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LETTERS

Tagging "Infiltrators"

It is an unwelcome sign of the times that Bill Wattenburg's Policy Forum "Fluorescent barriers to infiltration" appeared in *Science* (26 Aug., p. 1184), instead of in a military ordnance journal. Wattenburg proposes to block illegal immigration across the U.S.-Mexico border by aerial dusting with fluorescent chemicals, thereby tagging would-be "infiltrators" into San Diego. The danger in such a scheme is less in its dubious practical outcome than in its invitation to scientists to collaborate with xenophobia. Those of us who find human tagging distasteful, whether with fluorescent dyes or yellow stars, must question his premises.

Immigrants, legal or otherwise, have long been a convenient scapegoat for troubled economies. California's fiscal crisis stems from long-term decline in manufacturing, politically expedient tax cuts, and an overheated real estate market. The widespread belief, echoed by Wattenburg, that illegal immigrants are a drain on the economy and cause unemployment among U.S. workers has been effectively challenged by demographers. Politicians from David Duke to California Governor Pete Wilson obscure these realities by targeting undocumented workers, invariably those with dark skin. One product of this movement, the "Save Our State" proposition on the November ballot, would deny schooling and basic medical care to California's undocumented immigrants.

Experience suggests that high-tech gadgets are no match for desperate and determined people. During the Vietnam War, the "Jason" group of elite scientists arranged for 20,000 sensors of various types to be dropped on the Ho Chi Minh Trail to interdict Northern "infiltration." This "McNamara Line" was notoriously ineffective; reportedly, the North Vietnamese decoyed "people sniffers" by hanging bags of urine in the trees. We can expect similar resourcefulness from Mexican immigrants, who after all are not criminals, but impoverished workers seeking a better life for their children. The real impact of Wattenburg's proposal would be to lend credibility to anti-immigrant hysteria, at present the leading edge of "respectable" racism in this country.

Miriam Golomb
Division of Biological Sciences, University of Missouri, Columbia, MO 65211, USA

Environmental Estrogens

The Environmental Protection Agency (EPA) was pleased to read "Environmental estrogens stir debate" by Richard Stone (News & Comment, 15 July, p. 308), where it was stated that in "the debate over hormone-modulating pollutants," EPA has increased its emphasis "on the noncancer effects of the chemicals it regulates." This approach is characterized as "a fresh concern for EPA, which in the past has crafted regulations based mainly on chemical carcinogenicity." While EPA has indeed often emphasized chemical carcinogenicity, noncancer health effects are also considered in most of EPA's regulatory actions, and some regulations are based solely on effects other than cancer.

All of the statutes under which EPA regulates provide authority to regulate for noncancer health effects. For example, the Clean Air Act designates six criteria pollutants (lead, particulate matter, ozone, nitrogen oxides, sulfur oxides, and carbon monoxide). Under this act, EPA must set a National Ambient Air Quality Standard (NAAQS) for each criteria pollutant for the entire United States "which in the judgment of [EPA], based on such criteria and allowing an adequate margin of safety, [is] requisite to protect the public health" (*1*).

The NAAQSs are based on a range of observed health effects which include respiratory effects, cognitive and neurobehavioral effects, reproductive effects, and death. None of the ambient air quality standards are based on carcinogenicity. In addition, the Clean Air Act lists 189 hazardous air pollutants that are subject to National Emission Standards for Hazardous Air Pollutants. A large proportion of these pollutants are not considered to be possible human carcinogens and are instead regulated on the basis of noncancer effects.

In addition, health effects testing authorities under the Toxic Substances Control Act and the Federal Insecticide, Fungicide and Rodenticide Act require testing of industrial chemicals and pesticides for multiple health endpoints, including mutagenesis, teratogenesis, behavioral disorders, and carcinogenesis. EPA uses this and other information in its ongoing evaluation of noncancer toxicity and regularly sets reference doses (RfDs), which are threshold levels of safe exposure for noncancer effects. These are routinely used in regulatory decisions.

Finally, addressing chemical estrogenicity is not a departure from past EPA practice. In our efforts to protect public health, we consider results from many types of tests and studies examining the full range of health endpoints before making regulatory decisions. As our understanding of the effects of chemicals on human health and the environment evolves, our regulatory decisions will continue to incorporate the latest science. Thank you for your efforts to inform the public understanding of these critical issues.

David Gardiner
Assistant Administrator,
U.S. Environmental Protection Agency,
Washington, DC 20460, USA

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1. Clean Air Act, Section 109(b)(1).

Stone's article "Environmental estrogens stir debate" is well researched and well balanced. The differences of opinion are justified, but in some cases persuasive logic can be used to reach opposite conclusions. The "two points . . . arguing against a demonstrable link between hormone-modulating pollutants and human health effects," when examined closely, actually support the possibility of human health effects.

1) Basic pharmacology seldom can be counted on to simply "add up." The "very weak [environmental] estrogens" such as polychlorinated biphenyls (PCBs), with 100- to 10,000-fold lower affinities for estrogen receptors, are present in human blood at about 2 to 8 nanograms (ng) per milliliter (ml); estrogen occurs in cycles in human female blood in amounts of between 0.03 and 0.50 ng/ml, and most of this is bound to plasma proteins or conjugated to disarming groups, or both. Along with the PCBs are found DDE (the principal metabolite of DDT), mirex, methoxychlor, phthalates, and other pesticides and plasticizers in concentrations at least equivalent to those of endogenous estrogens. If the lesser binding and greater membrane transport of the weak estrogens are factored in, the pharmacology begins to add up.

2) The acid-base argument is the more frightening possibility. Certainly, if a precise combination of estrogenic and antiestrogenic chemicals is present, "the net effect may be zero" (if one ignores the other disrupting effects cited by Earl Gray and Richard Peterson). However, dioxin is (justifiably) one of the most targeted environmental contaminants, and progress has been made in attenuating environmental concentrations. As the balance which may have been in place shifts, estrogenic and other subtle toxicities may be unmasked. This is amply illustrated in the comparison of Mary Wolff *et al.*'s study of the association of DDE with breast cancer (*1*) with Nancy Krieger *et al.*'s study (*2*), which did not support the association. The exposures Krieger *et al.* found were from before 1970 and included higher DDT concentrations than those in the more recent exposures found by Wolff *et al.* (G. Taubes, News & Comment, 22 Apr., p. 499). Before 1970, however, we were only becoming aware of dioxin toxicity and could not even detect it near the concentrations of today's regulations. Therefore, products such as 2,4,5-T, hexachlorophene, and pentachlorophenol (which were banned before Wolff *et al.*'s exposures were found) contained higher concentrations of dioxin. Dioxin is more potent as an antiestrogen than DDE is as an estrogen; therefore, the lack of association between DDE burdens and breast cancer before 1970 and the positive association after 1985 should, perhaps, have been expected. The below-normal incidence of breast cancer and endometrial cancer in the (dioxin-exposed) population near Seveso, Italy, *was* expected (*3*).

This is a small sample of the complicating factors among the environmental hormone disrupters and the endogenous hormones and neurotransmitters. Even if, as Stephen Safe has stated, "the [PCB] problem is under control" (R. Stone, Research News, 14 Feb. 1992, p. 798), the "other environmental problems" Safe referred to in 1992 all involve interactions. If we can successfully describe and quantify some of these interactions so that we can successfully predict the net biological effects of even a few simple mixtures, then the risk assessment of environmental mixtures, and thus the health and welfare of humans and wildlife, will have been well served.

Larry G. Hansen
Department of Veterinary Biosciences,
University of Illinois,
2001 South Lincoln Avenue,
Urbana, IL 61801, USA
Heiko T. Jansen
Department of Cell Biology,
Neurobiology and Anatomy,
University of Cincinnati,
231 Bethesda Avenue,
Cincinnati, OH 45267–0521, USA

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1. M. S. Wolff *et al.*, *J. Natl. Cancer Inst.* **85**, 648 (1993).
2. N. Krieger *et al.*, *ibid.* **86**, 589 (1994).
3. M. Holloway, *Sci. Am.* **270**, 25 (January 1994).

Stone reports on two recent epidemiologic studies of whether DDT exposure relates to a risk of breast cancer. In the first of these studies (*1*), one of us (M.S.W.), with Paolo Toniolo at New York University Medical Center and others, found strong evidence

for an association between the body burden of DDE and breast cancer risk in a population of primarily Caucasian women. For women with the highest body burdens of DDE, the risk of breast cancer was found to be four times higher than for those with the lowest burdens. In the second study (2) by Nancy Krieger and her colleagues at the Kaiser Foundation Research Institute, no association was found between DDT and breast cancer when women of all races were examined together. However, a positive association (although not statistically significant) was observed between DDT and breast cancer risk in Caucasian and African-American women. Among Asian women there was no evidence of an association.

There are several possible mechanisms by which DDT may increase risk of breast cancer. Hormonal modulation (estrogenicity and antiestrogenicity) is only one possibility. Another is induction of cytochrome P450 enzymes. These mechanisms may act independently or in concert with one another. Moreover, whatever the mechanisms of action, the carcinogenic potency of DDT and its metabolites may be enhanced by the persistence of these compounds in the human body.

To determine whether the apparent association between DDT and breast cancer risk is real, further epidemiologic investigations as well as complementary mechanistic studies must be carried out. The ultimate hope is that this research may identify a preventable environmental cause of breast cancer in women.

Mary S. Wolff
Philip J. Landrigan
Mount Sinai Medical Center,
One Gustave L. Levy Place,
New York, NY 10029–6574, USA

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Genetic Testing and Insurance Costs

In his letter about genetic testing and the costs of insurance (9 Sept., p. 1509), J. Alexander Lowden argues that "If . . . only the applicant [can] know his or her risk and may legally conceal it from the insurer, then insurance will become too expensive for all but those who know they will succomb at an early age." The argument is specious. The cost of insuring individuals who have genetic diseases has been included in insurance costs based on actuarial tables when there was no genetic testing, and the deaths due to inherited disease were part of the age-distributed probability of death due to randomly experienced causes. The insurance companies did not go broke during that period, as Lowden fears could happen in the future.

If one of the insured in that distribution knows that he or she will die of a certain disease, but the insurance company doesn't, little will have been changed for the insurance company. Lowden might fear that individuals who know they will die of a certain disease, perhaps prematurely, will stock up on insurance to better provide for their heirs. However, the levels of insurance individuals buy are dictated by affordability.

Even if the relatively small fraction of the population with heritable diseases were to double the amount of life insurance they buy, there would still be a random element to the payout periods. The cause of death might be predictable, but the age of death is not, within broad limits. By excluding people who have been diagnosed as having a genetic disease, the insurance companies would shift the mean age of death in their actuarial distributions to a higher age. This will defer benefit payments and help short-term profits. Long-term profits should not be affected significantly, as most of "the rest of us [with] perfect genes" will either collect their annuities or die while insured.

S. J. Deitchman
3606 Stewart Drive,
Chevy Chase, MD 20815, USA

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Children's Vaccine Initiative

The special *Science* issue "Frontiers in medicine: vaccines" (2 Sept. 1994) is an excellent and comprehensive report focusing on the high cost-effectiveness of vaccines and the need for new initiatives to bring their benefits to the children who need them most.

The News reports emphasize the need for intensified efforts to bring new research ideas to fruition in the form of vaccines against the most important diseases of the developing world, ensuring all the while that these vaccines will not be priced so high as to be unreachable for most children.

As Ann Gibbons points out (News, 2 Sept., p. 1376), the Children's Vaccine Initiative (CVI) has been slow to gain momentum. The recent reorganization in the World Health Organization (WHO) to centralize vaccine activities in the Global Programme for Vaccines and Immunization will now provide the strong leadership necessary to move ahead. The three units of the Global Programme for Vaccines and Immunization—Vaccine Research and Development, Vaccine Supply and Quality, and the Expanded Programme on Immunization—can address barriers to delivery of old vaccines