

Monsanto

FROM
(NAME-LOCATION-PHONE) D. R. Bishop - A3NB

DATE : December 13, 1982

cc. G. Roush - G2WG

SUBJECT :

REFERENCE :

TO : R. T. Berendt - E2ND
T. M. Bistline - E2ND

Please review the attached draft fact sheet and let me have your comments/approval by December 16.

Dr. Roush has asked me to review our lay statement with his Biohazards Committee on December 17 so I really need your input.

Please disregard the earlier draft which, as you'll note, has undergone substantial changes.


Dan R. Bishop

DRB:ec

Attachment

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"DIOXIN FACT SHEET"

Introduction

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 ~~the most toxic of the~~ dioxin impurities because of ^{their} ~~its~~ tendency to form as an unwanted contaminant^s in the manufacture of the defoliant, Agent Orange^{and Chlorobiphenyls,} Monsanto was one of the companies that made ^{Agent Orange} ~~this product~~ 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. ①

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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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} ~~Many~~ are not considered hazardous. One dioxin, however, has been found to be extremely toxic in tests with some laboratory animals, and, thus, has triggered human health concerns. 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. (2)

This dioxin is the focus of widespread controversy associated with its presence in the Vietnam war defoliant, Agent Orange, and its occurrence 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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of it used as fill dirt at residential sites, thus creating additional environmental contamination and the possibility of increased human exposure.

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 ^{about} ~~16 billionths of an ounce~~ ^{TCDD} equal to ~~about one-billionth~~ of the animal's body weight 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.

Studies have also shown that exposure to TCDD can cause a number of serious long-term effects in animals. It has increased the incidence of cancer in mice and rats ^{fed diets} ~~fed doses in~~ the low parts-per-trillion range. 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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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 animal species. Since no human is known to have ever died from TCDD poisoning, ^{the amount required to produce} its acute toxicity ⁱⁿ to people is unknown.

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 supported by findings from health studies, many done recently, on workers 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-form skin condition attributed to exposure to TCDD. The remaining question concerns soft tissue cancers.

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

A severe acne-form 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 a cluster of blackheads over the cheekbones, around the earlobes, on the face, chest and back. In more severe cases, puss pockets, large cysts and scarring can occur. 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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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 ^{in 1979} 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 indicates that chloracne will manifest itself in humans exposed to toxic levels of TCDD. In the absence of chloracne, the medical literature suggests, there is not likely to be any other adverse effect.

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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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.

The U.S. Food and Drug Administration has established 25 parts-per-trillion (ppt) as the maximum amount of TCDD permitted in fish for human consumption. The Canadian government's standard is 20 ppt. *These levels are not really different. They are based on different assumptions about the amount of fish eaten.*

Environmental Behavior

Normally, TCDD is not soluble in water and, thus, does not contaminate drinking water supplies. It has a high boiling point (about 500°F) which makes environmental exposure through inhalation highly unlikely. The most likely route of human exposure is through direct skin contact with contaminated soil. Obviously, ingestion is also a possibility, particularly with children who are prone to putting their fingers in their mouths.

Sunlight is capable of degrading TCDD relatively quickly on plant and soil surfaces. The length of time required for TCDD to break down into harmless substances in soil varies from several months to many years depending on the soil type, the amount of bacteria present in the soil and the depth of contamination. It tends to bind itself to soil particles, minimizing its ability to move about in the environment.

Three studies have been done to determine whether TCDD is transferred from the soil to plant life. Two of these found

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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. *Plants grown in contaminated or suspected soil should be washed, particularly root vegetables, to remove adhering soil.*

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