HEALTH & CLEAN AIR
newsletter
Spring-Summer 2004

Car Crazy

Our products create pollution, noise and waste.
AB Volvo; 1989[a]

From Bangkok to Berlin, China to California, the single most vexing challenge confronting health and environmental officials is the car, and its kin, trucks and buses. They already exact an immense and tragic toll, one that is sure to grow as the heat-induced levels of smog and fine particles rise with the worsening of global warming.

This Newsletter focuses on the health damage of traffic. Rather than taking a global view, however—for example, how many deaths in the United States and the world can be attributed to traffic emissions—the focus is on much narrower and more immediate concerns: what happens to children who live 50 yards from an Interstate highway, for example? Or to students in a school across the street from a marshaling yard for diesel buses? Does it make any difference whether you live the length of one football field or five from a heavily traveled highway?

Somewhat surprisingly, answering these questions requires a review of Dutch, German, Swiss and other European literature, for it is there that studies have focused not on specific pollutants, but simply "traffic." Such studies are now being launched in the United States and Canada as well, and for good reason: the European work—and, most recently a study of highway troopers in North Carolina—leaves no doubt that living, schooling or working in or near heavy traffic results in not only very clear, very serious illness but death.

The study of 9 Highway Patrolmen in North Carolina suggests that breathing in vehicles, even by healthy young men like the troopers, triggers changes that could pre-sage heart attack or stroke. The troopers were followed fours day in a row, as they worked the 3 p.m. to midnight shift in cars equipped with pollution monitors. Their heart beats changed, and blood levels of chemicals that indicate increased heart risk, such as C-reactive protein, increased. The team concluded traffic pollution was linked to "pathophysiologic changes that involve inflammation, coagulation, and cardiac rhythm."[1]

By any measure, the loss of life and wellness due to cars, trucks, buses (as well as aircraft, ships and so-called non-road engines) is immense.

There's the obvious, of course; namely, traffic accidents. Vehicles are the leading cause of death in the United States for age 2 and every age between 4 and 34.[2] In 2001, traffic injuries killed 43,423 Americans.[3]

[a] In the late 1980s and early 1990s, Volvo widely distributed a brochure with this statement emblazoned across its front in bold, black letters. The company went on to commit itself to "action at every conceivable level, as our contribution to creating a better environment for future generations." In fulfillment of that pledge, Volvo established 20 different goals, ranging from establishing environmental councils in companies and plants to an announced objective "To be, and considered, one of the most environmentally aware companies in the transport sector." See, e.g., http://www.rand.org/publications/MR/MR1009/MR1009.appa.pdf

Globally, the World Health Organization estimates that traffic injuries kill more than five million people, accounting for nearly 1 of every 10 deaths.[4]

Then there's the less obvious: air pollution generally. Air pollution claims more lives than traffic accidents, killing tens of thousands of Americans each year,[5, b] and tail pipes are the major source of that air pollution. Ozone, or smog, has been linked to deaths[6] and development of asthma.[7] Fine particles, such as diesel soot, are associated with premature death, as is sulfur dioxide.[8] As carbon monoxide levels rise, so do levels of death from congestive heart failure.[9] Cars, trucks and buses are, in the aggregate, the major source of fine particles and smog, and virtually all carbon monoxide. Because of the sulfur in gasoline and diesel fuel, vehicles are also a major source of sulfur dioxide in cities.

Finally, there is the incalculable: global warming. There can be little credible dispute that global warming has begun. By every measure—air, soil, stratospheric and ocean temperatures; sea level rise; sea ice, glacier and ice cap melting; as well as plant and animal population shifts—warming has begun, and the patterns are consistent only with a human signature, not natural events, such as sun spots.[10]

Because warmth boosts formation of ozone and, to a lesser degree fine particles, higher temperatures due to global warming will increase air pollution.[11] One group of scientists has projected that the warming associated with a doubling of carbon dioxide, which is the principal greenhouse gas, would triple the number of worst ozone days in southern California, increasing death, hospitalization and illness dramatically.[12]

Pollutants Associated With Traffic

There are essentially two fundamentally different ways of assessing the impacts of motor vehicles on human health:

One—and, by far, the most common—is to compare levels of air pollutants from motor vehicles with the prevalence of a wide variety of ills, ranging from subtle changes to severe and irreversible impacts, of which death is the most serious. The principal pollutants from motor vehicles are—

- *carbon monoxide* (from incomplete combustion of fuels);
- *oxides of nitrogen* (formed when combustion heat causes oxygen, which is roughly 20 percent of the air, and nitrogen, which is about 80 percent, to combine);
- *hydrocarbons* (essentially unburnt gasoline or diesel fuel);
- *particles* (i.e. diesel soot, which is a probable cause of cancer); and,
- *ozone* (formed when hydrocarbons and oxides of nitrogen react, especially in sunlight).

This approach requires extensive monitoring for a variety of pollutants, disentangling the effects of, say, carbon monoxide from ozone, controlling for a variety of unrelated factors, such as tobacco smoking in the home, socio-economic status, and the like.

Cars, trucks and buses cause a variety of serious injuries, including death, all of which can be reduced simply by taking vehicles off the road or curbing their emissions.

The other approach is, comparatively at least, simplicity itself: find places crowded with cars, trucks and buses—near interstate highways, for example—and look for illnesses that are higher there than in places with little or no traffic.

The United States has tended to follow the first approach, and so did Europe until about ten years ago, when a groundbreaking study in Munich, Germany appeared in the scientific literature. It was a study of 6,537 Munich fourth graders in which signs of ill health—difficulty breathing, for example—were compared to traffic counts. By today's standards, the findings were hardly extraordinary: as traffic volume increased, the ability of the children to exhale forcefully fell and difficulty in breathing increased.[13]

What was remarkable was the simplicity of technique and certainty of the outcome. Soon, researchers throughout western Europe—Amsterdam, Stockholm, London, to name but three—launched traffic studies. What they have developed in the intervening decade is a remarkable and irrefutable body of evidence showing that cars, trucks and buses cause a variety of serious injuries, including death, all of which can be reduced simply by taking vehicles off the road or curbing their emissions.

It makes sense that air pollution would be worse near busy streets and highways, and studies show that it is. For example, studies in Stockholm, Munich

[b] Indeed, one European study concluded that air pollution mortality is twice that of traffic fatalities. N Künzli, R Kaiser, S Medina, M Studnicka, O Chanel, P Filliger, M Herry, F Horak Jr, V Puybonnieux-Texier, P Quénel, J Schneider, R Seethaler, J-C Vergnaud, H Sommer. Public-health impact of outdoor and traffic-related air pollution: a European assessment *Lancet* 356, 9232, Sep. 2000.

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and various areas in the Netherlands all found that at sites with heavy traffic, levels of particles 2.5 microns (millionth of a meter) were roughly 18 percent higher than in the urban areas generally. The "reflectance" of filters, which is a measure of diesel-related particles, was 31 to 59 percent higher at traffic sites.[14]

If pollution is worse, shouldn't the same be true of the injuries it causes? Yes, and scores of studies demonstrate this.

Ten years after that first seminal study in Munich, researchers in the same city randomly selected 7,509 school children and compared the presence of various ailments with traffic counts. They found that where traffic levels were higher, so were cough, wheeze and, most alarmingly, asthma.[15] This, commented one prominent scientist, suggested "an important role for primary combustion products from traffic."[16] A study of 16,663 junior high school students in Jakarta also found a link between traffic levels and a variety of illnesses—cough, phlegm, persistent cough and, again, asthma.[17] Car, truck and bus pollutants also kill.

Mortality

In a study of residents of England and Wales aged 45 years and older, researchers compared 189,966 deaths from stroke with distance from a main road. In men living within 200 meters of such a road, stroke mortality was 7 percent higher compared with men living 1,000 or more meters away. For women, the increase was 4 percent. The scientists concluded that about 990 stroke deaths per year could be attributed to traffic pollution.[18]

In Amsterdam, adults living within 100 meters of a freeway or 50 meters of a busy road were almost twice as likely to die from lung or heart disease, causing researchers to conclude that "long-term exposure to traffic-related air pollution may shorten life expectancy.[19] In another Amsterdam study, scientists concluded that increases in two specific vehicular pollutants—"black smoke," which is a measure of particle concentrations, and oxides of nitrogen—were both linked to death the following day. This effect, they also concluded, was higher "in the summer and in the population living along busy roads."[20]

Virtually all gasoline and diesel fuels are contaminated with sulfur which, when burned, produces an invisible gas sulfur dioxide. It, in turn, forms sulfuric acid on contact with water (e.g. in the lungs) and, over time, extremely fine particle sulfates. In large cities, cars, trucks and buses are major sources of sulfur. That was the case in Hong Kong before the government ordered sharp reductions in sulfur content in 1990. Hong Kong's levels of sulfur dioxide immediately fell nearly 50 percent, accompanied by a 3.9 percent drop in respiratory deaths per year and a 2 percent decline in cardiovascular mortality.[21]

Birth Defects

As American researchers launched studies, they too found a relationship between traffic and ill health. A team at the University of California at Los Angeles looked at 9,000 babies born from 1987 to 1993. Pregnant women who were exposed to the highest levels of ozone and carbon monoxide because their homes were close to busy freeways were three times as likely to have a child with certain heart defects as women breathing the cleanest air.[22]

Cancer

When researchers in Denmark collected data from the nation's Cancer Registry on children who had been diagnosed with leukemia, tumors of the central nervous system or malignant lymphoma, they found that those children's exposures to both benzene and nitrogen dioxide (which are indicators of vehicle pollution) had been higher during and 9 months before birth than those of 5,506 other children selected at random. The researchers concluded that a doubling of vehicle pollution increased the risk of lymphomas by 25 percent. What specific pollutant is responsible? Nobody can be sure—but whatever it may be, it comes from vehicles.[23]

Bronx's Hunts Point and Asthma

Hunts Point, a one-square-mile peninsula at the edge of the south Bronx in New York City is home to what some claim is the world's largest produce and meat market in the world. About 80 percent of the city's produce and 40 percent of the region's meats pass through Hunts Point—and so, too, do up to 20,000 diesel trucks per week, or about two every minute.[24] Rates of infectious diseases and chronic diseases are well above average in Hunts Point. The death rate from pneumonia and flu is twice that of New York City as a whole. Deaths from chronic lung disease are 20 percent higher and from stroke 55 percent greater.[25] The asthma rate at Hunts Point is said to be the second highest in the United States and the third highest in the world.[26]

The relationship between truck traffic at Hunts Point and levels of fine particles composed of elemental carbon is linear: as truck traffic increases so, too, does the air pollution.[27]

Cough and Respiratory Illness

In The Netherlands, researchers compared various measures of respiratory health in 82 elementary school children who lived in either more-polluted Utrecht or in less-polluted Bilthoven. In Utrecht,

where concentrations of carbon monoxide and NO_2 (both markers of car and truck pollution) were 50 to 80 percent higher than Bilthoven, so, too, was the evidence of subtle but important damage. For example, the children in Utrecht coughed more and were unable to exhale as forcefully as those in Bilthoven. Levels of respiratory disease, which is the leading cause of death in children under five years of age, were also higher in Utrecht.[28]

In children living 100 meters or less from a freeway, cough, wheeze, runny nose and—again, alarmingly, doctor diagnosed asthma—were reported "significantly more often."

When their nasal passages were cleaned with a swab, several indicators of body damage—interleukin-98, urea, uric acid, albumin, and nitric oxide metabolites—were elevated in the Utrecht children.

Results in Munich, Germany were similar. There, dry cough at night and cough without infection were both more pronounced in a group of 1,756 infants exposed to traffic related air pollutants.[29]

Another Dutch study, this of 673 adults and 106 children between 0 and 15 years of age living near busy streets in Haarlem were compared to 812 adults and 185 children living along quiet streets. The adults living near busy streets reported only mild shortness of breath, but the children, especially boys, used more respiratory medicines, wheezed more, and suffered from other symptoms of illness. "Living along busy streets," the researchers concluded, "increases the risk of developing chronic respiratory symptoms in children."[30]

Yet another Dutch effort focused on children attending schools less than 1,000 meters from major freeways in the province of South Holland. The freeways carried between 80,000 and 150,000 vehicles per day, 8,000 to 17,500 of them trucks. A total of 13 schools and 1,498 children participated. In children living 100 meters or less from a freeway, cough, wheeze, runny nose and—again, alarmingly, doctor diagnosed asthma—were reported "significantly more often." In this case, the linkages were stronger in girls than in boys.[31]

When a team from California's Office of Environmental Health Hazard Assessment (OEHHA) examined possible links between traffic and respiratory illness, they found "spatial variability in traffic pollutants and associated differences in respiratory symptoms (which) support the hypothesis that traffic-related pollution is associated with respiratory symptoms in children."[32] This was so, even though the area was "a region with good air quality."

In a somewhat similar study in 1995 involving 2,160 children at 12 schools, the researchers administered pulmonary function tests and found a clear linkage between levels of black smoke—a marker for diesel trucks—and both declines in lung function and increased respiratory symptoms. They concluded that truck traffic "can lead to reduced lung function and increased prevalence of respiratory symptoms in children living near major roadways."[33] Comparable findings came from a study of German infants[34] and another of about 4,000 Dutch children.[35]

For six weeks, researchers followed 49 children with chronic respiratory symptoms who lived in Kuopio, Finland. Regardless of their size, particles were linked to increased cough. One day after exposure to particles ranging in size from 0.1 to 1.0 microns, the children's ability to exhale forcefully declined.[36]

The most common way of determining whether lung injury has occurred is to use spirometry, in which a person blows through a tube into a machine that measures how much air was exhaled, how forcefully and fast, and how the volumes vary at different points. Unfortunately, spirometry leaves as much information missing as it provides.

To further plumb the impacts of air pollution on the respiratory system requires collecting samples from within the lung itself and, sometimes, of it. Such samples can provide evidence of injury long before it may be manifested by reductions in the ability to breathe normally.

When 15 healthy volunteers exercised intermittently while breathing diesel exhaust for one hour, researchers found "well-defined and marked systemic and pulmonary inflammatory response ." These impacts were, they added, "underestimated by standard lung function measurements" using spirometry.[37]

They found not only changes in the lung itself—increases in neutrophils, mast cells, CD4+ and CD8+, and T lymphocytes, which are indicators of inflammation and other damages—but in blood. Blood levels of neutrophils and platelets, both produced by the body to ward off infections and both of which can increase the risk of heart attack and stroke, rose significantly.

Similarly, respiratory fluids drawn from 164 traffic officers in Rome contained not only 1,000 percent more particles, but larger numbers of macrophages and inflammatory cells, compared to samples from 109 residents of Perugia, a relatively unpolluted town.[38] A U.S. study of both healthy and asthmatic subjects exposed to diesel exhaust "demonstrated that modest concentrations of diesel exhaust have clear-cut inflammatory effects" on both asthmatics and non-asthmatics.[39] Some

vehicle pollutants also seem to increase susceptibility to illness.[40, 41]

Since the closer the traffic is, the higher the risk, those in greatest danger are people in the midst of traffic: namely, drivers and their passengers.

Those Inside Vehicles at Greater Risk

For example, inside the taxies of 29 Parisian drivers followed for about two months, levels of carbon monoxide, black smoke, and oxides of nitrogen were all higher than those recorded by monitors in the city.[42] In Copenhagen, drivers were breathing twice as much soot as bicyclists; three times as much benzene (which causes cancer in humans); and four times as much organic chemicals. In-car benzene levels were 40 to 50 times the limits recommended by the World Health Organization.[43] A Swedish study of 6,364 truck drivers found they were at increased risk of both lung and prostate cancer.[44]

The same was found in London: car drivers' in-car air pollution was twice that of bicyclists in summer, and 1.66 times as high in winter.[45]

Closer the Roadway, Greater the Effects

British researchers examined the respiratory fluids of 22 children from 3 months to 16 years of age, scheduled to undergo elective surgery. Ultrafine particles were found in all children, but the levels in youngsters living on busy roads was triple that as those on quiet streets.[46]

Similar results in Erie County, New York. There, living within 200 meters of heavily traveled roads were almost twice as likely to be hospitalized for asthma as children who lived 500 or more meters from them.[47]

Massachusetts veterans living within 50 meters of a main road were more likely to have persistent wheeze than those living more than 400 meters away. A study of three groups of 1,500 women in Tokyo found the same.[48]

Diesels—The Greater Threat

Quite a number of studies show that diesels—whose sales are rising rapidly because of higher gasoline prices—account for a substantial fraction of air pollution and, especially, the injuries.

In a Dutch study, for example, researchers studied children in six areas near busy roads, measuring their lung functions and assessing exposure to traffic pollution, separating cars from trucks. They also measured air pollution in the children's school. The density of truck traffic was linked to a decline in lung function, but the connection of car traffic was less. They concluded that traffic related air pollution, "in particular, diesel exhaust particles (DEP)," could cause reduced lung function,[49] evidence that pollution was literally changing the size or shape of the lungs.

A number of studies—some of human subjects, others in the laboratory—have left no doubt that diesel soot triggers an inflammatory response in the body that, in turn, can injure the body—a sort of self-inflicted injury. Inflammation—the crimson of sunburned skin, flu-induced fever or swelling after a sharp crack on the forearm—is a protective response. Water leaking into an injured area, for example, dilutes and washes away toxic substances while it carries in antibodies. Specialized blood cells, macrophages, engulf and kill invading bacteria or other foreign substances.

Trouble is, if inflammation continues day after day, the result can be worse than the initial injury. In joints, for example, the result of unabated inflammation—not caused by air pollution, but inflammation nonetheless—can be rheumatoid arthritis with ruptured tendons, eroded, deformed joints, and numbed fingers, hands and arms.[50] In the intestine it could be the fever, pain and diarrhea of Crohn's Disease.[51] In the case of chronic inflammation due to air pollution, the results can range from quite mild (an increase in the number of macrophages, for example) to profoundly serious: death, due to heart attack, stroke, congestive heart failure or asthma. And, there can be no doubt that inflammation follows exposure to tailpipe air pollutants, as surely as sunrise follows sunset.

Adolescents who reported constant truck traffic outside their homes were two times more likely to also report wheeze than were those who reported no truck traffic.

For instance, when diesel soot was applied to laboratory cultures of human bronchial cells, it triggered production of proteins that stimulate the growth and reproduction of two kinds of white blood cells, granulocytes and macrophages.[52] This is one of the body's earliest and most effective defenses against infection, and a sure signal of inflammation.[53]

Because of such studies, governments have redoubled their efforts in the past several years to curb soot emissions from diesels, but with unexpected results.

With Diesel Soot, Size Counts

The amount of soot emitted by a diesel engine can be measured by either its mass, or weight, or by the number of particles. Traditionally, regulations have targeted the mass of particles, not the num-

ber. This is an important distinction, because in at least some truck and bus engines, when particle mass drops, the number of particles move in precisely the opposite direction, jumping as much as 3,500 percent.[54]

Such increases can result in massive contamination of the air. For example, researchers measured for particles in Copenhagen and Odense, taking some samples at street level and other on rooftops, dividing them into 29 different size ranges. They found as many as 200,000 particles in a cubic centimeter of air, which is roughly the size of a pencil eraser. They also found that levels of these ultrafine particles corresponded closely to concentrations of carbon monoxide and oxides of nitrogen, both of which are emitted largely by motor vehicles.[55]

Whether it is the mere size of the particles or their composition (or both) that make them dangerous is unclear. Certainly, however, diesel soot contains substances that at higher doses are unquestionably poisonous.

The Size and Coatings of Particles

Analyzing particles from a 1985 diesel engine, scientists found them coated with a range of metals and other elements: manganese, phosphorous, calcium, chromium, iron, zinc, titanium, magnesium, molybdenum, barium, sodium, sulfur and silicon. There were individual spheres, chains, clusters and flakes. Measured by number, 99.5 percent of the particles were fine or ultrafine, small enough to not only reach the deepest areas of the lung, but pass through cell walls and enter the blood stream.[56]

Miles Traveled by Diesels is Increasing

The number of miles traveled by diesel engines is rising sharply and apparently inexorably. Between 1992 and 1997, the miles traveled by diesel-powered trucks jumped 46 percent, while that of smaller vehicles—step vans, pickups and cars, for example—rose 50 percent.[57]

Barring a resurgence of support for zero emitting vehicles in California and elsewhere, this trend toward diesels is likely to worsen. In Europe, where manufacturers and governments have used diesels to stave off the competitive threat of the Japanese vehicle manufacturers, they account for one-third of new car sales. Governments encourage diesels by pricing their fuel below that of gasoline-powered counterparts. And, with American consumers beginning to resist the single-digit fuel economy of sport utility vehicles, U.S. car makers are increasing the share of the more fuel efficient diesels.[58]

Control Measures

Because of an accidental experiment in Atlanta, there can be no question that reducing traffic yields immediate health benefits. Asked to park their cars and take the bus to reduce congestions during the 1996 Olympics, Atlantans did just that for 17 days.

Concentrations of traffic related pollutants—ozone, oxides of nitrogen, carbon monoxide and particles of 10 micron and smaller—fell by up to 50 percent from predicted values. The effects were seen in emergency rooms as well: in health maintenance organizations and Medicaid-accepting doctors, acute care visits for asthma fell 44 and 41 percent, respectively. Similarly, there were 11 percent drops in such visits to emergency pediatric departments and 19 percent in the Georgia Hospital Discharge Database.[59]

The economic benefits of lessening pollution can be immense. Banning lead in gasoline, for example, is estimated to have produced economic benefits ranging from $110 to $319 billion per year in the U.S. alone.[60]

Are There Solutions? Of Course.

Virtually every rational person would agree that cities should be friendlier to the people who live in them than to cars. And it possible to design cities that are friendly to their residents. In Curitiba, Brazil, for example—

> *buses carry 50 times more passengers than they did 20 years ago, but people spend only about 10 percent of their yearly income on transport. As a result, despite the second highest per capita car ownership rate in Brazil (one car for every three people), Curitiba's gasoline use per capita is 30 percent below that of eight comparable Brazilian cities. Other results include negligible emissions levels, little congestion, and an extremely pleasant living environment...*[61]

Similarly, vehicles that meet the needs of average drivers but produce no air pollution are technologically feasible. The response of the vehicle industry to the California mandate for zero-emitting vehicles made that clear beyond dispute. But are such vehicles too expensive? Nobody can say, because, amidst complaints from Detroit, Tokyo, Stuttgart and the other centers of auto manufacturing, the experiment was essentially abandoned (at least with respect to battery-electric cars) well in advance of the cost reductions that would have been achieved through experience.

Eventually, of course, both these measures and others will be required if vehicle use continues to grow. Still, there is the possibility that humanity will continue on a relentless march to its own destruction. ■

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

It is not often that the reader concerned with the adverse health effects of air pollution has to read journals in the field of economics, and most of these have concerned estimates of the economic burden of current air pollution levels. However, a recent paper in a British economics journal is an exception, as it calls into question the reliability of current estimates of the impact of particulate pollution on mortality.

Written by G. Koop and L. Tole[62] and published this year in the *Journal of Environmental Economics and Management*, the study analyzed PM and mortality data from the City of Toronto over the period 1992–1997.

There were six monitoring stations for the criteria gases, but only one for PM_{10} and $PM_{2.5}$ for most of the period. The authors noted that there was usually only one observation every three days, and that 66.29 percent of the raw daily data were missing. They used a Bayesian Model averaging method for their analysis, and included 48 explanatory variables, noting that if three lag periods were included, this would increase the number of models to 312. They argue that results from single regression models are inherently unreliable.

It is perhaps not surprising that they found that standard deviations were large and wrote: "Indeed they become so large that the hypothesis that air pollution has no effect on mortality is not implausible." They further argue that the uncertainties are so great, that the findings cannot reliably be used in policy formulation.

A critical discussion of this paper (and a few similar ones that have been published with similar conclusions) quickly mires the reader in a quagmire of biostatistical jargon so that it becomes difficult to define the real issues. This discussion is designed to provide some assistance in this task.

The construction of a model is a simple concept as it involves the combination of variables that best describes a given outcome—in this case the association between particulate parameters and daily mortality. However, with large numbers of possible variables, some choice has to be exerted on which model to choose. In such a question, the idea of averaging a very large number of possible models is attractive, as it might lead to a more reliable definition of a relationship than simply (and more or less arbitrarily) selecting one model for emphasis. This difficulty has been recognized by biostatisticians, and it is made more difficult by the fact that in many papers, the basis for individual model selection is not discussed. One of the largest datasets (NMAPS) from 88 cities in the U.S. did not involve model selection (a point not noted by these authors).

Another problem is the handling of missing data; there are formal statistical methods for doing this, but the influence of such methods on the final result is not usually well defined, though it is often assumed that they will necessarily weaken an association.

The authors of this paper infer that their results call into question the many other studies that have found an association between particulate pollution and premature mortality. In Canada, a two-inch headline in the *Financial Post*, quoting this paper, said: "Number of people who die from air pollution in Toronto = 0."

The reality is that this paper does not diminish the reliability of those studies in which very much larger datasets have been used, and which like the NMAPS analysis, the result does not depend on an arbitrary model selection. Other criticisms are:

1. The Toronto data are very limited.
2. The monitoring network for a large city can have only provided a very inexact exposure estimate for the whole population
3. The inclusion of many intercorrelated and related weather variables would have the effect of weakening any air pollution/mortality association.
4. The time-series data (which include studies from Europe, Australia and Canada) are strongly supported by the two major longitudinal studies that have shown a strong association between long-term residence in regions of higher pollution, and survival.

These studies indicate a higher level of risk than that deducible from time series estimates. They were not discussed in this paper. Also not discussed were the many emerging studies of the possible mechanisms that might explain the lethal nature of fine particles, many of which find that exposure triggers production of platelets, fibrinogen and other factors linked to heart attack and stroke. If Koop and Tole are correct, such objective measures of exposure impacts are of no consequence.

No doubt these over-interpreted studies will continue to appear; and no doubt their conclusions will be trumpeted by those who do not wish to see any action taken that would limit human exposure to particulate emissions.

I am grateful to Dr. Duncan Thomas of USC, Dr. Lianne Sheppard of the University of Washington, and Dr. Kaz Ito of New York University for their comments on this paper.

Worth Noting

Injuries to the Unborn

Few adults fail to be moved when children are injured, for they are among the most vulnerable and helpless. Even more vulnerable and unable to protect themselves, however, are the unborn, and the evidence continues to mount that their injuries by air pollution are both real and substantial.

Researchers in Vancouver, B.C. in Canada collected data from the government's statistical registry for 1985 to 1998 and compared pre-term low birth weight and intrauterine growth retardation daily average and one-hour levels of various air pollutants. They found low birth weight linked to exposure to sulfur dioxide (SO_2), which is emitted by refineries, coal fired power plants, sugar beet processors and other industrial facilities, in the first month of pregnancy. Pre-term birth was associated with not only SO_2, but carbon monoxide, while reduced intrauterine growth rate was tied to levels of SO_2 and nitrogen dioxide. Pollution levels were all well within standards that are supposedly health-based, leading the researchers to conclude that even relatively low levels of air pollution are associated with these injuries to the unborn.[63]

What Makes Corporations Tick?

As tobacco companies have begun settling lawsuits, long-secret internal documents have become public. When commentators reviewed them for the *Annual Review of Public Health*, they concluded that "the tobacco industry has been engaged in deceiving policy makers and the public for decades."[64] To some, that may not seem to be news.

However, the author went beyond the tobacco industry, examining comparable information related to the asbestos and lead industries, presumably on the grounds that something found once may be an exception, found twice a coincidence, but found three times is a pattern. What the author found was "historical similarities in behavior" suggesting that researchers "interested in the activities of chemical, pharmaceutical, or oil companies, for example, could learn much about how these industries operate" by studying the tobacco, asbestos and lead firms. ■

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