PART III

COSTS OF FOSSIL FUELS

Introduction to Part III

Part II documented the benefits reaped by mankind from the use of fossil fuels, including:

- Fossil fuels have vastly improved human well-being and safety by powering labor-saving and life-protecting technologies such as air conditioning, modern medicine, and cars and trucks.

- A warmer world would see a net decrease in temperature-related mortality and diseases in virtually all parts of the world, even those with tropical climates.

- The greater efficiency made possible by technologies powered by fossil fuels makes it possible to meet human needs while using fewer natural resources and land, thereby benefiting the natural environment.

Against these benefits must be balanced the costs imposed on humanity and the environment from the use of fossil fuels. The Working Group II contribution to Fifth Assessment Report of the United Nations' Intergovernmental Panel on Climate Change (IPCC) claims climate change causes a "risk of death, injury, and disrupted livelihoods" due to sea-level rise, coastal flooding, and storm surges; food insecurity, inland flooding, and negative effects on fresh water supplies, fisheries, and livestock; and "risk of mortality, morbidity, and other harms during periods of extreme heat, particularly for vulnerable urban populations" (IPCC, 2014, p. 7).

Environmental advocacy groups similarly claim the "hidden costs" of using oil and coal amount to billions and even trillions of dollars a year for the United States alone. For example, the Natural Resources Defense Council (NRDC) says "the total cost of global warming [which the NRDC attributes to fossil fuels] will be as high as 3.6% of gross domestic product (GDP). Four global warming impacts alone – hurricane damage, real estate losses, energy costs, and water costs – will come with a price of 1.8% of U.S. GDP, or almost $1.9 trillion annually (in today's dollars) by 2100. … [T]he true cost of all aspects of global warming – including economic losses, noneconomic damages, and increased risks of catastrophe – will reach 3.6% of U.S. GDP by 2100 if business-as-usual emissions are allowed to continue" (NRDC, 2008, pp. iv, vi). See also Lovins (2011, pp. 5–6) for a similar discussion.

These claims seem disconnected from reality. The predictions of "droughts, floods, famines, [and] disease spread" were shown in Parts I and II to be without any scientific basis, so we should be skeptical when seeing them included in cost-benefit analyses. As for the economic impact of "oil dependence," just one recent innovation in energy technology – combining horizontal drilling and hydraulic fracturing (fracking) to tap oil and natural gas trapped in shale deposits – has created 1.7 million new direct and indirect jobs in the United States, with the total likely to rise to 3 million in the next eight years (IHS Global Insight, 2012). It has added $62 billion to federal and state treasuries, with that total expected to rise to $111 billion by 2020. By 2035, U.S. fracking operations could inject more than $5 trillion in cumulative capital expenditures into the economy, while generating more than $2.5 trillion in cumulative additional government revenues (*Ibid*). And this is only one of many value-creating innovations occurring in the energy sector.

The NRDC and other advocacy groups like it have several things in common. First, they accept

uncritically the claims of the IPCC, invariably citing the *Summaries for Policymakers* of its Fourth or Fifth Assessment Reports while overlooking the caution and uncertainties expressed in the full reports. (This is especially ironic in the case of the NRDC since the organization infiltrated the IPCC, placing its own staffers on many of the IPCC's editing and peer-review committees, and so effectively *wrote* the reports they now cite as proof of their views. See Laframboise (2011).) The IPCC's computer models fail to replicate past temperature trends, meaning they cannot produce accurate forecasts of future climate conditions (Fyfe *et al.*, 2013). According to McKitrick and Christy (2018), for the period from 1958 to 2017 the models hindcast a warming of~ 0.33° C/decade while observations show only ~ 0.17°C/decade. (With a break term for the 1979 Pacific climate shift included the models hindcast ~ 0.39°C/decade and observations show ~ 0.14° C/decade.) This fact undermines all alleged cost-benefit analyses of climate change that rely on IPCC reports for forecasts of future climate conditions.

Second, the Rocky Mountain Institute, NRDC, and groups like them invariably exclude from their accounting any of the *benefits* of fossil fuels. As documented in Chapters 3, 4, and 5, these benefits are huge relative to any damages due to fossil fuels or to climate change. Ignoring those benefits is obviously wrong. Epstein (2016) notes,

> [I]t is a mistake to look at costs in isolation from benefits, or benefits apart from costs. Yet that appears to be the approach taken in these reports. … [A] truly neutral account of the problem must be prepared to come to the conclusion that increased levels of CO_2 emissions could be, as the Carbon Dioxide Coalition has argued, a net benefit to society when a more comprehensive investigation is made. The entire process of expanding EPA regulations and other Obama administration actions feeds off this incorrect base assumption.

Environmental groups also rely heavily on economic models and simulations, called integrated assessment models (IAMs) to reach their conclusions. Like the climate models relied on by the IPCC, these models hide assumptions and uncertainties, are often invalidated by real-world data, and fail the test of genuine scientific forecasts. They are merely scenarios based on their authors' best guesses, "tuned" by their biases and political agendas, and far from reliable. See Chapter 2 for the candid discussion of by a group of leading modelers of "the art and science of climate model tuning" (Hourdin *et al.,* 2017) and Green and Armstrong (2007) for an audit of the use of IAMs for forecasting. Real data are available to fact-check the models, but they are curiously absent from the claims of advocates and the academic literature they cite.

Chapters 6 and 7 of Part III set out an accurate accounting of the biggest alleged costs of fossil fuels, those attributable to chemical compounds released during the combustion of fossil fuels and what the IPCC calls "threats to human security" which includes famine, conflict, damage from floods and extreme weather, and forced migration. The authors find that in both cases, costs are exaggerated in the popular as well as the academic literature. Non-specialists feed these inflated cost estimates into their computer models apparently without understanding they are unsupported by real observational data and credible economic, scientific, and public health research. When these major sources of concern are addressed, any remaining costs are quite small or speculative.

Chapter 8 conducts cost-benefit analyses of climate change attributed by the IPCC to the combustion of fossil fuels, the use of fossil fuels, and regulations enacted or advocated in the name of slowing or stopping global warming. At the risk of overly simplifying what is a very complicated analysis, the conclusions of that chapter can be said to affirm the small and highly uncertain cost of man-made climate change, the net benefits of fossil fuels, and the very high cost of regulations aimed at reducing greenhouse gas emissions.

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6

Air Quality

Chapter Lead Author: John D. Dunn, M.D., J.D.

Contributors: Jerome C. Arnett, Jr., M.D., Charles Battig, M.D., M.S.E.E., James E. Enstrom, Ph.D., Steve Milloy, J.D., S. Stanley Young, Ph.D.

Reviewers: Kesten Green, Ph.D., Richard J. Trzupek, Art Viterito, Ph.D.

Key Findings

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Key Findings

The key findings of this chapter include the following:

An Air Quality Tutorial

- The combustion of fossil fuels without air pollution abatement technology releases chemicals known to be harmful to humans, other animal life, and plants.

Citation: Dunn, J.D. 2019. Air Quality. In: *Climate Change Reconsidered II: Fossil Fuels.* Nongovernmental International Panel on Climate Change. Arlington Heights, IL: The Heartland Institute.

- At low levels of exposure, the chemical compounds produced by burning fossil fuels are not known to be toxic.

- Exposure to potentially harmful emissions from the burning of fossil fuels in the United States declined rapidly in recent decades and is now at nearly undetectable levels.

- Exposure to chemical compounds produced during the combustion of fossil fuels is unlikely to cause any fatalities in the United States.

Failure of the EPA

- Due to its faulty mission, flawed paradigm, and political pressures on it to chase the impossible goal of zero risk, the U.S. Environmental Protection Agency (EPA) is an unreliable source of research on air quality and its impact on human health.

- The EPA makes many assumptions about relationships between air quality and human health, often in violation of the Bradford Hill Criteria and other basic requirements of the scientific method.

- The EPA has relied on research that cannot be replicated and violates basic protocols for conflict of interest, peer review, and transparency.

Observational Studies

- Observational studies are easily manipulated, cannot prove causation, and often do not support a hypothesis of toxicity with the small associations found in uncontrolled observational studies.

- Observational studies cited by the EPA fail to show relative risks (RR) that would suggest a causal relationship between chemical compounds released during the combustion of fossil fuels and adverse human health effects.

- Real-world data and common sense contradict claims that ambient levels of particulate matter kill hundreds of thousands of Americans and millions of people around the world annually.

- By conducting human experiments involving exposure to levels of particulate matter and other pollutants it claims to be deadly, the EPA reveals it does not believe its own epidemiology-based claims of a deadly threat to public health.

Circumstantial Evidence

- Circumstantial evidence cited by the EPA, World Health Organization (WHO), and other air quality regulators is easily refuted by pointing to contradictory evidence.

- EPA cannot point to any cases of death due to inhaling particulate matter, even in environments where its National Ambient Air Quality Standard (NAAQS) is exceeded by orders of magnitude.

- Life expectancy continues to rise in the United States and globally despite what should be a huge death toll, said to be equal to the entire death toll caused by cancer, attributed by the EPA and WHO to just a single pollutant, particulate matter.

Conclusion

- It is unlikely that chemical compounds released during the combustion of fossil fuels kill or harm anyone in the United States, though it may be a legitimate health concern in third-world countries that rely on burning biofuels and fossil fuels without modern emission control technologies.

Introduction

Data cited by Simon (1995, 1996), Lomborg (2001), Anderson (2004), Hayward (2011), Goklany (2007, 2012), Epstein (2014), Pinker (2018), and many others reported in Part II, much of it compiled by the U.S. Environmental Protection Agency (EPA) and

other government sources, document a dramatic improvement in public health since the beginning of the industrial revolution. *Do chemical compounds released by burning fossil fuels nevertheless pose a public health risk?*

In 2010, the EPA claimed just one kind of air pollutant, particulate matter (fine dust particles), caused approximately 360,000 and as many as 500,000 premature deaths in the United States in 2005, citing Laden *et al.* (2006) (EPA, 2010, p. G7). The high estimate would be more than one-fifth of all deaths in the United States that year and nearly as high as all deaths from cancer (Kung *et al.,* 2008). In 2011, then-EPA Administrator Lisa Jackson endorsed the highest estimate in testimony to Congress, saying, "If we could reduce particulate matter to levels that are healthy we would have an identical impact to finding a cure for cancer" (quoted in Harris and Broun, 2011, p. 2).

The World Health Organization (WHO) similarly claims air pollution is a major health problem globally, saying it caused 600,000 premature deaths in 2010 in Europe alone (WHO, 2015). A 2016 WHO report claimed "3.9 million premature deaths each year [are] attributable to outdoor air pollution" and exposure to household air pollution (HAP) "causes 4.3 million premature deaths each year" (WHO, 2016, p. ix).

These claims are reported and repeated without hesitation or scrutiny by environmental groups, the media, and even serious scholars in the climate change debate. But the EPA and WHO claims are based on weak epidemiological relationships and trends carelessly described without definition as "associations" or "trends." Much like assumptions, computer models, and circumstantial evidence are paraded by the United Nations' Intergovernmental Panel on Climate Change (IPCC) as evidence in the climate science debate, so too are these unscientific lines of reasoning presented as evidence by the EPA and WHO in the debate over air quality.

This chapter begins with a brief tutorial on air quality[1] and then explains why chemical compounds released during the combustion of fossil fuels do not present a significant human health threat in the United States or other developed countries. In developing countries, where exposure to pollutants is greater, a health risk may be present, though fossil fuels may prove to be a solution rather than the problem in many regions. Morrison (2018), for example, describes an effort to replace old biomass cookstoves in developing countries with "stoves that use propane, a fossil fuel, the same blue-flamed byproduct of gas drilling contained in cylinders under countless American backyard grills." The solution to air quality issues in developing countries lies in the prosperity, values, and technologies used by developed countries to solve their air quality problems.

[1] We use the term "air quality" rather than "air pollution" when possible because the public policy goal is to improve air quality, not necessarily to reduce or end "air pollution." Referring to chemical compounds created during the combustion of fossil fuels as "pollution" prejudges them as harmful. Emissions are not harmful unless they are present in concentrations sufficient to endanger human health.

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6.1 An Air Quality Tutorial

Critics of fossil fuels often attribute social costs to the public health consequences of emissions created by the combustion of fossil fuels without understanding basic facts about chemistry, alternative (often natural) sources of the same chemicals, evidence of human exposure and trends of the same, and how all these data are interpreted. This section offers a brief tutorial on these topics.

6.1.1 Chemistry

> *The combustion of fossil fuels without air pollution abatement technology releases chemicals known to be harmful to humans, other animal life, and plants.*

When burned, fossil fuels release carbon dioxide (CO_2), water (H_2O), carbon monoxide (CO), sulfur dioxide (SO_2), nitrogen oxides (NO_x), and particulate matter (PM). Another pollutant, ozone (O_3), is created through photochemical reaction with the other pollutants. Carbon dioxide and water, as Moore has observed, are "the two most essential foods for life" (Moore, 2015) and are not public health concerns, leaving five emissions of concern.

Carbon monoxide (CO) is a colorless, odorless gas formed when carbon in wood or fossil fuels is not burned completely. Approximately 80% or more of human outdoor CO emissions in the United States comes from motor vehicle exhaust while the remaining 20% comes from industrial processes and residential wood burning. CO is produced indoors by woodstoves, gas stoves, unvented gas and kerosene space heaters, and smoking.

Sulfur dioxide (SO_2) is formed when fossil fuels containing sulfur, such as coal and oil, are burned, when gasoline is extracted from crude oil, and when metals are extracted from ore. Sulfur dioxide dissolves in water, creating droplets that are less basic or alkaline than would otherwise occur, creating what is popularly and inaccurately called "acid rain."

Nitrogen oxides (NO_x) are a group of gases containing nitrogen and oxygen, most of which are colorless and odorless. Nitrogen oxides form when fuel is burned at high temperatures, as in a combustion process. Half of NO_x emissions in the United States come from motor vehicle exhaust and most of the rest from stationery generators.

Particulate matter (PM) is the general term used to describe a mixture of solid particles and liquid droplets found in the air. Some PM particles are large enough to be seen as dust or dirt. Others are so small they can be detected only with an electron microscope. $PM_{2.5}$ refers to particles less than or equal to 2.5 μm (micrometer) in diameter. PM_{10} refers to particles less than or equal to 10 μm in diameter (about one-seventh the diameter of a human hair). "Primary" PM is emitted directly into the atmosphere. Examples of primary particles are dust from roads or black carbon (soot) from burning wood or fossil fuels. "Secondary" particles, which are formed in the atmosphere from gaseous emissions, include sulfates (formed from SO_2), nitrates (formed from NO_x), and carbon (formed from CO_2).

Fossil fuels create PM in the form of soot when the supply of oxygen during combustion is insufficient to completely convert carbon to carbon oxides. This typically occurs during the combustion of coal and oil, not natural gas. PM also is produced by agriculture (plowing, planting, and harvesting activities), resuspension by wind or traffic of dust particles from roads, and many natural processes including forest fires, wind erosion, desert dust, volcanoes, sea salt aerosols (sodium chloride

(NaCl)), and biological aerosols (e.g., spores and pollen). The EPA estimates approximately 16% of U.S. PM_{10} emissions and 40% of $PM_{2.5}$ emissions are anthropogenic while the rest is "fugitive dust" (dust from open fields, roadways, storage piles, and other non-point sources) and "miscellaneous and natural sources" (EPA, 2018a). See Figure 6.1.1.1.

Ozone (O_3) is a triatomic oxygen molecule gas that occurs in Earth's upper atmosphere and at ground level. Ozone is not directly emitted into the atmosphere when fossil fuels are combusted, but it can be counted as a pollutant resulting from their use because fossil fuel use produces precursors to the photochemical reaction that creates ozone at ground level. Those precursors are carbon monoxide, nitrogen oxides, and particulate matter. Trees and other plants also produce ozone precursors, in particular hydrocarbons, but primarily in rural areas where their ratio to nitrogen oxides is too large to create the conditions in which ozone is formed.

Volatile organic compounds (VOCs) are often included in lists of pollutants attributable to the use of fossil fuels. All molecules containing carbon with high vapor pressure at ordinary room temperature are classified as VOCs, meaning they readily evaporate in air. This category necessarily duplicates or overlaps with others in this list of emissions. Nature, primarily plants, produces about ten times as much VOCs, by weight, as all human activities (1,150 versus 142 teragrams per year). The combustion of fossil fuels contributes only a small fraction of man-made VOCs, with carbon monoxide, gasoline fumes, and benzene being three examples.

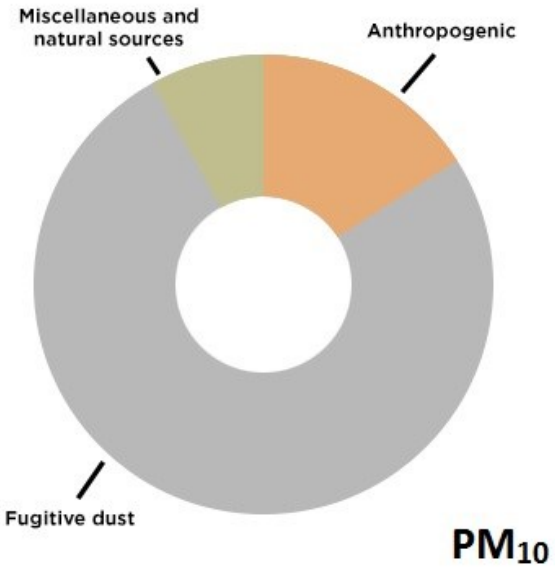
Lead (Pb) is often included as an emission from the combustion of fossil fuels, but it was a lead-containing compound called tetraethyllead added to petroleum to improve engine performance that was responsible for lead emissions from motor vehicles.

Lead is not found in appreciable amounts in coal or refined oil products. Due to the phase-out of leaded gasoline in the United States and other nations, lead in the air is no longer a public health hazard in the United States or other developed countries (von Storch *et al.,* 2003). The main sources of human lead emissions today are waste incinerators and lead-acid battery manufacturers.

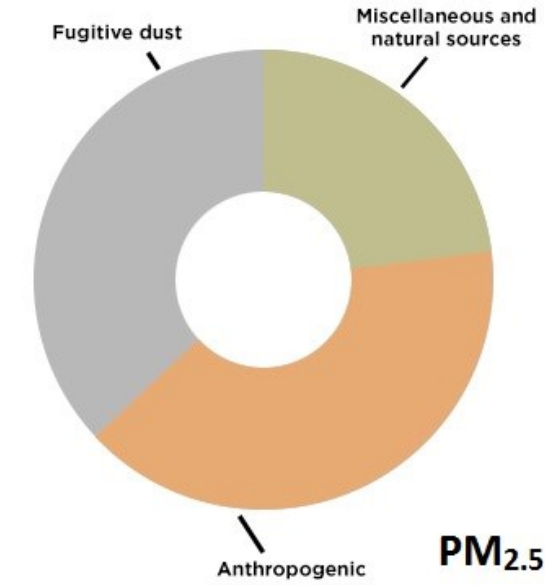
Some trace minerals in fossil fuels also are present in ash when fossil fuels are burned. The ash can become airborne or dissolved into and transported by water. One such compound is *mercury (Hg),* which in its organic form (methylmercury) can be poisonous to humans and other living creatures. Mercury is a naturally occurring substance, with

Figure 6.1.1.1
Sources of particulate matter (PM) in the United States

A. Relative amounts of U.S. PM_{10} emissions from anthropogenic and other sources, 2011



B. Relative amounts of U.S. $PM_{2.5}$ emissions from anthropogenic and other sources, 2011



Source: EPA, 2018a, data from the 2011 National Emissions Inventory, Version 1.

some 200 million tons present in seawater. Mercury emissions from the combustion of fossil fuels in the United States are very small relative to other sources: approximately 7 tons annually (EPA, 2018b) versus 5,000 to 8,000 tons from all sources globally, including volcanoes, subsea vents, geysers, forest fires, and other natural sources.

Gasoline evaporates quickly when exposed to air, a property that leads to rapid dispersal of spills above ground, but when spilled underground (say, from leaking gas station tanks) it can remain in place for years and pose a threat to drinking water. Finally, carbon monoxide and particulate matter from incomplete fuel combustion by automobile engines and NO_x can react with sunshine to create ozone (already discussed above) and a visible haze called "smog."

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6.1.2 Exposure

> *At low levels of exposure, the chemical compounds produced by burning fossil fuels are not known to be toxic.*

The most important lesson regarding air quality is what matters most is not the toxicity of a chemical but the *level of exposure.* As Paracelsus, a Swiss physician, observed some five centuries ago, "Everything is poison. There is nothing without poison. *Only the dose makes a thing not a poison*." Without exposure there can be no harm.

Emissions from the combustion of fossil fuels increased early in the modern era due to rising population and per-capita energy consumption, but have been falling since the 1940s. Today, most of the potentially harmful chemical compounds created during the combustion of fossil fuels for electricity generation are removed by pollution control technologies and never enter the air. According to the National Energy Technology Laboratory (NETL, 2015), an agency within the U.S. Department of Energy, pollution controls used by a "subcritical pulverized coal plant with a nominal net output of 550 MWe" reduce NO_x emissions by 83%, SO_2 emissions by 98%, mercury by 96.8%, and PM by 99.9% when compared with a similar plant with no pollution controls (p. 77). Catalytic converters on cars and trucks convert CO and unburned hydrocarbons in the combustion process into CO_2 and converts NO_x into harmless N_2.

This section begins with an explanation of the potential threat to human health posed by exposure to seven chemical compounds produced by the combustion of fossil fuels: carbon monoxide (CO), sulfur dioxide (SO_2), nitrogen oxides (NO_x), particulate matter (PM), ozone (O_3) (formed by the interactions of the previous four), volatile organic compounds (VOCs) (the vaporous state of some previous compounds), and elementary mercury (Hg). It then summarizes recent research on current levels of exposure to these chemicals. Particulate matter is reported briefly here, but is addressed in much greater detail in Section 6.3.

Potential threat to human health

Carbon monoxide (CO) can be poisonous at high levels of exposure not commonly found in ambient air. In the human body, hemoglobin (an iron compound) in the blood carries oxygen from the lungs to various tissues and transports carbon dioxide (CO_2) back to the lungs. Hemoglobin has 240 times more affinity toward CO than it does for oxygen. When the hemoglobin reacts with CO, it reduces the hemoglobin available for the transport of oxygen. This in turn reduces oxygen supply to the body's organs and tissues. Consequently, people who suffer from cardiovascular disease are most at risk from elevated levels of CO. There is also the potential for harm in pregnancy, because relative oxygen levels have a greater impact on the fetus, which depends on maternal blood oxygen. More commonly, exposure to elevated levels of CO may result in visual impairment, reduced manual dexterity, and difficulty in performing complex tasks. Figure 6.1.2.1 shows

6.1.2.1
Health effects associated with human exposure to carbon monoxide

CO concentration (parts per million)	Duration of exposure (hours)	Effect
100	10	Headache
300	10	Nausea, unconsciousness
600	10	Death
1000	1	Unconsciousness
1000	4	Death

Source: Radovic, 1992.

one estimate of the health effects associated with different levels of exposure to carbon monoxide.

Most of the sulfur in a fossil fuel combines with oxygen and forms *sulfur dioxide (SO_2)* in the combustion chamber. Unless captured by emission control technology, this SO_2 is emitted into the atmosphere where it oxidizes to sulfur trioxide (SO_3). SO_3 is soluble in water in the clouds and forms H_2SO_4 (sulfuric acid). Exposure to sulfuric acid irritates the mucous membranes of the respiratory tract, which causes airways to restrict and damages the cells of the mucous membranes, causing the release of inflammatory mediators that cause airway swelling and spasm, restricting airway size, causing an increase in work of breathing and decrease in available inspired air.

Exposure to a concentration of 1 part per million of SO_3 can cause coughing and choking; higher levels can result in temporary breathing impairment such as wheezing, chest tightness, or shortness of breath. Long-term exposure can aggravate existing cardiovascular disease and respiratory illnesses. SO_3 in the atmosphere also acts as a precursor to fine particulate matter.

Nitric oxide (NO) released during combustion of fossil fuels is oxidized in the atmosphere to *nitrogen dioxide* (NO_2). (NO_2 is also created from the nitrogen in the air during some high-temperature processes that do not involve fossil fuels.) Nitrogen dioxide is a noxious gas that can cause inflammation of the respiratory tract, similar to sulfuric acid, and, at high concentrations, even death.

NO_2 is soluble in water and forms HNO_3 (nitric acid). Like sulfuric acid, nitric acid constricts airways in humans and animals and can cause adverse health effects. Short-term exposure may lead to changes in airway responsiveness and lung function in individuals with preexisting respiratory illnesses. Long-term exposure may lead to increased susceptibility to respiratory infection and may cause irreversible alterations in lung structure.

The sulfuric and nitric acids created by SO_2 and NO_x in the atmosphere return to the surface in the form of dry deposition of particles or rain that is slightly more acidic than would otherwise occur, popularly referred to as "*acid rain*." Pure water is neither acidic nor basic, but natural rainfall even in the absence of human use of fossil fuels is slightly acidic because it dissolves carbon dioxide from the air. Nitrogen, like carbon dioxide, is a plant fertilizer, and therefore higher levels are generally beneficial to most types of plant life and, by expanding habitats, to animal life as well. However, the addition of nitrogen to lakes and rivers can cause excessive algae growth, which contributes to eutrophication (depletion of dissolved oxygen), which can harm fish and other aquatic life. This concern is addressed in Chapter 5.

Particulate matter (PM), whether produced by the combustion of fossil fuels or by other processes described in the preceding section, can enter lungs and get trapped in the very thin air passages, reducing the air capacity of the lungs. Reduced air capacity can lead to such breathing and respiratory problems as emphysema and bronchitis, as well as increased general susceptibility to respiratory diseases. People with heart or lung disease and the elderly are especially at risk. Depending on the composition of the particles, chemical or mechanical or even allergenic, the effect is directly on the tissues, like the chemical effects described above for nitrous and sulfuric compounds, but not as toxic. The effect of particles is determined by their composition since

they are not large enough to obstruct airways, even the terminal bronchioles that allow air into the air sacs that exchange oxygen and carbon dioxide. However, deposits of small particles can occur because the cleaning mechanisms in the alveoli and airways are not 100% efficient. There is no medical research establishing a mechanism for how small particles might cause death.

Exposure to ground-level *ozone (O_3)* can cause inflammation of the lining of the lungs, reduced lung function, and respiratory symptoms such as cough, wheezing, chest pain, burning in the chest, and shortness of breath. Longer-term exposure has been associated with the aggravation of respiratory illnesses such as asthma, emphysema, and bronchitis, leading to increased use of medication, absences from school, doctor and emergency department visits, and hospital admissions.

Volatile organic compounds (VOCs) related to fossil fuels include the compounds mentioned above, since the classification is determined by their ability to evaporate at relatively low temperatures. Most VOCs considered public health threats come from the use of cleaners, paints, and building materials in indoor spaces and not the combustion of fossil fuels. Outdoor levels of VOCs are monitored and regulated due to their role in the creation of ozone and smog.

Exposure to *mercury (Hg)* fumes can cause harmful effects on the nervous, digestive and immune systems, lungs and kidneys, and may be fatal. The inorganic salts of mercury are corrosive to the skin, eyes and gastrointestinal tract, and may induce kidney toxicity if ingested. Neurological and behavioral disorders may be observed after inhalation, ingestion or dermal exposure of different mercury compounds. Symptoms include tremors, insomnia, memory loss, neuromuscular effects, headaches and cognitive and motor dysfunction WHO, 2017).

Once in the environment, mercury can be transformed by bacteria into methylmercury, which bioaccumulates in fish and shellfish. Human consumption of seafood with high levels of methylmercury can cause some of the health effects described above. Methylmercury can pass through the placenta, exposing the fetus and causing birth defects, possibly manifested as lower IQ.

Current levels of exposure

The U.S. EPA was required by the Clean Air Act to establish National Ambient Air Quality Standards (NAAQS) setting the maximum level of exposure, measured in concentration of the pollutant in the air and time of exposure for substances believed to endanger public health or the natural environment. The EPA has set NAAQS for six pollutants, which it calls "criteria air pollutants," being the five identified in the previous section as attributable to fossil fuels plus lead (EPA, 2018a). The current NAAQS appear in Figure 6.1.2.2.

In its description of the table in Figure 6.1.2.2, the EPA says "The Clean Air Act identifies two types of national ambient air quality standards. ***Primary standards*** provide public health protection, including protecting the health of 'sensitive' populations such as asthmatics, children, and the elderly. ***Secondary standards*** provide public welfare protection, including protection against decreased visibility and damage to animals, crops, vegetation, and buildings."

EPA says of its NAAQS, "The primary standards are set at a level intended to protect public health, including the health of at-risk populations, with an adequate margin of safety. In selecting a margin of safety, the EPA considers such factors as the strengths and limitations of the evidence and related uncertainties, the nature and severity of the health effects, the size of the at-risk populations, and whether discernible thresholds have been identified below which health effects do not occur. In general, for the criteria air pollutants, there is no evidence of discernible thresholds" (EPA, 2018b, p. 1). EPA's use of "safety factors" and a "linear no-threshold dose-response relation" are controversial and are explored in Section 6.2.2.

The EPA has estimated the "percentage of children living in [U.S.] counties with pollutant concentrations above the levels of the current air quality standards" for the six EPA criteria pollutants in the most recent year, 2013. Its findings are summarized in Figure 6.1.2.3.

As shown in Figure 6.1.2.3, according to the EPA carbon monoxide in ambient outdoor air is a nonexistent threat, with 0% of children living in counties in which they might be exposed to harmful levels of that pollutant. Fewer than 1% of children live in counties where lead exposure might be a threat, 2% where nitrogen dioxide is a problem, and 3% for sulfur dioxide. Particulate matter and ozone seem to pose larger problems, with between 3% and 21% of children living in counties where they might be exposed to unhealthy levels of PM and 58% threatened by ozone.

EPA also has created an "Air Quality Index" combining and weighing its measures of exposure to

Figure 6.1.2.2
National Ambient Air Quality Standards

Pollutant [links to historical tables of NAAQS reviews]		Primary/ Secondary	Averaging Time	Level	Form
Carbon Monoxide (CO)		primary	8 hours	9 ppm	Not to be exceeded more than once per year
			1 hour	35 ppm	
Lead (Pb)		primary and secondary	Rolling 3 month average	0.15 µg/m^3 (1)	Not to be exceeded
Nitrogen Dioxide (NO_2)		primary	1 hour	100 ppb	98th percentile of 1-hour daily maximum concentrations, averaged over 3 years
		primary and secondary	1 year	53 ppb (2)	Annual Mean
Ozone (O_3)		primary and secondary	8 hours	0.070 ppm (3)	Annual fourth-highest daily maximum 8-hour concentration, averaged over 3 years
Particle Pollution (PM)	$PM_{2.5}$	primary	1 year	12.0 µg/m^3	annual mean, averaged over 3 years
		secondary	1 year	15.0 µg/m^3	annual mean, averaged over 3 years
		primary and secondary	24 hours	35 µg/m^3	98th percentile, averaged over 3 years
	PM_{10}	primary and secondary	24 hours	150 µg/m^3	Not to be exceeded more than once per year on average over 3 years
Sulfur Dioxide (SO_2)		primary	1 hour	75 ppb (4)	99th percentile of 1-hour daily maximum concentrations, averaged over 3 years
		secondary	3 hours	0.5 ppm	Not to be exceeded more than once per year

(1) In areas designated nonattainment for the Pb standards prior to the promulgation of the current (2008) standards, and for which implementation plans to attain or maintain the current (2008) standards have not been submitted and approved, the previous standards (1.5 µg/m3 as a calendar quarter average) also remain in effect.

(2) The level of the annual NO2 standard is 0.053 ppm. It is shown here in terms of ppb for the purposes of clearer comparison to the 1-hour standard level.

(3) Final rule signed October 1, 2015, and effective December 28, 2015. The previous (2008) O3 standards additionally remain in effect in some areas. Revocation of the previous (2008) O3 standards and transitioning to the current (2015) standards will be addressed in the implementation rule for the current standards.

(4) The previous SO2 standards (0.14 ppm 24-hour and 0.03 ppm annual) will additionally remain in effect in certain areas: (1) any area for which it is not yet 1 year since the effective date of designation under the current (2010) standards, and (2)any area for which an implementation plan providing for attainment of the current (2010) standard has not been submitted and approved and which is designated nonattainment under the previous SO2 standards or is not meeting the requirements of a SIP call under the previous SO2 standards (40 CFR 50.4(3)). A SIP call is an EPA action requiring a state to resubmit all or part of its State Implementation Plan to demonstrate attainment of the required NAAQS.

Source: EPA, 2018a.

Figure 6.1.2.3
Percentage of children living in counties with exposures above the EPA NAAQS in 2015

Percentage of children exposed	Pollutant	Measurement of Exposure
0	Carbon monoxide	Concentrations above the level of the current standard for carbon monoxide
0.1	Lead	Ambient lead concentrations above the level of the current three-month standard for lead
2	Nitrogen dioxide	Concentrations above the level of the current one-hour standard for nitrogen dioxide at least one day per year
3	Sulfur dioxide	Sulfur dioxide concentrations above the level of the current one-hour standard for sulfur dioxide at least one day per year
3	Particulate Matter (2.5 μm)	Average concentration above the level of the current annual $PM_{2.5}$ standard
7	Particulate Matter (10 μm)	PM_{10} concentrations above the level of the current 24-hour standard for PM_{10} at least one day per year
21	Particulate Matter (2.5 μm)	$PM_{2.5}$ concentrations above the level of the current 24-hour $PM_{2.5}$ standard at least once per year
58	Ozone	Ozone concentrations above the level of the current 8-hour ozone standard at least one day during the year

Source: EPA, 2018b, from text on p. 12.

the six criteria pollutants. The percentage of children living in counties where they might be exposed to what the EPA deems "unhealthy air" was only 3% in 2015, down from 9% 16 years earlier (EPA, 2018). A graph showing the decline appears as Figure 6.1.3.3 in the next section.

EPA versus Real-World Exposure

EPA's estimates of exposure to chemical compounds released during the combustion of fossil fuels are "stylized facts," simplifications of the very complex and uncertain data collected and interpreted to meet the needs of government regulators (and perhaps newspaper headline writers). Still, they can be shown to greatly overstate the real-world exposure to pollutants experienced by people living in the United States, including children.

Start with the EPA's assumption that every child living in a county is breathing the *worst* air quality reported by *any* air-quality monitoring station in that county over the course of a year. This is why the text above summarizing EPA's findings uses the clumsy phrase "percentage of children living in counties where they might be exposed to pollutant concentrations above the levels of the current air quality standards" instead of the percentage or number of children *actually* exposed. As Schwartz and Hayward reported in 2007,

> EPA and ALA [American Lung Association] get their inflated numbers by counting everyone in a county as breathing air that exceeds federal standards, even if most of the county has clean air. For example, only one rural area of San Diego County, with about 1% of the population, violates the EPA's 8-hour ozone standard. But the EPA and the ALA count all three million people in the county as breathing "unhealthy" air. This is akin to giving every student in a school a failing grade if just one gets an "F" on an exam (p. 7).

It gets worse. The "one day per year" appearing in Figure 6.1.2.3 is EPA shorthand for a complex way of measuring "exceedances" and "violations" (explained by Schwartz and Hayward, 2007, pp. 8-9).

If exceedances occurred one day a year, then some children living in counties where children could be exposed to a pollutant as little as 0.27% of the time (1 ÷ 365). So for the $PM_{2.5}$ standard, a one-day violation a year in counties where 21% of the children in the United States reside means the *average child* in the United States is exposed only 0.06% of the time (0.21 x 0.27), or for about five hours a year, to ambient levels of $PM_{2.5}$ above EPA's NAAQS.

When the EPA's faulty way of counting affected children is corrected, Schwartz and Hayward (2007, p. 10) found "about 11% of Americans live in areas that violate the 8-hour ozone standard, while about the same fraction live in areas that violate for $PM_{2.5}$." The authors were using data from 2006. Since then concentrations of $PM_{2.5}$ have fallen by about 24% (see Figure 6.1.3.1 below). So maybe only 8% of Americans (0.11 x (1 - 0.24)) live in areas that violate the $PM_{2.5}$ standard 0.27% of the time, so average exposure is 0.02% a year, or less than two hours a year.

EPA estimates anthropogenic emissions account for about 40% of $PM_{2.5}$ released into the air each year in the United States (EPA, 2018, see Figure 6.1.1.1 above). Fossil fuel-related activities account for approximately half of those emissions, so fossil fuels account for about 20% of human exposure to $PM_{2.5}$ in the United States. So maybe fossil fuels are responsible for exposing Americans to levels of PM_{10} that exceed EPA's NAAQS for about 24 minutes a year (0.02 x 0.2 x 60).

The same exercise could be performed for ozone and other pollutants and would arrive at similar conclusions: exposure to possibly harmful air pollutants due to the use of fossil fuels in the United States is probably too low to accurately measure or distinguish from background levels. This is according to the EPA's own monitoring stations and assuming *arguendo* that EPA's NAAQS actually are meaningful indicators of a possible threat to public health. That assumption is taken up (and refuted) in Section 6.1.4 and in later sections.

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6.1.3 Trends

> *Exposure to potentially harmful emissions from the burning of fossil fuels in the United States declined rapidly in recent decades and is now at nearly undetectable levels.*

Chemical compounds released during the combustion of fossil fuels in the United States and in developed countries around the world have fallen dramatically since the 1940s and 1950s as a result of technological change, public pressure for a cleaner environment, and government regulations. Air quality data for the United States are readily available from government agencies and are used to document these trends for the rest of this chapter. Data for Europe, readily available on the website of the European Environment Agency, show similar trends for that part of the world.

Figure 6.1.3.1 shows the trends for emissions and aerial concentrations in the United States during each of four periods: 1980 to 2016, 1990 to 2016, 2000 to 2016, and 2010 to 2016. Sulfur dioxide emissions fell by 90% since 1980, carbon monoxide emissions by 73%, and emissions of nitrogen oxides by 62%. The declines in just the most recent period, the six years from 2010 to 2016, were substantial for every pollutant except particulate matter. Aerial carbon monoxide concentrations have fallen 85% since 1980, lead 99%, and nitrogen dioxide between 61% and 62%. The trend analysis reveals much of the improvement took place in only the past 16 years, since 2000, and that major improvements occurred in the past six years.

As noted in Section 6.1.2, the EPA tracks the percentage of children in the United States living in counties where they might be exposed to pollutant

Figure 6.1.3.1
Change in criteria pollutants in the United States, 1980–2016

A. Percent change in emissions of five criterion pollutants plus VOCs in the United States, 1980-2016

	1980 vs 2016	1990 vs 2016	2000 vs 2016	2010 vs 2016
Carbon Monoxide	-73	-66	-52	-21
Lead	-99	-80	-50	-23
Nitrogen Oxides (NO_x)	-62	-59	-54	-30
Volatile Organic Compounds (VOC)	-55	-42	-21	-10
Direct PM_{10}	-57	-18	-15	-4
Direct $PM_{2.5}$	---	-25	-33	-6
Sulfur Dioxide	-90	-89	-84	-66

B. Percent change in aerial concentration of six criteria pollutants in United States, 1980–2016

	1980 vs 2016	1990 vs 2016	2000 vs 2016	2010 vs 2016
Carbon Monoxide	-85	-77	-61	-14
Lead	-99	-99	-93	-77
Nitrogen Dioxide (annual)	-62	-56	-47	-20
Nitrogen Dioxide (1-hour)	-61	-50	-33	-15
Ozone (8-hour)	-31	-22	-17	-5
PM10 (24-hour)	---	-39	-40	-9
PM2.5 (annual)	---	---	-42	-22
PM2.5 (24-hour)	---	---	-44	-23
Sulfur Dioxide (1-hour)	-87	-85	-72	-56

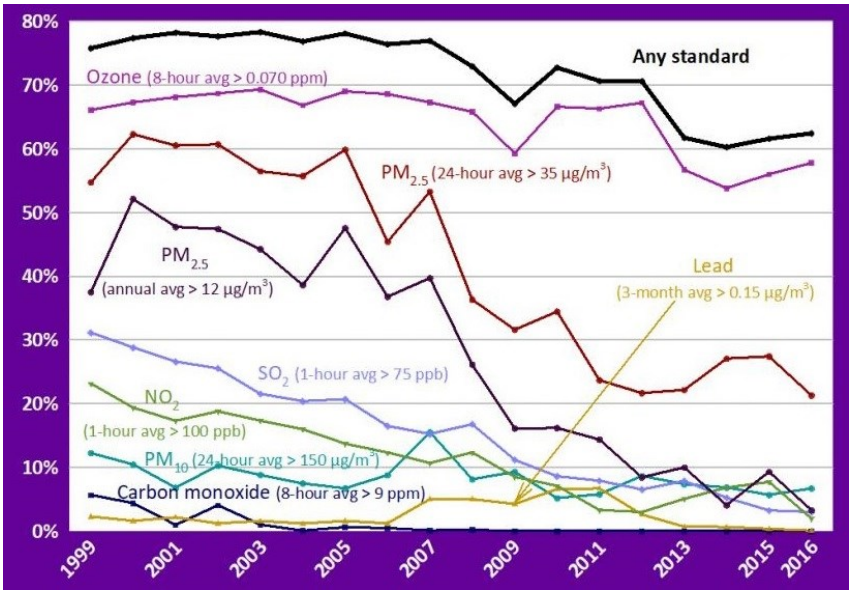
Source: EPA, 2018b.

concentrations higher than the levels of the current air quality standards. Its graph showing estimates for 1999–2016 appears as graph A in Figure 6.1.3.2. It shows exposure to what the EPA believes to be unsafe levels of exposure is in steep decline. For example, the percentage of children living in counties where they might be exposed to harmful levels of $PM_{2.5}$ decreased from 55% to 21%, to SO_2 from 31% to 3%, and to NO_2, from 23% to 2%. These are dramatic declines.

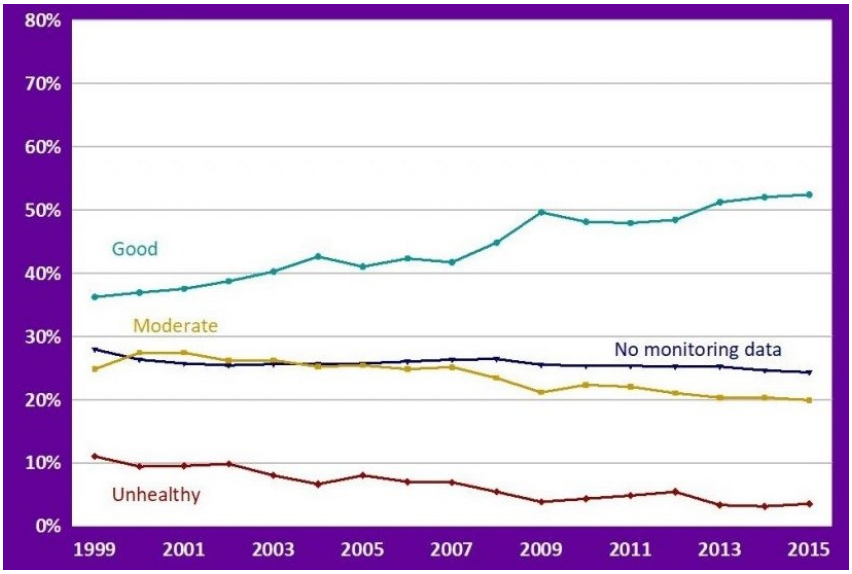
The EPA's "Air Quality Index," which combines and weights its measures of exposure to the six criteria pollutants, also shows a dramatic reduction in exposure to possibly harmful pollutants from 1999 to 2015. The EPA's graph showing changes in the percentage of days with "good," "moderate," or

Figure 6.1.3.2
Trends in U.S. Air Quality

A. Percentage of children ages 0 to 17 years living in U.S. counties with pollutant concentrations above the levels of the current air quality standards, 1999–2016



B. Percentage of days with good, moderate, or unhealthy air quality for children ages 0 to 17 years in the United States, 1999–2015



Source: EPA, 2018c.

"unhealthy" air quality for children from 1999 to 2015 appears in Figure 6.1.3.2 as graph B. The percentage of days during which children lived in counties where they might be exposed to what the EPA deems "unhealthy air" has declined from 9% in 1999 to 3% in 2015, while the percentage of children's days with "good" air quality increased from 36% in 1999 to 52% in 2015.

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6.1.4 Interpreting Exposure Data

> *Exposure to chemical compounds produced during the combustion of fossil fuels is unlikely to cause any fatalities in the United States.*

The chemistry, exposure data, and trends presented in Sections 6.1.1, 6.1.2, and 6.1.3 are a necessary though not sufficient basis for rendering findings on the possible health effects of emissions caused by the combustion of fossil fuels. We can begin by ruling out negative health effects due to "acid rain" and aerial emissions of mercury.

There is no biological mechanism whereby less basic rainwater would pose a threat to human health. Benarde (1987) reported that "an exhaustive search of the pertinent literature indicates that deleterious human health effects [of "acid rain"], if there are any, remain to be established. As a consequence of pollution abatement efforts the next 15 to 20 years should witness a reduction in acid levels. Accordingly, a worsening of current levels of chemical pollutants is not anticipated. Hence, a significant threat to public health via acid rain currently or in the foreseeable future, should not be expected." More recent efforts to link acid rain with human health effects focus on the contribution of SO_2 and NO_x emissions to the formation of fine particulate matter and not to acidification *per se* (Chestnut and Mills, 2005; Menz and Seip, 2004). Even EPA says "Walking in acid rain, or even swimming in a lake affected by acid rain, is no more dangerous to humans than walking in normal rain or swimming in non-acidic lakes" (EPA, n.d.).

Mercury is a genuine threat to human health. However, *exposure* to mercury in the United States and other developed countries is well within public safety levels. The National Research Council of the National Academies of Sciences determined in 2000 that 85 micrograms of mercury per liter (µg/L) or higher in cord blood was associated with early neurodevelopmental effects. According to the Centers for Disease Control and Prevention's *Fourth National Report on Human Exposure to Environmental Chemicals,* blood samples from 8,373 people taken in 2010 (the most current test data available) found 95% had mercury levels below 4.90 µg/L and "all blood mercury levels for persons in the Fourth Report were less than 33 µg/L" (CDC, 2018, Vol. 1, p. 319 and CDC, 2017).

The accumulation of methylmercury (MeHg) in fish tissue has been raised as a public health issue, but its accumulation depends on many environmental factors and is largely independent of concentrations of elemental mercury in the air (Mason *et al.,* 2005). Electricity generation using coal in the United States released an estimated 26.5 tons of mercury in 2011 and only 6.94 tons in 2016 (EPA, 2018). This is dwarfed by other emission sources: U.S. forest fires emit at least 44 tons per year; cremation of human remains, 26 tons; Chinese power plants, 400 tons; and volcanoes, subsea vents, geysers and other sources, approximately 9,000-10,000 tons per year (Soon and Driessen, 2011). Atmospheric concentrations of mercury do not coincide with changes in anthropogenic emissions, a reflection of the fact that humans account for less than 0.5% of all the mercury in the air and, as is the case with mercury in the oceans, the numerous natural cycles that affect its presence in the atmosphere. Soon and Monckton (2012) concluded an analysis of U.S. mercury control regulations as follows:

> The scientific literature to date strongly and overwhelmingly suggests that meaningful management of mercury is likely impossible,

> because even a total elimination of all industrial emissions, especially those from U.S. coal-fired power plants, will almost certainly not be able to affect trace, or even high, levels of MeHg that have been found in fish tissue over century-long time periods.

Globally, emissions of mercury have plummeted since governments around the world launched campaigns to reduce industrial emissions. Since 1990, nine European countries reduced their emissions by 85% or more and five (Sweden, Denmark, Norway, Ireland, and Croatia) now report zero emissions (European Environment Agency, n.d.) U.S. emissions from electricity generation fell 89% during the same period (Oakridge National Laboratory, 2017, figure ES1, p. viii). Exposure to mercury in the air (as opposed to ingesting paint chips that might contain lead and other avenues of exposure) is not a health threat in the United States or other developed countries today.

Regarding the remaining pollutants, EPA has established National Ambient Air Quality Standards (NAAQS) that it uses to determine which states, counties, and cities are "nonattainment" areas making them subject to EPA enforcement actions, and to define and report to Congress progress toward "good" air quality. To the public, failure to attain NAAQS may appear to be evidence of "unhealthy" air, and EPA encourages this perception. However, NAAQSs are set *orders of magnitude lower* than what the best available science suggests is a level where human health and public welfare are actually threatened (Belzer, 2012). This means failing to attain EPA's NAAQS does not mean an actual threat to human health exists (Belzer, 2012).

EPA standards are based on either the dose at which no adverse effect was observed (NOAEL) or the lowest dose at which an adverse effect was observed (LOAEL). When a LOAEL is used, the default safe threshold is reduced by a factor of ten to account for the unknown distance between the observed LOAEL and the unobserved NOAEL. If the LOAEL or NOAEL comes from an animal study, the default safe threshold is reduced by another factor of 10 to account for the possibility that humans are more sensitive than the most sensitive laboratory animal tested. Together, these two "safety adjustments" can reduce the safety threshold by a factor of 100.

A third default safety factor of ten is used to make sure the most susceptible members of the population are protected. A fourth factor of ten is applied when data is obtained from studies with less-than-lifetime exposure. A fifth factor of ten is applied when the database is incomplete. When all five safety factors are used, the composite safety factor is 10,000. This means the EPA standard would be 10,000 times more strict than what the actual public health research suggests is a dose that is dangerous to human health. Mercifully – and because such extreme precaution would subject it to ridicule in the public health community – EPA has adopted a policy whereby the total safety factor applied to any particular chemical is no more than 3,000, a still remarkably high risk multiplier (EPA, 2002, pp. 4-41).

Incredibly, this is not the only way the EPA errs on the side of setting its safety standards too low. Belzer (2012) identifies the following practices:

- Extrapolating human cancer risk at very low environmental levels from very high laboratory exposures to animals;
- Using default assumptions such as daily adult inhalation, drinking water consumption, and time spent outdoors that overstate the average;
- Reliance on simulation models instead of exposure data obtained from the risk scenario of interest;
- Estimating risks and benefits using exposures to a small fraction of the population, such as the 95th percentile, rather than the mean; and
- Extrapolation of risk from each step of a risk assessment means even small over-estimations produce very large reductions in the safety standard.

The result of these default options and assumptions is "cascading bias," which Belzer (2012, p. 13, fn. 28) defines as "when each of several terms in a point estimate of risk is upwardly biased, the point estimate is biased by the product of the biases." A bureaucracy's definition of acceptable risk is not a statement of relative risk based on toxicology or observation, or even derived from epidemiological associations, but the result of a political process that balances science with institutional goals, with the latter often influenced by subjective judgements about acceptable risk. As Belzer (2017) later observed, "EPA will strive for the highest estimate of risk that does not bring upon the Agency unbearable

ridicule. You simply cannot rely on the EPA risk assessment to give you an unvarnished perspective. When given an EPA risk assessment, all you know is risk can't be any worse" (p. 3). In short, the EPA's NAAQS should not be accepted as a definition of "safe" or "unsafe" air, even if they were arrived at by close attention to the science and with utmost integrity.

As the discussion of particulate matter later in this chapter demonstrates, the EPA's NAAQS were not determined by "close attention to the science and with utmost integrity." The process by which they were established demonstrates an almost shocking degree of manipulation, dishonesty, and refusal to acknowledge research findings that run counter to the agency's policy agenda. That they are still defended today by the EPA bureaucracy and the coterie of well-paid academics it has assembled to provide the appearance of scientific fact, if not by the agency's administrator, reveals a flawed culture inside a failed government agency.

Later in this chapter these issues – along with whether small-associations epidemiology is a legitimate basis for air quality standards at all, particularly when the EPA's philosophy is that there is no safe level of any primary air pollutant (the "linear no-threshold" (LNT) dose-response relationship) – are addressed in some depth. But even before those concerns are addressed, the evidence is clear that *very few people in the United States are exposed to pollutants at levels likely to pose a threat to human health.* The same is almost certainly sure for much of Europe and developed countries around the world. Further confirmation can be seen in the inability of the EPA to show any declines in mortality in the past two decades that could be attributed to the decline in particulate matter or other pollutants, a decline that should be apparent if the criteria pollutants were once a human health threat at levels higher than today's.

The very low and falling number of children who may be exposed to dangerous chemicals and the almost ridiculously low levels of exposure chosen by EPA for its NAAQS have never been reported by the press, but the EPA's highly speculative numbers of people "killed" every year by particulate matter and ozone appear countless times in headlines and the fundraising letters of environmental advocacy groups such as the American Lung Association (ALA, n.d.). They also appear in estimates of the "social cost" of fossil fuels and of future climate change and are used to justify anti-fossil fuel regulations. But as the analysis in this section shows, the real public health risks of exposure to EPA's six criteria pollutants are negligible.

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6.2 Failure of the EPA

The data presented in Section 6.1 show dramatic progress has been made in reducing emissions of possibly harmful chemical compounds produced during the use of fossil fuels, and more importantly reducing human exposure to those chemicals. While giving credit for this achievement to the government agencies most responsible for enforcing environmental protection laws might seem appropriate, this is not the case. As was shown in Chapter 1, Section 1.3.1, improvements in air quality in the United States began in the 1940s and 1950s, long before the national government and U.S. Environmental Protection Agency (EPA) got involved. Hayward writes,

> The chief drivers of environmental improvement are economic growth, constantly increasing resource efficiency, technological innovation in pollution control, and the deepening of environmental values among the American public that have translated to changed behavior and consumer preferences. Government regulation has played a vital role, to be sure, but in the grand scheme of things regulation can be understood as a lagging indicator, often achieving results at needlessly high cost, and sometimes failing completely (Hayward, 2011, p. 2).

Schwartz and Hayward (2007) note, "Improvements in air quality are not unique. Other environmental problems, such as water quality, were also improving before the federal government took over regulatory control. Likewise, other risks were dropping without federal regulation. Per mile of driving, the risk of dying in a car accident dropped 75% between 1925 and 1966 – the year Congress adopted the National Traffic and Motor Vehicle Safety Act and created the National Highway Traffic Safety Administration. Between 1930 and 1971 – the year that the Occupational Safety and Health Administration was created – the risk of dying in a workplace accident dropped nearly 55%." In all these cases – air quality, automobile safety, and workplace safety – the rate of improvement was about the same before and after the federal government nationalized policy. Without doubt, improvements would have continued in all these areas even if the federal government had not taken the regulatory reins away from the states.

As this section will show, the EPA has often been more of a hindrance than a help in advancing the cause of environmental protection in the United States. The discussion in Chapter 1, Section 1.4.3, of how government bureaucracies work (and do not work) provides good background for this discussion. This section begins by explaining how the EPA's mission has evolved over time in response to congressional and public pressure as well as the natural tendencies of bureaucracies, creating a culture that cannot concede the possibility that human emissions of toxic substances are *not* a major public health crisis in need of the EPA's expert attention. This bias contaminates all of its scientific research, making it unreliable. Next, the EPA's repeated and flagrant violation of the basic rules of the scientific method is documented. Finally, the loss of integrity and outright corruption that have affected the agency are documented. Along the way, parallels to the mission, methodology, and corruption of the United Nations' Intergovernmental Panel on Climate Change (IPCC), documented in Chapter 2, are identified.

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6.2.1 A Faulty Mission

> *Due to its faulty mission, flawed paradigm, and political pressures, the U.S. Environmental Protection Agency (EPA) is an unreliable source of research on air quality and its impact on human health.*

Chapter 2 explained how the mission of the United Nations' Intergovernmental Panel on Climate Change (IPCC) – to find and document the human impact on the global climate – blinded it to the possibility that natural variability could explain most or even all of the warming experienced in the late twentieth century and thus obviate the need for an organization tasked with solving the nonexistent problem. The EPA's mission similarly blinds it to the possibility that natural causes of cancer and other diseases may outweigh any effects of man-made chemical compounds.

EPA's website says "Born in the wake of elevated concern about environmental pollution, the EPA was established on December 2, 1970 to consolidate in one agency a variety of federal research, monitoring, standard-setting and enforcement activities to ensure environmental protection. Since its inception, the EPA has been working for a cleaner, healthier environment for the American people" (EPA, 2018). Elsewhere on its website, the EPA says its mission is simply "to protect human health and the environment."

The simple mission statement obscures profound conflicts of interest that prevent the EPA from making good on its promise. Like many government agencies, the EPA was given not one but three mandates: to identify, evaluate, and solve a social problem. But combining all three responsibilities in the same entity means the agency has no incentive to decide the social problem does not merit a significant investment of public monies to solve, or that the problem, should it exist, even could be solved. The agency is also charged with measuring its own success and then reporting it to those who control its funding and future existence. As explained in Chapter 1, Section 1.4.3, the heads of such agencies, no matter how honest or well-intended, cannot objectively evaluate their own performances (Savas, 2000, 2005). Schwartz and Hayward (2007) explained it the following way:

> The Clean Air Act charges the EPA with setting air pollution health standards. But this means that federal regulators decide when their own jobs are finished. Not surprisingly, no matter how clean the air, the EPA continues to find unacceptable risks. The EPA and state regulators' powers and budgets, as well as those of environmentalists, depend on a continued public perception that there is a serious problem to solve. Yet regulators are also major funders of the health research intended to demonstrate the need for more regulation. They also provide millions of dollars a year to environmental groups, which use the money to augment public fear of pollution and seek increases in regulators' powers. These conflicts of interest largely explain the ubiquitous exaggeration of air pollution levels and risks, even as air quality has steadily improved (2007, pp. 11–12).

The EPA quickly grew in size and influence. Its resources and power naturally attracted the attention of interest groups. Jay Lehr, Ph.D., a scientist who was involved in the founding of the EPA, wrote in 2014, "Beginning around 1981, liberal activist groups recognized the EPA could be used to advance their political agenda by regulating virtually all human activities regardless of their impact on the environment. Politicians recognized they could win votes by posing as protectors of the public health and wildlife. Industries saw a way to use regulations to handicap competitors or help themselves to public subsidies" (Lehr, 2014).

As reported by Chase (1995), in 1993 President Bill Clinton signed the International Convention on Biological Diversity and just months later created the President's Council on Sustainable Development, making "'ecosystem protection" the EPA's highest mandate. "Under the new rules [EPA's] primary goal would no longer be to protect public health. Rather, it would seek to save nature instead" (p. 91). Evidence that the change in mission affected the EPA's research since 1993 can be found in the fact that in 1987, 1990, and 1991 the agency produced a series of reports recognizing the impacts of pollution (not only air pollution but also impacts on water and food and exposure to toxic waste) were small relative to other human health risks (EPA, 1987, 1990, 1991), but since then it has embraced a "zero risk" paradigm whereby *any* human impact on the environment, no matter how small, is regarded as justification for government regulation (e.g., EPA, 2004, 2009). Protecting public health has become a pretense for stopping any human activity that has any impact at all on the environment. Such a broad definition of "environmental protection" gives the agency license to regulate virtually every human activity.

The War on Cancer

Much as the IPCC assumes only man can cause climate change, the EPA's mission leads it to assume that natural causes of cancer and other diseases either do not exist or do not matter to the regulatory process. In both cases the assumptions are false, and they contaminate and often invalidate much of what both the IPCC and the EPA do.

The EPA ignores and even hides from the public evidence that man-made chemicals are trivial contributors to the nation's disease and mortality rates. For example, Bruce N. Ames and Lois Swirsky Gold, two distinguished medical researchers at the University of California-Berkeley, pointed out that "99.99% of all pesticides in the human diet are natural pesticides from plants" (Ames *et al.,* 1990). "All plants produce toxins to protect themselves against fungi, insects, and animal predators such as humans. Tens of thousands of these natural pesticides have been discovered, and every species of plant contains its own set of different toxins, usually a few dozen. When plants are stressed or damaged (when attacked by pests), they greatly increase their output of natural pesticides, occasionally to levels that are acutely toxic to humans" (Ames and Gold, 1993, p. 157. See also Ames, 1983, and Ames *et al.*, 1990).

The EPA's focus on man-made chemical compounds as the cause of negative health effects was reinforced by political constraints placed on the agency. According to Kent and Allen (1994), "The strong political pressures in the Congress to legislate risk levels at or near zero can have a serious impact on the costs of environmental programs. To the extent that zero risk statutes are not feasible, they also threaten the overall credibility of the nation's environmental efforts. Statutory language pursuing 'zero discharge' and extremely low cleanup standards for superfund sites could force huge social investments that would divert scarce resources from even higher-risk problems" (Kent and Allen, 1994, p. 65).

The EPA's campaign to regulate away all risks is doomed to fail since risk is inherently subjective. Lash (1994) explained why this is so:

> Some people willingly die to protect their children; others abandon them. Some choose to die for religious faith, or honor, or country; others use those concepts as rhetorical symbols to achieve selfish ends. *It is the interaction of what we value with what we believe to be reality that determines how we act. Given identical information and alternatives, different people make different choices.* The debate over what the comparative risk process is, what it should be, and whether it is essential or pernicious as a tool for public policy is a debate about decisions, who should make them, and how (p. 70).

He added, "Whether the issue is smoking or global climate change, normative questions are inextricably woven into the assessment of risk" (Lash, 1994, p. 76). Furedi (2010) noted, "frequently, worst-case thinking displaces any genuine risk-assessment process. Risk assessment is based on an attempt to calculate the probability of different outcomes. Worst-case thinking – these days known as precautionary thinking – is based on an act of imagination. It imagines the worst-case scenario and demands that we take action on that basis. … In the absence of freedom to influence the future, how can there be human responsibility? That is why one of the principal accomplishment[s] of precautionary culture is the normalisation of irresponsibility. That is a perspective that we need to reject for a mighty dose of humanist courage."

Ames and Swirsky Gold warned, "Excessive concern for pollution will not improve public health – and, in the confusion, may cause us to neglect important hazards, such as smoking, alcohol, unbalanced diets (with too much saturated fat and cholesterol, and too few fruits and vegetables), AIDS, radon in homes, and occupational exposures to chemicals at high levels. The progress of technology and scientific research is likely to lead to a decrease in cancer death rates and incidence of birth defects, and an increase in the average human life span (Ames and Gold, 1993, p. 179).

The War on Coal

President Barack Obama understood clearly how the EPA could be used to advance his political agenda, which included penalizing manufacturers and the fossil fuel industry and rewarding high-tech companies and the alternative energy industry. When campaigning for president in January 2008, Obama told the editorial board of *The San Francisco Chronicle,* "If somebody wants to build a coal-fired power plant, they can. It's just that it will bankrupt

them," and later, "Under my plan … electricity rates would necessarily skyrocket" (Martinson, 2012).

Once elected, Obama proceeded to "weaponize" the EPA against the fossil fuel industry. His administration promulgated new rules and tightened older ones in an effort to strangle the coal industry. According to Orr and Palmer (2018) those efforts included:

- Clean Power Plan
- Cross-State Air Pollution Rule
- More stringent National Ambient Air Quality Standards (NAAQS) for mercury, particulate matter, and ozone
- Cooling Water Intake Rule
- Coal Combustion Residuals Rule
- Carbon Pollution Standards for New Plants
- Effluent Limitations Guidelines
- Stream Protection Rule
- Department of the Interior bans on new mines on public lands and mountaintop mining

Many of these regulations could not be justified by cost-benefit analysis, a point that will be documented in Chapter 8. They were adopted solely as part of a "war on coal" modeled after the war on cancer to force a transition from fossil fuels to alternative energy sources (wind and solar) or mandatory energy conservation. Wrote Orr and Palmer,

> The war on coal was very real. It was led from the White House and backed by hundreds of millions of dollars in funding from left-wing foundations including the Rockefeller Brothers, the Hewlett Foundation, the MacArthur Foundation, Bloomberg Philanthropies, and even Chesapeake Energy, a natural gas drilling company seeking to grow demand for its product. These millions were funneled to environmental activist groups including Greenpeace, the Sierra Club, and Natural Resources Defense Council. Just one donor, billionaire Michael Bloomberg, has given more than $168 million to the Sierra Club to support the effort (citing Suchecki, 2015, and Brown, 2017).

Members of the Obama administration sometimes acknowledged the real political objective of the campaign. EPA Administrator Gina McCarthy testified before the U.S. Senate Environment and Public Works Committee on July 23, 2014: "The great thing about this [Clean Power Plan] proposal is that it really is an investment opportunity. *This is not about pollution control*" (McCarthy, 2014, italics added). Secretary of State John Kerry described U.S. policy regarding coal-fueled power plants: "We're going to take a bunch of them out of commission" (Davenport, 2014). In a December 9, 2015 address at the United Nations conference where the Paris Accord was negotiated, Kerry was remarkably frank about *how the treaty was not, after all, about protecting the environment.* He said:

> The fact is that even if every American citizen biked to work, carpooled to school, used only solar panels to power their homes, if we each planted a dozen trees, if we somehow eliminated all of our domestic greenhouse gas emissions, guess what – that still wouldn't be enough to offset the carbon pollution coming from the rest of the world.
>
> If all the industrial nations went down to zero emissions – remember what I just said, all the industrial emissions went down to zero emissions – it wouldn't be enough, not when more than 65% of the world's carbon pollution comes from the developing world (Quoted in Watts, 2015).

The EPA was a willing accomplice in this political campaign to end the world's reliance on fossil fuels. An international climate treaty would have provided legal as well as political cover for exercising even more power over sectors of the economy that constitutionally and by tradition were the reserve of state governments or left unregulated. The Paris Accord would have been the capstone of an eight-year march to power under a president devoted to transforming the nation's energy, manufacturing, and agricultural sectors into a new system in which the agency would be empowered to regulate virtually every aspect of life in America. Today, the EPA has a budget of $8 billion and 12,000 full-time staff. Its regulations already account for more than half of the

total cost of complying with federal regulations (Crews, 2018). But like all bureaucracies, it wanted to grow.

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6.2.2 Violating the Bradford Hill Criteria

EPA makes many assumptions about relationships between air quality and human health, often in violation of the Bradford Hill Criteria and other basic requirements of the Scientific Method.

Belzer (1994) wrote: "Science involves a set of rigorous procedures for sorting out evidence from assertions, fact from fiction, and causation from association. Scientists develop theories of physical, biological, and human systems and craft testable hypotheses, all the while subjecting their efforts to critical review by their peers and the marketplace of ideas" (p. 176). As discussed in Chapter 2, Section 2.2.1, the scientific method requires researchers to formulate and disprove an alternative *null* hypothesis. In the case of man-made climate change, the hypothesis is that dangerous climate change is resulting, or will result, from human-related greenhouse gas emissions. A reasonable null hypothesis is that changes in global climate indices and the physical environment are the result of natural variability. Another null hypothesis could be that any hypothetical mechanism that produces some global warming will not produce a climate catastrophe. Climate scientists have failed to disprove either null hypothesis, meaning the original hypothesis has not been proven to be correct.

The scientific method imposes the same requirements on the debate over the human health effects of the chemical compounds produced during the combustion of fossil fuels. The EPA has compiled mountains of assumptions, observational studies, and circumstantial evidence in support of its implicit hypothesis that man-made chemical compounds cause measurable and harmful effects on human health, while failing to invalidate the null hypothesis that observed death rates and illnesses are the result of other causes including aging, genetics, naturally occurring carcinogens, unhealthy behaviors such as smoking and poor nutritional choices, and other forms of risky behavior. Instead of testing the elements of its hypothesis for validity, the EPA adopted the fallacies of anchoring (defending a previous decision or piece of information against new evidence), confirmation bias (interpreting all new evidence as confirmation of an existing belief), and cherry-picking arguments and information to support its hypothesis.

Bradford Hill Criteria

Much of the public concern over man-made chemicals is due to the assumption by policymakers, regulators, and advocates that evidence of an *association* between a chemical in the air or water and a human health effect is evidence that the chemical *causes* that effect. Because distinguishing between coincidence and correlation, on the one hand, and causal relationships on the other can be very difficult in matters of public health, an English epidemiologist named Sir Austin Bradford Hill (1897–1991) established in 1965 what has become known as the Bradford Hill Criteria (BHC), nine minimal conditions necessary to provide evidence of a causal relationship between an event (in this case exposure to an air pollutant) and a health effect (illness or mortality). The criteria are presented in Figure 6.2.2.1.

Similar standards have been proposed by other researchers (e.g., Henle-Koch-Evans postulates (Evans, 1976, 1977) and Susser, 1973, 1991). Commenting on the Bradford Hill Criteria, Foster *et al*. (1993) wrote,

> Most scientists would agree that they are not standards of scientific proof, or at least not the high standards that the HKE postulates are generally assumed to be. Nevertheless, Hill's criteria have been widely influential in epidemiology. The fact that epidemiologists feel it necessary to debate them at all underscores the frequent difficulty of interpreting epidemiological evidence. At the least, it points to the need for a holistic assessment of the data, and the recognition that the evidence will never be completely consistent (p. 10).

The Bradford Hill Criteria are endorsed by the Federal Judicial Center (FJC), an education and research agency of the United States federal courts established by an Act of Congress (28 U.S.C. §§ 620–629) in 1967, at the recommendation of the Judicial Conference of the United States. FJC's reference manual for judges, titled the *Reference Manual on Scientific Evidence*, provides expert advice for determining the admissibility of scientific evidence in U.S. federal courts and advises federal judges and lawyers practicing in federal courts to adhere to that advice in complying with the rules of evidence. The latest (third) edition is co-published by

Figure 6.2.2.1
Bradford Hill Criteria for establishing a causal relationship

1. *Strength of the association.* Relative risk (the incidence rate in the exposed population divided by the rate in the unexposed population) measures the strength of the association. The higher the relative risk, the greater the likelihood that the relationship is causal.

2. *Consistency of the observed association.* Has it been repeatedly observed by different persons, in different places, circumstances, and times?

3. *Specificity of the association.* Causation is most likely when the association is limited to specific occupations, particular sites, and types of diseases.

4. Temporal relationship of the association. The effect must occur after the cause.

5. *A dose-response curve.* The higher the dose, the higher the incidence of disease or mortality. A higher dose should not lead to less, rather than greater, harmful effects.

6. *Biological plausibility.* A plausible mechanism between cause and effect is helpful, but since it depends on the biological knowledge of the day, "this is a feature I am convinced we cannot demand."

7. *Coherence with current knowledge.* The cause-and-effect interpretation of the data should not seriously conflict with the generally known facts of the natural history and biology of the disease.

8. *Experimental evidence.* Before-and-after comparisons can reveal the strongest support for the causation hypothesis.

9. *Analogizing to similar known causes.* Knowing the effects of a drug such as thalidomide or a disease such as rubella on pregnant women makes it more plausible that other drugs and diseases might have similar effects.

Source: Hill, 1965.

the National Research Council of the National Academies (FJC, 2011).

The manual's chapter on epidemiology was coauthored by a distinguished legal scholar, Michael D. Green, J.D., the Bess & Walter Williams Chair in Law, Wake Forest University School of Law, and two distinguished epidemiologists: D. Michal Freedman, J.D., Ph.D., M.P.H., epidemiologist in the Division of Cancer Epidemiology and Genetics at the National Cancer Institute in Bethesda, Maryland, and Leon Gordis, M.D., M.P.H., Dr.P.H., professor emeritus of epidemiology at Johns Hopkins Bloomberg School of Public Health and professor emeritus of pediatrics at Johns Hopkins School of Medicine, Baltimore, Maryland.

The authors (on p. 566) define relative risk (the focus of BHC #1) as the ratio of the incidence rate (often referred to as incidence) of disease or mortality in exposed individuals to the incidence rate in unexposed individuals:

$$RR = \frac{\text{(Incidence rate in the exposed)}}{\text{(Incidence rate in the unexposed)}}$$

The FJC authors stressed, "The relative risk is one of the cornerstones for causal inferences. Relative risk measures the strength of the association. The higher the relative risk, the greater the likelihood that the relationship is causal" (p. 602). On the important question of how high a relative risk finding must be to pass the legally required threshold (in civil cases) of "more likely than not," or at least 51% probable, the FJC authors wrote:

> Some courts have reasoned that when epidemiological studies find that exposure to

> the agent causes an incidence in the exposed group that is more than twice the incidence in the unexposed group (i.e., a relative risk greater than 2.0), the probability that exposure to the agent caused a similarly situated individual's disease is greater than 50%. These courts, accordingly, hold that when there is group-based evidence finding that exposure to an agent causes an incidence of disease in the exposed group that is more than twice the incidence in the unexposed group, the evidence is sufficient to satisfy the plaintiff's burden of production and permit submission of specific causation to a jury. In such a case, the factfinder may find that it is more likely than not that the substance caused the particular plaintiff's disease. Courts, thus, have permitted expert witnesses to testify to specific causation based on the logic of the effect of a doubling of the risk (FJC, 2011, p. 612).

Since this is an important and contentious point in the air quality debate, it is worth quoting the FJC at greater length on this question:

> Having additional evidence that bears on individual causation has led a few courts to conclude that a plaintiff may satisfy his or her burden of production even if a relative risk less than 2.0 emerges from the epidemiological evidence. For example, genetics might be known to be responsible for 50% of the incidence of a disease independent of exposure to the agent. If genetics can be ruled out in an individual's case then a Relative Risk greater than 1.5 might be sufficient to support an inference that the substance was more likely than not responsible for the plaintiff's disease. ...
>
> Eliminating other known and competing causes increases the probability that the individual's disease was caused by the exposure to the agent. ...
>
> Similarly, an expert attempting to determine whether an individual's emphysema was caused by occupational chemical exposure would inquire whether the individual was a smoker. By ruling out (or ruling in) the possibility of other causes, the probability that a given agent was the cause of an individual's disease can be refined. Differential etiologies are most critical when the agent at issue is relatively weak and is not responsible for a large proportion of the disease in question.
>
> Although differential etiologies are a sound methodology in principle, this approach is only valid if general causation exists and a substantial proportion of competing causes are known. Thus, for diseases for which the causes are largely unknown, such as most birth defects, a differential etiology is of little benefit. And, like any scientific methodology, it can be performed in an unreliable manner (pp. 616–7).

The FJC's insistence on RRs of 2 (or at least 1.5) is lower than what other researchers in the field expect. Arnett (2006) wrote, "[O]bservational epidemiological studies, unless they show overwhelmingly strong associations – on the order of an increased relative risk of 3.0 or 4.0 – do not indicate causation because of the inherent systematic errors that can overwhelm the weak associations found. These errors include confounding factors, methodological weaknesses, statistical model inconsistencies, and at least 56 different biases" (p. 1).

The EPA and the voluminous research it claims in support of its regulations violate this first and most important of the Bradford Hill Criteria by relying on observational studies with RRs less than 4.0, 3.0, 2.0, and even the lowest standard of 1.5. Indeed, as shown in the next section, the studies on which the EPA relies often find zero or even negative RRs that are hidden in meta-analyses or simply left out of their reviews of the literature. The EPA simply assumes associations, even very weak ones, are proof of causation.

Another violation of the Bradford Hill Criteria is the EPA's reliance on animal experiments in which mice and rats are exposed to near-toxic doses of toxins. The EPA assumes, falsely, that such experiments produce reliable evidence of the risk to humans exposed to far lower levels of those toxins in daily life (Whelan, 1993). That assumption is contradicted by current toxicological knowledge (BHC #7). Ames and Gold (1993) wrote:

> Animal cancer tests are conducted at near-toxic doses of the test chemical that cannot

> predict the cancer risk to humans at the much lower levels to which they are typically exposed. The prediction of cancer risk requires knowledge of the mechanisms of carcinogenesis, which is progressing rapidly. Recent understanding of these mechanisms undermines many of the assumptions of current regulatory policy regarding rodent carcinogens and requires a reevaluation of the purpose of routine animal cancer tests (p. 154).

Commenting on the use of animal testing in the search for cures to cancer rather than possible causes, Mak, Evaniew, and Lost (2014) write, "there is a growing awareness of the limitations of animal research and its inability to make reliable predictions for human clinical trials. Indeed, animal studies seem to overestimate by about 30% the likelihood that a treatment will be effective because negative results are often unpublished. Similarly, little more than a third of highly cited animal research is tested later in human trials. Of the one-third that enter into clinical trials, as little as 8% of drugs pass Phase I successfully."

A third violation of BHC and the scientific method is EPA's default assumption of a linear no-threshold (LNT) dose-response relationship. For example, EPA assumes there is no safe threshold of exposure to fine particles ($PM_{2.5}$) so that even brief exposure to extremely low levels of $PM_{2.5}$ (like those calculated in Section 6.1.3) can cause illnesses and death within hours of inhalation (i.e., "short-term" or literally "sudden death") and that long-term (i.e., years or decades) exposure to low levels of $PM_{2.5}$ also can cause premature death (EPA, 2009; Samet, 2011, p. 199). EPA reasons that if exposure to large concentrations has negative health effects, then exposure to even tiny amounts also must have negative effects, albeit smaller ones. EPA's LNT assumption for $PM_{2.5}$ and other pollutants has been vigorously disputed (e.g., Calabrese and Baldwin, 2003; Calabrese, 2005, 2015).

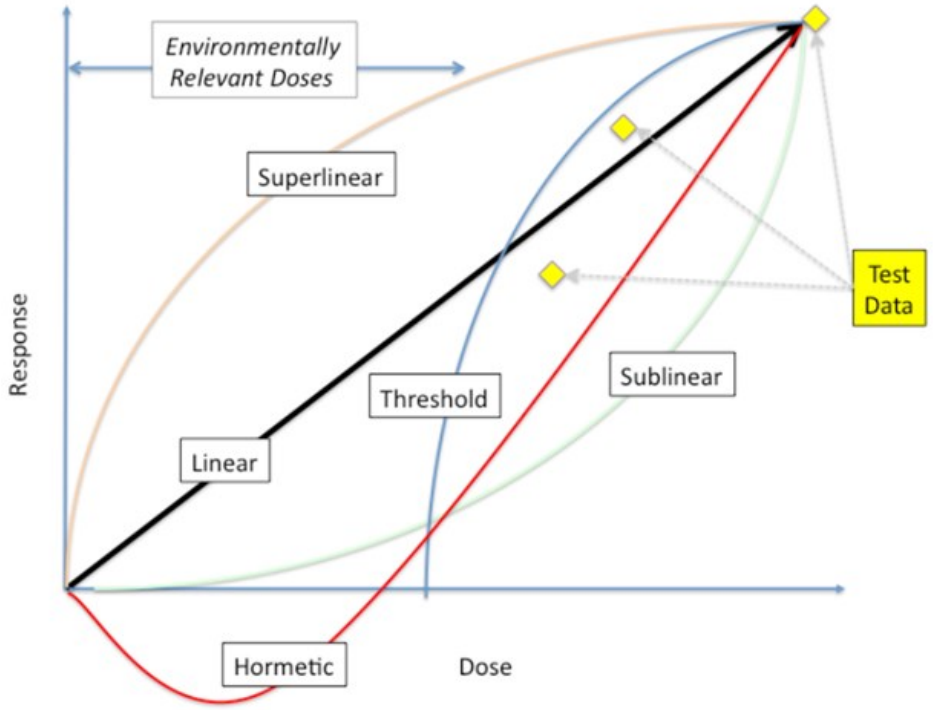
Calabrese and Baldwin (2003) explained, "The dose-response revolution is the changing perception that the fundamental nature of the dose response is neither linear nor threshold, but U-shaped," meaning extremely low exposures of some toxins may have *positive* health effects (called hormesis). This contradicts EPA's assumption that responses are linear all the way down to zero exposure, and if true it invalidates much of its health effects claims relying on this assumption. Figure 6.2.2.2 shows some of the alternative dose-response curves that EPA simply assumes away.

Calabrese and Baldwin continue,

> [A]cceptance that hormetic-like U-shaped dose responses are widespread and real has been difficult to achieve. The reasons for this are many, but in general include the following. First, the field of toxicology has become progressively and insidiously dependent on the role of government to set the national (and international) toxicological agenda. This agenda translates into designing and interpreting studies to fit into current risk assessment paradigms. That is, in the case of noncarcinogens, regulatory agencies design hazard assessment methodology to provide a NOAEL [no-observed-adverse-effect-level], whereas in the case of carcinogens, the study needs data that can be employed to estimate low-dose cancer risk. Such NOAEL and/or low-dose evaluations are dominating concerns. These controlling governmental regulatory perspectives have provided a seductive focus on toxicological thinking, providing the flow of financial resources and forcing private-sector and academic institutions to respond to such initiatives (*Ibid.*).

Calabrese and Baldwin's account is consistent with what we know about how government bureaucracies operate (see Chapter 1, Section 1.4) and how funding can bias research findings (see Chapter 2, Section 2.2.4). The EPA's linear no-threshold assumption means when large populations are involved, such as the population of the United States (approximately 326 million), simple math allows it to claim that even tiny amounts of an air pollutant with very small effects are responsible for thousands of deaths each year. Such claims generate favorable headlines, please political overseers, and justify a bigger research budget next year. But in fact, it is just as likely that those low levels of exposure have *positive* health effects or no effect at all. Government bureaucrats, politicians, the media, and environmental activists have no reason to let the public know that EPA's claims are implausible and even counterfactual (see Altman, 1980; Whelan, 1993; Avery, 2010; Milloy, 2001, 2016).

Figure 6.2.2.2
Alternative ways to extrapolate from high to low doses



Source: Belzer, 2012, Figure A, p. 8.

Finally, the EPA's paradigm assumes that reducing potential health threats by reducing emissions is superior to making investments in health promotion, even though the latter may be far more cost effective. Focusing only on small and hypothetical health benefits, often achieved at enormous costs by further reducing already de minimus emissions of toxins, the agency misses significant opportunities for protecting public health by reforming existing policies that perversely reward harmful behavior or by making targeted public investments in improving nutrition, safety, or education. For example, Gough (1990) determined that if the EPA's estimates of cancer risks from environmental exposures were correct and if its regulatory programs were 100% successful in controlling those exposures, the agency could eliminate only between 0.25 and 1.3% of all cancers. Hattis and Goble (1994) also expressed concern that the EPA is taking resources away from solving more urgent problems (p. 125).

Just as the IPCC and its allies in the climate change debate closed ranks against distinguished climate scientists who questioned their disregard of the basic requirements of the scientific method, the EPA and its allies attacked Ames, Feinstein, Calabrese, and other highly qualified critics. Writing in 1991, Feinstein observed,

> In previous eras of medical history, when major changes were proposed in customary scientific paradigms, the perceived threats to the status quo led to profound intellectual discomforts. Rational discussion of the proposed changes was sometimes replaced by passionate accusations about ethical behavior. A similar situation may arise in epidemiology today, as fundamental problems are noted in paradigmatic scientific methods, and as the available epidemiological evidence is used not only in public policy controversies, but particularly in adversarial legal conflicts. When the basic scientific quality of epidemiological evidence

> and methods is questioned, defenders of the status quo may respond by castigating the dissenters as fools or heretics, or by insinuating that they have been bribed with consultation honoraria (Feinstein, 1991, abstract).

* * *

In conclusion, the EPA assumes its task is to accumulate evidence in support of a self-serving hypothesis rather than disprove the null hypothesis that observed rates of death and illnesses are the result of causes other than the chemicals produced by modern industrial society. It assumes that association equals causation, administering massive doses of chemicals to laboratory animals predicts the human health impacts of much lower levels of exposure, and that even brief exposure to low levels of some pollutants can cause disease or death. All of these assumptions violate the Bradford Hill Criteria and other requirements of the scientific method, rendering EPA's science an unreliable guide for researchers and policymakers.

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6.2.3 Lack of Integrity and Transparency

The EPA has relied on research that cannot be replicated and violates basic protocols for conflict of interest, peer review, and transparency.

In 2018, EPA Administrator Scott Pruitt acknowledged his agency had been "weaponized" by the previous administration to wage a "war on fossil fuels." "The key to me," Pruitt told a reporter for *The Daily Signal*, "is that weaponization of the agency that took place in the Obama administration, where the agency was used to pick winners and losers. Those days are over" (Bluey, 2018).

Pruitt went on to say, "Can you imagine, in the first instance, an agency of the federal government, a department of the U.S. government, declaring war on a sector of your economy? Where is that in the statute? Where does that authority exist? It doesn't. And so to restore process and restore commitment to doing things the right way, I think we've seen tremendous success this past year" (Bluey, 2018) About the Paris Accord, which President Donald Trump had said the United States would exit, Pruitt said, "What was decided in Paris under the past administration was not about carbon reduction. It was about penalties to our own economy because China and India, under that accord, didn't have to take any steps to reduce CO_2 until the year 2030. So, if it's really about CO_2 reduction, why do you let that happen?"

As described in Section 6.2.1, the "war on coal" was real. Burnett (2018) writes, "Nearly a year into his presidency, Obama's Environmental Protection Agency (EPA) issued an endangerment finding ruling carbon dioxide, the gas plants need for life and every human and animal exhales, a danger to human health or the environment. Never before had EPA found a naturally occurring chemical dangerous at levels that have no toxic effect. During his tenure, Obama also successfully pressured Congress to increase the subsidies to wind and solar power plants and directed agencies such as EPA to expand their regulatory authority to tighten regulations on coal-fired power plants. Combined with competition from natural gas, these regulations and subsidies caused the premature closure of more than 250 coal-fired power plants nationwide."

Major regulatory decisions, such as the agency's finding that carbon dioxide endangered public health and therefore could be regulated by the EPA under the Clean Air Act, were rushed through without the documentation required for a major rule and even without approval by the EPA's Science Advisory Board. This follows a long history of the EPA refusing to respond to outside criticism, reliance on a small cabal of favored researchers, refusal to consider research that contradicts its findings, and general lack of transparency (see Expert Panel, 1992; GAO, 2008, 2011; NRC, 2011; Smith, 2014; Carna, 2015b).

Many authors have reported the lack of integrity and often outright corruption that have characterized the EPA. Lehr (2014) wrote, "The vague language of the federal environmental statutes and the corresponding massive delegation of authority to the EPA to make law, enforce law, and adjudicate violations concentrate tremendous power in the hands of the agency, breeding insensitivity, zealotry, and abuse. Experience has shown that regulatory agencies will tend to expand until checked, and the potential for regulatory expansion at the EPA, unbounded as it is by congressional language, is vast."

A sample of books documenting corruption inside the EPA appears in Figure 6.2.3.1. Following the table are brief reports of some especially egregious examples of corruption inside the agency.

The John Beale Case

One of the highest-salaried EPA officials responsible for setting NAAQS for particulate matter and ozone in the 1990s and for the "Endangerment Finding" for carbon dioxide in 2009 "is a convicted felon who

Figure 6.2.3.1
Exposés of lack of integrity and corruption inside the EPA

Ron Arnold, *Freezing in the Dark: Money, Power, Politics and the Vast Left Wing Conspiracy,* 2007.

Wilfred Beckerman, *Through Green-Colored Glasses: Environmentalism Reconsidered,* 1996.

Larry Bell, *Climate of Corruption: Politics and Power Behind the Global Warming Hoax*, 2011.

James T. Bennett and Thomas J. DiLorenzo, *Cancer Scam: Diversion of Federal Cancer Funds to Politics,* 1998.

Alex B. Berezow and Hank Campbell, *Science Left Behind: Feel-Good Fallacies and the Rise of the Anti-Scientific Left*, 2012.

Rupert Darwall, *The Age of Global Warming: A History*, 2013.

James V. DeLong, *Out of Bounds, Out of Control: Regulatory Enforcement at the EPA*, 2002.

Jeff Gillman and Eric Heberlig, *How the Government Got In Your Backyard,* 2011.

Indur M. Goklany, *The Precautionary Principle: A Critical Appraisal of Environmental Risk Assessment,* 2001.

Geoffrey C. Kabat, *Hyping Health Risks: Environmental Hazards in Daily Life and the Science of Epidemiology,* 2008.

Wallace Kaufman, *No Turning Back: Dismantling the Fantasies of Environmental Thinking,* 1994.

Aynsley Kellow, *Science and Public Policy: The Virtuous Corruption of Virtual Environmental Science*, 2007.

Jay H. Lehr, ed., *Rational Readings on Environmental Concerns*, 1992.

S. Robert Lichter and Stanley Rothman, *Environmental Cancer – A Political Disease?* 1999.

Christopher Manes, *Green Rage: Radical Environmentalism and the Unmaking of Civilization,* 1990.

A.W. Montford, *The Hockey Stick Illusion: Climategate and the Corruption of Science*, 2010.

Daniel T. Oliver, *Animal Rights: The Inhumane Crusade,* 1999.

James M. Sheehan, *Global Greens: Inside the International Environmental Establishment,* 1998.

Julian Simon, *Hoodwinking the Nation,* 1999.

Rich Trzupek, *Regulators Gone Wild: How the EPA Is Ruining American Industry,* 2011.

Source: Lehr, 2014.

went to great lengths to deceive and defraud the U.S. government over the span of more than a decade," according to Alisha Johnson, press secretary to Gina McCarthy, the EPA administrator at the time (Isikoff, 2013).

John C. Beale, a high-ranking career bureaucrat in the EPA's Office of Air and Radiation and said to be the person most responsible for the EPA's rulings on ozone, particulate matter, and carbon dioxide, was convicted of felony theft of government property in 2014 and sentenced to 32 months in prison for fraud and stealing nearly $900,000 from American taxpayers (*Wall Street Journal,* 2013). Mark Kaminsky, an investigator for the Office of the Inspector General, testified that Beal is a pathological liar who "lied across all aspects of his life" (Gaynor, 2014). During his deposition, Beale said he lied to his friends and colleagues because he felt "an excitement about manipulating people or convincing them of something that's not true" (Hayward, 2014).

Patrick Sullivan, assistant inspector general for investigations at the EPA, told NBC News "he

doubted Beale's fraud could occur at any federal agency other than the EPA. 'There's a certain culture here at the EPA where the mission is the most important thing,' he said. "They don't think like criminal investigators. They tend to be very trusting and accepting'" (Isikoff, 2013). According to NBC News, the scandal was "what some officials describe as one of the most audacious, and creative, federal frauds they have ever encountered."

Much of Beale's work at the EPA was in furtherance of agendas promoted by liberal environmental organizations, the use of collusive lawsuits with a result of sue and settle for new environmental regulations, and promotion of more burdensome air regulations with the objective of imposing maximum harm on industry in general and the coal industry in particular. A minority report issued by the U.S. Senate Committee on Environment and Public Works painted a vivid picture of manipulation and corruption:

> Before his best friend Robert Brenner [Deputy Director of the Office of Policy, Analysis, and Review (OPAR) within the office of Air and Radiation (OAR)] hired him to work at the EPA, Beale had no legislative or environmental policy experience and wandered between jobs at a small-town law firm, a political campaign, and an apple farm. Yet at the time he was recruited to the EPA, Brenner arranged to place him in the highest pay scale for general service employees, a post that typically is earned by those with significant experience.
>
> What most Americans do not know is that Beale and Brenner were not obscure no-name bureaucrats housed in the bowels of the Agency. Through his position as head of the Office of Policy, Analysis, and Review, Brenner built a "fiefdom" that allowed him to insert himself into a number of important policy issues and to influence the direction of the Agency. Beale was one of Brenner's acolytes – who owed his career and hefty salary to his best friend.
>
> During the Clinton Administration, Beale and Brenner were very powerful members of the EPA's senior leadership team within the Office of Air and Radiation, the office responsible for issuing the most expensive and onerous federal regulations. Beale himself was the lead EPA official for one of the most controversial and far reaching regulations ever issued by the Agency, the 1997 National Ambient Air Quality Standards (NAAQS) for Ozone and Particulate Matter (PM). These standards marked a turning point for the EPA air regulations and set the stage for the exponential growth of the Agency's power over the American economy. Delegating the NAAQS to Beale was the result of Brenner's facilitating the confidence of the EPA elites, making Beale the gatekeeper for critical information throughout the process. Beale accomplished this coup based on his charisma and steadfast application of the belief that the ends justify the means (U.S. Senate Committee on Environment and Public Works, 2014, p. i).

According to Reynolds (2018), "Beale had his hand in another fishy tactic utilized by the EPA. The creation of the particulate matter regulations came about as a result of the first instance of "sue and settle," in which friendly bureaucrats negotiate settlements with activist groups. In the case of the particulate matter regulations, the American Lung Association had sued the EPA to expedite the creation of the regulations, and a court order imposed a deadline on the agency. The Obama administration stuffed the EPA with former employees of radical environmental organizations, and then put them in charge of negotiating settlements when those organizations sued. This allowed the EPA to bypass the normal rulemaking process with congressional oversight because they were under court order. Incidentally, Pruitt put an end to this practice in October 2017, another reason he's been targeted for destruction by the Left."

Anyone who claims the EPA's ozone and PM NAAQS are based on the scientific method and "best available science" should read this account carefully and reconsider.

Richard Windsor

While conducting research for a book, Christopher Horner, an attorney and author affiliated with the Competitive Enterprise Institute, found an EPA memo from 2008 describing "alias" email accounts created by former EPA Administrator Carol Browner

(1993–2001). Those accounts created a "dual account structure" used by high-level officials inside the EPA to correspond with one another and with outside environmental groups without fear that the messages would be "leaked" to the public. Many of the accounts were apparently set to "auto-delete" (Horner, 2012a).

More recently, EPA Administrator Lisa Jackson (2009–2013) invented the name "Richard Windsor" for emails sent and received to evade federal transparency laws. The scandal resulted in her abrupt resignation in December, 2013 just days after the Justice Department announced it would begin releasing the secret emails. She was never formally charged with a crime.

Federal law requires all government employees to use only official email accounts. If they use a private account to do official business, they are required to make those accounts available to their employing department or agency. Why would two EPA administrators and their senior staff seek to hide their professional (not personal) emails from the public? The Competitive Enterprise Institute, which filed Freedom of Information Act (FOIA) requests and eventually launched a lawsuit leading to a judge's decision to order the release of Jackson's emails, said in a news release on the date of her resignation announcement, "the emails relate to the war on coal Jackson was orchestrating on behalf of President Obama outside the appropriate democratic process" (Hall, 2012). The news release continued,

> But this scandal cannot end with Jackson's resignation. She appears to have illegally evaded deliberative procedures and transparency requirements set in law – as did the federal appointees and career employees with whom she communicated through her alias email account. She must be held to account, as must those others – both to assure the peoples' business is done in public and to send a signal to other high-level government officials this conduct cannot and will not be tolerated.
>
> Meanwhile, CEI will continue to try to get to the bottom of Jackson's efforts to evade public scrutiny of her actions. We have and will continue to pursue what we have determined to be widespread similar behavior including private email accounts, private computers and privately owned computer servers used to hide discussions that, by law, must be open to scrutiny and be part of the public record. The administration has admitted the agency has destroyed documents in apparent violation of the federal criminal code, and we intend to continue to investigate and expose these attempts to hide the agency's actions.

Regarding Carol Browner, the Clinton-era EPA administrator, Horner wrote: "You remember Ms. Browner? She's the lady who suddenly ordered her computer hard drive be reformatted and backup tapes be erased, just hours after a federal court issued a 'preserve' order that her lawyers at the Clinton Justice Department insisted they hadn't yet told her about? She's the one who said it didn't matter because she didn't use her computer for email anyway?" (Horner, 2012b).

Regrettably, the corruption didn't end with Jackson's resignation. In 2015, EPA Administrator Gina McCarthy (2013–2017) repeatedly refused to turn over to congressional investigators records of the agency's interactions with environmental advocacy groups, leading the chairman of the House Science, Space and Technology Committee to issue a subpoena for the records in March (Carna, 2015a). In October, the EPA again refused to turn over records to congressional investigators, this time concerning its collaboration with environmental groups to alter global temperature records, leading to another subpoena (Warrick, 2015).

McCarthy also was subpoenaed for hiding and deleting text messages just days after being told by a House committee that she may have been violating federal document retention laws (Miller, 2015). Twenty-one members of Congress introduced legislation to impeach her, saying "Administrator McCarthy committed perjury and made several false statements at multiple congressional hearings, and as a result, is guilty of high crimes and misdemeanors – an impeachable offense" (Gosar, 2015).

Human Experiments

> *By conducting human experiments involving exposure to levels of particulate matter and other pollutants it claims to be deadly, the EPA reveals it doesn't believe its own epidemiology-based claims of a deadly threat to public health.*

Another EPA scandal pertains to life-endangering experiments performed on human subjects in violation of international standards and medical ethics (Bell, 2013; Dunn, 2012, 2015; Milloy, 2013, 2016; Milloy and Dunn, 2012, 2016). The EPA has tested a variety of air pollutants – including very high exposures to $PM_{2.5}$ – on more than 6,000 human volunteers. Many of these volunteers were elderly or already health-compromised – the very groups the EPA claims are most susceptible to death from $PM_{2.5}$ exposure. $PM_{2.5}$ exposures in these experiments have been as high as 21 times greater than allowed by the EPA's own air quality rules (Milloy, 2012).

It is illegal, unethical, and immoral to expose experimental subjects to harmful or lethal toxins. *The Reference Manual on Scientific Evidence* (FJC, 2011), published by the United States Federal Judicial Center and cited previously in Section 6.2.2, on page 555 declares that exposing human subjects to toxic substances is "proscribed" by law and cites case law. The Nuremberg Code and the Helsinki Accords on Human Experimentation by the World Medical Association prohibit human experiments that might cause harm to the subjects. The EPA's internal policy guidance on experimental protocols prohibits, under United States law (the "Common Rule"), experiments that expose human subjects to any harm, including exposure to lethal or toxic substances.

The EPA human experiments were conducted from January 2010 to June 2011, according to information obtained by JunkScience.com from a Freedom of Information Act request, and ended three months before then-EPA Administrator Lisa Jackson testified to Congress claiming $PM_{2.5}$ was possibly the most deadly substance known to mankind, killing as many people as die from cancer in the United States every year. If the EPA believed its own rhetoric about the health threats of $PM_{2.5}$, then it also should have believed these experiments could have resulted in serious injury or death, and so were illegal and unethical.

What could have possessed these EPA researchers to conduct these illegal experiments? Robert Devlin, a senior EPA research official who supervised human experiments at the University of North Carolina School of Medicine, said in an affidavit, "Controlled human exposure studies conducted by the EPA scientists and the EPA funded scientists at multiple universities in the United States fill an information gap that cannot be filled by large population studies. … These studies are done under conditions that are controlled to ensure safety, with measurable, reversible physiological responses. They are not meant to cause clinically significant adverse health effects, but rather reversible physiological responses can be indicators of the potential for more serious outcomes (Devlin, 2012).

Devlin either did not believe EPA Administrator Jackson's claims that exposure to even low levels of $PM_{2.5}$ could cause instant death, or he knowingly violated the provisions of the Nuremberg Code, the Helsinki Accords on Human Experimentation, and the U.S. Common Rule. Either Jackson is wrong, or Devlin and scores of other doctors and researchers who participated in these illegal experiments should be in prison.

The EPA refused to respond to FOIA requests filed by medical researchers Steve Milloy and John Dale Dunn, M.D. (note both are contributors to this chapter). When sued, it claimed the EPA-funded researchers were immunized from any requirement to produce their data because the data were the private property of the researchers. Then the EPA's inspector general took up the case in October 2012. Eighteen months later, the inspector general concluded the agency had indeed failed to warn study subjects that it believed the experiments could kill them – but the inspector general inexplicably ignored the issue of whether the experiments were fundamentally illegal and unethical (EPA, 2014).

Embarrassed by negative publicity from the case, the EPA quietly paid the National Research Council of the National Academies of Sciences to produce a report that it expected would exonerate the agency. A committee of mostly academics, many of them recipients of government grants to find evidence favoring the government's hypothesis that man-made chemicals threaten human health, was formed and began meeting on June 1, 2015. There was no public notice of the formation of the committee or its meeting, so the legally required "public" meeting was attended only by the committee members and EPA and NRC staff.

In June 2016, Milloy and Dunn learned of the NRC investigation for the first time from a congressional aide who just happened to see information about it. They learned five meetings had been held, the last one in April 2016, none open to the public. Milloy and Dunn hurriedly provided comments to the committee docket (record) and requested an opportunity to present oral and written information to the committee. They were allowed to participate remotely in one meeting (Milloy *et al.,* 2016).

The NRC released its report in March 2017 (NRC, 2017a). As Milloy and Dunn had feared, it

was a whitewash. From NRC's announcement of the report's release:

> The committee concluded that the societal benefits of CHIE [controlled human inhalation exposure] studies are greater than the risks posed to the participants in the eight studies considered, which are unlikely to be large enough to be of concern. EPA applies a broad set of health-evaluation criteria when selecting participants to determine that there is no reason to believe that their participation in the study will lead to an adverse health response (NRC, 2017b).

The first sentence in the NRC's statement ought to be shocking to all readers. Since at least the end of World War II, the ethics of human experimentation was never about balancing "societal benefits" against individual risks. The consensus of ethicists around the world is that *no* societal benefit can justify human experimentation where serious physical harm is a possibility. Even informed consent is not a permission slip to conduct such experiments. This sentence demonstrates how the NRC failed to properly frame its investigation from the very start.

The second sentence from the NRC's summary directly contradicts the EPA's claims about the health effects of exposure to low concentrations of $PM_{2.5}$. Whereas the EPA repeatedly claims there is "no safe level of exposure" to $PM_{2.5}$, that even tiny exposures raise the risk of adverse health effects up to and including sudden death, the NRC says experiments exposing volunteers to such levels do not "lead to an adverse health response." To avoid having scores of medical doctors and researchers working under its management go to jail for violating medical ethics, the EPA apparently admitted to the NRC that PM is not the deadly pollutant it has been saying it is to the public, Congress, and the public health research community.

The EPA's response to the concerns expressed by Milloy and Dunn illustrates the same aversion to transparency, defiance of the law, and opposition to transparency that were demonstrated in the previous examples in this section. EPA's motive for conducting the experiments, from Devlin's testimony and the circumstances, seems clear. The EPA knew its claims about the health effects of $PM_{2.5}$ and other pollutants are vulnerable to challenge because the underlying studies – all dubious epidemiological statistical correlation studies – do not actually show that particulate matter kills *anyone.* Neither do animal toxicology studies, no matter how much PM the laboratory animals inhale. So the EPA decided to break the rules – of the international community as well as of the agency itself – and bolster its claims about particulate matter by conducting human experiments.

The Current Administration

> *While the new administration has pledged to improve matters, some current regulations and ambient air standards are based on flawed data.*

On February 17, 2017, Scott Pruitt became EPA Administrator, although he resigned effective July 9, 2018. (At the time of this writing, a permanent replacement has not been named.) During his years as attorney general for the State of Arkansas, Pruitt grew familiar with the EPA's misuse of science, lack of transparency, and outright corruption of the regulatory process. With other state attorneys general, he sued the EPA 14 times for exceeding its constitutional authority by attempting to federalize state environment and energy regulation. As administrator, Pruitt proposed a 2018 budget for EPA that was $2.6 billion below the agency's 2017 funding level. The opening pages of the proposed budget state:

> This resource level and the agency FTE [full-time equivalent] level of 11,611 supports the agency's return to a focus on core statutory work and recognizes the appropriate federal role in environmental protection. The budget addresses our highest environmental priorities and refocuses efforts toward streamlining and reducing burden. Responsibility for funding local environmental efforts and programs is returned to state and local entities, while federal funding supports priority national work (EPA, 2017a, pp. 1–2).

Under Pruitt's leadership, the EPA began to unravel the "war on coal" waged by his predecessors. Specific regulatory changes are discussed in some detail in Chapter 8, as part of the cost-benefit analysis of regulations, and so won't be raised here. However, in light of the abuses of transparency and process documented above, three Pruitt initiatives should be mentioned here. First, on October 16, 2017, Pruitt

issued an agency-wide directive designed to end the "sue and settle" practice that was used to set the PM and ozone NAAQSs. In the announcement of the directive, Pruitt is quoted as saying,

> The days of regulation through litigation are over. We will no longer go behind closed doors and use consent decrees and settlement agreements to resolve lawsuits filed against the Agency by special interest groups where doing so would circumvent the regulatory process set forth by Congress. Additionally, gone are the days of routinely paying tens of thousands of dollars in attorney's fees to these groups with which we swiftly settle (EPA, 2017b).

Also in October 2017, Pruitt announced the EPA would no longer appoint to its advisory boards individuals who receive funding from the agency. According to the directive, "members [of advisory committees] shall be independent from EPA, which shall include a requirement that no member of any of EPA's federal advisory committees be currently in receipt of EPA grants, either as principal investigator or co-investigator, or in a position that otherwise would reap substantial direct benefit from an EPA grant" (EPA, 2017c). "It is very, very important to ensure independence, to ensure that we're getting advice and counsel independent of the EPA," Pruitt told *The New York Times*. He pointed out that members of just three boards – Scientific Advisory Board, Clean Air Science Advisory Committee, and Board of Scientific Counselors – had collectively accepted $77 million in EPA grants over the previous three years. "He noted that researchers will have the option of ending their grant or continuing to advise EPA, 'but they can't do both'" (Dennis and Eilperin, 2017).

On April 30, 2018, the EPA issued a notice of a proposed rule for "strengthening transparency in regulatory science." That notice said, "Today, EPA is proposing to establish a clear policy for the transparency of the scientific information used for significant regulations: Specifically, the dose response data and models that underlie what we are calling 'pivotal regulatory science'" (EPA, 2018). The proposed rule calls for ending the use of "secret science" – research utilizing databases that are not made available to independent scholars to replicate findings – and challenges the EPA's most controversial assumption, the linear no-threshold dose-response. The rule also calls for more complete disclosure of confounding factors and model uncertainty.

These three initiatives are bold departures from "business as usual" at the EPA, and if successful they would address the most important reasons the agency has lost nearly all its credibility in the air quality debate (Johnston, 2018). It will take years for these reforms to change the agency's culture and lead to corrections of its faulty scientific and public health claims. Until that time, no one should rely on any public health research conducted by the EPA in justification of its regulations.

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6.3. Observational Studies

When EPA-sponsored toxicological research fails to prove a particular exposure is harmful, the agency turns to observational studies in which the researcher is not able to control how subjects are assigned to the "treated" group or the "control" groups or the treatments each group receives. Such studies are frequently used in the field of epidemiology, a branch of medicine that studies the incidence and distribution of diseases. Section 6.3.1 describes EPA's history of relying on such studies and their shortcomings, and Section 6.3.2 presents a case studying featuring EPA's reliance on flawed studies to justify regulation of fine particulate matter.

6.3.1 Reliance on Observational Studies

> *Observational studies are easily manipulated, cannot prove causation, and often do not support a hypothesis of toxicity with the small associations found in uncontrolled observational studies.*

Just as the academic literature on climate science is clogged with multi-author reports based on unreliable computer models generally aimed at supporting the federal government's "war on fossil fuels,' so too is the literature on air quality is clogged by government-funded observational studies, sometimes called epidemiological studies or simply epidemiology. Such studies generally compare the observed health outcomes of subjects thought to have been exposed to a relatively high level of a chemical compound in an uncontrolled setting, typically determined by air quality monitors located in or near the area where the subjects live or work, to a control group that is either larger (e.g., all residents of the country) or whose members live or work in an area with lower levels of exposure. Observational studies differ from experiments, in which subjects are randomly assigned to a treated group or a control group. Wolff and Heuss (2012) reported EPA's increased reliance on such studies beginning in 1996:

> In considering the establishment of NAAQS, EPA relies on three types of health effect studies: controlled human exposures ("clinical"), animal toxicology ("toxicology") and epidemiology studies. In all NAAQS reviews prior to the 1996 PM review, EPA relied most heavily on controlled human exposures, which establish health effect endpoints as a function of exposure and demonstrate causality, and the toxicology studies which provide insights as to the mode of the damage caused by an exposure. Epidemiology studies were used if they supported the findings in the other two types of studies because epidemiology studies can only identify statistical associations between air pollutant concentrations and health endpoint incidence and cannot be used to demonstrate causality (cause-effect relationships).

For the PM NAAQS review that ended in 1996, Wolff and Heuss (2012) wrote, the EPA for the first time subordinated human exposure and toxicological studies to epidemiological studies "because they [the toxicological studies] showed no evidence of effects at concentrations near the level of the existing NAAQS." To make a case for a lower NAAQS for PM_{10} and a new NAAQS for $PM_{2.5}$, the EPA had to turn to epidemiology studies that found "very weak statistical associations" between exposure and mortality. "EPA promulgated new annual and 24-hour $PM_{2.5}$ NAAQS based on the epidemiology findings" (*Ibid.*).

EPA's reliance on epidemiology represented a major step away from sound science. The Federal Judicial Center, whose authors were introduced and quoted earlier in this chapter, stress, "epidemiology cannot prove causation; rather, causation is a judgment for epidemiologists and others interpreting the epidemiological data. Moreover, scientific determinations of causation are inherently tentative. The scientific enterprise must always remain open to reassessing the validity of past judgments as new evidence develops" (FJC, 2011, p. 598).

Foster *et al.* (1993), commenting on more than a dozen cases of what they call "phantom risks" ("cause-and-effect relationships whose very existence

is unproven and perhaps unprovable" (p. 1)), concluded, "The epidemiological studies are frequently inconsistent; the animal studies often show clear toxic effects, but at levels that vastly exceed any reasonable human exposure. Although the issues vary, similar themes constantly reappear" (p. 13) and "despite hysterical claims that were widely publicized during the 1970s, typical environmental exposures to most chemicals are too low to be a major (or even detectable) source of illness" (p. 14).

A major shortcoming of observational studies is their failure to replicate results, a violation of BHC #2 requiring consistency of the observed association. Young and Karr (2011) wrote,

> It may not be appreciated how often observational claims fail to replicate. In a small sample in 2005 [citing Ioannidis, 2005], of 49 claims coming from highly cited studies, 14 either failed to replicate entirely or the magnitude of the claimed effect was greatly reduced (a regression to the mean). Six of these 49 studies were observational studies, and in these six, in effect, randomly chosen observational studies, five failed to replicate. This last is an 83% failure rate. In an ideal world in which well-studied questions are addressed and statistical issues are accounted for properly, few statistically significant claims are false positives. Reality for observational studies is quite different (p. 117).

Young and Karr continued,

> We ourselves carried out an informal but comprehensive accounting of 12 randomised clinical trials that tested observational claims. … The 12 clinical trials tested 52 observational claims. They all confirmed no claims in the direction of the observational claims. We repeat that figure: 0 out of 52. To put it another way, 100% of the observational claims failed to replicate. In fact, five claims (9.6%) are statistically significant in the clinical trials *in the opposite direction* to the observational claim. To us, a false discovery rate of over 80% is potent evidence that the observational study process is not in control. The problem, which has been recognised at least since 1988, is systemic (*Ibid.*).

Alvan R. Feinstein, a Yale epidemiologist, produced a series of devastating critiques of research relied on by the EPA and other regulatory agencies (Feinstein, 1988, 1991; Feinstein and Massa, 1997). In a 1988 article published in *Science* he observed:

> Many substances used in daily life, such as coffee, alcohol, and pharmaceutical treatment for hypertension, have been accused of "menace" in causing cancer or other major diseases. Although some of the accusations have subsequently been refuted or withdrawn, they have usually been based on statistical associations in epidemiological studies that could not be done with the customary experimental methods of science. With these epidemiological methods, however, the fundamental scientific standards used to specify hypotheses and groups, get high-quality data, analyze attributable actions, and avoid detection bias may also be omitted. Despite peer-review approval, the current methods need substantial improvement to produce trustworthy scientific evidence (Feinstein, 1988, abstract).

James Enstrom, an epidemiologist long associated with the Jonsson Comprehensive Cancer Center at the University of California, Los Angeles and now head of the Scientific Integrity Institute, observed the following flaws in epidemiological studies relied on by the EPA and air quality regulatory agencies in California to estimate the health effects of particulate matter (PM):

- mobile populations
- unreliable, non-continuous, and fixed monitor information
- no monitor information on some pollutants all the time (2.5 micron particulate matter, for example) or part of the time (10 micron and others)
- an attempt to assess long-term chronic health effects of air quality by death studies, an acute phenomenon
- death certificates and raw death data used without autopsies

- inside air quality ignored for populations living indoors, particularly during old age, advanced medical illness, and terminal illness

- no biological plausibility because the deaths are in the setting of non-toxic levels of air pollution (Enstrom, 2005)

Each of these flaws can lead to violations of BHC standards and make such studies unreliable guides for public policy. Observational studies are easily manipulated, cannot prove causation, and often do not support a hypothesis of toxicity with the small associations in uncontrolled observational studies. And yet, an important part of the case against fossil fuels – that they produce emissions that threaten human health – relies entirely on such research. The flawed results are often fed, without criticism or skepticism, into the computer models used to predict future health effects and the "social cost of carbon" (see, e.g., Bosello *et al.*, 2006). This is a critical mistake that careful researchers should avoid.

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6.3.2 The Particulate Matter Scare

> *Real-world data and common sense contradict claims that ambient levels of particulate matter kill hundreds of thousands of Americans and millions of people around the world annually.*

The studies relied on by the EPA to support its "war on coal" frequently fail to show relative risks (RR) that would suggest a causal relationship between the chemical compounds released during the combustion of fossil fuels and adverse human health effects. Particularly egregious is the agency's claim, against real-world data and common sense, that small particles in the air kill hundreds of thousands of Americans annually.

EPA's Research

The EPA first asserted authority to regulate fine particulate matter ($PM_{2.5}$) as a pollutant in 1997. The U.S. Senate Committee on the Environment and Public Works reported how John Beale, now a convicted felon, played a major role in the decision:

> In the case of the 1997 NAAQS, the Playbook started with a sue-and-settle agreement with the American Lung Association, which established a compressed timeline to draft and issue PM standards. This timeline was further compressed when EPA made the unprecedented decision to simultaneously issue new standards for both PM and Ozone. Issuing these standards in tandem and under the pressure of the sue-and-settle deadline, Beale had the mechanism he needed to ignore opposition to the

> standards – EPA simply did not have the time to consider dissenting opinions.
>
> The techniques of the Playbook were on full display in the "Beale Memo," a confidential document that was leaked to Congress during the controversy, which revealed how he pressured the Office of Information and Regulatory Affairs to back off its criticism of the NAAQS and forced them to alter their response to Congress in 1997. EPA also brushed aside objections raised by Congress, the Office of Management and Budget, the Department of Energy, the White House Council of Economic Advisors, the White House Office of Science and Technology Policy, the National Academy of Sciences, and EPA's own scientific advisers – the Clean Air Science Advisory Committee.
>
> These circumstances were compounded by EPA's "policy call" to regulate $PM_{2.5}$ for the first time in 1997. $PM_{2.5}$ are ubiquitous tiny particles, the reduction of which the EPA used to support both the PM and Ozone NAAQS. In doing so, the Playbook also addressed Beale's approach to EPA's economic analysis: overstate the benefits and underrepresent the costs of federal regulations. This technique has been applied over the years and burdens the American people today, as up to 80% of the benefits associated with all federal regulations are attributed to supposed $PM_{2.5}$ reductions (U.S. Senate Committee on the Environment and Public Works, 2014, p. ii).

Fourteen years later, in 2011, EPA Administrator Lisa Jackson claimed in testimony before Congress, "If we could reduce particulate matter to levels that are healthy we would have an identical impact to finding a cure for cancer" (quoted in Harris and Broun, 2011, p. 2; see also Congressional Record, 2011). Cancer kills approximately 570,000 people in the United States annually, making this an astounding and incredible claim.

In 2014, then EPA Administrator Gina McCarthy told reporters, "John Beale walked on water at EPA." The U.S. Senate Committee on Environment and Public Works commented on that remark: "This unusual culture of idolatry has led EPA officials to blind themselves to Beale's wrongdoing and caused them to neglect their duty to act as public servants. As such, to this day EPA continues to protect Beale's work product and the secret science behind the Agency's NAAQS and PM claims" (U.S. Senate Committee on Environment and Public Works, 2014, p. iii).

As reported in the introduction to this chapter, in 2010, the EPA claimed PM caused approximately 360,000 and as many as 500,000 premature deaths in the United States in 2005, citing Laden *et al.* (2006) (EPA, 2010, p. G7). Figure 6.3.2.1 reproduces Table G-1 from the EPA report supporting the agency's claim. In 2012, approximately the same team of authors who produced the estimates relied on by the EPA for the estimates in Figure 6.3.2.1 updated their analysis to account for changes in the cohort population and air quality up to and including 2009. They reported, "Each 10-μg/m3 increase in $PM_{2.5}$ was associated with a 14% increased risk of all-cause death [95% confidence interval (CI): 7%, 22%], a 26% increase in cardiovascular death (95% CI: 14%, 40%), and a 37% increase in lung-cancer death (95% CI: 7%, 75%)" (Lepeule *et al.,* 2012). They went on to report, "Given that there were 2,423,712 deaths in the United States in 2007 (Xu *et al.* 2010) and that the average $PM_{2.5}$ level was 11.9 $\mu g/m^3$ (U.S. EPA 2011), our estimated association between $PM_{2.5}$ and all-cause mortality implies that a decrease of 1 $\mu g/m^3$ in population-average $PM_{2.5}$ would result in approximately 34,000 fewer deaths per year" (*Ibid.*).

The EPA's claim that $PM_{2.5}$ causes long-term death is grounded in two long-term epidemiological studies: the Harvard Six Cities study (Dockery *et al.*, 1993; Pope *et al.,* 2002) and the American Cancer Society (ACS) study (Pope *et al.,* 1995, 2002, 2009). The original Harvard Six Cities study tracked the health of 8,111 subjects in six cities between 1974 and 1991 and found an RR of 1.26 for those living in cities with the highest reported levels of air pollution compared to those living in the city with the lowest reported level of air pollution. The authors concluded, "fine particulate air pollution … contributes to excess mortality in certain U.S. cities."

Besides the obvious problem of a small sample size and failing to consider many possible confounding factors, the study found subjects with more than a high school education showed no association of PM exposure with mortality and even found for that group a slight *decrease* in mortality rates due to respiratory disease (Arnett, 2006, p. 5). This finding violates BHC #2 requiring consistency of the observed association and #3 requiring specificity of the association.

Figure 6.3.2.1
Estimated $PM_{2.5}$-related premature mortality associated with incremental air quality differences between 2005 ambient mean $PM_{2.5}$ levels and lowest measured level from the epidemiology studies or policy relevant background

Air Quality Level	Estimates Based on Krewski et al. (2009)		Estimates Based on Laden et al. (2006) *(90th percentile confidence interval)*
	'79-'83 estimate (90th percentile confidence interval)	*'99-'00 estimate (90th percentile confidence interval)*	
10 µg/m³ (LML for Laden et al., 2006)	26,000 (16,000—36,000)	33,000 (22,000—44,000)	**88,000 (49,000—130,000)**
5.8 µg/m³ (LML for Krewski et al., 2009)	**63,000 (39,000—87,000)**	**80,000 (54,000—110,000)**	210,000 (120,000—300,000)
Policy-Relevant Background	110,000 (68,000—150,000)	140,000 (94,000—180,000)	360,000 (200,000—500,000)
Bold indicates that the minimum air quality level used to calculate this estimate corresponds to the lowest measured level identified in the epidemiological study			

Source: EPA, 2010, Table G-1.

The original ACS study compared air quality levels with mortality in more than 500,000 people from 151 U.S. metropolitan areas between 1982 and 1989. It found RRs of 1.17 for PM and 1.15 for sulfate, once again comparing the most polluted city with the least polluted city. Even this weak association – far below the RR of 2 or even 1.5 required by the Federal Judicial Center – is suspect. According to Arnett (2006), "health information was obtained only once, at entry into the study in 1982 and it considered only a few of the 300 known risk factors that have been associated with cardiovascular disease. None of the data obtained was verified by review of medical records or by other means" (p. 6).

Not surprisingly, given the small associations they found and lack of supporting science, the EPA's own scientific advisory committee refused to approve a PM standard. In 1995, in response to a request from the agency, researchers for the National Institute of Statistical Science investigated the possible relationship between airborne particulate matter and mortality in Cook County, Illinois, and Salt Lake County, Utah. "We found no evidence that particulate matter < or = 10 microns (PM_{10}) contributes to excess mortality in Salt Lake County, Utah. In Cook County, Illinois, we found evidence of a positive PM_{10} effect in spring and autumn, but not in winter and summer," they reported. "We conclude that the reported effects of particulates on mortality are unconfirmed" (Styer *et al.,* 1995).

In its 2013 estimate of the "social cost of carbon," which has since been rescinded, the EPA claimed public health is endangered by chemical compounds released during the combustion of fossil fuel, principally particulate matter, ozone, nitrogen dioxide (NO_2), sulfur dioxide (SO_2), mercury, and hydrogen chloride (HCl) (EPA, 2013). Other harms it cited included visibility impairment (haze), corrosion of building materials, negative effects on vegetation due to ozone, acid rain, nitrogen deposition, and negative effects on ecosystems from methylmercury

In 2014 and 2015, the EPA relied on the same sources (Laden *et al.,* 2006 and Lepeule *et al.,* 2012) for its regulatory impact statement regarding the proposed Clean Power Plan (EPA, 2014, 2015), which has since been rescinded (EPA, 2018a). The EPA claimed benefits of the new regulations would be worth an estimated $55 billion to $93 billion in

2030. Virtually all the benefits would come from reducing particulate matter emissions and exposure to ozone, which the EPA said would avoid 2,700 to 6,600 premature deaths and 140,000 to 150,000 asthma attacks in children annually (EPA, 2015). From 2009 to 2011, EPA claimed reducing PM emissions amounted to 99% or more of the benefits of eight of twelve new rules (Smith, 2011).

Despite much research, there is no generally accepted medical or biological explanation for how $PM_{2.5}$ at concentrations close to U.S. ambient levels could cause disease or death. No laboratory animal has ever died from $PM_{2.5}$ in an experimental setting, even though animals have been exposed to levels of $PM_{2.5}$ as much as 100+ times greater than human exposures to $PM_{2.5}$ in outdoor air (Arnett, 2006). The EPA assumes without providing clinical evidence that exposure to ambient levels of $PM_{2.5}$ causes disease and mortality. This violates BHC #6, requiring biological plausibility, and #8, requiring experimental evidence.

As reported earlier in Section 6.2, the EPA *has* tested a variety of air pollutants – including very high exposures to $PM_{2.5}$ – on more than 6,000 human volunteers. Many of these volunteers were elderly or already health-compromised – the very groups the agency claims are most susceptible to death from $PM_{2.5}$ exposure. The agency has admitted there have been no deaths or any dangerous adverse events clearly caused by these $PM_{2.5}$ exposures, which were as high as 21 times greater than the exposures allowed by the agency's own air quality rules (Milloy, 2012).

Recently EPA reduced the size of claims made in the past with regards to the small particle and ozone co-benefits of the Clean Power Plan (Saiyid, 2018), but a more far-reaching review of the EPA's methodology and integrity is needed. Such a review would likely result in dramatic changes in NAAQS and other EPA policies. According to the EPA, average exposure in the United States to both PM_{10} and $PM_{2.5}$ has fallen steeply since the 1990s and is now below the agency's NAAQS (EPA, 2018b). Figure 6.3.2.2 reproduces the EPA's graphs for $PM_{2.5}$ and PM_{10} concentrations for the period 2000–2016.

Independent Research

The Health Effects Institute (HEI), a nonprofit research organization jointly funded by the EPA and the automobile industry, has conducted several studies on the health effects of air quality, reanalyzing data from the Harvard Six Cities and ACS studies as well as a newer database called the National Morbidity, Mortality and Air Pollution Study (NMMAPS) (Krewski *et al.,* 2000; Krewski *et al.,* 2005; HEI, 2008). While generally confirming the findings of the earlier reports, they also reported considerable heterogeneity in the data, indicating exposure to identical levels of particulate matter was correlated with different health outcomes in different parts of the country, a violation of BHC #2, which requires consistency of the observed association across different places. In 2008, HEI reported, "We have re-done our analyses with more stringent convergence criteria for the GAM [generalized additive models] estimation procedure and found that estimates for individual cities changed by small amounts and that the estimate of the average particulate pollution effect across the 90 largest U.S. cities changed from a 0.41% increase to a 0.27% increase in daily mortality per 10 micrograms per cubic meter of PM10," a significant reduction.

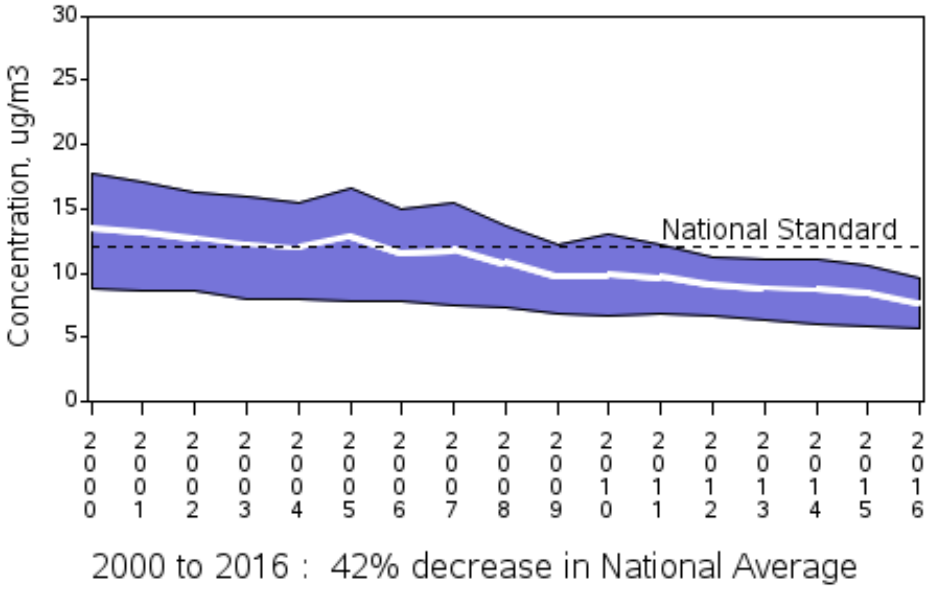
Enstrom (2005) surveyed observational studies on the health effects of PM in the United States up to that year. His table summarizing the findings appears as Figure 6.3.2.3.

None of the studies in Enstrom's table found an RR for $PM_{2.5}$ greater than 1.15 (at the 95% confidence level), far below the Federal Judicial Center requirement of an RR of 2 or more to pass the legal requirement for evidence showing exposure to a chemical compound is "more likely than not" to cause an adverse health effect (FJC, 2011). Recall that an RR = 1 means no association at all, and a negative RR means a possible *positive* effect on health outcomes. In the same article, Enstrom presented the results of his original study of the health effects of $PM_{2.5}$ in California. He described his methodology as follows:

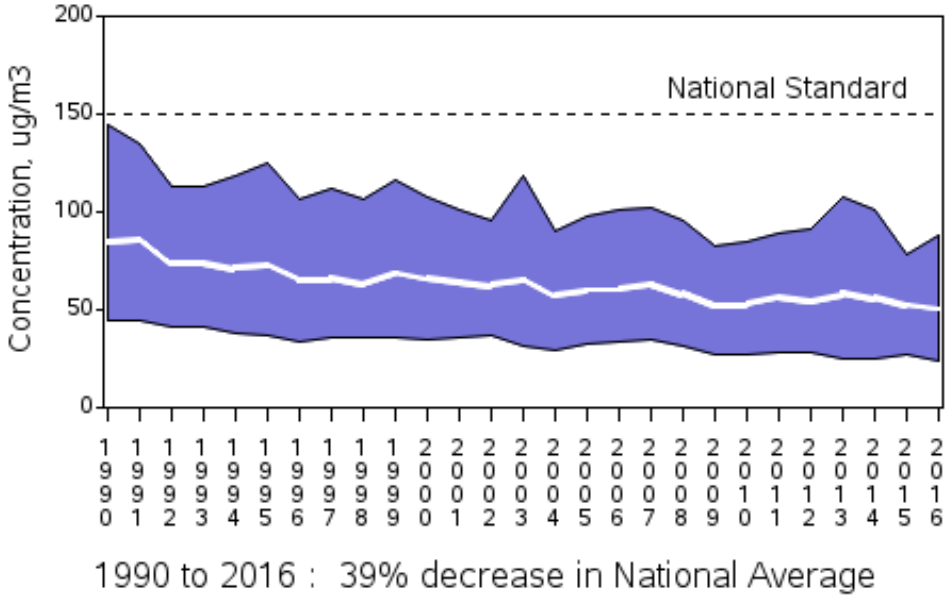
> [T]he long-term relation between fine particulate air pollution and total mortality was examined in a cohort of 49,975 elderly Californians, with a mean age of 65 [years] as of 1973. These subjects, who resided in 25 California counties, were enrolled in 1959, recontacted in 1972, and followed from 1973 through 2002; 39,846 deaths were identified. Proportional hazards regression models were used to determine their relative risk of death (RR) and 95% confidence interval (CI) during 1973–2002 by county of residence. The models adjusted for age, sex, cigarette smoking, race, education, marital status,

Figure 6.3.2.2
Declining aerial concentrations of $PM_{2.5}$ and PM_{10} in the United States, 2000–2016

A. PM2.5 seasonally weighted average annual concentration in the United States, 2000–2016



B. PM_{10} seasonally weighted average annual concentration in the United States, 1990–2016.



Source: EPA, 2018b.

Figure 6.3.2.3
Relative risk and 95% confidence interval (CI) for long-term all-cause mortality per 10-ug/m^3 increase in $PM_{2.5}$ for U.S. cohort studies based on $PM_{2.5}$

TABLE 10
Relative risk (RR) and 95% confidence interval (CI) for long-term all-cause mortality per 10-μg/m^3 increase in $PM_{2.5}$ for U.S. cohort studies based on $PM_{2.5}$ data, circa 1980

	$PM_{2.5}$		Study characteristics					
Study (author, year)	Data period	Mean (range) (μg/m^3)	Cohort geographic definition	Follow-up period	Mean entry age for period	Number entered in cohort	Deaths in follow-up period	RR (95% CI)
Males								
Dockery et al., 1993	1979–1985	19 (11–30)	6 U.S. cities	1975–1989	~50	3671[a]	830[a]	1.15 (1.02–1.30)[b]
Pope et al., 1995	1979–1981	18 (9–34)	50 U.S. SMSAs	1982–1989	57	130,310[a]	~12,400[a]	1.07 (1.03–1.11)[b]
McDonnell et al., 2000	1973–1977	32 (17–45)	9 CA airsheds	1976–1992	58	≤1347	≤375	1.09 (0.98–1.21)[b]
Lipfert et al., 2000	1979–1981	24 (6–42)	42 U.S. counties	1975–1981	51	26,067	~4600[c]	0.95 (0.89–1.01)[c]
	1982–1984	22 (8–41)		1982–1988	57	~21,467	~6100[c]	0.94 (0.90–0.98)[c]
	1982–1984	22 (8–41)		1989–1996	63	~15,367	~5765[c]	0.89 (0.85–0.95)[c]
Pope et al., 2002	1979–1983	21 (10–30)	61 U.S. SMSAs	1982–1998	57	~159,000[a]	~36,000[a]	1.05 (1.01–1.10)
Enstrom, 2005	1979–1983	24 (11–42)	11 CA counties	1973–1982	66	15,573	4701	1.03 (0.99–1.07)
	1979–1983	24 (11–42)		1983–2002	74	10,872	8831	0.97 (0.95–1.00)
Females								
Dockery et al., 1993	1979–1985	19 (11–30)	6 U.S. cities	1975–1989	~50	4440[a]	599[a]	1.12 (0.96–1.30)[b]
Pope et al., 1995	1979–1981	18 (9–34)	50 U.S. SMSAs	1982–1989	57	164,913[a]	~8365[a]	1.06 (1.01–1.12)[b]
McDonnell et al., 2000	1973–1977	32 (17–45)	9 CA airsheds	1976–1992	58	≤2422	≤568	~1.00 (assumed)
Pope et al., 2002	1979–1983	21 (10–30)	61 U.S. SMSAs	1982–1998	57	~200,000[a]	~24,000[a]	1.02 (0.98–1.06)
Enstrom, 2005	1979–1983	24 (11–42)	11 CA counties	1973–1982	65	20,210	4094	1.05 (1.01–1.10)
	1979–1983	24 (11–42)		1983–2002	73	16,116	10,815	1.02 (0.99–1.04)
Both Sexes								
Dockery et al., 1993	1979–1985	19 (11–30)	6 U.S. cities	1975–1989	~50	8111	1430	1.13 (1.04–1.23)[b]
Pope et al., 1995	1979–1981	18 (9–34)	50 U.S. SMSAs	1982–1989	57	295,223	20,765	1.07 (1.04–1.10)[b]
Pope et al., 2002	1979–1983	21 (10–30)	61 U.S. SMSAs	1982–1998	57	~359,000	~60,000	1.04 (1.01–1.08)
Enstrom, 2005	1979–1983	24 (11–42)	11 CA counties	1973–1982	65	35,783	8795	1.04 (1.01–1.07)
	1979–1983	24 (11–42)		1983–2002	73	26,988	19,646	1.00 (0.98–1.02)

[a] Obtained from supplementary data (Krewski et al., 2000).
[b] Recalculated from published data (US EPA, 2004).
[c] Obtained from the author.

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Source: Enstrom, 2005.

> body mass index, occupational exposure, exercise, and a dietary factor. For the 35,789 subjects residing in 11 of these counties, county-wide exposure to fine particles was estimated from outdoor ambient concentrations measured during 1979–1983 and RRs were calculated as a function of these $PM_{2.5}$ levels (mean of 23.4 $\mu g/m^3$) (abstract).

Enstrom (2005) described his findings as follows:

> For the initial period, 1973–1982, a small positive risk was found: RR was 1.04 (1.01–1.07) for a 10-$\mu g/m^3$ increase in $PM_{2.5}$. For the subsequent period, 1983–2002, this risk was no longer present: RR was 1.00 (0.98–1.02). For the entire follow-up period, RR was 1.01 (0.99–1.03). The RRs varied somewhat among major subgroups defined by sex, age, education level, smoking status, and health status. None of the subgroups that had significantly elevated RRs during 1973–1982 had significantly elevated RRs during 1983–2002. The RRs showed no substantial variation by county of residence during any of the three follow-up periods. Subjects in the two counties with the highest $PM_{2.5}$ levels (mean of 36.1 $\mu g/m^3$) had no greater risk of death than those in the two counties with the lowest $PM_{2.5}$ levels (mean of 13.1 $\mu g/m^3$). These epidemiological results do not support a current relationship between fine particulate pollution and total mortality in elderly Californians, but they do not rule out a small effect, particularly before 1983.

In later writing on this study, Enstrom (2006) said, "The methodology used in my study is completely consistent with the methodology used in the 2002 Pope study. For instance, my study controlled for smoking at entry and presented results for never smokers. Furthermore, fully adjusted relative risks hardly differed from age-adjusted relative risks. My study used the same 1979–1983 $PM_{2.5}$ data that was used in the Pope studies." Enstrom also noted his findings were consistent with those of Krewski *et al.* (2005) who found "no excess mortality risk in California due to $PM_{2.5}$ among the ACS CPS II cohort during 1982–1989."

Moolgavkar (2005) wrote a lengthy review and criticism of the EPA's reliance on epidemiology in *Regulatory Toxicology and Pharmacology*. He wrote, "the results of observational epidemiology studies can be seriously biased, particularly when estimated risks are small, as is the case with studies of air pollution. The Agency [EPA] has largely ignored these issues." He continued, "I conclude that a particle mass standard is not defensible on the basis of a causal association between ambient particle mass and adverse effects on human health."

Smith *et al*. (2009) conducted a reanalysis of data from the NMMAPS to test intercity variability and sensitivity of the ozone-mortality associations to modeling assumptions and choice of daily ozone metric, reasoning that such analysis could reveal confounders and "effect modifiers." They report finding "substantial sensitivity. We examined ozone-mortality associations in different concentration ranges, finding a larger incremental effect in higher ranges, but also larger uncertainty. Alternative ozone exposure metrics defined by maximum 8-hour averages. Smith *et al.* concluded, "Our view is that ozone-mortality associations, based on time-series epidemiological analyses of daily data from multiple cities, reveal still-unexplained inconsistencies and show sensitivity to modeling choices and data selection that contribute to serious uncertainties when epidemiological results are used to discern the nature and magnitude of possible ozone-mortality relationships or are applied to risk assessment" (*Ibid.*).

Enstrom returned to the issue with a paper presented in 2012 at a meeting of the American Statistical Association (Enstrom, 2012). Part of that presentation included a new table summarizing more recent California-specific studies of $PM_{2.5}$ and total mortality in California. That table appears below in Figure 6.3.2.4.

While one study in Enstrom's table shows an RR of 1.84 it is clearly an outlier: None of the other studies shows an RR greater than 1.11 and several show RRs *less* than 1.0, suggesting a *positive* health effect from PM. Recent research plainly shows no support for claims by the EPA and other air quality regulators that PM poses a threat to human health. Commenting on his findings, Enstrom wrote, "There is now overwhelming epidemiological evidence that particulate matter (PM), both fine particulate matter ($PM_{2.5}$) and coarse particulate matter (PM_{10}), is not related to total mortality in California" (p. 2324).

Krstic (2013) conducted a reanalysis on the dataset used by Pope *et al.* (2009) of 51 metropolitan regions. He found "a visual analysis of Figure 4 presented on page 382 of their article indicates that data-point number 46 (Topeka, Kansas) is a

Figure 6.3.2.4
Epidemiological cohort studies of $PM_{2.5}$ and total mortality in California

Relative risk of death from all causes (RR and 95% CI) associated with increase of 10 µg/m³ in PM2.5

Krewski 2000 & 2010 (N=40,408 [18,000 M + 22,408 F]; 4 MSAs; 1979-1983 PM2.5; 44 covariates)	CA CPS II Cohort	RR = 0.872 (0.805-0.944)	1982-1989
McDonnell 2000 (N~3,800 [1,347 M + 2,422 F]; SC&SD&SF AB; M RR=1.09(0.98-1.21) & F RR~0.98(0.92-1.03))	CA AHSMOG Cohort	RR ~ 1.00 (0.95 – 1.05)	1977-1992
Jerrett 2005 (N=22,905; 267 zip code areas; 1999-2000 PM2.5; 44 cov + max confounders)	CPS II Cohort in Los Angeles Basin	RR = 1.11 (0.99 - 1.25)	1982-2000
Enstrom 2005 (N=35,783 [15,573 M + 20,210 F]; 11 counties; 1979-1983 PM2.5; 25 county internal comparison)	CA CPS I Cohort	RR = 1.039 (1.010-1.069) RR = 0.997 (0.978-1.016)	1973-1982 1983-2002
Enstrom 2006 (N=35,783 [15,573 M + 20,210 F]; 11 counties; 1979-1983 & 1999-2001 PM2.5)	CA CPS I Cohort	RR = 1.061 (1.017-1.106) RR = 0.995 (0.968-1.024)	1973-1982 1983-2002
Zeger 2008 (3.1 M [1.5 M M + 1.6 M F]; Medicare enrollees in CA+OR+WA (CA=73%); 2000-2005 PM2.5)	MCAPS Cohort "West"	RR = 0.989 (0.970-1.008)	2000-2005
Jerrett 2010 (N=77,767 [34,367 M + 43,400 F]; 54 counties; 2000 PM2.5; KRG ZIP; 20 ind cov+7 eco var; Slide 12)	CA CPS II Cohort	RR ~ 0.994 (0.965-1.025)	1982-2000
Krewski 2010	CA CPS II Cohort		
(N=40,408; 4 MSAs; 1979-1983 PM2.5; 44 cov)		RR = 0.960 (0.920-1.002)	1982-2000
(N=50,930; 7 MSAs; 1999-2000 PM2.5; 44 cov)		RR = 0.968 (0.916-1.022)	1982-2000
Jerrett 2011 (N=73,609 [32,509 M + 41,100 F]; 54 counties; 2000 PM2.5; KRG ZIP Model; 20 ind cov+7 eco var; Table 28)	CA CPS II Cohort	RR = 0.994 (0.965-1.024)	1982-2000
Jerrett 2011 (N=73,609 [32,509 M + 41,100 F]; 54 counties; 2000 PM2.5; Nine Model Ave; 20 ic+7 ev; Fig 22 & Tab 27-32)	CA CPS II Cohort	RR = 1.002 (0.992-1.012)	1982-2000
Lipsett 2011 (N=73,489 [73,489 F]; 2000-2005 PM2.5)	CA Teachers Cohort	RR = 1.01 (0.95 – 1.09)	2000-2005
Ostro 2011 (N=43,220 [43,220 F]; 2002-2007 PM2.5)	CA Teachers Cohort	RR = 1.06 (0.96 – 1.16)	2002-2007
replaced Ostro 2010	Incorrect 2010 Result:	RR = 1.84 (1.66 – 2.05)	2002-2007

Source: Enstrom, 2012.

potentially influential statistical outlier when the 51 metropolitan areas only are considered" and "the statistical significance of the correlation between the reduction in $PM_{2.5}$ and population-weighted life expectancy in the 51 largest U.S. metropolitan areas should not be affected by the removal of a single data point. Unfortunately, it appears that the statistical significance of the correlation is lost after removing Topeka, Kansas, from the regression analysis" (p. 133).

Specifically, Krstic found "removing data point number 46 (Topeka, Kansas), as an observed potentially influential statistical outlier, yields weak and not statistically significant correlation (i.e., ~0.35 years per 10 mg/m^3; r2 = 0.022; p = 0.31) between the studied variables" (*Ibid.*). He further reported, "Similar and statistically not significant results are obtained on the basis of the complete data kindly provided by the authors for the 211 counties from the 51 metropolitan areas." Krstic's scatter diagrams, shown in Figure 6.3.2.5, clearly show the outlier (Topeka, in the bottom left of the first scatter diagram) and the lack of correlation when it is removed. Krstic concluded:

> The results of the presented reanalysis on the basis of the data from Pope *et al.* (2009) show that the statistical significance of the association between the reduction in $PM_{2.5}$ and the change in life expectancy in the United States is lost after removing one of the metropolitan areas from the regression analysis. Hence, the observed weak and statistically not significant correlation between the studied variables does not appear to provide the basis for a meaningful and reliable inference regarding potential public health benefits from air pollution emission reductions, which may raise concern for policymakers in decisions regarding further reductions in permitted levels of air pollution emissions (p. 135).

Young and Xia (2013) observed, "At one point or another, the Environmental Protection Agency (EPA) and the California Air Resources Board (CARB) speak of thousands or more than 160,000 deaths attributable to $PM_{2.5}$. … The EPA and CARB base their case on statistical analysis of observational data. But if that analysis is not correct, and small-particle air pollution is not causing excess statistical deaths, then the faulty science is punishing society through increased costs and unnecessary regulation" (p. 375). They reported the results of their reanalysis of data used in Pope *et al.* (2009) as follows: "We compute multiple analyses sweeping across the county from west to east and show that one can 'cut' along the longitude passing just west of Chicago and find no effect of $PM_{2.5}$ to the west and a small effect of $PM_{2.5}$ on statistical deaths to the east. Both Styer *et al*. and Smith *et al*. make the point if the effect of the pollutant is not consistent, then it is unlikely that you have a causative agent. We agree" (p. 376).

Beyond their finding of heterogeneity, Young and Xia reported, "The association between $PM_{2.5}$ with mortality, when compared to the associations between other variables and mortality, shows that the importance of $PM_{2.5}$ is relatively small. There is no measurable association in the western United States, although it accounts for about 11% of the variance in the eastern United States. The Pratt regression analysis across the entire United States has $PM_{2.5}$ explaining about 4% of the standard deviation" (p. 383). The authors conclude, "All analysis indicates that changes in income and several other variables are more influential than $PM_{2.5}$, so policymakers might better focus on improving the economy, reducing cigarette smoking, and encouraging people to pursue education" (p. 384).

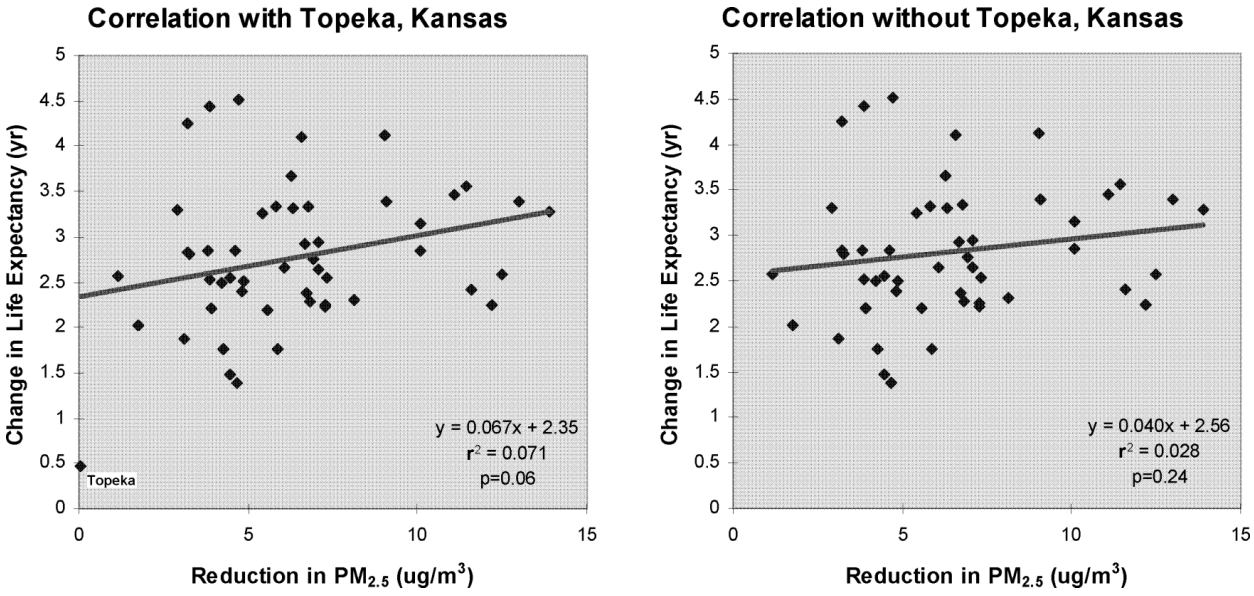
Milloy (2013) reported the results of his analysis of daily air quality and daily death data in California for 2007–2010. According to the author's executive summary, "Based on a comparison of air quality data from the California Air Resources Board and death certificate data for 854,109 deaths from the California Department of Public Health for the years 2007–2010, no correlation was identified between changes in ambient $PM_{2.5}$ and daily deaths, including when the analysis was limited to the deaths among the elderly, heart and/or lung deaths only, and heart and/or lung deaths among the elderly."

Milloy concluded, "Although this is only an epidemiological or statistical study that cannot absolutely exclude the possibility that $PM_{2.5}$ actually affects mortality in some small and as yet unknown way, these results also illustrate that it would be virtually impossible to demonstrate through epidemiological study that such an effect actually exists" (*Ibid.*).

* * *

Observational studies funded by and relied upon by the EPA and other air quality regulators fail to show relative risks (RR) that would suggest a causal

Figure 6.3.2.5
Change in life expectancy vs. reduction in $PM_{2.5}$ concentration with and without Topeka, Kansas as an influential outlier



Source: Krstic, 2013. Data from Pope *et al.*, 2009.

relationship between the chemical compounds created during the combustion of fossil fuels and adverse human health effects. Independent researchers have examined the data and found no such relationship exists, meaning tens and even hundreds of billions of dollars have been wasted trying to solve a problem that did not exist. Objective research reveals aerial particulate matter poses little or no threat to public health. Similar analyses of EPA's other NAAQS and regulatory initiatives reach similar conclusions.

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6.4 Circumstantial Evidence

Circumstantial evidence cited by the EPA and other air quality regulators is easily refuted by pointing to contradictory evidence.

The EPA and other air quality regulators cite observational studies with small sample sizes (such as the Harvard Six Cities report), historical incidents where cases of extremely poor air quality appeared to have caused a spike in illness or mortality, and laboratory experiments showing physiological responses to high levels of exposure that might be indicative of human health effects in the real world. These are all examples of circumstantial evidence being cast as proof of causation and are easily refuted by contradictory evidence.

6.4.1 Sudden Death

Real-world evidence that fine particulate matter ($PM_{2.5}$) does not cause sudden death is readily available. Everyone is constantly and unavoidably exposed to $PM_{2.5}$ from both natural and manmade sources. Natural sources include dust, pollen, mold, pet dander, forest fires, sea spray, and volcanoes. Manmade sources primarily are smoking, fossil fuel combustion, industrial processes, wood stoves, fireplaces, and indoor cooking. Indoor exposures to $PM_{2.5}$ can easily exceed outdoor exposures by as much as a factor of 100. Although the EPA claims almost 25% of annual U.S. deaths are caused by ambient levels of $PM_{2.5}$, no death has ever been medically attributed to such exposure.

Much higher exposures to $PM_{2.5}$ than exist even in the "worst" outdoor air are not associated with sudden death. The level of $PM_{2.5}$ in average U.S. outdoor air – air the EPA claims can cause sudden death – is about 10 millionths of a gram (microgram) per cubic meter. In one day, a person breathing such air would inhale about 240 micrograms of $PM_{2.5}$. In contrast, a cigarette smoker inhales approximately 10,000 to 40,000 micrograms of $PM_{2.5}$ *per cigarette*. A pack-a-day smoker inhales 200,000 to 800,000 micrograms every day.

A marijuana smoker inhales 3.5 to 4.5 times more $PM_{2.5}$ than a cigarette smoker – i.e., 35,000 to 180,000 micrograms of $PM_{2.5}$ per joint (Gettman, 2015). Typical water pipe or "hookah" smokers inhale the equivalent $PM_{2.5}$ of 100 cigarettes per session. Yet there is no example in published medical literature of these various types of short-term smoking causing sudden death despite the very high exposures to $PM_{2.5}$ (Goldenberg, 2003). Sudden deaths due to high $PM_{2.5}$ exposures were not reported when Beijing experienced $PM_{2.5}$ levels of 886 micrograms per cubic meter – some 89 times greater than the U.S. daily average (Milloy, 2013).

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6.4.2 Life Expectancy

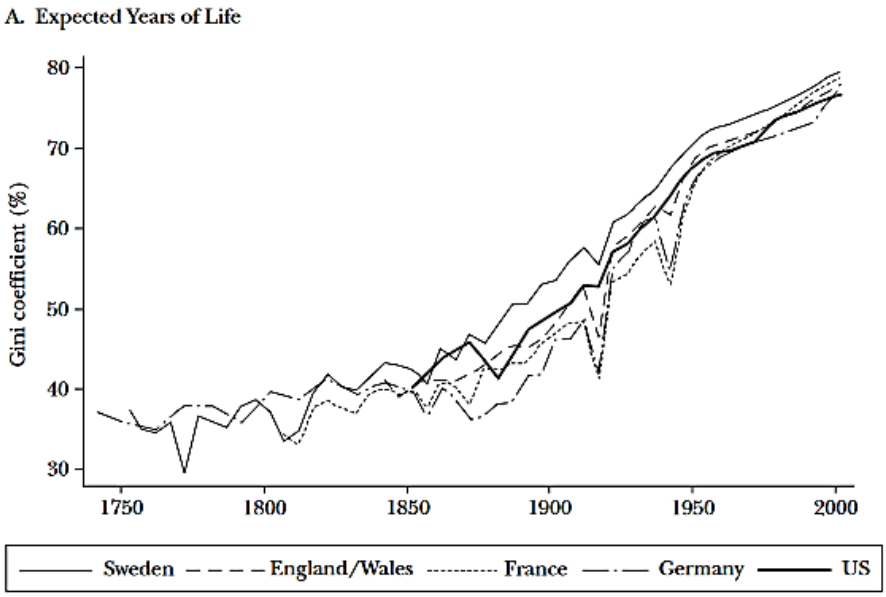
The sources cited in the introduction to this chapter leave no doubt that the chemical compounds created during the combustion of fossil fuels are not causing an epidemic of illnesses. Further evidence is easy to find. Life expectancy in the world's wealthiest countries – all of them with the highest levels of energy consumption and fossil-fuel use in the world – rose rapidly since the beginning of the Industrial Revolution, as shown in Figure 6.4.2.1. According to the U.S. Census Bureau:

- "The world average age of death has increased by 35 years since 1970, with declines in death rates in all age groups, including those aged 60 and older (Institute for Health Metrics and Evaluation, 2013; Mathers *et al.,* 2015).

- "From 1970 to 2010, the average age of death increased by 30 years in East Asia and 32 years in tropical Latin America, and in contrast, by less than 10 years in western, southern, and central Sub-Saharan Africa (Institute for Health Metrics and Evaluation, 2013; Figure 4-1).

- "In the mean age at death between 1970 and 2010 across different WHO regions, all regions have had increases in mean age at death, particularly East Asia and tropical Latin America.

Figure 6.4.2.1
Expected life expectancy for five rich countries, 1742–2002



Source: Peltzman, 2009, p. 180, Figure 2.

- "Global life expectancy at birth reached 68.6 years in 2015 (Table 4-2). A female born today is expected to live 70.7 years on average and a male 66.6 years. The global life expectancy at birth is projected to increase almost 8 years, reaching 76.2 years in 2050.

- "Northern America currently has the highest life expectancy at 79.9 years and is projected to continue to lead the world with an average regional life expectancy of 84.1 years in 2050." (U.S. Census Bureau, 2016, pp. 31–33).

Life expectancy in the United States rose from 47 years in 1900 to 77 years in 1998 (Moore and Simon, 2000, p. 26). Life expectancy rose for every age group in the United States during that time, as shown in Figure 6.4.2.2.

According to a landmark study on the causes of cancer commissioned by the U.S. Office of Technology Assessment and published by the National Cancer Institute in 1981, comparisons of cancer rates in countries with different levels of air quality as well as of urban and rural residents found "little or no effect of air pollution. To distinguish between 'little' and 'no' from such direct comparisons is not of course possible, as any real effects will probably be undetectably small, while even if there are no real effects it is impossible to prove a negative" (Doll and Peto, 1981). The authors estimated "combustion products of fossil fuels in ambient air ... would ... account for about 10% of lung cancer in big cities or 1% of all cancer. These crude estimates probably provide the best basis for the formation of policy." In the three decades since the Doll and Peto report, air quality in the United States and in many other developed countries has improved dramatically, with aerial concentrations of potentially harmful man-made chemical compounds often falling to background (natural) levels. It is logical therefore to assume that the health risks of exposure to such chemicals, barely detectible when Doll and Peto were writing, are indistinguishable from zero today. This in fact is what more recent researchers have found. According to Ames and Gold (1993), "cancer death rates in the United States (after adjusting the rates for age and smoking) are steady or decreasing" and "In the United States and other industrial countries life expectancy has been steadily increasing, while infant mortality is decreasing. Although the data are less adequate, there is no evidence

Figure 6.4.2.2
Life expectancy in the United States has increased at every age

Age	1901	1954	1968	1977	1990	2014
0	40	70	70	73	76	78.9
15	62	72	72	75	77	79.5
45	70	74	75	77	79	81.1
65	77	79	80	81	82	84.4
75	82	84	84	85	86	87.3

Source: Data through 1990 from Moore and Simon, 2000, p. 27. 2014 data from Arias, 2017, Table A, p. 3.

that birth defects are increasing. Americans, on average, are healthier now than ever" (pp. 153, 154). Ames and Gold specifically reject popular claims that man-made toxins are responsible for significant human health risks:

> Epidemiology and toxicology provide no persuasive evidence that pollution is a significant cause of birth defects or cancer. Epidemiological studies of the Love Canal toxic waste dump in Niagara Falls, New York, or of dioxin in Agent Orange, or of air pollutants from refineries in Contra Costa County, California, or of contaminated well water in Silicon Valley, California or Woburn, Massachusetts, or of the pesticide DDT, provide no persuasive evidence that such forms of pollution cause human cancer. In most of these cases, the people involved appear to have been exposed to levels of chemicals that were much too low (relative to the background of rodent carcinogens occurring naturally or produced from cooking food) to be credible sources of increased cancer (p. 175).

Michael Gough (1997) wrote, "When EPA was established in 1970, there was a clear expectation that removing chemicals from the air, water, and soil would reduce cancer rates. Now, almost three decades later, scientists are almost uniform in their opinion that chemicals in the environment are associated with only a tiny proportion of cancer. Moreover, there is no evidence that EPA's efforts have had any effect on cancer rates." He concludes,

> There is no cancer epidemic. Cancer mortality from all cancers other than lung cancers has been dropping since the early 1970s, and lung cancer mortality began dropping in 1990. The contribution of environmental exposures to cancer is small – two percent or less – and regulation of those exposures can reduce cancer mortality by no more than one percent. Some of that reduction, even if realized, might be offset by increased food prices that would decrease consumption of fresh fruits and vegetables that are known to protect against cancer.

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6.5 Conclusion

> *It is unlikely that the chemical compounds created during the combustion of fossil fuels kill or harm anyone in the United States, though it may be a legitimate health concern in third-world countries that rely on burning biofuels and fossil fuels without modern emission control technologies.*

Ray Hyman's Categorical Directive, mentioned in Chapter 1, says "before we try to explain something, we should be sure it actually happened." Scientists, economists, and others attempting to incorporate the damages caused by chemical compounds created during the combustion of fossil fuels should pause and consider whether such damages exist at all. Economists and computer modelers dutifully enter stylized facts provided by the U.S. Environmental Protection Agency and World Health Organization into their integrated assessment models to calculate a "social cost of carbon," but it is unlikely such emissions kill or harm *anyone* in the United States or in other developed countries, though it may have been a concern at one time. Air quality may still be a legitimate health concern in developing countries that rely on burning biofuels and fossil fuels without emission control technologies.

The best available evidence suggests levels of exposure to the chemicals created by the combustion of fossil fuels is too low in the United States, and higher but still too low in other developed countries, to produce the public health effects alleged by environmentalists and many government agencies. Even those low levels of exposure are falling fast in the United States thanks to prosperity, technological change, and government regulation. As Paracelsus said many centuries ago, the dose makes the poison. Without exposure, there can be no harm.

The most influential source of claims that air pollution is a public health hazard in the United States, the U.S. Environmental Protection Agency (EPA), is simply not credible on this issue. Given its constrained mission, flawed paradigm, political pressures, and evidence of actual corruption, there is no reason to believe any science produced by the EPA in justification of its regulations. The EPA makes many assumptions about relationships between air quality and human health often in violation of the Bradford Hill Criteria and other basic requirements of the scientific method. These assumptions, such as a linear no-threshold dose-response relationship and that injecting mice and rats with massive doses of chemicals can accurately forecast human health effects at ambient levels of air pollution, have been debunked again and again.

Observational studies cited by the EPA and other air quality regulators are not designed to test hypotheses and cannot establish causation. Most observational studies cannot be replicated, and in nearly one case in ten, efforts to replicate results find *benefits* where the previous study found harms or vice versa. In any case, observational studies fail to show relative risk (RR) ratios that would suggest a causal relationship between air quality and adverse human health effects.

Finally, the circumstantial evidence cited by the EPA and other air quality regulators is easily refuted by pointing to contradictory evidence. From everyday experience where we do not see people dropping dead in the street from exposure to cigarette fumes that contain orders of magnitude more particulates than ambient air, to the extensive evidence that human health and longevity are increasing over time, the circumstantial evidence is overwhelming that emissions produced from the use of fossil fuels are *not* a threat to human health.