Health and Environmental Concerns Associated With Diesel Vehicle Emissions

Air pollution remains a very serious health problem around the world. According to the World Health Organization, exposure to urban air pollution is responsible for approximately 800,000 premature deaths, annually. The most serious and widespread air pollution problems are related to particulate matter and ozone, each of which are the principle air pollution problems in Chinese cities and each of which is most closely linked to diesel vehicle emissions. The adverse health effects of each of these pollutants will be briefly reviewed below.

A. Particulate

Tiny enough for between 40 and 1,000 of them to fit on a human hair, particles cause death and illness over both the short and long term. These particles find their way into even the youngest lungs. In Leicester, England, for example, physicians analyzed macrophages, specialized blood cells found in the lung, from children undergoing elective surgery. Ultrafine carbon particles—the sort that typify diesel engine exhaust—were present in the macrophages of all of the children, even a 3-month-old infant. Macrophage particle levels in children who lived close to busy roads were roughly three times higher than those of children living by quiet roads.

Mortality The evidence that exposure to particulate kills is beyond dispute. Studies have shown that increases in daily particle levels are followed by increases in daily deaths in many cities around the world. The largest of these studies tracked the health histories of 552,138 adults in 151 metropolitan areas in the US from 1982 through 1989 and accounted for smoking, obesity, age, alcohol use, and other potential confounding factors. The association between mortality and fine particulate matter is, in the words of one notable researcher, Dr. Douglas Dockery of the Harvard University School of Public Health, “consistent [and] robust.”

These effects are associated with chronic and acute exposures alike. In a study of 772 patients in Boston who had suffered heart attacks, researchers found increases in ambient concentrations of particles —both PM_{10} and $PM_{2.5}$— preceded an increase in the frequency of heart attacks over a period of few hours to one day later. Longer term exposures—years or even decades—are equally dangerous. Analysis of data from the previously noted Harvard study, researchers found that a 10 $\mu g/m^3$ increase of PM_{10} concentrations can be associated with a four percent increase in death (regardless of cause), a six percent increase in cardiopulmonary illness related deaths and an eight percent increase in lung cancer related deaths.

A brief review of similar, notable studies provides a sense of the breadth and depth of the research that makes the conclusion that particulate kills unavoidable. Consider the following:

■ Researchers from Johns Hopkins, Harvard and other universities examined data from 90 cities in different regions of the United States, covering all geographic areas. Daily levels of air pollution from 1987 to 1994 were compared to death and hospital

records. The researchers found not only a link between exposure to particles and death, but "strong evidence of association between PM_{10} levels and exacerbation of chronic heart and lung disease sufficiently severe to warrant hospitalization."

■ Responding to the rapidly accumulating body of evidence from the United States that air pollution was linked to mortality, the European Union founded the "Air Pollution and Health—A European Approach," or APHEA, study. Eleven teams of researchers from 10 different nations studied European cities with a total population of 25 million. As in North America, increases in particle concentrations were followed by similar increases in mortality and hospital admissions.

Illness In addition to their linkage to death, fine particles are associated with a litany of lesser ills, including runny or stuffy noses, sinusitis, sore throat, wet cough, head colds, hay fever, burning or red eyes, wheezing, dry cough, phlegm, shortness of breath, and chest discomfort or pain, as well as hospital admissions for asthma and bronchitis. Increases in fine particle levels are accompanied by higher rates of chronic cough, asthma, and emphysema, even among non-smoking Seventh-Day Adventists. In Utah, elevation in particulate matter concentration resulted in a tripling of hospital admissions of children for respiratory illnesses.

The strongest evidence for ambient PM exposure health risk is derived from epidemiologic studies. Many epidemiologic studies have shown statistically significant associations of ambient PM levels with a variety of human health endpoints in sensitive populations, including mortality, hospital admissions and emergency room visits, respiratory illness and symptoms, and physiologic changes in mechanical pulmonary function. The epidemiologic science points to fine inhalable $PM_{2.5}$ as being more strongly associated with some health effects, such as premature mortality, than inhalable fraction PM_{10}, which is associated with other health effects.[1]

The Health Effects Institute (HEI), a research organization jointly funded by the US EPA and the auto industry, released the results of two major studies that are central to the debate over the adverse impact of particulate on human health. The first report is a re-analysis of two long-term community health studies: the Harvard Six Cities Study (1993), and the American Cancer Society Study (1995). The second, called the National Morbidity, Mortality, and Air Pollution Study (NMMAPS), is original research on hospitalization and deaths associated with air pollution in major U.S. cities.

i. The Particle Epidemiology Reanalysis Project

The Harvard Six City Study and the American Cancer Society study examined the long-term effects of exposure to particulate air pollution on mortality. The Harvard Six-Cities Study by Dr. Douglas Dockery of the Harvard School of Public Health, and others, was published in December 1993 in the New England Journal of Medicine. Researchers followed the health of more than 8,000 people in six small cities that fell along a gradient of air pollution concentrations for a period of 14 to 16 years. As particle concentrations

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[1] Time-series analyses strongly suggest a positive effect on daily mortality across the entire range of ambient PM levels. Relative risk (RR) estimates for daily mortality in relation to daily ambient PM concentration are consistently positive, and statistically significant (at P. 0.05), across a variety of statistical modeling approaches and methods of adjustment for effects of relevant covariates such as season, weather, and co-pollutants.

increased, there was an almost directly proportional increase in the death rate in the residents studied. Residents of the most polluted city in the study, Steubenville, Ohio, had a 26 percent increased risk of premature mortality, compared to the residents of the cleanest city studied, Portage, Wisconsin. According to study authors, this translates into a shortened life expectancy of one to two years for residents of Steubenville compared to residents of Portage.

The March 1995 American Cancer Society study, by Dr. Arden Pope of Brigham Young University, and others, found an association between chronic exposure to fine particle air pollution and premature death in a study group of over half a million people in 151 cities. Sulfate pollution was also associated with early death. The study reported strong associations between sulfates and fine particles and death by cardio-pulmonary causes.

These original studies used statistical techniques to adjust for age, and to control for the effects of smoking, diet, and occupational exposure.

Dr. Daniel Krewski of the University of Ottawa and his associates conducted a reanalysis of these two studies for the Health Effects Institute. First, the HEI - funded researchers undertook a reanalysis of the original studies and a quality audit of the underlying data. Second, researchers performed an extensive sensitivity analysis using alternative statistical methods, and considering the role of 20 potential confounders such as other pollutants, climate, and socio-economic factors on study results. The reanalysis by independent investigators validated the original studies.

ii. The US-National Morbidity, Mortality and Air Pollution Study (NMMAPS)

The Health Effects Institute also commissioned an original nationwide study of the short-term effects of air pollution on human health in the 90 largest American cities. A team of investigators led by Dr. Jonathan Samet and Dr. Scott Zeger of the Johns Hopkins University School of Hygiene and Public Health examined short-term increases in mortality rates caused by short-term elevations in particulate air pollution. Harvard School of Public Health researchers Dr. Douglas Dockery and Dr. Joel Schwartz studied effects on hospitalization in a subset of these cities. NMMAPS developed a new standardized methodology for examining pollution effects across many cities. Investigators developed state-of-the-art statistical techniques to examine the effects of multiple pollutants and the extent of life-shortening.

Some critics have argued that short-term increases in the death rate are unimportant because the individuals affected are very frail and near death, even in the absence of air pollution. NMMAPS dispels this “harvesting” notion. NMMAPS investigators report that life is not shortened by a matter of days, but that life shortening is on the order of months or more.

Critics have also argued that other pollutants may be responsible for observed health effects. NMMAPS found strong evidence linking daily increases in particulate pollution to increases in death, in the twenty largest U.S. cities. The association between particulate matter and mortality persisted even when other pollutants were included in the analysis.

In addition, NMMAPS found stable and robust associations between particulate pollution and increased hospital admissions for cardiovascular disease, pneumonia, and chronic obstructive pulmonary disease.

In their final report to HEI, the investigators concluded "these complementary analyses of mortality and morbidity provide new and strong evidence linking particulate air pollution at current levels to adverse health effects."

iii. Special Diesel Health Concerns

Diesel emissions deserve a special discussion, as they tend to be a dominant source of mobile source cancer risk. The most compelling information to suggest a carcinogenic hazard is the consistent association that has been observed between increased lung cancer and diesel exhaust exposure in certain occupationally exposed workers working in the presence of diesel engines. Approximately 30 individual epidemiological studies show increased lung cancer risks of 20 to 89 percent within the individual study populations. Analytical results of pooling the positive study results show that on average the lung cancer risks were increased by 33 to 47 percent.

The concern for the carcinogenic health hazard resulting from diesel exhaust exposures is widespread and several national and international agencies have designated diesel exhaust or diesel particulate matter as a 'potential' or 'probable' human carcinogen.[2] [3] The International Agency for Research on Cancer (IARC) in the late 1980s concluded that diesel exhaust is a 'probable' human carcinogen.[4] Based on IARC findings, the State of California identified diesel exhaust in 1990 as a chemical known to the State to cause cancer and after an extensive review in 1998 listed diesel exhaust as a toxic air contaminant.[5] The World Health Organization recommends that "urgent efforts should be made to reduce [diesel engine] emissions, specifically of particulates, by changing exhaust train techniques, engine design and fuel composition."

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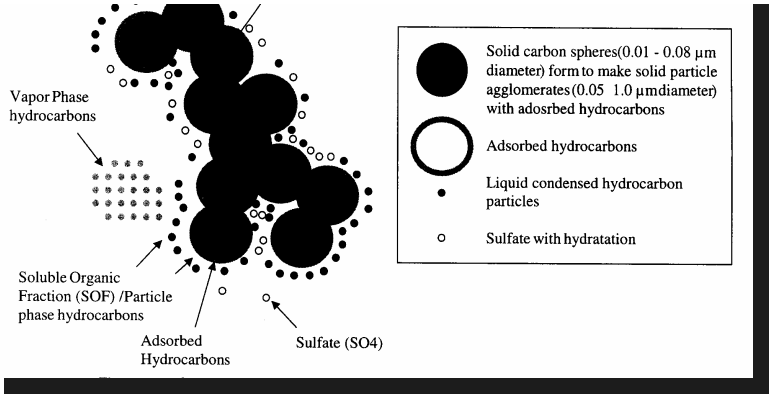
[2] National Institute for Occupational Safety and Health (1988) Carcinogenic effects of exposure to diesel exhaust. NIOSH Current Intelligence Bulletin 50. DHHS (NIOSH) Publication No. 88-116. Centers for Disease Control, Atlanta, GA.

[3] World Health Organization (1996) Diesel fuel and exhaust emissions: International program on chemical safety. World Health Organization, Geneva, Switzerland.

[4] International Agency for Research on Cancer (1989) Diesel and gasoline engine exhausts and some nitroarenes, Vol. 46. Monographs on the evaluation of carcinogenic risks to humans. World Heath Organization, International Agency for Research on Cancer, Lyon, France.

[5] California EPA (1998) Proposed Identification of Diesel Exhaust as a Toxic Air Contaminant Appendix III Part A: Exposure Assessment. California Environmental Protection Agency. California Air Resources Board April 22, 1998.



In addition to these concerns about carcinogenicity, recent studies by Dr. David Diaz-Sanchez of the University of California at Los Angeles, and others, suggest that exposure diesel exhaust can exacerbate human response to common allergens, causing a substantial increase in the body's immune response.

Diesel particulate matter is mainly attributable to the incomplete combustion of fuel hydrocarbons as well as engine oil and other fuel components such as sulfur. Diesel PM is also emitted along with semi-volatile organics that may also contribute to diesel health effects. Diesel exhaust contributes ubiquitously to ambient $PM_{2.5}$, as diesel engines are used to power numerous types of equipment in many places. While diesel particulate matter contributes to ambient levels of $PM_{2.5}$, diesel PM is distinguished from other $PM_{2.5}$ constituents by a high content of elemental carbon (to which organic compounds adsorb) and a high number of ultrafine particle constituents (both organic carbon and sulfate). Additionally, mobile source diesel particulate matter is emitted into the breathing zones of humans and thus has a greater potential for human exposure (per kg of emissions) when compared to other combustion sources such as stacks.

B. Ozone (O_3)

Ground-level ozone, the main ingredient in smog, is formed by complex chemical reactions of volatile organic compounds (VOC) and nitrogen oxides (NOx) in the presence of heat and sunlight. VOCs are emitted from a variety of sources, including motor vehicles, chemical plants, refineries, factories, consumer and commercial products, and other industrial sources. VOCs also are emitted by natural sources such as vegetation. NOx is emitted largely from motor vehicles, nonroad equipment, power plants, and other sources of combustion. Diesel vehicles are a major source of NOx emissions in China.

The science of ozone formation, transport, and accumulation is complex. Ground-level ozone is produced and destroyed in a cyclical set of chemical reactions involving NOx, VOC, heat, and sunlight. As a result, differences in NOx and VOC emissions and weather patterns contribute to daily, seasonal, and yearly differences in ozone concentrations and differences from city to city. Ozone is associated with stagnant periods (periods of low wind, allowing VOC and NOx to concentrate) of elevated ambient temperature and bright sunlight (on the order of several days), more so than a single high temperature day. Further complicating matters, once formed, ozone can be transported to downwind areas, hundreds of kilometers away from the original source of the pollution, affecting air quality in regions with extremely low VOC or NOx emissions. This last phenomenon has been termed, "air mass transport."

Based on a large number of recent studies, it is clear that serious adverse health effects result when people are exposed to the levels of ozone found today in SPMR.[6],[7]

While it is well known that the proportion of VOC/NOx concentration plays an important role in O_3 formation, variables such as meteorology and air mass transport imply that the only way to reduce O_3 is a severe control of both pollutants, especially in large urban areas where this ratio cannot be controlled.

i. Short-Term Exposures

A large body of evidence shows that ozone can cause harmful respiratory effects including chest pain, coughing, and shortness of breath. People with compromised respiratory systems are the most severely affected. When inhaled, ozone can cause acute respiratory problems, aggravate asthma, cause significant temporary decreases in lung function of 15 to 20 percent in some healthy adults, cause inflammation of lung tissue and impair the body's immune system defenses, making people more susceptible to respiratory illnesses. All of which result in increased hospital admissions and emergency room visits.

Short-term exposures (1-3 hours) to high ambient ozone concentrations have been linked to increased hospital admissions and emergency room visits for respiratory problems. For example, studies conducted in the northeastern U.S. and Canada show that ozone air pollution is associated with 10-20 percent of all of the summertime respiratory-related hospital admissions. Repeated exposure to ozone can make people more susceptible to respiratory infection and lung inflammation and can aggravate preexisting respiratory diseases, such as asthma. Exposure to ozone can cause repeated inflammation of the lung, impairment of lung defense mechanisms, and irreversible changes in lung structure, which could lead to premature aging of the lungs and/or chronic respiratory illnesses such as emphysema, chronic bronchitis and chronic asthma.

Children are most at risk from ozone exposure because they are typically active outdoors, playing and exercising during the summer when ozone levels are highest. For example, summer camp studies in the eastern U.S. and southeastern Canada have reported significant reductions in lung function in children who are active outdoors. Further, children are more at risk than adults from ozone exposure because their respiratory systems are still developing. Adults who are outdoors and moderately active during the summer months, such as construction workers and other outdoor workers, also are among those most at risk. These individuals, as well as people with respiratory illnesses such as asthma, especially asthmatic children, can experience reduced lung function and increased respiratory symptoms, such as chest pain and cough, when exposed to ozone during periods of moderate exertion.

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[6] U.S. EPA, 1996, Review of National Ambient Air Quality Standards for Ozone, Assessment of Scientific and Technical Information, OAQPS Staff Paper, EPA-452/R-96-007.

[7] U.S. EPA, 1996, Air Quality Criteria for Ozone and Related Photochemical Oxidants, EPA/600/P-93/004aF.

ii. Prolonged and Repeated Exposures

A large body of scientific literature has made associations between certain patterns of lower level ozone exposure and health effects. These studies do not include any hourly ozone concentrations above the 0.12 parts per million (ppm) level of the 1-hour US national ambient air quality standard (NAAQS).[8] Studies of 6 to 8 hour exposures showed health effects from prolonged and repeated exposures at moderate levels of exertion to ozone concentrations as low as 0.08 ppm.

Studies of acute health effects have shown transient pulmonary function responses, transient respiratory symptoms, effects on exercise performance, increased airway responsiveness, increased susceptibility to respiratory infection, increased hospital and emergency room visits, and transient pulmonary respiratory inflammation. Such acute health effects have been observed following prolonged exposures at moderate levels of exertion at concentrations of ozone well below the current standard of 0.12 ppm. The effects are more pronounced at concentrations above 0.09 ppm, affecting more subjects or having a greater effect on a given subject in terms of functional changes or symptoms.

iii. Other Effects

In addition to the effects on human health, ozone is known to adversely affect the environment in many ways. These effects include reduced yield for commodity crops, for fruits and vegetables, and commercial forests; ecosystem and vegetation effects in such areas as National Parks; damage to urban grass, flowers, shrubs, and trees; reduced yield in tree seedlings and non-commercial forests; increased susceptibility of plants to pests; materials damage; and visibility. In the US state of California alone, the economic impact of ozone on agriculture is estimated as US$ 490 million/yr.[9]

C. Other Pollutants

i. Sulfur Dioxide (SO_2)

Sulfur dioxide (SO_2) exacerbates pre-existing respiratory illnesses, as well as contributing to their development. Sulfur dioxide is an irritant that is generally associated with increases in such respiratory diseases as bronchitis and emphysema. As SO_2 comes into contact with humid surfaces, it is transformed into sulfuric acid, hence its irritant properties.

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[8] The US national ambient air quality standard (NAAQS) designates the USEPA position on concentrations of pollutants that are deemed unhealthy to the general public.

[9] "Research Note 94-18 - Cost of ozone damage to California crops," California Air Resources Board, 1994, http://www.arb.ca.gov/research/resnotes/notes/94-18.htm, downloaded 10 February 2004.

In addition to producing respiratory system irritation, prolonged exposure to this gas has been linked to an increase in cardiovascular death rate in the elderly, as well as to causing hypertrophy of glandular cells - specifically mucous membranes, an increase in the sensitivity of the trachea, and reduction in muco-ciliar movement resulting in bronchitis (Amdur, 1990).

Sulfuric acid is one of the most important acid species contributing to widespread ecosystem damage. Sulfate particulate matter is a significant health concern as well as one of the primary pollutants responsible for impaired visibility.

ii. Carbon Monoxide (CO)

Carbon monoxide -- an odorless, invisible gas generated during the incomplete combustion of carbon based fuels -- poses a serious threat to human health. Fetuses and those afflicted with heart disease are especially at risk. Because the affinity of hemoglobin in the blood is 200 times greater for carbon monoxide than for oxygen, carbon monoxide hinders oxygen transport from blood into tissue. Therefore, more blood must be pumped to deliver the same amount of oxygen. Numerous studies in humans and animals have demonstrated that those individuals with weak hearts are placed under additional strain by the presence of excess CO in the blood. In particular, clinical health studies have shown a decrease in time to onset of angina pain in those individuals suffering from angina pectoris and exposed to elevated levels of ambient CO.[10]

Healthy individuals are also affected, but only at higher CO concentrations. Exposure to elevated CO levels is associated with impairment of visual perception, work capacity, manual dexterity, learning ability and performance of complex tasks.

iii. Nitrogen Dioxide (NO_2)

Nitrogen dioxide (NO_2) is an invisible gas however has a noticeable smell and is highly irritating. An exposed person sensitive to this pollutant will immediately experience burning eyes, nose and mucous membranes. Nitrogen dioxide has been linked with increased susceptibility to respiratory infection, increased airway resistance in asthmatics, and decreased pulmonary function.[11, 12]

iv. Conclusions

It is no longer debateable that air pollution is a serious source of adverse health effects and even premature death. The most serious air pollution problems in

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10"Air Quality Criteria For Carbon Monoxide", US Environmental Protection Agency, Second External Review Draft, October 1999.

[11] U.S.EPA, 1993, Air Quality Criteria for Oxides of Nitrogen, EPA/600/8-91/049aF.

[12] U.S. EPA, 1995, Review of National Ambient Air Quality Standards for Nitrogen Dioxide, Assessment of Scientific and Technical Information, OAQPS Staff Paper, EPA-452/R-95-005

China are related to particulate matter and ozone. Each pollutant is closely linked to emissions from diesel vehicles and adverse public health outcomes.