Revisiting 1 ppb as a "Level of Concern" for Dioxin in Soil

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ABSTRACT

A November 1989 conference organized by the Center for Risk Management revieved information about estimating exposures from dioxin in soil.[1] It concluded that site-by-site evaluations are necessary and that the "Times Beach risk assessment" overestimated exposures.

INTRODUCTION

The authors of the "Times Beach Risk Assessment" varned that their conclusion that 1 ppb of dioxin in soil was a level of concern should not be applied to other situations and other locations and that the assessment was based on many assumptions.[2] With the passage of time, however, 1 ppb has become the dividing line between acceptable and unacceptable levels, and in the five years since publication of that assessment, a number of scientists investigated the bases of the most important assumptions. The conference revieved those investigations and assumptions.

RESULTS AND DISCUSSION

Renate Kimbrough (U.S. Environmental Protection Agency) described the risk assessment[2] that established 1 ppb of dioxin in soil as a level of concern. She also advanced the opinion that the risks were overestimated because the soil was not uniformly contaminated at 1 ppb and because no one was likely to live on the contaminated soil for 70 years. She and other speakers reviewed what is known about concentrations of dioxin in soil at Times Beach, Seveso, and an industrial site in Camden, New Jersey. The highest levels of contamination, up to 32,000 ppb, were detected in horse arenas in Missouri,[3] where horses became sick and died. The contamination in the town of Times Beach was largely confirmed to the sprayed roads; concentrations of more than 1 ppb were found in only 2 of more than 1,000 measurements of dioxin in off-road samples after a flood hit the town in 1982.[4] Contamination was more general at Seveso, and Zone A, the most contaminated area, had an average of 2 ppb.[3] Concentrations at the Camden site were 2200 ppb.[5] No health effect has been linked to residence in Times Beach, and chloracne in Seveso was related to exposures soon after the accident, not to exposures from dioxin in soil.

Brendan Birmingham (Ontario Ministry of the Environment) was unable to attend the conference but submitted information about dioxin (expressed as TEFs) in various soils in North American.[6] Concentrations were lower in rural soils (30 samples, 0.4 ± 0.6 ppt) than in urban soils (47 samples, 11.3 ± 21.8 ppt), and industrial soils were highest (20 samples, 40.8 ± 33.1 ppt). His data indicate that "background" concentrations are well below those as found at Seveso or Times Beach.

Armon Yanders (University of Missouri) discussed his studies of the behavior of dioxin in Times Beach soil.[7] Dioxin cochromatographed with oil and moved downward into soil. Its halflife is very long, maybe 100 years, and it does not move laterally in the soil. In contrast, measurements at some locations in Seveso showed that up to 80 percent of dioxin disappeared from the soil within 18 months. Such short halflives were not observed everywhere at Seveso; no decreases were observed in some locations. After 18 months, no further decreases were seen because the dioxin penetrated downward into the soil where it is unavailable for volatilization and photodegradation.[8]

Dennis Paustenbach (ChemRisk) emphasized the quantitative importance of the Times Beach risk assessment assumption that children between 1.5 and 3.5 years of age ingest 10 g soil/d. Edward Calabrese (University of Massachusetts) described his studies of soil ingestion[9] and referred to others[10] that have examined a total of about 250 children. The mean soil ingestion rate is <40 mg/d, and 95 percent of the studied children ingest <70 mg/d. One studied child, however, ingested 5 to 8 g soil/d. These results indicate that the assumption of 10 g soil ingested/d is much too high for almost all children, but about right for geophagic children.

Thomas Umbreit (University of Medicine and Dentistry of New Jersey) reviewed studies of the bioavailabilty of dioxin from soil.[11] Liberation of dioxin in the digestive systems of guinea pigs was measured by chemical and biological means; up to 30 percent of the dioxin in Times Beach soil was bioavailable, compared to only 1.6 percent from the Camden soil.

Methods to measure dioxin in soil and in serum samples make it possible to look for associations between residence on contaminated soils and exposures as determined by concentrations of dioxin in serum. Larry Needham (Centers for Disease Contro)l presented data that show no clear correlations between levels of dioxin in soil and body burdens or between time of residence on contaminated soils and body burdens at Times Beach or Seveso.[3] On the other hand, it is clear that people who lived at Times Beach (5 to 60 ppt dioxin lipid) or rode horses in contaminated arenas in Missouri (5 to 557 ppt) were more exposed than other Missouri residents (non detectable to 20 ppt). Needham reported that the exposure factor most clearly linked to higher levels at Times Beach was being present at the time of spraying or soon after, which fits with observations reported by Umbreit[5] that dioxin becomes more tightly bound to soil over time.

For persons who do not live in contaminated areas, the most important source of dioxin is the food chain. George Fries (U.S. Department of Agriculture) discussed possible uptake of dioxin by plants,[12] and Craig McFarland (U.S. Environmental Protection Agency) reported that uptake and translocation within plants are so low that they are inconsequential in contributing to exposure.[13] Data are insufficient to draw conclusions about the amount of contamination of tubers by dioxin in soil. Uptake by leaves of vapor phase dioxin was also discussed, but there are few data on this point. Fries emphasized the importance of making local measurements and observations to determine how soil ingestion rates of beef and dairy cattle are influenced by different methods of animal husbandry.

Contaminated soil can wash into waterways, be deposited in sediment, and injested by fish, which are then consumed by humans. Russell Keenan[14] (ChemRisk) discussed use of the Bio-Concentration Factor, Bio-Accumulation Factor, and Bioavailability Index to estimate concentrations in fish based on knowledge of concentrations in water or sediment. Although the bio-concentration factor is most often used, he favors the bioavailability index, but convincing information to force a choice is apparently missing. An equally perplexing problem in estimating exposures from fish is the limited information available about fish consumption rates.

Allan Smith[15] (University of California, Berkeley) presented calculations of the contribution of dioxin in mother's milk to lifetime exposures.[16] His calculations differ from those of a World Health Organization Committee,[17] but were there good information about the transfer of dioxin from soil to the mother, either set of calculations could be employed to estimate exposures to the baby.

The presentations and the general discussion led by Tom Burke (State of New Jersey Department of Health) highlighted the impossibility of using standard assumptions to estimate exposures. Halflives, bioavailability, time since deposition of dioxin on soil, and soil ingestion rates by food animals can vary from site to site and affect exposure estimates and risk assessments. The estimate for children's soil ingestion rates in the Times Beach risk assessment was far higher than the rate for the average child, but not much different from the rate for a geophagic child. Risk assessment cannot instruct risk managers about how they should consider geophagic children. Should risk management decisions be based on them or average children? Most conference participants agreed that exposures would be lower on commercial and industrial sites, but there was disagreement about whether deeds and covenants could guarantee that properties would not revert to residential use in the future. The last two points--how to consider geophagic children and whether legal documents can be counted on in risk management--go well beyond risk assessment and illuminate the importance of policy decisions.

REFERENCES

1. Center for Risk Management. 1990. Human Exposures from Dioxin in Soil. Resources for the Future: Washington, DC (in preparation).

2. Kimbrough, R.D. et al. 1984. J. Toxicol. Environ. Health 14:47-93.

3. L. Needham, U.S. Centers for Disease Control. 1989. Paper presented at Center for Risk Management, November (to be reported in ref. 1).

4. V. Houk, U.S. Centers for Disease Control. 1989. Remarks at Center for Risk Management, November (to be reported in ref. 1).

5. T. Umbreit, University of Medicine and Dentistry of New Jersey. 1989. Paper presented at Center for Risk Management, November (to be reported in ref. 1).

6. B. Birmingham. 1989. Paper presented at Dioxin 89 and submitted to Conference at Center for Risk Management, November. In press, Chemosphere.

7. A. Yanders. 1989. Paper presented at Center for Risk Management, November (to be reported in ref. 1).

8. Di Domenico, A. et al. 1980. Ecotoxicol. Environm. Safety 4:327-338.

9. Calbreese, E.J. et al. 1989. Regul. Toxicol. Pharmacol. 10:123-137.

10. Binder, S. et al. 1986. Arch. Environ. Health 41:341-345; Clansing, P. et al. 1987. Internat. Arch. Occupat. Environ. Med. 59:73-85.

11. Umbreit, T.H. et al. 1986. Science 232:497-499; Umbreit, T.H. et al. 1988. Drug Chem. Toxicol. 11:405-418; McConnell, E.E. et al. 1984. Science 223:1077-1079; Shu, H. et al. 1988. Fundam. Appl. Toxicol. 10: .

12. Fries, G, U.S. Department of Agriculture. 1989. Paper presented at Center for Risk Management, November (to be reported in ref. 1).

13. McFarland, C., U.S. Environmental Protection Agency. 1989. Remarks at Center for Risk Management, November (to be reported in ref. 1).

14. Keenan, R., ChemRisk. 1989. Paper presented at Center for Risk Management, November (to be reported in ref. 1).

15. Smith, A.H. 1987. Risk Analysis 9:377-383.

16. Smith, A.H. 1989. Paper presented at Center for Risk Management, November (to be reported in ref. 1).

17. Report of a WHO Working Group. 1988. Assessment of health risks in infants associated with exposures to PCBs, PCDDs, and PCDFs in breast milk. WHO:Copenhagen.