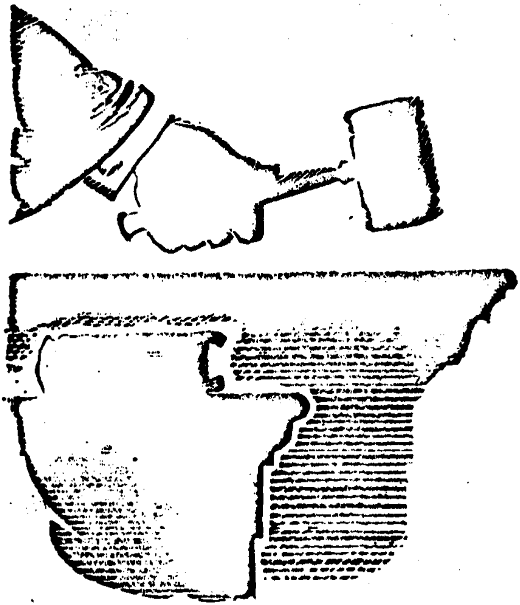
Guilty until proven innocent

Carcinogens in the environment



by ROBERT PAEHLKE

illustrations by ERIC NASMITH

Cancer is never an easy subject to confront. Given the choice, most of us would prefer not to think too much about the disease which has become the number two killer in Canada. Sometimes its causes are unknown. But research *has* identified certain carcinogenic chemicals at large in our environment. In fact, it is now generally acknowledged that the great majority of cancer cases are environmentally caused. Can there by any chemical which is so important to our way of life that we cannot make do with less of it, or simply do without it?

Global concern among environmentalists regarding PCBs (polychlorinated biphenyls) began in 1966 when a Swedish scientist found trace amounts in fish from the Baltic and in the feathers of museum eagles captured before 1944. Through the late 1960s dozens of scientific papers were published on the effects of PCBs on birds, on fish and on other animal species. In 1969 Coho salmon in Lake Michigan were found to contain five ppm (parts per million) PCBs and were banned for eating. The then-leading manufacturer of PCBs, Monsanto Chemical, voluntarily reduced production from 34 thousand tonnes in 1970 to 16 thousand tonnes in 1971. In 1972 the company restricted PCB use to confined systems. Seemingly action was swift and effective.

PCBs, it is now generally accepted, are carcinogens. They are still present in the water and fish of the Great Lakes. Today we debate whether to burn them in Mississauga cement kilns; traces are sometimes present in the oil used on county roads; a Regina 'storage depot' recently leak-

ed PCBs into surrounding soil. One is left wondering whether the action taken in response to the PCB danger, whatever it seemed at the time, was, in retrospect, adequate.

Barry Commoner in a milestone *Environment* magazine article published in 1973 revealed a hidden side to the history of PCBs. PCBs were first manufactured in 1929 and the first occupational health incident was reported in 1930. In Commoner's words: "By October, 1933, 23 of the 24 men in the (manufacturing) plant suffered with chloracne (a severe skin disorder)." Shortly thereafter it was found that PCBs affect liver function and in 1945 the New York department of labour advised that PCB exposures be "controlled insofar as humanly possible." PCBs in effect had been tested on human beings, failed the test, and then were put into mass manufacture. In the period between 1940 and 1968 PCBs found their way into the wider environment.

Alas, the PCB story is not unique. In fact the history of asbestos, benzene, vinyl chloride monomer (VCM) and dozens if not hundreds of other hazards is similar in many ways. In the period during or immediately following World War II total production of many hazardous substances grew rapidly as the number of industrial and consumer uses for each substance proliferated. Many of these substances were found to be occupational hazards before they became or were discovered to be hazards in the wider environment. Almost none had been tested for safety prior to their introduction into the workplace. From the workplace they entered the national and international productive process, consumer products and ultimately, in perhaps small quantities, the wider environment. Studying the history of carcinogens leads one quickly to what should perhaps be the new first principle of environmental protection: EVERYTHING LEAKS.

This principle means that we must simply accept that any substance used in large quantities will one way or another, sooner or later, be released in some amount into the environment. So-called 'contained' uses are a myth. The 'sealed' condensers in fluorescent light fixtures (which contain PCBs) ultimately go to the dump, rust, and spill their contents. Production facilities age, warehouses burn down, byproducts and waste products are never perfectly handled, all known modes of transportation have unanticipated accidents, older workers become habituated, newer workers are imperfectly trained, and many consumers pay no attention to instructions regarding use and disposal. Small amounts are released — and in nature remarkably dilute amounts of some substances can return to haunt us in unexpected ways. If we are to keep carcinogens in the environment to an absolute minimum we must learn to detect their hazardous character *prior* to mass manufacture. We must straightforwardly limit the development of some industries by simply disallowing the production and use of carcinogenic substances.

Before taking a closer look at this assertion, we should consider why we have not acted more effectively in the past. How have we come to the point where we cannot eat the fish of many of our finest waters? Here are five reasons.

First, we as a society are not clear that, when we must choose, we prefer health to ever greater wealth. This generalization has many manifestations in the way we live and work. Many corporations do everything in their power to avoid regulation of their production and use of carcinogenic substances. Our agriculture ministries openly encourage the extensive use of chemical biocides as, on balance, necessary and safe when the evidence is not as clear as it might be. Medical agencies and the medical profession have been extremely slow in shifting their efforts from cancer cure to cancer prevention. Many workers and unions have hesitated to push occupational and community health concerns to the forefront of their bargaining demands.

Second, environmentalists for the most part have not considered occupational health to be a central concern. This, of course, is the real meaning of Barry Commoner's article. Today's occupational health problems are tomorrow's environmental problems. If we are serious about preventive as opposed to reactive environmental protection, Canadian environmentalists must orient their concern towards the Canadian workplace.

Third are the difficulties arising from the considerable time lag between exposure to a carcinogen and onset of visible health effects. This period is in most cases from 15 to 30 years. Given that sort of delay it is extraordinarily difficult to pin down the several contributory causes of disease. In the case of asbestos it took decades to establish conclusively the extent to which the substance is hazardous.

Fourth, it is only very recently that we have been able to measure the presence of some carcinogens at dilutions where they have toxic effects. With this knowledge we have come to dismiss the myth that there are safe thresholds for carcinogens in the workplace or in the environment. The greater the dilution, we have learned, the fewer the cancer cases, but it is likely there are no measurable levels at which a carcinogen will not affect some individual.

Fifth, cancer strikes on a 'statistical basis' and this allows human nature the opportunity to ignore the possibility of personal or family impact. Some individuals, for reasons we do not understand, can both smoke heavily and live and work in the most hazardous of communities and industries and never seemingly be affected. All we know is that it is 'statistically' safer to avoid exposure to certain substances.

For these reasons we have come to the point that it seems every week we learn of a new carcinogen at large in the environment. Some carcinogens

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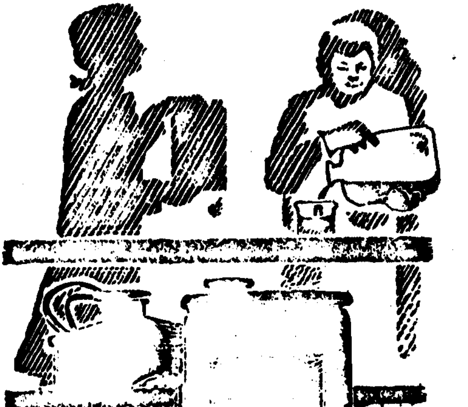
have been there for a long time. For example, polycyclic aromatic hydrocarbons are the product of the incomplete combustion of any organic matter from tobacco to fossil fuels to wood. Other carcinogens are deliberately dispersed into the wider environment. For example, 19 of 25 organochlorine pesticides are known to be carcinogenic. Many of these pesticides are still produced and used in large quantities. Kepone, DDT and Mirex are all organochlorine pesticides. Other carcinogens are strictly post-World War II phenomena. For example, Radon-222 or Radium-226 are by-products of uranium mining, milling and processing. Radium-226 seeping from the Elliott Lake mining complex has contaminated the Serpent River system of Northern Ontario. Dioxin, one of the most potent toxins known, has been widely dispersed as an impurity within the herbicide 2,4,5T. —

Generally carcinogens are produced or encountered in the asbestos, plastics, petrochemical, paint, pesticide, rubber, metal smelting, solvent, welding and mining industries. Often these industries exercise a great deal of caution with these dangerous substances. Sometimes they do not. Often workers are not as cautious as they might be because they are not able to learn, try as they might, the dangers associated with the substances with which they work. And many workers, being human, are hesitant to find out the full truth of what may be happening to their health. Carcinogens are carried home on workers' clothes and cars; small amounts are flushed down drains in the process of 'cleaning up'; residues are removed with 'empty' containers. Releases occur when safety regulations are not fully followed. These 'errors' notwithstanding, probably the majority of carcinogens in the environment are released in the 'normal' functioning of the industrial process. Releases occur either prior to any knowledge of danger or prior to corporate acceptance of that knowledge. These are generally very different points in time; the communication and decision process from independent research to expensive action is a complex one.



Industrial releases, then, are ordinarily small and the substances are rapidly diluted by air or water to ppm, ppb, or ppt levels. Some few carcinogens are highly dangerous even at these levels. Most carcinogens at these levels affect only a very small number of highly susceptible individuals. Sadly one cannot effectively predict in advance which individuals are susceptible and therefore precautions cannot be taken. Nevertheless it is argued that low level, localized dangers associated with certain industries are a tolerable 'price of progress'. This argument collapses in the face of the alterations that dynamic nature adds to the equation. Four key natural processes within living ecosystems wreak havoc with this all too commonly put argument.

The first process is bioaccumulation — the building up of toxic substances in food chains. In *Silent Spring* Rachel Carson recounted the story of Clear Lake, California. The lake was treated in 1949, 1954 and 1957 with DDD, an organochlorine insecticide at .02 ppm in an anti-mosquito campaign. After a time the

DDD residue in the water was so low that it was not detectable. In the plankton, however, the DDD concentration mounted to 5 ppm. In small plankton-eating fish, concentrations reached 40 to 300 ppm, and in larger (fish-eating) fish they reached 2500 ppm. DDD is fat-soluble (as are PCBs and many other carcinogens) and accumulates in the fatty tissues of fish. At the top of the food chain at Clear Lake were the grebes who, at 1600 ppm, were eliminated from the Clear Lake region.

It is not necessary to look far to find other examples. Cows graze up to 50 sq metres of field in a day and build up a large portion of the contaminants thereby gathered in their milk. Molluscs filter enormous quantities of water and some can, for example, concentrate heavy metals by a factor of 300,000 and organochlorine insecticides by 70,000 times over their concentration in the water. PCBs are highly susceptible to such bioamplification. By the time PCBs reach carnivorous sea mammals, having been concentrated ten to one hundred-fold at each step in the chain, they may be concentuated ten million-fold over levels in the surrounding water. Human diets which concentrate heavily on the lower end of food chains are clearly prudent.

The second process, natural resistance, is related to the first. Lower-order species breed in massive multiples and eventually shrug off concentrations of toxins to which higher-order, slower breeding, longer living species — such as humans — cannot adapt. The plankton and fish of Clear Lake seemingly remained healthy while the grebes disappeared. One can eliminate 99 per cent of a given insect population with an insecticide, but those remaining, and their millions of descendants, may be resistant to the toxin which killed their siblings. The mosquitoes of the Mississippi Delta ignore doses of poisons 120 times stronger than those which once would have killed them. We who constantly up the dosage of all deliberately spread toxins are among the most vulnerable of species.

The third process is natural dispersion. High concentrations of DDT have been found in penguins and polar bears living great distances from any possible direct source of the substance. Toxins are dispersed in the environment in a wide variety of ways. They travel in the wind and are washed out in the rain. Those buried on land are leached out by water. All ultimately travel the watercourses and ocean currents, and migrate within the bodies of fish, birds and mammals. Many species, having picked up toxins, migrate thousands of kilometres before becoming dinner for a member of another species. A carcinogen that is persistent will soon find its way to the ends of the earth. Once in the environment persistent substances cannot be retrieved.

The fourth process is the biochemical interaction of toxic substances in nature. Chemical reactions occur in nature without human permission, plan or even awareness. Metallic mercury was converted to harmful methyl (organic) mercury in the bottom mud of Canadian rivers and Japanese bays. In addition to such chemical changes, synergistic and catalytic chemical and biological effects occur within the human body. Asbestos and cigarette smoke are more than additively carcinogenic; the substances seem somehow to enhance each other's deadly effects. Some chemicals are carcinogenic primarily in combination with other chemicals: the pesticide kepone and alcohol are suspected of acting in this manner. We have hardly begun to investigate interactive effects for either the workplace or the wider environment. We may never understand them fully.

Together these four processes distinguish persistent carcinogens and other toxins from other forms of pollution. They must, then, be treated differently legislatively and administratively. As we have seen, these substances are dangerous even if highly diluted in air or water. They can, as at the Love Canal, turn up as extreme hazards decades after being buried, seemingly carefully, in the ground. Those locally released can spread throughout the globe and can interact with other substances in unknown ways.

The burden of evidence regarding all persistent substances must be reversed. Any hint of danger certifies guilt; innocence must be proven to a tough jury. In the case of carcinogens in particular there is the compounding problem of long delays between exposure and health impact. This is merciful to the individual, but complicates detection of danger enormously. For all the reasons discussed above we simply cannot wait un-

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til more carcinogenic substances are at large in the environment for certain proof. The only humane and rational way to proceed is in a preventative manner. The best preventative technique involves the acceptance of guilt on indicative evidence unless innocence is demonstrated irrefutably.

Animal tests and such tests as the Ames bacterial mutagenicity test give us the technical ability to screen potentially hazardous substances from our workplaces and thereby from the wider environment. Perhaps nine of ten substances carcinogenic in humans can be detected using these techniques. The Ames test is quicker and less expensive, but less reliable than animal tests. Animal tests should be used for every suspect chemical and all new chemicals introduced into workplaces. Both types of tests will occasionally condemn relatively harmless substances. Considering the risks run in not acting forcefully this is a small price to pay. Substances found suspect should not be produced. If there are compelling arguments for exceptions let them be made. Even so hazardous a substance as asbestos, when used as a fireproof material in construction or in firemen's coats, might well be excepted. These uses, by the way, do not account for the largest portion of asbestos production. A good case for yet to be utilized substances would be harder to make, and a case for substances for which less harmful substitutes can be found would be impossible to make other than for brief transition periods.

Back-up to the screening tests could come from large scale epidemiological (disease distribution) studies built into the national health care system. These studies can and must be done in a way that protects the anonymity of all individuals. It should be easy and cheap to correlate medical, life history and occupational data and detect carcinogens that get by the initial screening process. This sort of research effort would be at least as productive a long term investment as research on surgical or chemical cancer cure techniques.

Beyond research we need to encourage new legislation. The research is almost pointless if we continue to allow carcinogenic substances to be produced in large quantities. Another approach to the problem might be the development of legislative and legal ways to make it easier for cancer victims and their surviving relatives to gain compensation from the producers of carcinogens. Those who benefit from the production of carcinogens should at least bear the monetary costs of the disease. The Japanese have a law called "Compensation of Pollution-Related Health Damage" which is a useful beginning in this area. If the real cost of the production and release of carcinogens were even partially borne by the producers they would soon find their products priced out of the market.

Finally, workplace union-management health committees should be supported by environmentalists, scientists and the medical profession. Those committees should be provided access to the latest in scientific and medical knowledge. Protecting worker health today could save the health of all our children tomorrow.

Cancer, as the slogan goes, can be beaten. But the solution is at least as much political, legal and economic as it is medical. □

Robert Paehlke is an associate professor of political studies at Trent University in Peterborough, Ontario.