Preventive environmental health strategies: from laboratory studies to public health practice.

Sánchez L, Espinosa-Torres F, Jiménez C, Cebrián M, Waliszewski S, Saldate O. Is DDT use a public health problem in Mexico? Environ Health Perspect 104:584–588 (1996).

Eriksson P, Archer T, Fredriksson A. Altered behaviour in adult mice exposed to a single low dose of DDT and its fatty acid conjugate as neonates. Brain Res 514:141–142 (1990).

Eriksson P, Nilsson-Hakansson I, Nordberg A, Aspberg A, Fredriksson A. Neonatal exposure to DDT and its fatty acid conjugate: effects on cholinergic behavioural variables in the adult mouse. Neurotoxicol 11:345–354 (1990).

Eriksson P, Ahlbom J, Fredriksson A. Exposure to DDT during a defined period in neonatal life induces permanent changes in brain muscarinic receptors and behaviour in adult mice. Brain Res 582:277–281 (1992).

ATSDR. Toxicological Profile for 4,4’-DDT, 4,4’-DDE and 4,4’-DDD. (U.S. H.H.S.-A.T.S.D.R.-Draft, 1993). Atlanta, GA:Agency for Toxic Substances and Disease Registry, 1993.

Kotter HBWM. Behavioural teratology of exogenous substances: regulation aspects. Prog Brain Res 73:59–67 (1988).

Rodier PM. Comparative postnatal neurological development. In: Prenatal exposure to toxicants (Needleman HL, Billinger D, eds). Baltimore, MD:Johns Hopkins University, 1996; 3–23.

Rodier PM. Critical periods for behavioral anomalies in mice. Environ Health Perspect 18:79–93 (1976).

Rodier PM. Time of exposure and time of testing in developmental neurotoxicology. Neurotoxico-cology 7:69–76 (1986).

Rodier P. Chronology of neuron development: animal studies and their clinical implications. Dev Med Child Neurol 22:525–545 (1980).

Ruppert PH. Postnatal exposure. In: Neurobehavioral toxicology (Annau Z, ed). Baltimore, MD:Johns Hopkins University Press, 1986; 170–189.

U.S. Geological Survey. Surface-water-quality assessment of the Yakima River basin, Washington: pesticide and other trace-organic-compound data for water, sediment, soil, and aquatic biota, 1987–91. USGS Report 92-644. Portland, OR:U.S. Geological Survey, 1992.

Waliszewski SM, Pardio Sedas VT, Infanzon RM, Rivera J. Determination of organochlorine pesticide residues in human adipose tissue: 1992 study in Mexico. Bull Environ Contam Toxicol 55:43–49 (1995).

Waliszewski SM, Pardio Sedas VT, Infanzon RM, Rivera J. Organochlorine pesticide residues in human breast milk from tropical areas in Mexico. Bull Environ Contam Toxicol 57:22–28 (1996).

**Lead in Drinking Water: A Preventive Solution**

Many children are at risk from lead (Pb) poisoning. One study found that one in five children in North Carolina had tested positive for elevated levels of Pb in their blood (J). In a separate study in Missouri, it was reported that 5.7% of 528 schools and 2.4% of 1,123 day care centers exceeded the EPA’s action level of Pb in drinking water (2). These numbers and conclusions justify major concern, and efforts to curtail Pb consumption should be rigorously investigated.

One source that certainly contributes to this widespread problem is permanently installed drinking water fountains (3); of notable concern are water fountains found in elementary schools (1,2,4). Many old school buildings probably contain Pb-contaminated supply pipes or Pb solder from which the Pb leaches into drinking water and is then passed into human tissues, causing various physiological and neurological damage. As water in these buildings rests in Pb-contaminated plumbing overnight, throughout the summer months, and during school vacations when there is little movement of water, Pb accumulates and levels increase, causing a potential health threat. However, leaching of Pb is unpredictable, and strategies for the elimination of it from drinking water have been difficult to develop and evaluate. Although various approaches have been devised to reduce Pb in water to safe levels, i.e., adding calcium carbonate and legislating stringent Pb piping standards, these endeavors are not sufficient for complete safety (3). Temporary efforts to reduce Pb concentration in drinking water include morning flushing of the water source or permanently installed water coolers (J), use of Pb filters, or switching to bottled water dispensed in free-standing coolers. It has been reported that one-time morning flushing of drinking water coolers in elementary schools may not provide day-long Pb exposure protection for children (4). Flushing is tedious and time consuming and offers only temporary reduction of Pb because, in many cases, the Pb leaches back into the water from Pb-contaminated plumbing; therefore, many people have switched to bottled water dispensed in free-standing coolers. Until recently, it was not known whether chemical contaminants such as Pb would accumulate in bottled water dispensed in free-standing coolers.

We have examined bottled water dispensed from free-standing coolers and found Pb levels to be less than 5 ppb without any evidence of Pb accumulation in water remaining in contact with the plastic plumbing materials and the stainless steel water reservoir cooling tank during periods of non-use up to 28 days (unpublished observations). These free-standing water coolers with plastic plumbing and a stainless steel reservoir tank may be one way to provide school children with Pb-free drinking water.

**Bill Jirles**

**Julius Thigpen**

**Diane Forsythe**

National Institute of Environmental Health Sciences

Research Triangle Park, North Carolina

**REFERENCES**

1. Mass RP, Patch SC, Gagnon AM. The dynamics of lead in drinking water in U.S. workplaces and schools. Am Ind Hyg Assoc J 55:829–832 (1994).

2. Gnaedinger RH. Lead in school drinking water. J Environ Health 55(6):15–18 (1993).

3. Davis WF. A case study of lead in drinking water: protocol, methods, and investigative techniques. Am Ind Hyg Assoc J 51:620–624 (1990).

4. Murphy EA. Effectiveness of flushing on reducing lead and copper levels in school drinking water. Environ Health Perspect 101:240–241 (1993).

**Calculation of Cancer Risk**

Recently the EPA proposed changes in how it determines which chemicals and pollutants cause cancers, relying less on animal tests and more on new techniques of molecular biology. Acknowledging recent advances in molecular biology and other fields, EPA’s new proposal would give more weight to a broad range of evidence, including details about precisely how toxic agents wreak their harm on human cells and on genetic material that control cells’ reproduction. By taking the mechanics of cancer into account, the new approach will more precisely measure a chemical’s cancer potential. At the same time, the new proposal opens the way for new statistical analyses about the effects that chemicals might have at very small doses that people are exposed to, rather than at very large doses given to animals to test their effects.

In summary, the EPA will rightly draw more on improved understanding of the mechanism by which toxic effects are produced. Over the years it has been recognized that the ultimate value of toxicological information relates to its use in the development of formal risk or safety assessments. Thus, a broad array of research is focused on the development of mechanistic information that will have value in assessing the potential human health risk of environmental pollutants and consumer products and assessing the safety of pharmaceuticals.

From this research, there emerged significant advances in our understanding of the mechanism of carcinogenesis, which justifies EPA’s effort to rethink cancer calculations. Among these advances are the following:

- Significant developments in science of how humans metabolize cancer-causing substances. Most molecules identified as carcinogenic do not produce their detrimental effects themselves. They have to be metabolized, usually into a form that can react irreversibly with sites on DNA, altering gene expression. The role of two important sets of enzymes—the
cytochrome P450 family and the glutathione S-transferase—are now well known from both human and animal studies to play an important role in the metabolism of carcinogens.

- A better understanding of the potential differences in a tumor suppressor gene pathway in chemically induced and spontaneous kidney cancer.
- An understanding of the different capacities of cells for DNA repair. Two types of DNA repair exist: repair pathways and a tolerance mechanism. In repair mechanism, the DNA damage is removed, while tolerance mechanism circumvents the damage without fixing it.
- Increased information on the role of viruses in carcinogenesis. For example, recent reports describe how an adenovirus that has a key gene deleted can no longer reproduce itself in normal cells, but does just fine in cancer cells lacking the p53 tumor suppressor gene. As a result, the virus kills the cancer cell, apparently without harming the normal cells.
- Advances in our knowledge of signal transduction that have led to major insights into the fundamental pathway that governs growth regulation of cells. These discoveries fulfill the long-sought ability to delineate a sequence of events from extracellular signals to nuclear responses. Additionally, key molecules in the pathway are evolutionarily conserved and mediated in an eclectically array of signals. Alterations in the pathway are important in tumorigenesis and tumor progression.
- Discovery that apoptosis—programmed cell death—is highly regulated at the molecular level by oncogenes and anti-oncogenes. Understanding the biochemical and molecular pathway that controls apoptosis is central to the cancer problem.
- The characterization of the relationship between peroxisome proliferators and cancer, including the role of cell receptors, DNA oxidation, and gene expression.
- A better understanding of the mechanism through which the leukenomagen benzene affects bone marrow. Metabolites of benzene are found to be highly concentrated in bone marrow of exposed animals.
- The elucidation of generic mechanisms of action common to multiple chemicals. These studies have included nitroaromatic compounds, automotive fuels, dioxin, butadiene, chloroform and chlorine, dimethylamine, ethylene oxide, and furans.
- Increased expertise in the development of methods for conducting carcinogenic studies, including numerous assays and techniques in a range of disciplines.

In 1996, there was evidence of high momentum in both privately funded and government supported research aimed at better understanding risks to human health from exposure to environmental agents. Clearly, environmental health research including risk assessment science is at a remarkable point in time. There is a wonderful record of accomplishments, and this accounting is only a partial list. There has been enormous progress against cancer, yet so much more remains to be done in improving the basis for understanding and assessing potential adverse effects of chemicals and consumer products on human health.

The more we know about the mechanisms involved in environmental chemical interactions with complex mammalian organisms, including both laboratory animals and humans, the more confident the public will become in estimates of human health risks, and the firmer the scientific foundation for environmental health policy formulation will become for the prevention and control of cancer and related diseases, dysfunction, and premature death. This policy and the related regulations and intervention programs shape our society and suggest priorities for investment of public health resources. The importance of these issues cannot be overstated, and the EPA appears to be moving in the right direction.

Bailus Walker
Howard University Cancer Center
Washington, D.C.

**Responsible Care and the Third World**

As you point out, many environmental health scientists remain skeptical that the chemical industry’s Responsible Care initiative is mainly a public relations ploy to improve the industry’s dismal public image [EHP 104:1138(1996)]. Nowhere is the challenge greater than in the rapidly industrializing countries of the Third World, where corporate responsibility is not compelled by public awareness, regulation, and compensation laws.

The double standards of global corporations that operate more polluting and dangerous plants in Third World countries were described not only by industry critics but also the International Labor Office and the United Nations Center on Transnational Corporations [1,2]. After the disaster at Union Carbide’s plant in Bhopal, India, giant chemical producers based in the United States and Europe have been obliged to issue policy statements to the effect that they do not have lower standards for the protection of human health and safety and the environment in their Third World operations.

When pressed, however, leading firms have been reluctant to disclose toxic release inventory data for pollution from their foreign plants as they have had to do by law in the United States since 1988. Similarly, U.S. law requires a process hazards analysis in the event of failure of safety systems, including worst-case accident scenarios—are the big companies willing to release similar analyses for their affiliates’ plants in Africa, Asia, and Latin America? What about meeting modern standards for disposal of hazardous wastes from plants located in countries with no facilities available for disposing of these wastes in a manner that would meet the standards the companies face in Europe and North America? Where a control limit is opposed by a corporation as unnecessarily strict, does the company comply with the limit outside the country where the limit is in effect but being challenged in court? If a pesticide is banned for certain uses or voluntarily withdrawn from markets in the United States, does that mean it will be similarly withdrawn elsewhere? If teratogenic glycol ether solvents are withdrawn from uses in the United States because of liability considerations, will they be withdrawn from sale in other countries where no such liability exists?

Product stewardship is the most challenging area that the chemical corporations have tried to address through the Responsible Care initiative. But closer examination has shown that, in 1991, DuPont's putative product steward was none other than the company’s sales representatives. Obviously salespeople have neither the incentive nor the training to critically evaluate the industrial hygiene and pollution control measures of their customers.

To some extent, industry is being forced to develop cleaner and safer processes and products in North America and Europe. Will the companies who are making these advances in some countries rapidly transfer them around the world? Or will the companies take many years to reformulate additives, sealants, and paints made with toxic solvents and heavy metal pigments in Third World countries?

Responsible Care does not deal with the very sensitive subject of compensation. Bayer has operated highly hazardous chrome facilities in Mexico and South Africa and many workers and members of the surrounding communities have been harmed. Lung cancer has been recognized as a compensable occupational disease in chromate workers in Germany since 1936, but this was not entered in the schedule of occupational diseases in South Africa until 1994. When a Natal doctor attributed some work-