Coal Power: Providing Energy, Asthma, Cardiovascular Disease, and Free Abortions

Ethan Helm* and Benjamin N. Larsen* Department of Biology Lake Forest College Lake Forest, Illinois 60045

Over 23,600 people die from it yearly. It causes more than 554,000 asthma attacks and 38,200 nonfatal heart attacks. (Schneider, 2004). It has left twenty-five percent of New York's Adirondack Lakes uninhabitable (Wu, 2003). It is a large contributor the global warming trend which is melting the artic and glaciers world wide and pushing many species to the brink of extinction (Schneider, 2004). Coal power plants are not only affecting our cities by cloaking them with ominous clouds of pollution, but also our children, us, and the world we live in.

Most of this is preventable. Over half of the electricity in the United States comes from coal power and 548 power plants have not been modified to reduce emissions in the last thirty years (Schneider, 2004). In this time period, huge technological advances have been made that could significantly reduce pollution. Unfortunately, such technology has mainly been implemented in newly constructed power plants and current legislation does not require old power plants to be modified (Corrigan, 2005). As such, over 70% of the sulfur dioxide and 62% of nitrogen oxides released from these plants could have been prevented (Wu, 2003). Additionally, large amounts of ozone, particulate matter, and carbon dioxide could also be reduced if older plants implemented extant technology.

Part of the problem with coal power is that it does not produce just one pollutant. Carbon dioxide, sulfur dioxide, nitrogen oxides, ozone, and particulate matter are all released through burning coal. Nitrogen oxides, ozone, sulfur dioxides, and particulate matter have all been linked with respiratory problems. Additionally, particulate matter has been associated with cardiac disease, and all of the pollutants have been shown to be harmful and potentially fatal to children (Schneider, 2004).

While there are many similarities, pollutants work in a variety of different ways. For example, particulate matter, nitrogen oxides, and sulfur dioxides form acids with other compounds, which cause scarring in the lungs (Schneider, 2004). Also, ozone is damaging because it catalyzes oxidation inside the body. Through oxidation, it can produce free radicals, which cause muscle pain, inhibit hormone function, and disrupt neural impulses (Wu, 2003).

While coal power pollution affects people in a variety of ways, we are focusing on the direct physiological effects of pollution on humans. As such, we are not researching the heat trapping capabilities of carbon dioxide. Additionally, we will not be focusing on the environmental impacts on biodiversity due to nitrogen oxides. Instead, we concentrated on the cardiac, pulmonary, and developmental problems associated with pollution from coal power.

Pulmonary Effects of Coal Power Pollution

Nearly eighty years ago, a dense mist settled to the east of Belgium's third largest metropolitan area, Liege (Roholm, 1937). Soon after the emergence of the mist, doctors in the area began to realize something was wrong. Several thousand acute pulmonary attacks had been caused in the area and there were sixty confirmed deaths (Firket, 1931). In the small town of Meuse, people did not know what hit them. They would later find out that a poison in the form of sulfur dioxide was being produced by a local factory (Roholm, 1937). This discovery was met with much skepticism, and sulfur dioxide emission standards were not increased until the 1970s. Since then, the scientific world has investigated the role of the pollutants released through industrialized processes, and as a result, we know that sulfur dioxide, nitrogen oxides, particulate matter, and ozone all have deleterious pulmonary effects (Pope et al., 2002). A recent study conducted by Kan and Chen in Shanghai found that mortality caused by pulmonary problems was associated pollution from coal power plants; these findings lead to the conclusion that current pollution levels are a threat to the general population's health in Shanghai (2003).

General Effects

Pollution does not even have to affect people in the form of disease. In fact, exposure to air borne pollutants can reduce lung function (Ackermann-Liebrich, 1997). This should be of concern to those whom enjoy the outdoors or breathing in general. Reduced lung function may also be a concern for athletes during competitions in which the body needs oxygen to produce ATP that fuels muscle contractions. An increase in particulate matter of just 10 μ g/m³ corresponded to a 3.4% decrease in FEV (forced expiratory volume, or the amount of air your lungs exhale after a deep breath). Not to be left out, nitrogen dioxide and sulfur dioxide were also correlated with decreased pulmonary function (Ackermann-Liebrich, 1997).

Asthma

Particulate matter has been linked to both cardiac and pulmonary dysfunction. In Barcelona, for example, there was a strong correlation between levels of particulate matter and the number of asthma related hospital visits over a three year period (Llorca et al., 2005). Research by Castellsague et al. also describes a correlation between particulate matter exposure and emergency room hospitalizations for asthma (1995). Some of particulate matter's health impacts may be actually be underestimated due to "masking" by nitrogen dioxide (Llorca et al., 2005).

Also, Nitrogen dioxide has been linked to asthma related symptoms. It was shown to decrease tolerance to allergens among allergic asthmatic individuals. Subjects exposed to NO_2 for short periods of time experienced allergic symptoms when subsequently exposed to allergen levels that had previously left them asymptomatic. Both early and late phase airway responses were more severe after nitrogen dioxide exposure and the histamine response doubled. The increase in symptom intensity may be

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even more pronounced in individuals with more severe asthma or other health problems (Strand, 1998). The findings of Castellsague et al. support this trend; they found nitrogen dioxide increased symptoms of brachial asthma among adults (1995) and Llorca et al. noticed more emergency room treatments for asthma when ambient levels of nitrogen dioxide were higher. The number of emergency room visits increased by about 70% for each additional 100 μ g/m³ of nitrogen dioxide in the air (Llorca et al., 2005).

People who do not have preexisting conditions can also be affected by coal power plant pollution. Chronic obstructive pulmonary disease makes breathing difficult for those inflicted. Generally, the disease is associated with smokers suffering from emphysema and or chronic bronchitis. However, Tolbert et al. found a correlation between chronic obstructive pulmonary disease and exposure to ambient nitrogen dioxide, ozone, and particulate matter (2000). Death due to chronic disease has been linked to sulfur dioxide (Kan, 2003), although the affected individuals are probably sickened by the effect of multiple pollutants.

Pollution Hinders Development

In early December of 1952, a stationary front moved through London thereby reducing wind. At the same time, a thermal inversion trapped coal smoke in the Thames Valley (Schwartz, 2004). The combination of these two events caused a prolific build up of pollution, which resulted in the deaths of around 4,000 people over a four-day period, and many others in the following weeks (Anderson, 1999). Of those who died, the mortality rate of infants was twice that of adults (Schwartz, 1994). In fact, from the time of birth to 4 years of age, the number of alveoli in the human lungs increases by over ten fold (Schwartz, 2004). Thus. infants are not able to obtain oxygen as readily as adults. This demonstrates that development extends past the amniotic sac, and as such, children are even more susceptible to the effects of pollution (Schwartz, 2004).

Lung Development in Children

Children spend more time outdoors, are more active, and breathe more rapidly than adults (Gauderman et al., 2000). Thus, children are more susceptible to inhaling pollutants, all while their bodies are still developing. Gauderman et al. investigated this by monitoring the large and small airways of 3035 fourth graders around Los Angeles, California over four years (2000). By comparing the development of the lungs and the amount of ozone, particulate matter, and nitrogen oxides, they found that each pollutant except ozone decreased lung development. Nitrogen oxides and particulate matter reduced the maximum volume of large air pathways by 0.77% and 0.90% annually (2000). Both values are larger than the 0.2% annual decrease believed to be caused by secondary smoking (Berkey et al., 1986). This demonstrates that the effect of coal power pollution on lung development in children could be physiologically significant (Gauderman et. al., 2000) and cause as much as a 16.7% decrease in lung capacity over a ten year period if both pollutants are independent.

Other studies have confirmed that particulate matter obstructs proper lung development. For example, Jedrychowski et al. found similar results to Gauderman and his associates (1999). Woodward et al. went a step farther by linking particulate matter to infant mortality due to respiratory illness (1997).

Research on the affect of nitrogen oxides on lung development has not been as consistent. In 1990, Dijkstra et al. did a study on children in Holland and found that nitrogen oxides do not prevent lung development. Conversely, Ware et al. found that nitrogen oxides do affect large air pathways (1984). The same lab later refuted these claims (Berkey, 1986). The involvement of nitrogen oxides by Gauderman and his coworkers regarding lung development in children represents a debate in the scientific community that has not been resolved (2000).

While Gauderman et al. did not find that ozone had a statistically significant effect on lung development in children; other scientists had conflicting results (2005). For example, Bates (1995) found that long-term exposure to ozone caused developmental problems associated with the lungs (1995). Frischer et al. confirmed this by analyzing the affect of ambient ozone on children in Austria (1999). The entire debate on ozone's effect was questioned by Tager whom stated that scientists were not adequately addressing co-pollutants of ozone (1999). This critique can be broadly applied to much of pollution research, and many researchers, such as Gauderman, choose to acknowledge the limitations of their studies (2000).

Preterm Delivery and Associated Mortality

Xu et al. (1994) linked the use of coal stoves to low birth rate and premature birth. Low birth weight has been established to be the most important factor for predicting neonatal mortality (McCormick, 1985). How pollution affects premature delivery, however, is not fully understood. It is known, however, that infections can be passed from mother to child causing premature birth (Xu et al., 1995). If disease can be passed through the amniotic sac, pollution can affect the fetus as well. In 1995. Xu et al. noticed that there were significant seasonal changes in concentrations of sulfur dioxide and particulate matter around Beijing, China. Based on this, they looked at medical data to determine whether gestation periods were lower during the periods of higher pollution. Using 24,370 pregnant women from four parts of Beijing, Xu et al. were able to determine that there is a statistically significant correlation between concentration of SO₂ and particulate matter and average gestational age of new born children (1995).

This result was confirmed by Wang et al. Using the same area over a longer period of time, they analyzed the gestational age of 74,621 births (1997). The data showed that for every 100 μ g/m³ of sulfur dioxide there was an 11% greater chance of premature birth with an estimated reduction of birth weight of 7.3g for each 100 μ g/m³ increase. Likewise, for every 100 μ g/m³ of particulate matter preterm delivery was 10% more likely to result in a reduction of birth weight by approximately 6.3g (Wang et al., 1997).

Additionally, Bobak and Leon found mortality increased from the lowest to highest measured amounts of particulate matter in the Czech Republic. However, no correlation between infant mortality and nitrogen oxides and sulfur dioxides was found (1992). In Brazil, particulate matter was more indirectly related to infant mortality through pneumonia (Penna and Duchaide, 1991). While these studies do not demonstrate that pollution from coal power directly causes preterm mortality, there is undoubtedly a correlation between preterm mortality and increased sulfur dioxide and particulate matter.

Coal Powered Pollution Hurts the Heart

Plasma Viscosity

An increase in mortality has been associated with Air pollution (Bobak, 1992; Kan, 2003; Penna and Duchaide, 1991; Peters and Doring, 1997). Much of the mortality is related to cardiac dysfunction rather than pulmonary trouble (Pope et al., 2004). Peters and Doring investigated hospitalization and mortality during pollution episodes due to cardiac disease. A large cross-sectional survey revealed that during pollution episodes, plasma viscosity increased dramatically. In fact, there was nearly a 25% chance of plasma viscosity exceeding the 95th percentile among men. Such considerable thickening may be part of the physiological chain reaction linking ambient air pollution to hospitalization and mortality from cardiovascular illness (Peters and Doring, 1997) also observed by Tolbert et al. (2000) and Pope et al. (2004). Another link in this chain may be vaso and arterial constriction caused by exposure to particulate matter and ozone (Brooke et al., 2004). The effects of such constriction would likely be compounded by atherosclerosis (Pope et al., 2004). quickened

Cardiovascular Disease

Other cardiac problems have also been associated with air pollution. In Atlanta, a survey of over two million emergency room visits revealed an association between cardiovascular disease and ambient air pollution, especially particulate matter (Tolbert et al., 2000). Dysrythmia, cardiac arrest, and heart failure were all associated with exposure to particulate matter (Tolbert et al., 2000; Pope, 2004). As suggested by Peters and Doring, symptoms of cardiovascular disease are probably related to thickening of the blood (1997). Smokers are at particular risk of cardiac disease and exposure to air borne pollutants may increase the risk of disease synergistically (Pope et al., 2004).

Ischemic Strokes

Scientists have demonstrated that pollution can also cause death through strokes (Tolbert et al., 2000). There are two types of strokes: ischemic and hemorrhagic. Hemorrhagic strokes occur because of a burst blood vessel in the brain, while ischemic strokes are a result blockage of blood flow to the brain. Moreover, an increase in plasma viscosity and heart rate variability cause an increased risk for ischemic strokes (Hong et al., 2002a; Peters and Doring, 1997; Peters et al., 1999).

Unfortunately, the affect of pollution on strokes has not been widely studied. In South Korea, strokes are much more common than in the United States. Additionally, rapid industrialization in the form of cars and coal power plants has marked a huge increase in pollution in South Korea, Taiwan, India, and China (Hong et al., 2002a). By comparing hospital records in relation to pollution levels over time, Hong et al. found that particulate matter, nitrogen oxides, sulfur dioxide, and ozone are all correlated with an increase in ischemic stroke mortality (2002a). Another study in Hong Kong found similar results (Wong et al., 2002). Further studies in South Korea illustrated that ozone and particulate matter have a stronger correlation (Hong et al., 2002b). Conversely, Tsai et al. postulated that particulate matter and nitrogen oxides are more closely associated with ischemic strokes in Taiwan (2003). The preponderance of the evidence suggests that coal power pollution is linked to ischemic strokes, but more research must be done to determine which pollutants have the most effect.

Discussion

There is a large body of research linking pollutants released by coal power plants to pernicious health effects. Exposure to pollutants like nitrogen dioxide, sulfur dioxide, particulate matter, and ozone has been correlated to increasing emergency room visits for asthmatics (Castellsague et al., 1995; Llorca et al., 2005), heightened allergic response (Strand, 1998), decreased lung function in healthy individuals (Ackermann-Liebrich, 1997), chronic obstructive pulmonary disorder (Tolbert et al., 2000), and even death from chronic disease (Kan, 2003). These pollutants have also been tied to pulmonary developmental retardation in children (Gauderman, 2000; Jedrychowski et al., 1999). Further studies have linked some pollutants to lowered birth weight and or premature birth (Wang et al., 1997; Xu et al., 1994) while others link particulate matter to infant mortality (Bobak and Leon, 1992; Penna and Duchaide, 1991).

In addition to pulmonary problems, coal power pollution can be dangerous to the circulatory system. Hospitalization due to cardiac dysfunction also increased with ambient air pollution (Pope et al., 2004; Tolbert et al., 2000). Such hospitalization is likely related to high blood plasma viscosity correlated to increasing ambient particulate matter (Peters and Doring, 1997).

While not all of these studies were directly linked to point source pollution from coal power plants, the fact that many pollutants released when coal is combusted in power plants are associated with negative health impacts is alarming. The potential damage caused by coal power pollution should reinforce pleas to maintain or increase existing emissions standards as declared in the Clean Air Act. Moreover, attempts to losen regulations might cause an increase in chronic health problems, interference with children's development, and premature death.

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References

Ackermann-Liebrich, U., P. Leuenberger, J. Schwartz, C. Schindler, C. Monn, G. Bolognini, J. P. Bongard, O. Brandli, G. Domenighetti, S. Elsasser, L. Grize, W. Karrer, R. Keller, H. Keller-Wossidlo, N. Kunzli, B. W. Martin, T. C. Medici, A. P. Perruchoud, M. H. Schoni, J. M. Tschopp, B. Villiger, B. Wuthrich, J. P. Zellweger and E. Zemp. "Lung function and long term exposure to air pollutants in Switzerland. Study on Air Pollution and Lung Diseases in Adults (SAPALDIA) Team." *American Journal of Respirartory Critical Care Medicine*; 1997; **155**: 122-129.

Anderson, H. R. Health effects of air pollution episodes. In: Holgate S. T., Samet J. M., Koren, H. S., Maynard R. L., eds. *Air Pollution and Health* 1999; London. UK. Academic Press: 461-484. Bates, D. Ozone: a review of recent experimental, clinical, and epidemiological evidence, with notes on causation—Part 1. *Can. Respir.* 1995; **2**:25-31, 1995.

Berkey, C., J. Ware, D. Dockery, B. J. Ferris, and F. Speizer. Indoor air pollution and pulmonary function growth in preadolescent children. *Am. J. Epidemiol.* 1986; **123**:250-260. Bobak, M., and Leon, David A. Air Pollution and infant mortality in the Czech Republic,1986-88. *Lancet* 1992; **340**.

Brook, R. D., J. R. Brook, B. Urch, R.Vincent, S. Rajagopalan, and F. Silverman. Inhalation of fine particulate air pollution and ozone causes acute arterial vasoconstriction in healthy adults. *Circulation* 2002.

Castellsague, J, J. Sunyer, M. Saez and J.M. Antonio. "Shortterm association between air pollution and emergency room visits for asthma in Barcelona." *Thorax* 1995; **50**: 1051-1056.

Corrigan, Z. Pollution on the Rise: Local Trends in Power Plant Pollution. US PIRG Education Fund, Clear the Air 2005.

Dijkstra, L., D. Houthuijs, B. Brunekreer, I. Akkerman, and J. Boleij. Respiratory Health Effects of the indoor environment in a population of Dutch Children. *Am. Rev. Respir. Dis.* 1990; **142**:1172-1178.

Firket, J. The cause of the symptoms found in the Meuse Valley during the fog of December, 1930. *Bull Acad. R. Med. Belgium* 1931; **11**:638-741.

Frischer, T., M. Studnicka, C. Gartner, E. Tauber, F. Horak, A. Veiter, J. Spengler, J. Kuhr, and R. Urbanek. Lung function growth and ambient ozone: a three-year population study in school children. *Am. Journal of Respiratory Critical Care Medicine* 1999; **160**:390-396.

Gauderman, J. W., R. McConnell, F. Gilliland, S. London, D. Thomas, E. Avol, H. Vora, K. Berhane, E. B. Rapparport, F. Lurmann, H. G. Margolis, and J. Peters. Association between Air Pollution and Lung Function Growth in Southern California Children. *American Journal of Respiratory and Critical Care Medicine* 2000; **162**: 1383-1390

Hong, Y., J. Lee, H. Kim, and H. Kwon. Air Pollution: A New Risk Factor in Ischemic Stroke Mortality. *Stroke* 2002a; **33**:2165-2169.

Hong, Y., J. Leel H. Kim, E. Ha, J. Schwartz, and D. Christiani. Effects of Air Pollutants on Acute Stroke Mortality. *Environmental Health* Perspectives 2002b; **101**: 187-191.

Jedrychowski, W., E. Flak, and E. Mroz. The adverse effect of low levels of ambient air pollutants on lung function growth in preadolescent children. *Environ. Health Perspective* 1999; **107**:669-674.

Kan, H., and Chen, B. Air pollution and daily mortality in Shanghai: a time-series study. *Archives of Environmental Health* 2003; **58**(6):360-367.

Llorca, J., A. Salas, D. Prieto-Salceda, V. Chinchon-Bengoechea, and M. Delgado-Rodríguez. Nitrogen dioxide increases cardiorespiratory admissions in Torrelavega (Spain). *Journal of Environmental Health* 2005; **68**(2):30-35.

McCormick, M. C. The contribution of low birth weight to infant mortality and childhood morbidity. *New England Journal of Medicine* 1985; **312**:82-90.

Penna, M. L. F. and Duchiade, M. P. Air pollution and infant mortality from; pneumonia in the Rio de Janeiro metropolitan area. *Bull Pan Am Health Organ* 1991; **25**: 47-54.

Peters, A., and A. Doring. Increased plasma viscosity during an air pollution episode: a link to mortality? *Lancet* 1997; **349**: 1582-1587.

Peters, A., S. Perz, A. Doring, J. Stieber, W. Koenig, and H. E. Wichmann. Increases in heart rate during an air pollution episode. *American Journal of Epidemiology* 1999; **138**: 890-899.

Pope, A. C. III, R. Burnett, G. D. Thurnston, M. J. Thun, E. Calle, D. Krewski, and J. J. Godleski. Cardiovascular mortality and long-term exposure to particulate air pollution: epidemiological evidence of general pathways of disease. *Circulation* 2004; **109**: 71-77

Strand, V., M. Svartengren, S. Rak, C. Barck, and G. Bylin. Repeated exposure to an ambient level of NO2 enhances asthmatic responses to a nonsymptomatic allergen dose. *European Respiratory Journal* 2000; **12**: 6-12.

Roholm, Kaj. The Fog Disaster in the Meuse Valley, 1930: A Fluorine Intoxication. The *Journal of Industrial Hygiene and Toxicology* 1937; **19**:126-137.

Schneider, C. Dirty Air, Dirty Power. *Clear the Air* 2004; Mount Vernon Printing.

Schwartz J. What are people dying of on high air pollution days? *Environ Res.* 1994; **64**:26–35.

Schwartz, J. Air Pollution and Children's Health. *Pediatrics* 2004; **113**:1037-1043.

Tager, I. Air pollution and lung function growth: is it ozone? *Am. J. Respir. Crit. Care Med.* 1999; **160**:387-389.

Tolbert, P. E., M. Klein, K. B. Metzger, J.Peel, W. D. Flanders, K. Todd, J. Mulholland, P. B. Ryan, and H. Frumkin. Interim results of the study of particulates and health in Atlanta (SOPHIA). *Journal of Exposure Analysis and Environmental Epidemiology* 2000; **10**: 446-460.

Tsai, S., W. B. Goggins, H. Chiu, and C. Yang. Evidence for Association Between Air Pollution and Daily Stroke Admissions in Kaohsiung, Taiwan. *Stroke* 2003; **34**: 2612-2616.

Wang, X., H. Ding, L. Ryan, and X. Xu. Association between Air Pollution and Low Birth Weight: A Community-based Study. *Environmental Health Perspectives*. 1997; **105**: 514-520.

Wong, T. W., W. S. Tam, T. S. Yu, and A. H. S. Wong. Associations between daily mortalities from respiratory and cardiovascular diseases and air pollution in Hong Kong, China. *Occupational and Environmental Medicine* 2002; **59**:30-35.

Woodruff, T. J., J. Grillo, C. Schoendor. The relationship between selected causes of postneonatal infant mortality and particulate air pollution in the United States. *Environ. Health Prospect* 1997; **105**:608-612.

Wu, B. Lethal Legacy: A Comprehensive Look at America's Dirtiest Power Plants. *US PIRG Education Fund, Clear the Air* 2003.