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LETTERS

Sources of Dioxin

A substantial portion of Philip H. Abelson's editorial "Chlorine and organochlorine compounds" (26 Aug., p. 1155) revolves around the assertions that "Two research groups have concluded that forest and brush fires are major sources of PCDDs [polychlorinated dioxins]" and "Anthropogenic production of dioxin has decreased during the past two decades and is smaller than that created by combustion of wood." We estimate that, in the United States, forest fires produce about 20 kilograms (kg) of "dioxin" [a term that includes polychlorinated furans as well as dioxins, all with equal weighting], that residential wood burning accounts for another approximately 20 kg, and agricultural fires for about 10 kg. The total annual dioxin production is estimated to be approximately 400 kg, most of it from incinerators (*1*). It is true that anthropogenic production has decreased, thanks largely to controls of municipal waste combustion and to the banning of formerly high-volume organochlorine pesticides and herbicides. This trend is likely to continue, as incinerator emissions are controlled further. But a reduction of an order of magnitude is required before forest fires, or biomass burning generally, become the dominant source of dioxin in the United States.

In support of the forest fire claim, Abelson cites a review by G. W. Gribble (*2*), which in turn cites two primary articles (*3*, *4*) from the early 1980s. T. J. Nestrick and L. L. Lamparski (*3*) reported one of the first measurements of dioxin emission from wood burning, but made no estimate of the magnitude of this source. A. Sheffield (*4*) produced the first comprehensive assessment of dioxin sources, estimating annual emissions from Canada. He estimated about 60 kg annually from forest fires and 25 to 110 kg from other sources. These numbers are not incongruent with those for the United States, if one takes into account that Canada has an order of magnitude fewer people than the United States and a similar forested area.

Sediment core data from Siskiwit Lake (*5*), located on an island in Lake Superior, confirm the dominance of anthropogenic dioxin. The dioxin deposition rate was constant between 1920 and 1940, then increased by an order of magnitude from 1940 to 1970. This was the period of great expansion in industrial chlorine use. Between 1970 and 1983, the dioxin sedimentation rate has declined by 30%, in parallel with the decreased anthropogenic production, mentioned by Abelson. Other sediment cores and soil samples from the Great Lakes region and from Europe show a similar pattern (*6*).

These considerations do not address the extent to which dioxins in the environment, whether anthropogenic or not, are actually harmful, nor the extent to which organochlorine commodities are responsible for anthropogenic dioxin. These are separate facets of the chlorine debate. But it should be clear that people, not forest fires, are responsible for most of the dioxin in the United States, and most likely in other industrialized areas as well.

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Abelson describes the U.S. EPA as a "tool of Greenpeace." Greenpeace would love to take credit for EPA's consideration of a chlorine reduction, abruptly muffled though it was. But credit must go to those who deserve it. We petitioned EPA for zero dioxin discharge and lost; the chlorine industry pushed for the dioxin reassessment (*1*) and won. So, in many ways, the industry deserves the credit for the logical outcome of the reassessment—a call for a chlorine phase-out.

To his credit, Abelson does not deny the link between dioxins and the chlorine industry. Instead, he resurrects Dow's old "trace chemistries of fire" theory (*2*), contending that forest and brush fires are "major sources" of dioxins. Neither Abelson nor his source, Gribble, acknowledges that this theory has been "largely discounted" by scientists (*3*).

Humans have always coexisted with forests and forest fires, but modern human

tissues carry 50 times more dioxin than ancient tissues (4). Today, people in agrarian regions burn wood for heating and cooking; nonetheless, their body burdens of dioxins are much smaller than those of people in industrialized regions (4). Dioxin levels in dated sediment cores rise in concert with the post-1940 growth of the chlorine industry (5). Likewise, archived soil and vegetation samples carry dioxin levels that increase in parallel with the chlorine industry's expansion (6).

A chlorine phase-out is not a new idea, not even to industry. In 1989, delegates from the Commission of the European Communities and Europe's largest chemical manufacturers agreed that "one may argue that the only fundamental solution for the environmental problems caused by organohalogenated products and their waste is to drastically reduce their production and restrict their use to closed systems" (7). More recently, the International Joint Commission on the Great Lakes has recommended "sunsetting" chlorine (8), while the 14 nations of the Oslo and Paris Conventions have agreed that organohalogens should be reduced with the aim of elimination (9).

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8. *Fifth Biennial Report on Great Lakes Water Quality* (International Joint Commission on the Great Lakes, Washington, DC, 1991); *Sixth Biennial Report on Great Lakes Water Quality* (International Joint Commission on the Great Lakes, Washington, DC, 1993).
9. "Final declaration, Ministerial Meeting of the Oslo and Paris Commissions, Oslo and Paris Conventions for the Prevention of Marine Pollution, 21–22 September 1992" (Oslo and Paris Commissions, London, 1993).

There is an alternative to the chlorine "sunset policy" being considered by EPA that would achieve the same societal goal of significant health risk reduction without unreasonable economic disruption. Randomly chlorinated organic byproducts in the waste, air, and water and solids from each chlorine-based manufacturing process and each process that treats or incinerates these wastes could be fractionated by molecular weight, hydrophobicity, and toxicity with the use of gas chromatography–mass spectrometry, high-performance liquid chromatography, and in vitro indicator assays. Chlorine-based processes that generate randomly chlorinated organic byproducts that are highly chlorinated, highly hydrophobic, and that assays show are strongly mutagenic, arylhydrocarbon hydroxylase inducing, or estrogenic could be "sunsetted" in 10 years, unless specific actions were taken within 5 years. These actions would include installing the best available treatment technology, producing a complete set of test data for the compounds and their treatment and incineration byproducts, and preparing a cradle-to-grave risk assessment demonstrating that the risks are de minimis or that the benefits to society of their continued manufacture outweigh their irreducible risks. Those that produce a mix of intermediate results and a mix of lowest results could be "sunsetted" in 15 years and 20 years, respectively, unless the same actions were taken in 10 and 15 years, respectively.

Chlorination of drinking water could continue, because the public health benefits can be demonstrated to outweigh the public health risks; but financial incentives could be provided for communities to add carbon filtration or equally efficacious alternatives could be found to reduce these avoidable risks if it is determined to be in the public interest to do so. Pharmaceuticals manufactured with chlorine-based compounds would not be "sunsetted" automatically. However, because there are few demonstrable benefits associated with wastewater chlorination, it could be "sunsetted" in the next round of reissuance of 5-year National Pollutant Discharge Elimination System permits to industries and municipalities under the Clean Water Act.

This alternative approach would strike an appropriate balance between the rights of the public to be protected from unnecessary exposure to highly persistent, bioaccumulative, and toxic byproducts of random chlorination and the rights of commercial entities to be protected from overly broad government restrictions that amount to unreasonable restraint of trade.

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Response: Smoke emitted during the global annual combustion of about 2 to 3 billion metric tons of plant materials contains nu-

merous toxic chemicals. Some of them are dioxins. The amounts of the individual substances emitted is a function of variables that include chlorine content of the fuel and flame temperatures. If flame temperatures exceed 1000°C, essentially no dioxins are emitted. However, most of the world's plant materials are burned under conditions favorable to the formation of dioxins. In one study of the burning of wood, the total dioxins produced were as much as 160 micrograms per kilogram (μg/kg) of wood [T. J. Nestrick and L. L. Lamparski, *Anal. Chem.* **54**, 2292 (1982)]. This result was obtained when various specimens of wood were burned in different stoves. Soot was collected and analyzed by well-designed and documented procedures. Tetrachlorinated, hexachlorinated, heptachlorinated, and octachlorinated dioxins were present. The isomers of the dioxins were separated and quantitated. The highly chlorinated dioxins were the major components. In the soot from a series of experiments, their total content ranged from 10 to 167 μg/kg of fuel. The total yields of tetrachlorinated dioxins (TCDDs) ranged from 0.1 to 7.8 μg/kg of fuel. The TCDDs have 22 isomers, of which the 2,3,7,8 isomer (dioxin) is the most toxic. Its yield averaged about 0.05 μg/kg of fuel.

In the developing countries biomass—including fuel wood, twigs, dung, and agricultural residues—is a major source of domestic energy. Minimum energy requirements for cooking and heating are estimated at 6 to 10 gigajoules or 0.5 to 1.0 cubic meters of fuel wood per person per year [M. R. de Montalembert and J. Clement, *FAO Forestry Paper No. 42* (Food and Agriculture Organization of the United Nations, Rome, 1983)]. During this century, the exponentially increasing populations have used a correspondingly greater amount of fuel. A few hundred years ago the people then living required only small amounts of biomass energy.

In 1978 fuel wood alone (1.67×10^9 cubic meters) accounted for a fifth of the total domestic energy consumption of the developing countries (M. R. de Montalembert and J. Clement, *ibid.*). But amounts burned in the destruction of forests in such places as the Amazon basin probably exceed that figure. A further source of dioxins is slash-and-burn agriculture.

When wood is burned in stoves in the United States, most of the dioxins formed are captured in the hardware. Emissions to the atmosphere from burning of biomass in the developing countries are relatively far greater. Their order of magnitude can be estimated by reference to the earlier-mentioned experiments with stoves. The burning of 2 to 3 billion metric tons leads to smoke containing on the order of 100 kg of

2,3,7,8 tetrachlorinated dioxin. This exceeds the EPA estimates of total current anthropogenic emissions in the United States by an order of magnitude.

—*Philip H. Abelson*

■

The D_2 Receptor Gene

Kenneth Blum and Ernest P. Noble (Letters, 2 Sept., p. 1346) incorrectly imply that we have demonstrated (Articles, 17 June, p. 1715) that the gene encoding the dopamine D_2 receptor locus (*Drd2*) influences several responses to alcohol, morphine, and cocaine in the mouse. We would like to clarify our interpretation of the data we presented (in our original figure 2) and reiterate our intent in creating the composite figure representing genetic influences on drug responses.

The method of quantitative trait locus (QTL) gene mapping allows identification of the tentative chromosomal positions of the genes influencing traits showing multigenic inheritance (*1*). We applied the QTL method to data from our own and others' laboratories to seek patterns of association that might suggest hypotheses regarding commonality of genetic control of multiple drug responses. We indeed reported preliminary evidence suggesting that several drug-related behaviors are tentatively associated with marker loci in a region of mouse chromosome 9 near the gene *Drd2* (*2*). However, we are rather less sanguine than Blum and Noble appear to be about the interpretation of this pattern of results. As we tried to make clear, the composite map we presented was designed to stimulate hypothesis generation, not to serve as a springboard for jumping to conclusions.

We briefly reiterate here the important reasons for not assuming that this pattern of association shows that the *Drd2* gene is the QTL mapped in each case (our original note 76). First, the associations presented in the figure represent tentative assignments of genetic association and need to be verified in a segregating F_2 or backcross population [our original notes 69 and 74; see also (*3*)]. While we expect that the majority of the provisional QTLs will be confirmed by further, rigorous tests with F_2 or backcross populations, only two QTLs for responses in the *Drd2* region have thus far been tested and verified. Contiguity of multiple verified QTLs in a relatively small region of chromosome 9 could reflect several causes (our original note 76). Each trait could be influenced by a different gene. Some (or all) could be affected by the same gene, or a gene cluster. Finally, all could be affected by