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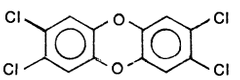
Chlorinated Dioxins

Chlorinated dioxins have become widely recognized as dangerous substances. What are some of their properties, how do they behave in the environment, and what hazards do they present for humans? In the chemical sense, there are actually 75 dioxins that contain chlorine, but the most notorious is 2,3,7,8-tetrachlorodibenzo-*p*-dioxin (TCDD), which has the structural formula



The compound is thermally very stable, melts at 307°C, has a vapor pressure of 1.7×10^{-6} mmHg at 25°C, and is practically insoluble in water. TCDD is an unwanted by-product of the manufacture of trichlorophenol, which subsequently is used to produce other compounds such as (2,4,5-trichlorophenoxy)acetic acid (2,4,5-T). At an earlier time, TCDD constituted several parts per million of some 2,4,5-T, but it is now present at 0.1 ppm or less. Another source of TCDD is the incomplete burning of municipal and industrial wastes as well as wood. When exposed to solar ultraviolet light in the presence of a hydrogen donor (foliage), TCDD is destroyed in days. When it becomes incorporated in soils, it is only slowly degraded. Because of its low solubility in water and strong adsorption to soil, the chemical moves very slowly.

When TCDD is orally administered to animals, the median lethal dose (LD50) ranges from 1 microgram per kilogram of body weight in guinea pigs to 5000 μg/kg in hamsters. The hazard to humans is assessable only by study of some industrial accidents. One of the most extensively investigated of these occurred in 1976 at Seveso, Italy. Excessive pressure built up in a reaction vessel, the safety disk ruptured, and a cloud containing sodium hydroxide, 2,4,5-trichlorophenol, and an estimated 2 kg of TCDD slowly drifted over a neighboring area depositing much of its contents. An area of 87.3 hectares received most of the material; the 736 people who lived in the area were moved 16 to 23 days later. None of the people died; clinical examinations indicated some effects on liver and nervous system, but later studies did not reveal a continuation of symptoms. The primary medical effect observed was a total of 187 cases of chloracne, a dermatitis resembling common acne. In most instances the symptoms were mild, and in all cases the disorder had disappeared in 2 years. Herbivorous animals were much more severely attacked and thousands died. The presumptive major cause was ingestion of TCDD on grass and foliage.

Observations at Seveso seem to indicate that ingestion of TCDD is necessary to bring out its full toxic effect. They have also been interpreted as showing that chloracne is the most definitive human indicator of exposure to environmental TCDD. No significant change was observed in the incidence of spontaneous abortions, congenital malformations, or postnatal development. It seems unlikely that in future an unusually high incidence of cancer will occur. This guess is based on experience of chemical workers who have been involved in accidents similar to that at Seveso, but whose exposure as indicated by intensity of chloracne was greater. Some of the accidents, notably one in 1949, occurred long enough ago that were cancer to be associated with them, it would now be evident. Some 121 workers were involved in the 1949 accident, and while ironclad proof of a null effect is missing, so too is a basis for believing that TCDD is a dangerous carcinogen in humans. It is clear that, when administered orally, TCDD is highly toxic, but when bound to soil it does not pose much of a hazard. It is also clear that TCDD is unwelcome. In the matter of risk versus benefit, the balance is completely one-sided: all risk and no benefit. Success in curtailing the amount of unwanted industrial production should be followed by vigilance in controlling TCDD formation in the incineration of municipal wastes. With time, natural degradation of the chemical will attenuate its presence in the environment.—PHILIP H. ABELSON