Health Consultation

Residential Dioxin Contamination

EASY GOER LANE

GREENBRIER, ROBERTSON COUNTY, TENNESSEE

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U.S. DEPARTMENT OF HEALTH AND HUMAN SERVICES
Public Health Service
Agency for Toxic Substances and Disease Registry
Division of Health Assessment and Consultation
Atlanta, Georgia 30333

Health Consultation: A Note of Explanation

An ATSDR health consultation is a verbal or written response from ATSDR to a specific request for information about health risks related to a specific site, a chemical release, or the presence of hazardous material. In order to prevent or mitigate exposures, a consultation my lead to specific actions, such as restricting use of or replacing water supplies; intensifying environmental sampling; restricting site access; or removing the contaminated material.

In addition, consultations may recommend additional public health actions, such as conducting health surveillance activities to evaluate exposure or trends in adverse health outcomes; conducting biological indicators of exposure studies to assess exposure; and providing health education for health care providers and community members. This concludes the health consultation process for this site, unless additional information is obtained by ATSDR which, in the Agency's opinion, indicates a need to revise or append the consultations previously issued.

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Prepared by:

Tennessee Department of Health
Under a Cooperative Agreement with
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Background and Statement of Issues

On March 17, 2003, the Tennessee Department of Environment and Conservation (TDEC) Division of Superfund (DSF) contacted the Tennessee Department of Health (TDH) Environmental Health Studies and Services (EHSS) concerning a complaint that chemicals had been improperly disposed of at a residence in Greenbrier, Robertson County, Tennessee, by a past property owner. This possible release of unknown substance(s) seems to have occurred sometime prior to April 2001.

This consultation involves two residences in the same subdivision: Residence A, where the suspected release occurred, and Residence B, where the complaint originated. Both these neighboring properties are approximately ¾-acre lots with single-family homes. The subdivision was built on land formerly used for agriculture. In December 2001, Residence A was vacated, and First Tennessee Bank took possession of the property. The house has since remained vacant.

The complaint involved an area of dead grass in the yard of Residence B. After first noticing the problem in April 2001, the property owner speculated that the dead grass was the result of a chemical release by the previous Residence A property owner. It is purported that the previous Residence A property owner participated in a landscaping business and emptied a tank of unknown substance(s) into his back yard, resulting in an area of bare dirt. By April 2002, the area of dead grass in the Residence A yard had increased significantly.

The adjacent property, Residence B, is down-gradient from the suspected release point. Its owner contends that the unknown substance(s) has migrated onto the property and caused grass to die there. The Residence B property owner wanted to know the cause of the dead grass and, if the cause was a chemical release, whether the chemicals pose a health hazard. Two adults (including the complainant), a two-year-old child, and three dogs live at the Residence B property.

On April 23, 2002, the Residence B property owner contacted Tennessee Emergency Management (TEMA) and was referred to the Robertson County TEMA. A Robertson County TEMA representative contacted First Tennessee Bank, owner of the property from which the problem seemed to originate. In May 2002, per the bank's request, Vita Environmental Company collected and analyzed surface soil samples from both properties. Test results indicated no chemicals, although non-specified compounds were noted. An unidentified Vita engineer speculated that the grass may have been killed by a commercial herbicide called Roundup. Vita recommended that both yards be sodded and indoor carpets cleaned. These recommendations were performed, and the grass sod was watered and grew well the rest of the summer of 2002.

In February 2003, the Residence B owner again noticed the grass dying in a pattern similar to that of the previous spring. The property owner contacted an attorney, who recommended having Southern Environmental Contracting, LLC, analyze surface soil samples. The samples were collected in February 2003. Results of these samples showed

no chemicals in targeted testing. Tentatively identified compounds (TICs), substances not present in the list of targeted compounds, were reported; however, not all TICs were identified and quantified using individual standards.

On March 10, 2003, the Residence B property owner's attorney contacted TDEC DSF seeking resolution regarding the possible release of hazardous substance(s). On March 18, 2003, Barry Brawley, TDEC DSF Field Office Manager, contacted TDH EHSS regarding review of the lab data and determination of health risk.

TDH reviewed the lab results and determined the TICs to be non-specific and agreed with TDEC that more specific testing should be performed. Two new surface soil samples (0–6 in.) were extracted from the original site by the TDEC DSF. They obtained one sample from the center of the suspected release area and a second from the gradient surface near the adjacent landowner's fence. Soil was not collected directly from the Residence B yard because the property owner did not allow TDEC employees on the property at that time.

On April 28, 2003, TDEC escorted TDH to the site. The site was visually inspected and several photos were taken. On the Residence A property, there was indeed a bare area approximately 12' x 20' ft at the side of the driveway and extending into the backyard. It was observed that weeds were growing in the area, suggesting that vegetation was returning.

Discussion

Neither the surface soil samples collected in May 2002 by Vita Environmental Company nor those collected in February 2003 by Southern Environmental Contracting, LLC, detected pesticides/herbicides, while the general chemistry tests noted non-specific TICs.

The surface soil samples collected from the suspected release area and along the fenced property line on March 2003 by TDEC DSF, however, indicated the presence of dioxins, furans, DDT, and dieldrin. These became the chemicals of potential concern due to the possible health hazards to the neighboring family, especially their young child.

Dioxins and Furans

Polychlorinated dibenzodioxins and dibenzofurans, the main chemicals of concern, are similar classes of chlorinated aromatic chemicals. They are usually produced inadvertently as byproducts, and neither polychlorinated dibenzodioxins nor dibenzofurans have known commercial or natural uses. Processes that contribute to their production include incineration or burning of waste, pulp and bleaching processes used in pulp and paper mills, and the chemical syntheses of trichlorophenoxyacetic acid, hexachlorophene, polychlorinated biphenyls, vinyl chloride, and pentachlorophenol. Dioxins and dibenzofurans are also produced naturally during forest fires and by burning

wood in stoves and fireplaces. Dioxins in very small concentrations are ubiquitous in the environment and have been found worldwide, even in remote areas (ARB 1986a).

The most important routes of human exposure to dioxins are from inhalation and ingestion, while dermal exposure is a less important route. The most often-noted health effect resulting from exposure to large amounts dioxins is chloracne. Such exposure has occurred in occupational settings at much higher amounts than what has been found the yards discussed in this health consultation. Chloracne is a severe skin disease with acne-like lesions that occur mainly on the face and upper body. Changes in blood and urine that may indicate liver damage have also been reported. Exposure to high concentrations may induce long-term alterations in glucose metabolism and subtle changes in hormone levels (ATSDR 1998).

The toxicity of dioxins and dibenzofurans is dependent on the chemical structure of the individual compound. The toxicity of various tetrachlorodioxins, for example, may vary by a factor of 100,000 or more. The most commonly known of these compounds—and perhaps one of the most toxic—is 2,3,7,8-tetrachlorodibenzo-p-dioxin (TCDD). TCDD has been classified by the International Agency for Research on Cancer (IARC) as a known human carcinogen. Other polychlorinated dibenzodioxins and dibenzofurans have not been studied sufficiently to determine their carcinogenicity (IARC 1998).

Once dioxins enter the body, a small amount is metabolized and eliminated. The rest bio-accumulates in the body. The body's concentration is dependent on the rates of ingestion, elimination, and storage capacity for dioxins. The approximate half-life for dioxins in humans is estimated to range from 6–10 years (Pirkle 1989).

Because general population exposure to dioxins occurs as exposure to a mixture of different congeners, effects due to individual congeners are difficult to determine. The various congeners are also not equally toxic, nor are they considered equally potent as carcinogens. For the purpose of assessing the cancer risk associated with these chemicals, the Environmental Protection Agency (EPA) adopted a procedure in 1989 which uses the concept of toxic equivalency factors (TEF). The cancer potency of all congeners chlorinated in the 2,3,7,8 positions are related to TCDD and tetrachlorodibenzo-p-furan (TCDF). The concentrations of the various congeners are multiplied by the appropriate TEF, and then the adjusted concentrations are summed. The sum is called the TCDD toxicity equivalent (TEQ). If TEQ≤50 parts per trillion (0.05 parts per billion [ppb]), no action is indicated. If TEQ≥0.05 ppb but <1 ppb, then the situation is evaluated. If the TEQ≥1 ppb, action is taken (DeRosa 2002).

The TEQs of the two surface soil samples collected by TDEC DSF from Residence A were calculated using this method and are below the ATSDR screening level (Tables 1 and 2). Therefore, adverse health effects are not likely to occur.

Table 1—Range of Contaminant Dioxin and Furan Concentrations Measured at the Suspected Area of Chemical Release

Site # 1 Chemical	Concentration (ppb)	Equivalency Factor	Tox Adjusted. Concentration (ppb)	ATSDR Screening Level (ppb)
2378 tetra…furan	5.10E-04	0.1	5.10E-05	
2378 tetra...dioxin	2.80E-04	1.0	2.80E-04	
12378 penta...furan	Not detected	0.05	0.00E+00	
23478 penta...furan	Not detected	0.5	0.00E+00	
12378 penta…dioxin	1.00E-03	0.5	5.00E-04	
123478 hexa…furan	5.30E-04	0.1	5.30E-05	
123678 hexa...furan	2.60E-04	0.1	2.60E-05	
234678 hexa…furan	Not detected	0.1	0.00E+00	
123789 hexa...furan	3.40E-04	0.1	3.40E-05	
123478 hexa...dioxin	1.70E-03	0.1	1.70E-04	
123678 hexa...dioxin	2.30E-03	0.1	2.30E-04	
123789 hexa...dioxin	3.00E-03	0.1	3.00E-04	
1234678 hepta...furan	4.10E-03	0.01	4.10E-05	
1234789 hepta...furan	Not detected	0.01	0.00E+00	
1234678 hepta...dioxin	1.50E-01	0.01	1.50E-03	
Octa...furan	1.10E-02	0.001	1.10E-05	
Octa…dioxin	1.50E+01	0.001	1.50E-02	
			TEQ = 1.82E-02	**5.00E-02**

Table 2—Range of Contaminant Dioxin and Furan Concentrations measured along the fenced property line

Site # 2 Chemical	Concentration ppb	Equivalency Factor	Tox Wted Conc (ppb)	ATSDR screening level (ppb)
2378 tetra...furan	3.30E-04	0.1	3.30E-05	
2378 tetra...dioxin	Not detected	1.0	0.00E+00	
12378 penta...furan	Not detected	0.05	0.00E+00	
23478 penta...furan	Not detected	0.5	0.00E+00	
12378 penta…dioxin	6.30E-04	0.5	3.15E-04	
123478 hexa...furan	4.10E-04	0.1	4.10E-05	
123678 hexa...furan	Not detected	0.1	0.00E+00	
234678 hexa…furan	Not detected	0.1	0.00E+00	
123789 hexa...furan	Not detected	0.1	0.00E+00	
123478 hexa...dioxin	9.50E-04	0.1	9.50E-05	
123678 hexa…dioxin	1.30E-03	0.1	1.30E-04	
123789 hexa…dioxin	1.60E-03	0.1	1.60E-04	
1234678 hepta...furan	1.80E-03	0.01	1.80E-05	
1234789 hepta...furan	Not detected	0.01	0.00E+00	
1234678 hepta…dioxin	1.10E-01	0.01	1.10E-03	
Octa…furan	3.50E-03	0.001	3.50E-06	
Octa…dioxin	1.40E+01	0.001	1.40E-02	
			TEQ = 1.59E-02	**5.00E-02**

DDT (C_{14}-H_9-C_{l5})

DDT (p,p'-dichlorodiphenyltrichloroethane) is an organochlorine insecticide that was used in a broad range of agricultural and nonagricultural applications worldwide beginning in 1939. In 1972, DDT was banned in the United States and in many parts of the world, except for use in controlling emergency public health problems. DDT is still used in certain parts of the world to control vector-borne diseases, such as malaria. The release of DDT into the environment occurs primarily through spraying applications onto agricultural crops, forest lands, other nonagricultural land, and homes. Exposures in homes usually occur through the use of DDT as a mothproofing agent, to control lice, and, in some parts of the world, to control mosquitoes and other disease-bearing vectors. The long-range transport of DDT has resulted in the wide dispersion of insecticide and its metabolites throughout the world, even into remote areas, such as the Artic or Antarctic regions (ATSDR 2002).

DDT can be degraded through atmospheric photo-oxidation in air or photolysis on the surface of water or soil. DDT can undergo slow biodegradation through reductive dechlorination to form DDE and DDD, and then be further degraded into other metabolites. The persistence of DDT and its metabolites, in combination with their high lipophilicity, have contributed to the bioaccumulation and biomagnification of DDT and its products in the environment. DDT, DDE, and DDD accumulate in fatty tissues, with tissue concentrations typically increasing with the trophic level of the organism (ATSDR 2002).

Numerous studies have been conducted on DDT and related compounds in a variety of animal species, but data for humans are somewhat limited. Most of the information on health effects in humans comes from studies of workers in DDT manufacturing plants or of spray applicators who had exposure over an extended period. Due to these limitations, disease causality cannot be determined from these studies (ATSDR 2002).

DDT's most well-known effect is impairment of nerve-impulse conduction. Effects of DDT on the nervous system have been observed in both humans and animals and can vary from mild sensations to tremors and convulsions. Humans have been reported to tolerate doses as high as 285 mg/kg without fatal result, although vomiting has occurred. There are no documented, unequivocal reports of a fatal human poisoning occurring exclusively from ingestion of pure DDT, but deaths have been reported following ingestion of commercial products containing DDT and other substances. Animal deaths following high exposure to DDT are usually caused by respiratory arrest.

In addition to being a neurotoxicant, DDT is capable of inducing marked alterations to reproduction and development in animals. These changes have been attributed to hormone-altering effects of DDT isomers and/or metabolites.

Some studies in humans suggest that high DDT/DDE burdens may be associated with alterations in end points that are controlled by hormonal function, such as lactation, maintenance of pregnancy, and fertility. High blood levels of DDE during pregnancy have also been associated with increased odds of having a pre-term and small-for-gestational-age infants, and height abnormalities in children (ATSDR 2002).

Studies in animals have shown that DDT can also cause cancer, primarily in the liver. The possible association between exposure to DDT and various types of cancers in humans, particularly breast cancer, has been studied extensively. Thus far, no conclusive evidence links DDT and related compounds to cancer in humans (ATSDR 2002).

In residential land use, the health risk of DDT is assumed to be limited to direct ingestion of contaminated soil only. While soil ingestion is just one of several possible pathways (including inhalation, dermal, etc.), the theoretical added toxicity from the other pathways seems negligible. Results of both surface soil samples taken March 18, 2003 were far below ATSDR's Cancer Risk Evaluation Guide (CREG) of 2 ppm and ATSDR's Reference Dose Media Evaluation Guide (RMEG) of 30 ppm for a child (Table 3). Therefore, adverse health effects are not likely to occur.

Table 3—Range of DDT Contaminant Concentrations

Sample Area	Concentration (ppm)	ATSDR CREG (ppm)	ATSDR RMEG, child (ppm)
Site #1: Release Area	0.023	2	30
Site #2: Fence Line	0.031	2	30

Dieldrin (C_{12}-H_8-Cl_6-O)

From the 1950s until 1970, dieldrin was widely used as a pesticide for crops such as corn and cotton. Dieldrin was also used to control locusts and mosquitoes and as a wood preservative. Because of concerns about damage to the environment and potentially to human health, EPA banned all uses of dieldrin in 1974, except to control termites. In 1987, EPA banned all uses. EPA considers dieldrin to be a persistent, bioacculumative, toxic pollutant. Sunlight and bacteria biodegrade the related pesticide, aldrin, to dieldrin. Both pesticides bind tightly to soil and evaporate slowly. In both soil and water, dieldrin breaks down very slowly.

Dieldrin is widespread in the environment, but at very low levels. Ingestion of foods such as fish, shellfish, dairy products, and meat from affected areas is the most common source of dieldrin exposure.

Animals exposed to high levels of aldrin or dieldrin experienced nervous system effects. Oral exposure to lower levels for a long period of time also affected the liver and immune system. Whether dieldrin has the same immunosuppressive effect on humans is not known. Studies in animals give conflicting results regarding dieldrin's effect on reproduction, and whether it affects reproduction in humans is unknown.

No conclusive evidence shows that aldrin or dieldrin causes cancer in humans. Studies indicate they can cause liver cancer in mice. The International Agency for Research on Cancer (IARC) has determined that aldrin and dieldrin are not classifiable as human carcinogens. The EPA has determined that aldrin and dieldrin are probable carcinogens, however, based on studies in mice (ATSDR 2000).

In residential land use, the risk of dieldrin and aldrin exposure is assumed to be limited to direct ingestion of contaminated soil only. While soil ingestion is just one of several possible pathways (including inhalation, dermal, etc.), the possibility of added toxicity

from the other pathways seems negligible. Results of both surface soil samples taken March 18, 2003 were slightly above ATSDR's Environmental Media Evaluation Guide (EMEG) of 3 ppm for chronic exposure of a child (Table 4).

One sample was slightly above ATSDR's Cancer Risk Evaluation Guide (CREG) of 0.04 ppm for a child. The CREG represents a theoretical risk of one excess cancer in a million people; in this case, the higher concentration of dieldrin found (0.15 ppm) represents a theoretical risk of 3.8 excess cancers in a million people. Although this risk is slightly greater, it is based on two samples and is below the acceptable level for chronic exposure.

Table 4—Range of Dieldrin Contaminant Concentrations

Sample Area	Concentrations (ppm)	ATSDR CREG, child (ppm)	ATSDR Chronic EMEG, child (ppm)
Site #1: Release Area	0.055	0.04	3
Site #2: Fence Line	0.150	0.04	3

Children's Health Considerations

The many physical differences between children and adults demand special emphasis when considering environmental exposure. Children could be at greater risk from certain kinds of exposure to hazardous substances. Children play outdoors and engage in hand-to-mouth behaviors that increase their exposure potential. Smaller than adults in stature, children breathe dust, soil, and vapors closer to the ground. In the event of exposure, a child's lower body weight and higher intake rate result in a higher dose of the hazardous substance per unit of body weight. If exposure levels are high enough and occur during critical growth stages, a child's developing body systems can sustain permanent damage.

In 1996, ATSDR launched an initiative to place a special, agency-wide emphasis on environmental hazards to children's health and to emphasize child health in all agency programs and activities (ATSDR 1997, 1998).

The concerns expressed by the Residence B property owner regarding the small child's possible exposure to chemicals was carefully considered as this health consultation was prepared.

Conclusions

No apparent public health hazard exists at Residence A. The TEQs of the two surface soil samples collected by TDEC DSF from Residence A are below the ATSDR screening level). Therefore, adverse health effects are not likely to occur. Since surface soil samples have not been collected from the Residence B property, data is not available to evaluate. Therefore, the soils at Residence B are classified by ATSDR as posing an indeterminate public health hazard.

Recommendations

Further attempts to obtain access and soil samples from the Residence B property should occur.

Public Health Action Plan

The Tennessee Department of Health will review future data and provide health education to the families involved as requested.

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CERTIFICATION

This Residential Dioxin Contamination Health Consultation was prepared by the Tennessee Department of Health under a cooperative agreement with the Agency for Toxic Substances and Disease Registry (ATSDR). It is in accordance with approved methodology and procedures existing at the time the health consultation was begun.

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The Division of Health Assessment and Consultation, ATSDR, has reviewed this public health consultation and concurs with the findings.

Roberta Erlwein

Chief, State Program Section, SSAB, DHAC, ATSDR

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