The Public Health Response to 2,3,7,8-TCDD Environmental Contamination in Missouri

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Synopsis

In 1971, waste oil containing 2,3,7,8-tetrachlorodibenzo-p-dioxin (TCDD) was sprayed for dust control on a number of residential, recreational, and work areas in Missouri. In several of them, the level and extent of environmental contamination were not known until late 1982 or 1983. Extrapolation from existing toxicological data indicated the potential for substantial adverse health effects in highly exposed populations.

As a result, the Missouri Division of Health and the Centers for Disease Control initiated close collaboration with the Environmental Protection Agency (EPA) on review and evaluation of environmental data, the development of health advisories to EPA on the need for remedial or preventive actions at specific contaminated sites, a health education effort for the medical community and general public, establishment of a dermatological screening clinic, establishment of a central listing of potentially exposed persons through administration of a health effects survey questionnaire, and a pilot medical study of a "highest risk" cohort.

Strategies for additional interventions will continue to be based on findings derived from this first phase of the investigation.

In 1971, approximately 29 kilograms of 2,3,7,8-tetrachlorodibenzo-p-dioxin (TCDD)-contaminated sludge wastes, which originated as a result of the hexachlorophene production in a southwest Missouri plant, were mixed with waste oils and sprayed for dust control on areas in the eastern part of the State. Although current knowledge of the implications for the health of potentially exposed populations is incomplete (*1–3*), the contamination was of sufficient magnitude and extent to initiate environmental and public health investigations. As of November 1984, about 250 residential, work, and recreational areas (including several horse arenas) in Missouri were suspected of being contaminated. Forty sites have been confirmed as having at least 1 part per billion (ppb) in soil, more than 100 sites have not shown contamination at this level, and the remaining sites are still under investigation. At first, levels as high as 35,000 ppb of TCDD were measured in soil at 1 of the 40 sites; currently, isolated levels as high as 2,200 ppb exist in these contaminated areas, but most levels in soil samples with detectable TCDD range from less than 1 ppb to several hundred ppb.

The earlier phases of this investigation focused on several sites in eastern Missouri, but later activities included all 40 contaminated sites throughout the State. The Centers for Disease Control (CDC) had previously worked with the Missouri Division of Health (MDH) in 1971, the time of the initial con-

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tamination; in 1974, this work culminated in the laboratory identification of TCDD in the waste oil (*4*). With further discoveries of widespread contaminations in mid-1982, MDH and CDC in consultation reinitiated public health activities on the basis of new information and additional environmental data.

Under the Comprehensive Environmental Response, Compensation, and Liability Act of 1980 (CERCLA, more commonly known as "Superfund"), responsibilities for assessing the public health impact and the development and implementation of intervention strategies at hazardous waste sites are shared primarily by State health and environmental agencies, the U.S. Environmental Protection Agency (EPA), and CDC. The environmental agencies are primarily responsible for investigating and determining the composition, extent, and magnitude of contamination at suspected sites. In addition, they are responsible for developing and executing remedial strategies to mitigate or eliminate any further threat to the environment or the public health. Public health agencies are responsible for assessing the potential and actual public health impact of such situations of environmental contamination. CERCLA mandates as part of these responsibilities that public health advisories serve as part of the basis for decisions on such matters as relocation of affected residents or immediate remedial actions; EPA is responsible for the specific decisions about all remedial activities.

Reliable laboratory data on environmental samples from the contaminated sites played an integral part in the assessment of the presence and extent of possible TCDD contamination. An important consideration in the quantitation of dioxin is that the low part-per-billion concentrations required to make public health decisions approach the analytical detection limits of the methods routinely employed. EPA and CDC cooperatively developed a quality control-quality assurance protocol that would ensure specific congener verification of the TCDD and provide information on the accuracy and precision of the analytical system. Because TCDD binds tightly to soil, it can be substantially dispersed in the environment only by the contaminated soil being moved by erosion or on purpose; few analyses for TCDD in other media (for example, water) have, therefore, been done.

Approximately half of the 40 identified sites are contaminated with peak levels in excess of 100 ppb, and two-thirds of the contaminated sites are in residential areas. The lack of uniformity in geography, topography, geology, and characteristic land use at these sites presented difficult public health policy decisions. Sites where the levels of contamination were high and where there was frequent and regular access constituted the greatest public health risk; however, at other sites, TCDD contamination was in clearly circumscribed areas, at subsurface depths exceeding 15 feet, under paved areas, or in areas with limited land use. Similarly, characteristic land use patterns were important considerations in estimating likely routes of uptake and degrees of exposure. All of these considerations were taken into account in assessing the risk of exposure for an estimated 4,600 persons from these contaminated areas from 1971 to 1984.

Risk to Human Health

The case of TCDD accentuates many difficulties encountered in assessing immediate or delayed health risks following long-term exposure to environmental chemical contaminations. As yet, there is no reliable, widely available method for directly measuring TCDD uptake by humans; furthermore, there are no referent ranges from well-characterized populations with which to compare individual results. In this investigation, the lack of such direct measures of exposure to TCDD substantially hindered attempts to assess the degree of exposure to and concomitant health risk posed by environmental TCDD.

We, therefore, had to estimate the long-term risk of adverse health effects as a result of an estimated total cumulative dose. These calculations, as well as the subsequent risk evaluations, focused on the health risks associated with contamination of soils in residential areas and have been detailed by Kimbrough and coworkers (*5*).

Exposure assessment. The effective dose is a function of (*a*) the concentration of environmental

Relative carcinogenic potencies among selected chemicals evaluated by the Carcinogen Assessment Group as suspect human carcinogens

Compounds	Slope[1] (mg per kg per day)$^{-1}$	Molecular weight	Potency index[2,3]	Order of magnitude index
TCDD[4]	4.25×10^{5}	322	$1 \times 10^{+8}$	+8
Aflatoxin B_1	2,924	312.3	$9 \times 10^{+5}$	+6
Benzo(a)pyrene	11.5	252.3	$3 \times 10^{+3}$	+3
DDT[4]	8.42	354.5	$3 \times 10^{+3}$	+3
Ethylene dibromide	8.51	187.9	$2 \times 10^{+3}$	+3
PCBs[4]	4.34	324	$1 \times 10^{+3}$	+3
Beryllium	4.86	9	$4 \times 10^{+1}$	+2
Carbon tetrachloride	1.3×10^{-1}	153.8	$2 \times 10^{+1}$	+1
Epichlorhydrin	9.9×10^{-3}	92.5	9×10^{-1}	0
Vinyl chloride	1.75×10^{-2}	62.5	1×10^{0}	0

[1] Animal slopes are 95 percent upper-limit slopes based on the linearized multistage model calculated on the basis of animal oral studies, except for the slope for vinyl chloride, which was based on animal inhalation studies; human slopes are point estimates based on the linear nonthreshold model.

[2] The potency index is a rounded-off slope in (mMol per kg per day)$^{-1}$ and is calculated by multiplying the slopes in (mg per kg per day)$^{-1}$ by the molecular weight of the compound.

[3] Not all of the carcinogenic potencies presented in this table represent the same degree of certainty; all are subject to change as new evidence becomes available.

[4] NOTE: TCCD = Tetrachlorodibenzo-p-dioxin, DDT = dichlorodiphenyltrichloroethane, PCBs = polychlorinated biphenyls.

SOURCE: Adapted from Environmental Protection Agency, Carcinogen Assessment Group (*15*).

TCDD contamination, (*b*) location of and access to contaminated areas, (*c*) types of activities conducted in contaminated areas, and (*d*) duration of exposure. Indirect assessment by estimating exposure to and uptake of TCDD is inherently more difficult for soil and dust contaminations than for more easily predicted exposures by air, food, or water. In this case, principal routes of uptake were thought to be through dermal absorption, ingestion, and inhalation of contaminated dirt or dust particles.

For each route of uptake, the daily dose depends on the amount of soil present (for example, amount of soil on the skin), the TCDD concentration in the soil, and the percent of TCDD absorbed from soil by that route of exposure (for example, percent of the ingested TCDD absorbed in the gastrointestinal tract). Estimates of the amount of soil were based on conservative assumptions about activity patterns and degree of exposure. Ingestion, followed by percutaneous absorption, are estimated to be the largest sources of TCDD uptake; inhalation is estimated to be a negligible factor except in extremely dusty settings such as horse riding arenas.

Risk evaluation. Animal studies have shown great species variability in both acute and chronic responses to TCDD exposures; where humans fit on this response scale is not clear (*6*). However, common findings from both animal toxicological work and limited data on cases of high-dose, accidental exposures of humans have indicated prominent effects on several organ systems. Liver changes include diminished function, hepatocellular necrosis, tumor induction (in animals), and microsomal enzyme induction. Other effects include chloracne, depressed cell-mediated immunity, and peripheral neuropathy (*7–11*). Some studies have suggested that occupational exposures to TCDD may induce an excess risk of developing soft tissue sarcomas (*3,12*).

Results of human epidemiologic studies of delayed health effects after TCDD exposure in other settings are sufficiently inconclusive and do not provide precise dose-response data to be used as the basis for estimating risk. The only available dose-response data for TCDD come from animal toxicological work in studies of its carcinogenicity in rodents (*13,14*). These studies suggest that TCDD is a potent animal carcinogen at doses that are several orders of magnitude lower than those for other known carcinogens (table) (*15*). This has led to concern about continuing exposure to even minute amounts of environmental TCDD contamination (although the degree of human sensitivity to TCDD and the nature and extent of risk for long-term exposures need to be fully clarified). Thus, the only adequate dose-response data available from animal carcinogenicity studies were used in our risk assessment caclulations.

A linear, nonthreshold dose-response model was used to calculate increased lifetime cancer risk, and the calculation methods incorporated guidelines that a group of outside consultants recommended to CDC (*5*).

Risk management. In considering the foregoing sets of calculations, we concluded that TCDD levels of 1

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ppb or more in residential soil pose a level of concern for the development of delayed health risks. It is critical that in highly contaminated areas (soil contamination levels > 100 ppb), with a high degree of access and concomitant exposure, the estimated excess lifetime cancer risk may accumulate rapidly and be orders of magnitude higher than 10^{-6}. Therefore, on the basis of exposure and risk determinations, MDH and CDC advised that continued exposure to people living over the long term in residential areas with 1 ppb or more of TCDD contamination in the soil would lead to a risk of developing adverse health effects.

These public health advisories and consideration of the available remedial options were the basis on which EPA decisions to eliminate or mitigate these exposures were made. Decisions were made on a site-specific basis, as indicated by the complexities and variability of circumstances characteristic of each site. The time frame for such decisions was based on the degree of contamination and estimated exposure and on the degree to which continued exposures could be prevented while temporary or permanent remedial actions were considered, executed, or both. In most cases, recommendations were for quick, temporary environmental cleanup or stabilization or restriction of access to contaminated areas because of such characteristics as limited, well-defined areas of contamination, relatively low TCDD soil levels, or relative inaccessibility of contaminated areas.

The situation at Times Beach, however, was unique. Times Beach is in an urban, residential area with a history of recurring floods. In 1971, the then unpaved streets of the town were sprayed for dust control with TCDD-contaminated oil. Environmental samples taken in late 1982 identified TCDD contamination along the shoulders of the streets and in ditches on the sides of the street in a variable pattern and at different levels throughout parts of the town. The population of approximately 2,100 was, therefore, potentially exposed to TCDD environmental contamination along roadways at concentrations ranging from undetectable, at less than 1 ppb, to 980 ppb.

Immediately after the initial environmental sampling, but before the laboratory analyses of the samples were completed, a flood struck a major portion of the town and led to the near total evacuation of the community. The flooding raised the possibility of TCDD-contaminated soil being moved from washed-out roadways into the debris and, conceivably, into the houses. Therefore, upon receiving the results of the initial laboratory analyses and while awaiting further environmental sampling to clarify the location, extent, and level of contamination, CDC issued an advisory on December 23, 1982. The advisory recommended that the evacuated residents not return to the town and that cleanup efforts be halted (or be performed with full protective gear only on an emergency basis) until additional environmental sampling could delineate the extent of TCDD contamination. This advisory, unlike those at other sites, was issued on an emergency basis to prevent temporarily the repopulation of the town, especially since the main activity of the returning people would have been intensive cleanup of the potentially contaminated muddy soil and debris.

Post-flood environmental sampling results available in February 1983 showed only limited movement of TCDD-contaminated soil. However, on the basis of the extensive amount of contaminated soil in and near the roadbeds, the continued threat of flooding, and the available remedial strategies, EPA decided to offer to the inhabitants of this affected area a phased plan of permanent relocation.

Public Health Activities

In addition to ongoing review and assessment of EPA environmental sampling data, in January 1983 MDH and CDC began four distinct public health actions.

- *Providing health education for both the medical and public health communities and the general public about current understandings of the health effects of dioxin exposures.* To this end, a summary of the medical-epidemiologic literature was prepared and sent to physicians in eastern Missouri (*1*). On January 18, 1983, experts from Government, academic institutions, and industry were brought together to give a seminar for the local medical community. Individual consultations and toll-free hotlines were established to answer questions from and concerns of the general public.

• *Providing a dermatological screening clinic to the general public.* This clinic was intended to screen for cases of chloracne as an indication of possible dioxin exposure. In February 1983, on consecutive weekends, all residents of eastern Missouri who had reason to suspect that they had been exposed to TCDD and who had current skin problems were invited to these screening clinics.

• *Creating and maintaining a central list of potentially exposed individuals.* This list will enable public health agencies to keep in touch with and locate potentially exposed persons for educational purposes or possible epidemiologic and clinical followup. Specifically, when a reliable screening method for TCDD in serum becomes available, we will be better able to assess a person's exposure status and concomitant health risks. Baseline and identifying information was collected in the form of a health effects survey questionnaire designed to elicit information on possible routes of exposure, lifestyle habits, residential and occupational histories, and medical history. It was also intended to serve (*a*) as a screening tool for identifying a "highest risk" cohort on whom intensive medical evaluations were focused, and (*b*) in compiling a community-based data set with a sufficient sample size from which epidemiologic inferences might be drawn.

• *Designing and implementing a pilot medical study of a "highest risk" cohort.* The primary purpose of this study was to place potential current effects in perspective and to identify areas for more detailed epidemiologic studies. Results of this investigation suggested several organ systems for further study (renal-urinary tract, immunologic, hepatic, neurological) as discussed more fully by Stehr and coworkers (*16*).

Summary

These actions are the first phase in the investigation of dioxin contaminations in Missouri. The public health agencies involved continue to review environmental sampling data on new suspected sites and to develop public health advisories. In addition, more comprehensive and definitive epidemiologic studies are currently underway, and others are being planned, of potentially exposed cohorts in localized, discrete areas of highest contaminations with adequate comparison groups. Ultimately, our goal is to obtain more complete information on the etiologic role, if any, of environmental TCDD contaminations in human disease. In addition, an intensive effort is underway to develop analytic methods for determining TCDD body-burden levels and establishing background referent ranges. Such information will provide a better basis for making future public health decisions.

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