DIOXIN: ENVIRONMENTAL IMPACTS AND POTENTIAL HUMAN HEALTH EFFECTS

ISSUE BRIEF NUMBER IB83079

AUTHOR:

Michael M. Simpson

Science Policy Research Division

THE LIBRARY OF CONGRESS

CONGRESSIONAL RESEARCH SERVICE

MAJOR ISSUES SYSTEM

DATE ORIGINATED 04/18/83
DATE UPDATED 10/21/83

FOR ADDITIONAL INFORMATION CALL 287-5700

ISSUE DEFINITION

Animal studies indicate that dioxin is a highly toxic compound; however, there have been no human deaths attributed to exposure to dioxin. Current concerns about dioxin center around the potential adverse environmental impacts as well as the possible harmful human health effects of trace dosages encountered as environmental contaminants. The environmental impact and human health effects of such exposure to dioxin are not clear at this time.

Conflicting statements regarding the effects of dioxin exposure are found in both scientific and popular journals. This controversy stems from the lack of conclusive findings from well-defined exposure studies. Policymakers are consequently confronted with deciding whether or not to take regulatory or other action to address fears about the threat to health and the environment, in the absence of confirmed findings on the risks associated with dioxin.

This issue brief presents a short background on the physical/chemical properties of dioxin, describes several existing sources of possible human exposure, and highlights what is currently known about its environmental impacts and human health effects. Congressional interest is intense at this time because of large numbers of Vietnam veterans' claims for benefits associated with use of herbicides in that war as well as because of certain incidents of potential significance to health involving disposal of wastes containing dioxin. (See also IB83043 -- Agent Orange: Veterans' Complaints and Studies of Health Effects.)

BACKGROUND AND POLICY ANALYSIS

The compound dioxin was identified when it was synthesized by a research technician in the mid-1950s. The technician was exposed to the dioxin and subsequently developed a severe case of chloracne which required hospitalization. This event provided the first clue as to the potential human health effects of dioxin exposure.

There are many different forms of dioxin; the form most commonly referred to as dioxin is 2,3,7,8,-tetrachlorodibenzo-p-dioxin (TCDD). Dioxin is a contaminant that occurs in the production of TCP (2,4,5,-trichlorophenol). When TCP is used to make other compounds, a dioxin contaminant may be passed along to the new substance. The compound TCP is used in the production of several compounds, including the herbicide 2,4,5,-T (2,4,5,-trichlorophenoxyacetic acid) and the compound hexachlorophene (bis-trichoro-2-hydroxyphenol methane), which is used in soaps and disinfectants.

The level of dioxin contamination in 2,4,5-T until 1970 averaged 1-5 parts per million (ppm) with a few samples as high as 4-7 ppm. After-1970, levels of dioxin contamination were required to be reduced to less than 0.1 ppm in 2,4,5-T. The U.S. Pharmacopeia has established a maximum detectable limit of 0.05 ppm dioxin for hexachlorophene; the actual content measured in hexachlorophene has been less than 0.005 ppm. (A physical/chemical description of the compound dioxin is provided in the appendix.)

Policymakers have often been called upon to make risk-regulating decisions

in the absence of sufficient scientific data. Frequently such data as may be available are conflicting. In making such policy decisions, Congress considers a number of factors in determining allowable risk to public health and the environment, as well as the economic impact and net social benefits involved. Policy decisions about the regulation of dioxin may affect the general population, several industries, and foreign trade. At present, there are several organizations, activities, agencies, and interest groups concerned about the issue of dioxin. Their positions are reflected by their individual policy statements.

o The Veterans Administration (VA) has a long-standing and continuing concern over the effects of dioxin exposure, stemming from the use of Agent Orange in Vietnam. The VA's review of available literature and ongoing studies indicates that, as yet, no consensus exists within the scientific community concerning the causal relationship between dioxin exposure and those health problems experienced by some Vietnam veterans.

o The Dow Chemical company acknowledges that dioxin is highly toxic and produces ill health effects in laboratory animals; however, at concentrations found in the environment, the company believes dioxin does not pose a health hazard to humans.

o The American Medical Association (AMA) has not confirmed any chronic adverse human health effects related to environmental exposure to dioxin other than chloracne, but has encouraged further research in this area.

o As a result of studies conducted at sites contaminated with dioxin, the Centers for Disease Control (CDC) has established a safe exposure limit for dioxin in the environment at 1.0 part per billion (ppb). The CDC's position is that below 1.0 ppb the individual is not at risk; above 1.0 ppb, inadequate data exist to determine if any risk is present. The exposure limit is both site and condition specific -- i.e., this limit may vary from location to location, depending upon the level of dioxin contamination, and the condition of exposure. CDC is presently conducting research on Vietnam veterans exposed to dioxin (phenoxy herbicides) to better define the health risks from exposure.

o The National Institute for Occupational Safety and Health (NIOSH) claims that evidence is increasing to support a link between occupational exposure to dioxin and soft tissue sarcoma.

o The Veterans of Foreign Wars (VFW), in its concern for the health of veterans in general, finds that although no confirmed relationship has been established between dioxin exposure and specific adverse health effects, there are sufficient trends reported in the scientific journals to suggest some health risk. The VFW is seeking compensation for any veteran suffering from specific conditions reported to result from dioxin exposure.

o Also supporting the idea of compensation is the Vietnam Veterans of America (VVA). The VVA maintains that scientific evidence supports the association of adverse human health effects and dioxin exposure.

o The Sierra Club and the Environmental Defense Fund (EDF) have taken stronger positions against dioxin. The Club and EDF believe dioxin presents a health hazard and recommend further research to determine the degree of hazard to man. They call for the removal of all uses of chemicals contaminated with dioxin and stricter control over production of chemicals that could have dioxin as a contaminant.

The diversity of these policy positions reflects the lack of conclusive scientific data.

Dioxin Research

Dioxin research is presently being conducted in several private and Federal research facilities. Federal agencies have budgeted over $10,000,000 for dioxin research during fiscal year 1983 (FY83) and more than $50 million is scheduled to be spent on dioxin research in FY84. The major purpose of this research is to determinate the human health effects and environmental impacts associated with dioxin exposure.

In addition to Federal expenditures, the Dow Chemical Company has made $3,000,000.00 available to researchers to identify the human health effects resulting from dioxin exposure.

Concern has been expressed concerning the coordination and control of research efforts by individuals, interest groups, researchers, and Federal agencies.

Superfund

At present, funds used to temporarily or permanently relocate residents living in dioxin-contaminated areas come from the Superfund budget. If the trend of buying these contaminated areas continues, additional funding of the Superfund program will become necessary. (See IB83064 -- Superfund: Hazardous Waste Clean-up -- for details of the complete issues associated with the Superfund.)

Sources of Exposure to Dioxin

Public concern about exposure to dioxin has been heightened as a result of increased awareness of several sources of proven or possible exposure: 1) the use of the herbicide "Agent Orange" in aerial spraying operations during the Vietnam conflict (there were also occasional uses of herbicides such as 2,4,5-T for brush control in civilian applications until 1979. Since 1979, uses of 2,4,5-T have been limited to rice crops and rangelands; 2) the Seveso, Italy, chemical plant accident; 3) emissions from municipal incinerators and some fossil fuel power plants; 4) the; spreading of dioxin contaminated oil for dust control in Missouri. and 5) unknown sources of dioxin in Michigan, New Jersey, Massachusetts, and Oregon.

1. Herbicide Aerial Spraying.

From 1962 to 1971, herbicide aerial spraying operations were conducted by the U.S. in the Republic of Vietnam. The peak periods of spraying were 1968 and 1969. According to the U.S. Air Force, the herbicide most often sprayed was "Agent Orange," a half-and-half mixture of the herbicide 2, 4,-D (2,4-dichlorophenoxyacetic acid) and 2,4,5-T (2,4,5-trichlorophenoxyacetic acid). Dioxin is a contaminant of 2,4,5-T. Average dioxin levels in "agent orange" were estimated at 2 ppm with a high of 47 ppm and a low of 0.02 ppm. The Environmental Protection Agency (EPA) has since recommended the limit for the contaminant dioxin in 2,4,5-T at 0.1 ppm (May 1971). Both U.S. air and ground forces were subjected to possible dioxin exposure while in specific

regions of the Republic of Vietnam (see IB83043 -- Agent Orange: Veterans' Complaints and Studies of Health Effects).

2. Seveso, Italy, Chemical Plant Accident.

The Seveso, Italy, accident (July 10, 1976) involved an explosion at a chemical plant where TCP was manufactured. A cloud of TCP with the contaminant dioxin escaped from the chemical plant as a result of the explosion. The cloud, estimated to contain roughly 3 grams of dioxin, was dispersed by the wind across a residential area adjacent to the plant. Several hundred people were exposed to the chemical cloud. Within the first year after the accident 187 cases of chloracne were reported; 164 were children and 23 were adults. Eight of the children with chloracne also had severe chemical burns; eleven other children had chloracne and mild chemical burns. Several other children that did not develop chloracne did develop erythema (red skin patches). Very few adults developed chloracne or chemical burns. Studies are still being conducted on the exposed population to determine the chronic health effects.

3. Power Plant and Incinerator Emissions.

Dioxin has also been identified as an emission from some fossil fuel power plants and municipal incinerators. The dioxin is produced during the incomplete combustion of fuel or waste that contains such compounds as chlorophenols, chlorinated benzenes, and biphenyl ethers. The amount of discharge (smoke stack emissions) is dependent upon incinerator operation, fuel, and combustion temperature.

4. State of Missouri Contamination.

Dioxin contamination of soil in Missouri dates back to 1971, when dioxin-contaminated waste oil was applied to the soil for dust control. At a horse arena where the dioxin-contaminated waste oil was applied several horses died. Several other sites in the State also were sprayed with the dioxin-contaminated waste oil for dust control. Not all sites have been positively identified or tested. Dioxin contamination sites in the State have been identified to have levels between 1.0 ppb-1,800 ppb. Some estimates for suspected dioxin contamination sites are as high as 1.0 ppm. The problem of dioxin site identification and cleanup has been complicated by rainfall, surface run-off, and flooding which spreads the dioxin contaminated soil. At present, efforts are underway to identify all dioxin sites in the State and either clean up the site or possibly relocate the residents.

As a result of the soil being contaminated with dioxin, the EPA has requested the Federal Emergency Management Agency (FEMA) to permanently relocate the residents of Times Beach and 11 families of Imperial, MO. The EPA has also requested FEMA to temporarily relocate three St. James, MO Families.

5. Michigan and New Jersey Contamination.

Certain sites in Michigan and New Jersey have recently been identified as having been contaminated with dioxin. Dioxin has been detected in rivers around the Dow Chemical Plant in Midland, Michigan, and at three sites near the Diamond Alkali Corp. plant in New Jersey. Both plants are reported to have produced Agent Orange contaminated with dioxin during the Vietnam War. At present only one site has been identified in Michigan. In New Jersey there are 50 additional sites that are scheduled for dioxin contamination

testing. The specific source or sources of dioxin contamination remain to be determined in both States. Further investigations will be conducted in each State to identify any additional dioxin contaminated sites.

6. Identification of Sites Possibly Contaminated with Dioxin.

In 1980, the Environmental Protection Agency compiled a list of sites in 33 States possibly contaminated with dioxin. These sites will be tested to determine if they are actually contaminated.

Massachusetts is one of the suspect States. Several ponds and lakes in the State will be tested during the summer of 1983. The ponds and lakes may have been contaminated when herbicides were sprayed on them to control water weeds in the 1950s and 1960s.

It has been reported that dioxin-contaminated sites also exist in Oregon, with dioxin concentrations up to six times greater than those at Times Beach, Mo. The source of the dioxin in Oregon is unknown at this time.

Environmental Effects

1. Biological Accumulation and Elimination.

Studies have demonstrated that accumulation and elimination of dioxin occurs in many animal species. It has been reported that dioxin, at concentrations found in the environment, does not accumulate in animal tissue to any significant degree.

It would appear that the rates of accumulation and elimination are dependent upon the environmental concentration, conditions of exposure, and animal species.

Few studies have been conducted to determine whether dioxins are accumulated in plants. Those studies that have addressed this question appear to indicate that very little dioxin accumulation occurs in plants. When accumulation did occur the lowest levels were recorded in the fruits and leaves with the highest levels found on the surface of roots. It is interesting to note that when plants were transplanted from dioxin-contaminated soil to uncontaminated soil, the dioxin levels accumulated in the plant disappeared.

2. Soil Contamination.

Some studies have been conducted to determine if dioxin is persistent in most types of soils. Those studies that evaluate dioxin persistence in soil found that over 90% of the dioxin could be detected in the soil one year after the original contamination occurred. This would indicate that a strong bond is established between dioxin and most soils. The persistence of dioxin in the environment has been attributed to these strong bonds.

3. Water Contamination.

Dioxin is not very soluble in water (highly insoluble). The greatest danger of contamination of the rivers and streams is from dioxin-contaminated soil run-off. The eroded dioxin-contaminated soil particles could settle on the bottom of the stream or could be carried with the flow of the stream.

Disposal Methods

Small quantities of dioxin can be disposed of by physiochemical, chemical, and microbiological means. Physiochemical means include: ultraviolet light; gamma rays; or hydrogen donation. An ultraviolet light treatment was developed recently for commercial disposal of dioxin-contaminated liquid wastes. The treatment is 98% efficient. Chemical disposal is possible utilizing alkaline dehydrochlorination, reduction with iron chlorides, oxidation with ruthenium tetroxide, chloridides, and chlorolysis.

Alkaline dehydrochlorination uses anhydrous alkali metal salts of polyhydroxy alcohols to remove the chlorine from the chlorinated dioxins. This is accomplished by mixing dioxins, alcohol, and a water solution of any (alkaline) metal hydroxide.

Reduction of dioxin with iron chlorides is being studied; this method has had limited success.

Oxidation with ruthenium tetroxide is an acceptable method for destroying small amounts of dioxin. This method is usually used to destroy dioxin produced in the laboratory.

Chloriodide research is being conducted in the hopes of using chloriodides to destroy dioxin in contaminated soil. Micellar catalysts are used un the process.

Chlorolysis is a process currently being investigated. Final research results remain to be determined. Microbiological means depend on microbes to destroy the dioxin. This method has been field tested with limited success; due to its high cost it may not be considered practical for bulk disposal. Research is underway to develop a better microbial degradation method. Such degradation has not proven successful to date. Larger quantities of dioxin can be disposed of using more traditional means such as: a) incineration; b) perpetual repository storage (not true disposal); c) secure landfill (presently permitted but may still present an environmental hazard later); and d) entombment (concrete mixed with dioxin -- this is not actually disposal).

Dioxin is easily destroyed by natural sunlight in a very short time. The difficulty in this form of disposal results from the fact that natural light destruction of dioxin is only surface active (i.e., it destroys only the layer on the surface directly exposed to the natural sunlight.)

General Exposure Standards

Two agencies have established exposure standards for dioxin and a third has recommended a maximum exposure limit. The Environmental Protection Agency's Scientific Advisory Panel in 1980 established the "no observable effect level" (NOEL) for dioxin at 0.001 micrograms dioxin per kilogram of body weight per day (mg/kg/day). The NOEL is defined as a dose below which no carcinogenic, teratogenic, or reproductive effect is observed.

The Federal Food and Drug Administration (FDA) set the dioxin levels in edible fish as not to exceed a limit of 50 ppt (parts per trillion). It has been recommended by the Centers for Disease Control (CDC) that individuals not be exposed to levels of dioxin greater than 1 ppb in soil for any

extended periods of time.

The chemical industrial exposure limit for dioxin of 0.0007 mg/kg body weight was set by the EPA to protect workers in the chemical industry and related fields.

Biological Effects -- Animals

Experiments on laboratory animals show interspecies differences (i.e., no two species react in the same way) in response to a high concentration acute exposure to dioxin. This response would indicate that dioxin has a different acute toxic effect in each animal species. However, when exposure levels are high enough to cause death, the length of time between exposure and death (the latent period) is similar for each species. How the dioxin causes death in experimental animals is not known. When laboratory animals are exposed to lower concentrations of dioxin, where death is not expected to occur, two sets of symptoms are generally observed. The first set of symptoms, common to all species, includes: a) chloracne; and b) the development of horny skin layer (hyperkeratosis). The second set of symptoms which is specific to each individual species includes: a) in pregnant female mice, dioxin exposure results in the production of some abnormal offspring (teratogenic effect); b) in pregnant female mice and some other species, dioxin exposure causes the death of some of the unborn offspring (fetotoxic effect); c) in chickens a unique lesion is developed (hydropericordium); d) in some animal species an infiltration of tissues with fluid occurs (edema); e) in rabbits liver disturbances are common; f) in monkeys conversion of the meibomian gland occurs (the gland in the eyelid changes from a sebaceous fluid producer to a keratin producer); and g) in guinea pigs suppression of the immune system occurs. Although reproductive disorders were observed in female mice, no reproductive disorders were observed in male mice exposed to dioxin at identical concentrations.

Biological Effects -- Man

No human deaths have been reported after an acute exposure to dioxin. The most prominent effect observed in man from an acute or chronic exposure to dioxin is chloracne. Chloracne can range in severity and extent. The more severe cases may involve extensive acne-like eruptions of blackheads, abcesses, and cysts initially on the face, giving the appearance of grayish sheets. The most severe cases may involve the total body. Chloracne is not a condition that is unique to dioxin exposure; it may also occur from exposure to many other chlorinated compounds. The chronic effects of exposure to dioxin (other than chloracne) are not well understood. Although several symptoms, diseases, and conditions have been described by individuals chronically exposed to dioxin, there is not sufficient information to establish a cause-and-effect association. The diseases or conditions that have been reported include: 1) enlarged liver; 2) liver function abnormality; 3) neuromuscular effects; 4) deranged porphyrin (a disturbance in production of porphyrin -- a natural pigment produced by the body); 5) spontaneous abortion; 6) congential malformation; 7) cancer; and 8) chromosomal aberrations.

1. Liver Enlargement.

Enlargement of the liver has been reported in some exposed individuals. However, mortality among the dioxin-exposed individuals who displayed an

enlarged liver was not statistically different from that of non-exposed individuals (individuals with normal livers). Any relationship between dioxin exposure and the liver enlargements is unclear at this time.

2. Liver Function Abnormality.

Liver dysfunction has been observed in some individuals who also were accidently exposed to dioxin. Individuals exposed to low levels of dioxin may have an abnormal liver function, yet no temporary or permanent damage may occur. Exposures to very high levels of dioxin may cause an abnormal liver function with some signs of permanent damage. Any relationship between dioxin exposure and liver function abnormality is unclear at this time.

3. Neuromuscular Effects.

Some individuals exposed to dioxin report a weakness in their lower limbs. This effect has not been observed in any animal trials. The impairment reported seems to be slight, affecting muscular coordination without any other apparent side effects. Any relationship between dioxin exposure and the neuromuscular effect is unclear at this time.

4. Deranged Porphyrin.

Porphyrin derangement, which may result in spotting of the skin, is known to have a genetic as well as an environmental cause (e.g., environmental exposure to hexachlorobenzene, polychlorinated biphenyls and dioxin). Any relationship between dioxin exposure and the porphyrin effect is unclear at this time.

5. Spontaneous Abortions.

Spontaneous abortions have been reported as a possible adverse health effect of dioxin exposure. A review of recent studies indicates that there appears to be no difference between the rate of spontaneous abortions within the dioxin-exposed populations and the non-exposed populations.

6. Congenital Malformations.

It has been reported that as a result of dioxin exposure congenital malformations have occurred. There appears to be no difference in the number of congenital malformations occurring in dioxin exposed and non-exposed populations. This was confirmed in a comparison made between the rate of congenital malformations observed after the Seveso, Italy, accident and the estimated world rate.

7. Cancer.

Cancer has been suggested as an effect of dioxin exposure. It is difficult to demonstrate a cause-and-effect relationship between dioxin exposure and cancer. There appears to be no difference in the overall cancer death rate between the dioxin-exposed and the non-exposed populations. According to some studies, there does appear to be some increase in the incidence of soft tissue sarcoma in dioxin-exposed populations. NIOSH claims evidence is increasing to support a link between occupational exposures to dioxin and soft tissue sarcoma. Other studies, conducted both in this country and overseas, have shown no increase in soft tissue sarcomas rates as a result of dioxin exposure.

It has recently been reported that an association may exist between stomach cancer and dioxin exposure. Evidence in support of either association is not sufficient to make a positive confirmation of cause and effect.

8. Chromosomal Aberrations.

Cytogenetic studies (those relating to the function and structure of the cell) to date do not indicate an increase in the frequency of chromosomal aberrations as a result of dioxin exposure. When the prevalence of chromosomal aberrations is compared between the dioxin-exposed individuals and the total population at large no significant difference is observed.

Summary

Studies indicate that dioxin is a highly toxic chemical that may persist for many years in the environment. Plants do not appear to accumulate great amounts of dioxin. Animals appear to accumulate and eliminate dioxin at different rates depending on environmental conditions and animal species. Bodies of water generally become contaminated from dioxin-contaminated soil run-off as a result of erosion. Soil forms a strong bond with dioxin which is mainly responsible for the persistence of the dioxin in the environment.

Dioxin may be disposed of by traditional methods such as incineration, perpetual storage, landfill, and entombment. Dioxin molecules are destroyed by sunlight on exposed surfaces.

Animal exposure studies demonstrate species differences for the effects of dioxin. Chronic exposures appear to result in specific health effects for each animal species.

Chloracne is the most consistent condition of dioxin exposure in humans. Other symptoms have been reported, yet no cause-and-effect association has been confirmed.

Points for Further Consideration

Future issues which may confront policymakers include: (a) identification of cause-effect relationships with regard to the human health effects of dioxin exosure; (b) proposed establishment of dioxin industrial discharge requirements; (c) establishment of final dioxin disposal requirements (changing the chemical character of dioxin); (d) initiation of further chronic exposure studies with animals; (e) development of new waste discharge methodologies; (f) requirement for extensive acute and chronic testing of new chemicals prior to marketing; (g) identification of one Federal agency to coordinate all dioxin and Agent Orange research efforts; (h) examination of the need for some form of a victim compensation program, based upon a chosen level of acceptable risk, to protect the individual from environmental sources of dioxin (a hazard insurance program); and (i) examination of the need for the development of a risk analysis program for all hazardous waste sites.

HEARINGS

U.S. Congress. House. Committee on Energy and Commerce. Subcommittee on Oversight and Investigations. Defining health emergencies under the superfunded statute: recent PCB and dioxin cases. Hearing, 97th Congress, 2d session. Nov. 19, 1982. Washington, U.S. Govt. Print. Off., 1983. 370 p.

U.S. Congress. House. Committee on Interstate and Foreign Commerce. Subcommittee on Oversight and Investigations. Agent orange: exposure of Vietnam veterans. Hearing, 96th Congress, 2d session. Sept. 25, 1980. Washington, U.S. Govt. Print. Off., 1981. 249 p.

----- Involuntary exposure to agent orange and other toxic spraying. Hearings, 96th Congress, 1st session. June 26-27, 1979. Washington, U.S. Govt. Print. Off., 1980. 256 p.

U.S. Congress. House. Committee on Veterans Affairs. Subcommittee on Oversight and Investigations. Current status of agent orange studies. May 6, 1981. Washington, U.S. Govt. Print. Off., 1981. 385 p.

U.S. Congress. House. Committee on Veterans Affairs. Subcommittee on Medical Facilities and Benefits. Scientific community report on agent orange. Hearings, 96th Congress, 2d session. Sept. 16, 1980. Washington, U.S. Govt. Print. Off., 1981. 145 p.

----- Oversight hearing to receive testimony on agent orange. Hearing, 96th Congress, 2d session. Feb. 25, 1980. Washington, U.S. Govt. Print. Off., 1980. 121 p.

----- Oversight hearing to receive testimony on agent orange. Hearing, 96th Congress, 2d session. July 22, 1980. Washington, U.S. Govt. Print. Off., 1981. p. 459 p.

U.S. Congress. House. Committee on Veterans Affairs. Ad Hoc Subcommittee. Status of Vietnam veterans in the Bay area. Hearing, 96th Congress, 2d session. Apr. 10, 1980. Washington, U.S. Govt. Print. Off., 1980. 64 p.

CHRONOLOGY OF EVENTS

10/20/83 -- U.S. EPA confirmed the presence of dioxins and furans in municipal incinerator emissions.

07/00/83 -- Contaminated sites reported in Massachusetts and Oregon.

06/00/83 -- Soil contamination with dioxins found in Michigan and New Jersey.

02/00/83 -- Missouri clean up of dioxin spills begins.

11/19/82 -- Hearings held by Subcommittee on Oversight and Investigations of the Committee on Energy and Commerce. Hearing on the hazards of dioxin.

12/03/80 -- P.L. 96-510, Comprehensive Environmental Response Compensation and Liability Act 1980 (CERCLA), "Superfund" -- clean up of hazardous waste sites.

10/21/76 -- P.L. 94-580, Resource Conservation and Recovery Act (RCRA), control of hazardous wastes.

10/11/76 -- P.L. 94-469, Toxic Substance Control Act, the control of all toxic substances not covered by other toxic substances control.

07/10/76 -- Seveso, Italy chemical plant explosion, dioxin contaminant spread over local region.

07/19/75 -- P.L. 94-56, Hazardous Material Transportation Act. (Amended 1976 to cover the transport of toxic materials.)

12/16/74 -- P.L. 93-523, Safe Drinking Water Act. (Amended 1977 to eliminate contaminants from discharge or entry into water sources.)

10/18/72 -- P.L. 92-500, Clean Water Act. (Federal Water Pollution Control Act.) (Amended 1977 to cover discharge of toxic pollutants into water.)

00/00/71 -- Missouri sites were contaminated with dioxin.

12/31/70 -- P.L. 91-604, Clean Air Act. (Amended 1977 to cover discharge of hazardous pollutants into the air.)

1960-70 -- Agent orange contaminated with dioxin used in Republic of Vietnam for aerial spraying operations.

00/00/56 -- Dioxin identified.

Unknown -- Incinerator production of dioxin.

ADDITIONAL REFERENCE SOURCES

Axelrod D. Dioxin exposure report to the Governor and legislative April 1982. New York State Department of Health. 1982.

Barnes, D.G. Human health aspects of accidental chemical exposure of dioxin -- strategy for environmental reclamation and community protection. International Academy of Environmental Safety, International Society of Ecotoxicology and Environmental Safety. Oct. 5-7, 1981.

Bartleson, F.D., Harrison, D.D., and Morgan, J.D. Field Studies of Wildlife exposed to TCDD contaminated soils. U.S. Air Force report, AFATL-TR-75-49, Mar. 1975.

Beljan, J.R. (Council of Scientific Affairs, JAMA), Health effects of agent orange and dioxin contaminants. Journal of the American Medical Association, v. 248, no. 15, Oct. 15, 1982.

Blain, E.H. Chlorodioxins -- origin and fate. American Chemical Society Washington, D.C. 1973.

Cocucci, S., DiGerolamo, F., Verderio, A., Covallaro, A., Colli, G., and Gorni, A., Invernizzi, G., and Luciani, L. Absorption and translocation of tetrachlorodibenzo-p-dioxin by plants from polluted soil. Experientin, v. 35, Fasc 4, May 79: 482-484.

Crosby, D.G., Wong A.S. Environmental degradation of 2,3,7,8-tetrachlodibenzo-p-dioxin (TCDD). Science, v. 195, Mar. 25, 1977: 1337-1338.

Crow, K.D. Chloracne. Seminars in Dermatology, v. 1, no. 4, December 1982: 305-314.

Esposito, M.P., Tierman, T.O., and Dryden, F.E. Dioxin. Environmental Protection Agency, Cincinnati, OH. EPA-600 2-80-197. November 1980.

Franelli, R., Cheabrando, C., and Bonaccorsi, A. TCDD contamination in the Seveso incident. Drug Metabolism Reviews, v. 13, no. 3, 1982: 407-422.

Kimble, B.J. and Gross, M.L. Tetrachlorodibenzo-p-dioxin quantilation in Stack-Collected coal fly ash. Science, v. 207, Jan. 4, 1980: 59-61.

Kriebel, David. The dioxins--toxic and still troublesome. Environment, v. 23, no. 1, Jan./Feb. 1981: 6-13.

Kociba, R.J. and Schwetz, B.A. Toxicity of 2,3,7,8,-tetrachlorodibenzop-dioxin (TCDD). Drug Metabolism Review, v. 13, no. 3, 1982: 387-406.

Reggiani, G. Toxicology of 2,3,7,8-tetrachlorodibenzo-p-dioxin (TCDD): short review of its formation, occurrence, toxicology, and kinetics, discussing, human health effects, safety, measures, and disposal. Regulatory Toxicology and Pharmacology, v. 1, 1981: 211-243.

Sun, Marjorie. Missouri's costly dioxin lesson. Science, v. 219, Jan. 28, 1983: 367-369.

Thomasson, W.A. Deadly legacy: dioxin and the Vietnam veteran. The Bulletin, v. 35, no. 5, May 1979: 15-19.

Townsend, J.C., Bodner, K.M., Van Peenen, P.F.D., Olsen, R.D., and Cook, R.R. Survey of reproductive events of wives of

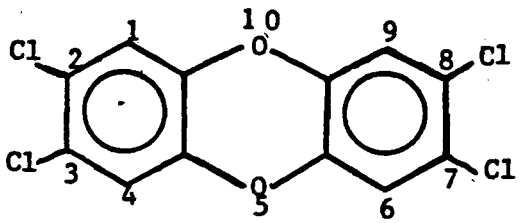
employees exposed to chlorinated dioxins. American Journal of Epidemiology, v. 115, no. 5: 695-713.

Young, A.L., Calcagni, J.A., Thalken, C.E., and Tremblay, J.W. The toxicology environmental fate and human risk of herbicide orange and its associated dioxin. U.S. Air Force Report, October 1978. OEHL TR-78-92

APPENDIX 1

Structure and physical/chemical characteristics of 2,3,7,8-tetrachlorodibenzo-*p*-dioxin, TCDD or dioxin.

Structure



Cl=Chlorine
O=Oxygen
⬡=Benzene

2,3,7,8-tetrachlorodibenzo-*p*-dioxin (TCDD)

Physical Characteristics

molecular weight	322	
melting point,	303 - 305°C	577 - 581°F
decomposition point,	980 - 1,000°C	1796 - 1832°F

Chemical Characteristics

Solubility, grams/liter

ortho-dichlorobenzene	1.40
chlorobenzene	0.72
Orange Herbicide	0.58
benzene	0.57
chloroform	0.37
acetone	0.11
normal-octanol	0.05
lard oil	0.04
methanol	0.01
water	2×10^{-7}

REFERENCE:
Young, A.L., Calcagni, J.A., Thalken, C.E., and Tremblay, J.W. The toxicology environmental fate and human risk of herbicide orange and its associated dioxin. U.S. Air Force Report, OEHL TR-78-92, October 1982. page I-22