the volume of material requiring treatment will be limited. However, these measures address only part of the problem: except for the use of alternatives, they are all associated with some level of discharge and they cannot influence contamination associated with goods used outside manufacturing or processing sites. In some cases, AFFFs being a notable example, dispersion to the environment is immediate and total, upon use.

Costing the first set of options set out above is beyond the scope of this report. Costs of improving containment and use efficiency<sup>54</sup> will be internalised by producers and business consumers. In many cases any improvement and associated costs will reflect legislative demands and the permit conditions that facilities are required to operate to. There will be some cost to government, for example through the use of PFAS alternatives at military airbases. Analysis of these costs would require a counterfactual scenario to be developed where alternatives to PFAS were defined, along with differences in likely permit conditions, extending the current assessment to a full cost-benefit analysis.

Environmental remediation may deal with:

- contaminated soils
- contaminated groundwater
- contaminated surface water
- targeted collection of goods containing PFAS at end-of-life to reduce the volume of material that needs to be treated.

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<sup>53</sup> Some "alternatives" are not impact-free. The BREF document on the tanning of hides and skins notes a trend to using shorter chain (C<sub>4</sub> or C<sub>6</sub>, rather than C<sub>8</sub> perfluoro compounds). The shorter chain fluorocarbon resins are described (as of 2013) as being more favourably assessed toxicologically but are as persistent in the environment as the longer chain PFAS.

<sup>54</sup> Increased use efficiency will lead to at least some payback through reduced demand for new material.

Targeted collection of goods containing PFAS at end-of-life is not a viable option in most cases. Some applications, such as the use of aqueous film-forming foams (AFFFs) lead to direct contamination of the environment. The range of other applications has become so extensive that separation of goods, for example at waste processing sites, would be impractical, as is evident from the problems faced across Europe with respect to the efficient recycling of another widely dispersed material, plastic. Even if it were practicable to some extent, the option would still leave some level of contamination present as collection processes would never capture all contaminated material and emissions will arise during the use phase.

Environmental remediation of PFAS contamination is not straightforward. Concawe<sup>55</sup>, an environmental research and advisory body for the oil industry, notes that many remediation techniques used for other contaminants are ineffective for PFAS because of their low volatility (preventing the use of gas stripping) and their resistance to biological degradation. With this in mind, the main techniques for removing PFAS from the environment involve:

- removal of soils; and
- groundwater extraction and PFAS adsorption onto activated carbon or resins.

Soil removal is a reasonably straightforward process, though it can clearly create a large quantity of soil that needs either further treatment or storage. Storage of PFAS at authorised landfills may lead to leaching into surrounding areas because standard leachate treatment plants are not able to effectively treat these substances.<sup>56</sup> The use of landfill would also place a burden on space at landfills, particularly if contaminated material is required to be stored at the limited number of sites that are licensed to deal with hazardous wastes.

One alternative to storage at landfill is to destroy PFAS through incineration, though this itself is not straightforward (or inexpensive) as it requires use of very high temperatures. Complete destruction of PFOS requires a temperature of 1,000 to 1,200 °C.<sup>57</sup> Associated costs are high because of the significant energy inputs that are required and the likelihood that soil (etc.) volumes will be large. Modern municipal and hazardous waste incinerators can reach high temperatures, e.g. 800 °C or more, and at such temperatures the PFAS on a treated consumer product may break down. The use of lower temperatures can lead to the generation of hazardous by-products.<sup>58</sup>

The Concawe report also discusses stabilisation of PFAS within soils using additives such as activated carbon, and solidification of soils using concrete mixes. Both have been shown to greatly reduce the potential for leaching. However, neither approach provides

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<sup>55</sup> Concawe (2016). Environmental fate and effects of poly- and perfluoroalkyl substances (PFAS). Report no: 8/16.

<sup>56</sup> Oliaei F *et al.* (2013). PFOS and PFC releases and associated pollution from a production plant in Minnesota (USA). *Environmental Science Pollution Research*. 20: 1977–1992.

<sup>57</sup> Schultz M *et al.* (2003). Fluorinated alkyl surfactants. *Environmental Engineering Science* 20(5) 487–501. Yamada T *et al.* (2005). Thermal degradation of fluorotelomer treated articles and related materials. *Chemosphere* 61, 974–984.

<sup>58</sup> Yamada T and PH Taylor (2003). Final Report: Laboratory scale thermal degradation of perfluorooctanyl sulfonate and related precursors. Final Report for 3 M Company.

a permanent solution to the problem of PFAS contamination. These approaches could be seen as creating a potentially large number of hazwaste landfills across Europe.

A further option is soil washing, moving PFAS to the aqueous phase where it can be filtered and retrieved. Concawe reports trials showing a reduction in concentrations *below target levels* after two washing cycles.<sup>59</sup> No information was available on the quantities of sludge or filtrate generated, materials that would need further storage or treatment. Concentrating the PFAS up would have the benefit of reducing the quantity of contaminated material, which would in turn reduce demand for storage or incineration.

The most commonly applied treatment for contaminated groundwater is extraction and use of granular activated carbon (GAC). The efficiency of extraction of PFOS is in the order of 90%, though efficiency for other PFAS (e.g. PFOA) can be much lower especially for short-chained PFAS like PFBA. The characteristics of the absorptive medium can be adapted to specific PFAS, though this leads to trade-offs with improved recovery of some species and lower recovery of others. Spent recovery media are typically incinerated at high temperature.

Other effective treatments are reverse osmosis which is commonly used for preparation of drinking water<sup>60</sup>, ion exchange and nano-filtration. All cases will generate wastes that require either specialised storage or high temperature incineration.

Concawe reports on a number of other innovative methods currently being explored, such as photolysis/ photocatalysis, reductive decomposition, advanced oxidation and sonolysis. However, none seem close to application. In particular, these technologies are unlikely to be feasible for high flowrate, low concentration applications, precisely the conditions faced for environmental remediation.

The potential for contamination of surface waters may also need to be considered. In many cases, contamination would be better treated at source (i.e. at the factory or at a site of soil contamination) than downstream where concentrations will be more dilute. Information from the Veneto Region below provides a case where contamination may be from diffuse sources across the Region. A first response should be to ensure that the emitting industries either treat their own waste water or discharge to sewer for treatment at a waste water treatment plant (WWTP) if this does not already happen. This may further require upgrading of the WWTP and where contamination of surface waters persists, further remedial action may be necessary.

In summary, a number of techniques are available for PFAS remediation, but they are not straightforward and as such are likely to be costly. The information above has been used to check the validity of options adopted at various locations in the case study material presented below.

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<sup>59</sup> The report does not state what these target values were. Other parts of the report discuss a range of 0.1 to 0.5 µg/l, though elsewhere reference is made to 0.023 µg/l.

<sup>60</sup> Tang C Y *et al.* (2007). Effect of Flux (Transmembrane Pressure) and Membrane Properties on Fouling and Rejection of Reverse Osmosis and Nanofiltration Membranes Treating Perfluorooctane Sulfonate Containing Wastewater. *Environmental Science and Technology* 41: 2008–2014.

### 3.2.2 *Quantifying the costs of environmental remediation*

Building on the information presented in the previous section, the costs to regulatory and other stakeholders (excluding direct impacts on health and the environment) relate to the following activities:

- survey work to identify sites that are likely not to meet regulatory criteria on PFAS contamination;
- liaison with stakeholders using or living near contaminated resources (public meetings, etc.);
- monitoring concentrations of PFAS before, during and after remediation;
- removal of contaminated soils followed by:
  - storage of these soils at controlled landfill sites, or
  - high temperature incineration, or
  - soil washing to extract PFAS
    - storage or high temperature incineration of contaminated filters, etc.
- extraction of groundwater followed by:
  - use of GAC to absorb PFAS, or
  - use of ion-exchange (IX) resins to absorb PFAS, or
  - reverse osmosis or nanofiltration, which may require expansion of water treatment facilities given the need to generate an additional 15–20% of water for use in the treatment process<sup>61</sup>
    - all of which would need to be followed by storage of collected pollutants or high temperature incineration of contaminated filters, etc.

Stabilisation of PFAS within soils is not considered here to be a long-term solution to the problem for the vast majority of cases, and so is not considered further.

Cost data for the above activities have been taken from the case study examples provided in Chapter 4 and various sources in the grey literature (see Annex 3 for more on this).

Using data from reported cases, analysis can be carried out in two stages:

1. Quantification of the costs of environmental remediation for a standardised situation, e.g.
  - a. release of a standardised quantity of PFAS into soil (e.g. 10kg); and
  - b. contamination of a drinking water supply
    - i. leading to the provision of water from alternative sources for a standardised population (e.g. 100,000 people)

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<sup>61</sup> Black and Veatch (2018) Alternatives Evaluation Report. Emerging contaminants treatments strategies study.

- ii. leading to the requirement for upgrading of water treatment works for a standardised population (e.g. 100,000 people)
2. Extrapolation of the “standardised cases” to national or European level, accounting for:
  - a. the number of sites where soils are contaminated by PFAS and where soils will need to be decontaminated or stabilised;
  - b. the number of communities served by contaminated water supplies in need of remediation;
  - c. the probability that these sites will be remediated; and
  - d. the population served by contaminated drinking water supplies.

The analysis that follows identifies a number of uncertainties that affect the analysis, including:

- the quantities of PFAS released;
- the number of contaminated sites;
- the representativeness of case study material; and
- Limited data availability for some activities.

The extrapolation of existing data to new cases is clearly prone to uncertainty. The easiest way of dealing with this would be to define plausible ranges for each variable used in the extrapolation, and to use these to calculate absolute minimum and maximum estimates of cost. Such an approach has three problems:

1. It can provide an extremely broad spread of results.
2. It biases attention to the extremes of the range rather than to the most likely estimate, providing no reason to prefer any value between the two.
3. It typically does not account for possible correlation between input values.

Consideration was given to the use of Monte Carlo analysis to deal with uncertainty. However, it was concluded in the course of the work that this could give a false impression of the quality of the data adopted for analysis. An alternative developed for the presentation of results was to adopt a series of plausible and well-defined scenarios to demonstrate the likely and potential magnitude of damage and reasonable ranges for the results. The assumptions followed for each scenario would be clearly stated with the results in order that readers can understand why and how differences arise.

### 3.2.3 Quantifying the costs related to loss of ecosystem services

Ecosystem services are typically regarded as falling into four categories:

- supporting services, such as nutrient cycling and soil formation
- provisioning services, such as the production of food, fishing opportunity, raw materials, novel compounds of possible medicinal use and clean water
- regulating services, such as carbon sequestration, water purification, waste cycling, pollination and pest control; and
- cultural services, such as the spiritual and historic significance of natural resources.

Whilst the possibility of PFAS affecting a number of these services is not ruled out, there is a lack of data available for describing associated impact pathways. The most significant omissions concern information on response functions for specific ecosystems and data on stock at risk for specific sites affected by PFAS contamination.

An alternative route, proceeding from emission straight to valuation, is possible using results of studies undertaken to quantify individual willingness to pay (WTP) to avoid the impacts of either PFAS or other substances with PBT or vPvB properties. Two examples have been identified:

1. A study by Sunding (2017) assessing damage to Minnesota's natural resources resulting from 3M's disposal of PFCs in Washington County, Minnesota<sup>62</sup>.
2. A UK survey undertaken not on PFAS specifically but for the REACH restriction dossier for D4/D5<sup>63</sup> that considered WTP regarding PBT and vPvB properties.

Sunding's study from the USA covered damage to groundwater, surface water and increased costs of water purification. The groundwater assessment compared the difference in house prices in areas where groundwater was contaminated and used for drinking water, with areas that were uncontaminated. Results showed a decline in house prices of 7.3% in the most affected areas (4.4% in other contaminated communities). The average lost value per home was USD 17,400 or EUR 12,657 (USD 14,000 or EUR 10,184 in other affected communities) with an annualized loss of USD 288 or EUR 209 (USD 231 or EUR 209). Total past and future damage for the period 1971 to 2050 for the 57,000 houses affected was estimated to USD 1.5 billion, or EUR 1.1 billion.

Results were corroborated with surveys of residents and consideration of defensive expenditures via bottled water sales. As part of the assessment WTP to avoid fishing in PFOS contaminated surface waters was estimated from a survey at USD 19 (EUR 14) to USD 45 (EUR 33) per trip depending on the type of fish present ("popular"

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<sup>62</sup> Sunding D L, Damage to Minnesota's natural resources resulting from 3M's disposal of PFCs in Washington County.

<sup>63</sup> RAC/SEAC (2016) Background Document to the Opinion on the Annex XV dossier proposing restrictions on Octamethylcyclotetrasiloxane (D4) and Decamethylcyclopentasiloxane (D5). Committee for Risk Assessment and Committee for Socio-Economic Analysis, European Chemicals Agency, Helsinki.

or “unpopular” species). Total loss to anglers over the period 2008 to 2040 was estimated at USD 121 million (EUR 88 million).

Further to these calculations, restoration costs related to public water supply and point-of-entry treatment units for 2018 to 2050 was valued at USD 396 million (EUR 288 million). It is unclear in the Sunding study what is covered by these restoration costs. Nevertheless, it is presumed that the Sunding study concern the upgrading of water treatment facilities to remove PFAS.

The D<sub>4</sub>/D<sub>5</sub> study using the UK survey, notes the following

“Quantification of environmental impacts of regulatory policy changes is difficult. In the case of D<sub>4</sub>/D<sub>5</sub> the benefits of the proposed restriction are estimated by considering society’s Willingness to Pay (WTP) for a reduction in potential risks to the aquatic environment. ... A representative sample of the UK population (sample size = 829) stated that they were willing to pay €46 per year per person to reduce the risks associated with the PBT substance - D<sub>4</sub>, and €40 per year per person to reduce the risks associated with the vPvB substance - D<sub>5</sub>. The WTP for superior quality personal care products (i.e. those that use D<sub>4</sub>/D<sub>5</sub>) was estimated at €5 per year per person. This indicates that respondents value the precautionary benefits of reduced potential risk of accumulation of D<sub>4</sub>/D<sub>5</sub> in the aquatic environment at around seven times the value of the loss of beneficial properties provided by D<sub>4</sub> and D<sub>5</sub> in personal care products.”

The application of the results of the survey recognizes that significant uncertainty exists. Rather than seeking to provide a total estimate of damage to compare against any increase in cost of alternatives, the study compares two alternative WTP estimates. The first WTP estimate is linked to avoidance of environmental harm and the second to the useful properties that are linked to the presence of D<sub>4</sub>/D<sub>5</sub> in personal care products, providing a general indication of societal preference.

These results are discussed further below, though a full quantification by country is not presented. To make such a quantification based on the Sunding analysis of house prices and fishing trips would require additional information that is not available on the scale of the current assessment (e.g. knowledge of angling behavior in areas where there is PFAS contamination). The results of the D<sub>4</sub>/D<sub>5</sub> assessment are based on a relatively small survey, the authors of the work applied them only in a limited way in development of the restriction dossier, not quantifying a total cost but comparing WTP to avoid damage with WTP for the properties that they provide in the end product.

#### 3.2.4 Value transfer

Value transfer, often called benefits transfer in environmental economics, is a practice used to estimate economic values by transferring information available from an already completed study in one context to another, for example by transferring valuation of fishing trips in the USA to a European country.<sup>64</sup> The need for value transfer arises because of the limited amount of valuation data available.

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<sup>64</sup> The methods are described in detail in Navrud S. and Ready R. (eds.) (2007). *Environmental Value Transfer: Issues and Methods*. Springer.

At its most basic, values are simply transferred from one location to another, with no adjustment. Such an approach is reasonable where the differences between locations are expected to be small. However, in most cases the process is not as simple, where people in one location may respond differently to those in another location for example relating to differences in income, culture, or availability of alternative resources. The validity of the transfer should therefore be evaluated carefully.

Three approaches have been described<sup>65</sup>:

- unit value transfer
- value function transfer; and
- meta-analysis.

Unit value transfer is typically made with adjustment for variation in income:

$$B_{p'} = B_S \left( \frac{Y_p}{Y_S} \right)^\beta$$

Where  $B_{p'}$  = adjusted benefit estimate for new location,  $B_S$  = original estimate from study site,  $Y_p$  and  $Y_S$  are the income levels at the new location and study site respectively and  $\beta$  = elasticity (typically 0.4–1.0). Jacobsen and Hanley<sup>66</sup> found that GDP/capita (i.e. societal income) was a better predictor of WTP than respondents' income, which simplifies the calculations.

International transfer in this context is carried out using exchange rates adjusted for purchasing power parity (PPP). Purchasing power parities are the rates of currency conversion that equalise the purchasing power of different currencies by eliminating the differences in price levels between countries. The production of PPPs is a multilateral exercise involving the National Statistical Institutes of the participating countries, Eurostat and the Organisation for Economic Co-operation and Development (OECD), and is governed by the PPP Regulation.<sup>67</sup> Data are available from OECD.<sup>68</sup>

In our study setting a question that arises is how reliable extrapolation from the USA to Europe might be, given that a significant part of the data identified for this research comes from North America. One response to this question is that there is likely as much or more variation between European countries, as there is between the USA and Europe. Another response concerns the way that the US data are used, often alongside European data and serving to extend the evidence base for the analysis. Consideration has been given to whether there is any clear disparity in US and European estimates.

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<sup>65</sup> Navrud S. (2016). Possibilities and challenges in transfer and generalization of monetary estimates for environmental and health benefits of regulating chemicals. (Presentation at OECD Workshop on socioeconomic impact assessment of chemicals management).

<sup>66</sup> Jacobsen J B and Hanley N (2009). Are There Income Effects on Global Willingness to Pay for Biodiversity Conservation? *Environmental and Resource Economics*, Volume 43, Issue 2, pp 137–160.

<sup>67</sup> Regulation (EC) No 1445/2007 of the European Parliament and of the Council of 11 December 2007 establishing common rules for the provision of basic information on Purchasing Power Parities and for their calculation and dissemination.

<sup>68</sup> OECD Database: Purchasing power parities. Accessed 10.11.2018.

Value function transfer and meta-analysis add further explanatory variables into the equation to explain how values may vary from site to site. For the present analysis, however, neither technique has proved necessary in a formal sense because of the nature of the data gathered. However, the derivation of ranges and best estimates takes account of some of the principles of value function transfer by recognising non-linearities in costs according to (e.g.) the number of households affected.<sup>69</sup>

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<sup>69</sup> Upon request the excelspread sheets used for the monetarisation and valuation in this report can also be provided along with a guidance on how to use the estimation of costs for value transfer.

## 4. The case studies

The case studies have multiple roles in this study. One role is to illustrate the impact pathways, i.e., how the production of PFAS and the manufacture of PFAS-containing products, as well as the use and end-of-life disposal of those products, result in emissions to the environment and human exposures. This provides a basis for identifying the impact pathways for similar instances of contamination in other locations.

An additional purpose of the case studies is to gather concrete cost data based on actual instances of PFAS exposure and to translate these into costs per incident. They also help to identify the additional information needed to quantify the negative impacts in a way that will enable them to be extrapolated – if possible – to the Nordic region and then to Europe overall.

The case studies follow the life cycle of PFAS from their production at chemical production facilities, their application in manufacturing of products, the use phase and the impact pathways for wastewater discharges, and then the end-of-life phase and their disposal in landfills. Incineration is not reviewed per se, as the PFAS emissions from this disposal method (air-borne as well as via the bottom ash) are not yet well studied.<sup>70</sup> Several case studies also provided opportunities for getting human epidemiological data on health effects as well as concrete health-related costs.

### 4.1 Case Study 1: Exposures due to production of PFAS

Case Study 1 looks at how the production of PFAS results in emissions to the environment, resulting in human and environmental exposure (impact pathways).

#### 4.1.1 Background and context

The production of PFAS can generate extensive emissions. Most PFAS are colorless and odorless<sup>71</sup>, and initially they were produced without consideration of the impacts of emissions during their production and processing.

The US company 3M was the first to manufacture a PFAS commercially – the C8, PFOA. It licensed the technology to another US company, Dupont, which developed the polymer Teflon. In the 1970s and 1980s 3M scientists became concerned because laboratory animals exposed to C8s were developing health problems, including birth defects. They alerted Dupont scientists and Dupont started to take measures to protect

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<sup>70</sup> Hansson *et al.* (2016). Sammanställning av befintlig kunskap om föroreningskällor till PFAS-ämnen i svensk miljö. Report Number C 182.

<sup>71</sup> The odor of PFBA has been described as "sharp, similar to butyric acid."

workers exposed in their jobs. Measures to reduce emissions to the environment were not taken until considerably later. Contamination levels at production sites can be high.

A number of lawsuits are underway to prompt action from manufacturers to clean-up contamination. The level of damages and settlements reached depend on a number of factors which include the identified harm to the environment and human health. Some settlements include punitive damages and funds to support the clean-up of the contaminated water.

In February 2018, a settlement of USD 850 million (EUR 618 million) was reached between 3M and the state of Minnesota.<sup>72</sup> The funds are expected to support the development of alternative water supplies, treat existing water supplies, finance water conservation and efficiency projects and support groundwater recharge projects.<sup>73</sup> While it is the largest settlement to date in the United States for a case of PFAS contamination, the amount originally sought by the plaintiff was USD 5 billion.<sup>74</sup> The settlement amount diminishes in comparison to the company's market value (about 1%) and the profits made on the PFAS-containing products.<sup>75</sup> 3M is facing other legal actions: a February 2018 news article reported that 37 cases related to PFC contamination against 3M are underway.<sup>76</sup>

#### 4.1.2 Cases of contamination

##### Chemours factory complex in Dordrecht, Netherlands

The Chemours plant in the Netherlands belonged to the company Dupont until 2015, when Dupont spun off its specialty chemicals division, which included production of fluorochemicals such as the fluoropolymer Teflon. The Dordrecht plant is Chemours' biggest production site in Europe, employing over 550 employees.<sup>77</sup> In addition, Chemours has a total of 35 production sites worldwide, 25 in North America, four in Europe, Africa and Middle East, four in Asia and two in South America.

In 2012 Dupont had replaced production of C8 with GenX, a perfluoroalkyl ether carboxylate alternative that includes FRD-902, FRD-903 and E1. While GenX seems to be less bioaccumulative than PFOA<sup>78</sup>, reports filed by Dupont with the USEPA indicated that the replacement chemical may cause some of the same health problems as PFOA.<sup>79</sup>

Although the Dordrecht facility supposedly stopped PFOA production in 2012, toxicological studies found concentrations of PFOA in grass surrounding the plant that

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<sup>72</sup> Kary T, 3M Settles Minnesota Lawsuit for \$850 million. Bloomberg, 20.02.2018. Accessed 08.13.2018.

<sup>73</sup> Marten Law (website). The True Cost of Scotchgard: 3M to Pay Minnesota \$850 Million in Perfluorochemical Settlement. Accessed 10.10.2018.

<sup>74</sup> Marcotty J, State alleges 3M chemicals caused cancer and infertility, alleges \$5 billion in damage. Star Tribune, 21.11.2017. Accessed 10.10.2018.

<sup>75</sup> Alder C, 3M settles Minnesota groundwater lawsuit for \$850 million. 21.02.2018. Accessed 09.09.2018.

<sup>76</sup> Marcotty J, 3M settles groundwater lawsuit for \$850 million, Star Tribune, 20.02.2018. Accessed 15.10.2018.

<sup>77</sup> Scott A, Dutch Chemical Plant Under Investigation, Chemical and Engineering News, 18.04.2016. Accessed 10.11.2018.

<sup>78</sup> Beekman M *et al.* (2016). Evaluation of substances used in the GenX technology by Chemours, Dordrecht, RIVM Letter. Report 2016-0174.

<sup>79</sup> Lerner S (2016). New Teflon Toxin Causes Cancer in Lab Animals, The Intercept, 03.06.2016. Accessed 18.09.2018.

were inconsistent with that claim.<sup>80</sup> Today the facility discharges both FRD-902 and FRD-903 into wastewater, which is pre-treated before being discharged into sewage treatment. FRD-903 is also released into the air via the factory's chimneys.<sup>81</sup>

In 2016, the Dutch government asked the Dutch National Institute for Public Health and the Environment (RIVM) to evaluate the existing knowledge of the toxicity and health effects of PFOA (C8), which had been released from the Chemours facility from 1970 until 2012.<sup>82</sup> RIVM estimated that 750,000 people were exposed to high levels of PFOA due to their residence in cities close to the Dordrecht plant and the Merwede river downstream.<sup>83</sup> RIVM also detected PFAS and GenX in vegetable gardens within 1 kilometer of the Dordrecht plants, suggesting pathways into humans.<sup>84</sup> While levels of contamination do not exceed advisory thresholds, residents also face exposure from the air directly as well as drinking water. Thus, residents are advised to consume produce from their vegetable gardens in moderation.

The Chemours facility used PFOA to produce the fluoropolymer known as Teflon, a production process similar to that carried out at a Chemours (formerly Dupont) plant in West Virginia.<sup>85</sup> The West Virginia plant has been the target of legal action since 2001, when residents brought a class action against Dupont concerning their exposure to PFOA. Under a 2004 settlement, Dupont agreed to fund a medical monitoring program for 70,000 persons as well as new water treatment systems. After ten years, the C8 panel of scientists following the medical monitoring program concluded that six illnesses were probably linked to the exposure to PFOA: kidney and testicular cancer, ulcerative colitis, thyroid disease, pregnancy-induced hypertension and high cholesterol. More than 3,500 personal injury claims were pending against Dupont and Chemours when agreement was reached to settle the claims for USD 671 million (EUR 488 million).

The Netherlands government requested a medical monitoring study of the health effects resulting from the exposure in Dordrecht, following the approach taken with the Dupont case in West Virginia. The study undertaken by RIVM found the levels of PFOA in Dordrecht's drinking water to have been lower than in the US case. However, concentrations in the atmosphere between 1970 and 2012 had exceeded the legal amount. RIVM's risk assessment concluded that under the most unfavourable scenario for concentration levels, some residents might experience impacts on the liver. However, risks of cancer and to the unborn child were considered to be limited.<sup>86</sup>

Chemours was required to reduce GenX emissions from 6,400 kg/year to 2,000 kg/year. In September 2018, the company announced it would invest EUR 75 million in

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<sup>80</sup> No Author, Another Chapter: Chemours releases GenX and PFOAs into waters globally, *Encore*, 08.08.2018. Accessed 10.10.2018.

<sup>81</sup> RIVM (2016). GenX (Website in Dutch).

<sup>82</sup> RIVM (2016). Risicoschatting emissie PFOA voor omwonenden (In Dutch).

<sup>83</sup> No Author (2018). Another Chapter: Chemours releases GenX and PFOAs into waters globally, *Encore*, 08.08.2018. Accessed 10.10.2018.

<sup>84</sup> RIVM (2018). Risicobeoordeling van GenX en PFOA in moestuingewassen in Dordrecht, Papendrecht en Sliedrecht. (In Dutch with English Summary).

<sup>85</sup> Nair A S, DuPont settles lawsuits over leak of chemical used to make Teflon. *Reuters Business News*, 13.02.2017. Accessed 10.09.2018.

<sup>86</sup> RIVM (2018). Risicobeoordeling van GenX en PFOA in moestuingewassen in Dordrecht, Papendrecht en Sliedrecht. (In Dutch with English Summary).

reducing emissions of GenX and organic fluorinated substances, by installing active carbon filters and other technical solutions which are expected to eventually remove up to 99% of the targeted substances.<sup>87</sup>

Note that a Chemours (formerly Dupont) facility in Fayetteville, North Carolina is also the target of legal action for polluting a wide geographical area. The plant discharged large amounts of GenX into the Cape Fear River, the major source of drinking water for downstream cities, including Wilmington (110,000 pop.). A lawsuit filed against Dupont and Chemours in February 2018 consolidated three class action suits and is seeking funds for environmental cleanup (cost of water filtration), monitoring, and punitive damages for illness and reduced property values.<sup>88</sup> In November 2018, state authorities and Chemours announced they had reached an agreement which will cost the company USD 12 million (EUR 8.7 million) in civil penalties, in addition to USD 1 million (EUR 727,400) in investigation costs. Furthermore, if the agreement is approved by the court in its current state, Chemours has committed to reduce its emissions significantly. If reduction targets are not achieved additional fines will be paid.<sup>89</sup>

### Veneto region, Italy

A large-scale contamination of PFAS was discovered in the Veneto Region of Italy in 2013, directly affecting groundwater, surface water, drinking water and land in an area of over 200 square kilometers.<sup>90</sup> The contamination was attributed to emissions from a facility operated by the company Miteni since 1964. The chemical company produced several PFAS-containing products such as herbicides and pharmaceuticals. The company reported on their website that production of PFOS and PFOA stopped in 2011<sup>91</sup>, but their product catalog still includes PFHxS and PHxSF.<sup>92</sup>

The Veneto Region's response to the incident drew on a wide range of stakeholders across sectors. Several monitoring studies were undertaken to gather information on the levels of contamination and their impacts. Because of this contamination, standards were introduced for water, agriculture and food. In April 2018, a legal case was brought against Miteni<sup>93</sup> by the Public Prosecutor's Office in Vicenza.<sup>94</sup>

The contamination included a number of PFAS compounds. Monitoring data collected between 2013 and 2015 identified the following specific compounds: perfluorobutanoic acid (PFBA), perfluorobutane sulfonate (PFBS), perfluorodecanoic

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<sup>87</sup> Chemours, The Chemours Company Takes Significant Action to Minimize Emissions. Accessed 08.09.2018.

<sup>88</sup> Reisch M S, Merged lawsuit filed against DuPont and Chemours in North Carolina, Chemical and Engineering News, 05.02.2018, Accessed 08.09.2018.

<sup>89</sup> Wagner A, Chemours to pay \$12 Million fine as part of GenX Agreement, StarNews Online, 21.11.2018. Accessed 01.12.2018.

<sup>90</sup> WHO Europe (2016). Keeping our water clean: The case of water contamination in the Veneto Region, Italy.

<sup>91</sup> As claimed by Miteni Company on their website, available here.

<sup>92</sup> This information was obtained by an NGO (IPEN and Alaska Community Action on Toxics (ACAT)) submission of information specified in Annex E of the Stockholm Convention pursuant to Article 8 of the Convention, and confirmed with a product research on the company's website using CAS numbers for PFHxS and PHxSF.

<sup>93</sup> In October 2018, the company filed bankruptcy.

<sup>94</sup> No author, La procura di Vicenza indaga sulle sostanze cancerogene nell'acqua, Le Iene, 21.05.2018, Accessed October 2018 (in Italian).

acid (PFDA), perfluorododecanoic acid (PFDoDA), perfluoroheptanoic acid (PFHpA), perfluorohexanoic acid (PFHxA), perfluorohexane sulfonate (PFHxS), perfluorononanoic acid (PFNA), perfluoropentanoic acid (PFPeA) and perfluoroundecanoic acid (PFUnDA).<sup>95</sup> Another source indicated that PFPeA, PFHxA, PFHpA, PFHxS, PFNA, PFDA, PFUnDA, PFDoDA, PFBA, and PFBS were present in the contaminated waters.<sup>96</sup>

Residents in the surrounding areas were exposed to the contamination for decades. The highest combined concentration levels of PFAS (estimated to be 1214 ng/l) were found in the municipalities of Brendola, Lonigo and Sarego<sup>97</sup> among others.<sup>98</sup> Estimates for the number of affected individuals vary from 120,000<sup>99</sup> to 350,000 people.<sup>100</sup>

The Veneto Region Environmental Agency carried out studies identifying two main pathways for the contamination in the area. One pathway was through contaminated wastewater emitted from the chemical factory directly into a creek and the surrounding groundwater. The second pathway was from the wastewater plant to a canal that drained into the surface waters of the Brenta river.<sup>101, 102</sup> This river basin, which is characterised by a 100–200 metre deep bed of pebbles and gravel, is highly permeable, increasing the spread of the contamination.

The contaminated water was drunk by residents in the area and also made its way into the food chain via contaminated water used for irrigation. Contamination was found in foods such as eggs and fish. Agriculture in the Veneto Region represents about 10% of national production suggesting a high risk for contaminated foods being shipped and consumed in other parts of the country or even in other countries.<sup>103</sup>

Information on health risks was collected through a biomonitoring study undertaken between July 2015 and April 2016 for different population groups including people working in the agricultural sector and people residing in uncontaminated areas, which served as a control group. Agricultural workers were given greater attention due to their greater risk of exposure to crops and livestock. An analysis found a higher risk of mortality, diabetes, cerebrovascular diseases, myocardial infarction and Alzheimer's disease. Females had an increased morbidity for several conditions. The results are summarized in Table 8.

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<sup>95</sup> Mastrantonio M *et al.* (2017). Drinking water contamination from perfluoroalkyl substances (PFAS): an ecological mortality study in the Veneto Region, Italy. *The European Journal of Public Health*. Feb 1;28(1):180–185.

<sup>96</sup> ISS (National Institute of Health). Dept of Environmental Health (no date). Perfluorooctanoic acid (PFOA) pollution of groundwater: the case study of Veneto, Italy.

<sup>97</sup> *Ibid.*

<sup>98</sup> Regione di Veneto (2018). Piano di Sorveglianza Sanitaria Sulla Popolazione Esposte alle Sostanze Perfluoroalchiliche (in Italian), Rapport no 04, p.10.

<sup>99</sup> WHO Europe (2016). Keeping our water clean: The case of water contamination in the Veneto Region, Italy.

<sup>100</sup> No author, Another Chapter: Chemours releases GenX and PFOAs into waters globally, *Encore*, 08.08.2018. Accessed 10.10.2018.

<sup>101</sup> WHO Europe (2016). Keeping our water clean: The case of water contamination in the Veneto Region, Italy.

<sup>102</sup> ISS (National Institute of Health). Dept of Environmental Health (no date). Perfluorooctanoic acid (PFOA) pollution of groundwater: the case study of Veneto, Italy.

<sup>103</sup> No author (2018). La procura di Vicenza indaga sulle sostanze cancerogene nell'acqua, *Le Iene*, 21.05. 2018, Accessed October 2018 (in Italian).

**Table 8: Disease risk ratios – Veneto region**

	Males		Females	
	Number of deaths	Relative risk (95% confidence interval)	Number of deaths	Relative risk (95% confidence interval)
General mortality	21,149	1.19 (1.17–1.21)	20,692	1.21 (1.19–1.23)
Diabetes	292	1.21 (1.06–1.38)	595	1.48 (1.34–1.62)
Cerebrovascular disease	1,871	1.34 (1.27–1.41)	2,271	1.29 (1.23–1.34)
Myocardial infarction	1,900	1.22 (1.16–1.28)	1,458	1.24 (1.17–1.32)
Alzheimer’s disease	89	1.33 (1.05–1.70)	178	1.35 (1.09–1.67)
Kidney cancer		1.07 (0.90–1.28)		1.32
Breast cancer		n.a.		1.11
Parkinson’s disease				1.35

Source: Mastrantonio M *et al.* (2017). Drinking water contamination from perfluoroalkyl substances (PFAS): an ecological mortality study in the Veneto Region, Italy. *The European Journal of Public Health*. Feb 1;28(1):180–185.

Monitoring studies also found a higher rate of mortality and disease (liver, bladder and kidney cancer and cirrhosis) among employees at the chemical plant.<sup>104</sup>

Within three months of the 2013 announcement of PFAS contamination in the Veneto Region, authorities installed activated carbon filters in drinking water treatment plants. The estimated cost of the installation was EUR 2 million, which was paid for by the Veneto Region’s government and taxpayers. Activities related to the Health Surveillance Plan cost an additional EUR 4.3 million. These modifications resulted in a substantial decline in the concentration of PFOA in water from about 1475 ng/l to 386 ng/l, and a decline in PFOS from 117 ng/l to 36 ng/l.

Maintaining the carbon filters will also incur costs. One source estimated the cost of a changed set of filters at EUR 150,000 while chemical monitoring costs EUR 750,000, leading to a total cost of the filter installation of EUR 900,000 per year.

The short-term cost (5 years) was estimated to be EUR 6.5 million while investment in the medium-term to improve water treatment plants was estimated to be EUR 4.2 million. An alternative solution to invest in new water pipelines was estimated to be EUR 61.7 million. Table 9 translates the cost findings to the production cost of drinking water.

**Table 9: Production cost of drinking water**

	2014 (EUR cent/m3)	2015 (EUR cent/m3)
Cost without PFAS pollution	4.7–8.3	4.0–8.5
Cost with PFAS pollution	10.0–18.7	6.6–21.0

Source: ISS (National Institute of Health). Dept of Environmental Health (no date). Perfluorooctanoic acid (PFOA) pollution of groundwater: the case study of Veneto, Italy.

<sup>104</sup> WHO Europe (2016). Keeping our water clean: The case of water contamination in the Veneto Region, Italy.

Between 2013 and 2014, the chemicals company also installed activated carbon filters for wastewater treatment.<sup>105</sup>

### Fluorochemical production sites – data limitations and assumptions

As the case studies indicate, fluorochemical production plants have historically been major sources of PFAS exposure. One of the challenges of this study was to estimate the current number of fluorochemical production sites in the Nordic countries and in Europe. The constant changes within the fluorochemicals/fluoropolymers industry made it difficult to use data sources from the past that identified sites by company. For example, Elf Atochem had merged with Total/Fina to become Alofina which later became, Arkema. Similarly, ICI Fluorochemicals was sold to INEOS, which in turn sold that part of its business to Mexichem.

In addition, production is shifting from Europe and North America to Asia, particularly China, and other regions. Among the major producers are companies such as Daikin, Asahi Glass, Honeywell, Mitsui Chemicals, Gujaratfluorochemicals, and Dongyue Chemicals. BASF, Solvay, and Dupont are also global players.

The US-based FluoroCouncil represents a number of PFAS producers internationally. Members include Archroma, Arkema France, AGC Inc., Chemours, Daikin Industries, and Solvay Specialty Polymers. Dynax Corporation and Tyco Fire Products are associate members.

Regulatory pressure has also contributed to changes in the industry. Production of the long-chain PFOA and PFOS was phased out years ago by companies like 3M, Dow-Dupont, and BASF, which for the most part switched to production of short-chain PFAS. However, company websites seldom provide information on which fluorochemicals are produced and at which sites.

The list presented in Table 10 is the result of the search efforts carried out for this study. It assumes that production of fluorochemicals and/or fluoropolymers is taking place in 2018 at the following sites within Europe:

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<sup>105</sup> ISS (National Institute of Health). Dept of Environmental Health (no date). Perfluorooctanoic acid (PFOA) pollution of groundwater: the case study of Veneto, Italy.

**Table 10: List of manufacturers of fluorochemicals and/or fluoropolymers**

Country	Company and site of plant	What is being produced
Belgium	3M (Zwijndorf)	Fluorochemicals
France	Arkema (Pierre-Bénite)	Fluoropolymers (PVDF)
	Solvay Solexis (Tavaux)	Fluoropolymers (PVDF)
	Daikin Chemical France S.A.S. <sup>1</sup>	Fluorochemicals
Germany	Dyneon (Gendorf)	Fluorochemicals, fluoropolymers (PTFE, FEP, PFA, THV)
	BASF (Ludwigshafen)	n.a.
Italy	Solvay Solexis (Spinetta-Argeno)	Fluoropolymers ) – PTFE, MFA
	Heroflon S.p.A. (Collebeato)	Fluoropolymers (PTFE compounds and micropowders)
	Miteni (Trissino) <sup>2</sup>	Fluorinated intermediates; performance fluorinated products
Netherlands	Chemours (Dordrecht)	Fluoropolymers (PTFE, FEP)
	Daikin Chemical Netherlands (Oss) – Pre-compounding of fluoroelastomers	Fluorochemicals
United Kingdom	AGC (Blackpool)	Fluoropolymers – PTFE, PFA

Source: 1) Daikin Europe, manufacturing of fluoroelastomer base polymer and polymer processing aids.

2) Miteni files bankruptcy in October 2018.

Based on this list, it is further assumed that the number of PFAS production sites in Europe is between 12 and 20 plants. However, in this study the authors have not been able to identify any PFAS production facilities in the Nordic countries.

## 4.2 Case Study 2: Exposures due to manufacture and commercial use of PFAS-containing products

### 4.2.1 Background and context

The many facilities where PFAS are used in the manufacture of consumer goods and other products also constitute major sources of PFAS emissions to the environment. A 3M study from 2000 estimated that 15% of all indirect emissions of POSF (perfluorooctane sulfonyl fluoride, compound that is used for producing PFOS) occurred during manufacturing from secondary applications.<sup>106</sup> In addition, the use of PFAS as surfactants and coatings in providing commercial services, such as professional cleaning, has also been linked to hotspot contamination.

A mapping of PFAS pathways in Sweden concluded that quantifying the emissions released from the many uses of PFAS in products might not be possible due to lack of data and the “diffuse character” of certain areas of usage.<sup>107</sup> To increase information on how and where PFAS are used, the Swedish Chemicals Agency has recently introduced

<sup>106</sup> Alexandre G. Paul *et al.* (2008). A First Global Production, Emission, And Environmental Inventory For Perfluorooctane Sulfonate. *Environmental Science & Technology* 2009 43 (2), 386–392.

<sup>107</sup> Hansson *et al.* (2016). Sammanställning av befintlig kunskap om föroreningskällor till PFAS-ämnena i svensk miljö. Report Number C 182.

a requirement for companies to report all products containing added PFAS compounds and their use to the Swedish "Products Register"<sup>108</sup>, starting from 1 January 2019.

This case study identifies several industrial activities that have the potential to release PFAS to the environment, either directly or indirectly, e.g., through discharges to wastewater treatment works on site or off-site via sewerage systems. It provides brief descriptions of cases where contamination has been identified, leading to costs for remediation.

#### 4.2.2 Cases of contamination

##### Textile and leather manufacturing

The textile industry is one of the most extensive users of PFAS. The water repellency and stain resistance qualities of PFAS have led to widespread PFAS treatment of items intended for use outdoors such as raincoats, snowsuits for children, ski jackets, shoes and umbrellas as well as outdoor equipment such as tents, awnings and sails. Other items frequently treated with PFAS include carpets, upholstery, and leather products. Textile applications can account for an estimated 35% of the demand for fluorotelomers. This demand is projected to grow by 14% by 2020.<sup>109</sup>

Awareness within the textile industry concerning the health and environmental impacts of PFAS is growing and alternative non-fluorinated methods of treating fabrics and leather for water repellency have been analysed.

Though the case study of PFAS contamination in the Veneto region above focused on the facility producing fluorochemicals operated by Miteni, sources of the contamination may also include hundreds of smaller textile and leather companies. In fact, the Veneto Region is known for its model of "industrial clusters" where multiple companies producing the same good are located in the same area.

As shown in Figure 3 below, several industrial clusters producing goods that could contain PFAS were present in the contaminated area.

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<sup>108</sup> Swedish Chemicals Agency (2018). (News) The Swedish Chemicals Agency is introducing a requirement to report PFAS to the Products Register. Accessed September 2018.

<sup>109</sup> Press release (No date) Fluorotelomers Market to Reach USD 539.3 Million Worldwide by 2020, Digital Journal. Accessed 10.11.2018.



### Metal plating, including chromium plating

The use of PFOSs as a wetting agent/fume suppressant in chromium plating was first reported in 1954<sup>112</sup>, and additional types of PFAS have been developed for such use since then.<sup>113</sup> PFAS are also used to improve the stability of the baths used in electroplating of copper, nickel and tin and the overall performance of the process. In addition, PFAS are used to treat metal surfaces to prevent corrosion, reduce mechanical wear, or enhance aesthetic appearance, as well as to promote flow of metal coatings and prevent cracks during drying.<sup>114</sup>

The chrome plating industry is estimated to use around 32 to 40.7 tonnes of pure PFOS globally. The estimations are likely to cover different applications within metal plating, and not only chromium plating.<sup>115</sup> For Denmark, this number was estimated to be 10 kg/year in 2009 with 28 kg/year as purchased quantity.<sup>116</sup> In Sweden, the figure (from 2013) was 180 kg/year for PFOS.<sup>117</sup>

Again, the proportion of metal plating facilities in Europe using PFAS is not known. Statistics from Eurostat<sup>118</sup> on the number of establishments of treatment and coating of metals and machining in the EEA indicate 163 establishments with more than 250 employees in 2015, and another 151,455 establishments with less than 250 employees, i.e., SMEs.

### Paper and paper products

Paper mills and paper products industry using PFAS to produce waterproof and grease-proof paper products may be a significant source of PFAS contamination released to water and air as well as to soil.<sup>119</sup> For example, a mapping of PFAS pathways to Norway's Tyrifjorden identified paper mills as among the main sources of the fjord's PFAS contamination. As per Case Study 4.5.2.1 below, paper mill waste has also been implicated in PFAS-contaminated substrate sold as compost.

The PFAS are typically added to wet wood fibers that are subsequently made into paper. A US NGO obtained notifications to the US Food and Drug Administration concerning PFAS for use in food contact materials.<sup>120</sup> The FDA notifications estimated releases to the environment of the PFAS based on a *typical paper mill producing 825 tonnes of PFAS-coated paper per day and discharging 26 million gallons (around 99,000 m<sup>3</sup>) of water per day*. One notification from the company Chemours estimated wastewater discharges of 95 pounds/day (43 kg/day–15,965 kg/y) in discharges contain-

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<sup>112</sup> German Environment Agency (2017). Use of PFOS in chromium plating – Characterisation of closed-loop systems, use of alternative substances. Report No. (UBA-FB) 002369/ENG.

<sup>113</sup> Danish Environmental Protection Agency (2011). Substitutions of PFOS for use in non-decorative hard chrome plating.

<sup>114</sup> Kissa E (2001). Fluorinated Surfactants and Repellents. Surfactant Science Series. 97. Marcel Dekker, New York

<sup>115</sup> Danish Environmental Protection Agency (2011). Substitutions of PFOS for use in non-decorative hard chrome plating.

<sup>116</sup> Ibid.

<sup>117</sup> Hansson *et al.* (2016). Sammanställning av befintlig kunskap om föroreningskällor till PFAS-ämnen i svensk miljö. Report Number C 182.

<sup>118</sup> Eurostat Annual enterprise statistics by size class for special aggregates of activities (NACE Rev. 2) [sbs\_sc\_sca\_r2].

<sup>119</sup> Neltner T and Maffini M, Paper mills as a significant source of PFAS contamination, but who's watching? EDF, 21.05.2018 Accessed 10.11.2018.

<sup>120</sup> Ibid.

ing 43,000 ppt. Another notification for the same PFAS estimated higher concentrations in the paper, resulting in 183 pounds/day (83 kg/day–30,295 kg/year) and wastewater discharges containing concentrations of 83,000 ppt.

Paper and cardboard treated with PFAS are frequently used in products such as plates, popcorn bags, pizza boxes, and food containers and wraps (see Case Study 4 on food contact materials). Non-food applications may include folding cartons and masking papers.<sup>121</sup>

Statistics from Eurostat<sup>122</sup> on the number of manufacturers of paper and paper products in the EEA provide totals of 488 manufacturers employing more than 250 employees in 2015, and another 19,477 establishments with less than 250 employees, i.e., SMEs. It is not known how many of these manufacturers use PFAS to treat their products.

### Paints and varnishes

PFAS have long been used in coating, paint, and varnish to reduce surface tension for substrate wetting and levelling, as dispersing agents, and for improving gloss and anti-static properties. The protective properties of anticorrosive paints can be enhanced by perfluorinated urethanes. PFAS can also be used as aids in pigment grinding and to address pigment flotation problems.<sup>123</sup>

Eurostat statistics<sup>124</sup> indicate that 104 manufacturers of paints and varnishes in the EEA employ more than 250 employees in 2015, and another 4,027 establishments employ less than 250 employees. Again, it is not known how many of these use PFAS surfactants in their products.

### Cleaning products

The surfactant properties of PFAS have made them useful in industrial and household cleaning products. They have been used to lower surface tension and improve wetting and rinse-off in products such as carpet spot cleaners, alkaline cleaners, denture cleaners and shampoos, floor polish, and dishwashing liquids. They are sometimes added to cleaners containing strong acids and bases, such as those for cleaning concrete, masonry, and metal surfaces (e.g. airplanes). They may also be used in nonaqueous cleaning agents to aid in removal of adhesives and in dry cleaning of textiles or in the cleaning of metal surfaces. PFAS can also enhance cleaning formulations for removal of calcium sulfate scale from reverse osmosis membranes.

The relevant Eurostat category here is “Manufacturers of soap and detergents, cleaning and polishing preparations, perfumes and toilet preparations”.<sup>125</sup> Under this category, some 178 manufacturers in the EEA employ more than 250 employees in 2015, and another 9,402 establishments employ less than 250 employees. Again, it is not known how many of these use PFAS surfactants in their products.

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<sup>121</sup> Kissa E (2001). Fluorinated Surfactants and Repellents. Surfactant Science Series. 97. Marcel Dekker, New York.

<sup>122</sup> Eurostat Annual enterprise statistics by size class for special aggregates of activities (NACE Rev. 2) [sbs\_sc\_sca\_r2].

<sup>123</sup> Ibid.

<sup>124</sup> Ibid.

<sup>125</sup> Ibid.

### Plastics, resins, and rubbers

PFAS are used to manufacture certain plastics or applied plastics such as polytetrafluoroethylene (PTFE) and polyvinylidene fluoride (PVDF). PTFE is best known by the brand name Teflon (Dupont, now Chemours). It has hundreds of uses in consumer and industrial products such as textiles, medical equipment, cookware, and so on.

PVDF is used in a range of industrial applications such as automotive fuel hoses, electrical cable insulation and jacketing, high purity piping, and semiconductor piping.<sup>126</sup> They are also used as mold-release agents for thermoplastics, polypropylene, and epoxy resins, polyurethane elastomer foam molding, in formulations for antiblocking agents for vulcanized and unvulcanized rubbers, in silicone rubber sealants for soil resistance, and to improve wetting of fibers or fillers in composite resins.<sup>127</sup>

The closest Eurostat category here is “Manufacturers of basic chemicals, fertilisers and nitrogen compounds, plastics and synthetic rubber in primary forms”.<sup>128</sup> Under this category, 340<sup>129</sup> manufacturers in the EEA had more than 250 employees in 2015, and another 8,650 establishments employ less than 250 employees. Again, it is not known how many of these are manufacturers using fluorochemicals or fluoropolymers.

### Car washes

The surfactant properties of PFAS make them useful for a wide range of industrial cleaning products and surface treatments. For example, they are used in car wash products and automobile waxes, which makes car washes potential sources of PFAS contamination.

In 2018, a car wash facility in the US state of New Hampshire was cited as one of the sources of PFAS contamination in wells serving several nearby towns.<sup>130</sup> Investigators tested wells on the car wash property and found levels for PFAS higher than expected – up to 158.8 ppt, compared to the USEPA lifetime advisory level of 70 ppt. The facility will be required to take measures to prevent the contamination from continuing.

According to the International Car Wash Association 79,000 car wash facilities are operating in Europe. These are likely to be SMEs employing less than 250 workers.<sup>131</sup> It is not known how many of these that use products containing PFAS.

### Manufacturers of PFAS-treated products – data limitations and assumptions

Table 11 present the number of manufacturers carrying out activities that may involve the use of PFAS. These statistics are taken from Eurostat and, in the case of car washes, from the industry trade association. The numbers in column 1 represent small-sized manufacturers with less than 250 employees, while column 2 represent medium-sized-manufacturers with more than 250 employees.

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<sup>126</sup> The European Commission-DG Enterprise and Industry (2010). Analysis of the risks arising from the industrial use of Perfluorooctanoic acid (PROA) and Ammonium Perfluorooctanoate (APFO) and from their use in consumer articles.

<sup>127</sup> Kissa E (2001). Fluorinated Surfactants and Repellents. Surfactant Science Series. 97. Marcel Dekker, New York.

<sup>128</sup> Eurostat Annual enterprise statistics by size class for special aggregates of activities (NACE Rev. 2) [sbs\_sc\_sca\_r2].

<sup>129</sup> Figures for CZ, IE, EL and SK are missing for enterprises with 250 or more employees.

<sup>130</sup> Sullivan M, Car wash cites for PFAS pollution, Sea Coast Online, June 4 2018. Accessed 19 August 2018.

<sup>131</sup> International Car Wash Association. Industry Information.

**Table 11: Number of manufacturers carrying out activities that may have involved use of PFAS**

Industrial activity	Manufacturers <250 employees	Manufacturers >250 employees
Textiles	61,685	262
Leather	37,120	159
Carpets	no data	-
Paper	19,477	488
Paints and varnishes	4,027	104
Cleaning products	-	178
Metal treatments	151,455	163
Car washes	79,000	-
Plastic, resins, rubbers	-	340
Totals	352,764	1694
3% of total	10,583	51
10% of total	35,276	169

It is not known how many of the products manufactured by these companies that have been produced or treated with PFAS, or how many commercial service industries that are using products containing PFAS. In the absence of more data, it was assumed that between 3% and 10% of the manufacturers in these industries have used or are using PFAS in the manufacturing process or in the commercial service provided. These activities would have the potential for releasing PFAS to air, water and soil during the manufacturing process as well as afterwards, in the form of industrial waste.

Note that these numbers do not include manufacturers from other industrial activities known to use PFAS, such as makers of electronic chips, cosmetics and personal care products, photography films and mineral extraction. Note also that non-fluorinated alternatives are available for some of these applications, e.g., the consumer textile industry.

### 4.3 Case Study 3: Contamination from use of aqueous film-forming foams

#### 4.3.1 Background and context

Aqueous film-forming foams (AFFFs), a specific sub-type of firefighting foam<sup>132</sup>, are one of the many industrial products that contain PFAS. PFAS-containing AFFFs were found to be particularly useful in extinguishing petroleum-based fires (also known as class B liquid fires).<sup>133</sup> The surfactant properties of the PFAS serve to form a coating or blanket that deprives the fire of oxygen until the flames die out. Because of their

<sup>132</sup> Norden (2013). Per- and polyfluorinated substances in the Nordic Countries: Use, occurrence and toxicology. TemaNord 2013:542.

<sup>133</sup> Swedish Environmental Institute (2015). Risks and Effects of the dispersion of PFAS on Aquatic, Terrestrial and Human populations in the vicinity of International Airports: Final Report of the RE-PATH project. p.17.

effectiveness, they have been widely used around the world since the 1960s<sup>134</sup> both at airports and at fire training facilities.

The biggest concern related to the use of AFFFs is the contamination of drinking water sources.<sup>135</sup> This concern is also related to long-lasting impacts of these substances in the water. For instance, PFOAs are said to have a hydrolytic half-life of more than 97 years.<sup>136</sup> Contamination of the environment due to AFFFs might take place in different ways in specific locations due to local, complex interactions within the ecosystems. Various exposure pathways have been identified.<sup>137</sup>

The hazardous substances in the AFFFs usually find their way to surface waters through direct runoff, in ground waters through infiltration and in soil through soil diffusion or dispersion. Drinking water can be contaminated as a result of ground water and surface water contamination.<sup>138</sup> Groundwater and surface water contamination can also lead to contamination of agricultural produce through uptake into biota. Contaminated surface waters and biota (fish) can create further health risks for humans and other animals.<sup>139</sup> A Swedish study (“RE-PATH”) investigated contamination around two airports where AFFFs had been used for many years.<sup>140</sup> The results clearly indicated increased levels of PFAS in the surrounding soil, surface/ground water and biota – up to 100 times higher than in control areas.

Water pollution (whether it is groundwater or surface waters) related to the use of AFFFs following a fire, accidental leaks or fire-fighting trainings is considered to be severe.<sup>141</sup> Cases of groundwater, soil and surface water contamination have been documented near airports, military bases and fire drill sites in Germany, the Scandinavian countries<sup>142</sup>, the UK and others. One report suggests that *many classes of PFAS are observed in groundwater, essentially every AFFF impacted site investigated to date.*<sup>143</sup>

As the environmental and health risks of PFAS became apparent in the 1980s, the role of AFFFs in PFAS-related environmental contamination started to draw attention. In Europe, AFFFs containing PFOS were banned in 2006 with a complete phase-out in

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<sup>134</sup> Kärrman A *et al.* (2011). Environmental levels and distribution of structural isomers of perfluoroalkyl acids after aqueous fire-fighting foam (AFFF) contamination. *Environ. Chem.*, 8, 372–380.

<sup>135</sup> Field, J *et al.* (2017). FAQs Regarding PFAS Associated with AFFF Use at U.S. Military Sites, Report for Environmental Security Technology Certification Program (ESTCP).

<sup>136</sup> Franko *et al.* (2012). Dermal Penetration potential of Perfluorooctanoic Acid (PFOA) in Human and Mouse Skin, *Journal of Toxicology and Environmental Health, Part A*. 75:50–62.

<sup>137</sup> The National Academies of Science Engineering and Medicine (2017). Use and Potential Impacts of AFFF Containing PFAS at Airports.

<sup>138</sup> Interstate Technology Regulatory Council (no date). Environmental Fate and Transport for Per- and Polyfluoroalkyl Substances.

<sup>139</sup> The National Academies of Science, Engineering, Medicine (2017). Use and Potential Impacts of AFFF Containing PFAS at Airports.

<sup>140</sup> Swedish Environmental Protection Agency (2015). Risks and Effects of the dispersion of PFAS on Aquatic, Terrestrial and Human populations in the vicinity of International Airports: Final Report of the RE-PATH project. Report number: B 2232.

<sup>141</sup> Eschauzier C *et al.* (2012). Polyfluorinated Chemicals in European Surface Waters, Ground and Drinking Waters. In: Knepper T., Lange F. (eds) *Polyfluorinated Chemicals and Transformation Products. The Handbook of Environmental Chemistry*, vol 17. Springer, Berlin, Heidelberg.

<sup>142</sup> Danish Environmental Protection Agency (2014). Screeningsundersøgelse af udvalgte PFASforbindelser som jord- og grundvandsforurening i forbindelse med punktkilder (In Danish).

<sup>143</sup> Field J *et al.* (Report for Environmental Security Technology Certification Program-ESTCP) (2017). FAQs Regarding PFASs Associated with AFFF Use at U.S. Military Sites. p:8.

2011.<sup>144</sup> Starting from 2020, the use of AFFFs containing more than 25 ppb of PFOA or its salts as well as those containing more than 1000 ppb of one or a combination of PFOA related substances will be restricted within the EU.<sup>145</sup> The restrictions will not apply to AFFFs placed on the market before July 2020.<sup>146</sup>

The new generation of AFFFs may still contain shorter chain PFAS.<sup>147</sup> However, only limited data is available on the chemical composition of these newer generation AFFFs, previous studies found high concentrations of PFHxA, C6 and 6:2 FTS in the products.<sup>148</sup> Little information is also available for the impact of these fluorinated replacement substances on the environment and human health. Furthermore, because the older generation PFAS (such as PFOS) are highly toxic and persistent; the impacts of the historical releases continue to present serious risk to human health and to the environment. On the positive side, high-performance non-fluorinated AFFFs have been developed and are now on the market.<sup>149</sup>

#### 4.3.2 Cases of contamination

##### Kallinge-Ronneby Military and Civilian Airbase

Between 1980 and 2003, AFFFs containing PFOS were used in Sweden. In 2003, these were replaced a new type containing other PFAS but old stocks containing PFOS continued to be used.<sup>150</sup> Swedish airports and Swedish armed forces started using a fluorine-free alternative to fluorine-based fire-fighting foams as of June 2011.<sup>151</sup>

During a 2013 groundwater quality survey, high concentrations of PFAS were detected in the Bredåkra delta (Ronneby). The testing was expanded to a larger area, confirming the PFAS contamination in the outgoing water from one of the two municipal waterworks, Brantafors, which supplied water to around 5,000 people.<sup>152</sup>

Table 12 presents the levels for some of the PFAS compounds detected in Brantafors compared to Kärragården, the second source of drinking water in the municipality. It can be seen that PFHxS, PFOA and PFOS were sometimes 100–300

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<sup>144</sup> Swedish Chemicals Agency (2015). Survey of Fire Fighting Foam.

<sup>145</sup> Commission Regulation (EU) 2017/1000 of 13 June 2017 amending Annex XVII to Regulation (EC) No 1907/2006 of the European Parliament and of the Council concerning the Registration, Evaluation, Authorisation and Restriction of Chemicals (REACH) as regards perfluorooctanoic acid (PFOA), its salts and PFOA-related substances.

<sup>146</sup> Ibid.

<sup>147</sup> Swedish Environmental Protection Agency (2015). Risks and Effects of the dispersion of PFAS on Aquatic, Terrestrial and Human populations in the vicinity of International Airports: Final Report of the RE-PATH project. Report number: B 2232.

<sup>148</sup> Swedish Chemicals Agency (2014). Chemical Analysis of Selected Fire-fighting Foams on the Swedish Market.

<sup>149</sup> IPEN (2018). Fluorine-free firefighting foams (3f) viable alternatives to fluorinated aqueous film-forming foams (AFFF).

<sup>150</sup> Swedish Environmental Protection Agency (2015). Risks and Effects of the dispersion of PFAS on Aquatic, Terrestrial and Human populations in the vicinity of International Airports: Final Report of the RE-PATH project. Report number: B 2232.

<sup>151</sup> Swedish Chemicals Agency (2015). Occurrence and use of highly fluorinated substances and alternatives: Report from a government assignment. Report 7/15.

<sup>152</sup> Ronneby Municipality Website, page dedicated to PFAS contamination, available here.

times higher in the contaminated water source. Given that firefighting foams containing PFOS were phased out between 2003 and 2008<sup>153</sup>, these highly elevated levels underline the persistent nature of PFAS.<sup>154</sup>

**Table 12: PFAS levels (ng/l) in outgoing drinking water from the waterworks in Ronneby, Sweden on Dec 10, 2013**

	Brantafors	Kärragården	
PFPeA (Perfluoropentanoic acid)	38	10	The Swedish National Food Agency sets the recommended action level of 90 ng/l for a combined sum for 11 different PFAS, highlighted in red in the table.
PFHxA (Perfluorohexanoic acid)	320	3.6	
PFHpA (Perfluoroheptanoic acid)	32	1.4	
PFOA (Perfluorooctanoic acid)	100	1	
PFBS (Perfluorobutane sulfonic acid)	130	<2.6	
PFHxS (Perfluorohexane sulfonic acid)	1700	4.6	
PFOS (Perfluorooctane sulfonic acid)	8000	27	
PFHpS (Perfluoroheptane sulfonic acid)	60	<1	

Source: University of Goteborg, The Sahlgrenska Academy of Institute of Medicine (Li Ying *et al.*), Technical Report: Half-lives of PFOS, PFHxS and PFOA after end of exposure to contaminated drinking water, 2017.

The source of the contamination was identified as the fire drill site located in the nearby military airport.<sup>155</sup> The contamination is estimated to have started in the mid-1980s.<sup>156</sup> The quantity released into the environment is unknown. The PFAS leaked from the site to the surrounding area into soil, eventually reaching the ground waters. The contaminated water source was equipped with a carbon filter at the time and according to the authorities, this filtering system might have reduced contamination of the drinking water, until it became saturated.<sup>157</sup>

Brantafors waterworks was closed right after the contamination was detected, and reopened a year later in 2014 after being equipped with charcoal filters and using only two of its four abstraction points (the remaining two having too high levels of PFAS). The water was monitored until October 2014, when the PFAS levels showed an upgoing trend which led to the re-closing of the waterwork, despite levels never exceeding the Swedish recommended action level of 90 ng/l. To secure drinking water supply, new pipes were built to provide Brantafors with uncontaminated water from the area Karlsnäs.

Biomonitoring between 2014 and 2016 resulted in testing of 3418 persons to determine exposure to PFAS from the drinking water. In addition, a smaller sub-sample of 106 individuals was created for a panel study to estimate half-life of the substances. These individuals gave regular blood samples and the monitoring will continue in the

<sup>153</sup> Banzhaf S *et al.* (2016). A review of contamination of surface-, ground-, and drinking water in Sweden by perfluoroalkyl and polyfluoroalkyl substances (PFASs). The Royal Swedish Academy of Sciences. p.337.

<sup>154</sup> Li Ying *et al.* (2017). Technical Report: Half-lives of PFOS, PFHxS and PFOA after end of exposure to contaminated drinking water.

<sup>155</sup> Swedish Chemicals Agency (2015). Survey of Fire Fighting Foam.

<sup>156</sup> Li Ying *et al.* (2017). Technical Report: Half-lives of PFOS, PFHxS and PFOA after end of exposure to contaminated drinking water.

<sup>157</sup> Ronneby Municipality Website, page dedicated to PFAS contamination, available here.

coming years.<sup>158</sup> The concentrations for the Ronneby main group and for the sub-group were much higher when compared to the reference group, a sample of 242 people living in a nearby community not affected by the contamination, see Table 13.

**Table 13: Median levels for PFAS concentrations in ng/ml in serum samples, tested 6th months after end of exposure**

Contaminants	Reference Population (n=242)	Main Ronneby Group (n=3418)	Panel Study Group (n= 106)
PFHxS	0.84	152	277
PFOA	1.59	10.4	17.5
PFOS	4.21	176	345

Source: University of Goteborg, The Sahlgrenska Academy of Institute of Medicine (Li Ying *et al.*), Technical Report: Half-lives of PFOS, PFHxS and PFOA after end of exposure to contaminated drinking water, 2017

Another pilot study was conducted among school children comparing those who had been drinking the contaminated water and those who had not. The children exposed to contamination were found to have 20–50 times higher levels of different PFAS substances, particularly PFOS, PFOA and PFHxS.<sup>159</sup>

Ronneby and other incidents led the Swedish authorities and other responsible parties (like the Swedish Air forces) to conduct national scale monitoring of drinking waters as well as further investigations in known contaminated areas.<sup>160</sup> The Swedish Chemical Agency (KEMI) and the National Food Agency set up a national PFAS network which brings together a wide range of stakeholders to advance existing knowledge on the issue.<sup>161</sup> In one of the few examples of national monitoring of PFAS in the environment, around 6,000 measurements of surface and ground water were compiled (existing data or new measurements) across the county by the Swedish Environmental Protection Agency. All water supplies with contamination levels that exceeded the safety level of 90 ng/l were found to be located close to an individual fire training site or to one located within an airport. The use of fire extinguishing foams was identified as the *largest direct point source* of contamination.<sup>162</sup>

Between 2013 and 2015, new water pipe connections were built between uncontaminated wells in Karlsnäs and Brantafors.<sup>163</sup> The cost of changing the water supply from Brantafors to Karlsnäs is roughly estimated to have cost Ronneby municipality

<sup>158</sup> Li Ying *et al.* (2017). Technical Report: Half-lives of PFOS, PFHxS and PFOA after end of exposure to contaminated drinking water.

<sup>159</sup> Jakobsson K *et al.* (2014). Exponering för perfluorerade ämnen (PFAS) i dricksvatten i Ronneby kommun (In Swedish). Rapport 8:2014.

<sup>160</sup> Banzhaf S *et al.* (2016). A review of contamination of surface-, ground-, and drinking water in Sweden by perfluoroalkyl and polyfluoroalkyl substances (PFASs), The Royal Swedish Academy of Sciences.

<sup>161</sup> Sahlin S (2017). PFAS in the Baltic Sea Region.

<sup>162</sup> Swedish Environmental Protection Agency (2016). Högfluorerade ämnen (PFAS) och bekämpningsmedel en sammantagen bild av förekomsten i miljön. (report in Swedish with English Summary).

<sup>163</sup> Ronneby Municipality (No date). Frågor och svar om PFAS. Accessed August 2018.

SEK 60 million (incl. VAT) (EUR 5.8 million).<sup>164</sup> The additional annual cost for increased monitoring is calculated to around SEK 50,000 (incl VAT) (EUR 4,800).

According to the latest information available, the investigations are in progress as to how to clean up the contaminated soil in Ronneby. The Swedish Armed Forces is leading the process. Significant water resources remain unusable for an unforeseeable future due to PFAS contamination. The loss of these valuable resources has not been monetised, but should nevertheless be taken into account.

### Jersey Civilian Airport, Channel Islands

The Jersey Airport case is one of the earliest and well documented examples in Europe of contamination of groundwater and surrounding areas due to AFFFs. In 1991, the fire training site started using AFFFs to meet the requirements of UK Airport Fire Services.<sup>165</sup> They were regularly using AFFFs until 1993, when foaming water started to emerge from a land drain excavated near the training site.<sup>166</sup> *A brown coloration and substantial foaming* was also identified in a farm's private water supply that was found to be contaminated.<sup>167</sup>

According to the accounts of the airport, 78 properties were within the plume area. Groundwater in 36 of these properties tested positive for PFOS.<sup>168</sup> Although at some of the sites, concentrations of PFOS have shown signs of decline, they have remained at high levels for seven years in private wells.<sup>169</sup> For instance, one private well continued to show high levels of PFOS since the first publicly available recording began in 1999 until 2006, between 2.7 µg/l (2700 ng/l) and 9.5 µg/l (9500 ng/l). In another area where samples were taken, one property had levels as high as 98 µg/l (98,000 ng/l).<sup>170</sup> As regards groundwater, the level detected in a borehole was 96 µg/l (96000 ng/l).<sup>171</sup> The fire-training site was identified as the origin of the contamination. The foam used at the site during training exercises was discharged regularly without monitoring<sup>172</sup>, dissolving into the ground and rainwaters.<sup>173</sup> Contamination subsequently found its way into the St.Ouen's aquifer and the beach of St.Ouen's Bay.<sup>174</sup>

A monitoring program was put in place to regularly test the ground waters for contamination levels, starting from 1994. The impact on agricultural products (potatoes

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<sup>164</sup> Schyberg, I. (2018). Ronneby Miljö och Teknik AB, personal communication.

<sup>165</sup> States of Jersey, States Greffe Logs, Jersey Airport: fireground remediation – Deed of settlement, 19 October 2004. Accessed 08.09.2018.

<sup>166</sup> Foundation for Water Research (2008). Survey of the prevalence of perfluorooctane sulphonate (pfos), perfluorooctanoic acid (pfoa) and related compounds in drinking water and their sources.

<sup>167</sup> States of Jersey, States Greffe Logs, Jersey Airport: fireground remediation – Deed of settlement, 19 October 2004. Accessed 08.09.2018.

<sup>168</sup> Jersey Airport Website dedicated to the PFOS, available here: <http://www.jerseyairport.com/PFOS/Pages/Questions-and-Answers.aspx>

<sup>169</sup> Foundation for Water Research (2008). Survey of the prevalence of perfluorooctane sulphonate (pfos), perfluorooctanoic acid (pfoa) and related compounds in drinking water and their sources.

<sup>170</sup> Ibid.

<sup>171</sup> Ibid.

<sup>172</sup> Robins N S (British Geological Survey)(2000). The water resources of Jersey: an overview.

<sup>173</sup> States of Jersey, States Greffe Logs, Jersey Airport: fireground remediation – Deed of settlement, 19 October 2004. Accessed 08.09.2018.

<sup>174</sup> Foundation for Water Research (2008). Survey of the prevalence of perfluorooctane sulphonate (pfos), perfluorooctanoic acid (pfoa) and related compounds in drinking water and their sources.

and cauliflowers etc.) was also analysed. At the time, the monitoring committee could not find any laboratory to test the relevant substances in Europe and the samples had to be sent to the US. In 1999 the authorities started to conduct their own analysis.<sup>175</sup> The initial response was to inform the residents using the water supplies of the possibility of contamination and to provide the particular farm mentioned above with a new borehole. Domestic water supplies were tested and free bottled water was offered in cases of contamination.<sup>176</sup> In the aftermath of the contamination, Jersey Airport has funded remediation and other related costs.<sup>177</sup> To ensure safe drinking water to the residents in the affected area, 67 among 78 properties in the plume area have been connected to the main water supply.<sup>178</sup>

Long-term remediation works began in 2002 and finished in 2004 with the aim of addressing the current contamination and preventing future problems.<sup>179</sup> The total cost was GBP 7.4 million (EUR 10.6 million). Table 14 presents the main breakdowns.

**Table 14: Details of clean-up costs from the Jersey Civilian Airport**

Expenditure Type	Total – Euros*
Investigation (incl. installing and monitoring boreholes, ongoing sampling and analysis)	1,427,167
Connection to water supplies	208,237
Payments of Water Rates to Jersey Water	523,386
Remedial Works associated to Old Fire Training Ground	446,739
Professional Fees	791,778
Finance Costs	122,282
Jersey Water Mains Connection Costs	808,303
Capital Works – Fire Training Ground	6,292,376
Total	10,620,271

Note The original figures were in British Pounds. They were converted to Euros using the rates from 19 October 2004, the day the Settlement Deed was approved between the parties involved.

Source: The Jersey Airport Website

### Schiphol Airport, The Netherlands

In July 2008, an error in the sprinkler-system at a KLM hangar at Schiphol-Ost released 10,000 liters of aqueous fire-fighting foam, containing 143 kg of PFOS, into the surrounding environment.<sup>180</sup> This fed into a larger reserve of waste water (100 million liters) kept in five reserve reservoirs, several of which leaked and caused substantial contamination of the soil and surface water.<sup>181</sup> A later study found the water resources

<sup>175</sup> Ibid.

<sup>176</sup> States of Jersey, States Greffe Logs, Jersey Airport: fireground remediation – Deed of settlement, 19 October 2004. Accessed 08.09.2018.

<sup>177</sup> According to the Jersey Airport Website, accessible here

<sup>178</sup> Jersey Airport Website dedicated to the PFOS, available here

<sup>179</sup> States of Jersey, States Greffe Logs, Jersey Airport: fireground remediation – Deed of settlement, 19 October 2004. Accessed 08.09.2018.

<sup>180</sup> Brandveilig, Zorgen om verontreiniging door incident met sprinklerinstallatie 07.09.2011 and No Author, Bodemvervuiling bij Schiphol snel aanpakken, De Volkskrant, 12 October 2011. Accessed 09.11.2018.

<sup>181</sup> The Government of Netherlands (2018). Beantwoording Kamervragen over berichten over met PFOS en PFOA verontreinigde grond bij Schiphol. (In Dutch).

to contain over 12 times the average amount of PFOS otherwise found in several reference sites in the Netherlands.<sup>182</sup> Residents were warned in 2008 not to swim in or consume fish from the nearby “Ringvaart” canal until the contaminated waste-water could be drained from the overfull reservoirs.<sup>183</sup>

Contaminated soil from this incident also resulted in delays of over a year to a project to build a new bus lane in Schiphol-Oost in 2017. Over 50,000 m<sup>3</sup> of the soil dug up was found to be contaminated and thus difficult to dispose.<sup>184</sup> The cost of the remediation is estimated at EUR 30–40 million.<sup>185</sup>

### Information on AFFFs releases – data limitations and assumptions

According to the latest Eurostat data from 2015, Europe has a total of 455 civilian airports (28 MS and Iceland, Norway and Switzerland) with a passenger capacity of 15,000 and more per year), among those 318 are considered main airports with 150,000 or more passengers by year.<sup>186</sup>

On-line country by country research found an estimated 239 military airfields in the EEA countries and Switzerland.<sup>187</sup> The numbers are likely to be an underestimate, because they do not contain non-active military bases, which may be historical sources of AFFF contamination. Also, the US military uses firefighting foams during training and emergencies, as well as in automated fire suppression systems.<sup>188</sup> Under US military specifications, AFFFs purchased for use at US military sites must be based on PFAS chemistry to conform to military specifications (MIL-F-24385F).<sup>189</sup> Some military airfields in Europe would be in countries participating in NATO and therefore it is reasonable to assume that fluorinated AFFFs conforming to the US military specification have been used.

Countries have differed in their approaches to addressing problems related to the use of AFFFs. Swedish authorities developed guidance on how to avoid PFAS containing foams and how to handle residues from firefighting foams. The state-owned airport operator Swedavia went fluorine free as of 2008.<sup>190</sup> The Swedish Armed Forces still uses AFFFs, but only in one location (Halmstads Garnison) in which the foam and firewater is collected to avoid emissions to the environment.<sup>191</sup> The aim is to go completely PFAS free as soon as equally good alternatives are available. The Danish Armed Forces and the Royal Danish Air Force no longer uses fluorinated AFFFs, nor do the international airports in Copenhagen and Billund.<sup>192</sup> Only limited information has been found regarding

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<sup>182</sup> Kotterman MJJ and Kwadijk C.J.A.F (2009). PFOS onderzoek in waterbodem en vis. Report No: Co64/09 (in Dutch).

<sup>183</sup> Sportvisserij Netherlands (News) Rijnland hervat waterafvoer Schiphol terrain, 21 July 2008, Accessed: August 2018. (In Dutch).

<sup>184</sup> Boele B, Schiphol Logistics Park blijkt vervuild, Haarlems Dagblad, 03.01.2018. Accessed 02.09.2018.

<sup>185</sup> Schiphol Airport (2017). Annual Report (In Dutch).

<sup>186</sup> Eurostat Transport Statistics [AIRP\_TYP] Accessed 14/06/2018.

<sup>187</sup> These numbers are approximate and do not include all EU-28 MS. Some air fields might be inactive or no-longer in existence.

<sup>188</sup> Field J *et al.* (Report for Environmental Security Technology Certification Program-ESTCP) (2017). FAQs Regarding PFASs Associated with AFFF Use at U.S. Military Sites.

<sup>189</sup> Anderson H *et al.* (2016). Occurrence of select perfluoroalkyl substances at U.S. Air Force aqueous film-forming foam release sites other than fire-training areas: Field-validation of critical fate and transport properties, Chemosphere 150-678e685.

<sup>190</sup> Swedavia Airports (2018). PFAS på Swedavias flygplatser.

<sup>191</sup> Minister of Defence Hultqvist P. (2017) Answer to question on removal of PFAS. Dnr F62017/01275/MFI

<sup>192</sup> IPEN (2018). Fluorine-free firefighting foams (3f) viable alternatives to fluorinated aqueous film-forming foams (AFFF).

whether fluorinated AFFFs are used by municipal rescue services or what types of AFFFs are used at other Danish airports. In Finland, the military no longer uses AFFFs during training and fluorinated AFFFs are prohibited at the fire training areas of airports.<sup>193</sup>

In Norway, the state-owned Avinor (which operates the majority of Norway’s civilian airports) abolished PFOS-containing fire foam in 2001, and went fluorine free in 2011.<sup>194</sup> The state-owned oil company Equinor has also shifted to fluorine free foams.<sup>195</sup> An important usage area remains offshore installations. Offshore platforms accounted for 54 out of 57.6 tonnes of PFOS released in Norway up until 2005 (the amounts released from airports and fire drilling areas on land could not be estimated).<sup>196</sup> The Norwegian offshore sector has now consciously reduced the amount of PFOS-containing foam – from 4 tonnes in 2014 to 1.1 tonne in 2017.<sup>197</sup>

In the UK, Heathrow Airport transitioned to fluorine free foam in 2012.<sup>198</sup> Though Germany has no national restrictions on the use of AFFFs, the Umweltbundesamt (UBA) has published guidelines on the issue and some federal states have set threshold values for PFAS in foams. The number of sites in the EEA where fluorinated AFFFs may have been used are presented in Table 15.

**Table 15: The number of sites in the EEA where fluorinated AFFFs may have been used.**

Sector	Activity	Total
Aviation	Main passenger airports	318
	Medium passenger airports	137
	Small airports	no data
	Military airbases	239
Other fire control	Fire stations	84,099
	Site emergency services	no data

The number of fire stations across Europe is at least 85,000. However, it is not known whether all of these stations have been engaged in training involving AFFFs. The use of AFFFs, i.e., whether it is used during the trainings, accidents or both, might vary from country to country. A survey of AFFFs in Sweden by the Swedish Chemical Agency mentions firefighting training centres in Sweden, which used various types of AFFFs and notes that some types were not used after 2011 because they contained PFOS and PFOA.<sup>199</sup> An inventory carried out in 2017 aimed to locate all non-airport fire-fighting training sites in Norway active within the past 40 years, and identified 249 previously unknown sites.<sup>200</sup>

Another possible source of contamination is release of AFFFs during efforts to extinguish fires. Figures are available for different types of fires across the EU on a yearly

<sup>193</sup> Sahlin S (2017). PFAS in the Baltic Sea Region.

<sup>194</sup> Avinor (2018). PFOS I Focus. (In Norwegian).

<sup>195</sup> IPEN (2018). Fluorine-free firefighting foams (3f) viable alternatives to fluorinated aqueous film-forming foams (AFFF).

<sup>196</sup> Climate and Pollution Agency (2012). Inventory of PFOS and PFOS-related substances in fire-fighting foams in Norway.

<sup>197</sup> A. Heggelund, Norwegian EPA (2018) Personal communication.

<sup>198</sup> Ibid.

<sup>199</sup> Swedish Chemicals Agency (2015). Survey of Fire Fighting Foam. Report 5/15.

<sup>200</sup> Heggelund, A. Norwegian EPA. Personal communication, Sep 2018.

basis (latest data from 2015) but AFFFs are specifically used to extinguish class B fires (fires involving gasoline, petroleum greases, tars, oils, oil-based paints, solvents, alcohols) and the number of class B fires could not be separated out from the total number of fires. Fluorinated AFFFs may also be present in fire suppression systems in place in industrial facilities where flammable and explosive substances are stored or used.

Some studies have tried to estimate the total amounts of AFFFs found in the market (sold, or in stocks of manufacturers as well as potential users like airports, firefighting training centres or petroleum refineries). For instance, a report from 2009 estimated the quantities of PFOS containing AFFFs sold in the Dutch market in 2002 as 212,000 l and concluded that around 25 million l of foam have been placed in the Dutch market in 20 years. Some 75% of these stocks were estimated as still unused. Around 18% of the stocks were thought to have been sold to the aviation industry, and another 11% sold to the fire prevention/protection industry.<sup>201</sup> Another study from 2008 estimated that in order to manufacture AFFFs between 1970 and 2002, 10,000 tonnes of POSF were produced/used. The amounts released into air/water were 9150 tonnes of POSF and between 91 to 460 tonnes of PFOS.<sup>202</sup> A more recent study estimated that between 1.13 to 3.81 tonnes of firefighting foam (monomers) is used annually in the EU.<sup>203</sup>

A recent US study identified 40 novel groups of anionic, zwitterionic and cationic PFAS that had been never observed before, using non-target screening of groundwater from the areas near 13 firefighting training sites. Water samples were collected between 2011 and 2015. Their chemical composition suggested that they were either produced via electrochemical fluorination (a process of manufacturing of AFFFs by 3M which was phased out in 2002) or were compounds found in AFFFs prior to 1988, for which the composition is not known.<sup>204</sup> It is therefore reasonable to assume that both previous and new generation AFFFs will continue to be a major source of pollution into the future.<sup>205</sup>

#### 4.4 Case Study 4: Exposures during the use phase of PFAS-treated products

In order to have an overall view of the long-term costs of inaction, it is important to keep in mind the impact pathways of exposure from those products during the use phase of the PFAS life cycle. A 2000 3M study estimated that 85% of the indirect emissions of POSFs result from losses during the use and disposal stages.<sup>206</sup>

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<sup>201</sup>RIVM (2009). Estimation of emissions and exposures to PFOS used in industry. Report 601780002/2009.

<sup>202</sup>Alexandre G. Paul *et al.* (2008). A First Global Production, Emission, And Environmental Inventory For Perfluorooctane Sulfonate, *Environmental Science & Technology* 2009 43 (2), 386-392.

<sup>203</sup>Ministry of Infrastructure and Environment of Netherlands and Public Waste Agency of Flanders (2016). Inventory of awareness, approaches and policy: Insight in emerging contaminants in Europe.

<sup>204</sup>Krista A. Barzen-Hanson *et al.* (2017). Discovery of 40 classes of per- and polyfluoroalkyl substances in historical aqueous film-forming foams (AFFFs) and AFFF-impacted groundwater, *Environmental Science and Technology*.

<sup>205</sup>Banzhaf S *et al.* (2016). A review of contamination of surface-, ground-, and drinking water in Sweden by perfluoroalkyl and polyfluoroalkyl substances (PFASs), *The Royal Swedish Academy of Sciences*.

<sup>206</sup>Alexandre G. Paul *et al.* (2008). A First Global Production, Emission, And Environmental Inventory For Perfluorooctane Sulfonate. *Environmental Science & Technology* 2009 43 (2), 386-392.

A 2015 study by KEMI provides a useful compendium of PFAS in products<sup>207</sup> and lists a number of areas of application (see Table 16):

**Table 16: Products and areas of applications for PFAS**

Textiles and leather, including impregnating agents
Paper- and food-packaging
Fire-fighting foam
Cosmetic products
Household products
Paint, printing ink and lacquer
Cleaning agents and polish
Non-stick products
Ski wax
Hard- and decorative chrome plating
Hydraulic systems in the aviation industry
Photographic and electronic equipment and components
Photographic surface layers
Photoresistors and anti-reflective coatings for semiconductors
Synthesis chemicals (intermediates)
Medical devices
Building materials
Oil and mining production
Plant protection agents

For many of the applications above, the primary pathways of exposure will occur during the manufacturing of the product or at end-of-product disposal. For example, the application of PFAS as an anti-vapour suppressant during chrome plating will result in releases during the manufacture of the chrome-plated product, and virtually no PFAS will be released when the final product is being used or disposed of at end of life. The use of PFAS in manufacture of medical devices will similarly have the greatest impact during the manufacturing and disposal of the product. These impact pathways are addressed in Case Study 2 on manufacturing and Case Study 4 on end-of-life disposal.

However, for other products, the use phase will also have significant impacts on people and the environment, e.g., the use of PFAS in cosmetic products will have environmental impacts when the product is washed off by the user and the PFAS enters sewers, sewage treatment plants and eventually waterways. The laundering and re-impregnation of textiles and cleaning of leather treated with PFAS and the use of PFAS surfactants in cleaning agents or as agents in pharmaceuticals will likewise result in releases of PFAS to sewers and waterways.

This case study focuses on three categories of products containing PFAS where the use of the product leads to human exposure and to releases to the environment, where they then accumulate.

<sup>207</sup>Swedish Chemicals Agency (2015). Occurrence and use of highly fluorinated substances and alternatives: Report from a government assignment. Report 7/15.

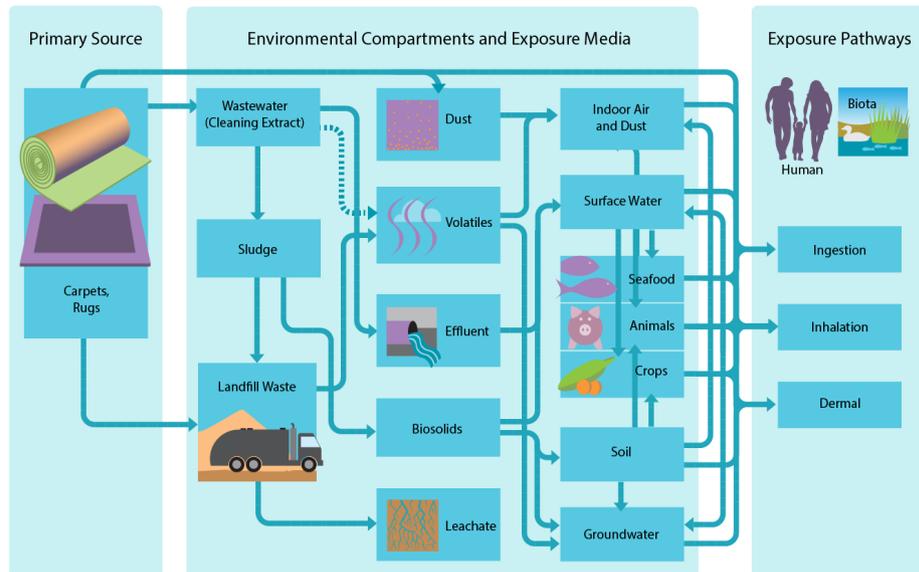
- PFAS-treated carpets
- PFAS-treated food contact materials
- cosmetic products containing PFAS.

It concludes with a look at how PFAS from both domestic and industrial sources will be collected into sewers and then channelled to urban waste water treatment plants where they end up in the wastewater discharges and sewage sludge.

#### 4.4.1 Product 1 – PFAS-treated carpets

PFAS have been used to give stain, soil and oil resistance to carpets and rugs since the 1980s.<sup>208</sup> PFAS can be applied to carpets at four different points: (1) during the manufacture of fibers for carpets, (2) during the process of manufacturing carpets and rugs, (3) at a separate finishing facility after the carpet or rug has been manufactured, or (4) post-manufacture and sale through treatment by the consumer or professional cleaner.<sup>209</sup> Figure 4 show impact pathways for PFAS in carpets.

Figure 4: Impact pathways for PFAS in carpets



Source: From CA DTSC, 2018.

PFAS used to treat carpets and rugs is a major source of human exposure. Surface scuffing during normal use can release PFAS on fibers in the form of tiny particulates which can be resuspended and become part of indoor air and settle into dust. Human exposure takes place when these fine particulates are inhaled or when household or office

<sup>208</sup> California Department of Toxic Substances Control (CADTSC) (2018). Product – Chemical Profile for Perfluoroalkyl and Polyfluoroalkyl Substances (PFASs) in Carpets and Rugs (Discussion draft).

<sup>209</sup> Note that stain-resistant sprays are also sold to consumers for treating upholstered furniture and other textiles.

dust is ingested. Exposure from treated carpets via ingestion is significantly higher in toddlers than adults because of greater hand-to-mouth behaviour.<sup>210</sup> Under one scenario, this pathway was estimated to contribute 40–60% of total uptake of PFAS in infants (0–1 years), toddlers (1–4 years), and children (5–11 years). Other vulnerable populations include industrial workers, carpet installers, carpet cleaners and workers in stores selling furnishings, carpets and outdoor clothing.<sup>211</sup>

Most commercial and residential carpets sold in the U.S. are treated with PFAS, especially carpets made of synthetic materials like nylon, polypropylene, acrylic and polyester, which are prone to absorbing liquids.<sup>212</sup> A 2000 study commissioned by 3M (a producer of PFAS treatments for carpets) estimated loss of PFAS from carpets at 50 percent over a typical lifespan of nine years, due to walking and vacuuming of the carpet.<sup>213</sup> After 2000, improvements made by PFAS-producers and carpet manufacturers considerably reduced the loss of fluorinated treatments.

After the US, the European Union is the second-biggest market in the world for carpets (both treated and untreated).<sup>214</sup> Belgium, the Netherlands and the United Kingdom are the leading countries where carpet manufacturing takes place. Some 1.6 million tonnes of carpet are disposed of in the EU each year. Most of this ends up being incinerated or deposited in landfills. Less than 3% of carpets marketed in the EU are subsequently recycled.

Side-chained fluorinated polymers are now the most commonly used carpet and rug PFAS treatment in the U.S. and in Europe. However, longer-chain PFAS continue to be manufactured in China, Russia and India, and carpets imported from those countries as well as carpets made from recycled materials may still contain long-chain PFAS.

A Danish survey of rugs marketed for children found PFAS in five of the 21 carpets screened for total-fluorine in the textile surface.<sup>215</sup>

#### 4.4.2 Product 2 – Food contact materials treated with PFAS

PFAS are used in the paper industry for producing papers that resist grease and water. These are used for manufacturing food packaging, e.g. plates, popcorn bags, and

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<sup>210</sup>Trudel D *et al.* (2008). Estimating Consumer Exposure to PFOS and PFOA. *Risk Analysis: An International Journal* 28.2: 251–269.

<sup>211</sup> California Department of Toxic Substances Control (CADTSC) (2018). Product – Chemical Profile for Perfluoroalkyl and Polyfluoroalkyl Substances (PFASs) in Carpets and Rugs (Discussion draft).

<sup>212</sup> California Department of Toxic Substances Control (CADTSC) (2018). Product – Chemical Profile for Perfluoroalkyl and Polyfluoroalkyl Substances (PFASs) in Carpets and Rugs (Discussion draft).

<sup>213</sup> Battelle Memorial Institute 2000, as cited in CA DTSC, 2018.

<sup>214</sup> Onyshko, J and Hewlett R (Anthesis Consulting Group) (2018). Toxics in carpet in the European Union.

<sup>215</sup> Danish Environmental Protection Agency (2016). Survey and risk assessment of chemical substances in rugs for children.

other packaging materials, e.g., cartons, masking tape.<sup>216</sup> The end product may contain 1–1.5% PFAS by weight.<sup>217</sup> PFAS in food packaging can leach into food, increasing dietary exposure.<sup>218</sup>

Information on PFAS in paper- and food-packaging is hard to come by as it can be regarded as confidential business information. However, a recent U.S. study of food contact materials (FCM) used in fast food restaurants found detectable fluorine in 46% of food contact papers and 20% of paperboard samples analysed of the 400 samples analysed for total organofluorine content.<sup>219</sup> The presence of fluorinated chemicals in fast food packaging was seen as indicative of FCMs being a significant source of dietary PFAS exposure and environmental contamination.<sup>220</sup>

Similar levels of PFAS were found in fast food packaging gathered in Europe in 2017 and tested by the Danish Consumer Council's Think Chemicals programme.<sup>221</sup>

The use of PFAS in compostable food packaging might present an additional source of contamination for soil and vegetation.<sup>222</sup> Contamination of composts from PFAS and related substances has been documented.<sup>223</sup>

#### 4.4.3 Product 3 – Cosmetic products containing PFAS

PFAS are used in various cosmetic and hygiene products such as sun screens, body lotions, shaving creams, dental floss and a variety of make-up products (e. g. lipsticks, eyeshadows).<sup>224, 225</sup> The use of PFAS in cosmetics is related to their surfactant qualities which help with the penetration of the products through the skin (such as creams) or their capacity to make the skin brighter and absorb<sup>226</sup> more oxygen. They are also used to make the cosmetic products oil- and water-repellent, and weather resistant.<sup>227, 228</sup>

Compared to other uses of PFAS such as in fire-fighting foams, research in the area of PFAS in cosmetics remains limited.<sup>229</sup> Information on the quantities of PFAS used in

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<sup>216</sup> Trier X *et al.* (2011). Polyfluorinated surfactants (PFS) in paper and board coatings for food packaging. *Environ Sci Pollut Res Int* 18(7):1108–1120. DOI: <https://doi.org/10.1007/s11356-010-0439-3>

<sup>217</sup> UNEP/POPS/POPRC.9/INF/11 2013.

<sup>218</sup> Begley TH *et al.* (2008). Migration of fluorochemical-paper additives from food-contact paper into foods and food simulants. *Food Additive and Contaminants: Part A* 25(3):384–390.

<sup>219</sup> Laurel A *et al.* (2017). Fluorinated Compounds in U.S. Fast Food Packaging. *Environmental Science & Technology Letters*.

<sup>220</sup> Tittlemier SA *et al.* (2007). Dietary exposure of Canadians to perfluorinated carboxylates and perfluorooctane sulfonate via consumption of meat, fish, fast foods, and food items prepared in their packaging. *Journal of Agricultural and Food Chemistry*. 55(8):3203–3210.

<sup>221</sup> Chemical Watch (10 March 2017). "High levels of fluorinated substances found in EU fast food packaging."

<sup>222</sup> Center for Environmental Health (2018). Avoiding Hidden Hazards.

<sup>223</sup> Fuchs Jacques G (FiBL) (2008). Compost and digestate: sustainability, benefits, impacts for the environment and for plant production.

<sup>224</sup> Fujii *et al.* (2013). Occurrence of perfluorinated carboxylic acids (PFCAs) in personal care products and compounding agents. *Chemosphere* 93 (2013) 538–544.

<sup>225</sup> Danish EPA. Risk Assessment of fluorinated substances in cosmetic products (Not yet published).

<sup>226</sup> *Ibid.*

<sup>227</sup> Fujii *et al.* (2013). Occurrence of perfluorinated carboxylic acids (PFCAs) in personal care products and compounding agents. *Chemosphere* 93 (2013) 538–544.

<sup>228</sup> Danish EPA. Risk Assessment of fluorinated substances in cosmetic products (Not yet published).

<sup>229</sup> Fischer S *et al.* (2016). Poly- and perfluoroalkyl substances on the market and in the Swedish environment. *Norman Bulletin Issue 5*.

cosmetic manufacturing is not available and cosmetic companies usually do not disclose information on the fluorine content.<sup>230</sup> The current body of knowledge about PFAS in cosmetics mostly stems from individual studies that surveyed a sample of products in the market to identify different types of PFAS in their composition.

Exposure to toxic substances in cosmetics can occur directly from the skin. For instance, a number of studies carried out on mice and human skin suggest that PFOA penetrates both human and mice skin.<sup>231</sup> Absorption of these chemicals through skin may not be a significant route of exposure, but absorption can increase when used on or around the eyes, posing a greater hazard.<sup>232</sup> Although the research in this area remains very limited, it has been suggested that significant variations in absorption may occur depending on the type of PFAS used in the products, and the other chemicals present.<sup>233</sup>

One of the PFAS identified in cosmetics is PTFE, known more widely under the brand name Teflon. The Skin Deep® database compiled and maintained by the U.S.-based Environmental Working Group provides ingredient lists and safety ratings for almost 75,000 cosmetics and personal care products.<sup>234</sup> Teflon was found in 66 different products from 15 different brands. In all, 13 different PFAS chemicals were found in nearly 200 products from 28 brands, including shampoo and shaving cream.

CosIng – the European Commission's public database on substances that may be found in cosmetic products – lists 76 PFAS.<sup>235</sup> Their technical functions lend them to a broad range of uses, such as anticaking agents, emulsifiers, anti-statics, emulsion stabilizers, surfactants, film formers, viscosity regulators and solvents. Many of the products are for use on the skin and hair. Chemicals used in personal cosmetics include perfluoropolyethers (PFPE), PFOA and perfluorodecaline.<sup>236</sup>

A survey of a wide range of consumer products by the Nordic Council of Ministers published in 2017 found concentrations of TOF (total organic fluorine) over the limits of detection for many of the products. The highest concentrations were detected in two dental floss samples (the only personal care products included in the sample). They both showed a 310,000,000 µg/m<sup>2</sup> for TOF and 3 µg/m<sup>2</sup> and 19.5 µg/m<sup>2</sup> concentrations for PFAS respectively.<sup>237</sup> Table 17 shows the concentrations of different PFAS detected.

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<sup>230</sup> Ibid.

<sup>231</sup> Franko *et al.* (2012). Dermal Penetration potential of Perfluorooctanoic Acid (PFOA) in Human and Mouse Skin. *Journal of Toxicology and Environmental Health, Part A*. 75:50–62.

<sup>232</sup> EWG Cosmetics Database, Is Teflon in your Cosmetics? Published March 14, 2018. Accessed 08.08.2018.

<sup>233</sup> Ibid.

<sup>234</sup> EWG Cosmetics Database, Is Teflon in your Cosmetics? Published March 14, 2018. Accessed 08.08.2018.

<sup>235</sup> Swedish Chemicals Agency (2015). Occurrence and use of highly fluorinated substances and alternatives: Report from a government assignment. Report 7/15.

<sup>236</sup> Jamberg U and Holmstrom, K (2007). Perfluoroalkylated acids and related compounds (PFAS) in the Swedish environment.

<sup>237</sup> Nordic Council of Ministers (2017). Analysis of PFASs and TOF in products.

**Table 17: PFCA concentrations (µg/kg) detected in dental floss samples**

	PFBA	PFHxA	PFHpA	PFOA	PFNA	PFDA	PFUnD A	PFDoD A	PFTriA	PFTeD A
LOD <sup>238</sup>	0.05	0.10	0.06	0.06	0.08	0.08	0.08	0.05	0.05	0.09
Dental floss 1	< LOD	< LOD	< LOD	0.104	< LOD	< LOD	< LOD	< LOD	< LOD	< LOD
Dental floss 2	< LOD	< LOD	3.47	13.1	< LOD	< LOD	< LOD	< LOD	< LOD	< LOD
LOD	0.05	0.10	0.06	0.06	0.08	0.08	0.08	0.05	0.05	0.09

Source: Nordic Council of Ministers (2017). Analysis of PFASs and TOF in products.

A study carried out for a master's thesis at Lund University in 2017 investigated cosmetic products to identify PFAS. Products were grouped into larger categories as sunscreen, moisturizing creams, and foundation and eye make-up. PFAS were detected in 59 products among the 1354 surveyed (4.4% of total). The brands that had products for which tests showed PFAS content included L'Oréal (4 out of 41 products tested), IsaDora (8 out of 94), The Body Shop (18 out of 98), Maybelline (6 out of 72), Biotherm (1 out of 45), Lumene (22 out of 43). Large variations of PFAS concentrations among different brands were found.<sup>239</sup>

The Danish Environment Protection Agency carried out an ingredients survey based on the information found in the database of Kemiluppen app. The Kemiluppen app enables consumers to scan the barcodes of cosmetic products in Denmark, which are then assessed by the Danish Consumer Council. The survey evaluated 11,108 products that had been scanned (some products may have been scanned multiple times) and found 78 (in 20 different types of products) with fluoroalkyl substance or other fluorinated compounds listed as contents. It was also possible to gauge a product's market size, based on how many times consumers had scanned a particular product. PTFE (polytetrafluoroethylene) was present in 13 types of products that were scanned 16,641 times. C9-15 fluoroalcohol phosphate was present in four types of products that were scanned 7,826 times. Creams and lotions contained the highest number of fluoroalkyl substances (six), followed by BB/CC creams and foundations (three in each)

Another study conducted by the US Breast Cancer Fund found PFOS concentrations above detection levels in six out of 17 products tested, mainly anti-aging creams, moisturizers and skin powders, all belonging to the biggest manufacturers such as L'Oréal and Proctor & Gamble.<sup>240</sup> Perfluorinated compounds have also been found in hair and skin conditioners.<sup>241</sup>

<sup>238</sup> LOD stands for level of detection.

<sup>239</sup> Henricsson C (Master Thesis) (2017). The Presence of PFAS in Cosmetic Products (Förekomst av PFAS i kosmetiska produkter). University of Lund.

<sup>240</sup> Breast Cancer Fund (2015). Anti-aging secrets exposed: chemical linked to breast cancer found in skin care.

<sup>241</sup> Nordic Ecolabelling (2018). About Nordic SAWN Ecolabelled Cosmetic Products. Version 3.3.

A campaign launched by a Swedish NGO in 2017 has resulted in six global cosmetic companies pledging to phase out the use of PFAS in their products.<sup>242</sup>

The use of personal care products leads to the products, after bathing, entering into sewerage and wastewater treatment plants. This type of contamination has received little attention, but in the case of products such as shampoos and shaving creams, it might be highly relevant. For example, one study found PFCA in 89% of the sunscreens surveyed.<sup>243</sup> This suggests that these products are potential contaminants for the aquatic environment.

The research carried out for this study was not able to find information on the quantities of PFAS used in manufacturing cosmetics. It is therefore not possible to provide an overall estimate of the scope of the problem. Though data is available on the concentrations of certain PFAS in the groundwater and drinking water systems, how much of this contamination stems from personal care products is unknown.

#### 4.4.4 Discharges from waste water treatment plants

Conventional wastewater treatment is not effective in removing PFAS from waste streams.<sup>244</sup> Thus municipal wastewater treatment plants (WWTP) are major point sources for PFAS contamination of the aquatic environment.<sup>245</sup> The release of PFAS to the biosphere through WWTPs can be a result of both industrial activities and their domestic usage.<sup>246</sup> Given the extreme persistence of PFAS, these substances may end up in sewage sludge applied to agricultural land and subsequently taken up into produce for human consumption. One study found that

“Often PFAS concentrations increase in wastewater treatment plants as a result of biodegradation of precursors during the activated sludge process. PFOA is generally fully discharged into receiving rivers, while about half of PFOS is retained in the sewage sludge.”<sup>247</sup>

Other studies have confirmed this. A 2016 study for the Swedish Environmental Protection Agency concluded that the main transport route of PFAS from societal use of goods and chemical products into the environment was via sewage treatment plants and waste management facilities, which could constitute locally significant point sources. PFAS-containing sewage from industry could also enter the environment via sewage treatment and waste management facilities. For Sweden, emissions of PFAS into the environment

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<sup>242</sup> H&M, L’Oreal, Lumene, Body Shop, Isadora and Kicks. See: Chemical Watch: Cosmetics giant L’Oréal to eliminate PFASs in products, July 2018.

<sup>243</sup> Fujii *et al.* (2013). Occurrence of perfluorinated carboxylic acids (PFCAs) in personal care products and compounding agents. *Chemosphere* 93 (2013) 538–544.

<sup>244</sup> Adler, A and Van der Voet J (2015). Occurrence and point source characterization of perfluoroalkyl acids in sewage sludge, *Chemosphere* 129: 62–73.

<sup>245</sup> Ahrens L *et al.* (2016). Screening of PFASs in groundwater and surface water, Uppsala Report No: 2016:2 as cited in in CA DTSC, 2018.

<sup>246</sup> Eriksson U *et al.* (Report for Swedish Environmental Protection Agency) (2015). Screening of PFASs in sludge and water from waste water treatment plants.

<sup>247</sup> Loos *et al.* (2009; 2010), as cited in the Concawe report (2016). Environmental fate and effects of polyand perfluoroalkyl substances (PFAS).

from sewage treatment plants (336) were estimated at 70 kg/yr via water discharges and 5 kg/yr via sludge since 2004. The level in outgoing wastewater had increased since 2009, most likely due to an increase in the use of PFAS in consumer products.<sup>248</sup>

Another study from Switzerland detected PFAAs in all the sewage sludge samples collected from 20 different WWTPs in 2008 and tested a number of historical samples from 1993–2002. Concentrations of PFOS were between 15 to 600 µg/kg.<sup>249</sup> In the same vein, a study sampling sewage sludge from 45 different WWTPs in Switzerland found elevated PFOS concentrations (median 2290 µg/kg). In total, these 45 WWTPs constitute approximately ¼ of the total production of sludge in the country, with 55 000 tonnes. Total quantities of PFOS at the selected WWTPs was estimated as 7.5 kg for the year 2011 (median value), which is extrapolated as 30 kg per year for the whole country. Based on these findings, the study estimated per capita emissions for Switzerland (µg/pers/day) in sewage sludge, for PFOA and PFOS, as 0.2 and 4.8 respectively (median values). It is important to note that these WWTPs were selected for being close to potential industrial pollution sources and therefore the mean emissions might be overestimated.<sup>250</sup>

A study of several small rivers in Germany concluded that discharges of waste waters were the largest contributor of PFOS to surface waters. The study sampled waste water at different stages from treatment plants between 2005 and 2006.<sup>251</sup> The WWTPs had different industries in their proximity with different daily flows. The WWTPs received inflows from domestic, industrial (breweries, tobacco, food and plastics) and commercial sources, with domestic inflows having much lower levels of PFAS (PFOS and PFOA). The study also found that PFOA is able to pass fully from the WWTP without diminishing in concentration and to find its way to rivers, while half of the PFOS is retained in the sludge. In general the study states that PFOA concentrations found are similar to those reported for US, but PFOS concentrations were higher.<sup>252</sup>

In an Austrian<sup>253</sup> study, investigating concentrations of PFAS in effluent samples from 21 municipal WWTPs, findings indicate varying degrees of concentrations ranging from 0 to 280 ng/l for PFHxA, 10 to 220 ng/l for PFOA, and 4 to 340 ng/l for PFOS.<sup>254</sup>

A joint 2013 European study, analysed effluents from 90 European WWTPs, and concluded that despite the phasing out of PFOS, detection levels of this substance (among others) indicates an on-going release of these substances from PFAS containing products, and that release can be not *solely classified as historical*.

On the basis of these findings, it seems reasonable to conclude that PFAS concentrations are found in the discharges of most of Europe's wastewater treatment plants.

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<sup>248</sup> Swedish Environmental Protection Agency (2016). Högfluorerade ämnen (PFAS) och bekämpningsmedel en sammantagen bild av förekomsten i miljön (In Swedish).

<sup>249</sup> Adler A and Van der Voet J (2015). Occurrence and point source characterization of perfluoroalkyl acids in sewage sludge. *Chemosphere* 129: 62–73.

<sup>250</sup> Ibid.

<sup>251</sup> Becker M *et al.* (2008). Perfluoroactane surfactants in waste waters, the major source of river pollution. *Chemosphere* 72(1): 115–21.

<sup>252</sup> Ibid.

<sup>253</sup> The location of sampling sites is not mentioned in the study, but we assume it is Austria since the scientists publishing the study are based at the University of Vienna.

<sup>254</sup> Clara M *et al.* (2008). Emissions of perfluorinated alkylated substances (PFAS) from point sources-identification of relevant branches. *Water Science and Technology* 58(1):59–66.

## 4.5 Case Study 5: Impacts at end of life of PFAS-treated products

### 4.5.1 Background and context

At the end of their useful life, consumer products containing PFAS are discarded or, in some instances, recycled. If the product is a solution such as a cleaning fluid or coating, it will frequently be disposed of down a household drain where the PFAS will flow with the other fluids into a sewer system and to an urban waste water treatment plant.

If the product is an article, it will end up in a municipal solid waste stream. At that point it may be diverted for recycling or material reuse, or disposed of in a landfill or via an incinerator.

Globally, many municipalities and industries still rely on landfilling for final disposal of PFAS-containing products. The latest figures from the US EPA state that in 2015 53% of municipal waste was landfilled and 13% incinerated (the rest was recycled or composted).<sup>255</sup>

In the Nordic countries, landfilling household waste is done to a very low extent; e.g. 0.5% in Sweden (2017)<sup>256</sup>, 1% in Denmark (2016)<sup>257</sup> and 3% in Norway (2017).<sup>258</sup> Incineration is the more common method for final treatment, with Sweden incinerating around 50% of its household waste in 2017, Denmark 51% (2016) and Norway 57% (2017). The EU-28 countries as a whole are also moving from landfilling to incineration. In 1995 64% of municipal solid waste was landfilled and 14% incinerated. In 2016, 24% of household waste went to landfills while 27% was incinerated.<sup>259</sup>

Knowledge about the necessary conditions for destruction of PFAS and what happens if those conditions are not achieved is still limited. PFAS can be broken down, but only under conditions that are so harsh, e.g. incineration at very high temperatures, that they do not occur in the normal environment.<sup>260</sup> A 2014 study for the USEPA found that a thermal reactor system operating at 1000 °C was able to destroy fluorotelomer-based polymers without resulting in the formation of detectable levels of PFOA.<sup>261</sup> However, such temperatures may not be typical. The EU rules for municipal waste incineration require a temperature of 850 °C.<sup>262</sup> Experiments in temperatures similar to municipal waste

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<sup>255</sup> Environment Protection Agency (2018). Advancing Sustainable Materials Management: 2015 Fact Sheet.

<sup>256</sup> Avfall Sverige (2018). Hushållsavfall – behandlad och insamlad mängd (In Swedish).

<sup>257</sup> Danish Environmental Protection Agency (2016). Affaldsstatistikken 2016 (In Danish).

<sup>258</sup> Statistisk sentralbyrå (2018). 426 kilo avfall per innbyggjar (In Norwegian).

<sup>259</sup> Calculated from Eurostat: Municipal waste landfilled, incinerated, recycled and composted in the EU-28, 1995 to 2016.

<sup>260</sup> Wang Z *et al.* (2017). A never-ending story of per- and polyfluoroalkyl substances (PFASs)? *Environmental Science & Technology*, Mar 7;51(5).

<sup>261</sup> Taylor P S *et al.* (2014). Investigation of waste incineration of fluorotelomer-based polymers as a potential source of PFOA in the environment. *Chemosphere* 110: 17–22.

<sup>262</sup> Directive 2010/75/EU of 24 November 2010 on industrial emissions (integrated pollution prevention and control) (Recast), OJ L 334/17 (17.12.2010). Article 50 requires operating conditions for a waste incineration plant to reach at least 850 °C for at least two seconds.

incineration plants (800 °C -1000 °C) have shown that certain fluorine products remain after combustion.<sup>263</sup> The knowledge gap as regards the fate of these substances in incineration processes is significant and an area where the knowledge base needs to improve.

If a product is disposed of through landfill, the matrix (material of the product) may break down, but the PFAS will remain. Over time (and depending on the design of the landfill and the durability of the barriers between the waste and the underlying earth), the PFAS will migrate into any liquid in the landfill and drain into leachate collection systems or directly into soil and groundwater.

PFAS have been detected in landfill leachate around the world.<sup>264</sup> A Swedish study of 26 PFAS in samples from groundwater, surface water, WWTP effluent, and landfill leachate found that landfill leachates had the highest average total PFAS concentrations (487 ng/l).<sup>265</sup> Shorter-chain PFAAs tend to be the most abundant PFAS in landfill leachate.<sup>266</sup> Another factor raised by the study is that only a small fraction of the total number of PFAS compounds are being analysed. A screening of the total organic fluor (TOF) content in sewage sludge showed that the individually analysed PFAS compounds only accounted for a few percent of the TOF. The authors conclude that the picture is likely to be the same for landfill leachate.

PFAS release from solid waste is slow, compared to the amount of PFAS manufactured and used in consumer products each year. A study of PFAS releases from carpet and clothing using a model landfill reactor found that for the most part, the releases did not take place until >200 days of operation.<sup>267</sup> In an actual landfill, the process of breaking down the underlying substrate to release the PFAS will take much longer. Thus, the PFAS in solid waste sitting in landfills today will continue to be released into leachate for many years into the future.<sup>268</sup> Moreover, a review of the fate and transformation of PFAS in landfills noted that ambient air around landfill sites had found elevated concentrations of PFAS compared to upwind sites used as controls, which suggested that landfills are also potential sources of PFAS in the atmosphere.<sup>269</sup>

The stability of PFAS compounds also means that they tend to remain in other materials where they have been used. PFAS used to ensure low surface tensions during plastics moulding<sup>270</sup> are likely to remain in the plastic waste stream during materials recycling. This has implications for the circular economy.

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<sup>263</sup> Huber S *et al.* (Norwegian Institute for Air Research) (2009). Emissions from incineration of fluoropolymer materials. Report number: OR112/2009.

<sup>264</sup> Fuertes I *et al.* (2017), Perfluorinated alkyl substances (PFASs) in northern Spain municipal solid waste landfill leachates, *Chemosphere* 168: 399–407. See also Hamid H *et al.* (2018). Review of the fate and transformation of per- and polyfluoroalkyl substances (PFASs) in landfills. *Environmental Pollution*, 235: 74–84.

<sup>265</sup> Ahrens L *et al.* (2016). Screening of PFASs in groundwater and surface water, Uppsala Report No: 2016:2.

<sup>266</sup> Hamid *et al.* (2018).

<sup>267</sup> Lang J R *et al.* (2016). Release of Per- and Polyfluoroalkyl Substances (PFASs) from Carpet and Clothing in Model Anaerobic Landfill Reactors. *Environmental Science & Technology*, 50, 5024–5032.

<sup>268</sup> Lang J R *et al.* (2017). National Estimate of Per- and Polyfluoroalkyl Substance (PFAS) Release to U.S. Municipal Landfill Leachate. *Environmental Science and Technology*, 21;51 (4):2197–2205.

<sup>269</sup> Hamid H (2018). Review of the fate and transformation of per- and polyfluoroalkyl substances (PFASs) in landfills. *Environmental Pollution*, 235: 74–84.

<sup>270</sup> Kennebunk, Kennebunkort and Wells Water District (2017). Per and Polyfluoroalkyl Substances (PFASs) Usage (Draft for informational purposes). See also Hamid H (2018). Review of the fate and transformation of per- and polyfluoroalkyl substances (PFASs) in landfills. *Environmental Pollution*, 235: 74–84.

PFAS are frequently applied to paperboard used for food contact products, to make waterproof and greaseproof containers. For communities with active programs aimed at diverting biodegradable materials into composting systems, it has been an unwelcome surprise to learn that compost developed from feedstocks of mixed food and yard waste and including compostable food service ware may have high levels of PFAS, with the most prevalent being the short chain PFAS (C<sub>4</sub> – C<sub>6</sub>).<sup>271</sup>

A Swedish study found that only a small fraction of the total number of PFAS compounds are being analysed. A screening of the total organic fluor (TOF) content in sewage sludge showed that the individually analysed PFAS compounds only accounted for a small percentage of the TOF.<sup>272</sup>

The application of compost or other soil enhancers such as biosolids (sewage sludge) in which PFAS are present to agricultural soil is leading to concern that the PFAS may be taken up by edible plants and end up bioaccumulating in the food chain. A study of greenhouse lettuce and tomatoes grown in a soil amended with biosolids affected by industrial chemicals found that the plants had taken up PFAS<sup>273</sup>, and that perfluorobutanoic acid (PFBA) and perfluoropentanoic acid (PFPeA) seemed to have bioaccumulated to a degree. The short-chain PFAS seemed to be less attached (more mobile) to the agricultural soil and to have higher crop uptake potential. The study indicates that plants grown on soil amended with sewage sludge containing PFAS can bioaccumulate PFAS, with the extent of the bioaccumulation varying depending on the concentration of PFAS, the properties of the soil, the type of crop, and the chemical under analysis.

The finding that short-chain PFAS had the highest potential to bioaccumulate in produce was duplicated in a recent Minnesota study.<sup>274</sup> The study investigated whether home gardens irrigated with PFAS-contaminated water would result in contaminated produce. Among the conclusions: PFAS in water can enter into the food chain under real-world conditions. Short-chain PFAS in water had a greater impact on levels in produce than long-chain PFAS in soil. Finally, PFAS concentrations varied according to plant part with florets found to have the highest concentrations of PFAS.

#### 4.5.2 Cases of contamination

##### Arnsberg, Germany

In 2006, a high level of PFAS contamination was detected in the conjunction of the rivers Rhine, Ruhr and Moehne as well as nearby public water supplies. This had consequences

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<sup>271</sup> Lee L and Trim H (2018). Summary Sheet Evaluating Perfluoroalkyl Acids in Composts with Compostable Food Service-ware Products in their Feedstocks.

<sup>272</sup> Avfall Sverige (2018). Hushållsavfall – behandlad och insamlad mängd (In Swedish).

<sup>273</sup> Blaine AC *et al.* (2013). Uptake of Perfluoroalkyl Acids into Edible Crops via Land Applied Biosolids: Field and Greenhouse Studies. *Environmental Science & Technology*. 47(24), pp. 14062–14069.

<sup>274</sup> Scher D *et al.* (2018). Occurrence of perfluoroalkyl substances (PFAS) in garden produce at homes with a history of PFAS-contaminated drinking water. *Chemosphere* 196: 548–555.

for the whole Ruhr valley catchment and Lake Moehne, which supply 5–6 million people.<sup>275</sup> An estimated 40,000 people were exposed to drinking water that was contaminated from this source.<sup>276</sup> The water was contaminated with PFOA, perfluorohexanoate (PFHxA), perfluorohexane sulfonate (PFHxS), perfluoropentanoate (PFPA) and perfluorobutane sulfonate (PFBS). 110.5 kg of PFAS were calculated to have entered Lake Moehne.<sup>277</sup> This was gradually released into the Ruhr and Rhein, to make its way to the North Sea.

The source of the contamination was PFAS contaminated sludge containing industrial waste, which was sold under the name of “bio-solids” and was applied at farmland at the head of the Moehne valley.<sup>278</sup>

Several actions were taken to manage the contamination. First, a monitoring system was put into place to assess the level and spread of contamination in drinking water. Several biomonitoring studies were launched to measure the pathways into mothers and children (including breastmilk), and men, as well as the Activated carbon filters were installed in water works. Recommendations were developed to reduce the consumption of fish.<sup>279</sup> Carbon filters were installed in July 2006. The carbon filters were reactivated about every 6 months. The reactivation was undertaken in specialized centers that involved treatment of the filters in a furnace heated up to over 800 degrees Celsius.

A biomonitoring study was undertaken to determine the level of blood concentration in a sample of men, women and children.<sup>280</sup> In total, 138 children, mothers and men participated in the study. Measurements were taken before and in subsequent years after the installation of activated charcoal filtering system to remove PFOA from the drinking water. Repeat blood samples were analysed for about 20 to 25 individuals for each group (children, mothers and men). Notable decreases were detected over the two year period (see Table 18). For example, the serum concentration level of PFOA in children decreased 39%. The study suggest that the reduction may be due in part to a reduced consumption of fish from local sources.

**Table 18: Relative reduction of PFC plasma levels (%) between 2006 and 2008**

	PFOA	PFOS	PFHxS
Children	39.2 (31.6–48.5)	20.1 (14.7–27.6)	18.7 (10.4–33.7)
Mothers	39.4 (33.5–46.3)	21.7 (16.0–29.4)	29.6 (24.7–35.4)
Men	25.5 (21.3–30.5)	25.0 (21.5–29.0)	14.3 (10.4–19.7)

<sup>275</sup> IPEN (2018). Fluorine-free firefighting foams (3f) viable alternatives to fluorinated aqueous film-forming foams (AFFF).

<sup>276</sup> Hölzer J *et al.* (2009). One-year follow-up of perfluorinated compounds in plasma of German residents from Arnsberg formerly exposed to PFOA-contaminated drinking water, *International journal of hygiene and environmental health*, 212(5), pp.499–504.

<sup>277</sup> Weber R (2016). Presentation for Science and Policy of Organohalogen pre-Dioxin Symposium.

<sup>278</sup> Skutlarek D *et al.* (2006). Perfluorinated surfactants in surface and drinking waters. *Environmental science and pollution research international*, 13(5), p.299. IPEN (2018). Fluorine-free firefighting foams (3f) viable alternatives to fluorinated aqueous film-forming foams (AFFF).

<sup>279</sup> WHO Europe (2016). Keeping our water clean: The case of water contamination in the Veneto Region, Italy.

<sup>280</sup> Brede E *et al.* (2010). Two-year follow-up biomonitoring pilot study of residents’ and controls’ PFC plasma levels after PFOA reduction in public water system in Arnsberg. *International journal of hygiene and environmental health*, 213(3), pp.217–223.

As of 2006, 100 million Euros had been spent on investments in the regional water works.<sup>281</sup> Charges were pressed against the German company providing the sludge, which declared bankruptcy as a consequence of the contamination scandal, and the CEO was taken to court. The following year, national monitoring activities by the competent authorities were initiated.

### Baden-Wuerttemberg, Germany

Following the Arnsberg case (see previous case study), the state of Baden-Wuerttemberg analysed samples from 41 locations potentially subject to PFAS contamination in 2006.<sup>282</sup> Measure points with elevated levels were followed up subsequent years.

In 2013, PFAS was found during a routine analysis in a well belonging to Landkreis Rastatt's drinking water supply.<sup>283</sup> Further investigations unravelled a contamination situation of unprecedented dimensions, making it the greatest contamination case in Germany both in terms of surface affected and the complexity of contaminant composition.<sup>284</sup> As of August 2018, 644 hectares of soil in Landkreis Rastatt and Stadtkreis Baden-Baden, as well as 240 hectares in Mannheim, are expected to be contaminated by PFAS.<sup>285</sup> Below the area affected runs one of the largest underwater rivers in Europe, the Oberrhein-Aquifer, adding to the level of graveness of the pollution incident.

Although the reason for contamination remains somewhat disputed, the explanation which mainly is put forward is the use of compost blended with contaminated paper mill waste which was applied on agricultural land between 2005 and 2008.<sup>286</sup> An additional source would be a fire extinguishing event in 2010. The total amount of PFAS which entered the environment is hard to appreciate; an (uncertain) estimate received from a member of staff at Landkreis Rastatt reads 1,000–5,000 kg.<sup>287</sup>

Following the PFAS discovery, two waterworks providing Rastatt drinking water were taken out of use due to PFAS in the water, leaving Rastatt with one single waterwork available. To ensure safe drinking water, the local water company Stadtwerke Rastatt has invested millions of Euros in new infrastructure, groundwater monitoring, treatment methods such as active carbon filters or reversed osmosis, and in securing alternative sources of water.<sup>288</sup> This has enabled one of the waterworks to reopen in Februari 2018. The reconstruction of pipes, water works, and installation of activated carbon filters have cost the company EUR 3.6 million by the end of 2017. The same activities are estimated to cost the company another EUR 6.2 million during 2018–2020

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<sup>281</sup> Weber R (2016). Presentation for Science and Policy of Organohalogenes pre-Dioxin Symposium.

<sup>282</sup> LUBW (2018). Grundwasserüberwachungsprogramm Ergebnisse der Beprobung 2017 (in German).

<sup>283</sup> Regierungspräsidium Karlsruhe (No date). Überblick zur PFC-Problematik in Mittel- und Nordbaden.

<sup>284</sup> Regierungspräsidium Karlsruhe (2017). Antworten auf häufig gestellte Fragen zur PFC-Belastung im Landkreis Rastatt und den Stadtkreisen Baden-Baden und Mannheim Stand August 2017.

<sup>285</sup> Ministerium für Umwelt, Klima und Energiewirtschaft Baden-Württemberg (2018). Neuer Erlass schreibt Beurteilungsgrundlage für mögliche PFC-Belastungen von Grund- und Sickerwasser fort.

<sup>286</sup> Klatt P and Frey A (2016). Woher kam das Zeug bloß?, Frankfurter Allgemeine, 4 September 2016. Accessed September 2018.

<sup>287</sup> R. Söhlmann (2018). Landratsamt Rastatt, personal communication

<sup>288</sup> Stadtwerke Rastatt (2018). So schützen wir unser Trinkwasser in Rastatt (website in German) and personal communication

(much depending on how frequently the carbon filter will need to be changed). These costs have so far been borne by the company without the contribution of any state funds, and thus the water price for the consumers in Rastatt has been raised from EUR 1.64 to EUR 1.86 per m<sup>3</sup> in the course of 2017.

As regards the further securement of uncontaminated drinking water, there has been an ongoing discussion between the main water company (Stadtwerke Rastatt) and the responsible authorities at municipal and state level concerning the next steps to take. Though the water company called for immediate action in order to prevent a worsening of the situation, the public authorities proposed to spend the year up to 2021 to improve the knowledge base, and to await further action until then.<sup>289</sup> In the meanwhile, efforts to assess the extent and potential development of the contamination profile are being taken.

To improve knowledge on health effects, the state and local health authorities are carrying out a blood sampling study of the population, the results of which are scheduled to be presented at the end of 2018.<sup>290</sup> The study has cost around 257,000 Euros during 2017–2018, which includes costs for meetings with the experts, laboratory costs and the time spent on planning, carrying out and assessing the study. Two repetitions of the study are planned: one in 2020 and one in 2023, with an estimated budget of 408,000 Euro.

In the region in question, growing vegetables and fruit is a major part of the local economy. Today, even certain farmers who did not use the contaminated compost are unable to grow and sell their goods, as the PFAS has spread to reach their land as well.<sup>291</sup> To ensure that no produce with unacceptable levels of PFAS reach the consumers, the state of Baden-Württemberg has integrated a pre-harvest monitoring program targeting PFAS into their foodstuff monitoring framework. This is financed by the state and its costs have already amounted to more than one million Euros.<sup>292</sup> The state Environmental Department (LUBW) has also initiated pilot studies of PFAS in groundwater, focusing on potential contamination deriving from agriculture. As a result, PFAS were included into the regular state water monitoring program from 2015, with a planned timeline of four years. As of 2017, around 50% of the measure points showed PFAS contamination (around half of them at levels below 10 ng/l). Monitoring in general is likely to prove necessary for decades to come.

No clear verdict in terms of responsibility has yet been reached. Nevertheless, a verdict from Mannheim administrative court required the party who provided the contaminated compost to farmers in the region to pay for the preliminary investigation of the soils (35,000 Euros).<sup>293</sup> The court considered his provisioning of the papermill waste compost

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<sup>289</sup> Klatt P and Frey A (2016). Woher kam das Zeug bloß?, Frankfurter Allgemeine, 4 September 2016. Accessed September 2018.

<sup>290</sup> Gesundheitsamt Rastatt (No date). Informationen zum Stand der PFC-Blutkontrolluntersuchung im Landkreis Rastatt.

<sup>291</sup> Klatt P and Frey A (2016). Woher kam das Zeug bloß?, Frankfurter Allgemeine, 4 September 2016. Accessed September 2018.

<sup>292</sup> Ministerium für Umwelt, Klima und Energiewirtschaft Baden-Württemberg (No date). PFC-Belastung in Baden-Baden und im Landkreis Rastatt.

<sup>293</sup> Söhlmann, R. (2018), Landratsamt Rastatt. Personal communication.

mix to farmers as a cheap way of disposing of industrial waste, rather than providing useful soil improver. For the rest, the state has covered most of the costs related to the initial investigations and measures. 900,000 Euros has been spent on modelling groundwater remediation, and 1.5 million Euros on other research projects.<sup>294</sup>

As regards public spending, 4.4 million Euros of state funds and almost 1.7 million Euros of municipal funds have so far been used in relation to the PFAS contamination. How much expenses are yet to come remains unknown, but it has been estimated that a complete remediation of the affected soils might amount to 1–3 billion Euro.<sup>295</sup> A cost analysis dating from 2015 estimated that each hectare would cost 5.5 million Euros to remediate. With today's 640 contaminated hectares in Landkreis Rastatt, this would mean a total cost of around 3.5 billion Euro. To actually perform this enormous soil exchange is however not realistic, partly due to the vast quantities of soil that would be "lost".<sup>296</sup> In addition, the 2015 study estimated that the cost of groundwater remediation would amount to 150 million Euros. As today even more PFAS are likely to have reached the groundwater, it is probable that this figure will not suffice.

### Rockford, Michigan

The leather tannery complex owned by the footwear company Wolverine World Wide treated leather for manufacturing into shoes sold under the brand HushPuppy. PFAS purchased from the company 3M were applied to the leather (along with other chemicals) and the shoes were marketed as being waterproof. The chemicals were stored in drums at outdoor locations. A 2000 summary of hazardous chemicals on site stated that 16,590 pounds (7,525 kg) of Scotchgard FC-3573 and 64,409 pounds (29,215 kg) of Scotchgard FX-3573 were kept on site. In the early 2000s, 3M reformulated its product (Scotchgard) to remove PFOS from the formulations because of evidence of the chemical's toxicity, bioaccumulability and persistence in the environment.

High concentrations of both PFOA and PFOS have now been found in soil and groundwater at the now unused manufacturing site, and the chemicals have now migrated into a creek that runs into the nearby Rogue River.<sup>297</sup> Testing of surface foam at a local dam also found very high levels of PFAS, as did testing of fish caught in the Rogue River. The State of Michigan has now issued a fish consumption advisory to warn local anglers not to eat fish from the most contaminated stretch of the river. In 2017 local residents alerted environmental authorities to a former licensed disposal facility owned and operated by Wolverine as well as several unregulated dumpsites where leather scraps and other manufacturing waste were deposited, and requested testing of nearby wells.<sup>298</sup> In May 2017, PFAS contamination was detected, and Wolverine began a more extensive well sampling program. It also started to provide bottled water and water filters for affected households.

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<sup>294</sup>Ministerium für Umwelt, Klima und Energiewirtschaft Baden-Württemberg (No date). PFC-Belastung in Baden-Baden und im Landkreis Rastatt.

<sup>295</sup> IPEN (2018). Fluorine-free firefighting foams (3f) viable alternatives to fluorinated aqueous film-forming foams (AFFF).

<sup>296</sup> Söhlmann, R. (2018). Landratsamt Rastatt, personal communication.

<sup>297</sup> See State of Michigan website on its PFAS response for more information.

<sup>298</sup> Ibid.

Later in 2017, the State of Michigan developed a groundwater criterion for PFOA and PFOS of 0.07 µg/l (70 ppt) for protection of drinking water.

In one of the areas close to the now closed landfill, 561 homes were tested for PFAS in drinking water; PFOA and PFOS was detected in 197 homes, 70 of which had concentrations over 70 ppt. The highest concentration found was 62,500 ppt. In another affected area, 690 homes were sampled, 391 had detected levels of PFOA and PFOS, 38 homes had concentrations over 70 ppt, and the highest concentration was 49,200 ppt.<sup>299</sup>

Blood tests of residents have shown high levels of PFAS. One long-term resident whose well tested at 38,000 ppt learned that four different PFAS were found in her blood for a combined total of 5 million ppt. One chemical, PFOS, was found at 3.2 million ppt – about 750 times the national blood level average of 4,300 ppt.<sup>300</sup> While it is not possible to pin specific health impacts to a specific exposure of PFAS, the resident knew of epidemiology studies that had found links to the thyroid problems she suffered from and the liver cancer from which her husband had died in 2016.

#### 4.5.3 *Extent of the exposure due to PFAS disposal to land*

A study<sup>301</sup> of US municipal landfills calculated that the total mass of PFAS from landfill leachate to wastewater treatment plants in 2013 was between 563 and 638 kg. The researchers measured concentrations of 70 PFAS in 95 samples of leachate from 18 landfills in the USA of varying climates and deposit ages, then linked estimates of total annual leachate volumes in the US with the concentrations measured for the 19 PFAS where >50% of samples had quantifiable concentrations. Participating landfills were publicly owned, so they contained mainly municipal solid waste and some sewage sludge, but probably did not receive industrial waste. FTCA (5:3 fluorotelomer carboxylic acid) was the dominant PFAS in the majority of samples collected.

The ~600 kg/yr estimate for total PFAS mass release in US landfill leachate in 2013 has limitations. It is based on PFAS concentrations from 18 sites around the US, and then extrapolated to cover the total of 1540 landfills in the US that year. While none of the landfills in the study reported accepting waste from PFAS, textile or carpet production, some municipal solid waste landfills may accept such waste. For example, PFOS concentrations from leachate at a landfill that received wastewater treatment sludge from a 3M facility in Minnesota<sup>302</sup> were measured at 136 micrograms/L, compared to the ranges of concentrations (2–29 micrograms/L) in the leachate from the 18 landfills sampled in the study. In addition, it does not account for historic landfills (the US had some 6000 landfills in 1988) that may be unlined and

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<sup>299</sup> Ibid.

<sup>300</sup> Ellison G (2018). Belmont woman's blood is 750 times national PFAS average, Mlive, 11.01.2018 Accessed 15.10.2018.

<sup>301</sup> Lang JR *et al.* (2017). National Estimate of Per- and Polyfluoroalkyl Substance (PFAS) Release to U.S. Municipal Landfill Leachate. *Environmental Science and Technology*, 21;51(4):2197–2205.

<sup>302</sup> Oliaei F *et al.* (2013). PFOS and PFC releases and associated pollution from a production plant in Minnesota (USA). *Environmental Science Pollution Research*. 20: 1977–1992.

closed without low permeability covers.<sup>303</sup> These unlined landfills will continue to pose a risk of PFAS leakage to groundwater for many years to come.

The following examples illustrate that municipal and industrial landfills are sources of legacy PFAS, primarily affecting groundwater and run-off surface waters:

- a 2010 study calculated around 90 kg/yr for 44 PFAS in treated leachate from all (~1700) landfills in Germany<sup>304</sup>;
- a 2016 study of PFAS in the Swedish environment from different sources estimated 70 kg/year of PFAS emissions via leachate from 365 landfills.<sup>305</sup> Except for 8 kg of PFAS spread to land, the leachate was sent for sewage treatment;
- a 2017 study of 4 municipal waste landfills across northern Spain gathered data on the occurrence and concentration of 16 PFAS in the leachate from those sites. The landfills served 1.8 million people. Based on the volume of leachates from the landfill sites, it was estimated that the combined discharge of the 16 PFAS was 1.2 kg/year.<sup>306</sup>

The costs of remediation for removing PFAS contamination from affected waters would therefore be the same as for other sources of PFAS contamination.

#### 4.6 Other costs related to PFAS contamination

Some of the non-quantifiable costs of exposure to PFAS are the experiences of the individuals and communities affected, i.e., as they come to understand that the drinking water they have been consuming has contained a contaminant that may result in health impacts that do not become evident until years hence. Those experiences are part of the overall story concerning the socioeconomic costs of PFAS.

“For years, folks trusted that their water providers were delivering a completely safe product and knew exactly what was in it. At the same time, the providers trusted that regulators were adequately protecting water sources and knew exactly what was in our rivers. That trust is gone.”

Editorial in *Wilmington NC Star News*, 24 June 2018<sup>307</sup>

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<sup>303</sup> Lang JR *et al.* (2017). National Estimate of Per- and Polyfluoroalkyl Substance (PFAS) Release to U.S. Municipal Landfill Leachate. *Environmental Science and Technology*, 21;51(4):2197–2205.

<sup>304</sup> Busch J (2010). Polyfluoroalkyl compounds in landfill leachates. *Environmental Pollution*, Vol 158:5, 1467–1386.

<sup>305</sup> Swedish Environmental Protection Agency (2016). Högluorerade ämnen (PFAS) och bekämpningsmedel en sammantagen bild av förekomsten i miljön.

<sup>306</sup> Fiertes I *et al.* (2017). Perfluorinated alkyl substances (PFASs) in northern Spain municipal solid waste landfill leachates. *Chemosphere* 168: 399–407.

<sup>307</sup> Editorial, We must take control of our water quality, *Starnews Online*, 25.06. 2018. Accessed 11. 09. 2018.

Other more quantifiable costs of inaction may include loss of property value, reputational damages to a polluting company, (e.g, the recent Miteni bankruptcy), court awarded damages and financial settlements. For example, the Australian government announced AU\$73.1 million budgeted to support those affected by PFAS contamination, of which AU\$55.2 million will be spent across five years to give people access to safe drinking water.<sup>308</sup> The government has already spent more than AU\$100 million on PFAS, and has not yet started any remediation work. Some of the measures taken have addressed the human impacts of the contamination, e.g., public outreach, a help desk, and counselling services for affected communities.

Table 19 summarises a number of indemnities paid in legal settlements by companies with facilities that led to PFAS contamination of the environment as well as PFAS-related health effects.

**Table 19: Amounts of some indemnities paid in legal settlement in relation to PFAS contamination due to different sources**

Incident	Year <sup>1</sup>	Description	Amount (EUR) <sup>2</sup>
Contamination of drinking water in Hoosick Falls NY due to industrial production (US)	2014-Ongoing	Legal case on-going between the municipality and Saint-Gobain Performance Plastic and Honeywell International (as a part of on-going fees to be paid to the municipality)	443,000
Contamination of natural resources in Minnesota due to the 3M plant (US)	2018	Legal settlement between 3M and the State of Minnesota	710,400,000
Contamination of water supplies in Ohio (US)	2017	Agreement reached between Dupont Chemicals and Chemours in class action law suit with the residents	595,000,000
Contamination of Hyannis MA water supply due to AFFFs (US)	2017	Legal settlement between the town of Barnstable and Barnstable country	2,617,000
Contamination of agricultural fields in Baden-Wuerttemberg	2013	Initial investigations payed for by compost salesman who had provided contaminated compost to farmers	35,000

Note: 1) Year might refer to year of detection, or the year costs have incurred.  
 2) Costs in other currencies are converted to Euro, using average annual rates for the year they incurred.

## 4.7 Summary of case study findings

As the five case studies illustrate, PFAS are released to the environment from many sources, from production and manufacturing plants to specialist uses such as AFFFs for firefighting and everyday consumer products such as clothing, pizza boxes and cosmetics.

<sup>308</sup> Swanson M, People, not politics, must be PFAS priority, The Herald, May 2018. Accessed 12.11.2018.

The first three case studies cover the activities that account for a large proportion of the PFAS released directly into the environment. Case Study 1 looks at how the industrial facilities producing the fluorochemicals and fluoropolymers, while relatively limited in number, are significant individual emitters of PFAS into the air, soil and waterways. The study estimates that 12 to 20 facilities actively produce fluorochemicals in Europe, that these facilities are significant sources of PFAS released to the environment, and that exposure to workers at these plants is high. The study did not identify fluorochemical production facilities in the Nordic countries.

Other industrial activities with the potential to release PFAS to the environment take place throughout Europe, including the Nordic region. Case Study 2 considers the manufacture and commercial use of PFAS-containing products, including textile and leather manufacturing; metal plating, including chromium plating; paper and paper product manufacturing; paints and varnishes; cleaning products; plastics, resins and rubbers; and car wash establishments. Releases of PFAS occur via the air or effluent entering sewerage and wastewater treatment plants, before discharge into waterways. The case study gathers Eurostat figures for the number of large companies and SMEs carrying out the industrial activities reviewed. In the absence of information concerning how many of the companies use PFAS in their manufacturing, a range of 3% to 10% of facilities is suggested. The third major source of direct emissions is the widespread use of aqueous film-forming foams (AFFFs) used to extinguish fires in emergencies or during training, especially around airports and military bases. Where the AFFFs have migrated to groundwater and other sources of drinking water, nearby communities have been affected by elevated levels of PFAS in their drinking water. It is noted that other uses of AFFFs for fire-fighting, especially at major industrial facilities, may also be a significant source, but one that has so far received little attention. High performance non-fluorinated AFFFs are now available, but legacy emissions from PFAS in AFFFs used in the past will continue to be a problem for years to come.

The two case studies on the use and end-of-life phases of consumer products account for the remaining releases. They can be direct as well as indirect sources of exposure to PFAS. A 2000 study carried out for 3M estimated that 85% of the indirect emissions of POSFs (a precursor of PFOS) would result from losses during the use and disposal stages. More recent information on the proportion of indirect emissions of PFAS during the use and disposal stages of the chemicals' life cycle was not found.

Case Study 4 considered PFAS-treated carpets, PFAS-treated food contact materials, and cosmetics as examples of how a product's use is likely to lead to human exposure through ingestion and dermal absorption. Consumer products can also lead to releases to the environment when the product is washed off or laundered, entering sewers, treatment plants, and eventually waterways. The availability of suitable non-fluorinated alternatives makes the use of PFAS in many of these products unnecessary. Case Study 5 looks at end-of-life impacts of PFAS-treated products. Waste incineration may destroy PFAS in products if 1000 °C operating temperatures are reached, but such temperatures are not typical of most incineration capacity (the EU's Industrial Emissions Directive, for example, requires a temperature of 850 °C). If landfilled, the PFAS will remain even after the product's core materials break down. The compounds will

eventually migrate into liquids in the landfill, then into leachate collection systems or directly into the natural environment. They may then enter drinking water supplies, be taken up by edible plants and bioaccumulate in the food chain.

A number of other costs related to PFAS contamination include loss of property value, reputational damage to a polluting company (as in the case of the recent Miteni bankruptcy), and the costs incurred by public authorities in responding to affected communities – including public outreach, surveys of contamination, and remedial measures.

For future investigations of this nature, it would be useful to have more information concerning the sites where production of PFAS and/or where manufacturing of products involving PFAS is occurring – both current and legacy activities. National inventories of such sites, including where fluorinated AFFFs have been used, would help estimates of the numbers of affected populations, and the extent of contamination where remediation may be needed. Another suggestion is to include industries producing or using PFAS in the European Pollutant Release and Transfer Register, so that information on the location and amount of releases to air and to water is available.

National registries of products containing PFAS would help inform how PFAS are used and contribute to better characterisation of the major sources of exposure from products. Finally, more research is needed concerning what happens to PFAS discharged from wastewater treatment plants and during incineration of PFAS.



## 5. Estimates of costs of inaction linked to exposure to PFAS

### 5.1 Health-related costs of exposure to PFAS

This section presents findings for the health-related costs of PFAS exposure at three different levels – occupational (high) exposure, elevated (medium) exposure and background (low) exposure. The quantification was carried out for a selection of scenarios and health endpoints. Health-related costs that could not be quantified are reviewed qualitatively.

#### 5.1.1 *Occupational (high) exposure scenario: PFAS production and manufacture of PFAS-containing products*

Individuals who are regularly exposed to PFAS through their occupation (e.g. workers in manufacturing plants producing PFAS or PFAS-treated products) may face greater risk of developing illnesses that affect their health and well-being. The desk research identified two studies that investigated elevated health risks due to PFAS exposure among workers in Europe. One study investigated serum concentrations of PFOA and liver enzymes from 56 workers in a fluorochemical production plant. The study found that PFOA serum concentration was associated with higher ALT, GGT and ALP enzymes and lower bilirubin.<sup>309</sup> Another 2001 study conducted by a staff epidemiologist from 3M reported that occupational exposure to PFOA and PFOS in chemical plants in Antwerp and another site in the United States was associated with an increased level of cholesterol and triglycerides.<sup>310</sup> However, the findings were more cautiously stated in the study published several years later.<sup>311</sup>

One of the most well-known studies of occupational exposure to PFAS was carried out under the C8 Health Project, which gathered data from workers from the Dupont Washington Works facility in West Virginia from 1952–2008. The sample of workers was known as the worker cohort while the C8 Health Project also gathered data from a community cohort. A multi-mortality study was conducted for this worker cohort, which

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<sup>309</sup> Costa G *et al.* (2009). Thirty years of medical surveillance in perfluorooctanoic acid production workers. *Journal of Occupational and Environmental Medicine* 51:364–372.

<sup>310</sup> Olsen G W (2001). A longitudinal analysis of serum perfluorooctanesulfonate (PFOS) and perfluorooctanoate (PFOA) levels in relation to lipid and hepatic clinical chemistry test results from male employee participants of the 1994/95, 1997 and 2000 fluorochemical medical surveillance program. Final report *Epidemiology*.

<sup>311</sup> Olsen G W *et al.* (2003). Epidemiologic assessment of worker serum perfluorooctanesulfonate (PFOS) and perfluorooctanoate (PFOA) concentrations and medical surveillance examinations. *Journal of Occupational and Environmental Medicine*, 45(3), pp.260–270 and Lerner, S. (2018), 3M knew about the dangers of PFOA and PFOS decades ago, internal documents show, *The Intercept*, 31.07.2018.

had a target population of 6,026.<sup>312</sup> The study found evidence of a potential elevated risk of death from kidney cancer due to prior exposure to PFOA in the workplace.

Other sources suggest that occupational exposure to PFAS may have developmental toxicity. For example, a pregnant female worker who had direct contact with PFOA through her employment at the Dupont facility in West Virginia gave birth to a child with multiple birth defects. Soon afterwards, the company did not allow women of reproductive age to work directly with the chemical.<sup>313</sup> This endpoint, however, was not confirmed in the C8 Health Project in West Virginia.<sup>314</sup> Another study conducted among workers from a 3M plant in Decatur, Alabama did not find a relationship with pregnancy outcomes.<sup>315</sup> The small number of individuals in this category of pregnant workers may limit the detection of a statistical pattern between exposure to PFOA and pregnancy outcomes.

Overall, the evidence suggests that occupational exposure to PFAS may lead to an elevated risk of kidney cancer, a disease that can lead to significant costs in terms of health care expenditures, reduced quality of life, and premature death. The valuation for the health-related costs due to occupational exposure to PFAS focused on the kidney cancer endpoint for which there is epidemiological evidence.

An assessment was made by drawing on the findings from the cohort mortality study of workers from West Virginia<sup>316</sup> extrapolated to the European context. Like other epidemiological studies carried out under the C8 Health Project, the cohort mortality study had a robust design and was overseen by a Science Panel of three epidemiologists. The sample for the study was restricted to individuals who had worked at least one day at the Dupont chemical plant in West Virginia between 1948 and 2002. The sample was divided into four groups based on their estimated level of exposure to PFOA. The study found that high occupational exposure to PFOA was associated with an increased risk of death due to kidney cancer.

As explained in the Chapter 4, it was not possible to develop a firm estimate of the number of plants that manufacture PFAS or products using PFAS in Nordic and EU countries. Some assumptions were made in order to generate estimates to support the valuation of the health-related costs for the study. The first assumption is that 20 manufacturing plants produce PFAS in European countries (see conclusion to Case Study 1 at page 64). The second assumption relies on Case Study 2's research that identified a total of 352,764 small (less than 250 employees) and 780 large manufacturers (more than 250 employees), which may use or emit PFAS, in EEA countries. As Case Study 2 explains, no information was available concerning which plants used PFAS in manufacturing their products nor how many workers were employed at each plant. In the absence of concrete

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<sup>312</sup> Steenland K and Woskie S (2012). Cohort mortality study of workers exposed to perfluorooctanoic acid. *American journal of epidemiology*. 176(10) pp.909–917.

<sup>313</sup> Mordock J, C8 suspected in birth defects: One woman's story, Delaware Online, April 2 2016. Accessed 12.10.2018.

<sup>314</sup> An overview of the probable link evaluations from the C8 Health Project can be found here

<sup>315</sup> Grice M *et al.* (2007). Self-reported medical conditions in perfluorooctanesulfonyl fluoride manufacturing workers. *Journal of Occupational and Environmental Medicine* 49:722–729.

<sup>316</sup> Steenland K and Woskie S (2012). Cohort mortality study of workers exposed to perfluorooctanoic acid. *American journal of epidemiology*. 176(10) pp.909–917.

information, the calculations assume that 3% to 10% of these plants use PFAS. As a last step, in order to generate the size of the exposed population, it was assumed that small plants had 30 workers on average while large plants had 300 workers on average. The population with occupational exposure in Europe was therefore estimated to range between 334,508 and 1,091,692 (see Annex 2 for more information).

The analysis assumed that these individuals suffered an elevated risk of mortality from kidney cancer as documented from the West Virginia study.<sup>317</sup> The number of additional deaths due to PFAS exposure in this population was then estimated to be between 3.6 and 11.8. These deaths were then monetised using the lower bound of the ECHA “value of a statistical life”.

Table 20 presents a summary of the findings, which give an indication as to the potential scale of the health impacts for the scenario of occupational exposure to PFAS. A key uncertainty in constructing these estimates was the number and distribution of worker exposure to PFAS.

**Table 20: Occupational exposure scenario: Monetised annual health impact for manufacturing worker exposure to PFAS**

Findings	Annual estimates
Exposed population in Europe	335 thousand to 1.1 million
Population experiencing elevated health risk	83,627–273 thousand
Deaths due to kidney cancer linked to PFAS exposure	3.6 to 11.8 lives lost
Value of life lost	EUR 12.7 million–41.4 million

Note: For more information on this calculation please refer to Annex 2.

### 5.1.2 Elevated (medium) exposure scenario

The case studies identify two populations that are at risk for elevated exposure to PFAS. The first population are communities that are in close proximity to chemical plants that produce PFAS or PFAS-treated products (Case Studies 1 and 2). Contaminated water from these plants may enter the drinking water system serving the communities. The second population are communities that live close to sites contaminated by aqueous fire-fighting foams (Case Study 3). PFAS in the foam can seep into the ground and groundwater, leading to contamination of local drinking water supplies.

A Swedish study found that up to 300,000 of residents in the country – or about 3% of the total population – are or have been exposed to levels of PFAS above the action value, via municipal drinking water.<sup>318</sup> The study highlights two main sources for the contamination: close proximity to a plant producing PFAS or PFAS-treated products, and close proximity to areas with high utilisation of aqueous fire-fighting

<sup>317</sup> Steenland K and Woskie S (2012). Cohort mortality study of workers exposed to perfluorooctanoic acid. *American journal of epidemiology*. 176(10) pp.909–917.

<sup>318</sup> Holmström *et al.* (2014). Nationell screening av perfluorerade föreningar (PFAA) i dricksvatten. Rapport no 2014/20 (In Swedish). It should be noted that 1.4 million of Sweden’s 9.9 million residents are not connected to a municipal water system. It is assumed nonetheless that all residents have contact with municipal waste water at least occasionally.

foams. These same individuals may also suffer higher exposure to PFAS due to contaminated surface water or air emissions.

The case studies provide other estimates for the number of individuals with elevated exposure. For example, Case Study 1 notes that 750,000 individuals living downstream from the Dordrecht Chemours plant had elevated exposure to PFAS via air and drinking water, and were advised to limit the consumption of vegetables grown in the area.<sup>319</sup> On the other hand, the affected population may be minimal if the surrounding vicinity has few inhabitants. For example, in the case of the 3M manufacturing plant in Antwerp, the PFAS contamination was considered to have concentrated on the area of the port where few people reside.

Elevated levels of PFAS in affected communities is highlighted in several studies (see Table 21). The blood serum concentration levels and the type of PFAS compounds, however, vary. Studies from the C8 Health Project in West Virginia found serum concentration levels of PFOA at 350 ng/ml among the nearby community.<sup>320</sup> The serum concentration level of PFOA in the general population in the United States was estimated to be 3.07 ng/ml in 2010.<sup>321</sup>

A study from a case of aqueous fire-fighting foam contamination in Sweden also found an elevated level of PFAS exposure among residents nearby.<sup>322</sup> Blood samples were taken from 3,660 persons, of whom 3,412 were in the contaminated area (Ronneby) and 242 people from a reference population in a nearby community (Karlshamn) that did not receive the contaminated water. Of the seven PFAS compounds identified in the contaminated water, three were identified at elevated levels in the blood serum of the sampled population and also elevated levels in the groundwater (above 90 ng/ml). The three main PFAS compounds identified in the blood serum were PFHxS, PFOA, and PFOS.

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<sup>319</sup>No Author, Another Chapter: Chemours releases GenX and PFOAs into waters globally, Encore, 08.08.2018. Accessed 10.10.2018.

<sup>320</sup>Frisbee S J *et al.* (2009). The C8 health project: design, methods, and participants. *Environmental health perspectives*, 117(12), 1873–82. DOI: <https://doi.org/10.1289/ehp.0800379>

<sup>321</sup>Center for Disease Control and Prevention (2018). Fourth National Report on Human Exposure to Environmental Chemicals. Updated Tables Volume1.

<sup>322</sup>Li Ying *et al.* (2017). Technical Report: Half-lives of PFOS, PFHxS and PFOA after end of exposure to contaminated drinking water.

**Table 21: Review of serum concentration levels (ng/ml) in contaminated and outside contaminated areas – three examples**

	Ronneby, Sweden (AFFF contamination)		West Virginia, USA (chemical plant)		Veneto Region, Italy <sup>1</sup> (chemical plant)	
	Outside contaminated area	Contaminated area	Outside contaminated area <sup>2</sup>	Contaminated area <sup>3</sup>	Outside contaminated area	Contaminated area
Sample size	242	3,418	2000+	32,254	250	257
Study year	2014–2016		2009–2014	2005–2006	2015–2016	
PFHxS	0.84	152	1.35	n.a.	n.a.	2.98
PFOA	1.59	10.4	3.07	32.9	0.01	13.77
PFOS	4.21	176	4.99	19.6	0.01	8.69

Source: 1) Ingelido A M *et al.* (2018). Biomonitoring of perfluorinated compounds in adults exposed to contaminated drinking water in the Veneto Region, Italy. *Environment international*, 110, pp.149-159.  
 2) Center for Disease Control and Prevention (2018). Fourth National Report on Human Exposure to Environmental Chemicals. Updated Tables Volume 1.  
 3) Frisbee S J *et al.* (2009). The C8 health project: design, methods, and participants. *Environmental health perspectives*, 117(12), 1873–82.

Epidemiological studies on the health impacts of elevated levels of exposure are available from West Virginia and the Veneto Region. One West Virginia study<sup>323</sup> found an increased risk of high cholesterol, while other studies found a higher risk of cancer (kidney and testicular) and hyperuricemia.<sup>324</sup> A study from the Veneto Region found that residents suffered an increased risk of overall mortality due to exposure to PFAS from a nearby manufacturing plant.<sup>325</sup>

The valuation for health-related costs for population of affected communities focused on the all-cause mortality endpoint using the increased risk factor found in the Veneto Region study.<sup>326</sup> The calculation relied on the 3% estimate from Sweden and assumed that the distribution of contaminated sites in Sweden is comparable to other European countries and that a similar share of the population is exposed at medium levels. In reality this is likely to be a low estimate – the prevalence of elevated exposure may be higher in countries with more manufacturers of PFAS-treated products and countries with higher population density. The assumption of 3% is however bolstered by findings from the United States where national drinking water monitoring data suggests that a higher share of the population (about 4.5%) is exposed to elevated levels of PFAS.<sup>327</sup>

<sup>323</sup> Steenland K *et al.* (2009). Association of perfluorooctanoic acid and perfluorooctane sulfonate with serum lipids among adults living near a chemical plant. *American journal of epidemiology*, 170(10), pp.1268–1278.

<sup>324</sup> Steenland K *et al.* (2010). Association of perfluorooctanoic acid (PFOA) and perfluorooctane sulfonate (PFOS) with uric acid among adults with elevated community exposure to PFOA. *Environmental health perspectives*, 118(2). p.229. and Barry V *et al.* (2013). Perfluorooctanoic acid (PFOA) exposures and incident cancers among adults living near a chemical plant. *Environmental health perspectives*, 121(11–12), p.1313.

<sup>325</sup> Mastrantonio M *et al.* (2017). Drinking water contamination from perfluoroalkyl substances (PFAS): an ecological mortality study in the Veneto Region, Italy. *The European Journal of Public Health*. Feb 1;28(1):180–185.

<sup>326</sup> *Ibid.*

<sup>327</sup> The Groundwater Association (2017). PFAS Top 10 Facts.

The calculation then assumes that the exposed adult population would face an elevated risk of mortality on the order of magnitude found in the Veneto region study. Using the baseline death rate reported by Eurostat<sup>328</sup>, the calculation suggests that more than 12,000 deaths annually could be attributed to elevated PFAS exposure in the EU with an estimated loss of EUR 43 billion. A similar calculation for only the Nordic countries<sup>329</sup> suggests that almost 600 deaths annually are linked to PFAS, with a total value of EUR 2 billion.

Table 22 presents a summary of the calculations, which indicate the potential scale of the health-related costs of elevated (medium) PFAS exposure in adults living in affected communities.

**Table 22: Monetised annual costs due to elevated risk of all-cause mortality for adults living close to PFAS contamination**

	Nordic countries	EEA countries
"Exposed" population (3%)	621 thousand	12.5 million
Annual deaths linked to PFAS	587–692	11,745–13,843
Valuation of life lost	EUR 2.1–2.4 billion	EUR 41.1–48.5 billion

Note: For more information on this calculation please refer to Annex 2.

Pregnancy outcomes may also be affected in communities with elevated exposure. As seen in Table 23 low birth weight (weight less than 2,500 grams) is a health endpoint pointed out in several studies. The EFSA report notes that *there is an overall tendency towards an inverse correlation between concentrations of PFOS/PFOA and birth weight*<sup>330</sup> whereas the five-year retrospective study conducted as part of the C8 Health Study in West Virginia<sup>331</sup> found that PFOS, but not PFOA, to be associated with low birthweight.

The prevalence of low birth weight in Europe is estimated to be 6.8%.<sup>332</sup> Assuming that 3% of births take place in areas with elevated (medium) levels of exposure to PFAS, an estimated 3,544 births in EEA countries are low birth weight due to exposure to PFAS. In the Nordic countries, the prevalence of low birth weight is less (4.57%); in applying the same relative risk function, the analysis suggests that 271 births each year in Nordic countries are low birthweight due to medium level PFAS exposure.

Low birth weight may be associated with a higher risk of developing diseases in adulthood such as cardiovascular disease, respiratory disease and diabetes.<sup>333</sup> Low birthweight

<sup>328</sup> Eurostat provides the rate of deaths per 100,000 for Europe. For the Nordic Countries, a weighted average was used.

<sup>329</sup> Aside from the smaller population/exposed population, the key difference is the lower death rate for the Nordic countries, compared to the overall EU death rate.

<sup>330</sup> EFSA CONTAM Panel (EFSA Panel on Contaminants in the Food Chain), Knutsen HK *et al.*, 2018. Scientific opinion on the risk to human health related to the presence of perfluorooctane sulfonic acid and perfluorooctanoic acid in food. EFSA Journal 2018;16(12):5194, 128 pp. DOI: <https://doi.org/10.2903/j.efsa.2018.5194>

<sup>331</sup> Stein C R *et al.* (2009). Serum levels of perfluorooctanoic acid and perfluorooctane sulfonate and pregnancy outcome. American journal of epidemiology, 170(7), pp.837–846.

<sup>332</sup> OECD, 2016. Health status statistics.

<sup>333</sup> Almond D and Currie J (2011). Killing me softly: The fetal origins hypothesis. Journal of economic perspectives, 25(3), 153–72. And Bharadwaj P *et al.* (2017). Birth Weight in the Long Run. Journal of Human Resources, 53(1), 189–231.

is also associated with other important outcomes such as impaired cognitive development. For example, one study found that low birth weight was associated with a 25% lower likelihood of passing high school exit exams and a higher risk of unemployment at age 33 years.<sup>334</sup> Other studies have found that birth weight is positively associated with earnings.<sup>335</sup> For example, one study found that low birth weight was associated with lower income for men 30 years of age and for women between 50 and 60 years of age.<sup>336</sup> The researchers found that part of this effect could be explained by a higher use of sick leave, which suggests a higher susceptibility to illness. This increased risk of illness could be relevant for 271 low birth weight persons born each year in the Nordic countries and for the estimated 3,544 low birth weight persons born each year in EEA countries.

**Table 23: Number of births per year that are low birth weight in areas close to PFAS contamination**

	Nordic countries	EEA countries
Births in "exposed" areas	8,843 births	156,344 births
Births of low birth weight linked with PFAS	271 births	3,544 births

Note: For more information on this calculation please refer to Annex 2.

### 5.1.3 Background (low) exposure scenario

PFAS are widely present in consumer goods as highlighted in Case Study 3 (see Section 3.3). Consumer goods can contribute to human exposure to PFAS through several pathways. First, it can enter humans through direct hand-to-mouth transfer through products such as food contact materials. Second, PFAS-treated consumer goods like cosmetic products may end up in wastewater that then affects the water supply.

Some research suggests that children have an elevated exposure to PFAS and that this is due to frequent hand-to-mouth transfer and proximity to dust on the floor.<sup>337, 338</sup> Young children may also have greater exposure to PFAS-treated carpets.<sup>339</sup> While this conclusion of greater exposure for children is not supported by population-level bio-

<sup>334</sup> Currie J and Hysom R (1999). Is the impact of health shocks cushioned by socioeconomic status? The case of low birth-weight. *American Economic Review*, 89(2), 245–250.

<sup>335</sup> Black S *et al.* (2007). From the cradle to the labor market? The effect of birth weight on adult outcomes. *The Quarterly Journal of Economics*, 122(1), 409–439 and Bharadwaj P *et al.* (2017). Birth Weight in the Long Run. *Journal of Human Resources*, 53(1), 189–231.

<sup>336</sup> Bharadwaj P *et al.* (2017). Birth Weight in the Long Run. *Journal of Human Resources*, 53(1), 189–231. Table F.

<sup>337</sup> Winkens K *et al.* (2017). Early life exposure to per- and polyfluoroalkyl substances (PFASs): A critical review. *Emerging Contaminants*. 3: 55–68. DOI: <https://doi.org/10.1016/j.emcon.2017.05.001>

<sup>338</sup> Rappazzo K *et al.* (2017). Exposure to perfluorinated alkyl substances and health outcomes in children: a systematic review of the epidemiologic literature. *International journal of environmental research and public health*, 14(7). p. 691. Jun 27;14(7). pii: E691. DOI: <https://doi.org/10.3390/ijerph14070691>

<sup>339</sup> Trudel, D., Horowitz, L., Wormuth, M., Scheringer, M., Cousins, I. T., & Hungerbühler, K. (2008). Estimating consumer exposure to PFOS and PFOA. *Risk Analysis: An International Journal*, 28(2), 251–269.

monitoring data from the United States for three PFAS compounds (see Table 24) nevertheless a low level exposure to chemicals at critical periods of development such as infancy and childhood may have serious, irreversible impacts.<sup>340</sup>

**Table 24: Serum concentration levels by age level (USA, 2013–2014)**

	PFNA	PFOA	PFOS	Sample size
3–5 years	0.764	2.00	3.38	181
6–11 years	0.809	1.89	4.15	458
12–19 years	0.599	1.66	3.54	401
20 years and older	0.685	1.98	5.22	1,764

Source: CDC, 2018. Fourth National Report on Human Exposure to Environmental Chemicals. Updated tables, Volume 1.

A review of studies on the health impacts of exposure to PFAS among children identifies four key health endpoints<sup>341</sup>, namely high cholesterol, depressed immunity (including lower vaccine response and higher risk of asthma), reduced renal function, and younger age at menarche. These endpoints are reflected in the overall review of endpoints presented in Table 25.

It is challenging to monetise the increased risk of these health endpoints among children. It requires a life course view and a clear understanding of the complex relationships between the endpoints, other factors and the consequent impacts on development and well-being. For example, reduced renal function among children is associated with having delayed motor skills and language development, and trouble with concentration and self-esteem. Such issues could have cost implications in terms of more doctor visits, as well as poorer educational achievement. Among the four conditions mentioned above, depressed immunity was found to be the most amenable to quantification. As stated in Rappazzo *et al.*, 2017, *the studies of vaccine response were well done cohort study designs and despite the small number offer compelling evidence. The asthma studies are less consistent and include a broader range of study designs and quality.*<sup>342</sup>

Several regulatory bodies highlight immunotoxicity as a likely health consequence of PFAS exposure (e.g. EFSA, ATSDR and US-EPA). However, a closer review of the evidence for specific endpoints presents a more mixed picture. For example, the US-EPA validation studies conclude that the relationship between prenatal and maternal exposure to PFOA and PFOS and infectious disease is limited. While some studies identified a relationship with common childhood infections such as otitis media, allergies, common colds and the flu, the evidence was not consistent. For example, the C8

<sup>340</sup> As stated in Rappazzo *et al.* (2017): *It is increasingly understood that exposure to environmental chemicals during sensitive windows has the potential to permanently alter a child's risk of future morbidity, even at doses that have little effect in adults.*

<sup>341</sup> Rappazzo K *et al.* (2017). Exposure to perfluorinated alkyl substances and health outcomes in children: a systematic review of the epidemiologic literature. *International journal of environmental research and public health*, 14(7). p.691. Jun 27;14(7). pii: E691.

<sup>342</sup> Ibid.

Science Panel concluded that there is no probable link between PFOA and common infections.<sup>343</sup> If greater susceptibility to illness is indeed a health endpoint, PFAS exposure may then be linked to costs related to more doctor’s visits, fewer days attending school and lower workplace engagement for caretakers. Increased sickness among children may be due to a wide range of factors other than PFAS exposure and thus it would be important to base any estimation on studies with a robust design.

A Danish study explored the potential scale of the immunotoxicity endpoint. The study found that higher prenatal exposure to PFOS was associated with more days of fever among children ages 1 to 4 years.<sup>344</sup> For this study a calculation was made assuming that the level of PFAS exposure and the level of risk of fever could be directly extrapolated to the Nordic and the EEA countries. The estimated number of days of fever that may be attributable to PFAS exposure are presented below.

**Table 25: Additional fever days among children ages 1–4 years due to PFAS exposure**

	Nordic countries	EEA countries
“Exposed” children	45,229	784,794
Fever days in exposed population – overall	212,576	3,688,533
Fever days in exposed population – linked to PFAS exposure	83,742	1,453,059

Note: For more information on this calculation please refer to Annex 2.

#### 5.1.4 Background exposure (low) scenario

Discharges of PFAS from waste water treatment plants and landfills may lead to contamination of food and water, which are the two main sources of exposure to PFAS.<sup>345</sup> Case Study 5 (section 3.5) notes that PFAS released from products in landfills can migrate directly into soil and groundwater. The discharge may include a high level of long-chain PFAS compounds such as PFOA even if their production has been restricted in recent years. Contaminated water can travel long distances and be taken up in agricultural produce and drinking water, potentially affecting all persons in Europe. Lastly, evidence suggests that wastewater treatment is not effective at fully removing PFAS within the general population.

This study assessment found that exposure to PFAS through the pathway of waste water treatment plants and landfills may be most closely linked to background levels of PFAS in drinking water and food. Other factors may contribute to background levels of PFAS, for example, the disposal of consumer goods and exposure to dust.

The blood serum concentration of European populations with background levels of exposure to PFAS is not well-known. Some biomonitoring studies have been conducted

<sup>343</sup> Environment Protection Agency (2016). Health effects support document for perfluorooctanoic acid (PFOA). Document no: EPA822R16003 and Health effects support document for perfluorooctane sulphate (PFOS). Document No: EPA822R16002.

<sup>344</sup> Dalsager L *et al.* (2016). Association between prenatal exposure to perfluorinated compounds and symptoms of infections at age 1–4 years among 359 children in the Odense Child Cohort. *Environment international*, 96, pp.58–64.

<sup>345</sup> Trudel D *et al.* (2008). Estimating Consumer Exposure to PFOS and PFOA. *Risk Analysis: An International Journal* 28.2: 251–269.

among small populations as shown in Table 26. While the generalisability of these findings is limited due to the small sample sizes, the findings nevertheless provide insight into the levels of background PFAS exposure in different contexts.

**Table 26: Cross-country estimates of background PFAS levels in blood serum of adult populations**

	Italy <sup>1</sup>	Norway <sup>2</sup>	Sweden <sup>3</sup>	Belgium <sup>4</sup>	Spain <sup>5</sup>	Germany <sup>6</sup>	Greece <sup>7</sup>	USA <sup>8</sup>
Year	2015–2016	2006	1997–2000	1998, 2000	2006	2005	2008	2009–2014
Sample size	257	57	66	20	48	356	86	2,000+
PFOA	1.64	2.7	5.0	4.1	3.4	13.7	2.05 (male) 1.92 (female)	3.07
PFNA	0.58	0.55	--	--	--	--	--	0.675
PFDA	0.32	0.22	--	--	--	--	--	0.185
PFUnDA	0.18	0.14	--	--	--	--	--	--
PFDoDA	0.04	--	--	--	--	--	--	--
PFHxS	2.49	1.4	3.0	1.3	5.8	--	--	1.35
PFOS	5.84	12	34.2	17.2	15.2	5.7	13.63 (male) 9.28 (female)	4.99

Source: 1) Ingelido A M *et al.* (2018). Biomonitoring of perfluorinated compounds in adults exposed to contaminated drinking water in the Veneto Region, Italy. *Environment international*, 110, pp.149–159.  
 2) Haug L S *et al.* (2009). Time Trends and the Influence of Age and Gender on Serum Concentrations of Perfluorinated Compounds in Archived Human Samples. *Environ. Sci. Technol.* 43:6, 2131–2136.  
 3) Kärrman A *et al.* (2004). Levels of perfluoroalkylated compounds in whole blood from Sweden. *Organohalogen Compd.* 66, 4008–4012.  
 4) Kannan K *et al.* (2004). Perfluorooctanesulfonate and related fluorochemicals in human blood from several countries. *Environ. Sci. Technol.* 38, 4489–4495.  
 5) Ericson I *et al.* (2007). Perfluorinated chemicals in blood of residents in Catalonia (Spain) in relation to age and gender: a pilot study. *Environ. Int.* 33, 616–623.  
 6) Holzer J *et al.* (2008). Biomonitoring of perfluorinated compounds in children and adults exposed to perfluorooctanoate (PFOA) contaminated drinking water. *Environ. Health Perspect.* 116, 651–657.  
 7) Holzer J *et al.* (2008). Biomonitoring of perfluorinated compounds in children and adults exposed to perfluorooctanoate (PFOA) contaminated drinking water. *Environ. Health Perspect.* 116, 651–657.  
 8) Center for Disease Control and Prevention (2018). Fourth National Report on Human Exposure to Environmental Chemicals. Updated Tables Volume 1.

In contrast to these small-scale biomonitoring studies, a large, nationally-representative population-based survey that includes biomonitoring is conducted every two years in the United States. The survey is known as the National Health and Nutrition Examination Survey (NHANES). One of the survey modules involves a health examination and the collection of a blood sample. The blood samples are analysed for a wide range of chemicals including PFAS. One analysis concluded that the levels of

PFOS, PFOA and PFHxS in blood serum in the United States were comparable to several European countries.<sup>346</sup> This conclusion supports the transferability of findings from studies conducted in the United States on the health impacts of background exposure to PFAS to the European context.

Several analyses using the NHANES data suggest that PFAS exposure at background levels can increase the likelihood of cardiovascular disease. One study found a positive correlation with uric acid<sup>347</sup> while another found an association with total cholesterol<sup>348</sup>, which are both risk factors for cardiovascular disease. Another study found an association between PFOA and risk of developing hypertension.<sup>349</sup> The level of consensus was found to be highest for serum cholesterol. The epidemiological evidence relating elevated cholesterol and cardiovascular disease, however, is quite mixed. Another complicating factor is that studies investigating PFAS exposure do not assess the relative risk of cholesterol over the acceptable thresholds. Rather, they provide estimates for the elevation of cholesterol, which may remain below the acceptable threshold.

Due to challenges related to the serum cholesterol endpoint, the quantitative assessment focused on hypertension. The risk relationship between PFAS and hypertension was based on findings from a study from the United States (see Table 27).<sup>350</sup> Individuals with a systolic blood pressure greater than 140 mm Hg, a diastolic blood pressure greater than 90 mm Hg or a self-reported medical diagnosis were considered to have hypertension.

**Table 27: Risk of developing hypertension as a function of exposure to PFOA contamination**

Serum PFOA concentration	Odds-ratios (95% confidence interval)
Quartile 1 (<2.6 ng/ml)	1 (Reference)
Quartile 2 (2.7–3.9 ng/ml)	1.24 (0.89–1.74)
Quartile 3 (4.0–5.5 ng/ml)	1.63 (1.20–2.20)
Quartile 4 (>5.6 ng/ml)	1.60 (1.15–2.22)

Note: The analysis of hypertension is based on a sample of 2,208 adults (20 years and older) who provided a blood sample between 2003 and 2006 to the National Health and Nutrition Examination Survey.

Source: Min *et al.*, 2012.

<sup>346</sup> Kato K *et al.* (2011). Trends in exposure to polyfluoroalkyl chemicals in the US population: 1999–2008. *Environmental science & technology*, 45(19), pp.8037–8045.

<sup>347</sup> Shankar A *et al.* (2012). Perfluorooctanoic acid and cardiovascular disease in US adults. *Archives of Internal Medicine*, 172(18), pp.1397–1403.

<sup>348</sup> Nelson J *et al.* (2010). Exposure to polyfluoroalkyl chemicals and cholesterol, body weight, and insulin resistance in the general US population. *Environmental health perspectives*, 118(2), 197.

<sup>349</sup> Min J Y *et al.* (2012). Perfluorooctanoic acid exposure is associated with elevated homocysteine and hypertension in US adults. *Occup Environ Med*, 69(9):658–62.

<sup>350</sup> *Ibid.*

The analysis assumes that the entire adult population in Europe is exposed to background level exposure of PFOA through drinking water, and that about half have a level of exposure that is associated with a higher risk of developing hypertension. The latter assumption follows from a finding from the US study, where the risk for developing hypertension was elevated for individuals in the highest two quartiles of exposure. Several additional assumptions were made. For example, an estimated 6.1 million new cases of cardiovascular disease were diagnosed in the EU in 2015<sup>351</sup>, of which about half were assumed to be specifically related to hypertension.<sup>352</sup> The assessment then considered the increased risk of mortality due to hypertension.<sup>353</sup> These assumptions are presented in greater detail in Annex 2 and the findings are presented in Table 28. The analysis finds an estimated 12,655 to 41,417 cases of hypertension linked to PFAS exposure in the Nordic countries and about 153 to 500 deaths linked to hypertension and PFAS exposure. The estimated number of deaths that could be attributed to PFAS exposure in the EEA countries ranged from 3,066 to 10,035. Key uncertainties in developing these estimates is the underlying risk relationship between PFAS exposure and hypertension and the elevated risk of mortality associated with hypertension.

**Table 28: Monetised health impact (EUR) for background PFAS exposure leading to increased risk of developing hypertension**

	Nordic countries	EEA countries
Population at elevated risk of hypertension	10.3 million	207.8 million
Cases of hypertension linked to PFAS	12,655–41,417	254,167–831,818
Deaths linked to hypertension and PFAS	153–500	3,066–10,035
Valuation of life lost	EUR 687 million–2.2 billion	EUR 10.7 billion–35 billion

Note: For more information on this calculation please refer to Annex 2.

These figures do not capture all the costs associated with hypertension. Hypertension can also lead to other costs including health care costs, productivity lost and the cost of uncompensated care.<sup>354</sup> Moreover, in general, these figures do not include utility costs, i.e. the benefits of being fit and well and enjoying life to the fullest.

<sup>351</sup> European Heart Network (2017). European Cardiovascular Disease Statistics 2017.

<sup>352</sup> In the same report it is stated that high systolic blood pressure contributes for about half of all cardiovascular diseases.

<sup>353</sup> Zhou *et al.* (2018). Uncontrolled hypertension increases risk of all-cause and cardiovascular disease mortality in US adults: the NHANES III Linked Mortality Study. Scientific reports, 8(1), p.9418.

<sup>354</sup> European Heart Network (2017). European Cardiovascular Disease Statistics 2017. An estimated 53 percent of the cost of cardiovascular disease (which includes hypertension) in the EU is accounted for by health care costs (EUR 111 billion), while 26 percent is due to productivity loss (EUR 54 billion) and the remainder is due to the provision of uncompensated care (EUR 45 billion). CVD can account for about 19 percent of all DALYs lost in the EU annually.

### 5.1.5 Summary of health-related costs of exposure to PFAS

Table 29 presents an overview of the preliminary quantitative assessments of the health-related costs from PFAS exposure by the source.

**Table 29: Health-related costs (of exposure to PFAS)**

Exposure level	"Exposed" population and source	Health end-point	Nordic countries		All EEA countries	
			Population at risk	Annual costs	Population at risk	Annual costs
Occupational (high)	Workers at chemical production plants or manufacturing sites	Kidney cancer	n.a.	n.a.	84–273,000	EUR 12.7–41.4 million
Elevated (medium)	Communities near chemical plants, etc. with PFAS in drinking water	All-cause mortality	621,000	EUR 2.1–2.4 billion	12.5 million	EUR 41–49 billion
		Low birth weight	8,843 births	136 births of low weight	156,344 births	3,354 births of low weight
		Infection	45,000 children	84,000 additional days of fever	785,000 children	1,500,000 additional days of fever
Background (low)	Adults in general population (exposed via consumer products, background levels)	Hypertension	10.3 million	EUR 0.7–2.2 billion	207.8 million	EUR 10.7–35 billion
<b>Totals</b>			<i>Nordic countries</i>	<i>EUR 2.8–4.6 billion</i>	<i>All EEA countries</i>	<i>EUR 52–84 billion</i>

### 5.2 Non-health costs of environmental contamination with PFAS

Environment-related costs are considered here to cover the following elements:

1. Monitoring to assess PFAS contamination where it is suspected.
2. Provision of a temporary uncontaminated drinking water supply.
3. Upgrading of water treatment works and ongoing costs for maintenance and replacement and disposal of filters.
4. Excavation and treatment of soils.
5. Health assessments where contamination is found (health management costs, rather than costs of damage to health).
6. Impacts on biodiversity.

Data collected during the study on elements 1 to 5 of this list are provided in Annex 3, Part 1, with summary estimates provided in this Section. The Annex also includes information on the Purchasing Power Parity (PPP)-adjusted exchange rates used and factors applied to account for inflation.

### 5.2.1 *Impacts on, and public aversion to, risks to the natural environment*

Direct assessment of impacts on biodiversity is not possible, given a lack of data on the stock at risk, exposure-response, and other elements of the analysis. However, it is noted that various ecological impacts may be associated with the release of PFAS, ranging from impacts to the endocrine and immune systems of animals to restrictions on human fishing activity (e.g. in the cases of the contamination at Schiphol Airport and at Arnsberg in Germany, referred to above). For contamination of the River Rhine from Dusseldorf Airport, affected usages have been listed as being associated with the water works, local water rights including private wells, anglers, surfing club, and agricultural uses.<sup>355</sup>

For specific cases it may be possible to value elements such as lost fishing opportunity. Valuation data were generated for a specific case in the USA by Sunding as noted above, but this requires information on levels of fishing activity, the extent to which recommendations not to consume locally caught fish affects angling activity, the duration of any such recommendation, etc. and is beyond the scope of a generic study such as this. It is, however, important to note that Sunding's estimate based on willingness to pay to avoid fishing in PFOS contaminated surface waters for the one case of contamination arising from 3M's disposal of PFCs in Washington County, Minnesota amounted to the equivalent of over EUR 90 million for the period 2008–2040. This supports the view that loss of amenity associated with contamination of fish purely from the perspective of anglers is substantial.

Section 3.2.3 reported on a UK study undertaken to inform the development of the REACH Restriction on D4 and D5 in wash-off personal care products. This found a willingness to pay of EUR 46 per year per person to reduce the risks associated with the PBT substance – D4, and EUR 40 per year per person to reduce the risks associated with the vPvB substance – D5. The results of the survey could be used to provide an estimate of the total willingness to pay to avoid contamination with PBT/vPvB substances including PFAS. Such a result could be useful in the context of evaluation of a REACH Restriction or Authorisation where the costs of alternatives are substantially less (e.g. an order of magnitude) than the WTP estimate, or where the WTP for specific beneficial properties associated with the substance under investigation are also provided for comparison (as was the case with the D4/D5 Restriction).

For the purpose of the present study it seems better not to include the figures given the uncertainties that are associated with them, but to acknowledge firstly that economic estimates exclude ecological damage, and secondly that the costs linked to aversion to PBT and vPvB substances are likely to be substantial. The results of the UK study applied at a European scale suggest this aversion has an economic cost running into the billions of Euro.

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<sup>355</sup> Weber R (2016). Some lessons learned from PFOS/PFAS management in Germany.

### 5.2.2 Summary of the non-health unit costs associated with environmental contamination with PFAS

Data from Annex 3, Part 1 on the non-health costs associated with environmental contamination with PFAS have been reviewed and collated to provide best estimates and associated ranges, see Table 30. The logic for defining best estimates and ranges is discussed below. Where possible, estimates are provided as cost per person to enable aggregation at a later stage.

About half of the data that has been collected comes from the USA, raising questions about its transferability to Europe. Inspection of the data suggests that there is broadly as much variation within the European and US data sets, so both are included. Comparison of the US and European data could be taken to indicate that attitudes to risk aversion in the two regions are broadly similar (acknowledging the high level of variation in both the European and US datasets).

An important factor constraining the ranges (even though many are broad) is the fact that when scaling up from information collected in the case studies, the analysis needs to adopt representative best estimates and associated ranges around those best estimates. The costs of individual schemes may well lie outside these ranges, but the analysis needs to deal with average costs, not the extremes from specific cases which by their nature would lead to an over- or under-estimation of costs.

**Table 30: Summary of cost data for non-health expenditures. For units, see second column**

Activity	Unit	Best estimate	Range from studies	Adopted range
Monitoring	Cost/sample	EUR 340	EUR 278–402	EUR 278–402
	Cost/case	EUR 50,000	EUR 4.0 thousands –6.1 million	EUR 25 thousands –500 thousands
Health assessment (including biomonitoring)	Cost/person	EUR 50	No range	EUR 5–95 (+/-90%)
	Total biomonitoring and health assessment per case where it is considered appropriate	EUR 3.41 million	EUR 2.5 million –4.3 million	EUR 1 million –5 million
Provision of temporary uncontaminated supply	Cost/person	No relevant data: Hoosick Falls information rejected as it does not appear to be for a true “temporary” solution (see text)		
Provision of a new pipeline	Cost/person	EUR 800	EUR 37–5,000	EUR 500–1,500
Upgrading water treatment works (capital)	Cost/person	EUR 300	EUR 8–2,200	EUR 18–600
Upgrading water treatment works (maintenance)	Cost/person	EUR 19	EUR 8–30	EUR 8–26
Excavation and treatment of soils	Cost/kg PFAS	EUR 280,000	EUR 100 thousands –4.3 million	EUR 100 thousands –1 million
	Cost/case	EUR 5 million	EUR 100 thousands –3 billion	EUR 300 thousands –50 million

With respect to monitoring, costs per sample concern the collection of individual samples of groundwater (etc.) and the analysis of those samples. Associated costs do not account

for additional monitoring related activities, such as management of the monitoring regime. The reported costs per sample for monitoring were from a single study, but provided with a range indicating economies of scale as more monitoring was done. Relative to other parameters the range for monitoring is narrow, and the mid-point is adopted as the best estimate. A more important question for the analysis concerns how much monitoring would need to be carried out in total and in any country and with what frequency. This could vary from none or a few samples to several thousand. Figures expressed as average monitoring cost per case could therefore be more reliable. The range here is much larger, reflecting differences in the degree of contamination, and the extent of the population affected. Issues relating to the data will concern the activities included under "monitoring": in some cases these will cover only sampling and analysis, whilst in others they may include development of plans for public protection as well.

Two sources of information concerning the costs of carrying out health assessments are considered<sup>356</sup>, WHO's evaluation of a health assessment scheme around the Veneto site of contamination, and biomonitoring costs incurred around Ronneby Airport in Sweden. Costs in both cases run into the millions of EUR. It is unknown how representative these cases are, but it is possible (given that this cost category has not been identified for other cases) that they are higher than similar costs incurred elsewhere if indeed such activities have been undertaken. For the Ronneby case there is no information on the scale of the biomonitoring undertaken, whereas WHO provide data on the number of individuals potentially affected in Veneto. Accordingly, the health assessment costs are taken from the Veneto case and cover surveillance of the population once significant exceedance has been observed. The Ronneby data are useful for indicating that where health assessment of some kind is considered appropriate, costs can run into millions of EUR.

The best estimate is equal to the figure derived from the Veneto data (in this case, the number of people factored into the calculation of the average cost per person was the total exposed population, not the number of individuals undergoing testing of any kind). The extent of health assessment could vary significantly, theoretically from none at all to detailed and regular assessment of all exposed individuals. This variation could reflect national attitudes to pollution, public concern over a particular incident, the extent of exceedance of limit values, etc. A range of +/-90% is adopted in the costs analysis, reflecting possible variability.

Two cases were identified for the costs of providing a temporary uncontaminated supply, from Hoosick Falls, New York State and Peterson, Colorado, both in the USA. Hoosick Falls was home to the Saint-Gobain Performance Plastics and Honeywell Manufacturing Plant, whilst Peterson contains a US Air Force Base. For Hoosick Falls a 'temporary filtration system' was installed. Associated costs also include investigation of alternative water sources. The Hoosick Falls estimate of EUR 7.4 million for provision of a temporary water filtration system is of a similar magnitude to the costs reported elsewhere for a permanent system. This suggests that the responsible authorities are seeking an alternative source with no contamination but that this will take several years to

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<sup>356</sup> The health assessment costs are considered in this section, rather than Section 5.1 as they relate to management of problems of PFAS contamination rather than health impacts.

come online. In the meantime they have introduced a system that is similar to the permanent solutions elsewhere. Bottled water has been supplied to residents in both areas. A cost of EUR 81,000 equivalent is cited for Peterson, but no cost data for bottled water have been identified for Hoosick Falls.

Considering estimation of the costs of provision of temporary clean water supplies elsewhere, it is noted that costs will vary substantially according to the following factors:

- the number of people served by a scheme;
- the duration over which alternative supplies are required; and
- the form of the temporary measures that are put in place (e.g. additional filtration, provision of bottled water, provision of water tankers, temporary piping).

On the basis that PFAS contamination will not be resolved quickly, the duration over which alternative supplies are required seems likely to be in the order of months or years rather than days or weeks. This in turn starts to rule out very prolonged use of some of the quick fixes such as provision of bottled or tanked water.

Four of the incidents for which cost data were obtained provide estimates indicating the costs of providing permanent new pipelines, these being the cases for Jersey, Kallinge, Stadtwerke Rastatt and Veneto. There are two orders of magnitude difference in the reported costs which show a strong dependence on the number of people presumed to be affected. Factors affecting costs include:

- whether a new connection is made to a single point that goes on to serve a larger area, as assumed for Rastatt, or to individual buildings as was the case for Jersey;
- the number of people affected;
- the distance over which new pipework is required; and
- complications in laying pipes associated with geology and local infrastructure

The costs in Jersey are significantly higher than the other two locations, reflecting the small number of houses affected and the need to connect to individual homes rather than a single point. The likely range for an average cost per person is taken from the lower bound to the upper bound of costs excluding Jersey (EUR 100–EUR 1,500/person) with the best estimate taken as the mid-point of this range (EUR 800).

For upgrading of water treatment plant the cost range is again broad, EUR 8 to EUR 2,200 when normalised against population (the "high" estimate is notably the supposed cost of the temporary solution at Hoosick Falls). Excluding the two highest and lowest values gives a reduced range of EUR 18 to EUR 940/person, with remaining values spread rather evenly over this range. Taking the mean of the 9 data points excluding the highest and lowest gives a best estimate of EUR 300/person.

For maintenance costs, there are four estimates for which normalisation against population has been performed, from EUR 8/person/year to EUR 30/person/year. The range is adopted from these studies and the best estimate (EUR 19) is taken as the midpoint.

For excavation of soils the range is again broad, with costs per kg of PFAS of EUR 100,000, EUR 200,000–280,000 and EUR 4.3 million. It is unclear whether all of these costs can be attributed to PFAS or to other contaminants.

Soil remediation costs can also be expressed on a per case basis. The range is again very broad, EUR 100,000 to EUR 3 billion, the very high upper bound referring to possible costs at Rastatt in Germany.<sup>357</sup> Adopting any value within this range is prone to very high uncertainty, though for the purpose of illustration, a best estimate of EUR 5 million/case is taken, with a range for the main cost of EUR 300,000 to EUR 50 million. Definition of the upper bound cost for soil remediation is extremely difficult, given the extreme variability in the cost data identified (see Annex 3). Most cases identified had costs ranging from EUR 1 million to EUR 10 million, but there are several that are substantially higher (Schiphol at EUR 30–40 million, Dusseldorf Airport where costs are estimated at up to EUR 100 million, and Baden-Wurtemberg where costs are estimated between EUR 1 and 3 billion).

A number of other costs have also been identified for individual case studies, such as lost opportunity from closure of a borehole (Buncefield, UK), fees and capital works at specific sites not specifically involved in water treatment (Jersey) and risk analysis and project management (Uppsala). In each case, the total costs under this “other” category are substantial, covering a range of EUR 320,000 to EUR 6.3 million. Whilst no attempt is made to extrapolate these figures, given that they are only mentioned for single locations, they provide further evidence that the costs associated with remediation of PFAS contamination are large.

### 5.2.3 *Aggregating the costs of environmental contamination with PFAS*

A first step in aggregation is to simply combine the results for each European country as provided in Annex 3 part 1 (see Table 31 below). The results given do not represent an estimate of total damage for any country, as such a total has not been estimated for any country. The country for which data appear most complete is Sweden, where significant contamination has been found at 20% (7) out of the country’s 35 airports (military and civilian combined). This figure of 20% is carried forward to the analysis that follows. The country for which highest costs are estimated is Germany, where the total is almost entirely due to soil remediation in Baden-Wurtemberg, which is understood to follow the use of waste material as a soil treatment on agricultural land.<sup>358</sup> It is further understood that the remediation of the soils in question has not been performed, so the cost estimate must be considered theoretical. The results demonstrate a variation in the estimated costs by around 2 orders of magnitude.

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<sup>357</sup> <http://greensciencepolicy.org/wp-content/uploads/2016/09/Rolland-Weber-PFOS-PFAS-German-activities-Final.pdf>

<sup>358</sup> Reuning A, Landschaft mit Gift, Deutschland Funk, 23.04.2017. Accessed 10.11.2018.

**Table 31: Summation of the costs identified in the available literature, EUR millions by country. Figures in brackets indicate the number of cases or plant for which data were collected**

Total costs	DK	DE	IT	NL	NO	SE	UK
Monitoring		EUR 7.8 (2)			EUR 0.50 (2)	EUR 2.6 (7)	
Upgrading treatment works		EUR 104 (2)	EUR 2.1 (1)			EUR 5.3 (5)	
Install new pipelines		EUR 1.81 (1)				EUR 11 (2)	EUR 1.0 (1)
20 year maintenance cost of water treatment works discounted at 4%		EUR 11 (1)				EUR 1.6 (1)	EUR 13 (1)
Soil remediation costs	EUR 15	EUR 3,112 (2)		EUR 35 (1)	EUR 4.1 (1)	EUR 2.5 (3)	EUR 7.1 (1)
Biomonitoring						EUR 2.6 (1)	
Other quantified costs						EUR 0.38 (1)	EUR 12 (2)
<b>Total quantified cost</b>	<b>EUR 15</b>	<b>EUR 3,236</b>	<b>EUR 2.1</b>	<b>EUR 35</b>	<b>EUR 4.6</b>	<b>EUR 26</b>	<b>EUR 33</b>
Sites affected	Copenhagen (AP)	Dusseldorf (fire), Rastatt (waste)	Veneto industry	Schiphol (AP)	Oslo Fjord, Tyri-fjorden	Arlanda, Bromma, Kallinger, Kiruna, Ronneby, Umea, Uppsala (all APs)	Buncefield (fire), Jersey (AP)

The results shown in Table 31 are incomplete because they omit most European countries and are based on limited knowledge of contamination across the continent. As such they describe the absolute minimum for addressing the PFAS problem to the extent that they have been incurred (noting the discussion above concerning the Baden-Wurttemberg case where soil remediation is still to take place). As a minimum estimate the figures are clearly not reliable. In the absence of a European wide systematic screening programme it is unlikely that all cases of contamination have been identified.

#### 5.2.4 Number of sites releasing PFAS

Aggregation of the cost data to provide some estimate of damage at Nordic and EU scales requires additional data, provided in Annex 3, Part 2, covering:

- population
- water consumption
- quantity of water supplied from groundwater and surface water
- number of wastewater treatment plant (WWTP)
- number of plant or sources providing drinking water
- number of PFAS producers

- number of companies potentially using PFAS
- number of airports
- number of landfill and incineration sites.

Information covers the EU28, Norway and Iceland to the extent that data are available. Data for the USA are also included for reference, given that much of the information used in this report is of US origin. A summary of the number of sites releasing or potentially releasing PFAS is provided in Table 32 below, drawing on the information in Annex 3, Part 2.

**Table 32: Number of installations working in the sectors that may use or emit PFAS for the EEA. Figures in brackets represent businesses with more than 250 employees**

Sector	Activity	Total
Waste water treatment plant	T1 (primary treatment)	7,279
	T2 (secondary treatment)	24,316
	T3 (tertiary treatment)	19,716
	Of which T3N (T3 + nitrogen removal)	11,502
	Of which T3P (T3 + phosphorus removal)	10,436
Drinking water treatment	Large	Thousands
	Small	Ten thousands
	Very small	Hundred thousands
Aviation	Main passenger airports	318
	Medium passenger airports	137
	Small airports	no data
	Military airbases	239
Other fire control	Fire stations	84,099
	Site emergency services	no data
Waste	Hazardous waste landfill	365
	Non-hazardous waste landfill	3,801
Manufacturing industry	Large incineration (as energy from waste)	808
	PFAS manufacturers <sup>1</sup>	12–20
	Textiles	61,685 (262)
	Leather	37,120 (159)
	Carpet	no data
	Paper	19,477 (488)
	Paints and varnishes	4,027 (104)
	Cleaning products	(178)
	Cosmetics and personal care	no data
	Electronics	no data
	Photography films	no data
	Metal treatments	151,455 (163)
	Car washes	79,000
Mining	no data	
Plastic, resins, rubbers	(340)	

Note: 1) It has not proven possible to identify the European PFAS manufacturers with confidence. From data collected, it is assumed that there are between 12 and 20 sites, best estimate 16, distributed as follows: Belgium (2), Czechia (1), France (3), Germany (3), Italy (2), Netherlands (1), Poland (1), UK (3).

Not all of these enterprises, particularly in manufacturing, produce, use or emit PFAS. However, it can be concluded that the total number of sites emitting PFAS in some quantity could be in the order of 100,000 or more for the EEA. Information presented in Annex 3 demonstrates that these activities are not concentrated in a few countries but are spread throughout the region.

### 5.2.5 *Extrapolation of costs, by country*

Noting the uncertainties identified elsewhere in this report, a precise quantification of costs associated with PFAS contamination is not possible. Estimation of damage is, however, still an important exercise as it provides opportunity to describe the likely magnitude of economic damage. With this in mind, a scenario based assessment has been carried out.

The following elements are estimated, with results in this section provided for each of the Nordic countries and the EU28+Switzerland combined:

- costs of a screening programme
- costs of monitoring
- costs of water treatment
- costs of soil remediation
- costs of health assessment studies.

A full breakdown of results by country is provided in Annex 3, Part 3.

A key input to the assessment is based around the Swedish National Food Agency's limit value for PFAS in drinking water of 0.09 µg/l (90 ng/l) and techniques considered appropriate to treat contaminated water to meet that limit, and specifically the proportion of people exposed to levels above the limit value. Swedish data<sup>359</sup> indicates that this applies to between 2% and 3% of the Swedish population. The higher figure is selected here as the analyses have focused on 7 PFAS compounds rather than the 11 currently covered by the limit value: increasing the number of compounds will clearly increase the reported concentration and make exceedance of the limit more likely. A range of 1% to 5% is applied around this estimate. The assessment identifies the number of sites where significant contamination (i.e. in excess of the Swedish limit value) has been found and applies the proportion of the population with significant exposure indicated in the Swedish data.

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<sup>359</sup> Holmström *et al.* (2014). Nationell screening av perfluorerade föroreningar (PFAA) i dricksvatten. Rapport no 2014/20 (In Swedish).

Results are aggregated in the following tables to provide estimates for:

- the costs of a basic screening programme (Table 33);
- costs for monitoring at sites where significant PFAS contamination has been found (Table 34);
- costs for improvements to water treatment works to reduce exposure to PFAS above possible limits (Table 35);
- costs for soil remediation (Table 36);
- costs for health assessments when significant contamination is found (Table 37); and
- total of the above (Table 38).

The estimates are based on a number of assumptions which are summarised in the text below each table. Definition of low, best and high estimates is not straightforward, given limited data. In several cases the lower bound is based on data for Sweden because it provides the most complete information available for any country. Since this information only relates to contamination associated with the use of AFFFs at airfields it is likely to provide a lower bound: as other data in this report show, there are numerous other sources of PFAS contamination present in Europe including the manufacturing processes, the use and the disposal of contaminated waste materials. Ranges are provided along with best estimates based on the data in Table 30.

Estimated costs for a basic monitoring program are shown in Table 33. The assumptions used for the best estimate, and the low and high bounds, are given below the table. The best estimate indicates a cost in the order of EUR 14 million, in a range of EUR 2.8 to EUR 54 million.

**Table 33: Estimated costs for a basic screening programme to assess PFAS levels**

	N facilities for best estimate	Best estimate, EUR millions	Low, EUR millions	High, EUR millions
Denmark	78	EUR 0.08	EUR 0.02	EUR 0.31
Finland	184	EUR 0.19	EUR 0.06	EUR 0.65
Iceland	7	EUR 0.01	EUR 0.00	EUR 0.02
Norway	179	EUR 0.18	EUR 0.04	EUR 0.68
Sweden	411	EUR 0.42	EUR 0.12	EUR 1.50
Other EU28+CH	12,914	EUR 13.17	EUR 2.52	EUR 50.98
<i>Total</i>	<i>13,772</i>	<i>EUR 14.05</i>	<i>EUR 2.77</i>	<i>EUR 54.13</i>

### Best estimate assumptions

1. All airports and PFAS manufacturing sites are screened, assume 3 samples, using best estimate of cost/sample for monitoring;
2. 5% of other facilities are screened (fire stations, waste water treatment works, large and small supplies, hazardous and MSW landfills), assume 3 samples;
3. Best estimate of costs adopted.

### Low estimate assumptions

1. All airports and PFAS manufacturing sites are screened, assume 3 samples, using low cost/sample for monitoring.
2. 1% of other facilities are screened (fire stations, waste water treatment works, large and small supplies, hazardous and MSW landfills), assume 3 samples.
3. Low estimate of costs adopted.

### High estimate assumptions

1. All airports and PFAS manufacturing sites are screened, assume 3 samples, using low cost/sample for monitoring.
2. 10% of other facilities are screened (fire stations, waste water treatment works, large and small supplies, hazardous and MSW landfills), assume 3 samples.
3. High estimate of costs adopted.

A number of factors could influence the costs estimated for such a screening programme, including the number of samples taken at each point, whether sampling is carried out once only or repeatedly over time, how the programme is organised, whether it is specific to PFAS or whether the opportunity is taken to investigate the presence of other contaminants, and so on.

Estimated costs for monitoring at contaminated sites are shown in Table 34.

Table 34: Estimated costs for monitoring at sites where significant PFAS contamination has been found

	N facilities for best estimate	Best estimate, airfields and PFAS manufacturing only, EUR-millions	Best estimate, all source categories included, EUR-millions	Low, EUR-millions	High, EUR-millions
Denmark	8	EUR 0.03	EUR 0.40	EUR 0.05	EUR 7.98
Finland	22	EUR 0.47	EUR 1.11	EUR 0.19	EUR 20.81
Iceland	1	EUR 0.04	EUR 0.05	EUR 0.01	EUR 0.86
Norway	19	EUR 0.20	EUR 0.97	EUR 0.13	EUR 18.87
Sweden	48	EUR 0.76	EUR 2.40	EUR 0.36	EUR 44.97
Other EU28+CH	1,327	EUR 5.46	EUR 66.36	EUR 7.57	EUR 1,322.66
<i>Total</i>	<i>1,426</i>	<i>EUR 6.96</i>	<i>EUR 71.28</i>	<i>EUR 8.30</i>	<i>EUR 1,416.13</i>

### Best estimate assumptions

1. Assumed 20% of airports and airfields (as in Sweden) and PFAS manufacturing sites require monitoring programme, using best estimate cost/case for monitoring.
2. 0.5% of other facilities require monitoring.
3. Best estimate of costs adopted.

### Low estimate assumptions

1. Assumed 10% of airports and PFAS manufacturing sites require monitoring programme, using low estimate cost/case for monitoring.
2. 0.1% of other facilities require monitoring.
3. Low estimate of costs adopted.

### High estimate assumptions

1. Assumed 30% of airports and PFAS manufacturing sites require monitoring programme, using high estimate cost/case for monitoring.
2. 1% of other facilities require monitoring.
3. High estimate of costs adopted.

Table 34 separates the best estimate of cost associated with airfields and PFAS manufacturing from the best estimate taking into account all source categories. The total for airfields and PFAS manufacturing is only 10% of the total for the “all source” best estimate, but broadly in line with the low estimate. Summing the data from the literature review (Annex 3, Part 1) for existing monitoring at European sites where significant contamination has been found gives a cost of EUR 10.9 million, also of a similar order of magnitude to the calculated lower bound, though dominated by the Baden-Wurtemberg case. The likelihood of PFAS contamination being restricted to airfields and the small number of PFAS manufacturers present in Europe is unrealistic, hence a figure towards the best estimate does not seem unreasonable. Under the high estimate, results are almost totally dominated by sources others than airfield and PFAS manufacture because of the large number of potential sites (even though only 1% are considered). This position seems highly unlikely based on those countries where extensive monitoring has already been undertaken. However, the potential for more extensive contamination than is currently recognised exists, as shown by the case of Rastatt in Baden-Wurtemberg, Germany, where widespread contamination appears to have arisen from the spreading of waste paper materials on agricultural land, ironically for the purpose of soil improvement.<sup>360</sup>

The quantification of the costs associated with improvements to water treatment is carried out by consideration of the fraction of the Swedish population exposed to levels of PFAS in excess of the Swedish limit value. In line with the estimate given above, the best estimate is taken as 3% of the population. Broadly similar results were obtained for the USA, providing some level of verification on the order of magnitude of the estimate (though accepting the potential for coincidence, given the limited evidence base). The analysis then uses the estimates of cost/person in affected areas given in Table 35. Costs include both the upgrading of water treatment works and operation and maintenance over a 20 year period. The period of 20 years is selected to reflect that in some cases, perhaps most, advanced treatment of water supplies will be needed for many years to come, running to many decades unless remediation actions are possible and implemented.

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<sup>360</sup> <https://www.faz.net/aktuell/wissen/baden-wuerttemberg-chemische-abfaelle-auf-dem-acker-14419295.html>

In some cases, however, the need to use advanced water treatment may be reduced through connection to alternative uncontaminated supplies.

**Table 35: Estimated costs for improvements to water treatment works to reduce exposure to PFAS above possible limits.**

	Population affected, best estimate	Best estimate, EURmillions	Low, EUR millions	High, EUR millions
Denmark	170,000	EUR 97	EUR 7	EUR 274
Finland	160,000	EUR 93	EUR 7	EUR 265
Iceland	10,000	EUR 6	EUR 0	EUR 16
Norway	150,000	EUR 88	EUR 7	EUR 250
Sweden	290,000	EUR 166	EUR 13	EUR 472
Other EU28+CH	15,000,000	EUR 8,456	EUR 650	EUR 23,982
<i>Total</i>	<i>16,000,000</i>	<i>EUR 8,906</i>	<i>EUR 684</i>	<i>EUR 25,258</i>

### Best estimate assumptions

1. Assumed 3% of the population are exposed to excess levels of PFAS via drinking water.
2. Assume 20 year maintenance programme for treatment works, based on best estimate.
3. Assume best estimate cost per case for water treatment.
4. Assume 4% discount rate on future maintenance costs.

### Low estimate assumptions

1. Assumed 1% of the population are exposed to excess levels of PFAS via drinking water.
2. Assume 20 year maintenance programme for treatment works, based on best estimate.
3. Assume low estimate cost per case for water treatment.
4. Assume 4% discount rate on future maintenance costs.

### High estimate assumptions

1. Assumed 5% of the population are exposed to excess levels of PFAS via drinking water.
2. Assume 20 year maintenance programme for treatment works, based on best estimate.
3. Assume high estimate cost per case for water treatment.
4. Assume 4% discount rate on future maintenance costs.

In comparison, the results from the identified literature gives a figure of EUR 136 million for improvements to water quality, only 20% of the lower bound calculated here. However, the costs identified in the literature are in many cases based on situations where measures are yet to be taken. Also, further costs are not considered in this estimate, for example, improvement of water treatment, the need to construct new water pipelines

to bring in clean water from other areas, or the costs of shutting down existing wells and losing the water resource that they provide. Therefore, the estimated lower bound may not be unrealistic, and it is possible that the costs are substantially higher.

The results for individual countries will be more uncertain than the total estimate for the EEA countries. For example relative to Sweden the levels of contamination found in Denmark are much lower. This is likely linked to the distance between PFAS use and the location of the groundwater sources that dominate water supply. Table A3.9 in Annex 3 part 2 shows that 99% of drinking water in Denmark comes from ground rather than surface sources. This is a much higher percentage than in other countries. Extrapolation based on the Danish situation would therefore not account for the costs incurred already across Europe.

Estimated costs of soil remediation are given in Table 36, again with details on assumptions for the best, low and high estimates provided below the table.

**Table 36: Estimated costs for soil remediation**

	Number of sites affected for the best estimate	Best estimate, EUR millions, air-fields and PFAS manufacturing only, EUR millions	Best estimate, EUR millions	Low, EUR millions	High, EUR millions
Denmark	8	EUR 3	EUR 40	EUR 0.5	EUR 798
Finland	22	EUR 47	EUR 111	EUR 2.2	EUR 2,081
Iceland	1	EUR 4	EUR 5	EUR 0.1	EUR 86
Norway	19	EUR 20	EUR 97	EUR 1.6	EUR 1,887
Sweden	48	EUR 76	EUR 240	EUR 4.3	EUR 4,497
Other EU28+CH	1,327	EUR 546	EUR 6,636	EUR 91	EUR 132,266
<i>Total</i>	<i>1,426</i>	<i>EUR 696</i>	<i>EUR 7,128</i>	<i>EUR 100</i>	<i>EUR 141,613</i>

### Best estimate assumptions

1. Assumed 20% of airports and PFAS manufacturing sites require remediation.
2. 0.5% of other facilities require remediation.
3. Best estimate of costs adopted.

### Low estimate assumptions

1. Assumed 10% of airports and PFAS manufacturing sites require remediation.
2. 0.1% of other facilities require remediation.
3. Low estimate of costs adopted.

### High estimate assumptions

1. Assumed 30% of airports and PFAS manufacturing sites require remediation.
2. 1% of other facilities require remediation.
3. High estimate of costs adopted.

Of the estimates presented, soil remediation costs are likely to be the most uncertain, which is reflected in the range of the costs. The costs identified in the literature review

for the Baden-Wurttemberg case alone are between EUR 1–3 billion<sup>361</sup>, substantially more than the low estimate and almost half the best estimate. However, that cost is an estimate, and it is possible that more cost-effective solutions will be found. As noted above, Baden Wurttemberg is not the only location where high costs have been reported, with Schiphol and Dusseldorf Airports also reporting high remediation costs.

It is necessary to ask to what extent the costs of additional water treatment and soil remediation should be considered additive. It could be argued that with soil remediation carried out, there should be no need for additional water treatment, and vice-versa. However, this would ignore the time taken to carry out soil remediation, time during which water would need to be treated, or brought in from outside areas, to avoid excess exposure of the population. A failure to clean the soils would mean that water treatment would need to persist into the far future, rather than the 20 year period assumed here. Whilst there is some overlap it is clearly not a simple binary choice to treat water or remediate the soil, and both will often be necessary. A further factor to consider is that the longer soils are left contaminated, the more the PFAS will spread, potentially making clean-up more difficult, far more extensive and more expensive. Estimated costs for health assessments are provided in Table 37.

**Table 37: Estimated costs for health assessments when significant contamination is found**

	Population affected, best estimate	Best estimate, EUR millions	Low, EUR millions	High, EUR millions
Denmark	170,000	EUR 8.5	EUR 0.28	EUR 27
Finland	160,000	EUR 8.2	EUR 0.27	EUR 26
Iceland	10,000	EUR 0.5	EUR 0.02	EUR 1.6
Norway	150,000	EUR 7.7	EUR 0.26	EUR 25
Sweden	190,000	EUR 15	EUR 0.49	EUR 46
Other EU28+CH	14,000,000	EUR 744	EUR 24.79	EUR 2,355
<i>Total</i>	<i>15,000,000</i>	<i>EUR 783</i>	<i>EUR 26.11</i>	<i>EUR 2,480</i>

### Best estimate assumptions

1. Assumed 3% of the population are exposed to excess levels of PFAS via drinking water.
2. Assume best estimate cost per case for remediation.

### Low estimate assumptions

1. Assumed 1% of the population are exposed to excess levels of PFAS via drinking water.
2. Assume low estimate cost per case for remediation.

<sup>361</sup> Cost data for this case are taken from <http://greensciencepolicy.org/wp-content/uploads/2016/09/Rolland-Weber-PFOS-PFAS-German-activities-Final.pdf>. A full, detailed account of the costing has not been identified. However, further accounts (e.g. <https://www.faz.net/aktuell/wissen/baden-wuerttemberg-chemische-abfaelle-auf-dem-acker-14419295.html>) refer to this case as the largest environmental scandal in Germany in terms of the area affected (currently around 400 hectares).

## High estimate assumptions

1. Assumed 5% of the population are exposed to excess levels of PFAS via drinking water.
2. Assume high estimate cost per case for remediation.

The need to undertake health assessment will vary from location to location. The cost of biomonitoring in Ronneby, Sweden averaged over EUR 0.5 million per year for a 3 year period. The WHO report health assessment costs for the Veneto region of EUR 4.6 million. On this basis, only a very small number of cases would be needed to reach the low estimate made here, so again, a figure between the low and best estimates is easily feasible.

### 5.2.6 Summary of environment-related costs of exposure to PFAS

Table 38 shows total costs from the preceding tables covering initial environmental screening, monitoring where contamination is found, water treatment, soil remediation and health assessment, with a more detailed breakdown for the Nordic countries in Table 39.

**Table 38: Aggregated costs covering environmental screening, monitoring where contamination is found, water treatment, soil remediation and health assessment**

	Best estimate, EUR millions	Low, EUR millions	High, EUR millions
Denmark	EUR 145	EUR 8	EUR 1,106
Finland	EUR 214	EUR 10	EUR 2,393
Iceland	EUR 12	EUR 1	EUR 105
Norway	EUR 194	EUR 9	EUR 2,181
Sweden	EUR 423	EUR 18	EUR 5,061
Other EU28+CH	EUR 15,915	EUR 776	EUR 159,976
<i>Total</i>	<i>EUR 16,902</i>	<i>EUR 821</i>	<i>EUR 170,821</i>

**Table 39: Detailed breakdown for non-health environmental costs per Nordic Countries**

	N people affected (3%)	Screening and monitoring	Health assessment	Upgrade treatment works and maintenance	Soil remediation	Total (EUR-millions)
Denmark	169,791	EUR 0.07–8.3	EUR 0.28–27	EUR 7.4–274	EUR 0–798	EUR 8–1,106
Finland	164,153	EUR 0.25–22	EUR 0.27–26	EUR 7.2–265	EUR 2.2–2,081	EUR 10–2,393
Iceland	10,102	EUR 0.01–0.9	EUR 0.02–1.6	EUR 0.4–1.6	EUR 0.1–86	EUR 1–105
Norway	154,995	EUR 0.17–20	EUR 0.26–25	EUR 6.8–250	EUR 1.6–1,887	EUR 9–2,181
Sweden	292,421	EUR 0.48–47	EUR 0.49–46	EUR 13–472	EUR 4.3–4,497	EUR 18–5,061
<i>Nordic total</i>	<i>791,462</i>					<i>EUR 46–10,846</i>

Parallel calculations for all 31 EEA Member Countries and Switzerland arrive at a range of costs for environmental remediation totalling EUR 821 million to 170 billion. The uncertainties associated with these estimates are clearly high, reflecting the level of variation in the unit cost estimates. For example, remediation of just the one case of soil contamination in Germany at Baden-Wurttemberg has been estimated at EUR 3 billion, though this work has yet to be undertaken.

Uncertainties have been reviewed in the context of each of the tables presented. The lower and upper bounds should be considered illustrative because of the limited information available. Further analysis may be able to limit the range beyond that estimated here. However, based on the information from the literature review there is a firm basis for concluding that the lower bound estimates would be exceeded. A best estimate in the order of EUR 10–20 billion is certainly feasible. Significantly higher costs are feasible if several cases similar to the contamination at Baden-Wurttemberg are identified.

It is noted that results for individual countries will be more uncertain than results for Europe as a whole. It is, for example, understood that the contamination levels in Denmark are lower than Sweden. The situation in other countries may vary, with different levels of contamination. The data available from Sweden only link contamination to airports, whereas problems elsewhere have been identified in relation to other sources such as PFAS manufacture and use (Veneto), fires (Dusseldorf and Buncefield), and spreading of waste paper materials on agricultural land as a soil improver (Rastatt).

A further source of uncertainty concerns those elements of the analysis that have not been quantified. Notable amongst these are potential ecological effects, especially given the extreme persistence of PFAS. From an economic perspective the material reviewed above demonstrates public willingness to pay to avoid exposure to persistent and bioaccumulative substances, even without detailed assessment of impacts. Aggregated across the European population the willingness to pay based on information in the restriction proposal for D4 and D5 would be substantial in the order of billions of Euro and broadly comparable with the best estimate made here for monitoring and clean-up. The significant uncertainties in those estimates are recognised. Other elements that have not been quantified include the costs of providing new pipelines to provide access to uncontaminated water supplies and various administrative costs. The former, in particular, could be substantial based on evidence from a limited number of sites (e.g. Jersey, UK), certainly adding weight to the overall best estimate.



## 6. Conclusions

### 6.1 Findings and discussion

This study investigates the socioeconomic costs that may result from impacts on human health and the environment from the use of PFAS. Better awareness of the costs and long-term problems associated with PFAS exposure will assist authorities, policy-makers and the general public to consider more effective and efficient risk management. The production of PFAS, manufacture and use of PFAS-containing products, and end-of-life disposal of PFAS have resulted in widespread environmental contamination and human exposure. PFAS have been found in the environment all around the world and almost everyone living in a developed country has one or more PFAS in his/her body.

Because of the extreme persistence of PFAS in the environment, this contamination will remain on the planet for hundreds if not thousands of years. Human and environmental exposure will continue, and efforts to mitigate this exposure will lead to significant socioeconomic costs – costs largely shouldered by governments and taxpayers.

The focus of this study is on costs of inaction with respect to regulation of PFAS in the countries comprising the European Economic Area (EEA), i.e. the costs that society will have to pay in the future if action is not taken to limit PFAS-emissions today.

The impact pathway concept provides an overall analytical framework for the study. Five case studies, based on literature reviews, following the life-cycle of PFAS are presented to illustrate the links between production, use and disposal of PFAS, health and environmental exposures, and impacts and their economic valuation. The pathway concept provides a template for assessment of each source, enabling the analyst to consider which impacts are relevant.

The first three case studies cover the activities that account for a large proportion of the PFAS released into the environment: their production, their use in product manufacturing, and the use phase of PFAS-containing products. The industrial facilities producing the fluorochemicals and fluoropolymers, while relatively limited in number, are significant emitters of PFAS into the air, soil and waterways. Case Study 1 estimates that up to 20 facilities actively produce fluorochemicals in Europe, that these facilities are significant sources of PFAS released to the environment, and that exposure to workers at these plants is high.

Other industrial activities with the potential to release PFAS to the environment take place throughout Europe, including the Nordic region. Case Study 2 considers the manufacture and commercial use of PFAS-containing products, including textile and leather manufacturing; metal plating, including chromium plating; paper and paper product manufacturing; paints and varnishes; cleaning products; plastics, resins and rubbers; and car wash establishments. Releases of PFAS occur via the air or effluent entering sewerage and wastewater treatment plants, before discharge into waterways.

The third major source responsible for PFAS released to the environment, which is the focus of Case Study 3, is the widespread use of aqueous film-forming foams (AFFFs). The AFFFs are used to extinguish fires in emergencies or during training, especially around airports and military bases. Where the AFFFs have migrated to groundwater and other sources of drinking water, nearby communities have been affected by elevated levels of PFAS in their drinking water. It is noted that other uses of AFFFs for fire-fighting, especially at major industrial facilities, may also be a significant source, but one that has so far received little attention.

Case Study 4 and 5 considers the use and the end-of-life phase of consumer products, which account for the remaining releases and direct sources of exposure to PFAS. Case Study 4 looks at PFAS-treated carpets, PFAS-treated food contact materials and cosmetics as examples of how a product's use is likely to lead to human exposure. Possible exposure occurs through ingestion and dermal absorption, or through releases to the environment when the product is washed off or laundered, entering sewers, treatment plants, and eventually waterways. The availability of suitable non-fluorinated alternatives makes the use of PFAS in many of these products unnecessary.

Case Study 5 looks at end-of-life impacts of PFAS-treated products. Waste incineration may destroy PFAS in products if 1000 °C operating temperatures are reached, but such temperatures are not typical of most incineration capacity (the EU Industrial Emissions Directive, for example, requires a temperature of 850 °C for municipal waste incineration). If landfilled, the PFAS will remain even after the product's core materials break down. The compounds will eventually migrate into liquids in the landfill, then into leachate collection systems or directly into the natural environment. They may then enter drinking water supplies, be taken up by edible plants and bioaccumulate in the food chain.

#### **6.1.1** *Health-related costs to society*

To calculate health-related costs to society, the study looked for consensus regarding health endpoints affected by exposure to PFAS. Some agreement has emerged concerning liver damage, increased serum cholesterol levels (related to hypertension), decreased immune response, increased risk of thyroid disease, decreased fertility, pregnancy-induced hypertension/pre-eclampsia, lower birth weight, and testicular and kidney cancer.

The methodology draws upon risk relationships developed in the course of specific epidemiological studies for populations exposed to PFAS at different levels. Workers exposed to PFAS in the workplace were used to exemplify a high level of exposure. Communities affected by PFAS, e.g. because of proximity to manufacturing sites or sites where fluorinated AFFFs were used were assumed to have been exposed at a medium level; this level of exposure was assumed to have been experienced by 3% of the European population. The general population was considered to have experienced exposure at low (background) levels.

Table 40 provides an overview of the estimated annual costs for just a few health end-points where risk ratios were available for affected populations. Despite the high level of uncertainty and the assumptions underlying the calculations, the findings suggest that the health-related costs of exposure to PFAS are substantial.

**Table 40: Estimated health-related costs of exposure to PFAS at different levels of exposure**

Exposure level	“Exposed” population and source of PFAS	Health end-point linked to PFAS	Nordic countries		All EEA countries	
			Population at risk	Annual costs	Population at risk	Annual costs
Occupational (high)	Workers at chemical production plants or manufacturing sites	Kidney cancer	n.a.	n.a.	84,000–273,000	EUR 12.7–41.4 million
Elevated (medium)	Communities near chemical plants, etc. with PFAS in drinking water	All-cause mortality	621,000	EUR 2.1–2.4 billion	12.5 million	EUR 41–49 billion
		Low birth weight	8,843	136 births of low birth weight	156,344	3,354 births of low birth weight
		Infection	45,000 children	84,000 additional days of fever	785,000 children	1.5 million additional days of fever
Background (low)	Adults in general population (exposed via consumer products, background levels)	Hypertension	10.3 million	EUR 0.7–2.2 billion	207.8 million	EUR 10.7–35 billion
<i>Totals</i>			<i>Nordic countries</i>	<i>EUR 2.8–4.6 billion</i>	<i>All EEA countries</i>	<i>EUR 52–84 billion</i>

The range of estimated annual health-related costs due to PFAS exposure is *EUR 2.8–4.6 billion for the five Nordic countries and EUR 52–84 billion for all EEA countries*. Some overlap occurs in the figures, because workers and affected communities are also exposed to background levels of PFAS. The actual costs are likely to be higher, since these calculations are for only a few of the health impacts linked to exposure to PFAS.

### 6.1.2 Non-health costs related to environmental contamination

The second methodology compiled information on direct costs incurred by communities taking measures to reduce PFAS exposure through remediation of drinking water. Based on these direct costs, ranges of cost per persons affected or per case were developed. These unit costs then became the foundation for aggregating the costs of remediation for environmental exposure over and above action levels for PFAS concentrations in drinking water.

As with the health-based estimates, the study assumes that 3% of the European population is exposed to drinking water with PFAS concentrations over regulatory action levels, such that the water treatment works serving them will require upgrading and maintenance over the next 20 years. Recognising the uncertainties that exist in the analysis and the available data, costs of remediation have been quantified using

a scenario-based approach. For each scenario a number of parameters are specified, relating for example to the size of the affected population and the duration of maintenance works, and results generated accordingly.

Table 4.1 shows the range of costs for the various categories of actions related to environmental remediation for the five Nordic countries. The overall range of estimated non-health costs is *EUR 46 million – 11 billion over the next 20 years*, just for the Nordic countries. The upper end of this range is dominated by soil remediation costs for which associated uncertainty must be considered high.

**Table 4.1: Detailed breakdown of ranges in quantified non-health costs for the Nordic countries.**

	N people affected (3%)	Screening and monitoring	Health assessment	Upgrade treatment works and maintenance	Soil remediation	Total
Denmark	169,791	EUR 70,000–8.3 million	EUR 280,000–27 million	EUR 7.4 million–274 million	EUR 0–798 million	EUR 8 million–1.1 billion
Finland	164,153	EUR 250,000–22 million	EUR 270,000–26 million	EUR 7.2 million–265 million	EUR 2.2 million–2.1 billion	EUR 10 million–2.4 billion
Iceland	10,102	EUR 10,000–900,000	EUR 20,000–1.6 million	EUR 400,000–1.6 million	EUR 100,000–86 million	EUR 1 million–105 million
Norway	154,995	EUR 170,000–20 million	EUR 260,000–25 million	EUR 6.8 million–250 million	EUR 1.6 million–1.9 billion	EUR 9 million–2.2 billion
Sweden	292,421	EUR 480,000–47 million	EUR 490,000–46 million	EUR 13 million–472 million	EUR 4.3 million–4.5 billion	EUR 18 million–5.1 billion
<i>Nordic total</i>	<i>791,462</i>					<i>EUR 46 million–11 billion</i>

The cost estimates provided in the table are likely to be more robust at the aggregate, European level than at the national level, given the potential for significant variation between countries in sensitivity and use of PFAS, that could not be accounted for here.

Parallel calculations for all 31 EEA Member Countries and Switzerland arrive at a range of non-health costs for environmental remediation totalling *EUR 821 million to EUR 170 billion*. Again, these cost estimates will be more robust at the aggregate, European level than at the national level. A review of the uncertainties concludes that the lower and upper bounds should be considered illustrative because of the limited information currently available, reflecting the level of variation in the unit cost estimates. However, based on the information from the literature review there is a firm basis for concluding that the lower bound estimates would be exceeded. A *best estimate in the order of EUR 10–20 billion* is certainly plausible. Significantly higher costs than that are likely if several cases similar to the contamination at Baden-Wurtemberg are identified, where costs of soil remediation have been estimated at up to EUR 3 billion.

A number of other costs related to PFAS contamination are outside the scope of the quantification carried out in this report. These include loss of property value, reputational damage to a polluting company, costs of short-term measures such as provision of bottled water, ecological damage and the costs incurred by public authorities in responding to affected communities – including public outreach, surveys of contamination, and remedial measures.

### 6.1.3 Conclusions

The work of estimating the health and environment-related costs to society related to PFAS exposure has relied on the development of assumption-based scenarios. This reflects the limited data available in the academic literature, government documents and press reports. Whilst the uncertainties of the analysis need to be acknowledged, it is also important to recognise that, for a number of issues, there is little or no uncertainty. For example, that the equivalent of hundreds of millions of EUR have already been spent on remediation of PFAS contamination, that PFAS use is widespread, and that PFAS will persist in the environment for an extremely long time. Other certainties include:

1. PFAS are ubiquitous in the environment, and almost all people have PFAS in their bodies today. Monitoring in both Sweden and the USA concludes that around 3% of the population are or have been exposed above proposed limit values, primarily through contamination of drinking water but also via other sources.
2. Many sources of PFAS exposure exist linked to specialist applications (e.g. AFFFs for firefighting at airports and some industrial locations) and non-specialist uses (e.g. use in consumer goods such as clothing, cosmetics and pizza boxes).
3. Non-fluorinated alternatives for many of these uses are already on the market, and therefore certain uses of PFAS can be reduced.
4. The costs for remediating some cases of contamination run to many millions of EUR. Total costs at the European level are expected to be in the hundreds of millions of EUR as a minimum.
5. A large and growing number of health effects have been linked to PFAS exposure and evidence is mounting that effects occur even at background level exposures.

Current and proposed limit values for drinking water may be further reduced in recognition of growing information on, health and environmental risks. This would increase the costs of environmental remediation estimated here.

As explained throughout the study, the calculations rest on a number of assumptions, though these have been checked against (e.g.) data on costs incurred to ensure that they are linked to real-world experience. As more information becomes available in the future, drawing on the framework provided here, calculations will become more precise. Moreover, these findings are conservative. The figures will only get larger, in that the numbers of PFAS on the market and the volumes produced keeps increasing. Further inaction will lead to more sources of contamination, more people exposed, and higher costs for remediation. The longer that PFAS contamination is left in the environment, the wider it will spread and the greater the quantity of soil or groundwater that will need to be decontaminated.

## 6.2 Next steps and proposals for further studies

As with other studies seeking to identify costs of inaction linked to chemicals exposure, our findings have been hampered by a lack of data concerning contamination and levels of human exposure. Overall calculations rest on a number of assumptions which may become more precise as more information is available in the future. Additional research to help gather this additional information could include:

- consideration of health endpoints due to exposures to groups of PFAS, since exposures are rarely limited to a single PFAS and since PFAS as a group share similar properties – most importantly, the property of extreme persistence in the environment;
- more information concerning the sites where production of PFAS and/or where manufacturing of products involving PFAS is, or have been occurring. National inventories of such sites are needed, including of sites where fluorinated AFFFs have been used. This would be of great help in estimating the numbers of affected populations, and the extent of contamination where remediation may be needed;
- systematic cataloguing of cost data where problems have been identified;
- inclusion of industries producing or using PFAS in the European Pollutant Release and Transfer Register, so that information on the location of releases to air and to water is available and so those releases can be tracked;
- national registries of products containing PFAS to help inform how PFAS are used and to contribute to better characterisation of the major sources of exposure from products;
- drinking water standards using group parameters for PFAS, so as to require better monitoring of drinking water. This will provide early warnings when elevated levels are found and enable more effective identification and timely containment of sources of contamination;
- better understanding of what happens to PFAS discharged from wastewater treatment plants and during incineration, including assessment of ecological risks from PFAS contamination; and
- more biomonitoring and epidemiological studies to characterize links between PFAS exposure and health endpoints, to enable better calculations of associated health costs.

As this study highlights, the release of PFAS into the environment and constant exposure of humans is ongoing throughout the Nordic countries and Europe. Large-scale monitoring efforts such as those carried out in Sweden can help to clarify sources of contamination and provide more certainty concerning the scale of the socio-economic costs related to PFAS exposure. This will help to better inform policymakers, industry and consumers concerning the actions needed.

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# Sammanfattning

Högfluorerade ämnen (PFAS) kan skada människors hälsa och miljön. Förutom att orsaka ett stort lidande för drabbade individer medför denna exponering stora kostnader för samhället. I den här studien uppskattas kostnader för samhället som användningen av PFAS kan orsaka på grund av dess påverkan på miljön och människors hälsa. En större medvetenhet om kostnader och långsiktiga problem som kan förknippas med användningen av högfluorerade ämnen kan hjälpa berörda myndigheter, beslutsfattare och allmänheten att ta beslut om effektiva och riskminskande åtgärder.

I studien granskas hur produktionen av PFAS, tillverkningen och användningen av produkter som innehåller PFAS samt hur sluthantering av dessa har resulterat i omfattande miljöförorening och exponering av människor. PFAS har hittats i miljön över hela världen och nästan alla som lever i ett land med utvecklad ekonomi har ett eller flera PFAS-ämnen i sin kropp.

Eftersom de högfluorerade ämnena är så långlivade i miljön kommer dessa ämnen att finnas kvar på vår planet i hundratals, om inte tusentals år. Människors och miljöns exponering för PFAS kommer att fortsätta, och åtgärder för att minska denna exponering kommer medföra stora kostnader för samhället. Det är kostnader som till stor del ska betalas av offentliga myndigheter och skattebetalare.

I rapporten studeras kostnader som samhället kommer att behöva betala i framtiden om åtgärder inte genomförs för att begränsa utsläppen av PFAS. Studien fokuserar på de länder som ingår i det Europeiska Ekonomiska Samarbetsområdet (EES). "Cost of inaction" definieras som de kostnader som samhället i framtiden kommer att behöva betala om inga åtgärder genomförs idag för att begränsa utsläppen av PFAS. De PFAS som omfattas i den här studien är fluorerade ytaktiva ämnen med en kolkedjelängd av C<sub>4</sub>-C<sub>14</sub>. Studiens målsättning var att ta fram:

1. Ett ramverk för att uppskatta samhällskostnader kopplade till negativa effekter på hälsa och miljö i samband med exponering för PFAS.
2. Monetära värden för dessa samhällskostnader, dokumenterade genom fallstudier.

## Slutsatser

För att uppskatta de hälsorelaterade och miljömässiga kostnader för samhället utgick vi från scenarier som bygger på antaganden, eftersom tillgången på data är så begränsad. Studien baserar sig på litteratur, information från myndigheter och nyhetsartiklar. Även om studien och dess analys innebär vissa osäkerhetsfaktorer finns det en relativt stor säkerhet i en rad frågor:

1. PFAS förekommer överallt i miljön och nästan alla människor har PFAS i sina kroppar. Mätningar från både Sverige och USA visar att cirka tre procent av befolkningen exponeras för halter som ligger över de rekommenderade gränsvärdena, framförallt via dricksvatten men andra källor förekommer också.
2. PFAS kan hamna i miljön och i människor på många olika sätt. En källa till spridning av PFAS i miljön är via, till exempel användningen av brandsläckningskum för brandbekämpning på flygplatser och på vissa industrianläggningar. PFAS sprids då till miljön och når slutligen dricksvattnet. PFAS kan också spridas via användningen i konsumentvaror så som exempelvis pizzakartonger, kläder och kosmetiska produkter.
3. För många av de områden där PFAS idag används finns redan icke-fluorerade alternativ tillgängliga på marknaden. Därför skulle vissa användningar redan idag kunna minskas.
4. Kostnaden för att sanera mark och vatten uppskattas till många miljoner EUR. Den totala saneringskostnaden för EUs medlemsländer uppskattas till minst hundratals miljoner EUR.
5. De negativa hälsoeffekter som kan bero på exponering för PFAS är redan många och fortsätter dessutom att öka i antal. Det finns också bevis för att det kan uppstå negativa effekter redan vid exponering för relativt låga halter även vid halter motsvarande låga (bakgrunds) nivåer.

De rekommenderade gränsvärdena för dricksvatten kan komma att sänkas ytterligare när mer information om risker för hälsa och miljö tas fram. Detta skulle öka kostnaderna för den miljösanering som uppskattats i denna studie.

Beräkningarna i studien grundar sig på ett antal antaganden. Dessa antaganden har stämts av mot faktiska kostnadsuppgifter för att säkerställa att de är kopplade till verkliga fall. Allteftersom mer information blir tillgänglig kommer beräkningarna kunna bli mer exakta. I den här studien har vi varit försiktiga i våra antaganden, resultaten visar därför på kostnader som sannolikt inte kan vara lägre, däremot skulle de kunna vara högre. Det är troligt att kostnaderna kommer att öka i takt med att förhöjda volymer och ett ökat antal PFAS finns tillgängliga på marknaden.

Att vänta ännu längre med att begränsa utsläppen av PFAS kan få stora konsekvenser. Det kan innebära att antalet utsläppskällor ökar, att fler personer exponeras och att kostnaderna för saneringen blir högre. Föroreningarna kommer också att sprida sig, vilket medför att större mängder mark och/eller grundvatten kommer att behöva saneras.

## Metod

Två metoder har utvecklats under arbetet med denna rapport. Båda metoderna är baserade på fallstudier som berör exponeringen av PFAS. Den ena metoden används för att bedöma hälsorelaterade kostnader. Den andra metoden används för att beräkna kostnaderna för miljösanering. Data specifik för de nordiska länderna har använts när

sådana har funnits, men uppskattningarna bygger också på information från andra europeiska länder, USA och Australien.

## Spridningsvägar (fallstudier)

För att illustrera hur exponeringen av människor och miljö ser ut har fem fallstudier som följer PFAS livscykel (från produktion och användning vid produkttillverkning till produkters användning och sluthantering) använts. För att få ytterligare uppgifter om direkta kostnader som uppstått i samband med till exempel behovet att sanera förorenat dricksvatten har även andra fall av PFAS-föroreningar studerats.

I fallstudie 1 studeras produktionen av PFAS i Europa och vilka föroreningar denna har orsakat. Här granskas föroreningar som är kopplade till Chemours fabriker i Dordrecht (Nederländerna); Mitenis anläggning i Veneto-regionen (Italien); och 3Ms anläggning nära Antwerpen (Belgien). Studien uppskattar att upp till 20 anläggningar aktivt producerar fluorokemikalier i Europa samt att dessa anläggningar är betydande källor till utsläpp av PFAS till miljön. Studien visar också att exponeringen för arbetare vid dessa anläggningar är hög.

Fallstudie 2 handlar om tillverkning samt användning av produkter som innehåller PFAS. Industriella aktiviteter som kan släppa ut PFAS till miljön inkluderar textil- och lädertillverkning; metallplätering, inklusive kromplätering; tillverkning av papper och pappersvaror; färger och lacker; rengöringsprodukter; plast, hartser och gummi; samt biltvättar. I studien antas att ett intervall på 3–10 procent av dessa anläggningar använder PFAS. Några fluorokemiska produktionsanläggningar i de nordiska länderna kunde inte identifieras. Däremot visar statistik från Eurostat att annan industriell verksamhet som riskerar att släppa ut PFAS till miljön äger rum i regionen, såsom metallplätering och tillverkning av papper- och pappersvaror.

I fallstudie 3 och 4 studeras användningen av kemiska produkter och varor som innehåller PFAS. Vattenbaserade filmbildande skum (AFFF) som innehåller PFAS har använts för att släcka petroleumbaserade bränder samt i brandövningar, vilket har orsakat att grundvatten förorenats – särskilt kring flygplatser och militära baser. Även närliggande samhällen har påverkats av förhöjda nivåer av PFAS i dricksvattnet. I fallstudie 4 studeras användningen av varor och produkter som innehåller PFAS så som mattor, livsmedelsförpackningar och kosmetiska produkter. Dessa varor och produkter används som exempel för att visa hur användningen av en vara sannolikt kan leda till att människor exponeras via livsmedel och absorption genom huden. Användningen leder också till utsläpp av PFAS till miljön när produkter sköljs av eller rengörs och når avloppsnet, reningsverk och så småningom recipient.

I fallstudie 5 studeras effekterna från sluthantering av produkter som innehåller PFAS. Kommunal avfallsförbränning kan destruera PFAS i produkter vid driftstemperaturer på 1000°C, men vid deponering kommer PFAS att finnas kvar även efter att produktens kärnmaterial brutits ner. Föreningarna kommer så småningom förflytta sig till vätskor i deponin, och därefter till uppsamlingsystem för lakvatten eller direkt till

grundvatten och mark. De kan sedan nå dricksvattentäkter eller tas upp av växter och bioackumuleras i livsmedelskedjan.

## Hälsorelaterade samhällskostnader

För att beräkna hälsokostnader för samhället har studien sökt efter konsensus om hälsoeffekter kopplade till exponering för PFAS. De vetenskapliga bevis som finns är motstridiga, men viss konsensus verkar råda vad gäller leverskador, höga blodtrycksnivåer, nedsatt immunförsvaret (högre infektionsrisk), ökad risk för sköldkörtelsjukdom, nedsatt fertilitet, graviditetsinducerad hypertoni, preeklampsi, lägre födelsevikt, samt testikel- och njurcancer.

Metoden bygger på riskrelationer från epidemiologiska studier för populationer som i olika hög grad exponerats för PFAS. Arbetare som exponerats för PFAS på arbetsplatsen är exempel på en grupp med en hög exponeringsnivå, medan samhällen som drabbats av PFAS på grund av närheten till produktionsanläggningar, eller där fluorerade AFFF använts, antas ha exponerats på medelnivå. Denna exponeringsnivå antas gälla för tre procent av den europeiska befolkningen, medan befolkningen i övrigt antas ha exponerats för låga (bakgrunds-) nivåer.

Tabell 42 ger en översikt över de uppskattade årliga kostnaderna för ett antal hälsoeffekter där riskkvoter fanns tillgängliga för berörda populationer. Exempelvis bedömdes den årliga hälsorelaterade kostnaden för förhöjd risk för njurcancer p.g.a. yrkesmässig exponering för PFASs uppgå till mellan EUR 12,7 och EUR 41,4 miljarder i EES-länderna.<sup>362</sup> Den uppskattade hälsorelaterade kostnaden blev betydligt högre för såväl förhöjda som bakgrunds nivåer p.g.a. det stora antalet personer som då berörs. Den årliga hälsorelaterade kostnaden för exponering vid tre olika nivåer av PFASs beräknades till minst EUR 2,8–4,6 miljarder i de nordiska länderna och EUR 52 till EUR 84 miljarder i EES-länderna. Trots stora osäkerheter och att beräkningarna till stor del baseras på antaganden så tyder resultaten på att hälsokostnaderna för PFASs-exponering är betydande.

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<sup>362</sup> P.g.a. ofullständiga data gällande antal och lokalisering av produktionsanläggningar för kemikalier och produkter innehållande kemikalier har de hälsorelaterade kostnaderna, orsakade av yrkesmässig exponering för PFAS i de nordiska länderna, inte kunnat beräknas.

Tabell 42: Uppskattade årliga hälsorelaterade kostnader vid exponering av PFAS

Exoneringsnivå	Exponerad population & källa	Hälsa endpoint	Nordiska länder		Alla EEA länder	
			Exponerad population	Årlig kostnad	Exponerad population	Årlig kostnad
Yrkesmässig (hög)	Arbetare på kemiska produktionsanläggningar eller tillverkningsställen	Njurscancer	Ej tillgängligt	Ej tillgängligt	84000–273000	EUR 12,7–41,4 miljoner
Förhöjd (medium)	Samhällen nära kemiska anläggningar etc. med PFAS i dricksvattnet	Dödlighet (alla orsaker)	621000	EUR 2,1–2,4 miljarder	12,5 miljoner	EUR 41–9 miljarder
		Låg födelsevikt	8843 födselar	136 födselar med låg födelsevikt	156344 födselar	3354 födselar med låg födelsevikt
		Infektion	45000 barn	84000 ytterligare dagar med feber	785000 barn	1,5 miljoner ytterligare dagar i feber
Bakgrund (låg)	Vuxna i den allmänna befolkningen (exponerade via konsumentprodukter, bakgrunds nivå)	Hypertoni	10,3 miljoner	EUR 0,7–2,2 miljarder	207,8 miljoner	EUR 10,7–35 miljarder
<b>Totalt</b>			<i>Nordiska länder</i>	<i>EUR 2,8–4,6 miljarder</i>	<i>Alla EEA länder</i>	<i>EUR 52–84 miljarder</i>

Anm.: Årligt antal berörda personer är det uppskattade antalet individer med en ökad risk för negativa hälsoeffekter på grund av olika exponeringsnivåer. En viss överlappning förekommer i uppgifterna ovan, eftersom arbetstagare och drabbade samhällen också utsätts för bakgrunds nivåer av PFAS. Samtidigt är dessa kostnader troligen underskattade på grund av bristen på epidemiologiska riskrelationer att använda för beräkning av andra hälsoeffekter och relaterade kostnader.

## Icke hälsorelaterade samhällskostnader (miljörelaterade)

Den andra metoden innefattade att samla information om direkta kostnader som uppstått i samhällen där åtgärder, såsom rening av dricksvatten, vidtagits för att minska människors exponering för PFAS. Med dessa sammanlagda kostnader som grund beräknades kostnadsintervall per person eller per fall. Dessa enhetskostnader användes sedan för att räkna samman kostnaderna för sanering när miljöexponeringen, till exempel koncentrationer i dricksvatten, överskrider vissa nivåer. Det bör noteras att intervallen som anges i tabellen är stora, även när de normaliserats mot populationen.

Avgörande för om intervallen för medelvärden kan beräknas beror på vilken mängd data som finns tillgängliga. När det exempelvis gäller kostnaderna för att rena vatten fanns ett flertal estimerade tillgängliga. I ett sådant fall är det osannolikt att det faktiska medelvärdet kommer att vara ett extremvärde i någon av intervallens ändar som hämtats från studierna. Det är därför rimligt att minska det observerade intervallet till exempel genom att ta bort de uppskattningar som redan i tillräckligt stor omfattning exkluderats från andra datakällor, det vill säga att de är att betrakta som avvikande värden. För vissa kostnader finns dock mycket få uppskattningar tillgängliga. Var och en av de tillgängliga uppskattningarna kan vara lika gällande för att ange medelvärdet. I ett sådant fall antas det observerade värdeintervallet som ett intervall för troliga medelvärden.

I de fall då inget intervall finns att hämta från källorna i litteraturstudien har ett intervall uppskattats som till exempel intervallet +/- 90 % som används för att upprätta ett hälsovårdsprogram (definieras ej som en hälsokostnad i denna rapport då den avser hantering av problemet och inte effekter på människors hälsa). I det här exemplet är det angivna intervallet stort av två anledningar; dels på grund av bristen på tillgängliga data samt på grund av den möjliga variationen i genomförande av hälsovårdsprogram.

Liksom för de hälsorelaterade uppskattningarna har studien antagit att 3 % av den Europeiska befolkningen är exponerad för dricksvatten med PFAS-halter över de reglerade åtgärdsnivåerna, vilket gör att de vattenreningsverk som förser denna del av befolkningen med dricksvatten kommer att behöva förbättras och underhållas de närmsta 20 åren. Antagandet om 20 år återspeglar möjligheten att saneringen ska kunna lösa problemen, kanske genom rening eller användning av alternativ eller möjligheten för att saneringsåtgärder pågår under många år. På grund av de osäkerheter som finns i analysen och tillgängliga data har kostnader för sanering kvantifierats med hjälp av en scenaribaserad metod. För varje scenario har ett antal parametrar specificerats, exempelvis storleken av den drabbade befolkningen och tidsåtgången för underhållsarbete.

Tabell 43 visar kostnadsintervallen för olika kategorier av åtgärder kopplade till arbetet med att återställa och rena miljön.

**Tabell 43: Summerade kostnadsuppgifter för icke-hälsorelaterade utgifter, över 20 år,**

Vidtagen åtgärd vid fynd av PFASs	Enhet	Bästa uppskattning	Intervall från studier	Använt intervall
Övervakning av kontaminering från industriell användning eller AFFF	Kostnad per vattenprov	EUR 340	EUR 278–402	EUR 278–402
	Kostnad/fall av kontaminering	EUR 50000	EUR 5200–5,8 miljoner	EUR 25000–500000
Hälsobedömning (inklusive bioövervakning)	Kostnad/person	EUR 50	Inget intervall	EUR 5–95 (+/-90 %)
	Total bioövervakning & hälsobedömning per fall där så ansetts lämpligt	EUR 3,4 miljoner	EUR 2,5 miljoner–4,3 miljoner	EUR 1 miljoner–5 miljoner
Temporärt tillhandahållande av oförorenad resurs	Kostnad/person	Inga relevanta data		
Installerad av ny pipeline	Kostnad/person	EUR 800	EUR 37–5000	EUR 100–1500
Uppgradering av vattenverk (kapital)	Kostnad/person	EUR 300	EUR 8–2200	EUR 18–600
Uppgradering av vattenverk (underhåll)	Kostnad/person	EUR 19	EUR 8–30	EUR 8–30
Utgrävning och behandling av jord kontaminerad genom industriell eller AFFF användning	Kostnad/kg PFASs	EUR 280000	EUR 100000–4,3 miljoner	EUR 100000–1 miljoner
	Kostnad/fall	EUR 5 miljoner	EUR 100000–3 miljarder	EUR 300000–50 miljoner

Tabell 44 visar kostnadsintervall för olika åtgärds-kategorier relaterade till miljösanering för de fem nordiska länderna. Sammantaget rör det sig om ett kostnadsintervall på 46 miljoner–11 miljarder EUR.

**Tabell 44: Detaljerad översikt av intervall för de Nordiska länderna, med antagande att 5 % (bästa uppskattning 3 %) av populationen/befolkningen exponeras med halter över gällande gränsvärden och att vattenrening behövs under en 20 års period.**

	Antal exponerade personer (3 %)	Screening & övervakning	Hälsobedömning	Uppgradering av vattenverk & underhåll	Marksanering	Totalt
Danmark	170000	EUR 70000–8,3 miljoner	EUR 280000–27 miljoner	EUR 7,4 miljoner–274 miljoner	EUR 0–798 miljoner	EUR 8 miljoner–1,1 miljarder
Finland	160000	EUR 250000–22 miljoner	EUR 270000–26 miljoner	EUR 7,2 miljoner–265 miljoner	EUR 2,2 miljoner–2,1 miljarder	EUR 10 miljoner–2,4 miljarder
Island	10000	EUR 10000–900000	EUR 20000–1,6 miljoner	EUR 400000–1,6 miljoner	EUR 100000–86 miljoner	EUR 1 miljoner–105 miljoner
Norge	160000	EUR 170000–20 miljoner	EUR 260000–25 miljoner	EUR 6,8 miljoner–250 miljoner	EUR 1,6 miljoner–1,9 miljarder	EUR 9 miljoner–2,2 miljarder
Sverige	290000	EUR 480000–47 miljoner	EUR 490000–46 miljoner	EUR 13 miljoner–472 miljoner	EUR 4,3 miljoner–4,5 miljarder	EUR 18 miljoner–5,1 miljarder
<i>Norden totalt</i>	<i>790000</i>					<i>EUR 4,6 miljoner–11 miljarder</i>

Kostnadsberäkningarna som anges i Tabell 44 är sannolikt mer robusta för den sammanlagda europeiska nivån än för den nationella nivån.

Tabell 45 visar sammanlagda kostnader för miljöscreening, övervakning (där föreningar har hittats), vattenrening, marksanering och hälsobedömning för de fem nordiska länderna samt för övriga EES-länder och Schweiz.

**Tabell 45: Sammanlagda kostnader för miljöscreening, övervakning när föreningar upptäckts, vattenrening, marksanering och hälsobedömningar**

	Bästa uppskattning	Låg	Hög
Danmark	EUR 145 miljoner	EUR 8 miljoner	EUR 1,1 miljarder
Finland	EUR 214 miljoner	EUR 10 miljoner	EUR 2,4 miljarder
Island	EUR 12 miljoner	EUR 1 miljoner	EUR 105 miljoner
Norge	EUR 194 miljoner	EUR 9 miljoner	EUR 2,2 miljarder
Sverige	EUR 423 miljoner	EUR 18 miljoner	EUR 5,1 miljarder
Övriga EES+CH	EUR 15,9 miljarder	EUR 776 miljoner	EUR 159,9 miljarder
<i>Totalt</i>	<i>EUR 16,9 miljarder</i>	<i>EUR 821 miljoner</i>	<i>EUR 170,8 miljarder</i>

Motsvarande beräkningar för alla 31 EES-länder och Schweiz ger ett kostnadsintervall för miljösanering på EUR 821 miljoner till EUR 170 miljarder. De lägre och övre gränserna bör betraktas som symboliska på grund av den begränsade information som är tillgänglig. Baserat på informationen från litteraturstudien finns det anledning att tro att de nedre gränserna kommer överskridas. En uppskattning i storleksordningen EUR 10–20 miljarder är säkerligen rimlig. Troligtvis kan betydligt högre utgifter än så komma ifråga om flera fall med miljardkostnader för marksanering identifieras. En uppskattning av kostnaderna för ett fall som identifierats i samband med en studie gällande staden Rastatt i

Baden-Württemberg i Tyskland ligger inom intervallet EUR 1 till EUR 3 miljarder, ett intervall som antas komma att öka i omfattning med tiden. I detta fall anses källan till utsläppen ha varit förorenat material av återvunnet papper som spreds ut på jordbruksmark. Detta visar att allvarliga problem inte alltid är kopplade till flygfält och tillverkning av PFAS.

Ett antal andra kostnader relaterade till förorening av PFAS ligger utanför denna studies avgränsning och kvantifiering. Dessa inkluderar minskat värde på egendom, försämrat rykte för företag som förorenar, ekologiska skador, samt kostnader för att åtgärda förorenade områden som belastar berörda myndigheter – vilket inkluderar att informera allmänheten, att analysera förekomst av föroreningar samt att vidta saneringsåtgärder.

# Annex 1: National / state screening for PFAS contamination

This annex reviews the efforts of several EEA Member States and the USA to screen their territories to determine where PFAS contamination may pose risks to health and the environment. Two approaches for such screening can be distinguished. One approach is to identify the types of uses of PFAS that might lead to releases to the environment and then test water and soil in nearby areas for contamination. The other approach is to carry out comprehensive testing of drinking water supplies or wastewater treatment plant discharges and, if PFAS contamination is detected at levels of concern, to work upstream to identify the source of the contamination.

## Denmark

Groundwater is the major source of drinking water in Denmark. Denmark therefore regularly monitors groundwater, under a program called GRUMO<sup>363</sup>, as well as the wells supplying waterworks. A 2014 screening investigation looked for the presence of 7 PFAS in groundwater where uses of PFAS may have led to contamination<sup>364</sup> (Denmark has no direct production of PFAS). It drew on a 2005 survey of annual consumption of PFAS that estimated, of a total of 9–16 tonnes used that year, 50% was used in various textile, leather and paper products. PFOS and other PFAS were also used in paints and varnishes, in cleaning and polishing products, and in electroplating. Chromium plating was also targeted, since it was considered the largest consumer of PFOS in Europe at the time. Table A1.1 lists the industries and activities identified as potential sources of contamination, the numbers of screening investigations, and any PFAS contamination of groundwater identified in the 2014 screening report.

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<sup>363</sup> Geological Survey of Denmark and Greenland, Monitoring Programmes.

<sup>364</sup> Danish Ministry of Environment (2014). Screeningsundersøgelse af udvalgte PFAS-forbindelser som jor- og grundvandsforureng i forbindelse med punktkilder, Miljøprojekt nr. 1600.

**Table A1.1: Number of sites identified as potential sources of contamination and overview**

Industry/activity	No. sites investigated	PFAS in groundwater?	Concentration levels
Fire training facilities	8 sites	Yes	2 sites - 100 ng/l 2 sites - >1000 ng/l 4 sites - none found
Chromium plating	2 sites	No	na
Carpet industry	1 site	Yes	1500 ng/l
Paint industry	1 site	No	na
Landfills	4 sites	No	na

The investigation confirmed that fire drill sites were confirmed as potential sources of PFAS contamination, and recommended surveying the other large fire drill sites in Denmark that had used PFAS-containing aqueous film-forming foams (AFFFs). It reported on a comprehensive site investigation performed at another site (Copenhagen airport) that found concentrations of PFOS and PFOA at 500 times the Danish summed criteria of 100 ng/l (for 12 PFAS) in drinking water. The contamination on that site is contained by pumping and treatment by activated carbon filters to avoid contamination of drinking water.

A 2016 study<sup>365</sup> reviewed the Danish Product Register for information on professional uses of PFAS from 1983 to 2016. The reported data covered 152 compounds distributed over 27 industries and showed a decline of PFAS used from 98.5 tonnes in 2003 to 13.9 tonnes in 2016. It expanded the list of uses where contamination might have occurred to include the wood industry and furniture industry; the chemical industry, iron and metal industry and rubber and plastics industry; and other locations where direct emissions may have occurred, e.g., sites of chemical/oil fires. The study recommended that investigations for possible site contamination also should be carried out at sites where these types of uses occurred.

Concerning drinking water, testing was carried out for a total of 446 samples from 318 waterworks wells from 2013 to 2018.<sup>366</sup> The levels for all samples were below the summed criteria of 100 ng/l for the 12 PFAS monitored in Denmark.

In 2017, the Danish Regions tested 1730 groundwater samples near potential sources of contamination for the content of the 12 PFAS in covered by the Danish drinking limit value.<sup>367</sup> PFAS were found above the sum criteria in approximately 10% of the samples. The groundwater samples were taken in the upper part of the water table to identify soil contamination, rather than for testing of drinking water.

<sup>365</sup> Danish EPA (2016). Kortlægning af brancher der anvender PFAS.

<sup>366</sup> Personal communication; report not yet published (September 2018). Denmark has approximately 6,000 waterworks wells.

<sup>367</sup> Danish Regions (2018). Forebyggelse & samarbejde.

## Sweden

According to a 2016 Swedish EPA report<sup>368</sup>, 1,893 public water supplies were in use in 2015, 660 of which have been analysed for PFAS. About half of Sweden's public drinking water supply comes from surface water<sup>369</sup>; more than 80% of all surface water samples analysed contained detectable levels of PFAS. This includes sites with varying distances to identified PFAS sources. Available information concerning PFAS in groundwater is less reliable. Around 40% of the samples from areas with diffuse pollution, and 80% of samples from fire training sites contained detectable levels of PFAS.

In 2014, a survey of local authorities controlling drinking water in Sweden carried out by Swedish National Food Administration showed that around 3.6 million Swedes drink PFAS-contaminated water<sup>370</sup> – mainly the facilities using surface water. The levels were in most cases below 10 ng/l and were not considered to pose any threats to the public health. Also in 2014, five municipalities took actions related to their water supply in order to respond to PFAS contamination: Båstad, Ronneby, Halmstad, Uppsala and Botkyrka. Table A1.2 shows the number of sites sampled for different sources, and the analytical results.

**Table A1.2: PFAS detected in areas affected by different sources of emissions**

Source of PFAS	No of sites sampled		No of PFAS detected		Average levels of PFAS detected (ng/l)		Average levels of PFOS detected (ng/l)	
	SW	GW	SW	GW	SW (10 PFAS <sup>1</sup> )	GW (7 PFAS)	SW	GW
Fire training site	214	271	18	-	3 193	43 927	1 207	24 463
Diffuse pollution	34	53	10	-	99,5	209,6	42,3	23,5
Waste facility	1	-	-	-	Not analysed for 10 PFAS	-	21,6	-
Background area	60	-	14	-	13,5	-	1,5	-

Note: SW = surface water.

GW = groundwater.

1) PFOS, PFOA, PFHxS, PFHxA, PFHpA, PFNA, PFDA, PFBS, 6:2 FTSA and FOSA.

The 2016 report confirmed that the presence of PFAS in water tends to increase with proximity to likely contaminated areas. Nevertheless, detectable levels are also found in background areas far from any identified sources. The same survey identified more than 2,000 potential sources from which PFAS might enter the environment; the most common types being WWTPs and major fire incidents.<sup>371</sup>

<sup>368</sup> Swedish Environmental Protection Agency (2016). Highly fluorinated substances (PFAS) and pesticide, an overview of the presence in the environment (report in Swedish with English Summary).

<sup>369</sup> Swedish Water & Wastewater Association (2016). Produktion av dricksvatten.

<sup>370</sup> Swedish National Food Administration (2014). PFAA i råvatten och dricksvatten (report in Swedish)

<sup>371</sup> IVL 2016 as stated in Swedish Environmental Protection Agency (2016) report.

Table A1.3 lists the industries and activities identified as potential sources of contamination, the numbers of sites, and the estimated amount of PFAS released from each type of source into the environment.

**Table A1.3: Overview of various sources of PFAS and their estimated contributions to PFAS in the Swedish environment 2016<sup>1</sup>**

Industry/activity	No. sites identified <sup>2</sup>	Estimated contribution of PFAS
Production of PFAS	0	-
Production of PFAS-containing fire foam	2	Na
Waste water treatment plants (WWTP)	336	70 kg/year, of which PFOS represents 20 kg via water and 5 kg/year via sludge
Landfills (emissions from other parts of the waste treatment process are unknown)	365	70 kg/year (4 kg PFOS). 8 kg of total enters the environment; remainder enters WWTPs
Civilian airports (connected fire drilling sites)	10	1 600 kg total (380 kg PFOS) 1970–2000s
Military airports (connected fire drilling sites)	18	9 700 kg total (2 300 kg PFOS) 1970–2000s
Other fire drilling sites	295	Unknown
Fire incidents between 1998–2014 (registered by the Swedish Civil Contingencies Agency)	9,000	660 kg total (150 kg PFOS)
Chromium plating industries	11 (of which 3 use PFOS)	180 kg PFOS/year (2013, per exemption) via waste water and air
Direct emissions from other industry	Na	Na
Atmospherical deposition	Across all of Sweden	650–1 700 kg/year (25–30 kg PFOS)

Note: 2) A follow-up study in 2018 increased the number of fire incidents where AFFFs were used to 13,500 and doubled the number of other sites where AFFFs had been handled to approximately 800<sup>3</sup>

Source: 1) Hansson *et al.* (2016). IVL-report C 182: Sammanställning av befintlig kunskap om föroreningskällor till PFAS-ämnen i svensk miljö (report in Swedish).

3 Swedish EPA (2018). Fördjupad miljöövervakning av högfluorerade miljögifter (s.k. PFAS) och av växtskyddsmedel i vatten NV-08978-16 (report in Swedish).

## Finland

Finland has recognized the use of AFFFs as a major source of dispersal of PFAS compounds. The OECD chemical safety portal<sup>372</sup> reports screenings of soil near airports and firefighting training centers along with screenings of drinking water.<sup>373</sup> The first screening in 2015 focused on military sites and use of AFFFs. It collected samples from surface (6) and ground (18) water, wastewater (5), soil samples (4) and sediment samples (3). Half of the samples contained PFCs above detection levels, with most dominant compounds being PFOS and PFOA and to a lesser extent PFBA, PFHxA AND PFHpA. The samples taken from areas where firefighting foams were used showed the highest concentrations.<sup>374</sup>

<sup>372</sup> OECD chemical safety portal (2018). Country information for Finland.

<sup>373</sup> Ibid.

<sup>374</sup> Ryyänen T (Construction Establishment of Finnish Defence Administration) (2017). Per- and polyfluorinated substances in the Finnish Defence Forces.

## Belgium (Flanders)

Flanders has undertaken stand-alone screening in order to determine the scope of contamination from PFAS in groundwater and soil. Various risk locations were identified, based on types of activities carried out in their vicinity. The activities included fire-fighting sites, industrial uses of PFAS (chromium plating, textile and paper manufacturers and the paint industry). A total of 24 sites were selected and 40 soil and 1 groundwater sample were tested. In 66% of the risk locations, sample concentrations were 10 times higher than the reporting limit. In 24% of the locations, concentrations were 1000 times higher than the reporting limit.<sup>375</sup>

## The USA (national monitoring)

From 2013 to 2015, under the third Unregulated Contaminant Monitoring Rule (URCM 3), the US Environmental Protection Agency carried out testing for PFAS at a representative sample of public water systems serving less than or equal to 10,000 persons. Some 37,000 samples from 5000 public water systems were tested for PFOS, PFOA, PFNA, PFHxS, PFHpA and PFBS.<sup>376</sup>

A spatial analysis of the PFAS (PFOS and PFOA) found in drinking water due to the URCM 3 monitoring arrived at an estimate of six million U.S. residents served by public water supplies at levels above the US EPA's lifetime health advisory limit (70 ng/l).<sup>377</sup> The number of industrial sites manufacturing or using PFAS, the number of military fire training areas, and the number of wastewater treatment plants were significant predictors of the concentrations of PFAS in the public water supplies tested.

As of April 2018, at least 126 military bases in the USA reported potentially harmful levels of PFAS.<sup>378</sup> A total of 401 active and base closure sites had at least one area where a known or suspected release of PFAS had occurred. Starting in 2017, the US Department of Defense (DoD) has undertaken a complete monitoring of its drinking systems throughout the country.<sup>379</sup> As of 2018, 524 DoD water drinking systems were tested and 24 showed PFOS/PFOA levels above the US EPA lifetime health advisory levels. Monitoring of off-base private drinking systems resulted in the testing of 2,445 wells; 564 among those showed levels above the US EPA advisory levels.<sup>380</sup> The DoD has already spent 200 million USD studying and testing water supplies and providing either filters,

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<sup>375</sup> OVAM (2018). Onderzoek naar Aanwezigheid van PFAS in Grondwater, Bodem en Waterbodem ter hoogte van Risicoactiviteiten in Vlaanderen.

<sup>376</sup> EPA (2017). The Third Unregulated Contaminant Monitoring Rule (UCMR 3): Data Summary.

<sup>377</sup> Hu, X *et al.* (2016). Detection of Poly- and Perfluoroalkyl Substances (PFASs) in U.S. Drinking Water Linked to Industrial Sites, Military Fire Training Areas, and Wastewater Treatment Plants, *Environmental Science & Technology Letters* (10)3, pp. 344–350.

<sup>378</sup> Copp T, DoD: At least 126 bases report water contaminants linked to cancer, birth defects, *Military Times*, 26 April 2018. Accessed 01.11.2018.

<sup>379</sup> US Department of Defense (2017). Aqueous Film Forming Foam Report to Congress.

<sup>380</sup> US Department of Defense (2018) Addressing Perfluorooctane Sulfonate (PFOS) and Perfluorooctanoic Acid (PFOA).

alternate wells or bottled water where supplies were contaminated. The cost of cleaning up PFAS-contaminated water at military sites was estimated at 2 billion USD.

## The US state of Michigan

In 2017, the state of Michigan set up the Michigan PFAS Action Response Team (MPART) – a multi-agency group linking health, environment and other state government agencies in an effort to investigate possible hot spots of PFAS contamination and to act to protect drinking water sources.<sup>381</sup> The program started after several incidents of contamination of water were discovered near military bases where AFFFs had been used as well as historic pollution at a former leather treatment factory.

The state established a health standard for PFOS and PFOA (70 ppt, the same level as the US EPA lifetime advisory standard) and tested drinking water from public water supplies and in schools across the state. At the end of August 2018, the program had tested 892 of the state's 1,841 public water systems along with schools that operated their own wells). Four public water systems were found to have problems, including the city of Flint. The state's Department of Environmental Quality looked for the source of the contamination where concentrations exceeded background levels.

Michigan is also testing effluent from 90 wastewater treatment plants, as part of a program monitoring industrial pre-treatment of wastewater. In addition, the Department of Environmental Quality is sampling for PFAS when investigating the state's Superfund sites. As of September 2018, the Michigan PFAS response website listed 34 sites where contamination had been identified.<sup>382</sup>

In April 2018, elevated levels of PFOS in the Flint River were linked to discharges from a plating and plastics company that had reportedly not used PFOS for years.<sup>383</sup> A few months later a creek that fed into the Huron River was found to have 5,500 ppt of PFOS, more than 450 times the state limit for surface water. The contamination was traced to another company<sup>384</sup> supplying chrome-plated plastic components to automobile manufacturers. At one time, total PFAS downstream on the Huron River tested at 56,868 ppt. Due to high levels of PFAS in fish caught in the Huron River, the state issued a "do not eat fish" advisory for 30 sq. miles of the river.

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<sup>381</sup> State of Michigan, Website dedicated to PFAS contamination.

<sup>382</sup> State of Michigan, PFAS Sites Being Investigated.

<sup>383</sup> No Author, Black Cloud of PFOS, The County Press, 15.04.2018. Accessed 08.11.2018.

<sup>384</sup> Gardner P, Astronomical PFAS level sets new Michigan contamination milestone, Mlive, 24.09.2018. Accessed 05.11.2018.

# Annex 2: Health impacts – additional information on evidence and calculations

This annex presents additional information regarding the health assessments.

*Part 1* explains the calculations for each monetised health impact in more detail as well as the country-specific estimates.

*Part 2* presents an overview of the key epidemiological studies drawn upon for the assessment. It provides information about the region and population studied, the PFAS compounds in the contamination, the relevant health endpoint, the time period and the sample size. For example, the first study mentioned (Mastrantonio *et al.*, 2017) was used for the calculation of health impacts among the population with elevated exposure through close residence to a chemical plant and AFFFs contamination.

Upon request the excel spreadsheets used for the monetisation and valuation in this report can also be provided along with a guidance on how to use the estimation of costs for value transfer.

## Part 1: Estimating health impacts in exposed populations

This section explains the calculations underpinning each of the quantitative estimates presented in Section 5.1.

### *Occupational (high) exposure scenario*

The estimation for the occupational exposure scenario relied on a number of parameters and assumptions, which are listed below:

- assume that the number of PFAS-producing plants in the EEA ranges from 12 to 20 (Case Study 1);
- assume there are 352,764 small manufacturing plants and 780 large manufacturing plants across the EEA in the categories of manufacturing and commercial uses identified in Case Study 2 (textiles, leather, metal plating, paper and paper products, paints and varnishes, soaps and detergents, plastics and resins, car wash establishments);
- assume that the number of employees (exposed population) in small manufacturing plants is on average 30 while the number of employees in large plants is on average 300;

- assume that an estimated 3% to 10% of the types of manufacturing plants identified in Case Study 2 use PFAS-treated products; and
- the elevated risk of death due to kidney cancer from occupational exposure to PFOA (relative risk = 3.67). This estimate was obtained from an epidemiological study carried out in West Virginia.<sup>385</sup> The elevated risk was assumed to apply to the exposed population in the Nordic and EEA countries.

The calculation began with an estimation of the exposed population of workers in the Nordic countries and the EEA using information from the first three assumptions. Table A2.1 presents the results.

**Table A2.1: Estimating the size of the population with occupational exposure to PFAS**

	Best estimate	Assumption for calculation	Ave. workers in each	Nordic*	EEA*	Estimated exposed population
PFAS plants	12–20	Assume upper estimate	500	-	✓	10,000
Manufacturing plants – small	352,764	Assume that 3–10% have PFAS treated products	30	✓	✓	31,749–105,829
Manufacturing plants – large	780		300	✓	✓	702–2,340
Total:						334,508–1,091,692

Note: \*The actual numbers are not known and therefore a best guess was made to support the calculation.

We considered the death rate in this population which might be attributed to kidney cancer due to PFAS exposure. The number of deaths was estimated and compared with a scenario where occupational PFAS exposure was not present. This calculation relied on the third parameter from Steenland and Woskie, 2012. The elevated risk of kidney cancer mortality was evident for a quartile of the exposed population. Similarly, the calculation for this study assumed that the only a quarter of the population with occupational exposure faced a higher risk of kidney cancer due to PFAS exposure.

Table A2.2 presents the calculation for the Nordic and EEA countries together. The lower bound is based on the assumption that 3% of selected manufacturing plants make PFAS-treated products while the upper bound uses the assumption of 10%. These assumptions were made due to the absence of actual figures.

<sup>385</sup> Steenland K and Woskie S (2012). Cohort mortality study of workers exposed to perfluorooctanoic acid. American journal of epidemiology. 176(10) pp.909–917.

**Table A2.2: Calculation of monetised impact of elevated mortality from kidney cancer due to occupational PFAS exposure – EEA countries**

	Lower bound	Upper bound	Explanation/source
Exposed population	334,508	1,091,692	See Table A2.1 above
Population that experiences elevated health risk	83,627	272,923	Assumes only a quartile of the exposed population experiences an elevated risk of kidney cancer mortality
General population death rate by kidney cancer	0.01%	0.01%	Eurostat, 2015 standardised death rate
Annual deaths in exposed population - baseline	4.98	16.27	Milieu calculations using the elevated risk information from (Steenland and Woskie, 2012).
Not linked to PFAS exposure:	1.4	4.4	
Linked to PFAS exposure:	3.6	11.8	
Total value per year	EUR 12.7 million	EUR 41.4 million	Milieu calculations using ECHA lower bound value of life (EUR 3.5 million)

### *Elevated (medium) exposure scenario*

Three calculations were made for the scenario. The first was related to the costs of the elevated risk of all-cause mortality among adults. The second was an estimation of the number of births of low birth weight. The third was related to the higher risk of common childhood infections among children (immunotoxicity endpoint). Each is described below.

The estimation for the elevated risk of all-cause mortality drew on two key sources of information. One was an epidemiological study from the Veneto region.<sup>386</sup> Another was an estimate from Sweden on the human exposure to elevated levels of PFAS in groundwater. The key assumptions were:

- a relative risk of all-cause mortality due to elevated PFAS exposure. For this, we relied on an estimate from an epidemiological study carried out in the Veneto Region.<sup>387</sup> The study found a relative risk of 1.11 and a 95% confidence interval of 1.10 to 1.12. The calculation assumes that the elevated risk found in the Veneto Region applies to the exposed population in the Nordic and EEA countries;
- up to 300,000 residents of Sweden (or about 3%) are exposed to source levels of PFAS above the limit value via municipal drinking water.<sup>388</sup> The calculation assumes that the level of exposure in Sweden is equivalent across the Nordic countries and the EEA.

<sup>386</sup> Mastrantonio M *et al.* (2017). Drinking water contamination from perfluoroalkyl substances (PFAS): an ecological mortality study in the Veneto Region, Italy. *The European Journal of Public Health*. Feb 1;28(1):180–185.

<sup>387</sup> *Ibid.*

<sup>388</sup> Holmström *et al.* (2014). Nationell screening av perfluorerade föroreningar (PFAA) i dricksvatten. Rapport no 2014/20 (In Swedish).

Table A2.3 presents the calculation. The lower and upper bounds are based on the values of the 95% confidence interval from the estimate from Mastrantonio *et al.*, 2017.

**Table A2.3: Calculation of annual monetised impact of elevated mortality due to elevated PFAS exposure – Nordic and EEA countries**

	Nordic countries		EEA countries		Explanation/source
Total population	20,698,030		415,697,178		Population ages 19 years and up (Eurostat, 2017)
Population with elevated exposure	620,941		12,470,915		3% parameter estimate applied
General mortality rate	1.0%				Eurostat, 2017
Annual deaths in exposed population - baseline	6,458		129,199		Milieu calculations using the elevated risk information from Mastrantonio <i>et al.</i> , 2017.
	Lower bound	Upper bound	Lower bound	Upper bound	
Not linked to PFAS exposure:	5,871	5,766	117,453	115,356	
Linked to PFAS exposure:	587	692	11,745	13,843	
<i>Total value per year</i>	<i>EUR 2.1 billion</i>	<i>EUR 2.4 billion</i>	<i>EUR 41.1 billion</i>	<i>EUR 48.5 billion</i>	<i>Milieu calculations using ECHA lower bound value of life (EUR 3.5 million)</i>

The estimation for the elevated risk of low birth weight drew on three sources of information:

- number of total live births and percentage of low birth weight by country (Eurostat);
- elevated risk of low birth weight due to PFAS exposure. The calculation assumed that an estimate from an epidemiological study could be extrapolated to the exposed population in the Nordic and EEA countries. The study found a relative risk of low birth weight of 1.50<sup>389</sup>;
- population exposure to elevated levels of PFAS, estimated to be 3%.<sup>390</sup> This parameter was also used in the previous calculation. The calculation assumes that the level of exposure in Sweden is equivalent across the Nordic countries and the EEA.

<sup>389</sup> Stein C R *et al.* (2009). Serum levels of perfluorooctanoic acid and perfluorooctane sulfonate and pregnancy outcome. *American journal of epidemiology*, 170(7), pp.837–846.

<sup>390</sup> Holmström *et al.* (2014). Nationell screening av perfluorerade föreningar (PFAA) i dricksvatten. Rapport no 2014/20 (In Swedish).

Table A2.4 presents the calculation.

**Table A2.4: Calculation of births of low birth weight due to elevated PFAS exposure – Nordic and EEA countries**

	Nordic countries	EEA countries	Explanation/source
Number of births per year	294,777	5,211,464	Eurostat, 2016
Births with elevated exposure	8,843	156,344	3% parameter estimate applied
Low birth weight rate	4.6 %	6.8 %	Eurostat, 2017
Annual low birth weight in exposed population - baseline	407	10,631	Milieu calculations using the elevated risk information from Stein <i>et al.</i> , 2009.
Not linked to PFAS exposure:	271	7,088	
Linked to PFAS exposure:	136	3,544	

The estimation for the elevated risk of fever is based on findings from a study from Denmark.<sup>391</sup> The study found that children ages 1–4 years of age with the highest serum concentration of PFAS (10.19–25.10 ng/ml) had an increased risk of fever. This serum concentration level most closely corresponds with the elevated exposure scenario. The calculation assumed that all children aged 1–4 years with an elevated exposure would face an increased risk of fever. The key pieces of information and assumptions were as follows;

- number of children ages 1–4 years of age in the Nordic countries and the EEA (Eurostat);
- elevated risk of infection. The calculation relies on the aforementioned Danish study, which finds a relative risk of fever of 1.65.<sup>392</sup> The calculation assumes that the elevated risk of additional days with fever found in the study from Denmark applies to the entire population at elevated exposure in the Nordic countries and the EEA;
- population exposure to elevated levels of PFAS, estimated to be 3%.<sup>393</sup> This parameter was also used in the two previous calculations. The calculation assumes that the level of exposure in Sweden is equivalent across the Nordic countries and the EEA.

<sup>391</sup> Dalsager L *et al.* (2016). Association between prenatal exposure to perfluorinated compounds and symptoms of infections at age 1–4 years among 359 children in the Odense Child Cohort. *Environment international*, 96, pp.58–64.

<sup>392</sup> Ibid.

<sup>393</sup> Holmström *et al.* (2014). Nationell screening av perfluorerade föroreningar (PFAA) i dricksvatten. Rapport no 2014/20 (In Swedish).

Table A2.5 presents the calculation.

**Table A2.5: Calculation of additional days of fever among children 1–4 years old – Nordic and EEA countries**

	Nordic countries	EEA countries	Explanation/source
Number of children ages 1–4 years	1,507,631	26,159,812	Eurostat, 2016
Children with elevated exposure	45,229	784,794	3% parameter estimate applied
Fever days per year in baseline	4.7 days	4.7 days	Eurostat, 2017
Fever days in exposed population	212,576	3,688,533	Milieu calculations using the elevated risk information from Dalsager <i>et al.</i> , 2016.
Not linked to PFAS exposure:	128,834	2,235,475	
Linked to PFAS exposure:	83,742	1,453,059	

### *Background (low) exposure scenario*

The estimation of health impacts from background exposure to PFAS focused on the health endpoint of hypertension. It drew on epidemiological evidence gathered from the National Health and Nutrition Examination Survey in the United States, which focused on the adult population.<sup>394</sup> The parameters and assumptions in the calculation include the following:

- the adult population in the Nordic countries and the EEA (Eurostat);
- the incidence rate of hypertension in the EU<sup>395</sup>;
- elevated risk of hypertension. The calculation relies on an estimate for the elevated risk of hypertension due to PFAS exposure.<sup>396</sup> The study found an odds ratio of 1.63 and a 95% confidence interval of 1.2 to 2.2;
- elevated risk of death due to hypertension. The calculation relies on an estimate from Zhou *et al.*, 2018.

Min *et al.*, 2012 find an increased risk of hypertension in half of the population. The calculation assumes that half of the exposed population in the Nordic countries and the EEA face an elevated risk of hypertension. Table A2.6 show figures from calculations of the monetised impact of elevated risk of hypertension due to background exposure to PFAS for the Nordic and EEA countries.

<sup>394</sup> Min, J. Y. *et al.* (2012). Perfluorooctanoic acid exposure is associated with elevated homocysteine and hypertension in US adults. *Occup Environ Med*, 69(9):658–62.

<sup>395</sup> European Heart Network (2017). *European Cardiovascular Disease Statistics 2017*.

<sup>396</sup> *Ibid.*

**Table A2.6: Calculation of monetised impact of elevated risk of hypertension due to background exposure to PFAS – Nordic and EEA countries**

	Nordic countries		EEA countries		Explanation/source
	Lower bound	Upper bound	Lower bound	Upper bound	
Exposed population	20,698,030		415,697,178		Adult population (19 years and up), Eurostat, 2017
Population at higher risk	10,349,015		207,848,589		About half have an increased risk of hypertension (Min <i>et al.</i> , 2012).
Incidence rate of hypertension	0.01				European Cardiovascular Disease Statistics, 2017
Number of new hypertension cases	75,931		1,525,000		Milieu calculations using the elevated risk information from Min <i>et al.</i> , 2012.
Elevated risk of hypertension due to PFAS exposure	1.2	2.2	1.2	2.2	
Not linked to PFAS exposure:	63,276	34,514	1,270,833	693,182	
Linked to PFAS exposure:	12,655	41,417	254,167	831,818	
Elevated risk of disease due to hypertension	0.012				Zhou <i>et al.</i> , 2018
Number of deaths	916		18,398		Milieu calculations using the elevated risk information from Zhou <i>et al.</i> , 2018.
Not linked to PFAS exposure:	763	416	15,331	8,363	
Linked to PFAS exposure:	153	500	3,066	10,035	
	EUR 0.7 billion	EUR 2.2 billion	EUR 10.7 billion	EUR 35 billion	Milieu calculations using ECHA lower bound value of life (EUR 3.5 million)

## Part 2: Key epidemiological studies

An overview of the epidemiological studies reviewed in this report is presented in Table A2.7.

**Table A2.7: Overview of epidemiological studies reviewed**

Study	Population	PFAS Compound	Health end-point	Time period	Sample size
Mastrantonio <i>et al.</i> , 2017	Veneto Region	PFOA, PFOS, PFHxS	Mortality	1980–2013	41,841 deaths; 143 605 residents in contaminated area; 588 012 residents in uncontaminated areas
Vieira <i>et al.</i> , 2013	C8 Health Project	PFOA	Cancer	1996–2005	29,118 cases of cancer; resident population over 500,000
Steenland <i>et al.</i> , 2010	C8 Health Project	PFOA, PFOS	Uric acid	2005–2006	54,951 adults
Barry <i>et al.</i> , 2013	C8 Health Project	PFOA	Cancer	2005–2006	32,254 adults; 3,589 cancer cases
Steenland <i>et al.</i> , 2009	C8 Health Project	PFOA, PFOS	Serum lipids	2005–2006	46,294 adults
Byrne <i>et al.</i> , 2018	St Lawrence Island, Alaska	PFOA, PFOS, PFUnA, PFNA	Thyroid hormone	2013–2014	85 adults
Min <i>et al.</i> , 2012	USA - background levels	PFOA	Hypertension	2003–2006	2,934 adults
Nelson <i>et al.</i> , 2010	USA - background levels	PFOA, PFOS, PFHxS, PFNA	Cholesterol	2003–2004	860 individuals
Bonfeld-Jorgensen <i>et al.</i> , 2011	Greenland	PFOA, PFOS	Breast cancer	2000–2003	31 breast cancer cases and 115 controls
Simpson <i>et al.</i> , 2013	C8 Health Project	PFOA	Stroke	2005–2006	32,254 individuals aged 12 years and above
Winquist and Steenland, 2014	C8 Health Project	PFOA	Hypertension and cholesterol	2005–2006	32,254 individuals aged 12 years and above
Steenland <i>et al.</i> , 2013	C8 Health Project	PFOA	Ulcerative colitis	2005–2011	32,254 individuals aged 12 years and above
Bonfeld-Jorgensen <i>et al.</i> , 2011	Greenland	PFOA, PFOS, PFHxS, PFNA, PFOSA	Breast cancer	1996–2002	250 breast control cases and 233 controls
Steenland and Woskie, 2012	C8 Health Project - workers	PFOA	Mortality	1952–2008	1,308 workers; 243 deaths
Brede <i>et al.</i> , 2010	Arnsberg, Germany	PFOA	None	2008	138 individuals
Jensen <i>et al.</i> , 2015	Denmark	PFOA, PFOS, PFNA, PFDA	Miscarriage	2010–2012	2,874 in relatively exposed area and 336 in a relatively unexposed area
Shankar <i>et al.</i> , 2012	USA - background levels	PFOA	Cardiovascular disease	1999–2003	1, 216 adults
Alexander <i>et al.</i> , 2003	Decatur, Alabama (USA)	PFOS	Mortality; Bladder cancer	1998	2,083 individuals; 145 deaths
Bach <i>et al.</i> , 2016	Denmark	PFOA, PFOS, PFHxS, PFUnA, PFNA, PFHpS, PFDA	Birth weight	2008–2013	1,507 mother-child dyads
Wang <i>et al.</i> , 2016	Taiwan	PFOA, PFUnA, PFNA, PDFeA, PFUnDA, PFDoDA	Fetal and post-natal growth	2000–2001	223 mother-child dyads
Yang <i>et al.</i> , 2016	Beijing, China	PFOA, PFOS, PFUnA, PFNA, PFDA	Thyroid hormones	2013	157 mother-child dyads
Ingelido <i>et al.</i> , 2018	Veneto Region	PFOA, PFOS	None	2015–2016	507 subjects, 257 in high exposure areas

## Annex 3: Data used for environment-related cost calculations

This annex presents the data used and the calculations carried out for the environment-related cost estimates in three parts:

- Part 1: The costs gathered via the case studies and additional research;
- Part 2: Data used in the aggregation of costs as presented in Section 5.2 of the study;
- Part 3: Full cost estimates by country.

Upon request the excelspread sheets used for the monetarisation and valuation in this report can also be provided along with a guidance on how to use the estimation of costs for value transfer.

### Part 1: The costs gathered via the case studies and additional research

This Annex reports the data collected for the following cost elements:

1. Monitoring to assess PFAS contamination where it is suspected (Table A3.2 and Table A3.3).
2. Provision of a temporary uncontaminated drinking water supply (Table A3.4).
3. Upgrading of water treatment works and ongoing costs for maintenance and replacement and disposal of filters (Table A3.5).
4. Excavation and treatment of soils (Table A3.6).
5. Health assessments where contamination is found (Table A3.7).

### Exchange rates used

All currency data have been converted to Euro in 2017 prices. The exchange rates used are based on Purchasing Power Parity (PPP), from OECD. Purchasing power parities are the rates of currency conversion that equalise the purchasing power of different currencies by eliminating the differences in price levels between countries. In their simplest form, PPPs show the ratio of prices in national currencies of the same good or service

in different countries. PPPs are also calculated for groups of products and for each of the various levels of aggregation up to and including GDP. The basket of goods and services priced is a sample of all those that are a part of final expenditure: household consumption, government services, capital formation and net exports, covered by GDP. The original indicator is measured in terms of national currency per US dollar. It will be noted that there is variation between PPP rates and market exchange rates. Inflation has been accounted for using the HICP indicator from Eurostat. Key data for currency conversion to 2017 EUR are given in Table A3.1. All costs that follow in the tables, etc. presented in the report have been updated to 2017 EUR.

**Table A3.1: Conversion rates used**

	EU28 HICP price index, 2015=100	EUR/Danish Krone	EUR/Norwe- gian Krone	EUR/Swe- dish Krona	EUR/USD	EUR/GBP
2008	89.82	0.0995	0.0892	0.0900	0.7904	1.1254
2009	90.71	0.0984	0.0838	0.0853	0.7609	1.0718
2010	92.60	0.1008	0.0836	0.0848	0.7644	1.0883
2011	95.47	0.1010	0.0830	0.0853	0.7543	1.0680
2012	97.99	0.0999	0.0836	0.0873	0.7556	1.0769
2013	99.47	0.0999	0.0813	0.0854	0.7345	1.0561
2014	100.01	0.1006	0.0794	0.0845	0.7370	1.0559
2015	100.00	0.1031	0.0776	0.0844	0.7551	1.0832
2016	100.25	0.1000	0.0727	0.0811	0.7360	1.0484
2017	101.97	0.1005	0.0715	0.0799	0.7274	1.0196

Source: PPP conversion rates: OECD, <https://data.oecd.org/conversion/purchasing-power-parities-ppp.htm>

Harmonised Index of Consumer Prices: Eurostat, <https://ec.europa.eu/eurostat/web/hicp/data/database>

## Monitoring to assess PFAS contamination where it is suspected

Data identified so far for monitoring costs, in most cases relating to early site investigations, are shown in Table A3.2 and Table A3.3 for Europe and the USA. The annual sampling cost cited for Ronneby Airport is a figure for the period after site investigation and remediation, and is understood to possibly persist for many years. For most sites considered, the total costs of monitoring alone are in excess of EUR 100,000, and in several cases exceed EUR 1 million. Only partial estimates of cost have been identified for some sites.

**Table A3.2: Costs for PFAS monitoring, European data**

Location	Sampling	Cost
Arlanda Airport	Sampling of groundwater, annual cost, to 2016	EUR 5,200
	Annual cost for site investigations, 2017–2022	EUR 8,000
Baden-Wurtemberg	Investigation and research following identification of PFAS contamination	EUR 6.1 million
Bromma Airport	Sampling and consultant costs for area where contamination was found	EUR 32,000
Dusseldorf (fire at bottle depot)	Total cost of assessment	EUR 1.2 million
Kallinge Airport	Additional cost for water sampling within the monitoring programme	EUR 4,000
Kiruna Airport	Investigation of a confined potential soil contamination	EUR 8,100
	Estimated cost for implementing action plan, including: identifying risk objects, technical soil and water investigation, assessment of risks and need for measures, etc.	EUR 1.6 million
Norwegian coastline	Annual costs for analysis only, does not include sampling and reporting	EUR 36,000
Oslo fjord		EUR 21,000
Atmospheric contaminants (Norway)		EUR 32,000
Screening of emerging contaminants (Norway)		EUR 36,000
Riverine inputs and direct discharges to Norwegian coastal waters		EUR 4,000
Contaminants in Norwegian lakes		EUR 14,000
Pollutants in the Norwegian terrestrial and urban environment		EUR 28,000
Ronneby Airport	Annual sampling cost after investigation and remediation	EUR 3,400
Stadtwerke Rastatt	Measuring groundwater wells in WSG Ottersdorf	EUR 440,000
Stadtwerke Rastatt	Measuring groundwater wells (WSG Raental)	EUR 110,000
Umeå Airport	Sampling and investigation	EUR 110,000
Uppsala	Future annual additional costs to investigate potential PFAS-sources	EUR 16,000 to EUR 56,000
Tyrifjorden	Field sampling	EUR 11,000
	Additional sampling of sediments, incl. reporting	EUR 3,600
	Admin, coordination fieldwork	EUR 720
	Analyses	EUR 13,000
	Analyses of additional sediment samples	EUR 3,600
	Reporting	EUR 11,000
	Budgeted future cost, fieldwork, analysis, reporting, admin	EUR 140,000
Uppsala	Sampling associated with contaminated water supply	EUR 44,000
	Average cost for 2012–2014	EUR 160,000
	Average cost for 2015–2017	

**Table A3.3: Costs for PFAS monitoring, US data**

Sampling		No. samples	Cost	Cost/sample
Washington State, USA	Monitoring	500	EUR 170,000	EUR 402
	Monitoring	8,000	EUR 1,900,000	EUR 278
	Monitoring	24,000	EUR 5,800,000	EUR 285
US Military sites	Additional testing e.g. of specific incidents		EUR 2,200,000	

For the US data, the sampling regime under these different options includes costs of sample analysis and staff time to oversee testing and assist communities with drinking water contamination. Washington State foresees a need to monitor all public water supplies, down to *Group B public water systems with fewer than 15 connections and fewer than 25 people per day*.

### Provision of a temporary uncontaminated drinking water supply

The only information obtained so far on the costs of supplying water temporarily is from the USA (Table A3.4). A widely reported figure of USD 10 million for provision of a temporary filtration system at Hoosick Falls, New York State and investigation into an alternative drinking water source is of a similar magnitude to the costs reported elsewhere for permanent systems. This suggests that the responsible authorities are seeking an alternative source with no contamination (as has been done at Bennington, just across the State border in Vermont), but that this will take several years to come online, essentially meaning that there is no difference between what is defined as a “temporary” solution for Hoosick Falls and permanent solutions elsewhere.

Another figure is available for the rental of two large filtration tanks at Hoosick Falls at a cost of USD 300,000.<sup>397</sup> It is unclear how this temporary installation differs to anything more permanent. A further temporary measure involved provision of free bottled water for collection from local supermarkets though there are no data available on the costs of this for Hoosick Falls. However, contamination at Peterson, Colorado, led to bottled water being provided. Costs for this are given as EUR 81,000, though it is unclear how many people were provided with the water which was intended specifically for residents using private wells or small drinking water systems.<sup>398</sup> The total population in the area of concern is given as 60,000, of which it is estimated that 10–15,000 received water at levels above the health advisory threshold.<sup>399</sup> Considering the likely costs of providing water per head, it seems likely that group served by the EUR 81,000 fund for bottled water was only a very small part of the 10–15,000 in the zone of highest concern.

<sup>397</sup> DeMasi M, Hoosick Falls water filtration still weeks away, Albany Business Review, Jan 29, 2016. Accessed 05.08.2018.

<sup>398</sup> KRRC (2016). Air Force Signs Contract for Bottled Water Distribution.

<sup>399</sup> KRRC (2016). Health & Water Officials Try to Reassure Residents in Areas of PFC Contamination.

**Table A3.4: Costs normalised against population for temporary improvements to water systems**

Site	Action	Population	Cost	Cost/head
Hoosick Falls	Provision of bottled water	3,400	No data	
Peterson, Colorado	Provision of bottled water		EUR 81,000	
<b>Temporary filtration system (1)</b>				
Hoosick Falls	Installation of temporary filters	3,400	EUR 270,000	EUR 79
<b>Temporary water filtration system (2)</b>				
Hoosick Falls	Temporary water filtration system and investigation into an alternative drinking water source	3,400	EUR 8.6 million	EUR 2,500

Press reports from Hoosick Falls indicate that when water supplies are declared clear of contamination there is still mistrust, with some people preferring to carry on drinking bottled water.<sup>400</sup> Irrespective of the rationality behind the decisions made by individuals, this will also represent a social cost.

### Upgrading of water treatment works and ongoing costs for maintenance and replacement and disposal of filters

Table A3.5 show costs normalised against population for improvements to water treatment in response to PFAS contamination, in cases where some reasonable estimate of the human population affected can be made. Cost data are also presented where no estimate of population is possible, for completeness. Within the table, categories of cost are grouped, starting with installation of advanced filters. Each group shows economies of scale, with cost/head reducing as the population served increases, as would be expected.

A general problem with the cost data lies in understanding what elements of cost are covered. For example, the cost given for upgrading a WWTP may cover the capital costs of plant upgrade and subsequent maintenance costs, or the capital costs alone. In some cases costs are reported as being inclusive or exclusive of VAT, whilst in others no comment is provided. It is also often unclear whether maintenance costs, when provided, are represented as additional annual maintenance costs, or total maintenance costs. In some cases the cost data are separated out so that it is (reasonably) clear what is covered. In others, costs are reported as total but without further explanation. Where possible, original sources have been consulted in order to understand the data better.

Estimates of the population served by each plant are approximate, based on the population of the nearest town or city. This introduces potentially significant error: for example, the low cost relative to size (EUR 8/person) for Dusseldorf may result from exaggeration of the population served. The issue is also highlighted in the case of Bennington (USA), for which two estimates of the cost per head for installation of new pipework to connect homes to an alternative water source are possible (not included in the table). The

<sup>400</sup>Ward C, Hoosick Falls free bottled water program comes to an end. News10 (01 September 2017). Accessed 18.10.2018.

total cost of this action is given as USD 20 million<sup>401</sup> (EUR 17 million). To derive a low estimate of damage per head, we divide by the total population of Bennington, 15,764, and reach a figure of EUR 1,085 per person. However, Saint-Gobain, the company funding the work as it is linked to a factory that they were responsible for, state that they have provided connection to 200 homes. Assuming an average occupancy rate of 2.5 people per home gives a total population 500. Dividing the USD 20 million cost of the work by this much lower population drives cost per head up to EUR 34,000 (and per property to EUR 85,000).

With respect to variation in estimates, a Swedish expert who has worked on PFAS issues at a number of airports<sup>402</sup> risk assessment and investigating measures might vary between SEK 5–15 million, remediating individual water bodies for drinking water abstraction could cost between SEK 0–15 million, and other measures to remediate the site could be estimated to SEK 20–50 million. Likewise, Avinor in Norway estimated that remediation might cost between NOK 3–30 million per airport.<sup>403</sup>

Accepting these uncertainties, the dataset provides reasonable ranges for feeding into the analysis. As before, the dataset may be improved by researching the costs of improved water treatment without specific reference to PFAS.

An issue for aggregation of results concerns the size of communities affected. Clearly, not all will be very small or very large or average. This may best be addressed through sensitivity analysis.

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<sup>401</sup> VPR News, Bennington Homes Contaminated With PFOA Connect To Clean Municipal System.

<sup>402</sup> Personal communication from Niklas Löwegren, Project leader contaminated sites, Swedish Transport Agency.

<sup>403</sup> Heggelund, A.(2017) Norwegian EPA, personal communication September 2018.

**Table A3.5: Costs for upgrading water treatment works, by activity**

Incident	Description	Population <sup>1</sup>	Cost	Cost/person
<b>Installation of filters</b>				
<b>Europe</b>				
Arlanda	Estimated cost of new treatment plant for surface water leaving site preventing contamination of Lake Malaren, used for drinking water		EUR 180,000	
Dusseldorf	Total cost of managing PFC in the contaminated drinking water	1,200,000	>EUR 13,000,000	>EUR 10
	Costs of remediation after AFFF use		>EUR 12,000,000	> EUR 8
	Total costs of remediation		>EUR 100,000,000	> EUR 80
Kallinge(SE)	Installation of new pipelines, filters	4,561	EUR 4,300,000	EUR 943
Landvetter	Operation (2016) and installation of new (2017) treatment plant for surface water		EUR 250,000	
Malmö	Establishing water treatment facility and analysing water		EUR 140,000	
	Cost of treatment facility, sampling and dike cleaning		EUR 110,000	
	Estimated cost for future remediation and monitoring in surrounding recipients		EUR 100,000	
Stadtwerke Rastatt	Raental water works		EUR 4,000,000	
Veneto	Installation of filters	120,000	EUR 2,100,000	EUR 18
<b>USA</b>				
Brunswick County	Upgrade treatment plant with activated carbon	107,000	EUR 72,000,000	EUR 673
Cape Fear	Upgrade treatment plant with granular activated carbon (Cape Fear)	360,000	EUR 33,000,000	EUR 92
Cape Fear River	Upgrading of treatment plant installation of reverse-osmosis system (Brunswick County)	107,000	EUR 72,000,000	EUR 672
Cape Fear River	Expansion of additional treatment works		EUR 28,000,000	
Hoosick Falls (NY)	"Temporary" water filtration system	3,400	EUR 7,400,000	EUR 2,200
Issaquah	Installation of water treatment systems	30,234	EUR 880,000	EUR 29
Moose Creek	Installation of granular activated carbon system	9,000	EUR 2,700,000	EUR 300
Peterson Air Force base	Contamination from AFFFs, installation of water treatment systems for known contaminated wells	60,000	EUR 3,300,000	EUR 55
Tennessee River (Decatur)	Installation of carbon filtration systems	55,000	EUR 3,700,000	EUR 67
Warrington	Installation of carbon filtration systems	23,000	EUR 12,000,000	EUR 522
<b>Temporary water filtration system</b>				
<b>USA</b>				
Hoosick Falls	Temporary water filtration system	3,400	EUR 270,000	EUR 79
<b>Annual maintenance cost of water treatment works</b>				
<b>Europe</b>				
Landvetter	Annual operating cost		EUR 40,000	
Stadtwerke Rastatt	Raental water works	25,000 (50% of Rastatt)	EUR 750,000	EUR 30

Incident	Description	Population <sup>1</sup>	Cost	Cost/person
Uppsala	Annual cost for activated charcoal filtration, 2012–2017 average		EUR 30,000	
			EUR 110,000	
Veneto	Annual operating cost	120,000	EUR 920,000	EUR 8
<b>USA</b>				
Moose Creek	Annual maintenance cost	9,000	EUR 230,000	EUR 26
Tennessee River (Decatur)	Annual maintenance cost	55,000	EUR 550,000	EUR 10
<b>Rebuilding of water treatment works</b>				
<b>Europe</b>				
Stadtwerke Rastatt	Ottersdorf water works	25,000 (50% of Rastatt)	EUR 3,900,000	
Uppsala	Reconstruction of treatment plant to deal with PFAS		EUR 260,000	
<b>Install new pipelines / connections</b>				
<b>Europe</b>				
Jersey <sup>2</sup>	Connection to water supplies	161	EUR 210,000	EUR 1,306
Jersey <sup>2</sup>	Jersey Water Mains connection costs	161	EUR 810,000	EUR 5,037
Kallinge	Installation of new pipelines, filters	4,561	EUR 6,650,000	EUR 1,458
Kallinge	Costs of providing alternative water supply via new pipe connections between 2013 and 2015 and use of a new set of carbon filters	4,561	EUR 4,300,000	EUR 943
Stadtwerke Rastatt	Water pipe between water works at Muggensturm and Rauental	49,100	EUR 900,000	EUR37 accounting for both pipelines
	Water pipe Rauental-Lochfeldstr		EUR 910,000	
Veneto	Installation of new pipelines (not carried out)	120,000	EUR 61,700,000	EUR 514
<b>Other costs</b>				
<b>Europe</b>				
Buncefield UK	Lost opportunity cost from closure of a borehole		EUR 2,600,000	
Jersey <sup>2</sup>	Site investigation	161	EUR 1,430,000	EUR 8,893
Jersey <sup>2</sup>	Remedial works to old fire ground	161	EUR 450,000	EUR 2,799
Jersey <sup>2</sup>	Fees, etc.	161	EUR 910,000	EUR 5,659
Jersey <sup>2</sup>	Capital works on Fire Training Ground	161	EUR 6,300,000	EUR 39,179
Uppsala	Risk analyses and planning of measures to safeguard Uppsala's drinking water from pollution (not only PFAS)		EUR 320,000	
	Time spent within the public water authority (2012–2017, average annual cost)		EUR 55,000	

Note: 1)Population estimates provided here are for the municipalities identified in the case studies and hence are not necessarily specific to the number of people served by water treatment works. There may be significant uncertainty in these figures, especially for the largest towns and cities included.

2)The Jersey Airport case provides a breakdown of cost elements, as shown. It is possible that these costs are included in other estimates, but without disaggregation.

## Excavation and treatment of soils

Costs relating to environmental remediation, mostly in relation to soil cleaning but including some groundwater cleaning also, are presented in Table A3.6.

**Table A3.6: Costs related to remediation of environment, after contamination from PFAS due the different sources**

Incident	Year <sup>1</sup>	Description	Cost (EUR) <sup>2</sup>
<b>European cases</b>			
Arlanda Airport (SE)	2016	Cost of testing soil clean-up	EUR 7,600
	Future	Est. cost of pilot study on remediation costs	EUR 80,000 to EUR 400,000
	Future	Estimated costs of remediation	EUR 800,000
Clean up of agricultural fields in Baden-Wurttemberg (DE)	Future	Estimate of changing contaminated soil	Up to EUR 3 billion
Bromma Airport (SE)	Future	Estimated future remediation costs	EUR 340,000
Copenhagen Airport	2016	Clean-up of site and reconstruction of fire training area	EUR 15 million
Contamination due to Dusseldorf Airport (DE) Population = 1.2 million urban area	2014	Cost of 3 wells controlling the point sources:	EUR 2 million
		Estimated total remediation cost, up to:	EUR 100 million
Contamination around Jersey Airport (UK) Population affected = 67 properties	1993	Estimated total remediation cost	7.08 million
Contamination around Nurnberg Airport (DE) <sup>3</sup>	NA	Initial budget set by Nurnberg Airport for PFAS remediation	EUR 10,000,000
Contamination around Oslo Airport (NO) <sup>4</sup>	NA	Removal of 0.6 kg PFAS from stony area	EUR 1.9 million
		Removal of 0.5 kg PFOS/year by treatment facility at fire drill sites	EUR 2.2 million
Contaminated soils, Schiphol (NL)	2008	Removal of 50,000 m <sup>3</sup> of soil, 143 kg PFOS	EUR 30–40 million
Visby Airport (SE)	Future	Estimated future remediation costs	EUR 800,000 to EUR 1.4 million
<b>US cases</b>			
Minnesota contamination due to 3M factory disposal sites (US) <sup>5</sup>	2002	Total cost for 10 years for treatment of surface/ground water, sediment and soil at 3 sites	EUR 36 million
Contamination in Warrington due to use of AFFFs (US)	2016	Estimation of total costs for environmental restoration	EUR 77.7 million

- Note:
- 1) Year might refer to year of detection, or the year costs were incurred.
  - 2) Costs in other currencies are converted to Euro, using average annual rates for the year they incurred.
  - 3) Weber R, (2016). Presentation for Science and Policy of Organohalogenes pre-Dioxin Symposium, accessed August 2018.
  - 4) Norwegian Environment Agency (2016). PFAS-forurensning i grunnen Oppsummering fra workshop 26. Rapport M-622.
  - 5) Legal settlement presented in table 19.

The costs of dealing specifically with contaminated soils reported in Table A3.6 equate to the following estimates per unit of material (for those sites where data on both costs and quantity of PFAS were available):

- Nurnberg Airport: EUR 100,000/kg mixed PFOS, PFHxS, PFBS
- Schiphol Airport: EUR 200,000–280,000/kg PFOS; EUR 600–800/m<sup>3</sup> soil
- Oslo Airport: EUR 2.1 to EUR 4.3 million/kg PFOS

Variability in the range is not surprising given that the costs for soil remediation are a function of several factors, including<sup>404</sup>:

- the quantity of PFAS that was spilled or emitted;
- the presence of other contaminants that need to be eliminated;
- the quantity of soil that has been contaminated;
- the type of soil and its qualities for retaining PFAS;
- variability in the use of sites and surrounding lands and waterbodies that will influence the desired level of remediation.

Three Norwegian airports have modelled the costs of PFAS removal (including both water and soil), using different combinations of methods and different levels of allowed remaining concentrations. For Kristiansand airport, the figures range from around NOK 29.5 to NOK 332.5 million (EUR 2.1–24 million); from NOK 6.3 to NOK 91.3 million (EUR 0.5–7.1 million) for Harstad/Narvik; and from NOK 5.7 to NOK 113.4 million (EUR 0.4–8.1 million) for Svalbard Longyearbyen. Due to the highly hypothetical nature of these cost figures, as well as the vast number of cost estimates generated by the various choices of method and target concentration, they have not been included into the cost tables presented above. For the full details of the cost of the various remediation scenarios, the reader should consult the original reports.<sup>405</sup>

## Health assessments where contamination is found

A final category of cost concerns health assessment of the population in cases where contamination above permitted levels is identified. This category of cost is accounted for here, rather than in the section on “health costs”, as associated costs relate to management of the problem rather than the health or environmental damage caused.

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<sup>404</sup> National Research Council (1997). *Innovations in Ground Water and Soil Cleanup: From Concept to Commercialization*. Chapter 6. National Academy Press. Chapter 6.

<sup>405</sup> Avinor (2018). *PFOS I Focus*, (In Norwegian) Accessed 05.09.2018.

For the Veneto Region, WHO provides the following information on a human biomonitoring study of the affected population:

- Dates: From July 2015 to April 2016
- Population: Exposed and unexposed population
- Lead organisations: National Institute of Health, the Regional Environmental Protection Agency and health care trusts in the areas identified as being most affected
- Also involved: six local health and social-care units and 14 municipalities (seven exposed and seven unexposed).
- Sampling: Serum samples were taken from 507 people aged 20–49 years. The participants were also requested to complete a questionnaire on their dietary habits, water-supply sources and consumption of local food. Because of the contribution of other factors to PFAS body burden, the biomonitoring study also included a subgroup of 120 people living and working in agricultural areas, or working with livestock.

Separately, WHO describes a Health Surveillance Plan:

- Dates: Started in December 2016.
- Population: The Plan covers five local health units and involves almost 85 000 people between the ages of 14 and 65 years
- Objectives: To identify areas of expected/possible health impact, using data on PFAS contamination of the water supply before the installation of filters.
- Screening activities: Biennial screening of the exposed population for cancer was introduced, starting with 14 year-olds in December 2016. The reason for choosing youth to begin with was that unhealthy lifestyles are not associated with this age group; thus, if high PFAS concentrations and/or significant metabolic changes were found, they could provide an insight into the correlation between exposure to PFAS and health outcomes. People with unhealthy lifestyles are informed of the risks to their health and provided with support in modifying their behaviour. Those with PFAS serum concentrations higher than the median for the Italian population, and/or showing biochemical or blood-pressure changes, are taken over by their family doctors and placed on a second-level care path for the timely diagnosis of diseases related to PFAS exposure. The Veneto Region has a regional PFAS screening information system, which manages the entire survey process, from the mailing of invitation letters to the delivery of the results and the development of the most representative health indicators. The programme is completely free of charge for the target population. An ad hoc surveillance plan is scheduled for pregnant women and those working in the manufacture of these substances.

A series of further studies were initiated:

- ecological study of pregnancy and birth outcomes;
- occupational retrospective cohort study of employees at the chemical plant;
- retrospective ecological study of the exposed population considering mortality and morbidity data in the region over the period 2007–2014;
- retrospective ecological studies on cancer incidence over the period 1997–2013.

Costs were reported above as EUR 4.3 million, equivalent to EUR 50 per person covered by the plan. WHO reports that this covers only the first two years of the Health Surveillance Plan.

Biomonitoring has been carried out around Ronneby Airport in Sweden since 2014, following identification of PFAS contamination. The following costs have been identified<sup>406</sup> (see Table A3.7):

**Table A3.7: Biomonitoring costs related to PFAS contamination around Ronneby Airport, 2014–2018.**

Year	Activity	Cost
2014–2015	Risk assessment, risk communication, advice related to bio-monitoring study	EUR 52,000
2014–2015	PFAS analyses related to bio-monitoring study	EUR 170,000
2014	Bio-monitoring study (not more specified)	EUR 420,000
2015	Bio-monitoring study (not more specified)	EUR 460,000
2016	Bio-monitoring study (not more specified)	EUR 700,000
2017	Bio-monitoring study (not more specified)	EUR 450,000
2018	Bio-monitoring study (not more specified)	EUR 340,000
2014–2018	Average annual cost of biomonitoring study (including analyses)	EUR 510,000

## Part 2: Data used in the aggregation of costs

Additional data have been used in the aggregation of costs presented in Section 4.2 of the study, as follows:

- Population (Table A3.8)
- Water consumption (Table A3.9)
- Number of wastewater treatment plant (WWTP) (Table A3.10)
- Number of plant or sources providing drinking water (Table A3.11)
- Number of airports (Table A3.12)
- Number of landfill and incineration sites (Table A3.13)

Information covers the EU28, Norway and Iceland to the extent that data are available. Data for the USA are also included for reference, given that much of the information used in this report is of US origin.

<sup>406</sup> Personal communication, K. Jakobsson, Oct 2018.

Much of the analysis is based on extrapolation against estimates of the population affected. The population data provided in Table A3.8 are taken from Eurostat and the median projections under the UN's World Population Prospects. Some countries show a significant increase in population over the coming years (e.g. Denmark, France, Iceland, Luxembourg, Norway and Sweden), others show little change (e.g. Czechia, Finland, Germany, Italy, Slovakia) and some others a fall in population (e.g. Bulgaria, Greece, Lithuania). These changes will affect any quantification based on extrapolation of existing data on population.

**Table A3.8: Population, 2015–2050. Source: Eurostat for EU28, UN median projections for others**

	2015	2020	2030	2040	2050
Austria	8,576,261	9,005,487	9,675,572	10,087,623	10,247,691
Belgium	11,208,986	11,580,268	12,264,124	12,844,259	13,273,155
Bulgaria	7,202,198	6,954,254	6,408,361	5,933,535	5,564,146
Croatia	4,225,316	4,091,559	3,954,893	3,819,863	3,674,791
Cyprus	847,008	869,041	919,997	954,320	984,402
Czechia	10,538,275	10,652,407	10,691,890	10,552,301	10,478,190
Denmark	5,659,715	5,887,449	6,298,421	6,564,333	6,685,016
Estonia	1,313,271	1,317,940	1,306,181	1,283,732	1,256,975
Finland	5,471,753	5,561,792	5,697,608	5,722,378	5,687,527
France	66,415,161	67,818,978	70,525,154	72,915,525	74,376,832
Germany	81,197,537	83,751,689	84,613,298	84,133,642	82,686,973
Greece	10,858,018	10,560,467	9,944,658	9,419,973	8,918,545
Hungary	9,855,571	9,789,630	9,665,170	9,471,313	9,287,196
Iceland	336,728	354,222	383,538	403,548	415,151
Ireland	4,628,949	4,852,123	5,146,475	5,396,380	5,693,430
Italy	60,795,612	60,718,572	60,350,475	59,982,002	58,968,137
Latvia	1,986,096	1,911,668	1,743,960	1,598,786	1,506,055
Lithuania	2,921,262	2,749,762	2,410,874	2,128,883	1,957,377
Luxembourg	562,958	628,950	754,522	860,808	938,416
Malta	429,344	452,542	488,632	505,921	513,081
Netherlands	16,900,726	17,410,756	18,393,443	19,035,643	19,235,467
Norway	5,166,493	5,403,704	5,878,930	6,268,216	6,568,489
Poland	38,005,614	37,930,818	37,213,790	35,840,028	34,372,849
Portugal	10,374,822	10,209,628	9,880,173	9,553,608	9,116,350
Romania	19,870,647	19,259,049	18,023,954	17,069,777	16,331,359
Slovakia	5,421,349	5,458,718	5,464,199	5,373,043	5,261,609
Slovenia	2,062,874	2,075,778	2,080,145	2,066,086	2,045,090
Spain	46,449,565	46,562,044	47,110,106	48,244,792	49,257,477
Switzerland	8,238,610	8,647,547	9,477,452	10,234,794	10,977,129
Sweden	9,747,355	10,293,412	11,237,236	11,994,364	12,681,084
UK	64,875,165	67,236,507	71,563,991	75,004,352	77,568,588
EU28	508,401,408	515,591,288	523,827,302	528,357,270	528,567,808
USA	325,127,634	337,983,029	362,628,830	383,165,322	400,853,042

Data on total water consumption are shown in Table A3.9 covering not only drinking water but also industrial, agricultural and commercial uses. Overall, most supplies (80%) are taken from surface water. However, data demonstrate significant variation between countries with respect to the reliance on ground water, with Denmark and Malta obtaining more than 90% of their water from groundwater, whilst Bulgaria, Romania and Finland take more than 90% of their water from surface sources.

Table A3.9: Water consumption by country and source, million m<sup>3</sup>

Million m <sup>3</sup>	Surface	Ground	Surface + ground	Total gross abstraction	% surface	% ground
Austria						
Belgium	4,480	632	5,082	4,480	88%	12%
Bulgaria	5,071	558	5,629	5,071	90%	10%
Croatia	225	428	653	225	34%	66%
Cyprus	82	150	232	82	35%	65%
Czech Republic	1,237	366	1,603	1,237	77%	23%
Denmark	9	737	746	9	1%	99%
Estonia	1,525	199	1,724	1,525	88%	12%
Finland	6,298	264	6,562	6,298	96%	4%
France	24,400	5,608	30,008	24,400	81%	19%
Germany	27,195	5,841	33,036	27,195	82%	18%
Greece	4,297	5,611	9,908	4,297	43%	57%
Hungary	4,516	492	5,051	4,516	89%	10%
Iceland			3,011			
Ireland	561	196	757	561	74%	26%
Italy						
Latvia	92	155	248	92	37%	63%
Lithuania	254	157	411	254	62%	38%
Luxembourg	20	26	45	20	43%	57%
Malta	3	43	45	3	6%	94%
Netherlands	8,465	1,016	9,482	8,465	89%	11%
Norway						
Poland	8,486	2,608	11,094	8,486	76%	24%
Portugal						
Romania	5,868	590	6,458	5,868	91%	9%
Slovakia	248	326	574	248	43%	57%
Slovenia	714	182	895	714	80%	20%
Spain	26,613	6,304	32,916	26,613	81%	19%
Sweden	2,342	348	2,690	2,342	87%	13%
Switzerland	1,000	1,005	2,005	1,000	50%	50%
United Kingdom	5,232	2,053	7,285	5,232	72%	28%

Table A3.10 shows the number of waste water treatment plants in the EU28, Iceland and Norway. Data are taken from Eurostat, and show information for the latest year for which data are available for each country. In most cases data are taken from the period 2010–2014. Older data are highlighted in red. Data for “urban” and “other” sites have been combined. The columns indicate different levels of treatment, as follows:

- T1: Primary treatment only, removing solid material
- T2: Secondary treatment, as T1 but also digesting dissolved and suspended organic materials, sometimes with disinfection to kill pathogenic bacteria
- T3: Tertiary treatment, as T2, but with a “polishing treatment” such as the use of microfiltration or synthetic membranes to further purify the water
- T3N: T3 plant with additional nitrogen removal (included in T3 total, and will include some plant also with phosphorus removal)
- T3P: T3 plant with additional phosphorus removal (included in T3 total, and will include some plant also with nitrogen removal).

**Table A3.10: Number of waste water treatment plant, category other + urban, in the EU28, Iceland and Norway**

	Total	T1	T2	T3 (total)	T3N	T3P
Austria	1,842	-	791	1,051	806	889
Belgium	1,222	412	485	325	255	251
Bulgaria	90	10	54	26	26	22
Croatia	316	195	109	12	15	1
<i>Cyprus</i>	191	-	133	88	7	6
Czechia	2,636	50	1,314	1,272	546	83
Denmark	906	177	273	456	311	446
Estonia	1,044	216	539	289	98	231
Finland	202	-	-	202	50	202
France	3,275	23	647	2,605	2,516	1,719
Germany	12,590	2,307	4,824	4,028	3,540	3,112
Greece	235	-	35	200	198	130
Hungary	739	10	278	451	338	369
<i>Iceland</i>	18	13	13	-	-	-
Ireland	1,063	217	536	310	30	215
Italy	2,717	178	510	1,876	1,345	838
Latvia	1,165	207	640	48	11	-
Lithuania	717	78	578	61	56	56
Luxembourg	251	130	98	24	2	1
Malta	4	1	3	-	3	-
Netherlands	555	4	158	393	332	311
Norway	2,240	1,565	83	592	-	-
Poland	4,296	363	2,970	963	-	-
<i>Portugal</i>	4,287	-	1,617	116	-	-
Romania	826	219	518	89	47	27
<i>Slovakia</i>	568	125	346	97	84	34
Slovenia	352	4	314	36	32	24
Spain	2,041	35	974	1,032	722	509
Sweden	1,243	-	325	918	132	960
<i>UK</i>	8,047	740	5,151	2,156	-	-
Total	55,678	7,279	24,316	19,716	11,502	10,436

Note: Italics used where latest data are from before 2010. Blank cells: no data or zero

Source: Eurostat Database, Wastewater treatment plants by treatment level [env\_ww\_plt].

Numbers of waters supply zones are shown in Table A3.11. It seems likely that the use of filters of some kind would be applied to larger sites, whilst for smaller sites it may be more economical to provide water from alternative sources. Significant variation is seen between countries. For example, in the Netherlands very few people are served by small supplies or very small supplies, whilst these serve around 50% of the population in Lithuania.

**Table A3.11: Number of water supply zones in the EU28, Iceland and Norway\***

	Total number of supply zones	Large zones (>1,000 m <sup>3</sup> /d), >5,000 persons	Small zones (10-1,000 m <sup>3</sup> /d), 50-5,000 persons	Small zones: number of consumers	Very small zones (<10m <sup>3</sup> /d), <50 persons	Very small zones: number of consumers
Austria	8,708	208	8,500	1,750,000	31,000 + 170,000	750,000
Belgium	1,113					
Bulgaria	42					
Croatia						
Cyprus	10					
Czech Republic	3,001	211	2,790	1,630,000	>500 public + 500,000 private wells	1.1 million to 3 million at weekends
Denmark			1,300		70,000	71,300
Estonia	1,220					
Finland	1,359	159	1,200	900,000	thousands <sup>5</sup>	
France	29,101		20,500	16,500,000	7,100	120,000
Germany	6,959			~500,000 people (0.7%) are not served by centralised supply <sup>3</sup>		
Greece	830	30	800	1,600,000		
Hungary	300		1,750	3,300,000	50	1,500
Iceland						
Ireland	6,900		2,275	560,000	200,000	700,000
Italy	2,000					
Latvia			497	142,095	19	2,373
Lithuania	2,044	208	1,836	500,000	300,000	900,000
Luxembourg	118	20	98	180,000	25	400
Malta	22	2	20	430,000	0	0
Netherlands	185	10	175	80,000	0	0
Norway						
Poland	26,710		10,815	11,074	8,956	
Portugal	3,356		2,068		1,000	
Romania	2,910		4,000	4,500,000	11,000	
Slovakia	2,872					
Slovenia	1,080	284	796	364,471		
Spain	5,552					
Sweden	4,300				800k private wells <sup>4</sup>	1.2 million permanent
Switzerland						
UK	2,914	1,433 <sup>2</sup>	266 <sup>1</sup>	222,488 <sup>1</sup>	24,000 <sup>1</sup>	95,000 <sup>1</sup>

Note: Cells in *Italics* calculated by subtraction of small from large zones where appropriate.

- 1) UK data for small and very small zones only available for Northern Ireland and Scotland.
- 2) Source: <https://www.gov.uk/government/publications/water-and-treated-water/water-and-treated-water>.
- 3) Source: <https://www.umweltbundesamt.de/en/topics/water/drinking-water/small-scale-drinking-water-supplies#textpart-2>
- 4) Source: Banzhaf *et al.* (2016) <https://link.springer.com/article/10.1007/s13280-016-0848-8>.
- \* Extracted from [http://nccph.netedit.info/docs/05\\_small\\_water\\_systems\\_ver\\_june2005.pdf](http://nccph.netedit.info/docs/05_small_water_systems_ver_june2005.pdf), with newer data added where available (see notes).
- 5) Finland estimates that thousands of very small supplies exists but also do not know the number of users.

Table A3.12 shows the number of sites that may use AFFFs in each country, totalling 694 airports and airbases, and 84,000 fire stations. Data are not complete, lacking small airports and possibly fire stations not intended primarily to serve the public and businesses generally, for example site emergency services at some industrial facilities, for

example oil refineries. The omission of small airports may or may not be significant to the analysis: they may be less likely to use AFFFs (e.g. fire training may be coordinated at larger airports), but are also less likely to have effective containment in place. The aviation industry is also a significant user of PFAS in hydraulic fluid <sup>407</sup>, though unlike AFFFs these are not deliberately released to the environment.

**Table A3.12: Number of public airports, military airbases and fire stations unrelated to aviation, by country**

Country	Main airports (>150k passenger/y) <sup>1</sup>	Other airports (between 15k and 150k passenger/y) <sup>2</sup>	Military (air) bases <sup>2</sup>	Total Airports + Airbases	Fire stations <sup>3</sup>
Total: EU,EFTA	318	137	239	694	84,099
Austria	6	0	5	11	5,199
Belgium	5	0	9	14	252
Bulgaria	3	1	8	12	220
Croatia	7	2	4	13	1,923
Cyprus	2	0	2	4	31
Czechia	3	2	7	12	7,561
Denmark	6	2	5	13	295
Estonia	1	1	1	3	187
Finland	9	8	7	24	988
France	44	18	38	100	6,897
Germany	25	16	25	66	33,460
Greece	20	14	13	47	275
Hungary	2	2	4	8	302
Iceland	1	0	1	2	
Ireland	5	2	1	8	220
Italy	33	2	12	47	902
Latvia	1	0	1	2	92
Lithuania	3	0		3	83
Luxembourg	1	0		1	
Malta	1	0		1	
Netherlands	5	0	12	17	1,206
Norway	1	29	12	42	597
Poland	12	0	7	19	16,805
Portugal	8	5	9	22	473
Romaia	8	4	1	13	282
Slovakia	2	2	4	8	116
Slovenia	1	0	1	2	1,359
Spain	34	4	6	44	
Sweden	19	11	5	35	1,002
Switzerland	18	1	6	25	1,319
UK	32	11	33	76	2,053

- Note: 1) Eurostat Air Transport Statistics AIRP\_TYP\_Number of Commercial Airports.  
 2) Sufficient official data has not been found, figures are based on Wikipedia for each country, and it is not clear which numbers include inactive bases.  
 3) International Association of Fire and Rescue Services (2017). World Fire Statistics.

The omission of industrial facilities could be significant. RPA/BRE (2004) reported that 0.76 tonnes of PFOS based substance was held in Fire Authority inventories, whilst 23.7 tonnes was held in emergency stores at industrial complexes. Training at these sites

<sup>407</sup> RPA and BRE Environment (2004). Perfluorooctane Sulphonate: Risk reduction strategy and analysis of advantages and drawbacks. Report no: J454/PFOS RRS.

may or may not involve use of PFAS-containing materials, containment in training areas may or may not be effective.

The number of landfill sites in each country is shown in Table A3.13, divided into landfills for hazardous waste, non-hazardous waste and inert waste. Hazardous waste covers materials that are toxic to humans, ecotoxic, carcinogenic, teratogenic, explosive etc (a complete list is provided in Annex III of the Directive on Hazardous Waste <sup>408</sup>, and material meeting any of definitions is classified as hazardous). Hazardous waste landfills would be appropriate to any material significantly contaminated with PFAS, including filters and soils. Inert waste covers any material that will not react, degrade, dissolve or burn, with criteria set for leachability limits and hence should not include anything containing PFAS. Non-hazardous waste includes any other materials and would contain materials such as carpets, shoes, etc. that are contaminated with PFAS, but at lower concentration than material sent for hazardous waste disposal.

**Table A3.13: Number of landfill sites and incinerators in the EU28, Iceland and Norway**

	Disposal - landfill (D1, D5, D12)	Disposal - landfill for HW	Disposal - landfill for non-HW	Disposal - landfill for inert waste	Disposal - incineration (D10)
Austria	189	0	153	36	1
Belgium	64	9	51	4	117
Bulgaria	187	8	176	3	
Croatia	146	0	145	1	2
Cyprus	7	1	4	2	1
Czech Republic	263	38	147	78	35
Denmark	41	5	30	6	3
Estonia	15	7	6	2	2
Finland	227	29	155	43	18
France	918	16	245	657	0
Germany	1,147	34	308	805	93
Greece	178	2	176	0	132
Hungary	111	13	92	6	13
Iceland					
Ireland	35				6
Italy	470	10	275	185	100
Latvia	13	2	11	0	4
Lithuania	14	0	11	3	2
Luxembourg	13	0	2	11	1
Malta	1	0	1	0	1
Netherlands	40	1	39	:	3
Norway	111	10	82	19	1
Poland	701	49	643	9	85
Portugal	60	2	54	4	7
Romania	129	7	122	0	20
Slovakia	118	11	92	15	11
Slovenia	37	1	26	10	4
Spain	520	35	302	183	51
Sweden	227	49	111	67	8
United Kingdom	594	26	342	226	87
<b>Total</b>	<b>6,582</b>	<b>365</b>	<b>3,801</b>	<b>2,381</b>	<b>808</b>

<sup>408</sup> Council Directive of 12 December 1991 on hazardous waste (91 / 689 /EEC).

### Part 3: Full cost estimates by country

Full cost estimates, by country, are provided in the following tables:

- the estimated costs for a basic screening programme (Table A3.14);
- the estimated costs of monitoring at contaminated sites (Table A3.15);
- estimated costs for water treatment works to reduce exposure to PFAS above possible limits (Table A3.16);
- estimated costs for soil remediation (Table A3.17);
- estimated costs for health assessment when contamination is found (Table A3.18);
- aggregated costs covering environmental screening, monitoring where contamination is found, water treatment, soil remediation and health assessment (Table A3.19).

**Table A3.14: Estimated costs for a basic screening programme**

	N facilities for best estimate	Best estimate, EUR million	Low, EUR million	High, EUR million
Austria	806	EUR 0.82	EUR 0.14	EUR 3.22
Belgium	148	EUR 0.15	EUR 0.04	EUR 0.59
Bulgaria	39	EUR 0.04	EUR 0.01	EUR 0.13
Croatia	141	EUR 0.14	EUR 0.03	EUR 0.54
Cyprus	25	EUR 0.03	EUR 0.01	EUR 0.07
Czechia	736	EUR 0.75	EUR 0.17	EUR 2.83
Denmark	78	EUR 0.08	EUR 0.02	EUR 0.31
Estonia	131	EUR 0.13	EUR 0.03	EUR 0.51
Finland	184	EUR 0.19	EUR 0.06	EUR 0.65
France	2024	EUR 2.06	EUR 0.37	EUR 8.04
Germany	2771	EUR 2.83	EUR 0.53	EUR 10.95
Greece	89	EUR 0.09	EUR 0.02	EUR 0.36
Hungary	119	EUR 0.12	EUR 0.05	EUR 0.39
Iceland	7	EUR 0.01	EUR 0.00	EUR 0.02
Ireland	413	EUR 0.42	EUR 0.07	EUR 1.66
Italy	300	EUR 0.31	EUR 0.05	EUR 1.22
Latvia	69	EUR 0.07	EUR 0.01	EUR 0.28
Lithuania	151	EUR 0.15	EUR 0.03	EUR 0.59
Luxembourg	21	EUR 0.02	EUR 0.00	EUR 0.08
Malta	19	EUR 0.02	EUR 0.01	EUR 0.04
Netherlands	111	EUR 0.11	EUR 0.03	EUR 0.42
Norway	179	EUR 0.18	EUR 0.04	EUR 0.68
Poland	2447	EUR 2.50	EUR 0.42	EUR 9.81
Portugal	422	EUR 0.43	EUR 0.08	EUR 1.67
Romania	209	EUR 0.21	EUR 0.04	EUR 0.84
Slovakia	191	EUR 0.19	EUR 0.04	EUR 0.75
Slovenia	165	EUR 0.17	EUR 0.04	EUR 0.62
Spain	539	EUR 0.55	EUR 0.11	EUR 2.11
Sweden	411	EUR 0.42	EUR 0.12	EUR 1.50
Switzerland	114	EUR 0.12	EUR 0.02	EUR 0.45
UK	714	EUR 0.73	EUR 0.15	EUR 2.80
Total	13,772	EUR 14.05	EUR 2.77	EUR 54.13

### ***Best estimate assumptions***

1. All airports and PFAS manufacturing sites are screened, assume 3 samples, using best estimate of cost/sample for monitoring.
2. 5% of other facilities are screened (fire stations, waste water treatment works, large and small supplies, hazardous and MSW landfills), assume 3 samples.
3. Best estimate of costs adopted.

### ***Low estimate assumptions***

1. All airports and PFAS manufacturing sites are screened, assume 3 samples, using low cost/sample monitoring.
2. 1% of other facilities are screened (fire stations, waste water treatment works, large and small supplies, hazardous and MSW landfills), assume 3 samples.
3. Low estimate of costs adopted.

### ***High estimate assumptions***

1. All airports and PFAS manufacturing sites are screened, assume 5 samples at each site, using high cost/sample for monitoring.
2. 10% of other facilities are screened (fire stations, waste water treatment works, large and small supplies, hazardous and MSW landfills), assume 3 samples.
3. High estimate of costs adopted.

**Table A3.15: Estimated costs of monitoring at contaminated sites**

	N facilities for best estimate	Best estimate, EUR millions, airfields and PFAS manufacturing only, EUR million	Best estimate, all source categories included, EUR million	Low, EUR millions	High, EUR millions
Austria	81	EUR 0.11	EUR 4.05	EUR 0.43	EUR 81.17
Belgium	16	EUR 0.16	EUR 0.81	EUR 0.11	EUR 16.22
Bulgaria	4	EUR 0.12	EUR 0.21	EUR 0.04	EUR 4.51
Croatia	15	EUR 0.12	EUR 0.73	EUR 0.09	EUR 14.73
Cyprus	4	EUR 0.13	EUR 0.19	EUR 0.04	EUR 3.14
Czechia	79	EUR 0.67	EUR 3.97	EUR 0.50	EUR 77.14
Denmark	8	EUR 0.03	EUR 0.40	EUR 0.05	EUR 7.98
Estonia	14	EUR 0.08	EUR 0.69	EUR 0.08	EUR 13.53
Finland	22	EUR 0.47	EUR 1.11	EUR 0.19	EUR 20.81
France	206	EUR 0.47	EUR 10.29	EUR 1.11	EUR 204.72
Germany	286	EUR 1.03	EUR 14.28	EUR 1.59	EUR 282.67
Greece	9	EUR 0.13	EUR 0.47	EUR 0.07	EUR 10.20
Hungary	16	EUR 0.47	EUR 0.81	EUR 0.15	EUR 14.35
Iceland	1	EUR 0.04	EUR 0.05	EUR 0.01	EUR 0.86
Ireland	41	EUR 0.02	EUR 2.07	EUR 0.21	EUR 41.40
Italy	29	EUR 0.05	EUR 1.45	EUR 0.16	EUR 30.77
Latvia	7	EUR 0.01	EUR 0.35	EUR 0.04	EUR 6.99
Lithuania	16	EUR 0.08	EUR 0.79	EUR 0.09	EUR 15.49
Luxembourg	2	EUR 0.01	EUR 0.11	EUR 0.01	EUR 2.14
Malta	4	EUR 0.17	EUR 0.18	EUR 0.04	EUR 2.79
Netherlands	12	EUR 0.12	EUR 0.61	EUR 0.08	EUR 11.75
Norway	19	EUR 0.20	EUR 0.97	EUR 0.13	EUR 18.87
Poland	243	EUR 0.22	EUR 12.17	EUR 1.27	EUR 246.24
Portugal	43	EUR 0.13	EUR 2.16	EUR 0.24	EUR 42.85
Romania	20	EUR 0.02	EUR 1.02	EUR 0.11	EUR 21.14
Slovakia	19	EUR 0.08	EUR 0.97	EUR 0.11	EUR 19.55
Slovenia	19	EUR 0.24	EUR 0.94	EUR 0.13	EUR 17.71
Spain	56	EUR 0.35	EUR 2.79	EUR 0.34	EUR 55.89
Sweden	48	EUR 0.76	EUR 2.40	EUR 0.36	EUR 44.97
Switzerland	11	EUR 0.02	EUR 0.57	EUR 0.06	EUR 11.52
UK	74	EUR 0.45	EUR 3.70	EUR 0.45	EUR 74.10
<b>Total</b>	<b>1,426</b>	<b>EUR 6.96</b>	<b>EUR 71.28</b>	<b>EUR 8.30</b>	<b>EUR 1,416</b>

#### **Best estimate assumptions**

1. Assumed 20% of airports and PFAS manufacturing sites require monitoring programme, using best estimate cost/case for monitoring.
2. 0.5% of other facilities require monitoring.
3. Best estimate of costs adopted.

#### **Low estimate assumptions**

1. Assumed 10% of airports and PFAS manufacturing sites require monitoring programme, using low estimate cost/case for monitoring.
2. 0.1% of other facilities require monitoring.
3. Low estimate of costs adopted.

### High estimate assumptions

1. Assumed 30% of airports and PFAS manufacturing sites require monitoring programme, using high estimate cost/case for monitoring.
2. 1% of other facilities require monitoring.
3. High estimate of costs adopted.

**Table A3.16: Estimated costs for water treatment works to reduce exposure to PFAS above possible limits**

	Population affected, best estimate	Best estimate, EUR millions	Low, EUR millions	High, EUR millions
Austria	257,288	EUR 146	EUR 11	EUR 415
Belgium	336,270	EUR 191	EUR 15	EUR 542
Bulgaria	216,066	EUR 123	EUR 9	EUR 348
Croatia	126,759	EUR 72	EUR 6	EUR 204
Cyprus	25,410	EUR 14	EUR 1	EUR 41
Czechia	316,148	EUR 180	EUR 14	EUR 510
Denmark	169,791	EUR 97	EUR 7	EUR 274
Estonia	39,398	EUR 22	EUR 2	EUR 64
Finland	164,153	EUR 93	EUR 7	EUR 265
France	1,992,455	EUR 1,133	EUR 87	EUR 3,213
Germany	2,435,926	EUR 1,385	EUR 106	EUR 3,928
Greece	325,741	EUR 185	EUR 14	EUR 525
Hungary	295,667	EUR 168	EUR 13	EUR 477
Iceland	10,102	EUR 6	EUR 0	EUR 16
Ireland	138,868	EUR 79	EUR 6	EUR 224
Italy	1,823,868	EUR 1,037	EUR 80	EUR 2,941
Latvia	59,583	EUR 34	EUR 3	EUR 96
Lithuania	87,638	EUR 50	EUR 4	EUR 141
Luxembourg	16,889	EUR 10	EUR 1	EUR 27
Malta	12,880	EUR 7	EUR 1	EUR 21
Netherlands	507,022	EUR 288	EUR 22	EUR 818
Norway	154,995	EUR 88	EUR 7	EUR 250
Poland	1,140,168	EUR 648	EUR 50	EUR 1,838
Portugal	311,245	EUR 177	EUR 14	EUR 502
Romania	596,119	EUR 339	EUR 26	EUR 961
Slovakia	162,640	EUR 92	EUR 7	EUR 262
Slovenia	61,886	EUR 35	EUR 3	EUR 100
Spain	1,393,487	EUR 792	EUR 61	EUR 2,247
Sweden	292,421	EUR 166	EUR 13	EUR 472
Switzerland	247,158	EUR 141	EUR 11	EUR 399
UK	1,946,255	EUR 1,107	EUR 85	EUR 3,138
<b>Total</b>	<b>15,664,297</b>	<b>EUR 8,906</b>	<b>EUR 684</b>	<b>EUR 25,258</b>

### Best estimate assumptions

1. Assumed 3% of the population are exposed to excess levels of PFAS via drinking water.
2. Assume 20 year maintenance programme for treatment works, based on best estimate.
3. Assume best estimate cost per case for remediation.
4. Assume 4% discount rate on future maintenance costs.

#### *Low estimate assumptions*

1. Assumed 1% of the population are exposed to excess levels of PFAS via drinking water.
2. Assume 20 year maintenance programme for treatment works, based on best estimate.
3. Assume low estimate cost per case for remediation.
4. Assume 4% discount rate on future maintenance costs.

#### *High estimate assumptions*

1. Assumed 5% of the population are exposed to excess levels of PFAS via drinking water.
2. Assume 20 year maintenance programme for treatment works, based on best estimate.
3. Assume high estimate cost per case for remediation.
4. Assume 4% discount rate on future maintenance costs.

**Table A3.17: Estimated costs for soil remediation**

	N facilities for best estimate	Best estimate, EUR millions, airfields and PFAS manufacturing only, EUR millions	Best estimate, all source categories included, EUR millions	Low, EUR millions	High, EUR millions
Austria	81	11.0	EUR 405	EUR 5.1	EUR 8,117
Belgium	16	16.0	EUR 81	EUR 1.3	EUR 1,622
Bulgaria	4	12.0	EUR 21	EUR 0.5	EUR 451
Croatia	15	12.0	EUR 73	EUR 1.1	EUR 1,473
Cyprus	4	13.0	EUR 19	EUR 0.5	EUR 314
Czechia	79	67.0	EUR 397	EUR 6.0	EUR 7,714
Denmark	8	3.0	EUR 40	EUR 0.5	EUR 798
Estonia	14	8.0	EUR 69	EUR 1.0	EUR 1,353
Finland	22	47.0	EUR 111	EUR 2.2	EUR 2,081
France	206	47.0	EUR 1,029	EUR 13.3	EUR 20,472
Germany	286	103.0	EUR 1,428	EUR 19.1	EUR 28,267
Greece	9	13.0	EUR 47	EUR 0.9	EUR 1,020
Hungary	16	47.0	EUR 81	EUR 1.8	EUR 1,435
Iceland	1	4.0	EUR 5	EUR 0.1	EUR 86
Ireland	41	2.0	EUR 207	EUR 2.5	EUR 4,140
Italy	29	5.0	EUR 145	EUR 2.0	EUR 3,077
Latvia	7	1.0	EUR 35	EUR 0.4	EUR 699
Lithuania	16	8.0	EUR 79	EUR 1.1	EUR 1,549
Luxembourg	2	1.0	EUR 11	EUR 0.1	EUR 214
Malta	4	17.0	EUR 18	EUR 0.5	EUR 279
Netherlands	12	12.0	EUR 61	EUR 1.0	EUR 1,175
Norway	19	20.0	EUR 97	EUR 1.6	EUR 1,887
Poland	243	22.0	EUR 1,217	EUR 15.2	EUR 24,624
Portugal	43	13.0	EUR 216	EUR 2.8	EUR 4,285
Romania	20	2.0	EUR 102	EUR 1.3	EUR 2,114
Slovakia	19	8.0	EUR 97	EUR 1.3	EUR 1,955
Slovenia	19	24.0	EUR 94	EUR 1.6	EUR 1,771
Spain	56	35.0	EUR 279	EUR 4.1	EUR 5,589
Sweden	48	76.0	EUR 240	EUR 4.3	EUR 4,497
Switzerland	11	2.0	EUR 57	EUR 0.7	EUR 1,152
UK	74	45.0	EUR 370	EUR 5.4	EUR 7,410
<i>Total</i>	<i>1,426</i>	<i>696</i>	<i>EUR 7,128</i>	<i>EUR 100</i>	<i>EUR 141,613</i>

**Best estimate assumptions**

1. Assumed 20% of airports and PFAS manufacturing sites require remediation.
2. 0.5% of other facilities require remediation.
3. Best estimate of costs adopted.

**Low estimate assumptions**

1. Assumed 10% of airports and PFAS manufacturing sites require remediation.
2. 0.1% of other facilities require remediation.
3. Low estimate of costs adopted.

### High estimate assumptions

4. Assumed 30% of airports and PFAS manufacturing sites require remediation.
5. 1% of other facilities require remediation.
6. High estimate of costs adopted.

**Table A3.18: Estimated costs for health assessment when contamination is found**

	Population affected, best estimate	Best estimate, EUR millions	Low, EUR millions	High, EUR millions
Austria	257,288	EUR 13	EUR 0.43	EUR 41
Belgium	336,270	EUR 17	EUR 0.56	EUR 53
Bulgaria	216,066	EUR 11	EUR 0.36	EUR 34
Croatia	126,759	EUR 6	EUR 0.21	EUR 20
Cyprus	25,410	EUR 1	EUR 0.04	EUR 4
Czechia	316,148	EUR 16	EUR 0.53	EUR 50
Denmark	169,791	EUR 8	EUR 0.28	EUR 27
Estonia	39,398	EUR 2	EUR 0.07	EUR 6
Finland	164,153	EUR 8	EUR 0.27	EUR 26
France	1,992,455	EUR 100	EUR 3.32	EUR 315
Germany	2,435,926	EUR 122	EUR 4.06	EUR 386
Greece	325,741	EUR 16	EUR 0.54	EUR 52
Hungary	295,667	EUR 15	EUR 0.49	EUR 47
Iceland	10,102	EUR 1	EUR 0.02	EUR 2
Ireland	138,868	EUR 7	EUR 0.23	EUR 22
Italy	1,823,868	EUR 91	EUR 3.04	EUR 289
Latvia	59,583	EUR 3	EUR 0.10	EUR 9
Lithuania	87,638	EUR 4	EUR 0.15	EUR 14
Luxembourg	16,889	EUR 1	EUR 0.03	EUR 3
Malta	12,880	EUR 1	EUR 0.02	EUR 2
Netherlands	507,022	EUR 25	EUR 0.85	EUR 80
Norway	154,995	EUR 8	EUR 0.26	EUR 25
Poland	1,140,168	EUR 57	EUR 1.90	EUR 181
Portugal	311,245	EUR 16	EUR 0.52	EUR 49
Romania	596,119	EUR 30	EUR 0.99	EUR 94
Slovakia	162,640	EUR 8	EUR 0.27	EUR 26
Slovenia	61,886	EUR 3	EUR 0.10	EUR 10
Spain	1,393,487	EUR 70	EUR 2.32	EUR 221
Sweden	292,421	EUR 15	EUR 0.49	EUR 46
Switzerland	247,158	EUR 12	EUR 0.41	EUR 39
UK	1,946,255	EUR 97	EUR 3.24	EUR 308
<i>Total</i>	<i>15,664,297</i>	<i>EUR 783</i>	<i>EUR 26</i>	<i>EUR 2,480</i>

**Table A3.19: Aggregated costs covering environmental screening, monitoring where contamination is found, water treatment, soil remediation and health assessment.**

	Best estimate, EUR millions	Low, EUR millions	High, EUR millions
Austria	EUR 569	EUR 17	EUR 8,656
Belgium	EUR 290	EUR 16	EUR 2,234
Bulgaria	EUR 155	EUR 10	EUR 838
Croatia	EUR 152	EUR 7	EUR 1,712
Cyprus	EUR 35	EUR 2	EUR 362
Czechia	EUR 597	EUR 21	
Denmark	EUR 145	EUR 8	EUR 1,106
Estonia	EUR 94	EUR 3	EUR 1,437
Finland	EUR 214	EUR 10	EUR 2,393
France	EUR 2,274	EUR 105	EUR 24,213
Germany	EUR 2,952	EUR 132	EUR 32,874
Greece	EUR 249	EUR 16	EUR 1,607
Hungary	EUR 264	EUR 15	EUR 1,973
Iceland	EUR 12	EUR 1	EUR 105
Ireland	EUR 295	EUR 9	EUR 4,429
Italy	EUR 1,275	EUR 85	EUR 6,339
Latvia	EUR 72	EUR 3	EUR 811
Lithuania	EUR 134	EUR 5	EUR 1,720
Luxembourg	EUR 21	EUR 1	EUR 246
Malta	EUR 26	EUR 1	EUR 305
Netherlands	EUR 375	EUR 24	EUR 2,085
Norway	EUR 194	EUR 9	EUR 2,181
Poland	EUR 1,937	EUR 69	EUR 26,899
Portugal	EUR 411	EUR 17	EUR 4,880
Romania	EUR 472	EUR 28	EUR 3,191
Slovakia	EUR 199	EUR 9	EUR 2,263
Slovenia	EUR 133	EUR 5	EUR 1,899
Spain	EUR 1,144	EUR 68	EUR 8,115
Sweden	EUR 423	EUR 18	EUR 5,061
Switzerland	EUR 210	EUR 12	EUR 1,601
UK	EUR 1,579	EUR 94	EUR 10,933
<i>Total</i>	<i>EUR 16,902</i>	<i>EUR 821</i>	<i>EUR 170,821</i>

#### **Best estimate assumptions**

1. Assumed 3% of the population are exposed to excess levels of PFAS via drinking water.
2. Assume best estimate cost per case for remediation.

#### **Low estimate assumptions**

1. Assumed 1% of the population are exposed to excess levels of PFAS via drinking water.
2. Assume low estimate cost per case for remediation.

### *High estimate assumptions*

1. Assumed 5% of the population are exposed to excess levels of PFAS via drinking water.
2. Assume high estimate cost per case for remediation.



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### THE COST OF INACTION

PFAS (per and polyfluoroalkylsubstances) are known to be extremely difficult to degrade in the environment and to be bioaccumulative and toxic. Exposure to PFAS is suspected to increase the risk of adverse health effects, such as impacts on the thyroid gland, the liver, fat metabolism and the immune system. This study estimates the socioeconomic costs that may result from impacts on human health and the environment from the use of PFAS. Better awareness of the costs and problems associated with PFAS exposure will assist decision-makers and the general public to make more efficient and timely risk management decisions. Findings indicate that the costs are substantial, with annual health-related costs estimated to 2.8 – 4.6 billion EUR for the Nordic countries and 52 – 84 billion EUR for all EEA countries. Overall non-health costs are estimated at 46 million – 11 billion EUR for the Nordic countries.

Upon request the excel spreadsheets used for the monetarisation and valuation in this report can also be provided along with a guidance on how to use the estimation of costs for value transfer. Please contact any of the consultants or members of the steering group from the Swedish Chemicals Agency or the Danish Environmental Protection Agency if you are interested in receiving these excel spreadsheets.



# Attachment 22

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# Mortality Risk Valuation

This page contains information on Frequently Asked Questions on Mortality Risk Valuation and EPA practices concerning the use and measurement of the "Value of a Statistical Life" as it is applied in EPA economic analyses.

- What does it mean to place a value on life?
- Why do Agencies attempt to value risk reductions in dollars?
- What is Benefit-Cost Analysis?
- What is Benefit-Cost Analysis used for?
- What is the "Value of a Statistical Life"?
- What value of statistical life does EPA use?
- What other values has EPA used in the past?
- What is the current process for updating the Agency's estimates?
- Why is EPA proposing to change the terminology it uses when valuing changes in mortality risk?
- How does the "Value of Mortality Risk" Differ from the Value of a Statistical Life?
- How will EPA Estimate the Value of Mortality Risk (VMR)?
- Is EPA proposing a numeric value for VMR?
- What is a Cancer Differential?
- What are Altruistic Preferences?
- When will revised Guidance on Mortality Risk Valuation be available?
- Related References



## **What does it mean to place a value on life?**

The EPA does not place a dollar value on individual lives. Rather, when conducting a benefit-cost analysis of new environmental policies, the Agency uses estimates of how much people are willing to pay for small reductions in their risks of dying from adverse health conditions that may be caused by environmental pollution.

In the scientific literature, these estimates of willingness to pay for small reductions in mortality risks are often referred to as the "value of a statistical life." This is because these values are typically reported in units that match the aggregate dollar amount that a large group of people would be willing to pay for a reduction in their individual risks of dying in a year, such that we would expect one fewer death among the group during that year on average. This is best explained by way of an example. Suppose each person in a sample of 100,000 people were asked how much he or she would be willing to pay for a reduction in their individual risk of dying of 1 in 100,000, or 0.001%, over the next year. Since this reduction in risk would mean that we would expect one fewer death among the sample of 100,000 people over the next year on average, this is sometimes described as "one statistical life saved." Now suppose that the average response to this hypothetical question was \$100. Then the total dollar amount that the group would be willing to pay to save one statistical life in a year would be \$100 per person  $\times$  100,000 people, or \$10 million. This is what is meant by the "value of a statistical life." Importantly, this is not an estimate of how much money any single individual or group would be willing to pay to prevent the certain death of any particular person.

## **Why do Agencies attempt to value risk reductions in dollars?**

Agencies use estimates of values of risk reductions when conducting a benefit-cost analysis of a new policy or regulation that may affect public health. For example, many of the air and water pollution control regulations that are implemented by the EPA will reduce the risks of certain types of cancers, respiratory illnesses, and other diseases among large portions of the general public. Benefit-cost analysis compares the total willingness to pay for the health risk reductions from these policies to the additional costs that people will bear if the policies are adopted. These costs may come in the form of increased taxes, or, more commonly, increased prices of goods and services whose production, use, or disposal contributes to environmental pollution. The results of a benefit-cost analysis are presented to policy-makers and the public to help inform their judgments regarding whether or not a proposed policy should be adopted.

Only one federal environmental statute, the Safe Drinking Water Act, explicitly calls for the kind of formal benefit-cost analysis describe here. Most environmental laws do not require benefit-cost analysis, and some prohibit it (e.g., the air quality standards provisions of the Clean Air Act). Nevertheless, Presidential Executive Orders have required or encouraged the use of benefit-cost analysis in policy evaluation since the early 1980's. For "major" regulations—those expected to have an impact on the economy of \$100 million or more—federal agencies are required by Executive Order 12866 to conduct a formal benefit-cost analysis as a way of informing both policy makers and the public.

## **What is Benefit-Cost Analysis?**

Benefit-cost analysis is an analytical tool used to evaluate public policy options. For environmental policies, benefits are determined by what individuals would be willing to pay for risk reductions or for other improvements from pollution prevention. Costs are determined by the dollar value of the resources directed to pollution reduction. If the total benefits exceed the total costs, then the policy is said to "pass a benefit-cost test."

Of course in most cases where the total benefits exceed total costs, it will *not* be true that the benefits exceed the costs for each and every person affected by the policy; rather, some individuals will gain and others will lose. However, if the total benefits are greater than the costs, then it is *in principle* possible for those who gain to compensate those who lose so that everyone could be better off with the policy. This is what it means for a policy to pass a benefit-cost test.

The benefit-cost test alone is not the only relevant criterion for evaluating public policies since it omits important aspects of the policy decision. In particular, the benefit-cost criterion does not consider the distribution of benefits and costs among the affected individuals. These distributional effects often will be important to policy-makers and the general, so benefit-cost analysis typically will need to be supplemented by other information.

## **What is Benefit-Cost Analysis used for?**

The primary purpose of benefit cost analysis is to provide policy makers and others with detailed information on a wide variety of consequences of environmental policies.

Benefit-cost analysis is only one of many inputs into policy evaluation. Other factors include environmental justice considerations; ethical concerns; enforceability; legal consistency; and technological and institutional feasibility.

## What is the "Value of a Statistical Life"?

See the previous section: "What does it mean to place a value on life?"

## What value of statistical life does EPA use?

EPA recommends that the central estimate of \$7.4 million (\$2006), updated to the year of the analysis, be used in all benefits analyses that seek to quantify mortality risk reduction benefits regardless of the age, income, or other population characteristics of the affected population until revised guidance becomes available (see "What is the current process for updating the Agency's estimates?" below). This approach was vetted and endorsed by the Agency when the 2000 *Guidelines for Preparing Economic Analyses* <<https://epa.gov/environmental-economics/guidelines-preparing-economic-analyses>> were drafted. Although \$7.4 million (\$2006) remains EPA's default guidance for valuing mortality risk changes, the Agency has considered and presented others (see "What Values Has EPA Used in the Past?" below.)

## What other values has EPA used in the past?

Few economic analyses prepared by EPA calculated monetary benefits until the mid-1980s. One of the earliest major EPA regulations that developed more detailed economic estimates of the benefits of proposed regulatory standards was the National Ambient Air Quality Standards for particulate matter (USEPA 1984). This analysis drew on a review of six wage-risk studies published during 1976-1981 with a central estimate of \$4.6 million (2001\$). Around this same time EPA issued its first economic guidance and reported a range of VSL estimates for use in policy analysis of \$0.7 to \$12.9 million (2001\$) (USEPA 1983). The next major review of mortality risk valuation came in the mid-1990s when EPA reported to Congress on the economic benefits and costs of the Clean Air Act (USEPA 1997). This report based its VSL findings on 26 studies, 21 from the wage-risk literature and five from stated preference studies. This study forms the basis of EPA's existing mortality risk valuation guidance discussed above.

Beginning in 2004 EPA's Office of Air and Radiation (OAR) used an estimate of \$5.5 million (1999 dollars; \$6.6 million in 2006 dollars) for the analysis of air regulations. This estimate was derived from the range of values estimated in three meta-analyses of VSL conducted after EPA's *Guidelines* were published in 2000 (Mrozek and Taylor (2000), Viscusi and Aldy (2003), and later, Kochi, et al. (2006).) However, the Agency neither changed its official

guidance on the use of VSL in rule-makings nor subjected the interim estimate to a scientific peer-review process through the Science Advisory Board (SAB) or other peer-review group.

While the Agency is updating its guidance by incorporating the most up-to-date literature and recent recommendations from the SAB-EEAC, it has determined that a single, peer-reviewed estimate applied consistently best reflects the SAB-EEAC advice until updated guidance is available. Therefore, EPA has decided to return to the value established in the 2000 *Guidelines* for all its actions until a revised estimate can be fully vetted within the Agency and by EPA's Science Advisory Board.

## **What is the current process for updating the Agency's estimates?**

EPA is committed to using the best available science in its analyses and is in the process of revisiting its guidance on valuing mortality risk reductions.

- EPA has engaged the Science Advisory Board Environmental Economics Advisory Committee (SAB-EEAC) on several issues related to mortality risk valuation, including the use of meta-analysis – a statistical technique used to combine results from individual studies addressing similar problems.
- Following advice of the SAB-EEAC, EPA formed an expert panel to explore issues of meta-analysis (see USEPA 2006).
- In addition, EPA commissioned reports on the various approaches used in the literature to estimate the value of mortality risk reductions (Alberini 2004, Black *et al.* 2003, and Blomquist 2004).

EPA took this information into account and prepared a 2010 white paper: *Valuing Mortality Risk Reductions in Environmental Policy* <<https://epa.gov/environmental-economics/valuing-mortality-risk-reductions-environmental-policy-white-paper-2010>> (PDF, 1795.3K, About PDF <<https://epa.gov/epahome/pdf.html>>) featuring EPA's latest review of important issues surrounding how to value the reductions in risk to human health from environmental regulations and other Agency decisions. EPA submitted the whitepaper to its Science Advisory Board for feedback and recommendations on several issues including:

- replacing the often misunderstood term "value of statistical life" with the more accurate term "value of mortality risk reduction;"

- accounting for potential differences in people's willingness to pay for cancer mortality risk reductions relative to mortality risks from workplace or other accidental deaths when estimating the benefits of actions that are expected to reduce cancer-causing pollutants;
- accounting for possible differences in people's willingness to pay for risk reductions that will be experienced by others due to altruistic preferences in benefit-cost estimation; and
- synthesizing the body of evidence of people's willingness-to-pay for reducing mortality risks to inform benefit-cost analysis.

In 2011, the SAB completed its  Review of “Valuing Mortality Risk Reductions for Environmental Policy: A White Paper.” (pdf) <[https://epa.gov/system/files/documents/2022-03/86189901\\_0.pdf](https://epa.gov/system/files/documents/2022-03/86189901_0.pdf)> and the agency revised its estimates used in benefit-cost analysis in light of these the recommendations.

## **Why is EPA proposing to change the terminology it uses when valuing changes in mortality risk?**

The Agency believes that its benefit-cost analyses would be more transparent and comprehensible if the term "value of statistical life" were replaced with an alternative term that more accurately describes the health risk changes that are being analyzed. The term "value of statistical life" can give the misleading impression that a "price" is being placed on individual lives--as a mugger who says, "Your money or your life!?" In reality, EPA regulations typically lead to small reductions in mortality risks (ranging up to 1 in 1,000 per year) for large numbers of people. A benefit-cost analysis attempts to estimate the total sum of money that a large number of people would be willing to pay to reduce their mortality risks by amounts in this general range. The term "value of mortality risk reduction" conveys this idea more clearly and should reduce the confusion that sometimes arises when discussing the "value of statistical lives." It is important to understand that by adopting new terminology the Agency is not changing the economic theory that underlies these valuations. Furthermore, no matter which term is applied, the same underlying data would be used to estimate the value, and these values would lead to the same aggregate benefits if applied to the same policy proposal.

## **How does the “Value of Mortality Risk” Differ from the Value of a Statistical Life?**

The Value of Mortality Risk (VMR) and the Value of Statistical Life (VSL) are indeed related. The underlying theoretical concept is the same, and the estimated values for either metric would be based on the same published literature. The difference lies in the choice of units used to aggregate and report the risk changes. The VSL is typically reported in units of dollars per statistical death per year. The VMR would be reported in units such as dollars per micro-risk per person per year, where a “micro-risk” represents a one in a million chance of dying. EPA is proposing using VMR because it should help to reduce the misunderstandings that are sometimes caused by the VSL terminology.

## **How will EPA Estimate the Value of Mortality Risk (VMR)?**

For decades economists have been studying how people make tradeoffs between their own income and risks to their health and safety. These tradeoffs can reveal how people value, in dollar terms, small changes in risk. For example, purchasing automobile safety options reveals information on what people are willing to pay to reduce their risk of dying in a car accident. Purchasing smoke detectors reveals information on what people are willing to pay to reduce their risk of dying in a fire. EPA will review all of the peer-reviewed scientific studies of these income and health risk trade-offs and will attempt to summarize the results in a single best central estimate or range of estimates to use in benefit-cost analyses.

## **Is EPA proposing a numeric value for VMR?**

No, EPA is not proposing a numeric value for VMR at this time. The 2010 White Paper reviewed by the SAB-EEAC proposed a methodology for both incorporating the latest scientific evidence on how people value small reductions in their risk of dying and combining the estimates in the over 80 studies in the literature. EPA has identified a set of criteria for selecting studies from the literature and outlined a method for identifying appropriate estimates from those studies. The White Paper highlights a number of statistical issues that are associated with combining estimates from the studies and is seeking SAB feedback on how best to address these issues. EPA has proposed several options for identifying the best estimate or set of estimates for a VMR, but does not propose a value in this White Paper.

## **What is a Cancer Differential?**

A cancer differential is the additional amount that people are willing to pay to reduce cancer risks relative to accidental or other categories of mortality risks. In part, this may reflect the extended period of illness that accompanies life-threatening cancer, but it may also include intangible factors such as the additional feeling of dread associated with cancer. If people value different types of risk differently, then benefits analysis for different types of policies would ideally reflect these preferences. As described in the *White Paper on Valuing Mortality Risk Reductions in Environmental Policy*, EPA believes there is now sufficient scientific evidence for including a cancer differential in economic analysis of policies that reduce exposure to cancer-causing pollutants. This issue is one of the subjects for EPA's upcoming consultation with the Environmental Economics Advisory Committee of the Science Advisory Board.

## **What are Altruistic Preferences?**

Altruism is the concern for others. We know from studies that individuals are often willing to pay more when there are reductions in risks to themselves as well as others. That is, many studies show that individuals express altruism when asked how much they would be willing to pay to reduce risks to themselves as well as other people. Since most environmental policy addresses public risks that we all face in common, then it may be important to capture these altruistic preferences in our benefit-cost analysis. This issue is one of the subjects for EPA's upcoming consultation with the Environmental Economics Advisory Committee of the Science Advisory Board.

## **When will revised Guidance on Mortality Risk Valuation be available?**

Producing Agency guidance on mortality risk valuation is a multi-step process and depends on the recommendations received from the Science Advisory Board. Clear guidance based on the best available scientific information that can be consistently applied across the Agency is the goal. Based on the 2011 SAB review, new guidance is being developed.

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# Attachment 23



# report

report no. 8/16

## Environmental fate and effects of poly- and perfluoroalkyl substances (PFAS)





# Environmental fate and effects of poly- and perfluoroalkyl substances (PFAS)

Prepared for the Concaawe Soil and Groundwater Taskforce (STF/33):

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## ABSTRACT

Aqueous Film Forming Foam (AFFF) and Fluoroprotein (FP)/ Film Forming Fluoroprotein Foam (FFFP) foam have been used since the 1960s and 1970s, respectively, for the suppression of class B (flammable liquid) fires at airports, refineries and other major petroleum facilities. In recent years, however, the use of these has been challenged due to concern that certain poly and perfluoroalkyl substances (PFAS) used in their formulation exhibit PBT characteristics (Persistent, Bioaccumulative and Toxic). While alternative PFAS-free foams are now commercially available, concerns have been raised that these may be less effective for fighting large-scale flammable liquid fires and that other issues such as shelf life, compatibility with conventional application equipment and suitability of different materials for storage have not been fully evaluated.

It is important that users of class B fire- fighting foams understand and manage both environmental and fire safety aspects of foam use. An assessment of site foam stocks is recommended to ensure that any legacy stocks containing >0.001wt% PFOS (banned for use in the EU since June 2011) are set aside for safe disposal by high temperature incineration. A similar assessment should be completed for foam stocks that may be brought to site from third parties in the event of an emergency. At locations where fluorochemical- based foams have been used for fire- fighting or fire-fighting training, users should consider how to manage the potential issues.

Fire- fighting foams designated “C6” by manufacturers are formulated using PFAS that cannot degrade to form PFOS or PFOA and so these seem of less concern from an environmental standpoint. It should be noted, however, that given the range of compounds present there is still uncertainty about their properties. In addition, low environmental concentration limits have been set for short chain PFAS (i.e. <C6 PFSA; <C7 PFCA) in many EU countries due to their persistence. Where possible, therefore, water containing PFAS- based fire- fighting foam residues should be captured for treatment and not discharged to the environment.

This report, which is a review of published literature on the environmental fate and effects of PFAS, has been produced to help Concaawe members understand and manage environmental and human health risks associated with current and legacy formulations of PFAS- based class B fire- fighting foams. It describes the main types of PFAS, their use, fate and transport properties, toxicity data, regulation, and gives an overview of chemical analysis and remedial techniques.

The report has been reviewed by members of the Concaawe Special Taskforce on Soil and Groundwater, and the Emerging Contaminant Working Group of the Network for Industrially Contaminated Land in Europe (NICOLE).

**KEYWORDS**

PFOS, PFOA, PFAS, perfluorooctane sulfonate, perfluorooctanoic acid, poly- and perfluoroalkyl substances, toxicity, bioaccumulation, environmental quality standard, environmental fate, regulation, chemical analyses, remediation

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## SUMMARY

### Background

Poly and perfluorinated substances (PFAS) are used in a wide range of industrial applications and commercial products due to their unique surface tension/levelling properties. These include textile stain guards, grease-proof paper, fluoropolymer manufacture, coatings, and aqueous film-forming foams. Relevant to the refining industry is the use of PFAS in class B (flammable liquid) fire-fighting foams, including Aqueous Film Forming Foam (AFFF), Fluoroprotein (FP) and Film Forming Fluoroprotein Foam (FFFP). PFAS are used in fire foam products because of their ability to wet the surface of liquid hydrocarbon, resulting in a much higher foam spreading rate than is possible using only hydrocarbon-based surfactants. At sites where fire-fighting foams have been used, PFAS source zones may include fire-fighting training areas, areas where large fires have occurred historically, foam storage and dispensing locations and locations where AFFF has been repeatedly used for flammable vapour suppression during 'hot work'.

### Regulation

Concern around the environmental effects of PFAS use began in the late 1990s when it was realised that, due to their resistance to biodegradation, perfluorooctane sulfonic acid (PFOS) and perfluorooctanoic acid (PFOA), two of the most abundant PFAS, were ubiquitous in various biological and environmental matrices, and could biomagnify. Simultaneously, it became clear that they could have effects on human health and the (aquatic) environment. The degree of biomagnification is proportional to perfluorocarbon chain length and so regulation to restrict the manufacture and use of PFAS substances has focussed on PFAS containing more than 6 fully fluorinated carbon atoms

In 2009, PFOS was added to Annex B of the Stockholm Convention on Persistent Organic Pollutants (POPs), meaning that measures must be taken to restrict its production and use. With global restrictions now in place for PFOS, further regulation is proposed in Europe and elsewhere to restrict the manufacture and use of any PFAS substance that contains a C7 or C8 perfluorocarbon moiety in its molecular structure. The use of legacy firefighting- foam products containing >0.001wt% PFOS has been banned in the EU since 27th June 2011.

In 2013, PFOS and its derivatives were included in the Directive on "Environmental Quality Standards" (EQSD). The EU annual average environmental quality standard (AA-EQS) for PFOS in surface freshwater is set at a very low criterion of 0,00065 µg/l, based on the potential for secondary poisoning in humans due to fish consumption. The AA-EQS of 0,00065 µg/l is derived from starting points that are considered by many as very conservative, and is lower than background levels typically recorded in surface waters. It is also lower than the limit of quantitation (LOQ) typically achieved by commercial laboratories. The date set for EU-wide compliance with the AA-EQS is 22<sup>nd</sup> December 2027, with member states required to submit to the Commission a supplementary monitoring programme and a preliminary programme of measures to achieve compliance by 22<sup>nd</sup> December 2018.

Provisional drinking water standards developed by EU member states are generally around 0.1 to 0.5 µg/l PFOS, which is 3 orders of magnitude higher than the AA-EQS. In those countries where target values for groundwater have been derived these are within a similar range. Environmental standards may also encompass a range of other both short and long chain poly- and perfluorinated compounds with limits set both for individual substances and also the total PFAS concentration.

**Toxicity**

Available data on PFAS toxicity is dominated by PFOS, PFOA and also perfluorohexane sulfonate (PFHxS) due to the widespread detection of these compounds in humans and the environment, and concern that these could biomagnify to a level whereby humans consuming fish may be adversely affected. Much less data is available on the toxicology of other PFAS, and this is often inconsistent and fragmentary. For the less investigated polyfluorinated chemicals, toxicology is often estimated based on structure- activity relationships, or structural homologues.

Human exposure to PFAS is mainly by ingestion of contaminated food or water. These compounds are not metabolised, bind to proteins (not to fats) and are mainly detected in blood, liver and kidneys. Elimination of PFOS, PFHxS and PFOA from the human body takes some years, whereas elimination of shorter chain PFAS is in the range of days. The half-life of PFOS and PFOA in rodents is in the range of months which differs significantly from humans and can cause extrapolation issues in tests. There is significant data available on the impact of (sub)chronic PFOS and PFOA exposure on reproductive and/or developmental and other types of effects in both humans and animals. However, the results from epidemiological studies are not always consistent.

Animal studies show mainly effects from PFOS and PFOA on the liver, the gastrointestinal tract and on thyroid hormone levels. In general, PFOS is more toxic compared to PFOA. Carcinogenic effects of PFOS and PFOA have also been studied (human and animal studies, no focus on other PFAS). Several authorities, including ATSDR, U.S. EPA and IARC do not classify PFOS and PFOA as “proven carcinogens”, but instead as “suggestive carcinogens” or “possibly carcinogenic to humans” because of existing uncertainties. PFOS has been categorised as moderately acute and slightly chronically toxic to aquatic organisms. The MAC EQS (Maximum Allowable Concentration Environmental Quality Standard) derived by the European Commission for European freshwater and saltwater are based on the lowest NOEC reported (No Observed Effect Concentration of < 2,3 µg/l for *Chironomus tentans*) to protect the most sensitive species.

**Environmental fate and effects**

Emissions of PFAS to the environment include stable perfluoroalkyl sulphonic and carboxylic acids (PFSA and PFCA) and also less stable precursor compounds that may undergo abiotic or biotic transformation to PFSA and PFCA. While many studies have been published on environmental concentrations of PFSA and PFCA, much less data is available for precursor substances due to the difficulty inherent in their identification and analysis. Precursors are likely to have different physical and chemical properties to their breakdown products, leading to differences in their transport behaviour. For example, cationic or zwitterionic precursors may bind to clay minerals through ion exchange.

PFSA (e.g. PFOS) and PFCA (e.g. PFOA) are widely distributed in the global environment due to their high solubility in water, low/moderate sorption to soils and sediments and resistance to biological and chemical degradation. Monitoring data from across the EU show the widespread occurrence of PFSA and PFCA in surface water, with the very low EQS for PFOS in freshwater (0,00065 µg/l) often exceeded.

Little or no breakdown of PFOS and PFOA by photolysis is anticipated under environmental conditions.

**Analysis**

While a range of standard methods are available for the analysis of PFASs and PFCAs, the quantitative analysis of other PFAS substances is often difficult due to a lack of appropriate reference materials. To address this difficulty, analytical techniques have been developed whereby PFAS are quantitatively oxidized to fluoride (adsorbable organic fluorinated compounds (AOF) method), or a mixture of PFASs and PFCAs (total oxidisable precursor (TOP) method). The TOP method is most sensitive, with a detection limit around 0,002 µg/l range, vs 1 µg/l for AOF. Whereas the regulatory limits applicable for PFOS in groundwater (typically 0.02 to 1 µg/L) can sufficiently and reliably be measured and are above background levels, the AA-EQS of 0,00065 µg/l is so low that background levels are higher in many cases, and the AA-EQS is beyond the operational range of most commercial laboratories.

Specific precautions have to be taken in the sampling of environmental media since PFAS adsorb strongly to glass. Teflon-containing materials can lead to increased blank values if AOF is analysed, and may also interfere with the analysis by adsorbing PFAS. Currently the most appropriate material for sampling seems to be polyethylene or polypropylene.

**Remediation**

The remedial options available to address PFAS contamination are limited by the unique physical and chemical properties of these compounds. Many remediation methods utilized to address hydrocarbon contamination, such as air stripping, sparging, soil vapour extraction and bioremediation, are ineffective due to the low volatility of these compounds and their resistance to microbial degradation. Technologies currently used for the remediation of PFAS in soil and groundwater include excavation to landfill for soil (where authorised), and abstraction combined with activated carbon or resin treatment for groundwater. Groundwater extraction volumes may be high if remediation is required to very low environmental quality standards (e.g. for PFOS). Current best practice disposal routes for spent PFAS adsorption media are high temperature incineration at >1000°C, or regeneration at a specialist facility. Possible alternative remedial techniques include soil washing, soil solidification and the use of in-situ permeable reactive barriers or funnel and gate systems.

Emerging water treatment technologies for PFAS, such as photolysis/ photocatalysis, reductive decomposition, advanced oxidation and sonolysis, require high energy input per unit water volume and long residence times. Careful monitoring of treatment performance is also required to ensure complete breakdown of the various PFAS substances that may be present. Consequently, these technologies are unlikely to be feasible for high flowrate, low concentration applications.

**Implications for users of class B fire fighting foams**

It is important that users of class B fire-fighting foams understand and manage both environmental and fire safety aspects of foam use. An assessment of site foam stocks is recommended to ensure that any legacy stocks containing >0.001wt% PFOS (banned for use in the EU since June 2011) are set aside for safe disposal by high temperature incineration. A similar assessment should be completed for foam stocks that may be brought to site from third parties in the event of an emergency. At locations where fluorochemical-based foams have been used for fire-fighting or fire-fighting training, users should consider how to manage the potential issues.

In response to global regulatory initiatives to limit the production and use of long-chain PFAS substances, class B fire-fighting foam suppliers have developed foams that are completely free of fluorochemicals, and also "C6" foams based on

fluorotelomers containing 6 or fewer fully- fluorinated carbon atoms. C6 foams cannot degrade to PFOS or PFOA and so they seem of less concern from an environmental standpoint. It should be noted, however, that given the range of compounds present there is still uncertainty about their properties. In addition, low environmental concentration limits have been set for short- chain PFAS (i.e. <C6 PFSA; <C7 PFCA) in many EU countries due to their persistence. Where possible, therefore, water containing PFAS- based fire- fighting foam residues should be captured for treatment and not discharged to the environment.

While many sites now use fluorine- free foams for fire- fighting training and other non-critical application, there is still an ongoing debate with regard to their performance on larger in-depth fires (e.g. storage tank fires). It is therefore important that sites give careful consideration to both safety and environmental risk factors, and consult with fire safety experts, when determining the optimal foam type for any given application.



## 1. INTRODUCTION

Poly- and perfluoroalkyl substances (PFAS) have been used globally since the 1960's for a wide range of industrial, commercial and domestic applications due to their unique surface-active properties. PFAS have the ability to repel both oil and water, which has led to their use in stain guard products for carpets and soft furnishings. They are also used as specialist surfactants for industrial processing and fluoropolymer production. The ability of PFAS to form an aqueous film that will wet and flow across the surface of liquid hydrocarbon has led to their application in high performance fire-fighting foams used at airports, refineries, bulk storage terminals and other facilities handling large volumes of flammable liquid hydrocarbons. Their unique properties make it difficult to find equally effective replacement compounds for some applications, including in fire-fighting foams.

The perfluoroalkyl moiety within a PFAS molecule is highly resistant to abiotic and biotic degradation, which has led to the accumulation of PFAS breakdown products (perfluoroalkyl sulphonic and carboxylic acids) in the environment. Some of these substances (principally perfluorooctane sulfonic acid (PFOS) and perfluorooctanoic acid (PFOA)) have been identified as PBT, being persistent in the environment and having bioaccumulative and toxic properties in humans and wildlife. Therefore PFAS have emerged as a group of Constituents of Potential Concern (COPC), with an increasing regulatory focus regarding use, clean-up and protection of human health and the environment.

This technical report has been produced to help Concaawe members understand and manage potential environmental risks associated with the presence of PFAS in current and legacy fire-fighting foam formulations. The information contained may be useful in the design of site investigations, the development of conceptual site models and in the evaluation of potential risks and risk-management options. The report is structured as follows:

- **Section 2:** PFAS types, production methods and use;
- **Section 3:** Physical-chemical properties, and fate and transport behaviour;
- **Section 4:** Toxicity and potential risks to human health, ecology and the wider environment;
- **Section 5:** Regulatory values and environmental quality standards;
- **Section 6:** Current condition of European waters;
- **Section 7:** Chemical analysis methods;
- **Section 8:** Remediation options;
- **Section 9:** Conclusions.

## 2. PFAS TYPES, PRODUCTION AND USE

Poly- and perfluorinated alkyl substances comprise a large group of compounds (> 6,000) consisting of a hydrophobic alkyl chain of varying length, typically 2 to 16 carbon atoms, which is completely fluorinated (perfluorinated alkyl substances) or partly fluorinated with at least two fully fluorinated carbons (polyfluorinated alkyl substances). In Buck et al. (2011) PFAS are defined as compounds containing the perfluoroalkyl moiety  $C_nF_{2n+1-}$ , or more specifically:

- Perfluoroalkyl substances: aliphatic substances for which all of the H atoms attached to C atoms in the non-fluorinated substance from which they are notionally derived have been replaced by F atoms, except those H atoms whose substitution would modify the nature of any functional groups present (e.g. hydroxyl -OH).
- Polyfluoroalkyl substances: aliphatic substances for which all H atoms attached to at least one (but not all) C atoms have been replaced by F atoms, in such a manner that they contain the perfluoroalkyl moiety  $C_nF_{2n+1-}$ .

The above definition encompasses the group of fluorinated compounds that are of concern to global regulators. In particular, the definition excludes compounds containing “scattered” multiple F atoms (such as in  $CH_2FCHFCHFCH_2OH$ ). The widely used term “PFCs” (perfluorinated compounds) is not adopted since it is non-specific and encompasses perfluorinated chemicals in general, e.g. the greenhouse gas ( $CF_4$ ).

PFAS addressed by this review include perfluorinated carboxylic acids (PFCAs), perfluorinated sulfonic acids (PFSAs) and perfluorinated phosphonic acids (PFPAs). Polyfluorinated compounds include fluorotelomer alcohols (FTOHs), fluorotelomer sulfonic acids (FTSs), polyfluorinated alkyl phosphates (PAPs), perfluorooctane sulfonamide (PFOSA) and their derivatives.

PFAS production prior to 2001 was dominated by the 3M electrochemical fluorination process, which yielded 30-45% perfluorooctane sulfonylfluoride (POSF) as the main product and a range of other PFCAs and PFSAs. Since 2001, when production of PFAS by electrochemical fluorination ceased due to environmental concerns around PFOS, the main route for PFAS synthesis has been fluoro-telomerisation, which produces no PFOS or PFOS precursors.

Because of their unique surface characteristics (surfactant properties) and resistance to degradation in the presence of heat or acids perfluorinated alkyl substances have been used extensively in a variety of products and industries since the 1960s. Relevant to the refining industry is the use of PFAS in class B fire-fighting foams, including Aqueous Film Forming Foam (AFFF), Fluoroprotein (FP) and Film Forming Fluoroprotein Foam (FFFP).

This section provides an overview of the different types of PFAS and the production and use of what are viewed as the most important PFAS.

### 2.1. TYPES OF PFAS

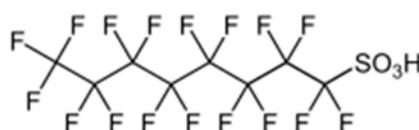
The family of PFAS comprises 42 families and subfamilies and several hundred compounds (Buck et al., 2011). The focus of this report is on the families of compounds described in the following sections.

### 2.1.1. Perfluoroalkyl sulfonic acids

Perfluorooctane sulfonate (PFOS) is a perfluoroalkyl sulfonic acid (PFSA), and is the most prominent PFSA (DEPA, 2013). It is the most commonly encountered perfluorinated compound in the environment and tissues of wildlife (Giesy, 2010). There are multiple other perfluorinated sulfonic acids with carbon chain lengths generally from C2 to C16.

PFOS consists of a chain of 8 fully fluorinated carbon atoms with a sulfonate group as the functional group on the terminal carbon. The structure of PFOS is given in **Figure 2.1**.

**Figure 2.1** Chemical structure of PFOS



In reality, PFOS is a mixture of linear (70%) and branched (30%) isomers<sup>1</sup> of PFOS, depending on the production process. PFSAs bearing a shorter perfluoroalkyl chain than PFOS can also be by-products of the production of PFOS. Furthermore, they are being introduced as alternatives for PFOS (Hori, 2006). For example, PFBS (perfluorobutane sulfonate; C<sub>4</sub>F<sub>9</sub>SO<sub>3</sub>-salt) is one important replacement substance for PFOS (Herzke, 2007).

PFOS is used as either the undissociated sulfonic acid or one of its sulfonate salts. The following salts are commonly known for PFOS:

- Ammonium salt,
- Potassium salt; and
- Lithium salt.

When dissolved in water, under most conditions, PFOS and its salts will dissociate to form the sulfonate anion.

### 2.1.2. Perfluoroalkyl carboxylic acids

Perfluoroalkyl carboxylic acids (PFCAs) are compounds that can contain a perfluorinated carbon chain of between 2 and 16 carbons in length with a terminal carboxylic acid functional group. Perfluorooctanoic acid (PFOA) is the most commonly encountered PFCA. PFCAs are widely used as products or raw materials for surfactants or surface treatment agents. PFOA is used as either the undissociated carboxylic acid or one of its carboxylate salts with ammonium perfluorooctanoate (APFO) as an example PFOA salt. PFOA has been widely used as an emulsion polymerization aid in the production of Teflon (Lindstrom, 2011).

PFOA consists of a chain of 7 perfluorinated carbon atoms, and a carboxyl head group. The structure of PFOA is given in **Figure 2.2**.

<sup>1</sup> Isomers: compounds with the same formula but different molecular structures.

**Figure 2.2** Chemical structure of PFOA



### 2.1.3. Potential PFSA and PFCA precursor compounds

Emissions of PFAS to the environment include stable PFASs and PFCAs and also less stable precursor compounds that may undergo abiotic or biotic transformation to PFASs and PFCAs. From a regulatory standpoint, precursors of long-chain PFASs and PFCAs are of greatest concern. OECD (2013) define long chain PFASs and PFCAs as:

- PFCAs with 7 and more perfluoroalkyl carbons, e.g. PFOA and PFNA
- PFASs with 6 and more perfluoroalkyl carbons, e.g. PFHxS and PFOS

In the OECD report the following classes of PFCA and PFSA precursors are identified:

- Substances that have the potential to degrade to long-chain PFCAs or PFASs, i.e. precursors such as PASF- and fluorotelomer-based compounds.
- Side-chain fluorinated polymers: fluorinated polymers consisting of variable compositions of non-fluorinated carbon backbones with polyfluoroalkyl (and possibly perfluoroalkyl) side chains. The fluorinated side-chains, including PASF- and fluorotelomer-based derivatives, are potential precursors of PFCAs.

PFSA and PFCA precursor compounds are reported (Backe et al., 2013; Martin et al., 2006 and Toms et al., 2009) to include:

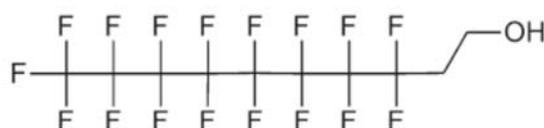
- fluorotelomers (polyfluorinated compounds);
- perfluoro sulfonamido carboxylates;
- perfluoro betaines;
- perfluoro sulphonamides;
- perfluoro sulfonamidoethanol;
- perfluoro thioamido amino carboxylates;
- perfluoro sulfonamido amines;
- perfluoro alkyl amido betaines;
- perfluoro sulfonamido amine oxides;
- perfluoro thioamido sulfonates;
- perfluoro thiohydroxyl ammonium;
- perfluoro sulfonamide ketones, aldehydes and ethers;
- perfluoro sulfonamide (acetic) acids.

The commercial analyses commonly used to quantify PFAS (e.g. US EPA method 537) only evaluate PFCAs and PFASs and do not detect the range of PFCA and PFSA precursors mentioned above. Research into the presence of precursors in urban runoff water in the San Francisco Bay area has shown that PFSA and PFCA represent less than 25% of the total amount of PFAS present (Houtz, 2012). Therefore, more comprehensive analytical methodologies are required.

### 2.1.3.1. Fluorotelomers

Fluorotelomers have an ethyl ( $\text{CH}_2\text{-CH}_2$ ) group between the fully fluorinated carbon chain and the functional group, and are therefore polyfluorinated molecules. In **Figure 2.3** an example is given of the fluorotelomer alcohol 8:2 FTOH, which has 8 fully fluorinated carbon atoms, an ethyl group, and an alcohol functional group. 8:2 FTOH is an example of a PFCA precursor: a number of studies have shown that it can transform to PFOA in the environment (Parsons et al., 2008).

**Figure 2.3:** Fluorotelomer alcohol 8:2 FTOH



Another widely- used fluorotelomer is the compound 1H-1H-2H-2H perfluorooctane sulfonate (also referred to as 6:2 FTS since it has 6 fully fluorinated carbon atoms, an ethyl group and a sulphonate functional group). Fluorotelomer sulfonates are used in place of PFOS for various applications, including class B fire- fighting foams and industrial surfactants.

Fluorotelomers are produced with a variety of different functional groups including alcohols, sulphonamides, sulfonamidoethylacrylates and methacrylates, and sulfonamidoacetic acids. The majority of the fluorotelomers are used for manufacturing various fluorotelomer-based products (e.g. building blocks for polymers, surfactants and side-chain fluorinated polymers). There is concern that many of these could eventually transform to PFSA and PFCA in the environment (Lindstrom et al., 2011).

### 2.1.3.2. Fluoropolymers

Fluorinated polymers may or may not be PFAS depending on whether they contain perfluoroalkyl moieties. The fluoropolymer polytetrafluoroethylene (Teflon, PTFE), is a PFAS and is used as a non-stick coating for cookware. It is virtually inert at normal temperatures, it starts to degrade above  $260^\circ\text{C}$ . Teflon resins contain part per million concentrations of hexafluoroacetone (HFA). PFOA is an essential processing aid in the formulation of these polymers. The manufacturing of non-stick cookware includes a sintering step at high temperature, which theoretically volatilizes residual PFOA (Herzke et al., 2007).

In PTFE-coated textiles (jackets, table-cloth etc.), primarily fluorotelomer alcohols and fluorotelomer carboxylic acids have been detected in relatively large quantities (up to  $11\text{ mg/m}^2$  fluorotelomers and  $0,4\text{ mg/m}^2$  PFCA, Berger and Herzke, 2006). During thermolysis of PTFE polymers trifluoroacetate and chlorodifluoroacetate can be produced (Herzke et al., 2007).

## 2.2. PRODUCTION PROCESSES

Historically, two processes have been used for the production of PFAS: electrochemical fluorination (ECF) and telomerization (TM). These synthesis routes result in different (isomeric) purities. In general the ECF process yields even- and odd-numbered, branched and linear chains of perfluoroalkyl compounds, whereas TM

produces only even-numbered, linear chains. Most production is nowadays undertaken via the telomerisation process (Buck et al., 2011).

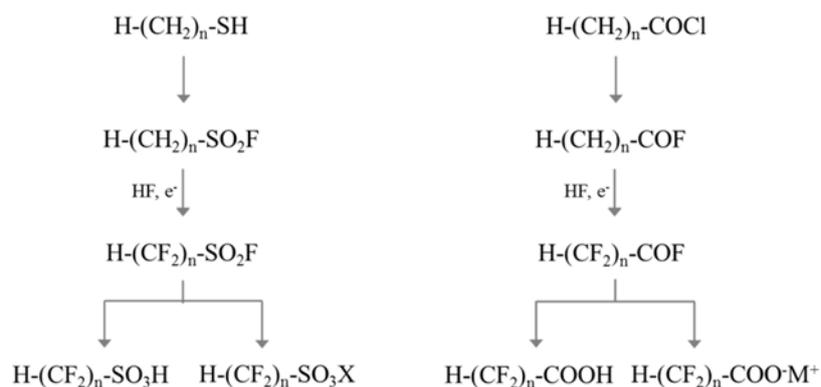
### 2.2.1. Electrochemical Fluorination

The source compound for manufacture of PFOS-related chemicals is perfluorooctane sulfonylfluoride (POSF). POSF is manufactured through a process known as electrochemical fluorination (**Figure 2.4**), in which an electric current is passed through a solution of anhydrous hydrogen fluoride (HF) and an organic feedstock of octane sulfonyl fluoride. The ECF process replaces the hydrogen within the carbon-hydrogen bonds of the organic feedstock with fluorine. Although for production of PFOS-related substances the starting material is linear octane sulfonyl fluoride, the product will contain some branched C8 compounds since the fluorination process is expected to lead to partial fragmentation and rejoining of the chain (EFSA, 2008).

The ECF process yields between 30% – 45% linear-chained POSF. The output of the ECF process thus is a mixture of isomers and homologues including shorter and longer linear-chained homologues; branched perfluoroalkyl fluorides of various chain lengths; linear-chained, branched, and cyclic perfluoroalkanes and ethers; and other by-products (OECD, 2002).

PFOA can also be produced through ECF using octanoyl fluoride as the feedstock with HF.

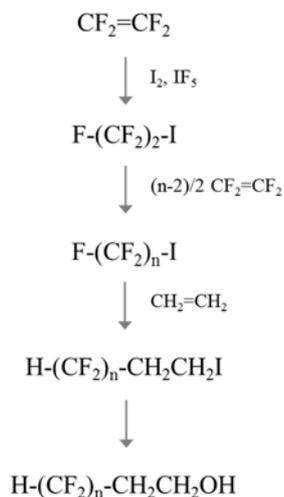
**Figure 2.4:** Production routes of PFOS and PFOA via electrochemical fluorination (Buck et al., 2011)



### 2.2.2. Fluoro Telomerization

The fluoro telomerization process (**Figure 2.5**) used by the industry leads to a successive addition of an ethyl group to the fluoroalkyl chain. It involves the reaction of perfluoroethylene (tetrafluorethene,  $\text{CF}_2=\text{CF}_2$ ) and perfluoroethyl iodide (trifluoroiodoethene,  $\text{CF}_3\text{-CF}_2\text{I}$ ) to produce linear-chained perfluorinated iodides with chain lengths that are generally even numbered. These perfluorinated iodides are then used as a feedstock to produce linear-chained perfluorinated carboxylic acids, fluorotelomer alcohols, and fluorotelomer olefins (Lindstrom, 2011). Through this process no PFOS or PFOS precursors are produced.

**Figure 2.5:** Production route of fluorotelomers via telomerisation (Buck et al., 2011)



## 2.3. USE

### 2.3.1. General Use

PFAS are used in a variety of products and production processes. From 1966 to the 1990s, the production and use grew due to their unique chemical stability and their surface tension/levelling properties. The annual production rate of PFOS increased significantly from 500 tonnes/year in the 1970's to almost 5000 tonnes/year in 2000 (Carloni, 2009). The use of PFOS included inks, varnishes, waxes, fire-fighting foams, metal plating and cleaning, coating formulations, lubricants, water and oil repellents for leather, paper and textiles (Paul et al., 2009). An overview of the uses of a selection of the different PFAS is given in **Appendix 1**.

In 2000, the key global producer 3M started to phase out the production of PFOS. Between 2000 and 2003, the global production dropped sharply as a consequence of 3M's initiative. In this period the production of PFOS in China increased, but not to the same global production level as before the year 2000 (Paul et al., 2009; Carloni, 2009).

In May 2009, PFOS was added to Annex B of the Stockholm Convention. Since that date, the use of PFOS and related compounds has been restricted in signatory countries to the Convention, although it is still being used for certain applications in which PFOS cannot be replaced by other chemicals (more information is included in Section 5).

PFAS have been found at a wide range of sites including manufacturing sites and within landfills, but is also encountered at airports, military sites and other large industrial (e.g., petrochemical) facilities where fires have occurred and/or fire-fighting trainings have been carried out.

### 2.3.2. Fire-fighting foam use

PFAS-based class B fire-fighting foams have been used since the 1970s for vapour suppression, firefighting and fire-fighting training at airports, refineries, bulk storage terminals and other facilities handling large volumes of flammable liquid hydrocarbon. PFAS are used in fire foam products because of their ability to produce a foam that will wet the surface of liquid hydrocarbon, resulting in a much faster foam spreading rate than is possible using only hydrocarbon-based surfactants.

Class B fire-fighting foam types likely to include PFAS include Aqueous Film Forming Foam (AFFF), Fluoroprotein (FP) and Film Forming Fluoroprotein Foam (FFFP). Supplier safety data sheets *may* list PFAS as “fluoroalkyl surfactant”, but the identity of the PFAS substances present is not usually provided (regarded as proprietary information).

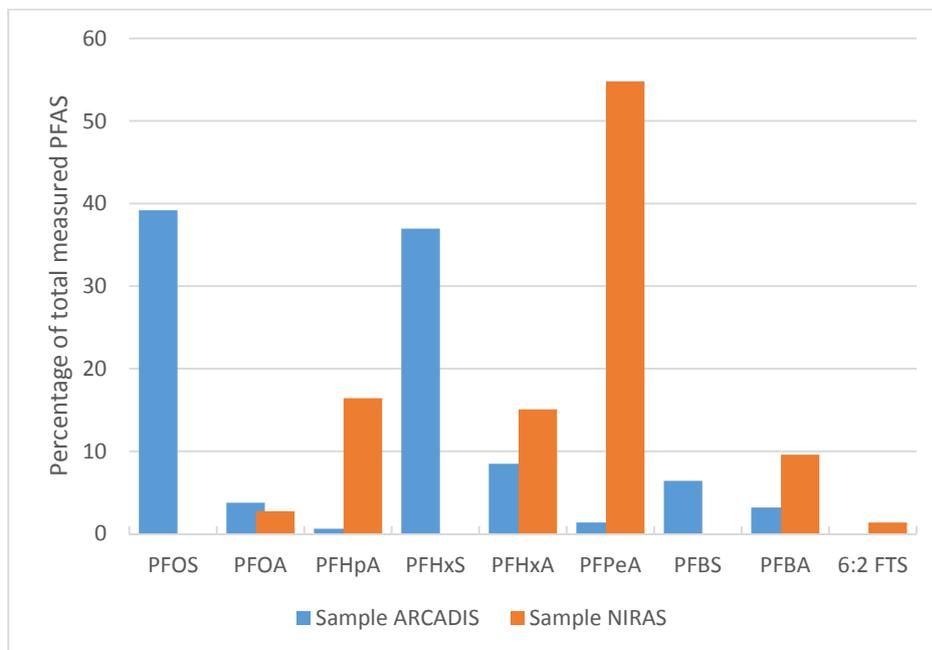
PFOS and its derivatives were used extensively in fire-fighting foam manufacture prior to 2001, when the production of PFOS was phased out in the USA due to environmental concerns. Since 2001 fire foams have been manufactured using fluorotelomer-based fluorosurfactants (Seow, 2013). However due to the long shelf-life of foam concentrates, it is likely that the use of PFOS-based foam products manufactured prior to 2001 continued after cessation of production: an EU ban on the use of foams containing PFOS as a primary component (>0,001 wt%) only came into force in June 2011. In countries not complying with the Stockholm convention, PFOS might still be used, and stockpiles of AFFF containing PFOS might still be present.

At sites where fire-fighting foams have been used residual PFAS may be present in soil and groundwater below fire-fighting training areas, areas where large fires have occurred, foam storage and dispensing locations and locations where PFAS-based foam has been repeatedly used for flammable vapour suppression during ‘hot work’. The Danish Environmental Protection Agency (DEPA) investigated the relationship between groundwater contamination and point sources of PFAS (DEPA, 2014) at both civil and military airports. The authors concluded that fire training is a high potential source for PFAS contamination of groundwater. During a further study conducted by NIRAS for the Danish Defence (military airfields), contaminated groundwater at a fire-fighting training area was analysed (Falkenberg et al., 2015). In this sample, PFPeA, PFHxA and PFHpA were the dominant PFAS in the groundwater, caused by contamination from the AFFF. PFOS was not observed in the groundwater.

ARCADIS analysed groundwater samples known to be contaminated with AFFF in a confidential study (the Netherlands, 2011). Based on this study, PFOS, PFHxS and PFHxA represented 82% of the total PFAS concentration detected in groundwater associated with contamination by this type of AFFF.

Results from both studies are illustrated in **Figure 2.6** and clearly show very different PFAS profiles in the groundwater, possibly reflecting the use of different fire-foam types.

**Figure 2.6:** PFAS concentrations in groundwater at two AFFF contaminated sites



These are just two examples of fire-fighting foam related PFAS impacts in groundwater. They are not to be assumed representative of typical impacts. A great deal of variability in PFAS mixtures used in fire-fighting foams and encountered in groundwater has been reported.

Backe et al. (2013) developed a new method to quantify an extensive range of PFAS in groundwater and fire-fighting foam. The authors concluded that “the profiles of PFAS in groundwater differ from those found in AFFF formulations, which potentially indicates environmental transformation of PFAS”.

In another study, Barzen-Hanson et al. (2015) analysed 5 different 3M AFFFs manufactured in the period 1989 – 2001, with focus on ultra-short PFSA (C2 PFSA: PFEtS, perfluoroethane sulfonate, and C3 PFSA: PFPrS, perfluoropropane sulfonate). The five types of AFFF were dominated by the following PFASs: PFOS, PFHxS and PFBS. However, relatively high concentrations of PFPrS (120 – 270 mg/l) and PFEtS (7 – 13 mg/l) were detected, representing 3,5% and 0,2% of the total PFSA concentration in AFFF. The relative ratio of these compounds in groundwater varies between sites and is different from the ratio detected in AFFF.

The relationship between fire-fighting foam type and potential impacts to groundwater quality can be summarized as follows:

- PFAS-based fire-fighting foam formulations have changed over time. Before 2001, the main PFAS compound was PFOS. After 2001, this changed to 8:2 FTS, 6:2 FTS and other fluorotelomer based PFAS. Recent investigations show also a portion of ultra-short PFSA (C2 and C3).
- Studies to date do not indicate a strong link between the ratio of PFAS in fire-fighting foam products and the ratio of PFAS in groundwater where they were used. Reasons for this could include: (1) Groundwater PFAS ratios being

dominated by a foam type other than the one tested, in the event that foam composition changed over time or different foams were used (2) Groundwater PFAS ratios changing due to differential transport during groundwater migration (3) Groundwater PFAS ratios being dominated by the degradation of precursors, rather than the PFAS present in the foam products (4) Interactions of PFAS with co-contaminants (e.g. differential partitioning into NAPL).

Further information on PFAS transport in groundwater is provided in Section 3.2.2.

## **2.4. ENVIRONMENTAL CONCERNS**

Although the use of PFOS is now restricted in many markets including the EU, PFOS can still be present in fire-fighting foam at levels up to 0,001 wt% (see Section 5.2.1).

PFOS is being replaced by alternatives, for example fluorotelomer derivatives based on mainly 6:2 FTS for fire-fighting (Seow, 2013) and smaller PFAS such as PFHxS and PFBS for their stain repelling properties (Stockholm Convention, 2014). Although these compounds are likely to be less toxic and have reduced bioaccumulative properties, concerns have raised about their transformation products becoming ubiquitously present in the global environment and about the lack of alternatives for PFAS (Scheringer, 2014). The unique PFAS properties make it difficult to find equally effective replacement compounds for some applications. Regarding fire-fighting foams specifically, there are concerns about finding the right balance between safe and effective fire-fighting and environmental protection.

Furthermore, although currently regulatory efforts are mainly focussed PFOS and PFOA, it is important to realize that several thousands of different PFAS are known to exist (Lindstrom, 2011). A few countries already regulate several additional PFAS (see Section 5.2). In addition proposed regulation specifically targeting fluorinated fire-fighting foam management may affect fire-fighting foam selection.

### 3. PROPERTIES, FATE AND BEHAVIOR

From the standpoint of environmental fate and effects, PFAS substances can be broadly divided into:

- Perfluoroalkyl sulphonic and carboxylic acids (PFSA and PFCA), for which environmental analysis is commercially available according to standardised test protocols. For these compounds a significant quantity of high-quality environmental fate data is available
- Other PFAS substances, including PFSA and PFCA precursors, for which very little environmental fate data is available due to the difficulties inherent in their analysis.

Perfluoroalkyl sulphonic and carboxylic acids (PFSA and PFCA) are widely distributed in the global environment due to their high solubility in water, low/moderate sorption to soils and sediments and resistance to biological and chemical degradation. While many studies have been published on environmental concentrations of PFSA and PFCA, little data is available for precursor substances due to the difficulty inherent in their identification and analysis.

Over the pH range normally found in soil, groundwater and surface waters (pH 5-9) PFSA and PFCA are normally present as anions, and this reduces sorption by soils and sediments, which usually carry a net negative charge. Their retardation during transport in groundwater increases with perfluorocarbon chain length and the fraction of organic carbon in the soil, with PFSA binding more strongly than PFCA of the same carbon number. The presence of co-contaminants has a variable impact on the mobility of PFAS, depending on PFAS chain length, PFAS concentrations and the characteristics of the co-contaminant. The environmental mobility of other PFAS substances is not well understood due to the lack of analytical data. Precursors are likely to have different physical and chemical properties to their breakdown products, leading to differences in their transport behaviour. For example, cationic or zwitterionic precursors may bind to clay minerals through ion exchange.

PFOS and PFOA have not been demonstrated to undergo significant biotransformation under normal environmental conditions. Little or no breakdown of PFOS and PFOA by photolysis is anticipated under environmental conditions.

More information is presented in the sections below.

#### 3.1. PHYSICOCHEMICAL PROPERTIES

Physicochemical properties for a number of PFAS, derived from scientific literature (Wang Z. et al., 2011), are summarized in **Appendix 2**, including:

- PFAS name and acronym;
- CAS registry number;
- Molecular formula;
- Molecular weight;
- Density;
- Solubility in water;
- Melting point;
- Boiling point;
- Vapour pressure;

- Henry's coefficient (i.e., air-water partition coefficient);
- Octanol-water partition coefficient ( $K_{ow}$ );
- Organic carbon-water partition coefficient ( $K_{oc}$ );
- Soil distribution coefficient ( $K_d$ );
- Dissociation constant ( $pK_a$ ).

As shown in **Appendix 2**, over 50 individual PFAS were identified for this review and fall into the following categories:

- Perfluorinated carboxylic acids; e.g. PFBA, PFPeA, PFHxA, PFHpA, and PFOA;
- Perfluorinated sulfonic acids; e.g. PFBS, PFPeS, PFHxS, PFHpS, and PFOS;
- Perfluorinated phosphonic acids (PFPA);
- Polyfluorinated compounds and/or precursors to PFSA and PFCAs, fluorotelomer alcohols (FTOHs), fluorotelomer sulfonic acids (FTSs), polyfluorinated alkyl phosphates (PAPs), perfluorooctane sulfonamide (PFOSA) and derivatives.

While PFOS and PFOA are comparatively well studied compared to other PFAS, many of which have not been studied at all, the available data is still relatively scarce. It should be noted that reported physicochemical properties vary in the literature. For example, 6:2 FTS exhibits a significant correlation between pH and solubility: the further the pH falls below pH 7 the greater the solubility decreases. This correlation is not likely to be due to the different form of a salt (carboxylate) or free acid, since this compound is already completely dissociated with a  $pK_a$  of less than 1,31.

Some of the parameters in **Appendix 2** are calculated parameters from literature. These parameters are based on the neutral form of the substances and not the conjugate base, which predominates for some PFAS at neutral pH (Wang Z. et al., 2011).

In addition, it is often observed that the physicochemical properties within a homologous PFAS series (i.e., the same terminal functional group with different  $CF_2$  chain length) change non-linearly. The reason may be that with increasing chain length, the geometry of the molecules changes (Wang Z. et al., 2011). When a PFAS molecule contains up to eight fluorinated carbon atoms, the molecule remains in a linear conformation. When a PFAS molecule contains more than eight fluorinated carbon atoms, a helix can be formed. The resulting increase in electron density leads to changes in physicochemical properties.

#### Structure of PFAS

Fluorine has the highest electronegativity of all atoms, a high ionization potential, and very low polarizability due to the low deformability of the outer electron shell. The covalent carbon-fluorine bond is one of the strongest bonds in organic chemistry (450 kJ / mol) due to the effective overlap of the molecular orbitals involved in the bond. Fluorine-carbon bonds are very infrequently found in naturally occurring organic compounds, although some plants and microorganisms synthesize organofluorine compounds (Murphy et al., 2003). The dense packing of fluorine electrons can also act as "shield", protecting PFAS from external attacks and thus causing the high thermal, chemical, photolytic (UV-radiation) and biological stability of these materials. The energy required for reduction of fluorine ( $F^- \rightarrow F + e^-$ ) is exceptionally high ( $E^0 = 3,6$  V).

The PFAS considered in this review generally consist of a hydrophobic, polyfluorinated or perfluorinated carbon chain and a hydrophilic functional consisting

of, for example, sulfonate or carboxylate or their salts. This amphiphilic (both hydrophobic and hydrophilic) characteristic of PFAS makes them ideal for use as surfactants. However, in contrast to conventional surfactants, the perfluorinated carbon chain also has a lipophobic characteristic which renders many PFAS coatings resistant not only to water, but also to oil, grease, other non-polar compounds and dirt particles. The surface activity of PFAS surfactants is higher than analogous hydrocarbon surfactants. This property is one of the reasons for the wide use of PFAS in industry. Both the length of the carbon chain and the configuration of the polar functional group can vary widely in different PFAS and results in a variety of different materials with different physicochemical properties. However, not all PFAS exhibit surfactant properties. For example, the hydrophilic influence of the hydroxyl group found on telomeric alcohols is too small to act as a surfactant.

PFAS surfactants have the ability, on the one hand, to group together at phase boundaries and on the other, to form micelles. Thus, in the environment, there can be accumulation of PFAS at the phase boundary between groundwater (hydrophilic) and soil air (hydrophobic).

## 3.2. FATE AND TRANSPORT

### 3.2.1. Fate

The following PFAS fate and transport characteristics are important:

#### Water Solubility

Solubility values for the PFAS listed in **Appendix 2** were derived from literature sources where available, either measured values or estimated based on molecular weight using standard environmental chemistry calculations (e.g. COSMOtherm).

As shown, solubility values for **PFCAs** (PFBA, PFPeA, PFHxA, PFHpA, and PFOA) vary between 4,2 g/l and fully miscible, and solubility values for **PFSAs** (PFBS, PFPeS, PFHxS, PFHpS, and PFOS) vary between 0,5 and 56,6 g/l. These relatively high solubility values in the gram per litre (g/l) range for the PFCAs and PFSAs are due to the carboxylate and sulfonate groups on these molecules, because these groups are hydrophilic. The solubility of PFCAs and PFSAs tends to decrease with molecular weight, which is due to the concomitant increase in the length of the perfluorinated alkyl chains which are hydrophobic.

In natural waters, the predominant species of PFCAs and PFSAs will be their anionic forms, which is due to the very low dissociation constants of these compounds (**Appendix 2**). At very low pH, PFCAs and PFSAs can exist in water in their fully protonated forms. However, most natural waters exhibit approximately neutral pH values and therefore it can be reasonably assumed that PFCAs and PFSAs exist as anions when dissolved in water.

The **fluorotelomer alcohols** (FTOHs) are very hydrophobic and are of relatively low solubility in water. For example, PFOA has a solubility of 3,4 to 9,5 g/l and perfluoroethylethanol (FTOH 4:2) has a solubility of 0,98 g/l (**Appendix 2**). Also, the water solubility decreases with increasing length of the alkyl chain. As with hydrocarbon-based surfactants, it can be assumed that the solubility of PFAS is affected by the chemical composition of the groundwater, particularly if the groundwater contains divalent ions.

The solubility of the **precursors** is estimated to vary over many orders of magnitude, as shown in **Appendix 2**, due largely to the significant variance in molecular type, structure, and weight of the various precursors. One important finding from this review is that very little research has been published on the water solubility for most PFAS.

#### Dissociation

When an acid dissolves in water, dissociation is the process by which the electronegative atom and a hydrogen atom, which are ionically bonded, separate into a proton ( $H^+$ ) and a negative ion. The extent of this dissociation in water is described by a chemical-specific dissociation constant ( $pK_a$ ). The  $pK_a$  value is a pH value at which half of the acid molecules dissociate into ions. The smaller the  $pK_a$  value is, the greater the extent of dissociation will occur at any pH.

Both PFOS and PFOA have negative  $pK_a$  values, which means both of these PFAS function as strong acids and exist as dissociated anions in aqueous solutions under almost all natural conditions. The tendency to release a hydrogen atom (proton) is a typical characteristic of an acid. The two compounds, PFOS and PFOA, are thus to be regarded as strong acids. In the salts of PFCAs and PFSA, the counter-ion (e.g. lithium) is also ionically associated with the carboxylate or sulfonate anion. In aquatic systems, these salts will dissociate into the positively charged cation and the negatively charged carboxylate or sulfonate ions.

Investigations at AFFF-impacted sites and other sites with PFAS concentrations in the range of  $\mu g/l$  up to  $mg/l$  did not show a decrease in the pH due to the presence of PFAS. As described above, the  $pK_a$  for PFOS and PFOA is negative, but pH is a function of the  $H^+$  concentration. PFOS and PFOA are normally not present at a very high concentration when tested in the environment ( $mg/l$  maximum) or are present as salts, which means the concentration of  $H^+$  (protons) in water is not sufficient to effectively influence the pH.

FTOHs are not acids and do not dissociate when dissolved in water.

#### Physical State

At typical environmental temperatures and pressures, PFAS and their salts exist predominantly as solids. Only the short-chain FTOH 6:2 exists as a liquid. The melting and boiling points of all PFAS in this review are comparatively high. PFOA has a relatively low melting point ( $59-60^\circ C$ ) and boiling point ( $192^\circ C$ ). For PFOS, the values are significantly higher. It is likely that shorter PFCAs melt and boil at lower temperatures than PFOS. FTOH 8:2 exists at room temperature as a solid, but sublimates from the solid form from open vessels and can volatilize from the liquid phase.

#### Vapour Pressure

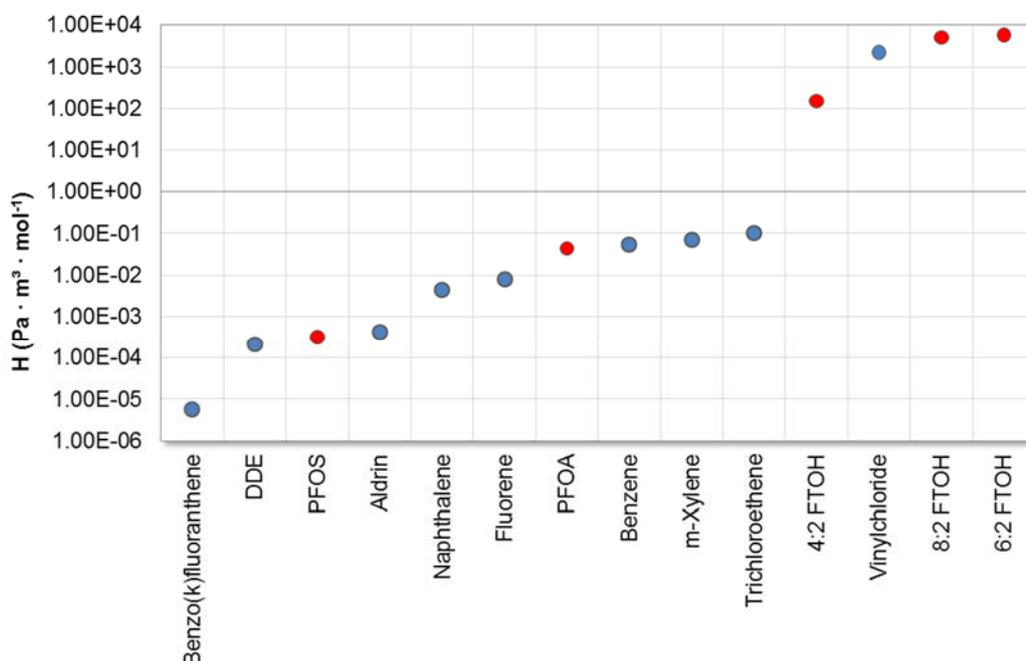
Vapour migration plays only a minor role in assessing the mobility of most PFAS in the environment due to the low to very low vapour pressure of the PFAS.

FTOHs are reported in the literature as having varying vapour pressures but, compared with other PFAS they have much higher vapour pressures and are therefore classified as volatile. It is therefore believed that FTOHs may migrate away from production/manufacturing processes in the atmosphere as a gas phase. FTOHs can, through various transformation processes discussed below, be transformed into PFOA and result in diffuse pollution of surface water and groundwater resources through precipitation.

**Henry's Coefficient (H)**

Henry's coefficient is an equilibrium partitioning coefficient that describes the extent to which a chemical partitions between the aqueous and gaseous phases. Henry's coefficients for PFAS, where known, are summarized in **Appendix 2**. Henry's coefficients for PFAS are also shown graphically on **Figure 3.1** along with values for some well-characterized hydrocarbons and solvents for comparison purposes.

**Figure 3.1:** Comparison of Henry coefficients for selected PFAS vs well-characterized hydrocarbons and solvents



As shown in **Appendix 2** and **Figure 3.1**, Henry coefficients for PFAS are quite variable, and range over nine orders of magnitude. For example, the Henry's coefficients for FTOH 8:2 and FTOH 6:2 are high and comparable to vinyl chloride. The Henry's coefficient for PFOA is comparable to those of benzene and xylenes. The Henry's coefficient for PFOS, on the other hand, is practically negligible and indicates that little PFOS will partition from the aqueous phase to the vapour phase from a fate and transport perspective. Because of this, volatilization of PFOS and PFOA from water is not considered to be a significant transport mechanism.

Since Henry's coefficients for most PFAS are not known, it is clear that more research is needed to understand the fate and transport of PFAS in the environment.

**3.2.2. Transport**

Mobility of PFAS in water will in part be influenced by the degree to which the PFAS sorb to sediments or soils during transport. The effect of PFAS sorption to sediments or soils during transport is to remove a portion of the PFAS from the aqueous phase, either permanently or temporarily, which can slow down or retard the velocity of the PFAS relative to the water velocity and attenuate PFAS concentrations over time and

distance. There are two sorption mechanisms which control the degree of PFAS sorption to sediments and soils during transport in water:

1. Hydrophobic sorption to naturally-occurring solid organic particles; and
2. Surface sorption to charged mineral surfaces.

Each of the sorption mechanisms is described below.

#### 1. Hydrophobic Sorption of PFAS to Naturally-Occurring Solid Organic Carbon

PFAS can sorb to naturally-occurring solid organic carbon particles present in sediment or soil during transport in water, in a manner analogous to sorption to granular activated carbon in water treatment systems. However, this mechanism also occurs naturally during transport because all soils and sediments typically contain some level of naturally-occurring solid organic carbon. The degree to which a PFAS sorbs to naturally-occurring solid organic carbon particles in sediment or soil during transport in water can be estimated by the PFAS-specific organic-carbon partition coefficient ( $K_{oc}$ ), the PFAS-specific octanol-water partition coefficient ( $K_{ow}$ ), or the PFAS- and soil-specific distribution coefficient ( $K_d$ ). Published values for these indicators are summarized in **Appendix 2**. It shall be noted however, that  $K_{ow}$  values for most PFAS are difficult to measure as they do not follow the typical lipid partition dynamics, due to their anionic or cationic charge. Therefore,  $K_{ow}$  is not an adequate parameter to predict sorption of PFAS.

The reason that three different indicators of PFAS sorption to sediments or soils were included in this review is that not all researchers measure or report each indicator, yet each indicator can provide some insight regarding the extent of PFAS sorption.

One implication regarding the degree of PFAS hydrophobic sorption and mobility in water from the information in **Appendix 2** is that there is a very wide range of reported values for all PFAS. Sorption of PFCAs and PFSAAs will increase with increasing chain length and with increasing solid phase fraction of organic carbon ( $f_{oc}$ ). In addition, sorption increases with decreasing pH and increasing concentration of  $Ca^{2+}$ . This finding suggests that the degree of PFAS hydrophobic sorption to soils and sediments is a site-specific phenomenon, and depends on the specific PFAS present at a site as well as the specific soil type.

Another implication regarding the degree of PFAS hydrophobic sorption and mobility in water from the information in **Appendix 2** is that no data were reported for hydrophobic sorption properties for more than half of the PFAS. This finding also indicates that more basic research is needed to determine the hydrophobic sorption properties of individual PFAS in soil and sediment. However, this issue may only be relevant for PFAS that are persistent in the environment. If a precursor exhibits rapid transformation in the environment, information on sorption properties is not that relevant.

#### 2. Surface Sorption of PFAS to Charged Mineral Surfaces

Because all of the PFCAs, PFSAAs, PFPAs, and some of the precursors are strong or weak acids that exist as anions in natural waters at almost all pH, surface sorption to charged mineral surfaces naturally present in soils or sediments may be a significant mechanism controlling the mobility of these PFAS in water during transport. While there are no numerical indicators of the extent to which anionic PFAS sorb to charged mineral surfaces that could be included in **Appendix 2**, several publications were

reviewed that provide some insight to this mechanism and implications for fate and transport.

Johnson et al. (2007) equilibrated several materials with solutions of PFOS to characterize surface sorption, including goethite, kaolinite, high iron sand and Ottawa sand (a silica sand produced by processing material obtained by hydraulic mining of massive orthoquartzite situated in deposits near Ottawa, Illinois). They found that PFOS sorption was significant, but lower than for many organic contaminants of similar molecular weight. The surface area normalized sorption of PFOS decreased for the materials in the following order: Ottawa sand > high iron sand > kaolinite > goethite.

Tang et al. (2010) investigated PFOS adsorption onto goethite and silica by batch adsorption experiments under various solution compositions. They found that PFOS adsorption onto silica surfaces was marginally affected by solution pH, ionic strength, and calcium concentration. However, in contrast, they found that PFOS uptake by goethite increased significantly at lower pH and higher calcium concentrations, which was likely due to enhanced electrostatic attraction between the negatively charged PFOS molecules and positively charged goethite surface.

Ferrey et al. (2012) investigated PFOS and PFOA sorption onto mineral surfaces by constructing laboratory microcosms with sediment from beneath a landfill and amending the microcosms with PFOS and PFOA. They found that sorption of PFOA and PFOS at near neutral pH was controlled by electrostatic sorption on ferric oxide minerals, and not by sorption to organic carbon, and that there was no evidence for degradation of the PFOA or PFOS. It should be noted that the batch microcosm experimental setup differs significantly from that typically used in batch sorption experiments, which may yield different results than batch sorption conditions designed to promote equilibrium conditions. Based on their results, the authors (Ferrey et al., 2012) recommended “that accurate predictions of PFOA and PFOS mobility in groundwater should be based on empirical estimates of sorption using affected soils or sediments.”

Lipson et al. (2013) investigated PFOS transport in bedrock groundwater at a well-characterized site where AFFF was released to the ground as part of fire-fighting activities during a catastrophic fire at a petroleum storage facility in the United Kingdom. Because the PFOS-containing AFFF was released concurrently with petroleum containing methyl-tert-butyl ether (MTBE) which has well-known fate and transport characteristics, the fate and transport of PFOS in a fractured chalk aquifer could be compared with that of MTBE. Based on mathematical fate and transport modelling results, they found that PFOS transport velocity was significantly lower than the average linear groundwater velocity and that the dual-porosity retardation factor for PFOS was lower than MTBE, indicating PFOS is more mobile than MTBE in this setting. The PFOS diffusion coefficient estimated through model calibration was significantly lower than the standard estimation method and it was hypothesized that PFOS transport was influenced by an anion exclusion effect associated with surface charge on the aquifer mineral surfaces.

One observation regarding the influence of surface sorption of PFAS to charged mineral surfaces during transport in water is that the results of the research in this area have been remarkably consistent, and demonstrate that surface sorption of PFAS to charged mineral surfaces during transport in water is an important mechanism controlling mobility of PFAS in water. However, very little research has been performed regarding this mechanism and what research has been published

has been focused on PFOS and PFOA. Clearly, more basic research is needed in this area.

Another observation regarding the influence of surface sorption of PFAS to charged mineral surfaces during transport in water is that site-specific information regarding soil mineralogy and groundwater geochemistry are required to understand and accurately predict PFAS mobility in water.

Apart from the two sorption mechanisms as discussed above, mobility of PFAS may also be influenced by the presence of co-contaminants (Lipson et al. 2013). Guelfo et al. (2013) studied the sorption of PFAS to multiple soils in the presence of (1) nonaqueous phase liquid (NAPL), which may be relevant at AFFF-impacted sites, and (2) non-fluorinated AFFF surfactants. PFAS with more than 6 CF<sub>2</sub> groups demonstrated variable sorption properties affected by the presence of NAPL and non-fluorinated AFFF. Shorter chain PFAS generally showed an increase in the sorption due to the presence of co-contaminants. The authors concluded that "PFAS groundwater transport at AFFF-impacted sites will depend on the solid phase characteristics as well as the PFAS concentration and chain length". In another study (Pan et al., 2009) the influence of cationic and anionic surfactants on the mobility of PFOS was investigated. The results showed that in the presence of a cationic surfactant, the sorption of PFOS on sediments increased due to hydrophobicity partitioning to the sorbed surfactant. The anionic surfactant on the other hand, increased the mobilisation of PFOS (concentration dependent), meaning that both types of surfactants have contrasting impacts.

### 3.2.3. PFAS Transformations

#### Biotic Transformations

**PFCAs and PFSA**s are generally considered to be recalcitrant to biodegradation via naturally-occurring microorganisms in water or soil. Biodegradation studies in which PFOS or PFOA were monitored for loss of parent compound have been conducted using a variety of microbial sources and exposure regimes (Parsons, 2008). Under aerobic conditions with activated sludge, no loss or biotransformation of PFOS or PFOA was observed. Under anaerobic circumstances, some removal of PFOS and PFOA has been observed, but no metabolites nor increase of fluoride was measured. To date, no laboratory data exist that demonstrates that PFCAs or PFSA undergo significant and complete biodegradation under environmental conditions.

**Precursors** are known to be transformed into PFCAs and PFSA under natural circumstances. Biotransformation of the **8:2 Telomer Alcohol** (FTOH 8:2) is relatively well studied (Parsons et al., 2008). The aerobic degradation of FTOH 8:2 begins with oxidation of the alcohol to an acid moiety, and then a subsequent  $\beta$ -oxidation to the complete degradation of the non-fluorinated aliphatic portion of the molecule. As a result, a PFCA is created as a by-product, in this case, PFOA. The removal of only the non-fluorinated radical to form the corresponding PFCA, in this case PFNA, is minor. In another study, these compounds were not detected (Wang et al., 2009).

Degradation studies using radiolabelled compounds [<sup>14</sup>C] on FTOH 8:2 molecules revealed a number of important results (Wang et al., 2009). After seven months of incubation, 35% of the <sup>14</sup>C molecules were irreversibly bound to the soil and could only be removed by combustion. This was confirmed by the fact that free fluoride (F) accounts for only a part of the mass loss (Dinglasan et al., 2004). A number of metabolites were identified, including:

- 3-OH-acid 7-3 F (CF<sub>2</sub>)<sub>7</sub>CHOH-CH<sub>2</sub>COOH;
- 7-2 FT-ketone F (CF<sub>2</sub>)<sub>7</sub>COCH<sub>3</sub>;
- 7-3 acid F (CF<sub>2</sub>)<sub>7</sub>CH<sub>2</sub>CH<sub>2</sub>COOH ;
- 2H-PFOA F (CF<sub>2</sub>)<sub>6</sub>CH<sub>2</sub>-COOH (11% after 7 days).

The formation of some of these metabolites and the fact that the <sup>14</sup>C-labeling could be dismissed after the formation of <sup>14</sup>CO<sub>2</sub> (6,8%) shows that multiple CF<sub>2</sub>-groups were reduced from FTOH 8:2. Three of the metabolites, PFOA (25%), 2H-PFOA (2%), and 7:3 acid (11%) were found to be stable. The remaining metabolites were detected only transiently. The ratio of PFOA to 7:3 acid (1,8 to 2,5) can be used as an indicator of the source of PFOS. PFOS was not observed as a transformation product from the degradation of FTOH 8:2 (Wang et al., 2009). Results also showed that degradation of FTOH 8:2 was relatively fast, with a half-life of approximately seven days. Partial mineralization of FTOHs to carbon dioxide during the study also shows that microorganisms can derive energy and grow from the removal of the non-fluorinated moiety.

Studies on the degradation of FTOH 8:2 in rat, mouse, trout, human hepatocytes, human liver microsomes and cytosol suggests that FTOH 8:2 in humans is converted only slightly, and that FTOH 8:2 is not a significant source of the formation of PFOA or other PFCAs (Nabb et al., 2007).

Microbial degradation of the **polyfluorinated alkyl phosphates** (PAPs) can occur by hydrolysis of the phospho-ester bond to form the respective FTOH as a by-product, which may then be converted according to further transformation processes (Lee et al., 2010). Short-chain PAPs were fully converted within ten days, but complete transformation of 2-mono-PAP after 90 days was not observed. PAPs can also be bio-transformed in higher organisms as demonstrated by experimental results with rats (D'Eon and Mabury, 2007).

To study the degradation of **industrial polymers**, a synthetic fluoroacrylate polymer was synthesized with different FTOH side chain lengths and incubated aerobically in soil over a period of two years (Russel et al., 2008). Terminal biotransformation by-products detected included PFOA, PFNA, PFDA, and PFUNa. However, a biodegradation half-life of 1.200 to 1.700 years was determined for these biotransformations. Thus it is concluded that microbial degradation of fluoroacrylate polymers hardly plays a role in the fate and transport of these compounds in the natural environment.

Biotic transformations of PFAS can be associated with substantial changes in the physicochemical properties of the compounds.

#### Chemical Transformations

PFCAs and PFSAs have shown to be very persistent in the environment (Wang et al., 2015). One study of Taniyasu et al. (2013) provided the first experimental evidence from field studies (at altitudes more than 2.500 m) that PFAS including PFOS can undergo photolysis. Taniyasu et al. (2013) states: "Long chain PFAS (PFCAs, PFSAs, FTOHs) can be successively dealkylated to short chain compounds such as perfluorobutanoic acid (PFBA) and perfluorobutane sulfonate (PFBS), but the short chain compounds were relatively more resistant to photodegradation". However, Wang et al. (2015) clearly doubt these results looking at the lack of information provided in the research.

Prior to the above mentioned study, photolysis was already investigated by many scientists (e.g. Chen et al., 2006, Hori et al., 2007, Giri et al., 2011), demonstrating photolysis of PFCAs. These studies were mostly performed with relatively high concentrations of PFAS and partly under extreme reaction conditions (e.g. under pressure, in combination with photochemical oxidants), not representing natural environmental conditions. No other studies were found that showed photolytic degradation of PFOS and PFOA under natural circumstances.

Regarding chemical degradation of **precursors**, volatile compounds such as FTOHs may react in the atmosphere and be oxidized by chlorine atoms, oxygen molecules, or photochemically generated OH radicals (Houtz et al., 2012). These authors concluded that photo-oxidation of FTOHs with chlorine atoms mainly produces by-products including fluorotelomer carboxylic acids (FTCAs), fluorotelomer aldehydes (FTALs), perfluoraldehyde (PFAL), carbonyl, PFOA and PFNA. It was also concluded that photo-oxidation of FTOH with hydroxyl radicals leads to the production of FTAL, PFAL and carbonyl. Abiotic transformations of PFAS can also be associated with substantial changes in the physicochemical properties of the compounds.

## 4. TOXICITY

The available data on PFAS toxicity is dominated by PFOS, PFOA and also PFHxS due to the widespread detection of these compounds in humans and the environment, and concern that these could biomagnify to a level whereby humans consuming fish may be adversely affected. Much less data is available on the toxicology of other PFAS, and this is often inconsistent and fragmentary. For the less investigated polyfluorinated chemicals, toxicology is often estimated based on structure-activity relationships, or structural homologues.

Human exposure to PFAS is mainly by ingestion of contaminated food or water. These compounds are not metabolised, bind to proteins (not to fats) and are mainly detected in blood, liver and kidneys. Elimination of PFOS, PFHxS and PFOA from the human body takes some years, whereas elimination of shorter chain PFAS is in the range of days. The half life of PFOS and PFOA in rodents is in the range of months which can cause extrapolation issues in tests.

There is significant data available on the impact of (sub)chronic PFOS and PFOA exposure on reproductive and/or developmental and other types of effects in both humans and animals. However, the results from epidemiological studies are not always consistent. Animal studies show mainly effects from PFOS and PFOA on the liver, the gastrointestinal tract and on thyroid hormone levels. In general, PFOS is more toxic compared to PFOA.

In 2008, the European Food Safety Authority derived a TDI (Tolerable Daily Intake) for PFOS of 150 ng/kg bw/day and for PFOA of 1.500 ng/kg bw/day. Later, taking into account more recent toxicity data, the U.S. EPA has proposed much lower RfDs (Reference Doses) of 30 ng/kg bw/day for PFOS and 20 ng/kg bw/day for PFOA (2014, draft).

Carcinogenic effects of PFOS and PFOA have also been studied (human and animal studies, no focus on other PFAS). Several authorities, including ATSDR, U.S. EPA and IARC do not classify PFOS and PFOA as “proven carcinogens”, but instead as “suggestive carcinogens” or “possibly carcinogenic to humans” because of existing uncertainties.

PFOS has been categorised as moderately acute and slightly chronically toxic to aquatic organisms. The MAC EQS derived by the European Commission for European freshwater and saltwater are based on the lowest NOEC reported (NOEC of < 2,3 µg/l for *Chironomus tentans*) to protect the most sensitive species.

The Sections below provide more detailed information about the exposure, toxicity and the bioaccumulation potential of PFOS and PFOA (Section 4.1 to 4.3). Information of other PFAS is included in Section 4.4.

### 4.1. UPTAKE, DISTRIBUTION IN TISSUE, BIOACCUMULATION AND ELIMINATION OF PFOS AND PFOA

#### 4.1.1. Uptake

Due to the physicochemical characteristics of perfluorinated compounds, exposure of PFAS is most likely via ingestion of contaminated food or water (dietary uptake/oral route) (Fromme et al., 2009, ATSDR, 2009). As PFAS have also been found in both

air and dust, exposure by breathing air, ingestion of dust, or dermal contact with dusts or aerosols of PFAS may also be a source of exposure (ATSDR, 2009).

Compared to data on ingestion, relatively little data are available on other paths of exposure, such as skin contact with PFAS-treated utensils or inhalation of indoor air (Stahl et al., 2011). The significance of these exposure pathways is unclear. ATSDR (2009) concluded that carpets treated with perfluoroalkyls can be a source of exposure for children.

#### **4.1.2. Distribution in tissue**

Perfluoroalkylated substances such as PFOS and PFOA have, contrary to most other persistent organic pollutants (POPs), a low affinity to lipids, but bind to proteins. PFOS is associated with cell membrane surfaces and accumulates in various, mainly high perfused, body tissues of exposed organisms (DEPA, 2013).

The highest concentrations are usually detected in blood, liver, kidneys, lung, spleen and bone marrow. Lower concentrations are detected in heart, testes, fat, brain and muscles. In the general public, PFOS concentrations in the blood range between sub-ppb levels up to the hundred ppb level. PFOA levels in blood are generally lower (sub-ppb levels up to tens of ppb levels, Loganathan et al., 2011). Although accumulation of PFAS in muscles is minimal (DEPA, 2013), accumulation in muscles may be an important exposure route when consuming fish and meat. Stahl et al. (2012) analysed PFOS and PFOA concentrations in liver and muscle tissue of wild boar to evaluate the potential health danger resulting from consumption of wild boar meat or liver. Both PFOS and PFOA were detected in liver and muscle tissue, whereas concentrations of PFOS were significantly higher in organs and tissues. Considering the TDI (see Section 4.2.2) for PFOS and PFOA, negative health effects from consumption of wild boar are not expected (Stahl et al., 2012). The very low Annual Average-Environmental Quality Standard EQS (see Section 5) however is based upon consumption of fish by humans.

Both in animals and humans, PFOS and PFOA cross the placenta, and are also excreted in breast milk (Stahl et al., 2011).

An unequivocal correlation between age and blood-PFAS concentrations is not evident. However, gender-dependent differences are as follows: men generally show higher concentrations of PFAS than women (Rylander et al., 2009). This gender related difference in concentration levels was also detected during other studies, such as the study of Calafat et al (2007), based on data of the U.S. population.

Neither PFOS nor PFOA are metabolized to any significant extent (Stahl et al., 2011).

#### **4.1.3. Bioaccumulation**

Conder et al. (2008) concluded that: “(1) bioconcentration and bioaccumulation of perfluorinated acids is directly related to the length of each compound’s fluorinated carbon chain; (2) PFASs are more bioaccumulative than PFCAs of the same fluorinated carbon chain length”.

The numerical criterion under REACH defining that a substance is bioaccumulative is a bioconcentration factor (BCF) in aquatic species higher than 2000 l/kg. (Commission Regulation (EU) No 253/2011). Bioconcentration factors > 1 l/kg indicate bioaccumulative potential only from a scientific standpoint.

Information about bioconcentration, bioaccumulation and biomagnification for PFOS and PFOA is presented below.

Overall, it should be noted that bioaccumulation can differ significantly between aquatic and terrestrial organisms. As PFASs and PFCAs are generally highly water soluble and have a low vapour pressure (Section 3), the efficiencies of biological depuration mechanisms (i.e. lungs vs. gill) and thus the values for bioaccumulation differ (PFOS depuration from fish is relatively rapid). As a consequence, studies may indicate a tendency for bioaccumulation based on data from terrestrial organisms while data from aquatic organisms may not be as conclusive, or even clearly indicate a lack of meaningful bioaccumulation (e.g., aquatic BAFs may be less than 2.000).

PFOS:

A selection of bioconcentration (BCF), bioaccumulation (BAF) and biomagnification (BMF) factors for PFOS is presented in **Table 4.1**<sup>2</sup>.

**Table 4.1:** A selection of BCFs, BAFs and BMFs for PFOS

<b>Bioconcentration Factor</b>		
Ratio between the chemical concentration in an organism to the concentration in water (exclusion of dietary intake)		
Bluegill	1.866 – 4.312	Drottar et al., 2001
Rainbow Trout	1.100 – 5.400	Drottar et al., 2001
Catfish and largemouth bass (Decatur, Alabama)	830 – 26.000	Giesy and Newsted, 2001
Rainbow Trout	2.900 (liver) 3.100 (blood)	Martin et al., 2003
<b>Bioaccumulation Factor</b> (within a trophic level)		
Increase of a chemical concentration in certain tissues of an organism due to absorption from food/environment		
Zooplankton/water	240	Houde et al., 2008
Mysis/water	1.200	Houde et al., 2008
Sculpin/water	95.000	Houde et al., 2008
Lake trout/water	16.000	Houde et al., 2008
<b>Biomagnification Factor</b> (across trophic levels)		
Increase of a chemical concentration in an organism compared to the chemical concentration in its diet		
Arctic cod/zooplankton (Western Canadian Arctic)	8,7	Powley et al., 2008
Caribou/lichen (Canada)	2,0 – 9,1	Müller et al., 2011
Wolf/caribou (Canada)	0,8 – 4,5	Müller et al., 2011
Dolphin/seatrout (2 U.S. locations)	0,9	Houde et al., 2006
Seatrout/pinfish (2 U.S. locations)	4,6	Houde et al., 2006

<sup>2</sup> In case that more information on bioaccumulation of PFOS is desired, following publications (a not limitative list) can be considered for review: Asher et al., 2012, Awad et al., 2011, De Silva et al., 2011, De Solla et al., 2012, Inoue et al., 2012, Jeon et al., 2010, Kwadijk et al., 2010, Labadie et al., 2011, Liu et al., 2011, Pan et al., 2014, Sakurai et al., 2013. Many of these publications also contain information on the bioaccumulation potential of PFOA and other PFAS.

Walrus/clam (Eastern Arctic Food Web)	4,6	Tomy et al., 2004
Narwhal/Arctic cod (Eastern Arctic Food Web)	7,2	Tomy et al., 2004
Beluga/Arctic cod (Eastern Arctic Food Web)	8,4	Tomy et al., 2004
Beluga/redfish (Eastern Arctic Food Web)	4,0	Tomy et al., 2004
Polar bear/seal (Canadian Arctic)	177	Martin et al., 2004

Note: due to the continuous improvements of the analytical methods for PFAS, it could be difficult to compare recent with older analytical results. Studies performed before 2007 may have considerable analytical inaccuracies and should be viewed in that light.

The data in **Table 4.1** show bioconcentration factors (BCF) > 2.000 l/kg, demonstrating the bioaccumulation properties of PFOS.

The BMF in **Table 4.1** highlight that predatory animals are recorded with greater concentrations in their bodies compared to the concentrations in their diets, demonstrating the biomagnification properties of PFOS. As a result, concentrations of PFOS are likely to be elevated within organisms at higher trophic levels.

In general, the bioaccumulation potential in the soil environment has been shown to be significantly lower than in the marine environment (DEPA, 2013).

PFOA:

A selection of bioconcentration, bioaccumulation and biomagnification factors for PFOA is presented in **Table 4.2**.

**Table 4.2:** A selection of BCFs, BAFs and BMFs for PFOA

<b>Bioconcentration Factor</b>		
Ratio between the chemical concentration in an organism to the concentration in water (exclusion of dietary intake)		
Water breathing animals	1,8 – 8,0	ECHA, 2014
Rainbow Trout	12 (liver) 25 (blood)	Martin et al., 2003
<b>Bioaccumulation Factor</b> (within a trophic level)		
Increase of a chemical concentration in certain tissues of an organism due to absorption from food/environment (e.g. water and food)		
Water breathing animals	0,9 – 266	ECHA, 2014
<b>Biomagnification Factor</b> (across trophic levels)		
Increase of a chemical concentration in an organism compared to the chemical concentration in its diet		
Water breathing animals	0,02 – 7,2 (most data below 1)	ECHA, 2014
Caribou/lichen (Canada)	0,9 – 11	Müller et al., 2011
Wolf/caribou (Canada)	0,9 – 3,8	Müller et al., 2011
Walrus/clam (Eastern Arctic Food Web)	1,8	Tomy et al., 2004
Narwhal/Arctic cod (Eastern Arctic Food Web)	1,6	Tomy et al., 2004

Beluga/Arctic cod (Eastern Arctic Food Web)	2,7	Tomy et al., 2004
Beluga/redfish (Eastern Arctic Food Web)	0,8	Tomy et al., 2004
Beluga whale/Pacific herring (Western Canadian Arctic Food Web)	1,3	Tomy et al., 2009
Arctic cod/marine arctic copepod (Western Canadian Arctic Food Web)	2,2	Tomy et al., 2009
Dolphin/seatrout (2 U.S. locations)	1,8	Houde et al., 2006
Seatrout/pinfish (2 U.S. locations)	7,2	Houde et al., 2006
Polar bears/ringed seal (2 U.S. locations)	45 – 125	Butt et al., 2008
Polar bear/seal (Canadian Arctic)	8,6	Martin et al., 2004

Note: due to the continuous improvements of the analytical methods for PFAS, it could be difficult to compare recent with older analytical results. Studies performed before 2007 may have considerable analytical inaccuracies and should be viewed in that light.

The results in **Table 4.2** show that the reported BCFs for PFOA are far below 2.000 l/kg. Also BAFs are well below 2.000. These data show that based on the REACH definition for “bioaccumulation”, this criterion is not met for PFOA. In Annex XV “Proposal for a Restriction of PFOA” (ECHA, 2014), it is concluded that the bioaccumulation criterion defined in the REACH regulation cannot be used to assess the bioaccumulation potential of PFOA. However, due to the long half-live times in humans and BMFs > 1, there is evidence for bioaccumulation of PFOA.

The revised Annex XIII of the REACH regulation (March 2011) was expanded with criteria for assessing the bioaccumulation potential: results regarding biomagnification, bioaccumulation in terrestrial species and concentrations in human body fluids could also be considered in the evaluation of the “bioaccumulation” criterion.

The Proposal Document for a restriction of PFOA (ECHA, 2014) concludes the following: “The bioaccumulative property is proven by studies from aquatic and terrestrial food webs, which clearly indicate accumulation of PFOA and APFO. In addition, human data strongly indicate that PFOA and APFO bioaccumulate in humans. It is of special concern that PFOA and APFO biomagnify in endangered species as shown for the polar bear and in animals which are likely to become endangered in the near future (narwhal and beluga whale). Additionally, human gestational and lactational exposure are of special concern as the foetus and newborn babies are highly vulnerable to exposure to toxic substances. Based on a weight of evidence approach, it is considered that the data from environmental species and humans shows that the B criterion of REACH Annex XIII is fulfilled”.

#### 4.1.4. Elimination

Both PFOS and PFOA are very slowly eliminated from the human body. The Toxicological Overview for PFOS and PFOA, published by the Public Health England (2009), documents a half life<sup>3</sup> from the human body of approximately 9 years for PFOS and 4 years for PFOA. Some data about half lives for PFOA and PFOS are summarized in **Table 4.3**.

**Table 4.3:** Half Life Times for PFOS and PFOA

<b>PFOS</b>		
Cynomolgus monkeys	132 days (males) 110 days (females)	Noker and Gorman, 2003
Cynomolgus monkeys (male and female)	200 days	Seacat et al., 2002
Rodents	1 – 2 months	Chang et al., 2012
Monkeys	4 months	Chang et al., 2012
Retired fluorochemical workers (U.S.A)	5,4 years	Olsen et al., 2007
<b>PFOA</b>		
Rats	5,63 days (males) 0,08 days (females)	Ohmori et al., 2003
Cynomolgus monkeys	33 days (males) 21 days (females)	Butenhoff et al., 2004
Retired fluorochemical workers (U.S.A)	2,3 – 3,8 years	Olsen et al., 2007
Population study (U.S.A)	2,9 – 8,5 years	Seals et al., 2011
Population study (U.S.A)	2,3 years	Bartell et al., 2010

In fluorochemical workers, PFHxS had the longest observed elimination half-life (8,5 years), followed by PFOS (5,4 years), and PFOA (2,3-3,8 years) (Olsen et al. 2007). Based on the studies listed above, the excretion of PFAS varies with the type of perfluorochemicals and also with the animal species and gender. The reason for the species and gender differences in elimination are not well understood (U.S. EPA, 2009).

In general, the blood half-lives of perfluorochemicals:

- are longer for sulfonates than for carboxylates;
- are shorter for branched isomers than straight chain;
- are often shorter in females than males. This may be due to the difference in renal clearance (and hormones) (DEPA, 2013). Sex differences documented for rats and monkeys are not always found in humans (DEPA, 2013);
- increase with chain length for carboxylates;
- vary a lot between species.

The primary clearance route for PFOS and PFOA is urine, rather than faecal elimination (Bull et al., 2014).

<sup>3</sup> Half life: the time required for a concentration to decrease by half compared to its initial concentration

## 4.2. HUMAN TOXICOLOGY OF PFOS AND PFOA

### 4.2.1. Health effects of acute exposure

The acute lethal toxicity of PFOS moderately corresponds to a classification as acute toxicity Category 4. In general, PFOS is more toxic compared to PFOA (DEPA, 2013).

Some data on acute toxicity of PFOS and PFOA are summarized in **Table 4.4**.

**Table 4.4:** A selection of acute toxicity data of PFOS and PFOA

<b>PFOS</b>				
<b>Inhalation</b>				
Rats		1,9 – 4,6 mg/l, 1 hour (PFOS dust in air)	Symptoms: Signs of emaciation Nasal discharge Stained urogenital region Breathing disturbances General poor condition	OECD, 2002
	LC50	5,2 mg/l (PFOS dust in air)		OECD, 2002
<b>Ingestion</b>				
Rat	Oral LD50	250 mg/kg bw	Symptoms: hypoactivity, stained urogenital region, decreased limb tone and ataxia, stomach distension, lung congestion	3M, 1999
Newborn mouse	Oral LD50	10 mg/kg bw/d		Lau et al., 2004
Rat	Oral LD50	Between 50 – 1500 mg/kg bw		OECD, 2002
<b>Dermal Exposure</b>				
No accurate data available. The only available dermal study is from Biesemeier and Harris (1974) (no detailed information available in this study)				
<b>PFOA</b>				
<b>Inhalation</b>				
No data about effects of acute exposure to humans and animals.				

Ingestion				
Rats	LD50	430 – 680 mg/kg bw	Symptoms: enlarged livers, gastrointestinal irritation, weight loss	PHE, 2009
Guinea Pig	LD50	200 mg/kg bw		PHE, 2009
Dermal Exposure				
New Zealand White rabbits	Dermal LD50	> 2000 mg/kg bw		Glaza, 1995
Rabbits	Dermal LD50	4300 mg/kg bw		Kennedy, 1985
Rats	Dermal LD50	7000 mg/kg bw (male) 7500 mg/kg bw (female)		Kennedy, 1985

LC: Lethal Concentration  
LD: Lethal Dosis

There are no data to assess the acute toxicity following high exposure by means of inhalation, ingestion, dermal or ocular contact in humans (PHE, 2009). Also the extensive literature search by Bull et al. (2014) did not identify data on the acute toxicity of PFOS and PFOA.

Public Health England (2009) states: “Animal data suggest that PFOS and PFOA have moderate acute oral toxicity with effects on the gastrointestinal tract and liver. Animal data suggest that they are mild skin and eye irritants”.

#### 4.2.2. Health effects of (sub)chronic exposure

There is much data on the impact of (sub)chronic PFOS and PFOA exposure on reproductive and/or developmental and other types of effects in both humans and animals.

##### Epidemiological studies (humans)

During the past few years, several epidemiological studies were conducted to investigate relations between PFOS/PFOA exposure and various health effects like fertility, growth, and developmental biomarkers (e.g. studies from workers at different 3M plants, population studies of residents from Ohio, West Virginia, Quebec, among others). Several of the human epidemiological studies have recently reported associations with PFOS and cholesterol, birth weight changes and various thyroid parameters. However, these studies show inconsistent results. Therefore, the U.S. EPA’s Science Advisory Board notes: “The results of existing epidemiology studies are not adequate for use in quantitative risk assessment” (U.S. EPA, 2014).

##### Animal Studies

Several studies have been carried out to examine chronic exposure<sup>4</sup> on animals, with focus on mice, rats and monkeys. The following toxic effects could be seen, following chronic exposure (PHE, 2009):

- Effects on the liver as primary target organ (Increase of the liver weight, liver cell hypertrophy)

<sup>4</sup> Chronic exposure experiments are long-term experiments in contrast to acute toxicity tests. Co-effecting factors may be influencing the results, i.e. lower stress-tolerance as compared to the reference animal

- Effects on the gastrointestinal tract
- Effects on thyroid hormone levels
- Body weight loss
- Effects on the lipid metabolism (Stahl et al., 2011)
- Reproductive and developmental toxic effects (e.g. reduction of foetal weight, oedema, delayed ossification of bones, cardiac abnormalities)

Some of the reported no-observed-effect-concentration (NOEC) and lowest-observed-adverse-effect-levels (LOAEL) are summarized in **Table 4.5**.

**Table 4.5:** Health effects of (sub)chronic exposure: NOEC and LOAEL for PFOS and PFOA exposure

<b>PFOS</b>				
Rats	Oral Diet, 14 weeks	NOEC: 0,4 mg/kg bw/d	Liver Effects	Seacat et al., 2003
Rats	Oral Gavage	NOEC: 1 mg/kg bw/d	Developmental Effects	Lau et al., 2003
Rats	Oral Diet, 90 days	LOAEL: 2 mg/kg bw/d	Liver Effects	Goldenthal, 1978
Rats	Oral gavage, 28 days	LOAEL: 5 mg/kg bw/d	Decrease in body weight	Cui et al., 2009
Rats	Oral gavage, 20 days	NOEC: 1,0 mg/kg bw/d	Maternal toxicity	Butenhoff et al., 2009
Rabbits	Oral gavage	NOEC: 0,1 mg/kg bw/day (maternal) NOEC: 1 mg/kg bw/day (foetal) LOAEL: 1 mg/kg bw/day (maternal) NOEC: 2,5 mg/kg bw/day (foetal)	Developmental maternal and foetal toxicity	Case et al., 2001
Cynomolgus Monkey	Oral Diet, 6 months	NOEC: 0,03 mg/kg bw/d LOAEL: 0,15 mg/kg bw/d	Effect on Thyroid hormone values	Seacat et al, 2002
<b>PFOA</b>				
Mice	Oral Gavage, 14 days	LOAEL: 0,3 mg/kg bw/day	Liver Weight	Loveless et al., 2006
Rats	Oral Gavage, 14 days	LOAEL :1 mg/kg bw/day NOEC: 0,3 mg/kg bw/day	Effect on hormone values	Loveless et al., 2006

Rats	Oral Diet, 14 days	LOAEL: 1,7 mg/kg bw/day (male) LOAEL: 76 mg/kg bw/day (female) NOEC: 0,6 mg/kg bw/day (male) NOEC: 22 mg/kg bw/day (female)	Liver Effects	Goldenthal, 1978
Rats	Oral Diet, 90 days	LOAEL: 0,6 mg/kg bw/day NOEC: 0,06 mg/kg bw/day	Liver Effects	Perkins et al., 2004
Mice	Oral Gavage	LOAEL: 1 mg/kg bw/day (maternal) LOAEL: 3 mg/kg bw/day (foetal) NOEC: 1 mg/kg bw/day (foetal)	Developmental Effects	Lau et al., 2006
Rats	Oral Gavage (Two generation study)	LOAEL: 1 mg/kg bw/day (F0, paternal) LOAEL: 1 mg/kg bw/day (F1, foetal) NOEC: > 30 mg/kg bw /day (F0, maternal)	Reproductive Effects	Butenhoff et al., 2004

Derivation of Reference Doses (RfDs<sup>5</sup>) / Tolerable Daily Intakes (TDIs)

**U.S. EPA**

In October 2009, the U.S. EPA issued provisional subchronic Reference Doses (RfDs) for PFOS and PFOA (U.S. EPA, 2009). The subchronic RfD for PFOS was 800 ng/kg bw/day and the subchronic RfD for PFOA was 200 ng/kg bw/day. The PFOS RfD was based on increases in liver weight in mice (Lau, et al., 2006), and the PFOA RfD was based on increased levels of thyroid stimulating hormone, reduced triiodothyronine, and reduced high density lipoproteins in monkeys (Seacat, et al., 2002).

In February 2014, the U.S. EPA released Draft Health Effects Documents for PFOS (U.S. EPA, 2014a) and PFOA (U.S. EPA, 2014b) which proposed chronic RfDs for these compounds of 30 ng/kg bw/day and 20 ng/kg bw/day, respectively.

For PFOS, the proposed RfD is based on a rat developmental neurotoxicity study by Butenhoff et al. (2009) that found increased motor activity and decreased habituation

<sup>5</sup> A Reference Dose (RfD) is the maximum amount of a substance that can be ingested daily over a lifetime without causing adverse non-cancer health effects

on Post Natal Day 17 in male offspring following a maternal dose of 1 mg/kg/day. No effects on pup body weight were reported. The selected proposed PFOS RfD is based on a pharmacokinetic approach that models human serum levels associated with developmental neurotoxicity in rat (Butenhoff et al. 2009) and is supported by the slightly higher 50 and 60 ng/kg bw/day RfD values for increases in liver weight and other developmental effects. Thus, co-occurring critical endpoints are protected by the chosen PFOS RfD.

For PFOA, the proposed RfD is based on modelled serum values from four different points of departure doses based on two rat studies (Palazzolo et al., 1993, York et al., 2002) and one mouse study (Lau et al., 2006) that showed consistent responses across studies. Reduced liver weight was used as a common denominator for loss of homeostasis and protection against co-occurring adverse developmental or kidney effects observed in two of the studies (York et al., 2002, Lau et al., 2006).

These proposed RfDs were subjected to peer review by independent scientists in August of 2014. The peer reviewers questioned the U.S. EPA's rationale for choosing reduced liver weight as the basis for the RfD for PFOA, and they requested further justification for the use of animal data as the basis for the RfD when human data are currently available. The proposed chronic RfD values will not be added to the U.S. EPA IRIS database until the Health Effects Documents are finalized.

### ***Europe***

European Food Safety Authority (2008): The Scientific Panel on Contaminants in the Food Chain (CONTAM) established a TDI for PFOS of 150 ng/kg bw/day. This TDI was based on the NOEC of 0,03 mg/kg bw/day from a subchronic study with Cynomolgus monkeys (Seacat et al., 2002. See **Table 4.5**). The TDI for PFOA of 1500 ng/kg bw/day was linked with the two-generation reproductive study with rats by Butenhoff et al. (2004, see **Table 4.5**).

## **4.2.3. Carcinogenic effects**

### ***Human studies***

The cancer incidence related to PFOS and PFOA exposure in worker-based populations was studied in several studies (e.g. at several 3M plants in U.S.A and Europe, DuPont's Washington Works Plant). In most cases, these human epidemiological studies could not find a direct correlation between the PFOS exposure and carcinogenicity, mainly due to the lack of information on other types of exposure (e.g. lifestyle information, influence from the use of other chemicals at the plants). Only in the DuPont's study (West Virginia Washington Works Plant, 2003) was a significant increase observed for cancer of kidney, bladder and urinary track organs, due to exposure to PFOA.

Studies within the general population (without occupational exposure to PFAS) did not reveal any direct correlation between PFOS/PFOA exposure and carcinogenicity (U.S. EPA, 2014a).

### ***Animal studies - PFOS***

Thomford et al. (2002) performed a study on carcinogenicity in which male and female rats were administered different concentrations of PFOS over a period of 104 weeks. A significant positive correlation was detected between PFOS exposure and the incidence of hepatocellular adenoma (liver) in male and female rats.

A comparable study was performed by Butenhoff et al. in 2012. Also in this study a significant increase in hepatocellular adenoma was observed in males and females. It was only in the female, 20 ppm dose group that a hepatocellular carcinoma was observed. There were no significant effects on kidney or bladder.

It has not been determined whether these results can also be extrapolated to humans.

#### Animal studies - PFOA

The studies of Butenhoff et al. (2012) and Biegel et al. (2001), both with rats, showed that PFOA exposure was correlated with liver adenomas or carcinomas, testicular Leydig cell adenomas and pancreatic acinar cell tumors (the latter, only showed in Biegel et al., 2001). In addition, ovarian tubular hyperplasia and adenomas were observed in the female rats in the Butenhoff et al. study (2012). In both studies, effects were detected in the 20 mg/kg/day-dose-group. Only the Leydig cell adenomas demonstrated a dose-response relationship.

There are no carcinogenicity studies using other animals than rats.

#### General conclusions on carcinogenicity

In regards to carcinogenesis, Stahl et al. (2011) concludes: "a genotoxic mechanism cannot be assumed for PFOS and PFOA, but rather a tumour promoting effect and/or epigenetic process comes into question".

ATSDR (2009) states: "The information available does not prove that perfluoroalkyls cause cancer in humans, but the evidence is not conclusive".

The U.S. EPA concludes that evidence of carcinogenicity of PFOS is "suggestive", but not definitive, because the tumour incidence does not indicate a dose response (U.S. EPA, 2014a). Based on the risk assessment study performed in 2005 (U.S. EPA, 2005), PFOA's carcinogenicity was also categorized as "suggestive". In the U.S. EPA 2014b study, a Human Equivalent Dose (HED) of 0,58 mg/kg bw/day and a slope factor of  $0,07 \text{ (mg/kg bw/day)}^{-1}$  was calculated (the basis for this calculation was the dose-response data of the Leydig cell tumours in rats, Butenhoff et al., 2012).

In June 2014, the International Agency for Research on Cancer (IARC), as part of the World Health Organization, assessed the carcinogenicity of PFOA. PFOA was classified as follows: "possibly carcinogenic to humans (Group B), based on limited evidence in humans that exposure to PFOA is associated with testes and kidney cancer and limited evidence in experimental animals" (IARC, 2014). Currently, PFOS is not yet classified by IARC.

### **4.3. TOXICITY OF PFOS AND PFOA TO ECOLOGICAL RECEPTORS**

Ecotoxicity data were primarily identified for aquatic organisms such as algae, aquatic plants, invertebrates and fish, and birds. Ecotoxicity tests of PFAS are mostly limited to PFOS and PFOA, and the dataset is small in comparison to established pollutants, but also to many other emerging chemicals of concern (Funkhouser, 2014).

#### PFOS

A good overview of PFOS' key acute and chronic aquatic ecotoxicological tests was provided in the "PFOS EQS Dossier" (2011), prepared for the revision of the Environmental Quality Standards Directive" (Directive 2013/39/EU), a daughter Directive of the Water Framework Directive (WFD), and it is shown in the tables in **Appendix 3**.

Based on this information, the EC<sub>50</sub> for freshwater algae and aquatic plants (acute tests/96h) ranges between 48 and 283 mg PFOS/l. The EC<sub>50</sub> for freshwater invertebrates (acute tests/48h) ranges between 4 and 124 mg PFOS/l. The NOEC for freshwater invertebrates ranges between < 0,002 and 12 mg PFOS/l. The differences in the measured EC and NOEC values are species dependent (for more information, see **Appendix 3**).

The following general conclusions can be derived from the PFOS aquatic ecotoxicological studies:

- Based on laboratory toxicity studies, PFOS can be generally categorized as “moderately acute and slightly chronically toxic to aquatic organisms” (Giesy et al., 2010);
- The most sensitive genus to PFOS exposure is the invertebrate (midge) *Chironomus tentans*. This genus is approximately 40-fold more sensitive compared to the next most sensitive genus (*Pimephalus*) (Giesy et al., 2010);
- Acute invertebrate toxicity data show that marine invertebrates are more sensitive to short-term PFOS exposure than freshwater invertebrates (Giesy et al., 2010).

Funkhouser (2014) states: “One considerable uncertainty with regard to PFOS ecotoxicity is a general lack of longer-term exposure studies. As an example, the vast majority of studies on PFOS toxicity to aquatic invertebrates have been less than a generation of particular study organisms and overall, less than 28 days. Because many PFAS and especially PFOS are persistent, longer-term exposures may occur in the environment”.

The MAC EQS derived by the European Commission for European freshwater and saltwater are based on the lowest NOEC reported (NOEC of < 0,0023 mg/l for *Chironomus tentans*) to protect the most sensitive species. The derived EQS are described in Section 5.2.

### PFOA

Following general conclusions can be derived from the PFOA aquatic ecotoxicological studies:

- Acute toxicity testing with aquatic species indicates that PFOA is generally less toxic than PFOS. There is a difference of about a factor 10 (DEPA, 2013). As an example, these effects were clearly shown in a marine species study with three different trophic levels, conducted by Mhadhbi et al. (2012);
- The most sensitive pelagic organism is *Pseudokirchneriella subcapitata* (a freshwater alga), with a 96-hour LOEC of 2,0 mg/l (Environment Canada, 2012);
- There are studies in aquatic organisms showing potential of PFOA to affect endocrine function. In minnows at PFOA concentrations of 3-30 mg/l, thyroid hormone biosynthesis was inhibited, vitellogenin expression was induced in males, oocytes developed in the testes of male fish, and ovary degeneration occurred in females. Other studies show hepatotoxicity, immunotoxicity and chemosensitivity in other different organisms such as mussels, seals, dolphins, turtles and rats (Environment Canada, 2012, cited from DEPA, 2013);
- PFOA exhibits low chronic toxicities in benthic organisms (> 100 mg/l) (Environment Canada, 2012).

A study with white leghorn chickens showed that PFOA had no effect on embryonic pipping success at concentrations up to 10 µg/g of embryos. However, there was a significant accumulation of PFOA in the liver of the embryos, compared to the initial whole-egg concentration (Environment Canada, 2012).

Currently, there is no EQS derived for PFOA by the European Commission.

#### Ecotoxicological effects to higher trophic level wildlife

Due to the multiple global sources of PFOS and PFOA and the persistency of these compounds (and therefore the wide-scale fate and transport pathways), both compounds are detected across the globe, even in remote places. Concentrations are detected in a variety of wildlife, such as seals, walrus, polar bears, dolphins, eagles, amongst others in all continents. PFOA concentrations in the liver of Canadian polar bears are about 13 µg/kg bw (Environment Canada, 2012). PFOA concentrations increase yearly by 2,3% in central East Greenland polar bears. In adult female sea otters, concentrations increased significantly over a 10-year period (Environment Canada, 2012).

Information about the accumulation and biomagnification potential of PFOS and PFOA is included in Section 4.1.3.

#### **4.4. TOXICITY, HALF LIFE TIMES AND BIOACCUMULATION POTENTIAL OF OTHER PFAS**

As mentioned previously, the most detailed studies of toxic and adverse health effects have been carried out for PFOS and PFOA. These two compounds, alongside PFHxS, are the compounds which are usually detected at the highest concentrations in human matrices (U.S. EPA, 2009). However, their use is currently being phased out and shorter-chain compounds are increasingly being used as replacements.

The data presently available regarding the toxicology of PFAS other than PFOS and PFOA is in comparison meagre, inconsistent, and fragmentary, particularly in light of the diversity of PFAS found in biological matrices. However, data for fluorotelomers and shorter chain homologues continue to be published. For the less investigated polyfluorinated chemicals, preliminary properties may be estimated based on their structure or from homologues.

A recent study of the Danish Environmental Protection Agency (DEPA, 2015c) describes the human toxicity of short-chain PFAS as follows: "The toxicokinetics and toxicity in humans for short-chain PFAS are mainly investigated for PFHxS, and that substance has rather similar properties as PFOS" and further "The other short-chain PFAS seem to be less toxic than PFOS/PFOA but the available data is insufficient for a final evaluation".

Another good overview of the toxicity of various long- and short- chain PFAS is included in the extensive literature review of Bull et al., 2014.

#### Short-chain PFAS

- Generally no or lower bioaccumulation potential in comparison to PFOS and PFOA although there may be some exceptions. The BCFs of PFBS and PFBA are about a factor 3 lower compared to the BCFs of PFOS and PFOA, respectively (based on modelling exercises) (Rayne et al., 2009). On the other hand, Lasier et al. (2011) states that "sulfonates with four to seven carbons may be as likely to

bioaccumulate as PFOS". In addition, it is difficult to extrapolate bioaccumulation data from animal studies to humans, as stated by DEPA (2015c) as follows: "The high presence of short-chain PFAS, especially PFBA, in human tissue including brain from deceased people is worrying, and it shows that the short-chain PFAS and a fluortelomer metabolite may be much more bioaccumulative in humans than the studies with experimental animals conclude".

- Persistent
- No data on carcinogenity for PFBA, PFHxA, PFBS, PFHxS
- Summary of information for the most common short-chain PFAS:
  - o PFBA
    - Half-life in fluorochemical workers: 1,2 – 4,6 days (Chang et al., 2008)
    - Half-life in retired fluorochemical workers: 1,9 – 6,3 days (Chang et al., 2008)
    - Half-life in male monkeys: 40,3 hours (Chang et al., 2008)
    - Half-life in female monkeys: 41,0 hours (Chang et al., 2008)
    - Urine is the main route of elimination of PFBA (Chang et al., 2008)
    - General low level of toxicity (Rickard, 2009)
  - o PFHxA
    - Half-life in male monkeys: 5 hours (Gannon et al., 2011)
    - Half-life in female monkeys: 2 hours (Gannon et al., 2011)
    - Half-life in rats: 2,5 hours, after oral dosing and 1 hour after in vitro administration (Gannon et al., 2011)
    - Urine is the main route of elimination of PFHxA (Gannon et al., 2011)
    - NOEC for subchronic toxicity: 20 mg/kg bw/day (rats) (Rickard, 2009)
    - NOEC for reproductive toxicity: 500 mg/kg bw/day (rats) (Rickard, 2009)
    - NOEC for developmental toxicity: 100 mg/kg bw/day (rats) (Rickard, 2009)
    - Not genotoxic (Rickard, 2009)
  - o PFBS
    - Half-life in retired fluorochemical workers: 13,1 – 45,7 days, with an average of 27,7 days) (Olsen et al., 2007)
    - Half-life in male rats: 2,1 hours (Chengelis et al., 2009)
    - Half-life in female rats: 0,64 hours (Chengelis et al., 2009)
    - Urine is the main route of elimination of PFBS (Chengelis et al., 2009, Olsen et al., 2007)
    - Based on the results of multiple acute ecotoxicity tests, PFBS is classified as an insignificant hazard by the U.S. National Institute of Occupational Safety and Health (NIOSH). No labelling required by the European Union (3M, Technical Data Bulletin)
    - PFBS acute oral LD50 (> 2000 mg/kg) in rat toxicity studies is classified by the U.S. EPA as "slightly toxic", by the European Union as "no hazard" (3M, Technical Data Bulletin)
    - Based on a NOEL of > 1000 mg/kg bw/day in a two-generation reproduction study with rats, PFBS is considered practically non-toxic in multi-generation reproduction (3M, Technical Data Bulletin)
    - BCF in Rainbow Trout (liver and blood): < 1 (no bioconcentration) (Martin et al., 2003)

- PFHxS
  - In a study with Swedish women, serum PFHxS concentrations (4,7 ng/ml) are lower than PFOS (20,7 ng/ml), but higher than PFOA (3,8 ng/ml) (Karman et al., 2007)
  - Half-life in retired fluorochemical workers: 8,5 years (Olsen et al., 2007)
  - Half-life in mice: 25 – 30 days (Sundström et al., 2012)
  - Half-life in male monkeys: 141 days (Sundström et al., 2012)
  - Half-life in female monkeys: 87 days (Sundström et al., 2012)
  - Urine is the main route of elimination of PFHxS (Sundström et al., 2012)
  - Studies that looked at the effects of maternal exposure levels during pregnancy and anthropometry of their new-born babies have been inconsistent (cited in Bull et al., 2014)

#### Long-chain PFAS

- Bioaccumulation potential: high (U.S. EPA, 2009)
  - Perfluorohexadecanoic acid (C16): BCF = 4.700 – 4.800 (Carp)
  - PFODA (Perfluorooctadecanoic acid) (C18): BCF = 320 – 430 (Carp)
- Environmental Toxicity testing: The acute toxicity of C9 –C20 PFCAs is low to moderate with acute EC/LC50 values between 8,8 – 285 mg/l (Environment Canada, 2012)
- Biochemical responses due to exposure to long-chain PFCAs in environmental toxicity testing: vitellogenin induction, oxidative stress and chemical sensitization in species such as marine mussels, rainbow trout and Baikal seals (Environment Canada, 2012)
- No data on carcinogenicity for the long-chain PFAS
- Summary of information for some long-chain PFAS:
  - PFNA (Perfluorononanoic acid) (C9)
    - Half-life in male mice: 34-68 days (Tatum-Gibbs et al., 2011)
    - Half-life in female mice: 25-68 days (Tatum-Gibbs et al., 2011)
    - Half-life in male rats: 29-30 days (Tatum-Gibbs et al., 2011)
    - Half-life in female rats: 1,4-2,4 days (Tatum-Gibbs et al., 2011)
  - PFDA (perfluorodecanoic acid) (C10)
    - Half-life in male rats: 40 days (Ohmori et al., 2003)
    - Half-life in female rats: 58 days (Ohmori et al., 2003)
  - PFDS (Perfluorodecane sulfonic acid) (C10)
    - No data available

#### Others (Precursors, Fluorotelomers)

- 8:2 FTOH (8:2 Fluorotelomer alcohol) (precursor of PFOA) (information from Bull et al., 2014):
  - Half-life in rats: < 5 hours
  - Excretion primarily via the faeces (> 70%)
  - Metabolism to PFOA, PFNA, PFDA, and other long chain PFCAs
  - Presence of the FTOH metabolites in blood following occupational exposure suggests metabolism of FTOHs to high levels of PFOA and PFNA in humans
  - NOEC (oral gavage, 90 days, rats, repeat dose toxicity): 5 mg/kg bw/day
  - NOEC(oral diet, 74 days, rats, reproductive toxicity): 25 mg/kg bw/day
  - NOEC (oral diet, 74 days, rats, developmental toxicity): 200 mg/kg bw/day

DEPA (2013) states: “Results from analyses of PFAS in polar bears indicate that fluorotelomers also contribute to the total bioaccumulation of per- and polyfluorinated compounds in these animals because perfluorononaic acid (PFNA) was almost only found in its linear form while both linear and branched isomers were observed for PFOA”.

## 5. REGULATION

Concern around the environmental effects of PFAS use began in the late 1990s when it was realised that, due to their resistance to biodegradation, PFOS and PFOA were ubiquitous in various biological (wildlife and humans) and environmental (water bodies) matrices, and could biomagnify. The degree of biomagnification is proportional to perfluorocarbon chain length and so regulatory initiatives to restrict the use of PFAS have focussed on the long chained PFAS. With global restrictions now in place for PFOS, further regulation is proposed in Europe and elsewhere to restrict the manufacture and use of any PFAS substance that contains a C7 or C8 perfluorocarbon moiety in its molecular structure. As there is a growing understanding of the properties of PFAS, it is clear that further information on their toxicology, persistence and bioaccumulation potential is required to further define which specific PFAS compounds pose a potential for risk to human health and the environment.

In 2009, PFOS was added to Annex B of the Stockholm Convention on Persistent Organic Pollutants (POPs), meaning that measures must be taken to restrict its production and use. In Europe, the use of PFOS is banned, although there are some exemptions. Substances or mixtures may not contain PFOS above 0,001 wt% (EU 757/2010). A derogation for the use of legacy fire-fighting foam stocks containing >0,001 wt% PFOS ended on June 27th 2011.

Since 26th June 2013, PFOA and its ammonium salt (APFO) have been identified as chemicals of “very high concern” and added to the candidate list of the European Chemicals Agency (ECHA). Since that time, four further long-chain PFCA (11 to 14 carbon atoms) have been identified as substances of very high concern. In a restriction proposal submitted to The European Chemicals Agency (ECHA) in 2014, Germany and Norway requested that the concentration of PFOA and possible PFOA precursors in products placed on the market be limited to <2 ppb, which is 5.000 times lower than the current limit for PFOS (0,001 wt%, or 10.000 ppb). The restriction proposal also covers substances having linear or branched perfluoroheptyl derivatives with the formula C<sub>7</sub>F<sub>15</sub>- as a structural element (ECHA, Annex XV Restriction Report, 2014). At the time of writing (Nov 2015), the second public consultation was ongoing (public consultation of the draft SEAC opinion). While the manufacture and use of short chain PFAS is still permitted, their persistence in the environment increases the risk of future use restrictions.

In 2013, PFOS and its derivatives were included in the EU Directive on Environmental Quality Standards (2013/39/EU amending 2008/105/EC). The EU annual average environmental quality standard (EQS) for PFOS in surface freshwater is set at a very low criterion of 0,00065 µg/l, based on the potential for secondary poisoning in humans due to fish consumption. The EQS of 0,00065 µg/l is derived from starting points that are considered by many as very conservative, and is lower than background levels typically recorded in surface waters (see Section 6). It is also lower than the LOQ typically achieved by commercial laboratories. The date set for EU-wide compliance with the EQS is 22nd December 2027, with member states required to submit to the Commission a supplementary monitoring programme and a preliminary programme of measures to achieve compliance by 22nd December 2018.

Provisional drinking water standards developed by EU member states are generally around 0.1 to 0.5 µg/l PFOS, which is 3 orders of magnitude higher than the Annual Average EQS. In those countries where target values for groundwater have been derived these are within a similar range. Environmental standards may also encompass a range of other PFCAs and PFSAs, with limits set both for individual

substances and also the total PFAS concentration. The available provisional drinking water, groundwater and soil guidelines are summarized in **Table 5.1**.

**Table 5.1:** Overview of (provisional) guidelines for drinking water, groundwater and soil in European countries and abroad

Drinking Water Criteria in µg/l in European Countries															
	PFOS	PFOA	PFOSA	PFBS	PFBA	PFPeA	PFHxA	PFHpA	PFNA	PFDA	6:2 FTS	PFHpS	PFHxS	PFPeS	Remark
Denmark	(0,1)	(0,1)	(0,1)	(0,1)	(0,1)	(0,1)	(0,1)	(0,1)	(0,1)	(0,1)	(0,1)	-	(0,1)	-	Sum of these 12 parameters: 0,1 µg/l
Germany	0,3	0,3	-	-	-	-	-	-	-	-	-	-	-	-	
The Netherlands	0,53	-	-	-	-	-	-	-	-	-	-	-	-	-	Values not included in legislation, but can be used in case of a PFOS contamination
Sweden	0,09	(0,09)	-	(0,09)	-	-	-	-	-	-	-	-	-	-	This limit is also applied for the sum of PFOS, PFHxS, PFBS, PFOA, PFHpA, PFHxA and PFPeA
U.K.	0,3	0,3	-	-	-	-	-	-	-	-	-	-	-	-	Tiered approach (concentrations of Tier 1 included)
Drinking Water Criteria in µg/l abroad															
	PFOS	PFOA	PFOSA	PFBS	PFBA	PFPeA	PFHxA	PFHpA	PFNA	PFDA	6:2 FTS	PFHpS	PFHxS	PFPeS	Remark
Minnesota	0,3	0,3	-	7	7	-	-	-	-	-	-	-	-	-	
New Jersey	-	0,04	-	-	-	-	-	-	0,013	-	-	-	-	-	
U.S. EPA	0,2	0,4	-	-	-	-	-	-	-	-	-	-	-	-	
Canada	0,3	0,7	-	-	-	-	-	-	-	-	-	-	-	-	
Groundwater Criteria in µg/l in European Countries															
	PFOS	PFOA	PFOSA	PFBS	PFBA	PFPeA	PFHxA	PFHpA	PFNA	PFDA	6:2 FTS	PFHpS	PFHxS	PFPeS	Remark
Denmark	(0,1)	(0,1)	(0,1)	(0,1)	(0,1)	(0,1)	(0,1)	(0,1)	(0,1)	(0,1)	(0,1)	-	(0,1)	-	Sum of these 12 parameters: 0,1 µg/l
Germany	-	-	-	-	-	-	-	-	-	-	-	-	-	-	
State of Bavaria	0,23	-	-	3,0	7,0	3,0	1,0	0,3	0,3	0,3	-	-	-	-	PFOS + PFOA + PFHxS: 0,3 µg/l
State of Baden-Württemberg	0,23	0,3	-	3,0	7,0	3,0	1,0	0,3	0,3	0,3	0,3	0,3	0,3	1	(1) In the case that PFOS, PFOA, H4PFOS, PFNA, PFDA, PFHpS, PFHpA, PFHxS, PFHxA, PFPeS, PFPeA, PFBS and PFBA occur at the same time: Concentration/limit value <1 (2) Each additional per- and polyfluorinated compound: 1 µg/l, each

Groundwater Criteria in µg/l in European Countries															
The Netherlands	0,023	-	-	-	-	-	-	-	-	-	-	-	-	-	Values not included in legislation, 4 different target values were derived for different site use scenarios. 0,023 is the most stringent value.
Groundwater Criteria in µg/l abroad															
	PFOS	PFOA	PFOSA	PFBS	PFBA	PFPeA	PFHxA	PFHpA	PFNA	PFDA	6:2 FTS	PFHpS	PFHxS	PFPeS	Remark
New Jersey	-	-	-	-	-	-	-	-	0,02	-	-	-	-	-	
Soil Criteria in mg/kg in European Countries															
	PFOS	PFOA	PFOSA	PFBS	PFBA	PFPeA	PFHxA	PFHpA	PFNA	PFDA	6:2 FTS	PFHpS	PFHxS	PFPeS	Remark
Denmark	(0,4)	(0,4)	(0,4)	(0,4)	(0,4)	(0,4)	(0,4)	(0,4)	(0,4)	(0,4)	(0,4)	-	(0,4)	-	Sum of these 12 parameters: 0,4 mg/kg ts
Germany	-	-	-	-	-	-	-	-	-	-	-	-	-	-	
State of Bavaria	Evaluation for pathway Soil -> Groundwater is based on Leachate Concentrations (µg/l) Evaluation for recycling of Soils is based on LAGA M20 Criteria														
The Netherlands	0,003	-	-	-	-	-	-	-	-	-	-	-	-	-	Values not included in legislation, 4 different target values were derived for different site use scenarios. 0,023 is the most stringent value.

More information about the global treaties and conventions, European legislation and national setting of guidelines is discussed in the sections below.

### 5.1. GLOBAL TREATIES AND CONVENTIONS

In May 2009, the parties of the Stockholm Convention - an international environmental treaty - decided to add PFOS to Annex B of the Stockholm Convention on POPs. This means that the parties must take measures to restrict the production and use of PFOS to those deemed acceptable purposes and/or specific exemptions listed in the Annex (Stockholm Convention, 2009). Acceptable purposes mean that there was no time limit put to the use, whereas specific exemptions meant that the exemption was only valid for 5 years after 2009:

- Acceptable purposes: Photo-imaging, photo-resistance and anti-reflective coatings for semi-conductors, etching agent for compound semi-conductor and ceramic filters, aviation hydraulic fluids, metal plating (hard metal plating) only in closed-loop systems, certain medical devices (such as ethylene tetrafluoroethylene copolymer (ETFE) layers and radio-opaque ETFE production, in-vitro diagnostic medical devices, and CCD colour filters), fire-fighting foams, insect baits for control of leaf-cutting ants from *Atta spp.* and *Acromyrmex spp.*
- Specific exemptions: Photo masks in the semiconductor and liquid crystal display (LCD) industries, metal plating (hard metal plating, decorative plating), electric and electronic parts for some colour printers and colour copy machines, insecticides for control of red imported fire ant, and termites, chemically driven oil

production, carpets, leather and apparel, textiles and upholstery, paper and packaging, coatings and coating additives, rubber and plastics.

In June 2015, the European Union has submitted a proposal to list PFOA, its salts (e.g. APFO) and PFOA-related compounds (e.g. 8:2 FTOH) in Annexes A, B and/or C<sup>6</sup> of the Stockholm Convention. The POPs review Committee (POPRC) will evaluate the proposal and will make recommendations to the Conference of the Parties. It will take at least 5 years to complete the procedures to list PFOA, its salts and PFOA-related compounds under the Stockholm Convention (Stockholm Convention, 2015).

## 5.2. EUROPEAN UNION LEGISLATION

### 5.2.1. EU Regulations regarding PFAS use

Legislation within the European Union (EU) is focused mainly on the use of PFOS and its derivatives. PFOS is currently classified under REACH (registration, evaluation, authorisation and restriction of chemicals) as a “PBT” substance (persistent, bioaccumulative and toxic).

The EU in effect banned the use of PFOS in finished and semi-finished products in 2006 (Directive 2006/122/EC). The maximum allowed concentration of PFOS in these products was 0,005%. Exemptions were made for certain industrial applications (e.g. photolithography, chromium plating industry, hydraulic fluids for aviation). In 2009 (Regulation EC 552/2009), this was incorporated into the existing REACH regulation (Annex XVII of REACH Regulation no. 1907/2006).

As described in Section 5.1, the parties of the Stockholm Convention decided in 2009 that the application and use of PFOS had to be restricted (Stockholm Convention, 2009). This was enforced through Regulation 850/2004/EC (relating to POPs) with PFOS added in 2010 (EU regulation 757/2010 dated 24 August 2010), and the threshold value lowered to or below 10 mg/kg (0,001 wt%) when it occurs in substances or in preparations.

Therefore, the following restrictions currently apply in the EU for PFOS and its derivatives ( $C_8F_{17}SO_2X$ ,  $X=OH$ , metal salts, halide, amide and others, including polymers):

- Substances or mixtures may not contain PFOS above 0,001 wt%;
- Semi-finished products or articles or components containing PFOS 0,1 wt% or greater are not allowed to be brought into circulation;
- New textiles or other coated new materials with a content of 1  $\mu\text{g}/\text{m}^2$  or more are not allowed to be brought into circulation.

Exemptions apply for the following applications as long as no alternatives are available:

- Photo-resistive or anti-reflective coatings for photolithographic processes;
- Photographic coatings applied to films, papers, or printing plates;

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<sup>6</sup> Annex A: measures must be taken to eliminate the production and use of these chemicals, Annex B; measures must be taken to restrict the production and use of these chemicals, Annex C: measures must be taken to reduce the unintentional releases of these chemicals.

- Mist suppressant for non-decorative hard chromium (VI) plating systems in closed loop systems;
- Hydraulic fluids for aviation and aerospace;
- PFOS-based wetting agents for controlled electroplating systems may still be used until August 26, 2015;
- Aqueous Firefighting Foams sold before December 27, 2006, could have been used until June 27, 2011. Currently firefighting foams have to contain less than 0,001 wt% PFOS.

For other PFAS, there are still no specific manufacturing or application restrictions in the EU, with the exception of Norway (see Section 5.3).

PFOA and its ammonium salt (APFO, perfluoro-ammoniumoctanate) recently have been identified as chemicals of “very high concern”, as defined under the European chemicals regulation, REACH (<http://echa.europa.eu/candidate-list-table>). From 26 June 2013 these substances were added to the candidate list of the ECHA.

Germany and Norway proposed a restriction that covers the following substances:

- PFOA, including its salts;
- Any other substance having linear or branched perfluoroheptyl derivatives with the formula  $C_7F_{15}$ - as a structural element, including its salts, except those derivatives with the formula  $C_7F_{15}-X$  where  $X = F, Cl, Br$ ;
- Any other substance having linear or branched perfluorooctyl derivatives with the formula  $C_8F_{17}$ - as a structural element, including its salts, except those derivatives with the formula  $C_8F_{17}-X$ , where  $X = F, Cl, Br$  or  $C_8F_{17}-SO_2X'$ ,  $C_8F_{17}-C(=O)OH$  or  $C_8F_{17}-CF_2-X'$  (where  $X'$ =any group, including salts).

The proposed restriction covers the manufacturing, use and placing on the market of the above mentioned substances (derivatives of C8 and C7) as a substance, as a constituent of other substances, or in mixtures, if the concentration is equal or greater than 2 ppb (2 µg/kg). Articles containing these substances in concentrations equal to or greater than 2 ppb are also proposed to be restricted (ECHA, Annex XV Restriction Report, 2014).

Currently, the second public consultation is ongoing (public consultation of the draft SEAC opinion).

The German Federal Environmental Agency (UBA), together with the Norwegian Environmental Authority (Klif, now Miljødirektoratet), identified four more perfluorinated compounds as substances of very high concern: long-chain perfluorocarboxylic acids with 11 to 14 carbon atoms (heneicosfluoroundecanoic acid, tricosfluorotridecanoic acid, pentacosfluorotridecanoic acid, heptacosfluorotetradecanoic acid). These compounds were added to the REACH candidate list on 19 December 2012 because of their very persistent and very bio-accumulating properties (vPvB). Consequently manufacturers and distributors must notify the ECHA, if their products contain more than 0,1 weight percent of these substances.

## 5.2.2. EU Environmental quality standards

The Directive on “Environmental Quality Standards” (EQSD) (Directive 2008/105/EC) is a daughter directive of the Water Framework Directive (WFD) and sets environmental quality standards for certain priority and priority hazardous substances. The list of these substances will be reviewed at regular intervals (currently set at six years) on the basis of scientific data and risk assessments. In Directive 2013/39/EC (12 August 2013) new priority hazardous substances were added, including PFOS and its derivatives, and a number of EU-wide environmental quality standards (EQS) were set. The EQS presented in the Directive were derived by RIVM (Dutch National Institute for Public Health and the Environment / Rijksinstituut voor Volksgezondheid en Milieu) in 2010 (RIVM, 2010).

The EQS for PFOS and derivatives are summarized in **Table 5.2**. The annual average EQSs (AA-EQS) set by the European Commission of 0,00065 µg/l (surface freshwater) or 0,00013 µg/l (coastal and transitional waters) are regarded as extremely challenging considering the current PFOS-levels recorded in European waters (see Section 6).

The derivation of the EQS is discussed in the PFOS EQS Dossier of 2011, prepared by the Sub-Group on Review of the Priority Substances List (under Working Group E of the Common Implementation Strategy for the Water Framework Directive).

The Maximum Allowable Concentration-EQS (MAC-EQS) was derived using a pooled freshwater-marine acute toxicity data set. The MAC-EQS for the freshwater environment (“Inland surface waters”) is based on an acute toxicity test result using the marine mysid *Mysidopsis bahia* (96h LC50: 3,6 mg/l, with an assessment factor of 100). For the marine environment (“Other surface waters”) the MAC-EQS is based on the same data point to which an additional assessment factor of 5 is applied (96h LC50: 3,6 mg/l, with an assessment factor of 500).

The AA-EQS was calculated based on 3 methodologies: (1) based on ecotoxicity, (2) based on secondary poisoning and (3) based on fish consumption by humans. The lowest calculated AA-EQS, in this case the one based on fish consumption by humans, was proposed as the AA-EQS for inland surface waters.

The following data were considered during derivation of the AA-EQS (based on fish consumption by humans): a TDI of 150 ng/kg bw/day (EFSA, 2008, see also Section 4.2.2), a human body weight of 70 kg, a daily consumption of 115 g fish, a maximum contribution of fish to the TDI of 10% and a BCF and BMF of 2.800 l/kg and 5 kg/kg (from water to fish), respectively.

For secondary poisoning (EQS Biota), the Cynomolgus monkey subchronic study (6 months, chronic effects are not known) of Seacat et al. (2002) was used.

**Table 5.2:** EQS of the European Commission for PFOS and its derivatives

Name of substance	AA-EQS* (µg/l)		MAC-EQS** (µg/l)		EQS (µg/kg)
	Inland surface waters	Other surface waters	Inland surface waters	Other surface waters	Biota
Perfluoro octane sulfonate and its derivatives (PFOS)	0,00065	0,00013	36	7,2	9,1

\* AA: Annual average

\*\* MAC: Maximum allowable concentration

The new EU Directive 2013/39/EC entered into force on 9 September 2013, and must be transposed into Member State legislation by 14 November 2015. The new EQS in the directive should be taken into account during the establishment of supplementary monitoring programmes during implementation of the Water Framework Directive. The 'programmes of measures' have to be submitted to the European Commission by the 22 December 2018. Based on the aim of achieving good surface water chemical status, in theory the EQS of the newly identified priority substances have to be met by 22 December 2027.

#### **Remarks on the EQS**

The AA-EQS for PFOS in surface water 0,00065 µg/l is considerably lower than the (provisional) drinking water standards (roughly between 0,1 and 0,5 µg/l for PFOS, see next paragraphs). There is a difference of nearly 3 orders of magnitude.

The low level of the AA-EQS is driven by the starting point for the calculation being the consumption of fish. Wilson (2015) demonstrated that with alternative (also defensible) values for: TDI (0,3 vs. 0,15 µg/kg day), contribution of fish to dietary uptake (73% vs. 10%), average fish consumption rate (0,028 kg/day vs. 0,115 kg/day), bioconcentration and biomagnification factors (1.124 vs 2.800 and 2 vs 5), the AA-EQS could have been derived 375 times higher (0,24 µg/l).

### **5.3. NATIONAL LEGISLATION AND GUIDANCE IN EUROPEAN COUNTRIES**

In most EU countries no additional national legal levels have been set. Most limits are still provisional and are being used as screening levels.

#### **5.3.1. Denmark**

The Danish Ministry of the Environment (DEPA, 2015) proposed the following health-based quality criteria in drinking water:

PFOS: 0,1 µg/l  
 PFOSA: 0,1 µg/l  
 PFOA (and salts e.g. APFO): 0,3 µg/l

Where PFOS, PFOA and PFOSA occur in the drinking water at the same time, the following criteria can be used (Concentration/Limit value < 1):

$$\text{PFOA (conc. } \mu\text{g/l)} / 0,3 \mu\text{g/l} + \text{PFOS (conc. } \mu\text{g/l)} / 0,1 \mu\text{g/l} + \text{PFOSA (conc. } \mu\text{g/l)} / 0,1 \mu\text{g/l} < 1$$

In the case that groundwater is directly used for drinking water consumption, the same criteria as for drinking water should be used for groundwater. In cases where contaminated soil affects the groundwater, the same health based drinking water quality criteria can be applied for the groundwater affected by the contamination (cited from DEPA, 2015).

DEPA (2015) has derived the following health-based soil quality criteria:

PFOS: 0,39 mg/kg soil  
 PFOSA: 0,39 mg/kg soil  
 PFOA: (and salts, e.g. APFO): 1,3 mg/kg soil

In the case that PFOS, PFOA and PFOSA occur in the soil at the same time, the following criteria can be used (Concentration/Limit value < 1):

$$\text{PFOA (conc. mg/kg)} / 1,3 \text{ mg/kg} + \text{PFOS (conc. mg/kg)} / 0,39 \text{ mg/kg} + \text{PFOSA (conc. mg/kg)} / 0,39 \text{ mg/kg}$$

More recently, on 27 April 2015, DEPA proposed new drinking water, groundwater and soil criteria for the sum of 12 PFAS (DEPA, 2015b). **Table 5.3** gives an overview.

**Table 5.3:** Most recent provisional drinking water, groundwater and soil criteria from DEPA (27 April 2015)

Sum of:	Provisional drinking water and groundwater criterion (µg/l)	Provisional soil criterion (mg/kg TS)
PFBS	0,1	0,4
PFHxS		
PFOS		
PFOSA		
6:2 FTS		
PFBA		
PFPeA		
PFHxA		
PFHpA		
PFOA		
PFNA		
PFDA		

### 5.3.2. Germany

The only national legal set value in Germany is related to the use of soil fertilizers. The legal limit for the use of soil fertilizers set in the German Fertilizer Ordinance (Düngemittelverordnung, DüMV, December 2012) is 100 µg/kg dry matter for the Sum of PFOS and PFOA.

The Umweltbundesamt (UBA) and the Drinking Water Commission (TWK) of the Federal Ministry of Health at the UBA recommend for the protection of human health a permanent tolerable, health-related indication value (HRIV) of 0,3 µg/l PFAS (adults, long life exposure). They regard a maximum yearly average value of 0,1 µg/l – as a precautionary value – for the sum total of highly accumulating PFAS as adequate (Umweltbundesamt, 2006). The above mentioned criteria are currently also used for the protection of groundwater.

There are no federal regulated values for discharge water or soil.

State of Bavaria

The Bavarian State office for Environment (Bayerische Landesamt für Umwelt, LfU) has published provisional evaluation criteria for selected PFAS in groundwater, surface water and soil (LfU, 2015). These provisional criteria, applicable in the State of Bavaria are discussed below.

The provisional threshold values for groundwater are summarized in **Table 5.4**.

**Table 5.4:** Provisional threshold values for groundwater (LfU, 2015)

Parameter	Provisional threshold value (µg/l)
PFOS	0,23
PFOS + PFOA + PFHxS	0,3
PFBA	7,0
PFBS	3,0
PFPeA	3,0
PFHxA	1,0
PFHpA	0,3
PFNA	0,3
PFDA	0,3

These threshold values were derived based on the HRIV-Concept of the Drinking Water Commission of the Federal Ministry of Health and are also based on the criteria of LAWA (Länder-Arbeitsgemeinschaft Wasser) to derive the “no-effect-levels” (called “Geringfügigkeitsschwellenwerte”). An exceedance of the groundwater threshold values highlights an adverse change of the groundwater status according to the Water Resources Law.

As long as the EU Directive 2013/39/EU is not implemented, PNEC<sub>aquatic</sub> (Predicted No Effect Concentrations) values shall be used to evaluate PFAS impacts in surface water. LfU derived PNEC values based on investigations with rainbow trout and are summarized in **Table 5.5**.

**Table 5.5:** PNEC<sub>aquatic</sub> values for surface water (LfU, 2015)

Parameter	PNEC (µg/l)
PFOS	0,05
PFOA	570

To calculate the risk potential for the pathway Soil → Groundwater, leachate values (elution according to DIN 38414-S4, water-solid ratio 10: 1) are to be used, as the sole determination of solids content is not meaningful, due to the mobility behaviour of the PFAS. The PFAS concentration in the eluate is transferred to the leachate at the sampling location. The assessment is based on the preliminary Level-1 and Level-2 values, as listed in **Table 5.6**, in accordance with the procedure described in LfU leaflet 3.8/1.

**Table 5.6:** Preliminary Level-1 and Level-2 values for PFAS for the Pathway Soil → Groundwater (LfU, 2015)

Parameter	Preliminary Level-1 Value (µg/l)	Preliminary Level-2 Value (µg/l)
PFOS	0,23	1,0
PFOS + PFOA + PFHxS	0,3	1,0
PFBA	7,0	28,0
PFBS	3,0	12,0
PFPeA	3,0	12,0
PFHxA	1,0	4,0
PFHpA	0,3	1,0
PFNA	0,3	1,0
PFDA	0,3	1,0

The preliminary Level-1-values correspond to the threshold values for groundwater (see **Table 5.4**). Further investigation or additional evaluation is triggered in the case that these levels are exceeded. The preliminary Level-2 values are used directly as a criterion for groundwater and leachate at the sampling location. When the Level-2 value is exceeded, risks cannot be excluded and remedial measures are usually required.

For the recycling/reuse of mineral residues / wastes outside of landfills only clean soil (Class Z0) may be used. For this scenario, for a number of PFAS concentration values were set according to LAGA M 20 (Länder-Arbeitsgemeinschaft Abfall (LAGA), 2003). Recycling/reuse is only allowed if the eluate concentrations do not exceed the levels as indicated in **Table 5.7**.

In case of recycling/reuse of soil material in “unrestricted incorporation” in technical buildings according to LAGA M 20 (Status of 6 November 1997), the concentrations shall fulfill the criteria of Class Z0. Any use of soil material in “restricted open installations” in technical buildings according to LAGA M 20 is only allowed if the PFC-concentrations fulfill the criteria of Class Z1.1.

**Table 5.7:** Criteria for the S4-Eluate (based on LAGA M 20) (LAGA, 2003)

Parameter	Z 0 (µg/l)	Z 1.1 / Z 1.2 (µg/l)	Z 2
Σ PFHxS, PFOS, PFOA, PFC <sub>C&gt;8</sub>	0,1	0,3	1,0
PFHxA	0,3	1,0	4,0
PFPeA	1,0	3,0	12,0
PFBS	1,0	3,0	12,0
PFBA	3,0	7,0	28,0

The adsorption of PFAS to soil is highly dependent on the soil matrix (see also Section 3). Therefore threshold concentrations for soil were not calculated.

According to a letter from the Bavarian Ministry of 7<sup>th</sup> January 2008 (updated on 23 June 2014), all sewage sludge with potential use on agricultural land or for landscape planning and a capacity of water treatment plants of 1.000 population equivalent, shall be analyzed for PFAS. A precautionary value of 100 µg/kg dry matter (+ 25% measurement tolerance) is applicable (LfU, 2015) (Germany, 2009).

### State of Baden-Württemberg

On 17 June 2015, the State of Baden-Württemberg (Ministerium für Umwelt, Klima und Energiewirtschaft) published provisional threshold values for groundwater. These provisional levels are summarized in **Table 5.8**.

**Table 5.8:** Provisional threshold values for Groundwater (Baden-Württemberg, 2015)

Nr	PFAS	Provisional Groundwater threshold (µg/l)
	PFOS	0,23
1	PFOS	0,3 <sup>1</sup>
2	PFOA	0,3
3	H <sub>4</sub> PFOS <sup>2</sup>	0,3
4	PFNA	0,3
5	PFDA	0,3
6	PFHpS	0,3
7	PFHpA	0,3
8	PFHxS	0,3
9	PFHxA	1,0
10	PFPeS	1,0
11	PFPeA	3,0
12	PFBS	3,0
13	PFBA	7,0
	Other per- und polyfluorinated compounds	Each 1,0

<sup>1</sup>: For the case that the compounds with Nr. 1 to 13 are present at the same time, following criterion shall be used: Concentration/Limit value < 1. For PFOS, the Limit Value of 0,3 µg/l shall be used, instead of the Limit value of 0,23 µg/l for the single compound.

<sup>2</sup>: H<sub>4</sub>PFOS = 6:2 Fluorotelomer sulfonic acid (6:2 FTS)

Most of the threshold values are taken from the UBA, TWK and LAWA.

### 5.3.3. The Netherlands

Following an accidental spillage of PFOS-contaminated AFFF in the Netherlands, the RIVM has derived risk based action levels for PFOS contamination (RIVM, 2011). These values have not been adopted into legislation, but can be used should an incident involving PFOS contamination take place.

A range of potential action levels have been defined based on background levels, and risk-based protection of ecology and human health. The four standards have been developed based on:

1. Reporting limit or (if higher) the background level of PFOS in soil and groundwater;
2. Eliminating ecological risks (based on evaluation being used in other frameworks, such as the Water Framework Directive). The values have been derived using two commonly used methods (**Table 5.2**);
3. Eliminating ecological effects (based on concentrations that effects have been observed in (ecotoxicological) experiments) and protection of groundwater as a drinking water resource;
4. Sustainable soil use (upper values based on risks to ecology and humans, based on evaluations used in other legislation such as re-use soil and sediment, including use for drinking water).

The RIVM-derived values are provided in **Table 5.9**:

**Table 5.9:** Risk based scenarios with derived action levels for PFOS

Scenario	Risk based value soil (µg/kg)	Risk based value groundwater (µg/l)
2a. Eliminating ecological risks (via established method preventive policy)	3,2	0,023
2b. Ecological protection (via sensitivity distribution species)	3,2	0,094
3. Ecological protection (ecotoxicological experiments) and quality of drinking water meets drinking water criteria	10	0,53
4. Permanent sustainable use of the soil (fit for use), groundwater quality meets drinking water criteria	100	4,7
Reporting Limit / Background Level		
1. Reporting Limit /Background Level	0,1	0,010

One of the scenarios above could be used to determine risk based target levels, depending on the site setting and presence of receptors.

#### 5.3.4. Norway

Norway is the only European Country where PFOA-containing consumer products are prohibited. The Environmental Agency of Norway restricted the use of PFOA in the Consumer Product Regulations (FOR 2004-06-01 nr 922, Section 2-32) as follows:

- Limit of PFOA in substances and mixtures with a maximum 0,001% PFOA, starting 1. June 2014
- Limit of PFOA in textiles, carpets and other coated consumer products with maximum 1 µg/m<sup>2</sup>, starting 1. June 2014.
- Further restrictions on adhesives, foil, or tape in semiconductors, and photographic coatings for film, paper, or screen are extended on 1 January 2016.

The Norwegian guideline value for PFOS in soil is 100 µg/kg dry weight (Norwegian Pollution Control Agency).

#### 5.3.5. Sweden

In 2014, Livsmedelsverket derived a maximum tolerable drinking water level of 0,09 µg/l for PFOS. As a precautionary measure, this limit was further applied for the sum of seven PFAS: PFOS, PFHxS, PFBS, PFOA, PFHpA, PFHxA and PFPeA (from DEPA, 2015).

### 5.3.6. United Kingdom

In 2009, the Drinking Water Inspectorate published guidance on the levels of PFOS and PFOA that water companies should act upon to fulfil their statutory obligations to ensure the safety of drinking water. The guidance is based on a multi-tiered approach and summarized in the table below (from Drinking Water Inspectorate, 2009):

**Table 5.10:** Guidance for PFOS and PFOA (from Drinking Water Inspectorate, UK)

Item	Regulatory requirement	Guidance value (concentration)	Minimum action to be taken
<b>Perfluorooctane sulphonate (PFOS)</b>			
Tier 1	Regulation 27 (Risk assessment)	potential hazard	• ensure considered as part of statutory risk assessment
Tier 2	Regulation 10 (Sampling: further provisions)	> 0,3µg/l	• consult with local health professionals; • monitor levels in drinking water.
Tier 3	Regulation 4(2) (Wholesomeness)	> 1,0µg/l	As tier 2 plus: • put in place measures to reduce concentrations to below 1.0µg/l as soon as is practicable.
Tier 4*	Water Industry (Suppliers' Information Direction) 2009 (Notification of events)	> 9,0 µg/l	As tier 3 plus: • ensure consultation with local health professionals takes place as soon as possible; • take action to reduce exposure from drinking water within 7 days.
*Note - notification to the Inspectorate may also be triggered at lower levels due to Tier 1, 2 or 3 activities			
<b>Perfluorooctanoic acid (PFOA)</b>			
Tier 1	Regulation 27 (Risk assessment)	potential hazard	• ensure considered as part of statutory risk assessment
Tier 2	Regulation 10 (Sampling: further provisions)	> 0,3 µg/l	• consult with local health professionals; • monitor levels in drinking water.
Tier 3	Regulation 4(2) (Wholesomeness)	> 5,0µg/l	As tier 2 plus: • put in place measures to reduce concentrations to below 5.0µg/l as soon as is practicable.
Tier 4*	Water Industry (Suppliers' Information Direction) 2009 (Notification of events)	> 45,0µg/l	As tier 3 plus: • ensure consultation with local health professionals takes place as soon as possible; • take action to reduce exposure from drinking water within 7 days.
*Note - notification to the Inspectorate under the Information Direction may also be triggered at lower levels due to Tier 1 2 or 3 activities			

## 5.4. LEGISLATION OUTSIDE EUROPE

For comparison reasons, further risk based values from outside of the European Union are included below.

### 5.4.1. U.S. EPA

In 2009, the U.S. EPA set the following drinking water guidance values (advisory levels):

- PFOA: 0,4 µg/l
- PFOS: 0,2 µg/l

If these provisional health advisory levels are exceeded, the use of water for drinking or cooking should be stopped. They reflect an amount of PFOS and PFOA that may cause adverse effects in the short term (weeks to months).

Currently, PFOS and PFOA are included by the U.S. EPA on the Draft Contaminant Chemical List 4 (CCL 4) (<http://www2.epa.gov/ccl/chemical-contaminants-ccl-4>), meaning that in the future regulation may be required under the Safe Drinking Water Act (SDWA).

#### Minnesota

More than ten years ago the Minnesota Department of Health (MDH) commenced the development of drinking water criteria for some PFAS. MDH published the following Health Risk Limits (HRLs) which are considered safe for people, including sensitive subpopulations

(<http://www.health.state.mn.us/divs/eh/hazardous/topics/pfcshealth.html>):

- PFOA: 0,3 µg/l
- PFOS: 0,3 µg/l
- PFBS: 7 µg/l
- PFBA: 7 µg/l

<http://www.pca.state.mn.us/index.php/view-document.html?gid=2869>

#### New Jersey

The Department of Environmental Protection of the State of New Jersey (NJ DEP) developed in 2009 a preliminary drinking water guidance value for PFOA, set at 0,04 µg/l (NJ DEP, 2009). This guidance level is the first phase of an ongoing process to establish a drinking water standard (MCL) for PFOA.

Related to this low drinking water guidance criteria, NJ DEP writes the following: "This value is the lower end of the range of values derived based on several non-cancer and cancer endpoints in different species, most of which cluster within a factor of two of this value. This drinking water concentration is expected to be protective of both non-cancer effects and cancer at the one in one million risk level. The recommendations provided here will be re-evaluated as additional data on PFOA's effects and kinetics in humans and animals become available".

In July 2015, the New Jersey Drinking Water Quality Institute proposed a drinking water maximum contaminant level (MCL) for PFNA (perfluorononanoic acid) of 0,013 µg/l, which is a protective level for chronic drinking water exposure and

technically feasible (NJ Drinking Water Quality Institute, 2015). The New Jersey Drinking Water Institute recommends “that NJ DEP propose and adopt an MCL of 13 ng/l for PFNA in drinking water”.

In 2014, NJ DEP developed a draft interim groundwater criterion for PFNA, set at 0,02 µg/l (NJ DEP, 2014).

Preliminary guidance values for PFOS are not available.

**5.4.2. Canada**

In 2010, Health Canada set the following provisional drinking water guidance values for PFOA and PFOS:

- PFOA: 0,7 µg/l
- PFOS: 0,3 µg/l

In 2013, Environment Canada developed draft Federal Environmental Quality Guidelines (FEQGs) for PFOS. These FEQGs are summarized in **Table 5.11**.

**Table 5.11** Draft Federal Environmental Quality Guidelines for PFOS in the environment in Canada (from Environment Canada, 2013)

Air	Sediment	Water (ng/l)	Fish Tissue (ng/g wet weight)	Wildlife Diet (ng/g wet weight food)		Bird Egg (ng/g wet weight)
				Mammalian	Avian	
N/A		6.000	8.300	4,6	8,2	1.900

These draft FEQGs are based on laboratory toxicity studies. If concentrations are detected above the FEQGs, Environment Canada conclude that adverse effects in the environment may occur.

## 6. CURRENT CONDITIONS OF EUROPEAN WATERS

Monitoring data from across the EU show the widespread occurrence of PFAS in surface water, with the very low EQS for PFOS in freshwater (0,00065 µg/l) often exceeded.

In an EU-wide survey, 122 water samples were collected in streams and rivers of 27 European countries (sampling in 2007, Loos et al., 2009). PFOS was detected in 93% of the samples with the highest concentration (1,371 µg/l) in the River Krka in Slovenia. PFOA was detected in 97% of the samples at a maximum concentration of 0,174 µg/l. In addition to PFOS and PFOA, a wide range of other PFCAs and PFSAAs were also detected.

A survey of 40 PFAS in surface water along the River Rhine watershed from Lake Constance to the North Sea found that total PFAS concentrations ranged from 0,00035 µg/l in the North Sea to 0,621 µg/l in the River Scheldt. PFOS, PFOA, PFBS and PFBA were usually the major compounds, with the C4-PFAS compounds PFBS and PFBA, accounting for up to 94% of the total.

In a recent European study of PFAS concentrations in 90 waste water treatment plant effluents (Loos et al., 2013), PFOA, PFHpA and PFOS were detected in more than 90% of the waters, with PFOA at the highest median concentration (0,0129 µg/l).

More information about the sources of PFAS in European waters and the occurrence of PFAS in European surface waters is included in the following sections. It highlights the wide spread occurrence of PFAS in the environment but is not intended to give a complete overview.

### 6.1. SOURCES OF PFAS TO EUROPEAN WATERS

The sources which can release significant quantities of perfluorinated alkyl acids to the environment are industrial and municipal wastewater treatment plants (e.g. from textile industry, chrome-plating industry, among others), landfill leachate treatment plants, fire-fighting incidents and fire-fighting training areas (e.g., at airports, fuel production and storage facilities) and landfills. Furthermore, indirect emissions are caused by atmospheric degradation of precursor compounds, which is likely the major source of pollution in remote areas, causing local “background” concentrations of PFAS.

Municipal wastewater treatment plant effluents and infiltration of urban runoff and leaching piping are probably the major source of diffuse pollution to rivers and aquifers (Eschauzier et al., 2012). Loos et al. (2013) stated: “Often PFAS concentrations increase in wastewater treatment plants as a result of biodegradation of precursors during the activated sludge process. PFOA is generally fully discharged into receiving rivers, while about half of PFOS is retained in the sewage sludge”.

Loos et al. (2013) investigated the sources of PFAS contamination in European rivers. They assessed the effluents of 90 European waste water treatment plants and their effect on emerging polar organic contaminants. The study primarily focused on municipal wastewater treatment plants, but some plants treated industrial wastewaters. The research was a follow-on study for the surveys for organic contaminants carried out previously by the European Commission’s Joint Research Centre (Loos et al., 2009, 2010). The results are summarized in the **Table 6.1**.

**Table 6.1:** PFAS Concentrations and Detection Frequency in 90 European Waste Water Treatment Plants (Loos et al., 2009, 2010)

	Detection Frequency (%)	Median Concentration (ng/l)	Highest (single) Maximum Concentration (mg/l) <sup>1</sup>
PFOA	99	12,9	15,9
PFHpA (C7)	94	5,1	3,0
PFOS	93	12,2	2,1
PFNA (C9)	89	2,3	2,7
PFDA (C10)	81	2,9	1,7
PFHxA (C6)	71	5,7	23,9
PFHxS (C6)	70	3,4	0,922

<sup>1</sup> These concentrations are relevant in relation to the MAC-EQS under the Water Framework Directive (see Section 5.2.2).

Note: No data are available about the waste water treatment plants participating in the sampling campaign (no data on waste water source, country, capacity, exact sampling procedure, etc.), although the data are considered representative for the EU.

Loos et al. (2013) stated: “Despite the voluntary phasing out of the production of perfluorooctane sulfonyl-based chemicals in the USA in 2002 (by the main producers), and European restrictions on marketing and use of products containing PFOS coming into force in 2006 (EC, 2006), the detection of PFOS in WWTPs indicates that products containing PFAS are still releasing these substances into the environment”. Low PFOS concentrations are still allowed (see Section 5.2.1), meaning that release of PFOS into the environment cannot be solely classified as “historical”.

## 6.2. PRESENCE IN EUROPEAN SURFACE WATERS

In an EU-wide survey, a range of polar organic persistent pollutants were analysed in unfiltered water samples collected in 2007 at 122 sampling locations in streams and rivers in 27 European countries (Loos et al., 2009). PFOS was detected in 93% of the samples (reporting limit 1 ng/l). The PFOS concentrations reported by Loos et al. (2009) are summarized in the table below.

**Table 6.2:** PFOS Concentrations in some European Rivers, studied by Loos et al., 2009

River	Country	Maximum PFOS Concentration (µg/l)
Krka	Slovenia	1,371 <sup>1</sup>
Scheldt	Belgium	0,154
Scheldt	The Netherlands	0,110
Seine	France	0,097
Rhine	Germany (Wesel)	0,032

<sup>1</sup> Average PFOS concentration: 39 µg/l, Median PFOS concentration: 0,006 µg/l

PFOA was detected in 97% of the samples. The maximum level was 0,174 µg/l. The average and median were 0,012 and 0,003 µg/l.

Eschauzier and coworkers investigated data concerning the presence of perfluoroalkyl acids in European surface waters, groundwater and drinking waters (Eschauzier et al., 2012). Additional data from a monitoring programme of the European Commission Joint Research Centre are given on their website. It gives an overview of concentrations of (emerging) contaminants measured in 2007 (JRC, 2007). The monitoring data confirm the widespread occurrence of PFAS in surface water. PFOS concentrations often exceed the new environmental quality standards for freshwater (see Section 5.2.2) meaning that an environmental risk especially to fish-eating birds and mammals at the highest trophic levels of the food chain could in theory be present.

An overview of the occurrence of PFAS in the different regions of Europe is given in the following sections.

### **6.2.1. Scandinavia**

Relatively low concentrations of PFAS have been found in the Nordic surface waters in comparison to the rest of Europe (Eschauzier 2012). This could be explained by the lower population density and reduced industrial activities. At locations near the larger cities (Oslo, Stockholm, Helsinki), higher values up to 0,050 µg/l have been measured (JRC, 2007).

Filipovic et al. (2015) investigated the distribution of some PFAS related to the usage of AFFFs at a military airport in Stockholm, Sweden. PFAS concentrations (as a sum-parameter) in the nearby groundwater ranged between 0,738 to 51 µg/l. Concentrations up to 0,079 µg/l were detected in surface water.

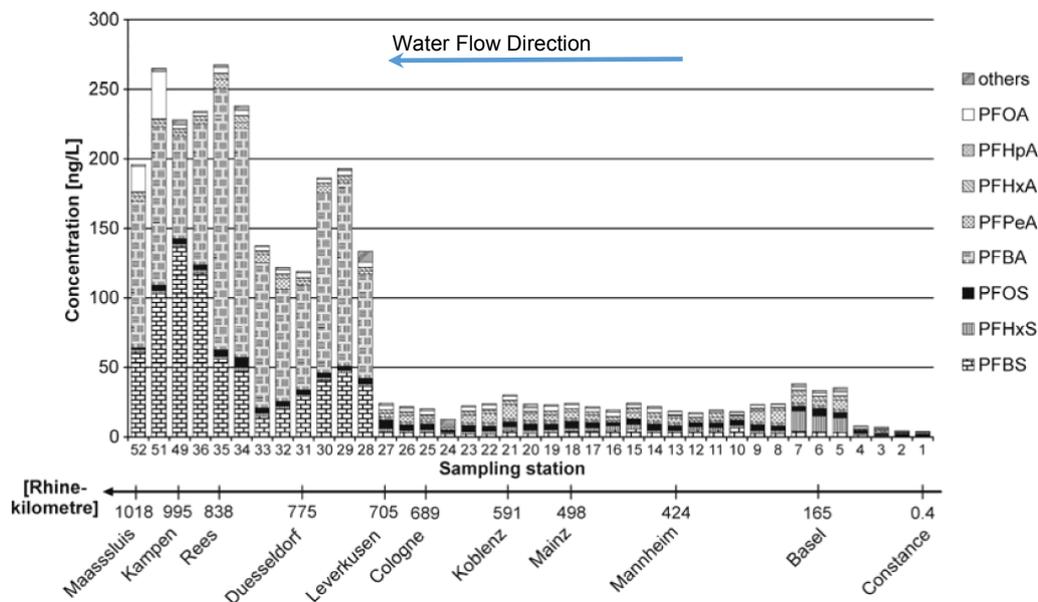
### **6.2.2. River Rhine and other big central European Rivers**

The central European rivers have higher concentrations and mass discharges of PFAS than those in the Northern European countries. The rivers Rhine, Rhone, Danube, Po and Scheldt have been studied extensively (e.g. Eschauzier et al., 2012, Moeller et al., 2010).

Moeller and co-workers studied the concentration profile of 40 PFAS in surface water along the River Rhine watershed from Lake Constance to the North Sea (Moeller et al., 2010). In the study, 75 water samples were taken along the course of the River Rhine as well as several major tributaries such as the Rivers Neckar, Main, Ruhr and waters from the Rhine-Meuse delta (Rivers Meuse and Scheldt). In this research, the concentrations of PFAS (total), measured in 2008, ranged from 0,00035 µg/l in the North Sea to 0,621 µg/l in the River Scheldt. PFOS, PFOA, PFBS and PFBA were usually the major compounds. The C4-based compounds, PFBS and PFBA, were found to be the predominating PFAS, with a percentage contribution of up to 94%.

In the River Rhine the concentrations of PFAS increase from 0,005 to 0,260 µg/l as the water flows downstream. Two large increases in concentrations have been measured, as can be seen in **Figure 6.1**.

**Figure 6.1:** PFAS concentration profile in surface water along the River Rhine (Moeller et al., 2010)



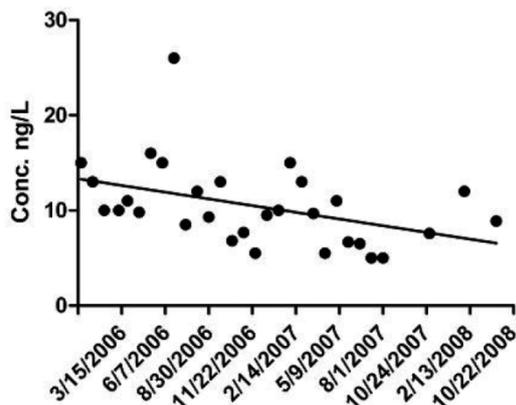
The first sharp increase occurs between station 4 and 5 by a factor of approximately 10 for PFHxS. The source could not be identified, but was likely caused by direct industrial emissions or indirectly via wastewater treatment plant effluents.

The second sharp increase occurs between station 27 and 28. This increase was found to be originating from the effluent of a wastewater treatment plant treating industrial wastewaters near the city of Leverkusen. By the end of 2008 measures had been taken to reduce the discharge of PFBS and PFBA at this wastewater treatment plant, which resulted in concentrations decreasing to about 0,010 µg/l at Station 28 in 2009 (Moeller et al., 2010).

In general, the concentrations PFOS and PFOA were lower in this study compared to earlier studies, but the concentrations of PFBS and PFBA were higher. This might be a result of the decreasing usage of PFOA and PFOS and the replacement of these compounds by the C4-based compounds PFBS and PFBA, although the difference may also be due to a variation in the time of sampling and the exact sampling locations.

Downstream along the River Rhine, at Nieuwegein (NL) (between Kampen and Maassluis in **Figure 6.1**), in the period of 2006-2009, the concentrations of PFOS and PFOA were below 0,030 µg/l for each compound. In this period, the concentrations of PFOS and PFOA show a decreasing trend (**Figure 6.2**).

**Figure 6.2:** Concentration of PFOA (ng/l) in the River Rhine at Lekkanaal, Nieuwegein (NL), sampled in the period of 2006 to 2008 (Eschauzier, 2012).



The River Moehne (Germany), which is a tributary of the River Ruhr, showed the highest concentrations of PFAS. The source of this contamination is related to the accidental release of PFAS via contaminated soil improvers applied on agricultural areas in the Moehne catchment in 2006 (Moeller et al., 2010).

In the River Scheldt (Belgium), the total PFAS concentration increased by a factor of 2.5 downstream of Antwerp (from 0,233 to 0,621 µg/l). Industrial plants located in the harbour area of Antwerp, including a fluorochemical manufacturing facility, have been reported as the likely sources (Moeller et al., 2010).

The mass discharge of PFAS into the European rivers was shown to correlate with the population of the catchment and thus (partly) explains the higher concentrations encountered in populated areas (Eschauzier, 2012).

Ahrens and coworkers (2010) examined the spatial distribution of 15 PFAS in surface water in the North Sea. The highest concentration was found near the coast, whereas the concentrations decreased rapidly from 0,018 to 0,00007 µg/l towards the open North Sea (past the coastal sampling points).

### 6.2.3. Italy

High concentrations of PFOA, with mean concentrations of 0,089 µg/l (Loos et al., 2008) and 0,200 µg/l (McLachlan et al., 2007), have been reported in the River Po, Italy. In a more recent study (Castiglioni, 2014), nine PFCAs and three PFAS have been monitored in the area of Milano. The mass balance of the emissions in the River Lambro basin showed continuously increasing contamination as the water moves downstream. The contamination originated mainly from industrial sources (90%) compared to urban sources. In the Veneto area, high concentrations have been measured, with total PFAS concentrations exceeding 1 µg/l (written question to the European Parliament, 2013).

#### **6.2.4. United Kingdom**

A UK study on the prevalence of PFOS, PFOA and related compounds in 2008 showed that PFOS and PFOA do not appear to be widespread background contaminants of drinking water in England. At sites where specific pollution incidents have occurred, contamination of environmental waters with PFOS has been encountered (Atkinson, 2008).

One of the known incidents in the U.K. occurred on 11 December 2005 at the Hertfordshire Oil Storage Terminal (known as the Buncefield Fire). More than 250.000 liter of AFFF was used to extinguish the fire, resulting in a considerable impact of soil and groundwater with PFAS and oil compounds.

Furthermore in the River Wyre high concentrations of PFOA (0,100 µg/l) have been encountered, and in the River Severn, high concentrations of PFOS have been encountered (0,238 µg/l) (Loos et al., 2009 / JRC).

Generally, minimal work has been done in the UK to understand background levels in groundwater or surface water.

#### **6.2.5. Poland**

A study in Poland reported concentrations of PFOS in rivers, lakes, streams in Poland and in the coastal region of the Baltic Sea. The concentrations varied between < 0,0005 and 0,150 µg/l. PFHxS was also reported (< 0,00025 – 0,110 µg/l) and PFOA occurred in concentrations of <0,0005 to 0,018 µg/l. Long-chained carboxylates could only be found in water of a drainage ditch close to an industrial area (Rostkowski et al., 2009).

## 7. CHEMICAL ANALYSIS METHODS

While a range of standard methods are available for the analysis of PFASs and PFCAs, many PFASs cannot be analysed readily due to the lack of appropriate reference materials. To address this difficulty analysis techniques have been developed whereby PFASs are quantitatively oxidized to fluoride (AOF method), or a mixture of PFASs and PFCAs (TOP method). The TOP method is most sensitive, with a detection limit around 1 ng/l range, vs 1 µg/l for AOF).

Whereas the target levels in groundwater for PFOS can sufficiently and reliably be measured and are above background levels, the AA-EQS of 0,00065 µg/l is so low that it lies beyond the operational range of commercial (and most other) laboratories.

In the subsequent sections a short overview of analytical possibilities and challenges is given. In **Appendix 4** more detail is given on this subject.

### 7.1. OVERVIEW OF STANDARD METHODS

Worldwide there are a variety of standard methods available applicable for the analysis of PFASs and PFCAs, including the international standard ISO 25101:2009(E) for the analysis of PFOS and PFOA. Most of the international available standards are based on liquid chromatography with a tandem MS/MS detector. Since the preparation of the samples starts with sorption of the compounds on an ion exchanger, only compounds with a polar group like the perfluorinated carboxyl acids (PFCAs) and perfluorinated sulfonic acids (PFASs) are captured. The German DIN procedure (HPLC-MS-MS) currently allows for the quantification of the highest number of compounds, and covers the analysis of PFOS and PFOA, and 8 other simple PFASs in soil and groundwater. However, currently (as of 2015) the analysis of up to 23 PFASs based on the DIN standard is offered by various commercial laboratories. The most challenging problem in extending this list is the availability of appropriate standards. In the scientific arena a number of other analytical methods are applied, such as the GC-MS-MS method for the determination of volatile facilitated telomers.

In commercial laboratories, the detection limit is in the range of 0.01 µg/l per compound. Only highly specialized laboratories are able to analyze the PFASs with one order of magnitude lower detection limit.

### 7.2. AOF AND TOP, TWO NEW SUM PARAMETERS

Many compounds used especially in fire extinguishing foams, but also in other industrial branches, are derivatives of the PFCAs or PFASs. Since many PFASs cannot be analysed readily, it is appropriate to consider analysing a “sum parameter”, similar to adsorbable organic halogenated compounds (AOX)<sup>7</sup>.

To determine the total PFAS content, the sum parameter AOF (absorbable organic fluorine compounds) has been developed. This analytical method, based on Combustion Ion Chromatography, is currently undergoing standardization. Because of the relatively high detection limit (1 µg/l fluoride) and the fact that the individual PFASs cannot be separated but show a substantially different toxicological potential, the AOF can only be used as guideline value and cannot be used to replace the

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The AOX (absorbable organic halogens) do not comprise any fluorinated compounds.

analysis of individual compounds. Furthermore, up to now, the correlation which could exist between AOF and PFAS has not been determined.

The other method (total oxidisable precursor, TOP) is an alternative which involves the oxidation of the precursors present in a sample during sample preparation to form PFCAs and PFSAs, which can then be quantified by a conventional analysis. An analysis of the sample with and without these oxidative pretreatment allows an estimate of the precursor content of the sample (Houtz, 2012)

### **7.3. SAMPLING**

Specific precautions have to be taken in the sampling of environmental media since PFAS adsorb strongly to glass. Teflon-containing materials can lead to increased blank values if AOF is analysed, and may also interfere with the analysis by adsorbing PFAS. Currently the most appropriate material for sampling seems to be polyethylene or polypropylene. However, it is not yet clear whether screening at sub- ng/l level is feasible using currently available field sampling techniques.

## 8. SOIL AND GROUNDWATER REMEDIATION

The remedial options available to address PFAS contamination are limited by the unique physico-chemical properties of these compounds. Many remediation methods utilized to address hydrocarbon contamination, such as air stripping, sparging, soil vapour extraction and bioremediation, are ineffective due to the low volatility of these compounds and their resistance to microbial degradation

Technologies currently used for the remediation of PFAS contaminated sites include soil incineration or excavation to landfill (where authorized) and groundwater extraction with PFAS adsorption onto activated carbon or resins. Landfilling or adsorptive techniques do not include a destruction of the PFAS molecules and may lead to leachate issues in the future

Groundwater abstraction volumes may be high if remediation is required to very low environmental quality standards (e.g. for PFOS). Although the degree of sorption of PFAS to sediment is generally low, it can be significant if organic material is present. Sorption of PFAS to sediment, leading to retardation of transport in groundwater, increases with perfluorocarbon chain length and may extend the duration of groundwater extraction. Possible alternative techniques include soil washing, soil solidification and the use of in-situ permeable reactive barriers or funnel and gate systems.

Current best practice disposal routes for spent PFAS adsorption media are high temperature incineration at  $>1000^{\circ}\text{C}$ , or regeneration at a specialist facility.

Emerging water treatment technologies for PFAS, such as photolysis/ photocatalysis, reductive decomposition, advanced oxidation and sonolysis, require high energy input per unit water volume and long residence times. Careful monitoring of treatment performance is also required to ensure complete breakdown of the various PFAS substances that may be present. Consequently, these technologies are unlikely to be feasible for high flowrate, low concentration applications

The following sections provide more information about remediation technologies with proven success or potential for success in the future.

### 8.1. PFAS-IMPACTED SOILS, SUB-SOILS AND SOLID MATERIALS

Currently there are no proven biological or chemical techniques which can cause mineralization of all PFAS. The most recalcitrant PFAS are reported to be PFASs such as PFOS, for which there are no proven methods causing mineralization *in situ*. Precursors and telomers (polyfluorinated compounds) may be broken down by microbial action or using certain chemical oxidants to form perfluorinated compounds as terminal "dead end" daughter products.

Excavation is the most commonly applied treatment method for PFAS impacts in the vadose zone. The excavated soil subsequently has to be placed into a landfill, or to be treated by other technologies. Looking to the future, excavation is not the preferred option for contaminated soil given the challenges faced with managing potential leachate generation or the high costs (financial, environmental) associated with other viable *ex situ* treatment options if the soils are not sent directly to landfill.

### 8.1.1. Landfills

While contaminated soil excavation and disposal to landfill is a remediation option, there may be challenges for the receiving landfill, because PFAS subsequently will become constituents of leachate (due to the high solubility of many PFAS) whereas the standard leachate treatment plants may not be able to effectively treat these substances. This is because they do not biodegrade (Oliaei et al., 2013). Landfills are already a source for release of PFAS to the environment since many consumer products are being placed into landfills at the end of their product life (e.g. impregnated carpets, textiles). Therefore, before sending soil contaminated with PFAS to landfills, checks should be undertaken to confirm that they are appropriately designed and managed so as to prevent further release into the environment.

### 8.1.2. Incineration

Excavated soil could also be treated by high temperature incineration. However, this can have significant cost implications alongside a large energy use requirement. Although PFOS was used as a fire suppressant, its thermal stability is limited (Giesy, 2010). This is based on the ease of cleavage of C-S bonds. However a very stable backbone remains with only C-F and C-C bonds (other PFAS). At 600°C, incineration of PFOS-contaminated material results in many by-products (Yamada and Taylor, 2003). In the same study, at higher temperatures (750 and 900°C) these by-products were not observed. Another study showed that a variety of reaction products can be formed at temperatures below 1.000°C (Yamada et al., 2005). For complete degradation, PFOS has to be destroyed with high temperature incineration at 1.000 – 1.200°C (Schultz, 2003; Yamada et al., 2005).

### 8.1.3. Immobilization (Solidification / Stabilization)

There is another alternative for vadose zone treatment. PFAS-contaminated soils can also be treated *in situ*. In this case, the contaminant will not be removed, but the leachability is reduced by immobilizing the contaminant(s). This can be done via stabilization and/or solidification. To stabilize the contaminant, additives such as activated carbon or other commercial products can be added, e.g. RemBind™ and MatCare™. Das et al., 2013 investigated the adsorption kinetics of PFOS on MatCARE™. This material displayed much faster kinetics (60 minutes) to reach adsorption equilibrium and significantly higher PFOS adsorption capacity (0,093 mmol g<sup>-1</sup>) when compared to a commercially-available activated carbon. Subsequent release of PFOS over an incubation period of 1 year was negligible (0,5-0,6%) (Das et al., 2013). Das et al. 2013 did not investigate the effectiveness of the methodology for other PFAS.

It is also possible to solidify soil with different cement mixtures. Obviously the outcome is no longer a granular geology but a monolith, and the leachate depends upon the type of cement and mixing ratios.

Immobilizing solid materials prior to landfill disposal might also be an option to reduce leachate concentrations.

### 8.1.4. Soil Washing

There is anecdotal evidence that Soil Washing is a possible technique for concentrating PFAS into sludge or washing water. Since the sorption of PFAS is low to moderate, PFAS tend to move to the aqueous phase. A non-reported trial from

DEC contractors (presented during the NICOLE meeting on unconventional contaminants in Manchester, June 2015 [www.nicole.org](http://www.nicole.org)), indicated that a significant part of the soil fraction was cleaned below target levels after two washing cycles. The amount of sludge or GAC that needed to be incinerated or transported to a landfill and the costs were not evaluated.

## 8.2. PFAS-IMPACTED GROUNDWATER

### 8.2.1. Pump and treat

Currently, groundwater extraction is the only viable *in situ* remediation technique to treat PFAS-contaminated water. The technique relies on extraction of water, with subsequent treatment of the water.

Water treatment techniques such as granular activated carbon (GAC), ion exchange and nanofiltration or reverse osmosis have been shown to be effective in removing selected PFAS from water as part of a pump & treat system. A subsequent destruction step such as incineration is required for complete remediation. Of these water treatment techniques, GAC is currently the most commonly applied technology.

#### *Granular Activated Carbon (GAC)*

This technique has been shown to be effective in removing PFOS and PFOA at parts per billion levels from relatively clean water (see **Figure 8.1**). GAC consistently removes PFOS at  $\mu\text{g/l}$  concentrations with an efficiency of more than 90% (Ochoa-Herrera, 2008, Eschauzier, 2011). However, GAC can be inefficient at removing PFOA and other PFAS (Oliaei, 2006). PFAS sorption is lower than organics with similar molecular weights (Qui, 2007), and other co-contaminants will compete for, and preferentially utilize, the adsorptive potential of the GAC media. The sorption velocity is faster for longer-chained PFAS and smaller diameter GAC particles; therefore, GAC that is optimized for PFOS removal will not optimally remove other PFAS (Qui, 2007, Eschauzier, 2011). Adsorption loadings for GAC are relatively low compared to other contaminants, and competition occurs when other contaminants are present.

Other types of adsorbents that have been used for PFAS include powdered activated carbon, polymers, maize straw derived ash, alumina and montmorillonite (Yu et al., 2011; Senevirathna et al., 2010; Hansen et al., 2010; Qu et al., 2009; Yu et al., 2009; Chen et al., 2011; Zhou et al., 2010). Commercial products have been developed for PFAS adsorption claiming better performance for shorter PFAS than conventional GAC. Spent adsorptive media are typically incinerated at high temperature ( $>1000^\circ\text{C}$ ) or thermally regenerated at a specialist facility, thereby adding to the overall management cost.

#### *Ion Exchange Resins*

Ion exchange resins or ion exchange polymers provide a large surface area onto which PFOS can attach. The contaminant removal from water is achieved by the attraction of the negatively charged functional to positively charged functional groups within the resin. The removal is stoichiometric, unlike sorption. A variety of resins containing different functional groups are available. Ion exchange resins are considered suitable for low concentration and high volume water treatment applications. Upon reaching maximum capacity of the resin, regeneration with NaCl solution, ethanol or hot water is possible and would produce a low volume concentrated PFOS waste stream ready for incineration. (Ochoa-Herrera 2008, Du et al., 2014).

For PFOS, different ion exchange resins can be suitable. Sorption using ion-exchange polymers is based on the attraction of the negatively charged functional group of PFOS, and also on the relatively negatively charged tail (due to electron negativity of the fluorine atoms). Non-ion exchange polymers usually show weaker bonding between the adsorbent and adsorbate, which makes regeneration easier and regeneration can occur, for example by solvent washing (Senevirathna et al., 2010). Anion-exchange resins exhibit higher adsorption capacity (Du et al., 2014).

In general sorption capacities decrease in the following order:

**ion-exchange polymers > non-ion-exchange polymers > GAC**

However, at lower concentrations (100 ng/l) non-ion exchange polymers showed higher adsorption capacity than other adsorbents. Adsorption kinetics highlight that GAC and ion-exchange polymers show fast sorption kinetics, much faster than non-ion exchange polymers (Senevirathna et al., 2010).

Chitosan beads have a high adsorption capacity of about 5.5 mmol/g for PFOS mainly due to the formation of micelles in porous materials. Anion-exchange resins show an adsorption capacity of about 4-5 mmol/g for PFOS (Du et al., 2014).

#### *Nano Filtration and Reverse Osmosis*

Nano filtration (NF) and reverse osmosis (RO) are relatively similar processes. Both allow the selective passage of a solvent, while the solutes are retained partially or completely. In a study the NF membranes in general had lower rejections than RO membranes. This was expected as NF membranes have larger pores and thinner rejection layers. Removal efficiencies for NF ranged from 90-99% (Tang, 2007; Schröder, 2010).

The use of RO membranes is a widely accepted filtration technique. Tang (2007) reports on a study of thin film composite polyamide RO membranes, where 99% removal of PFOS was achieved with several types of membranes at concentrations >1 mg/l. RO is normally used in the drinking water industry for removal of PFAS and other contaminants (Tang, 2007).

### **8.2.2. Permeable Reactive Barriers**

There is no experience available with Permeable Reactive Barriers (PRB) or Funnel and Gate systems, but there is no reason why some of the water treatment techniques, as described in the previous paragraph (GAC, Ion Exchange Resins) should not work in a GAC-sand PRB or a Funnel and Gate with exchangeable cassettes. Also other sorptive media like e.g. RemBind™ and MatCare™ might work in these systems. Currently research is being conducted about the applicability of several PRB technologies (e.g. SERDP/ESTCP dossiers ER-2423 and ER-2425).

### **8.3. DEGRADATION OF PFAS**

Research is currently being conducted on methods to achieve degradation of PFAS. A number of the key methods are summarized in this section. However there are still a number of concerns:

- Contaminated media often contain a complex mix of multiple PFAS. Often the amount of precursors is more than significant. Incomplete breakdown may result in an increase in PFCAs or PFSAs, an adverse effect.

- Most research is being conducted using demineralized water instead of environmental samples. Matrix effects can play a large role in the efficiency of treatment processes;
- Research is focused mainly on PFCAs (e.g. PFOA) but less on PFSA (e.g. PFOS), whilst degradation of PFSA is more difficult than PFCAs
- The studies mainly focus on the disappearance of the parent products (e.g. PFOS or PFOA), with less attention given to the reaction products and yield of fluoride.

#### Oxidation

According to Vecitis (2009), PFOS and PFOA oxidation is slow due to the high electronegativity of the fluorine atoms surrounding the carbon chain. They are recalcitrant towards oxidation due to the complete substitution of fluorine (C-F bond) for hydrogen (C-H bond). The perfluorinated backbone of PFOS and PFOA will also reduce the oxidizability of the ionic functional group ( $-\text{SO}_3^-$  for PFOS and  $-\text{CO}_2^-$  for PFOA), since it inductively reduces functional group electron density. Thus the perfluorination of PFOS and PFOA renders these compounds very difficult to degrade by advanced oxidation techniques. The presence of any other dissolved organic compound besides aqueous PFOS and PFOA will competitively inhibit degradation by oxidation, due to its low reaction rate (Buxton, 1988).

Nevertheless, several laboratory studies attest to the feasibility and varying degrees of effectiveness of chemical oxidation for PFOA destruction (Hori et al., 2005, 2008; Ahmad 2012; Hao, 2014). Several variations of oxidation processes using persulfate show promising results for degrading PFOA (Hori et al., 2005, 2008). PFOA was also effectively destroyed by ultraviolet-activated Fenton oxidation (Tang et al., 2012). Although the hydroxyl radical does not degrade PFOA, chemical oxidation systems can be effective in treating PFOA via alternative radical species (Ahmad, 2012). However, these studies focus mainly at treatment of PFOA and have not been validated for treatment of other PFAS too.

A challenge may be the complex composition of contaminated media and the presence of precursors which have large organic functional groups that can be oxidized via conventional oxidative processes (e.g. hydroxyl radical mediated) leaving PFCAs or PFSA.

#### Reduction

Perfluorinated compounds are difficult to defluorinate due to the low reduction potentials of fluorine ( $E < -2,7 \text{ V}$ ). Only the aqueous electron and alkaline metals have lower standard reduction potentials. Sub-critical elemental iron reduction (high temperature, high pressure) has been reported to degrade PFOS. However this is not feasible for *in situ* application.

The solvated electron is a powerful reductant ( $E = -2,87 \text{ V}$ ). Other reduction possibilities include alkaline 2-propanol photolytic reduction and vitamin B12 mediated reduction, however these options are costly (Vecitis, 2009).

#### Sonochemistry

Sonochemistry is the generation of chemical reactions by application of an acoustic field to a solution. High intensity ultrasound creates waves of compression and rarefaction, leading to the production and subsequent collapse of sub-microscopic bubbles. If the bubbles collapse within 1 microsecond and vapour temperatures near  $4.700 \text{ }^\circ\text{C}$  and high pressures are generated, then PFAS will pyrolytically decompose at the bubble-water interface (Moriwaki et al., 2005; Cheng et al., 2008, 2009). The

proposed reaction mechanism is degradation of PFOS due to oxidation after dissociation of the SO<sub>3</sub>-group, which generates PFOA. The PFOA will then undergo shortening of the perfluorocarbon chain caused by repetition of the COO-dissociation (Moriwaki et al., 2005).

In environmental media, in which more compounds are present than in demineralized water, lower degradation rates were observed for sonochemical degradation. For example, in landfill groundwater the degradation rate was reduced by 61% and 56% for PFOS and PFOA respectively, due to the presence of other organic constituents. (Cheng et al., 2008). The lower degradation rate was caused by other organic contaminants, rather than dissolved organic matter. A combined process of ozonation and sonolysis has shown to recover the rate loss for PFOS and PFOA.

Inorganic groundwater constituents also negatively affect PFAS sonochemical kinetics. Cheng and co-workers evaluated the effects of several inorganic species on sonochemical kinetics. It showed that the rate of reduction in the groundwater was primarily due to the presence of bicarbonate. Common cations had negligible effects (Cheng et al., 2009).

#### Photolysis

PFCAs and PFSA's have shown to be very persistent in the environment, there is no solid evidence that these compounds degrade photolytically under natural light conditions. There are references present that show that PFOS, PFOA and PFDA can degrade in the laboratory under circumstances in the UV-C range (Wang et al., 2015). The adsorption is weak up to 220 nm and even lower from 220 to 600 nm.

Adding FeCl<sub>3</sub> increases the applicable absorption region (Jin et al., 2014). In this research, PFOS concentrations decreased below the detection limit within 48 hours. A reaction mechanism was proposed, with intermediates of mainly C<sub>2</sub>-C<sub>8</sub> PFCAs. After 72 hours, 74% of the fluorine could be accounted for, with 58% as free fluoride.

## 9. CONCLUSIONS

- Poly- and perfluoroalkyl substances (PFAS) have been used since the 1970s in a wide range of industrial and commercial products as oil, water and stain repellents and surfactants. Relevant to the refining industry is the use of PFAS in class B (flammable liquid) fire-fighting foams, including Aqueous Film Forming Foam (AFFF), Fluoroprotein (FP) and Film Forming Fluoroprotein Foam (FFFP).
- The unique physical and chemical properties of PFAS mean they are difficult to replace with equally effective substitutes in many products, including class B fire-fighting foams.
- Limited physicochemical and toxicological data is available for many poly- and perfluoroalkyl substances (PFAS) and properties can vary greatly with respect to head group and chain length. Some PFAS have been identified as PBT; persistent, bio accumulative and toxic for humans and wildlife. PFOS and PFOA are the most well-known and studied compounds within this group.
- PFOS was added in 2009 to the Stockholm Convention on Persistent Organic Pollutants. While some PFAS can degrade in the environment, many end-products (including PFOS and PFOA) do not mineralize, making them very persistent. In addition, several PFAS bio-accumulate and many are highly soluble and mobile in the environment.
- PFAS sources to the environment include landfills, waste-water treatment plants, fire-fighting training areas and PFAS manufacturing plants. There are also numerous diffuse sources associated with the use of PFAS in consumer products.
- While there is ongoing debate around the toxicity of PFAS and whether they are carcinogens, there is sufficient evidence to trigger increasing regulatory focus in many parts of the world, including Europe.
- The European Union has set a very low annual average environmental quality standard (AA-EQS) for inland surface water of 0,00065 µg/l, based on the potential for secondary poisoning in humans due to fish consumption. The date set for EU-wide compliance with the AA-EQS is 22<sup>nd</sup> December 2027, with member states required to submit to the Commission a supplementary monitoring programme and a preliminary programme of measures to achieve compliance by 22<sup>nd</sup> December 2018
- Background PFOS concentrations in many European surface water bodies are higher than the AA-EQS, which presents major challenges for compliance. In addition, the analytical methods currently used by commercial laboratories yield quantification limits above or close to the AA-EQS.
- Environmental quality standards vary across EU member states and may encompass a range of other both short and long chain poly- and perfluorinated compounds, with limits set for both individual substances and also the total PFAS concentration.
- Commercial products (including AFFF) may contain PFAS substances for which commercial analysis methods are not yet available, and which may biotransform into PFAS of concern. The potential contribution from such precursor substances can be assessed by pre-treating environmental samples to convert unknown PFAS into a suite of readily analysable PFSAs and PFCAs.
- PFAS in soil and groundwater are currently difficult and expensive to remediate. Options include excavation to landfill for soil (where authorised), and abstraction combined with activated carbon or resin treatment for groundwater. Current best practice disposal routes for PFAS adsorption media are high temperature incineration at >1000°C, or regeneration at a specialist facility. Alternative water treatment techniques, such as sonolysis and advanced chemical oxidation, are being developed that may be more widely used in the future.

*The information provided in the body of the report can be used for risk assessment and evaluation of management options. It must be stressed that this is an active field of research, with regular advances in the science around PFAS toxicity, fate, transport and remediation technologies.*

## 10. GLOSSARY

AA-EQS	Annual Average Environmental Quality Standard
AFFF	aqueous-fire-fighting-foam
AOF	adsorbable organic fluorinated compounds
AOX	adsorbable organic halogens
APFO	perfluorooctanoic acid ammonium salt
BAF	bioaccumulation factor
BCF	bioconcentration factor
BMF	biomagnification factor
CIC	combustion ion chromatography
COM	Committee on Mutagenicity of Chemicals in Food, Consumer Products and the Environment
COPC	constituents of potential concern
ECF	electrochemical fluorination
ECHA	European Chemicals Agency
EPA	Environmental Protection Agency
EQS	environmental quality standards
FTA	fluorotelomer acid
FTOH	fluorotelomer alcohol
FTS	fluorotelomer sulfonic acid (6:2 FTS = H <sub>4</sub> PFOS)
GAC	granular activated carbon
HED	human equivalent dose
HF	hydrogen fluoride
HFA	hexafluoroacetone
Kd	soil distribution coefficient
Koc	organic carbon-water partition coefficient
Kow	octanol-water partition coefficient
IARC	International Agency for Research on Cancer
LOAEL	lowest observed adverse effect level
LOD	limit of detection
LD	lethal dose
MAC	maximum allowable concentration
MCL	maximum contaminant level
MTBE	methyl-tert-butyl ether
NF	nano filtration
NOEC	no observed effect concentration
PAP	polyfluorinated alkyl phosphate
PBT	persistent, bioaccumulative and toxic
PFAS	poly- and perfluoroalkyl substance
PFBA	perfluorobutanoic acid/ perfluorobutanoate
PFBS	perfluorobutane sulfonic acid/ perfluorobutane sulfonate
PFC	perfluorinated compound
PFCA	perfluoroalkyl carboxylic acid
PFDA	perfluorodecanoic acid/ perfluorodecanoate
PFDS	perfluorodecane sulfonic acid/ perfluorodecane sulfonate
PFHpA	perfluoroheptanoic acid/ perfluoroheptanoate

PFHxS	perfluorohexanoic acid/ perfluorohexane sulfonate
PFNA	perfluorononanoic acid/ Perfluorononanoate
PFOA	perfluorooctanoic acid/ perfluorooctanoate
PFOS	perfluorooctanesulfonic acid/ perfluorooctane sulfonate
PFOSA	perfluorooctane sulfonamide
PFPa	perfluorinated phosphonic acid
PFPeA	perfluoropentanoic acid
PFSA	perfluoroalkyl sulfonic acid
pKa	dissociation constant
PNEC	predicted no effect level
POP	persistent organic pollutant
POSF	perfluorooctane sulfonylfluoride
PTFE	polytetrafluoroethylene
REACH	registration, evaluation, authorization and restriction of chemicals
RfD	reference dose
RIVM	Dutch National Institute for Public Health and the Environment
RO	reverse osmosis
RP	reversed phase
SEAC	Committee of Socio-economic Analysis
SPE	solid phase extraction
TDI	total daily intake
TM	telomerization
TOP	total oxidisable precursor
vPvB	very persistent and very bio-accumulating properties
WFD	Water Framework Directive
WWTP	waste water treatment plant

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**APPENDICES CONTENT**

1. Historical uses of PFAS
2. Physicochemical properties
3. Acute and chronic aquatic ecotoxicity of PFOS
4. Chemical Analysis

## **APPENDIX 1 HISTORICAL USES OF PFAS**

Compound	AFFF	Paper industry (food packaging)	Textile industry	Chemical industry	Galvanic industry	Photolithographic industry	Electro industry (semiconductor)
POSF (perfluoro- octanesulfonyl fluoride)  Starter compound for the production of PFOS	X	X	X	X			
PFOS	X AFFF-foam	X Food packaging	X Textile,  Carpets, furniture, outdoor clothing, leather Impregnation	X Oil and gas industry  Polish  Dispersion media Ink Paint Varnish	X Metal and plastic coating, comprising;  Chromium, zinc, gold, copper, nickel, tin, brass, etc.	X Coating of photographic films, papers, printing plates	X
PFOA	X	X		X Polymer-production, Dyes Polishes Adhesives Lubricants		X	
APFO Ammoniumsalt of PFOA						X	
FOSE Perfluorsulfonam idethanol		X	X Fiber finishing	X Electro fluorination	X	X Photographic paper	
FOSA (perfluorooctanes ulfonamido)	X	X Paper equipment	X Leather equipment	X Electro fluorination	X Metal surface treatment Electroplating		X
PFOSE (N- alkylsulfonamido- ethanol)		X Coating of food packaging	X Coating of carpeting, clothing				
PFOSA (Perfluorooctyl- sulfonic acid)		X Paper, cardboard packaging	X Stain repellent  Water repellent Textiles, carpet, leather	X Oil repellent			
PTFE (Teflon)				X			
FTOH (Fluorotelomer alcohols)		X	X Water repellent	X Polymers  Paints Impregnating agents	X		
PAP (Polyfluorinated Alkyl Phosphates)		X Fastfood packaging					
Fluorocarbon resins		X	X				
N-alkyl- substituted perfluorooctan e-sulfonamide						X Photographic paper	
NETFOSA (N-Ethyl perfluorooctane sulfonamide)							
NETFOSE (N-ethyl perfluorooctane sulfonamido- ethanol)		X					

	Medical technology	Cleaning agents	Pesticide industry	Cosmetical industry	Cookware (non-stick)	Aviation industry
POSF (perfluorooctane-sulfonyl fluoride)  Starter compound for the production of PFOS						
PFOS	X Manufacture of video endoscopes	X Alkaline cleaning agents  Detergents Carpet cleaner	X Insecticides	X Cleaning fluids  Shampoos Handcremes		X Hydraulic fluids
PFOA			X Insecticides  Herbicides	X	X Teflon production	
APFO Ammoniumsalt of PFOA						
FOSE Perfluorsulfonamifethanol			X Pesticides			
FOSA (perfluorooctanesulfonamido)		X Alkaline cleaning agents Floor polish				
PFOSE (N-alkylsulfonamido-ethanol)						
PFOSA (Perfluorooctylsulfonic acid)						
PTFE (Teflon)	X Implantates				X	
FTOH (Fluorotelomer alcohols)		X			X	
PAP (Polyfluorinated Alkyl Phosphates)						
Fluorcarbon resins						
N-alkyl-substituted perfluorooctane-sulfonamide	X		X			
NEtFOSA (N-Ethyl perfluorooctane sulfonamide)			X Insecticides			
NEtFOSE (N-ethyl perfluorooctane sulfonamido-ethanol)						

## **APPENDIX 2 PHYSICOCHEMICAL PROPERTIES**

Name	Acronym	CAS Registry Number	Molecular Formula	Molecular Weight [g/mol]	Density <sup>a</sup> (20 - 25 °C) [g/ml]	Water Solubility <sup>b</sup> (20 - 25 °C) [g/L]	Melting Point <sup>a</sup> [°C]	Boiling Point <sup>a</sup> [°C]	Vapor Pressure <sup>b</sup> [Pa]	Henry-Coefficient [Pa·m <sup>3</sup> ·mol <sup>-1</sup> ]	log [Kow] <sup>b</sup> [-]	log K <sub>oc</sub> [L/kg]	K <sub>d</sub> (pH 7)	Dissociation Constant (pKa)
<b>Perfluoroalkyl Carboxylates / Perfluoroalkyl Carboxylic Acids</b>														
Perfluorobutanoic Acid	PFBA	375-224	F(CF <sub>2</sub> ) <sub>3</sub> COOH	214.04	1.65	Miscible	-17.5	121	1307	--	2.82	1.88	--	-0.2 to 0.7
Perfluoropentanoic Acid	PFPeA	2706-90-3	F(CF <sub>2</sub> ) <sub>4</sub> COOH	264.05	1.70	112.6	--	124.4	1057	--	3.43	1.37	--	-0.06
Perfluorohexanoic Acid	PFHxA	307-24-4	F(CF <sub>2</sub> ) <sub>5</sub> COOH	314.06	1.72	21.7	14	143	457	--	4.06	1.91	--	-0.13
Perfluoroheptanoic Acid	PFHpA	375-85-9	F(CF <sub>2</sub> ) <sub>6</sub> COOH	364.06	1.79	4.2	30	175	158	--	4.67	2.19	0.4 - 1.1	-0.15
Perfluorooctanoic Acid	PFOA	335-67-1	F(CF <sub>2</sub> ) <sub>7</sub> COOH	414.07	1.80	3.4 - 9.5	37 - 60	188 - 192	4 - 1300	0.04 - 0.09	5.30	1.31 - 2.35	0 - 3.4	-0.16 to 3.8
Perfluorononanoic Acid	PFNA	375-95-1	F(CF <sub>2</sub> ) <sub>8</sub> COOH	464.08	1.75	9.50	59 - 66	218	1.3	--	5.92	2.39	2.6 - 5.9	-0.17
Perfluorodecanoic Acid	PFDA	335-76-2	F(CF <sub>2</sub> ) <sub>9</sub> COOH	514.09	1.76	9.50	77 - 88	218	0.2	--	6.50	2.76	2.0 - 3.1	-0.17
Perfluoroundecanoic Acid	PFUnA	2058-94-8	F(CF <sub>2</sub> ) <sub>10</sub> COOH	564.09	1.76	0.004	83 - 101	160 - 230	0.1	--	7.15	3.30	12 - 103	-0.17
Perfluorododecanoic Acid	PFDoA	307-55-1	F(CF <sub>2</sub> ) <sub>11</sub> COOH	614.10	1.77	0.0007	107 - 109	245	0.01	--	7.77	--	24 - 269	-0.17 to 0.8
Perfluorotridecanoic Acid	PFTriA	72629-94-8	F(CF <sub>2</sub> ) <sub>12</sub> COOH	664.11	1.77	0.0002	--	--	0.3	--	8.25	--	--	--
Perfluorotetradecanoic Acid	PFTeDA	376-06-7	F(CF <sub>2</sub> ) <sub>13</sub> COOH	714.12	1.78	0.00003	--	276	0.1	--	8.90	--	--	--
Perfluoropentadecanoic Acid	PFPeDA	141074-63-7	F(CF <sub>2</sub> ) <sub>14</sub> COOH	764.12	--	--	--	--	--	--	--	--	--	--
Pentadecafluorooctanoic Acid Ammonium Salt (Ammonium Pentadecafluorooctanoate)	APFO	3825-26-1	C8 H4 NF15 NO2	445.11	--	14.2	157 - 165	--	0.01	--	--	--	--	2.5
<b>Perfluoroalkyl Sulfonates / PFSAs</b>														
Perfluorobutane Sulfonate	PFBS	375-73-5	F(CF <sub>2</sub> ) <sub>3</sub> SO <sub>3</sub> H	300.10	1.81	46.2 - 56.6	76 - 84	211	631	--	3.90	1.00	--	-6.0 to -5.0
Perfluorohexane Sulfonate	PFHxS	432-50-8	F(CF <sub>2</sub> ) <sub>5</sub> SO <sub>3</sub> H	400.11	--	2.3	--	--	58.9	--	5.17	1.78	0.6 - 3.2	-6.0 to -5.0
Perfluoroheptane Sulfonate	PFHpS	357-92-8	F(CF <sub>2</sub> ) <sub>6</sub> SO <sub>3</sub> H	450.12	--	--	--	--	--	--	--	--	--	--
Perfluorooctane Sulfonate	PFOS	1783-23-1	F(CF <sub>2</sub> ) <sub>7</sub> SO <sub>3</sub> H	500.13	--	0.52 - 0.57	54	> 400	6.7	<2e-6 to 3e-4	6.43	2.5 - 3.1	0.1 - 97	-6.0 to -2.6
Perfluorodecane Sulfonate	PFDS	333-77-3	F(CF <sub>2</sub> ) <sub>9</sub> SO <sub>3</sub> H	600.14	--	0.002	--	--	0.71	--	7.66	3.53	--	--
<b>Perfluoroalkyl Phosphonic Acids</b>														
Perfluorobutyl Phosphonic Acid	PFBPA	52299-24-8	F(CF <sub>2</sub> ) <sub>3</sub> P(O)(OH) <sub>2</sub>	350.02	--	14259.1	--	--	0.18	--	2.19	--	--	--
Perfluorohexyl Phosphonic Acid	PFHxPA	40143-76-8	F(CF <sub>2</sub> ) <sub>5</sub> P(O)(OH) <sub>2</sub>	400.03	--	515.3	--	--	0.04	--	3.48	--	--	--
Perfluoroheptyl Phosphonic Acid	PFOPA	40143-78-0	F(CF <sub>2</sub> ) <sub>6</sub> P(O)(OH) <sub>2</sub>	500.05	--	24.5	--	--	0.01	--	4.73	--	--	--
Perfluorodecyl Phosphonic Acid	PFDPA	52299-26-0	F(CF <sub>2</sub> ) <sub>9</sub> P(O)(OH) <sub>2</sub>	600.06	--	0.5	--	--	0.0002	--	5.98	--	--	--
<b>Perfluorooctane Sulfonamide and Derivatives</b>														
Perfluorooctane Sulfonamide	PFOSA	754-91-6	F(CF <sub>2</sub> ) <sub>7</sub> SO <sub>2</sub> NH <sub>2</sub>	499.14	--	-	154 - 155	--	--	--	-	2.5 - 2.62	35 - 56	--
Perfluorooctane Sulfonamidoethanol	FOSE	10116-92-4	F(CF <sub>2</sub> ) <sub>7</sub> SO <sub>2</sub> NH(CH <sub>2</sub> ) <sub>2</sub> OH	543.19	--	0.0009	--	--	0.00	--	5.78	--	--	--
N-Methyl-Perfluorooctane Sulfonamide	N-MeFOSA	31506-32-8	F(CF <sub>2</sub> ) <sub>7</sub> SO <sub>2</sub> NHCH <sub>3</sub>	513.17	--	0.0002	--	--	0.30	--	6.07	3.14	--	--
N-Ethyl-Perfluorooctane Sulfonamide	N-EtFOSA	4151-50-2	F(CF <sub>2</sub> ) <sub>7</sub> SO <sub>2</sub> NHCH <sub>2</sub> CH <sub>3</sub>	527.20	--	0.0001	--	--	0.12	--	6.71	3.23	--	--
N-Methyl-Perfluorooctane Sulfonamidoethanol	N-MeFOSE	24448-09-7	F(CF <sub>2</sub> ) <sub>7</sub> SO <sub>2</sub> NH(CH <sub>3</sub> )(CH <sub>2</sub> ) <sub>2</sub> OH	557.22	--	0.0003	--	--	0.0004	--	6.00	--	--	--
N-Ethyl-Perfluorooctane Sulfonamidoethanol	N-EtFOSE	1691-99-2	F(CF <sub>2</sub> ) <sub>7</sub> SO <sub>2</sub> NH(CH <sub>3</sub> )(CH <sub>2</sub> ) <sub>2</sub> OH	571.25	--	0.0001	55 - 60	--	0.002	--	6.52	--	--	--

Name	Acronym	CAS Registry Number	Molecular Formula	Molecular Weight [g/mol]	Density <sup>a</sup> (20 - 25 °C) [g/ml]	Water Solubility <sup>b</sup> (20 - 25 °C) [g/L]	Melting Point <sup>c</sup> [°C]	Boiling Point <sup>c</sup> [°C]	Vapor Pressure <sup>b</sup> [Pa]	Henry Coefficient [Pa·m <sup>3</sup> ·mol <sup>-1</sup> ]	log Kow <sup>b</sup> [-]	log Koc [L/kg]	Kd (pH 7)	Dissociation Constant (pKa)
<b>Fluorotelomer sulfonic acids</b>														
1H, 1H, 2H, 2H-Perfluorobutanesulfonic Acid	H4-PFBS (2:2 FTS)	149246-63-9	F(CF <sub>2</sub> ) <sub>2</sub> CH <sub>2</sub> CH <sub>2</sub> SO <sub>3</sub> H	228.13	--	-	--	--	--	--	--	--	--	--
1H, 1H, 2H, 2H-Perfluorohexanesulfonic Acid	H4-PFHxS (4:2 FTS)	757124-72-4	F(CF <sub>2</sub> ) <sub>4</sub> CH <sub>2</sub> CH <sub>2</sub> SO <sub>3</sub> H	328.15	--	27.9	--	--	0.33	--	3.21	--	--	--
1H, 1H, 2H, 2H-Perfluorooctanesulfonic Acid	H4-PFOS (6:2 FTS)	27619-97-2	F(CF <sub>2</sub> ) <sub>6</sub> CH <sub>2</sub> CH <sub>2</sub> SO <sub>3</sub> H	428.17	--	1.3	--	--	0.11	--	4.44	--	--	1.31
1H, 1H, 2H, 2H-Perfluorodecane sulfonic Acid	H4-PFDS (8:2 FTS)	39108-34-4	F(CF <sub>2</sub> ) <sub>8</sub> CH <sub>2</sub> CH <sub>2</sub> SO <sub>3</sub> H	528.18	--	0.06	--	--	0.01	--	5.66	0.01	--	1.32
1H, 1H, 2H, 2H-Perfluorododecane sulfonic Acid	H4-PFDS (10:2 FTS)	120226-60-0	F(CF <sub>2</sub> ) <sub>10</sub> CH <sub>2</sub> CH <sub>2</sub> SO <sub>3</sub> H	628.20	--	0.002	--	--	0.001	--	6.91	--	--	--
1H, 1H, 2H, 2H-Perfluorotetradecane sulfonic Acid	H4-PFTS (12:2 FTS)	149246-64-0	F(CF <sub>2</sub> ) <sub>12</sub> CH <sub>2</sub> CH <sub>2</sub> SO <sub>3</sub> H	728.21	--	0.0002	--	--	0.001	--	7.94	--	--	--
<b>Fluorotelomer Alcohols</b>														
Perfluoromethyl ethanol 2:2	2:2 FTOH	54949-74-5	F(CF <sub>2</sub> ) <sub>2</sub> CH <sub>2</sub> CH <sub>2</sub> OH	164.08	--	--	--	--	--	--	--	--	--	--
Perfluoroethyl ethanol 4:2	4:2 FTOH	2043-47-2	F(CF <sub>2</sub> ) <sub>4</sub> CH <sub>2</sub> CH <sub>2</sub> OH	264.09	--	0.98	--	--	214	--	3.30	0.93	--	--
Perfluorohexyl ethanol 6:2	6:2 FTOH	647-42-7	F(CF <sub>2</sub> ) <sub>6</sub> CH <sub>2</sub> CH <sub>2</sub> OH	364.11	--	0.02	-33	172	18.2	5726	4.54	2.43	--	--
Perfluorooctyl ethanol 8:2	8:2 FTOH	865-86-1	F(CF <sub>2</sub> ) <sub>8</sub> CH <sub>2</sub> CH <sub>2</sub> OH	464.12	--	0.0001	45	114	3.98	5039	5.58	3.84	--	--
Perfluorodecyl ethanol 10:2	10:2 FTOH	678-89-8	F(CF <sub>2</sub> ) <sub>10</sub> CH <sub>2</sub> CH <sub>2</sub> OH	564.14	--	0.00001	--	--	0.20	7776	6.63	6.20	--	--
Perfluorododecyl ethanol 12:2	12:2 FTOH	39239-77-5	F(CF <sub>2</sub> ) <sub>12</sub> CH <sub>2</sub> CH <sub>2</sub> OH	664.15	--	--	--	--	--	--	--	--	--	--
<b>Polyfluorinated Alkyl Phosphates</b>														
<b>monoPAP</b>														
4:2 Fluorotelomerphosphatemonoester	4:2 monoPAP	150065-76-2	F(CF <sub>2</sub> ) <sub>4</sub> CH <sub>2</sub> CH <sub>2</sub> OP(O)(OH) <sub>2</sub>	344.07	--	11.9	--	--	0.000	--	1.99	--	--	--
6:2 Fluorotelomerphosphatemonoester	6:2 monoPAP	57678-01-0	F(CF <sub>2</sub> ) <sub>6</sub> CH <sub>2</sub> CH <sub>2</sub> OP(O)(OH) <sub>2</sub>	444.09	--	2.6	--	--	0.000	--	3.39	--	--	--
8:2 Fluorotelomerphosphatemonoester	8:2 monoPAP	57678-03-2	F(CF <sub>2</sub> ) <sub>8</sub> CH <sub>2</sub> CH <sub>2</sub> OP(O)(OH) <sub>2</sub>	544.10	--	0.16	--	--	0.000	--	4.67	--	--	--
10:2 Fluorotelomerphosphatemonoester	10:2 monoPAP	57678-05-4	F(CF <sub>2</sub> ) <sub>10</sub> CH <sub>2</sub> CH <sub>2</sub> OP(O)(OH) <sub>2</sub>	644.12	--	0.01	--	--	0.000	--	5.92	--	--	--
12:2 Fluorotelomerphosphatemonoester	12:2 monoPAP	57678-07-6	F(CF <sub>2</sub> ) <sub>12</sub> CH <sub>2</sub> CH <sub>2</sub> OP(O)(OH) <sub>2</sub>	744.13	--	0.0003	--	--	0.000	--	7.21	--	--	--
<b>diPAP</b>														
4:2 Fluorotelomerphosphatediester	4:2 diPAP	135096-69-0	F(CF <sub>2</sub> ) <sub>4</sub> CH <sub>2</sub> CH <sub>2</sub> OP(O)(OH)OCH <sub>2</sub> CH <sub>2</sub> -	590.15	--	0.0004	--	--	0.000	--	6.16	--	--	--
6:2 Fluorotelomerphosphatediester	6:2 diPAP	57677-95-9	F(CF <sub>2</sub> ) <sub>6</sub> CH <sub>2</sub> CH <sub>2</sub> OP(O)(OH)OCH <sub>2</sub> CH <sub>2</sub> -	790.18	--	8.E-07	--	--	0.000	--	8.41	--	--	--
8:2 Fluorotelomerphosphatediester	8:2 diPAP	678-41-1	F(CF <sub>2</sub> ) <sub>8</sub> CH <sub>2</sub> CH <sub>2</sub> OP(O)(OH)OCH <sub>2</sub> CH <sub>2</sub> -	990.21	--	5.E-10	--	--	0.000	--	10.93	--	--	--
10:2 Fluorotelomerphosphatediester	10:2 diPAP	1895-26-7	F(CF <sub>2</sub> ) <sub>10</sub> CH <sub>2</sub> CH <sub>2</sub> OP(O)(OH)OCH <sub>2</sub> CH <sub>2</sub> -	1190.24	--	2.E-12	--	--	0.000	--	12.88	--	--	--
12:2 Fluorotelomerphosphatediester	12:2 diPAP	57677-99-3	F(CF <sub>2</sub> ) <sub>12</sub> CH <sub>2</sub> CH <sub>2</sub> OP(O)(OH)OCH <sub>2</sub> CH <sub>2</sub> -	1390.27	--	3.E-15	--	--	0.000	--	15.15	--	--	--
<b>Polytetrafluoroethylene (Teflon)</b>														
	PTFE	9002-84-0	(CF <sub>2</sub> ) <sub>n</sub>	--	--	--	327°C (Decomposes at 260°C)	--	--	--	--	--	--	--

Notes

Blank font indicates information from published literature sources.

Blue font indicates chemical formulas.

Red font indicates parameters estimated with published equations. Calculated parameters are based on the neutral form of the substances (and not the conjugate base, which predominates for some PFAS at neutral pH)

-- No data or not applicable.

<sup>a</sup> CAS database at <http://www.chemicalbook.com>

<sup>b</sup> Wang, et al., 2011.

Unless otherwise indicated, all parameter values obtained from literature sources listed separately.

**APPENDIX 3 ACUTE AND CHRONIC AQUATIC ECOTOXICITY OF PFOS  
(TABLES FROM THE PFOS EQS DOSSIER, 2011)**

ACUTE EFFECTS			Master reference
Algae & aquatic plants (mg.l <sup>-1</sup> )	Freshwater	<i>Selenastrum capricornutum</i> /96 h EC <sub>50</sub> : 71mg/l and 126mg/l	Environment Agency,2004
		<i>Selenastrum capricornutum</i> /96h EC <sub>50</sub> : 48.2mg/l *	Environment Agency,2008
		<i>Pseudokirchneriella subcapitata</i> / 72 h EC <sub>50</sub> : 120 mg/l	OECD, 2002 in RIVM 2010
		<i>Navicula pelliculosa</i> / 96 h EC <sub>50</sub> : 283 mg/l	OECD, 2002 in RIVM 2010
		<i>Chlorella vulgaris</i> /96h EC <sub>50</sub> : 81.6 mg/l	Environment Agency,2004 Boudreau et al, 2003b in RIVM 2010
		<i>Anabaena flos-aquae</i> / 96h EC <sub>50</sub> : 176 mg/l	Environment Agency,2004 OECD, 2002 in RIVM 2010
		<i>Lemna gibba</i> / 7d EC <sub>50</sub> : 31.1mg/l	Environment Agency,2004 Boudreau et al, 2003b in RIVM 2010
	Marine	<i>Skeletonema costatum</i> /96 h EC <sub>50</sub> : >3.2mg/l	Environment Agency,2004
Invertebrates (mg.l <sup>-1</sup> )	Freshwater	<i>Daphnia magna</i> / 48 h EC <sub>50</sub> : 27 mg/l	Environment Agency,2004
		<i>Daphnia magna</i> / 48 h EC <sub>50</sub> : 4 mg/l **	Environment Agency,2008
		<i>Daphnia magna</i> / 48 h EC <sub>50</sub> : 48 mg/l (geometric mean of 6 values)	OECD, 2002, Boudreau et al, 2003b, Ji et al 2008, and Li, 2009 in RIVM 2010
		<i>Daphnia pulicaria</i> / 48 h EC <sub>50</sub> : 124 mg/l	Boudreau et al, 2003b in RIVM 2010
		<i>Moina macrocopa</i> / 48 h EC <sub>50</sub> : 18 mg/l	Ji et al, 2008 in RIVM, 2010
		<i>Neocaridina denticulate</i> / 96 h EC <sub>50</sub> : 9.3 mg/l	Li, 2009 in RIVM 2010
		<i>Dugesia japonica</i> / 96 hr LC <sub>50</sub> : 18 mg/l (geometric mean of two values)	Li, 2008 and Li, 2009 in RIVM 2010
		<i>Physa acuta</i> / 96 hr LC <sub>50</sub> : 165 mg/l	Li, 2009 in RIVM 2010
		<i>Unio complamatus</i> / 96 hr LC <sub>50</sub> : 59 mg/l	Environment Agency,2004 OECD, 2002 in RIVM 2010
	Marine	<i>Mysid shrimp (Americamysis bahia)</i> / 96 h EC <sub>50</sub> : 3.6mg/l	Environment Agency,2004 OECD, 2002 in RIVM 2010
		<i>Brine shrimp (Artemia spp)</i> / 48hr LC <sub>50</sub> : 8.9 mg/l	Environment Agency,2004
		<i>Artemia spp</i> / 48 hr LC <sub>50</sub> : 8.3 mg/l	OECD, 2002 in RIVM 2010
		<i>Crassostrea virginica (Eastern oyster)</i> 96hr EC50 >3.0mg/l (Shell deposition)	Wildlife international (2000) referenced in OECD 2002
	Sediment	No data	

Fish (mg.l <sup>-1</sup> )	Freshwater	<i>Fathead minnow (Pimephales promelas)</i> /96 h EC <sub>50</sub> : 4.7mg/l ***	Environment Agency,2004
		<i>Fathead minnow (Pimephales promelas)</i> /96h LC50: 9.5mg/l	Environment Agency,2008
		<i>Pimephales promelas</i> / 96 h LC <sub>50</sub> : 6.6 mg/l (geometric mean of two values)	OECD, 2002 in RIVM 2010
		<i>Bluegill sunfish (Lepomis macrochirus)</i> / 96 h LC <sub>50</sub> : 6.9 mg/l	Environment Agency,2004
		<i>Lepomis macrochirus</i> / 96 h LC <sub>50</sub> : 6.4 mg/l	OECD, 2002 in RIVM 2010
		<i>Oncorhynchus mykiss</i> / 96h LC <sub>50</sub> : 7.8mg/l	Environment Agency,2008
		<i>Oncorhynchus mykiss</i> / 96 h LC <sub>50</sub> : 13 mg/l (geometric mean of two values)	OECD, 2002 in RIVM 2010
	Marine	<i>Sheepshead minnow (Cyprinodon variegatus)</i> / 96hr EC <sub>50</sub> : >15mg/l	Environment Agency,2004
	<i>Oncorhynchus mykiss</i> / 96h LC50: 13.7mg/l	Environment Agency,2004 OECD, 2002 in RIVM 2010	
Other taxonomic groups			

\* Noted that this study should be considered with care as it is based on nominal concentrations and the study duration is longer than the recommended test duration.

\*\* This value was generated in a static system with nominal concentrations and therefore the data should be treated with care.

\*\*\* This study was conducted in a static system with nominal test concentrations and should therefore be treated with care.

CHRONIC EFFECTS			Master reference
Algae & aquatic plants (mg.l <sup>-1</sup> )	Freshwater	<i>Selenastrum capricornutum</i> /96h EC <sub>10</sub> : 5.3mg/l *	Environment Agency, 2008
		<i>Lemna gibba</i> /7d NOEC: 15.1mg/l	Environment Agency,2004
		<i>Lemna gibba</i> /42d EC <sub>10</sub> : 0.2mg/l **	Environment Agency,2008
		<i>Chlorella vulgaris</i> / 96h EC <sub>10</sub> : 8.2mg/l	Environment Agency,2008 Boudreau et al, 2003b in RIVM, 2010
		<i>Navicula pelliculosa</i> / 96 h NOEC: 44mg/l	Environment Agency,2004 OECD, 2002 in RIVM 2010
		<i>Rhapidocelis subcapitata</i> /96h EC <sub>10</sub> : 53mg/l	OECD, 2002 in RIVM, 2010
		<i>Anabaena flos-aqua</i> /96h NOEC: 44mg/l	OECD, 2002 in RIVM, 2010
		<i>Lemna gibba</i> /7d EC <sub>10</sub> : 6.6mg/l	Environment Agency,2008 Boudreau et al., 2003b in RIVM, 2010
		<i>Myriophyllum sibiricum</i> / 42 d NOEC: 0.092mg/l	Hanson et al, 2005 in RIVM 2010
		<i>Myriophyllum spicatum</i> / 42 d NOEC: 3.2mg/l	Hanson et al, 2005 in RIVM, 2010
	Marine	<i>Skeletonema costatum</i> /96h NOEC : >3.2mg/l	Environment Agency,2004 OECD, 2002 in RIVM, 2010
Invertebrates (mg.l <sup>-1</sup> )	Freshwater	<i>Daphnia magna</i> / 21 d NOEC : 12 mg/l	Environment Agency,2004
		<i>Daphnia magna</i> /28d NOEC: 7mg/l ***	Environment Agency,2004
		<i>Daphnia magna</i> /21d NOEC: 5.3mg/l ***	Environment Agency,2004
		<i>Daphnia magna</i> / 21/28 d NOEC: 7.0 mg/l (geomean of 4 values)	Boudreau et al, 2003b, OECD, 2002 and Ji et al, 2008 in RIVM, 2010
		<i>Moina macrocopa</i> / 7 d EC <sub>10</sub> : 0.40mg/l	Ji et al, 2008 in RIVM 2010
		<i>Chironomus tentans</i> / 10d NOEC: 0.049mg	Environment Agency,2008
		<i>Chironomus tentans</i> / 36d NOEC: 0.049mg <0032mg/l LOEC with 32% effect	MacDonald et al, 2004 in RIVM, 2010
		<b><i>Chironomus tentans</i> / 36d</b> <b>NOEC: &lt;0.002mg</b> LOEC 0.002mg/l	MacDonald et al, 2004 in RIVM, 2010
		<i>Enallagma cyathigerum</i> / 120 d NOEC: <0.01mg/l LOEC with 18% effect	Bots et al, 2010 in RIVM 2010

	<b>Marine</b>	<i>Mysidopsis bahia</i> / 35 d NOEC : 0.25mg/l	Environment Agency,2004 OECD, 2002 in RIVM 2010
	<b>Sediment</b>	No data	
<b>Fish (mg.l<sup>-1</sup>)</b>	<b>Freshwater</b>	Fathead minnow ( <i>Pimephales promelas</i> ) / 42d NOEC : 0.3mg/l	Environment Agency,2004
		Fathead minnow ( <i>Pimephales promelas</i> ) / 21d NOEC: 0.028mg/l	Environment Agency,2008 Ankley et al, 2005 in RIVM, 2010
		<i>Oryzias latipes</i> / 14 d NOEC: <0.01mg/l LOEC with 80% effect	Ji et al, 2008 in RIVM, 2010
		Bluegill sunfish ( <i>Lepomis macrochirus</i> ) / 62d NOEC: <0.87mg/l	
	<b>Marine</b>	No data	
<b>Other taxonomic groups</b>		<i>Xenopus leavis</i> / 96 h NOEC: 5.0mg/l	

\*Noted that the algal study needs to be treated with care as based on nominal concentrations and also of 96hr duration rather than the test recommendation of 72 hrs.

\*\* Noted that this data generated in an outdoor microcosm study and the study details are incomplete

\*\*\* Noted that these studies were undertaken with nominal concentrations and therefore should be treated with care. Lowest valid datapoint is 12 mg/l.

## APPENDIX 4 ANALYICAL METHODS

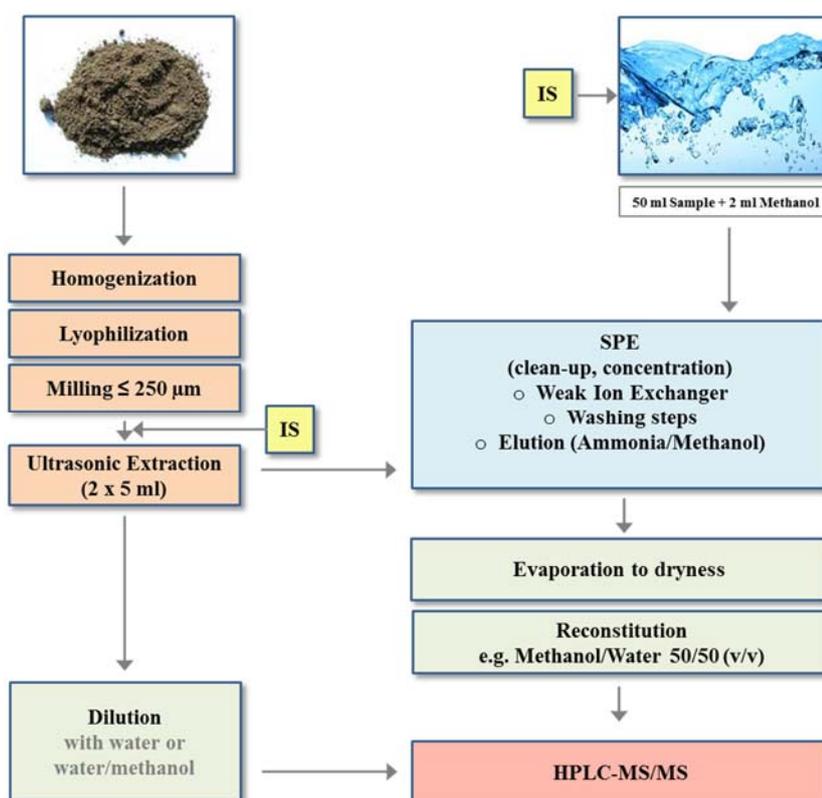
### Overview of standard methods

Worldwide there are a variety of methods available applicable for the analysis of PFAS including the international standard ISO 25101:2009(E) (Water quality – Determination of perfluorooctanesulfonate (PFOS) and perfluorooctanoate (PFOA) - Method for unfiltered samples using solid phase extraction and liquid chromatography/mass spectrometry). However, this method is applicable exclusively for the analysis of PFOS and PFOA. The US-standard (Method 537: Determination of selected perfluorinated alkyl acids in drinking water by solid phase extraction and liquid chromatography / tandem mass spectrometry (LC/MS/MS); EPA/600/R08/092) is applicable to analyse a large number of perfluorinated carboxyls and sulfonates. All methods are based on liquid chromatography with a tandem mass selective detection. The German standard (DIN-Method) currently allows the quantification of the highest number of contaminants:

- Water: DIN 38407-42:2011-03 (F 42) Analysis of selected perfluorinated compounds (PFC) in water – Method via high performance liquid chromatography – tandem mass spectrometry (HPLC-MS/MS) after solid phase extraction (DIN, 2011a);
- Soil: DIN 38414-14 (S14) Analysis of selected perfluorinated compounds (PFC) in sludge, compost and soil – Method via high performance liquid chromatography – tandem mass spectrometry (HPLC-MS/MS) (S14) (DIN, 2011b).

The S14-method is suitable for sediments, sewage sludge, compost, and soil.

**Figure 1. Analysis procedure according to DIN 38407-42:2011-03 (IS = Internal Standard)**



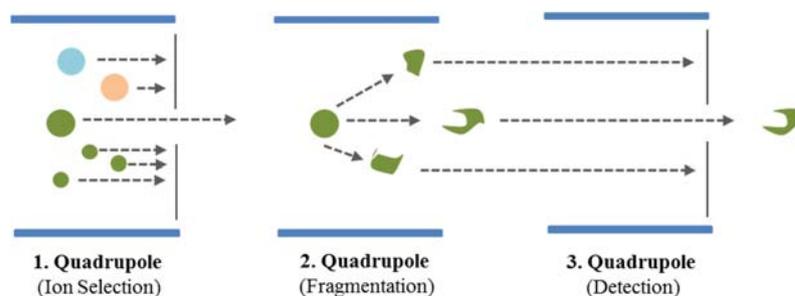
The general analytical procedure is shown in **Figure 1**. It consists in three steps: SPE-enrichment (*Solid Phase Extraction*), HPLC-separation and MS-MS-detection (DIN, 2011a). It is designed for the analysis of polar, low concentrated contaminants.

By selecting the appropriate solid (here: weak anion exchanger) for the SPE, it is possible to restrict the analysis to polar, negatively charged non-volatile substances, which bind to the ion exchange cartridge. The SPE serves to select and concentrate the contaminant and reduce matrix interference by dirty matrices. Other PFAS with no polar groups in the molecule cannot be detected with this analysis because they do not sorb to the ion exchanger during sample preparation.

It should be remarked that milling and ultrasonic extraction during sample preparation for solids analysis could destroy the PFAS. While this process is time-dependent (the destruction rate increases with time) and may not be significant, it may result in an underestimation of the true PFAS concentration.

The separation happens by Reversed Phase (RP) fluid-chromatography. The RP consists of alkyl chains covalently bonded to silica gel. The retention time of a substance depends on the retention in the stationary phase. The factor that limits the velocity of the process is the desorption back into the mobile phase. The branched isomers - especially occurring in PFOA, PFHxS and PFOS - usually elute just before the unbranched substances. For PFOS, several branched isomers are detected (DIN, 2011a).

**Figure 2. MS-MS-coupling principle**



The identification and quantification is conducted using the very selective and sensitive Negative-Ions Electrospray Tandem Mass spectrometry (ESI-MS-MS) (**Figure 2**) (Theobald et al., 2007). Mass spectrometry is a method to measure the mass/charge ratio ( $m/z$ ) of ions.

The ESI-Interface is the connection between the standard HPLC-system and the Tandem-MS. The mixed sample (liquid) is nebulized and becomes ionized in an electrical high voltage field. The Tandem-MS usually comprises three quadrupoles, although the measurement is done only in the first and the third. The central quadrupole (collision cell) is used for the fragmentation of the selected analyte. The quadrupole separation systems consist of four bar magnets. By applying electrical potential, the molecules with a precise mass (here: the molecules to be analysed) are accelerated, guided through the gap between the bars and filtered out. By doing so, undesired ions can be neutralized and therefore will not be detected. By changing the electrical field, the whole spectrum can be scanned using the first of the two MS systems. The ion to be analysed is then led to a collision cell, in which the molecule is energized by colliding with an inert gas like  $N_2$  or argon. In this process, the ion is split into lighter, very specific ions, which are identified in the second mass spectrometer.

It is possible to reconstruct the structure of the analyte from the pattern of the different mass fragments. **Figure 3** shows the parental compounds and the most important product ions generated in each ionization step for two examples (PFOA and PFOS). Only one product ion is obtained for the compound PFBA and the intensity of the second product ion for the compounds PFPeA and PFHxA is too low for reliable identification. The first production step serves to identify, the second to quantify the product ions.

**Figure 3. Example of typical fragmentation of PFOA and PFOS (DIN, 2011a)**

Compound	Initial Ion	1. Product-Ion	2. Product-Ion
PFOA	$\text{F}-\text{CF}_2-\text{CF}_2-\text{CF}_2-\text{CF}_2-\text{CF}_2-\text{CF}_2-\text{CF}_2-\text{CF}_2-\text{C}(=\text{O})\text{O}^-$ $m/z = 412,97$	$\text{F}-\text{CF}_2-\text{CF}_2-\text{CF}_2-\text{CF}_2-\text{CF}_2-\text{CF}_2-\text{CF}_2-\text{C}^+(\text{F})_2$ $m/z = 368,98$	$\text{F}-\text{CF}_2-\text{CF}_2-\text{C}^+(\text{F})_2$ $m/z = 168,99$
PFOS	$\text{F}-\text{CF}_2-\text{CF}_2-\text{CF}_2-\text{CF}_2-\text{CF}_2-\text{CF}_2-\text{CF}_2-\text{CF}_2-\text{CF}_2-\text{S}(=\text{O})_2\text{O}^-$ $m/z = 498,93$	$\text{S}(=\text{O})_2\text{O}^-$ $m/z = 79,96$	$\text{F}-\text{S}(=\text{O})_2\text{O}^-$ $m/z = 98,96$

The HPLC-MS/MS method is suitable to analyse the PFAS (shown in blue in **Table 4**) in drinking water, groundwater, and surface water with a detection level of 0.01 to 0.015 µg/l per compound. In treated wastewater the detection limit for PFAS analysed in commercial laboratories analysed is 0.025 µg/l. The detection limit is based on the health protection precautionary values for drinking water. In soil samples, detection limits of 2 µg/kg dry weight are reached in most cases. This value is substantially below the threshold value for the sum of PFOA and PFOS in sewage sludge (100 µg/kg DW), which cannot be exceeded in case of agricultural use.

The method can potentially be used for other water types - for example untreated water - but the applicability needs to be checked for each individual case (DIN, 2011a). The same applies to the solid analysis. While the method could be suitable for other sample materials, for example fertilizers, but this needs to be tested for the individual case.

In association with the solid phase extraction, the method can basically be used for other materials with a polar functional group in the molecule, for example the compounds PFOA, PFDoA, PFHpS, PFDS and H4PFOS. At present, the 23 compounds listed in **Table 1** can be analysed in commercial laboratories.

In addition, several other polar compounds can be analysed with this method. One example are the Telomer acids (degradation metabolites of Telomer alcohols) (Bayerisches Landesamt für Umwelt, 2012). However, this application has not yet been used as a commercial analysis method.

**Table 1. Analysed compounds (HPLC-MS/MS-method), available standards and limit of detection (LOD) HPLC-MS/MS and GC-MS (DIN, 2011a) (Bayerisches Landesamt für Umwelt, 2012). (the compounds explicitly named in the DIN norm are shown in blue, compounds where no internal standards are applicable are shown in red)**

Compound	Symbol	Internal/External Standard	LOD (Water) [µg/L]	LOD (Soil) [µg/kg]
Perfluoro-n-butyric acid	PFBA	<sup>13</sup> C <sub>4</sub> -PFBA	0,01	2
Perfluoro-n-pentanoic acid	PFPeA	<sup>13</sup> C <sub>4</sub> -PFHxA	0,01	2
Perfluoro-n-hexanoic acid	PFHxA	<sup>13</sup> C <sub>2</sub> -PFHxA	0,01	2
Perfluoro-n-heptanoic acid	PFHpA	<sup>13</sup> C <sub>4</sub> -PFOA	0,01	2
Perfluoro-n-octanoic acid	PFOA	<sup>13</sup> C <sub>4</sub> -PFOA	0,01	2
Perfluoro-n-nonanoic acid	PFNA	<sup>13</sup> C <sub>4</sub> -PFOA	0,01	2
Perfluoro-n-decanoic acid	PFDA	<sup>13</sup> C <sub>2</sub> -PFDA	0,01	2
Perfluoro-n-undecanoic acid	PFUnA	<sup>13</sup> C <sub>2</sub> -PFUnA	0,01	2
Perfluoro-n-dodecanoic acid	PFDoA	<sup>13</sup> C <sub>4</sub> -PFOA	0,01	2
Perfluoro-n-tridecanoic acid	PFTrA	<sup>13</sup> C <sub>4</sub> -PFOA	0,01	2
Perfluoro-n-tetradecanoic acid	PFTA	<sup>13</sup> C <sub>4</sub> -PFOA	0,01	2
Perfluoro-n-butyrsulfonic acid	PFBS	<sup>13</sup> C <sub>4</sub> -PFBA	0,015	3
Perfluoro-n-hexansulfonic acid	PFHxS	<sup>13</sup> C <sub>4</sub> -PFOS	0,015	3
Perfluoro-n-heptansulfonic acid	PFHpS	<sup>13</sup> C <sub>4</sub> -PFOS	0,01	2
Perfluoro-n-octansulfonic acid	PFOS	<sup>13</sup> C <sub>4</sub> -PFOS	0,01	2
Perfluoro-n-decansulfonic acid	PFDeS	<sup>13</sup> C <sub>4</sub> -PFOA	0,01	2
1H,1H,2H,2H-Perfluoro-n-octansulfonic acid	H4PFOS (6:2FTS; H.H PFOS)	<sup>13</sup> C <sub>4</sub> -PFOS	0,01	2
Perfluorooctansulfonamide	PFOSA	<sup>13</sup> C-MeFOSA	0,01	2
1H,1H,2H,2H-Perfluoro-n-decansulfonic acid	H4-PFDeS (8:2FTS)	<sup>13</sup> C <sub>4</sub> -PFOS	0,01	2
2H,2H-Perfluorodecanoic acid	H2PFDA	<sup>13</sup> C <sub>4</sub> -PFOS	0,01	2
7H-Dodecafluoroheptanoic acid	HPFHpA	<sup>13</sup> C <sub>4</sub> -PFOA	0,01	2
Perfluoro-3,7-dimethyloctanoic acid	PF37DMOA	<sup>13</sup> C <sub>4</sub> -PFOA	0,01	2
2H,2H,3H,3H-Perfluoroundecanoic acid	H4PFUnA	<sup>13</sup> C <sub>4</sub> -PFOA	0,01	2
2H,2H-Perfluorohexanoic acid	4:2 FTCA	<sup>13</sup> C <sub>2</sub> -4:2-FTCA	n.s.	n.s.
2H,2H-Perfluorooctanoic acid	6:2 FTCA	<sup>13</sup> C <sub>2</sub> -6:2-FTCA	n.s.	n.s.
2H,2H-Perfluorodecanoic acid	8:2 FTCA	<sup>13</sup> C <sub>2</sub> -8:2-FTCA	n.s.	n.s.
2H,2H-Perfluorododecanoic acid	10:2 FTCA	<sup>13</sup> C <sub>2</sub> -10:2-FTCA	n.s.	n.s.
2-Perfluorohexylethanol	6:2 FTOH	<sup>13</sup> C <sub>2</sub> D <sub>2</sub> -6:2-FTOH	n.s.	n.s.
2-Perfluorooctylethanol	8:2 FTOH	<sup>13</sup> C <sub>2</sub> D <sub>2</sub> -8:2-FTOH	n.s.	n.s.
2-Perfluorodecylethanol	10:2 FTOH	<sup>13</sup> C <sub>2</sub> D <sub>2</sub> -10:2-FTOH	n.s.	n.s.
2H,2H-Perfluorodecylacrylate	8:2 FTA	8:2 FTA	n.s.	n.s.
2H,2H-Perfluorodecylmethacrylate	8:2 FTMA	8:2 FTMA	n.s.	n.s.
2H,2H-Perfluorooct-1-en	6:2 FTen	6:2 FTen	n.s.	n.s.
2H,2H-Perfluorodec-1-en	8:2 FTen	8:2 FTen	n.s.	n.s.

n.s. = not specified

## Analysis methods for fluorinated precursors

Today, a number of precursors that have been identified in AFFF can be analyzed by HPLC-MS/MS-Method. However, the biggest challenge for any precursor analysis is the availability of standards. Once they are available, this methods will be the best way to analyse most of the PFAS (Backe et la, 2013).

On the other hand, telomer alcohols and other nonpolar PFAS cannot be analysed with the HPLC-MS/MS-Method, because they cannot be concentrated using solid phase extraction. For these compounds gas chromatography (GC-MS) is suitable, as previous experiences with wastewater have demonstrated. The extraction and concentration can be achieved with a good recovery rate by using high purity MTBE (liquid/liquid-extraction). The detection limits were 0,06 µg/l (6:2 FTOH), 0,3 µg/l (8:2 FTOH) und 0,6 µg/l (10:2 FTOH). With a sensitive mass spectrometer these detection limits can be further improved. The lower detection limits refer to the evaluation of mass fragments 31 m/z, which represent the highest signal in the mass spectrum. The disadvantage is that the fragment is less selective for the fluorotelomere alcohols than bigger fragments or fragments containing fluorine. Therefore, in case of particularly complex matrices, it is recommended to choose a different fragment. Since the matrix of a wastewater sample can have very different characteristics depending on its origin, a mass labelled "standard" should be used to correct analyte material losses during the sample preparation (Marzinkowski et a., 2013).

In addition to this, a Headspace-GC-MS-method has been developed, which can be used without an enrichment step. The detection limit is 0,01 µg/l for each compound. The GC-PCI-MS (gas chromatography positive chemical ionization and tandem mass spectrometry) is also suitable as a robust analytical method for volatile compounds, like FTOH, PFOSE and PFOSA (Reagen 2009). After extraction with DCM (or with methanol) the sample clean-up can be performed via SPE. Concentration of the sample should be avoided. If necessary, the sample should be filtered with a cellulose filter (0,45 µm) (Reagen 2009). Some of the compounds which can be analysed by GC-PCI-MS are listed below:

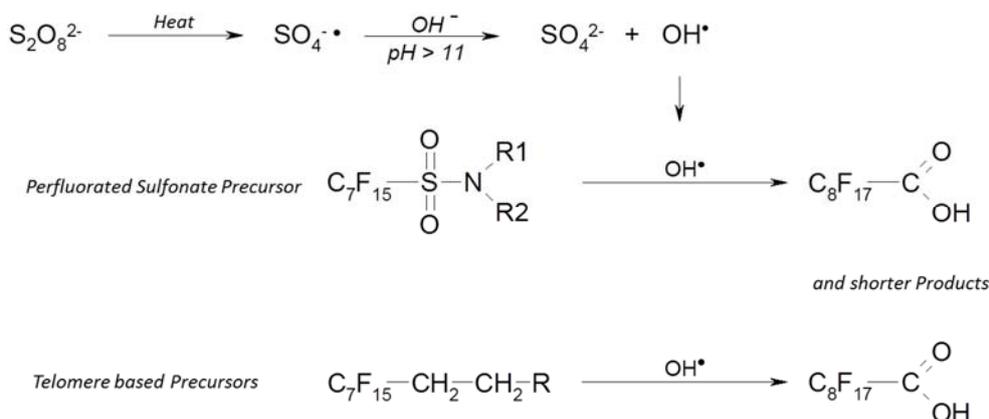
N-MeFOSA	(N-Methylperfluoro-1-octansulfonamide)
N,N-Me2FOSA	(N,N-Dimethylperfluoro-1-octansulfonamide)
4:2 FTOH	2-Perfluorobutylethanol
6:2 FTOH	2-Perfluorohexylethanol
8:2 FTOH	2-Perfluorooctylethanol
10:2 FTOH	2-Perfluorodecylethanol
7:2sFTOH	(1-Perfluoroheptylethanol)

Volatile PFAS - for example telomere alcohols - may also be present in the soil vapour. Up to now, no analytical methods for gas analysis were published. These could be developed based on the sampling method "*low flow sampling on Polyurethane foam in conjunction with a glass fiber filter in a stainless steel cartridge*". This procedure is described for the measurement of perfluorinated acids (Bayerisches Landesamt für Umwelt, 2012).

### Precursor Oxidation

A precursor by definition is a compound that has the potential to form PFCAs or PFSA. Precursor oxidation with hydroxyl radicals is a sample preparation step that allows for quantification of unknown precursors (Houtz and Sedlak, 2012). The hydroxyl radicals are generated by thermolysis of persulfate in a basic medium. The generated radicals lead to the elimination of all functional groups and non-fluorinated residuals to form perfluorocarboxylic acids, which are readily analysed using standard methods.

**Figure 4. Analysis of the total mass of oxidizable precursors**



The sample is analysed before and after oxidation and the change in concentration of PFCAs is indicative of the total precursor composition. Additional research is needed to determine if all precursors are transformed to perfluorocarboxylic acids during the high temperature oxidation; however, 62-100% of precursors in known formulations of AFFF are converted (Houtz and Sedlack, 2012). It may be possible to check the effectiveness of the preparation step using analysis of total fluorine.

### Adsorbable organic fluorinated compounds

Since many PFAS cannot be analysed with a reasonable effort, it is particularly interesting to analyse a “sum parameter”, similar to adsorbable organic halogenated compounds (AOX)<sup>8</sup>. The AOF-method (AOF: adsorbable organic fluorinated compounds), currently undergoing the standardisation process, is based upon the sorption of the fluorinated compounds on synthetic activated carbon with low fluorine content. The carbon is completely burnt without any soot production in presence of water at a temperature of 950 – 1000°C (hydropyrolysis). The combustion gases (HF, CO<sub>2</sub> and others) are adsorbed in a neutral or basic solution, which is injected into an ion chromatograph. The analysis is performed on fluoride. This method, called *Combustion Ion Chromatography* (CIC) (Wagner et al, 2013; Lange, 2014), achieves a detection limit of 1,0 µg/l Fluorine. This corresponds to a detection limit of 0,64 µg/l, with respect to PFOS only. Currently this method cannot be used for soil samples, but can be applied to extracts of soil samples.

It is assumed that the new AOF-Method, once published, will rapidly be established in the commercial laboratories, even though few laboratories have the required analytical instrumentation. Once established, the analysis price will likely drop, and this method could become a routine screening analysis. At present this analysis is probably going to be performed on selected samples only, with the intent to check if further PFAS are present in addition to the 23 commercially analysable compounds.

Up to now it could still not be determined if a correlation between AOF and PFAS actually exists. Furthermore, since not all PFAS can be analysed, verification of a potential correlation is only possible to a limited extent.

<sup>8</sup>

The AOX (adsorbable organic halogens) do not comprise any fluorinated compounds.

It should be noted that for the corresponding sum parameter AOX the halogenides bind to the organic polymer matrix in water with a high organic load (e.g. landfill leachate). As a consequence, the total AOX-value is very high, but this value does not correspond to the sum of the single compounds. A similar behaviour is expected for PFAS.

Furthermore, several other analytical methods for the quantification of fluorinated compounds exist. However, these are applied only in the research field (Gruber, 2011).

## sampling

### Sample Collection

For water sampling, teflon tubing should be avoided. Although high purity teflon tubing does not cause "blank contamination" in contrast to common teflon tubing, some researchers have found that Teflon could sorb PFAs. Usually polypropylen bottles rinsed with methanol and with PE screw caps are suitable for sampling; a minimum of 50 ml sample is needed to conduct the analysis.

If samples are taken for the of AOF it is very important to verify that the blank samples are clean. Therefore all materials containing PTFE are excluded, since suitable materials have not only to be PFAS-free, but also fluorine free. The use of silicone tubing for sampling and as gasket material is recommended.

The samples should be stored at the most for two weeks at a temperature of approximately 4°C. A longer storage time could lead to adsorption of compounds to the container and thus to losses (DIN, 2011a). By adding 5 Vol.-% Methanol to the samples, the losses by adsorption can be reduced. The sample dilution caused by the addition of methanol needs to be considered in the results interpretation.

For volatile compounds (e.g. FTOH) gas tight lockable glass bottles should be used and completely filled, avoiding a gas phase to be present in the full bottle. The samples have to be stored at a temperature of 4°C. Tests have shown that already after 24 hours of storage 10% loss could occur. (Bayerisches Landesamt für Umwelt, 2012). If possible, the sampling containers should be opened only once.

The soil samples should be liner samples or taken by core drilling and collected in wide-necked polyethylene (PE) or polypropylene (PP) bottles with screw cap and polyethylene gasket. It should be checked that the rim of the glasses is clean before closing them. The sample quantity necessary for the analysis depends on the grain size and has to be sufficient to allow the laboratory to conduct the preparation steps and store a back-up sample. Apart from that, the usual prescriptions for sampling of contaminated water and soil apply.

### Sample Preparation

For the preparation of the soil samples, the PFAS are extracted from the dry, homogenized sample via ultrasound-assisted extraction. Samples with high water content (sediments, sewage sludge) should preferably be dried by lyophilization. As an alternative, the drying can also be conducted at 40°C (more time consuming). To homogenize the sample, the dry material is crushed using an analytical mill to achieve a 95% throughput of the milled material through a 250 µm sieve. This allows obtaining a homogeneous sample from which representative samples for the analysis can be taken. Ultrasounds are applied (1 h, 40°C) (DIN, 2011b) to conduct the methanol extraction more efficiently. The extract is used for the next steps.

Coloured or turbid extracts (in most cases from sewage sludge, compost or sediments) or those, in which the contaminants have a very low concentration, are treated using solid phase extraction. The PFAS from the soil extracts (see **Figure 1**) or the water samples are concentrated on a weak anion-exchanger by performing a SPE on the unfiltered water sample (pH 6-8).

The solid phase is rinsed with water and solvents to separate the products that could interfere with the analysis. The adsorbed compounds are then eluted with methanol containing ammonia. Colourless extracts are analysed directly after dilution with water (Methanol: water 4:6). The leachate can be concentrated and dried by blowing off the solvent with nitrogen at a temperature of 40°C. The residual is dissolved again with a methanol-water mixture. The solution to be analysed can be filtered, if needed. Filtering will not cause losses. (DIN, 2011b).

The analysis of the nonpolar PFAS in water samples starts with the preparation using liquid-liquid extraction with MTBE. The extract is dried with sodium sulfate and concentrated in a rotary evaporator at 40°C and 400 mbar with acceptable losses (Bayerisches Landesamt für Umwelt, 2012).

For the concentration of Telomer acids, the rinsing step is usually avoided, because the losses are too high in this phase .

The volatile Telomer alcohols can theoretically be found in soil vapour, but operating procedures for the sampling do not yet exist. They might be adapted from the ambient air sampling protocol. Here, Perfluoralkanoic acids in the ambient air are adsorbed on Polyurethane foam in conjunction with a glass fiber filter in a stainless steel cartridge and then leached in the laboratory (Bayerisches Landesamt für Umwelt, 2012).

#### Separation

According to the standard, colourless and clear soil extract or very concentrated water samples can be analysed without further pre-treatment. Part of the extract has to be diluted with water without allowing the methanol content to drop below 40%. It should be considered that the direct injection method is only allowed as an alternative. Documentation and a demonstration that this method is equivalent to the standard methods are necessary.

If the only solvent for the solution to be analysed is water, high losses occur, especially in the case of PFOS, PFNA and PFDA (DIN, 2011a). In case of relevant matrix interference, the samples have to be cleaned via SPE.

There are no particular requirements regarding the chromatography. A complete separation of the single substances is not necessary, because they can be differentiated by their mass.

#### Calibration and Quantification

During analysis, analyte losses occur during different steps. The recovery rate is therefore much lower than 100%. Especially in soil analysis the recovery rate varies very strongly depending on the soil type. Therefore, a comparison standard<sup>9</sup> (internal standard) carrying heavier isotopes, e.g. <sup>13</sup>C<sub>4</sub>-PFBA, is usually added to the sample. This means that four carbon atoms of the molecule are exchanged with <sup>13</sup>C-Isotopes. Since this contains an extra neutron in comparison to the more common <sup>12</sup>C this isotope, the masses of the not-marked compounds are 4 u (u; unified atomic mass unit) lower than the masses of the marked compounds. Both behave in the same way as far as losses in the preparation, chromatography and ionization are concerned. They can be differentiated clearly in the detection because of the different molecular weights. The retention times of the resulting peaks are compared to those of the standards to identify the substance. For

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<sup>9</sup> According to the standard only the internal standardization is allowed for the sample analysis. To conduct the analysis, <sup>13</sup>C-marked standards have to be used at least for PFBA, PFHxA, PFOA und PFOS. If no internal standard is available for a substance, other internal standards can be considered, if the recovery rates are in the same range as the internal standards. This requirement is not always achieved, so that the use of additional standards, especially for substances that are often present, is basically recommended.

a reliable confirmation of the positive analysis results, the correspondence of the MS/MS Spectra of the sample to the standard should be checked.

For the quantification, the area ratio of the analyte in the sample to the corresponding internal standards is calculated. This means that for each compound to be analysed an internal standard marked with an isotope has to be available. However, for many PFAS this is not the case. To allow the determination of the recovery rate, another mass labelled standard is used, which behaves like the selected analyte as much as possible as far as sample preparation and analysis are concerned. An "external standard" is used in this case for the quantification. This is the same compound, but not mass labelled. In the worst case, even external standards are missing for the quantification (Gruber, 2011). For example, for PFPeA and PFBS there are no mass labelled standards available.  $^{13}\text{C}$ -PFBA is therefore used as internal standard for PFPeA and PFBS. PFBS measurement is based on  $^{13}\text{C}$ -PFHxS.

For the important metabolites 5:3 FTCA and 7:3 FTCA there are no comparison substances, but  $^{13}\text{C}$ -labelled Perfluorcarboxylic acids with an appropriate chain length are suitable as internal standards. According to the standard, only unbranched PFAS<sup>10</sup> can be used for calibration (DIN, 2011a). In the interpretation, the peak area of the linear and all the detected branched isomers is measured and evaluated on the basis of the calibration of the respective unbranched compounds. In the quantification it is assumed that the nonlinear isomers, which eluate just before the linear PFOS, show the same response-factor as the linear PFOS, although this is not completely correct. The analytical error is approx. 20%. This convention was agreed upon to allow the quantification of branched isomers. The reason is that their percentage, especially in the case of PFOS, can be significant and a chromatographic separation of all isomers is not possible in the usual conditions.

Moreover, the pure compounds for the calibration are not available for most of the isomers (DIN, 2011a). The indicated mass concentrations (in  $\mu\text{g/l}$  or  $\mu\text{g/kg DW}$ ) are based on the respective anions.

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<sup>10</sup> Branched Isomers occur especially in the PFOA, PFHxS and PFOS analysis.

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# Attachment 24

## The True Cost of PFAS and the Benefits of Acting Now

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**KEYWORDS:** PFAS, social costs, chemicals policy, remediation, prevention

### INTRODUCTION

Per- and polyfluoroalkyl substances (PFAS) are a class of over 9000 persistent hazardous chemicals used in industrial processes and consumer goods. They are ubiquitous in the environment and in people, who are exposed to PFAS via contaminated food and water, consumer products, and workplaces.<sup>1</sup> Exposure to several PFAS has been linked to a plethora of health effects in both animal and human studies, even at background levels. They are so environmentally persistent that they have been termed “forever chemicals.”

While in many ways PFAS contamination problems reflect broader issues with the chemicals regulatory system in the United States, a key feature of this industry is that only a handful of companies have produced the basic chemical building blocks for PFAS chemicals. These companies have known about the potential toxicity, human exposure, and extreme persistence of PFAS since the 1970s, yet have continued and expanded production.<sup>2</sup>

In the 2000s, in response to mounting pressure from the U.S. Environmental Protection Agency (EPA) about risks to

human and environmental health, PFAS manufacturers agreed to phase out U.S. production of perfluorooctanoic acid (PFOA), perfluorooctanesulfonate (PFOS), and some related PFAS. Replacement PFAS, including new chemicals developed by industry, are widely used in more than 200 use categories,<sup>3</sup> despite growing concerns about exposures, persistence, and toxicity.<sup>4</sup>

The PFAS industry claims that the chemicals’ use in consumer goods and industrial applications brings wide benefits, valuing the U.S. fluoropolymer segment at \$2 billion a year.<sup>5</sup> However, it fails to mention the costs of exposure, which are long-term, wide-ranging, routinely externalized onto

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the public, and disproportionately experienced. Focusing on a narrow, short-term view of PFAS benefits ignores how costs are displaced to communities and governments, despite existence of safer alternatives in most product sectors.

This review of the true costs of PFAS highlights the need to act now to ensure that exposures are capped at current levels by reducing the production and use of PFAS. It calls attention to systematic failures of U.S. chemical regulation, including inadequate premarket review of new compounds, data gaps that prevent and delay the regulation of existing chemicals, and the widespread externalization of social costs of pollution onto the public.

## ■ SNAPSHOT OF THE PROBLEM

**Shifting the Burden to Public Utilities.** Widespread contamination of surface water and groundwater due to industrial releases of PFAS or use of PFAS-containing firefighting foams is now a major problem in the United States and globally. An estimated 200 million U.S. residents, nearly two-thirds of the U.S. population, receive municipally provided drinking water that is contaminated with PFAS.<sup>6</sup>

Methods to reduce levels of PFAS in drinking water include filtration with granular activated charcoal treatment, reverse osmosis, ion exchange, or blending with less contaminated water from other sources, none of which fully eliminate PFAS. Municipalities may also opt to buy water from other distributors, but each method involves significant capital costs for new infrastructure and ongoing maintenance costs. For example, following extensive contamination by a PFAS manufacturer in the Cape Fear River watershed, Brunswick County, North Carolina spent \$99 million on a reverse osmosis plant and will incur \$2.9 million annually in operations expenses. Orange County, California estimates that the infrastructure needed to lower the levels of PFAS in its drinking water to the state's recommended levels will cost at least \$1 billion.

These costs of cleaning up PFAS contamination of water are rarely internalized by chemical manufacturers or other responsible parties. Instead, they are usually displaced onto public utilities, their ratepayers, and state and local governments.

Communities with PFAS-contaminated drinking water also incur expenses related to testing and monitoring the contamination, informing the public, gathering information on treatment alternatives, studying the feasibility of infrastructure investments, and staff time for these projects. Low-income communities may be unable to cover such expenditures and often have few options for cost recovery, especially when the source of the PFAS contamination has not been determined. Additionally, PFAS contamination is likely to disproportionately impact vulnerable communities due to historic racial discrimination in housing and occupational sectors, and inequitable enforcement of environmental regulations that concentrate point sources of pollution proximal to these communities.

PFAS in wastewater can lead to additional expenses for public utilities. Wastewater treatment plants are designed to remove solids and pathogens, not persistent chemicals, and so any PFAS coming into the treatment plant are largely discharged into receiving waters or left as contaminants in sewage sludge. Needed treatment to remove contaminants will result in increased costs, and failure to treat may decrease existing revenue streams. For example, the public utility

managing Merrimack, New Hampshire's wastewater currently earns \$400,000 annually from processing sludge into compost for public sale as fertilizer. If the utility can no longer sell the sludge due to PFAS contamination, it will instead have to spend \$2.4 million annually in landfill charges.

**Other Externalized Costs of PFAS.** Many other PFAS-related costs are routinely passed on to the public, rather than paid by the responsible polluters. For example, to prevent further contamination of water resources, the stock of fluorinated aqueous film-forming foams (AFFFs) still in place at military bases, airports, industrial sites, and local fire stations needs to be replaced with nonfluorinated foams. This requires collecting the AFFFs and then decontaminating or replacing equipment. The unused AFFFs and the PFAS-laden rinsewater must be contained, and no safe, permanent destruction methods currently exist.

The process of deciding what to do with hot spots of PFAS contamination is labor-intensive, time-consuming, and expensive. Testing of soil and water to determine the extent of contamination typically costs hundreds of dollars per sample, and few cleanup options exist. Landfilling of contaminated soil involves transportation costs and tip fees, and PFAS are only sequestered for the lifespan of the landfill. Incineration may destroy PFAS but only at extremely high temperatures, and has not been shown to work at large scale. Concerns about emissions from PFAS incineration, as well as public outrage at incineration testing in impacted communities, point to both health and political costs of PFAS incineration.

PFAS contamination may also reduce property values of homes and businesses. The discovery of water contamination, or even the perceived risk of potential contamination, can depress property values and stigmatize neighborhoods, potentially leading to lower home values and blocking residents' from selling properties, particularly when contamination achieves a level of public notoriety.<sup>7</sup>

Households and local businesses seeking to avoid exposure to contaminated drinking water may have to purchase bottled water or install and maintain home water filtration systems. In cases where the polluter is known, these costs may be recoverable through costly litigation. More often, however, the precise source of PFAS contamination is unclear, contested, or involves multiple polluters, making litigation or regulatory outcomes uncertain. Additionally, residents living outside of established boundaries or whose water is below specific action levels may not qualify for alternative water supplies, even if distribution systems exist.

Farms in areas with PFAS-contaminated water or soil may be forced to destroy harvests or products, or even to cease operation. As examples, dairy farms in more than one state were forced to dump milk contaminated with PFAS from agricultural applications of sludge and to euthanize their herds, while an organic farm near Colorado's Fort Peterson Air Force Base completely ceased production after learning that their irrigation water was highly contaminated.

Again, the governance and research expenses in such instances are substantial. In addition to technical expertise and staffing related to exposure assessment, human bio-monitoring, and cleanup efforts, local and state governments must invest significant resources in public engagement and communications, and in managing PFAS programs and task forces. For example, North Carolina has allocated over \$5 million for its PFAS Testing Network to address ongoing questions about PFAS exposure.

State and local governments may also incur significant legal expenses. States including New Hampshire and New Jersey have been sued by PFAS manufacturers opposed to health-protective drinking water regulations. States have occasionally received compensation from the companies responsible for PFAS pollution in their environs, including Minnesota (\$850 million), Alabama (\$39 million), and Michigan (\$168 million).<sup>8</sup> The number of lawsuits and the size of settlements indicates the nation-wide scope of PFAS contamination and the costs of exposure. Legal actions such as these require significant time and resources from state-employed and contracted lawyers, consultants, and other professionals.

Moreover, these legal actions happen after the damage has occurred. Since complete remediation of PFAS in the environment is impossible at this time, exposures will remain for generations to come.

**Health Impacts: The Biggest Externality.** Exposure to PFAS via contaminated drinking water has been linked to kidney and testicular cancer, ulcerative colitis, pregnancy and fertility problems, liver diseases, thyroid disease, and high cholesterol.<sup>1,9</sup> PFAS exposure is also linked to immunotoxic effects, including decreased response to vaccines and possible increases in COVID-19 severity.<sup>10</sup> Even low-level exposure is associated with serious health consequences. For example, multiple studies have linked prenatal PFAS exposure with low birth weight, a particularly concerning end point that is associated with higher risk of cardiovascular disease, respiratory disease, and diabetes in adulthood, as well as impaired cognitive development and lower lifetime earnings.<sup>11</sup>

The impacts on human health due to PFAS exposure are immense. A recent analysis of impacts from PFAS exposure in Europe identified annual direct healthcare expenditures at €52–84 billion.<sup>12</sup> Equivalent health-related costs for the United States, accounting for population size and exchange rate differences, would be \$37–59 billion annually. These costs are not paid by the polluter; they are borne by ordinary people, health care providers, and taxpayers.

Indirect social costs are also extensive, though more difficult to calculate. They include lost wages; lost years of life; reduced quality of life; increased stress, anxiety, and depression; and subsequent impacts on families and communities. Such social costs are quantifiable and can guide policy,<sup>13</sup> but no such analysis currently exists for health impacts from PFAS in the United States.

Finally, other significant health-related costs borne by government institutions and taxpayers include biomonitoring and health monitoring of exposed populations, and government research expenditures aimed at identifying PFAS toxicity and extent of exposures. In a more equitable world, this research would be carried out by the producer before the chemical came onto the market.

## DISCUSSION

The health, societal, and economic impacts of contamination from PFAS production and use are multifaceted and broadly distributed. The costs of these impacts are long-term, incompletely understood, and externalized onto individuals, communities, and government at all levels, while profits accrue to corporations shielded from these costs by the protections built into our chemical regulatory laws and practices.<sup>14</sup> The continued use of PFAS will lead to increases in contamination and exposures in the future. But these exposures can be capped if steps are taken now to reduce and eventually phase out

production and use of PFAS in all nonessential applications. In the meantime, the responsibility for paying for the legacy contamination should rest on the companies who continue to produce and market these chemicals even though they know about the chemicals' toxicity and extreme persistence.

Under a precautionary system of chemicals production in which companies had to demonstrate the safety of their products before accessing markets, costs could be substantially reduced by avoiding the production of toxic substances, and remaining costs would be internalized by PFAS producers into the price of their products. But in the United States, these costs are largely borne by the public and public institutions.

As this review of PFAS externalities shows, meaningful action must address not just remediation and cleanup of legacy contamination, but must also reduce current production and uses of PFAS, in order to limit the extent of future exposures. Class-based regulation of all PFAS is needed,<sup>15</sup> and California's recent action to regulate PFAS as a class in consumer products demonstrates that class-based restrictions are possible and desirable.<sup>16</sup>

Ubiquitous exposure to many toxic chemicals, not just PFAS, reflects a failure of regulatory systems to adequately reduce risk, and a privileging of short-term industry profits over long-term public health and environmental impacts. While the costs of drinking water treatment and PFAS remediation are substantial, the potential health-related costs of continued exposure to PFAS are much larger and will likely impact vulnerable communities disproportionately. Failing to take timely action to reduce the production and use of PFAS will result in exponentially higher costs to be paid by exposed populations for generations to come.

Understanding the true extent of these costs will clarify the benefits of improved regulatory controls and timely clean-ups. It will enable residents and policy makers to make informed decisions about who should rightfully bear responsibility for impacts and compensation. A strengthened regulatory system is needed, both in terms of enforcement of existing regulations and enactment of stronger, class-based laws to internalize the costs and reduce or eliminate the production of persistent, mobile, bioaccumulative, and toxic compounds. Only a strengthened regulatory system can adequately protect public health and the environment, and end the practice of forcing the public and future generations to bear the financial and health burden of pollution.

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## Notes

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# Attachment 25



**QuickFacts**  
**United States**

QuickFacts provides statistics for all states and counties, and for cities and towns with a **population of 5,000 or more**.

**Table**

All Topics	United States
<b>Population Estimates, July 1 2021, (V2021)</b>	331,893,745
<b>PEOPLE</b>	
<b>Population</b>	
<b>Population Estimates, July 1 2021, (V2021)</b>	331,893,745
Population estimates base, April 1, 2020, (V2021)	331,449,281
Population, percent change - April 1, 2020 (estimates base) to July 1, 2021, (V2021)	0.1%
Population, Census, April 1, 2020	331,449,281
Population, Census, April 1, 2010	308,745,538
<b>Age and Sex</b>	
Persons under 5 years, percent	6.0%
Persons under 18 years, percent	22.3%
Persons 65 years and over, percent	16.5%
Female persons, percent	50.8%
<b>Race and Hispanic Origin</b>	
White alone, percent	76.3%
Black or African American alone, percent (a)	13.4%
American Indian and Alaska Native alone, percent (a)	1.3%
Asian alone, percent (a)	5.9%
Native Hawaiian and Other Pacific Islander alone, percent (a)	0.2%
Two or More Races, percent	2.8%
Hispanic or Latino, percent (b)	18.5%
White alone, not Hispanic or Latino, percent	60.1%
<b>Population Characteristics</b>	
Veterans, 2016-2020	17,835,456
Foreign born persons, percent, 2016-2020	13.5%
<b>Housing</b>	
Housing units, July 1, 2019, (V2019)	139,684,244
Owner-occupied housing unit rate, 2016-2020	64.4%
Median value of owner-occupied housing units, 2016-2020	\$229,800
Median selected monthly owner costs -with a mortgage, 2016-2020	\$1,621
Median selected monthly owner costs -without a mortgage, 2016-2020	\$509
Median gross rent, 2016-2020	\$1,096
Building permits, 2020	1,471,141
<b>Families &amp; Living Arrangements</b>	
Households, 2016-2020	122,354,219
Persons per household, 2016-2020	2.60
Living in same house 1 year ago, percent of persons age 1 year+, 2016-2020	86.2%
Language other than English spoken at home, percent of persons age 5 years+, 2016-2020	21.5%
<b>Computer and Internet Use</b>	
Households with a computer, percent, 2016-2020	91.9%
Households with a broadband Internet subscription, percent, 2016-2020	85.2%
<b>Education</b>	
High school graduate or higher, percent of persons age 25 years+, 2016-2020	88.5%
Bachelor's degree or higher, percent of persons age 25 years+, 2016-2020	32.9%
<b>Health</b>	
With a disability, under age 65 years, percent, 2016-2020	8.7%
Persons without health insurance, under age 65 years, percent	10.2%
<b>Economy</b>	
In civilian labor force, total, percent of population age 16 years+, 2016-2020	63.0%
In civilian labor force, female, percent of population age 16 years+, 2016-2020	58.4%
Total accommodation and food services sales, 2012 (\$1,000) (c)	708,138,598
Total health care and social assistance receipts/revenue, 2012 (\$1,000) (c)	2,040,441,203
Total manufacturers shipments, 2012 (\$1,000) (c)	5,696,729,632

Total retail sales, 2012 (\$1,000) (c)	4,219,821,871
Total retail sales per capita, 2012 (c)	\$13,443
<b>Transportation</b>	
Mean travel time to work (minutes), workers age 16 years+, 2016-2020	26.9
<b>Income &amp; Poverty</b>	
Median household income (in 2020 dollars), 2016-2020	\$64,994
Per capita income in past 12 months (in 2020 dollars), 2016-2020	\$35,384
Persons in poverty, percent	△ 11.4%
<b>BUSINESSES</b>	
<b>Businesses</b>	
Total employer establishments, 2019	7,959,103
Total employment, 2019	132,989,428
Total annual payroll, 2019 (\$1,000)	7,428,553,593
Total employment, percent change, 2018-2019	1.6%
Total nonemployer establishments, 2018	26,485,532
All firms, 2012	27,626,360
Men-owned firms, 2012	14,844,597
Women-owned firms, 2012	9,878,397
Minority-owned firms, 2012	7,952,386
Nonminority-owned firms, 2012	18,987,918
Veteran-owned firms, 2012	2,521,682
Nonveteran-owned firms, 2012	24,070,685
<b>GEOGRAPHY</b>	
<b>Geography</b>	
Population per square mile, 2010	87.4
Land area in square miles, 2010	3,531,905.43
FIPS Code	1

[About datasets used in this table](#)

#### Value Notes

⚠ Estimates are not comparable to other geographic levels due to methodology differences that may exist between different data sources.

Some estimates presented here come from sample data, and thus have sampling errors that may render some apparent differences between geographies statistically indistinguishable. Click the Quick Info  icon to the left of each row in TABLE view to learn about sampling error.

The vintage year (e.g., V2021) refers to the final year of the series (2020 thru 2021). Different vintage years of estimates are not comparable.

Users should exercise caution when comparing 2016-2020 ACS 5-year estimates to other ACS estimates. For more information, please visit the [2020 5-year ACS Comparison Guidance](#) page.

#### Fact Notes

- (a) Includes persons reporting only one race
- (c) Economic Census - Puerto Rico data are not comparable to U.S. Economic Census data
- (b) Hispanics may be of any race, so also are included in applicable race categories

#### Value Flags

- Either no or too few sample observations were available to compute an estimate, or a ratio of medians cannot be calculated because one or both of the median estimates falls in the lowest or upper interval of an open ended distribution.
- F Fewer than 25 firms
- D Suppressed to avoid disclosure of confidential information
- N Data for this geographic area cannot be displayed because the number of sample cases is too small.
- FN Footnote on this item in place of data
- X Not applicable
- S Suppressed; does not meet publication standards
- NA Not available
- Z Value greater than zero but less than half unit of measure shown

QuickFacts data are derived from: Population Estimates, American Community Survey, Census of Population and Housing, Current Population Survey, Small Area Health Insurance Estimates, Small Area Income and Poverty Estimates, State and County Housing Unit Estimates, County Business Patterns, Nonemployer Statistics, Economic Census, Survey of Business Owners, Building Permits.

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# Attachment 26

United States  
Environmental Protection  
Agency

Office of Water  
(4607)  
Washington, DC 20460

EPA 815-R-00-026  
December 2000  
[www.epa.gov/safewater](http://www.epa.gov/safewater)

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# ARSENIC IN DRINKING WATER RULE ECONOMIC ANALYSIS

# **Arsenic in Drinking Water Rule Economic Analysis**

Developed for:

Office of Ground Water and Drinking Water  
U.S. Environmental Protection Agency  
401 M Street, S.W.  
Washington, DC 20460

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December 2000

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# Chapter 1: Executive Summary

## 1.1 Regulatory Background

An enforceable standard of 50 µg/L currently exists for arsenic in community water systems under the National Interim Primary Drinking Water Regulations (40 CFR 59566). In §1412(b)(12)(A) of the SDWA, as amended in 1996, Congress specifically directed EPA to issue a final rule by January 1, 2001. Congress recently changed the deadline for the final rule to June 22, 2001 (Public Law 106-377).

This document analyzes the impacts of the revised rule, which changes the current standard as follows:

- (1) Reduces the current MCL for arsenic in community water systems from 50 µg/L to 10 µg/L;
- (2) Requires non-transient non-community (NTNC) water systems to come into compliance with the new standard; and
- (3) Revises the current monitoring requirements to make them consistent with the Standard Monitoring Framework (40 CFR 141.23(c)).

## 1.2 Health Effects of Arsenic

Arsenic's carcinogenic role was noted over 100 years ago (NCI, 1999) and has been studied ever since. The Agency has classified arsenic as a Class A human carcinogen, "based on sufficient evidence from human data. An increased lung cancer mortality was observed in multiple human populations exposed primarily through inhalation. Also, increased mortality from multiple internal organ cancers (liver, kidney, lung, and bladder) and an increased incidence of skin cancer were observed in populations consuming drinking water high in inorganic arsenic."

A 1999 NRC report on arsenic states that "epidemiological studies ... clearly show associations of arsenic with several internal cancers at exposure concentrations of several hundred micrograms per liter of drinking water." Ten epidemiological studies covering eight organ systems have quantitative data for risk assessment (NRC, 1999, Table 4-1). The organ systems where cancers in humans have been identified include skin, bladder, lung, kidney, nasal cavity, liver, and prostate.

Table 10-6 of the same NRC report provides risk parameters for three cancers: bladder, lung, and liver cancer. Considering all cancers in aggregate, the NRC states that "considering the data on bladder and lung cancer in both sexes noted in the studies ... a similar approach for all cancers could easily result in a combined cancer risk on the order of 1 in 100" (at the current MCL of 50 µg/L).

New data provide additional health effects information on both carcinogenic and noncarcinogenic effects of arsenic. A recent study by Tsai et al. (1999) of a population that has been studied over many

years in Taiwan has provided standardized mortality ratios (SMRs) for 23 cancerous and non-cancerous causes of death in women and 27 causes of death in men at statistically significant levels in an area of Taiwan with elevated arsenic exposures (Tsai et al., 1999). SMRs are an expression of the ratio between deaths that were observed in an area with elevated arsenic levels and those that were expected to occur, based on the mortality experience of the populations in nearby areas without elevated arsenic levels. Drinking water (250-1,140 µg/L) and soil (5.3-11.2 mg/kg) in the Tsai et al. (1999) population study had very high arsenic content.

Tsai et al. (1999) identified “bronchitis, liver cirrhosis, nephropathy, intestinal cancer, rectal cancer, laryngeal cancer, and cerebrovascular disease” as possibly “related to chronic arsenic exposure via drinking water,” which had not been reported before. In addition, the study area had upper respiratory tract cancers previously only related to occupational inhalation. High male mortality rate (SMR > 3) existed for bladder, kidney, skin, lung, and nasal cavity cancers and for vascular disease. However, the authors noted that the mortality range was marginal for leukemia, cerebrovascular disease, liver cirrhosis, nephropathy, and diabetes. Females also had high mortalities for laryngeal cancer. There are, of course, possible differences between the population and health care in Taiwan and the United States. For example, arsenic levels in the U.S. are not as high as they were in the study area of Taiwan. However, the study gives an indication of the types of health effects that may be associated with arsenic exposure via drinking water.

Arsenic interferes with a number of essential physiological activities, including the actions of enzymes, essential cations, and transcriptional events in cells (NRC, 1999). A wide variety of adverse health effects have been associated with chronic ingestion of arsenic in drinking water, occurring at various exposure levels.

### **1.3 Regulatory Alternatives Considered**

In regulating a contaminant, EPA first sets a maximum contaminant level goal (MCLG), which establishes the contaminant level at which no known or anticipated adverse health effects occur. MCLGs are non-enforceable health goals. For this rulemaking, EPA is setting an MCLG of zero. EPA then sets an enforceable maximum contaminant level (MCL) as close as technologically possible to the MCLG. In addition, EPA may use its discretion in setting the MCL by choosing an MCL that is protective of public health while also ensuring that the quantified and non-quantified costs are justified by the quantified and non-quantified benefits of the rule. For this rulemaking, EPA is setting an MCL of 10 µg/L. Chapter 3 describes the process by which EPA determined both the MCLG and the MCL.

EPA considered a range of MCLs in developing the final Arsenic Rule, including MCLs of 3, 5, 10, and 20 µg/L. EPA evaluated the following five factors to determine the revised MCL:

- The analytical capability and laboratory capacity;
- The likelihood of water systems choosing various compliance technologies for several sizes of systems based on source water properties;
- The national occurrence of arsenic in water supplies;

- Quantified and non-quantified costs and health risk reduction benefits likely to occur at the MCLs considered; and
- The effects on sensitive subpopulations.

After evaluating the above factors, EPA considered an MCL of 3 µg/L since this is the level that has been determined to be as close to the MCLG as is feasible. However, the Agency is using its discretionary authority in §1412(b)(6)(A) to consider setting MCL at a less stringent level. The statute requires that the alternative less stringent level be one which maximizes health risk reduction at a level where costs and benefits are also considered. As a result, EPA considered the alternative MCL options of 5, 10, and 20 µg/L.

The Agency also considered two regulatory options related to the applicability of the revised MCL. Specifically, EPA investigated applying both the monitoring and treatment requirements of the Arsenic Rule to both community water systems (CWSs) and NTNCs. A CWS is defined as a system that provides piped water to at least 25 people or with at least 15 service connections year-round. An NTNC is a public water system that is not defined as a CWS and that regularly serves at least 25 of the same people for at least six months of the year. After considering the costs and benefits of the revised rule with regard to both CWSs and NTNCs, EPA is requiring both CWS and NTNC water systems to comply with all facets of the revised rule. The benefit-cost analysis upon which this decision is based is provided in Chapters 5, 6, and 7 of this Economic Analysis (EA). Transient non-community systems, which provide potable water to continuously changing populations, will not be subject to the revised rule.

The revised rule also includes modifications to the current monitoring requirements, including the availability of monitoring waivers. A detailed discussion of these changes can be found in Chapter 3.

#### **1.4 Benefits and Costs of the Proposed Rule**

Quantitative risk metrics (e.g., slope factors or reference doses) are necessary to evaluate cancer or non-cancer risks. Although arsenic causes numerous health effects, bladder and lung cancer are the only endpoints for which an Agency-approved metric for evaluating arsenic-related risk currently exists. This cancer slope factor (SF) for bladder and lung cancer is used to calculate cases potentially avoided due to the revised arsenic standard. Benefits estimates for avoided cases of bladder and lung cancer were calculated using mean population risk estimates at various MCL levels. Lifetime risk estimates were converted to annual risk factors and applied to the exposed population to determine the number of cases avoided. These cases were divided into fatalities and non-fatal cases avoided, based on survival information. The avoided premature fatalities were valued based on the VSL estimates discussed in Chapter 5, as recommended by EPA current guidance for cost/benefit analysis. The avoided non-fatal cases were valued based on the willingness to pay estimates for the avoidance of chronic bronchitis. The upper bound estimates include the possibility of the incidence rate being understated, depending on the survival rate for bladder cancer in the study area of Taiwan.

Numerous other health effects that are likely to be avoided as a result of this rule may generate significant benefits, and should not be discounted based on the fact that they cannot be quantified at this time. The estimated total national monetized benefits of the proposed rule and the other rule options considered are provided in Exhibit 1-1.

**Exhibit 1-1**  
**Total Annual Cost, Estimated Monetized Total Cancer Health Benefits and**  
**Non-Quantifiable Health Benefits from Reducing Arsenic in PWSs**  
**(\$ millions)**

Arsenic Level (µg/L)	Total Annual Cost (7%)	Annual Bladder Cancer Health Benefits <sup>1,2</sup>	Annual Lung Cancer Health Benefits <sup>1,2</sup>	Total Annual Health Benefits <sup>1,2</sup>	Potential Non-Quantifiable Health Benefits
3	\$792.1	\$58.2 - \$156.4	\$155.6 - \$334.5	\$213.8 - \$490.9	<ul style="list-style-type: none"> <li>• Skin Cancer</li> <li>• Kidney Cancer</li> <li>• Cancer of the Nasal Passages</li> <li>• Liver Cancer</li> <li>• Prostate Cancer</li> <li>• Cardiovascular Effects</li> <li>• Pulmonary Effects</li> <li>• Immunological Effects</li> <li>• Neurological Effects</li> <li>• Endocrine Effects</li> <li>• Reproductive and Developmental Effects</li> </ul>
5	\$471.7	\$52.0 - \$113.3	\$139.1 - \$242.3	\$191.1 - \$355.6	
10	\$205.6	\$38.0 - \$63.0	\$101.6 - \$134.7	\$139.6 - \$197.7	
20	\$76.5	\$20.1 - \$21.5	\$46.1 - \$53.8	\$66.2 - \$75.3 <sup>3</sup>	

<sup>1</sup> May 1999 dollars.

<sup>2</sup> These monetary estimates are based on cases avoided given in Exhibit 5-9 (a-c).

<sup>3</sup> For 20 µg/L, the proportional reduction from the lower level risk base case is greater than the proportional reduction from the higher level risk base case. Thus, the number of estimated cases avoided and estimated benefits are higher at 20 µg/L using the risk estimates adjusted for arsenic in cooking water and food.

For the revised MCL of 10 µg/L, the estimated monetized bladder and lung cancer health benefits range from \$139.6 million to \$197.7 million. More detail about these benefit estimates are found in Chapter 5. Exhibit 1-2 shows the estimated national cost of compliance of the revised rule and the other rule options that were considered. At the revised MCL of 10 µg/L, the estimated national cost of compliance is \$180.4 million at a discount rate of three percent, and \$205.6 million at a discount rate of seven percent.

**Exhibit 1-2  
Total National Cost of Compliance (\$ millions)**

Discount Rate	CWS		NTNC		TOTAL	
	3%	7%	3%	7%	3%	7%
<b>MCL = 3 mg/L</b>						
System Costs						
Treatment	\$665.9	\$756.5	\$27.2	\$29.6	\$693.1	\$786.0
Monitoring/ Administrative	\$2.2	\$3.0	\$1.0	\$1.4	\$3.2	\$4.4
State Costs	\$1.4	\$1.6	\$0.1	\$0.2	\$1.5	\$1.7
<b>TOTAL COST</b>	<b>\$669.4</b>	<b>\$761.0</b>	<b>\$28.3</b>	<b>\$31.1</b>	<b>\$697.8</b>	<b>\$792.1</b>
<b>MCL = 5 mg/L</b>						
System Costs						
Treatment	\$394.4	\$448.5	\$16.3	\$17.6	\$410.6	\$466.1
Monitoring/ Administrative	\$2.0	\$2.8	\$1.0	\$1.3	\$2.9	\$4.1
State Costs	\$1.1	\$1.3	\$0.1	\$0.2	\$1.2	\$1.4
<b>TOTAL COST</b>	<b>\$397.5</b>	<b>\$452.5</b>	<b>\$17.3</b>	<b>\$19.1</b>	<b>\$414.8</b>	<b>\$471.7</b>
<b>MCL = 10 mg/L</b>						
System Costs						
Treatment	\$169.6	\$193.0	\$7.0	\$7.6	\$176.7	\$200.6
Monitoring/ Administrative	\$1.8	\$2.5	\$0.9	\$1.3	\$2.7	\$3.8
State Costs	\$0.9	\$1.0	\$0.1	\$0.2	\$1.0	\$1.2
<b>TOTAL COST</b>	<b>\$172.3</b>	<b>\$196.6</b>	<b>\$8.1</b>	<b>\$9.1</b>	<b>\$180.4</b>	<b>\$205.6</b>
<b>MCL = 20 mg/L</b>						
System Costs						
Treatment	\$60.7	\$69.0	\$2.6	\$2.8	\$63.3	\$71.8
Monitoring/ Administrative	\$1.7	\$2.4	\$0.9	\$1.3	\$2.6	\$3.7
State Costs	\$0.7	\$0.8	\$0.1	\$0.2	\$0.9	\$1.0
<b>TOTAL COST</b>	<b>\$63.2</b>	<b>\$72.3</b>	<b>\$3.6</b>	<b>\$4.2</b>	<b>\$66.8</b>	<b>\$76.5</b>

The net benefits and benefit-cost ratios of each regulatory option are provided in Exhibit 1-3. At the revised MCL of 10 µg/L, the net benefits range from a high of \$17.3 million to a low of a negative \$40.8 million, at a discount rate of three percent. These net benefits correspond to benefit-cost ratios of 0.8 and 1.1 (also at a three percent rate of discount).

**Exhibit 1-3  
Net Benefits and Benefit-Cost Ratios of Each Regulatory Option  
(\$ millions)**

MCL (µg/L)		3	5	10	20
<b>3% Discount Rate</b>					
lower bound	Net Benefits	\$ (484.0)	\$ (223.7)	\$ (40.8)	\$ (0.6)
	Benefit/Cost Ratio	0.3	0.5	0.8	1.0
upper bound	Net Benefits	\$ (206.8)	\$ (59.2)	\$ 17.3	\$ 8.5
	Benefit/Cost Ratio	0.7	0.9	1.1	1.1
<b>7% Discount Rate</b>					
lower bound	Net Benefits	\$ (578.3)	\$ (280.6)	\$ (66.0)	\$ (10.3)
	Benefit/Cost Ratio	0.3	0.4	0.7	0.9
upper bound	Net Benefits	\$ (301.1)	\$ (116.1)	\$ (7.9)	\$ (1.2)
	Benefit/Cost Ratio	0.6	0.8	1.0	1.0

\*Costs include treatment, O&M, monitoring, and administrative costs to CWSs and NTNCs and State costs for administration of water programs.

As mentioned above, there are a number of important non-monetized benefits of reducing arsenic exposure that are not included in the net benefit and benefit-cost calculations. Chief among these are certain health impacts known to be caused by arsenic. Such nonquantifiable benefits may include skin cancer, kidney cancer, cancer of the nasal passages, liver cancer, prostate cancer, cardiovascular effects, pulmonary effects, immunological effects, neurological effects, endocrine effects, and customer peace-of-mind benefits from knowing their drinking water has been treated for arsenic. For example, a number of epidemiologic studies conducted in several countries (e.g., Taiwan, Japan, England, Hungary, Mexico, Chile, and Argentina) report an association between arsenic in drinking water and skin cancer in exposed populations. Early reports linking inorganic arsenic contamination of drinking water to skin cancer came from Argentina (Neubauer, 1947, reviewing studies published as early as 1925) and Poland (Tseng et al., 1968). However, the first studies that observed dose-dependent effects of arsenic associated with skin cancer came from Taiwan (Tseng et al., 1968; Tseng, 1977). These studies focused EPA's attention on the health effects of ingested arsenic. Studies conducted in the U.S. have not demonstrated an association between inorganic arsenic in drinking water and skin cancer. However, these studies may not have included enough people in their design to detect these types of effects.

The potential monetized benefits associated with skin cancer reduction would not change the total benefits of the rule to an appreciable degree, even if the assumption were made that the risk of skin cancer were equivalent to that of bladder cancer, using EPA's 1988 risk assessment. Skin cancer is highly treatable (at a cost of illness of less than \$3,500 for basal and squamous cell carcinomas versus a cost of illness of \$178,000 for non-fatal bronchitis) in the U.S., with few fatalities (less than one percent).

In addition to potentially reducing the risk of skin cancer, there are also a large number of other health-related benefits associated with arsenic reduction, as presented in Exhibit 1-1, which are not monetized in this analysis due to lack of appropriate data.

Other benefits not monetized in this analysis include customer peace of mind from knowing drinking water has been treated for arsenic and reduced treatment costs for currently unregulated contaminants that may be co-treated with arsenic. To the extent that reverse osmosis is used for arsenic removal, these benefits could be substantial. Reverse osmosis and activated alumina are the primary point-of-use treatments for small systems. (These benefits of avoided treatment cannot currently be monetized; however, they can be readily monetized in the future, as decisions are made about which currently unregulated contaminants to regulate.)

## Chapter 2: Need for the Revised Rule

### 2.1 Introduction

The Safe Drinking Water Act (SDWA), as amended in 1996, requires EPA to identify and regulate substances in drinking water that may have an adverse effect on public health and that are known or anticipated to occur in public water supplies. National Primary Drinking Water Regulations (NPDWRs) address risks to public health, and secondary regulations address aesthetic qualities (such as taste, odor, or color) that relate to public acceptance of drinking water. For NPDWRs, EPA must either establish a Maximum Contaminant Level (MCL) or, if it is not economically or technically feasible to monitor the contaminant in drinking water, specify a treatment technique to remove the contaminant or reduce its concentration in the water supply.

An enforceable standard of 50 µg/L currently exists for arsenic in community water systems under the National Interim Primary Drinking Water Regulations (40 CFR 59566). In §1412(b)(12)(A) of the SDWA, as amended in 1996, Congress specifically directed EPA to propose a NPDWR for arsenic by January 1, 2000, and issue the final regulation by January 1, 2001. Congress recently changed the deadline for the final rule to June 22, 2001 (Public Law 106-377).

This document analyzes the impacts of the rule, which revises the current standard as follows:

- 1) Reduces the current MCL for arsenic in community water systems from 50 µg/L to 10 µg/L;
- 2) Requires nontransient non-community water systems (NTNC) to comply with the new standard; and
- 3) Revises the current monitoring requirements to make them consistent with the Standard Monitoring Framework (40 CFR 141.23(c)).

Executive Order 12866, *Regulatory Planning and Review*, requires EPA to estimate the costs and benefits of the Arsenic Rule in an *economic analysis* document (EA). This chapter of the EA discusses the public health concerns being addressed by the rule, describes the history of regulatory efforts concerning arsenic, and discusses the economic rationale for the rule. Subsequent chapters will accomplish the following:

- Discuss the regulatory options considered by EPA (Chapter 3),
- Present the results of the baseline analysis (Chapter 4),
- Examine the benefits of the rule (Chapter 5),
- Present the results of the cost analysis (Chapter 6),
- Compare the costs and benefits of the rule and the regulatory options considered by EPA (Chapter 7), and
- Discuss the potential economic impacts of the rule (Chapter 8).

## 2.2 Public Health Concerns To Be Addressed

This section describes the public health concerns addressed by the final Arsenic Rule. A description of potential health effects associated with arsenic, including effects in sensitive subpopulations, along with the sources of human exposure to arsenic, is presented. In addition, the section describes current controls that address exposure to arsenic.

### 2.2.1 Health Effects of Arsenic

Arsenic is a naturally occurring element present in the environment in both organic and inorganic forms. Inorganic arsenic, considered to be the more toxic form, is found in ground water, surface water, and many foods. Chronic exposure to high levels of inorganic arsenic in drinking water has been found to result in a variety of adverse health effects, including skin and internal cancers and cardiovascular and neurological effects.

Exposures to organic forms of arsenic also occur through ingestion of food and metabolism of ingested inorganic arsenic. Experimental data on the effects of organic forms of arsenic are not as well characterized as those for inorganic arsenic, and thus are the subject for future research. Limited data on the primary organic forms in fish and shellfish (arsenobetaine and arsenocholine) suggest that these forms are relatively nontoxic. Other forms of organoarsenicals in foods have been even less well characterized. Recent *in vitro* toxicity evidence indicates that the trivalent form of monomethylarsonic acid is more toxic than either the trivalent (arsenite) or pentavalent (arsenate) forms of inorganic arsenic. Additional data are needed in this area before the toxicological significance of the trivalent form of monomethylarsonic acid is clear.

In 1996, EPA requested that the National Research Council of NAS conduct an independent review of the arsenic toxicity data. NRC was asked to review EPA's current criteria (50 µg/L and 0.018 µg/L), evaluate use of recent Taiwan data and other studies to assess the carcinogenic and non-carcinogenic health effects of arsenic, and recommend changes to EPA's risk characterization for arsenic. NRC issued its report on March 23, 1999 (NRC, 1999). The health effects of inorganic arsenic are summarized below and are described in more detail in Chapter 5.

#### **Cancer**

There is a large human database available for inorganic arsenic, unlike most environmental contaminants. However, there is substantial debate among the scientific community over the interpretation of these data and their application in risk assessment. NRC found that a number of epidemiologic studies conducted in several countries (e.g., Taiwan, Japan, England, Hungary, Mexico, Chile, and Argentina) report an association between arsenic in drinking water and skin cancer in exposed populations. Increased mortality from internal cancers of liver, bladder, kidney, and lung have also been reported.

EPA has identified arsenic as a group A “known” human carcinogen, based on increased risks of lung cancer in workers exposed to airborne arsenic and dose-dependent increases in skin cancer risk in Taiwan.

### ***Non-Cancer Health Effects***

In addition to cancer, NRC (1999) reported that arsenic exposures have been linked to other adverse health effects. These include thickening of the skin, effects on the nervous system such as tingling and loss of feeling in limbs, hearing impairment, effects on the heart and circulatory system, diabetes, developmental effects, and effects on the gastrointestinal system and liver. Many of these effects are observed at concentrations where cancer effects were observed in the epidemiology studies.

### ***Sensitive Subpopulations***

Certain sensitive individuals may be at a greater risk of serious illness from exposure to arsenic than the general population. The NRC report (1999) noted that human sensitivity to the toxic effects of inorganic arsenic exposure is likely to vary based on genetics, metabolism, diet, health status, sex, and other possible factors. For example, reduced ability to methylate arsenic (convert inorganic arsenic into less acutely toxic and more readily excreted forms) may result in retention of more arsenic in the body and increased risk of toxic effects. However, there is insufficient evidence at the present time to characterize the influence of such factors as age, sex, nutrition, and genetic polymorphism on the expression of arsenic toxicity (NRC, 1999).

The following groups have been cited in various studies as possibly being particularly susceptible to health effects from arsenic:

- **Children** are identified as especially susceptible because their dose of arsenic will be, on average, higher than that of adults exposed to similar concentrations due to their higher fluid and food intake relative to body weight. The NRC report cited one study that suggests that children may have a lower arsenic-methylation efficiency than adults.
- **Pregnant and lactating women** are especially vulnerable because of possible adverse reproductive and developmental effects of arsenic.
- **People with poor nutritional status** may have a reduced ability to methylate arsenic.
- **Individuals with pre-existing diseases that affect specific organs**—in particular, kidney and liver problems—may be more susceptible to the effects of arsenic because these organs act to detoxify arsenic in the body.  
In addition, arsenic can directly damage these and other organ systems, as described above. Individuals with pre-existing damage or congenital defects in these systems are more susceptible to health effects from exposure to arsenic. The elderly are more likely as a group to have pre-existing conditions in the susceptible organ systems.

Section 5.2.4 discusses the susceptibility of these subgroups in more detail. Due to a lack of available data, no quantitative analysis of the specific risks to sensitive populations was performed as part of this EA.

## **2.2.2 Sources and Mechanisms of Exposure**

Arsenic (As) is an element that occurs in the earth's crust. Accordingly, there are natural sources of exposure. Erosion and weathering of rocks deposit arsenic in water bodies and lead to the uptake of arsenic by animals and plants. Consumption of food and water is the major source of arsenic exposure for the majority of U.S. citizens. People may also be exposed from industrial sources, as arsenic is used in semiconductor manufacturing, petroleum refining, wood preservatives, animal feed additives, and herbicides.

Arsenic can combine with other elements to form inorganic and organic arsenicals. In general, inorganic derivatives are regarded as more toxic than the organic forms. While food contains both inorganic and organic arsenicals, primarily inorganic forms are present in water.

Recently, EPA developed estimates of human exposure to arsenic in drinking water, food, and air using data from numerous Federal sampling surveys analyzing the occurrence of arsenic in public water supplies, dietary foods, and ambient air. EPA's national air sampling databases indicate very low concentrations of arsenic in both urban and non-urban locations, at levels typically ranging from about 0.003 to 0.03  $\mu\text{g}/\text{m}^3$ . Air is therefore an insignificant source of arsenic intake, typically representing less than one percent of overall exposure.

EPA reviewed several local and regional studies for comparison purposes. Using the Total Diet Study of the Food and Drug Administration (FDA), recent dietary analyses indicate that the average adult's total arsenic intake is about 53  $\mu\text{g}/\text{day}$ . The FDA analytical methodology does not differentiate between the organic and inorganic forms of arsenic. For most people living in the U.S., inorganic arsenic exposure is primarily from food and water sources. Since the inorganic forms are considered to be more toxic, it is important to estimate the amount of inorganic arsenic in the diet. To accomplish this estimation, EPA used the FDA data along with a separate study that characterized arsenic species in foods. This separate characterization indicated that about 20 percent of daily intake of dietary arsenic is in the inorganic form. Conversely, most arsenic present in drinking water is in the form of inorganic arsenic species.

Accounting for the organic forms of arsenic in food, the dietary intake of inorganic arsenic was estimated to be approximately 14  $\mu\text{g}/\text{day}$ . An adult drinking 2 L/day of water containing 10  $\mu\text{g}/\text{L}$  of arsenic would obtain 20  $\mu\text{g}/\text{day}$  from drinking water, so that drinking water would contribute about 60 percent of total intake of inorganic arsenic. On the other hand, an adult drinking water containing 2  $\mu\text{g}/\text{L}$  of arsenic would obtain almost 80 percent of the daily inorganic arsenic from food.

## 2.3 Regulatory History

This section provides a chronology and overview of regulatory actions affecting arsenic in drinking water and recent efforts that have led to this rulemaking. It also summarizes the major studies and data collection efforts that highlighted the need for a new rule.

**Current MCL:** In 1975, EPA set the National Interim Primary Drinking Water Regulation at 50 µg/L (40 FR 59566, December 24, 1975). This standard was equal to the standard set in 1942 by the U.S. Public Health Service for interstate water carriers, which was not based on a risk assessment. EPA based the MCL on daily consumption of two liters of water providing approximately 10 percent of total ingested arsenic of 900 µg/day. Commenters recommended an MCL of 100 µg/L based on no observed adverse health effects. EPA noted long-term chronic effects at 300 to 2,750 µg/L, but no chronic effects at 120 µg/L (US EPA, 1975, pg. 59576, EPA-570/9-76-003).

**Water Quality Criteria:** In 1980, EPA announced the availability of Water Quality Criteria Documents to protect surface water bodies from pollutants under the Clean Water Act (45 FR 79318, November 28, 1980). These criteria are used as guidance to the States in establishing surface water quality standards and discharge limits for effluents. The criterion for protection of human health from ingestion of arsenic in contaminated water and aquatic organisms was 2.2 nanograms per liter (ng/L), or 0.0022 µg/L. In 1992, the Clean Water Act criterion was recalculated based on an updated risk assessment to yield 0.018 µg/L for arsenic (57 FR 60848, December 22, 1992).

**1983 Notice prior to proposal:** In an Advance Notice of Proposed Rulemaking (ANPRM) published October 5, 1983 (48 FR 45502), EPA requested comment on whether the arsenic MCL should consider carcinogenicity, other health effects, and nutritional requirements; and whether MCLs are necessary for separate valence states.

**1985 Proposed MCLG:** In 1985, EPA proposed a non-enforceable Maximum Contaminant Level Goal (MCLG) of 50 µg/L based on an NAS conclusion that 50 µg/L balanced toxicity and possible essentiality. EPA also requested comment on alternate MCLGs of 100 µg/L based on non-carcinogenic effects and 0 µg/L based on carcinogenicity (50 FR 46936, November 13, 1985).

**1986 SDWA Amendments:** The 1986 SDWA Amendments converted the 1975 interim arsenic standard to a NPDWR, subject to revision by 1989.

**1988 Risk Assessment Forum Report:** EPA's Risk Assessment Forum wrote the *Special Report on Ingested Inorganic Arsenic: Skin Cancer; Nutritional Essentiality* (EPA/625/3-87/013), in part, to evaluate the validity of applying the Taiwan 1968/1977 data to dose-response assessments in the U.S. At the 50 µg/L standard, the calculated U.S. lifetime risk ranged from  $1 \times 10^{-3}$  to  $3 \times 10^{-3}$ .

**1989:** After reviewing EPA's arsenic health effects studies in June 1988, the Science Advisory Board (SAB) stated in its August 14, 1989, report the following:

- The essentiality of arsenic is suggestive but not definitive;
- Hyperkeratosis may not be a precursor of skin cancer;
- The Taiwan data are adequate to conclude that high doses of ingested arsenic can cause skin cancer;
- The Taiwan study is inconclusive to determine cancer risk at levels ingested in the U.S.; and
- As (III) levels below 200 to 250 µg per day may be detoxified.

SAB concluded that the dose-response is non-linear and reported that the 1988 Forum Report did not apply non-linearity in its risk assessment.

**1989:** Uncertainty about arsenic risk assessment issues caused the Agency to miss the 1989 deadline for proposing a revised NPDWR, and a citizen suit was filed against EPA. A consent decree was entered by the court in June 1990 and was amended several times thereafter before being dismissed after passage of the 1996 SDWA Amendments.

**The Safe Drinking Water Act Amendments of 1996**, in §1412(b)(12)(A), directed EPA to take the following actions for arsenic:

- Develop an arsenic health effects research strategy within 180 days of enactment;
- Consult with the National Academy of Sciences, other Federal agencies, and interested public and private entities in conducting the studies;
- Propose a revised MCL by January 1, 2000; and
- Issue a final rule by January 1, 2001.

In addition SDWA, as amended in 1996, directed EPA to:

- Assess health effects for sensitive populations;
- List both compliance and/or variance treatment technologies for small systems;
- Evaluate the incremental costs and benefits of different regulatory options, accounting for the changes that may result from implementation of other rules;
- Issue an MCL that maximizes health benefits at a cost that is justified by the benefits;
- Review MCLs every six years or sooner.

The 1996 amendments also made the following changes:

- The effective date of MCLs is three to five years after promulgation of the final rule, rather than 18 months.

- Compliance for non-microbial contaminants can be achieved by use of point-of-use (POU) or point-of-entry (POE) devices that are maintained by the small public water system.

Congress authorized \$2.5 million per year from 1997 to 2000 for the studies. Congress appropriated \$1 million to EPA for arsenic research in 1996 and 1997 and \$1 million to the American Water Works Association Research Foundation in subsequent years.

EPA proposed the arsenic regulation on June 22, 2000, in the *Federal Register*. At the same time, EPA is proceeding with its Arsenic Research Plan, which will address a variety of issues related to exposure, treatment, and health effects.<sup>1</sup> In EPA's appropriations bill for 2001, Public Law 106-377, Congress directed EPA to issue the final arsenic rule by June 22, 2001, one year after proposal.

**NRC Report:** In 1996, EPA requested that the National Research Council of NAS conduct an independent review of the arsenic toxicity data and evaluate the scientific validity of EPA's 1988 risk assessment for arsenic in drinking water. In addition, NRC was asked to review EPA's current criteria (50 µg/L and 0.018 µg/L), evaluate use of recent Taiwan data and other studies to assess the carcinogenic and non-carcinogenic health effects of arsenic, and recommend changes to EPA's risk characterization for arsenic. NRC issued its report on March 23, 1999. The report had several main conclusions:

- The Taiwan studies provide the best available evidence on the human health effects of arsenic, and are supported by studies in Chile and Argentina that report similar results. These studies show that chronic ingestion of inorganic arsenic at high doses causes bladder and lung cancer, as well as skin cancer.
- Factors such as genetics, nutrition, and amount of arsenic in food can affect the U.S. risk assessment.
- Non-cancer chronic effects include skin effects, cardiovascular and cerebrovascular disease, diabetes, and reproductive effects.
- The molecular processes of arsenic toxicity are not well understood. Research can help characterize the dose-response relationship for both cancer and non-cancer endpoints, especially at low doses.
- The current 50 µg/L MCL is not adequately protective of human health and therefore requires downward revision as promptly as possible.<sup>2</sup>

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<sup>1</sup>The Arsenic Research Plan is published at <http://www.epa.gov/ORD/WebPubs/final/arsenic.pdf>.

<sup>2</sup>The NRC report is available at <http://www.nap.edu/readingroom/enter2.cgi?0309063337.html>.

## 2.4 Rationale for the Regulation

This section discusses the economic rationale for choosing a regulatory approach to address the public health consequences of drinking water contamination. EPA provides the economic rationale in response to Executive Order Number 12866, *Regulatory Planning and Review*, which states:

[E]ach agency shall identify the problem that it intends to address (including, where applicable, the failures of the private markets or public institutions that warrant new agency action) as well as assess the significance of that problem (§1, b(1)).

In addition, guidance from the Office of Management and Budget dated January 11, 1996, states that “in order to establish the need for the proposed action, the analysis should discuss whether the problem constitutes a significant market failure.” Therefore, the economic rationale presented in this section should not be interpreted as EPA’s approach to implementing the SDWA. Instead, it is EPA’s justification, as required by the Executive Order, for a *regulatory approach* to this public health issue.

### 2.4.1 Statutory Authority

Section 1412(b)(1)(A) of the SDWA requires EPA to establish National Primary Drinking Water Regulations for contaminants that may have an adverse public health effect; that are known to occur or that present a substantial likelihood of occurring once in public water systems (PWSs), at a frequency and level of public concern; and that present a meaningful opportunity for health risk reduction for persons served by PWSs. This general provision is supplemented by additional requirements that EPA proposed a revised MCL for arsenic by January 1, 2000 (§1412(b)(1)(A)), and issue a final regulation by June 22, 2001 (Public Law 106-377).

### 2.4.2 Economic Rationale for Regulation

In addition to the statutory directive to regulate arsenic, there is also economic rationale for government regulation. In a perfectly competitive market, market forces guide buyers and sellers to attain the best possible social outcome. A perfectly competitive market occurs when there are many producers of a product selling to many buyers, and both producers and buyers have complete knowledge regarding the products of each firm. Also, there must not be any barriers to entry into the industry, and producers in the industry must not have any advantage over potential new producers. Several factors in the public water supply industry do not satisfy the requirements for a perfect market and lead to market failures that may require regulation.

First, water utilities are natural monopolies. A natural monopoly exists because it is not economically efficient to have multiple suppliers competing to build multiple systems of pipelines, reservoirs, wells, and other facilities.<sup>3</sup> Instead, a single firm or government entity performs these functions generally under

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<sup>3</sup>Mansfield (1975) states that natural monopolies exist because the average cost of producing the product reaches a minimum at an output rate that is enough to satisfy the entire market at a price that is profitable. Multiple

public control. Under monopoly conditions, consumers are provided only one level of service with respect to the quality of the product, in this case drinking water quality. If consumers do not believe that the market of safety in public health production is adequate, they cannot simply switch to another water utility or perceived higher quality source of supply (e.g., bottled water) without incurring additional cost.

Second, high information and transaction costs impede public understanding of the health and safety issues concerning drinking water quality. The types of health risks potentially posed by trace quantities of drinking water contaminants involve analysis and distillation of complex toxicological data and health sciences. EPA recently developed the Consumer Confidence Report rule to make water quality information more easily available to consumers. The Consumer Confidence Report rule requires community water systems to mail their customers an annual report on local drinking water quality. However, consumers will still have to analyze this information for its health risk implications. Even if informed consumers are able to engage utilities regarding these health issues, the costs of such engagement, known as “transaction costs” (in this case measured in personal time and commitment), present another significant impediment to consumer expression of risk preference.

SDWA regulations are intended to provide a level of protection from exposure to drinking water contaminants that would not otherwise occur in the existing market environment of public water supply. The regulations set minimum performance requirements for all public water supplies in order to reduce the risk confronted by all consumers from exposure to drinking water contaminants. SDWA regulations are not intended to restructure market mechanisms or to establish competition in supply. Rather, SDWA standards establish the level of service to be provided in order to better reflect public preference for safety. The Federal regulations remove the high information and transaction costs by acting on behalf of all consumers in balancing the risk reduction and the social costs of achieving this reduction.

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producers competing would produce the product at higher than minimum long-run average cost. Competition to achieve lower average costs would drive prices down until a single supplier was victorious.

## Chapter 3: Consideration of Regulatory Alternatives

### 3.1 Regulatory Approaches

The Safe Drinking Water Act (SDWA) establishes EPA's responsibility for ensuring the quality of drinking water and defines the mechanisms available to the Agency to protect public health. Specifically, the SDWA requires EPA to set enforceable MCLs when technically or economically feasible or otherwise establish treatment technique requirements for specific contaminants in drinking water. In meeting this mandate, EPA sets water quality standards by identifying which contaminants should be regulated and establishing the levels of the contaminant that water systems must attain. This section discusses the approach EPA used in determining the regulatory alternatives that were considered.

#### 3.1.1 Determining the Standard

In regulating a contaminant, EPA first sets a maximum contaminant level goal (MCLG), which establishes the contaminant level at which no known or anticipated adverse health effects occur. MCLGs are non-enforceable health goals. For this rulemaking, EPA set an MCLG of zero. EPA then sets an enforceable maximum contaminant level (MCL) as close as technologically possible to the MCLG. In addition, EPA may use its discretion in setting the MCL by choosing an MCL that is protective of public health while also ensuring that the quantified and non-quantified costs are justified by the quantified and non-quantified benefits of the rule. For this rulemaking, EPA is setting an MCL of 10 µg/L. The following sections describe the process by which EPA determined both the MCLG and the MCL.

#### 3.1.2 Determining the MCLG

**Carcinogens:** For many years, Congress supported a goal of zero tolerance for carcinogens in food and water, and that goal was incorporated into the SDWA of 1974. Under this policy, contaminants that are classified as probable human carcinogens have had MCLGs set at zero. EPA's Office of Science and Technology (OST) (in the Office of Water) develops a cancer risk range that quantifies the probability that a person will develop cancer during a lifetime of ingesting water containing the regulated contaminant.

Data used in risk estimates usually come from lifetime exposure studies in animals. To predict the risk for humans, the oral doses used in animal studies are corrected for differences in animal and human size and surface area.

In 1986, EPA published *Guidelines for Carcinogen Risk Assessment* in the Federal Register (51 FR 33992). At that time EPA's default assumptions included low-dose linearity to extrapolate the cancer risk range, which assumes that carcinogenic effects do not exhibit a threshold and that carcinogens pose risks to humans at any concentration. EPA proposed revised *Guidelines for Carcinogen Risk Assessment* in 1996 (61 FR 17960).

**Non-carcinogens:** MCLGs for non-carcinogens are based on Reference Doses (RfDs) and their Drinking Water Equivalent Levels (DWELs).

The Reference Dose (RfD, formerly the Acceptable Daily Intake, or ADI), estimates the daily amount of chemical a person, including sensitive humans, can ingest over a lifetime with little risk of causing adverse health effects. RfDs are usually expressed in milligrams of chemical per kilogram of body weight per day (mg/kg/day). Data from chronic (usually two years) or sub-chronic (usually 90 days) studies of humans or animals provide estimates of the No- or- Lowest-Observed-Adverse-Effect Level (NOAEL or LOAEL). The NOAEL (or LOAEL) is divided by a total uncertainty factor (UF) of 1 to 10,000 to obtain the RfD. In the final National Primary Drinking Water Regulations published on January 30, 1991 (56 FR 3532), EPA applies a UF of 1, 3, or 10 when a NOAEL from a human study is used to account for intraspecies variation and an uncertainty factor of 100 to a human LOAEL to account for lack of a NOAEL and for species variation. The UFs provide a margin for variations in species responses, data gaps, and less than lifetime exposures. Scientific judgement is used to select the total UF for specific risk assessments.

The DWEL is calculated by multiplying the RfD by an assumed adult body weight of 70 kg (approximately 154 pounds) and dividing by an average adult water consumption of 2 liters per day (L/day). The DWEL assumes that 100 percent of the exposure comes from drinking water. The MCLG is then determined by multiplying the DWEL by the percentage of the total daily exposure contributed by drinking water (relative source contribution), set at 20 percent by default when adequate data are not available, but set between 20 and 80 percent when adequate data are available to estimate exposure. Based on the 1993 RfD (1993 Draft Criteria) for arsenic (0.3 µg/kg/day), the calculated DWEL would be 0.3 µg/kg/day times 70 kg divided by 2 L/day, or 10 µg/L. Due to the three-fold uncertainties noted in the Integrated Risk Information System (IRIS) file on arsenic, the DWEL could be 3 to 30 µg/L. It should be noted that the toxicological studies used to determine the effect level and the derivation of the RfD are different from the analysis conducted in 1975. Additionally, the current policy on relative source contribution, including the default policy, are also different from those used in 1975.

### **3.1.3 Determining an MCL**

Once an MCLG is established, EPA sets an enforceable standard—in most cases, a Maximum Contaminant Level (MCL). The MCL is the maximum permissible level of a contaminant in water that is delivered to any user of a public water system. EPA must set the MCL as close to the MCLG as feasible. The SDWA defines feasible as the level that may be achieved with the use of the best available technology, treatment techniques, and other means that EPA finds are available (after examination for efficacy under field conditions), taking cost to large systems into consideration.

After determining an MCL based on affordable technology for large systems, EPA must complete an economic analysis to determine whether the benefits of the standard justify the costs. If not, EPA may adjust the MCL to a level that “maximizes health risk reduction benefits at a cost that is justified by the benefits” (§1412(b)(6)).

### **3.1.4 Variances**

The 1996 SDWA identifies two classes of technologies for small systems: compliance and variance technologies. A compliance technology is one that achieves compliance with the MCL or treatment technique requirement. The 1996 Amendments require EPA to list affordable compliance technologies for three categories of small systems: those serving 25 to 500 people, those serving 501 to 3,300 people, and those serving 3,301 to 10,000 people. If EPA cannot identify an affordable compliance technology for a particular system category, it must then identify a variance technology instead. The variance technology must achieve the maximum reduction that is affordable, considering the size of the system and the quality of the source water, and must be protective of public health. If EPA lists such a variance technology, small systems will be eligible to apply to the States for a small system variance. States are authorized to grant variances from standards for systems serving up to 3,300 people if the system cannot afford to comply with a rule and the system installs the EPA-approved variance technology. States can grant variances to systems serving 3,301 to 10,000 people with EPA approval.

### **3.1.5 Analytical Methods**

The determination of an MCL depends on the ability of laboratories to reliably measure the contaminant at the MCL. The SDWA directs EPA to set an MCL “if in the judgement of the Administrator, it is economically and technologically feasible to ascertain the level of such contaminant in water in public water systems (§1401 (1)(c)(ii)).” EPA must therefore evaluate the available analytical methods to determine a Practical Quantitation Limit (PQL), which is the minimum reliable quantification level that most laboratories can be expected to meet during day-to-day operations. EPA has approved several analytical methods to support compliance monitoring of arsenic at the current MCL (40 CFR 141.23). In 1994, EPA evaluated available data and determined the PQL for arsenic to be 2.0 µg/L at an acceptance limit of ± 40 percent. In its July 1995 report, EPA’s Science Advisory Board recommended that EPA set the PQL for arsenic using acceptance limits similar to those applied for other inorganics. Based on more recent information and these recommendations from the SAB, in 1999 EPA derived a PQL of 3 µg/L using an acceptance limit of ± 30 percent for arsenic (EPA, 1999a).

Available data estimate that over 75 percent of EPA Regional and State laboratories and at least 62 percent of non-EPA laboratories are capable of achieving acceptable results at 3 µg/L within a 30 percent acceptance window. While the PQL represents a stringent target for laboratory performance, the Agency believes that most laboratories, using appropriate quality assurance and quality control procedures, have the capacity to achieve this level on a routine basis.

## **3.2 Regulatory Alternatives Considered and Final Rule**

This section describes the components of the final rule and the alternatives that were considered by the Agency.

### 3.2.1 Applicability

The Agency investigated applying the monitoring and treatment requirements of the proposed rule to both community water systems (CWSs) and non-transient non-community (NTNC) water systems. A CWS is defined as a system that provides piped water to at least 25 people or with at least 15 service connections year-round. An NTNC system is a public water system that is not defined as a CWS and that regularly serves at least 25 of the same people for at least six months of the year. After considering the costs and benefits of the proposed rule with regard to both CWSs and NTNC systems, EPA proposes to require both CWSs and NTNC water systems to comply with all facets of the proposed rule. The benefit-cost analysis upon which this decision is based is provided in Chapters 5, 6, and 7 of this EA. Transient non-community systems, which provide potable water to continuously changing populations, will not be subject to the proposed rule. The rule applies to CWSs and NTNC systems that produce water primarily from either ground or surface water sources.

### 3.2.2 Maximum Contaminant Level

EPA considered a range of MCLs in developing the proposed Arsenic Rule, including MCLs of 3, 5, 10, and 20 µg/L. EPA evaluated the following five factors to determine the proposed MCL:

- The analytical capability and laboratory capacity;
- The likelihood of water systems choosing various compliance technologies for several sizes of systems based on source water properties;
- The national occurrence of arsenic in water supplies;
- Quantified and non-quantified costs and health risk reduction benefits likely to occur at the MCLs considered; and
- The effects on sensitive subpopulations.

An MCL of 3 µg/L was considered since this is the level that has been determined to be as close to the MCLG as is feasible. However, the Agency is using its discretionary authority in §1412(b)(6)(A) to set MCL at a less stringent level. The statute requires that the alternative, less stringent level be one that maximizes health risk reduction at a level where costs and benefits are balanced.

As a result, EPA considered the alternative MCL options of 5, 10, and 20 µg/L.

### 3.2.3 Monitoring

The current monitoring requirements for arsenic (40 CFR 141.23(l)) apply to community water systems only. EPA is changing the current monitoring requirements to require systems to monitor for arsenic in accordance with the provisions of 40 CFR 141.23(c), the Standard Monitoring Framework (SMF). This change will make the arsenic requirements consistent with the requirements for inorganic contaminants (IOCs) regulated under the Phase II/V regulations. The revised rule would make the following changes to the monitoring requirements for arsenic:

- NTNC systems will be required to monitor for arsenic for the first time.
- MCL exceedances will trigger quarterly monitoring, as opposed to the current requirements for three additional samples within one month when exceedances occur.
- The State will determine when the system is “reliably and consistently” below the MCL, after a minimum number of samples following an exceedance (two samples for ground water systems and four for surface water systems), and can return to the default sampling frequency. (Currently, the system automatically returns to the default monitoring frequency when a minimum of two consecutive samples are below the MCL.)
- The State may grant a nine-year monitoring waiver to a system if it finds that arsenic detections are the result of natural occurrence and not of human activity. (Currently, no monitoring waivers are permitted.)

### 3.2.4 Compliance Technologies and Variances

EPA reviewed several technologies as best available technology (BAT) candidates for arsenic removal. Those technologies capable of removing arsenic from source water that fulfill the SDWA requirements for BAT determinations for arsenic are as follows:

- Anion exchange;
- Activated alumina (AA);
- Reverse osmosis (RO);
- Modified coagulation/filtration;
- Modified lime softening; and
- Oxidation/filtration (including greensand filtration).<sup>1</sup>

EPA has further determined that these technologies are affordable for all system size categories and has therefore not identified a variance technology for any system size or source water combination at the proposed MCL.

### 3.2.5 Monitoring Waivers

Under the final Arsenic Rule (§141.23(c)(3)), States may grant a nine-year monitoring waiver from sampling requirements to water systems based on the analytical results from previous sampling and a vulnerability assessment or the assessment from an approved source water assessment program (provided that the assessments were designed to collect all of the necessary information needed to complete a vulnerability assessment for a waiver). States issuing waivers must consider the requirements in 40 CFR 141.23(c)(2)-(6). In order to qualify for a waiver, there must be three previous samples from a sampling point (annual for surface water and three rounds for ground water) with analytical results reported below the MCL. Grandfathered data collected after January 1, 1990,

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<sup>1</sup>Oxidation/filtration is BAT only when the Fe/As ratio is > 20:1.

that are consistent with the analytical methodology and detection limits of the proposed regulation may be used for issuing sampling point waivers.

The current arsenic regulations §141.23(l)-(q) do not permit the use of monitoring waivers. However, a State could now use the analytical results from the three previous compliance periods (1993 to 1995, 1996 to 1998, and 1999 to 2001) to issue ground water sampling point waivers. Surface water systems must collect annual samples; thus, a State could use the previous three years' sampling data (1999, 2000, and 2001) to issue sampling point waivers. One sample must be collected during the nine-year compliance cycle in which the waiver is effective, and the waiver must be renewed every nine years. Vulnerability assessments must be based on a determination that the water system is not susceptible to contamination and arsenic is not a result of human activity (i.e., it is naturally occurring).

Not all States have required systems to report arsenic results below 50 µg/L. In this case, the States would not have adequate data to grant waivers until enough data are available to make the determinations.

EPA believes that some States may have been regulating arsenic under the proposed standardized inorganic framework. If so, those States will have to ensure that existing monitoring waivers have been granted using data reported below the new MCL. Otherwise, States will have to notify the systems of the new lower reporting requirements that need to be met to qualify for a waiver for the MCL.

### **3.2.6 Implementation**

The following schedule is proposed for implementation of the rule:

- States must submit applications for primacy revisions within two years after promulgation, unless a State requests and is granted a two-year extension.
- The rule will be effective five years after promulgation.
- All systems must complete initial sampling by December 31, 2007.

## Chapter 4: Baseline Analysis

### 4.1 Introduction

This chapter presents baseline information to describe the operational and financial characteristics of water systems in the absence of the Revised Arsenic Rule. The baseline information provides a basis for EPA's analysis of the costs, benefits and economic impacts of the regulatory options considered. This chapter includes data on the number of water systems regulated, the population affected, current treatment practices, raw and treated water quality, and socio-economic impacts.

The baseline is assumed to be current conditions, as reflected by the most recent available data. In some cases, changes in the industry have occurred or will occur that are not reflected in the available data; for example, changes in operations induced by a regulation that will take effect prior to the Arsenic Rule.

### 4.2 Industry Profile

#### 4.2.1 Definitions

According to EPA's definition, public water systems (PWSs) include community water systems (CWSs) and non-community water systems (NCWSs). NCWSs are further classified as either transient or non-transient. The rule will affect all public water systems except for transient non-community water systems. The following definitions will help the reader follow the discussion in this chapter:

- **Public water systems (PWSs)** serve 25 or more people or have 15 or more service connections and operate at least 60 days per year. A PWS can be publicly or privately-owned.
- **Community water systems (CWSs)** serve at least 15 service connections used by year-round residents, or regularly serve at least 25 year-round residents.
- **Non-community water systems (NCWSs)** do not have year-round residents, but serve at least 15 service connections used by travelers or intermittent users for at least 60 days each year, or serve an average of 25 individuals for at least 60 days a year.
- **Non-transient non-community water systems (NTNCs)** serve at least 25 of the same persons over six months per year (e.g., factories, schools, office buildings, and hospitals).
- **Transient non-community water systems (TNCs)** serve fewer than 25 of the same persons over six months per year (e.g., many restaurants, rest stops, parks).

Public water systems are also classified by their water source: surface water (e.g., drawn from lakes, streams, rivers, etc.) or ground water (e.g., drawn from wells or springs).

## 4.2.2 Sources of Industry Profile Data

EPA uses two primary sources of data to characterize the universe of water systems: the Safe Drinking Water Information System (SDWIS) and the Community Water System Survey (CWSS).

EPA's SDWIS contains data on all PWSs as reported by States and EPA Regions. This source reflects both mandatory and optional reporting components. States must report the system location, system type (CWS, NTNC, or TNC), primary raw water source (ground water or surface water), and violations. Optional reporting fields include type of treatment and ownership type. Because providing some data is discretionary, EPA does not have complete data on every system for these parameters. This is particularly common for non-community systems.

The second source of information, the CWSS, is a detailed survey of surface and ground water CWSs conducted by EPA in 1995 and published in 1997 (EPA, 1997b). The CWSS is stratified to represent the complete population of CWSs across the U.S. The CWSS includes information such as revenues, expenses, treatment practices, source water protection measures, and plant capacity. There is no equivalent survey such as the CWSS to define treatment practices in non-community water systems.

## 4.2.3 Number and Size of Public Water Systems

Exhibit 4-1 shows the number of systems in the U.S. by source water (ground or surface) and system size (measured by the number of people served), based on the December 1998 SDWIS data.<sup>1</sup> In the U.S. there are a total of 63,984 ground water systems and 11,843 surface water systems, including CWSs and NTNCs. All are potentially affected by the Arsenic Rule.

Some ground water sources (e.g., riverbank infiltration/galleries) are directly impacted by adjacent source water bodies and are separately identified in SDWIS as ground water under the direct influence of surface water (GWUDI). Since these systems would have similar occurrence as surface water systems, GWUDI systems are considered surface water systems in this analysis. SDWIS also provides system data by ownership. As previously described, PWSs include both publicly-owned and privately-owned systems. This detail is also provided in Exhibit 4-1, where any system referred to as "other" in the SDWIS database has been presented as a privately-owned system.

The majority (95 percent) of PWSs are small systems that serve fewer than 10,000 people. Eighty-nine percent of PWSs serve 3,300 people or fewer; 77 percent serve fewer than 1,000 people; 67 percent serve fewer than 500 people; and 34 percent serve fewer than 100 people.

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<sup>1</sup>The cost and benefit analyses are conducted using the 1997 SDWIS freeze. The 1998 SDWIS freeze is presented here, as it was the most recent representation of the regulated entities.

**Exhibit 4-1  
Total Number of Systems by Size, Type, and Ownership**

<b>SOURCE</b>	<b>&lt;100</b>	<b>101- 500</b>	<b>501- 1,000</b>	<b>1,001- 3,300</b>	<b>3,301- 10,000</b>	<b>10,001- 50,000</b>	<b>50,001- 100,000</b>	<b>100,001- 1,000,000</b>	<b>TOTAL</b>
<b>CWS</b>									
<b>Ground Water</b>									
Public	1,335	4,678	2,868	4,167	1,993	1,011	105	50	16,207
Private	12,942	10,380	1,821	1,547	466	205	26	11	28,303
Total	14,277	15,058	4,689	5,714	2,459	1,216	131	61	44,510
<b>Surface Water</b>									
Public	394	1,117	917	2,012	1,656	1,436	260	217	8,009
Private	698	886	303	408	188	171	40	44	3,053
Total	1,092	2,003	1,220	2,420	1,844	1,607	300	261	11,062
Total	15,369	17,061	5,909	8,134	4,303	2,823	431	322	54,352
<b>NTNCWS</b>									
<b>Ground Water</b>									
Public	1,725	3,108	1,163	337	23	9	0	0	6,365
Private	7,965	3,930	815	355	39	5	0	0	13,109
Total	9,690	7,038	1,978	692	62	14	0	0	19,474
<b>Surface Water</b>									
Public	58	63	19	24	6	3	1	1	175
Private	213	232	87	56	17	1	0	0	606
Total	271	295	106	80	23	4	1	1	781
Total	9,961	7,333	2,084	772	85	18	1	1	20,255

Source: Safe Drinking Water Information System (SDWIS), December 1998 freeze.

#### 4.2.4 System Size and Population Served

All PWSs are potentially subject to the requirements of the Arsenic Rule, with the exception of TNCs. The majority of systems to be regulated are community water systems, which also serve, on average, more people than NTNCs. Exhibit 4-2 provides information on the average populations served by CWSs for each system size category, and the total population served by NTNCs.

**Exhibit 4-2**  
**Total Population Served of Water Systems by**  
**Source Water, System Type, and Service Population Category**

Service Population Category	Community		Non-Transient Non-Community
	Ground Water	Surface Water	
< 100	859,777	61,450	-
101–500	3,741,017	570,448	-
501–1,000	3,457,163	921,449	-
1,001–3,300	10,631,422	4,797,855	-
3,301–10,000	14,095,015	10,995,980	-
10,001–50,000	25,004,779	36,819,575	-
50,001–100,000	8,609,455	20,500,370	-
100,001–1,000,000	14,575,556	65,375,183	-
> 1,000,000	2,855,494	28,658,586	-
<b>Total</b>	<b>83,829,678</b>	<b>168,700,896</b>	<b>31,968,181</b>

Source: EPA, Safe Drinking Water Information System (SDWIS), December 1998 freeze.

Those NTNCs determined to be affected by the Arsenic Rule are presented in Exhibit 4-3 by type of system. The NTNC populations were taken from the 1998 SDWIS freeze. The NTNCs are much smaller than CWSs on average and vary substantially in their characteristics. Schools account for more than half of the affected NTNCs (8,414 of 20,255), followed by office parks (950), daycare centers (809), food manufacturing facilities (768), and non-food related retailers (695). Prisons serve the largest number of people on average (1,820). All other system types serve an average of 500 people or fewer.

**Exhibit 4-3  
Characteristics of NTNC Systems Affected by the Revised Rule**

Service Area Type	SYSTEM CHARACTERISTICS			
	Number of Systems	Average Population Served Per System	Design Flow (mgd)	Average Daily Flow (mgd)
Daycare Centers	809	76	0.0051	0.0011
Highway Rest Areas	15	407	0.0089	0.0020
Hotels/Motels	351	133	0.0189	0.0045
Interstate Carriers	287	123	0.0029	0.0006
Medical Facilities	367	393	0.1166	0.0339
Mobile Home Parks	104	185	0.0262	0.0065
Restaurants	418	370	0.0039	0.0008
Schools	8414	358	0.0333	0.0085
Service Stations	53	230	0.0051	0.0011
Summer Camps	46	146	0.0218	0.0053
Water Wholesalers	266	173	0.1637	0.0494
Agricultural Products/Services	368	76	0.0199	0.0048
Airparks	101	60	0.0026	0.0005
Construction	99	53	0.0009	0.0002
Churches	230	50	0.0053	0.0011
Campgrounds/RV Parks	123	160	0.0214	0.0052
Fire Departments	41	98	0.0186	0.0045
Federal Parks	20	39	0.0065	0.0014
Forest Service	107	42	0.0014	0.0002
Golf and Country Clubs	116	101	0.0118	0.0027
Landfills	78	44	0.0053	0.0011
Mining	119	113	0.0123	0.0028
Amusement Parks	159	418	0.0171	0.0041
Military Bases	95	395	0.0695	0.0192
Migrant Labor Camps	33	63	0.0102	0.0023
Misc. Recreation Services	259	87	0.0025	0.0005
Nursing Homes	130	107	0.0411	0.0107
Office Parks	950	136	0.0077	0.0017
Prisons	67	1820	0.5322	0.1820
Retailers (Non-food related)	695	174	0.0038	0.0008
Retailers (Food related)	142	322	0.0058	0.0012
State Parks	83	165	0.0048	0.0010
Non-Water Utilities	497	170	0.0133	0.0031
Manufacturing: Food	768	372	0.0454	0.0120
Manufacturing: Non-Food	3845	168	0.0157	0.0038
<b>TOTAL</b>	<b>20,255</b>			

Source: EPA, 1999. Geometries and Characteristics of Public Water Systems, updated with the December 1998 SDWIS freeze.

## 4.2.5 Number of Entry Points

If water systems employ more than one water supply source, they may have more than one treatment facility. For estimation purposes this analysis assumes a treatment facility at every entry point to the distribution system. As a result, the total number of entry points is an important determinant of compliance costs. Exhibit 4-4 presents the distribution of entry points per ground water CWS by system service population category.

**Exhibit 4-4**  
**Average Number of Entry Points per Ground Water System**

Upper Bound 95% Confidence	Service Population Category							
	< 100	101- 500	501- 1,000	1,001 - 3,300	3,301 - 10,000	10,001 - 50,000	50,001 - 100,000	> 100,000
Percentile								
Mean	1	1	2	2	2	4	6	9
5th	1	1	1	1	1	1	1	1
50th (median)	1	1	1	1	2	3	4	5
95th	2	3	3	5	5	12	22	28

Source: EPA, 1999. *Geometries and Characteristics of Public Water Systems*, Table 5.2.

In this respect, surface water systems are unlike ground water systems in that little variation in the number of entry points was reported among surface water systems. Even for large population categories, the majority of surface water systems reported only one or two entry points. (EPA, 1999a). This finding was supported by data recently collected from the Information Collection Request for large surface water systems. Appendix C describes how the entry point distribution was incorporated into the cost analysis for this rule.

## 4.2.6 Number of Households

Another method for estimating the effect of regulations on customers is to determine the cost per household. This measure is often used instead of per capita cost because it is a more accurate representation of how customers are billed: per household, not per person.

Exhibit 4-5 shows that household consumption does not vary substantially across size category or ownership type. The mean water consumption ranges from 81,000 gallons per year to 127,000 gallons per year per household.

**Exhibit 4-5  
Water Consumption per Residential  
Connection**

Population	System Type	Mean Water Consumption* (kgal/yr)
< 100	Public	81
	Private	92
101-500	Public	93
	Private	110
501-1,000	Public	97
	Private	88
1,001-3,300	Public	82
	Private	102
3,301-10,000	Public	87
	Private	124
10,001-50,000	Public	108
	Private	110
50,001-100,000	Public	122
	Private	96
100,001-1,000,000	Public	127
	Private	114

Source: \*EPA, 1997. *CWSS, Vol. II: Detailed Summary Result Tables and Methodology Report*, Table 1-14;

#### **4.2.7 Production Profile**

Exhibit 4-6 shows the average design capacity (in thousands of gallons) of CWS plants by source, ownership, and system size categories. Design capacity is the maximum amount of water a plant can deliver. Exhibit 4-7 provides the daily production of CWSs (in thousands of gallons) for the same categories. Daily production is the average amount of water a plant delivers in a day.

**Exhibit 4-6**  
**Design Capacity of CWS Plants**  
**by Source, Ownership, and System Size**  
**(Thousands of Gallons)**

Primary Source/ Ownership Type	Service Population Category									
	<25	25-100	101-500	501-1,000	1,001-3,300	3,301-10,000	10,001-50,000	50,001-100,000	100,001-1,000,000	>1,000,000
<b>Ground Water</b>	6.27	21.86	86.86	251.0	619.5	1,864	6,673	20,785	67,379	392,939
Public	4.84	29.46	123.67	305.0	740.3	2,152	7,365	22,614	67,994	401,175
Private	6.50	21.34	77.30	232.1	560.6	1,683	6,347	18,234	75,629	-
Purchased-Public	-	5.71	27.37	81.4	223.0	801	3,380	19,796	26,765	-
Purchased-Private	0.89	4.99	24.78	79.5	200.6	824	2,748	8,690	-	-
<b>Surface Water</b>	1.30	20.32	92.60	239.3	617.9	1,818	6,682	19,707	69,224	554,759
Public	1.14	25.79	130.90	318.2	807.8	2,218	7,887	22,337	77,298	584,889
Private	3.19	18.13	75.69	214.2	527.3	1,582	6,165	15,869	61,381	296,609
Purchased-Public	0.04	5.71	29.01	81.8	241.1	854	3,698	13,206	43,650	-
Purchased-Private	1.12	4.99	24.65	73.6	213.8	719	2,933	12,788	29,270	-
<b>GW under influence</b>	-	22.16	87.20	247.5	631.6	1,779	7,499	18,482	-	-
Public	-	33.29	111.32	291.2	760.0	2,077	8,992	20,195	-	-
Private	-	21.53	81.77	227.4	618.5	1,802	-	-	-	-
Purchased-Public	-	-	30.21	97.1	209.3	461	2,319	-	-	-
Purchased-Private	-	2.54	29.83	94.3	-	905	-	-	-	-

Source: EPA, *Geometries and Characteristics of Public Water Systems*, Table B1.5.3.

**Exhibit 4-7  
Daily Production of CWS Plants  
by Source, Ownership, and System Size  
(Thousands of Gallons)**

Primary Source/ Ownership Type	Service Population Category									
	<25	25-100	101-500	501-1,000	1,001- 3,300	3,301- 10,000	10,001- 50,000	50,001- 100,000	100,001- 1,000,000	>1,000,000
<b>Ground water</b>	1.35	5.33	24.40	78.50	212	715	2,914	10,187	37,224	259,751
Public	0.96	6.72	33.20	90.50	243	796	3,129	10,900	37,095	267,256
Private	1.39	4.80	20.30	69.30	18	635	2,802	9,121	44,760	-
Purchased-Public	-	5.11	23.50	68.20	182	634	2,585	14,496	19,455	-
Purchased-Private	0.85	4.54	21.60	67.30	166	656	2,119	6,502	-	-
<b>Surface Water</b>	0.39	6.91	33.70	90.70	244	753	2,932	9,069	33,667	295,680
Public	0.28	7.51	41.60	106.20	284	823	3,133	9,387	34,749	293,439
Private	0.95	6.15	28.60	87.30	230	748	3,225	8,907	38,094	206,950
Purchased-Public	0.04	5.11	24.90	68.50	197	675	2,821	9,766	31,351	-
Purchased-Private	1.06	4.54	21.50	62.40	176	575	2,258	9,472	21,215	-
<b>GW under influence</b>	-	5.41	24.50	77.30	217	679	3,313	8,951	-	-
Public	-	7.70	29.50	86.00	250	765	3,907	9,611	-	-
Private	-	4.85	21.30	67.70	207	686	-	-	-	-
Purchased-Public	-	-	25.90	80.90	171	370	1,789	-	-	-
Purchased-Private	-	2.36	25.9	79.4	-	719	-	-	-	-

Source: EPA, *Geometries and Characteristics of Public Water Systems*, Table B1.5.1.

## 4.2.8 Treatment Profile

Exhibit 4-8 below presents information regarding in-place treatment technologies that affect arsenic concentrations in delivered water. The current treatment in-place will determine the likely remedy that systems will select in order to come into compliance with the new MCL.

**Exhibit 4-8  
Percentage of CWSs with Various Treatments in Place**

Primary Source/ Type of Treatments	Service Population Category								
	< 100	101-500	501-1,000	1,001-3,300	3,301-10,000	10,001-50,000	50,001-100,000	100,001-1,000,000	> 1,000,000
<b>Ground Water Systems</b>									
Ion Exchange	0.7%	1.6%	3.8%	1.9%	4.6%	3.3%	1.2%	0.0%	-
Reverse Osmosis	0.0%	1.2%	0.0%	0.9%	1.2%	0.7%	1.2%	0.0%	-
Coagulation/ Flocc.	1.5%	5.4%	4.2%	3.4%	8.1%	15.1%	24.2%	25.2%	-
Lime/Soda Ash Softening	2.1%	3.7%	4.1%	5.2%	7.0%	12.2%	17.4%	32.4%	-
Disinfection	52.8%	77.9%	84.0%	79.7%	86.8%	96.5%	86.3%	96.4%	-
<b>Surface Water Systems</b>									
Ion Exchange	0.0%	0.0%	0.0%	0.0%	0.0%	0.0%	0.0%	0.0%	-
Reverse Osmosis	0.0%	0.0%	0.0%	0.0%	0.0%	0.0%	0.0%	0.0%	-
Coagulation/ Flocc.	27.5%	52.6%	70.2%	78.5%	95.4%	94.5%	93.7%	99.5%	-
Lime/Soda Ash Softening	3.9%	8.1%	20.5%	17.5%	10.8%	6.9%	5.7%	5.1%	-
Disinfection	92.8%	94.1%	100.0%	100.0%	96.0%	98.0%	100.0%	100.0%	

Source: EPA, *Cost and Technology Document for the Arsenic Rule*, Tables 6-1 and 6-2.

## 4.3 Occurrences of Arsenic

EPA has relied on a variety of data sources to evaluate the occurrence of arsenic in community water systems and non-transient non-community systems. This information supports EPA's assessment of baseline conditions, including (1) the number of systems expected to exceed various MCL options, and (2) the population exposed to different levels of arsenic.

In 1992, EPA conducted an analysis of the number of systems that would be impacted by various arsenic MCL options, ranging from 0.5 µg/L to > 50 µg/L. These projections were based on the following national surveys:

- 1984-1986 National Inorganic and Radionuclide Survey (NIRS) for ground water systems;
- 1976-1977 National Organic Monitoring Survey for surface water systems;
- 1978-1980 Rural Water Survey for surface water systems; and
- 1978 Community Water Supply Survey for surface water systems.

These data sources have several limitations. First, the surveys used for surface water systems were conducted primarily before 1980. It is likely that arsenic occurrence has changed in the past two decades due to changes in raw water sources or the addition of filtration treatment to comply with the Surface Water Treatment Rule (SWTR). In addition, many of the survey responses had relatively high minimum reporting limits (5 µg/L). Therefore, it is statistically difficult to extrapolate low-level arsenic occurrence.

EPA (1999c) used the MCL compliance monitoring data from 25 States to develop an improved estimate of national baseline arsenic occurrence. The estimates based on this data are comparable to those based on the other sources listed above.

EPA used statistical techniques to assess:

- (1) the national distribution of mean arsenic concentrations in water systems,
- (2) the distribution of source means within systems, and
- (3) the number of systems with at least one source above various MCLs.

Exhibit 4-9 shows the percentage of systems with an arsenic occurrence in excess of ten different concentration levels, ranging from 2 µg/L to 50 µg/L. Less than one percent of ground water and surface water systems have a concentration level of arsenic greater than 50 µg/L. In contrast, 27 percent of ground water systems and 10 percent of surface water systems have an arsenic concentration greater than 2 µg/L. Exhibit 4-10 provides a summary of the number of systems expected to exceed various MCLs.

**Exhibit 4-9**  
**Arsenic Occurrence in CWSs at Various Concentration Levels (µg/L)**

Source	% of systems greater than (µg/L)									
	2	3	5	10	15	20	25	30	40	50
<b>GW</b>	27.3	19.9	12.1	5.3	3.1	2.0	1.4	1.1	0.64	0.43
<b>SW</b>	9.8	5.6	3.0	0.80	0.46	0.32	0.24	0.19	0.13	0.10

Source: EPA, 2000. *Arsenic Occurrence in Public Drinking Water Supplies*.

**Exhibit 4-10**  
**Statistical Estimates of Numbers of Systems with**  
**Average Finished Arsenic Concentrations in Various Ranges**

System size (population served)	Number of systems with mean arsenic concentration ( $\mu\text{g/L}$ ) in the range of:			
	>3 to 5	>5 to 10	>10 to 20	>20
<b>Ground Water CWS</b>				
Number of Systems	3,384	2,949	1,432	870
% of systems	7.8%	6.8%	3.3%	2.0%
<b>Surface Water CWS</b>				
Number of Systems	270	239	51	34
% of systems	2.5%	2.2%	0.5%	0.3%
<b>Ground Water NTNCWS</b>				
Number of Systems	1,677	1,995	635	405
% of systems	8.6%	10.3%	3.3%	2.1%
<b>Surface Water NTNCWS</b>				
Number of Systems	20	17	4	2
% of systems	2.5%	2.2%	0.5%	0.3%

## Chapter 5: Benefits Analysis

### 5.1 Nature of Regulatory Benefits

The benefits associated with reductions of arsenic in drinking water arise from a reduction in adverse human health effects. To a lesser degree benefits may also accrue from an avoidance of expensive consumer behaviors aimed at avoiding exposure, such as the purchase of bottled water.

The value to consumers of a reduction in the risk of adverse health effects includes the following components:

- The avoidance of medical costs and productivity losses associated with illness;
- The avoidance of the pain and suffering associated with illness;
- The losses associated with risk and uncertainty of morbidity, also called the “risk premium”; and
- The reduction in risk of premature mortality.

This conceptual valuation framework goes beyond valuing out-of-pocket medical costs and lost time to include the value consumers place on avoiding pain and suffering and the risk premium. The risk premium represents the damages associated with risk and uncertainty, captured in the expression of consumers’ willingness to pay for the reduction in risk of illness (Freeman, 1979).

This chapter first presents information on the multiple adverse health effects associated with arsenic, followed by a quantitative risk analysis of a two arsenic-related endpoints, bladder cancer and lung cancer. Because a large number of potential health effects cannot be quantified, it is likely that the estimated benefits associated with avoidance of bladder and lung cancer underestimate the total benefits of a reduction of arsenic in drinking water.

### 5.2 Health Effects

#### 5.2.1 Overview

Exposure to arsenic has many potential health effects, which have been described in two recent publications: *Arsenic in Drinking Water* by the National Research Council (NRC, 1999), and the Agency for Toxic Substances and Disease Registry’s *Draft Toxicological Profile for Arsenic* (ATSDR, 1998, updated September 2000). These two sources provide descriptions of health effects that are summarized in this section, along with additional information provided from the recent literature.

Ingestion of inorganic arsenic can result in both cancer and non-cancer health effects (NRC, 1999). Exposure may also occur via other routes of exposure including inhalation and dermal exposure. There is a large human effects database available for inorganic arsenic. However, the effects of organic forms of arsenic are not as well characterized as those of inorganic arsenic. Limited information suggests that the major organoarsenicals found in fish and shellfish (arsenobetaine and arsenocholine) have little or no toxicity.

It appears that some of the metabolites of inorganic arsenic may possess some toxicity. The final rule addresses both organic and inorganic forms of arsenic.

The nature of the health effects avoided by reducing arsenic levels in drinking water is a function of characteristics unique to each individual and the level and timing of exposure. Therefore, the relationship between exposure and response is quite complex. This section describes potential health effects but does not conclude that there are specific effects that occur due to the current levels of arsenic in our country's drinking water.

### **5.2.2 Carcinogenic Effects**

Arsenic's carcinogenic role was noted over 100 years ago (NCI, 1999) and has been studied since that time. The Agency has classified arsenic as a Class A human carcinogen, "based on sufficient evidence from human data. An increased lung cancer mortality was observed in multiple human populations exposed primarily through inhalation. Also, increased mortality from multiple internal organ cancers (liver, kidney, lung, and bladder) and an increased incidence of skin cancer were observed in populations consuming drinking water high in inorganic arsenic." (EPA, IRIS Web site, extracted 8/99).

The International Agency for Research on Cancer (IARC) concluded that inhalation of inorganic arsenic caused skin and lung cancer in humans. The 1999 NRC report on arsenic states that "epidemiological studies ... clearly show associations of arsenic with several internal cancers at exposure concentrations of several hundred micrograms per liter of drinking water" (NRC, 1999). Ten epidemiological studies, covering eight organ systems, present quantitative data useful for risk assessment (NRC, 1999, Table 4-1). The organ systems where cancers in humans have been identified include skin, bladder, lung, kidney, nasal, liver, and prostate.

Table 10-6 of the NRC report provides risk parameters for three cancers: bladder, lung, and liver cancer. Considering all cancers in aggregate, the NRC states in their Risk Characterization section that "considering the data on bladder and lung cancer in both sexes noted in the studies in chapter 4, a similar approach for all cancers could easily result in a combined cancer risk on the order of 1 in 100" (at the current MCL of 50 µg/L; NRC, 1999).

New data provide additional health effects information on both carcinogenic and non-carcinogenic effects of arsenic. A recently study by Tsai et al. (1999) of a population that has been studied over many years in Taiwan has provided statistically significant standardized mortality ratios (SMRs) for 23 cancerous and non-cancerous causes of death in women and 27 causes of death in men. SMRs are an expression of the ratio between deaths that were observed in an area with elevated arsenic levels and those that were expected to occur, based on the mortality experience of the populations in nearby areas without elevated arsenic levels. Drinking water (250-1,140 µg/L) and soil (5.3-11.2 mg/kg) in the Tsai et al. (1999) population study had very high arsenic content.

Tsai et al. (1999) identified "bronchitis, liver cirrhosis, nephropathy, intestinal cancer, rectal cancer, laryngeal cancer, and cerebrovascular disease" as possibly "related to chronic arsenic exposure via drinking water."

In addition, the study area had upper respiratory tract cancers previously only related to occupational inhalation. High male mortality rate (SMR > 3) existed for bladder, kidney, skin, lung, and nasal cavity cancers and for vascular disease. However, the authors noted that the mortality range was marginal for leukemia, cerebrovascular disease, liver cirrhosis, nephropathy, and diabetes. Females also had high mortalities for laryngeal cancer. The SMRs calculated by Tsai et al. (1999) used the one cause of death noted on the death certificates. Many chronic diseases, including some cancers, do not result in mortality. Consequently, the impact indicated by the SMR will underestimate the total impact of these diseases.

There are, of course, differences between the population and health care in Taiwan and the United States. For example, arsenic levels in the U.S. are not nearly as high as they were in the study area of Taiwan. However, the study gives an indication of the types of health effects that may be associated with arsenic exposure via drinking water.

### **5.2.3 Non-carcinogenic Effects**

Arsenic interferes with a number of essential physiological activities, including the actions of enzymes, essential cations, and transcriptional events in cells (NRC, 1999). A wide variety of adverse health effects have been associated with chronic ingestion of arsenic in drinking water, occurring at various exposure levels.

Effects on specific organ systems reported in humans exposed to arsenic are listed below in Exhibit 5-1 (NRC, 1999). Exhibit 5-1 provides descriptive information on the specific diseases and/or symptoms associated with categories of diseases.

**Exhibit 5-1**  
**Adverse Noncarcinogenic Health Effects Reported in Humans in NRC (1999) as Potentially Associated with Arsenic, by Organ System Affected\***

Cutaneous effects	1. hyperpigmentation 2. hyperkeratoses 3. melanosis
Gastrointestinal and hepatic effects	4. noncirrhotic portal hypertension 5. gastrointestinal hemorrhage secondary to esophageal varices 6. hepatic enlargement 7. splenic enlargement 8. periportal fibrosis of the liver 9. obliterative intimal hypertrophy of intrahepatic venules resulting in obstruction of portal venous flow, increased splenic pressures, and hypersplenism, and cirrhosis of the liver 10. diarrhea 11. cramping
Cardiovascular and peripheral vascular effects	12. peripheral vascular disease (blackfoot disease) 13. gangrene of the feet 14. coldness and numbness in the extremities 15. intermittent claudication 16. ulceration 17. spontaneous amputation 18. Raynaud's syndrome 19. acrocyanosis 20. ischemic heart disease
Cardiovascular and peripheral vascular effects (in children)	21. arterial spasms in fingers and toes 22. esenteric artery thrombosis 23. cerebrovascular disease 24. extensive coronary occlusions 25. cerebrovascular occlusions 26. ischemia of the tongue 27. Raynaud's syndrome 28. gangrene in extremities
Hematological effects	29. anemia - normocytic, megaloblastic 30. leukopenia - neutropenia, lymphopenia, eosinophilia 31. thrombocytopenia 32. reticulocytosis 33. erythroid hyperplasia
Pulmonary effects	34. chronic cough 35. restrictive and obstructive lung disease 36. emphysema
Immunological effects	37. impaired immune response (more specific effects observed in human cell studies and animal studies—see source)
Neurological effects	38. peripheral neuropathy
Endocrine effects	39. diabetes mellitus
Reproductive and developmental effects	40. spontaneous abortion 41. perinatal death 42. stillbirth 43. low birth weight 44. birth defects including coarctation of the aorta and others 45. neural tube defects 46. ophthalmic abnormalities 47. numerous skeletal abnormalities 48. urogenital abnormalities 49. growth retardation

Source: NRC (1999).

\*Notes in parenthesis indicate where health effects were observed in animal studies rather than human studies. NRC reports results of numerous animal reproductive and developmental studies and notes that there are "very few" human studies.

## 5.2.4 Susceptible Subgroups

This section discusses the nature of special susceptibilities and identifies population subgroups that may be at higher risk than the general population when exposed to arsenic.

### **Definition**

A susceptible subgroup exhibits a response that is different or enhanced when compared to the responses of most people exposed to the same level of arsenic (ATSDR, 1998). Many diseases affect certain subgroups of the population disproportionately. The subgroups may be defined by age, gender, race, ethnicity, socioeconomic status, pre-existing medical conditions, behavioral or physiological differences, or other characteristics. For example, there are pre-existing medical conditions that will increase susceptibility to most toxins, such as a pre-existing disease in the toxin's target organ. Very few diseases affect all population groups (ages, sexes, races) equally. For purposes of evaluating potential benefits to different segments of the population, it is useful to evaluate whether there are susceptible subpopulations that require consideration. The benefit of reducing their exposure may be considerably higher than the benefit associated with reducing exposure among the general population (on a per capita basis).

Special susceptibilities may be indicated by known differences in biological processes that are essential to detoxification of a toxin. In addition to identifying susceptible subgroups based on biological processes, susceptible subgroups are often identified by observing higher-than-average rates of the disease of interest. Increases in the rates of reported diseases may be due to a variety of factors. Some of these indicate an increased susceptibility; others are matters of personal choice and may not be considered relevant in a benefits analysis. One way to approach this issue is to evaluate increased susceptibility when it is based on an increased risk of disease due to factors *reasonably beyond the control of the subpopulation*. Factors that are usually beyond the control of the individual that may cause increased susceptibility include:

- Constitutional limitations (e.g., illnesses, genetic abnormalities, birth defects such as enzyme deficiencies);
- Concurrent synergistic exposures that cannot reasonably be controlled (e.g., at home or in the workplace); and
- Normal constitutional differences (i.e., differences based on sex, age, race, ethnicity, etc.).

Other factors that are not usually considered beyond the individual's control include personal choices, such as smoking, drinking, and drug use. Choice of place of residence or work may or may not be treated as a relevant factor. Ultimately, the types of factors that should be included in identifying susceptible subgroups is a matter of public policy.

No studies were located by ATSDR (1998) that focused exclusively on evaluating unusual susceptibility to arsenic. However, some members of the population are likely to be especially susceptible due to a variety of factors. These factors include increased dose (intake per unit of body weight) in children, genetic predispositions, and dietary insufficiency (ATSDR, 1998), as well as pre-existing health conditions.

## **Children**

One often-identified potential susceptible subgroup is children. Due to their increased fluid and food intake in relation to their body weight (NAS, 1995), their dose (milligrams per kilogram of body weight per day - mg/kg/day) of arsenic will be, on average, greater than that of adults. For example, an intake of 1.2 liters per day in a 70 kg adult yields an overall water intake of 0.017 liters per kg of body weight. An infant who consumes 1 liter per day and weighs 10 kg is consuming 0.1 liter per kg of body weight, which is more than 5 times the water intake per kg of an adult. Any contaminant that is present in the water will be delivered at a correspondingly higher level, on a daily basis. Foy et al. noted that in studies of some chronic exposures, children appear to be more severely affected, probably due to a higher exposure per body weight (1992 citation, reported in ATSDR, 1998). In certain circumstances, the increased daily dose in children can be effectively considered for non-carcinogenic effects because toxicity is evaluated in terms of exposures that can range from relatively short-term to long-term exposure. However, carcinogenic effects (i.e., bladder cancer) are evaluated based on a lifetime of exposure, which takes into consideration the elevated dose that occurs in children. Because the health effects measured in this benefits assessment are bladder and lung cancer, a sensitivity analysis to consider higher doses of arsenic during childhood was not necessary. However, the numerous potential non-carcinogenic effects listed in Exhibit 5-1 may be of greater concern for children than adults. Avoidance of these effects constitutes an unquantified benefit of the rule.

## **Genetic Predispositions and Dietary Insufficiency**

Methylation of arsenic plays a role in the detoxification of inorganic arsenic, and individuals who are deficient in essential enzymes for this process, or who have a dietary deficiency of methyl donors (choline or methionine), may be at greater risk following inorganic arsenic exposure (Buchet and Lauwerys, 1987; Vahter and Marafante, 1987; Brouwer et al., 1992 cited in ATSDR, 1998). However, liver disease may not increase risk at low levels of arsenic exposure since there is a greater production of DMA in these patients. (Buchet et al., 1982; Geubel et al., 1988 cited in ATSDR, 1998). Therefore, these factors are not expected to increase risk levels for a significant portion of the U.S. population.

## **Individuals with Pre-existing Organ Susceptibilities**

Individuals may have increased susceptibilities based on specific organ-related factors. Those with pre-existing diseases (e.g., kidney disease), as well as those with congenital defects (a single kidney) will be at greater risk from a toxin that either causes additional damage to that organ, or that relies on that organ for detoxification.

In the case of arsenic, both the kidneys and liver are used to detoxify and remove the contaminant. Both single high doses and long-term low doses may cause an accumulation of arsenic in the liver and kidneys, which can impair function. In addition, these organs may be directly damaged by arsenic exposure. A review of Exhibit 5-1 indicates that other organ systems are targets of arsenic toxicity, including the cardiovascular system (heart, veins, arteries), hematopoietic system, endocrine system, cutaneous system, pulmonary system, gastrointestinal system, immune system, and peripheral nervous system. In individuals with pre-existing damage

to these systems or congenital defects in the systems, the likelihood of risk is greater. Due to the higher incidence of most types of disease among the elderly, they are more likely to have pre-existing conditions in these organ systems.

### ***Individuals Exposed via Non-water Sources***

Although arsenic is ubiquitous at low levels, it is not generally found at levels of concern in food or air, in the absence of elevated local sources. Where background levels are high, however, (e.g., elevated levels in water) it is reasonable to consider the contribution to total exposure that may occur from soil, food, and other local sources. When anthropogenic sources are known to generate elevated arsenic levels in water (e.g., a local smelter), it is more likely that other media may be contaminated as well. The total exposure from all sources is a critical component of evaluating potential health risks and the benefits of avoiding contaminated drinking water in these cases. A reduction in arsenic in drinking water will reduce the overall exposure to individuals in living in contaminated areas (e.g., around certain Superfund sites) or workers exposed to arsenic on the job. Total exposure from all sources is of particular concern for non-cancer risks, because background levels from non-drinking water sources will determine whether the total exposure leads to an exceedence of a threshold for effects.

## **5.3 Quantitative Benefits of Avoiding Cancer**

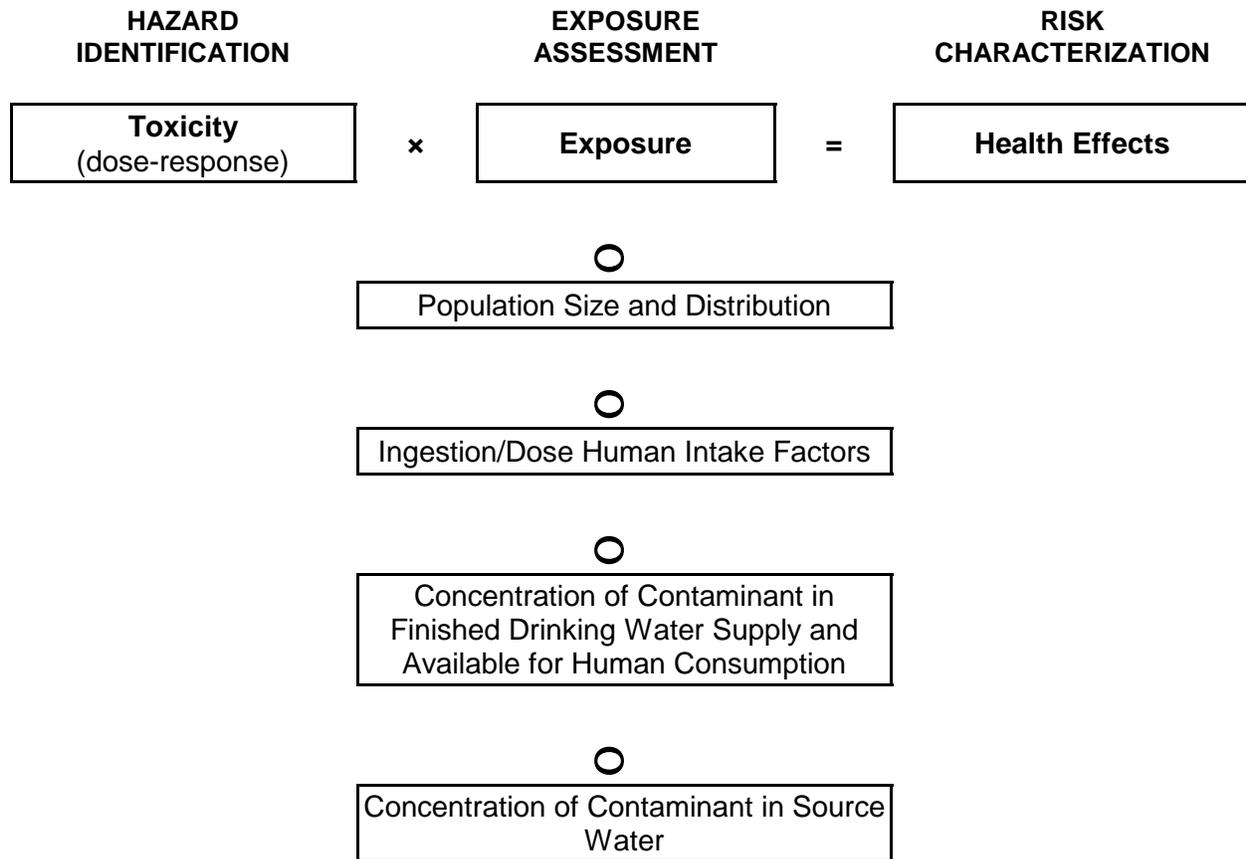
### **5.3.1 Risk Assessment for Cancer Resulting from Arsenic Exposure**

As noted, arsenic ingestion has been linked to a multitude of health effects, both cancerous and non-cancerous. These health effects include cancer of the bladder, lungs, skin, kidney, nasal passages, liver, and prostate. Arsenic ingestion has also been associated with cardiovascular, pulmonary, immunological, neurological, endocrine, and reproductive and developmental effects. A complete list of the arsenic-related health effects reported in humans has been shown in Exhibit 5-1. Of all the health effects noted above, current research on arsenic exposure has only been able to define scientifically defensible risks for bladder and lung cancer. That is, EPA has adequate data to perform a risk assessment on bladder and lung cancer. Because there is currently a lack of strong evidence on the risks of other arsenic-related health effects, the Agency has based its assessment of the quantifiable health risk reduction benefits on the risks of arsenic induced bladder and lung cancers.

Risk assessment is based on the analysis of scientific data to determine the likelihood, nature, and magnitude of harm to public health associated with particular agents, and involves three main analytical components: hazard identification (dose-response assessment), exposure assessment, and risk characterization.

Exhibit 5-2 illustrates the steps in a traditional risk assessment process for characterizing the potential human cancer associated with contaminants in drinking water.

**Exhibit 5-2  
Components of the Bladder Cancer Risk Assessment**



**5.3.2 Community Water Systems**

The following sections summarize how risk reductions were calculated for populations in community water systems exposed to arsenic concentrations. The approach for this analysis included five components. First, relative exposure factor distributions were developed, which incorporate data from the recent EPA water consumption study with age, sex, and weight data. Second, arsenic occurrence distributions were calculated for the population exposed to arsenic levels above 3 µg/L. Third, risk distributions for bladder and lung cancer were chosen for the analysis from Morales et al. (2000). Fourth, EPA developed estimates of the projected bladder and lung cancer risks faced by exposed populations using Monte-Carlo simulations, bringing together the relative exposure factor, occurrence, and risk distributions. These simulations resulted in upper bound estimates of the actual risks faced by U.S. populations exposed to arsenic concentrations at or above 3 µg/L in their drinking water. Finally, EPA made adjustments to the lower bound risk estimates to reflect exposure to arsenic in cooking water and in food in Taiwan. A more detailed description of the risk methodology is provided in Appendix B.

## ***Water Consumption***

EPA recently updated its estimates of per capita daily average water consumption (EPA, 1999). The estimates used data from the combined 1994, 1995, and 1996 Continuing Survey of Food Intakes by Individuals (CSFII), conducted by the U.S. Department of Agriculture (USDA). The CSFII is a complex, multistage area probability sample of the entire U.S. and is conducted to survey the food and beverage intake of the U.S. Per capita water consumption estimates are reported by source. Sources include community tap water, bottled water, and water from other sources, including water from household wells and rain cisterns, and household and public springs. For each source, the mean and percentiles of the distribution of average daily per capita consumption are reported. The estimates are based on an average of two days of reported consumption by survey respondents. The estimated mean daily average per capita consumption of community tap water by individuals in the U.S. population is 1 liter/person/day. For total water, which includes bottled water, the estimated mean daily average per capita consumption is 1.2 liters/person/day. These estimates of water consumption are based on a sample of 15,303 individuals in the 50 States and the District of Columbia. The sample was selected to represent the entire population of the U.S. based on 1990 Census data.

The estimated 90th percentile of the empirical distribution of daily average per capita consumption of community tap water for the U.S. population is 2.1 liters/person/day; the corresponding number for daily average per capita consumption of total water is 2.3 liters/person/day. In other words, current consumption data indicate that 90 percent of the U.S. population consumes approximately 2 liters/person/day, or less.

Water consumption estimates for selected subpopulations in the U.S. are described in the CSFII, including per capita water consumption by source for gender, region, age categories, economic status, race, and residential status and separately for pregnant women, lactating women, and women in childbearing years. The water consumption estimates by age and sex were used in the computation of the relative exposure factors discussed below.

## ***Relative Exposure Factors***

Lifetime male and female relative exposure factors (REFs) for each of the broad age categories used in the water consumption study were calculated, where the life-long REFs indicate the sensitivity of exposure of an individual relative to the sensitivity of exposure of an “average” person weighing 70 kilograms and consuming 2 liters of water per day, which is a “high end” water consumption estimate according to the EPA water consumption study referred to above (EPA, 1999). In these calculations, EPA combined the water consumption data with data on population weight from the 1994 Statistical Abstract of the U.S. Distributions for both community tap water and total water consumption were used because the community tap water estimates may underestimate actual tap water consumption. The weight data included a mean and a distribution of weight for male and females on a year-to-year basis. The means and standard deviations of the life-long REFs derived from this analysis are shown in Exhibit 5-3.

**Exhibit 5-3**  
**Life-Long Relative Exposure Factors**

	Community Water Consumption Data	Total Water Consumption Data
<b>Male</b>	Mean = 0.60 s.d. = 0.61	Mean = 0.73 s.d. = 0.62
<b>Female</b>	Mean = 0.64 s.d. = 0.6	Mean = 0.79 s.d. = 0.61

***Arsenic Occurrence***

EPA recently updated its estimates of arsenic occurrence and calculated separate occurrence distributions for arsenic found in ground water and surface water systems. These occurrence distributions were calculated for systems with arsenic concentrations of 3 µg/L or above. Arsenic occurrence estimates are described in more detail in Chapter 4.

***Risk Distributions***

In its 1999 report, *Arsenic in Drinking Water*, the NRC analyzed bladder cancer risks using data from Taiwan. In addition, the NRC examined evidence from human epidemiological studies in Chile and Argentina, and concluded that risks of bladder and lung cancer had comparable risks to those “in Taiwan at comparable levels of exposure” (NRC, 1999). The NRC also examined the implications of applying different statistical analyses to the newly available Taiwanese data for the purpose of characterizing bladder cancer risk. While the NRC’s work did not constitute a formal risk analysis, they did examine many statistical issues (e.g., measurement errors, age-specific probabilities, body weight, water consumption rate, comparison populations, mortality rates, choice of model) and provided a starting point for additional EPA analyses. The report noted that “poor nutrition, low selenium concentrations in Taiwan, genetic and cultural characteristics, and arsenic intake from food” were not accounted for in their analysis (NRC, 1999, p. 295). In the June 22, 2000, proposed rulemaking, EPA calculated bladder cancer risks and benefits using the bladder cancer risk analysis from the 1999 NRC report. We also estimated lung cancer benefits in a “What If” analysis based on the statement in the 1999 NRC report that “some studies have shown that excess lung cancer deaths attributed to arsenic are 2-5 fold greater than the excess bladder cancer deaths” (NRC, 1999).

In July 2000, a peer-reviewed article by Morales et al. (2000) was published, which presented additional analyses of bladder cancer risks as well as estimates of lung and liver cancer risks for the same Taiwanese population analyzed in the NRC report. EPA summarized and analyzed the new information from the Morales et al. (2000) article in a Notice of Data Availability published on October 20, 2000 (65 FR 63027). Although the data used were the same as used by the NRC to analyze bladder cancer risk in their 1999 publication, Morales et al. (2000) considered more dose-response models and evaluated how well they fit the Taiwanese data, for both bladder cancer risk and lung cancer risk. Ten risk models were presented in Morales et al. (2000). After consultation with the primary authors (Morales and Ryan), EPA chose Model 1 with no comparison population for further analysis.

EPA believes that the models in Morales et al. (2000) without a comparison population are more reliable than those with a comparison population. Models with no comparison population estimate the arsenic dose-response curve only from the study population. Models with a comparison population include mortality data from a similar population (in this case either all of Taiwan or part of southwestern Taiwan), whose exposure is assumed to be zero. Most of the models with comparison populations resulted in dose-response curves that were supralinear (higher than a linear dose-response) at low doses. The curves were “forced down” at zero dose because the comparison population consists of a large number of people with low risk and assumed zero exposure. EPA believes, based on discussions with the authors of Morales et al. (2000), that models with a comparison population are less reliable, for two reasons. First, there is no basis in the data on arsenic’s carcinogenic mode of action to support a supralinear curve as being biologically plausible. To the contrary, the conclusion of the NRC Panel (NRC, 1999) was that the mode of action data led one to expect dose responses that would be either linear or less than linear at low dose. However, the NRC indicated that available data are inconclusive and “...do not meet EPA’s 1996 stated criteria for departure from the default assumption of linearity” (NRC, 1999). Second, models that include comparison populations assume that the exposure of the comparison population is zero, and that the study and comparison populations are the same in all important ways except for arsenic exposure. Both of these assumptions may be incorrect: NRC (1999) notes that “the Taiwanese-wide data do not clearly represent a population with zero exposure to arsenic in drinking water”; and Morales et al. (2000) agree that “[t]here is reason to believe that the urban Taiwanese population is not a comparable population for the poor rural population used in this study.” Moreover, because of the large amount of data in the comparison populations, the model results are relatively sensitive to assumptions about this group. For these reasons, EPA believes that the models without comparison populations are more reliable than those with them.

Of the models that did not include a comparison population, EPA believes that Model 1 fits the data best, based on the Akaike Information Criterion (AIC), a standard criterion of model fit, applied to the Poisson models. EPA did not consider the multi-stage Weibull model for additional analysis, because of its greater sensitivity to the omission of individual villages (Morales et al., 2000) and to the grouping of responses by village (NRC, 1999), as occurs in the Taiwanese data. In Model 1, the dose effect is assumed to follow a linear function, and the age effect is assumed to follow a quadratic function. The Agency decided that the more exhaustive statistical analysis of the data provided by Morales et al. (2000), as analyzed by EPA, would be the basis for the new risk calculations for the final rule (with further consideration of additional risk analyses) and other pertinent information.

### ***Estimated Risk Reductions***

Estimated risk reductions for bladder and lung cancer at various MCL levels were developed using Monte-Carlo simulations. Monte-Carlo analysis is a technique for analyzing problems where there are a large number of combinations of input values, which makes it impossible to calculate every possible result. A random number generator is used to select input values from pre-defined distributions. For each set of random numbers, a single scenario’s result is calculated. As the simulation runs, the model is recalculated for each new scenario that continues until a stopping criterion is reached.

These simulations combined the distributions of relative exposure factors (REFs), occurrence at or above 3 µg/L, and risks of bladder and lung cancer taken from the Morales et al. (2000) article. The simulations resulted in upper bound estimates of the actual risks faced by populations exposed to arsenic concentrations at or above 3 µg/L in their drinking water.

### **Lower Bound Analyses**

Two adjustments were made to the risk distributions resulting from the simulations described above, reflecting uncertainty about the actual arsenic exposure in the Taiwan study area. First, the Agency made an adjustment to the lower bound risk estimates to take into consideration the effect of exposure to arsenic through water used in preparing food in Taiwan. The Taiwanese staple foods were dried sweet potatoes and rice (Wu et al., 1989). Both the 1988 EPA *Special Report on Ingested Inorganic Arsenic* and the 1999 NRC report assumed that an average Taiwanese male weighed 55 kg and drank 3.5 liters of water daily, and that an average Taiwanese female weighed 50 kg and drank 2 liters of water daily. Using these assumptions, along with an assumption that Taiwanese men and women ate one cup of dry rice and two pounds of sweet potatoes a day, the Agency re-estimated risks for bladder and lung cancer, using one additional liter of water consumption for food preparation (i.e., the water absorbed by hydration during cooking). This adjustment was discussed and used in the October 20, 2000 NODA (65 FR 63027).

Second, an adjustment was made to the lower bound risk estimates to take into consideration the relatively high arsenic concentration in the food consumed in Taiwan as compared to the U.S. The food consumed daily in Taiwan contains about 50 µg, versus about 10 µg in the U.S. (NRC, 1999, pp. 50–51). Thus, the total consumption of inorganic arsenic (from food preparation and drinking water) is considered, per kilogram of body weight, in the process of these adjustments. To carry them out, the relative contribution of arsenic in the drinking water that was consumed as drinking water, on a µg/kg/day basis, was compared to the total amount of arsenic consumed in drinking water, drinking water used for cooking, and in food, on a µg/kg/day basis.

Other factors contributing to lower bound uncertainty include the possibility of a sub-linear dose-response curve below the point of departure. The NRC noted “Of the several modes of action that are considered most plausible, a sub-linear dose response curve in the low-dose range is predicted, although linearity cannot be ruled out” (NRC, 1999). The recent Utah study (Lewis et al., 1999) provides some evidence that the shape of the dose-response curve may well be sub-linear at low doses. Because sufficient mode of action data were not available, an adjustment was not made to the risk estimates to reflect the possibility of a sub-linear dose-response curve. Additional factors contributing to uncertainty include the use of village well data rather than individual exposure data, deficiencies in the Taiwanese diet relative to the U.S. diet (selenium, choline, etc.), and the baseline health status in the Taiwanese study area relative to U.S. populations. The Agency did not make adjustments to the risk estimates to reflect these uncertainties because applicable peer-reviewed, quantitative studies on which to base such adjustments were not available.

Estimated risk levels for bladder and lung cancer combined at various MCL levels are shown in Exhibit 5-4 (a-c). The risk estimates without adjustments for exposure uncertainty through cooking water and food are shown Exhibit 5-4 (a). These estimates incorporate occurrence data, water consumption data, and male and female risk estimates. Lower bounds show estimates using community water consumption data; upper bounds show estimates using total water consumption data. Exhibit 5-4 (b) shows estimated risk levels for bladder and lung cancer combined at various MCL levels with adjustments for exposure uncertainty through cooking water and food. These estimates incorporate occurrence data, water consumption data, and male risk estimates, with lower bounds reflecting community water consumption data and upper bounds reflecting total water consumption data. There are no adjustments for other factors that contribute to uncertainty, such as the use of village well data as opposed to individual exposure data. Exhibit 5-4 (c) is a combination of Exhibit 5-4 (a) and Exhibit 5-4 (b), with the lower bounds taken from Exhibit 5-4 (b), and the upper bounds taken from Exhibit 5-4 (a). Thus Exhibit 5-4 (c) reflects the range of estimates before and after the exposure uncertainty adjustments for cooking water and for food, along with the incorporation of water consumption data, occurrence data, and cancer risk estimates. These estimates were used to estimate the range of potential cases avoided at the various MCL levels.

The upper bound risk estimates in Exhibits 5-4 (a-c) reflect the following:

- The total water consumption estimates from the EPA water consumption study;
- The occurrence distributions of arsenic in U.S. ground and surface water systems;
- Male and female risk estimates from Morales et al. (2000);
- Not adjusting for arsenic exposure from cooking water in Taiwan; and
- Not adjusting for arsenic exposure from food in Taiwan.

The lower bound risk estimates in Exhibits 5-4 (a-c) reflect the following:

- The community water system estimates of water consumption from the EPA water consumption study;
- The occurrence distributions of arsenic in U.S. ground and surface water systems;
- Male risk estimates from Morales et al. (2000);
- Adjusting for arsenic exposure from cooking water in Taiwan; and
- Adjusting for arsenic exposure from food in Taiwan.

**Exhibit 5-4 (a)**  
**Cancer Risks for U.S. Populations**  
**Exposed at or Above MCL Options, After Treatment<sup>1,2</sup>**  
**(Without Adjustment for Arsenic in Food and Cooking Water)**

<b>MCL (µg/L)</b>	<b>Mean Exposed Population Risk</b>	<b>90<sup>th</sup> Percentile Exposed Population Risk</b>
3	.93 - 1.25 x 10 <sup>-4</sup>	1.95 - 2.42 x 10 <sup>-4</sup>
5	1.63 - 2.02 x 10 <sup>-4</sup>	3.47 - 3.9 x 10 <sup>-4</sup>
10	2.41 - 2.99 x 10 <sup>-4</sup>	5.23 - 6.09 x 10 <sup>-4</sup>
20	3.07 - 3.85 x 10 <sup>-4</sup>	6.58 - 8.37 x 10 <sup>-4</sup>

<sup>1</sup>Actual risks could be lower, given the various uncertainties discussed, or higher, as these estimates assume that the probability of illness from arsenic exposure in the U.S. is equal to the probability of death from arsenic exposure among the arsenic study group.

<sup>2</sup>The estimated risks are male and female risks combined.

**Exhibit 5-4 (b)**  
**Cancer Risks for U.S. Populations**  
**Exposed at or Above MCL Options, After Treatment<sup>1,2</sup>**  
**(With Adjustment for Arsenic Exposure in Food and Cooking Water)**

<b>MCL (µg/L)</b>	<b>Mean Exposed Population Risk</b>	<b>90<sup>th</sup> Percentile Exposed Population Risk</b>
3	.11 - .13 x 10 <sup>-4</sup>	.22 - .26 x 10 <sup>-4</sup>
5	.27 - .32 x 10 <sup>-4</sup>	.55 - .62 x 10 <sup>-4</sup>
10	.63 - .76 x 10 <sup>-4</sup>	1.32 - 1.54 x 10 <sup>-4</sup>
20	1.1 - 1.35 x 10 <sup>-4</sup>	2.47 - 2.89 x 10 <sup>-4</sup>

<sup>1</sup>Actual risks could be lower, given the various uncertainties discussed, or higher, as these estimates assume that the probability of illness from arsenic exposure in the U.S. is equal to the probability of death from arsenic exposure among the arsenic study group.

<sup>2</sup>The estimated risks are for males.

**Exhibit 5-4 (c)**  
**Cancer Risks for U.S. Populations**  
**Exposed at or Above MCL Options, After Treatment<sup>1</sup>**  
**(Lower Bound With Food and Cooking Water Adjustment,  
Upper Bound Without Food and Cooking Water Adjustment)**

<b>MCL (µg/L)</b>	<b>Mean Exposed Population Risk</b>	<b>90<sup>th</sup> Percentile Exposed Population Risk</b>
3	.11 - 1.25 x 10 <sup>-4</sup>	.22 - 2.42 x 10 <sup>-4</sup>
5	.27 - 2.02 x 10 <sup>-4</sup>	.55 - 3.9 x 10 <sup>-4</sup>
10	.63 - 2.99 x 10 <sup>-4</sup>	1.32 - 6.09 x 10 <sup>-4</sup>
20	1.1 - 3.85 x 10 <sup>-4</sup>	2.47 - 8.37 x 10 <sup>-4</sup>

<sup>1</sup>Actual risks could be lower, given the various uncertainties discussed, or higher, as these estimates assume that the probability of illness from arsenic exposure in the U.S. is equal to the probability of death from arsenic exposure among the arsenic study group.

### 5.3.3 Non-Transient Non-Community Water Systems

#### ***Determination of System and Individual Exposure Factors***

In the past, the Agency has directly used SDWIS population estimates for assessing the risks posed to users of NTNC water systems. In other words, it was assumed that the same person received the exposure on a year-round basis. Under this approach it was generally assumed that all NTNC users were exposed for 270 days out of the year and obtained 50 percent of their daily consumption from these systems. As a comparison, TNC users are assumed to use the system for only ten days per year.

With the recent completion of *Geometries and Characteristics of Public Water Systems* (EPA, 1999a), however, the Agency has developed a more comprehensive understanding of NTNC water systems. These systems provide water in due course as part of operating another line of business. Many systems are classified as NTNC, rather than TNC, water systems solely because they employ sufficient workers to trigger the “25 persons served for over six months out of the year” requirement. Client utilization of these systems is actually much less and more similar to exposure in TNC water systems. For instance, it is fairly implausible that highway rest areas along interstate highways serve the same population on a consistent basis (with the exception of long distance truckers). Nevertheless, there are highway rest areas in both NTNC and TNC system inventories. The *Geometries and Characteristics of Public Water Systems* report suggests that population figures reported in SDWIS that have been used for past risk assessments generally appear to reflect the number of workers in the establishment coupled with peak day customer utilization.

Under these conditions, use of the SDWIS figures for population greatly overestimates the actual individual exposure risk for most of the exposed population and also severely underestimates the number of people exposed to NTNC water.<sup>1</sup> Adequately characterizing individual and population risks necessitates some adjustment of the SDWIS population figures. For chronic contaminants, such as arsenic, health data reflect the consequences of a lifetime of exposure. Consequently, risk assessment requires the estimation of the portion of total lifetime drinking water consumption that any one individual would receive from a particular type of water system. In turn, one needs to estimate the appropriate portions for daily, days per year, and year per lifetime consumption. These estimates need to be prepared for both the workers at the facility and the “customers” of the facility.

This adjustment was accomplished through a comprehensive review of government and trade association statistics on entity utilization by Standard Industry Classification (SIC) code.

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<sup>1</sup>For example, airports constitute only about a hundred of the NTNC water systems. Washington’s Reagan National and Dulles, Dallas/Fort Worth, Seattle/Tacoma, and Pittsburgh airports are the five largest of the airports. SDWIS reports that these five airports serve about 300,000 people. In actuality, the Bureau of Transportation Statistics (BTS) suggest that they serve about eleven million passengers per year. Examination of this information and other BTS statistics suggests that these airports serve closer to seven million unique individuals over the course of a year and that exposure occurs on an average of ten times per year per individual customer, not 270 times.

These figures, coupled with SDWIS information relating to the portion of a particular industry served by non-community water systems, made possible the development of two estimates needed for the risk assessment: customer cycles per year and worker per population served per day. These numbers are required to distinguish the more frequent and longer duration exposure of workers from that of system customers.<sup>2</sup> A more detailed characterization of the derivation of these numbers is contained in the docket. Exhibit 5-5 provides the factors used in the NTNC risk assessment to account for the intermittent nature of exposure.

**Exhibit 5-5  
Exposure Factors Used in the NTNC Risk Assessment**

NTNCWS	# cycles per yr	worker/pop/day	worker fraction daily	worker days/yr	worker exposure years	customer fraction daily	days of use/yr	customer exposure years
Water wholesalers	1.00	0.000	-	-	-	0.25	270	70
Nursing homes	1.00	0.230	0.50	250	40	1.00	365	10
Churches	1.00	0.010	0.50	250	40	0.50	52	70
Golf/country clubs	4.50	0.110	0.50	250	40	0.50	52	70
Food retailers	2.00	0.070	0.50	250	40	0.25	185	70
Non-food retailers	4.50	0.090	0.50	250	40	0.25	52	70
Restaurants	2.00	0.070	0.50	250	40	0.25	185	70

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<sup>2</sup>For example, travel industry statistics provide information on total numbers of hotel stays, vacancy rates, traveler age ranges, and average duration of stay. These figures can be combined with the SDWIS peak day population estimates to allocate daily population among workers, customers, and vacancies. The combination of these factors provides an estimate of the number of independent customer cycles experienced in a year.

**Exhibit 5-5  
Exposure Factors Used in the NTNC Risk Assessment (continued)**

NTNCWS	# cycles per yr	worker/pop/day	worker fraction daily	worker days/yr	worker exposure years	customer fraction daily	days of use/yr	customer exposure years
Hotels/motels	86.00	0.270	0.50	250	40	1.00	3.4	40
Prisons/jails	1.33	0.100	0.50	250	40	1.00	270	3
Service stations	7.00	0.060	0.50	250	40	0.25	52	54
Agricultural products/services	7.00	0.125	0.50	250	40	0.25	52	50
Daycare centers	1.00	0.145	0.50	250	10	0.50	250	5
Schools	1.00	0.073	0.50	200	40	0.50	200	12
State parks	26.00	0.016	0.50	250	40	0.50	14	70
Medical facilities	16.40	0.022	0.50	250	40	1.00	6.7	10.3
Campgrounds/RV	22.50	0.041	0.50	180	40	1.00	5	50
Federal parks	26.00	0.016	0.50	250	40	0.50	14	70
Highway rest areas	50.70	0.010	0.50	250	40	0.50	7.2	70
Misc. recreation service	26.00	0.016	0.50	250	40	1.00	14	70
Forest Service	26.00	0.016	1.00	250	40	1.00	14	50
Interstate carriers	93.00	0.304	0.50	250	40	0.50	2	70
Amusement parks	90.00	0.180	0.50	250	10	0.50	1	70
Summer camps	8.50	0.100	1.00	180	10	1.00	7	10
Airports	36.50	0.308	0.50	250	40	0.25	10	70
Military bases		1.000	0.50	250	40			
Non-water utilities		1.000	0.50	250	40			
Office parks		1.000	0.50	250	40			
Manufacturing: Food		1.000	0.50	250	40			
Manufacturing: Non-food		1.000	0.50	250	40			
Landfills		1.000	1.00	250	40			
Fire departments		1.000	1.00	250	40			
Construction		1.000	1.00	250	40			
Mining		1.000	1.00	250	40			
Migrant labor camps		1.000	1.00	250	40			

Once the population adjustment factors were derived, it was possible to determine the actual population served by NTNC water systems. Exhibit 5-6 provides a breakout of these figures by type of establishment.

Although not included in Exhibit 5-6, there are other equally important characteristics to note about these systems. With notable exceptions (such as the airports in Washington, DC, and Seattle), the systems generally serve a fairly small population on any given day. In fact, 99 percent of the systems serve fewer than 3,300 users on a daily basis. This means that water production costs will be relatively high on a per gallon basis.

**Exhibit 5-6**  
**Composition of NTNCs**  
**(Percentage of Total NTNCWS Population Served by Sector)**

Schools	9.7	Medical Facilities	8	Interstate Carriers	7.1	Campgrounds	1.3
Manufacturing	2.7	Restaurants	0.9	State Parks	8.6	Misc. Recreation	1.8
Airports	26.1	Non-food Retail	1.6	Amusement Parks	17.7	Other	3.5
Office Parks	0.6	Hotels/Motels	9.2	Highway Rest Area	1.0		

### ***Risk Calculation***

Calculations of individual combined risk for bladder and lung cancer were prepared for each industrial sector. Even within a given sector, however, risk varies as a function of an individual's relative water consumption, body weight, vulnerability to arsenic exposure, and the water arsenic concentration. Computationally, risks were estimated by performing Monte-Carlo modeling. The approach used was similar to the modeling technique applied in estimating the community water system risk estimation, but with two notable exceptions. First, each realization in a given sector was multiplied by the portion of lifetime exposure factor presented in Exhibit 5-6 to reflect the decreased consumption associated with the NTNC system. Second, relative exposure factors were limited to age-specific ratings where appropriate.<sup>3</sup> For example, in the case of school children, water consumption rates and weights for 6- to 18-year-olds were used.

To illustrate the process, it was assumed that a child would attend only NTNCWS-served schools for all twelve years, a somewhat improbable likelihood. Further, it was assumed that a child would get half of his or her daily water consumption at school (for an average first grader this would correspond to roughly nine ounces of water per school day). Finally, it was assumed that the child would have perfect attendance and attend school for 200 days per year.

The distribution of overall population risks was determined as part of the same simulation by developing sector weightings to reflect the total portion of the NTNCWS population served by each sector. Population weighted proportional sampling of the individual sectors provided an overall distribution of risk among those exposed at NTNC systems.

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<sup>3</sup>For example, water consumption among school children was weighted to reflect consumption between ages 6 and 18, while factory worker consumption was weighted over ages 20 to 64.

Exhibit 5-7 presents a summary of the risk analyses for regulation of arsenic in NTNC water systems. Exhibit 5-8 presents risk figures for three particular sets of individuals: children in daycare centers and schools, and construction workers. Construction and other strenuous activity workers comprise an extremely small portion of the population served by NTNC systems (less than 0.1 percent), but face the highest relative risks of all NTNCWS users (90<sup>th</sup> percentile risks of 0.4 to 2.3 x 10<sup>-4</sup> lifetime risk).

**Exhibit 5-7  
Mean Cancer Risks (Bladder and Lung combined),  
Exposed Population, and Annual Cancer Benefits in NTNCs**

Arsenic Level (µg/L)	Mean Exposed Population Risk (10 <sup>-4</sup> )		Total Bladder and Lung Cancer Cases Avoided per Year	
	lower bound	upper bound	lower bound	upper bound
3	0.0000657	0.000952	0.6	2.25
5	0.000162	0.00157	0.53	1.78
10	0.000374	0.00243	0.36	1.13
20	0.00064	0.00322	0.16	0.53
baseline	0.000853	0.00391	0.65	2.98

**Exhibit 5-8  
Sensitive Group Evaluation of Lifetime Combined Cancer Risks**

Group	Mean Risk	90 <sup>th</sup> Percentile Risk
Forest Service, Construction and Mining Workers	0.2 - 1.2 x 10 <sup>-4</sup>	0.4 - 2.3 x 10 <sup>-4</sup>
School Children	0.2 - 1.4 x 10 <sup>-5</sup>	0.5 - 2.8 x 10 <sup>-5</sup>
Day Care Children	1.1 - 7.3 x 10 <sup>-6</sup>	0.25 - 1.5 x 10 <sup>-5</sup>

However, there is considerable uncertainty about these exposure numbers, as it is quite likely that they overestimate consumption. It is not possible to determine from the analysis of NTNC systems the extent to which there is overlap of individual exposure between the various sectors. NTNC establishments generally constitute a small portion of their SIC sectors. In conjunction with the observation that NTNC populations would only serve about 11 percent of the total population if all sectors were mutually exclusive, it would seem reasonable to treat the SIC groups independently. However, it is equally plausible that there are communities where one individual might go from an NTNC day care center to a series of NTNC schools and then work in an NTNC factory. Unfortunately, the Agency presently has no basis for quantitatively estimating the extent to which this would occur.

## 5.4 Risk Assessment Results and Benefit Estimates

### 5.4.1 Cases Avoided

The lower and upper bound risk estimates from Exhibit 5-4 (c) were applied to the exposed population to generate cases avoided for CWS systems serving fewer than one million customers. Because the actual arsenic occurrence was known for the very large systems (those serving over a million customers), their system-specific arsenic occurrence distributions could be directly computed. The system specific arsenic distributions allowed direct calculation of avoided cancer cases. The process, described in detail in Appendix B, utilizes the same risk estimates from Morales et al. (2000) that were used in deriving the number of cases avoided in smaller CWS systems. Cases avoided for NTNC systems were also computed separately, utilizing factors developed to account for the intermittent nature of the exposure.

An upper bound adjustment was made to the number of bladder cancer cases avoided to reflect a possible lower mortality rate in Taiwan than was assumed in the risk assessment process described earlier. We also made this adjustment in the June 22, 2000, proposal. In the Taiwan study area, information on arsenic related bladder and lung cancer deaths was reported. In order to use these data to determine the probability of contracting bladder and lung cancer as a result of exposure to arsenic, a probability of mortality given the onset of arsenic induced bladder and lung cancer among the Taiwanese study population must be assumed. The study area in Taiwan is a section where arsenic concentrations in the water are very high by comparison to those in the U.S., and is an area of low incomes and poor diets, where the availability and quality of medical care is not of high quality by U.S. standards. In its estimate of bladder cancer risk, the Agency assumed that within the Taiwanese study area, the probability of contracting bladder cancer was relatively close to the probability of dying from bladder cancer (that is, that the bladder cancer incidence rate was equal to the bladder cancer mortality rate).

We do not have data on the rates of survival for bladder cancer in the Taiwanese villages in the study and at the time of data collection. We do know that the relative survival rates for bladder cancer in developing countries overall ranged from 23.5 percent to 66.1 percent in 1982-1992 (*Cancer Survival in Developing Countries*, International Agency for Research on Cancer, World Health Organization, Publication No. 145, 1998). We also have some information on annual bladder cancer mortality and incidence for the general population of Taiwan in 1996. The age-adjusted annual incidence rates of bladder cancer for males and females, respectively, were 7.36 and 3.09 per 100,000, with corresponding annual mortality rates of 3.21 and 1.44 per 100,000 (correspondence from Chen to Herman Gibb, January 3, 2000). Assuming that the proportion of males and females in the population is equal, these numbers imply that the mortality rate for bladder cancer in the general population of Taiwan, at present, is 45 percent. Since survival rates have most likely improved over the years since the original Taiwanese study, this number represents a lower bound on the survival rate for the original area under study (that is, one would not expect a higher rate of survival in that area at that time). This has implications for the bladder cancer risk estimates from the Taiwan data. If there were any persons with bladder cancer who recovered and died from some other cause, then our estimate underestimated risk; that is, there were more cancer cases than cancer deaths. Based on the above discussion, we think bladder cancer incidence could be no more than two times bladder cancer mortality; and that an 80 percent mortality rate would be plausible.

Thus, we have adjusted the upper bound of cases avoided, which is used in the benefits analysis, to reflect a possible mortality rate for bladder cancer of 80 percent. Because lung cancer mortality rates are quite high, about 88 percent in the U.S. (EPA, 1998b), the assumption was made that all lung cancers in the Taiwan study area resulted in fatalities.

The number of bladder, lung, and combined bladder and lung cases avoided at each MCL are shown in Exhibits 5-9 (a), 5-9 (b), and 5-9 (c). These cases avoided include both CWS and NTNC cases. The number of bladder cancer cases avoided range from 28.6 to 76.8 at an MCL of 3 µg/L, 25.6 to 55.7 at an MCL of 5 µg/L, 18.7 to 31.0 at an MCL of 10 µg/L, and 9.9 to 10.6 at an MCL of 20 µg/L. The number of lung cancer cases avoided range from 28.6 to 61.5 at an MCL of 3 µg/L, 25.6 to 44.5 at an MCL of 5 µg/L, 18.7 to 24.8 at an MCL of 10 µg/L, and 8.5 to 9.9 at an MCL of 20 µg/L. The number of combined bladder and lung cancer cases avoided range from 57.2 to 138.3 at an MCL of 3 µg/L, 51.1 to 100.2 at an MCL of 5 µg/L, 37.4 to 55.7 at an MCL of 10 µg/L, and 19.0 to 19.8 at an MCL of 20 µg/L.

The cases avoided were divided into premature fatality and morbidity cases based on U.S. mortality rates. In the U.S. approximately one out of four individuals who is diagnosed with bladder cancer actually dies from bladder cancer. The mortality rate for the U.S. is taken from a cost of illness study recently completed by EPA (EPA, 1998b). For those diagnosed with bladder cancer at the average age of diagnosis (70 years), the probabilities of dying of that disease during each year post-diagnosis were summed over a 20-year period to obtain the value of 26 percent. Mortality rates for U.S. bladder cancer patients have decreased overall by 24 percent from 1973 to 1996. For lung cancer, mortality rates are much higher. The comparable mortality rate for lung cancer in the U.S. is 88 percent.

**Exhibit 5-9 (a)**  
**Annual Bladder Cancer Cases Avoided**  
**from Reducing Arsenic in CWSs and NTNCs**

<b>Arsenic Level (µg/L)</b>	<b>Reduced Mortality Cases*</b>	<b>Reduced Morbidity Cases*</b>	<b>Total Cancer Cases Avoided*</b>
3	7.4 - 20.0	21.2 - 56.9	28.6 - 76.8
5	6.6 - 14.5	18.9 - 41.2	25.6 - 55.7
10	4.9 - 8.0	13.8 - 22.7	18.7 - 31.0
20	2.6 - 2.8	7.3 - 7.8	9.9 - 10.6

\* The lower-end estimate of bladder cancer cases avoided is calculated using the lower-end risk estimate from Exhibit 5-9(c) and assumes that the conditional probability of mortality among the Taiwanese study group was 100 percent. The upper-end estimate of bladder cancer cases avoided is calculated using the upper-end risk estimate from Exhibit 5-9(c) and assumes that the conditional probability of mortality among the Taiwanese study group was 80 percent.

**Exhibit 5-9 (b)**  
**Annual Lung Cancer Cases Avoided**  
**from Reducing Arsenic in CWSs and NTNCs**

<b>Arsenic Level (µg/L)</b>	<b>Reduced Mortality Cases*</b>	<b>Reduced Morbidity Cases*</b>	<b>Total Cancer Cases Avoided*</b>
3	25.2 - 54.1	3.4 - 7.4	28.6 - 61.5
5	22.5 - 39.2	3.1 - 5.3	25.6 - 44.5
10	16.4 - 21.8	2.2 - 3.0	18.7 - 24.8
20	7.4 - 8.7**	1.0 - 1.2**	8.5 - 9.9**

\* The lower and upper-end estimates of lung cancer cases avoided are calculated using the risk estimates from Exhibit 5-9 (c) and assume that the conditional probability of mortality among the Taiwanese study group was 100 percent.

\*\*For 20 µg/L, the proportional reduction from the lower level risk base case is greater than the proportional reduction from the higher level risk base case. Thus, the number of estimated cases avoided is higher at 20 µg/L using the estimates adjusted for uncertainty.

**Exhibit 5-9 (c)**  
**Annual Total Cancer Cases Avoided**  
**from Reducing Arsenic in CWSs and NTNCs**

<b>Arsenic Level (µg/L)</b>	<b>Reduced Mortality Cases*</b>	<b>Reduced Morbidity Cases*</b>	<b>Total Cancer Cases Avoided*</b>
3	32.6 - 74.1	24.6 - 64.2	57.2 - 138.3
5	29.1 - 53.7	22.0 - 46.5	51.1 - 100.2
10	21.3 - 29.8	16.1 - 25.9	37.4 - 55.7
20	10.2 - 11.3**	8.5 - 8.8	19.0 - 19.8**

\* The lower-end estimate of bladder cancer cases avoided and the lung cancer estimates assume that the conditional probability of mortality among the Taiwanese study group was 100 percent. The upper-end estimate of bladder cancer cases avoided is calculated using the assumption that the conditional probability of mortality among the Taiwanese study group was 80 percent.

\*\*For 20 µg/L, the proportional reduction from the lower level risk base case is greater than the proportional reduction from the higher level risk base case. Thus the number of estimated cases avoided is higher at 20 µg/L using the estimates adjusted for uncertainty.

#### **5.4.2 Economic Measurements of the Value of Risk Reduction**

The evaluation stage in the analysis of risk reductions involves estimating the value of reducing the risks. The following sections describe the use of benefits valuation techniques to estimate the value of the risk reductions attributable to the regulatory options for arsenic in drinking water. First, the approach for valuing the reductions in fatal risks is described, followed by a description of the approach for valuing the reductions in non-fatal risks.

The benefits described in the primary analysis of this Economic Analysis are assumed to begin to accrue on the effective date of the rule and are based on a calculation referred to as the “value of a statistical life” (VSL).

Of the many VSL studies, the Agency recommends using estimates from 26 specific studies that have been peer reviewed and extensively reviewed within the Agency.<sup>4</sup> These estimates, which are derived from wage-risk and contingent valuation studies, range from \$0.7 million to \$16.3 million and approximate a Weibull distribution with a mean of \$4.8 million (in 1990 dollars). Most of these 26 studies examine willingness to pay in the context of voluntary acceptance of higher risks of immediate accidental death in the workplace in exchange for higher wages. This value is sensitive to differences in population characteristics and perception of risks being valued. This value could also be updated to include changes in income from 1990 to 1999, which reflects the difference between the study population and the affected population, and would increase monetary benefits since income growth in that time period has been positive.

EPA updated the VSL estimate from *The Benefits and Costs of the Clean Air Act, 1970 to 1990* report to a value of \$5.8 million in 1997 dollars, according to internal guidance on economic analyses (Bennett, 2000). In order to directly compare the estimated national costs of compliance, the VSL used in this analysis was updated from the January 1997 value to \$6.1 million in May 1999 dollars, using the Consumer Price Index (CPI-U) for all items.

Several factors may influence the estimate of economic benefits associated with avoided cancer fatalities, including:

1. A possible “cancer premium” (i.e., the additional value or sum that people may be willing to pay to avoid the experiences of dread, pain and suffering, and diminished quality of life associated with cancer-related illness and ultimate fatality);
2. The willingness of people to pay more over time to avoid mortality risk as their income rises;
3. A possible premium for accepting involuntary risks as opposed to voluntary assumed risks;
4. The greater risk aversion of the general population compared to the workers in the wage-risk valuation studies;
5. “Altruism” or the willingness of people to pay more to reduce risk in other sectors of the population; and
6. A consideration of health status and life years remaining at the time of premature mortality.

Use of certain of these factors may significantly increase the present value estimate. EPA therefore believes that adjustments should be considered simultaneously. The Agency also believes that there is currently neither a clear consensus among economists about how to simultaneously analyze each of these adjustments, nor are there adequate empirical data to support definitive quantitative estimates for all potentially significant adjustment factors. As a result, the primary estimates of economic benefits presented in the analysis of this rule rely on the unadjusted estimate.

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<sup>4</sup> U.S. Environmental Protection Agency, *The Benefits and Costs of the Clean Air Act, 1970 to 1990*, October 1997, Appendix I; and U.S. Environmental Protection Agency, *Guidelines for Preparing Economic Analysis (Review Draft)*, June 1999, Chapter 7.

To assess the impacts of these other factors, EPA presents a sensitivity analysis that examines the impacts of changes in assumptions of the latency period and incorporation of income growth, etc. This sensitivity analysis is given in Section 5.5.

To estimate the monetary value of reduced fatal risks (i.e., risks of premature death from cancer) predicted under different regulatory options, VSL estimates are multiplied by the number of premature fatalities avoided. VSL does not refer to the value of an identifiable life, but instead to the value of small reductions in mortality risks in a population. A “statistical” life is thus the sum of small individual risk reductions across an entire exposed population. For example, if 100,000 people would each experience a reduction of 1/100,000 in their risk of premature death as the result of a regulation, the regulation can be said to “save” one statistical life (i.e.,  $100,000 \times 1/100,000$ ). If each member of the population of 100,000 were willing to pay \$20 for the stated risk reduction, the corresponding value of a statistical life would be \$2 million (i.e.,  $\$20 \times 100,000$ ). VSL estimates are appropriate only for valuing small changes in risk; they are not values for saving a particular individual’s life.

Estimates of the willingness to pay to avoid treatable, non-fatal cancers are the ideal economic measures used to value reductions in nonfatal risks. Unfortunately, this information is not available for bladder or lung cancer. However, willingness to pay (WTP) data to avoid chronic bronchitis is available and has previously been employed by the Office of Ground Water and Drinking Water (the microbial/disinfection by-product [MDBP] rulemaking) as a surrogate to estimate the WTP to avoid non-fatal bladder cancer. A WTP central tendency estimate of \$607,162 (in May 1999 dollars) is used to monetize the benefits of avoiding non-fatal cancers (this value was updated from the \$536,000 value EPA updated to 1997 dollars from the Viscusi et al. [1991] study).

To ground-truth the use of the chronic bronchitis WTP value as a proxy for WTP for the avoidance of non-fatal cases of bladder cancer, EPA has also developed cost-of-illness estimates for bladder cancer, as reported in Exhibit 5-10. These estimates of direct medical costs are derived from a study conducted by Baker et al. (1989), which uses data from a sample of Medicare records for 1974-1981. These data include the total charges for inpatient hospital stays, skilled nursing facility stays, home health agency charges, physician services, and other outpatient and medical services. EPA combined these data with estimates of survival rates and treatment time periods to determine the average costs of initial treatment and maintenance care for patients who do not die of the disease. This value of \$178,405, at a three percent discount rate, serves as a low-end estimate of the WTP to avoid bladder cancer and does not include the value of avoided pain and suffering, lost productivity, or risk premium.

**Exhibit 5-10**  
**Lifetime Avoided Medical Costs for Survivors**  
**(preliminary estimates<sup>1</sup>)**

<b>Type of Cancer</b>	<b>Date Data Collected</b>	<b>Number of Cases Studied</b>	<b>Estimated Mortality Rate</b>	<b>Mean Value per Non-fatal Case (Discount Rate)<sup>1</sup></b>
Bladder	1974-1981	5% of 1974 Medicare patients (sample from national statistics)	26% (after 20 years)	\$178,405 (3%) \$147,775 (7%) (for typical individual diagnosed at age 70)

Source: U.S. Environmental Protection Agency, *Cost of Illness Handbook (draft)*, September 1998.

<sup>1</sup> May 1999 dollars.

### 5.4.3 Estimates of Cancer Health Benefits of Arsenic Reduction

Benefits estimates were calculated based on the number of bladder and cancer cases avoided, as given in Exhibits 5-9 (a-c). The total cases avoided were divided into fatal and non-fatal cases, based on survival information (EPA, 1998b). The avoided premature fatalities were valued based on the VSL estimates discussed earlier, as recommended by current EPA guidance for cost/benefit analysis (EPA, 2000c). The avoided non-fatal cases were valued based on the willingness to pay estimates for the avoidance of chronic bronchitis.

The results of the benefits valuation are presented in Exhibit 5-11. Total annual health benefits resulting from bladder cancer cases avoided range from \$58.2 to \$156.4 million at an MCL of 3 µg/L, \$52.0 to \$113.3 million at an MCL of 5 µg/L, \$38.0 to \$63.0 million at an MCL of 10 µg/L, and \$20.1 to \$21.5 million at an MCL of 20 µg/L. Total annual health benefits from avoided cases of lung cancer range from \$155.6 to \$334.5 million at an MCL of 3 µg/L, \$139.1 to \$242.3 million at an MCL of 5 µg/L, \$101.6 to \$134.7 million at an MCL of 10 µg/L, and \$46.1 to \$53.8 million at an MCL of 20 µg/L. In addition, other potential non-quantifiable health benefits are summarized in Exhibit 5-11.

**Exhibit 5-11**  
**Total Annual Cost, Estimated Monetized Total Cancer Health Benefits and**  
**Non-Quantifiable Health Benefits from Reducing Arsenic in PWSs**  
**(\$ millions)**

Arsenic Level (µg/L)	Total Annual Cost (7%)	Annual Bladder Cancer Health Benefits <sup>1,2</sup>	Annual Lung Cancer Health Benefits <sup>1,2</sup>	Total Annual Health Benefits <sup>1,2</sup>	Potential Non-Quantifiable Health Benefits
3	\$792.1	\$58.2 - \$156.4	\$155.6 - \$334.5	\$213.8 - \$490.9	<ul style="list-style-type: none"> <li>• Skin Cancer</li> <li>• Kidney Cancer</li> <li>• Cancer of the Nasal Passages</li> <li>• Liver Cancer</li> <li>• Prostate Cancer</li> <li>• Cardiovascular Effects</li> <li>• Pulmonary Effects</li> <li>• Immunological Effects</li> <li>• Neurological Effects</li> <li>• Endocrine Effects</li> <li>• Reproductive and Developmental Effects</li> </ul>
5	\$471.7	\$52.0 - \$113.3	\$139.1 - \$242.3	\$191.1 - \$355.6	
10	\$205.6	\$38.0 - \$63.0	\$101.6 - \$134.7	\$139.6 - \$197.7	
20	\$76.5	\$20.1 - \$21.5	\$46.1 - \$53.8	\$66.2 - \$75.3 <sup>3</sup>	

<sup>1</sup> May 1999 dollars.

<sup>2</sup> These monetary estimates are based on cases avoided given in Exhibit 5-9 (a-c).

<sup>3</sup> For 20 µg/L, the proportional reduction from the lower level risk base case is greater than the proportional reduction from the higher level risk base case. Thus, the number of estimated cases avoided and estimated benefits are higher at 20 µg/L using the estimates adjusted for uncertainty.

## 5.5 Latency and Other Adjustments: A Sensitivity Analysis

For the final rulemaking analysis, some commenters have argued that the Agency should consider an assumed time lag or latency period in its benefits calculations. The term “latency” can be used in different ways, depending on the context. For example, health scientists tend to define latency as the period beginning with the initial exposure to the carcinogen and ending when the cancer is initially manifested (or diagnosed), while others consider latency as the period between manifestation of the cancer and death. Latency, in this case, refers to the difference between the time of initial exposure to environmental carcinogens and the actual mortality. Use of such an approach might reduce significantly the present value of health risk reduction benefits estimates.

In the Arsenic Rule, the Agency included qualitative language on the latency issue, including descriptions of other adjustments that may influence the estimate of economic benefits associated with avoided cancer fatalities. The Agency also agreed to ask the Science Advisory Board (SAB) to conduct a review of the benefits transfer issues and possible adjustment factors associated with economic valuation of mortality risks. A summary of the SAB’s recommendations is shown in the following section.

### 5.5.1 SAB Recommendations

EPA brought this issue before the Environmental Economics Advisory Committee (EEAC) of EPA’s SAB in a meeting held on February 25, 2000, in Washington, DC. The SAB submitted a final report on their findings and recommendations to EPA on July 27, 2000.

The EEAC report made a number of recommendations on the adjustment factors and benefit-cost analysis in general. A copy of the final SAB report has been placed in the record for this rulemaking.

The SAB EEAC noted that benefit-cost analysis, as described in the Agency's Guidelines (for economic analysis), is not the only analytical tool, nor is efficiency the only appropriate criterion for social decision making, but notes that it is important to carry out such analyses in an unbiased manner with as much precision as possible. In its report, the SAB recommended that the Agency continue to use a wage-risk based VSL as its primary estimate; any appropriate adjustments that are made for timing and income growth should be part of the Agency's main analysis, while any other adjustments should be accounted for in sensitivity analyses to show how results would change if the VSL were adjusted for some of the major differences in the characteristics of the risk and of the affected populations.

Specifically, the SAB report recommended that (1) health benefits brought about by current policy initiatives (i.e., after a latency period) should be discounted to present value using the same rate that is used to discount other future benefits and costs in the *primary* analysis; (2) adjustments to the VSL for a "cancer premium" should be made as part of a *sensitivity* analysis; (3) adjustments to the VSL for voluntariness and controllability should be made as part of a *sensitivity* analysis; (4) altruism should be addressed in a *sensitivity* analysis and separately from estimation of the value of a statistical cancer fatality, and the circumstances under which altruism can be included in a benefit-cost analysis are restrictive; (5) estimates of VSLs accruing in future years should be adjusted in the *primary* analysis to reflect anticipated income growth, using a range of income elasticities; (6) adjustments to the VSL for risk aversion should be made in a *sensitivity* analysis; (7) it is *theoretically* appropriate to calculate WTP for individuals whose ages correspond to those of the affected population, but more research should be conducted in this area; and (8) no adjustment should be made to the VSL to reflect health status of persons whose cancer risks are reduced.

After considering the SAB's recommendations, EPA has developed a sensitivity analysis of the latency structure and associated benefits for the Arsenic Rule, as described in the next section. This analysis consists of health risk reduction benefits that reflect adjustments for discounting, incorporation of a range of latency period assumptions, adjustments for growth in income, and incorporation of other factors such as voluntariness and controllability. Although the SAB recommended accounting for latency in a primary benefits analysis, the Agency believes that in the absence of any sound scientific evidence on the duration of particular latency periods for arsenic-related cancers, discounted benefits estimates for arsenic are more appropriately accounted for in a sensitivity analysis. Sensitivity analyses are generally reserved for examining the effects of accounting for highly uncertain factors, such as the estimation of latency periods, on health risk reduction benefits estimates.

Defining a latency period is highly uncertain because the length of the latency period is often poorly understood by health scientists. In some cases, information on the progression of a cancer is based on animal studies, and extrapolation to humans is complex and uncertain. Even when human studies are available, the dose considered may differ significantly from the dose generally associated with drinking water contaminants (e.g., involve a high level of exposure over a short time period, rather than a long-term, low level of exposure).

The magnitude of the dose may in turn affect the resulting latency period. Information on latency may be unavailable in many cases, or, if available, may be highly uncertain and vary significantly across individuals.

### 5.5.2 Analytical Approach

For the latency sensitivity analysis, the health benefits have been broken into separate treatments of morbidity and mortality. The mortality component of the total benefits is examined in this analysis because a cancer latency period (i.e., the time period between initial exposure to environmental carcinogens and the actual fatality) impacts arsenic-related fatalities only. For purposes of this analysis, the Agency examined the impacts of various latency period assumptions, adjustments for income growth, and incorporation of other adjustments, such as voluntariness and controllability, on bladder and lung cancer fatalities associated with arsenic in drinking water.

Because the latency period for arsenic-related bladder and lung cancers is unknown, EPA has assumed a range of latency periods from 5 to 20 years. While both lung and bladder cancer have relatively long average latencies, the lower end of the latency period is substantially less. As can be seen by inspection of the Surveillance, Epidemiology, and End Results (SEER) data of the National Cancer Institute, significant incidence of both cancers occurs in individuals in the 15- to 19-year-old age groups. This strongly indicates a short latency period for whatever the cause of the cancer may have been.

Moreover, the mode of action for arsenic is suspected to be one that operates at a late stage of the cancer process and that may advance the expression of cancers initiated by other causes (sometimes referred to as “promoting out” the cancerous effect). Therapeutic treatment with the drug cyclophosphamide, which causes cell toxicity, has been seen to induce bladder cancer in as little as 7 to 15 years in affected patients. This was of course a high dose treatment, but the example serves to illustrate the ability of an agent to advance the development of cancer.

For these reasons, we believe latency periods of 5, 10, and 20 years serve as reasonable approximations, in the absence of definitive data on arsenic-induced cancers, of the latency periods for the sensitivity analysis.

Exhibit 5-12 shows the sensitivity of the primary analysis VSL estimate (\$6.1 million, 1999 dollars) to changes in latency period assumptions and also with the incorporation of income growth and other adjustment factors. As is shown in Exhibit 5-12, the adjusted VSL is greater than the primary VSL (\$6.77 million versus \$6.1 million) at an income elasticity of 1.0, with adjustments for income growth only. The lowest adjusted VSL value (\$3.44 million) is yielded over a 20-year latency period that includes discounting and income growth only (income elasticity = 0.22). Assuming a seven percent discount rate, the highest adjusted VSL is also \$6.77 million (adjusted for income growth only [income elasticity = 1.0]). The lowest adjusted VSL is \$1.61 million (discounted over 20 years).

The first row of both the three and seven percent discount rate panels in Exhibit 5-12 shows the VSL used in the primary analysis. Because this value has not been adjusted for discounting over an assumed and *unknown* latency period, this value does not deviate from the original \$6.1

million used in the primary benefits analysis. The second and third rows of both the three and seven percent panels show the adjustments to the primary VSL to account for changes in WTP for fatal risk reductions associated with real income growth from 1990 to 1999. As real income grows, the WTP to avoid fatal risks is also expected to increase at a rate corresponding to the income elasticity of demand, as discussed below. This income growth, from the years 1990 to 1999, accounts for the differences in incomes of the VSL study population versus the population affected by the Arsenic Rule. This does not include any income adjustments over a latency period because of methodological issues that have not yet been resolved. However, pending the resolution of these issues, EPA may include an adjustment for income growth over a latency period in future analyses, as recommended by the SAB.

The fourth and fifth rows of both the three and seven percent panels illustrates the impacts of adjusting the primary VSL for discounting and income growth over a range of assumed latency periods. As is shown in Exhibit 5-12, this value decreases from \$5.84 million assuming a five-year latency period to \$3.75 million assuming a 20 year latency period (at a three percent discount rate and income elasticity of 1.0). At a seven percent discount rate, this value decreases from \$4.83 million to \$1.75 million.

**Exhibit 5-12**  
**Sensitivity of the Primary VSL Estimate to Changes in Latency Period Assumptions,**  
**Income Growth, and Other Adjustments**  
**(\$ millions, 1999)**

Adjustment Factor		Latency Period (Years)		
		5	10	20
<b>3 % Discount Rate</b>				
Primary Analysis (No VSL Adjustment)		6.1	6.1	6.1
Adjusted for Income Growth <sup>1</sup>	elasticity = 0.22	6.22	6.22	6.22
	elasticity = 1.0	6.77	6.77	6.77
Adjusted for Income Growth <sup>1</sup> and Discounting	elasticity = 0.22	5.37	4.63	3.44
	elasticity = 1.0	5.84	5.04	3.75
Adjusted for Income Growth <sup>1</sup> , Discounting, and 7% Increase for Voluntariness and Controllability	elasticity = 0.22	5.74	4.95	3.69
	elasticity = 1.0	6.25	5.39	4.01
Break-Even for Other Characteristics (as a percentage of the primary VSL estimate)				
elasticity = 0.22		6 %	19 %	40 %
elasticity = 1.0		-2 %	12 %	34 %
<b>7 % Discount Rate</b>				
Primary Analysis (No VSL Adjustment)		6.1	6.1	6.1
Adjusted for Income Growth <sup>1</sup>	elasticity = 0.22	6.22	6.22	6.22
	elasticity = 1.0	6.77	6.77	6.77
Adjusted for Income Growth <sup>1</sup> and Discounting	elasticity = 0.22	4.44	3.16	1.61
	elasticity = 1.0	4.83	3.44	1.75
Adjusted for Income Growth <sup>1</sup> , Discounting, and 7% Increase for Voluntariness and Controllability	elasticity = 0.22	4.75	3.38	1.72
	elasticity = 1.0	5.17	3.68	1.87
Break-Even for Other Characteristics (as a percentage of the primary VSL estimate)				
elasticity = 0.22		22 %	45 %	72 %
elasticity = 1.0		15 %	40 %	69 %
1. This adjustment reflects the change in WTP based on real income growth from 1990 to 1999.				

The sixth and seventh rows of the three and seven percent panels illustrate the effects of incorporating a seven percent increase for voluntariness and controllability as recommended for a sensitivity analysis in the SAB report on valuing fatal cancer risk reductions (SAB, 2000). One member of the SAB committee noted in the SAB report that this adjustment may be as high as two times the primary VSL, but this value is highly speculative. The seven percent adjustment accounts for empirical evidence in the literature that indicates individuals may place a higher willingness to pay (WTP) on risks where exposure is neither voluntary nor controllable by the individual.

In adjusting for both income growth and voluntariness and controllability, EPA used a range of income elasticities from the economics literature. Income elasticity is the percent change in demand for a good (in this case, WTP for fatal risk reductions) for every one percent change in income. For example, an income elasticity of 1.0 implies that a 10 percent higher income level results in a 10 percent higher WTP for fatal risk reductions. In a recent study (EPA, 2000c), EPA reviewed the literature related to the income elasticity of demand for the prevention of fatal health impacts. Based on data from cross-sectional studies of wage premiums, a range of elasticity estimates for serious health impacts was developed, ranging from a lower-end estimate of 0.22 to an upper-end estimate of 1.0.

There are several other characteristics that differ between the VSL estimates used in the primary analysis and an ideal estimate specific to the case of cancer risks from arsenic. These include a cancer premium, differences in risk aversion, altruism, age of the individual affected, and a morbidity component of the VSL mortality estimate. Very little empirical information is available on the impact that these characteristics have on VSL estimates; thus, they are not accounted for directly in this sensitivity analysis. A more complete discussion of the other characteristics identified by economists as having a potential impact on WTP to reduce mortality risks can be found in Chapter Seven of the Agency's *Guidelines for Preparing Economic Analyses* (EPA, 2000c), which is available in the docket for this final rulemaking.

However, it is possible to use a "break even" analysis to address the question: what would the impact on VSL of these additional characteristics need to be to produce the \$6.1 million VSL used in the primary benefits analysis (described earlier in this chapter). The last two rows of the three and seven percent panels of Exhibit 5-12 attempt to answer this question in percentage terms. For example, at a three percent discount rate over a ten year latency period and income elasticity of 1.0, a factor of 12 percent (as shown in the bottom row of the three percent panel of Exhibit 5-12) indicates that if accounting for these characteristics would increase VSL by more than 12 percent then the primary analysis will tend to understate the value of risk reductions. If accounting for these characteristics would not increase VSL by at least 12 percent then the primary analysis may overstate benefits (a negative percentage indicates that the primary analysis understates benefits unless the combined impact of these additional characteristics actually reduces VSL estimates).

Some researchers believe that the value of some of these characteristics will substantially add to the unadjusted VSL (one study suggests that a cancer premium alone may be worth an additional 100 percent of primary VSL value [Revesz, 1999]). Some researchers also believe that some of these characteristics have a negative effect on VSL, suggesting that some of these factors offset one another. Until we know more about these various factors we cannot explicitly make adjustments to existing VSL estimates.

The SAB noted in their report that these characteristics require more empirical research prior to incorporation into the Agency's primary benefits analysis, but could be explored as part of a sensitivity analysis.

### 5.5.3 Results

Exhibit 5-13 illustrates the impacts of changes in VSL adjustment factor assumptions on the estimated benefits for the range of fatal bladder and lung cancer cases avoided in the final Arsenic Rule, assuming a three percent discount rate. The results of this analysis at a seven percent discount rate are given in Exhibit 5-14. These results were calculated by applying the adjusted VSLs from Exhibit 5-12 to the lower- and upper-bound estimates of fatal bladder and lung cancer cases avoided as shown in Exhibit 5-9 (c). For purposes of this sensitivity analysis, EPA presented combined bladder and lung cancer cases avoided in Exhibits 5-13 and 5-14. Health risk reduction benefits attributable to reduced arsenic levels in both CWSs and NTNCWSs are presented in these exhibits as well.

It is important to note that the monetized benefits estimates shown in this section reflect *quantifiable* benefits only. As shown in Section 5.2, there are a significant number of *non-quantifiable* benefits associated with regulating arsenic in drinking water. As a result, the monetized benefits presented in the following exhibit represent a lower-bound estimate. Were EPA able to quantify some of the currently non-quantifiable health effects and other benefits associated with arsenic regulation, monetized benefits estimates could be significantly higher than what are shown in the exhibit.

**Exhibit 5-13. Sensitivity of Combined Annual Bladder and Lung Cancer Mortality Benefits Estimates to Changes in VSL Adjustment Factor Assumptions (\$ millions, 1999, 3% discount rate)<sup>1</sup>**

Arsenic Level (Fg/L)		3	5	10	20
<b>5 Year Latency Period Assumption</b>					
Primary Analysis (No VSL Adjustment)		199-452	176-328	130-182	62-69
Adjusted for Income Growth <sup>2</sup>	E = 0.22	203-461	181-334	133-186	63-70
	E = 1.0	221-502	197-364	144-202	69-77
Adjusted for Income Growth <sup>2</sup> and Discounting	E = 0.22	175-398	156-288	114-160	55-61
	E = 1.0	190-433	170-314	124-174	60-66
Adjusted for Income Growth <sup>2</sup> , Discounting, and 7% Increase for Voluntariness and Controllability	E = 0.22	187-425	167-308	122-171	59-65
	E = 1.0	204-463	182-336	133-186	64-71
<b>10 Year Latency Period Assumption</b>					
Primary Analysis (No VSL Adjustment)		199-452	176-328	130-182	62-69
Adjusted for Income Growth <sup>2</sup>	E = 0.22	203-461	181-334	133-186	63-70
	E = 1.0	221-502	197-364	144-202	69-77
Adjusted for Income Growth <sup>2</sup> and Discounting	E = 0.22	151-343	135-249	99-138	47-52
	E = 1.0	164-373	147-271	107-150	51-57
Adjusted for Income Growth <sup>2</sup> , Discounting, and 7% Increase for Voluntariness and Controllability	E = 0.22	161-367	144-266	105-148	50-56
	E = 1.0	176-399	157-289	115-161	55-61
<b>20 Year Latency Period Assumption</b>					
Primary Analysis (No VSL Adjustment)		199-452	176-328	130-182	62-69
Adjusted for Income Growth <sup>2</sup>	E = 0.22	203-461	181-334	133-186	63-70
	E = 1.0	221-502	197-364	144-202	69-77
Adjusted for Income Growth <sup>2</sup> and Discounting	E = 0.22	112-255	100-185	73-103	35-39
	E = 1.0	122-278	109-201	80-112	38-42
Adjusted for Income Growth <sup>2</sup> , Discounting, and 7% Increase for Voluntariness and Controllability	E = 0.22	120-273	107-198	79-110	38-42
	E = 1.0	131-297	117-215	85-119	41-45
<p>1. The lower- and upper-bound benefits estimates correspond to the lower- and upper-bound risk estimates and cancer cases avoided as shown in section III.D.2 of this preamble.</p> <p>2. This adjustment reflects the change in WTP based on real income growth from 1990 to 1999. E = income elasticity.</p>					

**Exhibit 5-14**  
**Sensitivity of Combined Annual Bladder and Lung Cancer Mortality Benefits Estimates to**  
**Changes in VSL Adjustment Factor Assumptions**  
**(\$ millions, 1999, 7% discount rate)<sup>1</sup>**

Arsenic Level (Fg/L)		3	5	10	20
<b>5 Year Latency Period Assumption</b>					
Primary Analysis (No VSL Adjustment)		199-452	178-328	130-182	62-69
Adjusted for Income Growth <sup>2</sup>	E = 0.22	203-461	181-334	133-186	63-70
	E = 1.0	221-502	197-364	144-202	69-77
Adjusted for Income Growth <sup>2</sup> and Discounting	E = 0.22	145-329	129-238	95-132	45-50
	E = 1.0	157-358	141-259	103-144	50-55
Adjusted for Income Growth <sup>2</sup> , Discounting, and 7% Increase for Voluntariness and Controllability	E = 0.22	155-352	138-255	102-142	49-54
	E = 1.0	168-383	150-278	110-154	53-58
<b>10 Year Latency Period Assumption</b>					
Primary Analysis (No VSL Adjustment)		199-452	178-328	130-182	62-69
Adjusted for Income Growth <sup>2</sup>	E = 0.22	203-461	181-334	133-186	63-70
	E = 1.0	221-502	197-364	144-202	69-77
Adjusted for Income Growth <sup>2</sup> and Discounting	E = 0.22	103-234	92-170	67-94	32-36
	E = 1.0	112-255	100-185	73-103	35-39
Adjusted for Income Growth <sup>2</sup> , Discounting, and 7% Increase for Voluntariness and Controllability	E = 0.22	110-251	98-182	72-101	35-38
	E = 1.0	120-273	107-198	78-110	38-42
<b>20 Year Latency Period Assumption</b>					
Primary Analysis (No VSL Adjustment)		199-452	178-328	130-182	62-69
Adjusted for Income Growth <sup>2</sup>	E = 0.22	203-461	181-334	133-186	63-70
	E = 1.0	221-502	197-364	144-202	69-77
Adjusted for Income Growth <sup>2</sup> and Discounting	E = 0.22	53-119	47-86	34-48	16-18
	E = 1.0	57-130	51-94	37-52	18-20
Adjusted for Income Growth <sup>2</sup> , Discounting, and 7% Increase for Voluntariness and Controllability	E = 0.22	56-127	50-92	37-51	18-20
	E = 1.0	61-139	54-100	40-56	19-21
<p>1. The lower- and upper-bound benefits estimates correspond to the lower- and upper-bound risk estimates and cancer cases avoided as shown in section III.D.2 of this preamble.</p> <p>2. This adjustment reflects the change in WTP based on real income growth from 1990 to 1999. E = income elasticity.</p>					

As shown in Exhibits 5-13 and 5-14, the highest range of adjusted benefits estimates at the 10 µg/L MCL (\$144 - \$202 million at three percent and seven percent) are yielded when benefits are adjusted for income growth only with an income elasticity of 1.0. The lowest adjusted benefits estimates at the 10 µg/L MCL (\$73 - \$103 million at three percent, \$34 - \$48 million at seven percent) are yielded under the assumption of a 20-year latency period that includes adjustments for discounting and income growth (income elasticity = 0.22). These results indicate the high degree of sensitivity of benefits estimates to different assumptions of a latency period and income elasticity and also the inclusion of adjustments for income growth and voluntariness and controllability.

## **5.6 Other Benefits of Reductions in Arsenic Exposure**

It is well established that the public often avoids the use of tap water that is suspected to be contaminated. In this context, contamination may suggest biological, chemical, or other water quality issues. When public perception of water quality declines, consumers purchase bottled water if they have the means to do so.

In addition or as an alternative, they may avoid the use of tap water, ingesting and cooking with other liquids, substituting pre-mixed baby formula, and using other strategies to limit ingestion. Consumer avoidance of tap water sources usually results in costs to the consumers, either in the cost of obtaining substitute fluids or potential health impacts of reduced fluid intake. In addition, there are numerous cases where government agencies have provided bottled water due to biological or chemical contamination. The levels of contamination at which the government activities occur vary depending on a variety of factors.

The relationship between arsenic in tap water and changes in consumer behavior or government interventions is a complex one. Factors that impact the choice to avoid tap water depend on public information that is provided on levels of contamination, potential health effects, individual aversions to risk taking, and other considerations. A quantitative evaluation of these responses and the potential benefits of avoiding associated costs to the consumer or governments is not included in this benefits assessment. However, it is clear that many consumers purchase bottled water (a multimillion dollar industry) or invest in other methods of improving drinking water quality, such as point-of-use (POU) devices, specifically to avoid ingestion of contaminants such as arsenic. Thus, it is reasonable to conclude that a reduction in arsenic contamination will have the long-term effect of restoring some level of consumer confidence in the water supply.

## Chapter 6: Cost Analysis

### 6.1 Introduction

This chapter presents the national cost estimates for the Arsenic Rule. The costs associated with the rule include: (1) costs borne by water systems to comply with the new MCL standard and modified monitoring requirements, and (2) costs to the States to implement and enforce the rule. Section 6.2 describes the inputs and methodologies used to estimate costs, including the following:

- A description of the technologies that may be used by systems to achieve the MCL (Section 6.2.1);
- The unit costs of different technologies for complying with the MCLs (Section 6.2.2);
- System and State unit costs for monitoring and administration functions (Section 6.2.3); and
- The methods used to predict systems' compliance methods (Section 6.2.4) and the methods used to calculate costs (Section 6.2.5).

Section 6.3 presents the results of the cost analysis, including the following:

- A summary of national costs for the different regulatory options (Section 6.3.1);
- Costs by system size and type for the MCL options (Section 6.3.2); and
- Household costs (Section 6.3.3).

Section 6.4 discusses the uncertainty inherent in the distribution of estimated national compliance costs.

### 6.2 Methodology

#### 6.2.1 Description of Available Technologies

In 1993, EPA developed a document entitled *Treatment and Occurrence—Arsenic in Potable Water Supplies* (EPA, 1993), which summarized the results of pilot-scale studies examining low-level arsenic removal, from 50 micrograms per liter ( $\mu\text{g/L}$ ) down to 1  $\mu\text{g/L}$  or less. EPA convened a panel of outside experts in January 1994 to review this document and comment on the ability of the technologies to achieve various MCLs. The Agency has since sought stakeholder input on the use of various technologies for arsenic removal under different conditions, and has incorporated that input into its estimates of technology performance and costs. The results are documented in the *Cost and Technology Document for the Arsenic Rule* (EPA, 2000d). The technology cost functions and removal efficiencies presented in that document are used as inputs for the cost analyses presented in this EA.

Some technologies generate wastes that require disposal or pre-treatment (e.g., pre-oxidation or corrosion control) in order to be effective. These associated requirements were identified for

different technologies and system types, and their costs were included in the costs of treatment where relevant.

In addition to these centralized treatment options, small systems may elect to use point-of-use (POU) devices to achieve compliance with the MCLs. POU involves treatment at the tap. The available POU technologies for arsenic removal are essentially smaller versions of reverse osmosis and activated alumina. These technologies will have to be maintained by the water system, involving some additional recordkeeping and maintenance costs.

The result of the review of technologies that would effectively remove arsenic and bring a water system into compliance is summarized in Exhibit 6-1. The list includes 13 treatment trains available to systems, consisting of various combinations of compliance technologies, waste disposal technologies, or pre-treatment technologies as required.

**Exhibit 6-1**  
**Arsenic Rule Treatment Trains by Compliance Technologies Component**  
**with Associated Removal Efficiencies**

Treatment Technology		Waste Disposal Technology				Corrosion Control	Pre-Oxidation <sup>o</sup>	Removal Efficiency
		POTW	Non-Hazardous Landfill	Mechanical De-Watering	Non-Mechanical De-Watering			
1	Modify Lime Softening						H	90%
2	Modify Coagulation/Filtration						H	95%
3	Anion Exchange (<20 mg/L SO4)	H					H	95%
4	Anion Exchange (20-50 mg/L SO4)	H					H	95%
5	Coagulation Assisted Microfiltration		H	H			H	90%
6	Coagulation Assisted Microfiltration		H		H		H	90%
7	Oxidation Filtration (Greensand)	H*					H	50%
8	Activated Alumina (pH 7 - pH 8)		H**				H	95%
9	Activated Alumina (pH 8 - pH 8.3)		H**				H	95%
10	Activated Alumina (23,100 BV) with pH adjustment (pH 6)		H**			H	H	95%
11	Activated Alumina (15,400 BV) with pH adjustment (pH 6)		H**			H	H	95%
12	POU Activated Alumina						H	90%
13	POU Reverse Osmosis						H	90%

<sup>o</sup> pre-oxidation incorporated into treatment trains based on a separate decision tree

\* POTW for backwash stream

\*\* non-hazardous landfill (for spent media)

## 6.2.2 Unit Costs and Compliance Assumptions

EPA estimated the costs of the various compliance technologies, including the centralized treatment technologies associated waste disposal technologies, and POU treatment technologies, excluding pre-treatment costs. Pre-treatment costs were separate treatment costs that apply to a particular set of systems (some systems would already have pre-treatment in place). Costs of each treatment train are estimated as functions of system size, design flow (used to calculate capital costs) and average flow (used to calculate operating and maintenance costs). Exhibits 6-2 (a) and 6-2 (b) presents a summary of unit compliance technology costs by cost component for the treatment trains listed in Exhibit 6-1, annualized over 20 years at a seven percent discount rate. Costs are in May 1999 dollars and are based on average and design flows for median populations of each system size category.

The unit costs are provided to demonstrate the range of costs across the treatment technologies for an MCL of 10 $\mu$ g/L, assuming either an influent arsenic concentration of 11 $\mu$ g/L (low range estimates shown in Exhibit 6-2(a)) or an influent arsenic concentration of 50 $\mu$ g/L (high range estimates shown in Exhibit 6-2(b)). EPA calculated these average unit costs for a single contaminated entry point, assuming a publicly-owned ground water system with the average number of entry points per system in that size category. Note that the capital and operating ad maintenance (O&M) cost components are listed separately for the treatment and waste disposal components of the treatment train. These costs are annualized over 20 years at a seven percent discount rate. Detailed descriptions of the assumptions and methodologies used to develop the underlying cost curves are available in the *Cost and Technology Document for the Arsenic Rule* (EPA, 2000d).

**Exhibit 6-2a**  
**System Compliance Technology Costs Assuming Influent Concentration of 11 µg/L and MCL of 10 µg/L (Dollars)**

Size Category	Treatment Train No.	1	2	3	4	5	6
< 100	Treatment Capital Costs	\$ 8,999	\$ 7,483	\$ 21,957	\$ 22,724	\$ 127,885	\$ 127,885
	Treatment O&M Costs	\$ 484	\$ 260	\$ 5,104	\$ 8,604	\$ 22,361	\$ 20,585
	Waste Disposal Capital Costs	\$ -	\$ -	\$ 3,955	\$ 3,955	\$ 29,900	\$ 20,686
	Waste Disposal O&M Costs	\$ -	\$ -	\$ 381	\$ 387	\$ 6,946	\$ 2,131
	Annual Costs (7%)	\$ 1,333	\$ 966	\$ 7,930	\$ 11,510	\$ 44,200	\$ 36,740
101-500	Treatment Capital Costs	\$ 13,688	\$ 8,966	\$ 21,957	\$ 37,150	\$ 265,526	\$ 265,526
	Treatment O&M Costs	\$ 1,416	\$ 482	\$ 5,104	\$ 9,470	\$ 23,619	\$ 22,400
	Waste Disposal Capital Costs	\$ -	\$ -	\$ 3,955	\$ 3,955	\$ 43,354	\$ 118,165
	Waste Disposal O&M Costs	\$ -	\$ -	\$ 412	\$ 455	\$ 12,863	\$ 2,177
	Annual Costs (7%)	\$ 2,708	\$ 1,328	\$ 7,962	\$ 13,804	\$ 65,638	\$ 60,795
501-1,000	Treatment Capital Costs	\$ 14,756	\$ 9,316	\$ 21,957	\$ 40,669	\$ 295,452	\$ 295,452
	Treatment O&M Costs	\$ 1,766	\$ 565	\$ 5,104	\$ 9,791	\$ 24,090	\$ 23,081
	Waste Disposal Capital Costs	\$ -	\$ -	\$ 3,955	\$ 3,955	\$ 46,424	\$ 141,947
	Waste Disposal O&M Costs	\$ -	\$ -	\$ 424	\$ 480	\$ 14,929	\$ 2,195
	Annual Costs (7%)	\$ 3,159	\$ 1,444	\$ 7,974	\$ 14,483	\$ 71,290	\$ 66,563
1,001-3,300	Treatment Capital Costs	\$ 24,087	\$ 12,655	\$ 38,991	\$ 120,712	\$ 526,687	\$ 526,687
	Treatment O&M Costs	\$ 4,760	\$ 1,266	\$ 5,104	\$ 12,431	\$ 28,088	\$ 28,088
	Waste Disposal Capital Costs	\$ -	\$ -	\$ 3,955	\$ 3,955	\$ 73,454	\$ 330,519
	Waste Disposal O&M Costs	\$ -	\$ -	\$ 525	\$ 696	\$ 29,102	\$ 2,364
	Annual Costs (7%)	\$ 7,034	\$ 2,460	\$ 9,682	\$ 24,894	\$ 113,839	\$ 111,366

NOTE: Average costs were calculated assuming a publicly-owned groundwater system with a single contaminated entry point, based on median population and the average number of entry points per system in the service size category, for the treatment train technologies described in Exhibit 6-1.

**Exhibit 6-2a (continued)**  
**System Compliance Technology Costs Assuming Influent Concentration of 11 µg/L and MCL of 10 µg/L (Dollars)**

Size Category	Treatment Train No.	1	2	3	4	5	6
3,301-10,000	Treatment Capital Costs	\$ 64,447	\$ 40,103	\$ 38,991	\$ 211,802	\$ 1,069,210	\$ 1,069,210
	Treatment O&M Costs	\$ 14,961	\$ 4,833	\$ 5,104	\$ 20,403	\$ 38,522	\$ 38,522
	Waste Disposal Capital Costs	\$ -	\$ -	\$ 3,955	\$ 3,955	\$ 121,208	\$ 762,407
	Waste Disposal O&M Costs	\$ -	\$ -	\$ 869	\$ 1,434	\$ 32,307	\$ 9,170
	Annual Costs (7%)	\$ 21,045	\$ 8,618	\$ 10,027	\$ 42,203	\$ 183,196	\$ 220,583
10,001-50,000	Treatment Capital Costs	\$ 247,207	\$ 168,801	\$ 38,991	\$ 362,184	\$ 1,793,771	\$ 1,793,771
	Treatment O&M Costs	\$ 35,250	\$ 17,001	\$ 5,104	\$ 31,688	\$ 55,413	\$ 55,413
	Waste Disposal Capital Costs	\$ -	\$ -	\$ 3,955	\$ 3,955	\$ 209,000	\$ 1,610,846
	Waste Disposal O&M Costs	\$ -	\$ -	\$ 1,593	\$ 2,984	\$ 45,793	\$ 50,349
	Annual Costs (7%)	\$ 58,584	\$ 32,934	\$ 10,750	\$ 69,233	\$ 290,253	\$ 427,134
50,001-100,000	Treatment Capital Costs	\$ 455,707	\$ 315,625	\$ 38,991	\$ 529,645	\$ 2,368,818	\$ 2,368,818
	Treatment O&M Costs	\$ 61,149	\$ 32,533	\$ 5,104	\$ 54,032	\$ 59,325	\$ 59,325
	Waste Disposal Capital Costs	\$ -	\$ -	\$ 5,085	\$ 5,085	\$ 309,158	\$ 2,381,322
	Waste Disposal O&M Costs	\$ -	\$ -	\$ 2,516	\$ 4,962	\$ 52,569	\$ 66,722
	Annual Costs (7%)	\$ 104,165	\$ 62,326	\$ 11,780	\$ 109,470	\$ 364,675	\$ 574,426
100,001-1,000,000	Treatment Capital Costs	\$ 1,462,373	\$ 918,353	\$ 38,991	\$ 1,873,015	\$ 6,887,505	\$ 6,887,505
	Treatment O&M Costs	\$ 309,897	\$ 177,044	\$ 5,104	\$ 168,459	\$ 96,658	\$ 96,658
	Waste Disposal Capital Costs	\$ -	\$ -	\$ 5,085	\$ 5,085	\$ 954,312	\$ 9,517,736
	Waste Disposal O&M Costs	\$ -	\$ -	\$ 12,132	\$ 25,570	\$ 178,509	\$ 237,418
	Annual Costs (7%)	\$ 447,935	\$ 263,730	\$ 21,396	\$ 371,308	\$ 1,015,379	\$ 1,882,614

NOTE: Average costs were calculated assuming a publicly-owned groundwater system with a single contaminated entry point, based on median population and the average number of entry points per system in the service size category, for the treatment train technologies described in Exhibit 6-1.

**Exhibit 6-2a (continued)**  
**System Compliance Technology Costs Assuming Influent Concentration of 11 µg/L and MCL of 10 µg/L (Dollars)**

Size Category	Treatment Train No.	7	8	9	10	11	12	13
< 100	Treatment Capital Costs	\$ 15,023	\$ 13,629	\$ 13,629	\$ 45,787	\$ 45,787	\$ 4,671	\$ 13,619
	Treatment O&M Costs	\$ 7,711	\$ 4,414	\$ 6,944	\$ 6,050	\$ 6,643	\$ 6,725	\$ 4,433
	Waste Disposal Capital Costs	\$ 3,955	\$ -	\$ -	\$ -	\$ -	\$ -	\$ -
	Waste Disposal O&M Costs	\$ 446	\$ 12	\$ 22	\$ 5	\$ 8	\$ -	\$ -
	Annual Costs (7%)	\$ 9,949	\$ 5,712	\$ 8,253	\$ 10,377	\$ 10,972	\$ 7,390	\$ 6,372
101-500	Treatment Capital Costs	\$ 63,059	\$ 29,131	\$ 29,131	\$ 62,507	\$ 62,507	\$ 27,027	\$ 78,866
	Treatment O&M Costs	\$ 8,540	\$ 6,065	\$ 10,087	\$ 7,494	\$ 8,437	\$ 39,804	\$ 26,552
	Waste Disposal Capital Costs	\$ 3,955	\$ -	\$ -	\$ -	\$ -	\$ -	\$ -
	Waste Disposal O&M Costs	\$ 571	\$ 78	\$ 150	\$ 34	\$ 51	\$ -	\$ -
	Annual Costs (7%)	\$ 15,437	\$ 8,892	\$ 12,986	\$ 13,428	\$ 14,388	\$ 43,652	\$ 37,781
501-1,000	Treatment Capital Costs	\$ 73,464	\$ 32,912	\$ 32,912	\$ 66,586	\$ 66,586	\$ 34,915	\$ 101,897
	Treatment O&M Costs	\$ 8,904	\$ 6,684	\$ 11,265	\$ 8,036	\$ 9,110	\$ 51,591	\$ 34,475
	Waste Disposal Capital Costs	\$ 3,955	\$ -	\$ -	\$ -	\$ -	\$ -	\$ -
	Waste Disposal O&M Costs	\$ 618	\$ 103	\$ 197	\$ 44	\$ 67	\$ -	\$ -
	Annual Costs (7%)	\$ 16,830	\$ 9,893	\$ 14,569	\$ 14,365	\$ 15,462	\$ 56,562	\$ 48,983
1,001-3,300	Treatment Capital Costs	\$ 170,709	\$ 60,846	\$ 60,846	\$ 97,616	\$ 97,616	\$ 97,980	\$ 286,071
	Treatment O&M Costs	\$ 12,006	\$ 11,930	\$ 21,255	\$ 12,627	\$ 14,814	\$ 146,709	\$ 98,728
	Waste Disposal Capital Costs	\$ 3,955	\$ -	\$ -	\$ -	\$ -	\$ -	\$ -
	Waste Disposal O&M Costs	\$ 1,016	\$ 313	\$ 602	\$ 135	\$ 203	\$ -	\$ -
	Annual Costs (7%)	\$ 29,509	\$ 17,986	\$ 27,600	\$ 21,977	\$ 24,231	\$ 160,659	\$ 139,458

NOTE: Average costs were calculated assuming a publicly-owned groundwater system with a single contaminated entry point, based on median population and the average number of entry points per system in the service size category, for the treatment train technologies described in Exhibit 6-1. In Treatment Trains 8 -11, waste disposal O&M costs include only non-hazardous landfill tipping fees, and therefore, are quite low.

**Exhibit 6-2a (continued)**  
**System Compliance Technology Costs Assuming Influent Concentration of 11 µg/L and MCL of 10 µg/L (Dollars)**

Size Category	Treatment Train No.	7	8	9	10	11	12	13
3,301-10,000	Treatment Capital Costs	\$ 421,562	\$ 159,129	\$ 159,129	\$ 205,374	\$ 205,374	\$ 296,207	\$ 865,248
	Treatment O&M Costs	\$ 22,659	\$ 29,916	\$ 55,506	\$ 28,369	\$ 34,369	\$ 449,875	\$ 305,030
	Waste Disposal Capital Costs	\$ 3,955	\$ -	\$ -	\$ -	\$ -	\$ -	\$ -
	Waste Disposal O&M Costs	\$ 2,381	\$ 1,033	\$ 1,988	\$ 447	\$ 671	\$ -	\$ -
	Annual Costs (7%)	\$ 65,205	\$ 45,970	\$ 72,514	\$ 48,202	\$ 54,426	\$ 492,048	\$ 428,222
10,001-50,000	Treatment Capital Costs	\$ 787,837	\$ 324,276	\$ 324,276	\$ 386,442	\$ 386,442	\$ 682,321	\$ 1,993,842
	Treatment O&M Costs	\$ 45,012	\$ 67,654	\$ 127,369	\$ 61,397	\$ 75,399	\$ 1,047,475	\$ 714,269
	Waste Disposal Capital Costs	\$ 5,085	\$ -	\$ -	\$ -	\$ -	\$ -	\$ -
	Waste Disposal O&M Costs	\$ 5,951	\$ 2,545	\$ 4,895	\$ 1,102	\$ 1,653	\$ -	\$ -
	Annual Costs (7%)	\$ 125,809	\$ 100,809	\$ 162,873	\$ 98,976	\$ 113,530	\$ 1,144,622	\$ 998,147
50,001-100,000	Treatment Capital Costs	\$ 1,168,062	\$ 512,683	\$ 512,683	\$ 593,012	\$ 593,012	\$ 1,150,447	\$ 3,362,537
	Treatment O&M Costs	\$ 73,549	\$ 122,590	\$ 231,264	\$ 109,500	\$ 134,983	\$ 1,778,028	\$ 1,216,748
	Waste Disposal Capital Costs	\$ 5,085	\$ -	\$ -	\$ -	\$ -	\$ -	\$ -
	Waste Disposal O&M Costs	\$ 9,606	\$ 4,476	\$ 8,608	\$ 1,938	\$ 2,906	\$ -	\$ -
	Annual Costs (7%)	\$ 193,892	\$ 175,459	\$ 288,265	\$ 167,413	\$ 193,866	\$ 1,941,826	\$ 1,695,498
100,001-1,000,000	Treatment Capital Costs	\$ 4,098,917	\$ 2,257,773	\$ 2,257,773	\$ 2,506,335	\$ 2,506,335	\$ 5,567,338	\$ 16,283,352
	Treatment O&M Costs	\$ 370,791	\$ 629,270	\$ 1,199,321	\$ 548,870	\$ 682,540	\$ 8,780,565	\$ 6,073,580
	Waste Disposal Capital Costs	\$ 5,085	\$ -	\$ -	\$ -	\$ -	\$ -	\$ -
	Waste Disposal O&M Costs	\$ 47,683	\$ 24,581	\$ 47,274	\$ 10,642	\$ 15,962	\$ -	\$ -
	Annual Costs (7%)	\$ 805,863	\$ 866,968	\$ 1,459,713	\$ 796,092	\$ 935,082	\$ 9,573,229	\$ 8,391,963

NOTE: Average costs were calculated assuming a publicly-owned groundwater system with a single contaminated entry point, based on median population and the average number of entry points per system in the service size category, for the treatment train technologies described in Exhibit 6-1. In Treatment Trains 8 -11, waste disposal O&M costs include only non-hazardous landfill tipping fees, and therefore, are quite low.

**Exhibit 6-2b**  
**System Compliance Technology Costs Assuming Influent Concentration of 50 µg/L and MCL of 10 µg/L (Dollars)**

Size Category	Treatment Train No.	1	2	3	4	5	6
< 100	Treatment Capital Costs	\$ 8,999	\$ 7,483	\$ 26,970	\$ 29,332	\$ 193,923	\$ 193,923
	Treatment O&M Costs	\$ 484	\$ 260	\$ 5,365	\$ 8,924	\$ 21,251	\$ 21,251
	Waste Disposal Capital Costs	\$ -	\$ -	\$ 3,955	\$ 3,955	\$ 36,236	\$ 65,339
	Waste Disposal O&M Costs	\$ -	\$ -	\$ 392	\$ 412	\$ 9,187	\$ 2,148
	Annual Costs (7%)	\$ 1,333	\$ 966	\$ 8,676	\$ 12,478	\$ 52,164	\$ 47,872
101-500	Treatment Capital Costs	\$ 13,688	\$ 8,966	\$ 43,632	\$ 117,795	\$ 508,282	\$ 508,282
	Treatment O&M Costs	\$ 1,416	\$ 482	\$ 5,365	\$ 11,527	\$ 26,696	\$ 26,696
	Waste Disposal Capital Costs	\$ -	\$ -	\$ 3,955	\$ 3,955	\$ 71,219	\$ 316,779
	Waste Disposal O&M Costs	\$ -	\$ -	\$ 490	\$ 621	\$ 24,844	\$ 2,301
	Annual Costs (7%)	\$ 2,708	\$ 1,328	\$ 10,346	\$ 23,640	\$ 106,241	\$ 106,877
501-1,000	Treatment Capital Costs	\$ 14,756	\$ 9,316	\$ 43,632	\$ 126,653	\$ 564,187	\$ 564,187
	Treatment O&M Costs	\$ 1,766	\$ 565	\$ 5,365	\$ 12,469	\$ 28,148	\$ 28,148
	Waste Disposal Capital Costs	\$ -	\$ -	\$ 3,955	\$ 3,955	\$ 77,743	\$ 358,515
	Waste Disposal O&M Costs	\$ -	\$ -	\$ 526	\$ 699	\$ 29,269	\$ 2,367
	Annual Costs (7%)	\$ 3,159	\$ 1,444	\$ 10,383	\$ 25,497	\$ 118,010	\$ 117,611
1,001-3,300	Treatment Capital Costs	\$ 24,087	\$ 12,655	\$ 43,632	\$ 218,240	\$ 1,103,278	\$ 1,103,278
	Treatment O&M Costs	\$ 4,760	\$ 1,266	\$ 5,365	\$ 19,699	\$ 37,737	\$ 37,737
	Waste Disposal Capital Costs	\$ -	\$ -	\$ 3,955	\$ 3,955	\$ 124,926	\$ 793,148
	Waste Disposal O&M Costs	\$ -	\$ -	\$ 836	\$ 1,362	\$ 40,238	\$ 8,074
	Annual Costs (7%)	\$ 7,034	\$ 2,460	\$ 10,692	\$ 42,035	\$ 193,909	\$ 224,820

NOTE: Average costs were calculated assuming a publicly-owned groundwater system with a single contaminated entry point, based on median population and the average number of entry points per system in the service size category, for the treatment train technologies described in Exhibit 6-1.

**Exhibit 6-2b (continued)**  
**System Compliance Technology Costs Assuming Influent Concentration of 50 µg/L and MCL of 10 µg/L (Dollars)**

Size Category	Treatment Train No.	1	2	3	4	5	6
3,301-10,000	Treatment Capital Costs	\$ 64,447	\$ 40,103	\$ 43,632	\$ 490,994	\$ 2,255,079	\$ 2,255,079
	Treatment O&M Costs	\$ 14,961	\$ 4,833	\$ 5,365	\$ 45,678	\$ 56,923	\$ 56,923
	Waste Disposal Capital Costs	\$ -	\$ -	\$ 5,085	\$ 5,085	\$ 285,807	\$ 2,123,020
	Waste Disposal O&M Costs	\$ -	\$ -	\$ 1,897	\$ 3,637	\$ 48,028	\$ 42,687
	Annual Costs (7%)	\$ 21,045	\$ 8,618	\$ 11,860	\$ 96,141	\$ 344,793	\$ 512,872
10,001-50,000	Treatment Capital Costs	\$ 247,207	\$ 168,801	\$ 43,632	\$ 923,917	\$ 3,571,834	\$ 3,571,834
	Treatment O&M Costs	\$ 35,250	\$ 17,001	\$ 5,365	\$ 75,188	\$ 65,568	\$ 65,568
	Waste Disposal Capital Costs	\$ -	\$ -	\$ 5,085	\$ 5,085	\$ 513,238	\$ 4,281,260
	Waste Disposal O&M Costs	\$ -	\$ -	\$ 4,124	\$ 8,409	\$ 72,683	\$ 95,250
	Annual Costs (7%)	\$ 58,584	\$ 32,934	\$ 14,087	\$ 171,288	\$ 523,853	\$ 902,095
50,001-100,000	Treatment Capital Costs	\$ 455,707	\$ 315,625	\$ 43,632	\$ 1,378,931	\$ 5,074,043	\$ 5,074,043
	Treatment O&M Costs	\$ 61,149	\$ 32,533	\$ 5,365	\$ 110,599	\$ 76,604	\$ 76,604
	Waste Disposal Capital Costs	\$ -	\$ -	\$ 5,085	\$ 5,085	\$ 717,287	\$ 6,653,715
	Waste Disposal O&M Costs	\$ -	\$ -	\$ 6,967	\$ 14,501	\$ 110,698	\$ 145,696
	Annual Costs (7%)	\$ 104,165	\$ 62,326	\$ 16,930	\$ 255,741	\$ 733,963	\$ 1,329,318
100,001-1,000,000	Treatment Capital Costs	\$ 1,462,373	\$ 918,353	\$ 43,632	\$ 3,623,972	\$ 18,245,297	\$ 18,245,297
	Treatment O&M Costs	\$ 309,897	\$ 177,044	\$ 5,365	\$ 328,792	\$ 203,223	\$ 203,223
	Waste Disposal Capital Costs	\$ -	\$ -	\$ 5,571	\$ 5,571	\$ 2,275,373	\$ 28,628,219
	Waste Disposal O&M Costs	\$ -	\$ -	\$ 36,579	\$ 77,955	\$ 477,280	\$ 672,537
	Annual Costs (7%)	\$ 447,935	\$ 263,730	\$ 46,588	\$ 749,350	\$ 2,617,509	\$ 5,300,288

NOTE: Average costs were calculated assuming a publicly-owned groundwater system with a single contaminated entry point, based on median population and the average number of entry points per system in the service size category, for the treatment train technologies described in Exhibit 6-1.

**Exhibit 6-2b (continued)**  
**System Compliance Technology Costs Assuming Influent Concentration of 50 µg/L and MCL of 10 µg/L (Dollars)**

Size Category	Treatment Train No.	7	8	9	10	11	12	13
< 100	Treatment Capital Costs	\$ 24,983	\$ 20,733	\$ 20,733	\$ 53,449	\$ 53,449	\$ 4,671	\$ 13,619
	Treatment O&M Costs	\$ 7,747	\$ 5,021	\$ 8,098	\$ 6,580	\$ 7,302	\$ 6,725	\$ 4,433
	Waste Disposal Capital Costs	\$ 3,955	\$ -	\$ -	\$ -	\$ -	\$ -	\$ -
	Waste Disposal O&M Costs	\$ 464	\$ 36	\$ 69	\$ 16	\$ 23	\$ -	\$ -
	Annual Costs (7%)	\$ 10,943	\$ 7,014	\$ 10,125	\$ 11,641	\$ 12,371	\$ 7,390	\$ 6,372
101-500	Treatment Capital Costs	\$ 104,869	\$ 57,733	\$ 57,733	\$ 94,204	\$ 94,204	\$ 27,027	\$ 78,866
	Treatment O&M Costs	\$ 9,495	\$ 10,104	\$ 17,779	\$ 11,029	\$ 12,829	\$ 39,804	\$ 26,552
	Waste Disposal Capital Costs	\$ 3,955	\$ -	\$ -	\$ -	\$ -	\$ -	\$ -
	Waste Disposal O&M Costs	\$ 694	\$ 240	\$ 461	\$ 104	\$ 156	\$ -	\$ -
	Annual Costs (7%)	\$ 20,462	\$ 15,794	\$ 23,690	\$ 20,025	\$ 21,877	\$ 43,652	\$ 37,781
501-1,000	Treatment Capital Costs	\$ 104,869	\$ 57,733	\$ 57,733	\$ 94,204	\$ 94,204	\$ 27,027	\$ 78,866
	Treatment O&M Costs	\$ 9,495	\$ 10,104	\$ 17,779	\$ 11,029	\$ 12,829	\$ 39,804	\$ 26,552
	Waste Disposal Capital Costs	\$ 3,955	\$ -	\$ -	\$ -	\$ -	\$ -	\$ -
	Waste Disposal O&M Costs	\$ 694	\$ 240	\$ 461	\$ 104	\$ 156	\$ -	\$ -
	Annual Costs (7%)	\$ 20,462	\$ 15,794	\$ 23,690	\$ 20,025	\$ 21,877	\$ 43,652	\$ 37,781
1,001-3,300	Treatment Capital Costs	\$ 283,894	\$ 166,171	\$ 166,171	\$ 213,095	\$ 213,095	\$ 97,980	\$ 286,071
	Treatment O&M Costs	\$ 15,866	\$ 28,169	\$ 52,180	\$ 26,840	\$ 32,470	\$ 146,709	\$ 98,728
	Waste Disposal Capital Costs	\$ 3,955	\$ -	\$ -	\$ -	\$ -	\$ -	\$ -
	Waste Disposal O&M Costs	\$ 1,511	\$ 963	\$ 1,853	\$ 417	\$ 626	\$ -	\$ -
	Annual Costs (7%)	\$ 44,547	\$ 44,818	\$ 69,718	\$ 47,372	\$ 53,211	\$ 160,659	\$ 139,458

NOTE: Average costs were calculated assuming a publicly-owned groundwater system with a single contaminated entry point, based on median population and the average number of entry points per system in the service size category, for the treatment train technologies described in Exhibit 6-1. In Treatment Trains 8 -11, waste disposal O&M costs include only non-hazardous landfill tipping fees, and therefore, are quite low.

**Exhibit 6-2b (continued)**  
**System Compliance Technology Costs Assuming Influent Concentration of 50 µg/L and MCL of 10 µg/L (Dollars)**

Size Category	Treatment Train No.	7	8	9	10	11	12	13
3,301-10,000	Treatment Capital Costs	\$ 701,070	\$ 468,896	\$ 468,896	\$ 545,004	\$ 545,004	\$ 296,207	\$ 865,248
	Treatment O&M Costs	\$ 35,414	\$ 90,018	\$ 169,031	\$ 81,254	\$ 99,783	\$ 449,875	\$ 305,030
	Waste Disposal Capital Costs	\$ 5,085	\$ -	\$ -	\$ -	\$ -	\$ -	\$ -
	Waste Disposal O&M Costs	\$ 4,721	\$ 3,183	\$ 6,122	\$ 1,378	\$ 2,067	\$ -	\$ -
	Annual Costs (7%)	\$ 106,791	\$ 137,461	\$ 219,413	\$ 134,077	\$ 153,294	\$ 492,048	\$ 428,222
10,001-50,000	Treatment Capital Costs	\$ 1,310,195	\$ 977,574	\$ 977,574	\$ 1,102,719	\$ 1,102,719	\$ 682,321	\$ 1,993,842
	Treatment O&M Costs	\$ 76,430	\$ 207,386	\$ 393,274	\$ 183,031	\$ 226,620	\$ 1,047,475	\$ 714,269
	Waste Disposal Capital Costs	\$ 5,085	\$ -	\$ -	\$ -	\$ -	\$ -	\$ -
	Waste Disposal O&M Costs	\$ 9,975	\$ 7,840	\$ 15,079	\$ 3,394	\$ 5,091	\$ -	\$ -
	Annual Costs (7%)	\$ 210,559	\$ 307,502	\$ 500,628	\$ 290,514	\$ 335,800	\$ 1,144,622	\$ 998,147
50,001-100,000	Treatment Capital Costs	\$ 1,942,521	\$ 1,557,893	\$ 1,557,893	\$ 1,738,984	\$ 1,738,984	\$ 1,150,447	\$ 3,362,537
	Treatment O&M Costs	\$ 128,791	\$ 357,213	\$ 679,531	\$ 312,954	\$ 388,534	\$ 1,778,028	\$ 1,216,748
	Waste Disposal Capital Costs	\$ 5,085	\$ -	\$ -	\$ -	\$ -	\$ -	\$ -
	Waste Disposal O&M Costs	\$ 16,683	\$ 13,785	\$ 26,512	\$ 5,968	\$ 8,952	\$ -	\$ -
	Annual Costs (7%)	\$ 329,314	\$ 518,052	\$ 853,098	\$ 483,070	\$ 561,634	\$ 1,941,826	\$ 1,695,498
100,001-1,000,000	Treatment Capital Costs	\$ 6,816,616	\$ 6,933,014	\$ 6,933,014	\$ 7,632,284	\$ 7,632,284	\$ 5,567,338	\$ 16,283,352
	Treatment O&M Costs	\$ 674,190	\$ 1,917,857	\$ 3,661,282	\$ 1,666,275	\$ 2,075,085	\$ 8,780,565	\$ 6,073,580
	Waste Disposal Capital Costs	\$ 5,598	\$ -	\$ -	\$ -	\$ -	\$ -	\$ -
	Waste Disposal O&M Costs	\$ 86,549	\$ 75,712	\$ 145,611	\$ 32,778	\$ 49,166	\$ -	\$ -
	Annual Costs (7%)	\$ 1,404,708	\$ 2,647,996	\$ 4,461,320	\$ 2,419,486	\$ 2,844,684	\$ 9,573,229	\$ 8,391,963

NOTE: Average costs were calculated assuming a publicly-owned groundwater system with a single contaminated entry point, based on median population and the average number of entry points per system in the service size category, for the treatment train technologies described in Exhibit 6-1. In Treatment Trains 8 -11, waste disposal O&M costs include only non-hazardous landfill tipping fees, and therefore, are quite low.

## 6.2.3 Monitoring and Administrative Costs

### *Monitoring Costs*

Monitoring under the current Arsenic Rule occurs annually for surface water systems, and triennially for ground water systems. Currently, when triggered by a violation the system must perform three additional tests within the month. Under the revised rule to be promulgated in January 2001, systems will still perform monitoring annually (for surface water systems) or every three years (for ground water systems); however, when triggered by a violation, the system will perform quarterly monitoring rather than three more samples in one month. All surface water systems must collect samples no later than December 31, 2006, and all ground water systems must collect samples by December 31, 2007, to demonstrate compliance with the revised MCL.

If quarterly monitoring is required it will continue until the State determines that the system is “reliably and consistently” below the MCL or until the PWS installs treatment. States are able to make this determination after ground water systems have taken two quarterly samples and surface water systems have taken four quarterly samples. Additionally, States may grant a nine-year monitoring waiver to qualifying systems, an option not previously available. To be eligible for a waiver, a system must meet the following criteria:

1. Demonstrate adequate source water protection by completing a vulnerability assessment; and
2. Demonstrate that three previous samples were below the MCL.

The monitoring requirements will impose new costs for some systems as follows:

- NTNCs will incur the full costs of the monitoring requirements for the first time, unless they are located in States that already require NTNCs to monitor for arsenic. For NTNCs that are currently required to monitor for arsenic, the incremental monitoring costs will depend on how the revised national requirements compare with the current State requirements. (It is assumed that States currently require NTNCs to monitor using the ground water requirements. It is also assumed that 96 percent of NTNCs use ground water sources, and 4 percent use surface water.)
- CWSs may incur additional costs if they find exceedances more frequently at the revised MCL.

The cost of monitoring includes preparing and analyzing the sample. Collecting the sample, arranging for delivery to the laboratory, and reviewing the results of the analysis is assumed to require one hour of the system operator’s time (at an estimated cost of \$28 per hour). EPA has assumed that all systems are equipped to collect samples. Therefore, no additional costs are assumed for installation of taps, re-piping of wells or other investments to permit sampling. EPA has assumed that systems will utilize one of two laboratory methods: (1) stabilized temperature platform graphite furnace atomic absorption (STP-GFAA) or (2) graphite furnace atomic absorption (GFAA). Both techniques cost \$40 per sample.

Total net monitoring costs were estimated over a 20-year period at discount rates of three and seven percent. The net costs are equal to the difference between the cost of the revised monitoring requirements and the cost of the current monitoring requirements. Cost and hour burden to the system and the State are listed below in Exhibit 6-3. The cost of routine monitoring, triggered monitoring, waiver application and public notification are all included in the total system costs. Miscellaneous costs related to sending samples to be analyzed and sending public notification to customers are also included in the system cost.

**Exhibit 6-3  
Unit Resources Required for Monitoring, Implementation, and Administration\***

<b>System Size Category</b>	<b>&lt; 10,000 people</b>		<b>&gt; 10,000 people</b>	
<b>State Activity</b>	<b>Hours</b>	<b>Rate</b>	<b>Hours</b>	<b>Rate</b>
Review a waiver application	8	\$41.47	8	\$41.47
Record monitoring of a sample result	1	\$41.47	1	\$41.47
Issue a single violation letter	4	\$41.47	4	\$41.47
Review a single permit	16	\$41.47	32	\$41.47
	<b>&lt;3,000 people</b>		<b>&gt;3,300 people</b>	
<b>System Activity</b>	<b>Hours</b>	<b>Rate</b>	<b>Hours</b>	<b>Rate</b>
Apply for a waiver	16	\$15.03	16	\$29.03
Take a sample	1	\$15.03	1	\$29.03
Report a sample	1	\$15.03	1	\$29.03
Prepare and Send Public Notification	8	\$15.03	8	\$29.03

Source: *Information Collection Request for the Public Water System Supervision Program.*

\*Estimates are provided in May 1999 dollars, updated from 1997 dollars using the CPI-U for all items.

States will also be required to spend time responding to systems that report MCL exceedances or systems that request a waiver (Exhibit 6-3). Hour burdens for States to review waiver applications, record monitoring of a sample, and issue a violation letter are the same for small and large systems. The number of hours required to review a single permit is twice as large for systems serving more than 10,000 people than for systems serving less than 10,000 people. The unit cost for all activities is consistent across all activities and size categories (\$41.47 per hour) (EPA, 1997).

Exhibit 6-3 also shows that the number of hours required at the system level to perform the responsibilities related to monitoring is the same for systems serving fewer than 3,300 people and systems serving more than 3,300 people. However, the hourly rate for systems serving more than 3,300 people (\$29.03) is almost double the rate for systems serving fewer than 3,300 people (\$15.03).

During the first year of implementation all systems will incur costs related to routine monitoring. In addition, systems in violation will incur costs related to triggered quarterly monitoring. Under the revised rule, a percentage of the systems will have monitoring waivers in subsequent years when monitoring is otherwise required. Monitoring waivers are not granted under the existing rule; therefore, the number of systems required to conduct routine monitoring under the revised rule is less than that under the existing rule. For this reason, the annual net cost of monitoring between the revised rule and the existing rule may be negative, or less expensive, after the initial year of implementation. The inputs and methodology associated with this analysis are presented in detail in the *Information Collection Request for the Proposed Arsenic in Drinking Water Rule*.

### **Administrative Costs**

States and systems will incur administrative costs to implement the revised arsenic program under the Arsenic Rule. States and systems will need to allocate time for their staff to establish and maintain the programs necessary to comply with the revised arsenic standard and the new monitoring requirements. Exhibit 6-4(a) lists the one-time State activities involved in starting up the program following promulgation of the rule. For example, start-up activities may include developing and adopting State regulations that meet the new Federal arsenic requirements. Resources are estimated in terms of full-time equivalents (FTEs), which EPA has assumed to cost \$64,480 per FTE, including overhead and fringe. Systems also have start-up costs for reviewing the rule and training operators. Exhibit 6-4(b) lists the one-time system start-up activities. The two primary activities that systems will perform to comply with the revised arsenic rule are reading and understanding the rule and operator training. For all systems the estimated time required to review the rule is eight hours. Systems serving fewer than 10,000 people require an estimated time of 16 hours to train operators; the estimated time for systems serving more than 10,000 people is 32 hours. The rate for all start-up activities for systems serving fewer than 10,000 people is \$15.03 per hour and \$29.03 per hour for systems serving more than 10,000 people.

**Exhibit 6-4(a)**  
**Estimated One-Time State Resources Required for Initiation of the Arsenic Rule**

Administrative Activity	Estimated State Resources (FTE)	Estimated Cost
<b>One Time Start-up Activities</b>		
Regulation Adoption and Program Development	0.2	\$12,900
System Training and Technical Assistance (CWS)	0.5	\$32,240
System Training and Technical Assistance (NTNC)	0.5	\$32,240
Staff Training (CWS)	0.12	\$7,740
<b>National Total*</b>	73.92	<b>\$4,767,840</b>

\*National totals include estimates for all States, territories, and Tribes.

### Exhibit 6-4(b)

#### Estimated One-Time System Resources Required for Initiation of the Arsenic Rule

System Size Category	< 10,000 people		> 10,000 people	
	Hours	Rate	Hours	Rate
Reading and Understanding Rule	8	\$15.03	8	\$29.03
Operator Training	16	\$15.03	32	\$29.03

Source: *Information Collection Request for the Public Water System Supervision Program.*

#### 6.2.4 Predicting Compliance Decisions (Compliance Decision Tree)

There is substantial variability in how systems will elect to comply with the Arsenic Rule. Choices of compliance method will vary depending on baseline source water arsenic concentrations, system size and location, types of treatment currently in place, and availability of alternative sources. In addition, the source water pH, total dissolved solids, sulfides, and other salts can change the effectiveness of technologies in removing arsenic.

The EA reflects this variability by predicting a range of compliance responses for different system types and sizes. The compliance decision tree specifies the percentage of systems in different categories that will choose specific compliance options, given the removal required by the MCL option and the baseline occurrence of arsenic in source water. For example, for a target MCL of 10 µg/L, the decision tree specifies the probability of different compliance choices for systems with different baseline influent concentrations (e.g., <10 µg/L, 10-20 µg/L, etc.), different sizes (e.g., population < or > 1,000), different sources (ground water or surface water), and different existing treatment facilities. The compliance choices are defined by a treatment technology and (where relevant) a waste disposal option, and/or pre-treatment technology.

EPA presented a draft of the compliance decision tree at an American Water Works Association (AWWA) technical workgroup meeting in February 1999 and made revisions based on the comments received at that meeting. The final compliance decision tree, as well as a discussion of the assumptions made during its development, is provided in Appendix A (“Cost Analysis Appendix”) by system size and type.

#### 6.2.5 Calculating Costs

Different methods were used to assess costs for three different categories of systems. A Monte-Carlo simulation model (SafeWaterXL) was used to estimate costs for community water systems, excluding the largest CWSs. A deterministic spreadsheet analysis was performed for NTNC water systems, while a separate case-by-case analysis were performed for the very large systems (serving more than one million people) that are expected to exceed one or more MCL options in the baseline. The costs for the three system categories were then summed to calculate total national costs.

The methodology for calculating the costs for each of these system categories is described separately below, beginning with a description of the SafeWaterXL model. In addition, a detailed description of the SafeWaterXL model is provided in Appendix C.

### **CWS Costs**

The national cost of compliance across CWSs (except those serving over one million people) was estimated using SafeWaterXL, a Monte-Carlo simulation model developed in Microsoft® Excel using the Crystal Ball® Monte-Carlo simulation add-in. SafeWaterXL forecasts a distribution of costs around the mean compliance cost expected for each system size category. The Monte-Carlo model provides the flexibility to incorporate as many data as are available, while maintaining uncertainty bounds to prevent any individual input from skewing the results. When sample data are not available as single point estimates, this technique is an invaluable tool.

Historically, most drinking water regulatory impact analyses used point estimates to describe the average system-level costs. By using SafeWaterXL, this analysis contains more detailed descriptions of system-level cost. SafeWater XL describes system-level costs in terms of a distribution. From the distribution, mean and median costs are available, as well as percentile costs.

### *Model Structure*

SafeWaterXL determines regulatory compliance costs for individual systems and subsequently calculates a national average. To do so, each system is assigned a random concentration from an occurrence distribution. This system concentration is distributed across the number of sites of possible contamination for that system. The average number of sites per system is determined based on the distribution of system intake sites for the size category as estimated from the CWSS. However, SafeWaterXL does not assume that all sites are equally likely to exceed the MCL standard. The likelihood of contamination is determined on a site-by-site basis. The sum of the mean arsenic concentration of all sites within a system must equal the mean arsenic concentration of the system. Given this upper bound, each site is assigned a concentration based on the assumed relative standard deviation around the mean system occurrence.

The model then compares the concentration at each site to the revised MCL standard; no costs are incurred for those sites whose concentrations fall below the specified MCL. If the site is determined to be in violation of the MCL, then SafeWaterXL calculates the percent reduction in arsenic concentration required to reduce the site concentration to 80 percent of the MCL standard (this is a safety factor that includes a 20 percent excess removal to account for system over-design).<sup>1</sup> A treatment train is then assigned to the site based on a decision tree for the size and type of the system. The decision tree and the selected treatment train reflect the removal efficiencies of the chosen technology. For example, a technology is chosen based on matching

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<sup>1</sup>No blending is assumed for the POU technologies.

the removal efficiencies and the percentage removal required at the site (SafeWaterXL identifies three categories of required removal: < 50 percent, 50-90 percent, > 90 percent).<sup>2</sup>

In this manner, capital and O&M costs are calculated at the site level for the selected treatment train. The system's cost of compliance is then determined by summing across the treating sites. For each system in SDWIS in which a violation is expected, a cost is calculated with this method, thereby creating an estimate of national compliance costs. Since household costs are also calculated for each system, a similar distribution of the cost of compliance at the household level is also created.

In order to develop more detailed results, the compliance decision tree is employed at the site level, so that only those sites requiring treatment would incur costs. The resulting total national compliance cost is expected to be a truer representation of the impact of the Arsenic Rule on systems. The sections below will describe the data needed to develop cost estimates for the entire universe of systems affected by the Arsenic Rule. After the discussion of data requirements, the SafeWaterXL model is described as it is used for this rule.

### *Model Inputs*

*Number of Systems:* The universe of public and private ground and surface water systems is taken from the Safe Drinking Water Information System (SDWIS), EPA's national regulatory database for the drinking water program. Based on data extracted in December 1998, a total of 54,352 CWSs and 20,255 NTNCs are subject to the new requirements under the Arsenic Rule. It is necessary to compile this data by system size, water source, and ownership, as costs may vary by these characteristics. SafeWaterXL calculates costs for public and private systems (the latter also includes "other" or "ancillary" systems), and surface and ground water systems. A summary table of this breakdown is provided in Chapter 4, "Baseline Analysis."

*Entry Points per System:* SafeWaterXL estimates each system's cost of compliance at the treatment site level. This modeling approach is used because a system may include more than one treatment site. Entry points are used as a proxy for potential or actual points of treatment. For example, a given water system may have three entry points: one entry point that currently treats, while two may not have treatment in place. Data on the distribution of the number of system entry points for each size category and type were extracted from the Community Water Supply Survey (CWSS). Linear interpolation was used to estimate values for the number of sites in cases where there were no survey data (see Chapter 4, "Baseline Analysis"). SafeWater XL uses this modified distribution of entry points for each system size and source water category.

*Population Served by System:* A system's size is determined by the number of people served by that system. These numbers were extracted from the SDWIS database (see Chapter 4, "Baseline Analysis"). Systems are grouped into eight categories to help identify systems with related characteristics so that any data or resources may be pooled during analysis.

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<sup>2</sup>The > 90 percent removal efficiency category is not relevant under the revised MCL of 10 µg/L.

SafeWaterXL recognizes the following size categories:

- < 100
- 101-500
- 501-1000
- 1,001-3,300
- 3,301-10,000
- 10,001-50,000
- 50,001-100,000
- 100,001-1,000,000.

*Flow Rate Parameters:* A system's size is further defined by its flow, which is calculated as a power law function of the population served. These functions were derived by EPA, and their derivation can be found in the Model Systems report (EPA, 1999b). The equation form is shown below.

$$\text{Average Flow} = a_A 4(\text{Population})^{b_A}$$

$$\text{Design Flow} = \max \left\{ \begin{array}{l} 2 \text{ Average Flow} \\ a_D 4(\text{Population})^{b_D} \end{array} \right.$$

*Where:*  $a_A, b_A, a_D, b_D$  = the regression parameters derived for flow vs. population  
 Population = the population served by the appropriate system type and primary source.

The regression parameters used in the cost model are provided in Exhibit 6-5. Values are provided for design and average flow for public and private ground water and surface water supplies. SafeWaterXL divides the resulting system design flow and average daily flow (kgpd) equally among all entry points. Treatment costs are calculated only at the sites that exceed the MCL and only for the minimum portion of flow that must be treated in order to achieve the new concentration standard, a process referred to as “blending.”

**Exhibit 6-5**  
**Flow Regression Parameters**  
**by Water Source and System Ownership**

	Average Flow		Design Flow	
	a	b	a	b
<b>Ground Water</b>				
Public	0.08558	1.05840	0.54992	0.95538
Private	0.06670	1.06280	0.41682	0.96078
Public-Purch	0.04692	1.10190	0.31910	0.99460
Private-Purch	0.05004	1.08340	0.32150	0.97940
<b>Surface Water</b>				
Public	0.14004	0.99703	0.59028	0.94573
Private	0.09036	1.03340	0.35674	0.96188
Public-Purch	0.04692	1.11020	0.20920	1.04520
Private-Purch	0.05004	1.08340	0.20580	1.00840

*Average Consumption per Household:* Household costs depend on the average annual consumption per residential connection. These mean estimates are provided in Chapter 4, “Baseline Analysis.” Depending on the system’s characteristics, SafeWaterXL multiplies the appropriate mean consumption per year (kgal) with the system’s computed cost per thousand gallons to arrive at the average annual cost of compliance per household for a community water system.

*Mean System Occurrence:* Arsenic occurrence data are based on EPA’s *Arsenic Occurrence in Public Drinking Water Supplies* report (EPA, 2000) and are represented by a lognormal distribution. Baseline occurrence is distinguished between ground and surface water systems and is provided in Chapter 4 (“Baseline Analysis”) as a lognormal distribution. The distribution is truncated at 50 µg/L, the current arsenic standard, because it is assumed that all arsenic reductions attributable to the new standard start at the previous standard (i.e., all systems are currently in compliance with the current standard).

For use in the SafeWaterXL model, EPA performed a regression analysis that weighted actual occurrence data by National Arsenic Occurrence Survey region. The analysis resulted in the distribution of ground and surface water systems exceeding arsenic concentrations greater than 3, 5, 10, and 20 µg/L as presented in Exhibit 6-6.

**Exhibit 6-6  
Arsenic Occurrence Distribution  
(Log-Normal Regression Results)**

Source	% of systems greater than (µg/L)			
	3	5	10	20
<b>GW</b>	19.7	12.0	5.3	2.0
<b>SW</b>	5.6	3.0	1.12	0.37

For ground water systems, the percentages displayed in Exhibit 6-6 above were based on a lognormal distribution with a mean of -0.25071 and a log standard deviation of 1.58257. Among surface water systems, the percentages were based on a lognormal distribution with mean -1.67805 and a log standard deviation of 1.7425.

*Relative Intra-System Standard Deviation of Arsenic Concentrations:* The relative intra-system standard deviation of the site concentrations within a system is calculated using data from a 25 State arsenic occurrence study (EPA, 2000b). SafeWaterXL uses a default value of 0.64. This standard deviation is applied to the mean system concentration to generate individual entry points concentrations within the system.

*Compliance Decision Trees:* The decision trees represent EPA’s best estimate of the treatment train technologies system operators will choose to achieve a particular percentage reduction in arsenic concentration. Decision trees are specific to the system’s size categories and source water. These are provided in Appendix A.

*Removal Efficiencies, Treatment Target, and Blending:* Each treatment train is associated with an arsenic removal efficiency that is assumed to be constant across system types. The removal efficiencies for the 13 treatment trains available under the Arsenic Rule were presented in Exhibit 6-1. SafeWaterXL employs these efficiencies, using the blending principle, to determine the amount of flow that requires treatment in order for the entry point to meet the treatment target. Blending uses the entry point concentration and treatment train removal efficiency to determine the fraction of flow required to obtain the treatment target. The treatment target is set at 80 percent of the MCL and represents the level to which systems will be over-designed to ensure compliance with the MCL.

SafeWaterXL employs the blending principle through the following equation at the entry point level:

$$\text{Fraction of flow treated} = \left\{ \frac{\left( \frac{\text{TreatmentTarget}}{\text{SiteConcentration}} - 1 \right) \cdot 4(\% \text{ Site Flow})}{\% \text{ Removal Efficiency}} \right.$$

Where: Treatment Target = the target MCL with 80 percent safety factor  
 Site Concentration = arsenic concentration at the site  
 % Removal Efficiency = percent removal efficiency of treatment train chosen  
 % Site Flow = percent of total flow at that site.

Note that the blending technique is used only for those systems expected to require less than 90 percent removal in order to achieve compliance with the new MCL standard. In addition, SafeWaterXL does not employ this technique for those systems that select treatment trains involving POU devices.

*Equipment Life, Discount Rate and Capitalization Rates:* System and State implementation costs are tracked for a 20-year period. This time frame was selected because water systems often finance their capital improvements over a 20-year period. This period of analysis may result in an overestimate of annualized costs because many types of equipment last longer than 20 years.

Two different adjustments are made in this analysis in order to render future costs comparable with current costs, reflecting the fact that a cost outlay today is a greater burden than an equivalent cost outlay sometime in the future. The first adjustment is made when the cost estimates that are derived are being used as an input in benefit-cost analysis. In this instance, costs are annualized using a social discount rate so that the costs of each regulatory option can be directly compared with the annual benefits of the corresponding regulatory option. Annualization is the same process as calculating a mortgage payment; the result is a constant annual cost to compare with constant annual benefits.

The choice of an appropriate social discount rate has been, and continues to be, a very complex and controversial issue among economists and policy makers alike. Therefore, the Agency compares costs and benefits using two alternative social discount rates, in part to determine the effect the choice of social discount rate has on the analysis. The annualized costs of each regulatory option are calculated and displayed using both a seven percent discount rate, required by the Office of Management and Budget (OMB), and a three percent discount rate, which the Agency believes more closely approximates the true social discount rate.

The second adjustment is made when the cost estimates that are derived are being used as an input into an economic impact analysis, such as an affordability analysis or an analysis of system-level costs or household-level costs. In these cases, rather than use a social discount rate when determining the annualized costs, an actual cost-of-capital rate is used instead. This rate should reflect the true after-tax cost of capital water systems face, net of any government grants or subsidies. The cost of capital rates used in this analysis are shown in Exhibit 6-7.

**Exhibit 6-7**  
**Summary of Recommended Cost of Capital Estimates**  
**(as of March 1998)**

Ownership Type	Size Category	Estimated After-Tax Cost of Capital
<b>NON-SMALL</b>		
Investor owned	10,001-50,000	5.26%
	>50,000	5.94%
Publicly owned	10,001-50,000	5.26%
	>50,000	5.23%
<b>SMALL</b>		
Private	1-500	4.17%
	501-10,000	4.17%
Public	1-500	5.10%
	501-10,000	5.20%
Source: <i>Development of Cost of Capital Estimates for Public Water Systems</i> (Draft Final Report). Prepared for U.S. EPA by Apogee/Hagler Bailly, Inc. under subcontract to International Consultants, Inc. June 1998.		

**NTNC Costs**

The cost for NTNCs is estimated using the mean values for system population for each system service category, as shown in Chapter 4. As with the CWSs, cost is annualized over a 20-year period, at discount rates of three and seven percent. Assumptions regarding the monitoring schedule correspond to the monitoring schedule for small ground water systems, including hour burdens and hourly labor rates. The remaining assumptions required for determining cost are described below.

*Number of Systems, Sites per System, and the Population Served:* The non-transient non-community water supply treatment decisions are modeled similarly to those for community water supplies. The number of non-transient non-community water supplies is taken from EPA's SDWIS, and include those systems as described in *Geometries and Characteristics of Public Water Supplies* (see Exhibit 6-8). For each service area type, the report lists the number of systems and the average population served. The non-food manufacturing service area combines 16 categories that were listed separately in the report. For this service area, the number of systems is the sum of the 16 categories, and the average population served is the mean of the individual populations weighted by the number of corresponding systems. Each of these systems has only a single site.

*System Flows and Treatment Choices:* For each service area, both design and average flows have been derived by the Agency using literature values and best engineering judgment. There are no primary survey data for non-community water systems that are equivalent to the CWSS-provided data for the community water system flow calculations (Smith, 1999). The design flow is used to calculate the treatment capital costs, while the average flow is used in the operating and maintenance cost equations.

For the non-transient non-community water supplies, one of two treatment technologies was chosen based on the level of the design flow. For service areas with design flows less than 2,000 gallons per day, POE activated alumina is used; for all others, centralized activated alumina is chosen (Kapadia, 1999a). Both treatment trains include pre-oxidation, and the centralized activated alumina also includes non-hazardous landfilling of the spent media (Kapadia, 1999a).

*Mean Arsenic Occurrence:* The arsenic occurrence distribution used for ground water community water supplies is also used for non-transient non-community water supplies. The number of systems exceeding the MCL for each service area was calculated from the percent of the distribution between the MCL and 100 µg/L. For this analysis, 100 µg/L was chosen as the upper concentration limit because the non-transient non-community supplies have not been previously regulated, and occurrence values above the 50 µg/L regulatory level are possible.

*Removal Efficiencies, Treatment Target, and Blending:* The removal efficiency associated with both POE activated alumina and centralized activated alumina is 95 percent. The NTNC model uses this efficiency with the blending principle in the case of centralized activated alumina to determine the amount of flow that requires treatment in order for the site to meet the treatment target. The treatment target is set at 80 percent of the MCL and represents the level to which systems will be over-designed to ensure compliance with the MCL. For POE activated alumina systems, all the flow is treated, which may result in finished water below the treatment target concentration.

*Equipment Life, Discount Rate, and Capitalization Rates:* As with the community water supplies, the system implementation costs are tracked for a 20-year period. For the two service areas using POE activated alumina, construction and forest service, the equipment is assumed to last ten years with purchases in year zero and year ten. For the centralized activated alumina the equipment is estimated to last 20 years. The cost estimates are annualized in the same manner as those for the community water supplies.

**Exhibit 6-8  
Non-Transient Non-Community System Characteristics and  
Compliance Decision Tree**

Service Area Type	SYSTEM CHARACTERISTICS				DECISION TREE	
	Number of Systems	Average Population Served Per System	Design Flow (mgd)	Average Daily Flow (mgd)	Activated Alumina Point of Entry	Centralized Activated Alumina
Daycare Centers	809	76	0.0051	0.0011		↓
Highway Rest Areas	15	407	0.0089	0.0020		↓
Hotels/Motels	351	133	0.0189	0.0045		↓
Interstate Carriers	287	123	0.0029	0.0006		↓
Medical Facilities	367	393	0.1166	0.0339		↓
Mobile Home Parks	104	185	0.0262	0.0065		↓
Restaurants	418	370	0.0039	0.0008		↓
Schools	8414	358	0.0333	0.0085		↓
Service Stations	53	230	0.0051	0.0011		↓
Summer Camps	46	146	0.0218	0.0053		↓
Water Wholesalers	266	173	0.1637	0.0494		↓
Agricultural Products/Services	368	76	0.0199	0.0048		↓
Airparks	101	60	0.0026	0.0005		↓
Construction	99	53	0.0009	0.0002	↓	
Churches	230	50	0.0053	0.0011		↓
Campgrounds/RV Parks	123	160	0.0214	0.0052		↓
Fire Departments	41	98	0.0186	0.0045		↓
Federal Parks	20	39	0.0065	0.0014		↓
Forest Service	107	42	0.0014	0.0002	↓	
Golf and Country Clubs	116	101	0.0118	0.0027		↓
Landfills	78	44	0.0053	0.0011		↓
Mining	119	113	0.0123	0.0028		↓
Amusement Parks	159	418	0.0171	0.0041		↓
Military Bases	95	395	0.0695	0.0192		↓
Migrant Labor Camps	33	63	0.0102	0.0023		↓
Misc. Recreation Services	259	87	0.0025	0.0005		↓
Nursing Homes	130	107	0.0411	0.0107		↓
Office Parks	950	136	0.0077	0.0017		↓
Prisons	67	1820	0.5322	0.1820		↓
Retailers (Non-food related)	695	174	0.0038	0.0008		↓
Retailers (Food related)	142	322	0.0058	0.0012		↓
State Parks	83	165	0.0048	0.0010		↓
Non-Water Utilities	497	170	0.0133	0.0031		↓
Manufacturing: Food	768	372	0.0454	0.0120		↓
Manufacturing: Non-Food	3845	168	0.0157	0.0038		↓
<b>TOTAL</b>	<b>20,255</b>					

Source: Geometries and Characteristics of Public Water Systems, EPA, May 1999.

## **Very Large CWS Costs**

EPA evaluated the regulatory costs of compliance for very large systems that will be subject to the new Arsenic Rule. The nation's 25 largest drinking water systems (i.e., those serving one million people or more) supply approximately 38 million people and generally account for about 15 to 20 percent of all compliance-related costs. Accurately determining these costs for future regulations is critical. As a result, EPA has developed compliance cost estimates for the arsenic and radon regulations for each individual system that serves more than one million persons. These cost estimates help EPA to more accurately assess the cost impacts and benefits of the Arsenic Rule. The estimates also help the Agency identify lower cost regulatory options and better understand current water systems' capabilities and constraints.

The system costs were calculated for the 24 public water systems that serve a retail population of more than one million persons and one public water system that serves a wholesale population of 16 million persons. The following are distinguishing characteristics of these very large systems:

- (1) A large number of entry points from diverse sources;
- (2) Mixed sources (i.e., ground and surface water);
- (3) Occurrence not conducive to mathematical modeling;
- (4) Significant levels of wholesaling;
- (5) Sophisticated in-place treatment;
- (6) Retrofit costs dramatically influenced by site-specific factors; and
- (7) Large amounts of waste management and disposal, which can contribute substantial costs.

Generic models cannot incorporate all of these considerations; therefore, in-depth characterizations and cost analyses were developed using several existing databases and surveys.

The profile for each system contains information such as design and average daily flows, treatment facility diagrams, chemical feed processes, water quality parameters, system layouts, and intake and aquifer locations. System and treatment data were obtained from the following sources:

- (1) The Information Collection Rule (1997);
- (2) The Community Water Supply Survey (1995);
- (3) The Association of Metropolitan Water Agencies Survey (1998);
- (4) The Safe Drinking Water Information System (SDWIS); and
- (5) The American Water Works Association WATERSTATS Survey (1997).

While these sources contained much of the information necessary to perform cost analyses, the Agency was still missing some of the detailed arsenic occurrence data in these large water systems. Where major gaps existed, especially in ground water systems, occurrence data obtained from the States of Texas, California, and Arizona; the Metropolitan Water District of Southern California Arsenic Study (1993); the National Inorganic and Radionuclides Study (EPA, 1984); and utility data were used. Based on data from the studies, detailed costs estimates were derived for each of the very large water systems.

Cost estimates were generated for each system at several MCL options. The total capital costs and operational and maintenance (O&M) costs were calculated using the profile information gathered on each system, conceptual designs (i.e., vendor estimates and *RS Means*), and modified EPA cost models (i.e., Water and WaterCost models). The models were modified based on the general cost assumptions developed in the Phase I Water Treatment Cost Upgrades (EPA, 1998c).

EPA consulted with the system operators to determine how each system would comply with various MCL options and to assess the costs of their compliance responses. Preliminary cost estimates were sent to all of the systems for their review. Approximately 30 percent of the systems responded by submitting revised estimates and/or detailed arsenic occurrence data. Based on the information received, EPA revised the cost estimates for those systems. EPA developed cost estimates for three very large systems that are expected to have arsenic levels above the revised MCL. These systems are located in Houston, TX, Phoenix, AZ, and Los Angeles, CA. This analysis resulted in the estimated costs listed in Exhibit 6-9.

**Exhibit 6-9**  
**Annual Treatment Costs for Three Large CWSs Expected to**  
**Undertake or Modify Treatment Practice to Comply with the Arsenic Rule**  
**(\$ millions)**

Large CWSs	Population Served	MCL ( $\mu\text{g/L}$ )			
		3	5	10	20
<b>Phoenix, AZ</b>	1,360,751				
Annual cost (3%)		\$ 11.6	\$ 5.5	\$ 2.2	\$ 0.0
Annual cost (7%)		\$ 13.2	\$ 6.3	\$ 2.5	\$ 0.0
<b>Houston, TX</b>	2,216,830				
Annual cost (3%)		\$ 15.0	\$ 2.7	\$ 0.9	\$ 0.5
Annual cost (7%)		\$ 16.0	\$ 2.9	\$ 1.0	\$ 0.5
<b>Los Angeles, CA</b>	3,700,000				
Annual cost (3%)		\$ 1.8	\$ 1.8	\$ -	\$ -
Annual cost (7%)		\$ 1.8	\$ 1.8	\$ -	\$ -

\* Exhibit updated on December 28, 2000 to reflect minor changes in cost estimates which have not been incorporated into subsequent exhibits. The impact is a \$0.07 million overestimation of national costs (less than 0.5% of total national costs)

## 6.3 Results

This section presents the results of the national cost analysis. Unless otherwise specified, national costs are presented in May 1999 dollars throughout this chapter.

### 6.3.1 National Costs

Exhibit 6-10 shows the total national cost breakdown across the four MCL options for the Arsenic Rule. The system and state cost components of the total annual compliance costs are

presented at discount rates of three and seven percent. Expected system costs include treatment costs, monitoring costs, and administrative costs of compliance. State costs include monitoring and administrative costs of implementation. These cost components are also displayed.

CWS costs are approximately \$668.0 million at the 3 µg/L MCL, \$396.0 million at the 5 µg/L MCL, \$171.4 million at the 10 µg/L MCL, and \$62.4 million at the 20 µg/L MCL (at a three percent discount rate). State costs associated with CWS administration, at a three percent discount rate, are approximately \$1.4 million at the 3 µg/L MCL, \$1.1 million at the 5 µg/L MCL, \$0.9 million at the 10 µg/L MCL, and \$0.7 million at the 20 µg/L MCL.

The cost to NTNCs ranges from \$28 million at the 3 µg/L MCL, \$16 million at the 5 µg/L MCL, \$7.9 million at the 10 µg/L MCL, and \$3.5 million at the 20 µg/L MCL (at a three percent discount rate). State costs associated with NTNC administration, at a three percent discount rate, are approximately \$0.1 million for each MCL.

**Exhibit 6-10  
Annual National System and State Compliance Costs  
(\$ millions)**

Discount Rate	CWS		NTNC		TOTAL	
	3%	7%	3%	7%	3%	7%
<b>MCL = 3 µg/L</b>						
System Costs						
Treatment	\$665.9	\$756.5	\$27.2	\$29.6	\$693.1	\$786.0
Monitoring/ Administrative	\$2.2	\$3.0	\$1.0	\$1.4	\$3.2	\$4.4
State Costs	\$1.4	\$1.6	\$0.1	\$0.2	\$1.5	\$1.7
TOTAL COST	\$669.4	\$761.0	\$28.3	\$31.1	\$697.8	\$792.1
<b>MCL = 5 µg/L</b>						
System Costs						
Treatment	\$394.4	\$448.5	\$16.3	\$17.6	\$410.6	\$466.1
Monitoring/ Administrative	\$2.0	\$2.8	\$1.0	\$1.3	\$2.9	\$4.1
State Costs	\$1.1	\$1.3	\$0.1	\$0.2	\$1.2	\$1.4
TOTAL COST	\$397.5	\$452.5	\$17.3	\$19.1	\$414.8	\$471.7
<b>MCL = 10 µg/L</b>						
System Costs						
Treatment	\$169.6	\$193.0	\$7.0	\$7.6	\$176.7	\$200.6
Monitoring/ Administrative	\$1.8	\$2.5	\$0.9	\$1.3	\$2.7	\$3.8
State Costs	\$0.9	\$1.0	\$0.1	\$0.2	\$1.0	\$1.2
TOTAL COST	\$172.3	\$196.6	\$8.1	\$9.1	\$180.4	\$205.6
<b>MCL = 20 µg/L</b>						
System Costs						
Treatment	\$60.7	\$69.0	\$2.6	\$2.8	\$63.3	\$71.8
Monitoring/ Administrative	\$1.7	\$2.4	\$0.9	\$1.3	\$2.6	\$3.7
State Costs	\$0.7	\$0.8	\$0.1	\$0.2	\$0.9	\$1.0
TOTAL COST	\$63.2	\$72.3	\$3.6	\$4.2	\$66.8	\$76.5

### 6.3.2 Costs by System Size and Type

This section presents the overall national compliance costs for water systems and for states at three and seven percent discount rates. Exhibit 6-11 shows a detailed breakout of national treatment costs by CWS size category for the various MCLs.

Exhibits 6-12 through 6-15 show the national treatment costs for NTNC systems by NTNC system service type for each MCL.

**Exhibit 6-11**  
**Total Annual CWS Treatment Costs Across MCL Options**  
**by System Size (\$ millions)**

System Size	MCL (µg/L)			
	3	5	10	20
<b>3% Discount Rate</b>				
<100	\$ 19.8	\$ 12.3	\$ 5.5	\$ 2.1
101-500	\$ 42.6	\$ 25.7	\$ 11.5	\$ 4.3
501-1,000	\$ 25.5	\$ 15.2	\$ 6.7	\$ 2.5
1001-3300	\$ 83.8	\$ 50.5	\$ 22.0	\$ 8.1
3,301-10,000	\$ 95.1	\$ 55.9	\$ 24.3	\$ 9.0
10,001-50,000	\$ 179.1	\$ 108.7	\$ 47.0	\$ 16.7
50,001-100,000	\$ 66.0	\$ 39.0	\$ 16.7	\$ 6.2
100,001-1,000,000	\$ 124.3	\$ 75.2	\$ 32.3	\$ 11.3
>1,000,000	\$ 29.7	\$ 11.8	\$ 3.8	\$ 0.6
Total	\$ 665.9	\$ 394.4	\$ 169.6	\$ 60.7
<b>7% Discount Rate</b>				
<100	\$ 21.3	\$ 13.2	\$ 5.9	\$ 2.3
101-500	\$ 46.4	\$ 28.0	\$ 12.5	\$ 4.6
501-1,000	\$ 28.9	\$ 17.2	\$ 7.6	\$ 2.8
1001-3300	\$ 97.4	\$ 58.8	\$ 25.6	\$ 9.4
3,301-10,000	\$ 109.2	\$ 64.2	\$ 27.9	\$ 10.3
10,001-50,000	\$ 205.4	\$ 124.7	\$ 53.9	\$ 19.2
50,001-100,000	\$ 75.0	\$ 44.3	\$ 19.0	\$ 7.0
100,001-1,000,000	\$ 140.5	\$ 85.0	\$ 36.5	\$ 12.7
>1,000,000	\$ 32.5	\$ 13.0	\$ 4.3	\$ 0.6
Total	\$ 756.5	\$ 448.5	\$ 193.0	\$ 69.0

**Exhibit 6-12**  
**Total Annual NTNC Treatment Costs at MCL 3 µg/L by System Service Type**  
**(3% Discount Rate)**

Service Area Type	# of Systems Above the MCL	Average Population Served Per System	Average Annual System Cost	Annual National Costs
Daycare Centers	159	76	\$5,217	\$831,099
Highway Rest Areas	3	407	\$5,466	\$16,144
Hotels/Motels	69	133	\$6,153	\$425,252
Interstate Carriers	57	123	\$5,074	\$286,723
Medical Facilities	72	393	\$13,540	\$978,452
Mobile Home Parks	20	185	\$6,666	\$136,496
Restaurants	82	370	\$5,140	\$423,058
Schools	1,657	358	\$7,177	\$11,890,922
Service Stations	10	230	\$5,217	\$54,445
Summer Camps	9	146	\$6,353	\$57,538
Water Wholesalers	52	173	\$16,456	\$861,907
Agricultural Products/Services	72	76	\$6,221	\$450,734
Airparks	20	60	\$5,059	\$100,600
Construction	19	53	\$4,733	\$92,258
Churches	45	50	\$5,229	\$236,789
Campgrounds/RV Parks	24	160	\$6,329	\$153,287
Fire Departments	8	98	\$6,132	\$49,505
Federal Parks	4	39	\$5,309	\$20,908
Forest Service	21	42	\$4,783	\$100,771
Golf and Country Clubs	23	101	\$5,661	\$129,308
Landfills	15	44	\$5,226	\$80,268
Mining	23	113	\$5,697	\$133,490
Amusement Parks	31	418	\$6,025	\$188,625
Military Bases	19	395	\$9,883	\$184,863
Migrant Labor Camps	6	63	\$5,554	\$36,090
Misc. Recreation Services	51	87	\$5,050	\$257,531
Nursing Homes	26	107	\$7,748	\$198,316
Office Parks	187	136	\$5,386	\$1,007,456
Prisons	13	1,820	\$45,861	\$605,012
Retailers (Non-food related)	137	174	\$5,133	\$702,366
Retailers (Food related)	28	322	\$5,261	\$147,101
State Parks	16	165	\$5,199	\$84,966
Non-Water Utilities	98	170	\$5,763	\$563,970
Manufacturing: Food	151	372	\$8,066	\$1,219,753
Manufacturing: Non-Food	757	168	\$5,944	\$4,500,232
<b>TOTAL</b>	<b>3,988</b>			<b>\$27,206,235</b>

**Exhibit 6-13**  
**Total Annual NTNC Treatment Costs at MCL 5 µg/L by System Service Type**  
**(3% Discount Rate)**

Service Area Type	# of Systems Above the MCL	Average Population Served Per System	Average Annual System Cost	Annual National Costs
Daycare Centers	97	76	\$5,196	\$504,051
Highway Rest Areas	2	407	\$5,428	\$9,763
Hotels/Motels	42	133	\$6,069	\$255,418
Interstate Carriers	34	123	\$5,062	\$174,207
Medical Facilities	44	393	\$12,959	\$570,242
Mobile Home Parks	12	185	\$6,547	\$81,640
Restaurants	50	370	\$5,124	\$256,824
Schools	1,009	358	\$7,024	\$7,086,564
Service Stations	6	230	\$5,196	\$33,020
Summer Camps	6	146	\$6,255	\$34,500
Water Wholesalers	32	173	\$15,679	\$500,052
Agricultural Products/Services	44	76	\$6,132	\$270,563
Airparks	12	60	\$5,048	\$61,134
Construction	12	53	\$4,733	\$56,180
Churches	28	50	\$5,207	\$143,590
Campgrounds/RV Parks	15	160	\$6,233	\$91,929
Fire Departments	5	98	\$6,050	\$29,740
Federal Parks	2	39	\$5,282	\$12,667
Forest Service	13	42	\$4,783	\$61,364
Golf and Country Clubs	14	101	\$5,610	\$78,033
Landfills	9	44	\$5,205	\$48,676
Mining	14	113	\$5,644	\$80,527
Amusement Parks	19	418	\$5,950	\$113,424
Military Bases	11	395	\$9,548	\$108,754
Migrant Labor Camps	4	63	\$5,511	\$21,804
Misc. Recreation Services	31	87	\$5,040	\$156,519
Nursing Homes	16	107	\$7,556	\$117,780
Office Parks	114	136	\$5,354	\$609,795
Prisons	8	1,820	\$43,104	\$346,270
Retailers (Non-food related)	83	174	\$5,117	\$426,424
Retailers (Food related)	17	322	\$5,237	\$89,168
State Parks	10	165	\$5,179	\$51,542
Non-Water Utilities	60	170	\$5,705	\$339,983
Manufacturing: Food	92	372	\$7,853	\$723,165
Manufacturing: Non-Food	461	168	\$5,874	\$2,708,131
<b>TOTAL</b>	<b>2,429</b>			<b>\$16,253,442</b>

**Exhibit 6-14**  
**Total Annual NTNC Treatment Costs at MCL 10 µg/L by System Service Type**  
**(3% Discount Rate)**

Service Area Type	# of Systems Above the MCL	Average Population Served Per System	Average Annual System Cost	Annual National Costs
Daycare Centers	43	76	\$5,168	\$222,846
Highway Rest Areas	1	407	\$5,377	\$4,299
Hotels/Motels	19	133	\$5,956	\$111,420
Interstate Carriers	15	123	\$5,047	\$77,207
Medical Facilities	20	393	\$12,174	\$238,133
Mobile Home Parks	6	185	\$6,387	\$35,405
Restaurants	22	370	\$5,103	\$113,692
Schools	448	358	\$6,818	\$3,057,578
Service Stations	3	230	\$5,168	\$14,599
Summer Camps	2	146	\$6,124	\$15,014
Water Wholesalers	14	173	\$14,628	\$207,398
Agricultural Products/Services	20	76	\$6,012	\$117,930
Airparks	5	60	\$5,034	\$27,101
Construction	5	53	\$4,733	\$24,974
Churches	12	50	\$5,177	\$63,471
Campgrounds/RV Parks	7	160	\$6,104	\$40,017
Fire Departments	2	98	\$5,938	\$12,977
Federal Parks	1	39	\$5,245	\$5,592
Forest Service	6	42	\$4,783	\$27,278
Golf and Country Clubs	6	101	\$5,542	\$34,263
Landfills	4	44	\$5,176	\$21,517
Mining	6	113	\$5,572	\$35,340
Amusement Parks	8	418	\$5,848	\$49,558
Military Bases	5	395	\$9,095	\$46,053
Migrant Labor Camps	2	63	\$5,452	\$9,589
Misc. Recreation Services	14	87	\$5,027	\$69,397
Nursing Homes	7	107	\$7,298	\$50,567
Office Parks	51	136	\$5,310	\$268,864
Prisons	4	1,820	\$39,380	\$140,629
Retailers (Non-food related)	37	174	\$5,097	\$188,796
Retailers (Food related)	8	322	\$5,205	\$39,394
State Parks	4	165	\$5,153	\$22,794
Non-Water Utilities	26	170	\$5,627	\$149,069
Manufacturing: Food	41	372	\$7,566	\$309,707
Manufacturing: Non-Food	205	168	\$5,780	\$1,184,505
<b>TOTAL</b>	<b>1,080</b>			<b>\$7,036,973</b>

**Exhibit 6-15**  
**Total Annual NTNC Treatment Costs at MCL 20 µg/L by System Service Type**  
**(3% Discount Rate)**

Service Area Type	# of Systems Above the MCL	Average Population Served Per System	Average Annual System Cost	Annual National Costs
Daycare Centers	16	76	\$5,135	\$83,500
Highway Rest Areas	0	407	\$5,318	\$1,603
Hotels/Motels	7	133	\$5,823	\$41,085
Interstate Carriers	6	123	\$5,029	\$29,013
Medical Facilities	7	393	\$11,259	\$83,054
Mobile Home Parks	2	185	\$6,201	\$12,962
Restaurants	8	370	\$5,078	\$42,666
Schools	169	358	\$6,577	\$1,112,336
Service Stations	1	230	\$5,135	\$5,470
Summer Camps	1	146	\$5,970	\$5,520
Water Wholesalers	5	173	\$14,025	\$74,986
Agricultural Products/Services	7	76	\$5,873	\$43,442
Airparks	2	60	\$5,018	\$10,187
Construction	2	53	\$4,733	\$9,418
Churches	5	50	\$5,143	\$23,777
Campgrounds/RV Parks	2	160	\$5,953	\$14,718
Fire Departments	1	98	\$5,808	\$4,786
Federal Parks	0	39	\$5,203	\$2,091
Forest Service	2	42	\$4,783	\$10,287
Golf and Country Clubs	2	101	\$5,462	\$12,734
Landfills	2	44	\$5,142	\$8,061
Mining	2	113	\$5,488	\$13,127
Amusement Parks	3	418	\$5,729	\$18,310
Military Bases	2	395	\$8,568	\$16,360
Migrant Labor Camps	1	63	\$5,383	\$3,570
Misc. Recreation Services	5	87	\$5,012	\$26,091
Nursing Homes	3	107	\$6,997	\$18,282
Office Parks	19	136	\$5,259	\$100,420
Prisons	1	1,820	\$35,041	\$47,189
Retailers (Non-food related)	14	174	\$5,073	\$70,861
Retailers (Food related)	3	322	\$5,167	\$14,748
State Parks	2	165	\$5,121	\$8,544
Non-Water Utilities	10	170	\$5,536	\$55,308
Manufacturing: Food	15	372	\$7,231	\$111,625
Manufacturing: Non-Food	77	168	\$5,670	\$438,184
<b>TOTAL</b>	<b>407</b>			<b>\$2,574,315</b>

### 6.3.3 Costs per Household

Household level costs are considered a good proxy for the affordability of rule compliance with regard to CWSs, since water systems recover costs at the household level through increased water rates. This of course assumes that non-residential customers of water systems, such as businesses, can pass along any increase in water costs to their customers through increased prices on their goods or services. In order to calculate the number of households served by systems that will treat, the expected number of treating systems is multiplied by the average number of households per system (varies by system type and size). Exhibit 6-16 presents the total number of households served by CWSs that treat, by size category.

**Exhibit 6-16**  
**Number of Households in CWSs Expected to Treat**  
**by Size Category and MCL (µg/L) Option**

	<100	101-500	501-1,000	1,001-3,300	3,301-10,000	10,001-50,000	50,001-100,000	100,001-1,000,000	Total
<b>3</b>	94,484	368,092	360,709	1,002,937	1,619,822	3,228,544	1,453,603	3,014,841	11,143,032
<b>5</b>	58,774	228,149	219,872	623,156	1,019,288	2,077,421	905,886	1,938,268	7,070,814
<b>10</b>	26,369	104,373	101,866	288,986	475,599	997,880	469,157	936,602	3,400,833
<b>20</b>	10,439	40,089	40,498	116,517	193,541	405,714	188,798	364,907	1,360,503

SafeWaterXL determines household costs separately for each affected CWS, by first dividing the CWS’s annual compliance cost by the CWS’s average daily flow (1,000 gallons per day), and then multiplied by 365 days to determine the CWS’s cost of compliance per 1,000 gallons produced. Finally, the CWS’s cost of compliance per 1,000 gallons (kgal) is multiplied by the average annual consumption per residential connection (kgal), to arrive at the average annual cost of compliance per household for the CWS. The estimates of average annual consumption per residential connection used in this analysis are provided in Chapter 4, “Baseline Analysis.”

Given expected household costs for each individual system, the average is then calculated for each size category. Exhibit 6-17 shows the average annual household costs by system size, across the four regulatory options.

The range of household costs for the MCL of 10 µg/L ranges from less than \$1 to approximately \$327; the costs for the MCL of 3 µg/L range from less than \$7 to \$317; the costs for the MCL of 5 µg/L, range from less than \$3 to \$318; and the costs for the MCL of 20 µg/L range from less than \$1 to \$351.

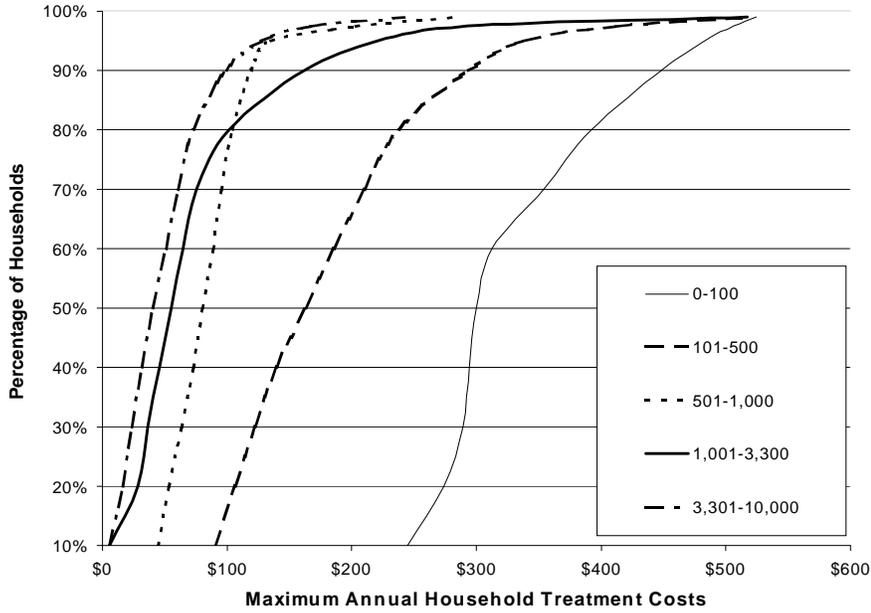
In the smallest two size categories, average household costs decrease as the MCL decreases. This somewhat counterintuitive result is due to the \$500.00 affordability cap assumed in the SafeWater XL simulations. As more systems are forced over the affordability cap, the systems’ costs are fixed at the costs associated with the POU technology. This results in lower average household costs for these systems.

**Exhibit 6-17**  
**Average Annual Household Costs Across MCL Options by System Size**

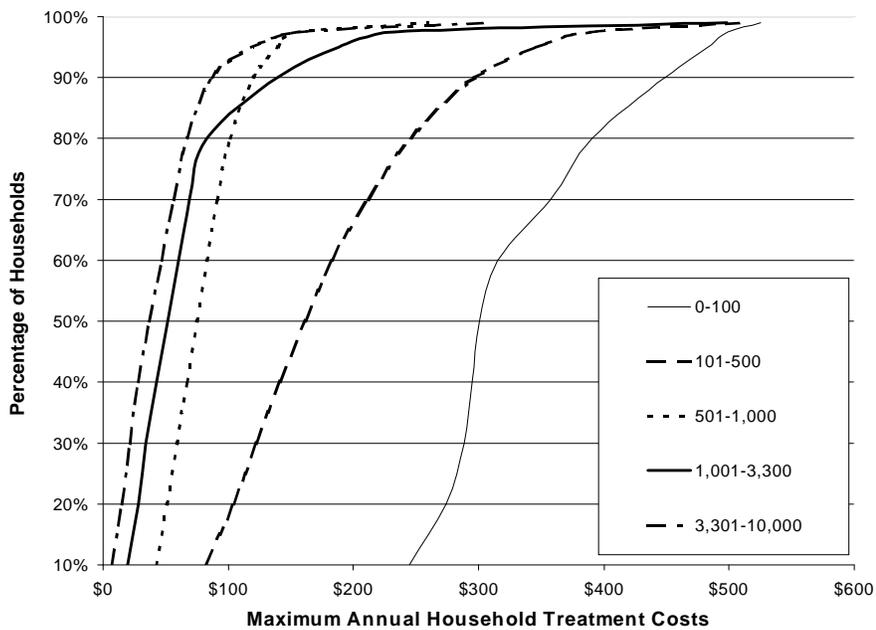
System Size	MCL ( $\mu\text{g/L}$ )			
	3	5	10	20
<100	\$317.00	\$318.26	\$326.82	\$351.15
101-500	\$166.91	\$164.02	\$162.50	\$166.72
501-1,000	\$74.81	\$73.11	\$70.72	\$68.24
1,001-3,300	\$63.76	\$61.94	\$58.24	\$54.36
3,301-10,000	\$42.84	\$40.18	\$37.71	\$34.63
10,001-50,000	\$38.40	\$36.07	\$32.37	\$29.05
50,001-100,000	\$31.63	\$29.45	\$24.81	\$22.63
100,001-1,000,000	\$25.29	\$23.34	\$20.52	\$19.26
>1,000,000	\$7.41	\$2.79	\$0.86	\$0.15
<b>All categories</b>	\$41.34	\$36.95	\$31.85	\$23.95

Exhibits 6-18 through 6-21 compare the distribution of annual household costs across public water systems serving fewer than 10,000 people, for MCLs of 3, 5, 10, and 20, respectively. The exhibits demonstrate the maximum annual costs that different percentages of households in treating systems face. Comparison of Exhibits 6-18 through 6-21 illustrates that regulatory compliance costs decrease across MCLs. This observation is depicted by the consistent shift to the left of cost curves across system size categories, when comparing incremental increases in the MCL.

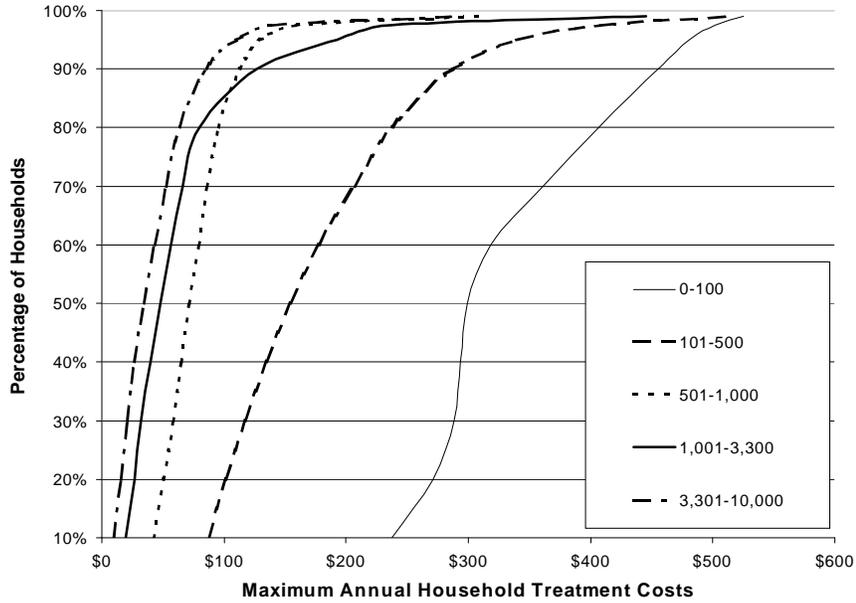
**Exhibit 6-18**  
**Annual Treatment Costs Per Household Across CWSs**  
**Expected to Treat and Serving < 10,000 People**  
**MCL 3 µg/L**



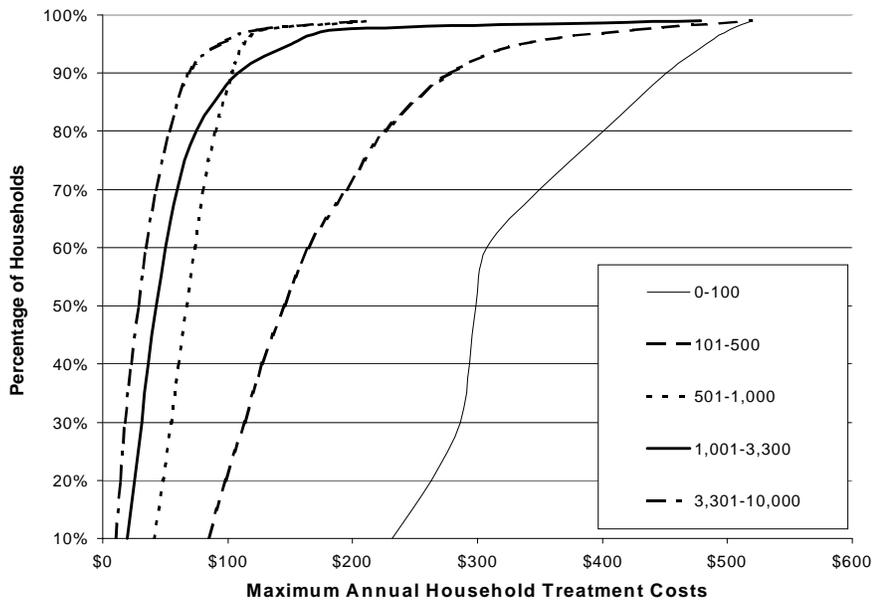
**Exhibit 6-19**  
**Annual Treatment Costs Per Household Across CWSs**  
**Expected to Treat and Serving < 10,000 People**  
**MCL 5 µg/L**



**Exhibit 6-20**  
**Annual Treatment Costs Per Household Across CWSs**  
**Expected to Treat and Serving < 10,000 People**  
**MCL 10 µg/L**



**Exhibit 6-21**  
**Annual Treatment Costs Per Household Across CWSs**  
**Expected to Treat and Serving < 10,000 People**  
**MCL 20 µg/L**



## 6.4 National Compliance Costs Uncertainty Analysis

The national cost estimates discussed throughout this chapter were developed within the SafeWaterXL modeling framework so that EPA could fully describe the variation in compliance costs among systems in a single size category (rather than just the average cost for systems within a size category). Hence, for each CWS size category, a distribution of compliance costs was estimated. These distributions are now used to access the uncertainty inherent in the national cost estimates.

A parametric bootstrap model was developed to estimate the distribution of national compliance costs.<sup>3</sup> The following steps were followed:

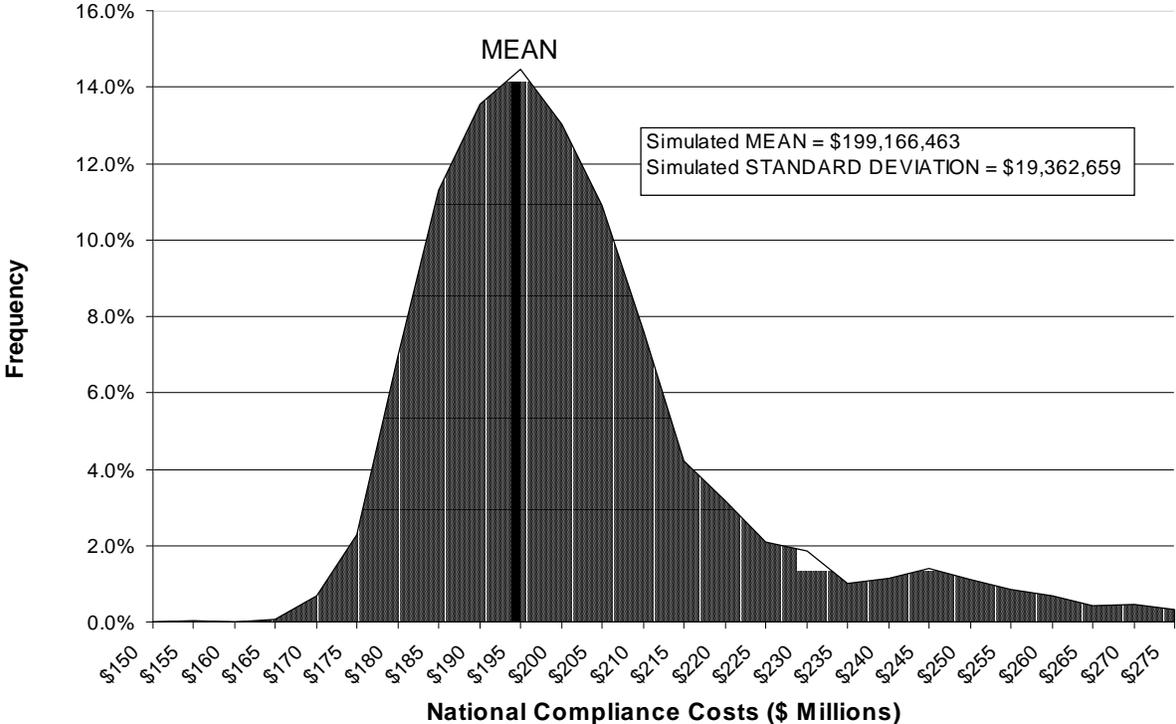
1. The distribution of costs for each CWS size and ownership cluster was pulled from the SafeWaterXL model results for an MCL of 10 µg/L.
2. The number of CWSs expected to modify or install treatment in each CWS size and ownership cluster was pulled from the SafeWaterXL model results for an MCL of 10 µg/L.
3. For each CWS size and ownership cluster, the model pulled a number of observations from the distribution of costs associated with that CWS size and ownership cluster (from step 1). The number of observations pulled was equal to the number of CWSs expected to modify or install treatment in each CWS size and ownership cluster (from step 2).
4. The observations (from step 3) were summed across all CWS size and ownership clusters to calculate a single estimate of national costs for CWSs.
5. No cost distributions are available for the NTNC systems and the very large CWSs. Therefore, after each single estimate of national costs for CWSs (from step 4) was calculated, the mean costs for very large CWSs and NTNC systems were added to it to calculate a single total national cost estimate.
6. Steps 3 through 5 were repeated 3,000 times to calculate a distribution of total national costs.

The distribution of total national costs is shown in Exhibit 6-22. The simulated mean national costs is \$199 million, and the simulated standard deviation is \$19 million. Also, the cumulative distribution of total national costs is shown in Exhibit 6-23. As this exhibit shows, the 10<sup>th</sup> and 90<sup>th</sup> percentile confidence interval for total national costs are \$190 million and \$227 million respectively.

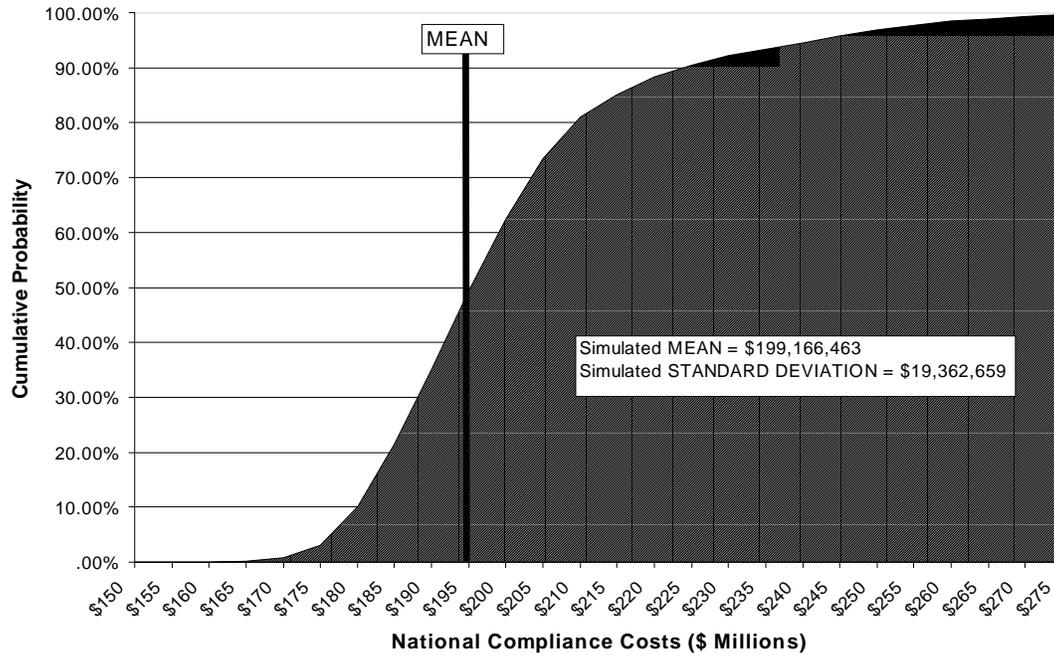
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<sup>3</sup> Only treatment costs were included in the uncertainty analysis. Also, the uncertainty analysis was conducted assuming a commercial discount rate. Although this commercial discount rate varies by CWS size and ownership, it approximates five percent for all PWSs.

**Exhibit 6-22**  
**National Compliance Costs Uncertainty Analysis**  
**Frequency Distribution (MCL 10 µg/L)**



**Exhibit 6-23**  
**National Compliance Costs Uncertainty Analysis**  
**Cumulative Distribution (MCL 10 µg/L)**



## **Chapter 7: Comparison of Costs and Benefits**

### **7.1 Introduction**

In this EA, EPA has analyzed the costs and benefits of regulating arsenic concentrations in drinking water to four different MCL standards. The four options considered reflect increasing levels of protection against exposure to arsenic in drinking water, employing a range of MCLs from 20 µg/L to 3 µg/L. As the MCL provisions for the four options become increasingly strict, the associated costs and benefits also increase incrementally. Chapter 5 (“Benefits Analysis”) describes in detail the estimated national health benefits of the Arsenic Rule options, while Chapter 6 (“Cost Analysis”) describes the projected national compliance cost estimates. This chapter presents a summary and comparison of the national costs and benefits and a cost-effectiveness analysis for each of the MCL options.

### **7.2 Summary of National Costs and Benefits**

#### **7.2.1 National Cost Estimates**

National compliance costs to public water systems (PWSs) for treatment (both annualized capital and operating and maintenance costs), monitoring and administrative activities, and costs to States, including any one-time start-up costs, for regulatory implementation and enforcement, were estimated and described in Chapter 6. The national costs for PWSs to comply with the four MCL options range from \$66.8 million (MCL=20 µg/L) to \$697.8 million (MCL=3 µg/L) annually based on a discount rate of three percent. Assuming a seven percent discount rate, the range of total national cost for community water systems ranges from \$76.5 million to \$792.1 million annually.

#### **7.2.2 National Benefits Estimates**

Chapter 5 contains a detailed summary of the methodology used to estimate a range of national health benefits from avoided cancer cases as a result of the four Arsenic Rule MCL options. The dollar value of the estimated health benefits associated with each of the four rule options was calculated based on lower and upper bound estimates of avoided bladder and lung cancer cases. The national benefits range from \$66.2 million (MCL=20 µg/L) to \$213.8 million (MCL=3 µg/L) annually, based on the lower bound estimates of cancer cases avoided. Under the upper bound scenario, the health benefits from avoided cancer increase from \$75.3 million at an MCL of 20 µg/L to \$490.9 million annually at an MCL of 3 µg/L.

### **7.3 Comparison of Benefits and Costs**

This section presents a comparison of total national benefits and costs for each of the Arsenic Rule options considered. Three separate analyses are considered, including a summary of benefit/cost ratios and net benefits, a direct comparison of aggregate national costs and benefits, and the results of a cost-effectiveness analysis of each regulatory option.

### 7.3.1 National Net Benefits and National Benefit-Cost Comparison

Exhibit 7-1 describes the net benefits and the benefit/cost ratios under various MCL options for PWSs at three and seven percent discount rates. Except for the upper bound benefit scenario at a discount rate of three percent, the net benefits are negative and decreasing as the Arsenic Rule MCL options become increasingly more stringent. For the same categories, the benefit/cost ratios are less than one and decrease as the MCL becomes more stringent. For nearly all of the options, costs outweigh the quantified benefits, with benefit/cost ratios all below or equal to one. For example, the ratios range from 0.3 (MCL=3 µg/L) to 1.0 (MCL=20 µg/L) at a seven percent discount rate. For the upper bound scenario at three percent the benefit/cost ratio exceeds one at an MCL of 10 µg/L and 20 µg/L. Of the MCL options examined, the net benefits and benefit/cost ratio are maximized at an MCL of 10 µg/L and a three percent discount rate.

**Exhibit 7-1**  
**Summary of Annual National Net Benefits and Benefit-Cost Ratios**  
**(\$ millions)**

MCL (µg/L)		3	5	10	20
<b>3% Discount Rate</b>					
lower bound	Net Benefits	\$ (484.0)	\$ (223.7)	\$ (40.8)	\$ (0.6)
	Benefit/Cost Ratio	0.3	0.5	0.8	1.0
upper bound	Net Benefits	\$ (206.8)	\$ (59.2)	\$ 17.3	\$ 8.5
	Benefit/Cost Ratio	0.7	0.9	1.1	1.1
<b>7% Discount Rate</b>					
lower bound	Net Benefits	\$ (578.3)	\$ (280.6)	\$ (66.0)	\$ (10.3)
	Benefit/Cost Ratio	0.3	0.4	0.7	0.9
upper bound	Net Benefits	\$ (301.1)	\$ (116.1)	\$ (7.9)	\$ (1.2)
	Benefit/Cost Ratio	0.6	0.8	1.0	1.0

\*Costs include treatment, O&M, monitoring, and administrative costs to CWSs and NTNCs and State costs for administration of water programs.

Exhibit 7-2 graphically depicts the absolute difference between the total value of national costs and benefits under each proposed MCL at a seven percent discount rate.

**Exhibit 7-2  
Comparison of Costs and Benefits  
(7% Discount Rate, in \$ millions)**

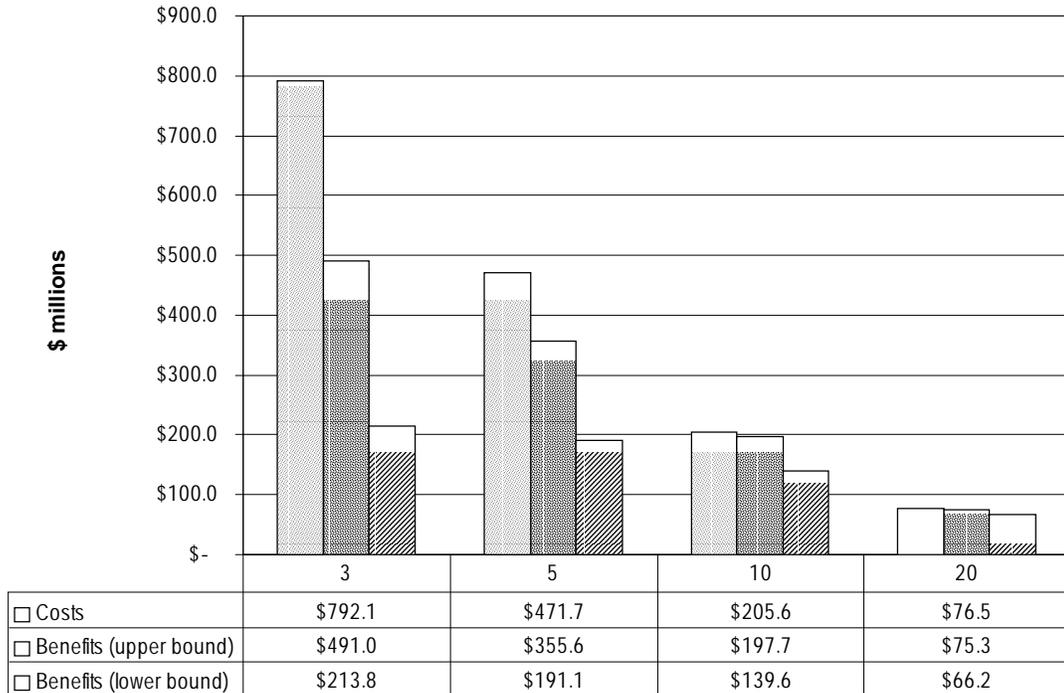


Exhibit 7-3 depicts the incremental costs and benefits of the rule as one moves from a less stringent standard to a more stringent standard. Moving to an MCL of 20 µg/L from the current MCL of 50 µg/L results in incremental costs of \$76.5 million and incremental benefits of between \$66.2 million and \$75.3 million. A move from 20 µg/L to 10 µg/L results in incremental costs of \$129.1 million and incremental benefits of between \$73.4 million and \$122.4 million. Moving beyond an MCL of 10 µg/L towards a more stringent standard results in incremental costs that far outweigh the incremental benefits, even under the upper bound benefits scenario.

**Exhibit 7-3  
Comparison of Incremental Costs and Benefits  
(7% Discount Rate, in \$ millions)**

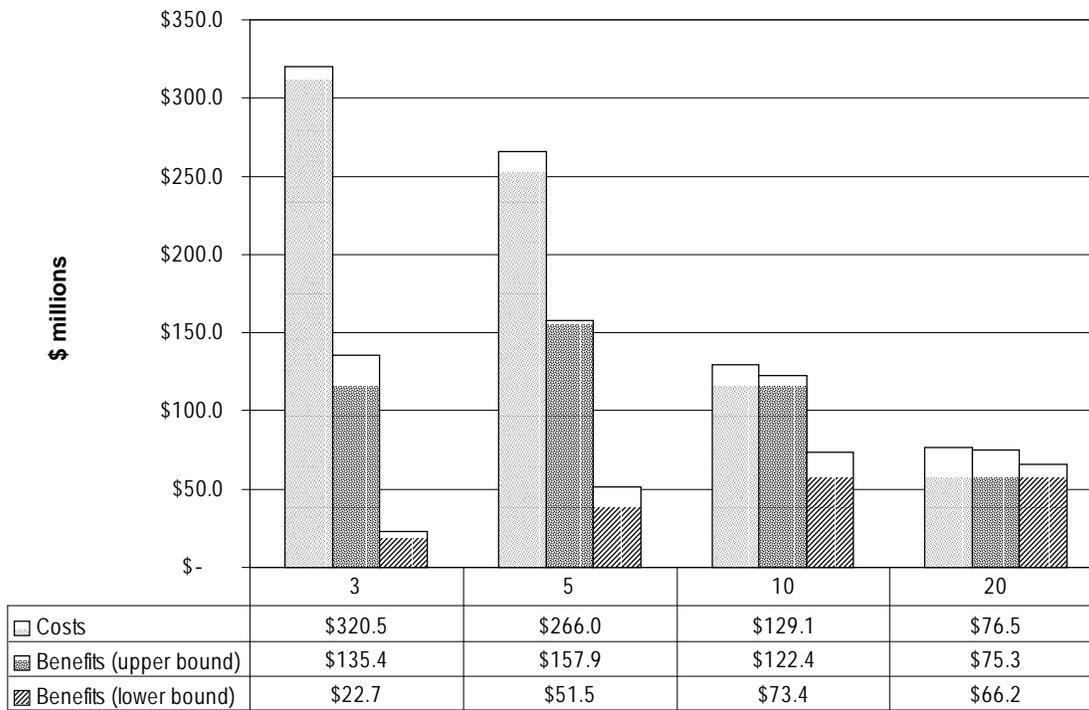


Exhibit 7-4 shows the results of an analysis in which the average national cost of achieving each unit reduction in cases of cancer avoided was calculated. The average annual cost per cancer case avoided was computed at each MCL option, for both three and seven percent discount rates. At a three percent discount rate, the cost per cancer case avoided ranges from \$5.0 million to \$12.2 million at an MCL of 3  $\mu\text{g/L}$ , from \$4.1 million to \$8.1 million at an MCL of 5  $\mu\text{g/L}$ , from \$3.2 million to \$4.8 million at an MCL of 10  $\mu\text{g/L}$ , and from \$3.4 million to \$3.5 million at an MCL of 20  $\mu\text{g/L}$ . At a seven percent discount rate, the cost per cancer case avoided ranges from \$5.7 million to \$13.8 million at an MCL of 3  $\mu\text{g/L}$ , from \$4.7 million to \$9.2 million at an MCL of 5  $\mu\text{g/L}$ , from \$3.7 million to \$5.5 million at an MCL of 10  $\mu\text{g/L}$ , and from \$3.9 million to \$4.0 million at an MCL of 20  $\mu\text{g/L}$ .

**Exhibit 7-4  
Cost per Cancer Case Avoided  
(\$ millions)**

Arsenic Level ( $\mu\text{g/L}$ )	lower bound**	upper bound**
<b>3% Discount Rate</b>		
<b>3</b>	\$ 12.2	\$ 5.0
<b>5</b>	\$ 8.1	\$ 4.1
<b>10</b>	\$ 4.8	\$ 3.2
<b>20</b>	\$ 3.5	\$ 3.4
<b>7% Discount Rate</b>		
<b>3</b>	\$ 13.8	\$ 5.7
<b>5</b>	\$ 9.2	\$ 4.7
<b>10</b>	\$ 5.5	\$ 3.7
<b>20</b>	\$ 4.0	\$ 3.9

\*\*Lower/upper bounds correspond to estimates of bladder cancer cases avoided.

### 7.3.2 Cost-Effectiveness

Cost-effectiveness analysis is another commonly used measure of the economic efficiency with which regulatory options are meeting the intended regulatory objectives. Exhibit 7-5 is a comparison of annual national costs (computed at a seven percent discount rate) and annual cases of cancer avoided at each MCL option. The two lines represent the cost per cancer case avoided under the lower and upper bound estimates of cancer cases avoided. These plotted lines depict the trend in marginal cost and benefits (expressed as health effects avoided) between each point on these curves (corresponding to each MCL option). Points along these lines represent each increment of cost that is incurred in order to achieve the next increment of risk reduction, i.e., additional cancer case avoided. The steepness of the curves under both benefits scenarios suggests that additional increments of risk reduction and benefits are achieved at increasingly greater cost to the nation.

**Exhibit 7-5  
Comparison of Annual Costs to Cases of Cancer per Year  
(7% Discount Rate)**

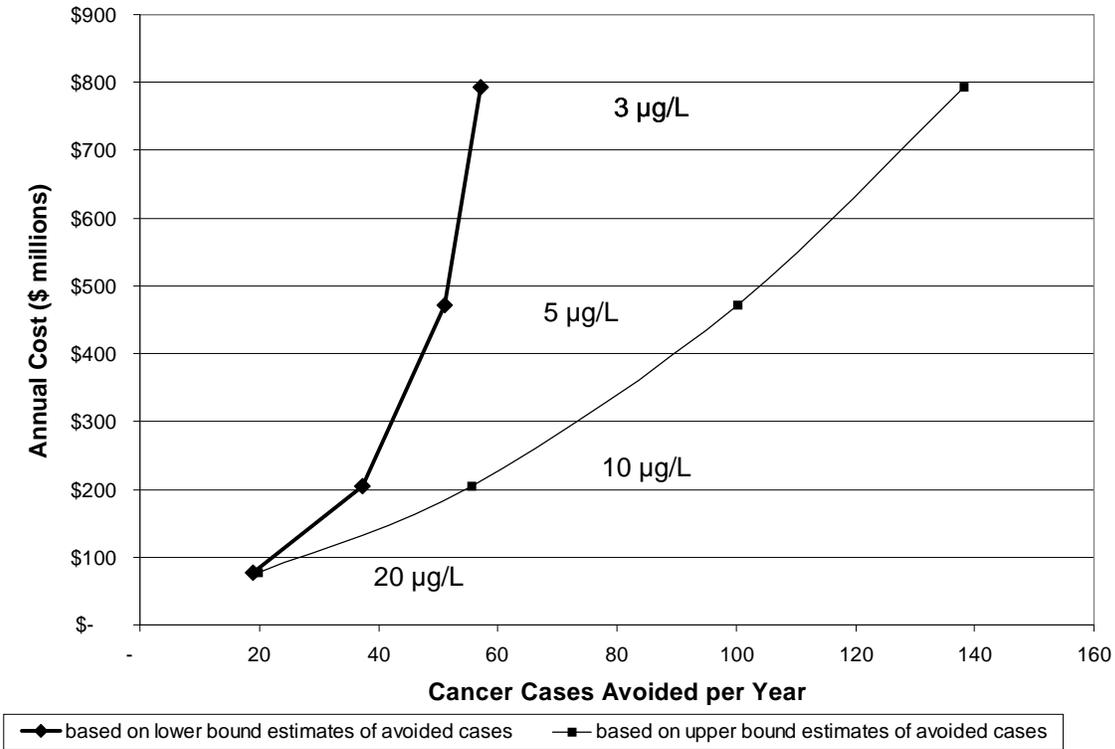
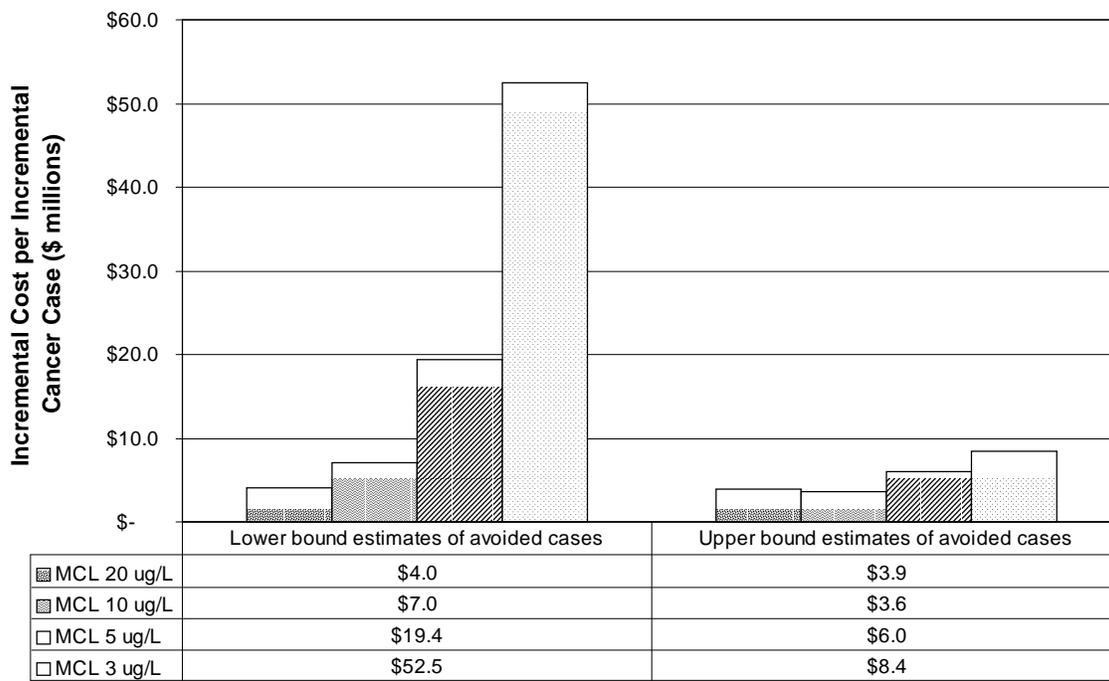


Exhibit 7-6 further reinforces the fact that as the MCL becomes more stringent, the incremental cost per cancer case avoided increases. For example, the additional cases of cancer avoided in moving from an MCL of 10 µg/L to 5 µg/L are achieved at a cost per case of \$3.6 million annually under the high bound and seven percent discount rate scenario. Similarly, in moving from an MCL of 5 µg/L to a more stringent MCL of 3 µg/L, the cost per case avoided increases to \$2.4 million per year under this same scenario.

**Exhibit 7-6  
Incremental Cost per Incremental Cancer Case Avoided  
(7% Discount Rate, in \$ millions)**



## 7.4 Other Benefits

Chapter 5 discusses a number of important non-monetized benefits of reducing arsenic exposure. Chief among these are certain health impacts known to be caused by arsenic. Such nonquantifiable benefits may include skin cancer, kidney cancer, cancer of the nasal passages, liver cancer, prostate cancer, cardiovascular effects, pulmonary effects, immunological effects, neurological effects, endocrine effects, and customer peace-of-mind benefits from knowing their drinking water has been treated for arsenic. For example, a number of epidemiologic studies conducted in several countries (e.g., Taiwan, Japan, England, Hungary, Mexico, Chile, and Argentina) report an association between arsenic in drinking water and skin cancer in exposed populations. Early reports linking inorganic arsenic contamination of drinking water to skin cancer came from Argentina (Neubauer, 1947, reviewing studies published as early as 1925) and Poland (Tseng et al., 1968). However, the first studies that observed dose-dependent effects of arsenic associated with skin cancer came from Taiwan (Tseng et al., 1968; Tseng, 1977). These studies focused EPA's attention on the health effects of ingested arsenic. Studies conducted in the U.S. have not demonstrated an association between inorganic arsenic in drinking water and skin cancer. However, these studies may not have included enough people in their design to detect these types of effects.

The potential monetized benefits associated with skin cancer reduction would not change the total benefits of the rule to an appreciable degree, even if the assumption were made that the risk of skin cancer were equivalent to that of bladder cancer, using EPA’s 1988 risk assessment. Skin cancer is highly treatable (at a cost of illness of less than \$3,500 for basal and squamous cell carcinomas versus a cost of illness of \$178,000 for non-fatal bronchitis) in the U.S., with few fatalities (less than one percent).

In addition to skin cancer, there are also a large number of other health effects associated with arsenic, as presented in Exhibit 7-7, which are not monetized in this analysis, due to lack of appropriate data.

**Exhibit 7-7**  
**Total Annual Cost, Estimated Monetized Total Cancer Health Benefits, and**  
**Non-Quantifiable Health Benefits from Reducing Arsenic in PWSs**  
**(\$ millions)**

Arsenic Level (µg/L)	Total Annual Cost (7%)	Annual Bladder Cancer Health Benefits <sup>1,2</sup>	Annual Lung Cancer Health Benefits <sup>1,2</sup>	Total Annual Health Benefits <sup>1,2</sup>	Potential Non-Quantifiable Health Benefits
3	\$792.1	\$58.2 - \$156.4	\$155.6 - \$334.5	\$213.8 - \$490.9	<ul style="list-style-type: none"> <li>• Skin Cancer</li> <li>• Kidney Cancer</li> <li>• Cancer of the Nasal Passages</li> <li>• Liver Cancer</li> <li>• Prostate Cancer</li> <li>• Cardiovascular Effects</li> <li>• Pulmonary Effects</li> <li>• Immunological Effects</li> <li>• Neurological Effects</li> <li>• Endocrine Effects</li> <li>• Reproductive and Developmental Effects</li> </ul>
5	\$471.7	\$52.0 - \$113.3	\$139.1 - \$242.3	\$191.1 - \$355.6	
10	\$205.6	\$38.0 - \$63.0	\$101.6 - \$134.7	\$139.6 - \$197.7	
20	\$76.5	\$20.1 - \$21.5	\$46.1 - \$53.8	\$66.2 - \$75.3 <sup>3</sup>	

<sup>1</sup> May 1999 dollars.

<sup>2</sup> These monetary estimates are based on cases avoided given in Exhibit 5-9 (a-c).

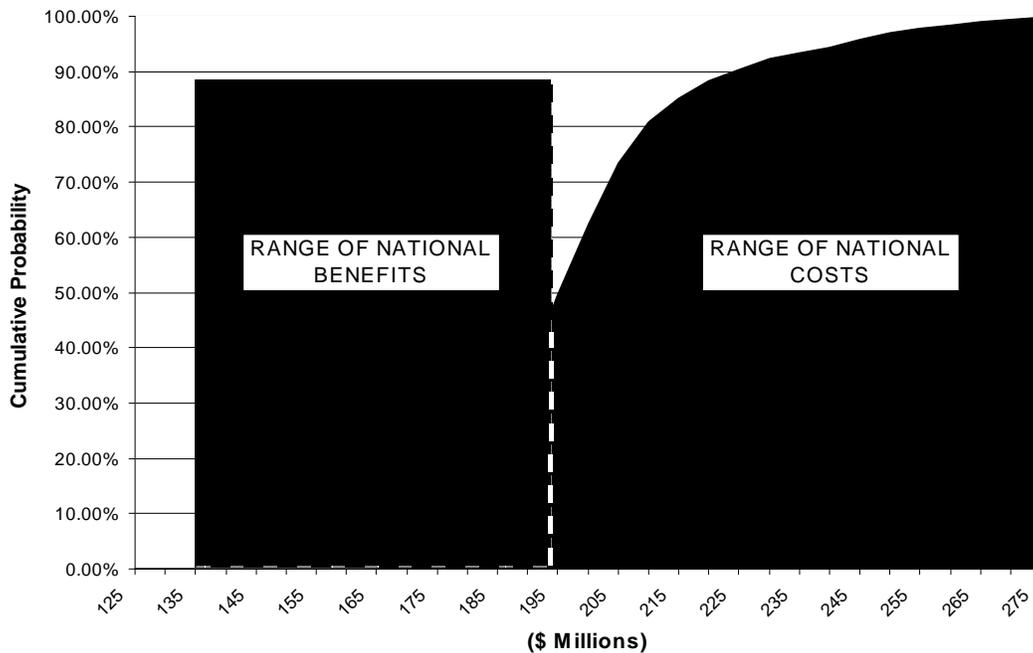
<sup>3</sup> For 20 µg/L, the proportional reduction from the lower level risk base case is greater than the proportional reduction from the higher level risk base case. Thus the number of estimated cases avoided and estimated benefits are higher at 20 µg/L using the estimates adjusted for uncertainty.

Other benefits not monetized in this analysis include customer peace of mind from knowing drinking water has been treated for arsenic and reduced treatment costs for currently unregulated contaminants that may be co-treated with arsenic. To the extent that reverse osmosis is used for arsenic removal, these benefits could be substantial. Reverse osmosis is the primary point-of-use treatment, and it is expected that very small systems will use this treatment to a significant extent. (These benefits of avoided treatment cannot currently be monetized; however, they can be readily monetized in the future, as decisions are made about which currently unregulated contaminants to regulate.)

## 7.5 Benefits-Costs Uncertainty Analysis

The uncertainty surrounding the national cost of compliance was described in Chapter 6. Exhibit 7-8 superimposes the distribution of national compliance costs onto the range of monetized benefits associated with the rule at an MCL of 10 µg/L. This exhibit illustrates that there is approximately a 50 percent probability that the costs of the rule will be lower than the monetized benefits of the rule under the upper bound benefit assumption.

**Exhibit 7-8**  
**National Compliance Costs and Benefits Uncertainty Analysis**  
**Cumulative Cost Distribution vs. Benefits Range (MCL 10 µg/L)**



## Chapter 8: Economic Impact Analyses

### 8.1 Introduction

The Environmental Protection Agency (EPA) is required to perform a series of analyses that addresses the distribution of regulatory impacts associated with the Arsenic Rule. This chapter presents analyses that support EPA's compliance with the following Federal mandates:

- Executive Order 12886 (Regulatory Planning and Review);
- Regulatory Flexibility Act (RFA) of 1980, as amended by the Small Business Regulatory Enforcement Fairness Act (SBREFA) of 1996;
- National Affordability determination required by the 1996 amendments to the Safe Drinking Water Act (SDWA);
- Unfunded Mandates Reform Act (UMRA) of 1995;
- Technical, Financial, and Managerial Capacity Assessment required by Section 1420(d)(3) of the 1996 amendments to the Safe Drinking Water Act (SDWA);
- Executive Order 13045 (Protection of Children From Environmental Health Risks and Safety Risks);
- Executive Order 12989 (Federal Actions to Address Environmental Justice in Minority Populations and Low-Income Populations);
- Paperwork Reduction Act;
- Health Risk Reduction and Cost Analysis (HRRCA) as required by Section 1412(b)(3)(C) of the 1996 SDWA Amendments; and
- Initial Regulatory Flexibility Analysis (IRFA).

These analyses draw on the cost analyses presented in Chapter 6 and an analysis of administrative requirements presented in a separate document, *Information Collection Request for the Arsenic Rule*.

Several of these Federal mandates require an explanation of why the rule is necessary, the statutory authority upon which it is based, and the primary objectives it is intended to achieve. Background information on the problems addressed by the rule, and EPA's statutory authority for promulgating the rule, are presented in Chapter 2. In this chapter, Section 8.2 presents the RFA and SBREFA analysis of impacts on small entities. Also described are the economic impacts of the rule on households. Section 8.3 discusses coordination of the Arsenic Rule with other Federal rules. The minimization of economic burden, UMRA, system capacity assessments, and the Paperwork Reduction Act are addressed in Sections 8.4, 8.5, 8.6, and 8.7, respectively. Section 8.8 discusses the rule's protection of children's health, Section 8.9 addresses environmental justice issues, and Section 8.10 contains the HRRCA.

### 8.2 Regulatory Flexibility Act and Small Business Regulatory Enforcement Fairness Act

The RFA provides that, whenever an agency promulgates a proposed or final rule under section 553 of the Administrative Procedure Act, after being required by that section or any other law to publish a general notice of rulemaking, the agency must prepare an initial and final regulatory

flexibility analysis. The agency must prepare such an analysis when proposing a rule (or promulgating a final rule) unless the head of the agency certifies that the rule will not have a significant economic impact on a substantial number of small entities. EPA did not certify that the proposed regulation would not have a significant economic impact on a substantial number of small entities. Consequently, the Agency prepared an initial analysis of the proposal and, because it has not certified the final rule, has now completed a final regulatory flexibility analysis. EPA prepared these analyses in compliance with the requirements of the RFA

Under the RFA, the term “small entity” means “small business,” “small governmental jurisdiction” and “small organization.” These terms are further defined by the Act. In the case of a “small business,” the term has the same meaning as a “small business concern” under section 3 of the Small Business Act. (Regulations of the Small Business Administration (SBA) at 13 CFR 121.201 have defined small businesses for Standard Industrial Classification (SIC) codes.) “Small governmental jurisdiction” means the government of cities, counties, towns and villages, among others, with a population of less than 50,000. A “small organization” is any not-for-profit enterprise that is independently owned and operated. The RFA authorizes an agency to establish other definitions of such terms which are appropriate to the agency’s activities and publish such definitions in the Federal Register after consultation with SBA and opportunity for public comment. 5 U.S.C. § 601(3), (4) & (5).

### **8.2.1 Description of the Initial Regulatory Flexibility Analysis**

The Regulatory Flexibility Act requires EPA to complete an Initial Regulatory Flexibility Analysis (IRFA) addressing the following:

1. The need for the rule;
2. The objectives of and legal basis for the rule;
3. A description of, and where feasible, an estimate of the number of small entities to which the rule will apply;
4. A description of the reporting, record keeping, and other compliance requirements of the rule, including an estimate of the types of small entities that will be subject to the requirements and the type of professional skills necessary for preparation of reports or records;
5. An identification, to the extent practicable, of all relevant Federal rules that may duplicate, overlap, or conflict with the rule; and
6. A description of “any significant regulatory alternatives” to the rule that accomplish the stated objectives of the applicable statutes, and that minimize any significant economic impact of the rule on small entities. Significant regulatory alternatives may include:

- Establishing different compliance or reporting requirements or timetables that take into account the resources of small entities;
- Clarifying, consolidating, or simplifying compliance and reporting requirements under the rule for small entities;
- Using performance rather than design standards; and
- Exempting small entities from coverage of the rule or any part of the rule.

If the initial assessment determines that a substantial number of small entities may face significant impacts as a result of the rule, then a formal regulatory flexibility analysis may be required.

### ***Defining “Small Entities” Affected by the Rule***

The Regulatory Flexibility Act (RFA) defines small entities as including “small businesses,” “small governments,” and “small organizations” (5 USC 601). The RFA references the definition of “small business” found in the Small Business Act, which authorizes the Small Business Administration (SBA) to further define “small business” by regulation. The SBA defines small business by category of business using Standard Industrial Classification (SIC) codes (13 CFR 121.201). For example, in the manufacturing sector, the SBA generally defines small business in terms of number of employees; in the agriculture, mining, electric, gas, and sanitary services sectors, the SBA generally defines small businesses in terms of annual receipts (ranging from \$0.5 million for crops to \$25 million for certain types of pipelines). The RFA also authorizes an agency to adopt an alternative definition of “small business” “where appropriate to the activities of the Agency” after consultation with the SBA and opportunity for public comment.

For the revised Arsenic Rule small entities are defined as those water systems that meet the following criteria:

- 7 A “small business” is any small business concern that is independently owned and operated and not dominant in its field as defined by the Small Business Act (15 USC 632). Examples of public water systems within this category include small, privately owned, public water systems and for-profit businesses where provision of water may be ancillary, such as mobile home parks or day care centers.
- 7 A “small organization” is any not-for-profit enterprise that is independently owned and operated, not dominant in its field, and operates a public water system. Examples of small organizations are churches, schools, and homeowners associations.
- 7 A “small governmental jurisdiction” is a city, county, town, school district or special district with a population of less than 50,000 (5 USC 601) that operates a public water system.

In 1998, EPA proposed that PWSs with populations of 10,000 or fewer persons be defined as “small entities” within the context of the Consumer Confidence Report (CCR) rulemaking (63 FR 7620, February 13, 1998). EPA requested public comments on this alternative definition.

For this rulemaking, the SBA Office of Advocacy agreed with the Agency's alternative definition. EPA intends to define "small entity" in the same way for RFAs under SBREFA for all future drinking water regulations, including the revised Arsenic Rule.

EPA selected this alternative definition for small water systems for several reasons:

- 7 A large proportion (94 percent) of all PWSs are small entities, although they serve a minority of the population. Larger PWSs (those serving over 10,000 persons) serve the majority of the population receiving water from public water systems.
- 7 Certain key financial ratios (e.g., total debt as a ratio of total revenue) show a distinct break point at the 10,000 or fewer system size level.<sup>1</sup> In general, the size of a PWS is an important financial characteristic, as larger systems can spread investments in fixed assets across a broader customer base. Smaller water systems typically serve primarily residential customers. Larger systems have fewer residential customers as a percentage of total water sales and more commercial customers. Annual sales revenue per connection is significantly higher for nonresidential than for residential connections.<sup>2</sup> Similarly, larger publicly owned systems are more likely to have rated bond issuances, another indicator of financial strength.<sup>3</sup>
- 7 In the 1996 Amendments to the Safe Drinking Water Act (SDWA), several measures creating regulatory relief defined small community water systems as those serving 10,000 or fewer customers. One provision allows for alternative means of delivery of the CCRs by systems serving 10,000 or fewer persons. Another used the same cutoff for modifications to monitoring requirements and for certain penalty provisions delegated to the States.<sup>4</sup>
- 7 EPA has previously used this criterion in both rulemaking and implementation activities pertaining to PWSs. The total trihalomethane (TTHM) rule promulgated by EPA in 1979 applied only to systems serving more than 10,000 persons. EPA chose the 10,000 cutoff in 1979 primarily out of a concern that smaller systems would have to divert resources from other activities to comply with the rule. In 1992, EPA initiated a regulatory negotiation process that resulted in regulatory actions to provide additional protection from microbial contaminants in drinking water while reducing health risks from disinfection byproducts. The Interim Enhanced Surface Water Treatment Rule promulgated from this process

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<sup>1</sup> *Community Water Systems Survey, Volume I: Overview*, U.S. EPA Office of Water, p. 26. January 1997.

<sup>2</sup> *Id.*, p. 14.

<sup>3</sup> *Id.*, p. 28.

<sup>4</sup> *House Report No. 104-632* (Commerce Committee), June 24, 1996 in *US Code Congressional and Administrative News* (USCCAAN), 1996, 4, pp. 1373, 1401 and 1409, discussing §§132(b) and 1418(a) of the House bill.

applied only to systems serving more than 10,000. The companion rule, the Stage 1 Disinfection Byproducts Rule, deferred compliance with part of the requirements for systems serving 10,000 or fewer persons.

For purposes of this analysis, therefore, “small entity” refers to any public water system that serves 10,000 or fewer persons. Exhibit 8-1 shows the universe of small PWSs potentially affected by the new arsenic standard.

**Exhibit 8-1  
Profile of the Universe of Small Water Systems  
Regulated Under the Arsenic Rule**

Type Water System	System Size Category				
	<100	101-500	501-1,000	1,001-3,300	3,301-10,000
<b>Publicly-Owned:</b>					
CWS	1,729	5,795	3,785	6,179	3,649
NCWS	1,783	3,171	1,182	361	29
<b>Privately-Owned:</b>					
CWS	13,640	11,266	2,124	1,955	654
NCWS	8,178	4,162	902	411	56
<b>Total Systems:</b>					
CWS	15,369	17,061	5,909	8,134	4,303
NCWS	9,961	7,333	2,084	772	85
<b>TOTAL</b>	<b>25,330</b>	<b>24,394</b>	<b>7,993</b>	<b>8,906</b>	<b>4,388</b>

Source: Safe Drinking Water Information System (SDWIS), December 1998 freeze.

### ***Determining What Number Constitutes a Substantial Number***

In this analysis approximately 71,013 PWSs are defined as small entities. EPA SBREFA guidance has several different criteria for what constitutes a substantial number of affected entities.<sup>1</sup> One of the criteria is that no more than 20 percent of systems affected by the revised Arsenic Rule may experience economic impacts of one percent of their revenues or greater.

### ***Measuring Significant Impacts***

To evaluate the impact that a small entity is expected to incur as a result of the rule, this analysis calculates the entity’s ratio of annualized compliance costs as a percentage of sales (for privately owned systems) or the entity’s ratio of annualized compliance costs as a percentage of annual governmental revenue or expenditures (for publicly owned systems). EPA guidance suggests using one percent as a threshold for determining significance, although additional factors may be considered. If compliance costs are less than one percent of sales or revenues, the regulation may in most cases be presumed to have no significant impact on a substantial number of small entities.<sup>5</sup>

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<sup>5</sup> *Id.*

## **Categorizing Systems**

EPA categorized affected small entities according to the categories identified in the SBREFA guidance (i.e., small business, small government, and small organization). Public water system inventories, managed by EPA and other organizations, traditionally categorize public water systems by size and by the characteristics of the population served (i.e., community water system, non-community water system). Therefore, detailed information by SIC or data on revenues or sales are not readily available.

## **Estimating Revenue by RFA Category**

The estimated revenues for small entities in Exhibit 8-2 are from the Bureau of the Census<sup>6</sup>; EPA chemical monitoring reform rulemaking; and additional data on independent privately owned CWSs, special districts, and authorities, which are from the CWS Survey. Exhibit 8-2 also shows the numbers of small businesses, governments, and organizations, obtained using information from EPA's Baseline Handbook.<sup>7</sup> These numbers were used to determine the weighted averages of estimated average revenue, as described in the column "Average Estimated Revenues per System."

Small government systems include municipal, county, State, Federal, military, and special district systems. Data on revenue for townships and municipalities were obtained from the *1992 Census of Governments*, converted to 1999 dollars by applying a conversion factor calculated from the national income and product account tables of the U.S. Bureau of Economic Analysis.<sup>8</sup> Specifically, the price deflators for 1992 and 1999 were obtained from Table 7.11, *Chain-Type Quantity and Price Indexes for Government*, Chain-Type Price Indexes for State and Local Governments. The average revenue for all small government PWSs was calculated at \$2,333,119.

Small businesses include both CWSs and NTNCWSs, such as privately owned community water systems, mobile home parks, country clubs, hotels, manufacturers, hospitals, and other establishments. For this analysis, all hospitals and day care centers were assumed to be businesses. Although some hospitals may be nonprofit, they have unusually high revenues and were included in the small business category to make the estimated revenue for small organizations more conservative. Estimated average revenue for the small businesses affected by the revised Arsenic Rule is \$2,675,582.

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<sup>6</sup>*1992 Census of Governments*, GC92 (4)-4: Finances of Municipal and Township Governments, U.S. Dept. of Commerce, Bureau of the Census.

<sup>7</sup>*Drinking Water Baseline Handbook* Second Edition, EPA Contract No. 68-C6-0039. Prepared by International Consultants, Inc.

<sup>8</sup>Methodology recommended by Bruce E. Baker, State and Local Governments, Government Division, U.S. Bureau of Economic Analysis.

**Exhibit 8-2**  
**Annual Cost of Compliance Costs as a Percentage of Revenues**  
**by Type of Small Entity**  
**(PWSs that are Expected to Modify or Install Treatment at an MCL = 10 µg/L)**

	Number of Systems	Average Estimated Revenues per System	Average Compliance Cost Per System	Cost to Revenue Ratio
<b>Water Systems that will Modify or Install Treatment</b>				
Small Government	1,116	\$2,333,119	\$41,999	1.8001%
Small Business	2,318	\$2,675,582	\$13,466	0.5033%
Small Organizations	472	\$5,990,914	\$6,828	0.1140%
All Small Entities	3,907	\$2,978,546	\$20,816	0.6989%
<b>Water Systems that will Only Monitor</b>				
Small Government	20,587	\$2,333,119	\$37	0.0016%
Small Business	38,131	\$2,675,582	\$39	0.0015%
Small Organizations	8,389	\$5,990,914	\$53	0.0009%
All Small Entities	67,106	\$2,984,958	\$40	0.0014%
<b>All Water Systems</b>				
Small Government	21,703	\$2,333,119	\$2,195	0.0941%
Small Business	40,449	\$2,675,582	\$809	0.0302%
Small Organizations	8,861	\$5,990,914	\$414	0.0069%
All Small Entities	71,013	\$2,984,605	\$1,183	0.0396%

Small organizations include primarily nonprofit NTNCWSs such as schools and homeowners associations. The estimates for small nonprofit organizations serving more than 500 people are actually higher than those for small businesses because the total number of such systems is small, and a large proportion of these organizations are schools and colleges with large budgets. This category also includes 50 percent of systems classified as “other.” The average estimated revenue for small organizations affected by the revised Arsenic Rule is \$2,978,546.

EPA also calculated the average estimated revenue for all small entities. This estimate is weighted to account for the number of small entities in each category (government, business, and organization) affected by the revised Arsenic Rule. This overall average is \$2,833,552.

**Conducting the Screening Analysis**

The final task of the initial assessment is to conduct the screening analysis and determine whether the rule is expected to result in significant economic impacts on a substantial number of small entities. The screening analysis involves the following three steps:

- (1) *Estimate the compliance cost of the rule to small PWSs.* Estimated average per-system compliance costs associated with the revised Arsenic Rule were taken from the estimate prepared by EPA and presented in Chapter 6.

- (2) *Obtain data on the number of small PWSs and their revenues or expenditures.* The number of small PWSs expected to modify or install treatment are found in Exhibit 8-2. These numbers are derived from the results of the SafeWaterXL model described in Chapter 6.
- (3) *Compute small entity impacts.* Using the data obtained in the preceding steps, EPA calculated the ratio of total annual compliance costs as a percentage of revenues or expenditures. These ratios, converted into percentages, are presented in Exhibit 8-2 in the column “Cost to Revenue Ratio.”

### 8.2.2 Initial Regulatory Flexibility Analysis Results

The results of the initial regulatory flexibility analysis are summarized below. As seen in Exhibits 8-2 and 8-3, at a maximum contaminant level (MCL) of 10 µg/L, 3,907 small PWSs are expected to have to modify or install treatment.

**Exhibit 8-3  
Number of CWSs Expected to Undertake or Modify Treatment Practice  
MCL 10 µg/L**

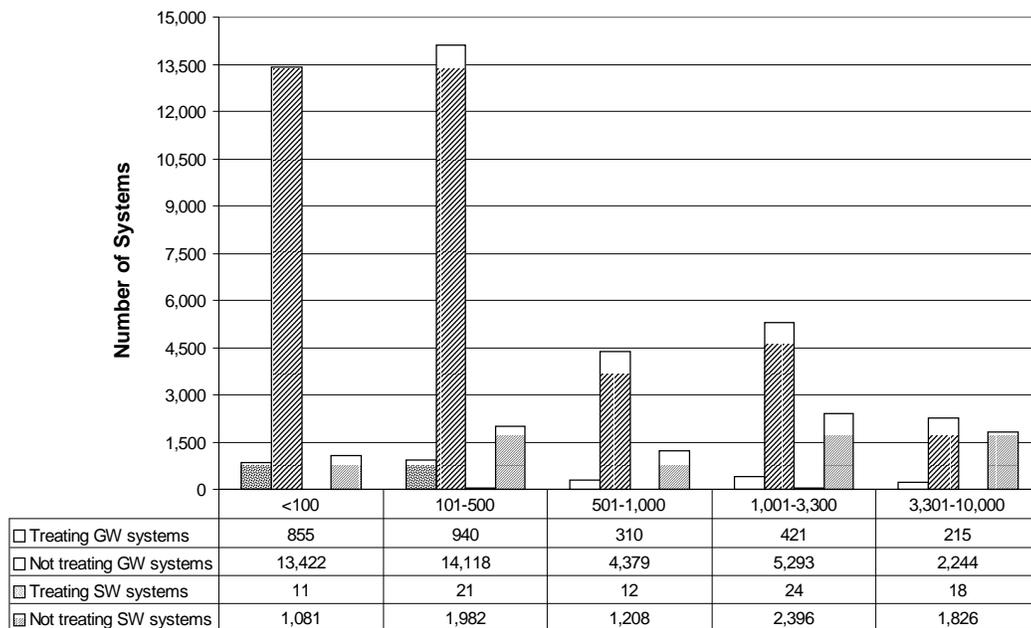


Exhibit 8-3 compares the number of CWSs expected to be affected by the promulgation of the new standard to the number of systems not expected to undertake or modify any of their existing treatment practices. Six percent of small CWSs and NTNC water systems are expected to have to modify or install treatment.

EPA compared the ratio of compliance cost to revenue to the threshold value for significant impacts of one percent under the revised arsenic standard of 10 µg/L. In Exhibit 8-2, the ratios

are displayed separately for small governments, small businesses, and small organizations, and cumulatively for all small entities.

A significant impact is generally defined as costs equal to or greater than one percent of revenues. Costs are equal to or greater than one percent of revenues only among small government entities that are expected to modify or install treatment at the revised MCL. The vast majority of water systems will see impacts less than one percent of their annual revenue. However, EPA's estimates show a number of small systems that will incur significant costs. Therefore, EPA is not certifying this rule as having no significant impact on small entities.

### **8.2.3 Summary of EPA's Small Business Consultations**

As required by section 609(b) of the RFA, as amended by SBREFA, EPA also conducted outreach to small entities and convened a Small Business Advocacy Review Panel to obtain advice and recommendations of representatives of the small entities that potentially would be subject to the rule's requirements.

EPA identified 22 representatives of small entities, in this situation small systems, that were most likely to be subject to the proposal. In December 1998, EPA prepared and distributed to the small entity representatives (SERs) an outreach document on the Arsenic Rule titled "Information for Small Entity Representatives Regarding the Arsenic in Drinking Water Rule" (EPA, 1998 ).

On December 18, 1998, EPA held a SER conference call for small systems from Washington, DC, to provide a forum for input on key issues related to the planned proposal of the Arsenic in Drinking Water Rule. These issues included, but were not limited to, issues related to the rule development, such as arsenic health risks, treatment technologies, analytical methods, and monitoring. Fifteen SERs from small water systems participated on the call from the following States: Alabama, Arizona, California, Georgia, Massachusetts, Montana, Nebraska, New Hampshire, New Jersey, Utah, Virginia, Washington, and Wisconsin.

Efforts to identify and incorporate small entity concerns into this rulemaking culminated with the convening of a SBAR Panel on March 30, 1999, pursuant to section 609 of RFA/SBREFA. The four person Panel was headed by EPA's Small Business Advocacy Chairperson and included the Director of the Standards and Risk Management Division within EPA's Office of Ground Water and Drinking Water, the Administrator of the Office of Information and Regulatory Affairs with the Office of Management and Budget, and the Chief Counsel for Advocacy of the SBA. For a 60-day period starting on the convening date, the Panel reviewed technical background information related to this rulemaking, reviewed comments provided by the SERs, and met on several occasions. The Panel also conducted its own outreach to the SERs and held a conference call on April 21, 1999, with the SERs to identify issues and explore alternative approaches for accomplishing environmental protection goals while minimizing impacts to small entities. Consistent with the RFA/SBREFA requirements, the Panel evaluated the assembled materials and small-entity comments on issues related to the elements of the IRFA (See Section 8.2.1) . A copy of the June 4, 1999, Panel report is included in the docket for the Arsenic Rule (U.S. EPA, 1999).

The revised rule addresses all of the recommendations on which the Panel reached consensus. In addition, to help small systems comply with the Arsenic Rule, EPA is committed to addressing several other Panel recommendations regarding guidance, which are discussed in detail in the pages to follow.

### ***Treatment Technologies, Waste Disposal, and Cost Estimates***

The Panel recommended the following: further develop the preliminary treatment and waste disposal cost estimates; fully consider these costs when identifying affordable compliance technologies for all system size categories; and provide information to small water systems on possible options for complying with the MCL, in addition to installing any listed compliance technologies.

In response to these recommendations, the Treatment and Cost document describes development of cost estimates for treatment and waste disposal; identification of affordable compliance technologies, including the consideration of cost; and options for complying with the MCL other than installing compliance technologies, such as selecting to regionalize.

Regarding point-of-use (POU) devices, the Panel recommended the following: continue to promote the use of POU devices as alternative treatment options for very small systems where appropriate; account for all costs, including costs that may not routinely be explicitly calculated; consider liability issues from POU/point-of-entry (POE) devices when evaluating their appropriateness as compliance technologies; and investigate waste disposal issues with POE devices.

In response to these recommendations, EPA included in the revised rule's preamble an expanded description regarding available POU compliance treatment technologies and conditions under which POU treatment may be appropriate for very small systems; a description of the components that contribute to the POU cost estimates; and a discussion that clarifies that water systems will be responsible for POU operation and maintenance to prevent liability issues from customers maintaining equipment themselves.

### ***Relevance of Other Drinking Water Regulations***

The Panel recommended the following: include discussion of the co-occurrence of arsenic and radon in the Arsenic Rule; take possible interactions among treatments for different contaminants into account in costing compliance technologies and determining whether they are nationally affordable for small systems; and encourage systems to be forward-looking and test for multiple contaminants to determine if and how they would be affected by the upcoming rules. In response, the revised rule's preamble includes a discussion on the co-occurrence analysis of radon and arsenic: the treatment section of the preamble describes the relationship of treatment for arsenic with other drinking water rules and how this issue was taken into account in cost estimates. In addition, the preamble encourages systems to consider other upcoming rules when making future plans for monitoring or treatment.

### ***Small Systems Variance Technologies and National Affordability Criteria***

The Panel recommended the following: include a discussion of the issues surrounding appropriate adjustment of its national affordability criteria to account for new regulatory requirements; consider revising its approach to national affordability criteria, to the extent allowed by statutory and regulatory requirements, to address the concern that the current cumulative approach for adjusting the baseline household water bills is based on chronological order rather than risk; and examine the data in the 1995 Community Water Supply Survey to determine if in-place treatment baselines can be linked with the current annual water bill baseline in each of the size categories for the revised Arsenic Rule.

In response to these recommendations, the treatment section of the revised rule's preamble includes an expanded discussion about the national affordability criteria and how it may be adjusted to account for new regulations. In addition, information regarding methodology and rationale is available to explain the national affordability approach.

### ***Monitoring and Arsenic Species***

The Panel recommended the following: that EPA consider allowing States to use recent compliance monitoring data to satisfy initial sampling requirements or to obtain a waiver; and that EPA continue to explore whether or not to make a regulatory distinction between organic and inorganic arsenic based on compliance costs and other considerations.

In response, the monitoring section of the rule's preamble describes the allowance of monitoring data that meet analytical requirements and have reporting limits sufficiently below the revised MCL and collected after 1990.

### ***Considerations in Setting the MCL***

The Panel recommended the following: in performing its obligations under SDWA, EPA should take cognizance of the scientific findings, the large scientific uncertainties, the large potential costs (including treatment and waste disposal costs), and the fact that this standard is scheduled for review in the future; give full consideration to the provisions of the Executive Order 12866 and to the option of exercising the new statutory authority under SDWA §1412(b)(4)(C) and §1412(b)(6)(A) in the development of the Arsenic Rule; and fully consider all of the "risk management" components of its rulemaking effort to ensure that the financial and other impacts on small systems are factored into its decision-making processes. The Panel also recommended that EPA take into account both quantifiable and non-quantifiable costs and benefits of the standard and the needs of sensitive sub-populations.

In response to all these recommendations, EPA has described in detail the factors that were considered in setting in the MCL and provides the rationale for this selection.

## ***Applicability of Proposal***

The Panel recommended that EPA carefully consider the appropriateness of extending the scope of the rule to non-transient non-community water systems (NTNCWSs).

In response, EPA has broadened the rule to include NTNCWSs. EPA has described the basis for this decision in the MCL section of the preamble, which includes a discussion of the incremental costs and benefits attributable to coverage of these water systems.

## ***Other Issues***

The Panel recommended that EPA encourage small systems to discuss their infrastructure needs for complying with the Arsenic Rule with their primacy agency to determine their eligibility for Drinking Water State Revolving Fund (DWSRF) loans, and if eligible, to ask for assistance in applying for the loans. In response, the UMRA analysis has been expanded to discuss funding options for small systems and to encourage systems to be proactive in communicating with their primacy agency.

Regarding health effects, the Panel recommended the following: further evaluate the Utah study and its relationship to the studies on which the NRC report was based and give it appropriate weight in the risk assessment for the revised arsenic standard; and examine the NRC recommendations in the light of the uncertainties associated with the report's recommendations, and any new data that may not have been considered in the NRC report. In response to these recommendations, the benefits analysis includes a discussion of the qualitative benefits evaluation and use of research data.

### **8.2.4 Small System Affordability**

Section 1415(e)(1) of SDWA allows States to grant variances to small water systems (i.e., systems having fewer than 10,000 customers) in lieu of complying with an MCL if EPA determines that there are no nationally affordable compliance technologies for that system size/water quality combination. The system must then install an EPA-listed variance treatment technology (§1412(b)(15)) that makes progress toward the MCL, if not necessarily reaching it. To list variance technologies, three showings must be made:

- (1) EPA must determine, on a national level, that there are no compliance technologies that are affordable for the given small system size category/source water quality combination.
- (2) If there is no nationally affordable compliance technology, then EPA must identify a variance technology that may not reach the MCL but that will allow small systems to make progress toward the MCL (it must achieve the maximum reduction affordable). This technology must also be listed as a small systems variance technology by EPA in order for small systems to be able to rely on it for regulatory purposes.

- (3) EPA must make a finding on a national level, that use of the variance technology would be protective of public health.

States must then make a site-specific determination for each system as to whether or not the system can afford to meet the MCL based on State-developed affordability criteria. If the State determines that compliance is not affordable for the system, it may grant a variance, but it must establish terms and conditions, as necessary, to ensure that the variance is adequately protective of human health.

In the Agency's draft national-level affordability criteria published in the August 6, 1998 *Federal Register*, EPA discussed the affordable treatment technology determinations for the contaminants regulated before 1996. The national-level affordability criteria were derived as follows. First an "affordability threshold" was calculated. The affordability threshold was based on the total annual household water bill as a percentage of household income. In developing this threshold value, EPA considered the percentage of median household income spent by an average household on comparable goods and services such items as housing (28 percent), transportation (16 percent), food (12 percent), energy and fuels (3.3 percent), telephone (1.9 percent), water and other public services (0.7 percent), entertainment (4.4 percent) and alcohol and tobacco (1.5 percent).

Another of the key factors that EPA used to select an affordability threshold was cost comparisons with other risk reduction activities for drinking water. Section 1412(b)(4)(E)(ii) of the SDWA identifies both point-of-entry and point-of-use devices as options for compliance technologies. EPA examined the projected costs of these options. EPA also investigated the costs associated with supplying bottled water for drinking and cooking purposes. The median income percentages that were associated with these risk reduction activities were: POE (> 2.5 percent), POU (2 percent) and bottled water (> 2.5 percent).

Based on the foregoing analysis, EPA developed an affordability criteria of 2.5 percent of median household income, or about \$750, for the affordability threshold (EPA, 1998). The median water bill for households in each small system category was then subtracted from this threshold to determine the additional expenditure per household that was considered affordable for new treatment. This difference is referred to as the "available expenditure margin." Based on EPA's 1995 Community Water System Survey, median water bills were about \$250 per year for small system customers. Thus, an average available expenditure margin of up to \$500 per year per household was considered affordable for the contaminants regulated before 1996. EPA next identified treatment technologies for all pre-1996 contaminants with average per household costs below \$500 per year. Therefore, it was not necessary to list any small system variance technologies for existing contaminant rules.

Applying this criterion to the case of arsenic in drinking water, EPA has determined that affordable technologies exist for all system size categories and has therefore not identified a variance technology for any system size or source water combination at an MCL of 10 µg/L (see Exhibit 8-4). In other words, annual household costs after installation of the compliance technology are projected to be below the available affordability threshold for all system size categories for MCLs of 3, 5, 10, and 20 µg/L.

**Exhibit 8-4  
Mean Annual Costs to Households Served by CWSs, by Size Category**

System Size	MCL (µg/L)			
	3	5	10	20
<100	\$317.00	\$318.26	\$326.82	\$351.15
101-500	\$166.91	\$164.02	\$162.50	\$166.72
501-1,000	\$74.81	\$73.11	\$70.72	\$68.24
1,001-3,300	\$63.76	\$61.94	\$58.24	\$54.36
3,301-10,000	\$42.84	\$40.18	\$37.71	\$34.63
10,001-50,000	\$38.40	\$36.07	\$32.37	\$29.05
50,001-100,000	\$31.63	\$29.45	\$24.81	\$22.63
100,001-1,000,000	\$25.29	\$23.34	\$20.52	\$19.26
>1,000,000	\$7.41	\$2.79	\$0.86	\$0.15
<b>All categories</b>	\$41.34	\$36.95	\$31.85	\$23.95

EPA recognizes that individual water systems may have higher than average treatment costs, fewer than average households to absorb these costs, or lower than average incomes, but believes that the affordability criteria should be based on characteristics of typical systems and should not address situations where costs might be extremely high or low or excessively burdensome. EPA believes that there are other mechanisms that may address these situations to a certain extent. In any case, EPA believes that small system variances should be the exception and not the rule.

EPA expects the available expenditure margin to be lower than \$500 per household per year for the Arsenic Rule because some sources of data, for example the Current Population Survey, indicate that water rates are currently increasing faster than median household income. Thus, the “baseline” for annual water bills will rise as treatment is installed for compliance with regulations promulgated after 1996, but before the Arsenic Rule is promulgated.

EPA notes, however, that high water costs are often associated with systems that have already installed treatment to comply with an NPDWR. Such in-place treatment facilities may facilitate compliance with future standards. EPA’s approach to establishing the national-level affordability criteria did not incorporate a baseline for in-place treatment technology. Assuming that systems with high baseline water costs would need to install a new treatment technology to comply with an NPDWR may thus overestimate the actual costs for some systems.

To investigate this issue, during the derivation of the national-level affordability criteria, EPA examined a group of five small surface water systems with annual water bills above \$500 per household per year. All of these systems had installed disinfection and filtration technologies to comply with the Surface Water Treatment Rule. If these systems were required to install

treatment to comply with the revised arsenic standard, modification of the existing processes would be much more cost-effective than adding a new technology. As a result, because these systems have already made the investment in treatment technology, and the cost is incorporated into current annual household water bills, costs to the household may not increase substantially.

Installing new technologies may interfere with in-place treatment or require additional treatment to address side effects, which will increase costs over the arsenic treatment technology base costs. For example, EPA assumed that CWSs would put corrosion control in place when the percent removal required was greater than 90 percent.

EPA believes that there is another mechanism in the SDWA to address cost impacts on small systems composed primarily of low-income households. Systems that meet criteria established by the State could be classified as disadvantaged communities under §1452(d) of the SDWA. They can receive additional subsidization under DWSRF, including forgiveness of principal. Under DWSRF, States must provide a minimum of 15 percent of the available funds for loans to small communities and have the option of providing up to 30 percent of the grant to provide additional loan subsidies to the disadvantaged systems, as defined by the State.

### **8.3 Coordination With Other Federal Rules**

Several Federal drinking water rules are under development involving treatment requirements that may relate to the treatment of arsenic for this drinking water rule. Although it is very difficult to determine how compliance with the Arsenic Rule might affect compliance with other drinking water regulations, the following briefly describes each rule, the impact the Arsenic Rule may have on that rule, and/or how each rule may impact the arsenic standard. The Arsenic Rule will be promulgated in a similar time frame as the Ground Water Rule, the Radon Rule, and the Microbial and Disinfection By-Product Rule.

#### **8.3.1 Ground Water Rule (GWR)**

The goals of the GWR are to: (1) provide a consistent level of public health protection; (2) prevent waterborne microbial disease outbreaks; (3) reduce endemic waterborne disease; and (4) prevent fecal contamination from reaching consumers. To ensure public health protection, EPA has the responsibility to develop a GWR that not only specifies the appropriate use of disinfection, but also addresses other components of ground water systems. This general provision is supplemented with an additional requirement that EPA develop regulations specifying the use of disinfectants for ground water systems as necessary. To meet these requirements, EPA is working with stakeholders to develop a final GWR by Spring 2001.

The GWR will result in more systems using disinfection. If a system does add a disinfection technology, it may contribute to arsenic pre-oxidation. This largely depends on the type of disinfection technology employed. For example, if a system chooses a technology such as ultraviolet radiation, it may not affect arsenic pre-oxidation. However, if it chooses chlorination, it will contribute to arsenic pre-oxidation. Arsenic pre-oxidation from arsenic (III) to arsenic (V) will enhance the removal efficiencies of the technologies. Another option is that systems may use membrane filtration for the GWR. In that case, depending on the size of the membrane,

some arsenic removal can be achieved. Thus, the GWR is expected to alleviate some of the burden of the Arsenic Rule.

### **8.3.2 Radon**

EPA proposed the Radon Rule in November 1999. One option for compliance with the Radon Rule that systems may employ is coagulation and assisted microfiltration. This technology will be sufficient to meet the revised arsenic standard as well. Thus, the Radon Rule is expected to alleviate some of the burden of the Arsenic Rule.

### **8.3.3 Microbial and Disinfection By-Product Regulations**

To control disinfection and disinfection by-products and to strengthen control of microbial pathogens in drinking water, EPA has developed a group of interrelated regulations, as required by the SDWA. These regulations, referred to collectively as the Microbial Disinfection By-product (M/DBP) Rules, are intended to address risk trade-offs between the two different types of contaminants.

EPA proposed a Stage 1 Disinfectants/Disinfection By-Products Rule (DBPR) and Interim Enhanced Surface Water Treatment Rule (IESWTR) in July 1994. EPA issued the final Stage 1 DBPR and IESWTR in November 1998.

The Agency has finalized and is currently implementing a third rule, the Information Collection Rule, that will provide data to support development of subsequent M/DBP regulations. These subsequent rules include a Stage 2 DBPR and a companion Long-Term 2 Enhanced Surface Water Treatment Rule (LT2ESWTR).

Stage 1 DBPR and IESWTR will primarily affect large surface water systems; thus, EPA does not expect much overlap with small systems treating for arsenic. Stage 2 DBPR and possibly the LT2ESWTR, however, could have significance as far as arsenic removal is concerned. For systems removing DBP precursors, systems may use nanofiltration. The use of nanofiltration would also be relevant for removing arsenic, and as a result, would ease some burden when systems implement these later rules.

## **8.4 Minimization of Economic Burden**

The revised Arsenic Rule includes several provisions that will insure that the economic burden to water systems is minimized, while still ensuring that the public health objectives of the rule are met. First, the rule is developed around the concept of a performance target known as the maximum contaminant level (MCL). Rather than prescribe a single treatment technique that must be installed in all water systems, EPA is only requiring those systems that currently provide finished water with an arsenic concentration above the target to undertake or modify treatment. As seen above, this will exclude the vast majority of systems from having to undertake any additional treatment under the revised Arsenic Rule. In addition, if a system does have to undertake or modify treatment, EPA is allowing systems to choose from a broad list of

technologies and is encouraging systems to choose the treatment technique that minimizes their total costs.

Second, EPA is allowing States to grant nine-year monitoring waivers to those systems that have a history of arsenic monitoring results below the revised MCL, and that do not show a substantial risk of future arsenic contamination. This provision of the rule will further reduce the cost to systems that currently provide finished water with low arsenic concentrations.

Finally, EPA is allowing small systems with finished water concentrations above the revised MCL to install POU technologies. This option will further allow small systems to minimize their total cost of compliance with the revised rule.

## **8.5 Unfunded Mandates Reform Act**

Title II of the Unfunded Mandates Reform Act of 1995 (UMRA), P.L. 104-4, establishes requirements for Federal agencies to assess the effects of their regulatory actions on State, local, and Tribal governments, and the private sector. Under UMRA Section 202, EPA generally must prepare a written statement, including a cost-benefit analysis, for proposed and final rules with “Federal mandates” that may result in expenditures to State, local, and Tribal governments, in the aggregate, or to the private sector, of \$100 million or more in any one year.

Before promulgating an EPA rule for which a written statement is needed, Section 205 of the UMRA generally requires EPA to identify and consider a reasonable number of regulatory alternatives and adopt the least costly, most cost-effective or least burdensome alternative that achieves the objectives of the rule. The provisions of Section 205 do not apply when they are inconsistent with applicable law. Moreover, Section 205 allows EPA to adopt an alternative other than the least costly, most cost-effective, or least burdensome alternative if the Administrator publishes an explanation why the more “costly” alternative was preferred for the final rule.

Prior to establishing any regulatory requirements that may significantly or uniquely affect small governments, including Tribal governments, EPA must develop a small government agency plan under Section 203 of the UMRA. The plan must provide for notifying potentially affected small governments; enabling officials of affected small governments to have meaningful and timely input in the development of EPA regulatory proposals with significant Federal intergovernmental mandates; and informing, educating, and advising small governments on compliance with the regulatory requirements.

EPA has determined that this rule contains a Federal mandate that may result in expenditures of \$100 million or more for State, local, and Tribal governments, in the aggregate and the private sector in any one year. Accordingly, under Section 202 of the UMRA, EPA is obligated to prepare a written statement addressing:

1. The authorizing legislation;
2. Cost-benefit analysis including an analysis of the extent to which the costs of State, local, and Tribal governments will be paid for by the Federal government;
3. Estimates of future compliance costs and disproportionate budgetary effects;
4. Macro-economic effects;
5. A summary of EPA's consultation with State, local, and Tribal governments and their concerns, including a summary of the Agency's evaluation of those comments and concerns; and
6. Identification and consideration of regulatory alternatives and the selection of the least costly, most cost-effective or least burdensome alternative that achieves the objectives of the rule.

The legislative authority for the Arsenic Rule is discussed in Chapter 2. Items two through five are addressed below, with the exception of future compliance costs, which are discussed in Chapter 6. Regulatory alternatives, the last item, are addressed in Chapters 3, 6, and 7.

### **8.5.1 Social Costs and Benefits**

Chapters 5, 6, and 7 contain a detailed cost-benefit analysis in support of the Arsenic Rule. At a seven percent discount rate, the Arsenic Rule is expected to have a total annualized cost of \$792.1 million for a MCL of 3 µg/L, \$471.7 million for a MCL 5 µg/L, \$205.6 million for a MCL of 10 µg/L, and \$76.5 million for a MCL of 20 µg/L.

EPA estimates that the Arsenic Rule will have total health benefits as a result of avoided bladder and lung cancer cases of approximately \$213.8 to \$490.9 million if the MCL were set at 3 µg/L, \$191.1 to \$355.6 million if the MCL were set at 5 µg/L, \$139.6 to \$197.7 million if the MCL were set at 10 µg/L, and \$66.2 to \$75.3 million if the MCL were set at 20 µg/L. These monetized health benefits of reducing arsenic exposures in drinking water are attributable to the reduced incidence of fatal and non-fatal bladder cancer and lung cancer. Currently under baseline assumptions (no control of arsenic exposure), there are annual fatal cancers and non-fatal cancers associated with arsenic exposures through CWSs. At an arsenic MCL level of 3 µg/L, an estimated 33 to 74 fatal cancers and 25 to 64 non-fatal cancers per year are prevented; at a arsenic level of 5 µg/L, an estimated 29 to 54 fatal cancers and 22 to 47 non-fatal cancers per year are prevented; at 10 µg/L, 21 to 30 fatal and 16 to 26 non-fatal cancers per year are prevented; and at 20 µg/L, 10 to 11 fatal and approximately 9 non-fatal cancers per year are prevented. A more detailed discussion of the total cancer risk and health benefits calculation may be found in Chapter 5, "Benefits Analysis."

In addition to quantifiable benefits, in Chapter 5, EPA has identified many potential non-quantifiable benefits associated with reducing arsenic exposures in drinking water. These potential benefits are not able to be quantified at this time, but may include reduced risk of skin

cancer and numerous non-cancerous health effects. In addition, certain non-health related benefits may exist, such as ecological improvements and an increase in consumers' perception of drinking water.

### **8.5.2 State Administrative Costs**

States will incur a range of administrative costs in complying with the Arsenic Rule. Administrative costs can include program management, inspections, and enforcement activities. EPA estimates that the total annual costs of State administrative activities for compliance with the MCL at a seven percent discount rate are approximately \$1.7 million for an MCL of 3 µg/L, \$1.4 million for an MCL of 5 µg/L, \$1.2 million for an MCL of 10µg/L, and \$1.0 million for an MCL of 20µg/L.

Various Federal programs exist to provide financial assistance to State, local, and Tribal governments in complying with this rule. The Federal government provides funding to States that have a primary enforcement responsibility for their drinking water programs through the Public Water Systems Supervision (PWSS) Grants program. Additional funding is available from other programs administered either by EPA or other Federal agencies. These include the Drinking Water State Revolving Fund (DWSRF) and Housing and Urban Development's Community Development Block Grant Program. For example, the SDWA authorizes the Administrator of the EPA to award capitalization grants to States, which in turn can provide low-cost loans and other types of assistance to eligible public water systems. The DWSRF also assists public water systems with financing the costs of infrastructure needed to achieve or maintain compliance with SDWA requirements. Each State will have considerable flexibility to determine the design of its program and to direct funding toward its most pressing compliance and public health protection needs. States may also, on a matching basis, use up to ten percent of their DWSRF allotments for each fiscal year to assist in running the State drinking water program.

Under PWSS Program Assistance Grants, the Administrator may make grants to States to carry out public water system supervision programs. One State use of these funds is to develop primacy programs. States may "contract" with other State agencies to assist in the development or implementation of their primacy program. However, States may not use program assistance grant funds to contract with regulated entities (i.e., water systems). PWSS Grants may be used by States to set up and administer a State program that includes such activities as public education, testing, training, technical assistance, development and administration of a remediation grant and loan or incentive program (excludes the actual grant or loan funds), or other regulatory or non-regulatory measures.

### **8.5.3 Future Compliance Costs and Disproportionate Budgetary Effects**

To meet the requirement in Section 202 of the UMRA, EPA analyzed future compliance costs and possible disproportionate budgetary effects of the MCL options. The Agency believes that the cost estimates, shown in Exhibit 8-5 and discussed in more detail in Chapter 6, accurately characterize future compliance costs of the revised rule.

With regard to the disproportionate impacts, EPA considered available data sources in analyzing the disproportionate impacts upon geographic or social segments of the nation or industry. To the extent that there may be disproportionate impacts to low-income or other segments of the population, EPA will prepare a small entity compliance guide, a monitoring/analytical manual, and a small systems technology manual that will assist the public and private sector. To fully consider the potential disproportionate impacts of this revised rule, EPA also developed three other measures:

- (1) Reviewing the impacts on small versus large systems;
- (2) Reviewing the costs to public versus private water systems; and
- (3) Reviewing the household costs for the revised rule.

The first measure, the national impacts on small versus large systems, is shown in Exhibit 8-5. Small systems are defined as those systems serving 10,000 people or less, and large systems are those systems serving more than 10,000 people.

The second measure of disproportionate impacts evaluated is the relative total costs to public versus private water systems, by size. Exhibit 8-5 also presents the annual system level costs for public and private systems by system size category for MCLs of 3  $\mu\text{g/L}$ , 5  $\mu\text{g/L}$ , 10  $\mu\text{g/L}$ , and 20  $\mu\text{g/L}$ . The costs are slightly lower for private systems across system sizes for all options. For example, for systems serving less than 100 people at the 10  $\mu\text{g/L}$  MCL public system costs are \$7,948, and private system costs are \$6,335.

**Exhibit 8-5  
Average Annual Cost per CWS Exceeding the MCL, by Ownership**

System Size	Treatment and Monitoring Costs		Total Cost
	Public	Private	All Systems
<b>MCL = 3 µg/L</b>			
<100	\$ 8,020	\$ 6,388	\$ 6,546
101-500	\$ 15,319	\$ 12,033	\$ 13,042
501-1,000	\$ 25,069	\$ 21,659	\$ 23,720
1,001-3,300	\$ 61,375	\$ 51,687	\$ 58,672
3,301-10,000	\$ 133,297	\$ 112,397	\$ 129,531
10,001-1,000,000	\$ 648,756	\$ 621,841	\$ 644,176
>1,000,000	\$ 10,360,933	--	\$ 10,360,933
<b>MCL = 5 µg/L</b>			
<100	\$ 8,065	\$ 6,384	\$ 6,551
101-500	\$ 14,845	\$ 11,762	\$ 12,712
501-1,000	\$ 24,406	\$ 21,175	\$ 23,146
1,001-3,300	\$ 59,998	\$ 49,055	\$ 56,911
3,301-10,000	\$ 124,483	\$ 103,388	\$ 120,621
10,001-1,000,000	\$ 601,335	\$ 584,831	\$ 598,488
>1,000,000	\$ 4,129,338	--	\$ 4,129,338
<b>MCL = 10 µg/L</b>			
<100	\$ 7,948	\$ 6,335	\$ 6,494
101-500	\$ 14,503	\$ 11,357	\$ 12,358
501-1,000	\$ 23,424	\$ 20,042	\$ 22,100
1,001-3,300	\$ 55,789	\$ 46,243	\$ 53,086
3,301-10,000	\$ 114,790	\$ 98,138	\$ 111,646
10,001-1,000,000	\$ 543,053	\$ 477,614	\$ 531,584
>1,000,000	\$ 1,340,716	--	\$ 1,340,716
<b>MCL = 20 µg/L</b>			
<100	\$ 7,785	\$ 6,209	\$ 6,361
101-500	\$ 13,814	\$ 11,065	\$ 11,902
501-1,000	\$ 21,733	\$ 18,877	\$ 20,595
1,001-3,300	\$ 51,116	\$ 42,869	\$ 48,779
3,301-10,000	\$ 105,155	\$ 85,201	\$ 101,374
10,001-1,000,000	\$ 482,300	\$ 443,463	\$ 475,909
>1,000,000	\$ 189,916	--	\$ 189,916

\*Costs were calculated at a commercial interest rate and include system treatment, monitoring, and administrative costs; note that systems serving over 1 million people are public surface water systems.

The third measure, household costs, can also be used to gauge the impact of a regulation and to determine whether there are disproportionately higher impacts in particular segments of the population. A detailed analysis of household cost impacts by system size is presented in Chapter 6. The costs for households served by public and private water systems are presented in Exhibit 8-6. As expected, cost per household increases as system size decreases. Cost per household is usually higher for households served by smaller systems than larger systems. This holds because smaller systems produce less water than large systems and are therefore unable to utilize economies of scale. Consequently, each household must bear a greater percentage share of the system's costs.

Exhibit 8-6 presents the costs per household for systems exceeding the MCL. For each size category there is a moderate difference in annual cost per household for 3  $\mu\text{g/L}$ , 5  $\mu\text{g/L}$ , 10  $\mu\text{g/L}$ , and 20  $\mu\text{g/L}$  across source and ownership. In general, costs per household are higher for private systems than for public systems. This difference could be attributable to a discrepancy in the cost of capital for public versus private entities. For public systems, the cost per household ranges from approximately \$5 to \$288 per year at 5  $\mu\text{g/L}$  and from approximately \$5 to \$285 per year at 10  $\mu\text{g/L}$  (excluding systems serving more than one million people). For private systems, the ranges are \$4 to \$317 per year, and \$4 to \$314 per year for an MCL of 5  $\mu\text{g/L}$  and 10  $\mu\text{g/L}$ , respectively.

To further evaluate the impacts of these household costs, the average costs per household were compared to median household income data for each system-size category. The result of this calculation, presented in Exhibit 8-7 for public and private systems, indicate a household's likely share of incremental costs in terms of its household income. For all system sizes and MCLs, average household costs as a percentage of median household income are less than one percent.

Among NTNCs, the average annual system cost ranges from approximately \$5,000 to \$39,000 at the revised MCL of 10  $\mu\text{g/L}$ . These results for systems exceeding the MCL are presented in Exhibit 8-8. At 3  $\mu\text{g/L}$ , 5  $\mu\text{g/L}$ , and 20  $\mu\text{g/L}$ , the average NTNC system cost ranges from \$5,000 to \$46,000, \$5,000 to \$43,000 and \$5,000 to \$35,000, respectively. More detail on the costs to NTNCs at these arsenic concentrations are presented in Chapter 6.

**Exhibit 8-6  
Annual Compliance Costs per Household for  
CWSs Exceeding MCLs**

System Size	Groundwater		Surface Water	
	Public	Private	Public	Private
<b>MCL = 3 µg/L</b>				
<100	\$ 285.93	\$ 319.62	\$ 218.47	\$ 231.50
101-500	\$ 134.47	\$ 190.51	\$ 54.75	\$ 72.52
501-1,000	\$ 79.11	\$ 76.64	\$ 15.22	\$ 13.98
1,001-3,300	\$ 64.50	\$ 84.32	\$ 5.77	\$ 7.52
3,301-10,000	\$ 45.79	\$ 65.42	\$ 3.74	\$ 4.33
10,001-1,000,000	\$ 40.77	\$ 39.67	\$ 5.39	\$ 4.62
>1,000,000	--	--	\$ 7.41	--
<b>MCL = 5 µg/L</b>				
<100	\$ 287.87	\$ 316.80	\$ 212.32	\$ 229.78
101-500	\$ 130.86	\$ 185.83	\$ 54.03	\$ 72.33
501-1,000	\$ 76.45	\$ 74.18	\$ 14.91	\$ 14.14
1,001-3,300	\$ 62.56	\$ 79.01	\$ 5.68	\$ 7.03
3,301-10,000	\$ 42.18	\$ 59.84	\$ 3.52	\$ 4.23
10,001-1,000,000	\$ 36.99	\$ 36.22	\$ 5.00	\$ 4.26
>1,000,000	--	--	\$ 2.79	--
<b>MCL = 10 µg/L</b>				
<100	\$ 285.03	\$ 314.11	\$ 214.23	\$ 229.02
101-500	\$ 126.46	\$ 180.21	\$ 52.72	\$ 71.01
501-1,000	\$ 72.51	\$ 69.87	\$ 14.23	\$ 13.93
1,001-3,300	\$ 56.76	\$ 73.42	\$ 5.51	\$ 6.81
3,301-10,000	\$ 38.08	\$ 55.35	\$ 3.13	\$ 4.03
10,001-1,000,000	\$ 31.72	\$ 30.78	\$ 4.55	\$ 3.99
>1,000,000	--	--	\$ 0.86	--
<b>MCL = 20 µg/L</b>				
<100	\$ 275.00	\$ 306.52	\$ 204.17	\$ 228.82
101-500	\$ 120.19	\$ 174.69	\$ 51.42	\$ 68.96
501-1,000	\$ 66.07	\$ 65.39	\$ 14.52	\$ 13.39
1,001-3,300	\$ 50.44	\$ 67.25	\$ 5.21	\$ 6.48
3,301-10,000	\$ 33.86	\$ 48.00	\$ 2.84	\$ 3.69
10,001-1,000,000	\$ 26.59	\$ 26.02	\$ 4.14	\$ -
>1,000,000	--	--	\$ 0.15	--

\*Costs to households were calculated at a commercial interest rate and include system treatment, monitoring, and administrative costs; note that systems serving over 1 million people are public surface water systems.

**Exhibit 8-7**  
**Annual Compliance Costs per Household for CWSs Exceeding MCLs,**  
**as a Percent of Median Household Income**

System Size	Groundwater		Surface Water	
	Public	Private	Public	Private
<b>MCL = 3 µg/L</b>				
<100	0.72%	0.81%	0.55%	0.58%
101-500	0.34%	0.48%	0.14%	0.18%
501-1,000	0.20%	0.19%	0.04%	0.04%
1,001-3,300	0.16%	0.21%	0.01%	0.02%
3,301-10,000	0.12%	0.17%	0.01%	0.01%
10,001-1,000,000	0.10%	0.10%	0.01%	0.01%
>1,000,000	--	--	0.02%	--
<b>MCL = 5 µg/L</b>				
<100	0.73%	0.80%	0.54%	0.58%
101-500	0.33%	0.47%	0.14%	0.18%
501-1,000	0.19%	0.19%	0.04%	0.04%
1,001-3,300	0.16%	0.20%	0.01%	0.02%
3,301-10,000	0.11%	0.15%	0.01%	0.01%
10,001-1,000,000	0.09%	0.09%	0.01%	0.01%
>1,000,000	--	--	0.01%	--
<b>MCL = 10 µg/L</b>				
<100	0.72%	0.79%	0.54%	0.58%
101-500	0.32%	0.45%	0.13%	0.18%
501-1,000	0.18%	0.18%	0.04%	0.04%
1,001-3,300	0.14%	0.19%	0.01%	0.02%
3,301-10,000	0.10%	0.14%	0.01%	0.01%
10,001-1,000,000	0.08%	0.08%	0.01%	0.01%
>1,000,000	--	--	0.00%	--
<b>MCL = 20 µg/L</b>				
<100	0.69%	0.77%	0.51%	0.58%
101-500	0.30%	0.44%	0.13%	0.17%
501-1,000	0.17%	0.16%	0.04%	0.03%
1,001-3,300	0.13%	0.17%	0.01%	0.02%
3,301-10,000	0.09%	0.12%	0.01%	0.01%
10,001-1,000,000	0.07%	0.07%	0.01%	0.00%
>1,000,000	--	--	0.00%	--

\*Costs to household were calculated at a commercial interest rate and include system treatment, monitoring, and administrative costs; median household income in May 1999 was \$39,648 updated from the 1998 annual median household income from the Census Bureau.

**Exhibit 8-8**  
**Total Annual NTNC Treatment Costs at MCL 10 µg/L by System Service Type**  
**(3% Discount Rate)**

Service Area Type	# of Systems Above the MCL	Average Population Served Per System	Average Annual System Cost	Annual National Costs
Daycare Centers	43	76	\$5,168	\$222,846
Highway Rest Areas	1	407	\$5,377	\$4,299
Hotels/Motels	19	133	\$5,956	\$111,420
Interstate Carriers	15	123	\$5,047	\$77,207
Medical Facilities	20	393	\$12,174	\$238,133
Mobile Home Parks	6	185	\$6,387	\$35,405
Restaurants	22	370	\$5,103	\$113,692
Schools	448	358	\$6,818	\$3,057,578
Service Stations	3	230	\$5,168	\$14,599
Summer Camps	2	146	\$6,124	\$15,014
Water Wholesalers	14	173	\$14,628	\$207,398
Agricultural Products/Services	20	76	\$6,012	\$117,930
Airparks	5	60	\$5,034	\$27,101
Construction	5	53	\$4,733	\$24,974
Churches	12	50	\$5,177	\$63,471
Campgrounds/RV Parks	7	160	\$6,104	\$40,017
Fire Departments	2	98	\$5,938	\$12,977
Federal Parks	1	39	\$5,245	\$5,592
Forest Service	6	42	\$4,783	\$27,278
Golf and Country Clubs	6	101	\$5,542	\$34,263
Landfills	4	44	\$5,176	\$21,517
Mining	6	113	\$5,572	\$35,340
Amusement Parks	8	418	\$5,848	\$49,558
Military Bases	5	395	\$9,095	\$46,053
Migrant Labor Camps	2	63	\$5,452	\$9,589
Misc. Recreation Services	14	87	\$5,027	\$69,397
Nursing Homes	7	107	\$7,298	\$50,567
Office Parks	51	136	\$5,310	\$268,864
Prisons	4	1,820	\$39,380	\$140,629
Retailers (Non-food related)	37	174	\$5,097	\$188,796
Retailers (Food related)	8	322	\$5,205	\$39,394
State Parks	4	165	\$5,153	\$22,794
Non-Water Utilities	26	170	\$5,627	\$149,069
Manufacturing: Food	41	372	\$7,566	\$309,707
Manufacturing: Non-Food	205	168	\$5,780	\$1,184,505
<b>TOTAL</b>	<b>1,080</b>			<b>\$7,036,973</b>

#### 8.5.4 Macroeconomic Effects

As required under UMRA Section 202, EPA is required to estimate the potential macro-economic effects of the regulation. These include effects on productivity, economic growth, full employment, creation of productive jobs, and international competitiveness. Macro-economic effects tend to be measurable in nationwide econometric models only if the economic impact of the regulation reaches 0.25 percent to 0.5 percent of Gross Domestic Product (GDP). In 1998, real GDP was \$7,552 billion; thus, a rule would have to cost at least \$18 billion annually to have a measurable effect. A regulation with a smaller aggregate effect is unlikely to have any measurable impact unless it is highly focused on a particular geographic region or economic sector. The macro-economic effects on the national economy from the Arsenic Rule should be negligible based on the fact that, assuming 100 percent compliance with an MCL, the total annual costs are approximately \$792 million at the 3 µg/L level, \$472 million at the 5 µg/L level, \$206 million at the 10 µg/L level, and \$77 million at the 20 µg/L level (at a seven percent discount rate).

#### 8.5.5 Consultation with State, Local, and Tribal Governments

Under UMRA section 204, EPA is to provide a summary of its consultation with elected representatives (or their designated authorized employees) of affected State, local, and Tribal governments in this rulemaking. EPA initiated consultations with governmental entities and the private sector affected by this rulemaking through various means. This included five stakeholder meetings announced in the *Federal Register* and open to anyone interested in attending in person or by phone, and presentations at meetings of the American Water Works Association (AWWA), the Association of State Drinking Water Administrators (ASDWA), the Association of California Water Agencies (ACWA), and the Association of Metropolitan Water Agencies (AMWA). Participants in EPA's stakeholder meetings also included representatives from the National Rural Water Association, AMWA, ASDWA, AWWA, ACWA, Rural Community Assistance Program, State departments of environmental protection, State health departments, State drinking water programs, and a Tribe. EPA also made presentations at Tribal meetings in Nevada, Alaska, and California.

To address the Arsenic Rule's impact on small entities, the Agency consulted with representatives of small water systems and convened a Small Business Advocacy Review Panel in accordance with the Regulatory Flexibility Act (RFA) as amended by the Small Business Regulatory Enforcement Fairness Act (SBREFA). Two of the small entity representatives were elected officials from local governments. EPA also invited State drinking water program representatives to participate in a number of workgroup meetings. In addition to these consultations, EPA participated in and gave presentations at AWWA's Technical Workgroup for Arsenic. State public health department and drinking water program representatives, drinking water districts, and ASDWA participated in the Technical Workgroup meetings. A summary of State, local, and Tribal government concerns on this rulemaking is shown in the next section.

In order to inform and involve Tribal governments in the rulemaking process, EPA staff attended the 16<sup>th</sup> Annual Consumer Conference of the National Indian Health Board on October 6-8, 1998, in Anchorage, Alaska. Over 900 attendees representing Tribes from across the country were in

attendance. During the conference, EPA conducted two workshops for meeting participants. The objectives of the workshops were to present an overview of EPA's drinking water program, solicit comments on key issues of potential interest in upcoming drinking water regulations, and to solicit advice in identifying an effective consultative process with Tribes for the future.

EPA, in conjunction with the Inter Tribal Council of Arizona (ITCA), also convened a Tribal consultation meeting on February 24-25, 1999, in Las Vegas, Nevada, to discuss ways to involve Tribal representatives, both Tribal council members and tribal water utility operators, in the stakeholder process. Approximately 25 representatives from a diverse group of Tribes attended the two-day meeting. Meeting participants included representatives from the following Tribes: Cherokee Nation, Nezperce Tribe, Jicarilla Apache Tribe, Blackfeet Tribe, Seminole Tribe of Florida, Hopi Tribe, Cheyenne River Sioux Tribe, Menominee Indian Tribe, Tulalip Tribes, Mississippi Band of Choctaw Indians, Narragansett Indian Tribe, and Yakama Nation.

The major meeting objectives were to:

- (1) Identify key issues of concern to Tribal representatives;
- (2) Solicit input on issues concerning current Office of Ground Water and Drinking Water regulatory efforts;
- (3) Solicit input and information that should be included in support of future drinking water regulations; and
- (4) Provide an effective format for Tribal involvement in EPA's regulatory development process.

EPA staff also provided an overview on the forthcoming Arsenic Rule at the meeting. The presentation included the health concerns associated with arsenic, EPA's current position on arsenic in drinking water, the definition of an MCL, an explanation of the difference between point-of-use and point-of-entry treatment devices, and specific issues for Tribes. The following questions were posed to the Tribal representatives to begin discussion on arsenic in drinking water:

- (1) What are the current arsenic levels in your water systems?
- (2) What are Tribal water systems' affordability issues in regard to arsenic?
- (3) Does your Tribe use well water, river water, or lake water?
- (4) Does your Tribe purchase water from another drinking water utility?

The summary for the February 24-25, 1999, meeting was sent to all 565 Federally recognized Tribes in the United States.

EPA also conducted a series of workshops at the Annual Conference of the National Tribal Environmental Council, which was held on May 18-20, 1999, in Eureka, California. Representatives from over 50 Tribes attended all, or part, of these sessions. The objectives of the workshops were to provide an overview of forthcoming EPA regulations affecting water systems; discuss changes to operator certification requirements; discuss funding for Tribal water systems; and discuss innovative approaches to regulatory cost reduction. Meeting summaries for EPA's Tribal consultations are available in the public docket for this rulemaking.

### **8.5.6 State, Local, and Tribal Government Concerns**

State and local governments raised several concerns, including the high costs of the rule to small systems; the burden of revising the State primacy program; the high degree of uncertainty associated with the benefits; and the high costs of including non-transient non-community water systems (NTNCWSs). EPA modified the revision of State primacy in order to decrease the burden of the revised arsenic regulation in response to State concerns, to minimize paperwork and documentation of existing programs that would manage the arsenic regulation.

Tribal representatives were generally supportive of regulations that would ensure a high level of water quality but raised concerns over funding for regulations. With regard to the revised Arsenic Rule, many Tribal representatives saw the health benefits as highly desirable, but felt that unless additional funds were made available, implementing the regulation would be difficult for many Tribes.

EPA understands the State, local, and Tribal government concerns with the above issues. The Agency believes the options for small systems in this rulemaking will address stakeholder concerns pertaining to small systems and will help to reduce the financial burden to these systems.

### **8.5.7 Regulatory Alternatives Considered**

As required under Section 205 of the UMRA, EPA considered several regulatory alternatives in developing an MCL for arsenic in drinking water. In preparation for this consideration, EPA evaluated arsenic levels of 3 µg/L, 5 µg/L, 10 µg/L, and 20 µg/L. EPA also evaluated national costs and benefits of States choosing to reduce arsenic exposure in drinking water. EPA believes that the regulatory approaches to arsenic described in the revised rule's preamble are the most appropriate to accomplish the SDWA objectives.

### **8.5.8 Impacts on Small Governments**

In developing this rule, EPA consulted with small governments pursuant to section 203 of the UMRA to address impacts of regulatory requirements in the rule that might significantly or uniquely affect small governments. In preparation for the revised Arsenic Rule, EPA conducted analysis on small government impacts and included small government officials or their designated representatives in the rulemaking process. EPA conducted stakeholder meetings on the development of the Arsenic Rule that gave a variety of stakeholders, including small governments, the opportunity for timely and meaningful participation in the regulatory development process. Groups such as the National Association of Towns and Townships, the National League of Cities, and the National Association of Counties participated in the rulemaking process. Through such participation and exchange, EPA notified potentially affected small governments of requirements under consideration during the development of the revised rule and provided officials of affected small governments with an opportunity to have meaningful and timely input into the development of the regulatory proposal.

In addition, EPA will educate, inform, and advise small systems, including those run by small governments, about the Arsenic Rule requirements. One of the most important components of this process is the Small Entity Compliance Guide, required by the Small Business Regulatory Enforcement Fairness Act of 1996 after the rule is promulgated. This plain-English guide will explain what actions a small entity must take to comply with the rule. Also, the Agency is developing fact sheets that concisely describe various aspects and requirements of the Arsenic Rule.

## **8.6 Effect of Compliance with the Arsenic Rule on the Technical, Financial, and Managerial Capacity of Public Water Systems**

Section 1420(d)(3) of the SDWA as amended requires that, in promulgating an NPDWR, the Administrator shall include an analysis of the likely effect of compliance with the regulation on the technical, financial, and managerial capacity of public water systems. The following analysis has been performed to fulfill this statutory obligation.

Overall water system capacity is defined in EPA guidance (EPA 816-R-98-006) (EPA 1998) as the ability to plan for, achieve, and maintain compliance with applicable drinking water standards. Capacity has three components: technical, managerial, and financial.

Technical capacity is the physical and operational ability of a water system to meet SDWA requirements. Technical capacity refers to the physical infrastructure of the water system, including the adequacy of source water and the adequacy of treatment, storage, and distribution infrastructure. It also refers to the ability of system personnel to adequately operate and maintain the system and to otherwise implement requisite technical knowledge. A water system's technical capacity can be determined by examining key issues and questions, including:

- Source water adequacy. Does the system have a reliable source of drinking water? Is the source of generally good quality and adequately protected?
- Infrastructure adequacy. Can the system provide water that meets SDWA standards? What is the condition of its infrastructure, including well(s) or source water intakes, treatment, storage, and distribution? What is the infrastructure's life expectancy? Does the system have a capital improvement plan?
- Technical knowledge and implementation. Is the system's operator certified? Does the operator have sufficient technical knowledge of applicable standards? Can the operator effectively implement this technical knowledge? Does the operator understand the system's technical and operational characteristics? Does the system have an effective operation and maintenance program?

Managerial capacity is the ability of a water system to conduct its affairs in a manner enabling the system to achieve and maintain compliance with SDWA requirements. Managerial capacity refers to the system's institutional and administrative capabilities. Managerial capacity can be assessed through key issues and questions, including:

- Ownership accountability. Are the system owner(s) clearly identified? Can they be held accountable for the system?
- Staffing and organization. Are the system operator(s) and manager(s) clearly identified? Is the system properly organized and staffed? Do personnel understand the management aspects of regulatory requirements and system operations? Do they have adequate expertise to manage water system operations? Do personnel have the necessary licenses and certifications?
- Effective external linkages. Does the system interact well with customers, regulators, and other entities? Is the system aware of available external resources, such as technical and financial assistance?

Financial capacity is a water system's ability to acquire and manage sufficient financial resources to allow the system to achieve and maintain compliance with SDWA requirements. Financial capacity can be assessed through key issues and questions, including:

- Revenue sufficiency. Do revenues cover costs? Are water rates and charges adequate to cover the cost of water?
- Credit worthiness. Is the system financially healthy? Does it have access to capital through public or private sources?
- Fiscal management and controls. Are adequate books and records maintained? Are appropriate budgeting, accounting, and financial planning methods used? Does the system manage its revenues effectively?

A complete technical, financial, and managerial capacity study is provided in the revised rule's preamble.

## **8.7 Paperwork Reduction Act**

The information collected as a result of this rule will allow the States and EPA to evaluate PWS compliance with the rule. For the first three years after promulgation of this rule, the major information requirements pertain to reading and understanding the rule and operator training. Responses to the request for information are mandatory (Part 141). The information collected is not confidential.

EPA is required to estimate the burden on PWSs for complying with the revised rule. Burden means the total time, effort, or financial resources expended by persons to generate, maintain, retain, disclose, or provide information to or for a Federal agency. This includes the time needed to review instructions; develop, acquire, install, and utilize technology and systems for the purposes of collecting, validating, and verifying information, processing and maintaining information, and disclosing and providing information; adjust the existing ways to comply with any previously applicable instructions and requirements; train personnel to be able to respond to a collection of information; search data sources; complete and review the collection of

information; and transmit or otherwise disclose the information. The Information Collection Rule for the revised Arsenic Rule estimated a total burden of 3.09 million hours for 10 µg/L.

## **8.8 Protecting Children from Environmental Health Risks and Safety Risks**

Executive Order (EO) 13045 (62 FR 19885, April 23, 1997) applies to any rule initiated after April 21, 1997, or proposed after April 21, 1998, that (1) is determined to be “economically significant” as defined under EO 12866 and (2) concerns an environmental health or safety risk that EPA has reason to believe may have a disproportionate effect on children. If the regulatory action meets both criteria, EPA must evaluate the environmental health or safety effects of the planned rule on children, and explain why the planned regulation is preferable to other potentially effective and reasonably feasible alternatives considered by EPA.

As described in Chapter 5 (“Benefits Analysis”), there are insufficient toxicological data to distinguish morbidity and mortality differences by age groups. No studies were located by ATSDR (1998) that focused exclusively on evaluating unusual susceptibility to arsenic. However, some members of the population are likely to be especially susceptible. For example, Chapter 5 describes several non-carcinogenic effects that may be of greater concern to children than adults, such as cardiovascular or reproductive effects. Similarly, arsenic has been suggested to pose significant problems in fetal development. This increased susceptibility may be due to a variety of factors. These factors include increased dose (intake per unit of body weight) in children, genetic predispositions, and dietary insufficiency (ATSDR, 1998), as well as pre-existing health conditions.

## **8.9 Environmental Justice**

Executive Order 12898 establishes a Federal policy for incorporating environmental justice into Federal agency missions by directing agencies to identify and address disproportionately high and adverse human health or environmental effects of its programs, policies, and activities on minority and low-income populations. The Executive Order requires the Agency to consider environmental justice issues in the rulemaking and to consult with Environmental Justice (EJ) stakeholders.

The Agency has considered environmental justice related issues concerning the potential impacts of this regulation and has determined that there are no substantial disproportionate effects. Because the Arsenic Rule applies to all community water systems, the majority of the population, including minority and low-income populations will benefit from the additional health protection.

## **8.10 Health Risk Reduction and Cost Analysis**

Section 1412(b)(3)(C) of the 1996 Amendments requires EPA to prepare a Health Risk Reduction and Cost Analysis (HRRCA) in support of any NPDWR that includes an MCL. According to these requirements, EPA analyzed each of the following in revising the Arsenic Rule:

1. Quantifiable and non-quantifiable health risk reduction benefits for which there is a factual basis in the rulemaking record to conclude that such benefits are likely to occur as the result of treatment to comply with each level;
2. Quantifiable and non-quantifiable health risk reduction benefits for which there is a factual basis in the rulemaking record to conclude that such benefits are likely to occur from reductions in co-occurring contaminants that may be attributed solely to compliance with the MCL, excluding benefits resulting from compliance with other proposed or promulgated regulations;
3. Quantifiable and non-quantifiable costs for which there is a factual basis in the rulemaking record to conclude that such costs are likely to occur solely as a result of compliance with the MCL, including monitoring, treatment, and other costs, and excluding costs resulting from compliance with other proposed or promulgated regulations;
4. The incremental costs and benefits associated with each alternative MCL considered;
5. The effects of the contaminant on the general population and on groups within the general population, such as infants, children, pregnant women, the elderly, individuals with a history of serious illness, or other sub-populations that are identified as likely to be at greater risk of adverse health effects due to exposure to contaminants in drinking water than the general population;
6. Any increased health risk that may occur as the result of compliance, including risks associated with co-occurring contaminants; and
7. Other relevant factors, including the quality and extent of the information, the uncertainties in the analysis, and factors with respect to the degree and nature of the risk.

This analysis summarizes EPA's estimates of the costs and benefits associated with various arsenic levels. The summary tables below characterize aggregate costs and benefits, impacts on affected entities, and tradeoffs between risk reduction and compliance costs.

### **8.10.1 Quantifiable and Non-Quantifiable Health Risk Reduction Benefits**

Arsenic ingestion has been linked to a multitude of health effects, both cancerous and non-cancerous. These health effects include cancer of the bladder, lungs, skin, kidney, nasal passages, liver, and prostate. Arsenic ingestion has also been attributed to cardiovascular, pulmonary, immunological, neurological, endocrine, and reproductive and developmental effects. A complete list of the arsenic-related health effects reported in humans is shown in Chapter 5. EPA has performed a risk assessment on bladder cancer and lung cancer. EPA then evaluated the health benefits attributable to these total cancer cases avoided.

The quantifiable health benefits of reducing arsenic exposures in drinking water are attributable to the reduced number of fatal and non-fatal bladder and lung cancers. The range of mean

bladder and lung cancer risks for exposed populations at or above arsenic levels of 3, 5, 10, and 20 µg/L in PWSs was described in Chapter 5. Exhibit 8-9 shows the health risk reductions (number of total bladder cancers avoided and the proportions of fatal and non-fatal bladder cancers avoided) at 3, 5, 10, and 20 µg/L, corresponding to the range of mean bladder cancer risks reported. Similarly, Exhibit 8-10 shows the total lung cancer cases avoided as a result of reduced arsenic exposure in PWSs. The sum of bladder cancer cases avoided and lung cancer cases avoided is shown in Exhibit 8-11.

**Exhibit 8-9**  
**Annual Bladder Cancer Cases Avoided from Reducing Arsenic in CWSs<sup>1</sup> and NTNCs**

Arsenic Level (µg/L)	Reduced Mortality Cases**	Reduced Morbidity Cases**	Total Bladder Cancer Cases Avoided*
3	7.4 - 20.0	21.2 - 56.9	28.6 - 76.8
5	6.6 - 14.5	18.9 - 41.2	25.6 - 55.7
10	4.9 - 8.0	13.8 - 22.7	18.7 - 31.0
20	2.6 - 2.8	7.3 - 7.8	9.9 - 10.6

\* The lower-end estimate of bladder cancer cases avoided is calculated using the lower-end risk estimate from Exhibit 5-9(c) and assumes that the conditional probability of mortality among the Taiwanese study group was 100 percent. The upper-end estimate of bladder cancer cases avoided is calculated using the upper-end risk estimate from Exhibit 5-9(c) and assumes that the conditional probability of mortality among the Taiwanese study group was 80 percent.

\*\*Assuming 20-year mortality rate in the U.S. of 26 percent.

\*\*\*Cases avoided from NTNCS are included.

**Exhibit 8-10**  
**Annual Lung Cancer Cases Avoided from Reducing Arsenic in CWSs and NTNCs**

Arsenic Level (µg/L)	Reduced Mortality Cases**	Reduced Morbidity Cases**	Total Lung Cancer Cases Avoided*
3	25.2 - 54.1	3.4 - 7.4	28.6 - 61.5
5	22.5 - 39.2	3.1 - 5.3	25.6 - 44.5
10	16.4 - 21.8	2.2 - 3.0	18.7 - 24.8
20	7.4 - 8.7***	1.0 - 1.2***	8.5 - 9.9***

\* The lower and upper-end estimates of lung cancer cases avoided are calculated using the risk estimates from Exhibit 5-9 (c) and assume that the conditional probability of mortality among the Taiwanese study group was 100 percent.

\*\*Assuming 20-year mortality rate in the U.S. of 88 percent.

\*\*\*For 20 ppb, the proportional reduction from the lower level risk base case is greater than the proportional reduction from the higher level risk base case. Thus the number of estimated cases avoided is higher at 20 using the estimates adjusted for uncertainty.

\*\*\*\*cases avoided from NTNCS are included.

**Exhibit 8-11**

**Annual Total Cancer Cases Avoided from Reducing Arsenic in CWSs and NTNCs**

<b>Arsenic Level (µg/L)</b>	<b>Reduced Mortality Cases**</b>	<b>Reduced Morbidity Cases**</b>	<b>Total Cancer Cases Avoided*</b>
3	32.6 - 74.1	24.6 - 64.2	57.2 - 138.3
5	29.1 - 53.7	22.0 - 46.5	51.1 - 100.2
10	21.3 - 29.8	16.1 - 25.9	37.4 - 55.7
20	10.2 - 11.3***	8.5 - 8.8	19.0 - 19.8***

\* The lower-end estimate of bladder cancer cases avoided and the lung cancer estimates assume that the conditional probability of mortality among the Taiwanese study group was 100 percent. The upper-end estimate of bladder cancer cases avoided is calculated using the assumption that the conditional probability of mortality among the Taiwanese study group was 80 percent.

\*\*Assuming 20-year mortality rate in the U.S. of 26 percent for bladder cancer and 88 percent for lung cancer.

\*\*\*For 20 ppb, the proportional reduction from the lower level risk base case is greater than the proportional reduction from the higher level risk base case. Thus the number of estimated cases avoided is higher at 20 using the estimates adjusted for uncertainty.

\*\*\*\*Cases avoided from NTNCs are included.

The Agency developed monetized estimates of the health benefits associated with the risk reductions from arsenic exposures. The approach used in this analysis for the measurement of health risk reduction benefits is the monetary value of a statistical life (VSL) applied to each fatal cancer avoided. For non-fatal cancers, willingness to pay (WTP) data to avoid chronic bronchitis is used as a surrogate to estimate the WTP to avoid non-fatal bladder cancers. A WTP central tendency estimate of \$607,162 (May 1999\$) is used to monetize the benefits of avoiding non-fatal cancers (this value was updated from the \$536,000 value EPA updated to 1997 dollars from the Viscusi et al. 1991 study).

The total national costs of the revised Arsenic Rule are summarized in Exhibit 8-12, along with the annual bladder cancer and lung cancer health benefits, and any non-quantifiable health benefits from other arsenic health effects. Total annual health benefits resulting from bladder cancer cases avoided range from \$58.2 to \$156.4 million at an MCL of 3 µg/L, \$52.0 to \$113.3 million at an MCL of 5 µg/L, \$38.0 to \$63.0 million at an MCL of 10 µg/L, and \$20.1 to \$21.5 million at an MCL of 20 µg/L. Total annual health benefits resulting from lung cancer cases avoided range from \$155.6 to \$334.5 million at an MCL of 3 µg/L, \$139.1 to \$242.3 million at an MCL of 5 µg/L, \$101.6 to \$134.7 million at an MCL of 10 µg/L, and \$46.1 to \$53.8 million at an MCL of 20 µg/L.

**Exhibit 8-12**  
**Total Annual Cost, Estimated Monetized Total Cancer Health Benefits and**  
**Non-Quantifiable Health Benefits from Reducing Arsenic in PWSs**  
**(\$ millions)**

Arsenic Level (µg/L)	Total Annual Cost (7%)	Annual Bladder Cancer Health Benefits <sup>1,2</sup>	Annual Lung Cancer Health Benefits <sup>1,2</sup>	Total Annual Health Benefits <sup>1,2</sup>	Potential Non-Quantifiable Health Benefits
3	\$792.1	\$58.2 - \$156.4	\$155.6 - \$334.5	\$213.8 - \$490.9	<ul style="list-style-type: none"> <li>• Skin Cancer</li> <li>• Kidney Cancer</li> <li>• Cancer of the Nasal Passages</li> <li>• Liver Cancer</li> <li>• Prostate Cancer</li> <li>• Cardiovascular Effects</li> <li>• Pulmonary Effects</li> <li>• Immunological Effects</li> <li>• Neurological Effects</li> <li>• Endocrine Effects</li> <li>• Reproductive and Developmental Effects</li> </ul>
5	\$471.7	\$52.0 - \$113.3	\$139.1 - \$242.3	\$191.1 - \$355.6	
10	\$205.6	\$38.0 - \$63.0	\$101.6 - \$134.7	\$139.6 - \$197.7	
20	\$76.5	\$20.1 - \$21.5	\$46.1 - \$53.8	\$66.2 - \$75.3 <sup>3</sup>	

<sup>1</sup> May 1999 dollars.

<sup>2</sup> These monetary estimates are based on cases avoided given in Exhibit 5-9 (a-c).

<sup>3</sup> For 20 µg/L, the proportional reduction from the lower level risk base case is greater than the proportional reduction from the higher level risk base case. Thus the number of estimated cases avoided and estimated benefits are higher at 20 µg/L using the estimates adjusted for uncertainty.

Reductions in arsenic exposures may also be associated with non-quantifiable benefits. EPA has identified several potential non-health non-quantifiable benefits associated with regulating arsenic in drinking water. These benefits may include any customer peace of mind from knowing that their drinking water has been treated for arsenic. To the extent that the Arsenic Rule can reduce households' perception of the health risks associated with arsenic in drinking water, household averting actions and costs to avoid these risks, such as buying bottled water or installing home treatment systems, could also be reduced.

### 8.10.2 Quantifiable and Non-Quantifiable Costs

The costs of reducing arsenic to various levels are summarized in Exhibit 8-13, which shows that as expected, aggregate arsenic mitigation costs increase with decreasing arsenic levels. Total national costs at a seven percent discount rate range are \$792.1 million per year at 3 µg/L; \$471.1 million per year at 5 µg/L; \$205.6 million per year at 10 µg/L; \$76.5 million per year at 20 µg/L.

**Exhibit 8-13**  
**Summary of the Total Annual National Costs of Compliance**  
**(\$ millions)**

Discount Rate	CWS		NTNC		TOTAL	
	3%	7%	3%	7%	3%	7%
<b>MCL = 3 µg/L</b>						
System Costs						
Treatment	\$665.9	\$756.5	\$27.2	\$29.6	\$693.1	\$786.0
Monitoring/ Administrative	\$2.2	\$3.0	\$1.0	\$1.4	\$3.2	\$4.4
State Costs	\$1.4	\$1.6	\$0.1	\$0.2	\$1.5	\$1.7
TOTAL COST	\$669.4	\$761.0	\$28.3	\$31.1	\$697.8	\$792.1
<b>MCL = 5 µg/L</b>						
System Costs						
Treatment	\$394.4	\$448.5	\$16.3	\$17.6	\$410.6	\$466.1
Monitoring/ Administrative	\$2.0	\$2.8	\$1.0	\$1.3	\$2.9	\$4.1
State Costs	\$1.1	\$1.3	\$0.1	\$0.2	\$1.2	\$1.4
TOTAL COST	\$397.5	\$452.5	\$17.3	\$19.1	\$414.8	\$471.7
<b>MCL = 10 µg/L</b>						
System Costs						
Treatment	\$169.6	\$193.0	\$7.0	\$7.6	\$176.7	\$200.6
Monitoring/ Administrative	\$1.8	\$2.5	\$0.9	\$1.3	\$2.7	\$3.8
State Costs	\$0.9	\$1.0	\$0.1	\$0.2	\$1.0	\$1.2
TOTAL COST	\$172.3	\$196.6	\$8.1	\$9.1	\$180.4	\$205.6
<b>MCL = 20 µg/L</b>						
System Costs						
Treatment	\$60.7	\$69.0	\$2.6	\$2.8	\$63.3	\$71.8
Monitoring/ Administrative	\$1.7	\$2.4	\$0.9	\$1.3	\$2.6	\$3.7
State Costs	\$0.7	\$0.8	\$0.1	\$0.2	\$0.9	\$1.0
TOTAL COST	\$63.2	\$72.3	\$3.6	\$4.2	\$66.8	\$76.5

EPA also assessed the cost impact of reducing arsenic in drinking water at the household level. Exhibit 8-14 examines the cost per household for each system size category. As shown in the table, costs per household decrease as system size increases. However, costs per household do not vary significantly across arsenic levels. This is because costs do not vary significantly with removal efficiency; once a system installs a treatment technology to meet an MCL, costs based upon the removal efficiency that the treatment technology will be operated under remain relatively flat. Usually, per household costs are, however, somewhat lower at less stringent arsenic levels. This is due to the assumption that some systems would blend water at these levels and treat only a portion of the flow in order to meet the target MCL. However, in the smallest two size categories, average household costs decrease as the standard becomes more stringent. This somewhat counterintuitive result is due to the \$500.00 affordability cap assumed in the SafeWater XL simulations. As more CWSs are forced over the affordability cap, the systems' costs are fixed at the costs associated with the POU technology. This results in lower average household costs for these systems.

**Exhibit 8-14  
Mean Annual Costs per Household in CWSs**

System Size	MCL (µg/L)			
	3	5	10	20
<b>&lt;100</b>	\$317.00	\$318.26	\$326.82	\$351.15
<b>101-500</b>	\$166.91	\$164.02	\$162.50	\$166.72
<b>501-1,000</b>	\$74.81	\$73.11	\$70.72	\$68.24
<b>1,001-3,300</b>	\$63.76	\$61.94	\$58.24	\$54.36
<b>3,301-10,000</b>	\$42.84	\$40.18	\$37.71	\$34.63
<b>10,001-50,000</b>	\$38.40	\$36.07	\$32.37	\$29.05
<b>50,001-100,000</b>	\$31.63	\$29.45	\$24.81	\$22.63
<b>100,001-1,000,000</b>	\$25.29	\$23.34	\$20.52	\$19.26
<b>&gt;1,000,000</b>	\$7.41	\$2.79	\$0.86	\$0.15
<b>All categories</b>	\$41.34	\$36.95	\$31.85	\$23.95

Exhibit 8-15 illustrates the cost per bladder cancer case avoided, based on national cost estimates which include all the costs of treatment, O&M, monitoring and administrative costs to CWSs and NTNCs, and all State start-up costs and State costs for administration of water programs. At a three percent discount rate, cost per case ranges from approximately \$12.2 million at an arsenic level of 3 µg/L (lower bound estimate of avoided bladder cancer cases) to \$3.4 million at an MCL of 20 µg/L (upper bound of avoided bladder cancer cases). Similarly, the range at a seven percent discount rate is \$13.8 million to \$3.9 million.

**Exhibit 8-15  
Cost per Cancer Case Avoided  
(\$ millions)**

Arsenic Level (µg/L)	lower bound**		upper bound**	
<b>3% Discount Rate</b>				
<b>3</b>	\$	12.2	\$	5.0
<b>5</b>	\$	8.1	\$	4.1
<b>10</b>	\$	4.8	\$	3.2
<b>20</b>	\$	3.5	\$	3.4
<b>7% Discount Rate</b>				
<b>3</b>	\$	13.8	\$	5.7
<b>5</b>	\$	9.2	\$	4.7
<b>10</b>	\$	5.5	\$	3.7
<b>20</b>	\$	4.0	\$	3.9

\*\*Lower/upper bounds correspond to estimates of bladder cancer cases avoided.

# Appendix A: Decision Tree and Large System Costs

## A.1 Introduction

The purpose of this appendix is to present the rationale behind the development of the decision tree and associated decision matrix. It includes an overview of the decision tree structure and major factors impacting the decision-making process. The following list outlines the contents of this appendix:

- **Background** - Presents a brief history of the arsenic regulation and the statutory requirements impacting EPA and the decision-making process.
- **Major Factors Affecting the Decision Tree** - Presents the rationale for selecting parameters which impact the decision tree, including MCL, population, water type, region, and co-occurrence of solutes.
- **Additional Factors Affecting the Decision Tree** - Presents other parameters in the process which impact the decision tree, including: corrosion control, pre-oxidation, regionalization, and alternative technologies.
- **Development of a Decision Tree** - Presents the logic used for developing the decision tree for treatment of arsenic to a final revised MCL of 10 µg/L.
- **Very Large System Methodology** - Discusses the cost estimates for the Nation's 25 largest drinking water systems.

## A.2 Background

In 1998 and 1999, EPA conducted technology and cost evaluations for the removal of arsenic from drinking water. These evaluations looked into the effectiveness of various removal technologies and the capital and operations and maintenance (O&M) costs associated with each process. The following were evaluated and determined effective to varying degrees:

- Modified Coagulation/Filtration (modifications to existing C/F plants);
- Coagulation Assisted Microfiltration (CMF);
- Modified Lime Softening (modifications to existing LS plants);
- Activated Alumina (AA);
- Ion Exchange (IX);
- Greensand Filtration (GF); and
- Point-of-Use (POU) Treatment Options.

The technology and cost evaluation yielded a document entitled *Technologies and Costs for the Removal of Arsenic From Drinking Water* (EPA, 2000c). The document includes detailed evaluations of the above technologies, capital and O&M cost estimates for each of the listed technologies, as well as other technologies that were considered ineffective or unproven.

EPA used the information contained in the technologies and costs (T&C) document to develop a regulatory decision tree. The decision tree was then used to fashion a decision matrix which

contains the probability that a given system will choose a treatment technology based on the percent removal required to meet the final revised MCL of 10 µg/L . The decision matrix, unit cost curves for treatment and waste disposal (illustrated in the T&C), treatment-in-place data and occurrence estimates were used to develop national cost of compliance estimates.

### **A.3 Major Factors Affecting the Decision Tree**

This section explains the rationale behind selecting each particular decision factor. Specifically, this section will discuss the following:

- the MCL target;
- influent arsenic concentration;
- population;
- region where the system is located;
- source water;
- whether a system has existing treatment in place;
- co-occurrence of solutes; and
- waste disposal issues.

#### **A.3.1 MCL Target**

Target treatment concentration (8 µg/L) which is equal to 80 percent of the final revised MCL of 10 µg/L was selected as the basis for the development of the Arsenic Rule decision tree. The selection of a target treatment concentration was the first step in the decision process and was essential for determining all other branches of the decision tree.

#### **A.3.2 Influent Arsenic Concentration**

Given the MCL, the influent arsenic concentration determines what percent removal of arsenic is needed, if any, and lays the groundwork for remaining decisions in the tree; therefore, the influent arsenic concentration was of major importance in developing the decision tree. Given the maximum influent arsenic level of 50 µg/L and at the final MCL of 10 µg/L, no systems would need to have a removal efficiency greater than 90 percent to treat for arsenic. Percent removal is critical for determining what additional technologies may be feasible. For example, if a ground water system has an influent arsenic level of 50 µg/L, and the target treatment concentration is 8 µg/L, then approximately 80 percent removal is required.<sup>1</sup>

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<sup>1</sup>Required removal percentages in the decision tree are based on worst cast scenarios and therefore correspond with the upper bound of the arsenic concentration range for each category.

### **A.3.3 System Size**

System size, or population, also plays a significant role in determining the treatment options available to a system, as well as the affordability of a particular technology. EPA established nine size categories to be used in the decision tree and EA process:

- 25 to 100;
- 101-500;
- 501-1,000;
- 1,001-3,300;
- 3,301-10,000;
- 10,001-50,000;
- 50,001-100,000;
- 100,001-1,000,000; and
- greater than 1,000,000.

Exceptions were made in the decision tree for particular systems. The Agency considered point-of-use (POU) treatment as a viable option only for the two smallest categories of groundwater systems. Systems serving greater than 1,000,000 were addressed on a case-by-case basis by EPA, and therefore, were not considered within the scope of the decision tree process.

In developing the probability of choosing a given technology for each of the size categories, the Agency considered several factors such as available data on in-place treatments from Community Water System Survey (CWSS). The logic used for developing the probabilities for each of the size categories is detailed in section A.5 below.

### **A.3.4 Region**

The region of the nation that a system resides in does not effect the treatment options available. Therefore, the decision tree is structured in such a way that, regardless of the region, the branches are identical, and in fact refer to the same pages within the decision tree. However, the number of systems that may select a particular option as defined in the decision matrix, is region-specific.

EPA has decided that the nation can be divided into three regions for the purpose of the decision making process: 1) Southwest Region; 2) Northwest Region; and 3) East Region. The regions were selected based upon availability of water (i.e., scarcity of water) and availability of land. In the Southwest Region, for example, water may be scarce and treatment technologies that generate large volumes of reject water, such as RO, may not be appropriate. In the East Region, water scarcity is much less a concern than the availability of land. Technologies or disposal options that require significant amounts of land are less likely to be utilized in the East Region. The Northwest Region, by comparison, is less affected by the scarcity of water or land availability than either of the other two regions.

### **A.3.5 Source Water**

The source of the system's raw water, either ground water or surface water, plays a major role in determining the technologies that may already be in use by a system and what treatment options are available if a system needs to install a new facility.

For example, greensand filtration is affected by the level of iron in the raw water. Influent levels greater than 300 mg/L (ppm) are conducive to removal of arsenic by greensand filtration. Surface waters typically have low iron content, whereas ground waters often have levels in excess of 300 mg/L (Subramanian, et al., 1997). Accordingly, greensand filtration was not considered a viable removal technology for surface water systems, but is viable for ground water systems.

To determine the types of treatment that are currently being utilized throughout the country by source, EPA reviewed the Community Water Systems Survey (CWSS). EPA determined there are few surface water systems utilizing RO, IX or AA. As a result, when approximating the treatment in place options, RO, IX, and AA were omitted for surface water systems.

Arsenic removal is significantly more efficient when arsenic is present as arsenate ( $\text{As}^{5+}$ ). Research has demonstrated many of the technologies considered perform poorly when arsenite ( $\text{As}^{3+}$ ) is the predominant form (EPA, 2000). Arsenite can be easily oxidized to arsenate using conventional oxidation methods, such as chlorination and potassium permanganate addition. Ground waters typically contain higher levels of  $\text{As}^{3+}$ , whereas  $\text{As}^{5+}$  is the dominant species in surface waters. As a result, ground water systems are more likely to install pre-oxidation and use higher oxidant doses, whereas surface water systems may be able to get by with little or no pre-oxidation capacity.

### **A.3.6 Systems with Treatment In-Place**

Information on in-place treatment technologies for all the flow categories of surface and ground water systems was obtained from Table 6.2 of "Geometries and Characteristics of Public Water Systems (EPA, 1999b)." The Agency determined that many existing treatment facilities will be able to achieve the necessary arsenic removal with little or no modification to their plant. Exhibit A- 1 below outlines the treatment technologies included in the decision tree, the percent removal assumed capable without modification or polishing, and the maximum percent removal.

### **A.3.7 Systems without Treatment In-Place**

Many factors affect the decision tree when considering the addition of a treatment option for systems with no current treatment in place. Source water type and quality, system size, required arsenic removal, and removal achievable by a particular technology are all major considerations. Many of these considerations have been discussed earlier in Section 4.

For ground water systems without treatment in-place, the most suitable treatment technologies are IX and AA. For surface water systems with no treatment in-place, AA with and without pH

adjustment and coagulation microfiltration are the most suitable. Modified CF and LS are for those surface water systems that already have CF or LS in-place.

The SDWA identifies POE and POU treatment units as potentially affordable technologies, but stipulates that POE and POU treatment systems “shall be owned, controlled and maintained by the public water system, or by a person under contract with the public water system to ensure proper operation and compliance with the maximum contaminant level or treatment technique and equipped with mechanical warnings to ensure that customers are automatically notified of operational problems.”

Preliminary affordability determinations have shown that POU technologies will only be considered viable for small systems. These determinations have shown the cost breakpoint to be in the area of 200 persons served. This estimate does not account for waste disposal costs, which would make central treatment estimates more expensive, thus increasing the breakpoint. As a result only POU AA and POU RO compliance strategies were included in the decision tree for the groundwater systems in the two smallest flow categories.

**Exhibit A-1  
Treatment Technologies for Systems with Treatment In-Place and Percent Removals Assumed and Achievable**

Treatment Technology	Percent Removal of In-Place System	Maximum Percent Removal <sup>1</sup>
Coagulation/Filtration <sup>2</sup>	50	95
Lime Softening <sup>2</sup>	50	90
Coagulation Assisted Microfiltration	NA	90
Ion Exchange	NA	>95
Activated Alumina	NA	>95
Reverse Osmosis	NA	>95
Greensand Filtration <sup>9</sup>	NA	80
POU Activated Alumina	NA	>90
POU Reverse Osmosis	NA	>90

1 - For Percent Removals of In-Place Systems that are very close to Maximum Percent Removals (e.g., 95 percent and > 95 percent) polishing steps may be required.

2 - Maximum Percent Removal involves modification to existing system in the form of additional chemical feed systems, pumping, piping, etc.

NA - Not Applicable

### A.3.8 Co-Occurrence of Solutes

There are a number of solutes and water quality parameters that may effect the viability of a particular treatment option. Total dissolved solids (TDS), silica, sulfate and iron can all be major

detractors/benefactors for the use of a particular technology. The decision tree simply cannot account for each individual situation where the influent water quality plays a role in selecting the treatment option. Utilities are encouraged to read the T&C document (EPA, 2000d) to gather additional information on parameters which impact the performance of a particular technology.

The decision tree uses influent sulfate and iron levels as decision factors in selecting treatment technologies. For ground water sources, both sulfate and iron levels are considered. Ion exchange is not considered a feasible treatment option when sulfate levels exceed 50 mg/L and greensand filtration is not considered viable when the iron level falls below 300 mg/L. Sulfate has been shown to decrease the effectiveness of ion exchange processes for arsenic removal; therefore, an upper bound sulfate concentration of 50 mg/L was used in the final rule for determination of ion exchange usage. Iron, on the other hand, significantly improves the effectiveness of greensand filtration (Subramanian, et al., 1997). Greensand filtration is best suited for ground waters (which typically contain higher levels of iron than surface waters) with high influent levels of iron (300 mg/L). For purposes of approximating national cost, greensand filtration is not considered a treatment option for surface water systems.

### **A.3.9 Waste Disposal**

Waste handling and disposal options are specific to the treatment technology selected, therefore the availability of disposal options does not vary by system size in the decision tree. However, the probability that a system will utilize a particular option does vary with system size.

#### **A.3.9.1 Mechanical Dewatering**

Mechanical dewatering processes include centrifuges, vacuum-assisted dewatering beds, belt filter presses, and plate and frame filter presses. Such processes generally have high capital, as well as high O&M costs, compared to similar capacity non-mechanical dewatering processes (e.g., storage lagoons). Due to the high costs, such processes are generally not suitable for very small water systems.

Filter presses have been used in industrial processes for years and have been increasing in the water treatment industry over the past several years. The devices have been successfully applied to both lime softening process sludge and coagulation/filtration process sludge. Filter presses require little land, have high capital costs, and are labor intensive.

Centrifuges have also been used in the water industry for years. Centrifugation is a continuous process requiring minimal time to achieve the optimal coagulation/filtration. Centrifuges have low land requirements and high capital costs. They are more labor intensive than non-mechanical alternatives, but less intensive than filter presses. Again, due to the capital and O&M requirements, centrifuges are more suitable for larger water systems.

### **A.3.9.2 POTW Discharge**

Indirect discharge (POTW discharge) is a commonly used method of disposal for filter backwash and brine waste streams. Coagulation/filtration and lime softening sludge materials have also been successfully disposed of in this manner. The primary cost associated with POTW discharge is that of the piping. Additional costs associated with POTW discharge may include lift stations, additional piping for access to the sewer system, and any cost incurred by the POTW in accommodating the increased demands on the POTW.

### **A.3.9.3 Sanitary Landfill Disposal**

Two forms of sanitary landfill are commonly used for disposal of water treatment byproducts: monofills and commercial nonhazardous waste landfills. In some parts of the country, decreasing landfill availability, rising costs, and increasing regulations are making landfill disposal more expensive. Costs associated with the development of monofills are generally less than those associated with commercial nonhazardous water landfill.

## **A.4 Additional Factors Affecting the Decision Tree**

### **A.4.1 Pre-Oxidation**

As mentioned above, inorganic arsenic occurs in two primary valence states, arsenite (As III) and arsenate (As V). As(III) is dominant in ground waters while surface waters more typically contain As(V). As(III) is easily oxidized to As(V) by conventional oxidation technologies such as chlorination and potassium permanganate addition. Each of the treatment technologies considered in the decision tree remove As(V) more readily than As(III) and as a result may require pre-oxidation.

In estimating national costs, it was assumed that only systems without pre-oxidation in-place would add the necessary equipment. It is expected that no surface water systems will need to install pre-oxidation for arsenic removal and that about fewer than 50 percent of the groundwater systems may need to install pre-oxidation for arsenic removal. Ground water systems without pre-oxidation should determine if pre-oxidation is necessary by determining if the arsenic is present as As (III) or As (V). Ground water systems with predominantly As (V) will probably not need pre-oxidation to meet the MCL. For single tap (POU) treatment options, centralized pre-oxidation is required. Exhibit A-2 shows the number of systems that were assumed to require addition of pre-oxidation.

## Exhibit A-2: Systems Needing to Add Pre-Oxidation

System Size	Percent of Ground Water Systems
25-100	54
101-500	30
501-1000	24
1001-3300	24
3300-10K	27
10001-50K	13
50,001-100K	41
100,001-1M	16

### A.4.2 Corrosion Control

Many of the treatment technologies considered in the decision tree (e.g. AA, and IX) remove hardness and alkalinity. Removal of hardness and alkalinity can reduce the pH of finished water and lead to corrosion problems within the system. Hardness and alkalinity, at the appropriate levels, act as buffers against corrosion in the treatment plant and distribution system. At these levels, alkalinity and hardness form protective coatings (metal hydroxides), control pH and enhance the buffer effect against corrosion. Where appropriate, corrosion control costs were included with arsenic treatment in the decision tree. It was assumed that the in-place lime softening and coagulation/flocculation plants had adequate corrosion control in-place.

### A.4.3 Alternative Technologies

*Technologies and Costs for the Removal of Arsenic from Drinking Water* (EPA, 2000) evaluated four arsenic removal technologies that were not included in the decision tree:

- Sulfur-Modified Iron,
- Granular Ferric Hydroxide,
- Iron Filings, and
- Iron Oxide Coated Sand.

The technologies were not included in the decision tree for reasons which are summarized below.

#### A.4.3.1 Sulfur-Modified Iron

A patented Sulfur-Modified Iron (SMI) process for arsenic removal has recently been developed. During this process, powdered iron, powdered sulfur, and the oxidizing agent ( $H_2O_2$  in preliminary tests) are thoroughly mixed and added to the water to be treated. The oxidizing agent serves to convert As(III) to As(V). Arsenic removal utilizing the SMI process seems to be

dependent on the iron to arsenic level as well as pH. Flow distribution problems were evident, as several columns became partially plugged during operation.

All experimentation on the SMI process has been at the bench-scale level, and involves only batch processes. The literature is unclear about removal efficiency since results varied from less than 10 to 99 percent, depending on conditions. It appears that O&M for such a system would be expensive and would require a highly trained operator. Finally, by the admission of the researchers, disposal costs might outweigh the increased adsorption capacity.

#### **A.4.3.2 Granular Ferric Hydroxide**

*Granular ferric hydroxide* is a technology that may combine very long run length without the need to adjust pH. The technology has been demonstrated for arsenic removal full-scale in England (Simms et al, 2000). A pilot-scale study for activated alumina was also conducted on that water and showed run lengths much longer than observed in pilot-scale studies in the United States. Due to the lack of published data showing performance for a range of water qualities, granular ferric hydroxide was not designated a BAT. In addition, there is little published information on the cost of the media, so it is difficult to evaluate cost. Granular ferric hydroxide is being investigated in several ongoing studies and may be an effective technology for removing arsenic.

#### **A.4.3.3 Iron Filings**

The Iron Filings process is essentially a filter technology, much like greensand filtration, wherein the source water is filtered through a bed of sand and iron filings. Unlike some technologies (i.e. ion exchange), sulfate is actually introduced in this process to encourage arsenopyrite precipitation.

While this process seems to be quite effective, its use as a drinking water treatment technology appears to be limited. There is no indication that this technology can reduce arsenic levels below approximately 25 ppb. This technology also suffers from a study design which failed to test its effectiveness at influent levels of concern in drinking water. Since the study design called for such high influent levels - 470 to 20,000 ppb - there is no data to indicate how the technology performs at normal source water arsenic levels, which most certainly are below the 470 ppb level used in experimentation. This technology needs to be further evaluated before it should be recommended as an approved arsenic removal technology for drinking water.

#### **A.4.3.4 Iron Oxide Coated Sand**

Iron oxide coated sand (IOCS) is a rare process that has shown some tendency for arsenic removal. IOCS consists of sand grains coated with ferric hydroxide which are used in fixed bed reactors to remove various dissolved metal species. Factors such as pH, arsenic oxidation state, competing ions, EBCT, and regeneration time have significant effects on the removals achieved with IOCS. Like other processes, the media must be regenerated upon exhaustion. IOCS has only been tested at bench-scale. High levels of arsenite could reduce IOCS effectiveness because the bonding is strong and may permanently damage the media. Natural organic matter may also

be problematic for arsenic removal. IOCS also takes a considerable amount of time to produce in a laboratory setting. At full-scale this would likely result in high capital cost.

## A.5 Development of a Decision Tree

### A.5.1 Surface Water Systems

The following describes the logic used for developing the decision tree for treatment of arsenic in surface water systems in order to comply with the final MCL. For actual breakout of percentages used in the decision tree, refer to the Exhibits A-7 to A-22.

1. Information on in-place treatment technologies for all the flow categories of surface water systems was obtained from Table 6.2 of “Geometries and Characteristics of Public Water Systems” (EPA, 1999b). This table is shown below as Exhibit A-3. Information provided in the document on in-place treatments was based on data from Community Water System Survey (CWSS), which EPA conducted in 1995 to obtain data to support its development and evaluation of drinking water regulations.
2. Exhibit A-3 shows the percentage of systems with in-place Lime/Soda Ash Softening. It was assumed that these systems would modify the existing treatment to comply with the final MCL.

**Exhibit A-3: Percent of Surface Water Systems with In-Place Treatment**

System Size	Lime/ Soda Ash Softening	Coagulation Flocculation	Filtration
25-100	3.9%	27.5%	78.5%
101-500	8.1%	52.6%	71.2%
501-1000	20.5%	70.2%	79.3%
1001-3300	17.5%	79%	81.7%
3300-10K	10.8%	95.4%	86.5%
10001-50K	6.9%	94.5%	96.3%
50,001-100K	5.7%	93.7%	88%
100,001-1M	5.1%	99.5%	93.4%

3. Exhibit A-3 was also used to estimate the percentage of systems with existing coagulation/filtration processes. In-place coagulation/flocculation was based on the smaller (in terms of percent use) of filtration and coagulation/flocculation. The Agency believes this is a conservative assumption for several reasons. The first is that the CWSS data on in-place treatments was gathered in 1995 and 1996, which may not be reflective of requirements under surface water treatment and disinfection by-product rules that were

adopted in later years. The second is that no arsenic removal is assumed for systems with filtration when the percentage is higher than the percentage for coagulation/flocculation.

4. The percent of remaining technologies likely to be used for arsenic treatment for each flow category was obtained by subtracting from 100, the percentages assigned for modified lime softening and modified coagulation /filtration per step 2 and 3 above. The remaining technologies that were considered in the decision tree for treatment of arsenic in surface water include coagulation microfiltration and activated alumina (AA). Systems choosing AA may also choose to pH adjust. This decision is primarily dependent on system size. Systems that serve less than 500 people (see step 6 below) are less likely to pH adjust their raw water supplies because of technical complexity and need for skilled labor. The Agency classified these systems in two natural pH categories. Systems that have raw water with pH between 7 and 8 and systems with pH in raw water greater than 8. For systems that are likely to adjust pH to 6, the Agency considered two run length options, low end (15,400 BV) and high end (23,100 BV).
5. Based on the Agency's best professional judgement, the Agency believes that for systems serving more than 500 people, the selection of treatment for arsenic would likely be distributed among pH adjusted AA with high end run length, pH adjusted AA with low end run length, and coagulation microfiltration in 40:40:20 ratio. Coagulation/ microfiltration is more expensive than activated alumina. However, some surface water systems may select it because they may get filtration credits or precursor removal along with arsenic removal. The benefits of this treatment approach could not be quantified.
6. For systems serving less than 500 people, it is assumed that there will be no usage of coagulation microfiltration technology, primarily because of its high capital cost, technical complexity and need for skilled labor. The Agency believes for this group, about 65 percent of systems with natural pH between 7 and 8 would likely use AA, about 23 percent systems with natural pH greater than 8 would likely use AA and remaining systems would evenly use pH adjusted activated alumina options.

## **A.5.2 Ground Water Systems**

The next section describes the logic used for developing the decision tree for treatment of arsenic to a MCL of 10 ug/L for ground water systems.

1. Information on in-place treatment technologies for all the flow categories of ground water systems was obtained from Table 6.1 of "Geometries and Characteristics of Public Water Systems" (EPA, 1999b). The information provided in the document on in-place treatments was based on data from Community Water System Survey (CWSS), which EPA conducted in 1995 to obtain data to support its development and evaluation of drinking water regulations.
2. For systems serving less than 10,000 people, the Agency selected roughly half the percentage of systems with in-place of lime softening and coagulation/ flocculation (Exhibit A-4). These systems would modify their existing treatment to meet arsenic

MCL. For systems serving more than 10,000 people, the Agency assumed 4 percent for each technology as the maximum percentage of systems with existing lime softening and coagulation/ flocculation treatments. There was a concern that the much higher percentages in might be due to mixed systems (groundwater and surface water) rather than groundwater systems. Thus much lower percentages were used to estimate existing treatment. Systems with existing treatment will modify it to meet the arsenic MCL.

**Exhibit A-4: Percent of Ground Water Systems with In-Place Treatment**

System Size	Lime/ Soda Ash Softening	Coagulation Flocculation
25-100	2.1%	1.5%
101-500	3.7%	2.0%
501-1000	4.1%	4.2%
1001-3300	5.2%	3.4%
3300-10K	7.0%	8.1%
10001-50K	12.2%	15.1%
50,001-100K	17.4%	24.2%
100,001-1M	32.4%	25.2%

3. For systems serving less than 100 people and requiring 50-90 percent removal of arsenic, the decision tree assumed a 5 percent usage for each POU option (RO and AA). For systems requiring less than 50 percent removal of arsenic, a 2 percent usage of each POU option was assumed. POU options were used less if lower removal of arsenic was desired because systems would have an opportunity for blending, which would make central treatment more cost effective.
4. In the decision tree, for systems serving between 100-500 people and requiring 50-90 percent removal of arsenic, the Agency assumed a 3 percent usage for each POU treatment option. For systems requiring less than 50 percent removal of arsenic, the Agency assumed a 1 percent usage of each POU option. The Agency’s assumption of POU usage for this size system is based on the fact that the economic feasibility of POU treatment for systems serving between 70 and 120 households. Therefore, this option would be less preferred by systems in this size in comparison to systems serving less than 100 people. With the increase in households, the management of this treatment strategy becomes progressively complex and cost prohibitive. For systems serving more than 500 people, the Agency did not consider any usage.
5. Anion Exchange (AX). The proposed rule decision tree utilized anion exchange to a great extent. The upper bounds were based on the co-occurrence of sulfate (Table IX-7 of the proposed rule). This table is replicated below as Exhibit A-5. Many comments on the

proposed rule noted other problems that would limit the use of anion exchange. The first was that the brine stream could be considered hazardous waste. Based on a review of this issue, the evaporation pond and chemical precipitation options were eliminated. Discharge to a POTW was not affected by this issue because of the domestic sewage exclusion in 40 CFR 261.4. In addition, the Agency received comments suggesting that stringent technically based local limits (TBLL) for arsenic and total dissolved solids (TDS) in various jurisdictions nationwide would limit the use of publicly owned treatment works (POTW) for discharge of anion exchange waste brine. Therefore, after examining other potential restrictions on POTW discharge of waste brine, the Agency believes lowering the usage of anion exchange with brine disposal to a POTW in the decision tree would be appropriate. In addition, the upper sulfate concentration has been reduced to 50 mg/L because of concerns about its effect on TDS increase.

**Exhibit A-5: Ground Water: Arsenic and Sulfate Co-occurrence**

Influent Arsenic	Likelihood of Sulfate (percent)		
	<25 mg/L	25-120 mg/L	>120 mg/L
<10 ug/L	48	33	19
10-20 ug/L	35	39	26
>20 ug/L	33	38	30

It was assumed that sulfate concentration and percent waste brine volume (in relation to background wastewater volume) are factors that would determine anion exchange selection for arsenic treatment. Percent waste volume was related to removal efficiency. Requiring lower removal efficiencies allow systems to treat a smaller volume of water than at a higher removal efficiency. Systems will blend an untreated portion with a treated portion of water to reduce costs while still complying with the MCL. Based on volume considerations, the option with sulfate less than or equal to 20 mg/L was selected about three times more frequently than the option with sulfate between 20 and 50 mg/L. The brine volume to background wastewater volume also contributed to correlation between anion exchange use and system size.

An increase in total dissolved solids from salt used for regeneration would likely restrict the use of anion exchange in the arid Southwest. However, arsenic occurrence is not limited to just the Southwest. There are areas in the mid-west and Northeast with arsenic above the MCL. The upper bound for systems (small systems) using anion exchange with POTW discharge was 7 percent. For many system size categories, anion exchange with sulfate less than 20 mg/L is the least expensive option. However, it is only be selected by 5 percent or less of the systems because of potential adverse impacts from disposing the brine in the sanitary sewer system.

6. Table IX-9 of the proposed rule presented the co-occurrence of iron and arsenic. This table is replicated below as Exhibit A-6. Approximately 18 percent of the systems had iron concentration above the secondary standard of 300 ug/L. One reference indicated that a 20:1 Fe/As ratio could remove up to 80 percent of the arsenic. It was assumed that two thirds of the systems above the secondary standard would have sufficient iron to achieve high arsenic removals.

**Exhibit A-6: Ground Water: Arsenic and Iron Co-occurrence**

Influent Arsenic	Likelihood of Iron (percent)	
	<300 ug/L	>300 ug/L
<10 ug/L	82	18
10-20 ug/L	81	19
>20 ug/L	71	29

Based on the Agency’s best professional judgement, the Agency believes that for groundwater systems serving less than 500 people, the selection of AA would likely be distributed among systems in a 3:1 ratio for systems with a raw water natural pH between 7 and 8 and systems with a raw water pH greater than 8. This is based on raw groundwater data from the USGS National Water Information System that was analyzed in the co-occurrence report. Projections on the percent of systems with raw water pH greater than 8 were made for each region. The highest percentage for any region was approximately 25 percent. As a conservative estimate, this was assumed nationwide.

For groundwater systems serving more than 500 people, the Agency believes that the selection of AA would likely be distributed evenly among pH adjusted AA with high end run length (23,100 BV) and pH adjusted AA with low end run length (15,400 BV). The Agency also believes that there would be a small percentage of systems serving more than 500 people that would continue to use AA without pH adjustment. However, the Agency believes the usage of AA technology without pH adjustment would decrease with increasing system size.

For groundwater systems serving 1,000 to 10,000 people, the Agency assumed a 10 percent usage of coagulation microfiltration distributed evenly among mechanical dewatering and non-mechanical dewatering options. For systems serving more than 10K people, the Agency assumed an increased usage (14 percent) of coagulation microfiltration with mechanical dewatering dominating in these size categories because of space consideration.

**Exhibit A-7**  
**Probability Decision Tree: Ground Water Systems Serving , 100 People**

No.	Treatment Technology Train	Percent of Treatment Required to Achieve MCL		
		<50%	50-90%	>90%
1	Modify Lime Softening and pre-oxidation	1.0	1.0	1.0
2	Modify Coagulation/Filtration and pre-oxidation	1.0	1.0	1.0
3	Anion Exchange (<20 mg/L SO4) and POTW w aste disposal and pre-oxidation	5.0	3.0	2.0
4	Anion Exchange (20-50 mg/L SO4) and POTW w aste disposal and pre-oxidation	2.0	1.0	1.0
5	Coagulation Assisted Microfiltration and mechanical dewatering/non-hazardous landfill w aste disposal and pre-oxidation	0.0	0.0	0.0
6	Coagulation Assisted Microfiltration and non-mechanical dewatering/non-hazardous landfill w aste disposal and pre-oxidation	0.0	0.0	0.0
7	Oxidation Filtration (Greensand) and POTW for backwash stream and pre-oxidation	12.0	0.0	0.0
8	Activated Alumina (pH 7 -pH 8) and non-hazardous landfill (for spent media) and pre-oxidation	56.0	63.0	70.0
9	Activated Alumina (pH 8 -pH 8.3) and non-hazardous landfill (for spent media) and pre-oxidation	19.0	21.0	23.0
10	Activated Alumina (23,100 BV) w ith pH adjustment (pH 6)/corrosion control and non-hazardous landfill (for spent media) and pre-oxidation	0.0	0.0	0.0
11	Activated Alumina (15,400 BV) w ith pH adjustment (pH 6)/corrosion control and non-hazardous landfill (for spent media) and pre-oxidation	0.0	0.0	0.0
12	POU Activated Alumina and pre-oxidation	2.0	5.0	0.0
13	POU Reverse Osmosis and pre-oxidation	2.0	5.0	2.0
	<b>Sum of Probabilities:</b>	<b>100.00</b>	<b>100.00</b>	<b>100.00</b>

**Exhibit A-8**  
**Probability Decision Tree: Ground Water Systems Serving 101-500 People**

No.	Treatment Technology Train	Percent of Treatment Required to Achieve MCL		
		<50%	50-90%	>90%
1	Modify Lime Softening and pre-oxidation	2.0	2.0	2.0
2	Modify Coagulation/Filtration and pre-oxidation	2.0	2.0	2.0
3	Anion Exchange (<20 mg/L SO <sub>4</sub> ) and POTW w aste disposal and pre-oxidation	5.0	3.0	2.0
4	Anion Exchange (20-50 mg/L SO <sub>4</sub> ) and POTW w aste disposal and pre-oxidation	2.0	1.0	1.0
5	Coagulation Assisted Microfiltration and mechanical dewatering/non-hazardous landfill w aste disposal and pre-oxidation	0.0	0.0	0.0
6	Coagulation Assisted Microfiltration and non-mechanical dewatering/non-hazardous landfill w aste disposal and pre-oxidation	0.0	0.0	0.0
7	Oxidation Filtration (Greensand) and POTW for backwash stream and pre-oxidation	12.0	0.0	0.0
8	Activated Alumina (pH 7 - pH 8) and non-hazardous landfill (for spent media) and pre-oxidation	56.0	63.0	64.0
9	Activated Alumina (pH 8 - pH 8.3) and non-hazardous landfill (for spent media) and pre-oxidation	19.0	21.0	22.0
10	Activated Alumina (23,100 BV) with pH adjustment (pH 6)/corrosion control and non-hazardous landfill (for spent media) and pre-oxidation	0.0	2.0	3.0
11	Activated Alumina (15,400 BV) with pH adjustment (pH 6)/corrosion control and non-hazardous landfill (for spent media) and pre-oxidation	0.0	0.0	3.0
12	POU Activated Alumina and pre-oxidation	1.0	3.0	0.0
13	POU Reverse Osmosis and pre-oxidation	1.0	3.0	1.0
	<b>Sum of Probabilities:</b>	<b>100.00</b>	<b>100.00</b>	<b>100.00</b>

**Exhibit A-9**  
**Probability Decision Tree: Ground Water Systems Serving 501-1,000 People**

No.	Treatment Technology Train	Percent of Treatment Required to Achieve MCL		
		<50%	50-90%	>90%
1	Modify Lime Softening and pre-oxidation	2.0	2.0	2.0
2	Modify Coagulation/Filtration and pre-oxidation	2.0	2.0	2.0
3	Anion Exchange (<20 mg/L SO4) and POTW w aste disposal and pre-oxidation	5.0	3.0	2.0
4	Anion Exchange (20-50 mg/L SO4) and POTW w aste disposal and pre-oxidation	2.0	1.0	1.0
5	Coagulation Assisted Microfiltration and mechanical dewatering/non-hazardous landfill w aste disposal and pre-oxidation	0.0	0.0	0.0
6	Coagulation Assisted Microfiltration and non-mechanical dewatering/non-hazardous landfill w aste disposal and pre-oxidation	0.0	0.0	0.0
7	Oxidation Filtration (Greensand) and POTW for backwash stream and pre-oxidation	12.0	0.0	0.0
8	Activated Alumina (pH 7 - pH 8) and non-hazardous landfill (for spent media) and pre-oxidation	25.0	30.0	31.0
9	Activated Alumina (pH 8 - pH 8.3) and non-hazardous landfill (for spent media) and pre-oxidation	2.0	2.0	2.0
10	Activated Alumina (23,100 BV) w ith pH adjustment (pH 6)/corrosion control and non-hazardous landfill (for spent media) and pre-oxidation	25.0	30.0	30.0
11	Activated Alumina (15,400 BV) w ith pH adjustment (pH 6)/corrosion control and non-hazardous landfill (for spent media) and pre-oxidation	25.0	30.0	30.0
12	POU Activated Alumina and pre-oxidation	0.0	0.0	0.0
13	POU Reverse Osmosis and pre-oxidation	0.0	0.0	0.0
	<b>Sum of Probabilities:</b>	<b>100.00</b>	<b>100.00</b>	<b>100.00</b>

**Exhibit A-10**  
**Probability Decision Tree: Ground Water Systems Serving 1,001-3,300 People**

No.	Treatment Technology Train	Percent of Treatment Required to Achieve MCL		
		<50%	50-90%	>90%
1	Modify Lime Softening and pre-oxidation	2.0	2.0	2.0
2	Modify Coagulation/Filtration and pre-oxidation	2.0	2.0	2.0
3	Anion Exchange (<20 mg/L SO <sub>4</sub> ) and POTW w aste disposal and pre-oxidation	5.0	3.0	2.0
4	Anion Exchange (20-50 mg/L SO <sub>4</sub> ) and POTW w aste disposal and pre-oxidation	2.0	1.0	1.0
5	Coagulation Assisted Microfiltration and mechanical dewatering/non-hazardous landfill w aste disposal and pre-oxidation	5.0	5.0	5.0
6	Coagulation Assisted Microfiltration and non-mechanical dewatering/non-hazardous landfill w aste disposal and pre-oxidation	5.0	5.0	5.0
7	Oxidation Filtration (Greensand) and POTW for backwash stream and pre-oxidation	12.0	0.0	0.0
8	Activated Alumina (pH 7 - pH 8) and non-hazardous landfill (for spent media) and pre-oxidation	17.0	16.0	17.0
9	Activated Alumina (pH 8 - pH 8.3) and non-hazardous landfill (for spent media) and pre-oxidation	0.0	0.0	0.0
10	Activated Alumina (23,100 BV) with pH adjustment (pH 6)/corrosion control and non-hazardous landfill (for spent media) and pre-oxidation	25.0	33.0	33.0
11	Activated Alumina (15,400 BV) with pH adjustment (pH 6)/corrosion control and non-hazardous landfill (for spent media) and pre-oxidation	25.0	33.0	33.0
12	POU Activated Alumina and pre-oxidation	0.0	0.0	0.0
13	POU Reverse Osmosis and pre-oxidation	0.0	0.0	0.0
	<b>Sum of Probabilities:</b>	<b>100.00</b>	<b>100.00</b>	<b>100.00</b>

**Exhibit A-11**  
**Probability Decision Tree: Ground Water Systems Serving 3,301-10,000 People**

No.	Treatment Technology Train	Percent of Treatment Required to Achieve MCL		
		<50%	50-90%	>90%
1	Modify Lime Softening and pre-oxidation	3.0	3.0	3.0
2	Modify Coagulation/Filtration and pre-oxidation	4.0	4.0	4.0
3	Anion Exchange (<20 mg/L SO <sub>4</sub> ) and POTW w aste disposal and pre-oxidation	5.0	3.0	2.0
4	Anion Exchange (20-50 mg/L SO <sub>4</sub> ) and POTW w aste disposal and pre-oxidation	2.0	1.0	1.0
5	Coagulation Assisted Microfiltration and mechanical dew atering/non-hazardous landfill w aste disposal and pre-oxidation	8.0	8.0	8.0
6	Coagulation Assisted Microfiltration and non-mechanical dew atering/non-hazardous landfill w aste disposal and pre-oxidation	2.0	2.0	2.0
7	Oxidation Filtration (Greensand) and POTW for backw ash stream and pre-oxidation	0.0	0.0	0.0
8	Activated Alumina (pH 7 - pH 8) and non-hazardous landfill (for spent media) and pre-oxidation	24.0	25.0	26.0
9	Activated Alumina (pH 8 - pH 8.3) and non-hazardous landfill (for spent media) and pre-oxidation	0.0	0.0	0.0
10	Activated Alumina (23,100 BV) w ith pH adjustment (pH 6)/corrosion control and non-hazardous landfill (for spent media) and pre-oxidation	26.0	27.0	27.0
11	Activated Alumina (15,400 BV) w ith pH adjustment (pH 6)/corrosion control and non-hazardous landfill (for spent media) and pre-oxidation	26.0	27.0	27.0
12	POU Activated Alumina and pre-oxidation	0.0	0.0	0.0
13	POU Reverse Osmosis and pre-oxidation	0.0	0.0	0.0
<b>Sum of Probabilities:</b>		<b>100.00</b>	<b>100.00</b>	<b>100.00</b>

**Exhibit A-12**  
**Probability Decision Tree: Ground Water Systems Serving 10,001-50,000 People**

No.	Treatment Technology Train	Percent of Treatment Required to Achieve MCL		
		<50%	50-90%	>90%
1	Modify Lime Softening and pre-oxidation	4.0	4.0	4.0
2	Modify Coagulation/Filtration and pre-oxidation	4.0	4.0	4.0
3	Anion Exchange (<20 mg/L SO <sub>4</sub> ) and POTW w aste disposal and pre-oxidation	3.0	1.0	0.0
4	Anion Exchange (20-50 mg/L SO <sub>4</sub> ) and POTW w aste disposal and pre-oxidation	0.0	0.0	0.0
5	Coagulation Assisted Microfiltration and mechanical dewatering/non-hazardous landfill w aste disposal and pre-oxidation	12.0	12.0	12.0
6	Coagulation Assisted Microfiltration and non-mechanical dewatering/non-hazardous landfill w aste disposal and pre-oxidation	2.0	2.0	2.0
7	Oxidation Filtration (Greensand) and POTW for backwash stream and pre-oxidation	0.0	0.0	0.0
8	Activated Alumina (pH 7 - pH 8) and non-hazardous landfill (for spent media) and pre-oxidation	11.0	11.0	11.0
9	Activated Alumina (pH 8 - pH 8.3) and non-hazardous landfill (for spent media) and pre-oxidation	0.0	0.0	0.0
10	Activated Alumina (23,100 BV) with pH adjustment (pH 6)/corrosion control and non-hazardous landfill (for spent media) and pre-oxidation	32.0	33.0	34.0
11	Activated Alumina (15,400 BV) with pH adjustment (pH 6)/corrosion control and non-hazardous landfill (for spent media) and pre-oxidation	32.0	33.0	33.0
12	POU Activated Alumina and pre-oxidation	0.0	0.0	0.0
13	POU Reverse Osmosis and pre-oxidation	0.0	0.0	0.0
<b>Sum of Probabilities:</b>		<b>100.00</b>	<b>100.00</b>	<b>100.00</b>

**Exhibit A-13**  
**Probability Decision Tree: Ground Water Systems Serving 50,001-100,000 People**

No.	Treatment Technology Train	Percent of Treatment Required to Achieve MCL		
		<50%	50-90%	>90%
1	Modify Lime Softening and pre-oxidation	4.0	4.0	4.0
2	Modify Coagulation/Filtration and pre-oxidation	4.0	4.0	4.0
3	Anion Exchange (<20 mg/L SO4) and POTW w aste disposal and pre-oxidation	3.0	1.0	0.0
4	Anion Exchange (20-50 mg/L SO4) and POTW w aste disposal and pre-oxidation	0.0	0.0	0.0
5	Coagulation Assisted Microfiltration and mechanical dewatering/non-hazardous landfill w aste disposal and pre-oxidation	12.0	12.0	12.0
6	Coagulation Assisted Microfiltration and non-mechanical dewatering/non-hazardous landfill w aste disposal and pre-oxidation	2.0	2.0	2.0
7	Oxidation Filtration (Greensand) and POTW for backwash stream and pre-oxidation	0.0	0.0	0.0
8	Activated Alumina (pH 7 - pH 8) and non-hazardous landfill (for spent media) and pre-oxidation	7.0	7.0	7.0
9	Activated Alumina (pH 8 - pH 8.3) and non-hazardous landfill (for spent media) and pre-oxidation	0.0	0.0	0.0
10	Activated Alumina (23,100 BV) with pH adjustment (pH 6)/corrosion control and non-hazardous landfill (for spent media) and pre-oxidation	34.0	35.0	36.0
11	Activated Alumina (15,400 BV) with pH adjustment (pH 6)/corrosion control and non-hazardous landfill (for spent media) and pre-oxidation	34.0	35.0	35.0
12	POU Activated Alumina and pre-oxidation	0.0	0.0	0.0
13	POU Reverse Osmosis and pre-oxidation	0.0	0.0	0.0
<b>Sum of Probabilities:</b>		<b>100.00</b>	<b>100.00</b>	<b>100.00</b>

**Exhibit A-14**  
**Probability Decision Tree: Ground Water Systems Serving 100,001-1,000,000 People**

No.	Treatment Technology Train	Percent of Treatment Required to Achieve MCL		
		<50%	50-90%	>90%
1	Modify Lime Softening and pre-oxidation	4.0	4.0	4.0
2	Modify Coagulation/Filtration and pre-oxidation	4.0	4.0	4.0
3	Anion Exchange (<20 mg/L SO4) and POTW w aste disposal and pre-oxidation	0.0	0.0	0.0
4	Anion Exchange (20-50 mg/L SO4) and POTW w aste disposal and pre-oxidation	0.0	0.0	0.0
5	Coagulation Assisted Microfiltration and mechanical dew atering/non-hazardous landfill w aste disposal and pre-oxidation	12.0	12.0	12.0
6	Coagulation Assisted Microfiltration and non-mechanical dew atering/non-hazardous landfill w aste disposal and pre-oxidation	2.0	2.0	2.0
7	Oxidation Filtration (Greensand) and POTW for backw ash stream and pre-oxidation	0.0	0.0	0.0
8	Activated Alumina (pH 7 - pH 8) and non-hazardous landfill (for spent media) and pre-oxidation	4.0	4.0	4.0
9	Activated Alumina (pH 8 - pH 8.3) and non-hazardous landfill (for spent media) and pre-oxidation	0.0	0.0	0.0
10	Activated Alumina (23,100 BV) w ith pH adjustment (pH 6)/corrosion control and non-hazardous landfill (for spent media) and pre-oxidation	37.0	37.0	37.0
11	Activated Alumina (15,400 BV) w ith pH adjustment (pH 6)/corrosion control and non-hazardous landfill (for spent media) and pre-oxidation	37.0	37.0	37.0
12	POU Activated Alumina and pre-oxidation	0.0	0.0	0.0
13	POU Reverse Osmosis and pre-oxidation	0.0	0.0	0.0
	<b>Sum of Probabilities:</b>	<b>100.00</b>	<b>100.00</b>	<b>100.00</b>

**Exhibit A-15**  
**Probability Decision Tree: Surface Water Systems Serving 100 People**

No.	Treatment Technology Train	Percent of Treatment Required to Achieve MCL		
		<50%	50-90%	>90%
1	Modify Lime Softening and pre-oxidation	4.0	4.0	4.0
2	Modify Coagulation/Filtration and pre-oxidation	28.0	28.0	28.0
3	Anion Exchange (<20 mg/L SO <sub>4</sub> ) and POTW waste disposal and pre-oxidation	0.0	0.0	0.0
4	Anion Exchange (20-50 mg/L SO <sub>4</sub> ) and POTW waste disposal and pre-oxidation	0.0	0.0	0.0
5	Coagulation Assisted Microfiltration and mechanical dewatering/non-hazardous landfill waste disposal and pre-oxidation	0.0	0.0	0.0
6	Coagulation Assisted Microfiltration and non-mechanical dewatering/non-hazardous landfill waste disposal and pre-oxidation	0.0	0.0	0.0
7	Oxidation Filtration (Greensand) and POTW for backwash stream and pre-oxidation	0.0	0.0	0.0
8	Activated Alumina (pH 7 -pH 8) and non-hazardous landfill (for spent media) and pre-oxidation	44.0	44.0	44.0
9	Activated Alumina (pH 8 -pH 8.3) and non-hazardous landfill (for spent media) and pre-oxidation	16.0	16.0	16.0
10	Activated Alumina (23,100 BV) with pH adjustment (pH 6)/corrosion control and non-hazardous landfill (for spent media) and pre-oxidation	4.0	4.0	4.0
11	Activated Alumina (15,400 BV) with pH adjustment (pH 6)/corrosion control and non-hazardous landfill (for spent media) and pre-oxidation	4.0	4.0	4.0
12	POU Activated Alumina and pre-oxidation	0.0	0.0	0.0
13	POU Reverse Osmosis and pre-oxidation	0.0	0.0	0.0
<b>Sum of Probabilities:</b>		<b>100.00</b>	<b>100.00</b>	<b>100.00</b>

**Exhibit A-16**  
**Probability Decision Tree: Surface Water Systems Serving 101-500 People**

No.	Treatment Technology Train	Percent of Treatment Required to Achieve MCL		
		<50%	50-90%	>90%
1	Modify Lime Softening and pre-oxidation	8.0	8.0	8.0
2	Modify Coagulation/Filtration and pre-oxidation	53.0	53.0	53.0
3	Anion Exchange (<20 mg/L SO <sub>4</sub> ) and POTW w aste disposal and pre-oxidation	0.0	0.0	0.0
4	Anion Exchange (20-50 mg/L SO <sub>4</sub> ) and POTW w aste disposal and pre-oxidation	0.0	0.0	0.0
5	Coagulation Assisted Microfiltration and mechanical dewatering/non-hazardous landfill w aste disposal and pre-oxidation	0.0	0.0	0.0
6	Coagulation Assisted Microfiltration and non-mechanical dewatering/non-hazardous landfill w aste disposal and pre-oxidation	0.0	0.0	0.0
7	Oxidation Filtration (Greensand) and POTW for backwash stream and pre-oxidation	0.0	0.0	0.0
8	Activated Alumina (pH 7 - pH 8) and non-hazardous landfill (for spent media) and pre-oxidation	24.0	24.0	24.0
9	Activated Alumina (pH 8 - pH 8.3) and non-hazardous landfill (for spent media) and pre-oxidation	9.0	9.0	9.0
10	Activated Alumina (23,100 BV) with pH adjustment (pH 6)/corrosion control and non-hazardous landfill (for spent media) and pre-oxidation	3.0	3.0	3.0
11	Activated Alumina (15,400 BV) with pH adjustment (pH 6)/corrosion control and non-hazardous landfill (for spent media) and pre-oxidation	3.0	3.0	3.0
12	POU Activated Alumina and pre-oxidation	0.0	0.0	0.0
13	POU Reverse Osmosis and pre-oxidation	0.0	0.0	0.0
	<b>Sum of Probabilities:</b>	<b>100.00</b>	<b>100.00</b>	<b>100.00</b>

**Exhibit A-17**  
**Probability Decision Tree: Surface Water Systems Serving 501-1,000 People**

No.	Treatment Technology Train	Percent of Treatment Required to Achieve MCL		
		<50%	50-90%	>90%
1	Modify Lime Softening and pre-oxidation	21.0	21.0	21.0
2	Modify Coagulation/Filtration and pre-oxidation	70.0	70.0	70.0
3	Anion Exchange (<20 mg/L SO <sub>4</sub> ) and POTW w aste disposal and pre-oxidation	0.0	0.0	0.0
4	Anion Exchange (20-50 mg/L SO <sub>4</sub> ) and POTW w aste disposal and pre-oxidation	0.0	0.0	0.0
5	Coagulation Assisted Microfiltration and mechanical dewatering/non-hazardous landfill w aste disposal and pre-oxidation	1.0	1.0	1.0
6	Coagulation Assisted Microfiltration and non-mechanical dewatering/non-hazardous landfill w aste disposal and pre-oxidation	0.0	0.0	0.0
7	Oxidation Filtration (Greensand) and POTW for backwash stream and pre-oxidation	0.0	0.0	0.0
8	Activated Alumina (pH 7 - pH 8) and non-hazardous landfill (for spent media) and pre-oxidation	0.0	0.0	0.0
9	Activated Alumina (pH 8 - pH 8.3) and non-hazardous landfill (for spent media) and pre-oxidation	0.0	0.0	0.0
10	Activated Alumina (23,100 BV) with pH adjustment (pH 6)/corrosion control and non-hazardous landfill (for spent media) and pre-oxidation	4.0	4.0	4.0
11	Activated Alumina (15,400 BV) with pH adjustment (pH 6)/corrosion control and non-hazardous landfill (for spent media) and pre-oxidation	4.0	4.0	4.0
12	POU Activated Alumina and pre-oxidation	0.0	0.0	0.0
13	POU Reverse Osmosis and pre-oxidation	0.0	0.0	0.0
	<b>Sum of Probabilities:</b>	<b>100.00</b>	<b>100.00</b>	<b>100.00</b>

**Exhibit A-18**  
**Probability Decision Tree: Surface Water Systems Serving 1,001-3,300 People**

No.	Treatment Technology Train	Percent of Treatment Required to Achieve MCL		
		<50%	50-90%	>90%
1	Modify Lime Softening and pre-oxidation	18.0	18.0	18.0
2	Modify Coagulation/Filtration and pre-oxidation	79.0	79.0	79.0
3	Anion Exchange (<20 mg/L SO4) and POTW w aste disposal and pre-oxidation	0.0	0.0	0.0
4	Anion Exchange (20-50 mg/L SO4) and POTW w aste disposal and pre-oxidation	0.0	0.0	0.0
5	Coagulation Assisted Microfiltration and mechanical dew atering/non-hazardous landfill w aste disposal and pre-oxidation	1.0	1.0	1.0
6	Coagulation Assisted Microfiltration and non-mechanical dew atering/non-hazardous landfill w aste disposal and pre-oxidation	0.0	0.0	0.0
7	Oxidation Filtration (Greensand) and POTW for backw ash stream and pre-oxidation	0.0	0.0	0.0
8	Activated Alumina (pH 7 - pH 8) and non-hazardous landfill (for spent media) and pre-oxidation	0.0	0.0	0.0
9	Activated Alumina (pH 8 - pH 8.3) and non-hazardous landfill (for spent media) and pre-oxidation	0.0	0.0	0.0
10	Activated Alumina (23,100 BV) w ith pH adjustment (pH 6)/corrosion control and non-hazardous landfill (for spent media) and pre-oxidation	1.0	1.0	1.0
11	Activated Alumina (15,400 BV) w ith pH adjustment (pH 6)/corrosion control and non-hazardous landfill (for spent media) and pre-oxidation	1.0	1.0	1.0
12	POU Activated Alumina and pre-oxidation	0.0	0.0	0.0
13	POU Reverse Osmosis and pre-oxidation	0.0	0.0	0.0
	<b>Sum of Probabilities:</b>	<b>100.00</b>	<b>100.00</b>	<b>100.00</b>

**Exhibit A-19**  
**Probability Decision Tree: Surface Water Systems Serving 3,301-10,000 People**

No.	Treatment Technology Train	Percent of Treatment Required to Achieve MCL		
		<50%	50-90%	>90%
1	Modify Lime Softening and pre-oxidation	11.0	11.0	11.0
2	Modify Coagulation/Filtration and pre-oxidation	87.0	87.0	87.0
3	Anion Exchange (<20 mg/L SO <sub>4</sub> ) and POTW w aste disposal and pre-oxidation	0.0	0.0	0.0
4	Anion Exchange (20-50 mg/L SO <sub>4</sub> ) and POTW w aste disposal and pre-oxidation	0.0	0.0	0.0
5	Coagulation Assisted Microfiltration and mechanical dewatering/non-hazardous landfill w aste disposal and pre-oxidation	0.0	0.0	0.0
6	Coagulation Assisted Microfiltration and non-mechanical dewatering/non-hazardous landfill w aste disposal and pre-oxidation	0.0	0.0	0.0
7	Oxidation Filtration (Greensand) and POTW for backwash stream and pre-oxidation	0.0	0.0	0.0
8	Activated Alumina (pH 7 - pH 8) and non-hazardous landfill (for spent media) and pre-oxidation	0.0	0.0	0.0
9	Activated Alumina (pH 8 - pH 8.3) and non-hazardous landfill (for spent media) and pre-oxidation	0.0	0.0	0.0
10	Activated Alumina (23,100 BV) w ith pH adjustment (pH 6)/corrosion control and non-hazardous landfill (for spent media) and pre-oxidation	1.0	1.0	1.0
11	Activated Alumina (15,400 BV) w ith pH adjustment (pH 6)/corrosion control and non-hazardous landfill (for spent media) and pre-oxidation	1.0	1.0	1.0
12	POU Activated Alumina and pre-oxidation	0.0	0.0	0.0
13	POU Reverse Osmosis and pre-oxidation	0.0	0.0	0.0
<b>Sum of Probabilities:</b>		<b>100.00</b>	<b>100.00</b>	<b>100.00</b>

**Exhibit A-20  
Probability Decision Tree: Surface Water Systems Serving 10,001-50,000 People**

No.	Treatment Technology Train	Percent of Treatment Required to Achieve MCL		
		<50%	50-90%	>90%
1	Modify Lime Softening and pre-oxidation	5.0	5.0	5.0
2	Modify Coagulation/Filtration and pre-oxidation	95.0	95.0	95.0
3	Anion Exchange (<20 mg/L SO <sub>4</sub> ) and POTW w aste disposal and pre-oxidation	0.0	0.0	0.0
4	Anion Exchange (20-50 mg/L SO <sub>4</sub> ) and POTW w aste disposal and pre-oxidation	0.0	0.0	0.0
5	Coagulation Assisted Microfiltration and mechanical dew atering/non-hazardous landfill w aste disposal and pre-oxidation	0.0	0.0	0.0
6	Coagulation Assisted Microfiltration and non-mechanical dew atering/non-hazardous landfill w aste disposal and pre-oxidation	0.0	0.0	0.0
7	Oxidation Filtration (Greensand) and POTW for backw ash stream and pre-oxidation	0.0	0.0	0.0
8	Activated Alumina (pH 7 - pH 8) and non-hazardous landfill (for spent media) and pre-oxidation	0.0	0.0	0.0
9	Activated Alumina (pH 8 - pH 8.3) and non-hazardous landfill (for spent media) and pre-oxidation	0.0	0.0	0.0
10	Activated Alumina (23,100 BV) w ith pH adjustment (pH 6)/corrosion control and non-hazardous landfill (for spent media) and pre-oxidation	0.0	0.0	0.0
11	Activated Alumina (15,400 BV) w ith pH adjustment (pH 6)/corrosion control and non-hazardous landfill (for spent media) and pre-oxidation	0.0	0.0	0.0
12	POU Activated Alumina and pre-oxidation	0.0	0.0	0.0
13	POU Reverse Osmosis and pre-oxidation	0.0	0.0	0.0
	<b>Sum of Probabilities:</b>	<b>100.00</b>	<b>100.00</b>	<b>100.00</b>

**Exhibit A-21**  
**Probability Decision Tree: Surface Water Systems Serving 50,001-100,000 People**

No.	Treatment Technology Train	Percent of Treatment Required to Achieve MCL		
		<50%	50-90%	>90%
1	Modify Lime Softening and pre-oxidation	6.0	6.0	6.0
2	Modify Coagulation/Filtration and pre-oxidation	88.0	88.0	88.0
3	Anion Exchange (<20 mg/L SO4) and POTW waste disposal and pre-oxidation	0.0	0.0	0.0
4	Anion Exchange (20-50 mg/L SO4) and POTW waste disposal and pre-oxidation	0.0	0.0	0.0
5	Coagulation Assisted Microfiltration and mechanical dewatering/non-hazardous landfill waste disposal and pre-oxidation	1.0	1.0	1.0
6	Coagulation Assisted Microfiltration and non-mechanical dewatering/non-hazardous landfill waste disposal and pre-oxidation	0.0	0.0	0.0
7	Oxidation Filtration (Greensand) and POTW for backwash stream and pre-oxidation	0.0	0.0	0.0
8	Activated Alumina (pH 7 - pH 8) and non-hazardous landfill (for spent media) and pre-oxidation	0.0	0.0	0.0
9	Activated Alumina (pH 8 - pH 8.3) and non-hazardous landfill (for spent media) and pre-oxidation	0.0	0.0	0.0
10	Activated Alumina (23,100 BV) with pH adjustment (pH 6)/corrosion control and non-hazardous landfill (for spent media) and pre-oxidation	2.0	2.0	2.0
11	Activated Alumina (15,400 BV) with pH adjustment (pH 6)/corrosion control and non-hazardous landfill (for spent media) and pre-oxidation	3.0	3.0	3.0
12	POU Activated Alumina and pre-oxidation	0.0	0.0	0.0
13	POU Reverse Osmosis and pre-oxidation	0.0	0.0	0.0
	<b>Sum of Probabilities:</b>	<b>100.00</b>	<b>100.00</b>	<b>100.00</b>

**Exhibit A-22**  
**Probability Decision Tree: Surface Water Systems Serving 100,001-1,000,000 People**

No.	Treatment Technology Train	Percent of Treatment Required to Achieve MCL		
		<50%	50-90%	>90%
1	Modify Lime Softening and pre-oxidation	5.0	5.0	5.0
2	Modify Coagulation/Filtration and pre-oxidation	93.0	93.0	93.0
3	Anion Exchange (<20 mg/L SO4) and POTW waste disposal and pre-oxidation	0.0	0.0	0.0
4	Anion Exchange (20-50 mg/L SO4) and POTW waste disposal and pre-oxidation	0.0	0.0	0.0
5	Coagulation Assisted Microfiltration and mechanical dewatering/non-hazardous landfill waste disposal and pre-oxidation	0.0	0.0	0.0
6	Coagulation Assisted Microfiltration and non-mechanical dewatering/non-hazardous landfill waste disposal and pre-oxidation	0.0	0.0	0.0
7	Oxidation Filtration (Greensand) and POTW for backwash stream and pre-oxidation	0.0	0.0	0.0
8	Activated Alumina (pH 7 - pH 8) and non-hazardous landfill (for spent media) and pre-oxidation	0.0	0.0	0.0
9	Activated Alumina (pH 8 - pH 8.3) and non-hazardous landfill (for spent media) and pre-oxidation	0.0	0.0	0.0
10	Activated Alumina (23,100 BV) with pH adjustment (pH 6)/corrosion control and non-hazardous landfill (for spent media) and pre-oxidation	1.0	1.0	1.0
11	Activated Alumina (15,400 BV) with pH adjustment (pH 6)/corrosion control and non-hazardous landfill (for spent media) and pre-oxidation	1.0	1.0	1.0
12	POU Activated Alumina and pre-oxidation	0.0	0.0	0.0
13	POU Reverse Osmosis and pre-oxidation	0.0	0.0	0.0
<b>Sum of Probabilities:</b>		<b>100.00</b>	<b>100.00</b>	<b>100.00</b>

## A.6 Very Large System Cost Methodology

EPA must conduct a thorough cost-benefit analysis, and provide comprehensive, informative, and understandable information to the public about its regulatory efforts. As part of these analyses, EPA evaluated the regulatory costs of compliance for very large systems, who would be subject to the new arsenic drinking water regulation. The nation's 25 largest drinking water systems (i.e., those serving a million people or more) supply approximately 38 million people and generally account for about 15 to 20 percent of all compliance-related costs. Accurately determining these costs for future regulations is critical. As a result, EPA has developed compliance cost estimates for the arsenic and radon regulations for each individual system that serves greater than 1 million persons. These cost estimates help EPA to more accurately assess the cost impacts and benefits of the arsenic regulation. The estimates also help the Agency identify lower cost regulatory options and better understand current water systems' capabilities and constraints.

The system costs were calculated for the 24 public water systems that serve a retail population greater than 1 million persons and one public water system that serves a wholesale population of 16 million persons. Exhibit A-23 lists these 25 public water systems. The distinguishing characteristics of these very large systems include:

- a large number of entry points from diverse sources;
- mixed (i.e. ground and surface) sources;
- occurrence not conducive to mathematical modeling;
- significant levels of wholesaling;
- sophisticated in-place treatment;
- retrofit costs dramatically influenced by site-specific factors; and
- large amounts of waste management and disposal which can contribute substantial costs.

Generic models cannot incorporate all of these considerations; therefore, in-depth characterizations and cost analyses were developed utilizing several existing databases and surveys.

The profile for each system contains information such as design and average daily flows, treatment facility diagrams, chemical feed processes, water quality parameters, system layouts, and intake and aquifer locations. System and treatment data were obtained from the following sources:

- The Information Collection Rule (1997);
- The Community Water Supply Survey (1995);
- The Association of Metropolitan Water Agencies Survey (1998);
- The Safe Drinking Water Information System (SDWIS); and
- The American Water Works Association WATERSTATS Survey (1997)

While these sources contained much of the information necessary to perform cost analyses, the Agency was still missing some of the detailed arsenic occurrence data in these large water

systems. Where major gaps existed, especially in groundwater systems, occurrence data obtained from the States of Texas, California, and Arizona, the Metropolitan Water District of Southern California Arsenic Study (1993), the National Inorganic and Radionuclides Study (EPA, 1984), and utilities were used. Based on data from the studies, detailed costs estimates were derived for each of the very large water systems.

**Exhibit A-23**  
**List of Large Water Systems That Serve More Than 1 Million People**

	<b>PWS ID #</b>	<b>Utility Name</b>
1	AZ0407025	Phoenix Municipal Water System
2	CA0110005	East Bay Municipal Utility District
3	CA1910067	Los Angeles-City Dept. of Water and Power
4	CA1910087	Metropolitan Water District of Southern California
5	CA3710020	San Diego- City of
6	CA3810001	San Francisco Water Department
7	CA4310011	San Jose Water Company
8	CO0116001	Denver Water Board
9	FL4130871	Miami-Dade Water And Sewer Authority-Main System
10	GA1210001	City of Atlanta
11	IL0316000	City of Chicago
12	MA6000000	Massachusetts Water Resource Authority
13	MD0150005	Washington Suburban Sanitation Commission
14	MD0300002	Baltimore City
15	MI0001800	City of Detroit
16	MO6010716	St. Louis County Water County
17	NY5110526	Suffolk County Water Authority
18	NY7003493	New York City Aqueduct System
19	OH1800311	City of Cleveland
20	PA1510001	Philadelphia Water Department
21	PR0002591	San Juan Metropolitan
22	TX0570004	Dallas Water Utility
23	TX1010013	City of Houston- Public Works Department
24	TX150018	San Antonio Water System
25	WA5377050	Seattle Public Utilities

Cost estimates were generated for each system at several MCL options. The total capital costs and operational and maintenance (O & M) costs were calculated using the profile information gathered on each system, conceptual designs (i.e., vendor estimates and RS Means), and modified EPA cost models (i.e., Water and WaterCost models). The models were modified based on the general cost assumptions developed in the Phase I Water Treatment Cost Upgrades (EPA, 1998).

Preliminary cost estimates were sent to all of the systems for their review. Approximately 30 percent of the systems responded by submitting revised estimates and/or detailed arsenic occurrence data. Based on the information received, EPA revised the cost estimates for those systems. Based on the results, only 3 of the very large systems had capital and/or O&M expenditures for complying with a MCL of 10 µg/L. More detailed costs estimates for each very large water system can be found in Radon and Arsenic Regulatory Compliance Costs for the 25 Largest Public Water Systems document, which is located in the water docket.

# Appendix B: Assumptions and Methodology for Estimating Cancer Risks Avoided and Benefits

## B.1 Community Water Systems Serving Fewer than One Million People

### B.1.1 Introduction

EPA's estimation of the number of cancer cases resulting from current levels of exposure to arsenic from drinking water in community water systems serving fewer than one million people, and the number of those cases that would be avoided following implementation of a specified arsenic MCL are obtained using the following basic risk algorithm.

$$R_{\text{Ind}} = C(\text{As})_{\text{Ind}} * [DW_{\text{Ind}} * DW_{\text{Adj}}] * R_{\text{Unit}} \quad \text{Equation B-1}$$

The components of this risk algorithm are as follows.

$C(\text{As})_{\text{Ind}}$  is the concentration of arsenic in drinking water that a given individual is exposed to, on average, over the course of his or her lifetime.  $C(\text{As})_{\text{Ind}}$  is obtained from the occurrence assessment distributions for surface water and ground water and is expressed in units of  $\mu\text{g/L}$ .

$DW_{\text{Ind}}$  is the daily drinking water consumption for a given individual, and is incorporated in this model as a lifetime weighted average expressed in units of L/kg-day. As a lifetime weighted average, this drinking water consumption value reflects differences in water consumption per kilogram body weight that is observed to occur over an individual's lifetime. This variable is also a function of the individual's sex.

$DW_{\text{Adj}}$  is an adjustment factor constant ( $= 70 \text{ kg} \div 2 \text{ L/day}$ ) that is applied to the weighted average drinking water consumption values for individuals to account for the fact that the unit cancer risk factor (as described below) is based upon an assumed lifetime average daily intake of 2 L/day and a lifetime average body weight of 70 kg.

It should be noted that the quantity  $[DW_{\text{Ind}} * DW_{\text{Adj}}]$  is also referred to in this modeling effort as the Lifetime Relative Exposure Factor (LREF). The LREF reflects a particular individual's lifetime exposure to arsenic from drinking water, given that person's  $DW_{\text{Ind}}$  value relative to an "average" individual consuming 2 L/day of water and weighing 70 kg. An LREF value less than one indicates the person has less lifetime exposure (and therefore less risk) than such an "average person" used to derive the unit risk factor; similarly a value greater than one indicates a higher lifetime exposure and greater risk than that "average person".

$R_{\text{Unit}}$  is the unit cancer risk factor for the specific endpoint of concern (e.g., bladder cancer, or lung cancer). This factor is in units of "expected cases per person per  $\mu\text{g/day}$ ." It is important to note that these unit risks, as derived from the Morales (2000) study are lifetime risks, that were developed with an underlying assumption of 70 years of exposure and a lifetime average water

consumption of 2 L/day and body weight of 70 kg. It should also be noted that the Morales (2000) cancer risk factors used in this modeling, which are derived from an analysis of the Taiwan data, are specific to a particular cancer endpoint (bladder, lung) and are sex-dependent.

The benefit modeling performed in support of the arsenic regulation utilizes Equation B-1 in a Monte Carlo simulation framework that provides information on the aggregate number of cases of cancer occurring (and avoided) in the overall population, as well as a characterization of the distribution of risks experienced by different individuals in the exposed population as a result of individual variability in exposure conditions. Because some of the factors that result in individual variability in exposure and risk are sex and source water dependent, the Monte Carlo model also incorporates information on fraction of males and females in the population, and on the proportion of individuals using surface water versus ground water as their primary community water supply source.

As an overview of how the simulation model operates, it can be viewed as being similar to taking a representative sample from the population exposed to arsenic in drinking water from community water systems and using the results obtained from that sample to characterize the overall risks of the population. In this modeling, a total of 2,000 iterations (samples) were used for each model run.

In each iteration, an individual is selected, and identified as male or female and as a ground water or surface water user, based on estimated probabilities associated with those characteristics. Then, a value is selected for each of the parameters in Equation B-1, based on the underlying probability distributions developed for each of those variables, and specific to the sex and source water as specified for that individual as appropriate.

The Equation B-1 calculation is carried out to determine that individual's lifetime cancer risk,  $R_{\text{Ind}}$ . The results of all 2,000 iterations are aggregated, and the average individual risk across all iterations is determined. This average risk value, multiplied by the number of individuals in the populations served by the affected water systems, provides the number of cases of cancer expected.

To complete the benefits modeling, a baseline with no reduction in the MCL (or arsenic levels in drinking water) is run first, with subsequent runs reflecting reductions in occurrence levels corresponding to the particular MCL being evaluated. The number of cancer cases estimated for these runs at the various MCL options is subtracted from the baseline cancer cases to obtain the estimate of cases avoided.

The following sections provide further discussion of the components of the model, including further information on how upper and lower bounds for the benefits estimates were established, how additional adjustments have been made to account for the differences in dietary intake of arsenic, and to reflect differences in cancer mortality rates between the affected US population and the Taiwan population that served as the basis of the unit risk factors.

### **B.1.2 Arsenic Concentrations in Finished Water of Community Water Systems Serving Fewer than One Million People**

This section provides further information on the variable  $C(\text{As})_{\text{Ind}}$  in Equation B-1.

EPA has developed lognormal arsenic occurrence distributions for the nation's community ground water and surface water systems serving fewer than one million people. These arsenic occurrence distributions, which reflect the probability of arsenic concentrations occurring at various levels in finished drinking water in surface and ground water systems, are used in the benefits model to characterize the variability in arsenic drinking water concentrations experienced by different individuals using these public water supplies.

Although the arsenic occurrence distributions were developed to characterize the full distribution of finished water arsenic concentrations, the benefits modeling focused only on the portion of those distribution exceeding  $3 \mu\text{g/L}$ , the lowest MCL option considered by EPA. EPA used the separate lognormal occurrence probability distributions for ground water and surface to first determine the number of people served by community water systems from each of those two source waters (and the total) expected to have arsenic present above  $3 \mu\text{g/L}$ .

In the Monte Carlo simulation model, the selection of a value for  $C(\text{As})_{\text{Ind}}$  of Equation B-1 in each iteration involved two steps. First, using relative probabilities derived from the lognormal occurrence distributions, an individual was selected and identified as being served by either ground or surface water having an arsenic above  $3 \mu\text{g/L}$ . In the second step, a specific finished water arsenic concentration was chosen at random from the appropriate ground or surface water occurrence distribution in the range exceeding  $3 \mu\text{g/L}$ .

By including a sufficient number of iterations in the Monte Carlo model, the full range of individual variability in exposure to different arsenic concentrations in the range of interest for both surface water and ground water sources is obtained.

In the baseline analysis (that is, with no change to the  $50 \mu\text{g/L}$  MCL), the selected finished water arsenic concentration value was used directly in the risk equation. In the model runs for various MCL options, that value was compared to the MCL. If that value was less than or equal to the MCL, it was also kept. If however the selected value exceeded the MCL, then it was multiplied by a factor of 0.8 of the MCL value reflecting an assumption that systems would treat to a level of 80% of the MCL. So, for example, if an iteration of a model run examining the  $10 \mu\text{g/L}$  MCL option produced a finished water arsenic value of  $25 \mu\text{g/L}$ , that value was changed to  $8 \mu\text{g/L}$ . If the model run were for the option of a  $20 \mu\text{g/L}$  MCL, that value would be changed to  $16 \mu\text{g/L}$ .

It should be noted that for the purposes of the benefits modeling, the concentration used is implied to be a lifetime average exposure level for the individual in that iteration.

### **B.1.3 Drinking Water Consumption**

This section provides further information on the variables  $DW_{\text{Ind}}$  and  $DW_{\text{Adj}}$  in Equation B-1.

The variable  $DW_{\text{Ind}}$  reflects the differences (variability) in individual water consumption within the exposed population. In Equation B-1, the variable  $DW_{\text{Ind}}$  is expressed in units of L/kg-day reflecting differences in consumption among individuals in the population as a function of body weight. This value is a lifetime average water consumption rate for individuals, recognizing that consumption of water per kg body weight changes over a lifetime, particularly between infancy, childhood and adulthood.

EPA obtained the distribution of individual weighted average lifetime water consumption values in terms of L/kg-day by integrating available data on the distribution of water consumption, in units of L/day, by males and females in the US in various age ranges with information on the distribution of body weights for males and females within those same age ranges.

The age and sex specific distributions of drinking water consumption in L/day are provided by data from the Continuing Survey of Food Intakes by Individuals (CSFII) for the years 1994-1996 conducted by the U.S. Department of Agriculture (USDA) and presented in EPA (1999). The data were collected from a sample population of 15,303 individuals in the 50 states and the District of Columbia that was chosen to be representative of the US population based on the 1990 census data.

The collection and analysis of drinking water consumption data in the CSFII provided the basis for several alternative ways of viewing drinking water consumption, in particular, how to include various direct water sources – for example, from community tap water, bottled water, household wells – consumed directly as a beverage, and indirect water that is water from such sources that is added to other foods during preparation at home or by food service establishments.

For the purposes of the arsenic benefits analysis, EPA chose to use two alternative sets of drinking water distributions to characterize lower and upper bounds of risk.

For the lower bound analyses, EPA used the CSFII drinking water distribution limited to the community tap water source, but which included both direct and indirect consumption of that water. This lower bound distribution reflects an overall average individual consumption (across all ages and both sexes) of approximately 1.0 L/day, with a 90<sup>th</sup> percentile value of approximately 2.1 L/day.

For the upper bound analyses, EPA used the CSFII drinking water distribution for total water, which includes community tap water, bottled water, and other sources, and also reflects both direct and indirect consumption of that water. This upper bound distribution reflects an overall average individual consumption (across all ages and both sexes) of approximately 1.2 L/day, with a 90<sup>th</sup> percentile value of approximately 2.3 L/day.

For the purposes of the arsenic benefits analysis, it was necessary to integrate the age and sex specific water consumption distributions (in L/day) with information available from Statistical Abstracts (1994) providing body weight distributions for the same sex-age categories included in the CSFII data. A submodel was run for this portion of the benefits analysis that effectively generated  $DW_{\text{Int}}$  values for individuals by “constructing” a lifetime weighted average water

consumption value in units of L/kg-day. Five age categories, based on the manner in which CSFII data were presented, were used for building these lifetime consumption values. These age categories were:

- < 1
- 1 – 10
- 11 – 19
- 20 – 64
- 65 – 70

Again, CSFII provided water consumption information separately for males and females in each of these categories, and Statistical Abstracts (1994) provided body weight distributions for these categories. In the simulation, an individual is selected, male or female according to the proportions of 51.9% male, 48.1% female. A value for water consumption in L/day and an average body weight for each of the five age categories is selected, and an average intake for each age category is computed by dividing the water consumption value selected by the body weight selected.

The individual's lifetime weighted average ( $DW_{\text{ind}}$  in Equation B-1) is then computed by averaging across the five age groups, weighting each appropriately for the number of years spent in that age range.

An additional adjustment factor had to be incorporated into Equation B-1 in order to account for the fact that the cancer unit risk factors used were calculated with an underlying assumption that it applied to an "average" person weighing 70 kg and consuming 2 L/day over the entire 70 year lifetime (or 0.0286 L/kg-day). Since drinking water consumption is being modeled in this analysis to explicitly account for the variability in water consumption as a function of body weight, proceeding without this adjustment would overestimate the cancer risk for those individuals with a lifetime weighted average consumption of less than 0.0286 L/kg-day, and similarly would underestimate it for those consuming more than 0.0286 L/kg-day as a lifetime average.

Because, as noted from the CSFII data, average water consumption across all age and sex groups is closer to 1.0 – 1.3 L/day and because lifetime average body weights are (especially for females) lower than 70 kg, failing to make this adjustment would in the aggregate overestimate cancer risk.

By applying the  $DW_{\text{Adj}}$  adjustment factor of 70 kg/(2 L/day) to the water consumption values obtained in the simulation, this correction for the underlying basis of the risk value is accomplished.

The water consumption and adjustment discussed above are described in greater detail in the RIA and its accompanying Appendix B. In that analysis, the product of the water consumption and the adjustment factor are described as the Lifetime Relative Exposure Factors (LREF), which reflects the exposure and risk relative to the 70 kg, 2 L/day (i.e., 0.0286 L/kg-day) person. In that

more detailed analysis, it is shown that the overall distribution of these factors tends to be lognormal with means and standard deviations as shown in Exhibit B-1 for both males and females and the lower and upper bound water consumption distributions. In essence, these LREF values indicate that, on average, individual exposure and risk are about 60% to 80% of what they would be if every individual were assumed to be a 70 kg, 2 L/day person.

**Exhibit B-1**  
**Summary of Lifetime Relative Exposure Factors (LREF):**  
**(Product of  $DW_{Ind}$  \*  $DW_{Adj}$  Overall Distributions are Lognormal)**

	Community Water Consumption Data	Total Water Consumption Data
Male	Mean = 0.60 s.d. = 0.61	Mean = 0.73 s.d. = 0.62
Female	Mean = 0.64	Mean = 0.79

#### B.1.4 Cancer Risk Factors

This section provides further information on the variable  $R_{Unit}$  in Equation B-1.

In its 1999 report, “Arsenic in Drinking Water,” the NRC analyzed bladder cancer risks using data from Taiwan. In addition, NRC examined evidence from human epidemiological studies in Chile and Argentina, and concluded that risks of bladder and lung cancer had comparable risks to those “in Taiwan at comparable levels of exposure” (NRC, 1999). The NRC also examined the implications of applying different statistical analyses to the newly available Taiwanese data for the purpose of characterizing bladder cancer risk. While the NRC’s work did not constitute a formal risk analysis, they did examine many statistical issues (e.g., measurement errors, age-specific probabilities, body weight, water consumption rate, comparison populations, mortality rates, choice of model) and provided a starting point for additional EPA analyses. The report noted that “poor nutrition, low selenium concentrations in Taiwan, genetic and cultural characteristics, and arsenic intake from food” were not accounted for in their analysis (NRC, 1999, pg. 295). In the June 22, 2000 proposed rule, EPA calculated bladder cancer risks and benefits using the bladder cancer risk analysis from the NRC report (NRC, 1999). We also estimated lung cancer benefits in a “What If” analysis based on the statement in the 1999 NRC report that “some studies have shown that excess lung cancer deaths attributed to arsenic are 2-5 fold greater than the excess bladder cancer deaths” (NRC, 1999).

In July, 2000, a peer reviewed article by Morales et al. (2000) was published, which presented additional analyses of bladder cancer risks as well as estimates of lung and liver cancer risks for the same Taiwanese population analyzed in the NRC report. EPA summarized and analyzed the new information from the Morales et al. (2000) article in a NODA published on October 20, 2000 (65 FR 63027; EPA, 2000). Although the data used were the same as used by the NRC to analyze bladder cancer risk in their 1999 publication, Morales et al. (2000) considered more dose-response models and evaluated how well they fit the Taiwanese data for both bladder cancer risk and lung cancer risk. Ten risk models were presented in Morales et al. (2000) used with and

without one of two comparison populations. After consultation with the primary authors (Morales and Ryan), EPA chose Model 1 with no comparison population for further analysis.

EPA believes that the models in Morales et al. (2000) without a comparison population are more reliable than those with a comparison population. Models with no comparison population estimate the arsenic dose-response curve only from the study population. Models with a comparison population include mortality data from a similar population (in this case either all of Taiwan or part of southwestern Taiwan) with low arsenic exposure. Most of the models with comparison populations resulted in dose-response curves that were supralinear (higher than a linear dose response) at low doses. The curves were “forced down” near zero dose because the comparison population consists of a large number of people with low risk and low exposure. EPA believes, based on discussions with the authors of Morales et al. (2000), that models with a comparison population are less reliable, for two reasons. First, there is no basis in data on arsenic’s carcinogenic mode of action to support a supralinear curve as being biologically plausible. To the contrary, the conclusion of the NRC panel (NRC, 1999) was that the mode of action data led one to expect dose responses that would be either linear or less than linear at low dose. However, the NRC indicated that available data are inconclusive and “...do not meet EPA’s 1996 stated criteria for departure from the default assumption of linearity.”(NRC, 1999)

Second, models that include comparison populations assume that the study and comparison populations are the same in all important respects except for arsenic exposure. Yet Morales et al. (2000) agree that “[t]here is reason to believe that the urban Taiwanese population is not a comparable population for the poor rural population used in this study.” Moreover, because of the large amount of data in the comparison populations, the model results are sensitive to assumptions about this group. Evidence that supports these arguments are that the risks in the comparison groups are substantially lower than in similarly exposed members of the study group and the shape of the estimated dose-response changes sharply as a result. For these reasons, EPA believes that the models without comparison populations are more reliable than those with them. Of the models that did not include a comparison population, EPA believes that Model 1 best fits the data, based on the Akaike Information Criterion (AIC), a standard criterion of model fit, applied to Poisson models. In Model 1, the relative risk of mortality at any time is assumed to increase exponentially with a linear function of dose and a quadratic function of age.

Morales et al. (2000) reported that two other models without comparison populations also fit the Taiwan data well: Model 2, another Poisson model with a nonparametric instead of quadratic age effect, and a multi-stage Weibull (MSW) model. Under Model 2, the points of departure for male and female bladder and lung cancer are from 1% to 11% lower than under Model 1, but within the 95% confidence bounds from Model 1. Model 2 therefore implies essentially the same bladder and lung cancer risks as Model 1. Under the MSW model, compared to Model 1, points of departure are 45% to 60% higher for bladder cancer and for female lung cancer, and 38% lower for male lung cancer. EPA did not consider the MSW model for further analysis, because this model is more sensitive to the omission of individual villages (Morales et al., 2000) and to the grouping of responses by village (NRC, 1999), as occurs in the Taiwanese data. However, if the MSW model were correct, it would imply a 14% lower combined risk of lung and bladder cancers than Model 1, among males and females combined.

Considering all of these results, the Agency decided that the more exhaustive statistical analysis of the data provided by Morales et al. (2000), as analyzed by EPA, would be the basis for the new risk calculations for the final rule (with further consideration of additional risk analyses) and other pertinent information. The Agency views the results of the alternative models described above as an additional uncertainty which was considered in the decision concerning the selection of the final MCL.

The specific lifetime risk measures provided in the Morales (2000) study that were used in this benefits analysis, and their conversion to the  $R_{Unit}$  values of cases per person per  $\mu\text{g/L}$  are shown in Exhibit B-2, below.

**Exhibit B-2**  
**Risk Measures from Morales (2000) and as Used in this Benefit Analysis**

	Bladder Cancer		Lung Cancer	
	Males	Females	Males	Females
$ED_{01}$ ( $\mu\text{g/L}$ )	395	252	364	258
Mean for $R_{Unit}$ (cases/person per $\mu\text{g/L}$ )	$2.53 \times 10^{-5}$	$3.97 \times 10^{-5}$	$2.75 \times 10^{-5}$	$3.88 \times 10^{-5}$
$LED_{01}$ ( $\mu\text{g/L}$ )	326	211	294	213
Upper 95% CL for $R_{Unit}$ (cases/person per $\mu\text{g/L}$ )	$3.07 \times 10^{-5}$	$4.74 \times 10^{-5}$	$3.40 \times 10^{-5}$	$4.69 \times 10^{-5}$

The  $ED_{01}$  values provided by Morales (2000) indicate that this is the arsenic concentration in drinking water that if consumed by an individual over a lifetime (with the assumption of 2 L/day and 70 kg body weight) has a 0.01 risk (i.e., 1% probability) of resulting in the indicated form of cancer. The  $LED_{01}$  is the lower 95% confidence bound on the dose producing that 0.01 risk

To be used in the benefits calculation shown in Equation B-1, these risk measures are converted to the units of cases/person per  $\mu\text{g/L}$  needed for  $R_{Unit}$  by simply dividing 0.01 by the corresponding  $ED_{01}$  or  $LED_{01}$   $\mu\text{g/L}$  values.

In the Monte Carlo simulation, the  $R_{Unit}$  value was incorporated as normal distribution with parameters based on the mean and upper 95% confidence limit as shown in Exhibit 3-D.2

### **B.1.5 Upper and Lower Bound Considerations**

In carrying out the arsenic benefits analysis, differing assumptions were used in an effort to establish upper and lower bounds on the estimated risks and avoided cases of cancer associated with the arsenic MCL. Some of the factors considered in the upper and lower bound estimates were noted in the preceding discussions. These are discussed more fully here.

For the upper bound analyses, EPA used:

- 7 The surface water and ground water occurrence distributions as provided in the occurrence analyses;
- 7 The drinking water consumption distribution using the total water consumption data from CSFII (i.e., averaging approximately 1.2 L/day)
- 7 The unit cancer risk factor distribution based on the  $R_{\text{Unit}}$  values shown in Exhibit 3.D.2.

For the lower bound analyses, EPA used:

- 7 The surface water and ground water occurrence distributions as provided in the occurrence analyses (same as upper bound);
- 7 The drinking water consumption distribution using the community (tap) water consumption data from CSFII (i.e., averaging approximately 1.0 L/day)
- 7 The unit cancer risk factor distribution based on the  $R_{\text{Unit}}$  values shown in Exhibit B-2 for males only (applied to both males and females), with further downward adjustments for potential contributions from water used in cooking and from food in the Taiwan population used to derive the risk factors

The use of the two different drinking water consumption distributions in establishing upper and lower bounds estimates were discussed previously. The other two adjustments noted in the third bullet for the lower bound estimates are described further here. Both of these adjustments reflect possible contributions to the cancer cases observed in the Taiwan study associated with arsenic in the water or food for that population that would not necessarily apply to the US population.

First, the Agency made an adjustment to the lower bound risk estimates to take into consideration the effect of exposure to arsenic through water used in preparing food in Taiwan. The Taiwanese staple foods were dried sweet potatoes and rice (Wu et al., 1989). Both the 1988 EPA “Special Report on Ingested Inorganic Arsenic” report and the 1999 NRC report assumed that an average Taiwanese male weighed 55 kg and drank 3.5 liters of water daily, and that an average Taiwanese female weighed 50 kg and drank 2 liters of water daily. Using these assumptions, along with an assumption that Taiwanese men and women ate one cup of dry rice and two pounds of sweet potatoes a day, the Agency re-estimated risks for bladder and lung cancer, using one additional liter water consumption for food preparation (i.e., the water absorbed by hydration during cooking). This adjustment was discussed and used in the October 20, 2000 NODA (65 FR 63027).

Second, an adjustment was made to the lower bound risk estimates to take into consideration the relatively high arsenic concentration in the food consumed in Taiwan as compared to the U.S. The food consumed daily in Taiwan contains about 50 Fg, versus about 10 Fg in the U.S. (NRC, 1999, pp. 50–51). Thus the total consumption of inorganic arsenic (from food preparation and drinking water) is considered, per kilogram of body weight, in the process of these adjustments. To carry them out, the relative contribution of arsenic in the drinking water that was consumed as drinking water, on a Fg/kg/day basis, was compared to the total amount of arsenic consumed in drinking water, drinking water used for cooking, and in food, on a Fg/kg/day basis.

Other factors contributing to lower bound uncertainty include the possibility of a sub-linear dose-

response curve below the point of departure. The NRC noted “Of the several modes of action that are considered most plausible, a sub-linear dose response curve in the low-dose range is predicted, although linearity cannot be ruled out.” (NRC,1999). The recent Utah study (Lewis et al., 1999), described in section 5.G.1(b), provides some evidence that the shape of the dose-response curve may well be sub-linear at low doses. Because sufficient mode of action data were not available, an adjustment was not made to the risk estimates to reflect the possibility of a sub-linear dose-response curve. Additional factors contributing to uncertainty include the use of village well data rather than individual exposure data, deficiencies in the Taiwanese diet relative to the U.S. diet (selenium, choline, etc.), and the baseline health status in the Taiwanese study area relative to U.S. populations. The Agency did not make adjustments to the risk estimates to reflect these uncertainties because applicable peer-reviewed, quantitative studies on which to base such adjustments were not available.

### B.1.6 Estimated Population Risk Values

The Monte Carlo simulation performed for this benefits analysis using the risk algorithm shown in Equation B-1 produce distributions of individual risk values ( $R_{Ind}$ ) for the baseline and the various MCL options considered, and for both the upper and lower bound sets of assumptions. Exhibit B-3 provides some summary statistics for the resulting distribution of risks. Note that the “exposed population” addressed in this table are those individuals using community ground or surface water supplies serving fewer than one million people having arsenic levels greater than 3 µg/L.

The key outputs resulting from this Monte Carlo simulation for estimating cancer cases avoided are the mean risk values shown in Exhibit B-3. The application of these mean risk values to estimate cases avoided is described in the following section.

**Exhibit B-3**  
**Cancer Risks for U.S. Populations**  
**Exposed At or Above MCL Options, after Treatment**  
**(Lower Bound With Food and Cooking Water Adjustment)**

	Mean Risk for Exposed Population (Lower and Upper Bounds)	90 <sup>th</sup> Percentile Risk for Exposed Population (Lower and Upper Bounds)
3	0.11 - 1.25 x 10 <sup>-4</sup>	0.22 - 2.42 x 10 <sup>-4</sup>
5	0.27 - 2.02 x 10 <sup>-4</sup>	0.55 - 3.9 x 10 <sup>-4</sup>
10	0.63 - 2.99 x 10 <sup>-4</sup>	1.32 - 6.09 x 10 <sup>-4</sup>
20	1.10 - 3.85 x 10 <sup>-4</sup>	2.47 - 8.37 x 10 <sup>-4</sup>

### B.1.7 Estimated Cancer Cases and Cases-Avoided

To estimate the number of cancer cases avoided for the various MCL options it is necessary to first calculate the number of cases expected at the baseline risk level (no change in the MCL, or 50 µg/L), and then for each MCL option. Baseline mean risk values and estimated mean risk levels for the various MCL options (shown in Exhibit B-3) are multiplied by the total number of people served by community ground and surface water systems serving fewer than one million people. Because the lower bound risk adjustments are also made to the baseline risk (the risk at 50 µg/L), the baseline number of expected cases in the adjusted risk scenario is not the same (it's lower, just as the adjusted risks are lower) as the baseline number of expected cases in the unadjusted risk scenario. The number of cases avoided at each MCL alternative is determined by subtracting the number of cases remaining at each option from the appropriate baseline number of cases. Thus, to estimate cases avoided, the number of remaining cases expected at the lower risk levels are subtracted from the number of cases expected at the lower baseline level, and the number of remaining cases expected at the higher risk levels are subtracted from the number of cases expected at the higher baseline level.

An upper bound adjustment was made to the number of bladder cancer cases avoided to reflect a possible lower mortality rate in Taiwan than was assumed in the risk assessment process described earlier. EPA also made this adjustment in the June 22, 2000, proposal. In the Taiwan study area, information on arsenic related bladder and lung cancer deaths was reported. In order to use these data to determine the probability of contracting bladder and lung cancer as a result of exposure to arsenic, a probability of mortality given the onset of arsenic induced bladder and lung cancer among the Taiwanese study population must be assumed. The study area in Taiwan is a section where arsenic concentrations in the water are very high by comparison to those in the U.S., and an area of low incomes and poor diets, where the availability and quality of medical care is not of high quality, by U.S. standards. In its estimate of bladder cancer risk, the Agency assumed that within the Taiwanese study area, the probability of contracting bladder cancer was relatively close to the probability of dying from bladder cancer (that is, that the bladder cancer incidence rate was equal to the bladder cancer mortality rate).

We do not have data on the rates of survival for bladder cancer in the Taiwanese villages in the study and at the time of data collection. We do know that the relative survival rates for bladder cancer in developing countries overall ranged from 23.5% to 66.1 % in 1982-1992 ("Cancer Survival in Developing Countries," International Agency for Research on Cancer, World Health Organization, Publication No. 145, 1998). We also have some information on annual bladder cancer mortality and incidence for the general population of Taiwan in 1996. The age-adjusted annual incidence rates of bladder cancer for males and females, respectively, were 7.36 and 3.09 per 100,000, with corresponding annual mortality rates of 3.21 and 1.44 per 100,000 (correspondence from Chen to Herman Gibb, January 3, 2000). Assuming that the proportion of males and females in the population is equal, these numbers imply that the mortality rate for bladder cancer in the general population of Taiwan, at present, is 45%. Since survival rates have most likely improved over the years since the original Taiwanese study, this number represents a lower bound on the survival rate for the original area under study (that is, one would not expect a higher rate of survival in that area at that time). This has implications for the bladder cancer risk estimates from the Taiwan data. If there were any persons with bladder cancer who recovered and died from some other cause, then our estimate underestimated risk; that is, there were more

cancer cases than cancer deaths. Based on the above discussion, we think bladder cancer incidence could be no more than 2 fold bladder cancer mortality; and that an 80% mortality rate would be plausible. Thus we have adjusted the upper bound of cases avoided, which is used in the benefits analysis, to reflect a possible mortality rate for bladder cancer of 80%. Because lung cancer mortality rates are quite high, about 88% in the U.S.(US EPA, 1998b), the assumption was made that all lung cancers in the Taiwan study area resulted in fatalities.

The total number bladder and lung cases avoided at each MCL are shown in Exhibit 3-D.3. These cases avoided include CWS and NTNC cases. The number of bladder and lung cancer cases avoided range from 57.2 to 138.3 at an MCL of 3 Fg/L, 51.1 to 100.2 at an MCL of 5 Fg/L, 37.4 to 55.7 at an MCL of 10 Fg/L, and 19.0 to 19.8 at an MCL of 20 Fg/L. The cases avoided were divided into premature fatality and morbidity cases based on U.S. mortality rates. In the U.S. approximately one out of four individuals who is diagnosed with bladder cancer actually dies from bladder cancer. The mortality rate for the U.S. is taken from a cost of illness study recently completed by EPA (US EPA, 1998b). For those diagnosed with bladder cancer at the average age of diagnosis (70 years), the probability for dying of that disease during each year post-diagnosis were summed over a 20-year period to obtain the value of 26 percent. Mortality rates for U.S. bladder cancer patients have decreased overall by 24 percent from 1973 to 1996. For lung cancer, mortality rates are much higher. The comparable mortality rate for lung cancer in the U.S. is 88% (US EPA, 1998b).

## **B.2 Community Water Systems Serving More than One Million People**

A separate analysis of the number of cancer cases and cases avoided was performed for community water systems serving more than one million people each. This analysis was based upon specific information available for each on the occurrence of arsenic in specific sources (entry points) for those systems, the flows for those entry points, and the number of people served by those specific systems.

Only three systems serving more than one million people were found to have arsenic levels in one or more entry point exceeding 3 µg/L: Phoenix, Houston, and Los Angeles.

The basic risk algorithm used for systems serving fewer than one million people as shown in Equation B-1 was also used for calculating cancer cases and cases avoided for the systems serving more than one million people.

There were two primary difference in the application of Equation B-1 for the systems serving more than one million people relative to its application for systems serving fewer than one million. First, the analysis was not done as a Monte Carlo simulation, but was based on average values for the variables in the equation. For example. the  $R_{Unit}$  values used were equivalent to the mean risk values for the upper and lower bound risks as shown previously in exhibit B-2 (with the various adjustments made to the lower bound value for the potential impacts of other intakes as described earlier).

The water consumption and adjustment factors  $[DW_{Ind} * DW_{Adj}]$  were simplified and used as average values rather than distributions.

The arsenic water concentrations  $[C(As)_{Ind}]$  used were calculated separately for each of the three very large systems using system-specific data. These calculations were carried out as follows.

Data was available on the arsenic concentration at each of the ground water and surface water entry points at each of these three very large systems. Data were also available on the average daily flow for the ground water and surface water sources in total.

EPA used that information to calculate an initial average arsenic concentration,  $C_{Initial}$ , for that portion of the system exceeding a particular MCL option as follows.

$$C_{Initial} = \frac{C_{GA} \left[ \frac{EP_{GA}}{EP_{GT}} \cdot \frac{F_G}{F_T} \right] + C_{SA} \left[ \frac{EP_{SA}}{EP_{ST}} \cdot \frac{F_S}{F_T} \right]}{\left[ \frac{EP_{GA}}{EP_{GT}} \cdot \frac{F_G}{F_T} \right] + \left[ \frac{EP_{SA}}{EP_{ST}} \cdot \frac{F_S}{F_T} \right]}$$

where:

$C_{GA}$  = the average arsenic concentration in the ground water entry points affected at that MCL option

$C_{SA}$  = the average arsenic concentration in the surface water entry points affected at that MCL option

$EP_{GA}$  = the number of ground water entry points affected at that MCL option

$EP_{GT}$  = the total number of ground water entry points in that system

$EP_{SA}$  = the number of surface water entry points affected at that MCL option

$EP_{ST}$  = the total number of surface water entry points in that system

$F_G$  = the total average daily flow from all ground water sources

$F_S$  = the total average daily flow from all surface water sources

$F_T$  = the total average daily flow from all water sources

These  $C_{Initial}$  values were used for  $C(As)_{Ind}$  in Equation B-1 to calculate the number of baseline cases in the population affected by the particular MCL option. The number of individuals in the population affected for a particular option at each of the very large systems was calculated as being the same portion of the total population served by that system as the portion of total flow affected at the given MCL option.

The post-regulatory cases remaining were calculated using the same procedure, except that a constant value was used for  $C(As)_{Ind}$  that was equal to  $0.8 * MCL$  value.

### B.3 Non-Transient Non-Community Water Systems

#### B.3.1 Data Inputs

Most of the data described above under the CWS risk model is also used in the NTNC risk model. This includes water consumption, body weight, and lifetime risk estimates. Also, the ground water arsenic concentrations at each MCL used in the CWS risk model are used in the NTNC risk model.

### **B.3.1.1 NTNC Service Categories, Population and Exposure Time**

The main differences between the CWS and NTNC risk models are how population is distributed among the different types of establishments that make up the NTNC category of systems, and the extent to which the worker and customer populations within a service category are exposed to arsenic (both in terms of length of exposure and drinking water consumed).

In addition to the CWS data already discussed, Exhibits B-4 and B-5 provide all of the data inputs necessary to model the bladder cancer risk associated with NTNC systems. First, note that in Exhibit B-4, the NTNC universe has been divided into 35 service categories. This was accomplished using the system descriptions in SDWIS (EPA, 1999b). For each service category, the total number of NTNCs and the population served by these NTNCs is taken from SDWIS. The population served by each NTNC often varies daily; the SDWIS population numbers are interpreted to mean the peak population served (both workers and customers).

The next data field in Exhibit B-4 is the number of customer cycles per year, or the number of times each year the customer base turns over. For example, if this parameter equals one, then the same customer's are served each day. If the value is seven, then seven sets of customers use the facility. The next field is the number of workers per person per day. For example, if the value is 0.1, as in the case of summer camps, then 10 percent of the peak population served (from SDWIS) is assumed to be workers. Both the number of customer cycles per year assumptions and workers per person per day data assumptions were made after investigating numerous data sources, including trade-journals and trade association information.

The next set of data fields in Exhibit B-4 are assumptions about the characteristics of the workers in each service type. The percent of workers' daily consumption is the percentage of drinking water consumed on a work day that is consumed at work. This value is assumed to be either 50 percent or 100 percent, depending on the service category. The number of days a person works is assumed to be 250 for all service categories. The number of years a person works at the NTNC establishment is assumed to be either 40 or 10, depending on the service category.

Information regarding customer behavior is provided in the next set of data fields in Exhibit B-4. The percent of customers' daily consumption is the percentage of total drinking water consumed on a day that the customer visits the NTNC, that is consumed at the NTNC. This value is assumed to be either 25 percent, 50 percent or 100 percent, depending on the service category. The number of days a customer visits the NTNC is provided for each service category. For example, the value for nursing homes of 365 indicates that nursing home customers are served by the nursing home year round, while the value for churches of 52 indicates that churches are assumed to serve their customers once per week. The number of years a person is assumed to visits each service category is also provided.

Finally, the total exposed worker and customer populations for each service category are provided in Exhibit B-4. These numbers are calculated as follows:

$$TC_c = (P_c * CC_c) * (1 - WP_c)$$

$$TW_c = P_c * WP_c$$

where:

TC = total number of customers

TW = total number of workers

P = SDWIS population

WP = workers per person per day

CC = number of customer cycles per year

c = NTNC service category

Exhibit B-5 provides the final set of data required to estimate bladder cancer risk from NTNCs. The percent of worker lifetime exposure is the percent of lifetime water consumption which is consumed at the NTNC by a worker. The percent of customer lifetime exposure is the percent of lifetime water consumption consumed at the NTNC by a customer. These numbers are calculated as follows:

$$PWLE_c = \frac{PWDC_c * DW_c * YW_c}{365 * 70}$$

$$PCLE_c = \frac{PCDC_c * DC_c * YC_c}{365 * 70}$$

where;

PWLE = percent of worker lifetime exposure

PCLE = percent of customer lifetime exposure

PWDC = percentage of workers daily consumption

PCDC = percentage of customers daily consumption

DW = worker days per year

DC = customer days per year

YW = worker years

YC = customer years

Returning to Exhibit B-5, the worker age bracket is the age range (corresponding to the age ranges used in the CWS risk analysis) that a NTNC worker is assumed to fall in. For all service categories, the worker age bracket is assumed to be 20-64 years of age. The customer age bracket is the age range (corresponding to the age ranges used in the CWS risk analysis) that a NTNC customer is assumed to be in. For most service categories, the customer age bracket is

assumed to be 0-70 years of age (all ages). However, certain service categories only serve certain age groups (e.g. nursing homes and schools), therefore more specific age ranges are assumed.

**Exhibit B-4  
NTNC Population and Exposure Time Data**

	Number of Systems	Total SDWIS Population	Number of Customer Cycles/Year	Worker Per Person Per Day	Percent of Worker's Daily Consumption	Worker Days Per Year	Worker Years	Percent of Customer's Daily Consumption	Customer Days Per Year	Customer Years	Total Worker Population	Total Customer Population
Water Wholesalers	266	66,018	1.00	0	n/a	n/a	n/a	25.0%	270.00	70.00	0	66,018
Mobile Home Parks	104	19,240	1.33	0.046	50.0%	250	40	100.0%	270.00	35.00	885	24,412
Nursing Homes	130	13,910	1.00	0.23	50.0%	250	40	100.0%	365.00	10.00	3,199	10,711
Churches	230	11,500	1.00	0.01	50.0%	250	40	50.0%	52.00	70.00	115	11,385
Golf and Country Clubs	116	11,716	4.50	0.11	50.0%	250	40	50.0%	52.00	70.00	1,289	46,923
Retailers (Food related)	142	45,724	2.00	0.07	50.0%	250	40	25.0%	185.00	70.00	3,201	85,047
Retailers (Non-food related)	695	120,930	4.50	0.09	50.0%	250	40	25.0%	52.00	70.00	10,884	495,208
Restaurants	418	154,660	2.00	0.07	50.0%	250	40	25.0%	185.00	70.00	10,826	287,668
Hotels/Motels	351	46,683	86.00	0.27	50.0%	250	40	100.0%	3.40	40.00	12,604	2,930,759
Prisons/Jails	67	121,940	1.33	0.1	50.0%	250	40	100.0%	270.00	3.00	12,194	145,962
Service Stations	53	12,190	7.00	0.06	50.0%	250	40	25.0%	52.00	54.00	731	80,210
Agricultural Products/Services	368	27,968	7.00	0.125	50.0%	250	40	25.0%	52.00	50.00	3,496	171,304
Daycare Centers	809	61,484	1.00	0.145	50.0%	250	10	50.0%	250.00	5.00	8,915	52,569
Schools	8,414	3,086,012	1.00	0.073	50.0%	200	40	50.0%	200.00	12.00	225,279	2,860,733
State Parks	83	106,895	26.00	0.016	50.0%	250	40	50.0%	14.00	70.00	1,710	2,734,802
Medical Facilities	367	163,631	16.40	0.022	50.0%	250	40	100.0%	6.70	10.30	3,600	2,624,510
Campgrounds/RV Parks	123	19,680	22.50	0.041	50.0%	180	40	100.0%	5.00	50.00	807	424,645
Federal Parks	20	780	26.00	0.016	50.0%	250	40	50.0%	14.00	70.00	12	19,956
Highway Rest Areas	15	6,105	50.70	0.01	50.0%	250	40	50.0%	7.20	70.00	61	306,428
Misc. Recreation Services	259	22,533	26.00	0.016	50.0%	250	40	100.0%	14.00	70.00	361	576,484
Forest Service	107	4,494	26.00	0.016	100.0%	250	40	100.0%	14.00	50.00	72	114,974
Interstate Carriers	287	35,301	93.00	0.304	50.0%	250	40	50.0%	2.00	70.00	10,732	2,284,963
Amusement Parks	159	76,462	90.00	0.18	50.0%	250	10	50.0%	1.00	70.00	13,763	5,642,896
Summer Camps	46	6,716	8.50	0.1	100.0%	180	10	100.0%	7.00	10.00	672	51,377
Airports	101	326,860	36.50	0.308	50.0%	250	40	25.0%	10.00	70.00	100,673	8,255,830
Military Bases	95	67,525	n/a	1	50.0%	250	40	n/a	n/a	n/a	67,525	0
Non-Water Utilities	497	84,490	n/a	1	50.0%	250	40	n/a	n/a	n/a	84,490	0
Office Parks	950	181,600	n/a	1	50.0%	250	40	n/a	n/a	n/a	181,600	0
Manufacturing: Food	768	285,696	n/a	1	50.0%	250	40	n/a	n/a	n/a	285,696	0
Manufacturing: Non-Food	3,356	588,792	n/a	1	50.0%	250	40	n/a	n/a	n/a	588,792	0
Landfills	78	3,432	n/a	1	100.0%	250	40	n/a	n/a	n/a	3,432	0
Fire Departments	41	4,018	n/a	1	100.0%	250	40	n/a	n/a	n/a	4,018	0
Construction	99	5,247	n/a	1	100.0%	250	40	n/a	n/a	n/a	5,247	0
Mining	119	13,447	n/a	1	100.0%	250	40	n/a	n/a	n/a	13,447	0
Migrant Labor Camps	33	2,079	n/a	1	100.0%	250	40	n/a	n/a	n/a	2,079	0
<b>Subtotal =</b>											1,662,407	30,305,774
<b>TOTAL =</b>												<b>31,968,181</b>

**Exhibit B-5**  
**NTNC Percent of Lifetime Exposure and Age at Exposure**

	<b>Percent of Worker Lifetime Exposure</b>	<b>Percent of Customer Lifetime Exposure</b>	<b>Worker Age Bracket</b>	<b>Customer Age Bracket</b>
Water Wholesalers	0.00%	18.49%	n/a	all
Mobile Home Parks	19.57%	36.99%	20 to 64	all
Nursing Homes	19.57%	14.29%	20 to 64	65+
Churches	19.57%	7.12%	20 to 64	all
Golf and Country Clubs	19.57%	7.12%	20 to 64	all
Retailers (Food related)	19.57%	12.67%	20 to 64	all
Retailers (Non-food related)	19.57%	3.56%	20 to 64	all
Restaurants	19.57%	12.67%	20 to 64	all
Hotels/Motels	19.57%	0.53%	20 to 64	all
Prisons/Jails	19.57%	3.17%	20 to 64	20 to 64
Service Stations	19.57%	2.75%	20 to 64	16 to 70
Agricultural Products/Services	19.57%	2.54%	20 to 64	all
Daycare Centers	4.89%	2.45%	20 to 64	<5
Schools	15.66%	4.70%	20 to 64	6 to 18
State Parks	19.57%	1.92%	20 to 64	all
Medical Facilities	19.57%	0.27%	20 to 64	all
Campgrounds/RV Parks	14.09%	0.98%	20 to 64	all
Federal Parks	19.57%	1.92%	20 to 64	all
Highway Rest Areas	19.57%	0.99%	20 to 64	all
Misc. Recreation Services	19.57%	3.84%	20 to 64	all
Forest Service	39.14%	2.74%	20 to 64	all
Interstate Carriers	19.57%	0.27%	20 to 64	all
Amusement Parks	4.89%	0.14%	20 to 64	all
Summer Camps	7.05%	0.27%	20 to 64	11 to 19
Airports	19.57%	0.68%	20 to 64	all
Military Bases	19.57%	0.00%	20 to 64	n/a
Non-Water Utilities	19.57%	0.00%	20 to 64	n/a
Office Parks	19.57%	0.00%	20 to 64	n/a
Manufacturing: Food	19.57%	0.00%	20 to 64	n/a
Manufacturing: Non-Food	19.57%	0.00%	20 to 64	n/a
Landfills	39.14%	0.00%	20 to 64	n/a
Fire Departments	39.14%	0.00%	20 to 64	n/a
Construction	39.14%	0.00%	20 to 64	n/a
Mining	39.14%	0.00%	20 to 64	n/a
Migrant Labor Camps	39.14%	0.00%	all	n/a

### B.3.2 The NTNC Risk Model

Just like the CWS risk analysis, the NTNC risk analysis is a Monte-Carlo based simulation model. This section will explain each step in the simulation. The Monte-Carlo simulation is conducted at each MCL option (50, 20, 10, 5 and 3 Fg/L). In addition, for each MCL option, the simulation is carried out for both the “Lower Bound” and “Upper Bound” scenarios just like in the CWS case. Therefore, the simulation model is carried out ten times. Each of these ten “runs” of the model is independent of the other, and can be discussed in isolation. Therefore, this section will include a generalized discussion of the model. The inputs that are used will depend on the MCL option and scenario being evaluated at the time. It is important not to confuse a “run” of the model as just described, and a model iteration. Each run of the model consists of 10,000 iterations. Within a single iteration, the model pulls a value for each variable from its input distribution (e.g. body weight) and calculates a value for each output variable (e.g. lifetime risk). This is done for 10,000 times for each model run. The results of the model run is the distribution of the 10,000 values for each output variable.

The first step of each iteration is to calculate the relative exposure factor for each sex and age category. This is done exactly as it was done in the CWS risk analysis. As shown in the following equations, the relative exposure factor is a function of daily water consumption and body weight.

$$REF_{mai} = \left( \frac{70}{2} \right) * \left( \frac{C_{mai}}{W_{mai}} \right)$$

$$REF_{fai} = \left( \frac{70}{2} \right) * \left( \frac{C_{fai}}{W_{fai}} \right)$$

where;

- REF = relative exposure factor
- C = daily water consumption (L)
- W = body weight (kg)
- i = model iteration number
- a = age category
- m = male
- f = female

Next, the lifetime risk of bladder cancer (1/100,000 people) is calculated for workers and customers of each sex for each service category. The next four equations, therefore are:

$$WLR_{fci} = PWLE_{ci} * AS_{gi} * (RF_i / 50) * \left( \frac{\sum_a (REF_{fai} * Z_{ac})}{\sum_a Z_{ac}} \right) * 100$$

$$WLR_{mci} = PWLE_{ci} * AS_{gi} * (RF_i / 50) * \left( \frac{\sum_a (REF_{mai} * Z_{ac})}{\sum_a Z_{ac}} \right) * 100$$

$$CLR_{mci} = PCLE_{ci} * AS_{gi} * (RF_i / 50) * \left( \frac{\sum_a (REF_{mai} * Z_{ac})}{\sum_a Z_{ac}} \right) * 100$$

$$CLR_{fci} = PCLE_{ci} * AS_{gi} * (RF_i / 50) * \left( \frac{\sum_a (REF_{fai} * Z_{ac})}{\sum_a Z_{ac}} \right) * 100$$

where;

WLR = worker lifetime risk (per 100,000 people)

CLR = customer lifetime risk (per 100,000 people)

AS = arsenic concentration (F g/L)

RF = risk of bladder cancer at 50 F g/L, 2 liters consumption per day, and 70 kg body weight

Z = years spent in age category

g = ground water

The sex of the worker and customer is then chosen for the iteration to determine the worker and customer risk for each service category:

$$WLR_{ci} = \begin{cases} WLR_{mci} & \text{if } RN_1 \leq MP \\ WLR_{fci} & \text{otherwise} \end{cases}$$

$$CLR_{ci} = \begin{cases} CLR_{mci} & \text{if } RN_1 \leq MP \\ CLR_{fci} & \text{otherwise} \end{cases}$$

where;

$RN_1$  = random number between 0 and 1

MP = percentage of the population that is male

Finally, the lifetime risk for the model iteration is determined by choosing among the 70 combinations of worker and customer risk over of the 35 service categories. This is accomplished using a population weighted probability distribution. First, the total worker and customer populations served are computed.

$$TC = \sum_c TC_c$$

$$TW = \sum_c TW_c$$

Next, the probability that the lifetime risk for the model iteration will be equal to the worker lifetime risk associated with a service category is calculated:

$$WPR_c = \frac{TW_c}{(TW + TC)}$$

where;

WPR = probability of choosing lifetime risk estimate for any iteration to be equal to the lifetime risk estimate of a worker in a given service category

Likewise, the probability that the lifetime risk for the model iteration will be equal to the customer lifetime risk associated with a service category is calculated:

$$CPR_c = \frac{TC_c}{(TW + TC)}$$

where;

CPR = probability of choosing lifetime risk estimate for any iteration to be equal to the lifetime risk estimate of a customer in a given service category

Given these probabilities, the lifetime risk estimate for each model iteration is chosen as follows:

$$LR_i = \begin{cases} WLR_{ci} & \text{with Probability } WPR_c \\ \vdots \\ CLR_{ci} & \text{with Probability } CPR_c \end{cases}$$

where;

LR = Lifetime risk (1/100,000)

In order to calculate the expected number of cancer cases associated with the model run, the mean lifetime risk is multiplied by the exposed population as follows:

$$CA = \left( \frac{\sum_{i=1}^N LR_i}{N} \right) * \frac{(TC + TW)}{100,000}$$

where;

CA = expected number of bladder cancer cases

N = number of iterations

## Appendix C. Cost Model Methodology

### C.1 Introduction

EPA used the regulatory cost model, SafeWaterXL, in estimating the annual national costs of compliance for the Arsenic in Drinking Water Rule. SafeWaterXL is a Monte-Carlo simulation model developed in Microsoft Excel using the Crystal Ball add-in.<sup>1</sup> The model is programmed in Visual Basic for Applications, the procedures and functions of which command for example, the user interface and much of the business logic required. These procedures and functions call on data and equations stored in Microsoft Excel spreadsheets, such as data on specific system characteristics (e.g., the number of people served, the type and source of the water system, the decision tree).

SafeWaterXL determines regulatory compliance costs for individual systems and subsequently calculates a national average cost based on the mean value of these data points. SafeWaterXL describes system-level costs in terms of a distribution, from which mean costs and percentile costs are available. Mean costs reflect the costs of treatment trains selected. Treatment trains consist of two main cost components, capital (the cost of constructing or installing equipment) and operation and maintenance (O&M, annual cost of operating equipment and performing routine maintenance) costs for: pre-treatment pre-oxidation technology (if necessary), treatment technology, and waste disposal technology. This modeling approach presents information critical to the assessment of system-level impacts and technology affordability by providing the average compliance costs for each water system type and size category, and the range of costs within each system size and type category.

In understanding how SafeWaterXL calculates annual national cost of compliance, it is important to distinguish between an “iteration” and a “run” of the model. A single iteration of the model represents a single system. This allows for variability in the water system configuration, current treatment in place, and source water quality to be captured in the compliance cost estimates. A model “run” uses data from the aggregate number of iterations to calculate summary cost information for different system size categories. For any individual “run,” only a single source water type may be evaluated, and the results are stratified by sixteen groups: 8 size categories and 2 ownership types (public/private).

### C.2 Data Inputs and Procedure (Single Model Iteration)

The fundamental steps required to conduct an iteration of SafeWaterXL are summarized below:

1. A system is selected from data files. A system is defined by the population it serves.
2. Each system is assigned a random concentration from an occurrence distribution.

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<sup>1</sup>For Windows 95/98/NT: Excel 2000, registered trademark of Microsoft Corporation; Crystal Ball Version 4.0, registered trademark of Decisioneering, Inc.

3. The selected arsenic concentration for the system is distributed across the number of sites (entry points) of possible contamination for that system based on the relative intra-system standard deviation (RSD).
4. The concentration at each site is compared to the revised MCL standard to determine if the site is in violation of the revised standard.
5. If the site is in violation of the revised MCL, the percentage removal of arsenic required in order to reach the treatment target is calculated.
6. Based on the percentage removal required to meet the treatment target and on the decision tree for the size and type of the system, a treatment train is then assigned to the site.
7. Using the removal efficiency of the treatment train chosen, the percentage of flow that must be treated in order for the entry point to meet the treatment target, is calculated.
8. The percentage of flow that needs to be treated is applied to the design flow, which is then used to derive the capital costs of the components of the treatment train (the sum of: treatment capital, waste disposal capital, and any pre-treatment capital costs).
9. Similarly, the percentage of flow that needs to be treated is also applied to the average flow, which is then used to derive the operation and maintenance costs of the components of the treatment train (the sum of: treatment O&M, waste disposal O&M, and any pre-treatment O&M costs).
10. The system's total annual treatment costs are calculated for the selected treatment train at various discount rates, by summing the treatment costs (annualized capital plus annual O&M cost components) across all treating sites.
11. This annual system cost is used to derive the cost per thousand gallons (cost/kgal) delivered by the water system.
12. Annual household costs are then calculated based on the system's unit cost of delivery (cost per thousand gallons) and the average annual household consumption per year.
13. If household costs are determined to exceed an affordability threshold of \$500, a less expensive treatment technology (POU device) is chosen and new costs are calculated (Steps 7-12 above are repeated using data for POU devices).
14. Otherwise, the results are forecasted for each iteration and another system is selected for the next iteration.

This procedure is conducted for all of the size categories and national costs are then calculated. Each step listed above is now described in detail.

- A system is selected from data files.

The basic unit of analysis within the cost model is an individual CWS. The SafeWaterXL model estimates regulatory cost based on a universe of CWSs using a December 1997 freeze of the Safe Drinking Water Information System (SDWIS) dataset, which allows costs to community water systems to be delimited by various system characteristics: source, ownership, and size. SDWIS contains data on all public water systems as reported by States and EPA Regions. This information is used to determine each system's primary raw water source (ground or surface water), its ownership type (public or private), and the population served by the system (service size category). Note that in SDWIS, systems under any influence of surface water are classified as surface water systems.

Included in this group are surface water systems that receive a portion of their flow from ground water sources. In SafeWaterXL, these “mixed systems” were reclassified as ground water systems if they were determined to rely on ground water for more than 50 percent of their water supply. Based on data from the Community Water System Survey (CWSS)<sup>2</sup>, systems were systematically reassigned in order to maintain the same average number of people served for the subset of systems. Approximately nine and twelve percent of non-purchased and purchased surface water systems were reclassified as a result.

The universe of systems modeled in SafeWaterXL also excludes the largest systems, those serving more than 900,000 people. These very large systems, although few in number, are significant contributors to the national cost of compliance estimate. Therefore, for the Arsenic in Drinking Water Rule, EPA did an independent analysis on the 25 very large systems (both ground and surface water source systems) to determine which would be affected at various MCL options. In addition, among the smallest systems (serving <100 people), approximately 150 ground water system were found to serve fewer than 25 people, but for modeling purposes were all assumed to serve 25 people. Due to the sheer number of systems in this size category (>14,000 systems), the effect of this modification was found to be insignificant.

In total, the resulting number of systems are distributed between two data files which the model calls on for system information. The criterion for these two files is source water: ground or surface. Then, within each file, CWSs<sup>3</sup> are first grouped by size category, resulting in eight different worksheets of data corresponding to each delimited category (25-100; 101-500; 501-1,000; 1,100-3,300; 3,301-10,000; 10,001-50,000; 50,001-100,000; 100,001-90,000). The resulting stratification of the 1997 SDWIS freeze used in SafeWaterXL is described in Exhibits C-1 and C-2 below for ground and surface water systems, respectively.

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<sup>2</sup>U.S. EPA. 1999. *Geometries and Characteristics of Public Water Systems*. Prepared for Office of Ground Water and Drinking Water by Science Applications International Corporation. EPA Contract No. 69-C6-0059.

<sup>3</sup>Note that public-purchased systems are analyzed as publicly-owned systems and similarly, private-purchased systems are analyzed as privately-owned system.

**Exhibit C-1.  
Stratification of Community Ground Water Systems**

System Size Category	Publicly-Owned		Privately-Owned		All GW Systems
	Non-Purchased	Purchased	Non-Purchased	Purchased	
<b>25-100</b>	1,217	125	12,893	197	14,432
<b>101-501</b>	4,141	480	10,242	385	15,248
<b>501-1,000</b>	2,574	300	1,798	115	4,787
<b>1,001-3,300</b>	3,847	347	1,599	100	5,893
<b>3,301-10,000</b>	2,027	229	493	35	2,784
<b>10,001-50,000</b>	1,078	207	259	17	1,561
<b>50,001-100,000</b>	126	26	27	1	180
<b>100,001-900,000</b>	74	15	18	--	107
<b>Total</b>	15,084	1,729	27,329	850	44,992

**Exhibit C-2.  
Stratification of Community Surface Water Systems**

System Size Category	Publicly-Owned		Privately-Owned		All SW Systems
	Non-Purchased	Purchased	Non-Purchased	Purchased	
<b>25-100</b>	150	209	404	293	1,056
<b>101-501</b>	348	634	396	490	1,868
<b>501-1,000</b>	331	476	131	212	1,150
<b>1,001-3,300</b>	873	930	225	280	2,308
<b>3,301-10,000</b>	771	567	102	104	1,544
<b>10,001-50,000</b>	724	387	114	35	1,260
<b>50,001-100,000</b>	133	61	31	3	228
<b>100,001-900,000</b>	136	33	33	3	205
<b>Total</b>	3,466	3,297	1,436	1,420	9,619

Systems in each worksheet are further defined by their ownership type and an exact number of people served. A separate decision tree also exists for each size category, such that there are sixteen in total available for analysis in SafeWaterXL, as presented in Appendix A.

For example in this step, a system is selected from one of the data files. Recall that when a model “run” is performed, only one source type may be analyzed at a time. The selection made by the user triggers which data file is utilized. Once designated, assuming all size categories are being analyzed, the model begins with the smallest size category (<100 people served).

- Each system is assigned a random concentration from an occurrence distribution.

The system selected in Step 1 has various associated system characteristics. Each system is also associated with an arsenic occurrence distribution based on the source water. However, these distributions define the universe of systems with the same type of source water using a mean and log standard deviation. To model a single system chosen from the data files, a random system occurrence is selected from this distribution.

In this manner, contaminant occurrence information determines the average system concentration given various system size and source water combinations. Exhibit 6-6 shows the estimated finished water arsenic occurrence distribution for ground and surface water systems. For use in the SafeWaterXL model, EPA performed a regression analysis that weighted actual occurrence data by National Arsenic Occurrence Survey region. On the basis of this, EPA replicated the estimated finished water distribution of ground and surface water systems through a log-normal fit using two sets of distribution parameters. The analysis resulted in the following distribution of systems exceeding various arsenic concentration levels:

**Exhibit C-3**  
**Arsenic Occurrence Distribution, Log-Normal Regression Results**

	3 µg/L	5 µg/L	10 µg/L	20 µg/L
<b>Ground water</b>	19.7%	12.0%	5.3%	2.0%
<b>Surface water</b>	5.6%	3.0%	1.12%	0.37%

\*Percentages represent systems exceeding the arsenic concentration

For ground water systems, the percentages displayed in Exhibit C-3 above were based on a lognormal distribution with a mean of -0.2507 and a log standard deviation of 1.5828. Among surface water systems, the percentages were based on a lognormal distribution of -1.6781 and a log standard deviation of 1.7425.

- The selected arsenic concentration for the system is distributed across the number of sites (entry points) of possible contamination for that system based on the relative intra-system standard deviation (RSD).

Once the system arsenic concentration is determined, the number of entry points, or sites of the system, are determined. The number of sites a system has is another important system characteristic to consider in the analysis because entry points are used as a proxy for the potential or actual points of treatment. Since not all sites in the system are equally likely to exceed the MCL standard, the likelihood of contamination is determined on a site-by-site basis. That is, each system may have more than a single site treating independently.

The average number of sites per system is determined based on the distribution of system intake sites for the size category as estimated from the CWSS. The range of number of sites per system is described in Exhibit C-4 for ground water systems, where a maximum of 37 possible sites was modeled. Linear extrapolation was used to estimate values for the number of sites in cases where survey data was not available.

**Exhibit C-4**  
**Distribution of Entry Points by Size Category Among Ground Water Systems**

<b>System Size Category</b>	<b>Mean</b>	<b>5<sup>th</sup> Percentile</b>	<b>50<sup>th</sup> Percentile (Median)</b>	<b>75<sup>th</sup> Percentile</b>	<b>95<sup>th</sup> Percentile</b>	<b>Maximum</b>
<-100	1	1	1	1	2	4
101-500	1	1	1	1	3	10
501-1,000	2	1	1	2	3	4
1,001-5,000	2	1	1	2	5	6
5,001-10,000	2	1	2	3	5	15
10,001-50,000	4	1	3	5	12	19
50,001-100,000	6	1	4	8	22	37
100,001-900,000	9	1	5	15	28	30

Source: U.S. EPA. 1999. *Geometries and Characteristics of Public Water Systems*. Prepared for Office of Ground Water and Drinking Water by Science Applications International Corporation. EPA Contract No. 68-C6-0059.

Among surface water systems, fewer sites per system exist. About 95 percent of the systems that serve fewer than 50,000 people have only a single entry point. Of the remaining surface water systems that serve greater than 50,000 people, the majority of the systems had fewer than three entry points, although some in the 50,001-100,000 and 100,001-900,000 service size categories were observed to have as many as six and four sites per system, respectively.

The SafeWaterXL model calculates potential costs of compliance at the entry point level, allowing for a maximum of 37, but modeling only the estimated number attributable to each system, based on the distribution described in Exhibit C-4. Once the number of sites within the system is determined from the distribution, the concentration of the contaminant at the site is calculated by applying the assumed relative intra-system standard deviation (RSD) around the mean system concentration. The average concentration of arsenic for that system (from Step 2) is assigned between all the system's sites using a log-normal distribution with the system concentration as the mean, and the intra-system deviation as the standard deviation, which is derived by multiplying the RSD by the system concentration. The RSD is an input ultimately used to distribute the system occurrence between the various entry points of the site. The RSD is a model input provided by the user that feeds into the calculation of the intra-system deviation based on the relationship expressed in Equation 1.

$$RSD = \frac{\text{Intra - System Standard Deviation}}{\text{System Concentration}} \quad (\text{Eq. 1})$$

This distribution used to assign site concentration is bound by zero at the lower limit and by the maximum site concentration (Eq. 2) at the upper limit. Note however, that the sum of the mean arsenic concentration of all sites within a system must still equal the mean arsenic concentration of the system.

$$\text{Max Site Conc.} = (\text{SysConc}) \times (\# \text{ of sites}) \quad (\text{Eq. 2})$$

where: SysConc = arsenic concentration for system

The maximum is set using the assumption that despite the number of entry points, if only one entry is contaminated, its individual concentration cannot exceed a limit such that when averaged across the number of possible sites, the overall concentration would exceed the original concentration determined for that system.

For any given system that has more than a single site, the average system concentration of arsenic for that system is assigned between all the system's sites using this method. Otherwise, if the system has only a single site, then the site concentration must equal the system concentration.

- The concentration at each site is compared to the revised MCL standard to determine if the site is in violation of the standard.

Although the system concentration could itself fall below the MCL, once the system concentration has been distributed between the possible number of entry points, one site may significantly exceed the MCL while the other falls below the MCL such that their average still equals the system concentration. For example, in a system with three sites, there may have two sites whose individual site concentrations are well below the MCL and one site whose concentration exceeds the MCL. In this example, only costs to the third site are calculated. However, if a system has only one site, then that single site is assigned the entire system concentration of arsenic.

For this reason, the concentration of each site of the system is individually compared to the MCL. No costs are incurred for those sites whose concentrations fall below the specified MCL, as no treatment is required. However, if the site is determined to be in violation of the MCL, then treatment costs for regulatory compliance will be calculated and the model must record the data and output information. To do so with the best approximation of the true costs of compliance, only the portion of the system's flow that must be treated to achieve the target MCL level is assigned a cost, as described in Step 5.

- If the site is in violation of the revised MCL, the percentage removal required in order to reach the treatment target is calculated.

If the site is determined to be in violation of the MCL, then SafeWaterXL calculates the percent reduction in the site’s arsenic concentration required to reduce the site concentration to 80 percent of the MCL standard. This is a safety factor which includes a 20 percent excess removal to account for system over-design. The percent of contamination reduction required can be expressed as:

$$\% \text{ removal} = \frac{(\text{SiteConc} - \text{TrtTarget})}{\text{SiteConc.}} \quad (\text{Eq. 3})$$

where:      % removal = percent removal required to meet treatment target  
               SiteConc = arsenic concentration at the treating site  
               TrtTarget = 80 percent of revised MCL

The magnitude of reduction required determines which treatment decision tree is used. A technology is chosen depending on the percentage removal required and treatment train removal efficiencies that will meet the target MCL. The model recognizes three categories of required reduction: <50 percent, 50-90 percent, and >90 percent. Each category is represented by a distinct decision tree of feasible technologies for the amount of removal required. For example, if a site has an influent arsenic level of 50 µg/L, and the target MCL is 2 µg/L, then 96 percent removal is required. Research indicates that lime softening is only capable of achieving approximately 80 percent removal, therefore lime softening would not be a viable treatment option for that site. Therefore, with information about the appropriate amount of removal required for the site to achieve compliance, the model is directed to the corresponding decision tree for a distribution of treatment trains from which to make a selection.

- Based on the percentage of removal required to meet the treatment target and on the decision tree for the size and type of the system, a treatment train is then assigned to the site.

Since entry points may have different site concentrations, it is likely that different treatment technologies would be applied at different sites to meet the target MCL depending on the percentage of removal required to meet the treatment target, and on the removal efficiency of the treatment train selected. The variability of treatment train selection among sites is based on probabilities defined in a decision tree, which contains a range of compliance responses for different system types and sizes, and represent EPA’s best estimate of the treatment train technologies that system operators will choose to achieve a particular percentage reduction in arsenic concentration. Specifically, the compliance decision trees are distributions that identify the percentage of systems in different categories that will choose specific compliance options. For example, the decision tree specifies the probability of different compliance choices for systems with different removal percentages required, baseline influent concentrations, different sizes (e.g., population served), and different sources (groundwater and surface water).

The decision trees are specific to the system’s size categories and source water, and vary according to the contaminant under consideration. SafeWaterXL uses sixteen distinct decision trees in total: one for each of the eight system size categories with ground water and surface water sources. Each decision tree contains a list of treatment trains with three sets of probabilities that would apply to the site, depending on which of three required treatment scenarios the site belongs (<50 percent, 50-90 percent, or >90 percent removal required as described in Step 5). The actual decision tree is illustrated as a flowchart, and is often summarized as a decision matrix, for a particular source water and size category. The matrices used in this analysis were developed for the Revised Arsenic Rule and may be found in Appendix A.

Appendix A describes the treatment technologies, their effectiveness, and the major factors that affected the composition of a particular decision tree. Among some of the centralized treatment options presented include: lime softening, anion exchange, activated alumina, reverse osmosis, and coagulation assisted microfiltration. Some associated waste disposal technologies are also described. Waste disposal technologies are specific to the treatment technology, although their availability does vary between size categories. In addition to these centralized treatment options, small systems may also elect to use point-of-entry (POE) devices to achieve compliance with the MCLs, identified as affordable technologies by the SDWA. The available POE technologies for arsenic removal are essentially smaller versions of reverse osmosis and activated alumina.

- Using the removal efficiency of the treatment train chosen, the percentage of flow that must be treated in order for the entry point to meet the treatment target, is calculated.

Once a treatment train is selected from the decision tree, the associated removal efficiency of the technology is used with information on system flow to determine the amount of flow at the site that must be treated in order to meet the treatment target. System flow is calculated as a power law function of the population served. EPA derived these functions, the derivation of which can be found in the *Geometries and Characteristics of Public Water Systems* report (U.S. EPA, May 1999). Both the equations, and the regression parameters employed in the SafeWaterXL cost model are presented in the following two equations and Exhibit C-5, respectively.

$$\text{Average Flow} = a_A \cdot 4(\text{Population})^{b_A} \quad (\text{Eq. 4})$$

$$\text{Design Flow} = \max \left\{ \begin{array}{l} 2 \cdot \text{Average Flow} \\ a_D \cdot (\text{Population})^{b_D} \end{array} \right. \quad (\text{Eq. 5})$$

where:  $a_A, b_A, a_D, b_D$  = regression parameters derived for flow vs. population  
 Population = population served by the system type and source

**Exhibit C-5.  
Flow Regression Parameters by System Source and Ownership Type**

System Source and Ownership Type	Average Flow		Design Flow	
	a <sub>A</sub>	b <sub>A</sub>	a <sub>D</sub>	b <sub>D</sub>
<i>Ground Water</i>				
Public	0.08558	1.05840	0.54992	0.95538
Private	0.06670	1.06280	0.41682	0.96078
Public-Purchased	0.04692	1.10190	0.31910	0.99460
Private-Purchased	0.05004	1.08340	0.32150	0.97940
<i>Surface Water</i>				
Public	0.14004	0.99703	0.59028	0.94573
Private	0.09036	1.03340	0.35674	0.96188
Public-Purchased	0.04692	1.11020	0.20920	1.04520
Private-Purchased	0.05004	1.08340	0.20580	1.00840

Source: U.S. EPA. 1999. *Geometries and Characteristics of Public Water Systems*. Prepared for Office of Ground Water and Drinking Water by Science Applications International Corporation. EPA Contract No. 68-C6-0059.

Based on these data, the system flow is determined in thousands of gallons per day (KGPD). The system flow is then divided equally among the possible sites of contamination, regardless of whether they are treating (i.e., violation of the revised MCL standard) or not. For example, a system with four potential sites of contamination is modeled to have four sites, each with 25 percent of the total system flow. However, even with this distribution of system flow between the number of sites, the resulting flow assumed at each site is further adjusted for treating sites, such that only the portion of flow that must be treated to lower the arsenic concentration is accounted for in the subsequent cost estimate.

SafeWaterXL employs a “blending” principle to determine the amount of flow that requires treatment in order for the entry point to meet the treatment target established by the MCL. The treatment target is considered 80 percent of the MCL and represents the contaminant level to which the design of systems will perform, to ensure adequate compliance with the MCL. To reach this target, data on the removal efficiencies of the chosen treatment trains, the contaminant occurrence at the site, and the percent of flow apportioned to that entry point are used to determine the fraction of flow needed to be treated, as expressed by the following relationship:

$$\text{Fraction of Flow Treated} = \frac{\left( \frac{\text{TrtTarget}}{\text{SiteConc}} - 1 \right)}{- \% RE} \times (\% \text{SiteFlow}) \quad (\text{Eq. 6})$$

where:            TrtTarget        =        80 percent of revised MCL  
                      SiteConc         =        arsenic concentration at the site  
                      % RE             =        % removal efficiency of treatment train chosen  
                      % Site Flow     =        % of total system flow attributable to that site

Notice that the blending technique is applied at the entry point level, but it is not used for systems selecting POU devices, as those options treat water at the tap rather than for the entire house. Since treatment costs to reduce such high levels of contamination can be significant, blending is an approach SafeWaterXL takes to best characterize the expected cost of compliance. In this manner, treatment costs are tallied only among the sites that are expected to treat, for the portion of the overall system flow that actually gets treated.

- The percentage of flow that needs to be treated is applied to the design flow, which is then used to derive the capital costs of the components of the treatment train (the sum of: treatment capital, waste disposal capital, and any pre-treatment capital costs).

Each treatment train is defined by a treatment technology and (where relevant in order to be effective) a waste disposal option, and/or pre-treatment technology. Therefore, the cost of the treatment trains is related to its constituent capital and O&M cost components. Capital costs are estimated as a function of design flow. When the treatment train has been selected, the overall capital costs of these various components are aggregated to derive an overall capital cost estimate. This is expressed in the following general treatment train cost functions at each site:

$$TrC_{cap} = T_{cap} + WD_{cap} + [(P_{PO})(PO_{cap})] \quad (\text{Eq. 7})$$

where:            TrC<sub>cap</sub>        =        Treatment train capital cost at treating site  
                      T<sub>cap</sub>            =        Treatment technology capital cost at treating site  
                      WD<sub>cap</sub>        =        Waste disposal technology capital cost at treating site  
                      P<sub>PO</sub>            =        Probability of using pre-oxidation at treating site  
                      PO<sub>cap</sub>        =        Pre-oxidation technology capital cost at treating site

Depending on the source water conditions and on the treatment technologies involved, EPA determined that some systems would require additional pre-oxidation. EPA developed a separate decision tree to approximate the number of systems that would implement pre-oxidation technologies when selecting a treatment train. The need for this separate decision tree was based in part on the distribution of systems with and without treatment-in-place. For technology trains in which pre-treatment is required, Exhibit C-6 summarizes the decision tree of probabilities by system size that a system would require these technologies.

Each of the treatment technologies considered in the decision tree remove As(V) more readily than As(III) and as a result, pre-oxidation may be necessary depending upon source water conditions. Systems without treatment in-place may already be chlorinating which may meet pre-oxidation requirements. For those systems, pre-oxidation may or may not need to be installed. Similarly, systems with treatment in-place may have pre-oxidation in-place, which could meet the pre-oxidation requirements.

**Exhibit C-6.**  
**Probability of a System Requiring Pre-Oxidation**

System Size Category	Pre-Oxidation (GW systems)	Pre-Oxidation (SW systems)
25-100	0.54	0.09
101-500	0.30	0.04
501-1,000	0.24	0
1,001-3,300	0.24	0
3,301-10,000	0.27	0.03
10,001-50,001	0.13	0.01
50,001-100,000	0.41	0.02
100,001-1,000,000	0.16	0

Source: Facsimile from Amit Kapadia, EPA OGWDW, July 27, 1999.

Similarly, the percentage of flow that needs to be treated is also applied to the average flow, which is then used to derive the operation and maintenance costs of the components of the treatment train (the sum of: treatment O&M, waste disposal O&M, and any pre-treatment O&M costs).

Unlike capital costs, which are expressed as a total cost, operation and maintenance costs are expressed as a cost per year, and are calculated as a function of average flow. The total O/M costs for each treating site are aggregated to derive an annual system O/M cost for the treatment technology. Treatment O&M cost, waste disposal O&M, and any pre-treatment O&M costs are tallied. These conditions are expressed in the following general treatment train cost functions at each site:

$$TrC_{O\&M} = T_{O\&M} + WD_{O\&M} + [(P_{PO})(PO_{O\&M})] \quad (\text{Eq. 8})$$

where:

- $TrC_{O\&M}$  = Treatment train O&M cost at treating site
- $T_{O\&M}$  = Treatment technology O&M cost at treating site
- $WD_{O\&M}$  = Waste disposal technology O&M cost at treating site
- $PO_{O\&M}$  = Pre-oxidation technology O&M cost at treating site

Since the treatment technologies produce residuals that may contain various levels of arsenic, the O&M costs associated with the treatment train are an important consideration in the overall cost of the technology chosen. The handling and disposal costs associated with these residuals can be significant, and depend on a number of factors, such as the size and flow of the water system. The amount of waste that is generated will affect which technology is implemented by a water system. For example, some methods may be impractical for larger systems due to land requirements. Alternatively, more expensive processes may be inappropriate for smaller systems due to the cost. Process oversight, transportation, and labor are all factors affecting the overall cost of the process. In general, the more complex the handling and the disposal methods, the more significant the maintenance requirements, and therefore the more costly.

- The system’s total annual treatment costs are calculated for the selected treatment train at various discount rates, by summing the treatment costs (annualized capital plus annual O&M cost components) across all treating sites.

Since operation and maintenance costs are annual, applying the amortization formula on the capital cost component (Step 8) over a specified period of repayment, results in an overall annual cost of treatment at a site:

$$TrC_{tot} = (TrC_{cap}) \left( \frac{r}{1 - (r + 1)^{-rp}} \right) + TrC_{O\&M} \quad (\text{Eq. 9})$$

where:

$TrC_{tot}$	=	Annual total treatment train cost at treating site
$TrC_{cap}$	=	Treatment train capital cost at treating site
$r$	=	Discount rate
$rp$	=	Repayment period
$TrC_{O\&M}$	=	Treatment train O&M cost at treating site

For the purposes of estimating the national cost of compliance, public water system and implementation costs are tracked over a 20-year period. This time frame is used because many public water systems often finance their capital improvements over 20 years. This may, however, result in an overestimate of annualized costs because many types of equipment last longer than 20 years. Capital and operational and maintenance (O&M) costs may be incurred at different points throughout the time period. For this reason, two adjustments were made to the estimated costs forecasted by SafeWaterXL in order to render future costs comparable with current costs, reflecting the fact that a cost outlay today is a greater burden than an equivalent cost outlay sometime in the future.

In the first instance, compliance costs that are subsequently used in cost-benefit analyses are annualized using a social discount rate so that regulatory option costs (e.g. costs for an MCL of 5 µg/L vs. an MCL of 10 µg/L) may be directly compared to the annual benefits of the corresponding regulatory option. Annualization is similar to the process involved in calculating a mortgage payment; the result is a constant annual cost as expressed in Equation 9. The Agency performs cost-benefit analyses using two social discount rates. As required by the Office of Management and Budget (OMB), a seven percent discount rate is used in estimating the national cost of compliance in a rulemaking. A three percent discount rate is also used to estimate the costs of compliance, as the Agency believes this rate more closely approximates the true social discount rate.

In the second instance, compliance costs that are subsequently used in various economic impact analyses as required by the SDWA and its Amendments, such as in affordability analyses, are annualized using an actual cost-of-capital discount rate rather than a social discount rate. Affordability analyses examine the costs of compliance to systems and individual households, rather than on a national level. Costs to households are considered a good proxy for determining the affordability of regulatory compliance, as described in the discussion on maximum allowable household cost in Step 11 below.

They are dependent on system costs to the extent that system costs are recovered through increased water rates. The cost-of-capital rate is used to reflect the true after-tax cost-of-capital that water systems face, net of any government grants or subsidies. The recommended cost-of-capital rates stratified by ownership, system type and size, as reported in *Development of Cost of Capital Estimates for Public Water Systems* (U.S. EPA, 1998), were used in SafeWaterXL. These were presented in Exhibit 6-7.

Together, the annualized capital and O&M cost components equal the annual cost of treatment. When these costs are summed across all the treating sites in a system, the annual system cost is calculated. In other words, the system's cost of compliance is determined by summing across the treating sites. For each system in which a violation of the revised MCL is expected, this overall cost is calculated:

$$SC_{ir} = \sum_{n=1}^n (TrC_{tot,n}) \quad (\text{Eq. 10})$$

where:

i	=	System/model iteration
n	=	Number of treating sites in the system
SC <sub>ir</sub>	=	Annual cost for system i at discount rate r
TrC <sub>tot</sub>	=	Annual total treatment train cost at treating site

- The annual system cost is used to derive the cost per thousand gallons (cost/kgal) delivered by the water system.

Once the annual cost per system is determined by summing the costs of all the treating sites of the system, this cost is used to determine the unit cost of delivery (cost per thousand gallons delivered) for the system as a result of the new treatment technology. The system cost annualized at the cost-of-capital discount rate is used in this calculation as it best represents the true cost impact on the system. The cost per thousand gallons delivered is calculated as:

$$Cost_{kgal} = SC_{i,coc} \div \left( AF_i \cdot \frac{365 \text{ days}}{1 \text{ yr}} \cdot \frac{1000 \text{ kgal}}{1 \text{ Mgal}} \right) \quad (\text{Eq. 11})$$

where:

Cost <sub>kgal</sub>	=	Cost per thousand gallons for the system
AF <sub>i</sub>	=	Average flow (MGD) of system i
SC <sub>i, coc</sub>	=	Annual cost for system i at the cost-of-capital discount rate

- Annual household costs are then calculated based on the system's unit cost of delivery (cost per thousand gallons) and the average annual household consumption per year.

The system's cost per thousand gallons delivered is used to calculate household costs according to Equation 12. The values used as estimates of the average annual tap water consumption per year are presented in Exhibit 7. More detail was given in Chapter 4.

$$Cost_{HHi} = Cost_{kgal} \cdot C_{HH} \quad (\text{Eq. 12})$$

where:  $Cost_{HHi}$  = Household cost per year for system i  
 $C_{HH}$  = Household consumption per year (kgal)

**Exhibit 7.  
Water Consumption per Residential Connection**

System Size Category	System Ownership Type	
	Public	Private
<100	81	92
101-500	93	110
501-1,000	97	88
1,001-3,300	82	102
3,301-10,000	87	124
10,001-50,000	108	110
50,001-100,000	122	96
100,001-1,000,000	127	114

Source: EPA. 1997. *CWSS, Vol. II: Detailed Summary Result Tables and Methodology Report*, Table 1-14.

- If household costs are determined to exceed an affordability threshold of \$500, a less expensive treatment technology (POU device) is chosen and new costs are calculated (Steps 7-12 above are repeated using data for POU devices).

SafeWaterXL employs a maximum allowable household cost of \$500, which forces systems who initially choose a treatment train with annual household costs in excess of \$500, to default to a POE device, thereby seeking a less expensive method of compliance. In general, the results of the model simulation showed that only the smallest systems (serving 25-500 people) are affected by this threshold. Based on the overall number of systems in these two size categories (see Exhibits 1 and 2), the number of systems affected is relatively small. SafeWaterXL does record the number of systems exceeding this affordability threshold.

- The system results are maintained in a database for further analysis.

### **C.2.1 Example Calculation (Single Iteration)**

In this section, we demonstrate the process by which SafeWaterXL calculates the annual cost of compliance for a single system assuming a target MCL of 5 µg/L. Each step in the procedure described in the previous section is addressed to exemplify how the many assumptions and data inputs are pooled together in a single iteration.

Given the following SafeWaterXL model setting selections by the user:

- Source water = ground water;
- Ownership type = public;
- MCL = 10 µg/L;

Then, a single iteration of the model proceeds as follows:

1. A system is selected from data files.

A publicly-owned community ground water system with three entry points serving 10,000 people is selected from the data files.

2. Each system is assigned a random concentration from an occurrence distribution.

Based on accompanying information in the data file for ground water systems, an average system concentration of 11.03 µg/L is selected from an occurrence distribution bound by a lognormal mean of -0.2507 and a log standard deviation of 1.5826.

3. The selected arsenic concentration for the system is distributed across the number of sites (entry points) of possible contamination for that system based on the relative intra-system standard deviation (RSD).

Since the system has three entry points, based on the average system concentration of 11.03 µg/L, the maximum site concentration is determined to be 33.10 µg/L (= 11.03 \* 3). Using the default RSD of 0.64 and this limitation on the maximum site concentration, the three sites are assigned concentrations of 8.89, 9.69, and 14.52 µg/L, respectively. These three concentrations keep the average system concentration at 11.03 µg/L.

4. The concentration at each site is compared to the revised MCL standard to determine if the site is in violation of the revised standard.

The first two sites are determined to have concentrations of 8.89µg/L and 9.69µg/L, both of which are below the user selected MCL of 10 µg/L. The final site of the system, however, exceeds the MCL with a concentration of 14.52 µg/L, and is the only site for which the remainder of the calculations are conducted.

5. If the site is in violation of the revised MCL, the percentage of removal required in order to reach the treatment target is calculated.

From Equation 3 above, the percent of removal required for a site with an influent concentration of 14.52 µg/L to reach the treatment target of 8 µg/L (80 percent of MCL 10 µg/L) equals 45 percent:

$$\% \text{ removal} = \frac{(\text{SiteConc} - \text{TrtTarget})}{\text{SiteConc.}} = \frac{(14.52 - 8)}{14.52} = 0.4490$$

6. Based on the percentage of removal required to meet the treatment target and on the decision tree for the size and type of the system, a treatment train is then assigned to the site.

Using the decision tree for ground water systems for the size category serving 3,301-10,000 people, a treatment train is selected based on the probabilities from the “<50%” removal column since this site requires 45 percent removal.

For this iteration, Treatment Train #6 (Coagulation/Microfiltration, Nonmechanical Dewatering, Non-Hazardous Landfill) is selected. This treatment train has a removal efficiency of 90 percent.

7. Using the removal efficiency of the treatment train chosen, the percentage of flow that must be treated in order for the entry point to meet the treatment target is calculated.

The system flow must now be determined. Since the system in this iteration of the model is a public groundwater system, using the flow equations (Equations 4 and 5) and the regression parameters from Exhibit C-5, the design flow equals 3.646 MGD:

$$\text{Design Flow} = a_D \cdot (\text{population})^{b_D} = (0.54992) \cdot (10,000)^{0.95538} = 3646.02393 \times \frac{1 \text{Mgal}}{1000 \text{kgal}} = 3.646$$

and the average flow equals 1.465 MGD:

$$\text{Average Flow} = a_A \cdot (\text{population})^{b_A} = (0.08558) \cdot (10,000)^{1.05840} = 1465.454309 \text{kgal} \times \frac{1 \text{Mgal}}{1000 \text{kgal}} = 1.465$$

As described in Step 7 above, the system’s total flow is evenly distributed among all the possible sites. In this case, since there are three sites, each receives 33.3 percent of the total system flow. Using the principle of blending, the fraction of the system’s total flow that must be treated in order for the site to meet the treatment target equals 16.6 percent:

$$\text{Fraction of Flow} = \frac{\left( \frac{\text{TrtTarget}}{\text{SiteConc}} - 1 \right)}{-\% \text{ RE}} \times (\% \text{SiteFlow}) = \frac{\left( \frac{8}{14.52} - 1 \right)}{-0.90} \times (0.333) = 0.1663$$

8. The percentage of flow that needs to be treated is applied to the design flow, which is then used to derive the capital costs of the components of the treatment train (the sum of: treatment capital, waste disposal capital, and any pre-treatment capital costs).

Applying the fraction of the system's total flow (16.6 percent) to the system's total design flow (3.646 MGD) from Step 7 above, the design flow at the treating site can be calculated:

$$(3.646 \text{ MGD}) \times (0.166) = 0.606 \text{ MGD}$$

This adjusted flow is then used to determine the capital costs of the treatment train using the various cost equations for the treatment capital and waste disposal capital. For this treatment train, the treatment capital cost is \$1,495,716 and the waste disposal capital cost is \$1,169,055, for a total capital cost of \$2,664,771.<sup>4</sup> For design flow (x), the cost (y) can be calculated:

<u>Treatment capital:</u>	x < 0.1	y = -11935465x <sup>2</sup> + 48800366x + 94324
	0.1 < x < 0.27	y = 2343199x + 228653
	0.27 < x < 1	y = -483591x <sup>2</sup> + 2308991x + 273143
	< 1x < 10	y = 1030810x + 1067733
	x > 10	y = 320x <sup>2</sup> + 921471x + 2129119

Based on a site design flow of 0.606 mgd, the third segment of the cost equation is used:

$$y = -483591(0.606)^2 + 2308991(0.606) - 273143$$

Similarly, for design flow (x) = the waste disposal capital cost (y) can be calculated from these equations:

<u>Waste Disposal capital:</u>	x < 0.085	y = 3069360x - 790
	0.085 < x < 1.8	y = 1749352x - 108017
	x > 1.8	y = 1627970x + 326504

For the waste disposal capital cost, the second cost segment is used:

$$y = 1749352(0.606) - 108017$$

In this example, based on the probability distribution listed in Exhibit 6, pre-oxidation was not selected, therefore the pre-oxidation capital costs are not calculated and included in the capital cost component of the treatment train.

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<sup>4</sup>Costs presented in this example are in April 1998\$, although post-processing of SafeWaterXL results updated these costs to May 1999\$ in the Regulatory Impact Analysis. Totals may not equal sample calculation provided due to rounding of input variables.

9. Similarly, the percentage of flow that needs to be treated is also applied to the average flow, which is then used to derive the operation and maintenance costs of the components of the treatment train (the sum of: treatment O&M, waste disposal O&M, and any pre-treatment O&M costs).

Similarly, by applying the fraction of the system's total flow (16.6 percent) to the system's total average flow (1.465 MGD) from Step 7 above, the average flow at the treating site can be calculated as follows:

$$(1.465 \text{ MGD}) \times (0.1663) = 0.244 \text{ MGD}$$

This flow is then used to determine the operation and maintenance costs of this treatment train using the various cost equations for treatment O&M and waste disposal O&M. The treatment O&M cost is \$46,500 and the waste disposal O&M cost is \$20,309, for a total annual O&M cost of \$66,809. For average flow (x), the O&M cost (y) is:

<u>Treatment O&amp;M:</u>	$x < 0.03$	$y = 196829x + 20264$
	$0.03 < x < 0.09$	$y = 136332x + 22139$
	$0.09 < x < 0.36$	$y = 80081x + 26977$
	$0.36 < x < 4.3$	$y = 13311x + 51014$
	$x > 4.3$	$y = 15236x + 42350$

Based on a site average flow of 0.244 MGD, the third segment of the cost equation is used:

$$y = 80081(0.244) + 26977$$

Similarly, for average flow (x), the waste disposal cost (y) is:

<u>Waste Disposal O&amp;M:</u>	$x < 0.085$	$y = -18812x^2 + 4686.1x + 2123.8$
	$0.085 < x < 0.72$	$y = 111819x - 6950.5$
	$x > 0.72$	$y = 16.966x^2 + 60792x + 28760$

For the waste disposal O&M cost, the second cost segment is used:

$$y = 111819(0.244) - 6950.5$$

Again, since pre-oxidation was not selected, no pre-oxidation O&M costs are calculated or included in the O&M cost component of the treatment train.

10. The system's total annual treatment costs are calculated for the selected treatment train at various discount rates, by summing the treatment costs (annualized capital plus annual O&M cost components) across all treating sites.

From Step 8, the total capital costs for this treatment train equal \$2,664,771. From Step 9, the total O&M costs for the treatment train equal \$66,809. Using a capital cost amortized at 5.26 percent<sup>5</sup> over 20 years, the annual cost to the system equals \$284,278:

$$= (TrC_{cap}) \left( \frac{r}{1 - (r+1)^{-tp}} \right) + TrC_{O\&M} = (\$2,664,771) \left( \frac{0.0526}{1 - (0.0526+1)^{-20}} \right) + \$66,809 = \$284,278$$

The example displayed here uses the commercial rate, which is a closer approximation to the cost of capital to water systems. Annual costs are also calculated at 3 and 7 percent, respectively, as \$245,923 and \$318,344.

11. This annual system cost is used to derive the cost per thousand gallons (cost/kgal) delivered by the water system.

The unit cost of water delivered by this system (cost per kgal per year) as a result of installing treatment is determined by dividing the system cost by the system average flow. The system cost that was derived using the commercial discount rate is used to arrive at a unit cost of \$0.53:

$$= SC_{ir} \div \left( AF_i \cdot \frac{365 \text{ days}}{1 \text{ yr}} \cdot \frac{1000 \text{ kgal}}{1 \text{ Mgal}} \right) = (\$284,278) \div \left( (1.465) \cdot \frac{365}{1} \cdot \frac{1000}{1} \right) = \$0.53$$

12. Annual household costs are then calculated based on the system's unit cost of delivery (cost per thousand gallons) and the average annual household consumption per year.

The cost per thousand gallons to the water system calculated in Step 11 is used to estimate the annual cost to households as a result of regulatory compliance, by multiplying it with the average annual household consumption of tap water for a system in that size category:

$$= Cost_{kgal,i} \cdot C_{HH} = (\$0.53) \cdot (108 \text{ kgal}) = \$46.24$$

The annual water consumption per household is presented in Chapter 4 and stratified by size category and ownership type.

13. If household costs are determined to exceed an affordability threshold of \$500, a less expensive treatment technology (POU device) is chosen and new costs are calculated (Steps 7-12 above are repeated using data for POE devices).

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<sup>5</sup>Commercial discount rates are presented in Exhibit 6-7 of Chapter 6 of the Regulatory Impact Analysis, and determined by size category and ownership type.

Since the estimated annual household cost for this system is \$46.24, this step does not affect the calculations already discussed. As described earlier, this affordability threshold affects only the smaller system size categories (<100 and 101-500). Therefore, the results of this iteration are recorded and the next iteration is triggered in Step 14.

14. The results are maintained in a database.

## **C.3 Model Run**

### **C.3.1 Number of Iterations**

Once a single iteration is completed, the calculated system data is recorded. Among the cost data forecasted for each iteration are the following:

- annual system cost (calculated at three discount rates: three percent, seven percent, cost-of capital);
- system capital cost (calculated at one discount rate: cost-of capital);
- system O&M cost (calculated at three discount rate: cost-of capital);
- cost per thousand gallons (calculated at one discount rate: cost-of capital); and
- household cost (calculated at one discount rate: cost-of capital).

Once complete, another iteration is started. This is repeated N times, until the total number of iterations (the total number of systems) for that size category is met, at which point the total annual national cost estimate for that size category is determined.

Next, once each size category is finished, the first iteration of the next size category begins. The cycles continue until all iterations of all eight size categories have been completed. The total annual national cost across all systems is therefore the sum of the annual national costs for each size category of systems, both publicly- and privately-owned.

If graphed against the estimated mean, the average system cost would generally fluctuate greatly between iterations at the beginning of a model run. However, as the number of data points increases, these fluctuations will dampen and should eventually converge on the estimated mean. The number of iterations must be a multiple of the number of systems that belong to each size category. This setting will avoid any systematic bias as the model cycles through all the systems within each size category from smallest to largest.

Each cycle therefore represents the universe of systems in that category as pulled from SDWIS (as summarized in Exhibits C-1 and C-2). Using this method, approximately the same number of non-zero data points should be generated when the same iteration settings are selected.

The anticipated number of non-zero data points is a function of the MCL, the occurrence distribution, and the number of systems in the size category, where a non-zero data point is a system that is required to treat and incurs treatment costs. For example, approximately eight cycles of the universe of ground water systems serving less than 100 people (14,432 systems, as

shown in Exhibit 1) are required to achieve 20,000 data points given an MCL of 3 µg/L, and an occurrence distribution where 19.7 percent of the systems are expected to exceed the MCL. For the purposes of regulatory analysis of arsenic in drinking water, a goal of 20,000 data points was used in SafeWaterXL.

### C.3.2 Model Outputs

The primary outputs of the SafeWaterXL model are national-level estimates of costs of compliance, as well as distributions of cost to systems or households, across various water system service size categories. To achieve these results, the output generated for each iteration, as stratified by water source, ownership, and service size category, are combined by SafeWaterXL at the conclusion of the model run.

#### Average Annual System Cost (Calculated at the Cost-of Capital Discount Rate)

Each iteration of the model describes the treatment and cost profile for a single system in a single size category. System cost is essentially equal to treatment cost, which is based on the treatment train technology chosen and the capital and operating and maintenance (O&M) costs of that selected treatment train. These costs are in turn a function of the amount of flow processed by the water system: capital costs are estimated as a function of design flow, while O&M costs are based on average flow. In addition to these treatment cost components, associated waste disposal capital and O&M costs are also included. A portion of these systems are then estimated to require pre-oxidation, which would add incremental costs to the total treatment cost.

In the case of calculating an average system cost, a commercial discount rate that is closer to the actual cost of capital that systems might face is used:

$$Avg.SC_{jr} = \frac{\sum_{i=1}^{m_j} (SC_{ir})}{m_j} \quad (Eq. 13)$$

where:

$SC_{jr}$	=	Annual system cost for size category j at discount rate r
$SC_{ir}$	=	Annual cost for system i at discount rate r
j	=	Size category
$m_j$	=	Number of systems in size category j

Although the equation above is used to calculate the average system cost for a particular size category, the result represents one ownership and source type (e.g. average system cost for public ground water systems serving <100). In order to combine the results for the two ownership types for a single run, each system cost must be weighted by its respective number of treating systems over the universe of systems in that size category:

$$Avg.SC_{j(total)} = \frac{(Avg.SC_{j(pub)}) (n_{j(pub)}) + (Avg.SC_{j(priv)}) (n_{j(priv)})}{(n_{j(pub)} + n_{j(priv)})} \quad (Eq. 14)$$

where:

- $SC_{j(\text{tot})}$  = Total annual system cost for size category j
- $SC_{i(\text{pub})}$  = Annual system cost for publicly-owned systems of size category j
- $SC_{i(\text{prv})}$  = Annual system cost privately-owned systems of size category j
- $n_{j(\text{pub})}$  = Number of publicly-owned treating systems in size category j
- $n_{j(\text{prv})}$  = Number of privately-owned treating systems in size category j

Average Annual Household Cost (Calculated at the Cost-of Capital Discount Rate)

Since household costs are also calculated for each system, a similar distribution of the cost of compliance at the system level are also calculated at the household level.:

$$Avg.Cost_{HHj} = \frac{\sum_{i=1}^{m_j} (Cost_{HHi})}{m_j} \quad (\text{Eq. 15})$$

where:

- $Cost_{HHj}$  = Annual household cost for size category j
- $Cost_{HHi}$  = Household cost for system i
- $m_j$  = Number of systems in size category j

Similarly, just as the average system cost was weighted across ownership types (Equation 14) the average household cost for a single size category must be a weighted average taking into consideration the number of households affected for each ownership type within the size category.

Annual National Cost (Calculated at Two Discount Rates , 3 percent and 7 percent)

Annual cost for a system size category is determined by adding the total cost of compliance across each treating system within that size category (e.g. the sum of all the system costs for each iteration in that size category). This is a function of the individual system cost not the average system cost, calculated at three and seven percent discount rates:

$$AC_{jr} = \sum_{i=1}^{m_j} (SC_{ir}) \quad (\text{Eq. 16})$$

where:

- $AC_{jr}$  = Annual cost for size category j at discount rate r
- $SC_{ir}$  = Annual cost for system i at discount rate r

Similarly, the annual national cost is total determined by adding the annual cost of compliance across all the size categories (e.g. the sum of all the system costs for all the iterations in the run):

$$ANC_r = \sum_{j=1}^8 (AC_{jr}) \quad (\text{Eq. 17})$$

where:  $ANC_r$  = Annual national cost at discount rate r

## Appendix D. *What-If* Cost Sensitivity Analysis

Chapter 6 of this report discusses the uncertainty associated with the National cost estimate. Lacking information on exactly which systems will need to undertake activities to achieve compliance, or what portion of those systems would require treatment, there will always be some uncertainty associated with the actual costs likely to be incurred. The Agency conducted a Monte Carlo simulation to provide a best estimate of probable costs and a sense of the relative precision of the estimate. None of that analysis addresses potential bias in Agency estimates.

A number of commenters asserted that there were factors in the Agency analysis that could significantly bias its estimate. The Agency disagrees with the issues raised for the reasons detailed in the response to comment document. This Appendix will not attempt to address all of those concerns. Rather, it describes a SafewaterXL simulation conducted to assess the sensitivity of the National cost estimate to changes in factors involving professional judgment and where there is uncertainty with respect to the status quo of the water supply industry. The factors considered relate to unit treatment costs and the compliance forecast (decision tree). Modeling was not conducted relating to water system entry point configurations since the Agency and commenters are in agreement that the entry point is the appropriate point for consideration of compliance costs and commenters have demonstrated that changes in such assumptions have minimal impact on national estimates. Likewise, factors, which could bias the Agency's cost estimates downward, are not evaluated.<sup>1</sup> These factors are not evaluated to give the clearest picture of the absolute magnitude of the potential for underestimation. The data discussed in this section are from a single Monte Carlo run of the Safewater XL model.

**Unit treatment costs-** The response to comment document contains a thorough critique of commenter unit cost estimates. There are four areas, however, where anecdotal evidence suggests costs beyond those evaluated by the Agency could be experienced by individual water systems in their compliance efforts. In an effort to provide some context on the significance of these concerns, modifications were made to the Agency's best estimate equations to incorporate these factors. The following changes were incorporated into this analysis:

**Accessory costs-** Some commenters asserted that the costs for installing clearwells or storage to achieve flow equalization after treatment, repiping around new treatment devices, and additional pumping needed after pressure breaks for treatment would be incurred by water systems, aside from the piping and pumping costs considered by the Agency. These commenters estimated that such costs could add up to 76 percent to the capital costs of compliance.

Technologies costed by the Agency do include ancillary piping costs. Further, technologies, which break pressure, like coagulation, included re-pumping costs. What neither the commenters, nor the Agency have information on, however, is the extent to which additional storage might be

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<sup>1</sup>A recently completed Agency report (Abt, 2000) suggests that many water systems achieve compliance with some rules without major treatment reconstruction. In some cases, as many as a third of all systems were able to achieve compliance without major reconstruction. Less capital intensive options than were costed in the Agency's decision trees could include drilling a new well, reconfiguring intakes to blend to the MCL level, or closing one, or more, wells and purchasing from a larger system can appreciably reduce costs.

required post treatment by water systems undertaking construction as part of their compliance effort. This impact was evaluated by increasing treatment capital costs by 76 percent for those systems that did not presently have disinfection (per EPA, 1999b). The Agency considered it highly improbable that a system which presently conducted disinfection would not have adequate storage or mixing zone capacity

**Land costs-** Some water systems undoubtedly will need to relocate entry points or acquire land for the building of new treatment facilities. Commenters agree with the Agency that there is no source of information for preparing a sound estimate of this impact. The issue is most likely to arise with currently untreated entry points. One commenter estimated that land acquisition could add five percent to compliance capital costs for ground water systems. While the Agency believes land acquisition will not be a common occurrence, the what-if analysis included a five percent increase in capital costs for land acquisition by ground water systems.

**Permitting and pilot testing-** The Agency has taken various approaches to the consideration of permitting and pilot testing requirements in past cost analyses. While such costs are not expected to be appreciable for most water systems, it is plausible that they could cause engineering costs to exceed the fifty percent of direct costs currently costed. For the purposes of the what-if analysis, the Agency is including three percent increases to direct capital costs for each factor per the recommendations of the Technology Design Panel (EPA, 1997).

**Compliance forecast/decision tree-** In developing its compliance decision trees, the Agency considers water quality factors, water availability, and cost. It is presumed that a water system will adopt the lowest cost technology it can feasibly use. Admittedly, systems sometimes select more expensive technologies, but do so to accomplish multiple treatment objectives. Lacking comprehensive information on co-occurrence, the Agency is unable to consider the benefits or costs of such actions. Regardless, they are not costs attributable to arsenic compliance.

The Agency made numerous modifications to the proposal decision tree in response to public comment. The use of ion exchange, for instance, was greatly reduced in response to residuals management concerns. To assess the impact of the decision tree upon National cost estimates, the what-if analysis eliminated ion exchange (a relatively inexpensive technology) and greatly increased the projected use of coagulation and microfiltration (the most expensive option for many strata). Tables D-1 through D-8 present the decision trees used in the analysis and can be compared to the primary analysis decision trees in Appendix A.

**Results-** Table D-9 depicts the results of the model run in comparison to those generated by the best estimate. It is interesting to note that, at the MCL option of 10, the 95 percent confidence interval on the best estimate is \$215 million dollars. The What-If estimate is less than ten percent greater than the Agency's original estimate. At the MCL option of five, however, the what-if assumptions generate a twenty-five percent increase in the National cost estimate. These results are consistent with those observed in the AWWARF Cost Implications Report (AWWARF, 2000) wherein lower options were much more volatile in the face of varying assumptions. While the Agency remains unpersuaded by many of the commenters arguments, this analysis does support their concern relating to uncertainty at options beneath the selected MCL.

**Exhibit D-1**  
**Probability Decision Tree: “What-If” Sensitivity Analysis**  
**Ground Water Systems Serving 100 People**

No.	Treatment Technology Train	Percent of Treatment Required to Achieve MCL		
		<50%	50-90%	>90%
1	Modify Lime Softening and pre-oxidation	1.0	1.0	1.0
2	Modify Coagulation/Filtration and pre-oxidation	1.0	1.0	1.0
3	Anion Exchange (<20 mg/L SO4) and POTW waste disposal and pre-oxidation	0.0	0.0	0.0
4	Anion Exchange (20-50 mg/L SO4) and POTW waste disposal and pre-oxidation	0.0	0.0	0.0
5	Coagulation Assisted Microfiltration and mechanical dewatering/non-hazardous landfill waste disposal and pre-oxidation	0.0	0.0	0.0
6	Coagulation Assisted Microfiltration and non-mechanical dewatering/non-hazardous landfill waste disposal and pre-oxidation	0.0	0.0	0.0
7	Oxidation Filtration (Greensand) and POTW for backwash stream and pre-oxidation	12.9	12.4	0.0
8	Activated Alumina (pH 7 -pH 8) and non-hazardous landfill (for spent media) and pre-oxidation	60.3	57.3	72.2
9	Activated Alumina (pH 8 -pH 8.3) and non-hazardous landfill (for spent media) and pre-oxidation	20.5	19.1	23.7
10	Activated Alumina (23,100 BV) with pH adjustment (pH 6)/corrosion control and non-hazardous landfill (for spent media) and pre-oxidation	0.0	0.0	0.0
11	Activated Alumina (15,400 BV) with pH adjustment (pH 6)/corrosion control and non-hazardous landfill (for spent media) and pre-oxidation	0.0	0.0	0.0
12	POU Activated Alumina and pre-oxidation	2.2	4.6	0.0
13	POU Reverse Osmosis and pre-oxidation	2.2	4.6	2.1
	<b>Sum of Probabilities:</b>	<b>100.00</b>	<b>100.00</b>	<b>100.00</b>

**Exhibit D-2**  
**Probability Decision Tree: “What-If” Sensitivity Analysis**  
**Ground Water Systems Serving 101-500 People**

No.	Treatment Technology Train	Percent of Treatment Required to Achieve MCL		
		<50%	50-90%	>90%
1	Modify Lime Softening and pre-oxidation	2.0	2.0	2.0
2	Modify Coagulation/Filtration and pre-oxidation	2.0	2.0	2.0
3	Anion Exchange (<20 mg/L SO4) and POTW w aste disposal and pre-oxidation	0.0	0.0	0.0
4	Anion Exchange (20-50 mg/L SO4) and POTW w aste disposal and pre-oxidation	0.0	0.0	0.0
5	Coagulation Assisted Microfiltration and mechanical dewatering/non-hazardous landfill w aste disposal and pre-oxidation	0.0	0.0	0.0
6	Coagulation Assisted Microfiltration and non-mechanical dewatering/non-hazardous landfill w aste disposal and pre-oxidation	0.0	0.0	0.0
7	Oxidation Filtration (Greensand) and POTW for backwash stream and pre-oxidation	12.9	12.5	0.0
8	Activated Alumina (pH 7 - pH 8) and non-hazardous landfill (for spent media) and pre-oxidation	60.4	57.2	66.1
9	Activated Alumina (pH 8 - pH 8.3) and non-hazardous landfill (for spent media) and pre-oxidation	20.5	19.1	22.7
10	Activated Alumina (23,100 BV) w ith pH adjustment (pH 6)/corrosion control and non-hazardous landfill (for spent media) and pre-oxidation	0.0	1.8	3.1
11	Activated Alumina (15,400 BV) w ith pH adjustment (pH 6)/corrosion control and non-hazardous landfill (for spent media) and pre-oxidation	0.0	0.0	3.1
12	POU Activated Alumina and pre-oxidation	1.1	2.7	0.0
13	POU Reverse Osmosis and pre-oxidation	1.1	2.7	1.0
<b>Sum of Probabilities:</b>		<b>100.00</b>	<b>100.00</b>	<b>100.00</b>

**Exhibit D-3**  
**Probability Decision Tree: “What-If” Sensitivity Analysis**  
**Ground Water Systems Serving 501-1,000 People**

No.	Treatment Technology Train	Percent of Treatment Required to Achieve MCL		
		<50%	50-90%	>90%
1	Modify Lime Softening and pre-oxidation	2.0	2.0	2.0
2	Modify Coagulation/Filtration and pre-oxidation	2.0	2.0	2.0
3	Anion Exchange (<20 mg/L SO <sub>4</sub> ) and POTW w aste disposal and pre-oxidation	0.0	0.0	0.0
4	Anion Exchange (20-50 mg/L SO <sub>4</sub> ) and POTW w aste disposal and pre-oxidation	0.0	0.0	0.0
5	Coagulation Assisted Microfiltration and mechanical dewatering/non-hazardous landfill w aste disposal and pre-oxidation	0.0	0.0	0.0
6	Coagulation Assisted Microfiltration and non-mechanical dewatering/non-hazardous landfill w aste disposal and pre-oxidation	0.0	0.0	0.0
7	Oxidation Filtration (Greensand) and POTW for backwash stream and pre-oxidation	12.9	12.5	0.0
8	Activated Alumina (pH 7 - pH 8) and non-hazardous landfill (for spent media) and pre-oxidation	27.0	27.2	32.0
9	Activated Alumina (pH 8 - pH 8.3) and non-hazardous landfill (for spent media) and pre-oxidation	2.2	1.8	2.1
10	Activated Alumina (23,100 BV) w ith pH adjustment (pH 6)/corrosion control and non-hazardous landfill (for spent media) and pre-oxidation	27.0	27.2	31.0
11	Activated Alumina (15,400 BV) w ith pH adjustment (pH 6)/corrosion control and non-hazardous landfill (for spent media) and pre-oxidation	27.0	27.2	31.0
12	POU Activated Alumina and pre-oxidation	0.0	0.0	0.0
13	POU Reverse Osmosis and pre-oxidation	0.0	0.0	0.0
<b>Sum of Probabilities:</b>		<b>100.00</b>	<b>100.00</b>	<b>100.00</b>

**Exhibit D-4**  
**Probability Decision Tree: “What-If” Sensitivity Analysis**  
**Ground Water Systems Serving 1,001-3,300 People**

No.	Treatment Technology Train	Percent of Treatment Required to Achieve MCL		
		<50%	50-90%	>90%
1	Mbdify Lime Softening and pre-oxidation	2.0	2.0	2.0
2	Mbdify Coagulation/Filtration and pre-oxidation	2.0	2.0	2.0
3	Anion Exchange (<20 mg/L SO4) and POTW waste disposal and pre-oxidation	0.0	0.0	0.0
4	Anion Exchange (20-50 mg/L SO4) and POTW waste disposal and pre-oxidation	0.0	0.0	0.0
5	Coagulation Assisted Mcrofiltration and mechanical dew atering/non-hazardous landfill waste disposal and pre-oxidation	5.4	4.5	5.2
6	Coagulation Assisted Mcrofiltration and non-mechanical dew atering/non-hazardous landfill waste disposal and pre-oxidation	5.4	4.5	5.2
7	Oxidation Filtration (Greensand) and POTW for backwash stream and pre-oxidation	12.9	12.5	0.0
8	Activated Alumina (pH7 - pH8) and non-hazardous landfill (for spent media) and pre-oxidation	18.3	14.5	17.5
9	Activated Alumina (pH8 - pH8.3) and non-hazardous landfill (for spent media) and pre-oxidation	0.0	0.0	0.0
10	Activated Alumina (23,100 BV) with pHadjustment (pH6)/corrosion control and non-hazardous landfill (for spent media) and pre-oxidation	27.0	30.0	34.1
11	Activated Alumina (15,400 BV) with pHadjustment (pH6)/corrosion control and non-hazardous landfill (for spent media) and pre-oxidation	27.0	30.0	34.1
12	FOU Activated Alumina and pre-oxidation	0.0	0.0	0.0
13	FOU Reverse Osmosis and pre-oxidation	0.0	0.0	0.0
<b>Sum of Probabilities:</b>		<b>100.00</b>	<b>100.00</b>	<b>100.00</b>

**Exhibit D-5**  
**Probability Decision Tree: “What-If” Sensitivity Analysis**  
**Ground Water Systems Serving 3,301-10,000 People**

No.	Treatment Technology Train	Percent of Treatment Required to Achieve MCL		
		<50%	50-90%	>90%
1	Modify Lime Softening and pre-oxidation	3.0	3.0	3.0
2	Modify Coagulation/Filtration and pre-oxidation	4.0	4.0	4.0
3	Anion Exchange (<20 mg/L SO <sub>4</sub> ) and POTW w aste disposal and pre-oxidation	0.0	0.0	0.0
4	Anion Exchange (20-50 mg/L SO <sub>4</sub> ) and POTW w aste disposal and pre-oxidation	0.0	0.0	0.0
5	Coagulation Assisted Microfiltration and mechanical dewatering/non-hazardous landfill w aste disposal and pre-oxidation	8.7	7.2	8.3
6	Coagulation Assisted Microfiltration and non-mechanical dewatering/non-hazardous landfill w aste disposal and pre-oxidation	2.2	1.8	2.1
7	Oxidation Filtration (Greensand) and POTW for backwash stream and pre-oxidation	0.0	12.5	0.0
8	Activated Alumina (pH 7 - pH 8) and non-hazardous landfill (for spent media) and pre-oxidation	26.0	22.6	26.9
9	Activated Alumina (pH 8 - pH 8.3) and non-hazardous landfill (for spent media) and pre-oxidation	0.0	0.0	0.0
10	Activated Alumina (23,100 BV) w ith pH adjustment (pH 6)/corrosion control and non-hazardous landfill (for spent media) and pre-oxidation	28.1	24.4	27.9
11	Activated Alumina (15,400 BV) w ith pH adjustment (pH 6)/corrosion control and non-hazardous landfill (for spent media) and pre-oxidation	28.1	24.4	27.9
12	POU Activated Alumina and pre-oxidation	0.0	0.0	0.0
13	POU Reverse Osmosis and pre-oxidation	0.0	0.0	0.0
<b>Sum of Probabilities:</b>		<b>100.00</b>	<b>100.00</b>	<b>100.00</b>

**Exhibit D-6**  
**Probability Decision Tree: “What-If” Sensitivity Analysis**  
**Ground Water Systems Serving 10,001-50,000 People**

No.	Treatment Technology Train	Percent of Treatment Required to Achieve MCL		
		<50%	50-90%	>90%
1	Modify Lime Softening and pre-oxidation	4.0	4.0	4.0
2	Modify Coagulation/Filtration and pre-oxidation	4.0	4.0	4.0
3	Anion Exchange (<20 mg/L SO4) and POTW w aste disposal and pre-oxidation	0.0	0.0	0.0
4	Anion Exchange (20-50 mg/L SO4) and POTW w aste disposal and pre-oxidation	0.0	0.0	0.0
5	Coagulation Assisted Microfiltration and mechanical dewatering/non-hazardous landfill w aste disposal and pre-oxidation	63.0	63.0	63.0
6	Coagulation Assisted Microfiltration and non-mechanical dewatering/non-hazardous landfill w aste disposal and pre-oxidation	2.0	2.0	2.0
7	Oxidation Filtration (Greensand) and POTW for backwash stream and pre-oxidation	0.0	4.1	0.0
8	Activated Alumina (pH 7 - pH 8) and non-hazardous landfill (for spent media) and pre-oxidation	4.0	3.3	3.8
9	Activated Alumina (pH 8 - pH 8.3) and non-hazardous landfill (for spent media) and pre-oxidation	0.0	0.0	0.0
10	Activated Alumina (23,100 BV) w ith pH adjustment (pH 6)/corrosion control and non-hazardous landfill (for spent media) and pre-oxidation	11.5	9.8	11.8
11	Activated Alumina (15,400 BV) w ith pH adjustment (pH 6)/corrosion control and non-hazardous landfill (for spent media) and pre-oxidation	11.5	9.8	11.4
12	POU Activated Alumina and pre-oxidation	0.0	0.0	0.0
13	POU Reverse Osmosis and pre-oxidation	0.0	0.0	0.0
	<b>Sum of Probabilities:</b>	<b>100.00</b>	<b>100.00</b>	<b>100.00</b>

**Exhibit D-7**  
**Probability Decision Tree: “What-If” Sensitivity Analysis**  
**Ground Water Systems Serving 50,001-100,000 People**

No.	Treatment Technology Train	Percent of Treatment Required to Achieve MCL		
		<50%	50-90%	>90%
1	Modify Lime Softening and pre-oxidation	4.0	4.0	4.0
2	Modify Coagulation/Filtration and pre-oxidation	4.0	4.0	4.0
3	Anion Exchange (<20 mg/L SO4) and POTW w aste disposal and pre-oxidation	0.0	0.0	0.0
4	Anion Exchange (20-50 mg/L SO4) and POTW w aste disposal and pre-oxidation	0.0	0.0	0.0
5	Coagulation Assisted Microfiltration and mechanical dewatering/non-hazardous landfill w aste disposal and pre-oxidation	63.0	63.0	63.0
6	Coagulation Assisted Microfiltration and non-mechanical dewatering/non-hazardous landfill w aste disposal and pre-oxidation	2.0	2.0	2.0
7	Oxidation Filtration (Greensand) and POTW for backwash stream and pre-oxidation	0.0	4.1	0.0
8	Activated Alumina (pH 7 - pH 8) and non-hazardous landfill (for spent media) and pre-oxidation	2.5	2.1	2.4
9	Activated Alumina (pH 8 - pH 8.3) and non-hazardous landfill (for spent media) and pre-oxidation	0.0	0.0	0.0
10	Activated Alumina (23,100 BV) w ith pH adjustment (pH 6)/corrosion control and non-hazardous landfill (for spent media) and pre-oxidation	12.2	10.4	12.5
11	Activated Alumina (15,400 BV) w ith pH adjustment (pH 6)/corrosion control and non-hazardous landfill (for spent media) and pre-oxidation	12.2	10.4	12.1
12	POU Activated Alumina and pre-oxidation	0.0	0.0	0.0
13	POU Reverse Osmosis and pre-oxidation	0.0	0.0	0.0
	<b>Sum of Probabilities:</b>	<b>100.00</b>	<b>100.00</b>	<b>100.00</b>

**Exhibit D-8**  
**Probability Decision Tree: “What-If” Sensitivity Analysis**  
**Ground Water Systems Serving 100,001-1,000,000 People**

No.	Treatment Technology Train	Percent of Treatment Required to Achieve MCL		
		<50%	50-90%	>90%
1	Modify Lime Softening and pre-oxidation	4.0	4.0	4.0
2	Modify Coagulation/Filtration and pre-oxidation	4.0	4.0	4.0
3	Anion Exchange (<20 mg/L SO <sub>4</sub> ) and POTW w aste disposal and pre-oxidation	0.0	0.0	0.0
4	Anion Exchange (20-50 mg/L SO <sub>4</sub> ) and POTW w aste disposal and pre-oxidation	0.0	0.0	0.0
5	Coagulation Assisted Microfiltration and mechanical dewatering/non-hazardous landfill w aste disposal and pre-oxidation	63.0	63.0	63.0
6	Coagulation Assisted Microfiltration and non-mechanical dewatering/non-hazardous landfill w aste disposal and pre-oxidation	2.0	2.0	2.0
7	Oxidation Filtration (Greensand) and POTW for backwash stream and pre-oxidation	0.0	4.1	0.0
8	Activated Alumina (pH 7 - pH 8) and non-hazardous landfill (for spent media) and pre-oxidation	1.4	1.2	1.4
9	Activated Alumina (pH 8 - pH 8.3) and non-hazardous landfill (for spent media) and pre-oxidation	0.0	0.0	0.0
10	Activated Alumina (23,100 BV) w ith pH adjustment (pH 6)/corrosion control and non-hazardous landfill (for spent media) and pre-oxidation	12.8	10.9	12.8
11	Activated Alumina (15,400 BV) w ith pH adjustment (pH 6)/corrosion control and non-hazardous landfill (for spent media) and pre-oxidation	12.8	10.9	12.8
12	POU Activated Alumina and pre-oxidation	0.0	0.0	0.0
13	POU Reverse Osmosis and pre-oxidation	0.0	0.0	0.0
	<b>Sum of Probabilities:</b>	<b>100.00</b>	<b>100.00</b>	<b>100.00</b>

**Exhibit D-9  
What-If Analysis Results**

<b>MCL Option</b>	<b>Best Estimate</b>	<b>What-If Estimate</b>
5	\$411 Million	\$515 Million
10	\$177 Million	\$192 Million

## Appendix E: Benefits and Costs by System Size Category

The drinking water supply industry is subject to considerable economies of scale with respect to the costs of treatment technologies. Per capita treatment costs steeply increase in inverse proportion to system size. This is illustrated earlier in this report by Exhibit 6-17 wherein a hundred-fold increase in household costs over the range of public water supplies can be observed at the chosen MCL. Because there is such a large increase in relative costs, benefit-cost ratios also show appreciable variation with system size. In response to comments received on the proposal, the Agency is providing a subcategorization of the benefits and costs associated with the various regulatory alternatives by system size.

Cost values for strata specific costs were taken from the National cost modeling effort and reflect use of a three percent interest rate for annualizing capital costs. Benefits were calculated as a product of the mean risk reductions (see Exhibit 5-4(c) and calculated as described in Appendix B ), populations served by impacted sites (shown in Exhibit E-1 and calculated per cost methodology described in Appendix C), and costs per case avoided (as described in Chapter 8 and Appendix B). For the latter element, \$6.1 million was assumed per cancer fatality and \$607,000 for non-fatal cancers. Exhibit E-1 depicts the benefits by system size category and Exhibit E-2 displays benefit cost ratios.

<b>Exhibit E-1</b>					
<b>Benefits by System Size</b>					
		<b>Population Stratum</b>			
<b>Type</b>	<b>MCL</b>	<b>25-500</b>	<b>500-3300</b>	<b>3300-10,000</b>	<b>10K-1000K</b>
<b>Upper</b>	20	2.41	7.90	9.09	45.18
<b>Upper</b>	10	7.13	23.34	26.85	133.50
<b>Upper</b>	5	12.45	40.75	46.89	233.11
<b>Upper</b>	3	16.67	54.57	62.80	312.18
<b>Lower</b>	20	2.72	8.91	10.25	50.97
<b>Lower</b>	10	5.15	16.85	19.39	96.40
<b>Lower</b>	5	7.01	22.94	26.39	131.20

<b>Exhibit E-2</b>					
<b>Benefit/Cost Ratios by System Size</b>					
		<b>Population Stratum</b>			
<b>Bound</b>	<b>MCL</b>	<b>25-500</b>	<b>500-3300</b>	<b>3300-10,000</b>	<b>10K-1000K</b>
<b>Impacted Population (thousands)</b>		<b>961</b>	<b>315</b>	<b>3,622</b>	<b>18,005</b>
<b>upper</b>	20	0.38	0.74	1.01	1.32
<b>upper</b>	10	0.42	0.81	1.11	1.39
<b>upper</b>	5	0.33	0.62	0.84	1.05
<b>upper</b>	3	0.27	0.50	0.66	0.85
<b>lower</b>	20	0.43	0.84	1.14	1.49
<b>lower</b>	10	0.30	0.59	0.80	1.00
<b>lower</b>	5	0.18	0.35	0.47	0.59

# Attachment 27

respondents into groups that align with the source categories identified in the rule.

Reporting facilities include, but are not limited to, those operating one or more units that exceed the CO<sub>2</sub>e threshold for the industry sectors listed in Table A-4 of 40 CFR 98.2(a)(2) or those in the categories in which all must report, such as petroleum refining facilities and all other large emitters listed in Table A-3 of 40 CFR 98.2(a)(1). Additionally, the GHGRP requires reporting of GHGs from certain suppliers as listed in Table A-5 of 40 CFR 98.2(a)(4) and of certain emissions information associated with mobile sources (e.g., for permit applications or emissions control certification testing procedures).

*Respondent's Obligation To Respond:* Mandatory (Sections 114 and 208 of the Clean Air Act provide EPA authority to require the information mandated by the Greenhouse Gas Reporting Program because such data will inform and are relevant to future policy decisions).

*Estimated Number of Respondents:* 11,080 (total).

*Frequency of Response:* Annual.

*Total Estimated Burden:* 739,187 hours (per year). Burden is defined at 5 CFR 1320.03(b).

*Total Estimated Cost:* \$99,831,931 per year, which includes \$30,621,791 for capital investment and operation and maintenance costs for respondents, labor cost of \$57,210,010 for respondents, and \$12,000,130 for the EPA.

*Changes in the Estimates:* This change in burden reflects an update in the number of respondents, an adjustment of labor rates to 2014 Bureau of Labor and Statistics (BLS) labor rates, an adjustment of capital costs to reflect 2013 dollars, a re-evaluation of the costs to monitor and report combustion emissions across the entire program, a re-evaluation of the activities and costs associated with Petroleum and Natural Gas Systems (Subpart W) and Geologic Sequestration of Carbon Dioxide (Subpart RR), and the addition of new segments and new reporters under Subpart W.

**Courtney Kerwin,**

*Acting Director, Collection Strategies Division.*

[FR Doc. 2016-12310 Filed 5-24-16; 8:45 am]

**BILLING CODE 6560-50-P**

## ENVIRONMENTAL PROTECTION AGENCY

[EPA-HQ-OW-2014-0138; FRL-9946-91-OW]

### Lifetime Health Advisories and Health Effects Support Documents for Perfluorooctanoic Acid and Perfluorooctane Sulfonate

**AGENCY:** Environmental Protection Agency (EPA).

**ACTION:** Notice of availability.

**SUMMARY:** The Environmental Protection Agency (EPA) announces the release of lifetime health advisories (HAs) and health effects support documents for Perfluorooctanoic Acid (PFOA) and Perfluorooctane Sulfonate (PFOS). EPA developed the HAs to assist federal, state, tribal and local officials, and managers of drinking water systems in protecting public health when these chemicals are present in drinking water. EPA's HAs, which identify the concentration of PFOA and PFOS in drinking water at or below which adverse health effects are not anticipated to occur over a lifetime of exposure, are: 0.07 parts per billion (70 parts per trillion) for PFOA and PFOS. HAs are non-regulatory and reflect EPA's assessment of the best available peer-reviewed science. These HAs supersede EPA's 2009 provisional HAs for PFOA and PFOS.

**FOR FURTHER INFORMATION CONTACT:** Jamie Strong, Health and Ecological Criteria Division, Office of Water (Mail Code 4304T), Environmental Protection Agency, 1200 Pennsylvania Avenue NW., Washington, DC 20460; telephone number: (202) 566-0056; email address: [strong.jamie@epa.gov](mailto:strong.jamie@epa.gov).

#### SUPPLEMENTARY INFORMATION:

##### I. General Information

*A. How can I get copies of this document and other related information?*

1. *Docket.* EPA has established a docket for this action under Docket ID No. EPA-HQ-OW-2014-0138. Publicly available docket materials are available either electronically through [www.regulations.gov](http://www.regulations.gov) or in hard copy at the Water Docket in the EPA Docket Center, (EPA/DC) EPA West, Room 3334, 1301 Constitution Ave. NW., Washington, DC. The EPA Docket Center Public Reading Room is open from 8:30 a.m. to 4:30 p.m., Monday through Friday, excluding legal holidays. The telephone number for the Public Reading Room is (202) 566-1744, and the telephone number for the Water Docket is (202) 566-2426.

2. *Electronic Access.* You may access this **Federal Register** document electronically from the Government Printing Office under the "Federal Register" listings FDSys (<http://www.gpo.gov/fdsys/browse/collection.action?collectionCode=FR>).

## II. What are perfluorooctanoic acid and perfluorooctane sulfonate and why is EPA concerned about them?

PFOA and PFOS are fluorinated organic chemicals that are part of a larger group of chemicals referred to as perfluoroalkyl substances. They were used to make carpets, clothing, fabrics for furniture, paper packaging for food and other materials (e.g., cookware) that are resistant to water, grease or stains. They are also used for firefighting at airfields and in a number of industrial processes. Both PFOA and PFOS are persistent in the environment and in the human body. Over time both chemicals have become widely distributed in the environment and have accumulated in the blood of humans, wildlife, and fish. Studies indicate that exposure to PFOA and PFOS over certain levels may result in adverse health effects, including developmental effects to fetuses during pregnancy or to breast-fed infants (e.g., low birth weight, accelerated puberty, skeletal variations), cancer (e.g., testicular, kidney), liver effects (e.g., tissue damage), immune effects (e.g., antibody production and immunity), and other effects (e.g., cholesterol changes).

## III. What are health advisories?

Under the Safe Drinking Water Act, EPA may publish HAs for contaminants that are not subject to any national primary drinking water regulation. SDWA section 1412(b)(1)(F). EPA develops HAs to provide information on the chemical and physical properties, occurrence and exposure, health effects, quantification of toxicological effects, other regulatory standards, analytical methods, and treatment technology for drinking water contaminants. HAs describe concentrations of drinking water contaminants at which adverse health effects are not anticipated to occur over specific exposure durations (e.g., one-day, ten-days, and a lifetime). HAs serve as informal technical guidance to assist federal, state and local officials, as well as managers of public or community water systems in protecting public health. They are not regulations and should not be construed as legally enforceable federal standards. HAs may change as new information becomes available.

#### IV. Information on the Drinking Water Health Advisories for PFOA and PFOS

EPA's HA levels, which identify the concentration of PFOA and PFOS in drinking water at or below which adverse health effects are not anticipated to occur over a lifetime of exposure, are: 0.07 parts per billion (70 parts per trillion) for PFOA and PFOS. Because these two chemicals cause similar types of adverse health effects, EPA recommends that when both PFOA and PFOS are found in drinking water the combined concentrations of PFOA and PFOS be compared with the 0.07 part per billion HA level.

EPA's lifetime HAs are based on peer-reviewed toxicological studies of exposure of animals to PFOA and PFOS, applying scientifically appropriate uncertainty factors. The development of the HAs was also informed by epidemiological studies of human populations that have been exposed to PFOA and PFOS. The HAs are set at levels that EPA concluded will not result in adverse developmental effects to fetuses during pregnancy or to breast-fed infants, who are the groups most sensitive to the potential harmful effects of PFOA and PFOS. EPA's analysis indicates that exposure to these same levels will not result in adverse health effects (including cancer and non-cancer) to the general population over a lifetime (or any shorter period) of exposure to these chemicals.

EPA's HAs for PFOA and PFOS are supported by peer-reviewed health effects support documents that summarize and analyze available peer-reviewed studies on toxicokinetics, human epidemiology, animal toxicity, and provide a cancer classification and a dose response assessment for noncancer effects. On February 28, 2014, EPA released draft versions of these health effects support documents for a 60-day public comment period and initiated a contractor-led, independent public panel peer review process (79 FR 11429). The peer review panel meeting occurred on August 21–22, 2014, and included seven experts in the following areas: Epidemiology, toxicology (liver, immune, neurological and reproductive and developmental effects), membrane transport, risk assessment, pharmacokinetic models, and mode-of-action for cancer and noncancer effects (79 FR 39386). Comments submitted to EPA's public docket during the 60-day public comment period were provided to the peer reviewers ahead of the meeting for their consideration. A peer review summary report and other supporting documents may be found at:

<http://www.regulations.gov> under the docket EPA-HQ-OW-2014-0138.

Dated: May 19, 2016.

**Joel Beauvais,**

*Deputy Assistant Administrator, Office of Water.*

[FR Doc. 2016-12361 Filed 5-24-16; 8:45 am]

**BILLING CODE 6560-50-P**

#### ENVIRONMENTAL PROTECTION AGENCY

[EPA-HQ-OPP-2015-0021; FRL-9946-40]

#### Pesticide Product Registration; Receipt of Applications for New Active Ingredients

**AGENCY:** Environmental Protection Agency (EPA).

**ACTION:** Notice.

**SUMMARY:** EPA has received applications to register pesticide products containing active ingredients not included in any currently registered pesticide products. Pursuant to the Federal Insecticide, Fungicide, and Rodenticide Act (FIFRA), EPA is hereby providing notice of receipt and opportunity to comment on these applications.

**DATES:** Comments must be received on or before June 24, 2016.

**ADDRESSES:** Submit your comments, identified by docket identification (ID) number and the File Symbol of interest as shown in the body of this document, by one of the following methods:

- *Federal eRulemaking Portal:* <http://www.regulations.gov>. Follow the online instructions for submitting comments. Do not submit electronically any information you consider to be Confidential Business Information (CBI) or other information whose disclosure is restricted by statute.

- *Mail:* OPP Docket, Environmental Protection Agency Docket Center (EPA/DC), (28221T), 1200 Pennsylvania Ave. NW., Washington, DC 20460-0001.

- *Hand Delivery:* To make special arrangements for hand delivery or delivery of boxed information, please follow the instructions at <http://www.epa.gov/dockets/contacts.html>.

Additional instructions on commenting or visiting the docket, along with more information about dockets generally, is available at <http://www.epa.gov/dockets>.

**FOR FURTHER INFORMATION CONTACT:** Robert McNally, Biopesticides and Pollution Prevention Division (7511P), Office of Pesticide Programs, Environmental Protection Agency, 1200 Pennsylvania Ave. NW., Washington, DC 20460-0001; main telephone

number: (703) 305-7090; email address: [BPPDFRNotices@epa.gov](mailto:BPPDFRNotices@epa.gov).

#### SUPPLEMENTARY INFORMATION:

##### I. General Information

###### A. Does this action apply to me?

You may be potentially affected by this action if you are an agricultural producer, food manufacturer, or pesticide manufacturer. The following list of North American Industrial Classification System (NAICS) codes is not intended to be exhaustive, but rather provides a guide to help readers determine whether this document applies to them. Potentially affected entities may include:

- Crop production (NAICS code 111).
- Animal production (NAICS code 112).
- Food manufacturing (NAICS code 311).
- Pesticide manufacturing (NAICS code 32532).

###### B. What should I consider as I prepare my comments for EPA?

1. *Submitting CBI.* Do not submit this information to EPA through [regulations.gov](http://www.regulations.gov) or email. Clearly mark the part or all of the information that you claim to be CBI. For CBI information in a disk or CD-ROM that you mail to EPA, mark the outside of the disk or CD-ROM as CBI and then identify electronically within the disk or CD-ROM the specific information that is claimed as CBI. In addition to one complete version of the comment that includes information claimed as CBI, a copy of the comment that does not contain the information claimed as CBI must be submitted for inclusion in the public docket. Information so marked will not be disclosed except in accordance with procedures set forth in 40 CFR part 2.

2. *Tips for preparing your comments.* When preparing and submitting your comments, see the commenting tips at <http://www.epa.gov/dockets/comments.html>.

##### II. Registration Applications

EPA has received applications to register pesticide products containing active ingredients not included in any currently registered pesticide products. Pursuant to the provisions of FIFRA section 3(c)(4) (7 U.S.C. 136a(c)(4)), EPA is hereby providing notice of receipt and opportunity to comment on these applications. Notice of receipt of these applications does not imply a decision by the Agency on these applications.

1. File Symbol: 91197-E. Docket ID number: EPA-HQ-OPP-2016-0251. Applicant: AFS009 Plant Protection,

# Attachment 28

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Organ Toxicity and Mechanisms |  
[Published: 03 December 2019](#)

# Subacute dermal toxicity of perfluoroalkyl carboxylic acids: comparison with different carbon-chain lengths in human skin equivalents and systemic effects of perfluoroheptanoic acid in Sprague Dawley rats

[Ji-Seok Han](#), [Sumi Jang](#), [Hwa-Young Son](#), [Yong-Bum Kim](#),  
[Younhee Kim](#), [Jung-Ho Noh](#), [Mi-Jeong Kim](#) & [Byoung-Seok Lee](#) 

[Archives of Toxicology](#) **94**, 523–539 (2020)

**771** Accesses | **7** Citations | **17** Altmetric | [Metrics](#)

## Abstract

Perfluoroalkyl and polyfluoroalkyl substances (PFASs) are used in various fields but raise concerns regarding human health and environmental consequences. Among PFASs, perfluorooctanoic acid (PFOA) and short-chain perfluoroalkyl carboxylic acids (SC PFCAs) are detectable in skin-contact consumer products and have dermal absorption potential. Here, we investigated the effects of dermal exposure to PFOA and SC PFCAs using in vitro and in vivo models. Human skin equivalents were topically

treated with 0.25 mM and 2.5 mM PFOA and SC PFCAs (perfluoropentanoic acid, PFPeA; perfluorohexanoic acid, PFHxA; and perfluoroheptanoic acid, PFHpA) for 6 days, and cell viability, interleukin (IL)-1 $\alpha$ , oxidative stress markers (malondialdehyde, MDA; and 8-hydroxydeoxyguanosine, 8-OHdG), and histopathology were examined. MDA levels were significantly higher in the PFASs groups than in controls. Compared with SC PFCAs, 2.5 mM PFOA caused more IL-1 $\alpha$  ( $p < 0.001$ ) release, decreased skin thickness and microscopic abnormalities. To evaluate systemic effects, Sprague Dawley (SD) rats were dermally treated with 250 and 1000 mg/kg PFHpA for 2 weeks and clinical and anatomic pathology were assessed. At 1000 mg/kg, 83% of the rats died, with severe ulcerative dermatitis at the application site. Adverse PFHpA-treated systemic changes were observed in the kidney, liver and testes, and histopathologic lesions such as renal tubular necrosis, hepatocellular necrosis, and germ cell degeneration were seen at 250 and 1000 mg/kg. Our study suggests that SC PFCAs have fewer effects on the skin than PFOA, but SC PFCAs can have adverse effects on major organs with systemic exposure at high concentrations.

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#### Ethics declarations

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#### Conflict of interest

The authors report no declarations of interest.

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# Attachment 29

# Perfluoroheptanoic acid affects amphibian embryogenesis by inducing the phosphorylation of ERK and JNK

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**Abstract.** Perfluoroalkyl compounds (PFCs) are globally distributed synthetic compounds that are known to adversely affect human health. Developmental toxicity assessment of PFCs is important to facilitate the evaluation of their environmental impact. In the present study, we assessed the developmental toxicity and teratogenicity of PFCs with different numbers of carbon atoms on *Xenopus* embryogenesis. An initial frog embryo teratogenicity assay-*Xenopus* (FETAX) assay was performed that identified perfluorohexanoic (PFHxA) and perfluoroheptanoic (PFHpA) acids as potential teratogens and developmental toxicants. The mechanism underlying this teratogenicity was also investigated by measuring the expression of tissue-specific biomarkers such as phosphotyrosine-binding protein, xPTB (liver); NKX2.5 (heart); and Cyl18 (intestine). Whole-mount *in situ* hybridization, reverse transcriptase-polymerase chain reaction (RT-PCR), and histologic analyses detected severe defects in the liver and heart following exposure to PFHxA or PFHpA. In addition, immunoblotting revealed that PFHpA significantly increased the phosphorylation of extracellular signal-regulated kinase (ERK) and c-Jun N-terminal kinase (JNK), while PFHxA slightly increased these, as compared with the control. These results suggest that PFHxA and PFHpA are developmental toxicants and teratogens, with PFHpA producing more severe effects on liver and heart development through the induction of ERK and JNK phosphorylation.

## Introduction

Environmental pollution emerged as a significant threat to human health early in the 20th century, as industrial societies became aware of an increased prevalence of a range of chronic illnesses and conditions. Environmental pollution is fueled by large-scale generation of synthetic chemicals that are normally employed in necessary industrial processes (1,2). Among these anthropogenic chemicals, perfluoroalkyl compounds (PFCs) are organofluorines, which have fluorine instead of hydrogen in their carbon chains. Strong bonds form between fluorine and carbon (Fig. 1A). The carbon-fluorine (C-F) bond is considered the strongest polar covalent bond, with a high dissociation energy reaching 117 kcal/mol. The strong polarity and high dissociation energy of C-F bonds make PFCs resistant to a range of biochemical processes such as thermal, chemical, hydrolytic, and biological degradation (2). Furthermore, the hydrophobic fluorinated alkyl chain and the lipophilic functional group make PFCs valuable in many industrial and commercial processes involved in the production of fabrics, electronics, pesticides, emulsifiers, paints and adhesives (3,4). This wide-ranging use of perfluorohexanoic (PFHxA) and perfluoroheptanoic (PFHpA) acids has resulted in their detection as pollutants in water, sediments, air, wildlife and humans, and these compounds are not degraded by any naturally occurring processes in the environment (5).

PFCs are present in atmospheric and aquatic systems on a global level (6). Environmental levels of PFCs were investigated in different world regions, identifying high PFC levels reaching 100 ng/l (7-9). The concentrations of various PFCs, which exert unpleasant effects in humans, have been determined in human blood samples from different world regions (10). High levels of PFCs in serum and liver samples from both humans and animals have also been detected (11). The levels of PFCs were elevated in individuals with regular occupational exposure to PFCs (4.3 ppb), as compared to the general population (12). Their unique physicochemical properties make PFCs highly persistent, increasing their bioaccumulation and toxicological effects (5,13). In addition, it has been shown that PFCs exert toxic reproductive and developmental effects on human and animal systems (12,14,15).

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Overexposure to these anthropogenic chemicals causes severe fetal abnormalities in amphibians and affects human development (12,16). The toxicity of PFCs is commonly determined by their carbon chain length and by the functional group.

The frog embryo teratogenicity assay-*Xenopus* (FETAX) utilizing anuran *Xenopus* embryos is a well-characterized method used to measure the developmental toxicity of chemicals. The FETAX assay is performed on fertilized *Xenopus* embryos in the middle of the blastula stage, at 96 h (17). Teratogenic effects are assessed by determining their influences on mortality, malformation, and growth inhibition, effects that clearly indicate developmental toxicity. This assay has been used to investigate teratogens and developmental toxicants that negatively affect human health and the ecosystem (18,19). FETAX is appropriate for the screening of chemicals (including nanomaterials), evaluating biotransformation, and can serve as a model of limb development. It is used to inform risk assessments of the effects of environmental hazards on amphibian and vertebrate populations. In addition to FETAX, reverse transcriptase-polymerase chain reaction (RT-PCR) and whole-mount *in situ* hybridization are useful in the investigation of the developmental toxicity of these compounds, as well as mechanisms underlying any effects on *Xenopus* embryos.

The toxicological potentials of PFHxA and PFHpA need to be investigated to determine the adverse effects of PFCs with different lengths. In the present study, we evaluated the developmental toxicity and teratogenicity of PFHxA and PFHpA using different biological assays. The results obtained from the *in vivo* model system showed that PFHpA is a more potent developmental toxicant and teratogen than PFHxA. In particular, PFHxA and PFHpA affected mortality and the growth rates of embryos, and caused severe developmental liver and heart defects. These results suggested that although PFHxA and PFHpA are developmental toxicants and teratogens, PFHpA has more severe effects on liver and heart development via the induction of extracellular signal-regulated kinase (ERK) and c-Jun N-terminal kinase (JNK) phosphorylation.

## Materials and methods

**Ethics statement.** We obtained a specific waiver from the Institutional Review Board of Kyungpook National University for the experimental use of amphibians or reptiles in Korea (no. KNU-2013-222). All the members of our research group attended educational and training courses on the appropriate care and usage of experimental animals. Adult *Xenopus laevis* were entrained in 12-h light/dark cycles at 18°C in containers provided by the Institutional Review Board of Kyungpook National University constructed to specifications for laboratory animal maintenance. There were no unexpected deaths of adult *Xenopus* during the study.

**Chemicals and reagents.** PFHxA and PFHpA were purchased from Sigma-Aldrich (St. Louis, MO, USA) and dissolved in dimethyl sulfoxide (DMSO) to produce 1 M stock solutions. The FETAX medium used in the experiment contained 10.7 mM NaCl, 1.14 mM NaHCO<sub>3</sub>, 0.4 mM KCl, 0.1 mM CaCl<sub>2</sub>, 0.35 mM CaSO<sub>4</sub>·2H<sub>2</sub>O, and 0.3 mM MgSO<sub>4</sub> and the levels of DMSO did not exceed 0.15%, which was below the

permissible level for this assay. PFC stocks were freshly made and diluted in FETAX medium.

***In vitro* fertilization.** Experiments on *Xenopus laevis* were conducted in accordance with documented standards of the Animal Care and Use Committee, in agreement with international laws and policies [National Institutes of Health (NIH) Guide for the Care and Use of Laboratory Animals, NIH publication no. 85-23, 1985]. Adult *Xenopus* were purchased from Nasco (Fort Atkinson, WI, USA) and kept in climate-controlled clear plastic aquariums with dechlorinated tap water at 18±2°C, with 12-h light/dark cycles. The housed frogs were fed only three times a week. For the induction of ovulation, human chorionic gonadotropin (1,000 IU) (Sigma-Aldrich) was injected under the skin of a female *Xenopus* in the evening. The following day, the females were made to lay eggs in 60-mm plastic dishes. The eggs were immediately fertilized in 0.1X modified Barth's saline (MBS) containing 88 mM NaCl, 5 mM Hepes, 2.5 mM NaHCO<sub>3</sub>, 1 mM KCl, 1 mM MgSO<sub>4</sub>, and 0.7 mM CaCl<sub>2</sub> (pH 7.8) after washing three times with 0.1X MBS. The frogs were sacrificed as follows: The anesthetic of choice for male frogs, tricaine methanesulfonate (MS222) was dissolved in dechlorinated water in an induction tank at a dose of 500 mg of MS222/l of water. *Xenopus* testes were obtained from the sacrificed males and minced in 1 ml of cold 1X MBS. Following successful fertilization, the jelly coat was removed from the embryos by gentle swirling in a 2% L-cysteine solution. The embryos were then transferred to 1X MBS containing 3% Ficoll 400 (GE Healthcare, Little Chalfont, UK). Unfertilized eggs and dead embryos were removed and the remaining embryos were maintained at 22±0.5°C until they reached blastula stage 8.5.

**FETAX assay.** FETAX assays were conducted to assess the developmental toxicity and teratogenic effects of the PFCs based on the American Society of Testing Material (ASTM) guide (ASTM E1439-98). Finely cleaved embryos in the blastula stage 8.5 were selected and used to exclude the effects of spontaneous embryonic developmental problems. Embryos were impartially allocated to 100-mm petri dishes (20-25 embryos/dish) and exposed to different concentrations of PFHxA (0.1, 0.5, 1, 1.5 and 2 mM) and PFHpA (0.25, 0.5, 0.75, 1 and 1.25 mM). DMSO (0.1%) and FETAX medium alone were used as the controls. Embryos were incubated at 23°C until the end of the assay, and the media were changed daily and dead embryos were removed. At the end of the experiment, embryo mortality was recorded and the surviving embryos were fixed in 4% formaldehyde to determine whether there was any malformation. Head-tail lengths and malformations were evaluated under a light microscope and images were analyzed using Axiovision software version 4.8 (Carl Zeiss, Munich, Germany) to measure growth inhibition.

**Whole-mount *in situ* hybridization.** *Xenopus* embryos were prepared for whole-mount *in situ* hybridization using a previously described method (20), with minor modification. Whole-mount *in situ* hybridization was conducted at the tadpole stage (stage 34-36) using digoxigenin-labeled RNA probes and BM purple staining. The probes were prepared in the following manner: RT-PCR products of phosphotyrosine-binding protein (xPTB; a liver marker) and NKX2.5 (heart marker)

Table I. Primers for RT-PCR.

Gene name	GeneBank accession no.	RT-PCR primer sequences (5'-3')	Product size (bp)	Annealing temperature (°C)	Cycles
xPTB	AY183756	F: ATGGATGCACTCAAGTCTGC R: TGGTAGCTTCAGAAGTGTTCAG	933	58	28
NKX2.5	NM_001172192	F: GTCAGCATCAACCCCTACAG R: TGCCACCCAGTCTTTAATC	491	53	28
Cyl18	AY188285	F: AGGCCGTAGCACCTGTGGGT R: GGTCCCACCAGCTCGGATCA	462	55	21
ODC	NM_001086698	F: GTGATGGGCTGGATCGTATC R: TCCATTGAATGTCGAGGCTG	386	55	21

F, forward; R, reverse; RT-PCR, reverse transcriptase-polymerase chain reaction.

Table II. Teratogenic index for PFHxA and PFHpA.

Acid	LC50 ( $\mu$ M) lethality	EC50 ( $\mu$ M) malformation	MCIG ( $\mu$ M)	TI=LC50/EC50
PFHxA	1523.5	1046.5	412.3	1.43
PFHpA	942.4	754.9	297.4	1.25

PFHxA, perfluorohexanoic acid; PFHpA, perfluoroheptanoic acid; TI, teratogenic index.

were sub-cloned into a T-easy vector (Promega, Fitchburg, WI, USA), linearized with *Apa*I and *Nco*I, respectively, and transcribed using a SP6 RNA polymerase kit (Invitrogen, Carlsbad, CA, USA).

**RT-PCR.** RNA was isolated from stage 34-46 embryos using TRIzol reagent, according to the manufacturer's instructions (Invitrogen), and quantified using an Optizen 320 UV spectrometer (Mecacys, Deajeon, Korea). An aliquot of RNA (1 g) was reverse transcribed using a PrimeScript First-Strand cDNA Synthesis kit, according to the manufacturer's instructions (Takara, Shiga, Japan). Reactions containing template RNA without reverse transcriptase were prepared as the negative controls (no-RT) and carrier was used as the loading control. cDNA was amplified using specific PCR primer sets (Table I) and 2X Emerald Amp PCR Master mix (Takara). The PCR products were separated in a 1-1.2% agarose gel and visualized on a transilluminator (Vilber Lourmat, Marne-la-Vallée, France) following ethidium bromide staining. The data were representative of at least three experiments. The Primer3 program was used to design primers and ODC was used as an internal control.

**Extraction and analysis of the liver.** Twenty-five embryos from each group were exposed to PFHxA and PFHpA, respectively, for 10 days. The surviving tadpoles were fixed in 4% formaldehyde for 2 h at room temperature. Tadpoles (3-5) were randomly selected from each group and their livers removed under a light microscope (Zeiss Stemi 2000-c; Carl Zeiss). Images were captured and analyzed to monitor changes in the liver size.

**Statistical analysis.** Data for the three endpoints from each FETAX assay were analyzed using GraphPad Prism software (GraphPad Software, Inc., La Jolla, CA, USA). Images from the RT-PCR and whole-mount *in situ* hybridization assays were analyzed by ImageJ software (NIH). Statistical analyses were conducted using SPSS 13.0 software (version 18; SPSS, Inc., Chicago, IL, USA).

## Results

**Developmental toxicity and teratogenicity assessment of PFCs by FETAX assay.** FETAX assays were performed to determine the potential teratogenicity and developmental toxicity of PFCs. FETAX assay results were expressed relatively in terms of the teratogenic index (TI)=LC50/EC50, where LC50 was the concentration that at which 50% of the embryos were killed and EC50 was the concentration at which 50% of the embryos were malformed. The minimum concentration that inhibits growth (MCIG) was also determined. Compounds were considered toxic when TI was  $\geq 1.2$  (17).

We performed an initial FETAX assay to determine whether PFHxA and PFHpA had embryonic lethal or teratogenic effects. This result confirmed that PFHxA and PFHpA were potential teratogens and developmental toxicants. The LC50 value for PFHxA in *Xenopus* embryos was 1523.5  $\mu$ M, while the EC50 value was 1046.5  $\mu$ M, and the MCIG value was 412.3  $\mu$ M, which produced a TI value of 1.43, indicating that this compound was a potential teratogen and developmental toxicant. The LC50 value for PFHpA was 942.4  $\mu$ M in the *Xenopus* embryos, with an EC50 of 754.9  $\mu$ M and an MCIG value of 297.4  $\mu$ M, producing a TI value of 1.25. Thus, this compound was also considered a teratogen and developmental toxicant (Table II). PFHxA had a significantly higher TI value (TI=1.43) than PFHpA (TI=1.25). Furthermore, the LC50 and EC50 values were higher for PFHxA, which had a shorter fluorinated carbon chain than PFHpA.

**Effects of PFHxA and PFHpA on *Xenopus* mortality and malformation rates.** Our FETAX assay results confirmed that PFHxA and PFHpA are potential teratogens and developmental toxicants. We then investigated the effects of

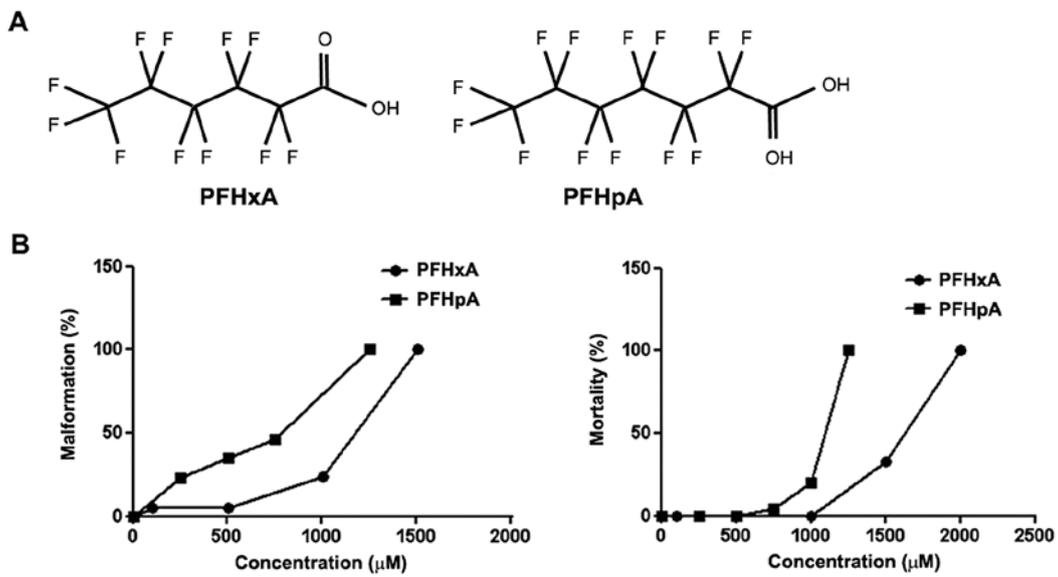


Figure 1. Lewis structures of PFCs. (A) Structures of PFHxA and PFHpA. (B) Effects of PFHxA and PFHpA on *Xenopus* mortality and malformation. Mortality and malformation rates were measured among embryos in stage 46 after exposure to PFHxA or PFHpA and the findings confirmed that the two compounds showed developmental toxicity and teratogenicity. PFCs, perfluoroalkyl compounds; PFHxA, perfluorohexanoic acid; PFHpA, perfluoroheptanoic acid.

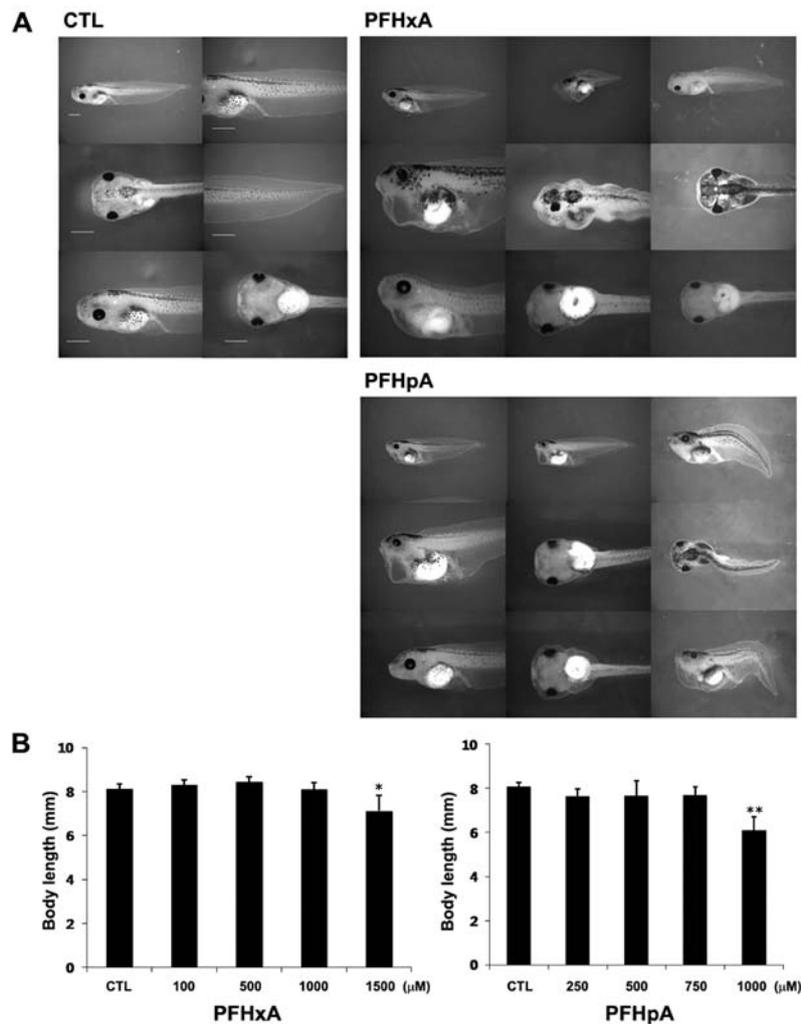


Figure 2. Abnormal phenotypes and shorter body lengths resulting from PFHxA and PFHpA exposure. Embryos developed various malformations including (A) stunted body, multiple edemas, gut mis-coiling, microcephaly, skeletal kinking and (B) shorter body length following exposure to PFHxA and PFHpA. Mean  $\pm$  SD; \* $P < 0.05$ , \*\* $P < 0.05$  compared with the control cells. PFHxA, perfluorohexanoic acid; PFHpA, perfluoroheptanoic acid; SD, standard deviation.

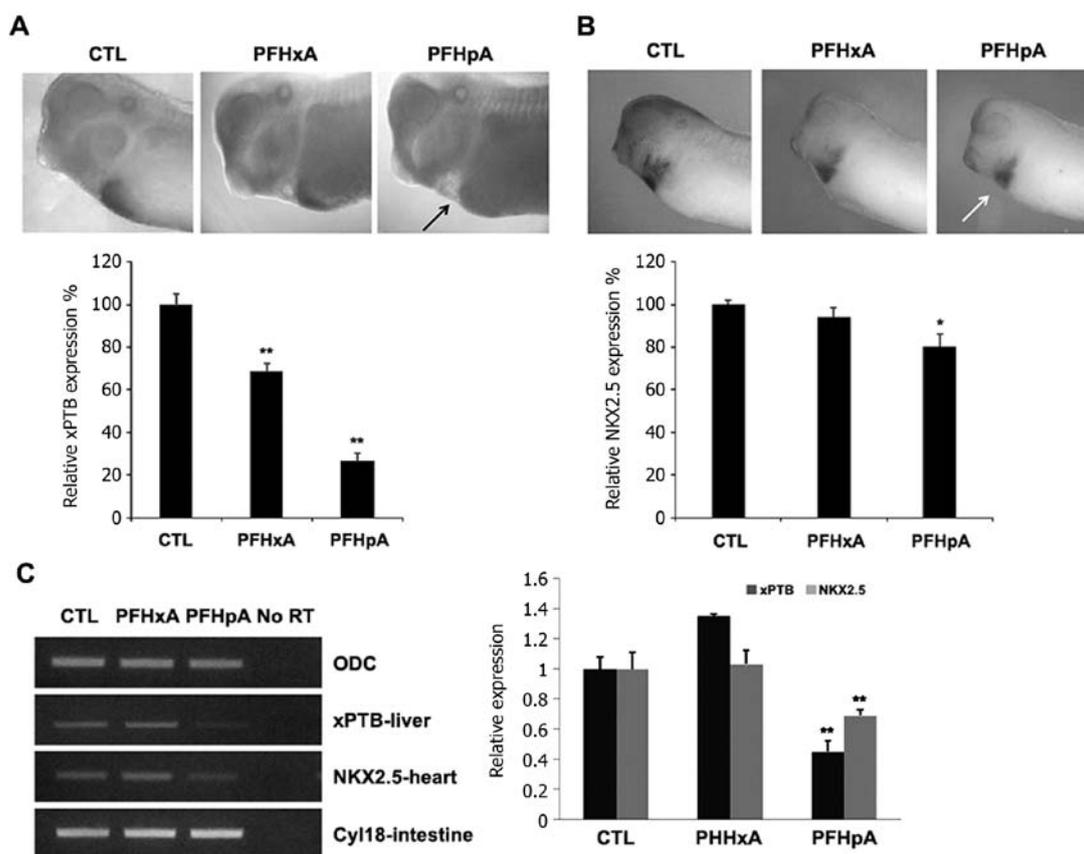


Figure 3. PFHpA severely affects liver and heart development. (A) PFHxA and PFHpA exposure altered the expression of tissue-specific mRNAs. Whole-mount *in situ* hybridization of stage 36 embryos exposed to 130  $\mu$ M PFHxA and PFHpA. xPTB expression levels were reduced in the liver and heart of exposed embryos (black arrow). The histogram shows the relative area and intensity of xPTB in the liver, expressed as a percentage of the control. Data are the mean of three independent experiments. (B) PFHxA and PFHpA exposure altered the expression of tissue-specific mRNAs. Whole-mount *in situ* hybridization of stage 36 embryos exposed to 130  $\mu$ M PFHxA and PFHpA. NKX2.5 expression levels were reduced in the heart of exposed embryos (white arrow). The histogram shows the relative area and intensity of NKX2.5 mRNA expression in the heart, expressed as a percentage of the control. Data are the mean of three independent experiments. (C) The levels of xPTB, NKX2.5, and Cyl18 mRNA expression were determined by RT-PCR. PCR was conducted without reverse transcriptase (No-RT) as a negative control and the carrier was used as the loading control. The histogram shows the relative expression levels of xPTB, NKX2.5, expressed as a percentage of the control ODC. Mean  $\pm$  SD; \*\* $P$ <0.05, \* $P$ <0.05 compared with the control cells. PFHxA, perfluorohexanoic acid; PFHpA, perfluoroheptanoic acid; SD, standard deviation.

different concentrations of PFHxA and PFHpA on mortality and malformation in developing embryos. As shown in Fig. 1B, exposure to PFHxA or PFHpA greatly increased the mortality and general malformation rates in the developing embryos. These concentration-dependent effects on mortality and malformation were more severe as the number of carbons increased. PFHpA significantly affected mortality and malformation rates within a narrow concentration range (Fig. 1B).

To investigate the mechanism underlying this toxicity, we assessed the phenotypes of embryos exposed to PFHxA and PFHpA. PFHxA and PFHpA were not highly toxic until early stage 26, as no significant differences were observed in the external phenotypes. PFC exposure induced certain embryonic abnormalities that were observed at the end of the study, such as pericardial edema, dorsal fin blisters, a curved body axis, lack of eye pigment, and disturbed facial cells (Fig. 2A). PFHxA-treated embryos developed mild and general malformations including gut mis-coiling and improper eye shape, whereas PFHpA induced more severe and diverse malformations (Fig. 2A). In agreement with this, the whole body length was reduced by 14 and 24% in tadpoles treated with 1,500  $\mu$ M of PFHxA or 1,000  $\mu$ M of PFHpA, respectively (Fig. 2B).

These results indicated that embryos exposed to PFHxA or PFHpA show a range of malformations and a reduced whole body length, which is a clear indicator of toxicity while PFHpA has slightly more severe defects compared to PFHxA.

*PFHpA modulates liver and heart development during embryogenesis.* We examined stage 36 tadpoles that had been continuously exposed to 130  $\mu$ M PFHxA or PFHpA using whole-mount *in situ* hybridization. The liver is a major metabolizing organ that detoxifies many compounds and is therefore vulnerable to the toxic effects of PFCs. We also measured a liver-specific marker, xPTB, which is expressed in the diverticulum region of the embryonic liver and plays an important role in low-density lipoprotein (LDL) receptor endocytosis in the liver (21). Livers of embryos treated with PFHxA developed abnormally and were unusually large (Fig. 3A). We also observed enlarged livers in tadpoles exposed to PFHxA although these effects were more pronounced in the presence of PFHpA (Fig. 2A). These results indicated that PFHxA and PFHpA induced liver defects and should be regarded as developmental toxicants and teratogens.

The heart-specific marker, NKX2.5, was used to examine the cardiovascular effects of PFC exposure. The amphibian

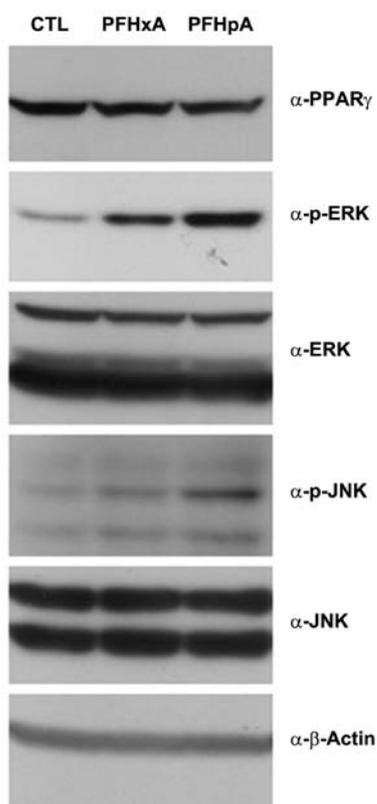


Figure 4. PFCs may induce liver toxicity by modifying ERK and JNK phosphorylation. *Xenopus* embryos were exposed to 130  $\mu$ M PFHxA or PFHpA. Total protein was isolated and analyzed by western blotting with antibodies raised against PPAR $\gamma$ , p-ERK, ERK, p-JNK, JNK, or  $\beta$ -actin. PFHxA and PFHpA treatment activated PPAR $\gamma$  and ERK/JNK phosphorylation. PFCs, perfluoroalkyl compounds; ERK, extracellular signal-regulated kinase; JNK, c-Jun N-terminal kinase; PFHxA, perfluorohexanoic acid; PFHpA, perfluoroheptanoic acid; PPAR $\gamma$ , peroxisome proliferator-activated receptor- $\gamma$ .

heart is composed of one ventricle, surrounded by atria on both sides, separated by a septum (22). Embryos treated with PFHxA and PFHpA showed enlarged atria and a loss of the atrial septum (Fig. 3B). Hearts from embryos exposed to either PFHxA or PFHpA were smaller and contained no trabeculae. Additionally, the pericardiac cavity was expanded and the atrial and ventricular walls were thinner than those observed in the control embryo hearts, which contained prominent trabeculae and the atria and ventricle were separated (Fig. 3B). This effect was more prominent in embryos

exposed to PFHpA, which reduced trabeculation of the ventricle as well as the thickness of the atrial and ventricular walls (Fig. 3B). The results showed that PFHxA and PFHpA treatment caused abnormal heart development.

*PFC exposure alters tissue-specific mRNA expression in vivo.* To investigate the effects of PFC exposure on the liver and heart further, the mRNA expression levels of three liver, heart, and intestine markers were determined by RT-PCR analysis. Previously, PFCs were shown to be tissue-specific toxicants (14,23,24). For this study, we exposed stage 36 embryos to 130  $\mu$ M PFHxA or PFHpA, a concentration that induced the development of various malformations, rather than embryonic lethality. As shown in Fig. 2C, mRNA expression of xPTB was considerably reduced in PFHpA-treated embryos, as compared to the control or PFHxA-treated embryos (Fig. 3A and C). Previous findings have also shown reduced xPTB expression following exposure to various PFCs (16,25,26). Furthermore, the heart-specific marker, NKX2.5, was used to examine the effects of PFCs on the heart. As shown in Fig. 2B and C, mRNA expression of the heart-specific marker, NKX2.5, was reduced in the PFHpA-treated embryos (Fig. 3B and C). NKX2.5 is critical for vasculogenesis and angiogenesis (27). We also assessed the mRNA levels of an intestinal marker, Cyl18, which is mainly expressed in the presumptive small intestine of embryos at the tailbud stage. As shown in Fig. 2C, we observed no significant effects of either PFHxA or PFHpA exposure on Cyl18 expression. These results suggested that PFHpA negatively affected liver and heart development (Fig. 3C).

*PFCs may induce liver toxicity by modifying ERK and JNK phosphorylation.* The roles of peroxisome proliferator-activated receptor- $\gamma$  (PPAR $\gamma$ ), ERK, and JNK pathways in mediating the developmental toxicity associated with exposure to PFCs remain to be elucidated. To investigate the effects of PFCs on PPAR $\gamma$ , ERK, and JNK pathways, stage 36 embryos were exposed to 130  $\mu$ M PFHxA or PFHpA and then analyzed by western blotting with anti-PPAR $\gamma$ , phosphorylated-ERK, ERK, p-JNK, JNK, and  $\beta$ -actin antibodies. PFHpA strongly induced ERK and JNK phosphorylation while PPAR $\gamma$  expression remained the same, which in turn activated signaling that caused liver toxicity, known as hepatocarcinogenesis (Fig. 4). These results clearly demonstrated that the observed developmental defects in the liver were associated with the PFHpA-mediated activation of ERK/JNK pathways.

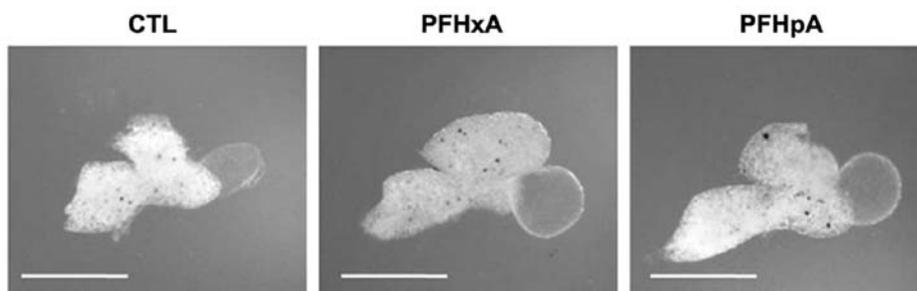


Figure 5. PFCs treatment induced hepatomegaly. Livers of tadpoles exposed to PFHxA or PFHpA. Livers of embryos treated with PFHxA and PFHpA were swollen, with severe morphological changes that were not observed in the control embryos. PFCs, perfluoroalkyl compounds; PFHxA, perfluorohexanoic acid; PFHpA, perfluoroheptanoic acid.

*PFC treatment induces hepatomegaly.* Hepatomegaly is often associated with chronic inflammatory liver disease. The liver is the major metabolizing organ and it is targeted following PFC exposure (28). Previous findings have shown that exposure to PFCs caused various chronic abnormalities, including hepatocellular hypertrophy and hepatomegaly (29,30). To investigate the effects of PFHxA and PFHpA on the liver, livers from 10-day old tadpoles that had been continuously exposed to 130  $\mu$ M PFHxA or PFHpA exhibited hepatomegaly (Fig. 5). These results suggested that high levels of exposure to PFHpA or PFHxA may induce hepatic disorders.

## Discussion

PFCs have emerged as persistent organic pollutants in the ecosystem and human exposure to these may increase the risk of developmental abnormalities (31). Several studies have examined the toxicity of different PFC in many aquatic and other animals (24,32,33). These studies have confirmed that exposure during particular developmental stages or under certain nutritional conditions produced adverse effects on neonates (34,35). Amphibians are more sensitive to environmental exposures during the fetal stage. The results of that study showed that the PFCs tested induced malformations in *Xenopus* embryos, including pericardial edema and hepatic defects. The industrial production of PFCs and their derivatives has been banned because of their toxicity and persistence in the environment. Given these conditions, developmental toxicity studies of PFCs remain in their infancy and further large-scale investigations are required.

*Xenopus laevis* is an amphibian species considered to be a suitable model for studying developmental defects induced by toxicants. In the present study, we conducted a FETAX assay using *Xenopus laevis* to examine the developmental toxicities of PFHxA and PFHpA. The FETAX assay provides a classical approach to detecting teratogenic effects of chemicals on fertilized *Xenopus* embryos in the middle of the blastula stage (17). When *Xenopus* embryos were exposed to C6-7 PFCs, various malformations and toxicity were observed, such as abnormal development, bent and uneven body axis deformation, multiple edemas, growth inhibition, and intestinal miscoiling (Fig. 2A). The PFCs tested produced different TI values, showing that the intensity of their effects on development were different, although they may operate via identical mechanisms.

Our results have demonstrated that PFHxA and PFHpA acted as developmental toxicants and teratogens, thereby producing lethal effects in *Xenopus* embryos. The difference between their effects may reflect the variation in the number of fluorinated carbons present in these compounds. In addition, PFHxA and PFHpA considerably modulated the expression levels of various tissue-specific genes that were relevant to the observed external and internal phenotypes. In particular, xPTB was used as a liver-specific marker and the protein encoded by this gene complexes with the LDL receptor to mediate hepatic LDL removal by endocytosis (35,36). Consistent with other reports, the xPTB mRNA levels were considerably modulated following exposure to either PFHxA or PFHpA (16). This specific reduction in the xPTB mRNA level may cause increased LDL accumulation in the liver, which can in turn lead to chronic cardiac disease, such as atherosclerosis and

myocardial infarction (37). Consistent with this, individuals with a high concentration of PFCs in their blood are more susceptible to LDL-associated adverse effects (5).

The amphibian heart develops from the mesoderm and is considered an essential organ for embryonic survival. During embryogenesis, NKX2.5 acts as a key transcription factor, regulating the transcription of downstream targets that are essential for heart formation (38,39). Studies have shown that NKX2.5 has an important role in the formation of the septum, which separates the ventricle from the atrium (15,38,39). Severe heart abnormalities such as atrial septal defects have been reported in association with the NKX2.5 mutation and downregulation. PFHxA and PFHpA treatments reduced the expression of NKX2.5 mRNA, which is critical for vasculogenesis and angiogenesis (32). Thus, PFCs may cause improper septum formation and impair cardiac function by downregulating the expression of NKX2.5. By contrast, no major change was observed in the intestine-specific marker, Cy118, following treatment with PFHxA and PFHpA.

Compelling evidence for PFC toxicity has emerged from pre-clinical and clinical studies (12,18). Activation of JNK and ERK pathway causes toxic effects that include hepatocarcinogenesis. Livers from PFHxA- and PFHpA-treated embryos exhibited hepatomegaly. This developmental and teratogenic effect was more pronounced in the presence of PFHpA, which may be due to its longer fluorinated carbon chain.

In conclusion, our results confirm that PFHxA and PFHpA are developmental toxicants and teratogens in *Xenopus*. In addition, PFHpA induced more severe pericardial edema, blisters on the dorsal fin, a curved body axis, lack of eye pigmentation, and disturbed facial cells. Furthermore, PFHpA produced more severe effects on liver and heart development through the induction of ERK and JNK phosphorylation, and exposure to elevated levels of PFHpA may therefore cause developmental toxicity and teratogenicity, inducing hepatic and cardiac disorders.

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