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AMERICAN LUNG ASSOCIATION®
of Pennsylvania

Comments of
Kevin M. Stewart
Director of Environmental Health
American Lung Association of Pennsylvania
regarding the
Pennsylvania Clean Vehicles Program
Regulation #7-398, (#2523)
before the
Environmental Quality Board
March 20, 2006
Harrisburg, Pennsylvania

Good evening. On behalf of the American Lung Association of Pennsylvania, I thank the Environmental Quality Board (EQB) for the invitation to submit comments today. We represent not only well over a million Pennsylvanians who suffer from chronic lung disease, but also the millions more who desire to breathe clean air and so protect their good health.

Founded over a century ago to fight tuberculosis, the Lung Association is the oldest voluntary health organization in the Commonwealth. Now we are dedicated to the prevention of lung disease and the promotion of lung health, a mission we carry out through education, community service, advocacy, and research. The Lung Association represents a body of scientific knowledge on the subject of lung disease, specifically on well-established links between air pollution and lung disease, both as a cause of new cases and as an exacerbating agent for existing conditions.

My name is Kevin Stewart. I hold a Bachelor of Science degree in chemical engineering from Princeton University, and I serve the American Lung Association of Pennsylvania as its director of environmental health.

Let me begin by stating our position clearly: The American Lung Association of Pennsylvania supports the Proposed Rulemaking on the Pennsylvania Clean Vehicles Program currently before the EQB (Regulation #7-398 (#2523)), as published in *The Pennsylvania Bulletin* on February 11, 2006 at 36 Pa.B. 715. Specifically, we agree with the three primary purposes of this rulemaking:

1) To postpone the compliance date from model year 2006 to model year 2008.
– Although the Lung Association's preference is that the rule would already have been in effect and that this hearing would have been largely moot, we recognize the value of this delay in establishing a consistent approach across many northeastern states that will assist vehicle manufacturers more smoothly achieve compliance.

2) To include a three-year early credit-earning period that will
   a) provide flexibility for the vehicle manufacturers during the implementation period, and
   b) help ensure that the regulation meets the necessary "identicality" requirements of the Federal Clean Air Act.

– The Lung Association is satisfied that these steps are appropriate to promote effective and comprehensive implementation of the Pennsylvania Clean Vehicles Program; and

3) To *update*, in the Pennsylvania Clean Vehicles Program, incorporation by reference to the California Low Emission Vehicles II (LEV II) Program contained in the California Code of Regulations. – The Lung Association recognizes, as do both the Commonwealth and the U. S. Environmental Protection Agency (EPA), that the LEV II Program, as understood to include neither Zero Emission Vehicle (ZEV) nor California fuel requirements, is *currently the legally effective program for Pennsylvania.*

Today, we are urging the members of the EQB to reject any proposals that would prevent the Commonwealth from implementing the Pennsylvania Clean Vehicles Program, as understood to include the LEV II Program described above, but rather to accept this proposed regulation as a sensible and appropriate step to help the Commonwealth move forward in the implementation of established policy. Here's why:

Poor Air Quality

Pennsylvania has one of the nation's most serious air pollution problems. Ozone smog and fine particle pollution are perhaps the two worst air pollutants plaguing the Commonwealth, leading to the exacerbation of respiratory and cardiovascular problems, including premature mortality in high risk groups and triggering hundreds of thousands of asthma attacks each year in Pennsylvania. According to our most recent *State of the Air* report, twelve Pennsylvania metro areas ranked among the worst fifty nationwide for ground level ozone, or microscopic particle pollution, or both. And seven of these metro areas ranked among the worst twenty-five for at least one pollutant.

While air quality has certainly been improving over the past three decades, it is *far* from being "very, very clean," as some have claimed. In 2003 and 2004, much of the improvement we experienced was associated with weather less favorable to the production of ozone smog. Last year's record of Air Quality Action Days and health advisory days is sufficient to show that our problem is far from being solved. Indeed, thirty-seven PA counties are in nonattainment for the national ozone smog standard, counties that comprise about five-sixths of the Commonwealth's population. Seventeen of these counties are also in nonattainment for the national standard for deadly fine particle pollution; parts of four counties contribute to the problem.

And in recognition of nearly a decade's worth of additional scientific study, the air quality standards need to be tightened – the current standards are inadequate to protect public health. In short, while we've made progress, we need to do much more to improve air quality.

Scientific Evidence for Adverse Health Effects

We are compelled to point out that the nonattainment status of more than half the counties in the Commonwealth has been determined with respect to what was once the "new" national air quality standards for ozone and particle pollution, standards that were promulgated by EPA in 1997. Those standards were established based on the *weight of the evidence* of what was known to medicine and science *at that time,* with findings that adverse consequences of exposure to elevated air pollution levels ranged from more lost school and work days, more frequent and severe asthma attacks, and increased use of medication, to irreversible lung damage, more hospitalizations, more emergency department visits, and more deaths.

In what has been nearly a decade since 1997, it is fair to state that *thousands* of new studies on the health and environmental effects of ozone and particle pollution have been published in peer-reviewed literature. Examples of some of the conclusions of studies released *after* the establishment of the 1997 standards include:

- Long-term exposure to ozone is related to asthma *development.*
- Ozone plus allergens exacerbates asthma.
- For adults with severe asthma, particle pollution and ozone combined worsened patients' health, and nitrogen dioxide and ozone combined increased their risk of death.
- Children with even mild asthma suffer adverse effects from air pollution.
- Even low levels of ozone contribute to hospitalization for respiratory disease.
- Inner-city asthmatic children born prematurely or with low birth weight have the greatest response to ozone.
- During the four-month period of the Summer Olympics in Atlanta, peak traffic decreased by 22%, ozone levels were reduced by 28%, and asthma incidents declined in children by as much as 44% in emergency room visits, and by 19% for hospitalizations.
- In the largest study of its kind as of 2000, increases in particle pollution and ozone were found to heighten the risk of pediatric emergency department visits for acute asthma "*at pollution levels that commonly occur in many U.S. cities.*"
- In landmark ongoing research on the chronic effects of air pollution in children,
    - Children living in high ozone communities who actively participated in several sports were more likely to develop asthma than children in these communities not participating in sports.
    - Children with asthma who were exposed to higher concentrations of particulate matter were much more likely to develop bronchitis.
    - Days with higher ozone levels resulted in significantly higher school absences due to respiratory illness.
    - Children living in communities with higher concentrations of acid vapor, ozone, nitrogen dioxide and particulate matter have significantly reduced lung growth and development. This decreased lung development may lead ultimately to increased risk of chronic respiratory illness in adulthood.
    - Children living nearer high-traffic areas were more likely to have developed asthma.

I have also provided the EQB with our recent report of *2005 Research Highlights*. I stress that this report, like the preceding litany of research conclusions, only scratches the surface of the extensive research being currently undertaken. A few examples from the current report are:

- Extended analysis of the landmark Harvard Six Cities study demonstrates that decline in average annual fine particle pollution levels, including to concentrations below the current air quality standard of 15 micrograms per cubic meter, leads to a reduction in risk for deaths due to cardiovascular and respiratory disease.
- Spatial analysis of fine particle pollution and mortality – i.e., use of a finer-toothed comb – shows that risk of premature mortality was nearly three times greater than was previously reported.
- Diabetics were found to be more vulnerable to particle pollution.
- Three independent research reviews (done at Yale, Harvard, and NYU) separately confirmed that increases in daily average ozone levels were linked with an increased risk of death, by up to nearly one percent for each 10 parts per billion of ozone, not only on the day of exposure but for up to two days following, even when controlled for confounders such as particle pollution and temperature.
- And many studies linking ozone air pollution with effects as disparate as heart arrhythmias, heart attacks, reduced birth weight, difficulty breathing (from exposure at levels below the current standard) among infants of asthmatic mothers, retardation of lung function growth, reduction in acute lung function among mail carriers at exposures below current standards, …

Again, I stress, this review merely scratches the surface of what is known about the health effects of air pollution.

Mobile Sources and LEV II Results

Mobile sources contribute about a third of the emissions in Pennsylvania. No other sector produces as much. Pollutants include ozone precursors volatile organic compounds (VOCs) and nitrogen oxides (NO_x), as well as fine particles, toxic and carcinogenic compounds. Keeping the Pennsylvania Clean Vehicles Program – that is, implementing the LEV II Program in Pennsylvania without adopting California's ZEV requirements or fuel standards – will provide significantly greater reductions in air pollution than would adopting the federal Tier II standards.

The American Lung Association of Pennsylvania estimates that over the period from 2010 through 2025, by which time full fleet turnover is expected, the Pennsylvania Clean Vehicles Program would prevent ozone precursors from being emitted in the amount of about 50,000 tons, and perhaps as much as 80,000 tons, beyond the reductions achievable by the federal vehicle standards. Of course, the benefits would continue in the amount of 6,000 tons and up to 9,000 tons for every year beginning with full implementation.

Benefits of Emission Reductions

The more that both VOC and NO_x emissions are decreased, the more we can expect to see decreased ozone air pollution levels. And there are millions of Pennsylvanians now at increased risk from exposure to air pollution who stand to benefit. It is my duty today to remind all of us that the reason we *have* air pollution control programs is that first and foremost, air pollution worsens and causes disease and even death for real people. In Pennsylvania, the populations at increased risk from air pollution include:

- 2.8 million infants, children, and youth under 18
- 1.9 million persons 65 and above
- 1 million individuals with asthma
- 400 thousand with chronic bronchitis
- 160 thousand with emphysema
- 3.4 million with heart disease
- 680 thousand with diabetes
- one-half million to 2 million persons who are found to be "responders" and who experience much larger declines in lung function than the population in general
- millions more who work or exercise outdoors.

Of course, some individuals are included in two or more of these categories, but a little analysis soon makes it plain that about half of the Commonwealth's population are described by at least one of these categories, so it is very probable that your family is home to one of these individuals, and practically certain if we expand "family" to include grandparents to cousins. That's why I must implore you to keep two things in mind:

- Every one of these millions is a real person, not a nameless statistic. Every one of these people is a family member, a neighbor, a coworker, a friend. Take a moment to remember those whom you know personally. Now multiply that to cover over 12 million Pennsylvanians.
- Asthma and other chronic lung diseases are potentially serious illnesses; among people who have them, air pollution can result in an increased need for medication, hospitalization, and even death. I ask you to think about your own last respiratory episode or one you witnessed when someone couldn't easily catch his or her next breath. Put yourself in that person's place, or into the place of the parent of a child with such an illness. Now ask yourself if that six to nine thousand tons a year of air pollution really ought to be allowed to go into the air.

Costs

... The vehicles

Vehicles meeting the California LEV II standards have been on the market in five states since the 2004 model year. As DEP has demonstrated and as auto industry representatives have confirmed, there is presently no discernible cost difference between federal vehicles and those of the Pennsylvania Clean Vehicles Program.

You've certainly heard various estimates for what the Pennsylvania Clean Vehicles Program will add to the sticker price of new cars when it is fully implemented, $3,000 being the number most frequently given. We must, however, remind you that it is clear from the history of regulation of the auto industry, that such estimates invariably significantly overstate the costs that consumers experience when such air pollution control measures are actually implemented.

Based strictly on the persistent historical pattern of such overestimates of the costs of implementation, to some extent by government regulators, but most significantly by the auto industry and its allies, we would not be surprised to find, when the new car standards are fully implemented ten years from now, that implementation costs would be in the ballpark of only $600 or $700 per vehicle. These are incremental initial costs on the order of 2%, a ratio, by the way, in the same range that some in this debate have claimed is small enough not to make a difference.

Indeed, we have seen no evidence that the public's buying habits are sensitive to so small an increase in initial vehicle cost. What is more, using the now quite conservative assumption that gasoline would cost $1.74 per gallon, the California Air Resources Board found that the increase in purchase price would be more than offset by savings in fuel expenditures over the life of the vehicle.

The inescapable conclusion here is that these air quality benefits are FREE. Even figuring in the additional initial vehicle cost, the true cost-effectiveness witnessed by the consumer over the life of the vehicle – i.e., how much additional money they're spending to reduce air pollution – will be negative (that's a savings) compared to the federal Tier II program. In air pollution control, deals like these don't come along all the time. Is it any wonder, then, that states whose vehicular emissions are a major component of their air pollution problems, and whose populations together comprise about one-third of the country's total, are willingly choosing this option?

... Health-related costs

I must remind the Board that even as we deliberate, we are all already paying, as a part of our medical expenses, insurance payments, and economic costs, for our failure to control air pollution. The serious consequences of outdoor air pollution in Pennsylvania, about one-third of which originates in the transportation sector, result in a total loss on the order of one billion dollars per year. Estimates range from a low of about $500 million to as much as several billion dollars. Over half of this total is accounted for by health care costs. The remainder includes costs due to lost work, lost productivity, and premature mortality. Estimates for premature mortality among Pennsylvanians due to air pollution range from about 2,000 a year to about 5,000. Of course, the preceding does not begin to characterize the human suffering involved.

In contrast, analysis shows that costs to the economy of implementing pollution control measures such as scrubbers on power plants, vehicle inspection and maintenance programs, and yes, even costs, if any, that are associated with clean car programs, are recouped many times over in savings in health, work, and lives.

Conclusion

The American Lung Association of Pennsylvania asks the Board not to underestimate the interest in and the importance of having the strongest vehicle emission standards available. We strongly encourage the EQB to support the Pennsylvania Clean Vehicles Program, and to reject any proposal that would have the effect of preventing its implementation. Please continue to give this issue your full and deliberate attention. We have encouraged a process that is both open and sober so that all parties can evaluate this issue with the full picture before it. Respect for our citizens requires it. The public's health demands it.

The American Lung Association of Pennsylvania appreciates the opportunity to contribute to the public deliberations on this matter and is pleased to respond to questions. For further information, please contact:

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Appendix: A few additional notes…

… on the status of the California Low Emission Vehicle program in Pennsylvania law:

We would simply cite EPA Region III Administrator Donald Welsh's December 2, 2005 communication in response to an inquiry by Chairman Richard Geist of the House Transportation Committee: "…because Pennsylvania's acceptance of [the National Low Emission Vehicle program] continued only up to the 2006 model year, it is our opinion that the [California Low Emission Vehicle] program is no longer a 'backstop,' but is the legally effective program for Pennsylvania. It is also our opinion that the Pa. Clean Vehicle Program is a 'federally enforceable part of the SIP,' as was represented by Secretary Biehler in his correspondence with you."

…on the question of sovereignty:

We refuse to believe that the legislatures and the executive branch officials of New York, New Jersey, Maine, Vermont, Massachusetts, Connecticut, Rhode Island, Washington and Oregon have all been hoodwinked or have all taken leave of their senses. Could it be that they know something some are refusing to see? – that California LEV II vehicles, both for fuel economy and for air quality, are a good deal for their citizens, too?

Indeed, given that states, not the federal government, have the primary responsibility under the Clean Air Act to meet the ambient air quality standards, the right of states to choose to adopt the California standards is a necessary feature of states' sovereignty. In this matter, the

Commonwealth has strongly defended its ability to protect the health of its citizens, and rightly has refused to be limited to the federal Tier II vehicle standards as its sole choice.

... on the ozone standard:

As a consequence of an American Lung Association lawsuit, the EPA has been required to abide by the law and is in the process of reviewing the national air quality standard for ground-level ozone. Although a decision is not required until the end of next year, the first draft of the EPA staff paper released on Nov. 14 not only

- confirmed that meeting the current 0.08 ppm ozone standard "would likely result in substantial reductions in exposures of concern and associated risks of serious health effects,"

but also

- found that "small but significant effects estimates have been reported" from exposures as low as 0.06 ppm," and
- called for assessments of potential standards set over the range of 0.06 to 0.08 ppm.

I would remind the Board that the federal Clean Air Act requires that ambient air quality standards be set solely according to the criterion that they "protect the public health and provide an *adequate margin of safety.*" Clearly, demonstrations of adverse health effects at levels below the current standard mean that the standard is inadequate and must be improved, something you can count on the Lung Association to fight to achieve. In this context, taking away one of the tools that we know works – that is, preventing the Pennsylvania Clean Vehicles Program from coming into effect – is just going to make the job of achieving future, more stringent standards that much harder.

...on the American Lung Association of Pennsylvania's support of Tier II standards during the Ozone Stakeholder processes conducted in south central Pennsylvania and in the Lehigh Valley - Berks region:

The California LEV II standards were not on the table when those stakeholder groups met. Those standards were developed later. At one time, Tier II was the best standard on the choice of options. That is no longer the case. Very simply, the LEV II standards are a stronger rule than the Tier II standards, and we see no compelling reason not to take advantage of the opportunity to clean the air even more.

AMERICAN LUNG ASSOCIATION.

January 30, 2006

2005 RESEARCH HIGHLIGHTS: HEALTH EFFECTS OF PARTICULATE MATTER AND OZONE AIR POLLUTION

This annotated bibliography presents brief summaries of selected research papers published in 2005 (or in press in January 2006) on the health effects of particulate and ozone air pollution. Some of the highlights of the new studies include:

- A long-term study showing risk of premature death attributable to PM is three times greater than previously reported;
- Studies linking daily exposures in PM with increased hospital admissions for strokes, congestive heart failure, heart attacks, COPD and other respiratory problems;
- A toxicology study showing links between exposure to $PM_{2.5}$ at levels near or below the current standards and development of atherosclerotic plaques;
- Many studies elucidating the biological mechanisms and pathways for cardiovascular effects;
- Studies linking prenatal exposure to air pollution with increased risk of low birth weight, preterm birth, infant mortality, and cancer;
- Research showing that coarse particles exacerbate respiratory disease;
- Three meta-analyses linking ozone air pollution with premature mortality and a multi-city study showing that effects are not due to temperature;
- Intervention studies showing that reductions in air pollution yield measurable improvement in children's respiratory health and reduction in premature deaths; and
- Policy analyses showing the need for strong annual and daily fine particle standards to protect susceptible populations and provide equivalent levels of protection to different regions of the country.

These summaries are in no way intended to substitute for medical information from a physician, nor are they intended to represent conclusions of the American Lung Association. Citations for all studies are provided.

PARTICULATE MATTER: *HEALTH EFFECTS OF SHORT-TERM EXPOSURES*

Premature Deaths

Fine Particles Linked to Daily Mortality in California

This multi-city study investigated associations between $PM_{2.5}$ and mortality in nine heavily populated California counties taking advantage of new $PM_{2.5}$ monitoring data collected from 1999 through 2002.

Statistical methods were used to control for effects of season, temperature, and humidity. The nine counties were: **Contra Costa, Fresno, Kern, Los Angeles, Orange, Riverside, Sacramento, San Diego, and Santa Clara**. Positive associations were observed with all-cause mortality, as well as mortality from respiratory disease, cardiovascular disease, and diabetes, and deaths in persons over age 65. *"Overall, this large, multi-county analysis provides evidence of significant associations of fine particles with daily mortality among nearly two-thirds of California's population,"* conclude the authors.

Ostro B, Broadwin R, Green S, Feng W-Y, Lipsett M. Fine Particulate Air Pollution and Mortality in Nine California Counties: Results from CALFINE. *Environ Health Perspec* 2006; 114:29-33. http://ehp.niehs.nih.gov/members/2005/8335/8335.pdf

Linear Relationship between PM and Mortality Reported in Multi-City European Study

This study investigated the exposure-response relationship in the **22 European cities** participating in the APHEA (Air Pollution and Health--A European Approach) project. The study concludes that the association between ambient particles and mortality can be adequately estimated using the linear model, confirming results previously reported for Europe and the United States. Different statistical models were used to explore the potential for a threshold level at 20 and 10 $\mu g/m^3$ but the linear models assuming no threshold gave a better fit. Thresholds for effects in individuals and subgroups may differ according to their sensitivity, and cannot be identified with the methodology used in this study. Investigators believe that measures focusing on lowering annual average pollution concentrations will have greater public health benefits than those focusing on a few days with the highest concentrations.

Samoli E, Analitis A, Touloumi G, Schwartz J, Anderson HR, Sunyer J, Bisanti L, Zmirou D, Vonk JM, Pekkanen J, Goodman P, Paldy A, Schindler C, Katsouyanni K. Estimating the Exposure-Response Relationships between Particulate Matter and Mortality within the APHEA Multicity Project. *Environ Health Perspect* 2005; 113:88-95. http://ehp.niehs.nih.gov/members/2004/7387/7387.pdf

Hospital Admissions and Emergency Room Visits

Air Pollution Linked to Ischemic Strokes

Particulate air pollution (PM_{10}) increases the risk for ischemic strokes -- those caused by a blood clot -- according to a study by Harvard University researchers. Hemorrhagic strokes, which occur when a blood vessel ruptures, were not affected by the level of pollution.

Researchers evaluated the link between daily levels of PM_{10} and hospital admission for strokes among Medicare recipients in nine U.S. cities: **Birmingham, Chicago, Cleveland, New Haven, Detroit, Minneapolis, Pittsburgh, Salt Lake City, and Seattle**. Compared to days with relatively low particulate air pollution levels, the risk of ischemic stroke -- the most common type of stroke -- was 1 percent higher on days with relatively higher air pollution. Similar associations were observed for carbon monoxide, nitrogen dioxide and sulfur dioxide.

"It appears that air pollution has only a small effect on acute ischemic events of either the heart or brain, but everybody in those cities is exposed. So, while the relative risk may be small, the absolute risk in terms of excess number of strokes can be quite high, especially when you realize that someone in the United States has a stroke every 45 seconds," according to the study authors.

Wellenius GA, Schwartz J, Mittleman MA. Air Pollution and Hospital Admissions for Ischemic and Hemorrhagic Stroke Among Medicare Beneficiaries. *Stroke 2005*; 36:2549-2553. http://www.ncbi.nlm.nih.gov/entrez/query.fcgi?cmd_=Retrieve&db=PubMed&list_uids=16254223&dopt=Citation

Low Levels of PM Trigger Hospital Admissions for Congestive Heart Failure

PM_{10} concentrations below current EPA standards are associated with an increased rate of hospital admissions for congestive heart failure, in a study of seven U.S. cities. The cities studied were **Chicago, Detroit, Cleveland, New Haven, Minneapolis, Birmingham, and Seattle**. Researchers evaluated the association between daily levels of PM_{10} and the rate of hospitalization for congestive heart failure in Medicare recipients (aged 65 or older). Overall, a 10 $\mu g/m^3$ increase in PM_{10} was associated with a 0.72% increase in hospital admissions on the same date. Researches conclude *"these results support the hypothesis that elevated levels of particulate air pollution, below the current limits set by the United States Environmental Protection Agency, are associated with an increase in the rate of hospital admission for exacerbation of CHF [congestive heart failure]."*

A related study in **Pittsburgh, Pennsylvania** evaluated the association between ambient air pollution and the rate of hospitalization for congestive heart failure among Medicare recipients in **Allegheny County** from 1987 to 1999. The study looked at 55,000 patients admitted with a primary diagnosis of congestive heart failure. PM_{10}, carbon monoxide, nitrogen dioxide and sulfur dioxide, but not ozone, were positively and significantly associated with the rate of admission on the same day, with the strongest associations observed with CO, nitrogen dioxide, and PM_{10}. The associations with carbon monoxide and nitrogen dioxide were the most robust in two-pollutant models. The results suggest that short-term elevations in air pollution from traffic-related sources may trigger acute cardiac decompensation in heart failure patients.

Wellenius GA, Schwartz J, and Mittleman MA. Particulate Air Pollution and Hospital Admissions for Congestive Heart Failure in Seven United States Cities. *Am J Cardiol* 2006; in press. http://www.ajconline.org/article/PIIS000291490501831X/abstract

Wellenius GA, Bateson TF, Mittleman MA, Schwartz J. Particulate Air Pollution and the Rate of Hospitalization for Congestive Heart Failure among Medicare Beneficiaries in Pittsburgh, Pennsylvania. *Am J Epidem* 2005; 161:1030-1036. http://aje.oxfordjournals.org/cgi/content/abstract/161/11/1030

Particulate Pollution Increases Risk of Heart Attacks in the Elderly

This multi-city study examined the association between PM_{10} and emergency hospitalization for heart attacks among elderly residents of 21 U.S. cities. Researchers obtained Medicare data on hospital admissions for 300,000 heart attacks over a 14-year period. The statistical model was able to control for possible confounding by weather.

The cities studied were **Birmingham, AL, Boulder, CO, Canton, OH, Chicago, IL, Cincinnati, OH, Cleveland, OH, Colorado Springs, CO, Columbus, OH, Denver Co, Detroit MI, Honolulu HI, Houston, TX, Minneapolis, MN, Nashville, TN, New Haven, CT, Pittsburgh, PA, Provo/Orem, UT, Salt Lake City, UT, Seattle, WA, Steubenville, OH, and Youngstown, OH.**

Overall, there was a small association between daily PM_{10} concentrations and increased risk of hospital admission for heart attack. The risk doubled for those with a previous admission for COPD. The relationship was nearly linear, but risks increased most sharply at daily concentrations less than 50 $\mu g/m^3$.

Zanobetti A, Schwartz J. The Effect of Particulate Air Pollution on Emergency Admissions for Myocardial Infarction: A Multicity Case-Crossover Analysis. *Environ Health Perspec* 2005; 113:978-982. http://ehp.niehs.nih.gov/members/2005/7550/7550.pdf

Children's Hospital Admissions Spike with Increases in Outdoor Air Pollution

A large-scale epidemiological study of respiratory hospital admissions in children was carried out in the

largest cities in Australia and New Zealand: **Brisbane, Canberra, Melbourne, Perth, Sydney, Auckland and Christchurch**. Positive associations were observed for $PM_{2.5}$, PM_{10}, nitrogen dioxide and sulfur dioxide, for hospital admissions for pneumonia and acute bronchitis, respiratory disease and asthma. The study found strong and consistent associations between various measures of outdoor air pollution and short-term increases in childhood hospital admissions. These changes were distinct from temperature effects.

Barnett AG, Williams GM, Schwartz J, Neller AH, Best TL, Petroeschevsky AL, Simpson RW. Air Pollution and Child Respiratory Health: A Case-Crossover Study in Australia and New Zealand. *Am J Resp Crit Care Med* 2005; 171:1272-1278. http://ajrccm.atsjournals.org/cgi/content/abstract/171/11/1272

Respiratory Emergency Department Visits Rise with Peaks in Air Pollution

A time-series study of a very large database of 4 million emergency department visits to 31 hospitals in **Atlanta** has shown positive relationships between various air pollutants and respiratory disease. In single-pollutant models examining 3-day moving averages of pollutants, visits for upper respiratory infections in infants and children were positively associated with PM_{10}, ozone, nitrogen dioxide and carbon monoxide. The association with ozone persisted in multipollutant models. Chronic Obstructive Pulmonary Disease visits were positively associated with nitrogen dioxide and carbon monoxide, while pneumonia was linked to $PM_{2.5}$ and organic carbon.

Peel JL, Tolbert PE, Klein M, Metzger KB, Flanders WD, Todd K, Mulholland JA, Ryan PB, Frumkin H. Ambient Air Pollution and Respiratory Emergency Department Visits. *Epidemiology* 2005; 16:164-174. http://www.epidem.com/pt/re/epidemiology/abstract.00001648_-200503000-00004.htm;jsessionid=DJ9K9yXFReTxbygYxCfQ8H2i6Px8bV2VPwblNhCEAMBDOBo6mjRK!400681292!-949856144!9001!-1

Respiratory Effects

Air Pollution Lowers Lung Function of Asthmatic Children in Detroit

African-American and Latino children on corticosteroid asthma maintenance medication or with upper respiratory infections are adversely affected by current levels of air pollution, according to a study of primary school age children with asthma in **Detroit**. The study explored the relationship between lung function and ambient levels of ozone and two measures of particulate matter -- PM_{10} and $PM_{2.5}$.

The study tracked 86 children in six 2-week seasonal assessments from winter 2001 through spring 2002. Two measures of lung function were measured -- peak flow, and forced expiratory volume in 1 second (FEV_1). For children on corticosteroids for their asthma, PM_{10} and 8-hour peak ozone were both associated with poorer lung function two days after exposure. For children with symptoms of respiratory infection, $PM_{2.5}$ and PM_{10} were associated with poorer lung function 3-5 days after exposure, while 8-hour peak ozone concentrations were associated with poorer lung function after 1-2 days.

"Our results emphasize the continued need for enforcement of existing standards as well as the importance of considering susceptible subgroups within the population when formulating new standards," concluded the University of Michigan researchers.

Lewis TC, Robins TG, Dvonch JT, Keeler GJ, Yip FY, Mentz GB, Lin X, Parker EA, Israel BA, Gonzalez L, Hill Y. Air Pollution-Associated Changes in Lung Function among Asthmatic Children in Detroit. *Environ Health Perspect* 2005; 113:1068-1075. http://ehp.niehs.nih.gov/members/2005/7533/7533.pdf

Cardiovascular Mechanisms and Effects

Inflammation and Coagulation Responses May Hold Clues to Mechanisms for PM and Coronary Effects

A panel study was conducted to measure the early physiological reactions characterized by blood biomarkers of inflammation, endothelial (the cells that line blood vessels) dysfunction, and coagulation in response to daily changes in air pollution in **Erfurt, Germany**. Blood parameters were measured repeatedly in 57 male patients with heart disease during winter 2000/2001. Hourly measurements were made of ultrafine particles, PM_{10}, $PM_{2.5}$, elemental and organic carbon, gaseous pollutants, and meteorological data at a central monitoring site.

Increased levels of C-reactive protein were observed with an increase in various sizes of particle pollution: ultrafine, fine, and PM_{10}. Clotting factor levels showed no consistent relationship to air pollution.

"This study adds to the evidence that elevated levels of ambient air pollution may cause systemic inflammatory and coagulation responses. These changes in blood markers could represent additional risk factors which in susceptible individuals such as patients with coronary heart disease, could increase the likelihood of serious arterial vascular thrombotic [blood clots in the arteries] events upon exposure to high levels of air pollutants," conclude the authors.

Rucker R, Ibald-Mulli A, Koenig W, Henneberger A, Woelke G, Cyrys J, Heinrich J, Marder V, Frampton M, Wichmann HE, Peters A. Air Pollution and Markers of Inflammation and Coagulation in Patients with Coronary Heart Disease. *Am J Resp Crit Care Med* 2005; Published ahead of print on November 17, 2005. doe:10.1164/rccm.200507 -1123OC http://ajrccm.atsjournals.org/cgi/content/abstract/200507_-1123OCv1

Vehicle Exhaust Contributes to Elevated Blood Pressure

Researchers exposed 23 healthy nonsmoking adults to concentrated ambient fine particles and fine particles plus ozone during 2-hour exposures. Blood pressure and heart rate were measured at 30-minute intervals during the controlled exposures. A significant increase in diastolic blood pressure was observed with the combined ozone and fine particle exposure. Follow-up investigations revealed that the magnitude of blood pressure change is associated with the $PM_{2.5}$ carbon content, leading investigators to suspect that pollution from vehicular traffic in urban centers may be causing cardiovascular adverse effects.

"Exposure to high ambient concentrations of air pollutants may initiate a rapid hypertensive response, thus promoting acute cardiovascular events in susceptible individuals. In conjunction, if this vasopressor response continues unabated, gradients in personal exposure to air pollution could contribute to long-term differences in interindividual blood pressure levels. Continued exposure to air pollution could thereby increase the risk for developing chronically elevated blood pressure and possibly overt hypertension," conclude the researchers.

Urch B, Silverman F, Corey P, Brook JR, Lukic KZ, Rajagopalan S, Brook RD. Acute Blood Pressure Responses in Healthy Adults During Controlled Air Pollution Exposures. *Environ Health Perpect* 2005; 113:1052-1055. http://ehp.niehs.nih.gov/members/2005/77_85/7785.pdf

Diesel Exhaust Impairs Blood Vessels

Breathing diesel exhaust at levels typically found in large cities for as little as an hour can disrupt important blood vessel functions, suggesting a possible mechanism for increased heart attack rates during periods of high air pollution. A chamber study by cardiologists at the University of Edinburgh used a double-blind, randomized, cross-over design to assess the effects of diesel fumes in two important and complementary aspects of vascular function: the regulation of vascular tone and the ability to remove of small blood clots.

Both functions were impaired and are plausibly related to the cardiovascular effects of air pollution.

The study involved 30 healthy, non-smoking men, aged 20 to 38, who were evaluated during two one-hour tests, two weeks apart. During each test, the men were exposed to either clean air or diesel exhaust from an idling engine, while riding a stationary bicycle for 15-minute stretches inside an exposure chamber. Participants received injections of a vasodilator and researchers measured their blood flow rates during and after exposure. The study found that exposure to diesel exhaust during exercise reduced the blood vessels' ability to dilate, or expand, and decreased levels on an enzyme that helps prevent clots from forming in the blood

According to the authors, these findings provide a plausible mechanism linking air pollution to the development of atherothrombosis and heart attacks.

Mills NL, Törnqvist H, Robinson, SD, Gonzalez M, Darnley K, MacNee W, Boon NA, Donaldson K, Blomberg A, Sandstrom T, Newby DE. Diesel Exhaust Inhalation Causes Vascular Dysfunction and Impaired Endogenous Fibrinolysis. *Circulation* 2005; 112:3930-3936.
http://circ.ahajournals.org/cgi/content/abstract/112/25/3930

Air Pollution Thickens the Blood

PM_{10} particles thicken the blood and boost inflammation, according to the results of an experimental study. Scientists tested the inflammatory and blood clotting responses of human lung cells, cells taken from the umbilical cord, and immune cells called macrophages. Each was tested six and 24 hours after exposure to particulate matter. The results showed that levels of clotting factors, which thicken the blood, were elevated in several of the cell types. The rate of death in immune cells also increased significantly, and exposure to PM_{10} boosted inflammatory activity.

Researchers concluded that *"PM_{10} has the ability to alter macrophage, epithelial, and endothelial cell function to favour blood coagulation via activation of the extrinsic pathway and inhibition of fibronolysis pathways."* This suggests a mechanism for the adverse cardiovascular effects caused by inhalation of particulate matter.

Gilmour PS, Morrison ER, Vickers MA, Ford I, Ludlam CA, Greaves M, Donaldson K, MacNee W. The Procoagulant Potential of Environmental Particles (PM_{10}). *Occup Environ Med* 2005; 62:164-171.
http://oem.bmjjournals.com/cgi/content/abstract/62/3/164

Susceptible Populations

Diabetics More Vulnerable to Particulate Air Pollution

A study of 270 diabetes patients in **Boston** used baseline data from earlier clinical trials to compare with 24-hour concentrations for $PM_{2.5}$, sulfates, and black carbon. Pollutant concentrations were evaluated for associations with vascular reactivity. The strongest and most robust finding was the association between sulfate particles, which represent long-range transport from coal-burning power plants, and decreased vascular reactivity.

"Our results link pollution exposure and physiological responses known to be along the pathway of adverse cardiovascular outcomes. We saw significant associations between vascular reactivity and exposure to particulate pollution, especially SO_4 [sulfates], and greater responses among people with diabetes. Higher rates of cardiac hospitalization and mortality on high-particulate days among people with diabetes may be partially explained by impairments in endothelial function, vascular smooth muscle function, and subsequent coronary artery vascular responses," conclude the authors. *"Diabetes confers vulnerability to particles associated with coal-burning power plants and traffic."*

O'Neill MS, Veves A, Zanobetti A, Sarnat JA, Gold DR, Economides PA, Horton ES, Schwartz J. Diabetes Enhances Vulnerability to Particulate Air Pollution-Associated Impairment in Vascular Reactivity and Endothelial Function. *Circulation* 2005; 111:2913-2920.
http://circ.ahajournals.org/cgi/content/abstract/111/22/2913

Prenatal Effects

Air Pollution Increases Risk of Preterm Births

Researchers at the University of North Carolina have reported an increased risk of preterm delivery associated with exposure to PM_{10} and sulfur dioxide during the last 6 weeks of pregnancy. The study population consisted of single infants born to mothers from 1997 to 2001 in four Pennsylvania counties: **Allegheny, Beaver, Lackawanna, and Philadelphia.**

The researchers used a time-series analysis, a study design which eliminates potential confounding by individual risk factors that do not change over short periods of time. The increased risk of preterm birth was small, but researchers note that if the effects are causal, the public health impact could be significant because of the large populations chronically exposed to high concentrations of air pollution.

Sagiv SK, Mendola P, Loomis D, Herring AH, Neas LM, Savitz DA, Poole C. A Time Series Analysis of Air Pollution and Preterm Birth in Pennsylvania, 1997-2001. *Environ Health Perspec* 2005; 113:602-606.
http://ehp.niehs.nih.gov/members/2005/7646/7646.pdf

Prenatal Exposure to Urban Air Pollutants Can Cause Genetic Alterations Linked to Increased Cancer Risk

A study of 60 newborns in **New York City** suggests that prenatal exposure to combustion-related urban air pollutants alters the structure of chromosomes of babies in the womb. Such genetic alterations have been linked to increased risk of cancer in children and adults.

The research involved a sample of infants and their non-smoking mothers in **Harlem, Washington Heights, and the South Bronx** -- three low-income neighborhoods in New York City. The mothers wore personal monitors to measure their exposure to polycyclic aromatic hydrocarbons (PAH) during the third trimester of their pregnancies. Chromosomal aberrations were measured in the cord blood cells and were found to be associated with PAH exposure. *"If confirmed, this finding may open new avenues for prevention,"* concluded the Columbia University researchers.

Bocskay KA, Orjuela MA, Dang D, Liu X, Warburton, DP, Perera FP. Chromosomal Aberrations in Cord Blood Are Associated with Prenatal Exposure to Carcinogenic Polycyclic Aromatic Hydrocarbons. *Cancer Epidemiology Biomarkers & Prevention* 2005; 14:506-511.
http://cebp.aacrjournals.org/cgi/content/abstract/14/2/506 and
http://www.mailman.hs.columbia.edu /ccceh/news-events/Chromosomal_Aberrations_Final_2_-15-05.pdf

Maternal Exposure to Environmental Pollutants May Alter Fetal Immune System

This study examined short-term associations of air pollution exposures with lymphocyte immunophenotypes in cord blood about nearly 1,400 deliveries in two regions of the Czech Republic. Researchers measured daily concentrations of $PM_{2.5}$ and 12 polycyclic aromatic hydrocarbons (PAHs) in the air, and various measures of the immune system in the cord blood at the time of birth. Ambient concentrations of PAHs and $PM_{2.5}$ during the last two weeks of gestation were associated with decreases in the percentages of T-lymphocytes in cord blood. Although the biological relevance of this finding is not

clear, it is significant that the fetal immune system may be altered by maternal exposure to air pollution.

Herz-Picciotto I, Herr CEW, Yap P-S, Dostal M, Shumway RH, Ashwood P, Lipsett M, Joad JP, Sram, RJ. Air Pollution and Lymphocyte Phenotype Proportions in Cord Blood. *Environ Health Perspec* 2005; 110:1391-1398. http://ehp.niehs.nih.gov/members/2005/7610/7610.pdf

PARTICULATE MATTER: *HEALTH EFFECTS OF LONG-TERM EXPOSURES*

Premature Deaths

Extended Analysis of Harvard Six Cities Study Shows Decline in Pollution Leads to Reduction in Death Rates

In 1993, the results of the landmark Harvard Six Cities cohort study were published reporting an association between long-term exposures to particulate air pollution and premature deaths. Now, researchers have extended the mortality follow-up in this study by eight additional years, during a period of reduced air pollution concentrations. Using estimates of pollution levels derived from ambient monitors, they found that reductions in death rates followed reductions in $PM_{2.5}$ levels, including to concentrations below the current annual average standard of 15 $\mu g/m^3$. Total mortality and deaths from lung cancer and cardiovascular causes were all positively associated with $PM_{2.5}$ concentrations. Reduction is risk was observed for deaths due to cardiovascular and respiratory disease, and not from lung cancer, a disease with a longer latency period and less reversibility.

Laden F, Schwartz J, Speizer FE, Dockery DW. Reduction in Fine Particulate Air Pollution and Mortality: Extended Follow-up of the Harvard Six Cities Study. *Am J Respir Crit Care Med* 2006; Published online January 19, 2006 as doi:10.1164/rccm.200503 -443OC.
http://ajrccm.atsjournals.org/cgi/content/abstract/200503_-443OCv1

Risk of Premature Death from Chronic Exposure to $PM_{2.5}$ in Los Angeles Three Times Greater than Previously Reported

Earlier studies of long-term health risks of air pollution relied on estimates of community average exposures which may entail measurement error, thus lowering the estimate of health risks attributable to poor air quality. This study used data from 23 $PM_{2.5}$ monitors and 42 ozone monitors to interpolate pollution exposures for nearly 23,000 residents of Los Angeles who are enrolled in the American Cancer Society cohort. After controlling for 44 different factors, the more accurate estimate of $PM_{2.5}$ exposure was associated with an increased relative risk for all cause mortality, deaths from ischemic heart disease, and lung cancer deaths. The results suggest that chronic health effects associated with within city gradients in exposure to $PM_{2.5}$ may be even larger than previously reported across metropolitan areas. Specifically, effects were nearly three times greater than reported in previous studies based on the American Cancer Society cohort.

Jerrett M, Burnett RT, Ma R, Pope III CA, Kerewski D, Newbold KB, Thurston G, Shi Y, Finkelstein N, Calle EE, Thun MJ. Spatial Analysis of Air Pollution and Mortality in Los Angeles. *Epidemiology* 2005; 16:727-736. http://www.epidem.com/pt/re/epidemiology/abstract.00001648_-200511000-00004.htm;jsessionid=DPSZd6wzb1HbyAEaAzG6pQyE20WpNDlbfSrRUkesL2UPdF8Z92gd!_-786779307!-949856145!9001!-1

Long-Term Effects of Air Pollution on Mortality Confirmed in French Study

The long term effects of air pollution on mortality were studied in 14,284 adults who lived in seven French

cities. Daily measurements of sulfur dioxide, total suspended particulate, black smoke, nitrogen dioxide and nitric oxide were available for a three year period. Statistical models controlled for individual confounders such as smoking, educational level, body mass index and occupational exposure. After excluding areas where local traffic pollution dominated, a positive association on the order observed in the long-term U.S. cites was reported for total suspended particulates, black smoke, nitrogen dioxide and nitric oxide and non-accidental mortality. Consistent patterns for lung cancer and cardiopulmonary causes were observed. Researchers conclude that *"urban air pollution assessed in the 1970s was associated with increased mortality over 25 years in France."*

Filleul L, Rondeau V, Vandentorren S, Le Moual N, Cantagrel A, Annesi-Maesano I, Charpin D, Declercq C, Neukirch F, Paris C, Vervloet D, Brochard P, Tessier J-F, Kauffmann F, Baldi I. Twenty Five Year Mortality and Air Pollution: Results from the French PAARC Survey. *Occup Environ Med* 2005; 62:453-460. http://oem.bmjjournals.com/cgi/content/abstract/62/7/453

Particle Pollution Increases Women's Risk of Developing and Dying from Coronary Heart Disease

A multi-decade study published in *Environmental Health Perspectives* reports that women who live in areas with greater coarse and fine particle concentrations have a higher risk of developing and dying from coronary heart disease. In this long-term follow-up of the Adventist Health Study (ASHMOG), a cohort of non-smokers in **California**, PM_{10}, $PM_{10-2.5}$, and $PM_{2.5}$ were associated with increased risk of fatal heart disease in women, especially older women, with the effect strongest for fine particles. No associations were found in males. This study is important because it explores the long-term effects of three different size fractions of particles .

Chen LH, Knutsen SF, Shavlik D, Beeson WL, Petersen F, Ghamsary M, Abbey D. The Association between Fatal Coronary Heart Disease and Ambient Particulate Air Pollution -- Are Females at Greater Risk? *Environ Health Perspec* 2005; 113:1723-1729.
http://ehp.niehs.nih.gov/members/2005/8190/8190.pdf

Long-Term Fine Particle Exposure in California Linked to Lower Birth Weight and Infant Mortality

A study of **California** infants who were born full term has shown a small but consistent effect of $PM_{2.5}$ on birth weight. Researchers matched air pollution monitoring data with California birth records. The pollution measurements were collected within 5 miles of the mother's residence, and were averaged for the time period corresponding to the pregnancy. California mothers who lived in areas with the highest $PM_{2.5}$ exposures during their pregnancy delivered slightly smaller babies, by 30 grams, compared to those with lower exposures, after controlling for demographic factors and carbon monoxide. No associations were observed between carbon monoxide and birth weight.

The authors noted several limitations of their study including difficulty in deciding on an appropriate time period for exposure measurements, and in assigning exposures to each mother based on residence, as well as the lack of data on maternal smoking.

"These findings have important implications for infant health because of the ubiquitous exposure to fine particulate air pollution across the United States," conclude the authors.

Particulate air pollution has been associated with infant mortality, particularly for respiratory causes and sudden infant death syndrome. A follow-up study by the same research group linked $PM_{2.5}$ monitoring data to infants born in **California** in 1999 and 2000 using the addresses of mothers who lived within five miles of a monitor. Each infant who died was matched to 4 infants who lived to age 1 by birth weight category and date of birth. For each matched set, researchers calculated exposure as the average $PM_{2.5}$ concentration

over the lifetime of the infant who died. The results *"add further evidence of a particle effect on respiratory related postneonatal infant mortality,"* according to researchers.

Parker JD, Woodruff TJ, Basu R, Schoendorf KC. Air Pollution an Birth Weight Among Term Infants in California. *Pediatrics* 2005; 115:121-128. http://pediatrics.aappublications.org/cgi/reprint/115/1/121

Woodruff TJ, Parker JD, Schoendorf KC. Fine Particulate Matter ($PM_{2.5}$) Air Pollution and Selected Causes of Postneonatal Infant Mortality in California. *Environ Health Perspec* 2006; doi:10.1289/ehp.8484. Online 13 January 2006. http://ehp.niehs.nih.gov/members/2006/8484/8484.pdf

Respiratory Effects

Researchers Link Childhood Asthma to Exposure to Traffic-Related Pollution

Living near a freeway may bring an increased risk of asthma according to researchers at the Keck School of Medicine of the University of Southern California. Scientists studying air pollution levels in **ten Southern California communities** found that the closer children live to a freeway, the greater their chance of having been diagnosed with asthma. Researchers also found that children who had higher levels of nitrogen dioxide -- commonly emitted by internal combustion engines -- in the air around their homes were more likely to have developed asthma. Researchers looked at the pollution-asthma link in 208 children who ware part of the Children's Health Study, the longest running investigation into air pollution and kids' health. Investigators concluded that the results *"strengthen an emerging body of evidence that air pollution can cause asthma and that traffic-related pollutants that vary within communities are partly responsible for this association."*

Gauderman WJ, Avol A, Lurmann F, Kuenzli N, Gilliland F, Peters J, McConnell R. Childhood Asthma and Exposure to Traffic and Nitrogen Dioxide. *Epidemiology* 2005; 16:737-743. http://www.epidem.com/pt/re/epidemiology/abstract.00001648_-200511000-00005.htm;jsessionid=DJkEWehOleZDuMfSbvT_cF8beu278S2Wu5QMzjsLcWqAaCqjgXUno!693302636!-949856145!9001!-1?index=1&database=ppvovft&results=1&count=10&searchid=2&nav=search

Cardiovascular Mechanisms and Effects

Air Pollution, High-Fat Diet Cause Atherosclerosis in Laboratory Mice

Test results with laboratory mice show a direct cause-and-effect link between exposure to fine particle air pollution and development of atherosclerosis, commonly known as hardening of the arteries. Mice that were fed a high-fat diet and exposed to air with fine particles had 1.5 more times plaque production than mice fed the same diet and exposed to clean air. Plaque, a fatty deposit on the inner lining of the blood vessels, can predispose individuals to conditions such as heart attacks and strokes. The fine particle exposure also led to increased inflammation of the artery walls and reduced function of the artery wall's inner lining.

The findings of the study may explain why people who live in polluted areas have a higher risk of heart disease. The findings are also important because the fine particle concentrations used in the study were well within the range of concentrations found in the air in the Northeastern U.S.. The average particle concentration over the course of the study was below the current 24-hour standard of 65 $\mu g/m^3$ and close to the annual average standard of 15 $\mu g/m^3$. *"These results suggest that repeated periods of short-term (eg, several hours) exposures to high particulate matter levels, such that may occur during rush hour traffic, is potentially capable of promoting progression of atherosclerosis, although the mean daytime particulate matter exposure concentration is within national recommendations. This may potentially have implications for the relevance of both the 24-hour and annual average National Ambient Air Quality Standards,"* concluded the study authors.

Sun Q, Wang A, Jin X, Natanzon A, Duquaine D, Brook RD, Aguinaldo J-GS, Fayad ZA, Fuser V, Lippmann M, Chen LC, Rajagopalan S. Long-term Air Pollution Exposure and Acceleration of Atherosclerosis and Vascular Inflammation in an Animal Model. *JAMA* 2005; 294: 3003-3010. http://jama.ama-assn.org/cgi/content/abstract/294/23/3003

Long-Term Exposure to Air Pollution Implicated in Clogging of the Arteries

Two large clinical trials in **Southern California** have been following the progression of atherosclerosis in participants by measuring the thickness of the carotid artery. Researchers compared this data with the subjects' annual ambient $PM_{2.5}$ exposures. After adjusting for age and other factors, as association was observed between a measure of hardening of the arteries and $PM_{2.5}$ exposures, suggesting a biological pathway for the relationship between particle exposure and premature death from heart disease. Researchers caution that $PM_{2.5}$ may be serving as a surrogate for the mixture of urban air pollution and constituents of PM and that follow up studies are needed to confirm these findings.

Künzli N, Jerrett M, Mack WJ, Beckerman B, LaBree L, Gilliland F, Thomas D, Peters J, and Hodis HN. Ambient Air Pollution and Atherosclerosis in Los Angeles. *Environ Health Perspect* 2005; 113:201-206. http://ehp.niehs.nih.gov/members/2004/7523/7523.pdf

Prenatal Effects

Child Cancers Linked to Pollutants

Researchers collected information on childhood deaths from leukemia or other cancers in Great Britain between 1953 and 1980. Children born near industrial emissions hotspots for carbon monoxide, PM_{10}, volatile organic compounds, nitrogen oxides, benzene, dioxins, 1,3-butadiene, and benz(a)pyrene had increased relative risk of dying from cancer. The study suggests that childhood cancers may be initiated in the womb, by industrial pollutants that have been inhaled by the mother.

Knox EG. Childhood Cancers and Atmospheric Carcinogens. *J Epidemiol Community Health* 2005; 59:101-105. http://jech.bmjjournals.com/cgi/reprint/59/2/101

COARSE PARTICLE HEALTH EFFECTS

Mortality

Particle Pollution Increases Women's Risk of Developing and Dying from Coronary Heart Disease

A multi-decade study published in Environmental Health Perspectives reports that women who live in areas with greater coarse particle concentrations have a higher risk of developing and dying from coronary heart disease. In this long-term follow-up of the Adventist Health Study (ASHMOG), a cohort of non-smokers in **California**, PM_{10}, $PM_{10-2.5}$, and $PM_{2.5}$ were associated with increased risk of fatal heart disease in women, especially older women, with the effect strongest for fine particles. No associations were found in males. This study is important because it adds to our understanding of the long-term effects of inhaling particulate air pollution.

Chen LH, Knutsen SF, Shavlik D, Beeson WL, Petersen F, Ghamsary M, Abbey D. The Association between Fatal Coronary Heart Disease and Ambient Particulate Air Pollution -- Are Females at Greater Risk? *Environ Health Perspec* 2005; 113:1723-1729. http://ehp.niehs.nih.gov/members/2005/8190/8190.pdf

Hospital Admissions

Coarse Particles Increase Hospitalization for Respiratory Infection in Children

A study published online in *Pediatrics* reported a detrimental effect of relatively low levels of coarse particulate matter on hospitalizations for respiratory infections in children. This study used a case-crossover design to examine the relationship between various air pollutants and hospitalization for respiratory infections among children younger than 15 years in **Toronto** over a 4-year period. When PM and gaseous pollutants were both taken into account, the effect remained pronounced for $PM_{10-2.5}$ in both boys and girls.

Lin M, Stieb DM, Chen Y. Coarse Particulate Matter and Hospitalization for Respiratory Infections in Children Younger Than 15 Years in Toronto: A Case-Crossover Analysis. *Pediatrics* 2005; 116:235 -240. http://pediatrics.aappublications.org/cgi/reprint/116/2/e235

Coarse Particles in Vancouver had Larger Effect on Elderly Hospital Admissions than Fine

A time-series study of nearly 9,000 adults over age 65 in **Vancouver, Canada** examined the relationship between hospital admissions for respiratory disease and daily measures of PM_{10}, $PM_{2.5}$, and $PM_{10-2.5}$. After adjustment for gaseous copollutants and meteorological variables, the study found that coarse particles have a larger effect on respiratory hospital admissions in the elderly than $PM_{2.5}$.

Chen Y, Qiuying Y, Krewski D, Burnett RT, Shi Y, McGrail KM. The Effect of Coarse Ambient Particulate Matter on First, Second, and Overall Hospital Admissions for Respiratory Disease Among the Elderly. *Inhalation Toxicology* 2005; 17:649-655. http://www.ncbi.nlm.nih.gov/entrez/query.fcgi?cmd=Retrieve&db=pubmed&dopt=Abstract&list_uids=16087571&query_hl=1&itool=pubmed_docsum

Systematic Review Supports Regulation of Coarse Particles

A systematic review of the epidemiological literature published in the *European Respiratory Journal* examined studies that have investigated the effects of both fine and coarse particles, and found that for some health endpoints, the effects are even stronger for coarse particles than for fine. Specifically, the paper finds that *"in studies of chronic obstructive pulmonary disease, asthma and respiratory admissions, coarse PM has a stronger or as strong short-term effect as fine PM, suggesting that coarse PM may lead to adverse responses in the lungs triggering processes leading to hospital admissions."* The review also found support for an association between coarse PM and cardiovascular hospital admissions.

With respect to the toxicology of coarse particles, the review concluded that *"studies clearly show that coarse PM exerts toxic effects in laboratory experiments, and that such effects are at least as potent as those observed in experiments using fine PM, when expressed on a mass basis,"* while cautioning that fine particles may deliver a higher dose of toxic material to the lungs.

Researchers concluded that the coarse particle fraction is of importance in the regulatory process as well as for control measures.

In an accompanying editorial, Swedish, German, and Dutch researchers argued that systematic review offers evidence for the separate regulation of the coarse particle fraction.

Brunekreef B, Forsberg B. Epidemiological Evidence of Effects of Coarse Airborne Particles on Health. *Eur Respir J* 2005; 26:309-318. http://erj.ersjournals.com/cgi/content/abstract/26/2/309

Sandström T, Nowak D, and van Bree L. Health Effects of Coarse Particles in Ambient Air: Messages for Research and Decision-Making. *Eur Respir J* 2005; 26:187-188. http://erj.ersjournals.com/cgi/content/full/26/2/187

Toxicological Mechanisms

Mechanisms for Coarse Particle Toxicity Developed

The results of laboratory toxicology study which exposed human alveolar macrophages and airway epithelial cells to particles in vitro and followed them for endpoints of inflammation and oxidant stress were reported in *Toxicology and Applied Pharmacology*. These are the two main airway cell types likely to interact with inhaled particles. This study found that the proinflammatory response in alveolar macrophages was driven by material present in the coarse PM. Cultures of bronchial epithelial cells also responded to the coarse fraction with higher levels of certain markers of inflammation than induced by fine or ultrafine PM. These epithelial cells also showed evidence of oxidant stress in response to coarse particle exposure, as well as to other size fractions of PM. This study adds to our understanding of potential mechanisms.

Becker S, Mundandhara S, Devlin RB, Madden M. Regulation of Cytokine Production in Human Alveolar Macrophages and Airway Epithelial Cells in Response to Ambient Air Pollution Particles: Further Mechanistic Studies. *Toxicol Appl Pharmacol* 2005; 207(2 Suppl):269-75. http://www.sciencedirect.com/science?_ob=ArticleURL&_udi=B6WXH-4GJK857-1&_coverDate=09%2F01%2F2005&_alid=354627336&_rdoc=1&_fmt=&_orig=search&_qd=1&_cdi=7159&_sort=d&view=c&_acct=C000050221&_version=1&_urlVersion=0&_userid=10&md5=bc7620603a455138eba195cbda74dac4

MISCELLANEOUS

Intervention Studies

Two Studies Report Children's Respiratory Health Improves When Pollution Declines

Before reunification of Germany in 1989, outdoor concentrations of total suspended particles were higher in **East Germany** than in **West Germany** due to emissions from industrial and domestic sources. Traffic-related air pollution was higher in West Germany than in East Germany. During the initial years after reunification, total suspended particulates (TSP) levels declined in East German and reached West German levels, but emissions from traffic increased. A German study compared two measures of lung function -- lung capacity and airway resistance in six year old East and West German children during this time of decreasing concentrations of TSP in East Germany.

Total lung capacity showed a clear association with long-term concentrations of TSP, while resistance was affected by short-term concentrations. Reduction in TSP was associated with better lung function when comparing repeated cross sections in six-year-old children. However, for children living near busy roads, this improvement was counteracted by increased air pollution associated with traffic during this period.

Reduction in air pollution exposures contributes to improved respiratory health in children, according to a new cross-sectional study of children in **nine communities in Switzerland**. Researchers investigated a moderate decline of PM_{10} air pollution in the 1990s in Switzerland. Some 9,600 children tracked their respiratory symptoms over a 9 year period.

After adjusting for socioeconomic, health-related, and indoor air factors, declines in PM_{10} were associated with declining prevalence of chronic cough, bronchitis, common cold, nocturnal dry cough, and conjunctivitis symptoms. Changes in prevalence of sneezing during pollen season, asthma, and hay fever

were not associated with the PM_{10} reduction.

No threshold with adverse effects of PM_{10} was apparent, because researchers observed the beneficial effects for relatively small changes of rather moderate air pollution levels.

"The larger reduction in symptom rates in areas with a stronger decrease in PM_{10} levels supports the causality of observed associations between air pollution and respiratory health in children," conclude the researchers.

Sugiri D, Ranft U, Schikowski T, Krämer U. The Influence of Large Scale Airborne Particle Decline and Traffic Related Exposure on Children's Lung Function. *Environ Health Perspect* 2006; 114:282-288. http://ehp.niehs.nih.gov/docs/2005/8180/abstract.pdf

Bayer-Oglesby L, Grize L, Gassner M, Takken-Sahli K, Sennhauser FH, Neu U, Schindler C, Braun-Fahrländer C. Decline of Ambient Air Pollution Levels and Improved Respiratory Health in Swiss Children. *Env Health Perspect* 2005; 113:1632-1637. http://ehp.niehs.nih.gov/members/2005/8159/8159.pdf

Biological Mechanisms

Biological Pathway for PM Damage Similar to Cigarette Smoking

This review article explores potential mechanisms of vascular disease induced by lung inflammation. Ambient particles and inhaled cigarette smoke are processed by alveolar macrophages and lung epithelial cells. These cells produce proinflammatory mediators such as cytokines that promote a local inflammatory response in the lung that are thought to contribute to the exacerbation of Chronic Obstructive Pulmonary Disease (COPD) and asthma and promote lung infection. These inflammatory mediators may also translocate into the circulation and induce a systemic inflammatory response. This response can include stimulation of the marrow to release leukocytes and platelets, activation of proteins such as C-reactive protein and fibroinogen that may increase coagulability, and activation of the vascular endothelium, the layer of cells that line the blood vessels and are in direct contact with the blood.

Together, researchers surmise, these effects could cause progression and instability of atherosclerotic plaques, precipitating or aggravating cardiovascular events.

van Eeden SF, Yeung A, Quinlam K, and Hogg JC. Systemic Response to Ambient Particulate Matter: Relevance to Chronic Obstructive Pulmonary Disease. *Proc Am Thorac Soc* 2005; 2:61-67. http://pats.atsjournals.org/cgi/content/abstract/2/1/61

Ultrafine Particles

Ultrafine Particles and PM Health Effects – A Review

Heart disease is the leading cause of death and hospitalization among older adults. Numerous community health studies have reported positive and consistent associations between cardiovascular hospital admissions and mortality with outdoor air pollution, particularly measures of $PM_{2.5}$ and PM_{10}. Panel studies have also reported positive associations between PM and risk of cardiac ischemia and arrhythmias, increased blood pressure, decreased heart rate variability, and increased markers of inflammation and clotting in the blood.

This review study focuses on ultrafine particles, those less than 0.1 microns, which dominate particle number concentrations and surface area, and are therefore capable of carrying large concentrations of adsorbed toxic air pollutants to cardiovascular target sites. The article reviews epidemiologic, panel, and

experimental studies of cardiovascular effects. Though most of these studies used measures of $PM_{2.5}$ or PM_{10}, reviewers hypothesize that it is the ultrafine fraction that may be the causal agent leading to a cascade of acute cardiovascular responses.

"High UFP [ultrafine particle] exposures may lead to systemic inflammation through oxidative stress responses to reactive oxygen species, and thereby promote the progression of atherosclerosis and precipitate acute cardiovascular responses ranging from increased blood pressure to myocardial infarction," state the authors.

They propose that components in ultrafine particles from fossil fuel combustion reach target sites in the lungs, veins, and heart to induce inflammation and oxidative stress, and suggest that future research focus on PM causal components and size fractions.

Delfine RJ, Sioutas C, Malik S. Potential Role of Ultrafine Particles in Associations between Airborne Particle Mass and Cardiovascular Health. *Environ Health Perspect* 2005; 113:934-946. http://ehp.niehs.nih.gov/members/2005/7938/7938.pdf

Policy Analyses

Benefits of an Annual Average $PM_{2.5}$ Standard in California

Analysts used the BENMAP model developed for U.S. EPA to assist in preparation of air pollution control policies to compare the impact of a the current annual average $PM_{2.5}$ NAAQS of 15 $\mu g/m^3$ with a more stringent standard of 12 $\mu g/m^3$ in **California**. The model estimates that achieving the current standard would avert approximately 4,000 premature deaths in California each year, and that approximately 2,000 additional fewer deaths would occur in California each year as a result of attainment of the more stringent standard.

Davidson K, Hallberg A, McCubbin D, Hubbell B. Analysis of $PM_{2.5}$ Using the Environmental Benefits Mapping and Analysis Program (BENMAP). *Journal of Toxicology and Environmental Health* 2005; 203-205. http://irr.uwaterloo.ca/rome/Proceedings/Davidson.pdf

Stringent Annual and Daily Standards Are Needed to Provide Uniform Protection Across the U.S.

An analysis of air quality monitoring data found that 78 percent of the U.S. population could be protected if EPA lowers both the daily and annual average air quality standards for fine particles. This level of protection would be achieved if the daily standard were lowered from 65 $\mu g/m^3$ to 30 $\mu g/m^3$, in conjunction with a lowering of the annual average standard of 15 $\mu g/m^3$ to a level of 12 $\mu g/m^3$.

The analysis found that the 30/12 $\mu g/m^3$ suite of standards provides nearly equivalent 24-hour and annual control of $PM_{2.5}$ distributions across the U.S., thus ensuring a more uniform and consistent level of protection than achieved by lowering only the daily standard.

Johnson PRS, Graham JJ. Analysis of Primary Fine Particle National Ambient Air Quality Standard Metrics. *JAWMA*. In press. http://bronze.nescaum.org/airtopics/pm_-naaqs/AW-05-00119_3.pdf

38 Percent of Northeasterners are Especially Susceptible to Particulate Air Pollution

Using susceptibility criteria compiled from major regulatory and research reports, this study found that within the New England, New Jersey, and New York study areas, 38 percent of the population is under age 18 or over age 65 -- age groups considered especially sensitive to PM pollution effects. Four to eighteen

percent of the adults in the region have cardiopulmonary conditions or diabetes, while 12-15 percent of the children have respiratory allergies or lifetime asthma.

The study finds that more protective air quality standards are needed to protect these sensitive populations.

Johnson PRS, Graham JJ. Fine Particulate Matter National Ambient Air Quality Standards: Public Health Impact on Populations in the Northeastern United States. *Environ Health Perspec* 2005; 113:1140-1147. http://ehp.niehs.nih.gov/members/2005/7822/7822.pdf

OZONE: *HEALTH EFFECTS*

Premature Deaths

Ozone Deaths Not Due to Changes in Temperature

Ozone air pollution has been associated with changes in daily mortality, but because high ozone days are generally quite hot, researchers seek to separate out the effect of temperature. The case-crossover approach allows each person to serve as his own control, by comparing the day of death with a day when the person did not die. This controls for season as well as individual risk factors by matching the control day with the day of the event.

This technique was used in a study of over one million deaths in 14 U.S. cities: **Birmingham, Boulder, Canton, Chicago, Cincinnati, Colorado Springs, Columbus, Detroit, Houston, New Haven, Pittsburgh, Provo, Seattle and Spokane**.

The study found that during the summer months, after controlling for temperature, a 10 ppb increase in maximum hourly ozone concentrations was associated with a small increase in the risk of death. Researchers concluded that *"the association between ozone and mortality risk is unlikely to be caused by confounding by temperature."*

Schwartz J. How Sensitive is the Association between Ozone and Daily Deaths to Control for Temperature? *Am J Respir Crit Care Med* 2005; 171:627-631. http://ajrccm.atsjournals.org/cgi/content/abstract/171/6/627

Three Meta-Analyses Find Short-Term Ozone Exposures Increase the Risk of Death

The U.S. Environmental Protection Agency commissioned 3 meta-analyses of studies of ozone and mortality. Three separate research groups were asked to conduct a meta-analysis, using their own methods and study selection criteria. All three studies report a small but substantial association between daily ozone levels and total mortality.

Researchers from Johns Hopkins University looked at 144 effect estimates from 39 time-series studies and estimated pooled effects by lags, age groups, cause-specific morality, and concentration metrics. They compared the results with pooled estimates from the National Morbidity, Mortality, and Air Pollution Study (NMMAPS) a time-series study of 95 large U.S. urban centers from 1987 to 2000. They found that the meta-analysis and NMMAPS results provided strong evidence of a short-term association between ozone and mortality, with large effects for cardiovascular and respiratory deaths, the elderly, and current-day ozone exposure.

Harvard University scientists gathered 71 time-series studies relating ozone to all-cause mortality, and selected 48 estimates from 28 studies for the analysis. They found a greater effect in the summer, and concluded that the "relationship between ozone and mortality should be considered for future regulatory impact analyses."

New York University researchers conducted a review of 43 short-term ozone mortality studies from around the world and conducted an additional time-series analysis for 7 U.S. cities: **Chicago, Detroit, Houston, Minneapolis-St. Paul, New York City, Philadelphia, and St. Louis**. Their results suggest short-term associations between ozone and mortality, although the estimates vary from city to city.

A commentary on the meta-analyses by Dr. David Bates concludes: *"The 2 new meta-analyses ... along with the recent European study, each have unique features and appear to resolve the question of whether ambient ozone levels are associated with increased mortality. It seems unlikely that $PM_{2.5}$ is an important confounder, and the effect of ozone appears to be independent of temperature. A final question -- that of biologic plausibility -- is in some ways the easiest to answer. Ozone is capable of causing inflammation in the lung at lower concentrations than any other gas. Such an effect would be a hazard to anyone with heart failure and pulmonary congestion, and would worsen the function of anyone with advanced lung disease."*

Bell ML, Dominici F, and Samet JM. A Meta-Analysis of Time-Series Studies of Ozone and Mortality with Comparison to the National Morbidity, Mortality, and Air Pollution Study. *Epidemiology* 2005; 16:436-445. http://www.yale.edu/environment/faculty/bell/publications/files/2005_ozone_meta_analysis.pdf

Levy JI, Chermerynski SM, Samat JA. Ozone Exposure and Mortality: An Empiric Bayes Metaregression Analysis. *Epidemiology* 2005: 16:458-468. http://www.epidem.com/pt/re/epidemiology/abstract.00001648_-200507000-00006.htm;jsessionid=DJt0tgmArXh4FlIwECoaRYSPm35YoJ4JK0MtraA1ta4uoXatHtOg!693302636!_-949856145!9001!-1

Ito K, De Leon SF, Lippmann M. Associations Between Ozone and Daily Mortality: Analysis and Meta-Analysis. *Epidemiology* 2005; 16:446-429. http://www.epidem.com/pt/re/epidemiology/abstract.00001648_-200507000-00005.htm;jsession id=DJtovQoc3oT2wet76WXwcOT7BxlYEizAoh513103nsbxp5grcATl!693302636!_-949856145!9001!-1

Bates DV. Ambient Ozone and Mortality. *Epidemiology* 2005; 16:427-429. http://www.epidem.com/pt/re/epidemiology/pdfhandler.00001648_-200507000-00002.pdf;jsessionid=DJtcCv2BAc441FRfqrmPOPLmtoZ9G0hztBBggwFOKwcXmB2mgpoY!693302636!-949856145!9001!-1

Goodman SN. The Methodologic Ozone Effect. *Epidemiology* 2005; 16:430-435. http://www.epidem.com/pt/re/epidemiology/fulltext.00001648_-200507000-00003.htm;jsessionid=DJuOO5EayBqoGFutBYstyE5XCOWFFtRZkzn6bBmcHauv4zjaZvUc!400681292!-949856144!9001!-1

Respiratory Effects

Infants at Risk of Respiratory Symptoms at Ozone Levels at or Near EPA Standards

Young children may be particularly sensitive to ozone because significant lung development continues after birth. This study followed close to 700 infants born in southwestern Virginia during the summer months in 1995 and 1996. Air quality levels were monitored in Vinton, Virginia, near **Roanoke**. Mothers were interviewed at enrollment and biweekly to report infants' daily respiratory symptoms.

Maximum 8-hour ozone and peak 1-hour ozone were associated with difficulty breathing, but not wheeze, in infants of asthmatic others. Ozone was not associated with cough in this study. The mean concentrations in this study (55 ppb 8-hour average, and 61 ppb peak 1-hour concentration) were below the EPA standards of 80 ppb and 120 ppb, respectively.

Infants of mothers who have asthma were found to have consistently higher risk of respiratory symptoms with increasing ozone exposure.

"At levels of ozone exposure near or below current EPA standards, infants are at increased risk of respiratory symptoms," concludes a study by Yale University medical researchers.

Triche EW, Gent JF, Holford TR, Belanger K, Bracken MB, Beckett WS, Laeher L, McSharry J-E, Leaderer BP. Low-Level Ozone Exposure and Respiratory Symptoms in Infants. *Environ Health Perspect*. Published online 29 December 2005. doi: 10.1289/ehp.8559.
http://ehp.niehs.nih.gov/members/2005/8559/8559.pdf

Mail Carriers Exposed to Ozone Below Current Standards Show Declines in Acute Lung Function

Outdoor workers are considered populations at risk of ozone exposure, because of the time they spend out of doors and the increased dose they receive due to exercise. A study in **Taichung City, Taiwan** has demonstrated that mail carriers exposed to daily ozone concentrations below the current 8-hour air quality standard experienced declines in lung function.

Ozone is a powerful oxidant that can induce pulmonary function impairment at low levels via several toxicological mechanisms. This study measured peak expiratory flow rates in 43 mail carriers twice daily for 6 weeks. A central monitoring station measured ozone, PM_{10} and nitrogen dioxide concentrations.

After working for 8 hours on a day with elevated ozone concentrations, the workers lost respiratory function in nighttime measurements for the same day and on the two following days.

Chan C-C, Wu T-H. Effects of Ambient Ozone Exposure on Mail Carriers' Peak Expiratory Flow Rates. *Environ Health Perspec* 2005; 113:735-738. http://ehp.niehs.nih.gov/members/2005/7636/7636.pdf

Lifetime Exposure to Ozone Stunts Lung Function in Young Adults

This study assessed effects of chronic exposure to air pollutants in University of California, Berkeley freshmen who were lifelong residents of the **Los Angeles** or **San Francisco Bay** areas. Students in the study had never smoked. Air pollution exposure was estimated based on spatial interpolation of PM_{10}, nitrogen dioxide, and ozone monitors to the students residences. Lung function measurements were gathered between February and May, when the students had not had recent exposure to increased levels of ozone.

The study found that lifetime exposure to ozone in adolescents 18-20 years old is associated with reduced levels of lung function measures that reflect the function of the small airways. The associations are independent of any effects related to PM and nitrogen dioxide.

Tager IB, Balmes, Lurmann F, Ngo L, Alcorn S, and Küenzli. Chronic Exposure to Ambient Ozone and Lung Function in Young Adults. *Epidemiology* 2005; 16:751-759.
http://www.epidem.com/pt/re/epidemiology/abstract.00001648_-200511000-00007.htm;jsessionid=DCOtFgaZNheSvMSLZ5yJjt16VKfpJOj49CWeX1Y6h1pgp4xeuWgk!600736187!-949856144!9001!-1

Cardiovascular Effects

Ozone Air Pollution Implicated in Heart Arrythmias

This study evaluated cardiac arrhythmias in patients with implanted cardioverter defibrillators in association with various measures of community air pollution.

Breathing increased ambient ozone concentrations during the previous hour was associated with increased risk of episodes of a particular type of cardiac arrythmia, suggesting that community air pollution may be a precipitant of these events. Associations with $PM_{2.5}$, nitrogen dioxide, and black carbon were positive, but not statistically significant. These episodes, known as atrial fibrillation, are not generally considered lethal, but are tied to an increased risk of premature death. People with this condition have a 5-fold increased risk of stroke if their episodes are not controlled by medication.

Rich DQ, Mittleman MA, Link MS, Schwartz J, Luttmann-Gibson H, Catalano PJ, Speizer FE, Gold DR, and Dockery DW. Increased Risk of Paroxysmal Atrial Fibrillation Episodes Associated with Acute Increases in Ambient Air Pollution. *Environ Health Perspec* 2006; 114:120-123. http://ehp.niehs.nih.gov/members/2005/8371/8371.pdf

New Evidence of Ozone and Pollution and Heart Attacks

French researchers have reported that short-term ozone exposure within a period of 1 to 2 days is related to acute coronary events in middle-aged adults without heart disease, and that nitrogen dioxide and sulfur dioxide are not. The study design allowed for control of long-term seasonal trends, and adjusted for temperature, relative humidity, and influenza epidemics.

Ruidavets J-B, Cournot M, Cassadou S, Giroux M, Meybeck M, Ferrières. Ozone Air Pollution is Associated with Acute Myocardial Infarction. *Circulation* 2005; 111:563-569. http://circ.ahajournals.org/cgi/content/abstract/111/5/563

Prenatal Effects

Prenatal Exposure to Ozone Linked to Reduced Birth Weight

This study investigated the effects of air pollutants on birth weight among term infants who were born in **California** during 1975-1987 and who participated in the Children's Health Study. Birth certificates provided material reproductive history and residence at time of birth. Information on sociodemographic factors and maternal smoking during pregnancy were collected by questionnaire. Monthly average air pollutant levels were interpolated from monitors to the zip code of the mother's residence at childbirth.

The researchers observed an association between lower birth weight and intrauterine growth retardation with ozone concentrations. Second- and third-trimester ozone levels were most strongly associated with deficits in birth weight, followed by carbon monoxide exposures during the first trimester. They reported a clear pattern of increasing deficits in birth weight with increasing levels of ozone for 24-hour ozone levels above 30 ppb.

Although the differences in birth weight were small on average, those in the highest ozone exposure group had deficits of a magnitude equivalent to those observed after exposure to cigarette smoke.

"Because exposures to the levels of ambient air pollutants observed in this study are common, and fetal growth is an important determinant for childhood and adult morbidity and mortality, or findings are likely to have important public health and regulatory implications," conclude the researchers.

Salam MT, Millstein J, Li Y-F, Lurmann FW, Margolis HG, Gilliland FD. Birth Outcomes and Prenatal Exposure to Ozone, Carbon Monoxide, and Particulate Matter: Results from the Children's Health Study. *Environ Health Perspec* 2005; 113:1638-1644. http://www.pubmedcentral.nih.gov/picrender.fcgi?artid=1310931&blobtype=pdf