

Sunlight destroys TCDD's toxic properties rapidly on plant and soil surfaces. The length of time required to break it down into harmless components below the surface ranges from several months to many years, depending on a number of variables.

Three studies have been done to determine whether TCDD is transferred from the soil to plant life. Two of these found no evidence that TCDD translocates from soil to plants, including food crops growing in contaminated soil. The other study suggested that it may translocate. More work needs to be done to resolve this question.

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The U.S. Environmental Protection Agency currently regulates only TCDD. It allows herbicides, such as 2,4,5-T, to contain up to 0.1 parts-per-million of this contaminant. Using evolving technology, the chemical industry now produces herbicides typically containing only one-tenth this amount.

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Environmental Behavior

In the environment, TCDD tends to bind itself to soil particles making it a fairly immobile substance. It is not mobile or soluble in water either. Rather, it settles to the bottom of rivers and streams, binding itself to the silt or sediment. This characteristic prevents it from contaminating drinking water supplies. However, fish that feed on the sediment can become contaminated. In recognition of this, the U.S. Food & Drug Administration has established 25 part-per-trillion as the maximum amount of TCDD permitted in fish for human consumption. TCDD has a high boiling point (about 500 degrees Fahrenheit), thus it is not volatile. This makes human exposure through inhalation highly unlikely. The most likely route of human exposure is by absorption through the skin from direct skin contact with contaminated soil. Ingestion is also a possibility, especially by children who tend to put their fingers in their mouths.

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the World Health Organization, found chloracne to be the most prominent, immediate health effect. Some cases of peripheral nerve impairment and mild liver disorder were reported initially, but these effects cleared with time. No increases in spontaneous abortion, birth defects or other long-term illnesses have been documented. Studies are continuing.

- Nitro, W. Va. -- 418 Monsanto Co. employees were exposed to dioxin from a March 1949 plant accident and/or through the ongoing manufacture of 2,4,5-T herbicide. They were examined by Raymond A. Suskind, M.D., of the University of Cincinnati. On Nov. 10, 1982, he reported finding no link between TCDD exposure and cancer, cardiovascular disease or reproductive abnormalities. He did find chloracne and a loss of skin elasticity around chloracne-affected areas.

In summary, the preponderance of medical evidence published in the scientific literature indicates that chloracne will manifest itself in humans exposed to toxic levels of TCDD. In the absence of chloracne, other adverse effects are improbable.

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Sources and Levels

Chemical plant operations are not the sole source of dioxins. Research has recently established that most combustion processes, like power plants, municipal trash incinerators, automobile engines, and even wood-burning fireplaces, create measurable amounts of dioxins, including TCDD.

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Different studies have suggested contradictory results. Additional work, designed to sort out this discrepancy, is continuing.

A severe acne-like skin condition, known as chloracne, is the hallmark (or visible indication) of both acute and chronic toxic exposure to TCDD in humans. Chloracne manifests itself shortly after exposure (usually within 2 to 3 weeks) and, in its milder forms, is characterized by clusters of blackheads over the cheekbones, around the earlobes, on the face, chest and back. In severe cases, pus pockets, large cysts and scarring have occurred. In some industrial workers, chloracne has persisted for up to 30 years, but mild cases usually clear up shortly after exposure ceases.

In some workers, with or shortly after the onset of chloracne, other clinical effects occurred. These included temporary liver and kidney disorders, severe pain in the muscles of upper and lower extremities, fatigue and nervousness. In all cases, these conditions began to subside with cessation of exposure and eventually cleared up altogether.

In short, a considerable amount of health information has been gathered from medical studies on groups of people known to have been exposed to TCDD. Following is a summary of findings from perhaps the two most significant studies done to date:

- Seveso, Italy -- More than 37,000 people potentially were exposed to several pounds of TCDD from a July 1976 chemical plant explosion. Studies of those people, including one done by

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cells. In summary, based on the animal data, TCDD is indeed a very toxic substance.

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Human Experience

From the human experience, it's clear that man is much more tolerant of TCDD than most, if not all, animal species. Since no human is known to have ever died from TCDD poisoning, the amount required to kill a human is unknown. However, much is known about other human health effects.

The Journal of the American Medical Association (JAMA) reported on Oct. 15, 1982, that there is no scientific evidence that TCDD has caused reproductive difficulties or hazards in humans. It further stated that there is no conclusive evidence that TCDD is mutagenic or teratogenic (capable of causing birth defects) in man. Regarding cancer, the JAMA article said that the herbicide 2,4,5-T, containing trace amounts of TCDD, has been used in U.S. agriculture for more than 30 years "with no recognized evidence of carcinogenicity or birth defects in humans."

These statements are based in part on findings from many health studies, some done recently, on sizeable groups of people known to have been exposed to TCDD going as far back as 30 years. Included in this data is the health experience of more than 500 workers who had been exposed to high peak loads of TCDD in some 11 recorded chemical plant accidents. With one question still to be resolved, these studies found no increased cancer rates or other long term health problems except a persistent acne-like skin condition attributed to exposure to TCDD. The remaining question concerns soft tissue cancers.

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of it used as fill dirt at residential sites, thus creating additional environmental contamination and the possibility of increased human exposure.

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Animal Data

The "acute toxicity" of TCDD -- that is the amount that will cause immediate poisoning -- differs considerably from one animal specie to another. In the guinea pig, the laboratory animal found to be the most sensitive, a single oral dose of an extremely minute amount (in the billionths of an ounce range), may prove fatal. On the strength of this finding, TCDD is commonly characterized as the most toxic synthetic substance known to man. However, other test animals, such as rabbits, dogs and hamsters, can tolerate doses ranging from 100 to more than seven thousand times more than that which can kill a guinea pig. It should be pointed out that a number of other commercially available substances are, in fact, more toxic to some of these other test animals than is TCDD.

Laboratory studies have also shown that test animals fed daily doses of TCDD in their diets for prolonged periods of time have experienced other serious health problems. It has increased the incidence of cancer in mice and rats. It has caused birth defects, such as cleft palate, in mice and hamsters, but showed no such effects in monkeys. TCDD has caused fetal damage in mice and rats. It has not been found mutagenic (capable of causing hereditary changes) in tests involving cells of mice, monkeys or people, but did show these properties in tests on bacterial

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Background

The word "dioxin" actually refers to a family of chemical impurities unintentionally formed as contaminants during the manufacture of certain pesticides and industrial chemicals. They have no commercial value. It is theoretically possible to form 75 different dioxin compounds depending on the number of chlorine atoms present and their arrangement in the molecule.

The toxicity of individual dioxins varies considerably across a broad spectrum of animal species. Some are not hazardous. One dioxin, in particular, is the source of considerable public concern because it is known to be highly toxic in tests with laboratory animals. It is this particular compound, 2,3,7,8 tetrachlorodibenzo-para-dioxin (commonly called "TCDD"), that is the subject of this paper. For purposes of brevity, the abbreviation, TCDD, is used throughout this discussion.

This dioxin is the focus of widespread controversy associated with its presence in the Vietnam war defoliant, Agent Orange, and its tendency to form during the production of other products including hexachlorophene.

TCDD from this latter source has sparked renewed public health worries in Missouri. In 1971, waste oil containing TCDD from a now defunct pharmaceutical plant was sprayed on the ground to control dust in several horse arenas and possibly on some roads as well. It has recently come to light that contaminated soil from these arenas was later removed and some

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"DIOXIN FACT SHEET"

Introduction

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As a major U.S. chemical company with world-class research expertise, Monsanto is frequently sought out by government officials, members of the news media and the general public for its views and scientific information on a wide range of chemical issues. One such issue, currently the center of public controversy and concern, focuses on a class of chemical impurities commonly referred to as "dioxins."

Monsanto scientists have had considerable experience dealing with dioxin impurities because of their tendency to form as unwanted contaminants in the manufacture of the defoliant, Agent Orange, and other chlorinated compounds. Monsanto was one of the companies that made Agent Orange for the U.S. government during the Vietnam War. In recent years, a number of company scientists have become well informed about the chemistry and the environmental, toxicological and human health effects of dioxin. We are further familiar with the substantial amount of scientific information that has been generated by independent researchers on this subject.

In preparing this information, it is not Monsanto's intention to "defend" dioxin or to minimize its potential for legitimate health concerns. Rather we seek to provide the kind of factual scientific perspective that is often lacking in political, courtroom and public discussions of this subject.

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