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Undergraduate

TINY BUT TOXIC: HOW INDUSTRIAL WASTE INFILTRATES THE BODY

Rachel Lew

During one of the most devastating episodes of air pollution in history, a 30-mile-wide cloud of smog settled on the city of London, incapacitating its residents. For five days, the smog was so thick that all transportation above ground was halted and Londoners were forced to walk the streets instead, dangerously exposing themselves to the pollution. The Great Smog of December 1952 was estimated to have caused 4,000 deaths within days. (Klein, 2012) Reflecting on the event fifty years later, a woman who lived through the Great Smog said, "It was the worst fog that I'd ever encountered. It had a yellow tinge and a strong, strong smell strongly of sulphur, because it was really pollution from coal fires that had built up. Even in daylight, it was a ghastly yellow colour" (Days of Toxic Darkness, 2002).

Ultra-fine particles not only exacerbate existing lung disease but also increase blood coagulability...clotted or thickened blood can easily clog major vessels.



Figure 1. The Great Smog of 1952 (London, England)

At the time, the smog was considered more of a visibility problem than a health hazard. Runners from Oxford and Cambridge University even persisted in their annual cross-country race, using track marshals to guide them through the yellow smog (Klein, 2012). Perhaps if they had been aware of the health effects associated with breathing polluted air, they would have decided to stay indoors that day. Exercising in polluted air has been proven to be particularly harmful not only to the throat and lungs, but also, surprisingly, to the heart (Pekkanen, 2002).

Upon inhaling the smog, Londoners in 1952 began choking and wheezing as toxic particles infiltrated and irritated their respiratory pathways. Weeks later, many found themselves afflicted with bronchitis, tuberculosis, and other

inflammatory pulmonary diseases. Worse, over the course of six months, more Londoners were plagued by pollution-caused cardiovascular disease, resulting in an estimated 8,000 additional deaths (Klein, 2012).

We now know that particulate matter air pollution, or PM, can damage both the pulmonary and cardiovascular systems of organisms, causing both short- and long-term health problems. In 2002, the number of PM-related deaths per year in the United States was about 60,000 (Verrier, 2002). Though this number only accounts for 2.4% of the total number of deaths in the US, by comparison, about the same number of people died from Alzheimer's disease that year (Kochanek, 2004).

Unfortunately, with the rise of industrialization, the problem of particulate air pollution has only intensified. In the 1900s, air pollution mainly consisted of emissions from coal combustion, such as sulfur oxides, nitrogen oxides, carbon monoxide, other organic compounds, and particulate matter made up of inorganic ash residues (Bituminous and

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Subbituminous Coal Combustion, 1998). Modern industrial processes have yielded even more forms of waste that are dispersed into the air. Diesel exhaust from heavy machinery, for example, contains DEP (diesel exhaust particles), which accumulate easily in the lungs and can hang in the air for long periods of time. Desertification in Mongolia and China due to industrial development on the land has given rise to ASD particles (Asian sand dusts), which are blown over as far as North America by wind currents (Takizawa, 2011).

DEP and ASD are examples of pollutant particles that affect the pulmonary system through airway irritation. Specifically, they heighten allergic symptoms; animals exposed in experiments to diesel exhaust exhibited enhanced airway hyperresponsiveness (AHR) and inflammation. Those exposed to ASD also showed inflammation in the mucous membrane of the nose. (Takizawa, 2011). In human children, greater

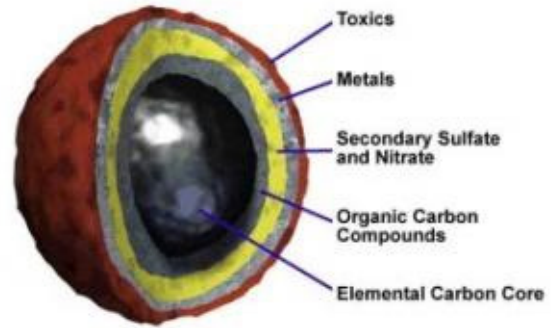


Figure 3. Composition of diesel exhaust particle (DEP) exposure to PM_{2.5}--pollutant particles with a diameter of 2.5 μm --NO₂, and soot is associated with increases in asthma symptoms (Gehring et al., 2010). Moreover, an eight-year study conducted in the Netherlands found that children who lived closer to roads with high traffic, and were thus exposed to higher levels of DEP and other traffic-related pollutants, were more likely to develop asthma during the first eight years of their lives (Gehring, 2010).

DEP and ASD are not the only particles contaminating ambient air, or air in its natural state. In general, PM is defined as “a mixture of combustive byproducts and resuspended crustal materials, as well as biological materials such as pollen, endotoxins, bacteria, and viruses” (Verrier, 2002). These particles are tiny; the largest are about 10 μm in diameter, about one-fifth the width of a human hair.

Some scientists believe that the smaller the PM, the more toxic it is to organisms. According to a 1995 article in

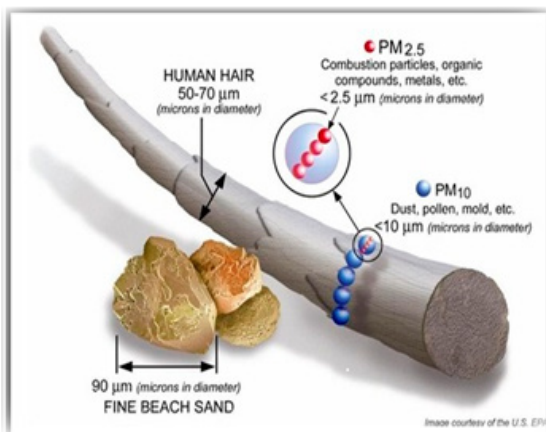
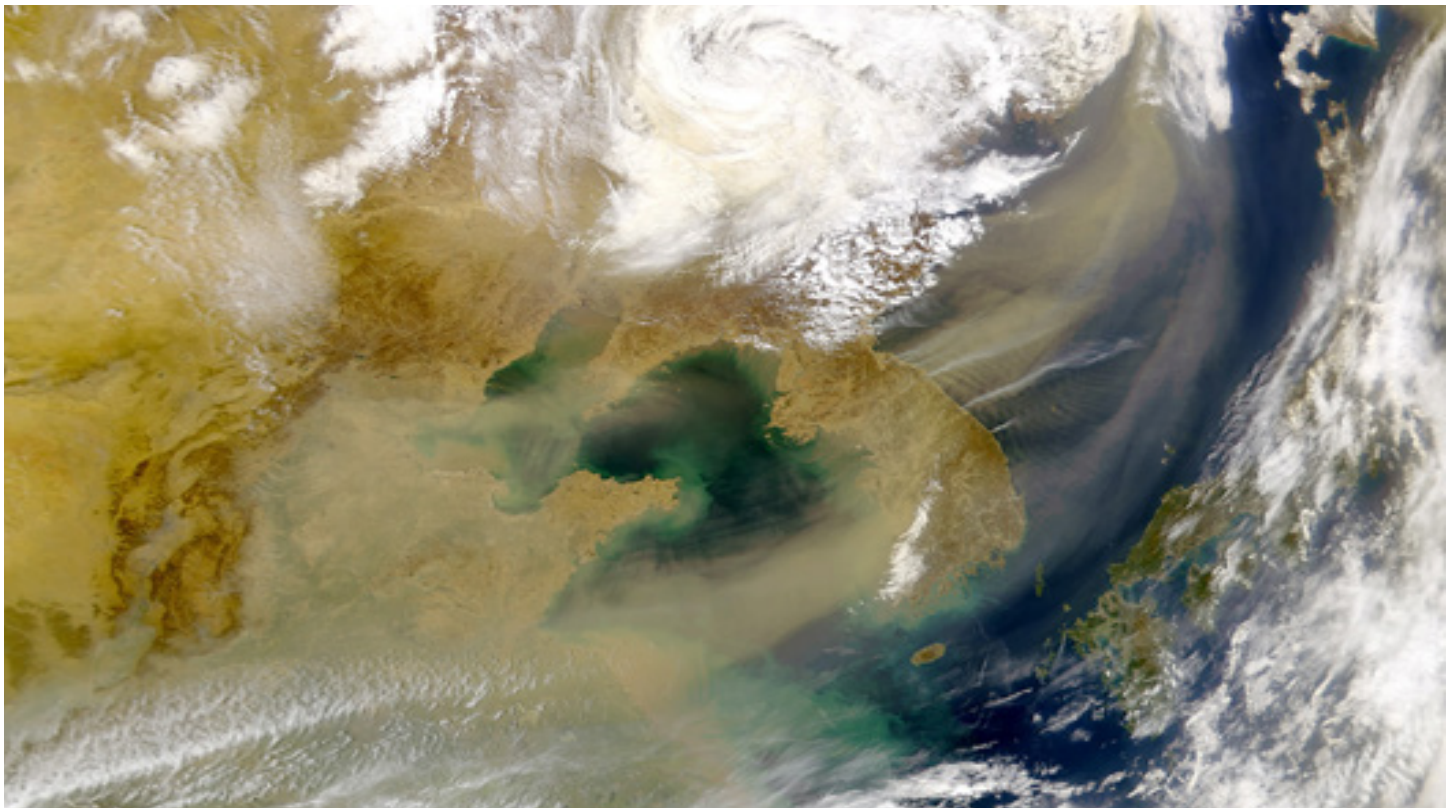


Figure 2. Relative size of particulate matter air pollution



Yellow cloud of Asian sand dusts (ASD) blows over from China

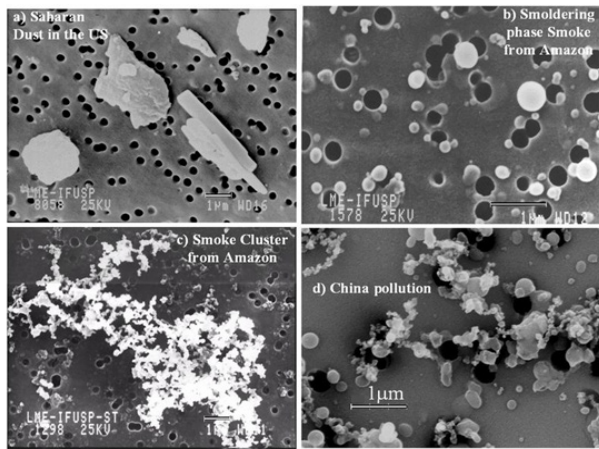


Figure 4. Various airborne pollutants under microscope

The Lancet by Seaton et al., soluble particles absorb water as they travel down the airway, grow in size, and can be easily rejected by the lung's defenses. However, ultra-fine particles penetrate these defenses and go on to cause inflammation in the alveoli of the lungs, "with release of mediators capable... of causing exacerbations of lung disease" (Seaton, 1995). These ultrafine particles are especially dangerous because they can enter systemic circulation. Then, as blood is circulated throughout the body, so are the ultrafine particles, spreading toxicity into areas outside of the lungs (Bhatnagar, 2004). The invasion of PM into circulating blood has been documented to cause constriction of arteries, cardiac arrhythmias, and myocardial infarction--the death of heart tissues as a result of inadequate oxygen supply (Verrier, 2002).

Interestingly, Seaton et al. also hypothesizes that these ultra-fine particles can transport chemicals on their surface, provoking an inflammatory reaction in the body. This low-grade inflammatory reaction would then cause an increase in "plasma viscosity, fibrinogen, factor VII, and plasminogen activator inhibitor" (Seaton, 1995). The protein fibrinogen aids in blood clotting, factor VII is a key initiator of blood coagulation, and plasminogen activator inhibitor inhibits the breakdown of blood clots. Therefore, ultra-fine particles not only exacerbate existing lung disease but also increase blood

In Beijing, for example, a citizen could check the Air Pollution Index one morning and leave the house knowing that he or she has a greater risk of incurring a heart attack that day.

coagulability. Corroborating this hypothesis, a study that exposed healthy human volunteers to CAPS (concentrated ambient air particles) saw results in which the volunteers' blood contained significantly more fibrinogen 18 hours after exposure (Ghio, 2000). If these hypotheses are correct, then PM inflicts cardiovascular disease indirectly: clotted or thickened blood can easily clog major vessels, and prevent blood flow to the heart--also known as ischaemic heart disease--and other vital organs. Lack of blood flow corresponds to lack of oxygen and nutrients; the resulting tissue death in these regions could manifest in a stroke or heart attack.

Currently, knowledge of the exact molecular mechanisms surrounding the effects of PM on the cardiovascular system remains limited. However, it is generally believed that this relationship exists. For one, the number of hospital visits for cardiovascular disease has been shown in many studies to be higher on days with higher levels of air pollution (Metzger, 2004; Pekkanen, 2002). Moreover, data collected on 500,000 people in the United States between 1979 and 2000 showed that "for every 10- $\mu\text{g}/\text{m}^3$ increase in fine particles (PM_{2.5}), all-cause mortality increased by 6% annually and cardiopulmonary mortality by 9%," a correlation which was also observed to a lesser extent with increases in density of coarser particles (Verrier, 2002). It is disturbing to consider that in highly polluted cities, pollution levels must be reported daily in the news to keep individuals aware of the hazards of going outdoors. In Beijing, for example, a citizen could check the Air Pollution Index one morning and leave the house knowing that he or she has a greater risk of incurring a heart attack that day (Chen, 2015).

In sum, exposure to air pollution can cause not only precipitous cardiopulmonary distress such as asthma attacks, heart attack, and stroke, but also chronic damage to the heart, lungs, and airway. Fortunately, however, it is possible to mitigate PM-related health risks. On the individual level, people living in heavily polluted areas can make lifestyle adjustments such as staying indoors when pollution levels are high. On the



Figure 5. Sunrise over a smog-covered Hong Kong

institutional level, it is important that governments make an effort to control common sources of pollution, since declines in city pollution levels have been shown to cause fewer deaths in the area. In Utah, for instance, a 13-month strike at a steel mill in Utah Valley--one of the most polluted cities in the US--caused PM10 concentrations to decrease and subsequently lower the city's mortality rate by 3%. Similarly, "[r]estrictions on the sulfur content of fuel oil in Hong Kong resulted in a 45% average reduction in SO2, and the average annual trend in deaths from all causes declined 2% and from respiratory causes declined 3.9%." (Laden, 2006). And if the pollution itself cannot be controlled, then efficient city planning can help increase the distance between housing and sites of pollution.

Any country engaging in industrialization necessarily contributes to the problem of pollution, and thus has a responsibility to relieve it. Heart disease and respiratory illness are two of the most common causes of death around the world; by striving to reduce air pollution, we are not only improving the Earth's health, but also securing our own.

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