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Health Effects of Diesel Exhaust Particulate Matter

Diesel engines emit a complex mixture of air pollutants, composed of gaseous and solid material. The visible emissions in diesel exhaust are known as particulate matter or PM, which includes carbon particles or "soot." Diesel exhaust also contains a variety of harmful gases and over 40 other known cancer-causing substances. In 1998, California identified diesel PM as a toxic air contaminant based on its potential to cause cancer, premature death, and other health problems. Those most vulnerable are children whose lungs are still developing and the elderly who may have other serious health problems. Each year in California, diesel PM contributes to 2000 premature deaths and thousands of hospital admissions, asthma attacks and other respiratory symptoms, and lost workdays. Overall, diesel engine emissions are responsible for the majority of California's known cancer risk from outdoor air pollutants. In addition, diesel soot causes visibility reduction and is a potent greenhouse agent involved in global warming.

Summary of the Health and Environmental Effects of Diesel PM Exposure for California*

- Premature deaths (2000 per year)
- Lung cancer (250 per year)
- Decreased lung function in children
- Chronic bronchitis
- Increased respiratory and cardiovascular hospitalizations
- Aggravated asthma
- Increased respiratory symptoms
- Lost workdays
- Reduction in visibility (10 to 75% of total)
- Global warming (2nd to carbon dioxide)

*Except for lung cancer, the health effects are based on the assumption that diesel exhaust PM is approximately as toxic as the general ambient PM mixture.

Diesel Particulate Matter (PM) Contributes to Premature Death

PM is a contributing factor to premature death from heart and/or lung diseases, based on studies of over 500,000 people (Pope et al., 1995, 2002), and independently verified with a reanalysis requested by industry and the U.S. Congress (Krewski et al., 2001). Average life expectancy was reduced by about 1.5 years, comparing the cities with highest and lowest high PM levels (Brunekreef, 1997). This translates to a loss of about 14 years of life for people who died from diseases associated with PM exposure (USEPA, 1999). These studies serve as the basis for PM air quality standards by ARB, U.S. EPA, the World Health Organization guidelines for Europe, and other countries.

Very few studies have investigated the responses of human subjects specifically exposed to diesel PM, and none of the available epidemiologic studies have measured the diesel PM content of the outdoor pollution mix. However, the extensive animal toxicology literature on the health impacts of diesel exhaust PM leads to the conclusion that diesel exhaust PM is at least as toxic as the general ambient PM mixture. ARB has made quantitative estimates of the public health impacts of diesel exhaust PM based on this equivalency assumption. We estimate that current Statewide levels of diesel PM contribute to 2,000 deaths (range: 970 to 3,000) annually, and that PM formed from diesel engine emissions of oxides of nitrogen (NO_X) contributes to an additional 900 deaths (range: 440 to 1,400) annually (Lloyd and Cackette, 2001).

Specific studies that link motor vehicle-related PM exposure to premature death include:

- Elderly people living near major roads had almost twice the risk of dying from cardiopulmonary causes (Hoek et al., 2000).
- PM from motor vehicles was linked to increased mortality (Tsai et al., 2000).
- Fine PM (PM2.5) from mobile sources accounted for three times the mortality as did PM2.5 from coal combustion sources (Laden et al., 2000).

PM Contributes to Illness

PM is also a contributing factor to hospital admissions and emergency room visits for cardiopulmonary causes (Pope, 1989; Schwartz et al., 2003; Sheppard, 2003; Zanobetti and Schwartz, 2003), asthma exacerbation (Whittemore and Korn, 1980), and lost work days (Ostro, 1987). We estimate thousands of hospital admissions for cardiopulmonary causes, emergency room visits, asthma attacks, and millions of lost work days each year in California due to PM (CARB, 2002). At least 10% of these impacts (see below) are related to diesel PM. In addition, preliminary evidence suggests that diesel PM exposure may facilitate development of new allergies (Diaz-Sanchez et al., 1999; Kleinman et al., 2005). By age 18, children exposed to higher levels of PM2.5, NO_X, acid vapor, and elemental carbon (all products of fossil fuel combustion, especially diesel) are five times more likely (7.9% versus 1.6%) to have underdeveloped lungs (80% of normal, equivalent to 40-year olds) compared to teenagers living in communities with lower pollutant levels, and will likely never recover (Gauderman et al., 2004).

In addition, several “intervention” studies report significant reduction in the number of adverse health impacts following either removal or reduction of a PM emission source. For example, the Southern California Children's Health Study reported improved lung function growth rates for young children who relocated from a high PM area to a lower PM area (Avol et al., 2001).

Diesel PM is a Significant Component of PM

There is no unique marker for diesel PM, so directly measuring outdoor levels is difficult. However, we have estimated that the average Statewide exposure to diesel PM in 2000 is 1.8 $\mu g/m^3$ (CARB, 1998). Thus, using an average Statewide PM2.5 exposure of 18.5 $\mu g/m^3$ (CARB, 2002), diesel PM makes up about 10% of total PM2.5.

Calculations performed for an air pollution episode in Southern California show that NO_X emitted from diesel engines could account for a significant fraction of the PM2.5 measured at inland locations. When both these diesel contributions are considered, the diesel PM contribution could increase to an upper limit of 40% during a severe PM2.5 episode (Mysliwiec and Kleeman, 2002).

Diesel PM is Emitted in Urban Areas Resulting in High Exposure

Many diesel emission sources are concentrated near densely populated areas such as ports, rail yards, and heavily traveled roadways. Thus, on average, every ton of diesel emissions in populated areas leads to higher exposures and greater health consequences than emission sources that are further removed from population centers. This point is illustrated by in-vehicle exposure studies conducted in California. Even though Californians average about 6% of their time on roadways, 30 to 55% of diesel PM exposures occurs in vehicles (Fruin et al., 2004). Moreover, self-pollution (i.e., pollution from the vehicle itself) has been observed on every school bus tested in California, regardless of the age of the bus. The cumulative exhaust inhaled by the 40 or so kids on a self-polluting bus is comparable to, or in many cases larger than, the cumulative amount inhaled by all the other people in the South Coast Air Basin (Marshall and Behrentz, 2005).

Diesel PM Deposits in the Lung and Components can be Absorbed in the Body

The majority of diesel PM is less than 1 μm in diameter ($1/70^{th}$ the diameter of a human hair). In general, particles 10 μm or less in diameter can be inhaled into the lungs (U.S. EPA, 2004). Not all inhaled particles deposit in the lung, and many are exhaled. Particles about 0.5 μm in diameter are minimally deposited in the airways, with higher deposition rates for particles both smaller and larger than 0.5 μm in diameter, Chemicals adsorbed on particles can dissolve in the fluid lining the airways, and then be absorbed into the body. Insoluble particles are cleared by more complex mechanisms.

Diesel PM Contains Compounds Known to Damage DNA and Cause Cancer

Diesel PM contains toxic chemicals including compounds that are known to cause damage to genetic material (DNA) and are considered to cause cancer. For example, one class of compounds typically present on diesel PM is polycyclic aromatic hydrocarbons, or PAHs. Some PAHs have been classified as probable human carcinogens by the U.S. EPA and by the International Agency for Research on Cancer (IARC, 1989), a World Health Organization group. These compounds have also been shown to damage DNA and also be absorbed into the bloodstream after diesel PM exposure, and are therefore considered to be available to damage cells in tissues such as the lung (U.S. EPA, 2002). Benzene, the first toxic air contaminant listed by the State, and a known human cancer causing agent for leukemia, has been reported not only in the gaseous phase of diesel exhaust, but also is present on diesel PM itself (U.S. EPA, 2002). Other cancer causing compounds such as formaldehyde, acetaldehyde, acrolein, and 1,3-butadiene are present in diesel exhaust (IARC, 1989; U.S. EPA, 2002) primarily in the gas phase. Diesel exhaust is also considered to pose a respiratory hazard to humans based on extensive studies that show that inflammation is

present in many animals exposed to diesel exhaust (U.S. EPA, 2002). Diesel exhaust is a complex mixture of toxic chemicals, many of which remain unidentified.

In addition to the health effects outlined above, it is estimated that exposure to diesel PM causes about 250 excess cancer cases per year in California (CARB, 2000). Over 30 human epidemiologic studies have investigated the potential carcinogenicity of diesel exhaust. These studies, on average, found that long-term occupational exposures to diesel exhaust were associated with a 40% increase in the relative risk of lung cancer (SRP, 1998). Other organizations have evaluated the carcinogenicity of diesel exhaust. For example, IARC (1989) concluded that diesel engine exhaust is a probable human carcinogen, and based on these IARC findings, the State of California under the Safe Drinking Water and Toxic Enforcement Act of 1986 (Proposition 65) identified diesel exhaust as a chemical known to the State to cause cancer. The U.S. EPA (2002) similarly concluded that diesel exhaust be considered a "probable" human carcinogen by inhalation exposure.

The Value of the Health Benefits of Reducing Diesel PM Exceed the Control Costs

Air pollution has a serious impact on the State's economy. The value of premature deaths resulting from exposure to direct diesel PM is estimated to be $16 billion per year (CARB, 2002; U.S. EPA. 1999). Further, an annual value of over $3.5 billion is associated with hospitalizations, the treatment of major and minor illnesses, and lost workdays each year (CARB, 2002; U.S. EPA. 1999). ARB diesel PM control measures provide health benefits (by avoiding premature deaths, hospitalizations, etc.) that exceed the cost of control. The ratio of benefits to costs for recently adopted measures range from 3 to 80 (CARB, 2003a, 2003b, 2003c, 2004a, 2004b, 2004c). Also, the U.S. EPA recently reported a ratio of 30 for off-road diesel vehicles (U.S. EPA, 2003).

Diesel PM Causes Visibility Reduction

The impact of diesel PM on visibility occurs at a wide range of scales, from large scale impacts, such as near-continental regional haze, to the small-scale impacts that occur from an individual vehicle's exhaust plume. Diesels constitute only about 5% of road vehicles; however, they could contribute from 10% to 75% of visibility degradation in urban areas, depending on surrounding source characteristics (Eldering and Cass, 1996). The peculiar composition of "conventional" diesel exhaust gives it an ability to reduce visibility nearly double that of most other particle sources. The net result is that the visibility impacts of the existing diesel fleet, though variable in time and location, are consistently far greater than their proportional fraction of vehicle mileage, and it is anticipated significant visibility benefits will accompany future reductions in diesel PM and NO_X emissions (Kleeman et al., 2001).

Diesel PM is a Potent Global Warming Agent

PM is an important component of the earth's climate system. Diesel engines emit soot, or black carbon particles which then become airborne. Diesel is responsible for more than half of black carbon emissions in the U.S. (Battye et al., 2002), and about 30% globally (Bond et al., 2004). Black carbon is a strong absorber of solar radiation. Scientists have known for many years that when black carbon particles combine with

dust and chemicals in air they become more efficient in absorbing solar radiation. These black carbon mixtures may be the second biggest contributor to global warming – about 60% of the global warming effects of carbon dioxide (Roberts and Jones, 2004).

Research on Diesel PM Health Effects

Several recent research publications have added to concerns regarding adverse health effects from exposure to diesel exhaust. First, a study of railroad workers employed between 1959 and 1996 found that lung cancer mortality was elevated in jobs associated with work on trains powered by diesel locomotives, suggesting that diesel exhaust contributed to lung cancer mortality in this study group. However, lung cancer mortality did not increase with increasing years of work in these jobs (Garshick and Laden, 2004).

A second study investigated transient exposures to diesel exhaust and their effects on cardiovascular function. Previous studies found a link between traffic-related pollution and cardiovascular effects, such as acute myocardial infarction (heart attacks). Mills and colleagues (2005) exposed 30 healthy men to diluted diesel exhaust in exposure chambers. The investigators found that inhalation of diesel exhaust at the levels found in urban environments impaired two important aspects of vascular function in humans: the regulation of vascular tone and endogenous fibrinolysis. This finding provides a potential mechanism that links air pollution to heart disease including heart attacks. (Mills et al., 2005)

The ARB has conducted a number of studies on the emissions from heavy-duty diesel engines. In one set of studies, toxic pollutant emissions were measured from an in-use 1998 model year diesel transit bus equipped with either an oxidative muffler or a catalyzed particulate filter (DPF) (Ayala et al., 2002; Kado et al., 2005). The emission rates of the measured PM-associated toxic compounds (micrograms per mile) were much lower for the DPF-equipped engine compared to the emission rate from the same diesel engine equipped with the oxidative muffler. The genetic toxicity of the emissions was similar in the two configurations above, both fueled with low sulfur diesel fuel, and depending on the test cycle used. In another related study, the toxicity for a similar engine (1998 model year), but with no aftertreatment (tested with CARB fuel) or with DPF (tested with low or ultraflow sulfur fuel) was determined (Kado and Kuzmicky, 2003). This was part of a larger multi-investigator project (Lev-On, et al. 2002). The highest relative toxicity was observed with the CARB-fueled diesel with no after-treatment, followed by the low sulfur fuel (in the diesel without after-treatment), followed by the low and ultralow sulfur diesel-fueled vehicles equipped with DPF.

Currently, a multi-disciplinary cooperative research effort to characterize and evaluate the health effects of advanced diesel engine systems and fuels is currently being developed by the Health Effects Institute (HEI) and the Coordinating Research Council (CRC). The Program entitled the “Advanced Collaborative Emissions Study” (ACES) is an eight-year, multi-million dollar research project for evaluating the health effects of new 2007 and 2010 engine emissions and is sponsored by several entities. Central to the health effects evaluation will be a chronic animal inhalation study initially modeled after the National Toxicology Program bioassay analyses. The bioassay is conducted

using two rodent species exposed over their lifetime to engine emissions, and they will be evaluated for carcinogenicity and for non-cancer endpoints. The engine emissions will also be chemically characterized in detail for toxic and criteria air pollutants.

References

Avol EL, Gauderman WJ, Tan SM, London SJ, Peters JM. Respiratory effects of relocating to areas of differing air pollution levels. Amer. J. of Resp. and Crit. Care Med. 164: 2067-2072, 2001.

Ayala, A., Kado, N.Y., Okamoto, R.A., Holmen, B.A., Kuzmicky, P.A., Kobayashi, R., and Stiglitz, K.E., 2002. Diesel and CNG Heavy-duty transit buses over multiple driving schedules: regulated emissions and project overview. *SAE Trans.J.Fuel Lub. 2002. 111, 735-747 (see also: SAE Paper* 2002-01-1722).

Battye W., Boyer K., Pace TG. Methods for improving global inventories of black carbon and organic carbon particulates. Report No. 68-D-98-046. Prepared for U.S. Environmental Protection Agency. Research Triangle Park, NC, by EC/R Inc., Chapel Hill NC, 2002.

Bond TC, Streets DG, Yarber KF, Nelson SM, Woo J-H, Klimont ZA. Technology-based global inventory of black and organic carbon emissions from combustion. J. of Geophys. Res.,109 D14203, 2004.

Brunekreef B. Air pollution and life expectancy: is there a relation? Occ. Environ. Med. 54:781-784, 1997.

CARB 1998 Proposed Identification of Diesel Exhaust as a Toxic Air Contaminant. Appendix III. Part A: Exposure Assessment, available at: http://www.arb.ca.gov/toxics/summary/diesel_a.pdf. 1998.

CARB 2002 California Air Resources Board and Office of Environmental Health Hazard Assessment. Staff Report: Public Hearing to Consider Amendments to the Ambient Air Quality Standards for Particulate Matter and Sulfates, available at: http://www.arb.ca.gov/research/aaqs/std-rs/pm-final/pm-final.htm. 2002.

CARB 2003a. California Air Resources. Staff Report: Proposed Diesel Particulate Matter Control Measure For On-Road Heavy-Duty Residential And Commercial Solid Waste Collection Vehicles, available at: http://www.arb.ca.gov/regact/dieselswcv/isor3.pdf. 2003.

CARB 2003b. California Air Resources. Staff Report: Proposed Airborne Toxic Control Measure For In-Use Diesel-Fueled Transport Refrigeration Units (TRU) And TRU Generator Sets, And Facilities Where TRUs Operate, available at http://www.arb.ca.gov/regact/trude03/isor.pdf. 2003.

CARB 2003c. California Air Resources. Staff Report: Airborne Toxic Control Measure For Stationary Compression-Ignition Engines, available at: http://www.arb.ca.gov/regact/statde/isor.pdf. 2003.

CARB 2004a. California Air Resources. Staff Report: Proposed Modifications To The Fleet Rule For Transit Agencies And New Requirements For Transit Fleet Vehicles, available at http://www.arb.ca.gov/regact/bus04/isor.pdf. 2004.

CARB 2004b. California Air Resources. Staff Report: Airborne Toxic Control Measure For Diesel-Fueled Portable Engines, available at: http://www.arb.ca.gov/regact/porteng/isor.pdf. 2004.

CARB 2004c. California Air Resources. Staff Report: Proposed Regulatory Amendments Extending the California Standards for Motor Vehicle Diesel Fuel to Diesel Fuel Used in Harborcraft and Intrastate Locomotives, available at: http://www.arb.ca.gov/regact/carblohc/isor.pdf. 2004.

Diaz-Sanchez D, Garcia MP, Wang M, Jyrala M, Saxon A. Nasal challenge with diesel exhaust particles can induce sensitization to a neoallergen in the human mucosa. J. Allergy Clin. Immunol. 104:1183-8, 1999.

Eldering A, Cass GR. Source-oriented model for air pollutant effects on visibility, J. of Geophys. Res., 101 (D14), 9,343-19,369, 1996.

Freeman III, A. M.; The Measurement of Environmental and Resource Values: Theory and Methods, Second Edition. Resources for the Future, Washington D.C. 2003.

Fruin SA, Winer AM, Rodes CE. Black carbon concentrations in California vehicles and estimation of in-vehicle diesel exhaust particulate matter exposure. Atmos. Environ. 34: 4123-4133, 2004.

Garshick E, Laden F, Hart JE, Rosner B, Smith TJ, Dockery DW, and Speizer F. Lung Cancer in Railroad Workers Exposed to Diesel Exhaust. *Environ Health Perspect* 112:1539–1543 (2004).

Gauderman WJ, Avol E, Gilliland F, Vora H, Thomas D, Berhane K, McConnell R, Kuenzli N, Lurmann F, Rappaport E, Margolis H, Bates D, Peters J. The Effect of air pollution on lung development from 10 to 18 years of age. N. Eng. J. Med. 351:1057-1067, 2004.

Hoek G, Brunekreef B, Goldbohm S, Fischer P, van den Brandt PA. Association between mortality and indicators of traffic-related air pollution in the Netherlands: A cohort study. Lancet 360:1203-1209, 2002.

IARC (International Agency for Research on Cancer) Monographs on the Evaluation of the Carcinogenic Risk of Chemicals to Humans: Diesel and Gasoline Engine Exhausts and Some Nitroarenes, Vol. 46, IARC, World Health Organization, Lyon, France. 1989.

Kado, N.Y., Okamoto, R.A., Kuzmicky, P.A., Kobayashi, R., Ayala, A., Gebel, M.E., Rieger, P.L., Maddox, C., and Zafonte, L. Emissions of Toxic Pollutants from Compressed Natural Gas (CNG) and Low Sulfur Diesel-Fueled Heavy-Duty Transit Buses Tested Over Multiple Driving Cycles, *Environmental Science and Technology, 39:7638-7649, 2005.*

Kado, N.Y. and Kuzmicky, P.A. Bioassay Analyses of Emissions from Compressed Natural Gas and Ultra-low Diesel Fueled Transit Buses. Final Report, South Coast Air Quality Management District, 2003.

Kittelson DB, Johnson J, Watts W, Wei Q, Bukoweicki N. Diesel aerosol sampling in the atmosphere. SAE Technical Paper No. 2000-01-2122, 2000.

Kleeman MJ, Eldering A, Hall JR, Cass GR. Effect of emissions control programs on visibility in southern California. Environ. Sci. Technol. 35, 4668-4674, 2001.

Kleinman MT C. Sioutas C, Stram D, Froines JR, Cho AK, Chakrabarti B, Hamade A, Meacher D, Oldham M. Inhalation of concentrated ambient particulate matter near a heavily trafficked road stimulates antigen-induced airway responses in Mice. J. Air Waste Management Assoc. 55:1277-88, 2005.

Krewski D, Burnett R, Goldberg MS, Koover K, Siemiatycki J, Jerrett M *et al*. Reanalysis of the Harvard Six Cities Study and the American Cancer Society Study of particulate air pollution and mortality. Research Report of the Health Effects Institute, 2001.

Laden F, Neas LM, Dockery DW, Schwartz J. Association of fine particulate matter from different sources with daily mortality in six U.S. cities. Environ. Health Persp. 108: 941-947, 2000.

Lev-On, M, LeTavec, C., Uihlein, J., Alleman, T.L., Lawson, D., Vertin, K., Thompson, G.J., Gautam, M., Wayne, S., Okamoto, R., Rieger, P., Yee, G., Ospital, J., Zielinska, B., Sagebiel, J., Chatterjeee, S., and Hallstrom, K. 2002.

Lloyd AC, Cackette TA. Diesel engines: Environmental impact and control. J. Air Waste Management Assoc. 51**:** 809-847, 2001.

Marshall JD, Behrentz E. Vehicle self-pollution intake fraction: children's exposure to school bus emissions. Environ. Sci.Technol. 39(8): 2559-2563, 2005.

Mills Nicholas L., Håkan Törnqvist, Simon D. Robinson, Manuel Gonzalez, Kareen Darnley, William MacNee, Nicholas A. Boon, Ken Donaldson, Anders Blomberg, Thomas Sandstrom, David E. Newby. Diesel Exhaust Inhalation Causes Vascular Dysfunction and Impaired Endogenous Fibrinolysis. *Circulation* 112:3930-3936, 2005.

Mysliwiec MJ, Kleeman MJ. Source apportionment of secondary airborne particulate matter in a polluted atmosphere. Environ. Sci. Technol. 36: 3806-3814, 2002.

Ostro BD. Air pollution and morbidity revisited: a specification test. J. Environ. Econ. Management. 14: 87-98, 1987.

Pope CA, Thun MJ, Namboodiri MM, Dockery DW, Evans JS, Speizer FE, Health CW. Particulate air pollution as a predictor of mortality in a prospective study of U.S. adults. Am. J. Respir. Crit. Car. Med. 151:669-674, 1995.

Pope, CA, Burnett RT, Thun MJ, Calle EE, Krewski D, Ito K, Thurston G. Lung cancer, cardiopulmonary mortality, and long-term exposure to fine particulate air pollution. JAMA. 287: 1123-1141, 2002.

Pope, CA . Respiratory disease associated with community air pollution and a steel mill, Utah Valley. Amer. J. Public Health. 79: 623-628, 1989.

Roberts DL, Jones A. Climate sensitivity to black carbon aerosol from fossil fuel combustion. J. Geophys. Res. 109, D16202, doi:10.1029/2004JD004676, 2004.

Schwartz J, Zanobetti A, Bateson T. Morbidity and mortality among elderly residents of cities with daily PM measurements. Revised Analyses of Time-Series Studies of Air Pollution and Health. Special Report. Health Effects Institute. Pgs. 25-72, 2003.

Sheppard L. Ambient air pollution and nonelderly asthma hospital admissions in Seattle, Washington, 1987-1994. Revised Analyses of Time-Series Studies of Air Pollution and Health. Special Report. Health Effects Institute. Pgs. 227-240, 2003.

SRP, 1998. Findings of the Scientific Review Panel: The Report on Diesel Exhaust, California Air Resources Board/ Office of Environmental Health Hazard Assessment. http://www.arb.ca.gov/toxics/dieseltac/de-fnds.pdf.

Tsai FC, Daisey JM, Apte MG. An Exploratory analysis of the relationship between mortality and the chemical composition of airborne particulate matter. Inhalation Toxicology 12 (Supplement 2): 121-135, 2000.

U.S. Environmental Protection Agency. Air Quality Criteria for Particulate Matter. Vol. 2, Chapter 6. EPA/600/P-99/002aF.

US EPA 1999. United States Environmental Protection Agency. The Benefits and Costs of the Clean Air Act 1990 to 2010. EPA-410-R-99-001. 1999. Available at http://www.epa.gov/air/sect812/copy99.html

US EPA (2002) Health Assessment Document for Diesel Engine Exhaust. National Center for Environmental Assessment, Office of Research and Development, U.S. Environmental Protection Agency, Washington, D.C.

US EPA (2003). Draft Regulatory Impact Analysis: Control of Emissions from Nonroad Diesel Engines. EPA420-R-03-008, U.S. Environmental Protection Agency, Washington, D.C.

Whittemore A, Korn E. Asthma and air pollution in the Los Angeles area. Amer. J. Public Health. 70:687-696, 1980.

Zanobetti A, Schwartz J. Airborne particles and hospital admissions for heart and lung disease. Revised Analyses of Time-Series Studies of Air Pollution and Health. Special Report. Health Effects Institute. Pgs. 241-248, 2003.