Death by Dust

Call it soot, haze, dust, black smoke, British Smoke, black carbon, fine particles, ultrafine particles, nanoparticles—call it whatever may suit you. But think of it for what it causes: death.

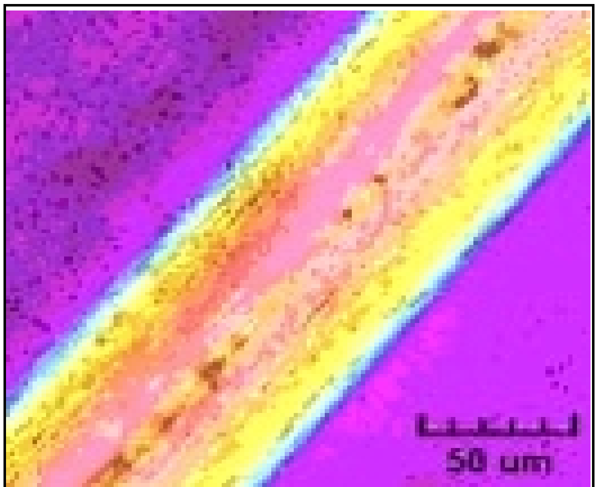

Figure 1 The tiny black specks against the purple background are diesel soot particles, magnified approximately 300 times, next to a human hair for comparison. (Source: South Coast Air Quality Management District, California)

It seems improbable that liquids or solids small enough for 40 to 1,000 to be laid side-by-side on the width of a human hair could cause arteries to harden and then, with a sudden upsurge, trigger fatal heart attacks and strokes. It seems equally improbable that the darkest of these solids and liquids could warm the entire planet and accelerate the melting of glaciers, snow packs and the polar icecaps. Improbable or not, however, it s also true

In reality, the lethal nature of soot and other particles makes sense. Such particles are largely the result of human activity, especially combustion. Animal lungs developed originally in an aquatic environment where such particles would not be found. When animals moved ashore, the air was pristine by today's standards so particles would have been rare.[1] Thus, as lungs, blood, and hearts evolved, resistance to particles would not have developed for the simple reason that they didn't, for practical purposes, exist. Today the human body can defend itself against bacteria, viruses, fungi and other attackers, but not soot—or, at the very least, not very well.

Particles are of two sorts:

1. So-called "primary" particles, which come straight from tailpipes, smokestacks, forest fires and the like, much of which are carbon, or soot; and,

2. "Secondary" particles, which are formed when other pollutants—such as sulfur dioxide, the principal cause of acid rain, for example—cook and age in the air, turning into new and different poisons.[2]

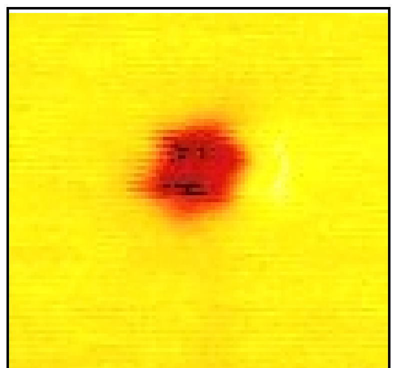

Figure 2 Ammonium sulfate, a fine particle formed after sulfur found in coal and fuel oil is burned and ages in the air. It is linked to death. (Institute for Atmospheric and Climate Science, Zurich, Switzerland).

All of these particles cause death and illness. Some, such as "black carbon," or soot, created when fuels like coal, diesel fuel or firewood, fail to burn completely, also cause global warming.

Particles cause death and illness over both the short and long term. On a daily basis, as their concentration rises, so do and illnesses, ranging from runny noses to emphysema. Over the longer term, particles cause grave illness, and injury, as well as death.

Fine particles billow by the millions of tons each year from gasoline, diesel and jet engines, coal fired power plants, steel mills, and hundreds of other types of smokestacks and tail pipes, literally clouding virtually the entire continent.

> *Only intense simultaneous efforts to slow CO_2 emissions and reduce non-CO_2 forcings can keep climate within or near the range of the past million years.*
> Hansen, J. et. al.,Climate change and trace gases.

Formed when coal, oil, wood and other fuels are burned incompletely,[3] the darkest component of soot, black carbon (BC), has a relatively short lifetime in the atmosphere—by some estimates from six to ten days[4]—because it is washed out by rain or other precipitation into or onto soil, oceans, snow and ice. It also simply settles out of the air. Once deposited, however, black carbon is extremely resistant to breakdown by heat, chemicals or organisms,[5] so it can reside for hundreds to thousands of years, acting as an enormous sink for carbon.[6] It is found virtually everywhere, even at remote and seemingly pristine islands.[7] Between 35 to 80 percent of total carbon in the air is in the submicron, or exceedingly fine, fraction, which is the most dangerous from a health perspective.[8] For one specific type of black carbon, diesel soot, 99.5 percent of the particles are either ultrafine (less than one-tenth of a micron (millionth of a meter)or fine (0.1-2.0 microns).[9]

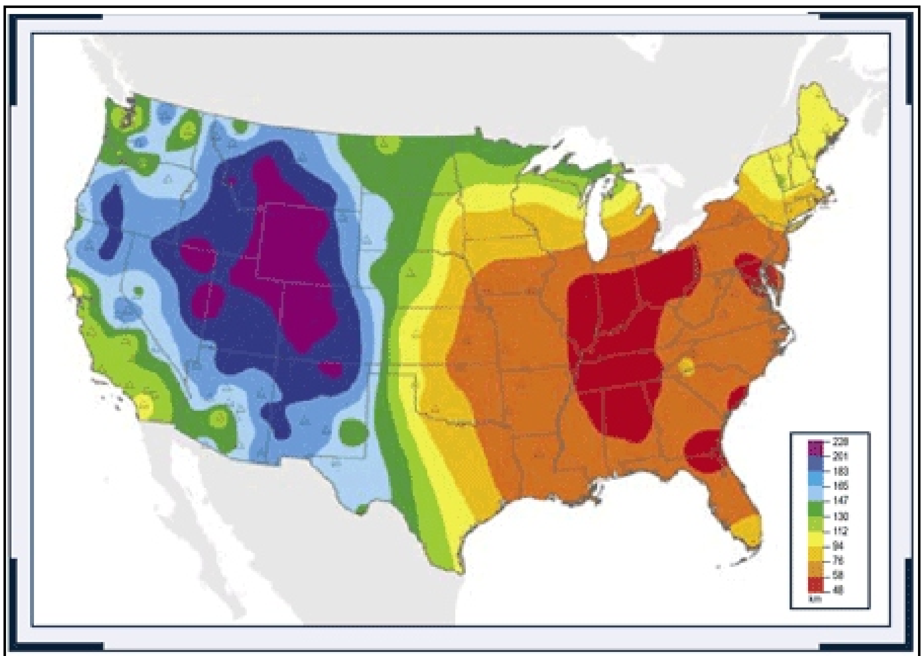

Figure 3 Limited visibility in the United States due to air pollution ranges from a low of 30 miles (48 kilometers) in the east to 138 miles (223 kilometers) in the Rocky Mountain west. (Source: U.S. EPA)

Most pollutants cause global warming by trapping heat from the Earth's surface, preventing it from being radiated into space. Black carbon causes warming by darkening things—droplets of cloud water, for example—and thus causing them to absorb incoming sunlight.[10, 11] The effect is particularly pronounced in snowy and icy environments, including the polar regions of the south,[12] as well as the north, where deposition in the early 20th century was so great that the warming effect was roughly 3 watts per square meter, or eight times the typical pre-industrial level.[13] Some climatologists calculate that black soot may be responsible for 25 percent of observed global warming over the past century.[14]

Visibility measurements from airport and other sites reflect concentrations of fine particles. On maps in which dense haze is shown in deepening shades of orange, that color has spread from a small, roughly circular area covering northern Ohio and bordering areas of Pennsylvania and Michigan in 1960 to a blanket over virtually every square mile east of the Mississippi River in 1990.[15] In a few locations—Southern California is the most notable—fine particle levels have fallen. Visibility has also improved somewhat in some areas of the eastern United States recently, principally because of acid rain controls, but visibility is still vastly worse than in the 1960s.[16]

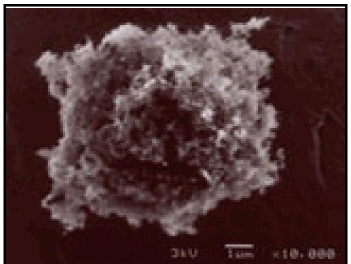

Figure 4 A particle of black carbon, or soot. These not only cause death and illness, but also global warming, especially in snow and icy areas like the north and south poles. (Source: NASA).

These particles find their way into even the youngest lungs, and a sobering study in the United Kingdom leaves little room for doubt about the toxic nature of black carbon. In Leicester, England, physicians analyzed macrophages—these are specialized white blood cells that attack invaders like bacteria and viruses and black carbon particles. Macrophages—the word literally means "big eater"— crawl through fluid in bodies searching bacteria, viruses and other things that don't belong there.

When a macrophage encounters an invader, its cell wall splits, allowing it to surround a particle; the cell membrane comes back together, with the particle ending up inside, engulfed, and killed by the macrophage.

When the Leicester scientists examined the macrophages of 64 children undergoing elective surgery, they found ultrafine, or extremely small, carbon particles—the sort from gasoline and diesel tailpipes—present in the macrophages of all of the children, even a 3-month-old infant.[17] The levels in children who lived close to busy roads were roughly three times those of children living by quiet roads.[18]

As levels in the air increased, so did the amount of carbon in the white blood cells. And with increases in black carbon content, there was a corresponding drop in the children's ability to breathe normally. The amount of air they could exhale forcibly in one second, called FEV1, fell 17 percent. More alarming, the total amount of air they could exhale forcibly from a full lung, called forced vital capacity dropped over 17 percent. Another measure, $FEV_{25\text{-}75}$ which is a test of the speed with which air is exhaled dropped by 34.7 percent.[1] This is an extraordinary drop, and one that continues for life. Such reductions in later years are considered by many doctors as second only to the exposure to tobacco smoke as a risk factor for death.[2]

There was a similar examination of 14 non-smoking Canadian workers, 11 utility employees and 3 non-maintenance employees of a university, all of whom had ultrafine particles,[19] evidence that the particles are literally everywhere—they just can't be seen, or if they can, it is as a slight, barely visible decrease in visibility, easily confused with fog.

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[1]Kulkarni, N., Pierse, N., Rushton, L. & Grigg, J. Carbon in Airway Macrophages and Lung Function in Children, p. 21, *N Engl J Med* 355:1 July 6, 2006.

[2]Gauderman, WJ, Air Pollution and Children—An Unhealthy Mix, p. 78, *N Engl J Med* 355:1 July 6, 2006.

Figure 5 The lung of a non-smoking city dweller that has been removed and dried, showing black carbon particles. In the lungs of Mexico City residents, investigators measured roughly two billion particles in every gram of dry lung tissue. Levels in the lungs of residents of Vancouver, British Columbia—a much less polluted city—were lower, but still remarkable: about 280 million per gram of dry lung tissue. (Source: University of California at San Francisco)

The numbers of particles have been rising for at least a half century, and there seems to be no end in sight. Consider, for example, that in 1950 there were no sales of jet fuel because there were no commercial jet aircraft. Today, however, the U.S. Energy Information Administration estimates that 1.7 million barrels of jet fuel are sold each day nationally, and when burned, each pound results in 100,000,000,000,000,000 particles.[20] Similarly, motor vehicles, especially diesels, emit immense amounts of soot—classified by the California Air Resources Board as a toxic air contaminant. Since 1960, the miles traveled nationally by 18-wheel, over the-road trucks has jumped 459 percent.[21]

Once in the air, particles reach the deepest recesses of the lung, and many stay there. There can be no doubt of this, because they have been counted by electron microscope in lung tissues of the dead. In residents of Mexico City, scientists found roughly two billion particles in every gram of dry lung tissue. Levels in the lungs of residents of Vancouver, British Columbia—a much less polluted city—were lower, but still remarkable: about 280 million per gram of dry lung tissue.

Though some of the pollution particles remain in the lung, others enter the blood stream or lodge themselves in the lymph, or immune, system. What happens next is something of a mystery that scientists are anxious to solve.[22]

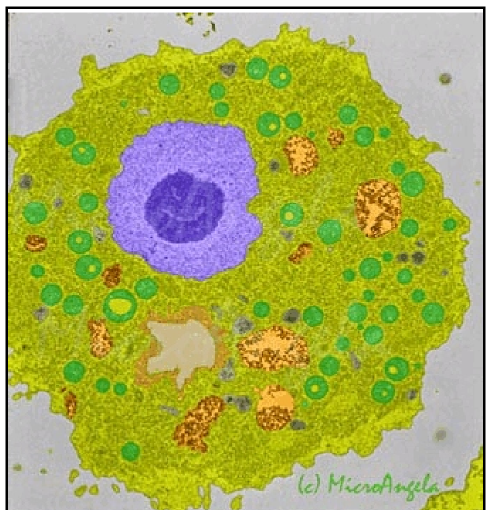

Figure 6 In this image, a macrophage, or "big eater," has engulfed metal particles, which are the black spots (source: University of Hawaii).

WHAT IS A PARTICLE?

"Particle" is a catch-all word. Defining it is not unlike trying to describe the contents of a Halloween goody bag after a night of trick-or-treating. "Particles" include solids and liquids alike, everything from diesel soot to ocean spray—dust and soil, metals ranging from arsenic to zinc, unburned and partially burned gasoline, tiny pieces of tires and brake pads, and scores of chemicals.

Defining particles is complicated even further by the fact that many of them are not emitted directly from a tailpipe or smoke stack, but instead are created by atmospheric chemical reactions. For example, when sulfur-containing coal, gasoline or diesel fuels are burned, a colorless gas, sulfur dioxide, is formed. It ages to form liquid fine particles that are droplets of acid rain, fog or snow, which in turn, turn into extremely fine, solid particle sulfates. Much the same happens to oxides of nitrogen. All these, in turn, react with the thousands of organic chemicals, yielding an atmospheric soup that simply cannot be completely described.

"No single analytical technique is currently capable of analyzing the entire range of organic compounds present in the atmosphere as particulate matter," explained the U.S. Environmental Protection Agency in a 2001 document.[23]

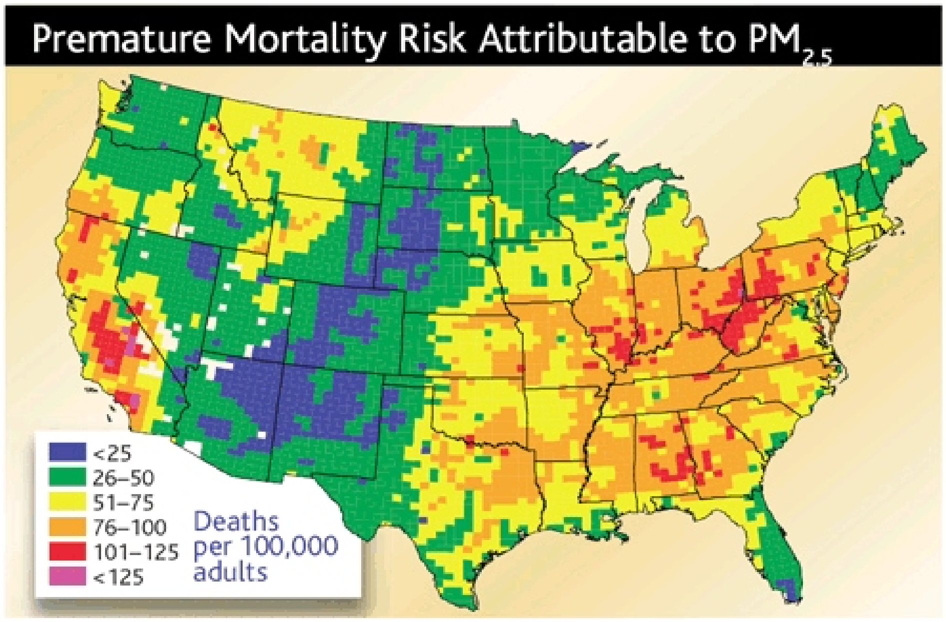

Figure 7 Fine particles kill 50,000 or more Americans each year. (Source: California Air Pollution Control Officers Association.)

Some particles can be sorted based on their chemical identities, such as sulfates and nitrates and metals such as mercury and lead. But most often particles are classified according to size. Part of the rationale for this is that whether a particle is an acid or metal, granite or gasoline, size determines how it will behave in the atmosphere and how far it will travel into the lung. Large particles—dust blown from fields or eroded from rocks, for example—travel shorter distances and are generally captured in the upper airways. Extremely small particles, emitted by the trillions times trillions from diesels, jet aircraft, powerplants, and others sources, can travel thousands of miles remaining suspended in air for weeks or months. When inhaled, they can penetrate to the very deepest parts of the lung.[24]

As a general matter—and like most generalizations, this one has exceptions—particles are described as coarse, fine, and ultrafine.

3. **Coarse particles** - larger than 2.5 microns or two and one-half one-millionths of a meter. Much of this larger fraction consists of soil, street dust, pollen, and mold, though it can include toxic metals and biologically hazardous materials.

4. **Fine particles** - 2.5 microns and smaller, and components include a wide variety of organic chemicals, compounds of lead, cadmium, vanadium and other metals, as well as black carbon from diesels and power plants, often coated with chemicals that can cause cancer and other serious illnesses.

5. **Ultrafine particles** - sometimes also referred to as nanoparticles, these can be as small as one-billionth of a meter. They include metals, organic compounds, and carbon spheres. Small does not mean safe, however: mice exposed to ultrafine particles build up 55 percent more artery-hardening plaque than those breathing clean air and 25 percent more than those exposed to fine particles.[25]

Fine particles rapidly enter the blood stream. They can be detected in blood within one minute, reach a peak within, 10 to 20 minutes, and stay at that level for up to 60 minutes.[26] What happens next is something of a mystery that scientists are anxious to solve.[27]

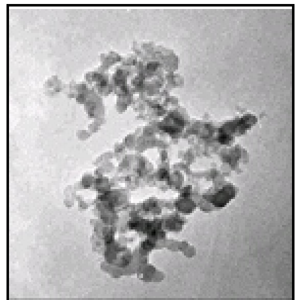
Figure 8 A diesel soot particle. (Source: Argonne National Laboratory)

One possibility is that particles somehow change levels of ingredients of the blood, as shown by a study in 112 persons over the age of 60 in Edinburgh and Belfast.[28] In a study of 47 Finnish patients with stable heart disease, exposure to fine and ultrafine particles was consistently associated with an electro-cardiogram abnormality often found in those with heart injury.[29] And, in Chapel Hill, North Carolina when elderly volunteers were exposed to concentrated Chapel Hill particles for two hours, their heart rate variability dropped significantly, which is often a predictor of heart illness and death.[30] Another study, this of people exposed to the Asian wildfires of 1998, suggests that particles somehow trigger an inflammatory reaction that, in turn, leads to a cascade of other adverse events.[31] One of the most provocative and useful studies found that PM_{10}–that is, particles smaller than 10 millionths of a meter–exposure resulted in an increase in C-reactive protein, an index of inflammation that is associated with increased rates of coronary artery disease.[32] One painstaking study at a time, the mechanism by which particles cause illness and death is being revealed. Notwithstanding these uncertainties, it is nevertheless clear that small particles—especially those from burning coal, oil, and other materials, but also some of those in the coarse fraction—trigger illness and death.

EFFECTS OF FINE PARTICLES

Mortality

The evidence that particulate kills is compelling. Studies have shown that increases in daily particle levels are followed by increases in daily deaths in Amsterdam, Athens, Barcelona, Basel, Berlin, Birmingham, Boston, Chicago, Cincinnati, Detroit, Dublin, Erfurt, Eastern Tennessee, London, Los Angeles, Lyon, Madison, Milan, Minneapolis, Mexico City, New York, Philadelphia, Provo, Rotterdam, Santiago, Santa Clara, Steubenville, St. Louis, Sao Paolo, Topeka, Valencia, and Zurich.[33]

The largest of these studies tracked the health histories of 552,138 adults in 151 metropolitan areas from 1982 through 1989 and accounted for smoking, obesity, age, alcohol use, and other potential confounding factors. In some studies, motor vehicle exhaust seems to play a critical role, but adverse health effects have also been linked to steel mills, peat-fired power plants, and a variety of other sources of particles.[34] The association between mortality and fine particulate matter is, in the words of Dr. Douglas Dockery of the Harvard University School of Public Health “consistent (and) robust.”[35]

The effects are associated with chronic and acute exposures alike. In a study of 772 patients in Boston who had suffered heart attacks, for example, researchers found that as concentrations of particles rose—both PM_{10} and $PM_{2.5}$—the frequency of heart attacks did the same a few hours to one day later.[36] Longer term exposures—years or even decades—are equally dangerous: when researchers analyzed data from more than 500,000 people in an average of 51 metropolitan districts in a study dating to 1982, they found that when PM_{10} concentrations increased by 10 micrograms per cubic meter, deaths from all causes rose 4 percent; from cardiopulmonary illness by 6 percent; and, from lung cancer by 8 percent.[37]

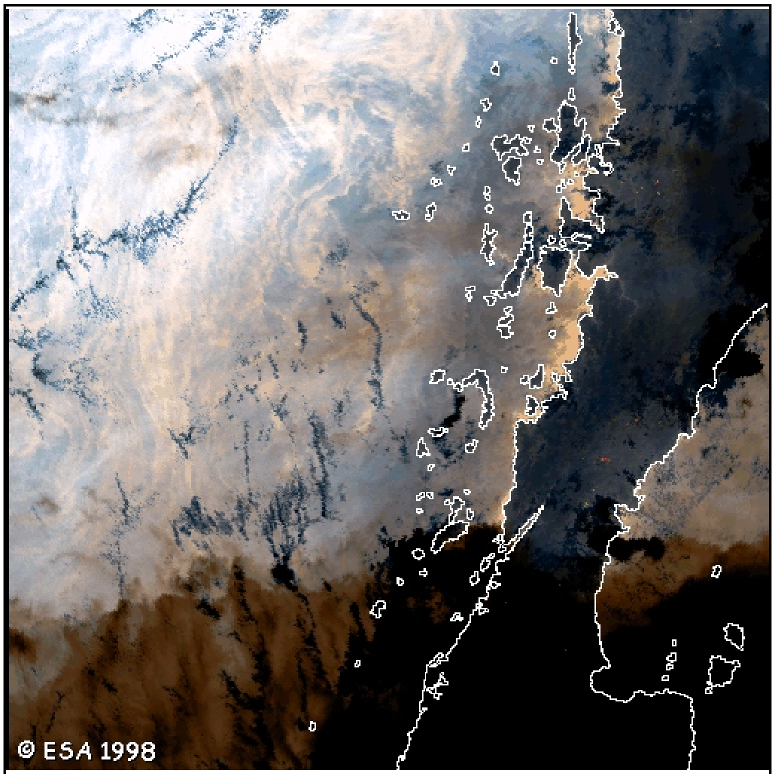

Figure 9 The haze of black carbon and other pollutants from Asian wildfires in 1997 and 1998 drifted across the Pacific Ocean to California and the American west. Investigators found evidence of heart and lung inflammation in Singaporean Army cadets exposed to the smoke. (Source: European Space Agency)

A brief review of some of these studies provides some sense of the breadth and depth of research that makes it possible for sober and conservative observers to accept the seemingly counter-intuitive proposition that something that can barely be seen, and even then only as haze, could be killing more people than automobile accidents. Consider the following:

6. Researchers from Johns Hopkins, Harvard and other universities examined data from 90 cities in different regions of the United States, covering all geographic areas. Daily levels of air pollution from 1987 to 1994 were compared to death and hospital records. The researchers found not only a link between exposure to particles and death, but "strong evidence of association between PM_{10} levels and exacerbation of chronic heart and lung disease sufficiently severe to warrant hospitalization."[38]

7. Responding to the rapidly accumulating body of evidence from the United States that air pollution was linked to mortality, the European Union founded the "Air Pollution and Health—A European Approach," or APHEA, study. Eleven teams of researchers from 10 different nations studied European cities with a total population of 25 million. As in North America, when particle levels rose, so did mortality and hospital admissions.[39] When British Smoke, or BS, a measure of particle concentrations, rose by 50 micrograms per cubic meter, mortality increased by 2.2 percent.[40]

8. In addition to comparing air pollution levels with deaths, researchers collected particle samples from the Canadian cities of Montreal, Ottawa-Hull, Toronto, Windsor, Winnipeg, Edmonton, Calgary, and Vancouver for chemical analysis. Carbon was the dominant constituent of the total particulate matter, while sulfur, which is a contaminant of coal and diesel fuel, had the highest correlation with fine particles. As in other studies, increases in mortality were linked to higher levels of particles, but the association was strongest with $PM_{2.5}$.[41]

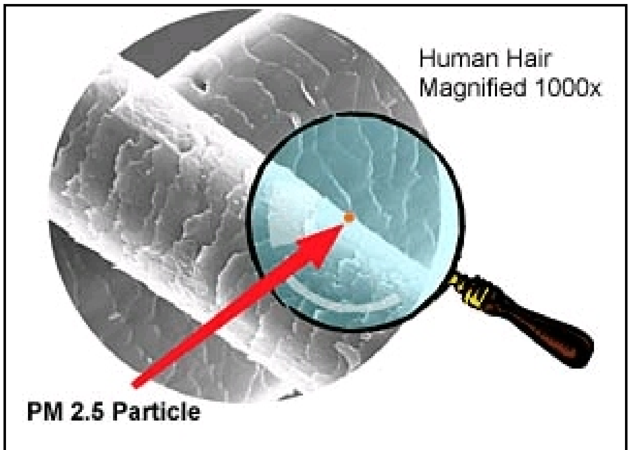

Figure 10 Source: U.S. Department of Energy

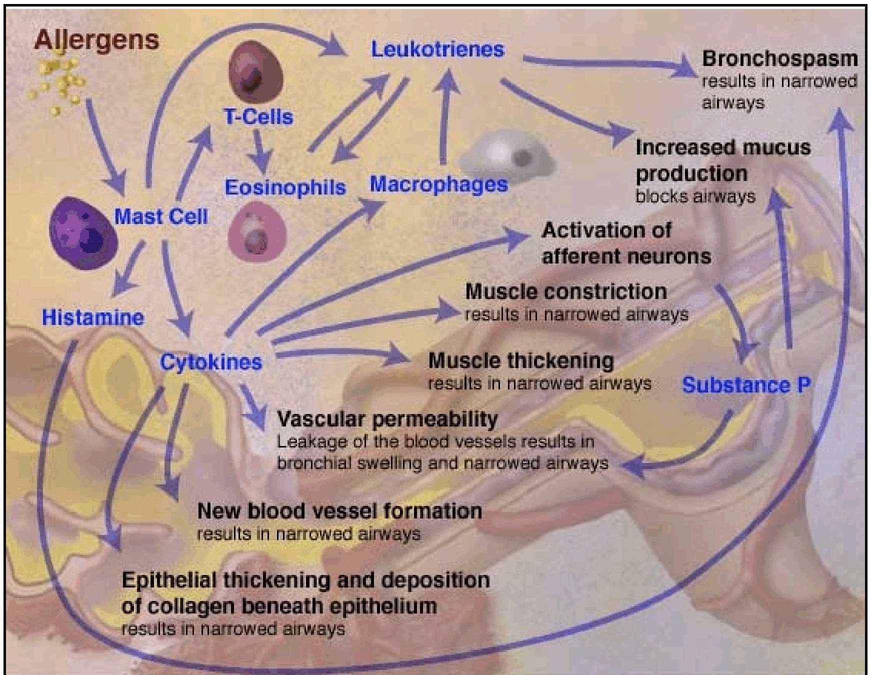

Figure 11 A depiction of how black carbon and other particles might, like common allergens such as pollen, trigger an inflamation, setting off a chain of events that ultimately leads to death from heart attack or stroke.

Some argue that particle-caused deaths are merely "harvesting"[42]—the acceleration by a few days of death in the elderly or frail that would have occurred in any event—but several studies have expressly addressed and discounted such claims.[43]

Two of the largest and most persuasive of the studies linking fine particles to illness and death are the Harvard Six Cities Study and the American Cancer Society Study (ACS). Published in the mid-1990s, they were sharply criticized by polluting industries and a few scientists who challenged the existence of a causal connection between fine particles and death. These criticisms, in turn, prompted two other studies (a) a re-analysis of the Six Cities and ACS work and (b) the National Morbidity, Mortality, and Air Pollution Study (NMMAPS), which was entirely new research on hospitalization and deaths associated with air pollution in major U.S. cities.

The re-analysis by independent investigators validated the original studies, confirming that they were sound science. In addition, NMMAPS found strong evidence linking daily increases in particle pollution in the twenty largest U.S. cities not only to increases in death, but increased hospital admissions for cardiovascular disease, pneumonia, and chronic obstructive pulmonary disease. These left no doubt that Americans were being placed at grave risk by particles. And, while the elderly and the ill may be at greatest risk from particles, the second largest group at risk are society's most defenseless: infants and children.

Infant and Child Mortality

The linkage between infant and child mortality and exposure to particles is the subject of considerably fewer studies than those of adults, but they are persuasive nonetheless. The results are consistent and they are reinforced by other studies of illness, suggesting a continuum of effects. In the Czech Republic, for example, researchers examined all births between 1989 and 1991. For each infant death, they randomly selected 20 other children of the same sex who had been born on the same day as the deceased, then examined 24-hour air pollution levels in the districts where each lived for the period between the birth and death. In all, they studied 2,494 infant deaths and, of these, the 133 due to respiratory causes were linked to increased levels of not only particles, but also sulfur dioxide and oxides of nitrogen. The researchers' conclusion was that "the effects of air pollution on infant mortality are specific for respiratory causes in [the period between one month and one year of age], are independent of socioeconomic factors, and are not mediated by birth weight or gestational age."[44]

Similarly, after scientists compared respiratory mortality in Sao Paulo, Brazil, of children under age five to daily levels of sulfur dioxide, carbon monoxide, ozone and PM_{10}, each was associated with an increased risk of death. As concentrations rose, so, too, did mortality and this was "quite evident after a short period of exposure (2 days)." The estimated proportions of respiratory deaths attributed to CO, SO_2, and PM_{10} when considered individually, were around 15 percent, 13 percent, and 7 percent, respectively.[45]

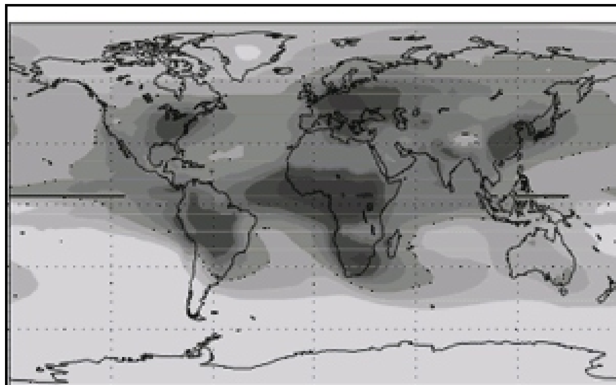

Figure 12 One year mean column burden of black carbon.

A quite intriguing study was done by economists at the National Bureau of Economic Research in Washington, D.C., who examined the relationship between infant mortality rates and decreases in particulate matter emissions because of a recession. They found that as levels of particles fell, so did neonatal mortality (death before the age of 28 days). Focusing specifically on Pennsylvania, they found that when the levels of total particulate matter dropped roughly 25 percent, infant deaths within one year of birth attributable to "internal" causes (e.g., respiratory and cardiopulmonary deaths) fell by 14 percent.[46]

Illness

In addition to their linkage to death, fine particles are associated with a litany of lesser ills, including runny or stuffy noses, sinusitis, sore throat, wet cough, head colds, hayfever, burning or red eyes, wheezing, dry cough, phlegm, shortness of breath, and chest discomfort or pain, as well as hospital admissions for asthma and bronchitis.[47] Increases in fine particle levels are accompanied by higher rates of chronic cough, asthma, and emphysema, even among non-smoking Seventh-Day Adventists.[48] Bronchitis and chronic cough increase in school children[49,50] as do emergency room and hospital admissions.[51,52] In Utah, when particulate levels rose, hospital admissions of children for respiratory illnesses tripled.[53]

The horrific toll of indoor black carbon in developing nations

The dominant source of black carbon in urban areas and developed nations is combustion in engines, especially diesels. Elsewhere, however, the burning of biomass is the culprit. This may be of crop resides in agricultural regions or fireplaces and woodstoves in rural areas. In less developed nations, however, the principal cause of black smoke is cooking and heating, which take a horrific toll on human health.

Throughout much of the developing world, cooking is done on primitive stoves in small rooms that frequently lack adequate ventilation. In one survey, 60 percent of households used a three-stone hearth or a U-shaped mud-plastered hearth known as a chulha. Only 28 percent of kitchens had chimneys, and only 32 percent had windows for ventilation. Under these circumstances, exposure to indoor air pollutants is unavoidable.[54]

Figure 13 Smoke from cooking, such as this scene in Nepal, kills about 2 million people each year. It causes acute respiratory infections (ARI), which are virtually unheard of in the United States and other developed nations, but are the single most important cause of mortality in children under age 5 in developing countries. (Source: Practical Action)

The pollution burden falls most heavily on women, who do most of the cooking, and on infants and children, who spend a great deal of time near their mothers and are highly susceptible to its damages. Acute respiratory infections (ARI) are the single most important cause of mortality in children under age 5,[55] and as indoor burning of biomass increases, so do ARIs. This has been documented generally,[56] as well as in the specific nations of South Africa,[57] Zimbabwe,[58] Nigeria,[59] Tanzania, [60] Gambia,[61, 62] Brazil,[63] Argentina,[64] and Nepal,[65] to name but a few. Indoor wood smoke is also associated with low birth weight[66] as well as infant mortality.[67]

By one estimate, the number of deaths because of indoor air pollution in India alone is as much as 400,000 per year.[68] Globally, it is estimated to account for two million deaths in developing countries and 4 percent of the world's disease burden.[69] Evidence also exists of associations with increased infant and perinatal mortality, pulmonary tuberculosis, nasopharyngeal and laryngeal cancer, cataract, and, specifically in respect of the use of coal, with lung cancer.[70]

BLACK CARBON (BC)

The combined emissions from wood, diesel, and coal represent more than 75 percent of the total global black carbon emissions.[71] Reducing emissions of black and other carbons is relatively straightforward. Diesel cars, trucks and buses can be fitted with trap oxidizers, which are devices that capture particles and burn them.[72] The same can be done with very large diesels, such as those found on ships.[73] Emissions from jet aircraft would drop sharply from replacing older aircraft with newer ones.[74]

Similarly, in China, which accounts for roughly one-quarter of the world's black carbon emissions, the dominant sources are neither vehicles nor electricity generating plants but instead indoor cooking and heating with coal or biomass. These uses account for roughly 83 percent of China's BC emissions, and they could virtually be eliminated by simply substituting coal briquettes or natural gas for raw coal, as well as some other measures.[75]

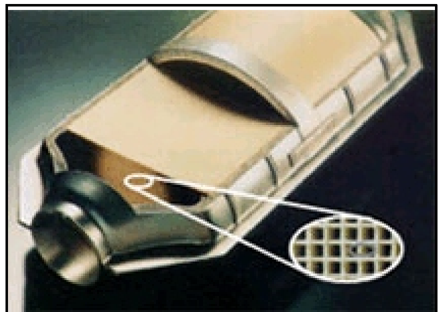

Figure 14 A trap oxidizer, a device that catches soot from car or truck exhaust, then destroys it.

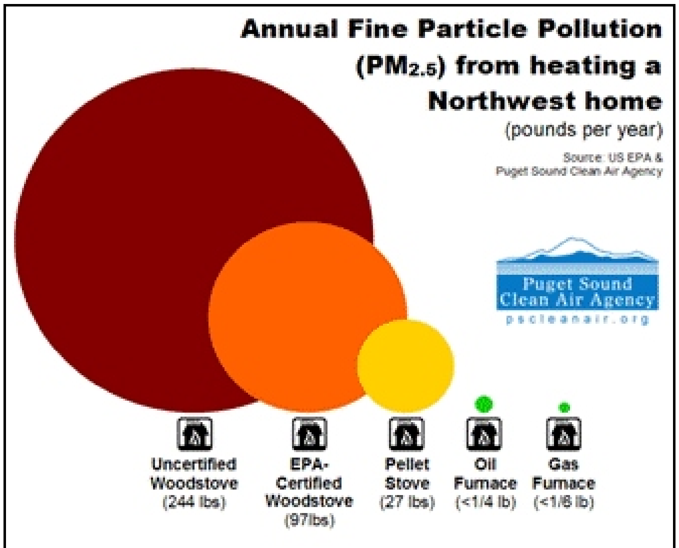

Figure 15 Want to eliminate soot from fireplaces? Get a pellet stove.

In the United States burning wood in older fireplaces or stoves is also a major source of black carbon. Newer versions of the stoves that burn pellets instead of conventional firewood or fireplace inserts that do the same can cut emissions 49 to 69 percent. Just as good, since the black carbon is burned instead of being vented to the air, more heat is extracted from the fuel. Where it's available, stove can be converted to natural gas, virtually eliminating black carbon emissions.[76] Upgrading stoves and fireplaces would not only provide almost immediate cooling benefits, but by some estimates, reduce annual air-pollution-related deaths in the United States by 2,000.[77]

1. Liem, K. F. Form and Function of Lungs: The Evolution of Air Breathing Mechanisms. American Zoologist 1988 28(2):739-759; doi:10.1093/icb/28.2.739.
Structural evolution of the vertebrate lung illustrates the principle that the emergence of seemingly new structures such as the mammalian lung is due to intensification of one of the functions of the original piscine lung. The configuration of the mechanical support of the lung in which elastic and collagen fibers form a continuous framework is well matched with the functional demands. The design of the mammalian gas exchange cells is an ingenious solution to meet the functional demands of optimizing maintenance pathways from nucleus to the cytoplasm while simultaneously providing minimal barrier thickness. Surfactant is found in the most primitive lungs providing a protective continuous film of fluid over the delicate epithelium. As the lung became profusely partitioned, surfactant became a functionally new surface-tension reduction device to prevent the collapse of the super-thin foam-like respiratory surface. Experimental analyses have established that in lower vertebrates lungs are ventilated with a buccal pulse pump, which is driven by identical sets of muscles acting in identical patterns in fishes and frogs. In the aquatic habitats suction is the dominant mode of feeding generating buccal pressure changes far exceeding those recorded during air ventilation. From the perspective of air ventilation the buccal pulse pump is overdesigned. However in terrestrial habitats vertebrates must operate with higher metabolic demands and the lung became subdivided into long narrow airways and progressively smaller air spaces, rendering the pulse pump inefficient. With the placement of the lungs inside a pump, the aspiration pump was established. In mammals, the muscular diaphragm represents a key evolutionary innovation since it led to an energetically most efficient aspiration pump. Apparently the potential energy created by contraction of the diaphragm during inhalation is stored in the elastic tissues of the thoracic unit and lung. This energy is released when lung and thorax recoil to bring about exhalation. It is further determined experimentally that respiratory and locomotory patterns are coupled, further maximizing the efficiency of mammalian respiration. Symmorphosis is exhibited in the avian breathing apparatus, which is endowed with a key evolutionary innovation by having the highly specialized lung continuously ventilated by multiple air sacs that function as bellows. Functional morphologists directly deal with these kinds of functional and structural complexities that provide an enormous potential upon simple changes in underlying mechanisms.

2. Khoder, M. I. Atmospheric conversion of sulfur dioxide to particulate sulfate and nitrogen dioxide to particulate nitrate and gaseous nitric acid in an urban area. Chemosphere
Volume 49, Issue 6, November 2002, Pages 675-684.
Sulfur dioxide, nitrogen dioxide, particulate sulfate and nitrate, gaseous nitric acid, ozone and meteorological parameters (temperature and relative humidity) were measured during the winter season (1999–2000) and summer season (2000) in an urban area (Dokki, Giza, Egypt). The average particulate nitrate concentrations were 6.20 and 9.80 ?g m-3, while the average gaseous nitric acid concentrations were 1.14 and 6.70 ?g m-3 in the winter and summer seasons, respectively. The average sulfate concentrations were 15.32 ug m-3 during the winter and 25.10 ug m-3 during the summer season. The highest average concentration ratio of gaseous nitric acid to total nitrate was found during the summer season. Particulate sulfate and nitrate and gaseous nitric acid concentrations were relatively higher in the daytime than those in the nighttime. Sulfur conversion ratio (Fs) and nitrogen conversion ratio (Fn) defined in the text were calculated from the field measurement data. Sulfur conversion ratio (Fs) and nitrogen conversion ratio (Fn) in the summer were about 2.22 and 2.97 times higher than those in the winter season, respectively. Moreover, sulfur conversion ratio (Fs) and nitrogen conversion ratio (Fn) were higher in the daytime than those in the nighttime during the both seasons. The sulfur conversion ratio (Fs) increases with increasing ozone concentration and relative humidity. This indicates that the droplet phase reactions and gas phase reactions are important for the oxidation of SO_2 to sulfate. Moreover, the nitrogen conversion ratio (Fn) increases with increasing ozone concentration, and the gas phase reactions are important and predominant for the oxidation of NO_2 to nitrate.

3. Mark Z. Jacobson, *Atmospheric Pollution: history, science and regulation*, Cambridge University Press, Cambridge, England (2002), p. 124.

4. Cooke, W.F.; Wilson, J.J.N. A global black carbon aerosol model. J of Geophys R. VOL. 101; ISSUE: D14 ; PBD: 27 Aug 1996.
A global inventory constructed for emissions of black carbon from fossil fuel combustion and biomass burning was implemented in a 3D global transport model and run for 31 model months, and results for January and July compared with measurements from the literature. The modeled values of black carbon mass concentration compared within a factor of 2 in continental regions and some remote regions but are higher than measured values in other remote marine regions and in the upper troposphere, explained by the coarse grid scale of the model, the simplicity of the current deposition scheme, and possibly too much black carbon being available for transport, which would also account for the disagreement in the upper troposphere. The disagreement may also be due to problems associated with the measurement of black carbon. Emissions from this database appear to provide a reasonable estimate of the annual emissions of black carbon to the atmosphere. Biomass burning emissions amount to 5.98 Tg and that from

fossil fuel to 7.96 Tg. A local sensitivity analysis showed that black carbon has a lifetime between 6 and 10 days, depending on the transformation rate between hydrophobic and hydrophilic black carbon.

5. Forbes, M.S., Raison, R.J., Skjemstad, J.O. 2006. Formation, transformation and transport of black carbon (charcoal) in terrestrial and aquatic ecosystems. Science of the Total Environment 370, 190–206. Also, Preston, C.M., Schmidt, M.W.I. 2006. Black (pyrogenic) carbon: a synthesis of current knowledge and uncertainties with special consideration of boreal regions. Biogeoscience 3, 397–420.

6. Brodowski S., Amelung, W., Haumaier, L., Abetz, C., Zech, W. 2005. Morphological and chemical properties of black carbon in physical soil fractions as revealed by scanning electron microscopy and energy-dispersive X-ray spectroscopy. Geoderma 128, 116–129. Also, Forbes, M.S., Raison, R.J., Skjemstad, J.O. 2006. Formation, transformation and transport of black carbon (charcoal) in terrestrial and aquatic ecosystems. Science of the Total Environment 370, 190–206.

7. Bhugwant, C., et. al. Impact of traffic on black carbon aerosol concentration at la Réunion Island (Southern Indian Ocean). Atmospheric Environment Volume 34, Issue 20, 2000, Pages 3463–3473.
To gain information on particle pollution by mobile sources, 3 experiments were conducted during the 1996–1998 period at Saint-Denis, the biggest urban site of La Réunion island (21.5˚S; 55.5˚E), situated in the Indian Ocean. Black Carbon (BC) concentrations were recorded with an Aethalometer which show high levels whatever the season (daily average: 270–650 ng m-3). At this site, a marked diurnal BC concentration variation is also evidenced in accordance with the observed traffic pattern. Measured daytime BC concentrations are 2–4 times greater than nighttime values. Neither MBL height obtained by radio soundings nor wind speed or direction could explain satisfactorily the BC variations. A comparison with BC concentrations measured at other more remote sites of the island (Sainte-Rose and the altitude site Piton Textor) suggests that the background concentrations of the island are of the order 50 ngC m-3. These background values are almost never encountered in the main city (range: 80–2800 ngC m-3). We show that due to a singular convergence of parameters (topography of the island, road network, movement of population, quality of fuel), the city of Saint-Denis appears as polluted as continental European big cities.

8. Novakov T.; Bates T.S.; Quinn P.K. Shipboard measurements of concentrations and properties of carbonaceous aerosols during ACE-2. Tellus, Volume 52, Number 2, April 2000
Mass concentrations of total, organic and black carbon were derived by analyzing the supermicron and submicron aerosol fractions of shipboard collected samples in the eastern Atlantic Ocean as part of the second Aerosol Characterization Experiment (ACE-2). These analyses were complemented by experiments intended to estimate the water-soluble fraction of the submicron carbonaceous material. Results: Depending on the sample, between 35% and 80% of total aerosol carbon is associated with the submicron fraction. Total submicron carbon was well correlated with black carbon, a unique tracer for incomplete combustion. These correlations and the approximately constant total to black carbon ratios, suggest that the majority of submicron total carbon is of primary combustion derived origin. No systematic relationship between total submicron aerosol carbon and sulfate concentrations was found. Sulfate concentrations were, with a few exceptions, significantly higher than total carbon. Experiments demonstrated that water exposure removed between 36% and 72% of total carbon from the front filter, suggesting that a substantial fraction of the total submicron aerosol organic carbon is water-soluble. An unexpected result of this study is that water exposure of filter samples caused substantial removal of, nominally insoluble, submicron black carbon. Possible reasons for this observation are discussed.

9. [59]
BERUBE, K.A., JONES, T.P., WILLIAMSON, B.J., WINTERS, C., MORGAN, A.J., & RICHARDS, R.J.
Physicochemical characterisation of diesel exhaust particles: factors for assessing biological activity
Atmospheric Environment 33 (1999) 1599–1614
From Cardiff. Source was a 1985 Japanese ISEKI tractor burning Esso 2000 Diesel and a 20/30 mixture of Esso light engine oil. Operated at 2000 rpm. Details of methodology.
Define four basic shapes:
1.Spherulites (individual particles); 2. Chains or clusters of spherulites; 3. Spherules (large bodies of spherulites; 4. Flake-like bodies.
Equivalent spherical diameter of spherulites was 0.23 microns; Distributions of particle size by number showed 10% were ultrafines; 89.5% were fine (0.1-2.0 microns) and 0.4% coarse (greater than 2.5 microns). But distribution by mass showed 0.01% ultrafine, 52.6% fine; and 47.4% coarse. Electron probe X-ray microanalysis showed presence of C,O,Na,Mg,K,Al,Si,P,S,Cl,and Ca along with a range of metals (Ti,Mn,Fe,Zn,&Cr). By analysis before and after sonication of particles in water, the mobile sorbed metals were Mg,P,Ca,Cr,Mn,Zn,Sr,Mo,Ba,Na,Fe,S, &

Si.
Stress differences between sonicated and impacted diesel particles - these differences are likely to affect toxicity. Excellent pictures. A definitive article.

10. Chýlek, P. , et. al. Black carbon and absorption of solar radiation by clouds. Journal of Geophysical Research, Volume 101, Issue D18, p. 23365–23372
The exact solution of the scattered electromagnetic field from a water droplet containing an arbitrarily located spherical black carbon particle is used to investigate the effect of black carbon on the absorption of solar radiation by clouds. When droplet absorption is averaged over all possible locations of black carbon within a droplet, the averaged absorption is close to the value calculated using the effective medium approximation. The preferential black carbon location on the top or close to the bottom of the droplet leads to an increased absorption. The estimated upper bound on the increased absorption of solar radiation (global and annual average) is 1-3 W/m^2 over the absorption of pure water clouds.

11. Jacobson, M.Z., Effects of absorption by soot inclusions within clouds and precipitation on global climate, J. Phys. Chem., 110, 6860–6873, 2006.

12. Hansen, A.D.A. Aerosol Black Carbon Measurements at the South Pole: Initial Results, 1986–1987. Geophysical Research Letters, VOL. 15, NO. 11, PAGES 1193–1196, 1988.
In December 1986 an aethalometer was installed at the NOAA/GMCC South Pole Observatory to measure concentrations of the combustion effluent tracer species aerosol black carbon (BC) with a time resolution of one hour. Hourly data covering a 1-year period from December 1986 through November 1987 showed infrequent events in which the concentrations increased greatly for periods of a few hours. These were attributed to local contamination. The remaining background data then yielded daily average BC concentrations generally ranging from 50 ng/m3 to 5 ng m/3, with a minimum in the early austral winter. The results imply long-range transport and suggest a minimum value of the order of 10 pg m/3 for its global background concentration.

13. McConnell, J.R., et. al. 20th-Century Industrial Black Carbon Emissions Altered Arctic Climate Forcing. 317 Science 5843, 7 Sep. 2007, pp. 1381–1384.
Black carbon (BC) from biomass and fossil fuel combustion alters chemical and physical properties of the atmosphere and snow albedo, yet little is known about its emission or deposition histories. Measurements of BC, vanillic acid, and non-sea-salt sulfur in ice cores indicate that sources and concentrations of BC in Greenland precipitation varied greatly since 1788 as a result of boreal forest fires and industrial activities. Beginning about 1850, industrial emissions resulted in a sevenfold increase in ice-core BC concentrations, with most change occurring in winter. BC concentrations after about 1951 were lower but increasing. At its maximum from 1906 to 1910, estimated surface climate forcing in early summer from BC in Arctic snow was about 3 watts per square meter, which is eight times the typical preindustrial forcing value.

14. Hansen, J. & Nazarenko, L. Soot Climate Forcing via Snow and Ice Albedos. PNAS.
Using the NASA GISS climate computer model to simulate effects of greenhouse gases and other factors on world climate and incorporating data from NASA spacecraft that monitor the Earth's surface, vegetation, oceans and atmospheric qualities, the calculated global warming from soot in snow and ice, by itself in an 1880–2000 simulation, accounted for 25 percent of observed global warming. NASA's Terra and Aqua satellites observe snow cover and reflectivity at multiple wavelengths, which allows quantitative monitoring of changing snow cover and effects of soot on snow. The researchers found that observed warming in the Northern Hemisphere was large in the winter and spring at middle and high latitudes. These observations were consistent with the researchers' climate model simulations, which showed some of the largest warming effects occurred when there was heavy snow cover and sufficient sunlight.

15. Maps can be found at http://capita.wustl.edu/CAPITA/CapitaReports/USVisiTrend/fig1.html, based on data collected by researchers at Washington University in St. Louis, MO., http://capita.wustl.edu/CAPITA/CapitaReports/USVisiTrend/usvstrd0.html.

16. U.S. Environmental Protection Agency, *National Air Quality and Emission Trends Report: 1999*, chapter 6, Visibility, www.epa.gov/oar/aqtrnd99/PDF%20Files/Chapter6.pdf.

17. BUNN, H.J., DINSDALE, D., SMITH, T., & GRIGG, J. "Ultrafine particles in alveolar macrophages from normal children." *Thorax* 2001; 56; 932–93422. Children aged 3 months to 16 years. No respiratory symptoms; Nonbronchoscopic BAL procedure before elective surgery. Data from Leicester, UK; proximity of home to busy

main road or residential street noted. All children's AM contained ultrafine carbonaceous particles (< 0.1 microns). Significantly more found in children who lived close to a busy traffic road. EM picture of carbonaceous ultrafine particles within a phagosome of an alveolar macrophage from a child aged 3 months. 10% of AM contained particles in AMs of children on busy roads, against 3.
BUNN, H.J., DINSDALE, D., SMITH, T., & GRIGG, J. Ultrafine particles in alveolar macrophages from normal children *Thorax* 2001; 56; 932–93422 children aged 3 months to 16 years. No respiratory symptoms; Nonbronchoscopic BAL procedure before elective surgery. Data from Leicester, UK; proximity of home to busy main road or residential street noted. All children's AM contained ultrafine carbonaceous particles (< 0.1 microns). Significantly more found in children who lived close to a busy traffic road. EM picture of carbonaceous ultrafine particles within a phagosome of an alveolar macrophage from a child aged 3 months. 10% of AM contained particles in AMs of children on busy roads, against 3.2% in children who lived on quiet roads.

18. KLEEMAN, M.J., SCHAUER, J.J., & CASS, G.R. "Size and Composition Distribution of fine particulate matter emitted from motor vehicles." *Environ Science & Technol:* Vol: 34; pgs. 132–1142:2000. Dilution source sampling system used to measure size-distributed chemical composition of fine particle emissions. It is impressive that from gasoline cars with and without catalytic converters, and from diesel engines, the size fraction is very constantly between 0.10 and 1.0 microns. Notes and observations by Bagley et al (*HEI Technical Report No 76* of 1996) that a 1991 diesel Cummins engine delivered lower overall particle mass emissions, but 15–35 times the number of particles as a 1988 Cummins engine (ultrafine particles made up the difference). For gasoline vehicles under various conditions, peaks of particle distributions seem to be between 0.1 and 0.2 microns, and the same is true of medium duty diesel vehicles. Sulfate particles seem to be a bit larger, peaking at about 0.9 microns. Interesting information.

19. HAUSER, R., GODLESKI, J.L., HATCH, V., & CHRISTIANI, D.C. "Ultrafine particles in human lung macrophages." *Arch Environ Health* 56; 150–156: 2001. 14 healthy current nonsmokers, of whom 11 were utility workers; 3 non-maintenance employees of a university. Macrophages isolated from BAL fluid. EM used to count particles within macrophages, and these were found in all subjects. Average was between 34 to 231 ultrafine particles per cubic microgram of cell cytoplasm. Numbers were associated with FEV1 as percent of predicted. Authors note: "The demonstration of ultrafine particles in all 14 subjects independent of occupational exposure, suggests that there is environmental exposure to ultrafine particles." Illustration of intracellular ultrafines ranging in size from 6–60 nm; these are compared to cultured macrophages which contain no particles. 4 of the subjects were welders, but they did not have more particles than the nonwelders.

20. Karcher, B., R.P. Turco, F. Yu, M.Yu, M.Y. Danilin, D.K. Weisenstein, R.C. Miake-Lye, and R. Busen. "A Unified Model for Ultrafine Aircraft Particle Emissions." *J. of Geophy. Res.*, 105, D24, pp. 29,379–29,386: 2002. Also, Material Safety Data Sheet, Chevron Jet Fuel, http://library.cbest.chevron.com/lubes/chevmsdsv9.nsf/db12c751d5603b418825681e007cddb3/0af8b9b4d5605ecf8825652c005f0c6f?OpenDocument.

21. *Ward's Motor Vehicle Facts and Figures 2001*, "Travel Trends, Vehicle Miles of Travel and Fuel Consumption." pp. 70–71.

22. BRAUER, M., AVILA-CASADO, C., FORTOUL, T.I., VEDAL, S., STEVENS, B., & CHURG, A. "Air pollution and retained particles in the lung." *Environ Health Perspect* 2001, 109; 1039–1043. Comparison between 11 autopsy lungs in never smoking women in Mexico City, and 11 control residents of Vancouver, BC. Average PM_{10} levels given as 66 micrograms/m3 in Mexico City and 14 micrograms/m3 in Vancouver. Particles counted on electron microscopy. Total retained particles were 2,055 x 10^6 particles/gram of dried lung from Mexico City lungs and 279 x 10^6 particles/g dry lung from Vancouver residents. Lungs from Mexico City contained numerous chain-aggregated, masses of ultrafine carbonaceous spheres, some of which contained sulfur and aggregates of ultrafine aluminum silicate. These constituted 25% of the total particles in the Mexico City lungs but were only rarely seen in the Vancouver lungs.

23. . U.S. Environmental Protection Agency, *Air Quality Criteria for Particulate Matter*, Volume I, p. 2–23 (March 2001).

24. U.S. Environmental Protection Agency, *Air Quality Criteria for Particulate Matter*, Volume I, p. 2–35 (March 2001).

25. Araujo, J. A. et. al. Ambient Particulate Pollutants in the Ultrafine Range Promote Early Atherosclerosis and Systemic Oxidative Stress. Circulation Research. 102(5):589-596, March 14, 2008.
Air pollution is associated with significant adverse health effects, including increased cardiovascular morbidity and mortality. Exposure to particulate matter with an aerodynamic diameter of <2.5 μ/m (PM2.5) increases ischemic cardiovascular events and promotes atherosclerosis. Moreover, there is increasing evidence that the smallest pollutant particles pose the greatest danger because of their high content of organic chemicals and prooxidative potential. To test this hypothesis, we compared the proatherogenic effects of ambient particles of <0.18 μ/m (ultrafine particles) with particles of <2.5 μ/m in genetically susceptible (apolipoprotein E-deficient) mice. These animals were exposed to concentrated ultrafine particles, concentrated particles of <2.5 μ/m, or filtered air in a mobile animal facility close to a Los Angeles freeway. Ultrafine particle-exposed mice exhibited significantly larger early atherosclerotic lesions than mice exposed to PM2.5 or filtered air. Exposure to ultrafine particles also resulted in an inhibition of the antiinflammatory capacity of plasma high-density lipoprotein and greater systemic oxidative stress as evidenced by a significant increase in hepatic malondialdehyde levels and upregulation of Nrf2-regulated antioxidant genes. The investigators concluded that "ultrafine particles concentrate the proatherogenic effects of ambient PM and may constitute a significant cardiovascular risk factor."

26. A. Nemmar, A. et. al. Passage of Inhaled Particles Into the Blood Circulation in Humans. Circulation. 2002;105:411-414.
To assess the mechanism by which particles cause increased cardiovascular morbidity and mortality, investigators recruited five healthy volunteers to inhale "Technegas," an aerosol consisting mainly of ultrafine 99mTechnetium-labeled carbon particles (100 nm). Radioactivity was detected in blood at 1 minute, reached a maximum between 10 and 20 minutes, and remained at this level up to 60 minutes. Thin layer chromatography of blood showed that in addition to a species corresponding to oxidized 99mTc, ie, pertechnetate, there was also a species corresponding to particle-bound 99mTc. Gamma camera images showed substantial radioactivity over the liver and other areas of the body. The investigators concluded that inhaled 99mTc-labeled ultrafine carbon particles pass rapidly into the systemic circulation, and this process could account for the well-established, but poorly understood, extrapulmonary effects of air pollution.

27. BRAUER, M., AVILA-CASADO, C., FORTOUL, T.I., VEDAL, S., STEVENS, B., & CHURG, A. "Air pollution and retained particles in the lung." *Environ Health Perspect* 2001, 109; 1039–1043. Comparison between 11 autopsy lungs in never smoking women in Mexico City, and 11 control residents of Vancouver, BC. Average PM_{10} levels given as 66 micrograms/m3 in Mexico City and 14 micrograms/m3 in Vancouver. Particles counted on electron microscopy. Total retained particles were 2,055 x 10^6 particles/gram of dried lung from Mexico City lungs and 279 x 10^6 particles/g dry lung from Vancouver residents. Lungs from Mexico City contained numerous chain-aggregated, masses of ultrafine carbonaceous spheres, some of which contained sulfur and aggregates of ultrafine aluminum silicate. These constituted 25% of the total particles in the Mexico City lungs but were only rarely seen in the Vancouver lungs.

28. SEATON, A., SOUTAR, A., CRAWFORD, V., ELTON, R., McNERLAN, S., CHERRIE, J., WATT, M., AGIUS, R., & STOUT, R. "Particulate air pollution and the blood." *Thorax* 54; 1027–1032: 1999. 112 individuals over the age of 60 in Edinburgh and Belfast provided blood samples over 18 months-108 provided the maximum of 12 samples. Estimated personal exposures to PM10 over the previous three days showed negative correlations with Hb concentration, PCV, and red blood cell count, platelets, and Factor VII levels. Rise of C-reactive protein also documented. Suggest that inhalation of PM_{10} may produce a sequestration of red cells in the circulation. Possible changes in red cell adhesiveness. Suggest that these changes may be linked to cardiovascular effects. PM_{10} levels in Belfast reached 80 micrograms/m3 in Belfast on two occasions, but averaged about 25–30. In Edinburgh, levels were lower as no peak exceeded 60 micrograms/m3, and the annual average was nearer 15–20 micrograms/m3. Possible occurrence of hemodilution was excluded by measurements of albumin in the blood which showed no dilution had occurred. Rise of C-reactive protein may indicate that an inflammatory response has occurred, although no increase in white cells was noted (NOTE - prevalence of banded neutrophils not measured). Link blood changes to possible ischemic damage in those with vulnerable coronary circulations.

29. PEKKANEN, J., TIMONEN, K.L., TITTANEN, P., MIRME, A., RUUSKANEN, J., & VANNINEN, E. "Daily variations of particulate air pollution and ST-T depressions in subjects with stable coronary heart disease. The Finnish ULTRA Study." *Am J Respir Crit Care Med* 161: A24: 2000. 23 women and 24 men aged 54–83 with stable coronary heart disease studied with biweekly clinic visits for 6 months. Ambulatory ECG recorded on these visits. PM2.5 and ultrafines recorded each day. Exercise test done and was more indicative of change. 94 ST-T depressions

observed. There was a consistent association between either ultrafine particles or $PM_{2.5}$ with an increased risk of ST-T depression.

30. DEVLIN, R.B., CASCIO, W., KEHRL, H., & GHIO, A. "Changes in heart rate variability in young and elderly humans exposed to concentrated ambient air particles." *Am J Respir Crit Care Med* 161: A239: 2000. 14 young (18–35) and 14 elderly (65–80) volunteers exposed to concentrated Chapel Hill particles for 2 hours. Changes in time and frequency domains measured. No changes in young subjects, but elderly group developed significant decrements in both time and frequency domains immediately after exposure. Changes persisted for at least 24 hours.

31. VAN EEDEN, S.F., TAN, W.C., SUWA, T., MUKAE, H., TERASHIMA, T., FUJII, T., QUI, D., VINCENT, R., & HOGG, J.C. "Cytokines involved in the systemic inflammatory response induced by exposure to particulate matter air pollutants (PM_{10})." *Am. J Respir Crit Care Med* 164; 826–830: 2001. Human alveolar macrophages (AM) harvested from bronchial lavage specimens (BAL) from a noninvolved segment or lobe of lungs resected for small peripheral tumours. These were more than 90% viable. All specimens tested for endotoxin contamination. Urban PM_{10} preparation (EHC 93) came from filters from Ottawa. Identified cytokines also measured in the blood of young army cadets exposed to Asian smoke in April 1998 in Singapore (see {10607}). AM Cells were incubated with residual oil fly ash (ROFA), ambient urban particles (EHC 93) inert carbon particles, and latex particles of different sizes (0.1, 1.0, and 10 microns) for 24 hours. The latex, inert carbon and ROFA particles all showed a similar maximum TNF (tumour necrosis factor alpha) response, whereas EHC 93 showed a greater maximum response that was similar to lipopolysaccharide (LPS). EHC 93 (Ottawa PM_{10}) also resulted in a broad spectrum of proinflammatory cytokines, (IL-6, MIP-1alpha, and GM-CSF), with no difference in the anti-inflammatory cytokine IL-10. Analysis of blood samples taken during the exposure of the army cadets in Singapore to the PM_{10} from the Asian fires of 1998, showed elevated levels of IL-1beta, IL-6, and GM-CSF during the exposure time. Authors conclude: "These results show that a range of different particles stimulate AM (human alveolar macrophages) to produce proinflammatory cytokines and these cytokines are also present in the blood of subjects during an episode of acute atmospheric air pollution. We postulate that these cytokines induced a systemic response that has an important role in the pathogenesis of the cardiopulmonary adverse health effects associated with atmospheric pollution."

32. DONALDSON, K., STONE, V., SEATON, A., & MACNEE, W. "Ambient particle inhalation and the cardiovascular system; potential mechanisms." *Environmental Health Perspectives* 109 (Supplement 4), 523–527: 2001. Notes that particles increase calcium flux on contact with macrophages. Oxidative stress is likely because of the very large particle surface area, and this can be augmented by oxidants generated by recruited inflammatory leukocytes. Notes that blood viscosity, fibrinogen, and C-reactive protein are elevated on PM_{10} exposure. Report on their study of elderly individuals in whom PM_{10} exposure resulted in an increase in C-reactive protein, which is an index of inflammation. Postulate that oxidative stress in the lungs may affect the cardiovascular system by increasing permeability, and by causing atheromatous plaque rupture or endothelial erosion; clotting factors might change and favor thrombus formation. Detailed discussion of blood viscosity and fibrinogen. Suggest that fibrinogen, CRP, and factor VII are part of the acute-phase response mediated by cytokines released during inflammatory reactions. Note that in a survey of 388 British men aged 50–69, the prevalence of coronary artery disease increased 1.5 fold for each doubling of C-reactive Protein level. They have also reported increased CRP in association with increases in urban PM_{10}. Useful and provocative review.

33. . This list was compiled by one of the most accomplished of fine particle researchers, Dr. Joel Schwartz of Harvard University and presented in testimony before the U.S. Congress. See Testimony of Joel Schwartz, http://www.senate.gov/~epw/105th/schwartz.htm.

34. Pope, C. Arden, III, Michael J. Thun, Mohan M. Namboodiri, Douglas W. Dockery, John S. Evans, Frank E. Speizer, and Clark W. Health, Jr. "Particulate Air Pollution as a Predictor of Mortality in a Prospective Study of U.S. Adults." *Am. J Respir Crit Care Med* 151, 669–74: 1995.

35. Dockery, Douglas W., C. Arden Pope III, Xiping Xu, et al., "An Association Between Air Pollution and Mortality in Six U.S. Cities." *New England Journal of Medicine* Vol 329, 1753–59: 1993.

36. PETERS, A., DOCKERY, D.W., MULLER, J.E., & MITTLEMAN, M.A. "Increased particulate air pollution and the triggering of myocardial infarction." *Circulation* 2001; 103; 2810–2815. 772 patients with MI in Greater Boston interviewed between Jan 95 and May 96. Hourly concentrations of $PM_{2.5}$, carbon black, and gaseous air pollutants were measured. Case-crossover approach used. For each subject, one case period was matched to 3 control periods exactly 24 hours apart. Conditional logistic regression analyses carried out, with Odds Ratios for a change in air pollution from the 5th to the 95th percentile calculated. Mean age 61.6 years; 164 were under age 50,

365 between 50–69; and 243 over 70 years of age. 63% were male. 31% had a prior myocardial infarction. 72% were “ever smokers” and 32% current smokers. Odds ratios for MI and $PM_{2.5}$ were above 1.2 for up to 2 hours before onset; and about 1.1 for 3–5 hours before. Using both the values of $PM_{2.5}$ in the previous 2 hour period, and a delayed response associated with a 24-hour average exposure 1 day before the symptoms, an odds ratio of 1.48 was associated with an increase of 25 micrograms/m3 in $PM_{2.5}$ during the 2 hour period, and of 1.69 for an increase of 20 micrograms/m3 $PM_{2.5}$ in the 24 hour period 1 day before the onset. Remarkable to see such a strong effect in what is a relatively small sample.

37. POPE, C.A. III., BURNETT, R.T., THUN, M.J., CALLE, E.E., KREWSKI, D., ITO, K., & THURSTON, G.D. “Lung Cancer, Cardiopulmonary mortality, and long-term exposure to fine particulate air pollution.” *JAMA* 2002; 287; 1132–1141. American Cancer Society cohort recruited in 1982. Analysis of over 500,000 people in an average of 51 metropolitan districts. Interesting data showing reductions in $PM_{2.5}$ from 1979–1983 and from 1999 to 2000, values ranging from 10 to 30 in the first period, and from 5 to 20 in the second. Nonparametric smoothed response functions shown for the three categories of diagnosis; conclude that for a 10 microgram/m3 change in PM_{10}, all cause mortality increased by 4%; cardiopulmonary mortality increased by 6%, and lung cancer mortality increased by 8%. 95% confidence levels of all indices of RR were above 1.0. Coarse particle fraction and TSP not consistently associated with mortality. Other pollutants considered were sulfate, sulfur dioxide, nitrogen dioxide, carbon monoxide, and ozone. Numbers of metropolitan areas that could be considered varied with the different pollutants. Cox proportional hazards model with inclusion of a metropolitan-based random effects component in a two stage analysis. The continuous smoking variables included nine different indices (such as “current smokers years of smoking squared” and eight others). Controls also devised for educational level and occupational exposures. A 2 dimensional term was inserted to account for spatial trends. Higher regressions were noted in men than in women, and lower educational status was associated with higher risks. Risks in never smokers were also generally higher than in former or current smokers. Authors conclude: “The findings of this study provide the strongest evidence to date that long-term exposure to fine particulate air pollution common to many metropolitan areas is an important risk factor for cardiopulmonary mortality.”

38. SAMET, J.M., ZEGER, S.L., DOMINICI, F., CURRIERO, F., COURSAC, I., DOCKERY, D.W., SCHWARTZ, J., & ZANOBETTI, A. “The National morbidity, mortality, and air pollution study: Part II Morbidity and mortality from air pollution in the United States Health Effects Institute.” *Research Report: Number 94*, Part II; June 2000 p. 82. 90 cities in different regions of the US, covering all areas. Daily PM_{10} values given for 1987 to 1994. Also O_3, SO_2, NO_2 and CO. Univariate analysis showed highest values for SO_2, with CO second, NO_2 third, and PM_{10} fourth. Distributed lag models give higher values, and authors note that the effects of pollution do not reach 0 until a lag of 5 days has occurred. Authors conclude: “Overall, this study provides strong evidence of association between PM_{10} levels and exacerbation of chronic heart and lung disease sufficiently severe to warrant hospitalization.” Effect of PM_{10} on mortality generally higher in Northeast, industrial Midwest, and southern California than in other regions.
This study is summarized in the EPA criteria document.

39. SAMOLI, E., SCHWARTZ, J., WOJTYNIAK, B., TOULOUMI, G., SPIX, C., BALDUCCI, F., MEDINA, S., ROSSI, G., SUNYER, J., BACHAROVA, L., ANDERSON, H.R., & KATSOUYANNI, K. “Investigating regional differences in short-term effects of air pollution on daily mortality in the APHEA project; a sensitivity analysis for controlling long-term trends and seasonality.” *Environmental Health Perspect* 109; 349–353: (2001).

40. Western European cities: Athens, Barcelona, Cologne, London, Lyon, Milan, and Paris; and five central eastern European cities Bratislava: Cracow, Lodz, Poznan, and Wroclaw—as previously analyzed in the APHEA study. Days with Black smoke > 200 micrograms/m3 were excluded. Poisson regression methods. Loess smoothing regression smoother, which is a generalization of a weighted moving average. Window chosen that minimized Akaike’s Information Criterion. Black smoke and SO_2 were the pollutants analyzed. Increase in BS of 50 micrograms/m3 associated with 2.2% increase in mortality when analysis restricted to days with BS less than 200, and 3.1% increase if restricted to days with BS less than 150 micrograms/m3. SO_2 effect for similar increase was 5.0% and 5.6% respectively. This increase occurred only in the eastern European cities. Authors conclude that “part of the heterogeneity in the estimates of air pollution effects between western and central-eastern cities reported in previous publications was caused by the statistical approach used and the inclusion of days with pollutant levels above 150 micrograms/m3.”

41. BURNETT, R.T., BROOK, J., DANN, T., DELOCIA, C., PHILIPS, O., CAKMAK, S., VINCENT, R., GOLDBERG, M.S., & KREWSKI, D. “Association between particulate- and gas-phase components of urban air pollution and daily mortality in eight Canadian Cities.” *Inhalation Toxicology*, 12 (Supplement 4): 15–39, 2000.

Analysis of time series data from Montreal, Ottawa-Hull, Toronto, Windsor, Winnipeg, Edmonton, Calgary, and Vancouver. Important feature is detailed compositional analysis of the PM_{10} collected in each city. $PM_{2.5}$ a stronger predictor of mortality that $PM_{10-2.5}$. Size-fractionated particulate mass explained 28% of the total health effect of the mixture, with the remaining effects being accounted for by the gases. Carbon represented half the mass of the particulate matter. 47 elemental concentrations reported. Good discussion of statistical issues and notes that slightly stronger associations resulted from co-adjustment method compared to pre-adjustment of both mortality and air pollution data. Correlations between pollutants are given. Separate risk relationships not given for different cities, but pollutant values are given for each city. Sulfur showed the highest correlation with fine mass, with Pb, Si, FE, K, Zn, Mn, P and SE modestly correlated with $PM_{2.5}$. Both particulates and gases associated with mortality.

42. Wollf G.T., In Response to the PM Debate, Regulation 20:1, 1997. The author, a General Motors Corporation scientist chaired the U.S. Environmental Protection Agency's Clean Air Science Advisory Committee.

43. (1)Schwartz J. "Is there harvesting in the association of airborne particles with daily deaths and hospital admissions?" *Epidemiology* 2001; 12:55–61. (2) Zanobetti A, Schwartz J, Samoli E et al. "The temporal pattern of mortality responses to air pollution: a multi-city assessment of mortality displacement." *Epidemiology* 2002; 13:87–93. (3) Zeger SL, Dominici F, Samet J. "Harvesting-resistant estimates of air pollution effects on mortality." *Epidemiology* 1999; 10:171–175. (4) Schwartz J. "Harvesting and long term exposure effects in the relation between air pollution and mortality." *Am J Epidemiol* 2000; 151:440–48.

44. . BOBAK, M., & LEON, D.A. "The Effect of Air Pollution on Infant Mortality appears specific for respiratory causes in the postnatal period." *Epidemiology* 1999; 10; 666–670. All births registered in the Czech Republic between 1989 and 1991. For each infant death, 20 controls randomly selected from infants of the same sex born on the same day and alive when the case died. Exposure assigned as the arithmetic mean of all 24-hour air pollution measurements in the district of residence of each case and control for the period between the birth and death of the index case. 2,494 infant deaths. Respiratory deaths analyzed. Risk Rate ratios for a 50 microgram/m3 increase in particles = 1.95; 1.74 for SO_2; and 1.66 for NO_2. Only particles showed a consistent association when all pollutants entered in one model. No evidence of an association between any pollutant and mortality from any other cause. Conclude: "the effects of air pollution on infant mortality are specific for respiratory causes in the postneonatal period, are independent of socioeconomic factors, and are not mediated by birth weight or gestational age."
The crude RR for SO_2 was 2.16; adjusted for socioeconomic was 1.94; adjusted for perinatal factors was 2.09; and adjusted for all covariates was 1.87. These values were higher than those for TSP. Similar RRs for NO_2 were 1.55; 1.71;; 1.60; & 1.78. Respiratory deaths numbered about 133. Important observations.

45. CONCEICAO, G.M.S., MIRAGLIA, S.G.E.K., KISHI, H.S., SALDIVA, P.H.N., & SINGER, J.M. "Air Pollution and child mortality: a time-series study in Sao Paulo, Brazil." *Environ Health Perspect* 109 (suppl 3):347–350: 2001. Mortality from respiratory causes of children under the age of 5. Daily levels of SO_2, CO, PM_{10}, and ozone used. Temperature and humidity data included. All three pollutants were associated with death rate, and the observed associations were dose dependent and "quite evident after a short period of exposure (2 days)." The estimated proportions of respiratory deaths attributed to CO, SO_2, and PM_{10} when considered individually, were around 15%, 13%, and 7% respectively. Reliability of death certification process is described.

46. CHAY, K.Y., & GREENSTONE, M. "The impact of air pollution on infant mortality: evidence from geographic variation in pollution shocks induced by a recession."
National Bureau of Economic Research, 1050 Massachusetts Avenue, Cambridge, MA 02138. Manuscript of about 40 typewritten pages with figures and tables sent to Dr. David Bates by Dr. Michael Brauer. It had been sent to him by his brother, who is an economist in Washington, DC. In 1980–1982, there was an economic recession, leading to substantial reductions in particulate pollution in some regions of the US. In this paper, neonatal mortality was assessed in relation to the reductions in TSP which occurred (regions being classified as those which had large reductions, medium reductions and small reductions). A large bank of data was analyzed, and it was shown that average income did not change over this period; that other variables that might have an influence on neonatal mortality did not change; and that mean birth weight and Apgar indices did not alter. It is shown that there was a reduction in infant deaths (within the period of one day and one month of birth) during the period of lower pollution. This amounted to 4–8 fewer infant deaths per 100,000 live births at the county level, for a 1 microgram/m3 reduction in TSP [note that in the Abstract and in the MS except on page 41 where it is correctly written, this is miss-spelled mg/m3"]. The authors, being economists, note that this corresponds to an elasticity of 0.35–0.45. In a sub analysis of Pennsylvania, where the drop in TSP was generally large, they note: "In all of Pennsylvania, mean TSP's pollution was relatively stable at about 70–74 units from 1978–1980 and then declined precipitously to about 53 units by 1982–83. At the same time, in 1978–80 infant deaths within one year of birth attributable to "internal" causes (eg.

respiratory and cardiopulmonary deaths) were stable and occurred at the rate of 1315–1380 per 1000,000 live births. But from 1980–82, the internal infant mortality rate declined from 1315 to 1131, and remained at this lower level in 1983–84. While not controlling for all changes that may have occurred in the absence of the pollution decline, these numbers imply that a 1 microgram/m3 decline in TSP's may result in about 10–11 fewer infant deaths per 100,000 births, which is an elasticity of 0.5–0.6." Also: "Based on our quasi-experimental research design, we find a significant impact of pollution reductions on decreases in infant mortality rates at the county level, with a 1 microgram/m3 decline in suspended particulates associated with about 4–8 fewer infant deaths per 100,000 live births (a 0.35–0.45 elasticity). The results are driven almost entirely by fewer deaths occurring within one month and one day of birth, suggesting that pollution exposure adversely impacts the fetus before birth." The paper contains figures that illustrate the changes, and many tables with a complex array of data from 1978 to 1984 addressing other factors that might have influenced the results. The data are convincing and the conclusions seem valid. The paper is written in a somewhat roundabout way (perhaps characteristic of the style of economists?) but the complex data are well presented and discussed.

47. Dockery, D. W., and C. A. Pope III. "Acute Respiratory Effects of Particulate Air Pollution," *Annual Review Public Health, 1994*, vol 15, 1994, 107–32.

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49 Ware J, Ferris Jr. B, Dockery D, Spengler J, Stram D, Speizer F. Effect of ambient sulfur oxides and suspended particles on respiratory health of preadolescent children. Am Rev Respir Dis 1986;133:834–42.

50 Dockery DW, Speizer FE, Stram DO, Ware JH, Spengler JD. Effects of inhalable particles on respiratory health of children. Am Rev Respir Dis 1989;139:587–94.

40Bates DV, Sizto R. Air pollution and hospital admissions in southern Ontario: The acid summer haze effect. Environ Res 1987;43:317–31.

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53. C. Arden Pope III, "Respiratory Disease Associated with Community Air Pollution and a Steel Mill, Utah Valley." *American Journal of Public Health*, Vol. 79 (May 1989), pgs. 623–628.

54. Wickramasinghe. 2001. Gendered Sights and Health Issues in the Paradigm of Biofuel in Sri Lanka Energia News 4: 4 14-16. Also, Manuel, J. The Quest for Fire: Hazards of a Daily Struggle. Env. Hlth. Persp. Volume 111, Number 1, January 2003.

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62 O'Dempsey, T., McArdle, T. F., Morris, J., Lloyd-Evans, N., Baldeh, I., Lawrence, B. E.,Secka, O. & Greenwood, B. M. Int. J. Epidemiol.1996 ; 25, 885-893.

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65. Pandey, M. R., Boleij, J. S., Smith, K. R. & Wafula, E. M. Lancet 1989; 1: 427-429.

66. Boy E., Bruce, N. & Delgado, H. Birth weight and exposure to kitchen wood smoke during pregnancy in rural Guatemala. Environ Health Perspect. 2002 Jan;110(1):109-14.

To assess whether domestic use of wood fuel is associated with reduced birth weight, independent of key maternal, social, and economic confounding factors, investigators studied 1,717 women and newborn children in rural and urban communities in rural Guatemala. They identified subjects through home births reported by traditional birth attendants in six rural districts (n = 572) and all public hospital births in Quetzaltenango city during the study period (n = 1,145). All were seen within 72 hr of delivery, and data were collected on the type of household fuel used, fire type, and socioeconomic and other confounding factors. Smoking among women in the study community was negligible. Children born to mothers habitually cooking on open fires (n = 861) had the lowest mean birth weight of 2,819 g [95% confidence interval (CI), 2,790-2,848]; those using a chimney stove (n = 490) had an intermediate mean of 2,863 g (95% CI, 2,824-2,902); and those using the cleanest fuels (electricity or gas, n = 365) had the highest mean of 2,948 g (95% CI, 2,898-2,998) (p< 0.0001). The percentage of low birth weights (< 500 g) in these three groups was 19.9% (open fire), 16.8% (chimney stove), and 16.0% (electricity/gas), (trend (p = 0.08). Confounding factors were strongly associated with fuel type, but after adjustment wood users still had a birth weight 63 g lower (p = 0.05; 95% CI, 0.4-126). This is the first report of an association between biofuel use and reduced birth weight in a human population. Although there is potential for residual confounding despite adjustment, the better-documented evidence on passive smoking and a feasible mechanism through carbon monoxide exposure suggest this association may be real. Because two-thirds of households in developing countries still rely on biofuels and women of childbearing age perform most cooking tasks, the attributable risk arising from this association, if confirmed, would be substantial.

67. Rinne, S.T. . et. al. Use of Biomass Fuel Is Associated with Infant Mortality and Child Health in Trend Analysis Am. J. Trop. Med. Hyg., 76(3), 2007, pp. 585-59.
Biomass fuel used for cooking results in widespread exposure to indoor air pollution (IAP), affecting nearly 3 billion people throughout the world. Few studies, however, have

tested for an exposure–response relationship between biomass fuel and health outcomes. To assess the relationship between biomass fuel, infant mortality, and children's respiratory symptoms. Eighty households in a rural community in Ecuador were selected based on their use of biomass fuel and questioned regarding a history of infant mortality and children's respiratory symptoms. Carbon monoxide (CO) and particulate matter (PM) were measured in a subset of these homes to confirm the relationship between biomass fuel use and IAP. Results showed a significant trend for higher infant mortality among households that cooked with a greater proportion of biomass fuel ($P = 0.008$). Similar trends were noted for history of cough ($P = 0.02$) and earache ($P < 0.001$) among children living in these households.

68. Kumar, S. & Mehra, S. ARI And Indoor Air Pollution: Its Burden And Correlation. The Internet Journal of Pulmonary Medicine. 2007. Volume 8 Number 2. http://www.ispub.com/ostia/index.php?xmlFilePath=journals/ijpm/vol8n2/ari.xml#r19

69. World Health Organization, Indoor air pollution in developing countries: a major environmental and public health challenge. Bulletin of the World Health Organization 2000.

70. Bruce N., Perez-Padilla R., & Albalak, R. Indoor air pollution in developing countries: a major environmental and public health challenge. Bull World Health Organ. 2000;78(9):1078-92.
Around 50% of people, almost all in developing countries, rely on coal and biomass in the form of wood, dung and crop residues for domestic energy. These materials are typically burnt in simple stoves with very incomplete combustion. Consequently, women and young children are exposed to high levels of indoor air pollution every day. There is consistent evidence that indoor air pollution increases the risk of chronic obstructive pulmonary disease and of acute respiratory infections in childhood, the most important cause of death among children under 5 years of age in developing countries. Evidence also exists of associations with low birth weight, increased infant and perinatal mortality, pulmonary tuberculosis, nasopharyngeal and laryngeal cancer, cataract, and, specifically in respect of the use of coal, with lung cancer. Conflicting evidence exists with regard to asthma. All studies are observational and very few have measured exposure directly, while a substantial proportion have not dealt with confounding. As a result, risk estimates are poorly quantified and may be biased. Exposure to indoor air pollution may be responsible for nearly 2 million excess deaths in developing countries and for some 4% of the global burden of disease. Indoor air pollution is a major global public health threat requiring greatly increased efforts in the areas of research and policy-making. Research on its health effects should be strengthened, particularly in relation to tuberculosis and acute lower respiratory infections. A more systematic approach to the development and evaluation of interventions is desirable, with clearer recognition of the interrelationships between poverty and dependence on polluting fuels.

71 Penner, J.E. Towards the development of a global inventory for black carbon emissions. International conference on carbonaceous particles in the atmosphere; 3-5 Apr 1991, Vienna, Austria.
The authors developed a global inventory for black carbon (BC) emi measured ambient concentration ratios of black carbon and SO_2 at locations throughout the world. They extended the data base for black carbon by using "smoke" measurements at a variety of monitoring sites and a calibration factor for black carbon derived herein from ambient smoke and BC

measurements. They demonstrate that BC to SO_2 ratios are well correlated at most sites and that distinct ratios of BC to SO_2 apply to source areas from economically distinct regions. However, within any one economic region, the ratio of BC to SO_2 appears to be relatively constant. These facts are used to construct a global inventory of black carbon emissions by using previously published inventories for the emissions of sulfur from fossil fuel use. The derived inventory totals nearly 24 Tg C/yr. This inventory is compared to a crude inventory based on emission factors and published fuel use statistics for wood and bagasse burning, diesel fuel, and domestic and commercial coal use. The combined emissions from wood, diesel, and coal can explain more than 75% of the total global emissions and usually are within a factor of two of the derived regional emissions from the BC/S ratio method. The black carbon inventory, totaling nearly 24 Tg C/yr, is used in the Lawrence Livermore National Laboratory global chemistry/climate model to simulate the world-wide distribution of black carbon. The predicted concentrations are compared with available measurements from throughout the world. This comparison supports the magnitude of the inventory which the authors have derived to within a factor of two, although significant uncertainties with respect to the treatment of scavenging and deposition in the model remain.

[72] A. M. Stamatelos A review of the effect of particulate traps on the efficiency of vehicle diesel engines. Energy Conversion and Management, V. 38, Issue 1, Jan. 1997, Pages 83-99.
Particulate traps are becoming more widely used on city buses, some delivery trucks and fork lift trucks. The possible use of diesel particulate traps will lead to a fuel consumption penalty imposed on the baseline engine that is due to the trap back pressure as well as to the energy requirements of the regeneration technique adopted to incinerate the collected soot at will. The combined effect of trap back pressure imposed on the engine and additional energy required for trap regeneration on the overall efficiency of the diesel power plant is examined in this paper. This effect varies according to engine type, trap type and size, regeneration system used, and the vehicle driving mode. Because of the strong interaction among the above parameters, optimization of trap systems on efficiency grounds is complicated. This complexity is even more pronounced in the case of diesel-powered passenger cars, where the full exploitation of their efficiency advantage over gasoline-powered cars is constrained by the necessity of an optimized solution of the particulate emission problem. The main diesel particulate trap regeneration philosophies existing today are reviewed in terms of their effect on the total efficiency of the diesel power plant. This is done by means of representative examples, concerning systems which may be suitable for large-scale application. The conclusions indicate that the price that must be paid for environmental protection, in the case of diesel particulate control systems, may be substantially reduced by system design optimization.

[73] Cherng-Yuan Lin. Reduction of particulate matter and gaseous emission from marine diesel engines using a catalyzed particulate filter. Ocean Engineering, V. 29, Issue 11, Sep. 2002, Pages 1327–1341
Diesel engines are used widely as the power sources of coastal ships and international vessels primarily due to their high thermal efficiency, high fuel economy and durable performance. However, the gaseous and solid substances exhausted from diesel engines during the combustion process cause air pollution, in particular around harbor regions. In order to effectively reduce particulate matter and gaseous pollution emissions, a catalyzed particulate filter was equipped in the tail pipe of a marine diesel engine. The engine's performance and emission characteristics under various engine speeds and torques were measured using a computerized engine data control and acquisition system accompanied with an engine dynamometer. The effectiveness of installing a catalyzed particulate filter on the reduction of pollutant emissions was examined. The experimental results show that the exhaust gas temperature, carbon monoxide and smoke opacity were reduced significantly upon installation of the particulate filter. In particular, larger conversion of carbon monoxide to carbon dioxide—and thus larger CO_2 and lower CO emissions—were observed for the marine diesel engine equipped with a catalyzed particulate filter and operated at higher engine speeds. This is presumably due to enhancement of the catalytic oxidation reaction that results from an exhaust gas with stronger stirring motion passing through the filter. The absorption of partial heating energy from the exhaust gas by the physical structure of the particulate filter resulted in a reduction in the exhaust gas temperature. The particulate

matter could be burnt to a greater extent due to the effect of the catalyst coated on the surface of the particulate filter. Moreover, the fuel consumption rate was increased slightly while the excess oxygen emission was somewhat decreased with the particulate filter.

74 Petzold, A. et. al.In situ observations and model calculations of black carbon emission by aircraft at cruise altitude. Journal of Geophysical Research, Volume 104, Issue D18, p. 22171–22182. http://www.agu.org/journals/jgr. *Publication Date:* 00/1999
The exhaust aerosol of two aircraft at cruise was extensively characterized in the size range from 0.003 to 2 _m for plume ages <=2 s. The black carbon (BC) exhaust aerosol of an older technology engine (Rolls-Royce/Snecma M45H Mk501) consisted of a primary BC mode with a modal diameter of 0.035 _m and a mode of coagulated BC particles with a peak near 0.15-0.16 _m in diameter. The total number density at the nozzle exit plane was 3(10^7 cm^-3 . In contrast, a modern technology engine (CFM International CFM56-3B1) emitted far smaller BC particles with a primary mode at 0.025 _m and a coagulated mode at 0.15 _m, as well as fewer particles by number with a concentration of 9(10^6 cm^-3 . The single-scattering albedo of the jet exhaust aerosol was 0.035+/-0.02 inside the plume, indicating a dominant contribution of ultrafine (D<0.1_m) BC particles to light extinction. Black carbon number emission indices EI(N) varied from 3.5(10^14 (CFM56-3B1) to 1.7(10^15 kg^-1 (M45H Mk501) with corresponding mass emission indices EI(BC) of 0.011 and 0.1 g kg^-1 . Previously reported corresponding values for a CF6-80C2A2 engine were 6(10^14 kg^-1 and 0.023 g kg^-1 , respectively. A comparison between EI(BC) values calculated by a new correlation method and measured data shows an excellent agreement, with deviations <10% at cruise conditions. By extending the EI(BC) calculation method to a globally operating aircraft fleet, a fleet-averaged emission index EI(BC)=0.038gkg^-1 is calculated.

75 Streets D.G. et. al. Black carbon emissions in China. Atmospheric Environment, Volume 35, Number 25, September 2001 , pp. 4281–4296(16). Emissions of BC in China are roughly one-fourth of global anthropogenic emissions. China's high rates of usage of coal and biofuels are primarily responsible for high BC emissions. Investigators calculate that BC emissions in China in 1995 were 1342 Gg, about 83% being generated by the residential combustion of coal and biofuels, and estimate that BC emissions could fall to 1224 Gg by 2020. This 9% decrease in BC emissions is contrasted with the expected increase of 50% in energy use; the reduction will be obtained because of a transition to more advanced technology, including greater use of coal briquettes in place of raw coal in cities and towns.

76 Timm, B. Air Quality Modeling and Health Benefits of a Woodstove Change-out Program, May 18, 2006, U.S. Environmental Protection Agency.

77 Timm, B. Air Quality Modeling and Health Benefits of a Woodstove Change-out Program, May 18, 2006, U.S. Environmental Protection Agency.