HEALTH & CLEAN AIR
newsletter
Spring 2003

Saving the Children

The sins of the fathers are to be laid upon the children.

Shakespeare, *Merchant of Venice*

Children are not ordinarily exposed to air pollution only once or twice, or even twenty or thirty times, even though the regulatory systems of most countries tend to focus on such exposures. Instead, children are exposed hundreds, thousands or even millions of times during the course of growing up, so one of the most pressing questions is what is the cumulative impact of breathing polluted air, year in and year out. Science is beginning to answer that question, and results are grim. Assembled, the studies show that the young—not merely asthmatics or those with chronic bronchitis, but otherwise healthy, robust infants and children—suffer a continuum of injuries ranging from reductions in their ability to breathe normally, to increased doctor visits, hospitalizations, school absences and, for some, death.

The studies demonstrating what is happening to the world's children span nearly four decades and a score of countries. Whether the children are Dutch, Brazilian, Chinese, Canadian or American, the results are the same.

Children are sickened and hospitalized by air pollution in their early years, their normal lung growth is retarded in middle and later years, and they enter early adulthood with symptoms of chronic illness. Throughout this time, they are placed at increased risk of death and of developing serious, even life-threatening, illnesses.

This *Newsletter* reviews these studies and attempts to describe the toll taken from children beginning, according to a few studies, before birth and continuing through puberty into early adulthood. The emphasis in this *Newsletter* is on describing the impacts, rather than attempting to disentangle the complex web of air pollution and striving to assign a specific illness to a specific pollutant. In some instances, it is quite clear that the cause of, say, increased school absences was smog, or ground-level ozone. In other cases it may be clear that fine particulate matter, or soot, is to blame for increased illness. Too often, however, humans breathe a complex mixture of various pollutants at a variety of concentrations in different places and it is unclear which pollutant or mixture is responsible for death or grave injury. That air pollution does, in fact, kill and cripple children is now clear beyond any credible dispute.

The first important study of the effect of air pollution on children was published in 1966 in Britain. Researchers identified a large number of children who had been adopted at birth, then compared those living in highly polluted areas with others in regions with less air pollution. In the more polluted areas, lower chest infections—bronchitis and pneumonia, for example—were triple the number of those in cleaner regions. A 1965 Philadelphia study pointed in the same direction. Comparing 1,346 patient visits to the Emergency

Department of the Children's Hospital, researchers found that on days with "noteworthy high pollution," bronchial asthma visits tripled.

Injured in the Womb: Low Birth Weight, Premature Birth and Birth Defects

A "low-birth-weight" newborn, one weighing less than 5.5 pounds, is vulnerable to a litany of ills: death, disease, physical and learning disabilities, and respiratory ailments to name but a few. Thus, finding a link between air pollution and low birth weight—and studies in California, the Northeastern United States, the Czech Republic, and Korea, have done precisely that—is profoundly troubling.

It is certainly plausible that air pollution could cause low birth weight. One of the most common air pollutants, carbon monoxide, binds with the blood's oxygen-carrying hemoglobin, with 220 times the affinity of oxygen itself. It, in effect, suffocates the body's large organs and, in the case of pregnant women, their developing babies in whom oxygen displacement is roughly 50 percent higher than in their mothers.

Carbon-rich fuels—gasoline or coal, for example—produce carbon monoxide when burned incompletely by out-of-tune or poorly designed engines or furnaces. It certainly causes low birth weight in the newborns of women smokers, where the carbon monoxide is created by burning tobacco: according to the World Health Organization, it is the leading cause of low-birth-weight infants.

If air pollution from cigarettes can cause low birth weight newborns, couldn't the same be true of women merely breathing air that has been polluted by cars, trucks and buses, which account for about 77 percent of carbon monoxide nationally? It could happen—and according to a number of studies, it does. Consider the following:

- In a Czech Republic study, researchers examined births and air pollution levels in 67 districts. Women exposed to sulfur dioxide (created when sulfur-rich fuels like coal and diesel are burned), especially in their first trimester, had higher numbers of both low birth weight newborns and premature babies. Newborns of women exposed to total suspended particulate matter (soot, also a product of coal combustion) were also linked to low birth weight.
- Exposure to sulfur dioxide also led to increased low-birth-weight newborns in a study of Boston, Philadelphia and four other northeastern cities, but so did carbon monoxide. The link between carbon monoxide and low birth weight was also found in southern California.
- All these pollutants—carbon monoxide, sulfur dioxide and total suspended particulate matter—were linked in Korea to newborns with increased low birth weight, and so was another, oxides of nitrogen (created by combustion, especially by power plants and vehicle engines). Scientists compared 276,763 births to pollution levels collected from 21 monitoring sites, where, as was the case in California and the Northeast cities, motor vehicles were the dominant sources of pollution.

Some studies have linked air pollution to other birth-related injuries. California, for example, has a Birth Defects Monitoring Program and when researchers compared its data to air pollution monitors they found a fit: heart defects (ventricular septal defects, or holes in the wall separating the heart's two lower chambers) increased in direct proportion to the mother's exposure to carbon monoxide in the second month of pregnancy. Other defects, such as those in the heart's arteries and valves, increased with exposure to ozone, or smog, in the second month.

That air pollution can indeed cause defects in the human fetus is demonstrated by another study, though of one of the most dangerous mixtures—tobacco smoke. Researchers studied 32 victims of sudden infant death syndrome, or SIDS, and found that in those exposed to cigarette smoke in the uterus the distance between alveolar attachments in their lungs increased. This, in turn, could allow their airways to narrow, cutting breathing efficiency.

Exactly how air pollution, especially when it is being transmitted to the child through the mother, could cause these defects is unclear. Nevertheless, low birth weight, premature birth and birth defects, seem to be merely the first of a succession of injuries inflicted on children by air pollution. The most serious of these injuries is death itself, which is clearly one of the effects of air pollutants.

Infant Death

The lethal effect of air pollution on children is dramatically illustrated by the infamous "Great Fog" that gripped London in 1952, officially killing 4,000, a toll that probably was closer to 8,000 or even, perhaps, 12,000. Often such episodes are described by skeptics as merely "harvesting," or accelerating deaths that would have occurred within a few days in any

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event, mostly in the elderly. A review of childhood death during the Great Fog belies that claim.

In the week before the Great Fog, 15 newborns four weeks or less in age died. During the Fog, that number jumped to 28, a 87 percent increase. Similarly, deaths among children between four weeks and one year of age jumped from 12 to 26, an increase of 116 percent. All told, the number of deaths of children under the age of 14 increased from 38 to 67, a 76 percent jump.

Still, pollution during the Great Fog, so dense that even in mid-afternoon drivers were forced to use their headlights, vastly exceeded anything seen today in most nations. Some legitimately question whether modern, presumably lower concentrations could be lethal. The answer to that is yes.

In the Czech Republic, for example, researchers not only examined each birth from 1989 to 1991, but each of the 2,494 infant deaths as well. Comparing these to air pollution levels, they found that without regard to the infant's birth weight or gestational age and the family's income or other socioeconomic factors, the deaths of newborns from respiratory causes were linked to levels of particulate matter. Other researchers examined infant mortality in 1980–1982, during an economic recession, which led to substantial reductions in particulate pollution in some regions of the U.S. As levels of total suspended particulate matter dropped, so did neonatal mortality, or deaths between one day and one month of birth. The scientists found a "significant impact of pollution reductions on decreases in infant mortality rates," which they concluded "suggest(s) that pollution exposure adversely impacts the fetus before birth."

Brazilian scientists linked child mortality to not only particulate matter, but to other pollutants as well: carbon monoxide was linked to a 15 percent increase in deaths from respiratory causes in children under five; sulfur dioxide with a 13 percent increase; and particulate matter, or soot smaller than 10 microns (about 50 of these would fit on the width of a human hair), a death increase of seven percent.

Low birth weight, premature birth and birth defects, seem to be merely the first of a succession of injuries inflicted on children by air pollution. The most serious of these injuries is death itself.

Concentrations of air pollution when biomass fuels—wood, for example—are burned are usually much high indoors than outside.

The lethal effect of pollutants is the same, however. Indeed, after an exhaustive review of studies, which included actual measurements of indoor air pollution, one researcher has estimated that there are 4.1 million deaths annually from acute respiratory illnesses in children under five in developing countries. This compares with 3.0 million deaths from intestinal disease, and 0.68 million from malaria.

The First Invisible Signs of Damage

Although air pollution can, and in too many cases does, cause death, its more subtle and insidious effects are vastly numerous. Some of these early injuries and diseases can be detected using spirometry—having a patient breathe as hard and fast as possible through a tube, then measuring the results. Other damages can be detected by swabbing or rinsing the nasal passages and searching for biological fingerprints of injury. And when scientists look for such evidence, it's usually there.

In The Netherlands, for example, scientists compared a group of elementary school students in Bilthoven to others in Utrecht, where the levels of "black smoke," or soot, carbon monoxide and nitrogen dioxide (principally emitted by cars, trucks and buses) were 50 to 190 percent higher. In Utrecht, the children could not breathe as well and their nasal rinses contained traces of uric acid, albumin, and nitric oxide metabolites—all signs of inflammation produced when the body is attempting to stave off injury.

There are literally hundreds of studies like this. They've been done in U.S. summer camps, other nations, as comparisons of rural children versus those in cities, and a wide variety of other circumstances. There is simply no question that the threshold indications of injury due to air pollution are reductions in the ability to breathe normally, often accompanied by evidence of lung and airway inflammation. At the next level, parents begin to see the damage.

Doctor Visits, School Absences and Hospitalization

The first outward sign of air pollution injury is likely to be a child's complaints of illness, triggering school absences, doctor visits and hospitalization. In Toronto, Canada, for example, when ozone levels rose during a 1980–1994 study period, hospitalization of children less than two years old for acute respiratory disease—asthma, acute bronchitis/bronchiolitis, croup (swelling of the windpipe and voice box) and pneumonia—increased sharply as well: hospitalization rates for acute bronchitis/bronchiolitis rose 45.7 percent, followed by croup at 45.3, asthma at 31.3 and 23.3 for pneumonia. Another study of hospital respiratory

admissions for infants in selected Ontario, Canada hospitals found that relatively low levels of summer pollutants—ozone, or smog, and sulfate particulate matter, often seen as summer haze—accounted for 16 percent of such admissions.

It seems clear that air pollution not only reduces the ability to breathe normally, but that it, in effect, stunts the growth of lung function.

These illnesses translate directly to missed days of school, and disruption of family life that accompany them. In southern California, for example, when ozone levels rose 20 parts per billion, a fairly common day-to-day variation there, school absences rose dramatically: an increase of 62.9 percent for illness-related absence rates (82.9 percent for respiratory illnesses, 45.1 percent for upper respiratory illnesses, and 173.9 percent for lower respiratory illnesses with wet cough). Researchers in Utah discovered that increases of inhalable particulate matter (PM10) resulted in a 40 percent increase in overall absences from school by children. In Seoul, Korea, researchers tracked 1,264 students for nearly four years. When air pollution levels rose, so did illness-related absences. The reverse is true as well: if air pollution declines, so does sickness.

In Atlanta, Georgia, for example, officials asked drivers to park their cars during the 1996 Olympics. They did, and as a result, levels of both ozone, or smog, and particulate matter fell sharply, and so did measures of illness. Citywide acute care visits and hospitalizations for asthma dropped, as did Medicaid claims (by 41 percent) and HMO visits (off 44 percent). There was a similar experience after East and West Germany reunified. Officials surveyed health in the State of Sachensen-Anhalt in 1992–1993, when air pollution levels were higher; 1998–1999, when lower; and 1995–1996, when they were at intermediate levels. As air pollution levels fell, so did bronchitis, sinusitis, and frequent colds.

Sadly, however, air pollution levels are, as a general matter, not falling; or, if so, not rapidly enough. Eventually, the repeated shocks to the children's maturing bodies takes a toll. One of the earliest effects is to slow the growth of respiratory systems, with permanent, perhaps irrevocable consequences. Children breathe more pound-for-pound than adults, spend more time outdoors and, perhaps most importantly, their respiratory, hormone and other systems are still growing and maturing and thus vulnerable to injury.

Slowing Lung Growth

It seems clear that air pollution not only reduces the ability to breathe normally, but that it, in effect, stunts the growth of lung function. This was clearly the case in a New Jersey study, where 797 children averaging 8.2 years of age were followed for two years. Pulmonary function tests were given four times to each child. Normally, as children grow, the amount of air they can breathe and the force with which they can inhale and exhale increases. That was the case with these children, but with a notable finding: ozone pollution seemed to slow lung growth.

These children lived in ten communities divided into low, medium and high ozone areas, and the more polluted the air, the slower the lung function growth. It was slowest in the high ozone areas, followed by medium and low ozone districts. This indicates that ozone not only can retard lung function growth, but that there is a "dose-response" relationship.

Researchers in Hong Kong found similar results when they compared the respiratory health of two groups of children 8 to 12 years old, one from a less polluted area and the other more highly polluted. Boys and girls were separately analyzed, and in both sexes the children from the more highly polluted areas couldn't exhale as fast or forcefully as those from the cleaner district—a sign of fibrosis, or stiffening of the lung.

California scientists also found "significant deficits in lung function growth" associated with children exposed to a variety of pollutants, principally from motor vehicles, over a four-year period. Although restricted to boys, the same slowed growth was found in 1,001 preadolescent children followed for two years in Krakow, Poland.

Are the Injuries Permanent?

A nagging question remains, however: will these changes follow children into adulthood and, if so, might all these changes be precursors to serious chronic disease? The answer to both these questions appears to be yes, based on several important studies. These must be viewed against the backdrop of animal studies —it would be unethical to use human subjects—finding grave injuries caused by air pollution, especially ozone, or smog.

When mammals of almost every sort have been exposed to ozone for long periods, they undergo strikingly similar changes, none of them good: normal cells with cilia, which are tiny whip-like organs that expel foreign particles and organisms, have been killed. They are replaced by thicker, stiffer, non-ciliated cells. Scars and lesions form, not unlike those found in smokers. In monkeys, whose respiratory systems most resemble those of humans,

these lesions are found in the deepest regions of the lung. They are worst when the exposures mimic what happens in real life—that is, for a few hours a day over a 90-day period. Most alarming, when the exposure halts, there is a "worsening" of the lesions. All of this suggests the very real possibility of permanent, structural change in the lungs of human children as well.

Since humans cannot be killed and autopsied, scientists have developed ingenious ways of determining whether air pollution injures human children in the same way it does animals. One of these has been to enroll children in long-term studies tracking their health and looking for correlations with air pollution levels. Perhaps the best of these is the Southern California Children's Health Study, a decade-long study tracking 5,500 students in 12 communities, about two-thirds enrolled as fourth-graders.

Southern California Children's Health Study

The striking findings of this study include the following:

- The 62.9 percent jump in illness-related absence rates for a 20 part per billion increase in ozone, mentioned earlier.
- A decrease in lung growth that is not only larger in children spending more time outdoors, but larger than those reported for exposure to environmental tobacco smoke.
- Most alarming, a three-fold increase in the development of asthma in children who live in high ozone, or smog, communities and play at least three sports. The number of children developing asthma in this study is relatively small, so the conclusion requires confirmation.

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Confirmation of CHS Findings

Other studies, conducted of other populations and in different ways, point in the same direction as the Children's Health Study. For example, of 520 non-smoking Yale University students, those who had lived for four or more years in a county with relatively high levels of ozone (these were areas of Arizona, Los Angeles, Connecticut, Maryland, New Jersey, New York, and Utah) wheezed and coughed more than students from cleaner air counties. Like the children in Hong Kong, the students from the more polluted counties could not exhale as fast or forcefully as their peers from less polluted areas.

Similarly, in a study of 130 Berkeley freshmen aged 17 to 21, those from more highly polluted southern California were less able to breathe normally than others from the less polluted Bay Area. In the jargon of scientists, the results were "consistent with biologic models of chronic effects of ozone in the small airways"—consistent, in other words, with precisely the kind of damage found in monkeys breathing ozone.

Policy Implications

Perhaps these conclusions are tissue-thin pieces of evidence stitched together to present an alarmist picture by strident activists. On the other hand, perhaps they are precisely what they seem: the slow emerging outline of a major threat to the health of the world's children. Or perhaps it's something in between.

What these studies do point to, however, are at least two important points:

First, because it is sometimes almost impossible to tease out the precise impacts of a single pollutant on an exact injury, the need to prefer solutions that reduce not just one, but all, pollutants.

Second, focus laws and regulations squarely on protecting human health, because the true threat posed to society is air pollution, not its control despite arguments from polluting industries and their defenders to the contrary.

It was this conviction that lead Congress in 1970 to rest the mandates of the Clean Air Act squarely on protection of human health. When the law's principal author, Sen. Edmund S. Muskie (D–Me) was confronted by claims from General Motors that it was technologically impossible to reduce tailpipe emissions from cars as much as proposed, he responded that—

> *The first responsibility of Congress is not the making of technological or economic judgments—or even to be limited by what appears to be technologically or economically feasible. Our responsibility is to establish what the public interest requires to protect the health of persons. This may mean that people and industries will be asked to do what seems to be impossible at the present time. But if health is to be protected, these challenges must be met. I am convinced they can be met.*

History proved Muskie right, and the challenges were met. If the evidence reviewed in this *Newsletter* is correct and new reductions in emissions are proposed to save the lives of children who are unnecessarily diseased and dying, there will no doubt be those who will say that society cannot afford the costs or that further reductions in air pollution are technologically impossible. History, however, is on the side of Muskie, and our children.

by David V. Bates, CM, MD, FRCP, FRCPC, FACP, FRSC

Unraveling Effects on Children

The first important study of the effect of air pollution on children was that published in 1966 by Douglas & Waller in Britain. They randomized genetic and SES factors by following a large cohort of infants adopted at birth into other families, and found a threefold difference in lower chest infections over a three-year period as between those living in high compared to very low air pollution regions of the country. This study had a considerable influence on the deliberations of the U.S. Senate Committee preparing the first U.S. Clean Air Act. In 1995, I reviewed the literature on the effects of air pollution on children for which I found 49 references. I was very surprised at the flood of reprint requests that followed this, perhaps because it was the first such review to be published. In it, I pointed out that the excess mortality of children in the London 1952 smog disaster was significantly increased to 67 during the week of the smog from the usual weekly total of 38 which it had been the week before. Of course, the numbers were small, and this was probably why the increase had been generally overlooked. The situation is now very different, as large numbers of studies of children have been published. This *Newsletter* brings together this information. One important question is why asthma prevalence varies in different regions, and whether current air pollution has anything to do with an increasing prevalence. In this connection, the following observations can be made:

1. Careful observations in Hartford Connecticut revealed that 19 percent of children in that city have asthma; prevalence rates were higher in Hispanic/Puerto Rican children, in those aged 5-10, in boys up to the age of 10; and in girls after the age of 15. This is about double the prevalence rate usually assumed for North America.
2. Changes in prevalence must be interpreted with caution, as these may occur through alterations in medical diagnostic practice.
3. A prospective study in Southern California indicated that the risk of developing asthma was increased in higher oxidant communities among children who engaged in outdoor sports.
4. Higher exposures to traffic exhaust may lead to a higher risk of developing asthma.
5. The effects of a "respiratory infection" in school aged children are shown by a significant decline in terminal airflow measurements even when the child is back in school, indicating residual small airway involvement.

Higher exposure to current air pollution probably does increase the risk of developing asthma, as defined as "doctor-diagnosed" asthma. It is not clear what proportion of the increased prevalence of the disease should be attributed to such exposures.

This question is complicated by the recent survey of 25,000 people over the age of 11 reported from Britain, which found that 32 percent of those reporting "wheezing" and 27 percent of those with "doctor-diagnosed asthma" had neither a raised serum IgE nor a raised IgE to house dust mites.

Can it be that current air pollution exposure is causing a small airway chronic bronchiolitis which in many cases is being confused with asthma?

Aggravation of Asthma and Other Effects

Whether or not modern air pollution exposures have a significant effect on asthma prevalence, there is no doubt from the data that asthma aggravation is a significant result of current levels of exposure. So are more frequent attacks of lower respiratory infection, and these cannot be assumed to be without long term consequences.

Effects on the fetus are difficult to interpret, since we have little idea of the mechanism; in fact the first clue on this problem may be the remarkable study of the lungs of infants exposed when in utero to the tobacco smoke of the mother, which found that there were structural differences in the lungs of the exposed infants. It is to be hoped that others follow up this lead, since if it is confirmed it would be a major clue.

Exposure to higher levels of coal burning pollution when in utero can lead to differences in some immune system parameters though once again we have little idea of possible mechanisms, nor of the long term significance. We have little idea of why exposure to current urban air pollution might be slowing down lung development in exposed children; this is because we know very little about the mechanisms involved in normal lung development.

By comparison with the paucity of information on these questions, we have no difficulty suggesting why exposure to vehicle emissions, or the chemicals derived from them, such as ozone, might have deleterious effects on the lung. There are not only easily demonstrable direct effects on lung function, together with the induction of lung inflammation as a result of such exposures, but in addition oxidants impair the function of alveolar macrophages, and this effect might well result in more severe respiratory infections of all types. It is the task of the macrophages to ingest and destroy invasive organisms, and both ozone and oxides of nitrogen have been shown to impair their ability to do this.

There is no reason to express much surprise at the finding that hospital admissions in the United States of infants with bronchiolitis more than doubled between 1980 and 1996. What is perhaps remarkable is that it did not occur to the authors that possibly increased urban exposure to particulate air pollution might be at least partly responsible.

Pope's pioneering study of hospital admissions in children for respiratory illness in Provo, Utah for two years when a local steel mill was operational, and for the intervening year in which it was shut down, led to one of the first studies integrating epidemiological findings with toxicology, when Ghio and Devlin showed that the particles from PM10 filters when the mill was operating were much more toxic than when it was shut down. This was probably due to their much higher metal content.

It has been pointed out above that the earliest studies of effects on the fetus are difficult to interpret; the same is true when one attempts to answer the question of what repetitive respiratory events in children lead to in the teen years and beyond. From the point of view of regulation, the obvious need to minimize current exposures does not have to rest on evidence of long-term damage, since the social disruption engendered by more frequent school absences or more severe exacerbations of asthma are sufficient in themselves to require the active prosecution of control strategies.

in future issues...

DISTINCTIONS WITHOUT A DIFFERENCE

There is no difference between pollutants that injure human beings and those that cause global warming, and making this artificial distinction handicaps efforts to control them all.

NOXIOUS NOX

Oxides of nitrogen play a critical role in the formation of both ground level ozone, or smog, and fine particles, so there is a tendency overlook the role they play in injuring human health.

Worth Noting

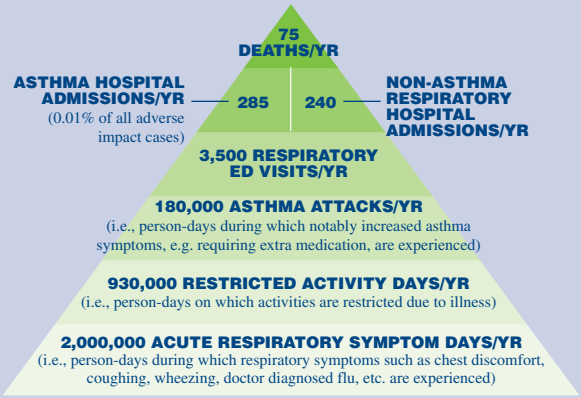
As the ability of scientists to quantify the death toll due to air pollution improves, some economists—the head of the Bush Administration's Office of Information and Regulatory Affairs (OIRA), John Graham, for example—are seeking to minimize the monetary value assigned to mortality.

Graham is pushing for government-wide application of "discounting" and "life years," concepts originally developed to compare investments that produce future income. Graham, former head of the Harvard University Center for Risk Analysis, which is heavily funded by ExxonMobil and other companies, wants them applied to life saving regulations.

The premise of "life years" is that the elderly are worth less because they have fewer years left to live, so a 70 year old is valued at, say, $258,000 versus $2.3 million. "Discounting" means that a life saved in the future is worth less than one saved today. Their combined effect is to devalue, and thus stave off, rules to protect against diseases with longer latency periods, such as lung, breast, brain and other cancers.

Mortality & Hospital Admissions are the "Tip of the Iceberg" of Pollution Effects

Source: Thurston,G.: U.S. Senate Testimony, 2/5/97
Pyramid of New York, NY Annual Adverse Ozone Impacts Avoided by the Implementation of the Proposed New Standard (vs. "As is"). Figure Sections sizes not drawn to scale



Discounting and life years also obscure a harsh reality: death is often the terminal event in a cascade of ill health effects. Based on the analysis of Dr. George Thurston of New York University (depicted in the accompanying pyramid), for every 75 deaths from air pollution, there are millions of bouts with sickness and ill health. These underscore the truth in the old saying that it is possible to know the price of everything, but the value of nothing.

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