

Regulation of PCBs

In his editorial "Excessive fear of PCBs" (26 July, p. 361) Philip H. Abelson distinguishes among the different industrial formulations of polychlorinated biphenyls (PCBs). He points out that the many less toxic congeners, especially the lower chlorinated ones, are eliminated by biological and chemical breakdown. However, more highly chlorinated congeners, non-ortho chlorinated congeners, and mono-ortho chlorinated congeners remain. These toxic congeners have been found in concentrated amounts in breast milk, fish, and meat (1). The Schaeffer study (2), cited by Abelson, is most pertinent for individuals who have been exposed to industrial mixtures (for example, for certain electrical workers).

A logical approach would be to identify the PCB congeners in food and environmental samples and calculate risk on the basis of actual PCB presence rather than on the basis of a comparison with a rather unrelated parent PCB formulation. Unfortunately, data about the long-term toxicity of individual PCB congeners and PCB mixtures in the food chain are lacking. Until further studies address these issues, adherence to current regulations based on Aroclor 1260 seems reasonable.

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Abelson argues that because PCBs with a chlorine content of 54% or less have not been shown to cause cancer at a rate which is statistically significant, their intensive regulation is not warranted. However, the non-cancerous health effects of PCBs and dioxins should be a major concern. Adverse physiological effects have been associated not only with 18 dioxin-like PCB congeners (1), but with a number of non-coplanar congeners, including lesser chlorinated congeners (2).

Current regulations are based on effects seen in adult organisms. Yet evidence from studies of wildlife and laboratory mammals shows that organisms exposed to PCBs and dioxins during embryogenesis

and the perinatal period suffer different health consequences than do adults exposed to these same chemicals (1, 3).

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Reproductive dysfunction has been demonstrated in studies in which PCBs were fed to mammals. Abortions, stillbirths, neonatal mortality, low birth weights, and other effects have been noted in mink, European ferrets, white-footed mice, monkeys, and seals—all of which are more sensitive to PCB exposure than are laboratory rats (1). These effects were produced with concentrations of PCB similar to those found in some wildlife habitats.

High concentrations of PCBs have been found in the tissues of pinnipeds and cetaceans at the top of marine food webs (2). Rather than slowly disappearing, PCB concentrations are expected to increase in the world's oceans from PCBs that have already been produced (3). Our knowledge about the environmental occurrence and toxicity of specific PCB congeners is in its infancy. Further research is required in these areas before the scientific community can provide regulators with a clean bill of health for PCBs. Formulations resulting in such effects include Aroclors 1254, 1248, and 1242; as well as the pure 3,4,5, 3',4',5'-hexachlorobiphenyl congener.

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Sources of Acidity in Surface Waters

On the basis of predominance of nitrate and nonmarine sulfate, L. A. Baker *et al.* (Reports, 24 May, p. 451) conclude that 75% of lakes and 47% of streams surveyed in the eastern United States (1) that are acidic (acid neutralizing capacity ≤ 0) are so because of acidic deposition. Yet they also acknowledge research showing that such deposition-dominated waters were critically acidic before receiving acid deposition.

Baker *et al.* contend that the number of affected surface waters is even greater than stated above because biological effects are observed "when pH declines below 6.0 or even 6.5." While they discussed it extensively, Baker *et al.* did not report the key finding of Phase II of the Paleolimnological Investigation of Recent Lake Acidification project (PIRLA II): the average Adirondack lake is no more acidic now (1984 median pH = 6.37) than before acidic deposition (1844 median pH = 6.32) (2).

The PIRLA II finding is consistent with that of the Direct/Delayed Response Project (DDRP), which did not find any correlation between acidic deposition and surface water acidity, but did find surface water acidity to be correlated with watershed soil chemistry and land use (3).

The DDRP findings are consistent with a comprehensive compilation of soil experiments which show that, contrary to the large pH depressions predicted by anion dominance, acidic deposition has little measurable effect on the pH of water issuing from acidic soils: the correlation between "clean rain" and "acid rain" leachate pH is $R^2 = 0.989$; $n = 20$ (4).

That critically acidic clear and colored surface waters are comparatively common in acid soil regions of the world in the absence of acid rain (4, 5) further indicates the need to revisit assessments of aquatic acidification.

Finally, eastern forests are recovering from essentially pervasive cutting and