HEALTH & CLEAN AIR
newsletter
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Noxious NO_x

Change Alone is Unchanging

Heraclitus, *Herakleitos & Diogenes*, pt. 1, fragment 23[a]

In nature, changing shape is an everyday event—the metamorphosis of a lowly, limb hugging caterpillar into a delicate, but wobbly butterfly, for example, or the gradual increase in girth and weight with age. Yet few things shape-shift into a succession of so many toxic forms as the lowly gas nitrogen, which is 80 percent of the air we breathe. Exposed to searing fire, nitrogen combines with oxygen to form the most common of conventional pollutants, oxides of nitrogen (NO_X, for short).[1] One of the most common NO_X is nitrogen dioxide, or NO_2.

Then, NO_X changes, again and again, forming acids, microscopically fine particles and ozone, or smog, for example. By the time this chain of reactions halts, NO_X and its progeny have injured and killed humans, devastated forests and lakes, artificially enhanced plant growth—and subsequent death—in bays and sounds,[2] destroyed stratospheric ozone, and warmed the Earth.[3] NO_X is truly, in the words of Dr. Bert Brunekreef of Utrecht University, "The gas that won't go away." For this, humanity is vastly worse off—and matters are getting worse.

Despite the litany of ills for which NO_X is responsible, emissions are rising virtually everywhere, with no end in sight. Current U.S. emissions, for example, are about 15 percent higher than 1970, despite ever-tightening tailpipe limits on cars and light trucks partly because emissions from coal-fired power plants increased 44 percent in the same period,[4] and partly because the number of miles being driven has jumped 140 percent since 1970.[5, b] Globally, emissions of NO_X are rising steadily and are predicted to more than triple by the end of the century.[6] Indeed, scientists at Harvard University have concluded that increased ozone due to NO_X emissions in China is blown to the United States and could effectively offset a 25 percent reduction in domestic emissions of NO_X. Similarly, NO_X emitted in the U.S. will increase ozone and other pollution in Europe and vice versa.[7]

Are emissions rising because of flaws in the Clean Air Act and other laws? Or is it a simple lack of political will amongst legislators and regulators? This Newsletter attempts to answer these questions by making sense of the hundreds, perhaps thousands, of studies that have been conducted on the health effects of NO_X, thus casting light on the underlying system of laws and regulations.

[a] Heraclitus (c. 535-c. 475 B.C.), Greek philosopher. *Herakleitos & Diogenes*, pt. 1, fragment 23 (1976; tr. by Guy Davenport).

[b] National emissions of NOx in 1970 were about 17 million tons, and in 1950 about 8 million tons, compared to about 23 million tons in 2002, according to the U.S. Environmental Protection Agency. That compares to about 25 million tons in 1983. Reducing emissions that much in 20 years is a tremendous achievement given the growth in vehicle miles traveled (VMT) and gross domestic product (GDP), but this must be placed in the context of a pollutant that is several orders of magnitude greater than natural levels, with escalating growth in both VMT and GDP. Some nations, most notably Germany, have required that state-of-the art emission controls be installed on motor vehicles and power plants alike. See Curtis Moore and Alan Miller, *Green Gold: Japan, Germany the United States and the Race for Environmental Technology* (Beacon Press, 1994).

Assessing the impacts of NO_X is a thorny challenge because it is rarely found by itself. Virtually always there are other pollutants—fine particles, ozone, benzene, to name but three. When researchers link NO_X to some adverse effect—whether it is death, increased visits to emergency rooms, pneumonia or some other ill—the question that is almost invariably raised is whether NO_X is truly the effector, or cause in fact of the injury; or, was NO_X a surrogate, a sort of stand-in, for one or more other pollutants that are the true source of injury?

In England, some tell the story of children who had to clear out the house after the last parent died, and found in the attic a box labeled: "Pieces of string too short to tie together." Disentangling the effects of NO_X from those of co-pollutants is a bit like tying together the strings, to wit:

The cascade of events triggered by NO_X begins with inhaling. Because it is a gas that doesn't readily dissolve in water, NO_X passes through the body's defenses —cilia, mucus, etc.—reaching the lung's deepest recesses, where air and blood are separated only by a cell wall. A number of studies in which healthy adult volunteers breathe NO_2 while exercising indicate that it, like the more powerful ozone, oxidizes lung tissue.[8] This triggers an inflammatory response, medical short hand for a collection of events that occurs as the body raises its defenses to what is, very roughly, comparable to a sunburn.

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The Early Changes

Generally, when inflammation occurs, blood vessels increase in diameter, boosting blood flow to the injured tissue. Some become leaky, allowing water, salts and some small proteins into the damaged area. One of the main proteins to leak out is fibrinogen, a protein synthesized by the liver that causes blood clotting.[9] In those breathing NO_X, fibrinogen increases, and so do levels of another substance that helps form blood clots: platelets, which are irregularly-shaped, colorless bodies found in blood.[10] Both platelets and fibrinogen are risk factors for heart attack. Again, however, because NO_X is almost invariably associated with other pollutants, disentangling its impacts can be challenging.

Generally, when inflammation occurs, circulating blood cells that ward off assaults by microorganisms rush to the injury.[11] Studies of those breathing NO_X find such an increase.[12]

The Next Changes: Less Serious Illnesses and Other Impacts

Like ozone, oxides of nitrogen oxidize and destroy organic matter. Animals exposed to NO_X are less able to ward off bacterial infections and die sooner.[13] Their susceptibility to viral infection increases,[14] and exposure to high levels of NO_X for weeks causes emphysema-like changes in animal lungs.[15]

Many children aged twelve and younger who are exposed to NO_X have more respiratory illnesses.[16] Those exposed to high levels of NO_X outdoors have more colds that settle in their chests, chronic wheezing and cough, bronchitis, chest cough with phlegm, and episodes of respiratory illness.[17] When those exposures occur indoors—which often happens because NO_X is created by unvented gas-fired space heaters, furnaces and stoves, as well as kerosene and gas heaters—children can suffer from shortness of breath, chronic wheezing and cough, phlegm, and bronchitis.[18]

Animal studies indicate that nitrogen dioxide (NO_2) facilitates the spread of blood-borne cancer cells to the lungs. Animals exposed to the pollutant develop a significantly larger number of cancer colonies in their lungs and die sooner than animals breathing clean air. This may be because nitrogen dioxide damages blood capillaries and cells of the immune system. Since most cancer patients have circulating cancer cells, they may be at an increased risk of cancer spread merely by breathing outdoor air.[19]

The Next Tier: Doctor Visits, Hospitals, Heart Attacks

Increased bronchitis and pneumonia are linked to NO_X exposure.[20] So are respiratory symptoms such as chronic phlegm, chronic cough and breathlessness;[21, 22] duration of respiratory symptoms;[23] visits to medical clinics for lower respiratory tract illness such as bronchitis and pneumonia;[24] and, respiratory drug sales.[25]

The increases in illnesses are reflected by a rise in visits with the first line of the medical community, general practice doctors;[26] hospital transport for asthma, bronchitis, pneumonia, angina and heart attack;[27] and, hospital emergency visits for those aged 61 or older.[28]

Given that the blood levels of factors that increase clotting have risen, it is

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perhaps not surprising to find that NO_X exposure is linked to increased heart attacks. It is also associated with angina, the so-called "old man's disease" of acute chest pain triggered when some part of the heart fails to receive enough blood.[29] Other studies find irregular heartbeat,[30] as well as hospital admissions for heart attack.[31]

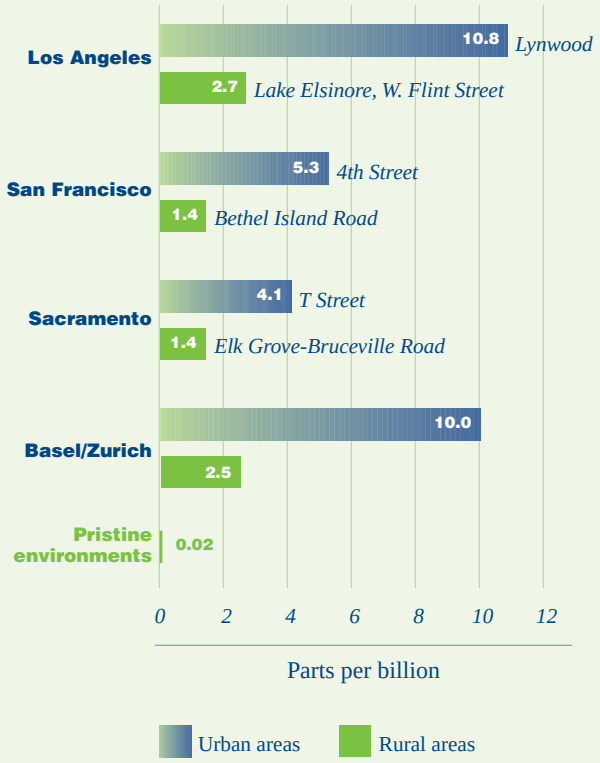
Development of Asthma

At least three studies have linked exposure to NO_X to the development of asthma.

In Taiwan, where asthma rates have more than tripled since 1973, scientists collected data from the national insurance bureau on 12,926 subjects from eight junior high schools, comparing the incidence rates of asthma with levels of various air pollutants. Asthma prevalence was linked to both NO_2 and ozone, while NO_2 and particulate matter pollution such as soot and dust were associated with monthly hospital admissions. The students also had declines in lung function, a measure of the ability to breathe normally, of 6 to 11 percent.[32]

In another study in Taiwan, 165,173 high school students aged 11 to 16 in two communities, Kaohsiung City and County, completed a video questionnaire. Some of the study regions were heavily polluted regions and others less so. (The highest NO_X levels were roughly one third of the maximum recorded in Los Angeles, about 50 percent lower than San Francisco's maximum and roughly the same as Sacramento concentrations.) Asthma prevalence varied at about 13 percent, but as levels of some pollutants rose, so did the disease frequency. It increased to 15.3 percent with higher levels of particulate matter, for example, and to 15.23 percent at NO_2 levels above 2.8 parts per billion.[33] This compares to a concentration of 10.8 parts per billion measured at Lynwood in Los Angeles.

In Montreal, Canada researchers recruited 457 asthmatic children three to four years of age at a hospital emergency room. A similar number of children without asthma were chosen and matched. Monitoring badges for the presence of NO_2 were worn by 20 percent of the children. Heavy smoking by the mother was correlated with development of asthma. There also was a dose-response relationship between NO_X and asthma, meaning that as levels of NO_X rose, so did the rate of the disease.[34]



Even in the least polluted rural areas, concentrations of NO_x are many orders of magnitude greater than they would be in the absence of industrial activity.

Aggravation and other Effects of NO_X on Asthmatics

Because of their compromised respiratory health, asthmatics are more vulnerable to pollution injuries.

In a study of 125 asthmatics in Australia, in children under age 14, exposure to NO_X was linked to tightness of the chest, breathlessness on exertion, and asthma attacks in day and night alike. In the adults, NO_X was associated with cough.[35] A London study analyzed data from 12 emergency departments. Daily visits ranged from 50 to 150, so the population base for this study was exceptionally large, thus increasing confidence in its results. Among its findings was an association between NO_2 levels and asthma visits by children that the researchers termed "particularly strong."[36]

Another large study, also in London, but of patient visits to general practitioners, reached similar conclusions. Examining the records of nearly 300,000 during the period 1992–94, researchers found that when NO_X levels rose, children's summertime doctor visits for asthma increased 13.2 percent one day later. In winter, visits by children for lower respiratory disease jumped 27.2 percent. Asso-

ciations with other pollutants were also found, but the link to NO_2 was the strongest.[37]

Deaths

A number of studies point to a link between NO_X and premature death.

Researchers analyzed 109 studies of air pollution and mortality from throughout the world: For every 24 part-per-billion increase in levels of NO_2, mortality from all causes rose 2.4 percent. The death rate increases for respiratory causes were even higher. They concluded that the "synthesis leaves little doubt that acute air pollution exposure is a significant contributor to mortality."[38]

In Santa Clara County in California, which is in the San Francisco Bay area, scientists examined eight years of data, comparing mortality with air pollution levels, attempting to determine whether federal standards that are supposed to protect America, in fact, do so. They found a link between pollution and deaths not only in the late 1980s, when the Bay area violated the standards, but continuing into the 1990s, when the mandates were being met. The strongest association was with particles, especially those smaller than 2.5 microns (about 1/50th the width of a human hair) and a nitrogen-based pollutant, ammonium nitrate. The link between pollution and deaths "calls into question the adequacy of national standards for protecting public health," they said.[39] Similarly, in Vancouver, British Columbia, a city with relatively low levels of air pollution, when researchers compared mortality with air pollution, wintertime levels of NO_2 were linked to death.[40]

In London, when levels of NO_2 rose, so, too, did deaths from cardiovascular reasons.

In London, when levels of NO_2 rose, so, too, did deaths from cardiovascular reasons, as well as pneumonia mortality in the elderly.[41] In Rouen and Le Havre, France, when NO_2 increased, there was a 6.1 percent jump in cardiovascular deaths.[42] Examining medical insurance files in Korea that covered 96 percent of the population, researchers found NO_2 connected to deaths from congestive heart failure.[43] In Seoul, a different study found NO_2 linked to acute stroke deaths.[44] In Barcelona, patients with severe asthma—that is, those with more than one admission to the emergency department for an asthma exacerbation—had a higher risk of dying on days with higher levels of NO_2.[45]

But is it NO_X?

Despite this body of evidence, the nagging question remains: is this litany of ills all due to NO_X? Or is the true cause one of the other pollutants almost invariably found with NO_X?

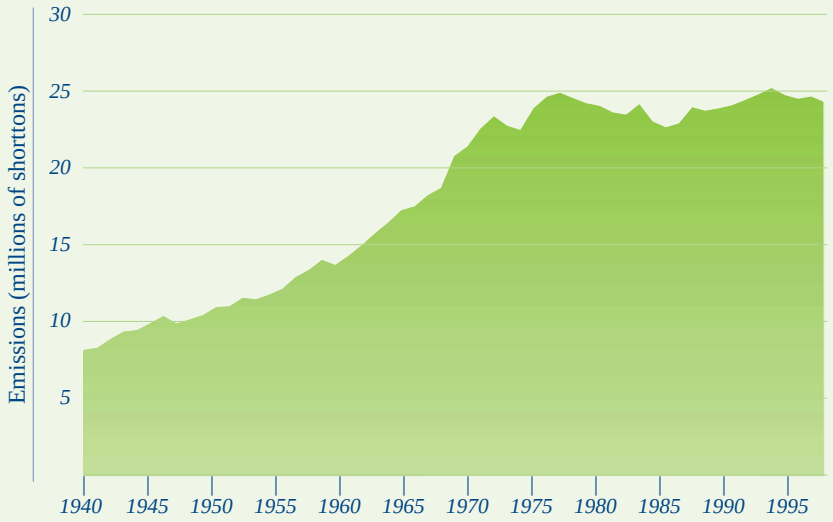
Clearly, when NO_X is present, so are other pollutants, especially particles, whether the place is an office or home,[46] a German city,[47] a Paris taxi[48] or a U.S. patrol car.[49] Indeed, several studies have found that NO_X is an excellent indicator of traffic pollution—so much so, in fact, that it is legitimate to ask whether the illness and injuries associated with NO_X are in reality due to the mix of car and truck emissions.[50]

Nevertheless, there are two important points that emerge from a review of the studies:

First, NO_X is, in and of itself, a threat to human health.

- The data from the studies of children cannot be dismissed and probably indicate that higher NO_2 exposure commonly has the effect of impairing lung defenses to common infections.
- The long-term studies probably indicate that higher NO_2 exposures can

Trend in Oxides of Nitrogen (NO_x) Emissions
1940-1998



After increasing steadily until 1975, NO_x emissions have stubbornly remained essentially unchanged, despite a roughly 90 percent reduction from cars on a per mile basis.

lead to a chronic low-grade respiratory bronchiolitis, impairing the expiratory flow rate; this effect is reversible if the level of NO_2 exposure is reduced.

- Acute exposures to high levels of NO_2 may cause changes within the lung that, in turn, could increase bronchial responsiveness, particularly in asthmatics.
- Any inhaled substance that can cause an inflammatory response, and this includes NO_2, will enhance the impacts of and susceptibility to allergens.
- It is possible that there is some sort of interaction between NO_2 and inhaled particulate material so that when both are present, the effect on lung defenses is greater than if either were present alone. As the respiratory bronchiole is a target organ in both cases, such a suggestion has considerable biological plausibility.

Second, the pollutants that NO_X creates—ozone, fine particles and acids—require aggressive controls to minimize death and serious irreversible illness.

Because the Clean Air Act has implemented targets specific to individual air pollutants and because the case against NO_2 has been confused, effective national controls on NO_X have been staved off by polluting industries, even though there is no doubt whatsoever that reducing emissions would save lives, reduce human illness and avoid injury to forests, lakes, streams and the climate. (California has been the notable exception to this rule, but it operates under its own California Clean Air Act.)

NO_X thus presents what some might consider a difficult question: in a case in which demonstrating an unequivocal cause-and-effect relationship between a specific pollutant and a given adverse health effect may be beyond the ability of science, what is the appropriate policy? Under current law, there is no affirmative action taken to reduce emissions. This policy thus places two burdens on the public (or, acting on the public's behalf, the government): first, that of proving that a pollutant causes injury; and, second, the risk of injury until and unless a cause-and-effect relationship can be established. Those who create the risk, and therefore the need for costly and time consuming studies, bear no burden at all, whether it is to reduce emissions or to demonstrate that their pollution poses no threat to human health or the environment.

Effective national controls on NO_x have been resisted by polluting industries, even though there is no doubt that reducing emissions would save lives, reduce human illness and avoid injury to forests, lakes, streams and the climate.

Europe has recently responded to this dilemma by articulating and adopting the "Precautionary Principle" (see "For Expert Readers"). The essence of the Precautionary Principle is very simple: better safe than sorry.

Suggestions that the Precautionary Principle be adopted in North America have incited vitriolic attacks. Two Canadian agricultural researchers, for example, termed the approach "Environmental McCarthyism" and compared it to the "trial by accusation" of the Inquisition of the Middle Ages, in which a mere charge of wrong doing was sufficient cause for execution.[51] The president of the U.S. Electric Power Research Institute, Dr. Chauncey Starr, attacked the principle as a "rhetorical statement that provides government a public welfare masquerade ...motivated by political pressures."[52] Instead of the Precautionary Principle, he believes that "in areas of public health and safety, comparative benefit/cost/risk analysis of all options should provide the judgmental base for decision making." Yet how does one conduct such a cost-benefit analysis when, by definition, science is incapable of identifying with precision all of the benefits?

The Bush Administration has said that NO_X "contributes to death and serious respiratory illness...acidifies surface water, reducing biodiversity and killing fish...damages forests...damages forest ecosystems, trees, ornamental plants, and crops...contributes to coastal eutrophication, killing fish and shellfish...contributes to decreased visibility (regional haze)...speeds weathering of monuments, buildings, and other stone and metal structures."[c] Yet in Washington, there is scant discussion of either immediate or substantial reductions in emissions. Indeed, Jeffrey Holmstead, E.P.A.'s assistant administrator for air, announced on August 27, 2003 a relaxation of federal law, that in effect, repeals what is known as new source review.[53] "For the first time," reported *The Washington Post*, "thousands of aging coal-fired power plants, oil refineries and factories will be able to upgrade their facilities—and extend their operational lives—without having to install costly anti-pollution equipment previously required under the Clean Air Act."[54]

As Heraclitus observed, the one constant in life is change. In the case of emissions of oxides of nitrogen, what is changing is the already immense toll on humanity and the environment—and for the worse.

[c] U.S. EPA, "Overview of the Human Health and Environmental Effects of Power Generation: Focus on Sulfur Dioxide (SO_2), Nitrogen Oxides (NO_X) and Mercury (Hg)," http://www.epa.gov/air/clearskies/pdfs/overview.pdf

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

ATTITUDES TOWARD ENVIRONMENTAL RISKS

We have, I believe, slowly been learning what lies behind the very diverse attitudes to risk that the public displays. This has become an important question since we have learned that we have to make judgements when the question involves a lot of uncertainty and cannot be resolved on purely "scientific" criteria. In such questions, no one can claim to be without bias; what is important is what predisposes people to align themselves closer to one or other poles of opinion on questions that are not amenable to a judgement based wholly on scientific data.

Recently, it has been suggested that as a society, we should adopt a "Precautionary Principle" when considering such questions. This idea apparently arose in Germany, and quickly became important in influencing attitudes and decisions in Europe. Last year, Rosner and Markowitz, from the Center for the History & Ethics of Public Health, Columbia University and the CUNY Graduate Center, New York, discussed the history of this principle in *Public Health Reports*.[55] They noted that the Business Roundtable founded in 1972 as an association representing 200 of the largest corporations in the U.S., vigorously opposed the Kyoto protocol, arguing that no policy commitments should be made until the economic consequences are thoroughly understood, and equating Precaution with economic and social stagnation.

A group of authors from the University of Massachusetts[56] laid out the basis for the Principle in a review published in *Environmental Health Perspectives*. They note four principal components of the Precautionary Principle:

1. Taking preventive action in the face of uncertainty;
2. Shifting the burden of proof to the proponents of an activity;
3. Exploring a range of alternatives to possibly harmful actions; and,
4. Increasing public participation in decision-making.

Recently, the President of the Electric Power Research Institute, Dr. Chauncey Starr, published in the journal *Risk Analysis* a spirited attack on the use of the Precautionary Principle.[57] He notes the strengths and weaknesses of adopting the Precautionary Principle as a basis for decision-making. In his view no such verifiable principle exists; "It is a rhetorical statement that provides government a public welfare masquerade for an indefinite deferment of a long-term policy response, or allows the deferment of disclosure of near-term actions motivated by political pressures." Instead, he recommends a "comparative benefit/cost/risk analysis, but notes "such early risk analyses have pragmatic uncertainties based on the limited available knowledge base and, accordingly, require judgmental application."

He also provides an interesting analogy to the Belmont 2002 horse race.

Whenever one looks up an entry in a dictionary, one usually finds that all meanings of the term are described. Starr is surely wrong in asserting that in the Precautionary Principle, the concept of "principle" is incorrectly used. My *Oxford Dictionary* notes that the use of the word Principle—in the sense of a personal philosophy that defines conduct—goes back at least to Oliver Cromwell, who apparently remarked: "If I were to choose any servant, I would choose a godly man that hath principles...." Furthermore, in physics, the word is used to characterize a "fundamental quality or attribute." Therefore, I can find no reason why, in defining the "Precautionary Principle," the word "Principle" has been misapplied.

What is being discussed surely is the attitude of mind we should, in the light of our recent past experience, bring to questions affecting the protection of public health. The Rosner and Markowitz article outlines the century of struggle preceding the modern arguments over the principle of precaution, in terms of protecting public health. Their analysis involves lead, silica, and vinyl chloride, documenting the fact that the hazards were well known to industry which, nevertheless, took

(continued on next page)

Worth Noting

Poisoned by Ethyl

When Ernest Oelgert ran shrieking from three imaginary figures, they knew something was wrong. That was October 21, 1924, when Oelgert first displayed this sign of lead-induced dementia. He was followed four days later by William McSweeney who was hauled from his home in a straitjacket. Then William Kresge threw himself out of a second story window. Walter Dymock was locked in a nearby home for the violently insane and died.[59] All worked at a Bayway, New Jersey refinery dubbed by *The New York Times* the "House of Butterflys" because of the hallucinations suffered by the workers, two-thirds of whom either died or were severely poisoned. The toxin was, tetraethyl lead, a new DuPont additive for boosting the octane of gasoline.

If ever there were a case for application of the Precautionary Principle, or erring on the side of safety (see "For Expert Readers"), this was it. In the wake of the Bayway poisonings, leaded gasoline, or "Ethyl,"[60] was removed from the market as the U.S. Surgeon General convened a blue ribbon panel to review the chemical's safety. Then, nine months later Ethyl was returned to the market, over the protest of some scientists who warned that the fuel might cause "chronic degenerative diseases."[61]

That warning proved prescient. Four generations of children have lost a portion of the essence of their humanity, intelligence, to the poison. Now, a new study demonstrates that it destroys intelligence even at levels below 10 micrograms (10 µg) per deciliter, now considered "safe." The five-year study determined that as concentrations of lead in the blood rose 1 to 10, IQ declined by 7.4 points.[62] Precaution in 1924 would likely have changed human history for the better.

Dangerous burning

Some of the most contentious decisions can surround the risks posed by waste incinerators. Frequently, proposals to build or operate an incinerator will be resisted by citizens concerned by the effect that burning might have on them and, especially, their children. Confronting these citizens will typically be a broad range of other interests, often including the government itself, sometimes condemning the attitudes of the citizens as knee jerk "not in my back yard," or NIMBY, environmentalism. To those caught up in such disputes a study from France may be of interest.

The study analyzed data from Besancon where an incinerator burned 67,000 metric tons of waste in 1998. Measurements of exhaust gas found levels of 16.3 nanograms (ng) of "international toxic equivalency factor," or TEQ, which is a way of sorting and ranking the danger posed by various poisonous chemicals. This is 163 times the European TEQ guideline of 0.1. In addition, the modeled dioxin exposures varied in different locations by a factor of 16 (<0.001 pg/m^3 to >0.004 pg/m^3 to a high of 0.0016 pg/m^3). More to the point, there was solid evidence of damage from the emissions: in those exposed to the incinerator's plume, the incidence of non-Hodgkin's lymphoma was over twice that of those not exposed.[63]

exceptional steps to prevent the scientific facts from being known. They note that Elizabeth Whelan of the American Council on Science and Health, which is mostly supported by industry, urged "Americans to recognize...the dramatically unpleasant side effects that a continuing embracing of environmental alarmism will have for our country."

The work of Prof. Paul Slovic has thrown important light on factors that influence where we individually choose to stand in such a spectrum.[58] Focusing on experimental work, he has shown, for example, that men and women differ in their perceptions of a wide range of risks, and that white Caucasian men tend to have a lower estimate of risk than white Caucasian women or African-American men. Race and sex are unlikely, however, to explain the hostility of Elizabeth Whelan and Chauncey Starr to the application of the Precautionary Principle. It is probable that some factor other than mere dialectics predisposes them to prefer public risk to private prudence.

Congratulations!

The Newsletter's Co-editor, Dr. David Bates was recently inducted into the Order of Canada, the highest award that can be bestowed on a civilian. His citation is below (and we could not have said it better ourselves). Congratulations!

David V. Bates, C.M.Vancouver, B.C.Member of the Order of Canada

He is a pioneer in the field of respiratory medicine in Canada. Founder of the country's first respiratory division at Montreal's Royal Victoria Hospital in the 1950s, he modernized the treatment of lung diseases. He helped to revolutionize the diagnosis of conditions such as chronic bronchitis and emphysema by demonstrating the importance of testing the lungs' ability to function. Committed to the study of the effects of air pollution on human health, he has constantly strived to have medical research influence public policy. He is an acknowledged leader in issues regarding air quality and has lent his expertise to several national and international committees and commissions.

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