COAL's ASSAULT *on* HUMAN HEALTH

By

Alan H. Lockwood, MD FAAN

Kristen Welker-Hood, ScD MSN RN

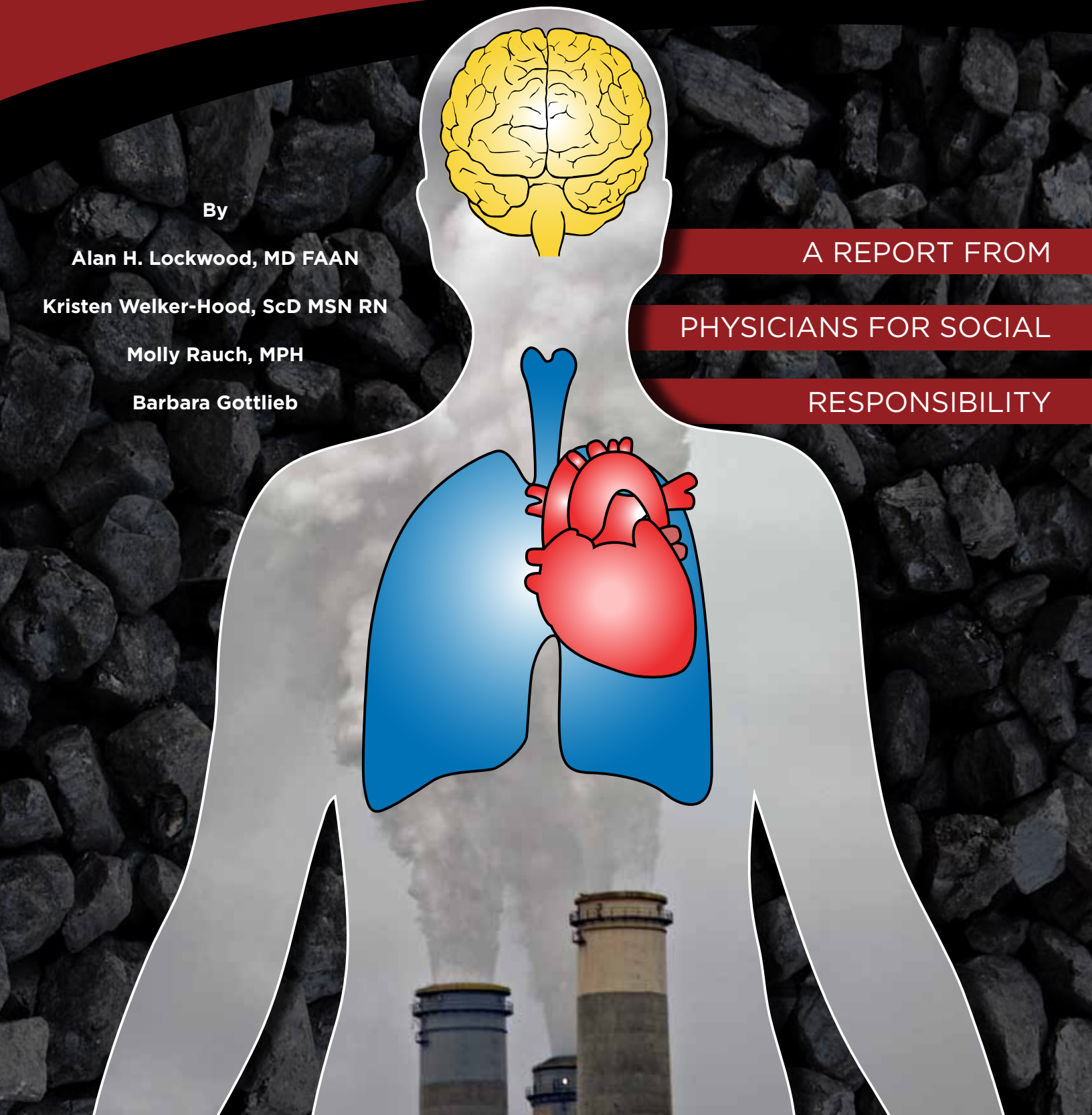
Molly Rauch, MPH

Barbara Gottlieb

A REPORT FROM

PHYSICIANS FOR SOCIAL

RESPONSIBILITY



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ACKNOWLEDGMENTS

The authors gratefully acknowledge the following experts who reviewed drafts of this report, sharing their insight and comments:

Paul Epstein, MD MPH
Howard Hu, MD MPH ScD
Philip Landrigan, MD MSc
Michael McCally, MD PhD
Cindy Parker, MD MPH
Jonathan Patz, MD MPH
Katherine Shea, MD MPH

Any remaining errors are entirely our own.

This report was made possible through the generous financial support of the Energy Foundation and the Compton Foundation.

Production was generously underwritten by PMG, a division of The Production Management Group, Ltd.

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ABOUT PHYSICIANS FOR SOCIAL RESPONSIBILITY

PSR has a long and respected history of physician-led activism to protect the public's health. Founded in 1961 by a group of physicians concerned about the impact of nuclear proliferation, PSR shared the 1985 Nobel Peace Prize with International Physicians for the Prevention of Nuclear War for building public pressure to end the nuclear arms race. Today, PSR's members, staff, and state and local chapters form a nationwide network of key contacts and trained medical spokespeople who can effectively target threats to global survival. Since 1991, when PSR formally expanded its work by creating its environment and health program, PSR has addressed the issues of global warming and the toxic degradation of our environment. PSR presses for policies to curb global warming, ensure clean air, generate a sustainable energy future, prevent human exposures to toxic substances, and minimize toxic pollution of air, food, and drinking water.

NOVEMBER 2009

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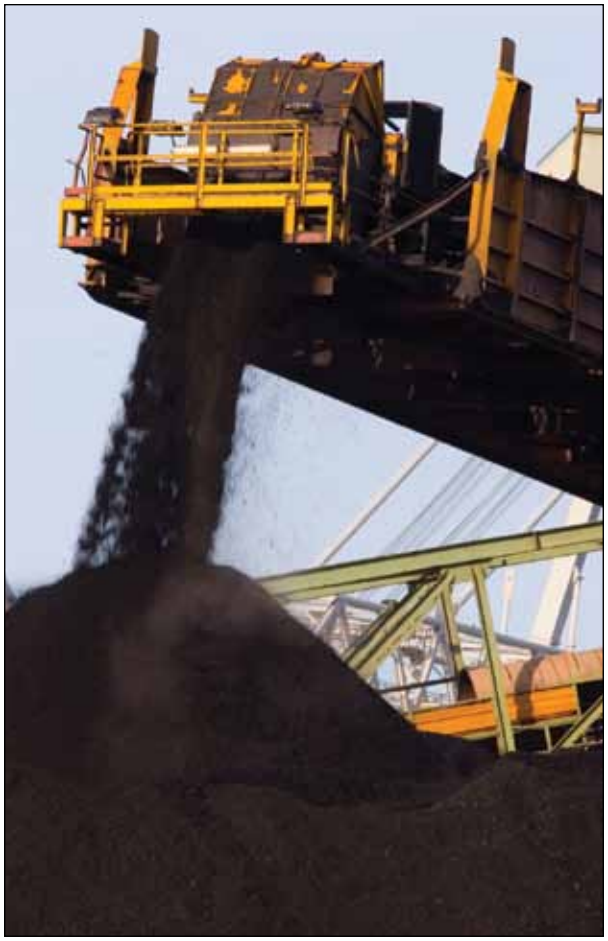
Executive Summary

Coal pollutants affect all major body organ systems and contribute to four of the five leading causes of mortality in the U.S.: heart disease, cancer, stroke, and chronic lower respiratory diseases. This conclusion emerges from our reassessment of the widely recognized health threats from coal. Each step of the coal lifecycle—mining, transportation, washing, combustion, and disposing of post-combustion wastes—impacts human health. Coal combustion in particular contributes to diseases affecting large portions of the U.S. population, including asthma, lung cancer, heart disease, and stroke, compounding the major public health challenges of our time. It interferes with lung development, increases the risk of heart attacks, and compromises intellectual capacity.

Oxidative stress and inflammation are indicated as possible mechanisms in the exacerbation and development of many of the diseases under review. In addition, the report addresses another, less widely recognized health threat from coal: the contribution of coal combustion to global warming, and the current and predicted health effects of global warming.



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THE LIFE CYCLE OF COAL

Electricity provides many health benefits worldwide and is a significant contributor to economic development, a higher standard of living, and an increased life expectancy.[1] But burning coal to generate electricity harms human health and compounds many of the major public health problems facing the industrialized world. Detrimental health effects are associated with every aspect of coal's life cycle, including mining, hauling,

preparation at the power plant, combustion, and the disposal of post-combustion wastes. In addition, the discharge of carbon dioxide into the atmosphere associated with burning coal is a major contributor to global warming and its adverse effects on health worldwide.

Coal mining leads U.S. industries in fatal injuries[2] and is associated with chronic health problems among miners, such as black lung disease, which causes permanent scarring of the lung tissues.[3] In addition to the miners themselves, communities near coal mines may be adversely affected by mining operations due to the effects of blasting, the collapse of abandoned mines, and the dispersal of dust from coal trucks. Surface mining also destroys forests and groundcover, leading to flood-related injury and mortality, as well as soil erosion and the contamination of water supplies. Mountaintop removal mining involves blasting down to the level of the coal seam—often hundreds of feet below the surface—and depositing the resulting rubble in adjoining valleys. This surface mining technique, used widely across southern Appalachia, damages freshwater aquatic ecosystems and the surrounding environment by burying streams and headwaters.[4]

After removal of coal from a mine, threats to public health persist. When mines are abandoned, rainwater reacts with exposed rock to cause the oxidation of metal sulfide minerals. This reaction releases iron, aluminum, cadmium, and copper into the surrounding water system[5] and can contaminate drinking water.[6]

Coal washing, which removes soil and rock impurities before coal is transported to power plants, uses polymer chemicals and large quantities of water and creates a liquid waste called slurry. Slurry ponds can leak or fail, leading to injury and death, and slurry injected underground into old mine shafts can release arsenic, barium, lead, and manganese into nearby wells, contaminating local water supplies. Once coal is mined and washed, it must be transported to power plants. Railroad engines and trucks together release over 600,000 tons of nitrogen oxide and 50,000 tons of particulate matter into the air every year in the process of hauling coal, largely through diesel exhaust.[7] Coal trains and trucks also release coal dust into the air, exposing nearby communities to dust inhalation.[8]

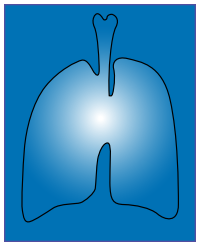
Coal combustion emissions damage the respiratory, cardiovascular, and nervous systems and contribute to four of the top five leading causes of death in the U.S.

The storage of post-combustion wastes from coal plants also threatens human health. There are 584 coal ash dump sites in the U.S.,[9] and toxic residues have migrated into water supplies and threatened human health at dozens of these sites.[10]

The combustion phase of coal's lifecycle exacts the greatest toll on human health. Coal combustion releases a combination of toxic chemicals into the environment and contributes significantly to global warming. Coal combustion releases sulfur dioxide, particulate matter (PM), nitrogen oxides, mercury, and dozens of other substances known to be hazardous to human health. Coal combustion contributes to smog through the release of oxides of nitrogen, which react with volatile organic compounds in the presence of sunlight to produce ground-level ozone, the primary ingredient in smog.

Table ES.1 (pages x–xi) describes the major health effects linked to coal combustion emissions. These health effects damage the respiratory, cardiovascular, and nervous systems and contribute to four of the top five leading causes of death in the U.S.: heart disease, cancer, stroke, and chronic lower respiratory diseases. Although it is difficult to ascertain the proportion of this disease burden that is attributable to coal pollutants, even very modest contributions to these major causes of death are likely to have large effects at the population level, given high incidence rates. Coal combustion is also responsible for more than 30% of total U.S. carbon dioxide pollution, contributing significantly to global warming and its associated health impacts.

RESPIRATORY EFFECTS OF COAL POLLUTION



Pollutants produced by coal combustion act on the respiratory system to cause a variety of adverse health effects. Air pollutants—among them nitrous oxide (NO_2) and very small particles, known as $PM_{2.5}$—adversely affect lung development, reducing forced expiratory volume (FEV) among children.[11] This reduction of FEV, an indication of lung function, often precedes the subsequent development of other pulmonary diseases.

Air pollution triggers attacks of asthma, a respiratory disease affecting more than 9% of all children in the U.S. Children are particularly susceptible to the development of pollution-related asthma attacks. This may be due to their distinct breathing patterns, as well as how much time they spend outside. It may also be due to the immaturity of their enzyme and immune systems, which assist in detoxifying pollutants, combined with incomplete pulmonary development.[12] These factors appear to act in concert to make children highly susceptible to airborne pollutants such as those emitted by coal-fired power plants.[13]

Asthma exacerbations have been linked specifically to exposure to ozone, a gas produced when NO_2 reacts with volatile organic compounds in the presence of sunlight and heat.[14] The risk to children of experiencing ozone-related asthma exacerbations is greatest among those with severe asthma. That risk exists even when ambient ozone levels fall within the limits set by the EPA to protect public health.

Coal pollutants trigger asthma attacks in combination with individual genetic characteristics.[15] This gene-environment interaction means that some individuals are more susceptible to the respiratory health effects of coal pollution. The genetic polymorphisms that appear to make people more susceptible include those that control inflammation and those that deal with oxidative stress, or the presence of highly reactive molecules, known as free radicals, in cells. (See text box.)

OXIDATIVE STRESS

Oxygen free radicals in biological systems are a normal cellular constituent and play critical roles in the control of many cellular functions. (Free radicals are atoms or molecules that contain at least one unpaired electron in an atomic or molecular orbit and are therefore unstable and highly reactive.)

The concentration of oxygen free radicals can be increased through exposure to environmental substances such as air pollution, tobacco smoke, pesticides, and solvents. When their concentration is excessive, these highly reactive molecules damage lipids, proteins, DNA, cell membranes, and other cellular components. "Oxidative stress" is the term used to describe that physiological state.

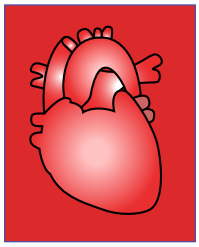
Oxidative stress is an important contributing factor in a variety of diseases, including atherosclerosis, hypertension, rheumatoid arthritis, diabetes mellitus, and neurodegenerative disorders such as Alzheimer's disease and Parkinson's disease, as well as normal aging. It is one of several mechanisms implicated in the pathogenesis of diseases caused or made worse by coal pollutants, such as cardiovascular and pulmonary disease.

Valko M, Leibfritz D, Moncol J, Cronin MTD, Mazur M, Telser J. Free radicals and antioxidants in normal physiological functions and human disease. Int J Biochem and Cell Biology 2007; 39: 44–84.

Coal pollutants play a role in the development of chronic obstructive pulmonary disease (COPD), a lung disease characterized by permanent narrowing of airways. Coal pollutants may also cause COPD exacerbations, in part through an immunologic response—i.e., inflammation.[16,17,18] PM exposure disposes the development of inflammation on the cellular level, which in turn can lead to exacerbations of COPD. COPD is the fourth leading cause of mortality in the U.S.

Exposures to ozone and PM are also correlated with the development of[19] and mortality from[20, 21, 22] lung cancer, the leading cancer killer in both men and women.

CARDIOVASCULAR EFFECTS OF COAL POLLUTION

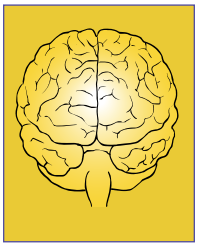


Pollutants produced by coal combustion damage the cardiovascular system. Coronary heart disease (CHD) is a leading cause of death in U.S., and air pollution is known to negatively impact cardiovascular health.[23] The mechanisms by which air pollution causes cardiovascular disease have not been definitively identified but are thought to be the same as those for respiratory disease: pulmonary inflammation and oxidative stress. Studies in both animals and humans support this theory, showing that pollutants produced by coal combustion lead to cardiovascular disease, such as arterial occlusion (artery blockages, leading to heart attacks) and infarct formation (tissue death due to oxygen deprivation, leading to permanent heart damage).

Recent research suggests that nitrogen oxides and $PM_{2.5}$, along with other pollutants, are associated with hospital admissions for potentially fatal cardiac rhythm disturbances.[24] The concentration of $PM_{2.5}$ in ambient air also increases the probability of hospital admission for acute myocardial infarction,[25] as well as admissions for ischemic heart diseases, disturbances of heart rhythm, and congestive heart failure.[26] Additionally, cities with high NO_2 concentrations had death rates four times higher than those with low NO_2 concentrations.[27] These studies show important immediate effects of coal pollutants on indicators of acute cardiovascular illness.

There are cardiovascular effects from long-term exposure as well. Exposure to chronic air pollution over many years increases cardiovascular mortality.[28] This relationship remains significant even while controlling for other risk factors, such as smoking. Conversely, long-term improvements in air pollution reduce mortality rates. Reductions in $PM_{2.5}$ concentration in 51 metropolitan areas were correlated with significant increases in life expectancy,[29] suggesting that air quality improvements mandated by the Clean Air Act have measurably improved the health of the U.S. population. Reducing exposure to the pollutants emitted by coal combustion is therefore an important aspect of improving cardiovascular health for the population at large.

NERVOUS SYSTEM EFFECTS OF COAL POLLUTION



In addition to the respiratory and cardiovascular systems, the nervous system is also a target for coal pollution's health effects. The same mechanisms that are thought to mediate the effect of air pollutants on coronary arteries also apply to the arteries that nourish the brain. These include stimulation of the inflammatory response and oxidative stress, which in turn can lead to stroke and other cerebral vascular disease.

Several studies have shown a correlation between coal-related air pollutants and stroke. In Medicare patients, ambient levels of $PM_{2.5}$ have been correlated with hospital admission rates for cerebrovascular disease,[30] and PM_{10} has been correlated with hospital admission for ischemic stroke.[31] (Eighty-seven percent of all strokes are ischemic.) $PM_{2.5}$ has also been associated with an increase in the risk of—and death from—a cerebrovascular event among post-menopausal women.[32] Even though a relatively small portion of all strokes appear to be related to the ambient concentration of PM, the fact that nearly 800,000 people in the U.S. have a stroke each year makes even a small increase in risk a health impact of great importance.[33]

Coal pollutants also act on the nervous system to cause loss of intellectual capacity, primarily through mercury. Coal contains trace amounts

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of mercury that, when burned, enter the environment. Mercury increases in concentration as it travels up the food chain, reaching high levels in large predatory fish. Humans, in turn, are exposed to coal-related mercury primarily through fish consumption. Coal-fired power plants are responsible for approximately one-third of all mercury emissions attributable to human activity.[34]

A nationwide study of blood samples in 1999–2000 showed that 15.7% of women of childbearing age have blood mercury levels that would cause them to give birth to children with mercury levels exceeding the EPA's maximum acceptable dose for mercury.[35] This dose was established to limit the number of children with mercury-related neurological and developmental impairments. Researchers have estimated that between 317,000 and 631,000 children are born in the U.S. each year with blood mercury levels high enough to impair performance on neurodevelopmental tests and cause lifelong loss of intelligence.[36]

A continued reliance on coal combustion for electricity will contribute to the predicted health consequences of global warming.

GLOBAL WARMING AND COAL POLLUTION

Coal damages the respiratory, cardiovascular, and nervous systems through pollutants acting directly on the body. But coal combustion also has indirect health effects, through its contribution to greenhouse gas emissions. Global warming is already negatively impacting public health and is predicted to have widespread and severe health consequences in the future. Because coal-fired power plants account for more than one third of CO_2 emissions in the U.S.,[37] coal is a major contributor to the predicted health impacts of global warming.

The effects of global warming already in evi-

Table ES.1: Coal's contributions to major health effects

	Disease or condition	Symptoms or result	Most-vulnerable populations	Total disease burden (coal is a suspected contributing factor in an unknown number of cases)	Coal pollutants implicated
RESPIRATORY	Asthma exacerbations	Coughing, wheezing, shortness of breath, and breathlessness with a range of severity from mild to requiring hospitalization	Children, adults	Number of visits to office-based physicians for asthma: 10.6 million in 2006. Number of hospitalizations with asthma listed first as diagnosis: 440,000.[38] School days missed per year attributable to asthma: 11.8 million.[39]	NO_2 Ozone Particulate Matter (PM)[40,41,42]
	Asthma development	New cases of asthma, resulting in coughing, wheezing, shortness of breath, and breathlessness with a range of severity from mild to requiring hospitalization	Children	Children with asthma: 6.7 million (9.1%). Adults with asthma: 16.2 million (7.3%).[43]	Suspected but not confirmed:[44,45,46] NO_2 Ozone $PM_{2.5}$
	Chronic Obstructive Pulmonary Disease (COPD)	Emphysema with chronic obstructive bronchitis; permanent narrowing of airways; breathlessness; chronic cough	Smokers, adults	Adults with COPD diagnosis in 2006: 12.1 million.[47] Deaths in 2005: 126,000.[48] Fourth leading cause of mortality in U.S.	NO_2 PM[49,50,51]
	Stunted lung development	Reductions in lung capacity; risk factor for development of asthma and other respiratory diseases	Children	Unknown	NO_2 $PM_{2.5}$[52]
	Infant mortality (relevant organ system uncertain; may be respiratory)	Death among infants age < 1 year	Infants	Deaths in 2005: 28,384. Almost 25% may have had respiratory causes: 2,234 deaths attributed to Sudden Infant Death Syndrome (SIDS), and 4,698 deaths attributed to short gestation and low birth weight.[53]	NO_2 PM[54,55]
	Lung cancer	Shortness of breath, wheezing, chronic cough, coughing up blood, pain, weight loss[56]	Smokers, adults	Deaths in 2005: 159,217. Leading cause of cancer mortality in U.S. among both men and women.[57]	PM[58,59,60]
CARDIOVASCULAR	Cardiac arrhythmias	Abnormal rate or rhythm of the heart; palpitation or fluttering; may cause fatigue, dizziness, lightheadedness, fainting, rapid heartbeat, shortness of breath, and chest pain[61]	Adults, hypertensives, diabetics, those with cardiovascular disease	Unknown	NO_2 $PM_{2.5}$[62]
	Acute myocardial infarction	Chest pain or discomfort; heart attack	Adults, diabetics, hypertensives	Deaths in 2006: 141,462.[63] Cases in 2006: 7.9 million.[64]	$PM_{2.5}$[65]
	Congestive heart failure	Shortness of breath, fatigue, edema (swelling) due to impaired ability of heart to pump blood; can result from narrowed arteries, past heart attack, and high blood pressure; can lead to death[66]	Adults, hypertensives, diabetics, those with cardiovascular disease	Deaths in 2006: 60,337.[67] Number of people living with heart failure: 5.7 million. New cases diagnosed each year: 670,000.[68]	$PM_{2.5}$[69]

Table ES.1: Coal's contributions to major health effects, *continued*

	Disease or condition	Symptoms or result	Most-vulnerable populations	Total disease burden (coal is a suspected contributing factor in an unknown number of cases)	Coal pollutants implicated
NEUROLOGICAL	Ischemic stroke	Artery supplying blood to the brain becomes blocked due to blood clot or narrowing;[70] may cause sudden numbness or weakness, especially on one side of body, confusion, trouble speaking, trouble seeing, trouble walking, dizziness, severe headache;[71] effects can be transitory or persistent	Elderly, hypertensives, diabetics	Deaths in 2005: 143,579. Number of strokes occurring each year: 795,000. NOTE: 87% of all strokes are ischemic; statistics are for all strokes.[72]	NO_2 $PM_{2.5}$ PM_{10} SO_2[73,74,75,76]
	Developmental delay	Reduced IQ; mental retardation; clinical impairment on neurodevelopmental scales; permanent loss of intelligence	Fetuses, infants, children	Babies born each year with cord blood concentrations of mercury >5.8 μg/L, the level above which mercury exposure has been shown to reduce IQ: 637,233 (15.7% of all babies born).[77]	Mercury[78]

dence include increases in global average land and ocean surface temperatures; increases in snow melt and receding glaciers; increases in the mean sea level; and changes in precipitation.[79] These global climate changes are already affecting human health. The World Health Organization estimated global warming to be responsible for 166,000 deaths in 2000, due to additional mortality from malaria, malnutrition, diarrhea, and drowning.[80]

In the future, global warming is expected to continue to harm human health. More frequent heat waves are projected to lead to a rise in heat exhaustion and heat stroke, potentially resulting in death, especially among elderly and poor urban dwellers. Declining air and water quality, an increase in infectious diseases, and a shrinking food supply are expected to contribute to disease and malnutrition, increase the migration of affected populations, and increase armed conflict and global instability. Table ES.2 (page xiii) describes the predicted health effects of global warming.

A continued reliance on coal combustion for electricity production will contribute to the predicted health consequences of global warming.

Carbon capture and sequestration (CCS) has been promoted as an effective way to keep CO_2 emissions out of the atmosphere, but substantial research and development are required before it can be used on the scale needed to mitigate global warming. Even then, the danger remains that CCS storage areas, whether underground or under the ocean, could leak, negating the value of CO_2 capture and storage. CCS also incurs other threats to health, including the danger of asphyxiation in the case of a large-scale CO_2 leak and the acidification of ocean waters. Moreover, the application of CCS would require continued coal mining, transportation, combustion, and waste storage, thus prolonging the emission of coal's toxic pollutants that harm human health.

POLICY RECOMMENDATIONS

The U.S. is at a crossroads for determining its future energy policy. While the U.S. relies heavily on coal for its energy needs, the health consequences of that reliance are multiple and have widespread and damaging impact. Coal combustion contributes to diseases already affecting large portions of the U.S. population, including asthma, heart disease, and stroke, thus compounding the major public health challenges of our time. Coal

combustion also releases significant amounts of carbon dioxide into the atmosphere. Unless we address coal, the U.S. will be unable to achieve the reductions in carbon emissions necessary to stave off the worst health impacts of global warming. Based on that assessment, PSR finds it essential to translate our concern for human health into recommendations for public policy.

- Emissions of carbon dioxide should be cut as deeply and as swiftly as possible, with the objective of reducing CO_2 levels to 350 parts per million, through two simultaneous strategies:
  - Strong climate and energy legislation that establishes hard caps on global warming pollution coming from coal plants.
  - The Clean Air Act (CAA). Carbon dioxide and other greenhouse gas emissions from coal plants have been designated pollutants under the CAA. The EPA should be fully empowered to regulate carbon dioxide under the CAA so that coal's contribution to global warming can be brought to an end.

Unless we address coal, the U.S. will be unable to achieve the reductions in carbon emissions necessary to stave off the worst health impacts of global warming.

- There should be no new construction of coal-fired power plants, so as to avoid increasing health-endangering emissions of carbon dioxide, as well as criteria pollutants and hazardous air pollutants.
- The U.S. should dramatically reduce fossil fuel power plant emissions of sulfur dioxide and nitrogen oxides so that all localities are in attainment for national ambient air quality standards.
- The EPA should establish a standard, based on Maximum Achievable Control Technology, for mercury and other hazardous air pollutant emissions from electrical generation.
- The nation must develop its capacity to generate electricity from clean, safe, renewable sources so that existing coal-fired power plants may be phased out without eliminating jobs or compromising the nation's ability to meet its energy needs. In place of investment in coal (including subsidies for the extraction and combustion of coal and for capture of carbon and other pollutants), the U.S. should fund energy efficiency, conservation measures, and clean, safe, renewable energy sources such as wind energy, solar, and wave power.

These steps comprise a medically defensible energy policy: one that takes into account the public health impacts of coal while meeting our need for energy. When our nation establishes a health-driven energy policy, one that replaces our dependence on coal with clean, safe alternatives, we will prevent the deterioration of global public health caused by global warming while reaping the rewards in improvements to respiratory, cardiovascular, and neurological health.

Table ES.2: Predicted health effects of global warming

Predicted human health effects	Contributing factors	Global warming mechanism	Most-vulnerable populations
Heat cramps, heat syncope, heat exhaustion, heat stroke	Heat waves	• Greenhouse effect	Children, the elderly, urban dwellers, those with underlying conditions such as cardiovascular disease, obesity, and respiratory disease
Diarrhea spread by water-borne bacteria including E Coli, Shigella, and cholera	Flooding, infrastructure damage	• Increase in extreme weather events and storm surges • Sea level rise	Children most vulnerable to death from diarrheal disease
Drowning	Flooding	• Increase in extreme weather events and storm surges • Sea level rise	Children, the elderly
Exacerbations of asthma, chronic obstructive pulmonary disease, and other respiratory diseases	Worsening air quality, heat waves	• Greenhouse effect • Heat increases production of ground-level ozone • Heat increases electricity demand and resulting particulate emissions from fossil fuel combustion • Airborne allergens (such as pollen) predicted to increase with global warming	Children, the elderly, those with preexisting respiratory disease
Infectious diseases: Malaria, dengue fever, yellow fever, West Nile virus, Lyme disease, and other insect-borne infections, as well as rodent-borne infections	Increased ranges and populations of disease-carrying insects and rodents	• Warming climate expands geographic range of insect and rodent vectors • High temperatures boost reproductive rates, lengthen breeding season, and increase bite frequency of insect vectors • High temperatures boost parasite development	Children, those with impaired immune systems, the developing world
Heart disease, heart attacks, congestive heart failure and other cardiovascular diseases	Worsening air quality	• Heat increases production of ground-level ozone • Heat increases electricity demand and resulting particulate emissions from fossil fuel combustion	Adults and the elderly
Hunger, malnutrition, starvation, famine	Reduced crop yields; crop damage; crop failure; disruptions in forestry, livestock, fisheries	• Changes in the water cycle leading to drought • Heat decreases reproductive lifecycle of some major food crops • Expanded range of some insect pests • Increase in extreme weather events • Changes in ecology of plant pathogens • Loss of agricultural land due to sea level rise	Children, the poor
Mass migration; violence; war	Societal instability; infrastructure damage; reduced crop yields	• All of the above	Children, the elderly, those with other underlying medical conditions
Mental health problems	All of the above	• All of the above	Varied

See sources on page xvi.

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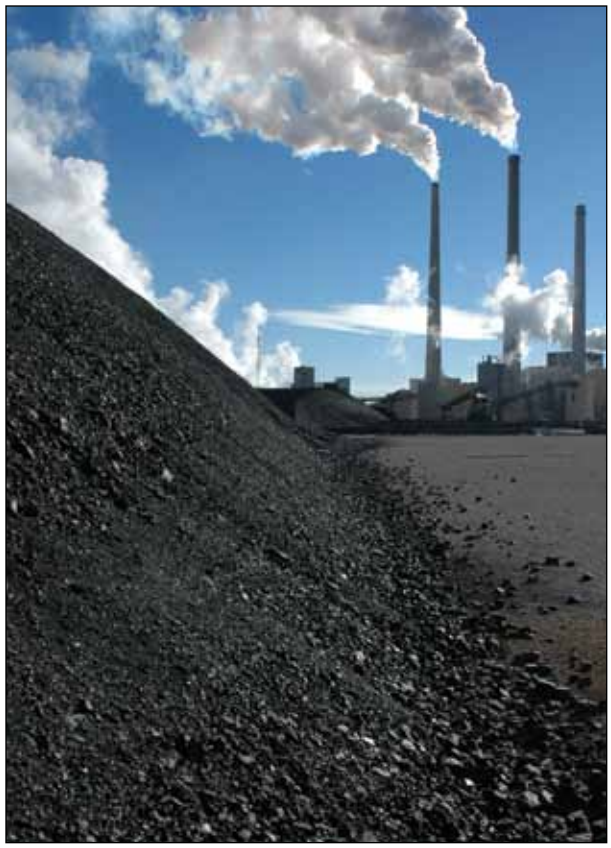
1. *Introduction*

Almost half of the energy used to generate electricity in the United States comes from burning coal, as shown in Figure 1.1. Coal is a major component of the economy and forms the center around which political, economic, health, and environmental considerations coalesce. The U.S. holds extensive coal reserves, although how much of that coal is accessible at a commercially viable cost is subject to debate. The high-end estimate 491 billion tons, which would be enough to last as much as 250 years at the current rate of consumption, earned the U.S. the title of the "Saudi Arabia of Coal."[1] In 2006, the electric power industry burned 1.026 billion tons of coal (see Figure 1.2). The electric industry currently plans to build as many as 100 new coal plants, adding to the approximately 600 large coal-burning power plants already in existence.

Using coal has a variety of major adverse impacts on health. Mining, transporting, burning, and disposing of the products of coal combustion all place human health at risk. With the passage of time, more and more adverse health effects have been attributed to the increasing reliance on coal. Studies of the health effects of hazardous air pollutants date clearly to 1872 with the publication of *Air and Rain: the Beginning of Chemical Climatology* by Robert Angus Smith. Since then, there have been a number of sentinel events that link episodes of severe air pollution to a variety of illnesses.[2] In October, 1948, almost half of the 14,000 residents of Donora, Pennsylvania were sickened when



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atmospheric conditions trapped toxic emissions from a nearby smelter: 20 died and 400 required hospitalization. In 1952, the infamous "killer fog" in London, lasting four days, sent death rates and hospital admissions soaring. Overall hospital admissions increased by 43%; those due to respiratory diseases rose by 163%. Almost 12,000 deaths

were attributed to this environmental disaster caused, in part, by burning coal.

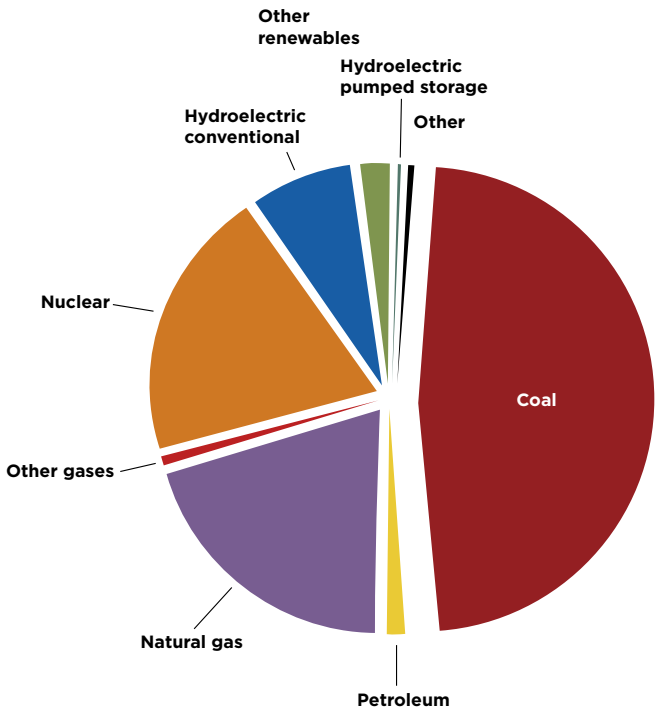
The link between burning coal and adverse health was made strikingly clear in Dublin, Ireland in the 1990s.[3] Because of increases in the cost of fuel oil in the 1980s, Dubliners switched from oil to bituminous coal to heat their homes and provide hot water. Subsequent increases in air pollution were associated with an increase in in-hospital deaths due to respiratory diseases. This led the Irish government to ban the marketing, sale, and distribution of bituminous coal on September 1, 1990. In the year that followed, black smoke concentrations declined by 70% (35.6 µg/m^3), respiratory deaths fell by 15.5%, and cardiovascular deaths fell by 10.3%. Approximately 450 lives were calculated to be saved that year by this measure, and hundreds of acute illnesses were prevented. Although burning coal was not the only cause of these illnesses, burning coal was clearly a major factor in the production of the complex mixture of airborne pollutants that had protean adverse effects on human health.

For each TerraWatt hour of electricity generated by coal, 24.5 deaths are expected in addition to 225 serious illnesses and 13,288 minor illnesses.

Many of coal's pollutants were identified by the U.S. Environmental Protection Agency in its 1998 report Report to Congress.[4] This report identified as many as 67 different hazardous air pollutants (HAPS) emitted from coal plants, but did not address particulates or oxides of nitrogen and sulfur (NO_X and SO_X), now referred to as criteria pollutants. Particulates, mercury, NO_X, SO_X, and the pollutants they give rise to, such as ozone, are now recognized as posing the greatest threats to health, and are the focus of much of this report.

Recent peer-reviewed reports provide estimates of the morbidity and mortality associated with burning coal. European data reported by Markandaya and Wilkinson show that for each TerraWatt hour of electricity generated (1 TWh = 10^{12} Watt hours), 24.5 deaths are expected (95% CI = 6.1–98) in addition to 225 serious illnesses (95% CI = 56.2–899) and 13,288 minor illnesses (95% CI = 3,322–53,150).[5] Burning lignite, a softer form of coal that yields more pollutants than bituminous coal, raises these numbers to 32.6 deaths (95% CI = 8.2–130), 298 serious illnesses (95% CI = 74.6–1,193), and 17,676 minor illnesses (95% CI = 4,419–70,704). To give these data perspective, consider the fact that nearly half of the 4,160 TWh of electricity generated in the United States in 2007 came from coal-fired power plants.[6] If these estimates are applied to the U.S., as many as 50,000 deaths per year may be attributable to burning coal. Although differences in population density between Europe and the U.S. are substantial and there are large boundaries on the 95% con-

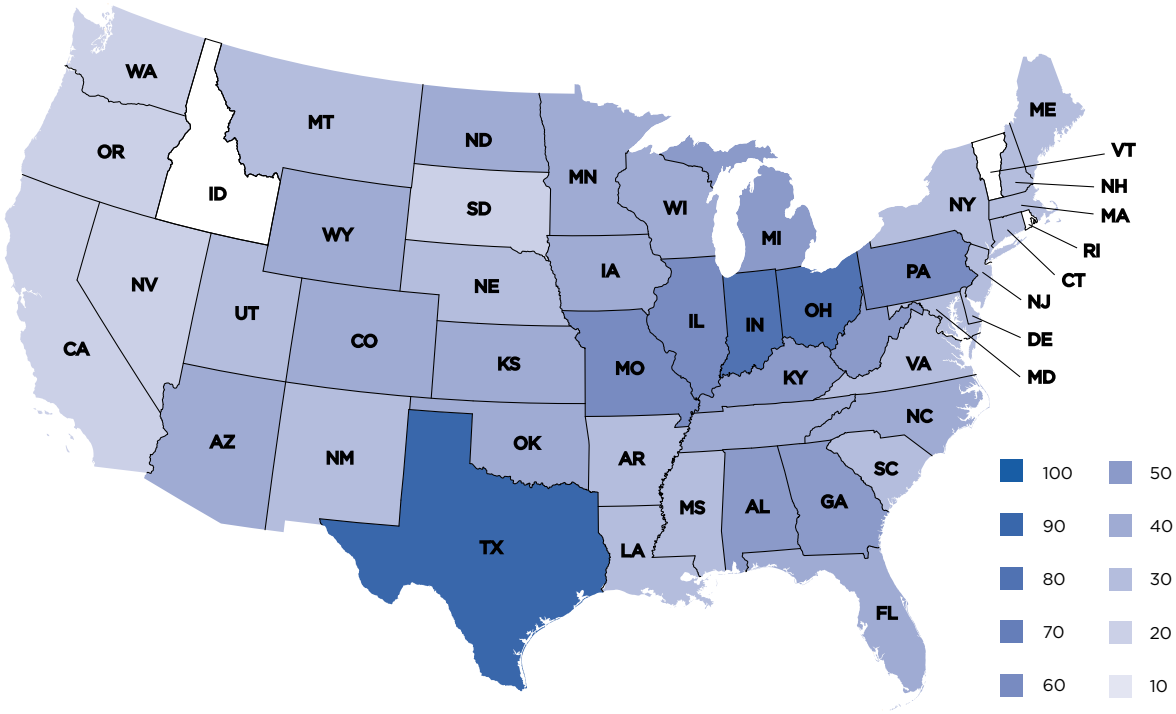
Figure 1.1: Sources of energy used for generation of electricity, 2006



Source: U.S. Energy Information Administration. Available from http://www.eia.doe.gov.

Figure 1.2: Coal consumption by U.S. utilities, 2006 (millions of tons)

By-state coal consumption by coal fired power plants. Texas led the nation, with states in the Ohio Valley that are close to coal fields and water transportation following closely behind.



Source: U.S. Energy Information Administration. Available from: http://www.eia.doe.gov/cneaf/coal/page/acr/table26.html.

fidence limits associated with these data, it is clear that burning coal has major adverse health effects.

In seeking to describe relationships between health and any single pollutant or any single source of the pollutant, notably burning coal, difficulties arise due to multiple sources of the pollutant in question and multiple health impacts. This is a particular issue with regard to SO_x, NO_x, and particulates, as there are many important sources of these pollutants in addition to burning coal. This is less of a problem in regard to mercury, where coal is the acknowledged largest single source of emissions. Thus, in this report we draw on literature that goes beyond that in which authors limit themselves to coal as the sole source of the pollutant in question.

In describing the health effects of coal combustion, this report utilizes an organ-system approach rather than a pollutant-based review. By considering coal's impact on the respiratory system, the cardiovascular system, and the central nervous system, we replace a piecemeal approach with a fuller and more integrated assessment of coal's overall effect on human health. To the best of our knowledge, this approach has not been taken in previous reviews of coal's health implications. To minimize bias, whenever possible we cite contemporary peer-reviewed medical literature and reports published by governmental agencies such as the U.S. Environmental Protection Agency and the Department of Energy. We hope that this report will provide physicians, other healthcare providers,

policy-makers, and concerned citizens with the information they need to make informed choices that affect the future of burning coal to produce electrical energy.

NOTES

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2. *Coal's Life Cycle*

Although people have burned coal for hundreds of years, the demand for coal exploded during the industrial revolution. Initially, coal powered the steam engine and therefore became the essential fuel for transportation during the nineteenth century, when steamships and railroads flourished. By fueling the steam shovel, coal became the vehicle for its own excavation. By the middle of the 1800s, coal replaced charcoal in the production of iron and steel, thus filling another key role in driving industrialization. Coal became a source of energy for the generation of electricity at the end of the 1800s.

Oil eventually replaced coal as the fuel of choice in the transportation industry. However, coal has once again become the dominant source of energy for the generation of electricity. Because more than 25% of the world's recoverable coal reserves are in the U.S. and because it is cheap, there has been a recent resurgence of coal as an energy source among utilities.[1] This modern coal boom is exemplified by the dozens of new coal plants currently in the planning or construction stage.

Today coal is the predominant source of energy used to produce electricity. Almost half of the energy used to generate electricity in the U.S. in 2007 came from coal, mined in such states as Wyoming, West Virginia, Kentucky, and Pennsylvania.[2] In addition to its major role in the generation of electricity, large amounts of coal are used by the steel industry. According to the World Coal Institute, almost 70% of global steel production is dependent on coal.[3]

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Coal is formed from fossilized prehistoric plants subjected to heat and pressure over millions of years. Coal is classified into four main types, or ranks, based on moisture and carbon content: lignite, sub-bituminous, bituminous, and anthracite (see Table 2.1). High-carbon coals produce the most energy when burned and low-carbon coals produce the least. Lignite is the lowest rank of coal, having the highest moisture content and the lowest energy content. Sub-bituminous coal is the next highest rank, with a lower moisture content and higher carbon content than lignite. Harder, black coals are higher in rank and include bituminous coal, the most abundant form of coal in the U.S., and anthracite, the hardest, richest in carbon and the rarest. Impurities such as sulfur and heavy

Table 2.1: The ranks of coal

Rank	Appearance	Percent carbon content	Uses	Percent of world reserves	Percent of U.S. production	Largest U.S. producers
Lignite	Brown, soft, flaky	25–35	Power generation	17	7	Texas, North Dakota
Sub-bituminous	Brownish-black, soft	35–45	Power generation, cement manufacture, industrial uses	30	44	Wyoming (Powder River Basin)
Bituminous	Black, hard	45–86	Power generation, cement manufacture, industrial uses	52	49	West Virginia, Kentucky, Pennsylvania
Anthracite	Black, hard, glossy	86–97	Domestic/industrial uses	1	Less than 0.5	Pennsylvania

metals are incorporated into coals as they are formed and are released when coals are burned or cleaned.

Electricity generation provides many benefits worldwide, and is synonymous with economic development, higher standards of living, and increased life expectancy.[4] However there are major health costs associated with the use of coal. Detrimental health effects are associated with every aspect of its life cycle, including mining, hauling, preparation at the power plant, combustion, and the disposition of post-combustion wastes. This section reviews in brief the human health effects of coal's life cycle.

MINING

Coal is extracted from underground and surface mines. The two main types of underground mines in the U.S. are longwall mines and room-and-pillar mines. In longwall mines, long sections of coal are removed without the use of supporting structures. This may lead to the subsidence of the land above. In room-and-pillar mines, sections of rock are not excavated (the "pillars") in order to provide structural support for the adjoining areas where all the coal is removed (the "rooms"). Both types of mines involve excavating shafts hundreds of feet deep, the installation of elevators, massive conveyance machinery, and air circulation technology.

Surface mining accounts for 69% of the coal mined in the U.S.[5] Used when the coal seam is close to the surface (less than 200 feet deep), it is cheaper than underground mining and often high-yield. In this method, vegetation, topsoil, and rock are blasted and removed down to the level of the coal seam, which is then mined. The top ten coal-producing mines in the U.S. are surface mines in the Powder River Basin of Wyoming.[6] "Mountaintop removal" is the name given to another type of surface mining, used to reach coal seams in mountainous terrain. It involves blasting down to the level of the coal seam—often hundreds of feet below the surface—and depositing the resulting rubble in adjoining valleys.

Coal mining leads U.S. industries in fatal injuries.[7] According to the National Institute for Occupational Safety and Health, the 2006 fatality rate in coal mining was 49.5 per 100,000 workers, more than 11 times greater than that in all private industry (4.2 per 100,000).[8] There were 47 occupational fatalities in coal mining in 2006, 34 in 2007, and 30 in 2008.[9] Underground coal mining is more dangerous than surface mining. Of 47 coal mining fatalities in 2006, 37 occurred in underground mining operations. The nonfatal injury rate in mining, of 3.9 per 100 full time workers in 2001, compares favorably to other private sector workers, where the average incidence rate of nonfatal injury was 5.4 in 2001.[10]

Coal mining is also associated with chronic health problems among miners. Black lung disease is caused by inhalation of respirable coal mine dust, which causes lung tissue scarring.

Although technology and prevention strategies have improved incidence and mortality rates in the past century, black lung disease still disables large numbers of ex-miners and claims many lives each year.[11] According to the National Institute of Occupational Safety and Health, black lung disease has been responsible for approximately 10,000 deaths in the past 10 years.

In addition to the miners themselves, communities proximate to coal mines may be adversely affected by mining operations. Injuries and even deaths may result from physical damage to surrounding communities due to blasting at surface mines and subsidence of underground mines. Surface mining also destroys forests and groundcover, leading to flood-related injury and mortality, as well as soil erosion and the contamination of water supplies. Rubble, or "overburden," is deposited on the surface, destroying plants and animals and introducing into the food web trace minerals and metals once deeply buried. One study of West Virginians found that people living in high coal-producing counties had higher rates of cardiopulmonary disease, chronic obstructive pulmonary disease, hypertension, lung disease, and kidney disease compared to people living in low coal-producing counties,[12] raising the possibility that coal mining operations may exacerbate a range of chronic health conditions among people living in nearby communities.

Coal mining operations may exacerbate a range of chronic health conditions among people living in nearby communities.

Flooding and contamination of water supplies are of particular concern in Appalachia, where mountaintop removal mining is widespread. The Environmental Protection Agency estimated in 2005 that mountaintop removal mining had adversely impacted 1,200 miles of streams in a study area that included parts of Kentucky, West Virginia, Virginia, and Tennessee. These 1,200 miles represented 2% of streams in the study area. The study further concluded that 724 miles of streams had been directly buried by valley fill related to mountaintop removal mining through 2001.[13] There are no official current estimates of the extent of the practice, but one advocacy organization estimated that by 2005, more than 450 mountain summits had been destroyed by mountaintop removal mining.[14] The human health effects of burying streams under piles of rubble have not been quantified, but include flood-related injury and mortality and contamination of drinking water and surface water resources with arsenic and other pollutants.[15]

After removal of coal from a mine, threats to public health persist. When mines are abandoned, rainwater reacts with exposed rock to cause the oxidation of metal sulfide minerals. These reactions generate acid and release contaminants such as heavy metals into the surrounding water system.[16] Red, orange, or yellow sediments in streams near abandoned mines are markers for this acidic mine drainage. The degraded water resulting from acid mine drainage renders the water undrinkable, and can corrode culverts and bridges.[17]

WASHING AND TRANSPORT

Coal is usually washed before it is transported to power plants to separate it from soil and rock impurities. Washing uses polymer chemicals and large quantities of water, and creates a liquid waste called slurry or sludge that must be stored. The slurry is the consistency of cement, and in addition to water, mud, and polymer chemicals, it contains heavy metals such as arsenic and mercury that are common in mined rock. Mine operators construct dams to impound the slurry in ponds, or inject it back into closed mines. Both slurry disposal strategies—the construction of surface impoundments and underground injection into closed mines—may leach chemicals into groundwater supplies. This is an aspect of mining that has not been examined closely.[18] In addition, both of these waste storage strategies can leak or break. Impoundment failures in the past have caused death and injury, including the 1972 Buffalo Creek, West Virginia, impoundment failure that killed 125 people and

injured 1,000. More recently, an impoundment breach in 2000 of about 250 million gallons of slurry near Inez, Kentucky, disrupted local water supplies but did not cause injuries or deaths.[19] Slurry injected underground into old mine shafts has the potential to release arsenic, barium, lead, and manganese into nearby wells, contaminating local water supplies.

Once coal is mined and washed, it must be transported to power plants. Coal is hauled to plants by train, truck, barge, and conveyor. Trains are the most economical way to move coal long distances and play the largest role in coal transport. In 2005, railroads accounted for 70% of coal shipments to power plants.[20] Together, railroad engines and trucks release over 600,000 tons of nitrogen oxide and 50,000 tons of particulate matter into the air every year in the process of hauling coal,[21] largely through diesel exhaust. Diesel engines currently produce approximately 1.8 million tons of NOx and 63,000 tons of small particles (less than 2.5 microns in diameter) each year.[22] These emissions adversely impact many organ systems, as this report will detail. Coal trains and trucks also release coal dust into the air as they move, degrading air quality and exposing nearby communities to dust inhalation.[23]

COMBUSTION

It is during the combustion phase of coal's lifecycle that our dependence on coal energy exacts the greatest toll on human health. Coal combustion releases over 70 harmful chemicals into the environment and contributes significantly to global warming (see Table 2.2). This section describes the pollutants emitted by coal combustion.

Coal combustion creates both solid and gaseous byproducts. Gas byproducts are emitted into the atmosphere through smokestacks. Some solids go into the atmosphere as well. Other solids are left behind at the plant as solid waste, also called coal ash. Some of the pollutants entering the air stay in the atmosphere for long periods; others fall to the earth and in turn pollute soil and water bodies. Some substances are not directly harmful but undergo chemical reactions in the atmosphere that create harmful secondary pollutants.

The pollutant composition of coal varies according to the geologic conditions of its formation. For example, plants that once lived and died in sea water formed coal with high sulfur content, while plants buried under fresh water formed low-sulfur coal. Thus, coals from different ranks and even from different mines differ not only in heat production and carbon content but also in pollutant composition. Such differences may affect local air quality concerns, as power plants may produce different pollution emissions depending on which coals are being burned.[24]

Notwithstanding local differences in pollutant composition, coal combustion causes pollution nationwide. Though coal supplies roughly 50% of the nation's electricity, it produces a disproportionate share of electric utility-related pollution. Coal plants emit approximately 87% of total utility-related nitrogen oxide pollution, 94% of utility-related sulfur dioxide pollution, and 98% of all utility-related mercury pollution.[25] Even across economic sectors, coal plants are responsible for a large share of human-caused air pollution: they are the single largest source of sulfur dioxide, mercury, and air toxic emissions and the second largest source of nitrogen oxide pollution.[26,27] Coal combustion is also responsible for more than 30% of total U.S. carbon dioxide pollution, contributing significantly to global warming.

Criteria air pollutants are a class of ubiquitous, harmful pollutants designated under the Clean Air Act. They are the only pollutants for which the EPA sets legal limits, called the National Ambient Air Quality Standards, on the amounts allowed in ambient air. These standards are based on health risk considerations. There are six criteria pollutants: nitrogen oxides, ozone, sulfur oxides, particulate matter, lead, and carbon monoxide. Coal combustion produces significant quantities of nitrogen oxides, sulfur oxides, and particulate matter, and contributes to the production of ground-level ozone.

Coal-fired power plants are second only to automobiles as the largest source of nitrogen oxide

Table 2.2: The health effects of power plant pollutants

Pollutant	What is it?	How is it produced?	Health effects	Most vulnerable populations
Ozone	Ozone is a highly corrosive, invisible gas	Ozone is formed when nitrogen oxides (NOx) react with other pollutants in the presence of sunlight.	Rapid shallow breathing, airway irritation, coughing, wheezing, shortness of breath. Makes asthma worse. May be related to premature birth, cardiac birth defects, low birth weight, and stunted lung growth.	Children, elderly, people with asthma or other respiratory disease. People who exercise outdoors.
Sulfur Dioxide (SO_2)	SO_2 is a highly corrosive, invisible gas. Sulfur occurs naturally in coal.	SO_2 is formed in the gases when coal is burned. SO_2 reacts in the air to form sulfuric acid, sulfates, and in combination with NO_x, acidic particles.	Coughing, wheezing, shortness of breath, nasal congestion and inflammation. Makes asthma worse. SO_2 gas can destabilize heart rhythms. Low birth weight, increased risk of infant death.	Children and adults with asthma or other respiratory disease.
Particulate Matter (PM)	A mixture of small solid particles (soot) and tiny sulfuric acid droplets. Small particles are complex and harmful mixtures of sulfur, nitrogen, carbon, acids, metals, and airborne toxics.	Directly emitted from coal burning. Formed from SO_2 and NO_x in the atmosphere.	PM crosses from the lung into the bloodstream resulting in inflammation of the cardiac system, a root cause of cardiac disease including heart attack and stroke leading to premature death. PM exposure is also linked to low birth weight, premature birth, chronic airway obstruction and remodeling, and sudden infant death.	Elderly, children, people with asthma.
Nitrogen Oxides (NO_x)	A family of chemical compounds including nitrogen oxide and nitrogen dioxide. Nitrogen occurs naturally in coal.	NO_x is formed when coal is burned. In the atmosphere can convert to nitrates and form fine acidic particles. Reacts in the presence of sunlight to form ozone smog.	NO_x decreases lung function and is associated with respiratory disease in children. Converts to ozone and acidic PM particles in the atmosphere.	Elderly, children, people with asthma.
Mercury	A metal that occurs naturally in coal.	Mercury is released when coal is burned.	Developmental effects in babies that are born to mothers who ate contaminated fish while pregnant. Poor performance on tests of the nervous system and learning. In adults, may affect blood pressure regulation and heart rate.	Fetuses and children are directly at risk. Pregnant women, children, and women of childbearing age need to avoid mercury exposure.
Carbon Dioxide	Coal has the highest carbon content of any fossil fuel.	Carbon dioxide is formed when coal is burned.	Indirect health effects associate with climate change including the spread of infectious disease, higher atmospheric ozone levels, and increased heat- and cold-related illnesses.	People of color, children, people with asthma.

Source: Clean Air Task Force. Cradle to grave: the environmental impacts of coal. June 2006.

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pollution, producing 18% of total U.S. nitrogen oxide emissions.[28] Nitrogen oxides are respiratory irritants.[29] They also pose a serious health risk as ozone precursors. Ground level ozone, also known as smog, is formed when nitrogen oxides react with volatile organic compounds in the presence of heat and sunlight. According to the American Lung Association, 175.4 million Americans live in counties with unhealthy ozone levels, representing more than half of the total U.S. population.[30] Ground level ozone is one of the nation's most pervasive air pollutants, and is particularly harmful to children and the elderly.

Sulfur occurs naturally in coal. Upon combustion, sulfur dioxide, a respiratory irritant, is emitted from coal plants and once in ambient air forms acid rain and particulate pollution. Coal-fired power plants are responsible for two thirds of the nation's sulfur dioxide emissions.[31]

Particle pollution is a complex combination of solids and aerosols suspended in the ambient air. It is categorized by researchers by size: PM_{10}, less than 10 microns in diameter, and $PM_{2.5}$, less than 2.5 microns in diameter. (By comparison, a human hair is about 70 microns in diameter.) Particle pollution is linked to asthma attacks as well as cellular inflammation, a risk factor for a range of chronic diseases. It has been estimated that coal plants in the U.S. will release 217,000 tons of PM_{10} and 110,000 tons of $PM_{2.5}$ in 2010.[32] These emissions estimates do not include secondary particle pollution, formed by the condensation of atmospheric gases such as oxides of nitrogen and sulfur with other pollutants, many of which are also released by coal plants.

The criteria pollutants produced by coal combustion carry large costs to society. The National Research Council has estimated the external costs associated with emissions of nitrogen oxides, sulfur dioxide, and PM from coal-fired power plants in the U.S. at $62 billion in 2005.[33]

In addition to the emissions of criteria pollutants, coal combustion is also a major source

of Hazardous Air Pollutants (HAPs), a class of harmful pollutants for which emissions limits, as opposed to allowable ambient air levels, are set by the EPA. These emissions limits are dictated by the technologies available to control pollution instead of by health risk considerations. There are 189 HAPs designated under the Clean Air Act.

In EPA smokestack tests released in 1998, coal plants were found to emit 67 different HAPs, many of which are known or probable human carcinogens, neurotoxins that can harm brain development, and reproductive toxins. These 67 HAPs include arsenic, beryllium, cadmium, chromium, lead, manganese, mercury, nickel, hydrogen chloride, hydrogen fluoride, acrolein, dioxins, formaldehyde, and radionuclides.[34] Based on exposure and risk estimates, the EPA identified four coal-related HAPs as posing potential risks to human health: mercury, dioxins, arsenic, and nickel. Mercury is the HAP of greatest concern emitted through coal combustion, due to its impacts on the nervous system. In 2007, electric utilities were responsible for more than 70% of all mercury air emissions.[35] Almost all of this mercury came from coal combustion.

Table ES.1 (see pages x–xi) describes the major health effects linked to coal combustion emissions. These health effects damage the respiratory, cardiovascular, and nervous systems and contribute to four of the top five leading causes of death in the U.S.: heart disease, cancer, stroke, and chronic lower respiratory diseases. These health effects are discussed further in subsequent sections. Although it is difficult to ascertain the proportion of this disease burden that is attributable to coal pollutants, even very modest contributions to these major causes of death are likely to have large effects at the population level, given high incidence rates.

POST-COMBUSTION WASTES

Coal's health effects extend beyond combustion. The potential hazards posed by post-combustion wastes are not a new problem, but one that has received little attention. In 2007, in response to complaints by interest groups, the EPA surveyed a number of sites where coal ash slurry, the residue left after burning coal, is stored. It found that damage to human health or the environment was inflicted at nine of the sites and potential damage was present at another 25.[36] A subsequent risk assessment showed that toxic residues from coal ash storage sites had migrated into water supplies and threatened human health at approximately 24 sites.[37] Then in December, 2008, a spill of approximately one billion gallons of coal ash slurry in Tennessee inundated hundreds of acres and threatened to contaminate drinking water and waterways with toxic metals, including lead and arsenic. Thus, coal poses risks to health from the point it is extracted from the ground, through combustion, and even afterwards as a toxic waste.

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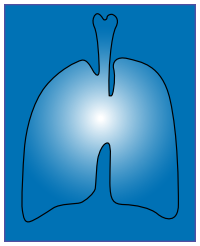
3. *Coal's Effects on the Respiratory System*



Virtually all airborne pollutants gain access to the body via the respiratory tract. Thus, it is no surprise that this important system is affected significantly by pollutants discharged into the atmosphere by electrical utilities that burn coal. These effects fall into several classes: *de novo* production of a condition, such as asthma, that did not exist prior to an exposure; an exacerbation of a previously-existing illness, again, such as asthma; and the development or progression of a chronic illness such as asthma, lung cancer, chronic obstructive pulmonary disease (COPD), and emphysema.

Data from the California Children's Health study have shown that air pollutants have clinically and statistically significant adverse effects on lung development.[1] In this prospective study, 1759 children were enrolled when they were in the fourth grade, when they were approximately 10 years old, and followed until age 18. Various measures of lung function were made periodically and correlated with their exposure to various pollutants. During normal development, the amount of air that can be forcibly exhaled in one second (FEV_1) increases with age. After controlling for various factors that could potentially confound the results, the investigators found that the FEV_1 failed to increase as predicted among children exposed to NO_2, acid vapor, and $PM_{2.5}$. Using a reduction of FEV_1 to 80% or less of the predicted value, children exposed to the highest levels of particulates were almost five times more likely to fall into the abnormal range than those with the lowest exposures. This impact on lung development is likely to be an additional risk factor for the subsequent development of other pulmonary diseases, such as asthma and chronic obstructive pulmonary disease.

ASTHMA

Asthma is a chronic disease of the lungs characterized by inflammation and narrowing of the airways. Patients with asthma experience recurrent episodes of dyspnea (shortness of breath), a sensation of tightness in the chest, wheezing, and coughing that typically occurs at night or early in the morning. Airway inflammation in asthmatics

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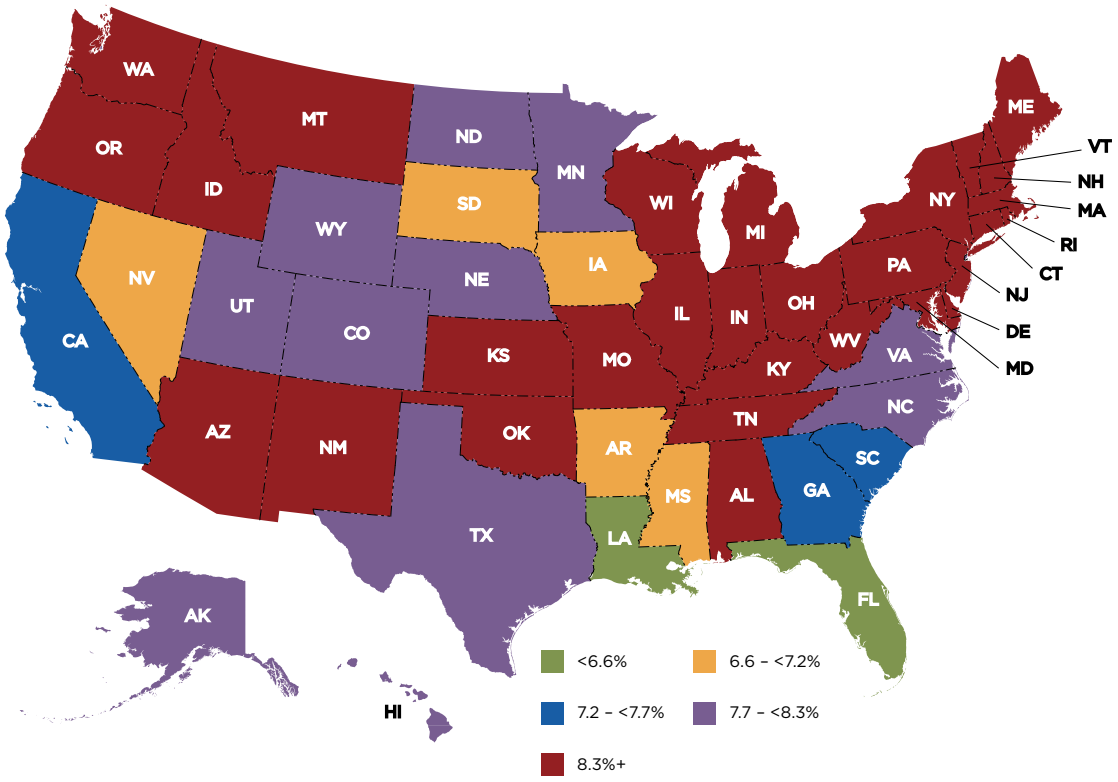
causes swelling that narrows a bronchial tree that has been previously sensitized to inhaled irritants, including many air pollutants. Exposure to an inhaled irritant causes further narrowing of the airways and the production of mucus that makes airways even narrower. During severe attacks, the lungs fail to perform their task of exchanging carbon dioxide, produced by metabolic processes in the body, for oxygen. This can lead to hypoxia (low blood oxygen level), hypercarbia (high blood carbon dioxide level), and respiratory acidosis (acidification of the blood caused by carbon dioxide retention) that may, in turn, cause cardiac arrhythmias and death. There are about 22 million asthmatics in the U.S., including 6 million children.[2] The Centers for Disease Control and Prevention report that the number of persons with asthma increased by 84% from 1980 to 2004. As shown in Figure 3.1, more than half of the states report that 8.6% or more of its inhabitants have asthma. These high-asthma states are clustered in the northeast and Midwest.

During an asthma attack, the airway is constricted due to inflammation and contraction or spasm of the muscles that surround the airway. This is associated with swelling of the tissues of the airway caused by triggers, or stimuli, which in turn cause an immune response. Asthmatics are more sensitive to these triggers than non-asthmatics, a condition known as hypersensitivity. There are many triggers, including dust, smoke, pollen, and volatile organic compounds. Some of the

Figure 3.1: CDC asthma prevalence by state

Adult self-reported current asthma prevalence rate by state, Behavioral Risk Factor Surveillance System 2007



Note: Ranges are based on quintiles of the overall prevalence estimates from year 2000 data.

Source: Air Pollution and Respiratory Health Branch, National Center for Environmental Health, Centers for Disease Control and Prevention

OXIDATIVE STRESS

The possibility that oxygen, or reactive forms of oxygen, might be toxic to certain cellular functions emerged in the 1950s. Subsequent research has focused on the importance of highly reactive forms of oxygen, known as oxygen free radicals, in biological systems. We now know that some of these free radicals exert critical controls over normal cellular metabolic process and cellular signaling. "Oxidative stress" is the term used to describe the physiological state characterized by an excessive concentration of these oxidizing free radical molecules.

Oxidative stress is one of several mechanisms implicated in the pathogenesis of diseases caused or made worse by pollutants formed by burning coal.

Free radicals are defined as atoms or molecules that contain at least one unpaired electron in an atomic or molecular orbit and are therefore unstable and highly reactive. Examples of reactive oxygen species (ROS) include the superoxide anion radical, formed by the addition of an electron to molecular oxygen (O_2); the hydroxyl radical, the neutral form of the hydroxl ion; and peroxyl radicals, the simplest of which is the hydroperoxyl radical, composed of one molecule of hydrogen and two molecules of oxygen. More complex peroxyl radicals have an organic group (abbreviated by an R) substituted for the hydrogen molecule.

ROS are a normal cellular constituent and play critical roles in the control of many cellular functions. However, the concentration of ROS can be increased through exposure to environmental substances such as air pollution, tobacco smoke, pesticides, and solvents. When the ROS concentration is excessive, these highly reactive molecules damage lipids, proteins, DNA, cell membranes, and other cellular components, producing oxidative stress, an important contributing factor in a variety of diseases.

In a contemporary review Valko, et al., summarize the current state of knowledge of oxygen free radicals and their importance in the production of a variety of diseases including cardiovascular and pulmonary disease, as well as other conditions including atherosclerosis, hypertension, rheumatoid arthritis, diabetes mellitus, neurodegenerative disorders such as Alzheimer's disease and Parkinson's disease, and normal aging.

Valko M, Leibfritz D, Moncol J, Cronin MTD, Mazur M, Telser J. Free radicals and antioxidants in normal physiological functions and human disease. Int J Biochem and Cell Biology 2007;39:44–84.

pollutants discharged by coal fired power plants may act as triggers and produce an asthma attack. These pollutants include sulfur dioxide, nitrogen oxides, and particulate matter. In addition, the carbon dioxide emissions from coal accelerate global warming, which is likely to increase the concentration in air of pollen from some plants, such as ragweed, and thereby contribute to the development of additional asthma attacks.

Genetic variability accounts for some of the differences in the sensitivity of individuals to asthma triggers.[3] Genetic studies have shown differences in the susceptibility to ozone that are due to polymorphisms (subtle differences in genes that control the expression of a trait) in the genes responsible for dealing with oxidative stress. Oxidative stress is created when oxygen ions, free radicals, or other reactive species are produced in excess of the body's ability to remove these molecules. Oxidative stress may be an important mechanism for the production of a variety of diseases (see text box). Genetic polymorphisms responsible for controlling the inflammatory response also increase an individual's susceptibility to the respiratory effects of ozone. Thus, the probability that an individual will develop asthma depends on exposure to a trigger, such as ozone, and the individual's susceptibility to that trigger, i.e., a complex combination and interaction between genetic and environmental factors. For a review of the genetic susceptibility

to the effects of air pollutants, such as ozone, particulates, nitrogen dioxide, and sulfur dioxide, on respiratory function see Yang, et al.[4]

Children appear to be more susceptible to the development of pollution-related asthma attacks than adults. There are several explanations for this increase in susceptibility. According to a review by Bateson and Schwartz, the susceptibility of children to the effects of air pollution is multifactorial and includes the following.[5] 1) Children have different rhythmic patterns of breathing than adults. 2) They are predominantly mouth-breathers, thereby bypassing the filtering effects of the nasal passages. This allows pollutants to travel deeper into the lungs. 3) They have a larger lung surface area per unit weight than adults. 4) They spend more time out of doors, particularly in the afternoons and during the summer months when ozone and other pollutant levels are the highest. 5) Children also have higher ventilation rates. i.e., volume of air per minute per unit body weight compared to adults. 6) When active, children may ignore early symptoms of an asthma exacerbation and fail to seek treatment, leading to attacks of increased severity. There are other factors that are important. The diameter of the airways in children is smaller than in adults and therefore airways may be more susceptible to the effects of the airway narrowing that is characteristic of asthmatic attacks. These factors, combined with the possible adverse impact of pollutants on lung development and the immaturity of enzyme and immune systems that detoxify pollutants, may all contribute to an increase in the sensitivity of children to pollutants produced by burning coal.[6]

CHRONIC OBSTRUCTIVE PULMONARY DISEASE (COPD), CHRONIC BRONCHITIS AND EMPHYSEMA

Asthma is a reversible condition. When permanent damage to the airway occurs, a chronic obstructive airway condition is present.

COPD is a condition characterized by narrowing of the airway passages. Unlike asthma, these changes are permanent rather than reversible. Like asthma, exposures to pollutants that produce an immunological response are critical in the pathogenesis of the condition. The response in larger airways is referred to as chronic bronchitis. A cough that produces sputum is characteristic of chronic bronchitis. In the alveoli, the inflammatory response leads to a destruction of tissue, or emphysema. These two conditions usually co-exist. Exacerbations of COPD may be triggered by pollutants or infections. Although current and ex-smokers account for 80–85% of all patients with COPD, exposure to air pollutants, including those that are produced by burning coal, plays an important role in the pathogenesis of acute exacerbations and the development of COPD.

PULMONARY INFLAMMATION AND AIR POLLUTANTS

Inflammation of pulmonary tissues is a critical element in the pathophysiology of illness caused by air pollution. Reactive oxygen species, such as free radicals and oxygen ions, appear to be central to this process. To avoid some of the difficulties associated with in vitro studies, several investigators have studied the response to particulate pollution in experimental animals. Roberts, et al., instilled particles into the lungs of rats treated with a compound (dimethylthiourea) that is believed to blunt the response to reactive oxygen species.[7] After treatment, the lungs of the animals were lavaged (rinsed with saline) and biomarkers of pulmonary injury were measured. Treated animals exhibited less evidence of damage to their lungs such as toxicity to cells, cytokine gene expression (genes that control cellular communications), pulmonary inflammation and other markers of pulmonary injury. In a subsequent study, Rhoden, et al., instilled standardized urban air particles (active agent) or saline (placebo control) into the lungs of rats.[8] Half of the animals in each group were treated with a reactive oxygen species inhibitor. Pretreatment with the inhibitor blocked the deleterious effects of the particles, as shown by reductions in several markers of pulmonary inflammation. These studies show that common

air pollutants such as particulates interfere with a variety of basic cellular mechanisms and dispose to the development of inflammation, a process that leads to diseases such as asthma, COPD, and emphysema.

These two studies are representative of many that have been performed using a variety of agents and techniques. Although performed in animals and not humans, they are consistent with a larger body of scientific evidence that helps establish a cause-and-effect relationship between particulates and pulmonary disease. As noted above, inflammation is a critical element in the pathogenesis of attacks of asthma and exacerbations of COPD. It matters little whether the inflammation is caused by particulates or other pollutants.

Additional evidence to support the hypothesis that air pollutants produce oxidative stress is derived from many studies. Recently, Fitzpatrick, et al., studied 65 children with severe asthma, including 35 with a reduction in baseline airway function as shown by a forced expiratory volume of less than 80% of that predicted, i.e., their ability to move air rapidly out of the lung was impaired.[9] Bronchoalveolar lavage (rinsing the airway with saline) was performed and metabolites and enzymes related to oxidative stress were measured. In the asthmatics, the concentration of glutathione, an antioxidant that protects cells from free radicals, was reduced and the concentration of the oxidized form (glutathione disulfide) was increased. This made the children less able to withstand oxidative stress and more susceptible to the development of an asthmatic attack.

OZONE, AIR POLLUTION, AND ASTHMA

Ozone, a highly reactive gas that consists of three atoms of oxygen (O_3), is formed by the reaction of volatile organic compounds (VOCs) with oxides of nitrogen (NO_X) in the presence of sunlight. Coal combustion does not produce ozone directly, but both the NO_X and the VOCs released by coal plants are essential contributors to the formation of ground-level ozone, the primary ingredient in urban smog. Ozone is a powerful oxidizing agent

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that irritates the lungs at concentrations typically encountered in urban settings, particularly in summer months. There are many studies linking increases in ozone to asthma and other pulmonary diseases (see Trasande and Thurston for review[10]).

One of the most compelling studies linking ozone with asthma exacerbations was performed by Gent, et al.,[11] who examined the effects of relatively low ozone levels on asthmatic children. Those authors conducted a prospective cohort study of 271 children younger than 12 who had physician-diagnosed asthma. The children were divided almost equally into groups who did or did not use daily maintenance medications. Rigorous statistical techniques were used to examine the relationship between ozone levels below EPA standards, respiratory symptoms, and the use of rescue medications as charted by the children's mothers on daily calendars. The authors found a significant association between ozone levels and symptoms, as well as the use of rescue medications in the children who used daily maintenance medications. No significant relationships were found between ozone levels and symptoms or medication use in the children who did not take daily maintenance medications. Thus, it appears that the threat to children posed by ozone is greatest among those with severe asthma, even when ambient ozone levels fall within the limits set by the EPA to protect public health.

Peel, et al., studied the relationship between a one standard deviation increase in ambient air pollutant levels and emergency room visits for various respiratory problems, including asthma.[12] They found the strongest association between increases in 24-hour PM_{10} levels, 24-hour increases in 10–100 nm particle concentrations, and 1-hour NO_2 concentrations and asthma attacks that occurred six to eight days after the peak. There were shorter delays between peaks of PM_{10}, ozone, NO_2, and carbon monoxide and emergency room visits for upper respiratory infections. During warm months, there was a 2.6% increase in asthma admissions after a 25 ppb increase in the ozone concentration. To give the 25 ppb increase perspective, the EPA eight-hour exposure standard in 2008 was set at 75 ppb. This study is one of many that establish a statistically rigorous link between a peak in the concentration of an air pollutant and the onset of a disease or disease symptom.

Increases in the risk of death from respiratory causes, including sudden infant death, were correlated with the concentration of PM_{10} and NO_2.

The evidence linking ozone levels to the development of asthma is less compelling than that linking ozone to asthma exacerbations. Gilmour, et al., reviewed five studies that address this issue.[13] A Dutch study of over 4,000 children enrolled at birth and followed for two years, focused on NO_2 and $PM_{2.5}$ attributed to traffic, found small but statistically significant associations between pollutant peaks and the development of symptoms of asthma.[14,15] Although this study focused on traffic as the source of the pollutants, burning coal can't be ignored as a source of NO_2 and $PM_{2.5}$. There were similar results from a second study of children in that age group from the Netherlands, Germany, and Sweden. The Children's Health Study of more than 6,000 children from southern California evaluated a wide range of ozone, particulates, oxides of nitrogen, and acids.[16] A significant association between ozone and asthma was confined to those children who participated in three or more sports. This result may be the consequence of the increases in the amount of air breathed per unit of time associated with exercise and the consequent increases in exposure to pollutants. Gilmour, et al., conclude that the results of all five of the studies they reviewed "support a modest increase in the risk for air pollution in relation…to asthma."[17]

The increase in susceptibility to pollutants among children appears to translate into pollution-related increases in infant mortality. Ritz, et al., reported increases in the risk of death from respiratory causes, including sudden infant death, with rises in the concentration of carbon monoxide, PM_{10}, and NO_2.[18] Bateson and Schwartz also cite a study reporting between 4 and 7 fewer infant deaths per 100,000 live births with a reduction in the concentration of total suspended particles of 1 $\mu g/m^3$.[19] To give this number perspective, Pope, et al., reported that there was a mean reduction in the $PM_{2.5}$ concentration of 6.52 $\pm$ 2.9 $\mu g/m^3$ in major U.S. metropolitan areas in the time interval between 1979–83 and 1999–2000.[20]

AIR POLLUTION AND COPD

Smoking tobacco is the most important risk factor for the development of COPD. Most authors report that approximately 85% of all cases of COPD can be attributed to this single, preventable cause. Data that have emerged during the past several years have shown that there is a smaller but important link between air pollution, including pollutants produced by burning coal, and the subsequent development of COPD exacerbations.

In a study of the residents of Helsinki, Finland, where coal-derived air pollutants account for a relatively small portion of total pollutant levels, pooled asthma and COPD emergency room visits increased on those days when there were increases in $PM_{2.5}$, coarse particles, and gaseous pollutants.[21] The Atlanta, Georgia, study of Peel, et al., found that when NO_2 or carbon monoxide increased by one standard deviation, emergency room visits for COPD increased by 2–3%.[22] Finally, in a study of hospitalization rates among Medicare enrollees, a 10 $\mu g/m^3$ increase in the concentration of $PM_{2.5}$

particles was associated with a same-day increase in COPD admissions of 2.5% (95% confidence interval (CI) = 2.1–3.2%).[23] These three studies of three different populations using different criteria all link increases in air pollutants to increases in exacerbations of COPD. Although they did not focus on pollutants derived exclusively from the combustion of coal, the pollutants they studied included those produced by coal burned by electrical utilities as well other sources.

LUNG CANCER

The National Cancer Institute estimates that in 2008 there were 215,020 new cases of lung cancer, the leading U.S. cancer killer in both men and women, with 161,840 deaths. While smoking tobacco, radon and other radioactive gases, secondhand smoke, asbestos, arsenic, nickel compounds, and other airborne organic compounds have been identified as risk factors for developing lung cancer, data from three large epidemiological studies show that air pollution may also be a risk factor.

First among these was a study of Seventh Day Adventists who lived in California.[24] This cohort of over 6,300 non-smoking white adults was followed from 1977 to 1992 and monitored for the development of lung cancer. These data were combined with monthly ambient air pollution data in various zip codes. For men, the interquartile range (the middle 50% of the range) increase for ozone of 100 ppb was associated with an increase in the relative risk (RR) for lung cancer of 3.56 (95% CI = 1.35–9.42). Lung cancer increases were also associated with significant increases in PM_{10} (RR = 5.21, 95% CI = 1.94–13.99) and for SO_2 (RR = 2.66, 95% CI = 1.62–4.39). Smaller increases among women were also found, however only the association with SO_2 was statistically significant. The difference between men and women was thought to be due to greater exposures among men.

In the Harvard Six Cities study, lung cancer death was positively associated with air pollution. The adjusted mortality rate ratio due to lung cancer for the most to least polluted cities was 1.26 (95% CI = 1.08–1.47).[25,26]

Complementary data were found in the American Cancer Society study.[27] This epidemiological study began with 1.3 million adults in 1982. From that set, approximately 500,000 adults were matched with air pollution data for their appropriate metropolitan area and vital statistics data through the end of 1998. Fine particulate increases of 10 $\mu g/m^3$ were associated with an 8% increase in lung cancer mortality. Increases in the concentration of oxides of sulfur were also associated with increases in lung cancer mortality.

These three large prospective epidemiological studies provide evidence that air pollution, particularly that due to particulates and ozone, may affect mortality due to lung cancer. Since some of the pollutants studied are formed directly or indirectly as the consequence of burning coal, it is possible that burning coal places those exposed to coal-related pollutants at greater risk for developing lung cancer.

NOTES

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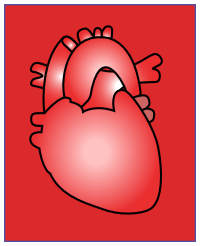
4. *Coal's Effects on the Cardiovascular System*



The American Heart Association (AHA), along with other organizations, has issued guidelines designed to aid health professionals as they seek to achieve primary and secondary prevention of the morbidity and mortality due to diseases of the cardiovascular system. Traditionally, these guidelines have focused on the control of hypertension, cholesterol levels, smoking, and other factors. More recently, these guidelines have been expanded to deal with lifestyle choices, such as diet, exercise, and avoidance of second-hand smoke. Controlling these risk factors has been the most important factor in the declines in death rates attributable to coronary heart disease over the past decades. However, because of accumulating evidence and a persistent concern that air pollutants are also linked to adverse cardiovascular health outcomes, the AHA convened an expert panel to evaluate this threat. The results of their deliberations, the single most authoritative review of this topic, were published in 2004.[1] In this section, we build on that prior publication, including studies published since it was written.

CARDIOVASCULAR DISEASE

Although death rates from coronary heart disease (CHD) have declined substantially during the past several decades, CHD remains a leading cause of death in the United States. Tables 4.1 and 4.2 summarize incidence rate and prevalence data from the National Heart Lung and Blood Institute 2009 Chartbook.[2] By any standard, the control of risk factors associated with CHD has important public health consequences even though the incidence and prevalence of CHD have fallen.

Figure 4.1 summarizes the pathophysiological mechanisms by which air pollutants, particularly particulate matter (PM), cause cardiovascular disease. Pulmonary inflammation and the presence of reactive oxygen species (ions, free radicals formed from oxygen) are both thought to be important mechanisms in the pathogenesis of cardiovascular disease. By convention, and for purposes of monitoring air to evaluate compliance with air quality standards, the PMs of greatest concern are those with a diameter of 2.5 μm or less ($PM_{2.5}$). These

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Table 4.1: Cardiovascular disease prevalence in the U.S. population, 2004 data

Hypertension	79,400,000
Coronary heart disease	15,800,000
Acute myocardial infarct	7,900,000
Angina pectoris	8,900,000
Congestive heart failure	5,200,00

Source: National Heart Lung and Blood Institute 2009

Table 4.2: Cardiovascular disease incidence rate and recurrence rate in the U.S. population, 2004 data (per year)

Myocardial infarct	1,200,000
First event	700,000
Recurrent event	500,000
Congestive heart failure	550,000
First event	550,000

Source: National Heart Lung and Blood Institute 2009

small particles are the most likely to penetrate deeply into the lungs, reach the alveoli, and initiate the pathophysiological sequences leading to acute and chronic manifestations of CHD.

The mechanisms depicted in Figure 4.1 suggest numerous possible therapeutic interventions. Reducing the exposure to airborne pollutants is the most obvious of these and forms the rationale for PSR's Code Black campaign to prevent the licensing and construction of new coal-fired power plants. Substantial efforts, beyond the scope of this report, are being invested in controlling the immunological and inflammatory responses and oxidative stress associated with the inhalation of pollutants.

IMMEDIATE IMPACTS OF AIR POLLUTANTS ON THE CARDIOVASCULAR SYSTEM

In a study supporting the hypothetical pathogenic mechanisms outlined in Figure 4.1, Brook, et al., evaluated the effects of fine particles and ozone on the diameter of the brachial artery in 25 healthy adults.[3] The behavior of brachial arteries is thought to be representative of the behavior of coronary and cerebral arteries. This double-blind, randomized, crossover study evaluated the cardiovascular response to a two-hour inhalation of fine particles (approximately 150 $\mu g/m^3$) and ozone (120 ppb). These concentrations, which are encountered routinely in urban settings, resulted in a significant reduction in the diameter of the brachial artery, implying narrowing of other arteries. In spite of this evidence, important questions remain, e.g., are these participants representative of the population at greatest risk (screened healthy controls versus patients with significant coronary artery disease)?

Animal studies are well suited to studying pulmonary inflammation and oxidative stress, mechanisms that may be important in cardiac disease pathogenesis. Roberts, et al., instilled particles into the lungs of animals pretreated with a drug (dimethylthiourea) believed to blunt the response to reactive oxygen species.[4] The treated animals showed less evidence of pulmonary injury, as evidenced by a blunted inflammatory response and other markers of pulmonary damage. There was also a reduction in the activity of genes controlling cytokines. Cytokines, molecules involved in cellular signaling and communication, are critical in the development and control of immunological responses. In a similar study, Rhoden, et al., instilled standardized urban air particles into the lungs of rats and measured the formation of superoxide ions, a reactive oxygen species.[5] Again, pretreatment with an inhibitor blocked the adverse effects of the particles, as shown by measured reductions in the level of several markers of lung inflammation. In a study of hyperlipidemic rabbits, Suwa, et al., found that a four-week exposure to PM_{10} was associated with acceleration of atherosclerosis, an increase in the turnover of cells in atherosclerotic

Figure 4.1: Pathophysiological mechanisms by which air pollution causes cardiovascular disease

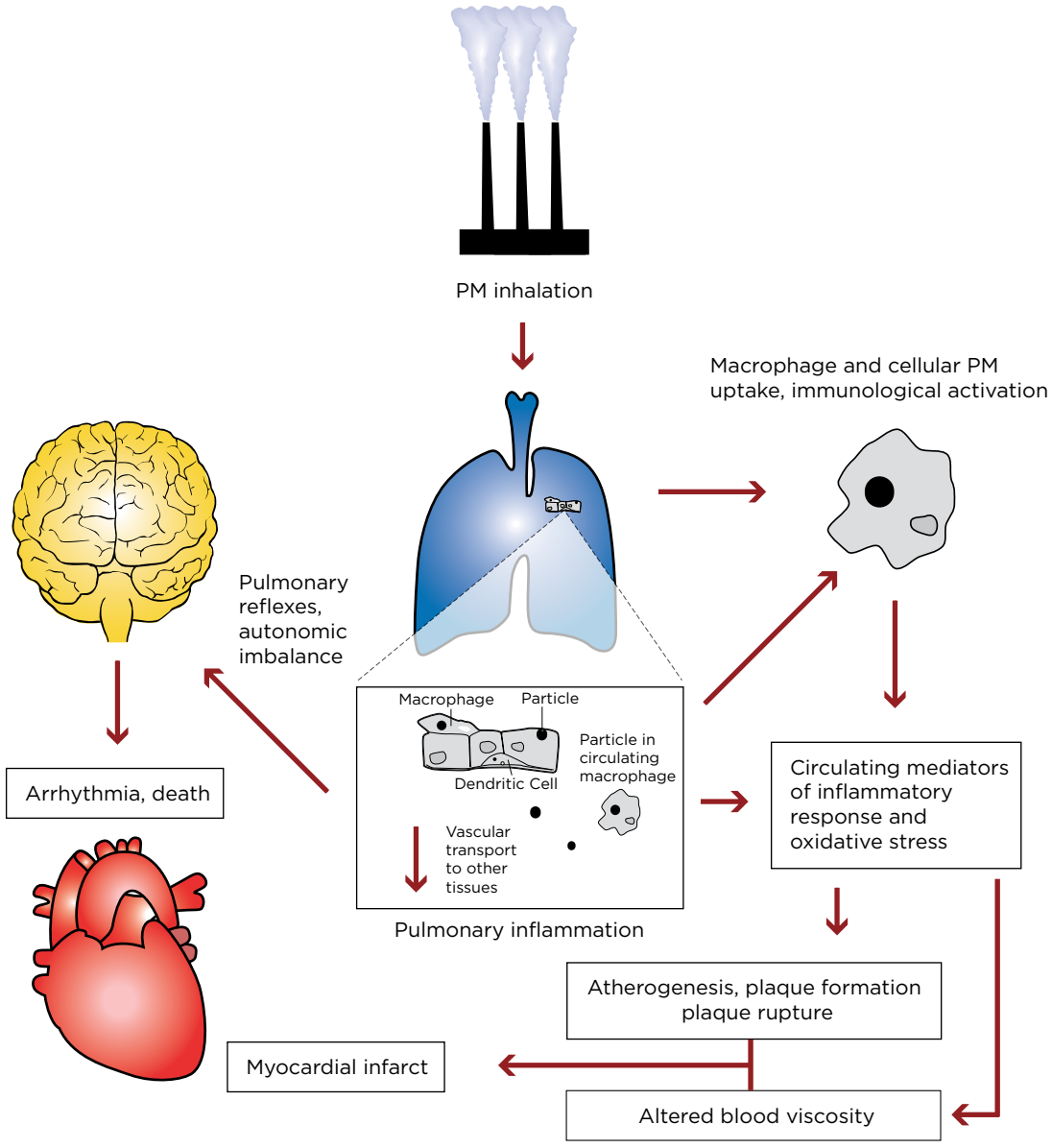

Figure legend:

Particles produced by burning coal are inhaled. Small particles, particularly those that are 2.5 μm or less in diameter, travel deep into the lungs, reaching the alveoli. Particles in the alveoli may have one of several fates: uptake by macrophages with subsequent stimulation of the inflammatory response and the immune system; uptake by dendritic cells, another pathway to immune system activation; or direct entry into the vascular compartment and subsequent transfer to other organs. Pulmonary reflexes, stimulated by inflammation, lead to imbalance of the autonomic nervous system and may potentiate cardiac arrhythmias such as ventricular fibrillation and atrial fibrillation. Inflammation and oxidative stress are important elements in the production and rupture of atherosclerotic plaques in coronary and cerebral arteries and may lead to myocardial or cerebral infarction. Increases in the viscosity of the blood also increase the risk of infarction of the heart or brain.

plaques, and an increase in the total lipid content of aortic lesions.[6] These selected studies show that pollutants produced by the combustion of coal have powerful effects on physiological processes that lead to disease endpoints, such as arterial occlusion and infarct formation.

Acute outcome studies typically focus on a single event, e.g., admission due to acute myocardial infarction or the discharge of an implanted cardioverter defibrillator (ICD). These events have a distinct time-of-occurrence that can found in hospital emergency room records or examination of defibrillator data extracted after a discharge. By examining these data in relationship to air quality data collected by monitoring stations, researchers have been able to examine the relationship between air pollution and hospital admissions for cardiovascular disease. Because of such studies, Peters, et al., hypothesized that there might be a link between transient increases in pollutant levels and therapeutic discharges of ICDs.[7] ICDs are permanently implanted in patients judged to be at risk for sudden death due to cardiac arrhythmias. These devices monitor heart rhythms continuously. When a rhythm disturbance such as ventricular fibrillation or ventricular tachycardia is detected, the ICD begins to pace or defibrillate the heart. Defibrillation is transient but quite painful, and patients are instructed to seek medical attention after an event. Modern ICDs typically include memory chips that store information for variable times. When the patient seeks medical attention after a discharge, technicians are able to retrieve relevant data, including the nature and time of the arrhythmia and the ICD discharge. Peters and her colleagues analyzed the ICD records from 100 events recorded in a single clinic in eastern Massachusetts and sought links between events and peaks in pollutant levels measured in that region. They considered daily average pollutant levels on the day of the event and one, two, and three days prior to the event. They found that an increase in the NO_2 concentration was followed by an increase in the probability of an ICD discharge two days later (odds ratio 1.8, 95% confidence interval (CI) = 1.1–2.9). Patients who experienced 10 or more ICD discharges (presumably an indication of more severe disease) exhibited associations with NO_2, CO, black carbon, and $PM_{2.5}$. Although they regarded this as a pilot study, they concluded that peaks in air pollution levels were associated with fatal or potentially fatal cardiac arrhythmias. They buttressed this claim by reviewing the results of animal studies linking pollutants to cardiac arrhythmias.

By examining how defibrillator discharges relate to air quality, researchers can explore how cardiovascular disease relates to air pollution.

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Peters and her colleagues also investigated the relationship between acute myocardial infarctions (MI) and air pollutants.[8] In this study, a total of 772 records of patient interviews conducted within four days of an acute MI (a step that minimizes recall bias) were evaluated in the context of air

pollutant concentrations. By pairing the data from the day of the MI with three other control sets measured at exactly the same time of the day (on days when the subject did not have an MI), patients served as their own controls. Compared to control periods, there was an increase in the probability of an MI in association with elevations in $PM_{2.5}$ levels measured two hours before the MI (odds ratio for an increase in $PM_{2.5}$ of 25 µg/m³ was 1.48, 95% CI = 1.09–2.02). In addition, there was a delayed response to a peak occurring a full day before an event (odds ratio for the same increase in $PM_{2.5}$ was 1.69, 95% CI = 1.13–2.34).

Two large studies using health outcomes such as mortality in relation to day-to-day changes in ambient air pollution levels have been critical in defining the health effects associated with pollutants. In a U.S. study, Dominici, et al., used ambient $PM_{2.5}$ concentrations and hospital admission rates in the Medicare National Claims History Files to look for associations between particulate levels and admissions for ischemic heart diseases, disturbances of heart rhythm, and congestive heart failure.[9] The data included 204 urban counties with a total of 11.5 million Medicare enrollees who lived an average of 5.9 miles from a $PM_{2.5}$ monitor. Using injuries as control, they found increases in all categories, with the largest found for congestive heart failure, where a 1.28% increase was found for an increase of 10 µg/m³ in $PM_{2.5}$ concentration. Increases in admissions for ischemic heart disease, heart failure, and disturbances of heart rhythm tended to be higher in those 75 years or older than in those 65–74. Additional details are shown in Table 4.3. The greatest effects were observed in the northeastern U.S. where coal-fired power plants are most plentiful. Although the increases in the rates appear small, on the order of a single percent, the large number of Medicare enrollees translates the result into a very large effect when measured in terms of total hospital admissions, patient morbidity and mortality, and the cost of health care and lost opportunities.

Katsouyanni, et al., reported the short-term effects of PM_{10} on the health of the residents of

Table 4.3: Percent change in hospitalization rate per 10 µg/m³ increase in $PM_{2.5}$ for all Medicare enrollees age > 65 years

Admission diagnosis	Lag days*	Percent rate increase (95% confidence interval)
Ischemic heart disease	2	0.44 (0.02–0.86)
Heart rhythm abnormality	0	0.57 (-0.01–1.15)
Heart failure	0	1.28 (0.78–1.78)

* Number of days between peak and greatest effect of $PM_{2.5}$

Source: Dominici F, Peng RD, Bell ML et al. Fine particulate air pollution and hospital admission for cardiovascular and respiratory diseases. JAMA 2006; 295(10):1127–1134.

29 European cities with a total population of over 43 million, extending over a period of approximately 5 years.[10] Unlike the U.S. study, they included all age groups. However, the results were remarkably similar to those observed in the U.S. Medicare population. They report a 0.6% increase (95% CI = 0.4–0.8%) in the daily number of deaths for a 10 µg/m³ increase in the PM_{10} concentration. They also found important modifiers of the PM_{10}-associated death rate, particularly with regard to NO_2 levels. Cities with high NO_2 concentrations had death rates that were approximately four times higher than found in cities with low NO_2 concentrations. Death rates in cities with warm climates were about 2.8 times higher than in cities with cold climates.

These examples extracted from the literature describing the effects of air pollution on acute morbidity and mortality are consistent: the small studies focused on individuals as well as large studies that rely on data extracted from large databases show adverse effects of pollutants on indicators of acute cardiovascular illness.

LONG-TERM IMPACT OF AIR POLLUTANTS ON THE CARDIOVASCULAR SYSTEM

Two studies linking the chronic effects of air pollutants on cardiovascular mortality are particularly relevant. The first of these is the Harvard Six Cities

study, reported by Dockery, et al.[11] This prospective cohort study followed over 8,100 adults for 14–16 years. The mortality rate in the most polluted city was 1.26% higher than the rate in the least polluted city (95% CI = 1.08–1.47%). This elevated rate persisted after controlling for important lifestyle confounders, including smoking cigarettes. Pope, et al., linked individual risk factor data from about 500,000 adults, metropolitan area air pollution data, and vital statistics and cause-of-death data. They found that an increase in the particulate concentration of 10 μg/m^3 was associated with a 6% increase in the risk of death due to cardiopulmonary causes. Because coal is a significant source of particulate pollution, these studies indicate that coal combustion has serious long-term impacts on the cardiovascular health of the U.S. population.

MITIGATING THE EFFECTS OF AIR POLLUTION

With the passage of the Clean Air Act in 1955 (with major revisions in 1970, 1977, and 1990), the U.S. embarked on a process to improve air quality in order to improve health. Pope, et al., evaluated the effects of the regulations and corresponding improvements in health in a recent paper.[12] In this study, the authors compared life expectancy in the late 1970s–early 1980s to life expectancy in the late 1990s–early 2000s in 211 counties in 51 metropolitan areas where fine particulate concentration data were available. They report a 0.61 + 0.20 year (+ standard error) increase in life expectancy after a decrease of 10 μg/m^3 in fine particle concentration. The two cities with the largest changes were Pittsburgh, PA, and Buffalo, NY, where major steel industries that consumed large amounts of coal were closed. A number of variables that could have confounded the results, such as smoking and socioeconomic class, did not have significant effects. This study showed that significant and measurable improvements in life expectancy followed the improvements in air quality mandated by the Clean Air Act.

NOTES

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5. *Coal's Effects on the Nervous System*

It is easy to understand that burning coal is likely to have an adverse impact on respiratory health: we inhale the products of combustion. It is less obvious that burning coal has important effects on the nervous system, particularly the brain. Cerebral vascular disease, i.e., stroke, and loss of intellectual capacity due to mercury are the two most important neurological consequences of burning coal.

THE BRAIN AND POLLUTION

The human brain is the organ that most clearly distinguishes us from other species. Our abilities to think abstractly, produce and enjoy music, art and literature, inquire about the nature of the universe, and a host of similar activities related to brain function are what makes us human. This uniquely human organ is also highly susceptible to disruption by what may appear to be relatively trivial acute or chronic abnormalities. Although the normal brain weighs 1,300–1,400 grams (about three pounds), the extremely high metabolic rate of the brain and the absence of significant energy stores within the brain mandate a high constant rate of blood flow in order to insure normal function and survival. When the body is at rest, between 15 and 20% of the cardiac output goes to the brain. Thus, even transient interruptions of the blood, oxygen, or glucose supplies to the brain may result in severe, permanent brain injury or brain death.

The complexity of the brain, coupled with its susceptibility to the effects of metabolic or physiological derangements or both, frequently leads to abnormalities of brain function that may be undetectable in an individual, but may have an enormous impact on the population as a whole. This, in turn, has public health consequences. This is illustrated in Figures 5.1 and 5.2, where the impact of a five-point decrement in IQ is depicted. The average IQ score is 100 and 95% of all individuals have IQ scores that fall between 70, the score below which one is considered to be retarded, and 130, the score above which one is considered to have superior intelligence. This is shown in Figure 5.1. Figure 5.2 demonstrates the effect of an across-the-board five-point decrement in IQ. For an individual, this

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Figure 5.1: Normal IQ distribution

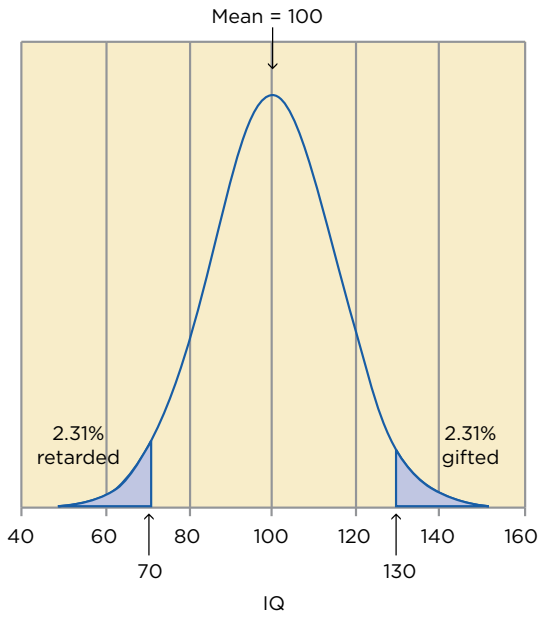
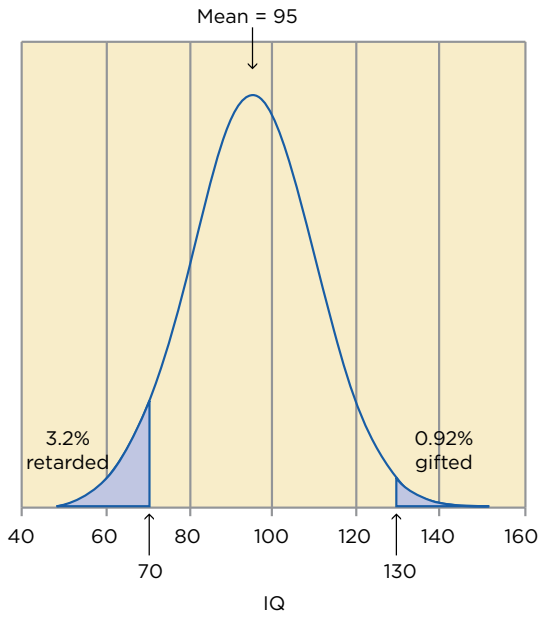


Figure 5.2: Change in IQ distribution with 5 point decrement



relatively small change would hardly be noticed. However, in a large population, substantial numbers of individuals are removed from the superior intelligence category and others are pushed down into the retarded category. The result is a smaller pool of individuals with outstanding intellects and a larger pool of individuals who require special resources to be able to function. It is this reality that makes it important to protect and preserve the brain's full potential.

CEREBRAL VASCULAR DISEASE

The same pathophysiological mechanisms that affect the coronary arteries and cause myocardial infarcts also apply to the arteries that nourish the brain, as shown in Figure 5.3. These common mechanisms include: stimulation of the inflammatory response in cerebral vessels leading to atherosclerotic plaque formation, rupture, and arterial occlusion; oxidative stress; and alterations in blood viscosity. In addition, arrhythmias may cause transient reductions of the cardiac output and cause hypoperfusion of the brain. The effects of reductions in the cardiac output are most prominent at the boundaries between major arteries (so-called watershed areas of the brain), and distal to sites of severe arterial stenosis.

The term stroke refers to a variety of acute cerebrovascular events including ischemic stroke, caused by occlusion of a cerebral artery by an atherosclerotic plaque or an embolus; cerebral hemorrhage, usually caused by rupture of a small artery in the brain; subarachnoid hemorrhage, often due to rupture of an aneurysm; or some other acute event due to a vascular cause. In the narrative that follows, unless otherwise stated, we will use the term stroke to mean an ischemic stroke caused by occlusion of a cerebral artery by an atherosclerotic plaque, the most common cause of stroke.

Although there have been major improvements in primary and secondary prevention of strokes in the past several decades, due to better care of

patients with hypertension and diabetes as well as improvements in smoking cessation efforts, stroke is still an important cause of death in the U.S. Current estimates indicate that the stroke death rate for men is 33.1 per 100,000 and 26.1 per 100,000 for women.[1]

Three large studies and several smaller studies have shown a correlation between air pollutants and acute strokes.

In their study of the relationship between fine particles ($PM_{2.5}$ or less) and hospital admission rates in the Medicare population, Domenici, et al., reported a 0.81% increase in the hospitalization rate for cerebrovascular disease (ICD 9 codes 430–438, 95% CI = 0.31–1.32%) for a 10 μg/m^3 increase.[2] This relationship was significant only on lag day zero (no lag between a peak and the admission) and not evident on lag days one and two. As with many of the other outcomes they considered in addition to cerebrovascular disease, this association was strongest in eastern parts of the U.S. when compared to western regions. This may be related to differences in the composition of PM due to the large number of coal-fired power plants in the east compared to the west.

In a second study of the Medicare population, Wellenius, et al., sought relationships between hospital admissions from 1986 to 1993 for ischemic and hemorrhagic strokes and increases in various air pollutants in nine major U.S. cities.[3] Admissions data were obtained from the Centers for Medicare and Medicaid Services and pollutant levels were obtained from the EPA. Pollutant levels are shown in Table 5.1. Note that PM_{10} levels were measured and not $PM_{2.5}$ levels as would be the case for more contemporary studies. They report that for an interquartile percentile increase in the PM_{10} concentration, there was a corresponding 1.03% increase in the admissions for ischemic stroke (95% CI = 0.04–2.04%) on the day of the increase. Similar results were observed for CO, NO_2, and SO_2 for ischemic strokes only. They did not find significant associations between pollutant levels and hemorrhagic strokes.

Figure 5.3: Pathophysiological mechanisms by which air pollution causes neurological disease

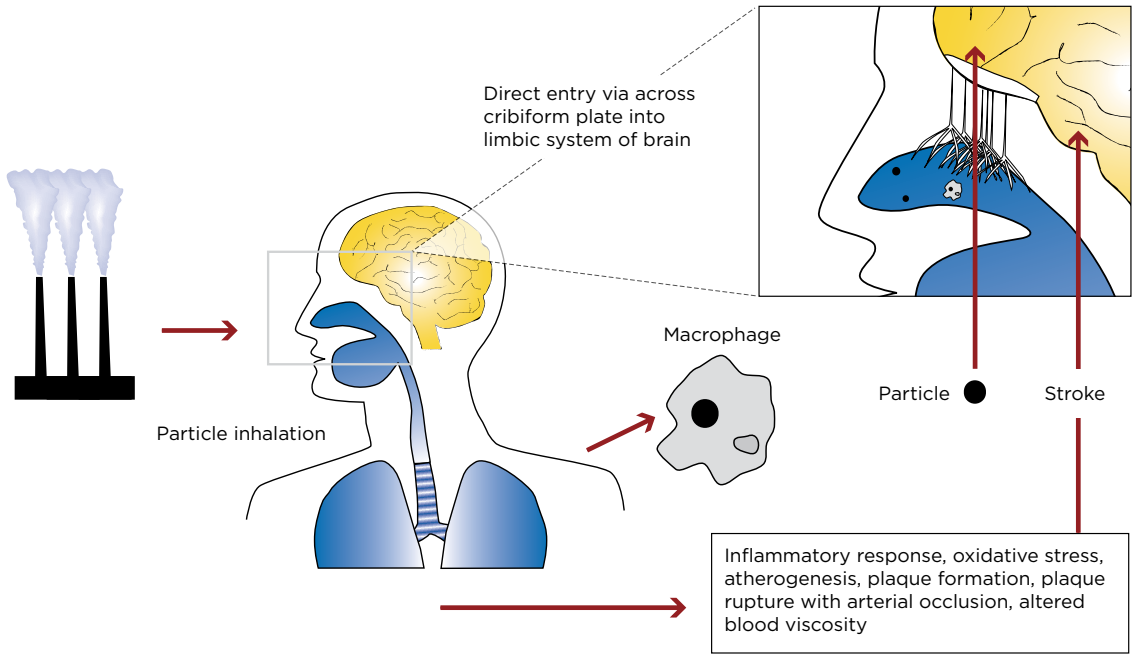


Table 5.1: Concentrations of various air pollutants in 9 U.S. cities

Pollutant	Percentile 25th	50th	75th
PM_{10} µg/m^3	18.88	28.36	41.84
CO ppm	0.73	1.02	1.44
NO_2 ppb	18.05	23.54	29.98
SO_2 ppb	3.57	6.22	10.26

Source: Wellenius GA, Schwartz J, Mittleman MA. Air pollution and hospital admissions for ischemic and hemorrhagic stroke among medicare beneficiaries. Stroke 2005; 36(12):2549–2553.

Finally, data from post-menopausal women enrolled in the Women's Health Initiative and collected in 2000 show that for each increase of 10 µg/m^3 in the $PM_{2.5}$ concentration, there was a 35% increase in the risk of a cerebrovascular event and an 83% increase in the risk of death from a cerebrovascular event.[4] The hazard ratio for the time to an acute cerebrovascular event, an indicator of relative risk, was reported as 1.35 (95% CI = 1.08–1.68). This observational study of post-menopausal women without a prior history of cardiovascular disease gains strength from the fact that the authors reviewed actual medical records, rather than relying on data from central databases. The restriction to this cohort of women loses some strength because the results may not be generalizable to men or younger women. They did not find any association between stroke and other air pollutants, including SO_2, NO_2, CO and O_3.

Two other studies conducted under more restricted circumstances have shown direct relationships between air pollutants and stroke. In a 2002 study of stroke mortality in Korea, Hong, et al., reported significant and increasing risk for death due to ischemic but not hemorrhagic stroke as same-day PM concentrations increased through four quartiles.[5] These authors also found significant temporal relationships between pollutants and stroke: same-day sulfur dioxide concentrations and ischemic stroke, a one-day lag between carbon monoxide and stroke, and a three-day lag between an ozone peak and stroke. In a more complex study of over 23,000 stroke admissions in Taiwan, Tsai, et al., reported significant positive associations between PM_{10}, NO_2, SO_2, CO, and O_3 when considered singly, and primary intracerebral hemorrhage and ischemic stroke admissions on days when the temperature was 20°C or greater.[6] On cooler days, the correlation between CO and ischemic stroke was the only factor that persisted. When they considered two pollutants together, there was a significant correlation between PM_{10} and NO_2 and both types of stroke.

In summary, even though a relatively small portion of all strokes appear to be related to concentration of PM, the fact that nearly 800,000 people in the U.S. have a stroke each year[7,8] makes the number of strokes attributable to PM a risk factor of importance. These studies emphasize the importance of measures designed to minimize PM concentrations in the air, including preventing the construction of new coal-fired power plants and developing and utilizing more effective technologies to reduce emissions from existing plants.

MERCURY

Coal contains trace amounts of mercury. When burned, this mercury evaporates and is emitted into the environment unless stringent control technologies are used to reduce those emissions. Coal-fired power plants are responsible for approximately one third of all emissions of mercury attributable to human activity, as shown in Table 5.2, making them the largest single source of mercury emissions. The 2007 Toxics Release Inventory listed point source releases of 7,935 pounds of mercury and 117,243 pounds of mercury compounds.[9] Point sources are stationary, single-site sources of a pollutant, such as a smoke stack at a power plant.

The mercury cycle is shown in Figure 5.4. Once mercury enters the atmosphere, it returns to the earth via rainfall, entering waterways. Mercury and other persistent toxicants in lakes and streams led various states, tribes, and territories to issue 3,221 advisories in 2004 urging caution when consuming fish from specific bodies of water, up from 3,089

Table 5.2: Anthropogenic sources of mercury

Source	Tons per year	Percent of total
Combustion	137.9	86.9
Electrical utilities	52.0	32.8
Municipal incinerators	29.6	18.7
Industrial boilers	28.4	17.9
Medical waste incinerators*	16.0	10.1
Other manufacturing	15.8	10.0

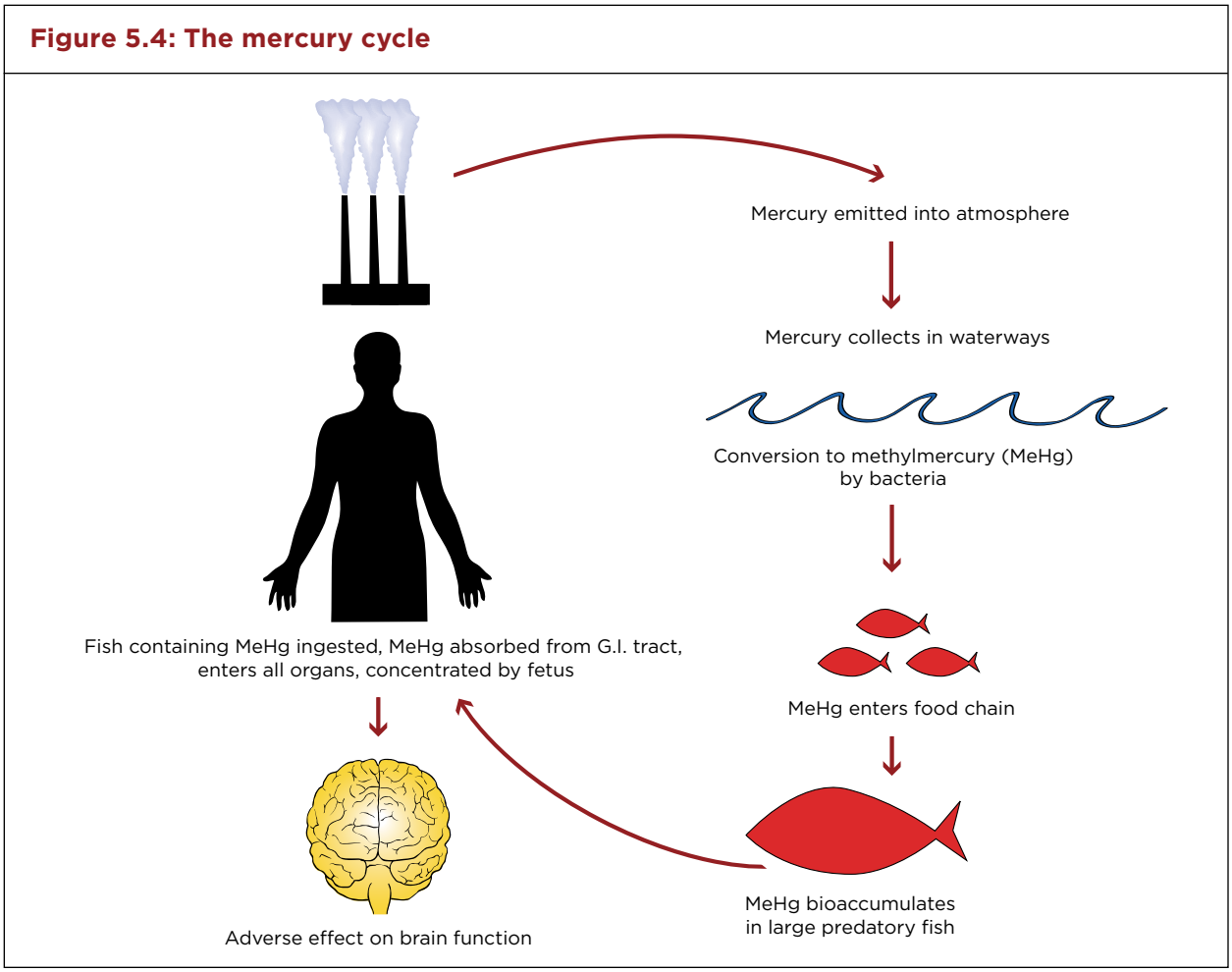
* Mercury emissions from medical waste incinerators are almost certainly lower at present due to regulations that have altered medical waste disposal methods.

Source: EPA Office of Air Quality Planning & Standards and Office of Research and Development. Mercury study report to Congress. Volume II: an inventory of anthropogenic mercury emissions in the United States; Dec 1997: EPA-452/R-97-004.

the year before.[10] In the water, bacteria convert elemental mercury into methylmercury (MeHg), a form that is persistent and bioaccumulates. The concentration of MeHg increases as it passes up the food chain, reaching high levels in large predatory fish. The fish with the highest mercury concentrations are large tuna, swordfish, king mackerel, and tile fish. Marine mammals that eat fish also may have a large mercury burden. Humans are exposed to coal-related mercury primarily through fish consumption.

Since mercury is recycled in the environment and substantial amounts are released during volcanic eruptions, opponents of stricter mercury controls attempt to downplay the importance of coal-related emissions. Nevertheless, minimizing or eliminating coal-related mercury emissions is an important and concrete action that can be taken

Figure 5.4: The mercury cycle



to prevent additional amounts of mercury from entering the environment and affecting health.

In the 1950s, outbreaks of mercury poisoning in Minamata and Niigata, Japan, were caused by eating fish contaminated with mercury from industrial discharges. As a result, there were 3,000 confirmed cases and 600 deaths. A second outbreak occurred in Iraq, caused by eating seed-grain that had been treated with a fungicide containing MeHg.[11] This caused 649 deaths among 6,530 affected individuals. Until recently, a dose-response curve, derived from these exposures and interpolated to more typical blood mercury levels, served as the basis for determining the permissible daily intake of mercury.[12] Because of deficiencies in these data and their analysis, alternate populations were examined in great detail.

Based on three large-scale, prospective studies of cohorts children exposed *in utero* to MeHg in the Seychelles, the Faroe Islands, and New Zealand, the National Research Council recommended establishing a benchmark dose of 58 µg/L of mercury in the cord blood of newborns. The benchmark dose is the lower 95% confidence interval for an estimated dose that results in doubling the prevalence of children with neurodevelopmental test scores that are in the clinically impaired range.[13] This somewhat arbitrary choice is thought to provide an adequate margin of safety and to provide a rational basis for regulatory actions. Based on the National Research Council recommendations, the EPA applied a tenfold safety factor, which is typical for EPA regulatory actions, and set the reference dose (RfD), the maximum tolerable daily dose, at 0.1 microgram of mercury per kilogram of body weight per day, the amount believed to lead to a mercury concentration of 5.8 µg/L mercury in cord blood.[14] To translate this standard into a practical form, it is necessary to know the maternal mercury blood level and the maternal-fetal mercury ratio. Fetal mercury levels are approximately 1.7 times those in the mother.[15]

Between 316,588 and 631,233 children are born in the U.S. each year with blood mercury levels high enough to cause lifelong loss of intelligence.

Based on the 1999-2000 National Health and Nutrition Examination Survey data,[16] approximately 15.7% of all American women of childbearing age were found to have blood mercury levels that would cause them to give birth to children with cord blood mercury levels of 5.8 µg/L mercury or more, i.e., above the target concentration. (Subsequent modeling by Stern suggested that the maternal:fetal ratio was overestimated in the earlier study.[17] However, direct measurements in a Swedish cohort suggest that the original ratio may be correct.[18]) Using these data and conservative estimates of blood mercury levels and their effect on intellectual performance, Trasande, et al., estimated that between 316,588 and 631,233 children are born in the U.S. each year with blood mercury levels high enough to impair performance on neurodevelopmental tests.[19] These authors further concluded that this lifelong diminution in intelligence costs society $8.7 billion per year (range $2.2–$43.8 billion in 2000 dollars). These cost estimates contrast sharply with others as low as $10 million dollars attributed to U.S. power plants by Griffiths, et al.[20]

HEALTH EFFECTS ON THE HORIZON

In prior sections, we have reviewed the peer-reviewed evidence published in leading medical journals that links pollutants produced by coal-burning power plants to diseases of the pulmonary, cardiovascular, and nervous systems. Emerging data that are based on smaller samples, and are therefore more speculative, suggest that there may be links between coal-derived pollutants and other diseases, such as Alzheimer's disease (AD) and diabetes mellitus, two of the most prevalent, costly, and debilitating chronic diseases of adults. If these early observations are upheld by more rigorous studies of large populations using contemporary epidemiological and statistical methods, such as those we refer to in earlier sections,

the public health implications will be enormous. Therefore, because of their possible importance, we will consider these potential links briefly.

ALZHEIMER'S DISEASE

The evidence linking air pollution and AD comes from studies comparing brains of dogs and humans living in highly polluted versus non-polluted cities in Mexico.[21,22] Animal data suggest that $PM_{2.5}$ crosses the nasal mucosa and enters the limbic system of the brain via the olfactory nerve.[23,24] Once in the brain, these PM cause inflammation and appear to lead to the deposition of amyloid, a neuropathological feature characteristic of AD. The authors suggest that "exposure to urban air pollution may cause brain inflammation and accelerate the accumulation of β-amyloid42, a putative mediator of neurodegeneration and AD pathogenesis." Similar findings were reported in animal experiments in which several strains of transgenic mice were exposed to PM.[25] The possible link between airborne pollutants and neurodegenerative diseases has not been firmly established. However it is potentially important because of the large and growing number of patients with AD and the disease's financial and societal impact.

DIABETES MELLITUS

The prevalence of diabetes mellitus (DM), particularly Type II DM, is increasing. In 2002, Lockwood observed a statistically significant relationship between the by-state prevalence of diabetes and the by-state air emissions of pollutants reported in the Toxics Release Inventory.[26]

In 2005, Brook postulated several mechanisms by which the inhalation of particulates might lead to the development of insulin resistance, a condition in which the body produces insulin but does not use it properly, thereby increasing the risk for the development of DM.[27] Again, the process begins with the inhalation of small particles that stimulate pulmonary inflammation and the generation of reactive oxygen species and oxidative stress. Subsequent pathways that involve the autonomic nervous system, the adrenal gland, and others are thought to lead to the development of insulin resistance and subsequently, to DM. This hypothesis gained credence with the publication of a paper linking NO_2 exposure with DM in women.[28] A positive relationship was found between NO_2 exposure and DM after controlling for several potential confounding factors such as age and body mass index. The authors conclude that their results "suggest that common air pollutants are associated with DM." That report used NO_2 levels as a surrogate marker for traffic-related air pollutants, including PM. Since substantial amounts of NO_2 and PM are emitted when coal is burned, there may be a link between NO_2 and PM derived from coal and DM.

DIABETES MELLITUS AND ALZHEIMER'S DISEASE

Converging evidence suggests that insulin resistance is a risk factor for a number of dementia-related conditions including Type II DM and impaired glucose tolerance, obesity, inflammation, ischemia, hypertension, cardiovascular disease, and abnormal lipid metabolism including hypercholesterolemia. To some extent, these associations blur the distinction between vascular causes of dementia and AD.[29] The putative mechanisms linking AD and DM include inflammation and oxidative stress. As discussed throughout this report, these two mechanisms are intimately related to exposure to various air pollutants, particularly fine particles, including those produced by burning coal.

SUMMARY

It is possible that the protean adverse health effects of burning coal may extend beyond those that are well established to include other common and costly chronic diseases. This possibility warrants vigilance and further investigation.

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6. *Coal, Global Warming, and Health*

COAL'S CONTRIBUTION TO GLOBAL WARMING

In previous sections we have discussed the relationship between coal plant pollutants and human health. Here, we address coal's contribution to global warming, and the likely impacts of that warming on human health.

With very high carbon dioxide emissions due to combustion, and significant methane emissions from mining activities, coal is a major contributor to the buildup of greenhouse gases in the atmosphere. These gases allow solar energy to reach the planet's surface but delay that energy's escape into space, effectively trapping heat in the lower atmosphere.[1]

Atmospheric concentrations of greenhouse gases have increased markedly since 1750 as a result of human activities, especially the combustion of fossil fuels. Carbon dioxide (CO_2) is the most abundant greenhouse gas, and almost all U.S. CO_2 emissions (close to 98%) are due to fossil fuel combustion.[2] The level of atmospheric CO_2 now far exceeds pre-industrial values: Whereas the preindustrial level was 280 ppm, it is now approximately 385 ppm[3] (see Figure 6.1). China emits the most greenhouse gas of any country, but the U.S. emits the most greenhouse gas per capita.[4] Historically, the U.S. is responsible for over one-quarter of all anthropogenic greenhouse gases emitted globally.[5]

Coal-fired power plants are a major greenhouse gas source in the U.S., accounting for more than one-third of our nation's CO_2 emissions (see Figure 6.2).[6] Within the electricity sector, coal generates roughly 50% of the electricity[7] yet emits over 80% of the sector's total emissions.[8] This disproportionate carbon footprint is due to the high carbon content of coal relative to other fossil fuels such as natural gas.

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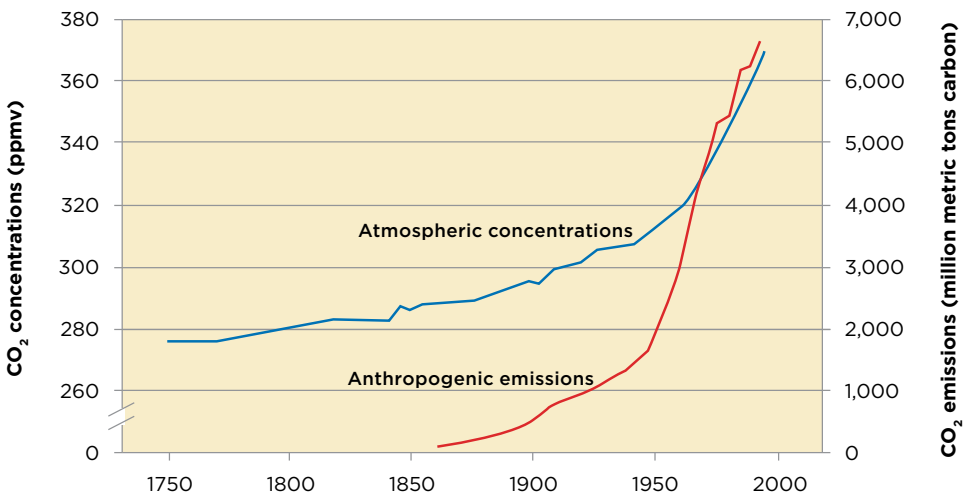
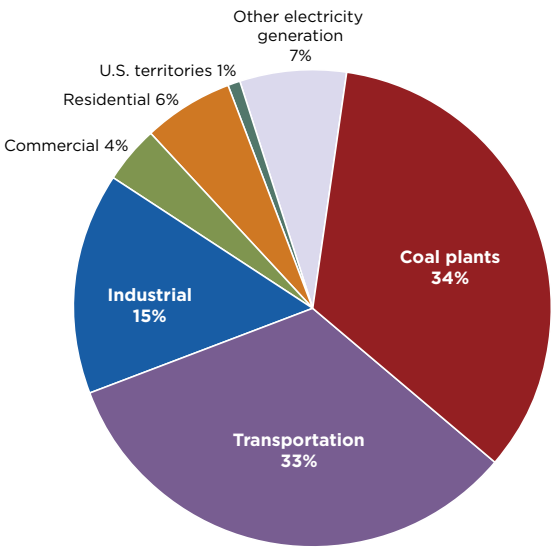
Methane emissions from coal mining are another important component of greenhouse gases.[9] Methane is produced when coal is formed and released when it is mined. Because methane is an occupational hazard in underground coal mines—it may explode or asphyxiate miners—it must be removed and ventilated into the atmosphere or burned. About 60% of methane emissions are now related to human activities,[10,11] with nearly 10% of that derived from coal mining and the remainder attributable largely to animal husbandry, waste management, and natural gas systems (see

Figure 6.1: CO_2 emissions and CO_2 concentrations (1750–2000)



Source: Oak Ridge National Laboratory. Carbon Dioxide Information Analysis Center. Available from: http://cdiac.esd.ornl.gov.

Figure 6.2: U.S. CO_2 emissions by source, 2007



Source: U.S. EPA. Inventory of U.S. greenhouse gas emissions and sinks: 1990–2007. 2009: EPA-430/R-09-004. Available from: http://www.epa.gov/climatechange/emissions/usinventoryreport.html.

Figure 6.3).[12] Although methane has a short lifetime in the atmosphere—9 to 15 years, compared to CO_2's 100 years or more—it is approximately 23 times more effective than CO_2 in trapping heat in the atmosphere.[13,14]

GLOBAL WARMING'S IMPACT ON HUMAN HEALTH

The accumulation of greenhouse gases in the atmosphere is making itself felt on earth in the form of increases in global average land and ocean surface temperatures, increases in snow melt and receding glaciers, thawing of permafrost, increases in the mean sea level, and changes in precipitation.[15] These effects create conditions that threaten human health directly and indirectly.

The high temperatures associated with global warming have direct implications for human health. Historically, global average temperatures have been quite stable. However, since 1909 the average temperature has risen 0.74°C (1.33°F).[16] In the U.S., the number of heat waves in the eastern and western regions rose by about 20%

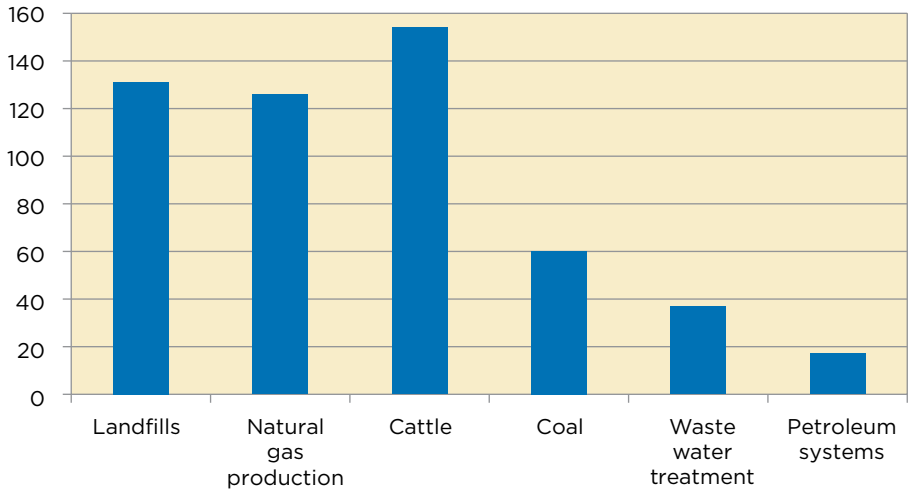
between 1949 and 1995.[17] Prolonged exposure to high temperatures can cause heat cramps, heat syncope, heat exhaustion, and heat stroke, which often leads to death. Advanced age is the most significant risk factor for heat-related deaths in the U.S., as the elderly are often less mobile, frequently home-bound and socially isolated, and may have thermoregulatory problems associated with multiple co-morbidities and medications that put them at higher risk for death during intense heat waves.[18] In addition, excessive heat exposure disproportionately affects people with certain pre-existing medical conditions, including cardiovascular disease, respiratory illnesses, and obesity.

It is difficult to quantify the number of deaths that result from heat waves, as heat-related deaths may be attributed to pre-existing conditions. However it is known that in 2003, between 22,000 and 35,000 people died as a direct result of the heat wave that swept Europe.[19,20] While it is impossible to attribute any specific heat wave to global warming, Stott, et al., concluded with a certainty of more than 90% that global warming more than doubled the probability of that heat wave occurring.[21]

In addition to the health effects directly associated with rising temperatures, global warming causes a profusion of interconnected public health problems.[22] Extreme-weather events and changing patterns of precipitation increase mortality from drowning. Flooding and infrastructure damage, along with temperature rise, increase the prevalence of insect- and water-borne diseases such as diarrhea, malaria, and dengue fever. High temperatures and continued fossil fuel consumption worsen air quality, impacting respiratory and cardiovascular health. Changing patterns of precipitation, rising temperatures, and extreme weather events cause crop damage and crop failure, affecting global food security. Competition for scarce resources such as food and water are predicted to cause mass migrations of environmental refugees, social destabilization, and war, while social destabilization and increasing global health problems are predicted to increase the risk of mental

Figure 6.3: Methane sources

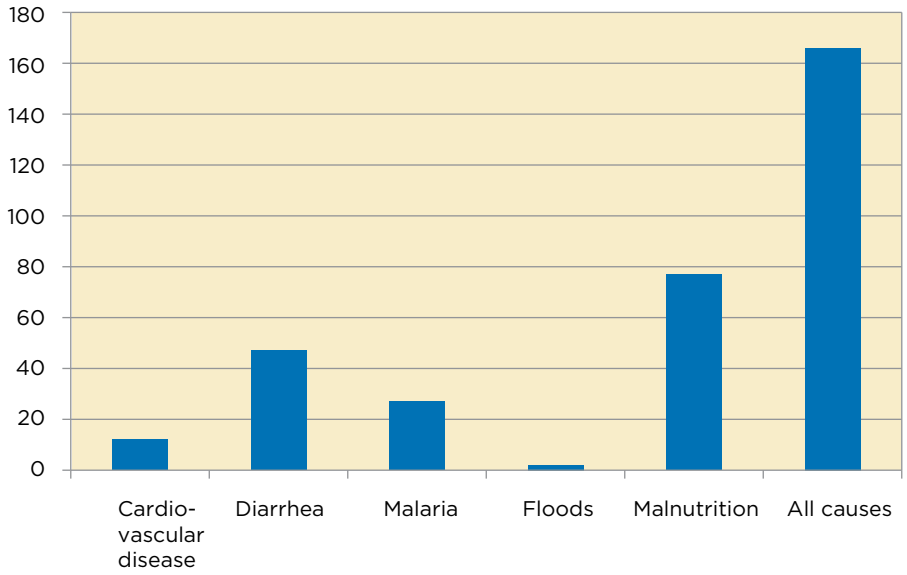
Methane emissions (Tg CO_2 equivalents). Contributions from coal include both active and abandoned mines. Contributions from cattle include emissions due to enteric fermentation and manure management.



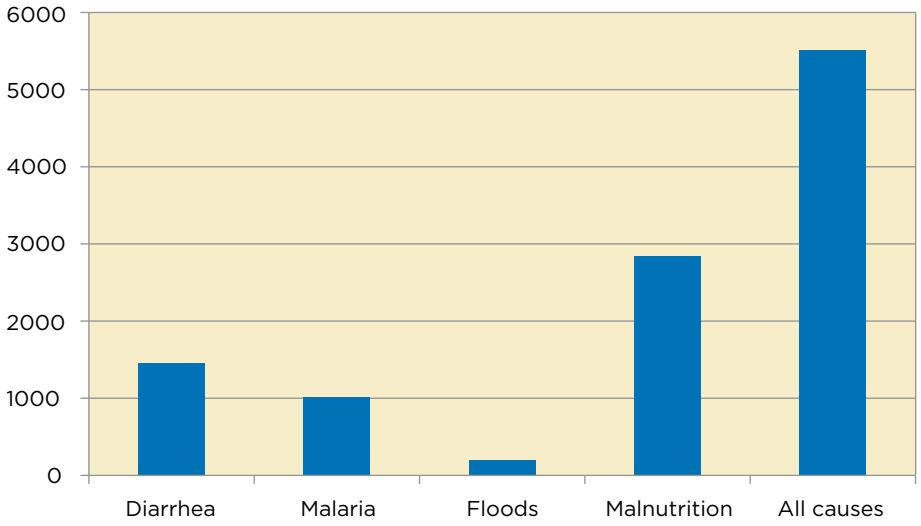
Source: U.S. EPA. Available from: http://www.epa.gov/methane/sources.html.

Figure 6.4: Morbidity and mortality due to global warming

Deaths in thousands due to global warming in 2000 compared to baseline climate recorded in 1961–1990



Morbidity, expressed in thousands of DALYs*



*DALYs = disability-adjusted life years, a measure of disease burden that combines years of life lost with years of life lived with illness or disability.

Source: 2004 World Health Organization data cited in Patz JA, Campbell-Lendrum D, Holloway T, Foley JA. Impact of regional climate change on human health. Nature 2005 Nov 17;438:310–317.

health problems, further adding to the burden on healthcare resources.

Many of these health effects are already evident. The World Health Organization (WHO) has quantified the annual impact of global warming on some health outcomes. WHO has estimated that global warming was responsible for 166,000 deaths in the year 2000 alone, due to additional mortality from malaria, malnutrition, diarrhea, and drowning. In addition, WHO estimated that in 2000, global warming caused increases in diarrhea, malaria, cardiovascular disease, and malnutrition that led to the loss of more than five million life years to disability from illness or premature death,[23] as shown in Figure 6.4.

The health burden of global warming, already large, is predicted to increase. Table ES-2 (see page xiii) shows the predicted health effects of global warming, the mechanisms that would drive these effects, and the populations most vulnerable to their implications.

RESPONDING TO GLOBAL WARMING

Events and activities that affect climate may not make themselves visible at once; rather, their impact may manifest years later. This is especially true of gradual phenomena such as the buildup of greenhouse gases in the atmosphere. The global warming effects that we currently experience were set in motion by greenhouse gases emitted over the past 250 years. By the same token, the greenhouse gases that we emit today might not affect us noticeably at the moment; they will manifest later—and, due to their persistence in the atmosphere and the substantial amount of heat absorbed by the oceans, will continue to be felt for decades to millennia to come. This delay in the felt consequences of current actions is known as "lag" in the climate system.[24]

The rise in average global temperatures has set in motion cycles of mutually reinforcing warming events. One example is the melting of polar ice. Polar ice melt reduces the amount of light-colored surface area that reflects the sun's rays, while expanding larger areas of dark land and ocean that absorb heat. This adds to overall warming, resulting in more ice melting. Rising temperatures in northern regions also cause the permafrost to melt. Permafrost is permanently frozen soil, sediment, or rock that remains at or below 0°C for at least two years. Because it is so cold, it can tolerate the introduction of considerable heat without thawing. However, when it does thaw, it releases potentially vast amounts of methane frozen in the mud and ice. Because methane is a potent greenhouse gas, this also accelerates global warming. Thus, one warming phenomenon feeds another, creating positive feedback cycles.[25]

These and other feedback cycles push us closer to a "tipping point": a point at which the accumulation of small changes in a steady state will force a change in that state that is sudden, significant, and usually irreversible.[26] In regard to climate, the tipping point will be reached when the gradual changes set in motion by burning fossil fuels—the accumulation of greenhouse gases, rising temperatures, and positive feedback loops—overwhelm our ability to offset them. At that point, the momentum of global warming will become irreversible on a human time scale. Therefore, action must be taken now to prevent such a scenario.

Steps that would prevent further global warming include replacing coal-based generation of electricity with clean, non-fossil fuel energy sources.

There are two categories of action that must be taken in response to global warming: mitigation and adaptation. Mitigation means preventing further global warming; it encompasses steps that would slow and stop the emission of greenhouse gases.[27] Significant forms of mitigation include replacing coal-based generation of electricity with clean, non-fossil fuel energy sources such as wind and solar; substituting non-fossil fuels for the gas and diesel we burn in vehicles; and reducing the need for energy by achieving increases in energy efficiency and conservation.

Adaptation refers to steps that would reduce vulnerability to the actual or expected negative

effects of climate change, such as building cities and healthcare infrastructure that can withstand extreme weather events, preparing emergency response plans, and developing drought-resistant crops.[28] With the effects of climate change already being felt, adaptation is necessary to protect vulnerable people and nations. At the same time, as a societal response adaptation is insufficient. If we continue to emit large amounts of greenhouse gases, global warming will continue and its effects will intensify. Eventually the damage wrought by floods, storms and sea level rise, drought and desertification, disease and hunger, habitat destruction and human displacement will overcome our capacity to protect ourselves. While adaptation measures are necessary, focusing our energies there to protect public health is insufficient. Adaptation does not prevent the triggering of climate tipping points such as those described above. The true protection of health and of life ultimately lies in prevention.

Lag in the climate system, especially when considered in conjunction with positive feedback

CARBON CAPTURE AND SEQUESTRATION

Carbon capture and sequestration (CCS) technology involves capturing the CO_2 from coal emissions, compressing it until it forms a liquid, and transporting the liquid CO_2 through pipelines to a geologically appropriate underground storage area where it would be stored ("sequestered") permanently.

Viewed through a public health lens, CCS poses several obstacles which would have to be resolved before it could be considered a viable option:

Concentrated CO_2 can be lethal. Concentrated CO_2 can asphyxiate people, as demonstrated in 1986 by the spontaneous release of CO_2 at Lake Nyos, Cameroon, that killed 1,700 people. Care must be taken to avoid pooling or leakage of CO_2 during both transport and storage.[29]

Permanent storage may not be possible. Storage would have to be leak-proof for geological periods of time.[30] If at any point in time stored CO_2 escaped into the atmosphere, it would contribute to global warming. If it were to leak into underground aquifers, it could dissolve and release contaminants such as arsenic, lead, mercury, and organic compounds, or could alter water acidity, affecting water quality.[31]

CCS would perpetuate coal pollution. During the time that CCS technology is being developed and safety and liability issues are being addressed, carbon emissions from coal plants would continue unabated. Even if CCS technology were successful, it would sustain the dependence on coal for the generation of electricity, perpetuating the release of other pollutants into the atmosphere, including particulates, sulfur dioxides, nitrogen oxides, and mercury, and contributing to the health-related problems associated with coal mining and the long-term storage of both pre- and post-combustion coal wastes.

CCS would divert funds from clean energy. The cost of research, development, construction, and implementation of CCS would be high.[32] The pipelines alone, reaching from every coal-powered plant in the country to appropriate storage areas, would require the construction of an extensive infrastructure system.[33] Funds spent on CCS development, construction, and deployment would be unavailable for investment in clean, safe energy from non-carbon-based sources.

Given the costs and difficulty of implementing CCS on a timeline and a scale that would effectively mitigate the health effects of global warming, the unreliability of permanent storage for geological periods of time, and the costs to health from coal's traditional pollutants, PSR has concluded that CCS is not a preferred option for developing the nation's energy future.

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loops and tipping points, has implications for our response to global warming. Even if CO_2 emissions cease today, average global temperatures will continue to rise. Therefore, current readings of CO_2 levels do not provide a full appraisal of the damage we have already inflicted on the climate system. This in turn means that we must be conservative in regard to future greenhouse gas emissions: Since the full impact of the emissions we have already released have not yet been felt, we should be cautious in assuming that we can predict the impacts of additional emissions.

The dynamics of climate change mean that we cannot take an unhurried approach to stopping global warming. We may be nearing climate tipping points; when we reach them, no human action or agency will be able to stop the climate change we will have set in motion. We must take action now, while there is still time to protect health and well-being on the planet.

NOTES

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7. *Policy Recommendations*

The U.S. is at a crossroads for determining its future energy policy. While the U.S. relies heavily on coal for its energy needs, the health consequences of that reliance are multiple and have widespread and damaging impact. Coal combustion contributes to diseases already affecting large portions of the U.S. population, including asthma, heart disease, and stroke, compounding major public health challenges of our time. It interferes with lung development, increases the risk of heart attacks, and compromises intellectual capacity. Coal pollutants affect all major body organ systems and contribute to four of the five leading causes of mortality in the U.S.: heart disease, cancer, stroke, and chronic lower respiratory diseases. Although it is difficult to ascertain the proportion of this disease burden that is attributable to coal combustion, even very modest contributions to these major causes of death are likely to have large effects at the population level, given high incidence rates.

The health effects of coal are not limited to diseases caused by combustion byproducts. Utilizing coal for energy also harms human health through the processes of mining, washing, transport, and post-combustion waste storage. Moreover, coal combustion is the largest point-source emitter of CO_2 in the U.S., contributing to the buildup of greenhouse gases that causes global warming. Although global warming is often framed as an environmental problem, it is likely to have significant public health consequences on a global scale, including increases in heat stroke, diarrhea, malaria, drowning, food shortages, mental health problems, and war. Many of these effects are already apparent. Reducing our nation's dependence on coal is essential if we are to achieve the reductions in carbon emissions necessary to stave off the worst health effects of global warming.

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Based on that assessment, PSR finds it essential to translate our concern for human health into

recommendations for public policy. The first of those recommendations is that emissions of CO_2 be cut as deeply and as swiftly as possible, with the objective of reducing CO_2 levels in the atmosphere to 350 parts per million. The reduction of CO_2 emissions, an urgent necessity for achieving satisfactory health outcomes, should be pursued through two simultaneous strategies: 1) strong climate and energy legislation that establishes hard caps on global warming pollution coming from coal plants; and 2) the Clean Air Act. Since its enactment, the Clean Air Act (CAA) as implemented by the EPA has been effective in reducing a wide variety of air pollutants, from nitrous oxides to volatile organic compounds. CO_2 and other greenhouse gases emitted by coal plants have been designated pollutants under the CAA. The EPA should be fully empowered to regulate these gases under the CAA so that coal's contribution to global warming can be brought to an end.

A medically defensible energy policy must take into account the public health impacts of coal while meeting our need for energy.

Secondly, PSR recommends that there be no new construction of coal-fired power plants, so as to avoid increasing health-endangering emissions of CO_2 as well as criteria pollutants and hazardous air pollutants. CO_2 emissions from coal could increase 60% by 2030 if current plans to invest hundreds of billions of dollars in new and old coal-fired power plants are realized. Those emissions would push greenhouse gas levels ever higher, potentially reaching a tipping point at which we would face the most extreme health consequences of global warming. For these reasons, PSR chapters across the nation have contributed to the effort to halt the permitting of new coal plants, adding the medical voice to licensing deliberations and helping to influence a number of decisions not to construct new plants.

The U.S. should dramatically reduce fossil fuel power plant emissions of sulfur dioxide and nitrogen oxides so that all localities are in attainment for national ambient air quality standards. In addition, we should establish a standard, enforceable by the EPA and based on Maximum Achievable Control Technology, for mercury and other hazardous air pollutant emissions from electrical generation.

Finally, the nation must develop its capacity to generate electricity from clean, safe, renewable sources so that existing coal-fired power plants may be phased out without eliminating jobs or compromising the nation's ability to meet its energy needs. In place of investment in coal (including subsidies for the extraction and combustion of coal and for capture of carbon and other pollutants), the U.S. should fund the improvement of energy efficiency, the expansion of conservation measures, and the research, development, and implementation of clean, safe, renewable energy sources such as wind energy, solar, and wave power.

These steps comprise a medically defensible energy policy: one that takes into account the public health impacts of coal while meeting our need for energy. When our nation establishes a health-driven energy policy, one that replaces our dependence on coal with clean, safe alternatives, we will prevent deterioration of global public health caused by global warming while reaping the rewards in improvements to respiratory, cardiovascular, and neurological health.

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