DIOXIN

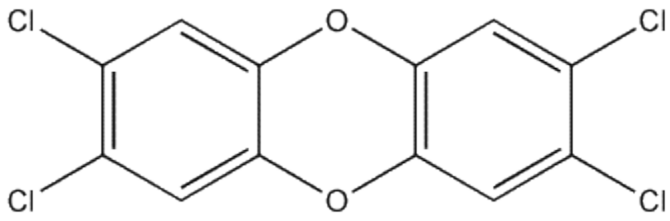
Overview

The term dioxin is commonly used to refer to a family of toxic chemicals that all share a similar chemical structure and a common mechanism of toxic action. This family includes eight of the chlorinated dibenzo dioxins (CDDs), ten of the polychlorinated dibenzo furans (PCDFs) and twelve of the polychlorinated biphenyls (PCBs)(#EPA). These are all organochlorine compounds. Dioxins are unintentionally produced by industrial, municipal, and domestic incineration and combustion processes, and there also are natural sources like brush and forest fires. As a consequence of industrialization, dioxin levels began increasing in the global environment. Declines in environmental levels began in the 1970s when dioxins were recognized as highly toxic chemicals and governments and industry took actions to prevent environmental pollution (#EPA). However, current exposure levels and dioxins' accumulation in the food chain, particularly in animal fat, still remain a concern.

As mentioned before, the CDD (chlorinated dibenzo dioxins) family is divided into eight groups of chemicals - their distribution is based on the number of chlorine atoms in the compound. The group with one chlorine atom is called the mono-chlorinated dioxin(s). The groups with two through eight chlorine atoms are called dichlorinated dioxin (DCDD), tri-chlorinated dioxin (TrCDD), tetra-chlorinated dioxin (TCDD), pentachlorinated dioxin (PeCDD), hexa-chlorinated dioxin (HxCDD), hepta-chlorinated dioxin (HpCDD), and octa-chlorinated dioxin (OCDD)(#ATSDR-Toxicological Profile for Dioxins).

2,3,7,8-TCDD is one of the most toxic and extensively studied of the CDDs and serves as a prototype for the toxicologically relevant or "dioxin-like" CDDs (#ATSDR-Toxicological Profile for Dioxins).

Chemical Description



2,3,7,8-TCDD (Tetrachlorinated Dibenzo-p-dioxin)(#PANNA)

In the pure form, CDDs are colorless solids or crystals. CDDs enter the environment as mixtures containing a variety of individual components and impurities (#ATSDR-Toxicological Profile for Dioxins).
2,3,7,8-TCDD is a by-product formed during the manufacture of 2,4,5-trichlorophenol (2,4,5-TCP). 2,4,5-TCP was used to produce hexachlorophene (used to kill bacteria) and the herbicide, 2,4,5-trichlorophenoxyacetic acid (2,4,5-T). Various formulations of 2,4,5-T have been used extensively for weed control on crops and range lands, and along roadways throughout the world. 2,4,5-T was a component of Agent Orange, which was used extensively by the U.S. military in the Vietnam War(#ATSDR-Toxic ological Profile for Dioxins).

Sources

CDDs are known to occur naturally, and are also produced by human activities. They are naturally produced from the incomplete combustion of organic material by forest fires or volcanic activity. CDDs are not intentionally manufactured by industry, except in small amounts for research purposes. They are unintentionally produced by industrial, municipal, and domestic incineration and combustion processes.
Currently, it is believed that CDD emissions associated with human incineration and combustion activities are the predominant environmental source (#ATSDR Toxicological Profile for Dioxins). CDDs also have been detected at low concentrations in cigarette smoke, home-heating systems, and exhaust from cars running on leaded gasoline or unleaded gasoline, and diesel fuel. Dioxins had been also detected during chlorine bleaching of pulp and paper. Burning of many materials that may contain chlorine, such as plastics, wood treated with pentachlorophenol (PCP), pesticide-treated wastes, other polychlorinated chemicals (polychlorinated biphenyls or PCBs), and even bleached paper can produce CDDs (#ATSDR Toxicological Profile for Dioxins).

Routes of Exposure and Metabolism

The substance can be absorbed into the body by inhalation of dust, through the skin and by ingestion (#ICSC).
Being highly lipophilic, dioxins dissolve in fat. They need to be transformed in the liver to become water soluble before they can be excreted. However, dioxins are metabolized slowly and therefore tend to bioaccumulate, especially in fat and in the liver.
The speed of elimination of dioxins can vary with dose, quantity of body fat, age and sex. The

process of elimination of dioxins and PCBs is similar in animals and man, but it is faster in most other mammals (#GreenFacts).
The developing fetus is most sensitive to dioxin exposure. The newborn, with rapidly developing organ systems, may also be more vulnerable to certain effects. Some individuals or groups of individuals may be exposed to higher levels of dioxins because of their diets (e.g., high consumers of fish in certain parts of the world) or their occupations (e.g., workers in the pulp and paper industry, in incineration plants and at hazardous waste sites, to name just a few)(#WHO).

Human Health Effects

Acute Health Effects

According to PANNA, short time exposure to dioxin (2,3,7,8 TCDD) can cause chloracne, redness and pain (#PANNA).

Chronic Health Effects

2,3,7,8 TCDD is listed as "known carcinogen" by IARC (International Agency for Research on Cancer) and also byU.S. National Toxicology Program Carcinogen List. According to the EU list (European Union Prioritization List) dioxin (2,3,7,8 TCDD) is an endocrine disrupting chemical (#PANNA).
In laboratory animals they have been linked to endometriosis (severe effects on the uterus), developmental and neurobehavorial effects (learning disabilities), developmental reproductive effects (low sperm count, genital malformations) and immunotoxic effects (#European commission).

Environmental Health Effects

In the environment dioxins can be commonly detected in air, soil, sediments and food. Dioxins are transported primarily through the air and are deposited on the surfaces of soil, buildings and pavement, water bodies, and the leaves of plants. Most dioxins are introduced to the environment through the air as trace products of combustion. The principal route by which dioxins are introduced to most rivers, streams and lakes is soil erosion and storm water runoff from urban areas (#EPA).
When released into the air, some dioxins may be transported long distances. Because of this, dioxins are found in most places in the world. When dioxins are released into water, they tend to settle into sediments where they can be further transported or ingested by fish and other aquatic organisms. Dioxins decompose very slowly in the environment and can be deposited on plants

and taken up by animals and aquatic organisms. Dioxins may be concentrated in the food chain so that animals have higher concentrations than plants, water, soil, or sediments. Within animals, dioxins tend to accumulate in fat (#FDA).

Due to its very low water solubility, most of the 2,3,7,8-TCDD occurring in water is expected to be associated with sediments or suspended material. Aquatic sediments may be an important, and ultimate, environmental sink for all global releases of TCDD. Two processes which may be able to remove TCDD from water are photolysis and volatilization.

The photolysis half-life at the water's surface has been estimated to range from 21 hr in summer to 118 hr in winter; however, these rates will increase significantly as water depth increases. Many bottom sediments may therefore not be susceptible to significant photodegradation.

The volatilization half-life from the water column of an environmental pond has been estimated to be 46 days; however, when the effects of adsorption to sediment are considered, the volatilization model predicts an overall volatilization removal half-life of over 50 years.

Various biological screening studies have demonstrated that TCDD is generally resistant to biodegradation. The persistence half-life of TCDD in lakes has been estimated to be in excess of 1.5 yr(#Technical Factsheet on Dioxin, EPA).

If released to soil, TCDD is not expected to leach. As a rule, the amount of TCDD detected more than 8 cm below the surface has been approximately 1/10 or less than that detected down to 8 cm. Being only slightly soluble in water, its migration in soil may have occurred along with soil colloids and particles to which it may have been bound. Soil cores collected from roadsides in Times Beach, MO in 1985 which had been sprayed with waste oils containing TCDD in the early 1970s indicated that most of the TCDD had remained in the upper 15 cm.. Tests conducted by the USDA determined that vertical movement of 2,3,7,8-TCDD did not occur in a wide range of soil types.

Being only slightly soluble in water, its migration in soil may have occurred along with soil colloids and particles to which it may have been bound. Photodegradation on terrestrial surfaces may be an important transformation process. Volatilization from soil surfaces during warm conditions may be a major removal mechanism. The persistence half-life of TCDD on soil surfaces may vary from less than 1 yr to 3 yrs, but half-lives in soil interiors may be as long as 12 years. Screening studies have shown that TCDD is generally resistant to biodegradation (#Technical Factsheet on Dioxin, EPA).

If released to the atmosphere, vapor-phase TCDD may be degraded by reaction with hydroxyl radicals and direct photolysis. Particulate-phase TCDD may be physically removed from air by wet and dry deposition (#Technical Factsheet on Dioxin, EPA).

Bioconcentration in aquatic organisms has been demonstrated. Mean bioconcentration factors (BCF) of 29,200 (dry wt) and 5,840 (wet wt) were measured for fathead minnows over a 28 day

exposure; the elimination half-life after exposure was found to be 14.5 days. Log BCFs of approximately 3.2 to 3.9 were determined for rainbow trout and fathead minnow in laboratory flow-through studies during 4-5 exposures. The following log BCFs have been reported for various aquatic organisms: snails, fish (Gambusia), daphnia 4.3-4.4; duckweed, algae, catfish, 3.6-3.95 (#Technical Factsheet on Dioxin, EPA).

Effects on wildlife

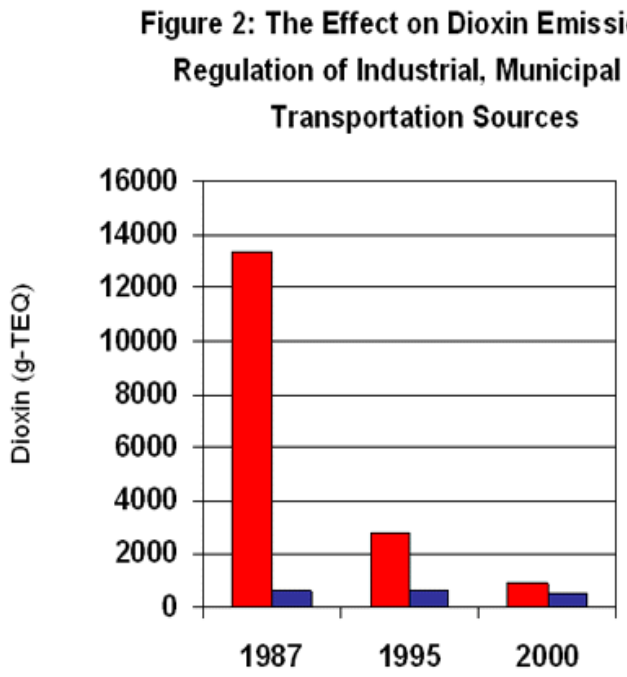
There is substantial evidence to indicate that populations of wildlife species high on the food chain are suffering health damage due to reproductive and developmental impairment due to background exposures to dioxins and related compounds. In the Great Lakes, exposure to dioxin-like compounds has been linked to large-scale hormonal, reproductive, and developmental impairment among numerous species of predator birds, fish and wildlife; these impacts are primarily transgenerational, affecting the offspring of the exposed organisms (#WFPHA, 2000).

- Lake trout embryos and sac fry are very sensitive to toxicity associated with maternal exposures to 2,3,7,8-tetrachlorodibenzo-p-dioxin (TCDD) and structurally related chemicals that act through a common aryl hydrocarbon receptor (AHR)-mediated mechanism of action. The loading of large amounts of these chemicals into Lake Ontario during the middle of the 20th century coincided with a population decline that culminated in extirpation of this species around 1960 (#Cook et al., 2003).
- Dioxin is responsible for inability of mink to reproduce around Lake Michigan (#Hochstein et al., 1998)

Regulation

EPA advises that children should not have more than 1 nanogram 2,3,7,8-TCDD per liter of water (ng/L) (ppt) in 1 day, or more than 0.01 ng/L per day for long-term exposure. For long-term exposure in adults, EPA recommends that there should not be more than 0.04 ng/L (ppt) in drinking water (#ATSDR-Toxicological Profile for Dioxins).

Recently the Environmental Working Group (EWG) has announced some alarming news regarding actual exposure to dioxins among Americans. EWG research found that the amount of dioxin a nursing infant ingests daily is up to 77 times higher than the level the agency has defined as harmless to the endocrine and immune systems. For cancer risk, the situation is even more dire: the general public is exposed (mostly through eating meat, dairy and shellfish) to up to 1,200 times more dioxin than regulatory agencies typically consider safe. EPA is now working on "Reanalysis of Key Issues Related to Dioxin Toxicity" in order to establish a safe daily dose for human exposure to this hazardous chemical (#EWG, 2010).



Source: U.S. EPA (Environmental Protection Agency). 2006. An inventory of sources and environmental releases of dioxin-like compounds in the United States for the years 1987, 1995, and 2000. National Center for Environmental Assessment, Washington, DC; EPA/600/P-03/002F. (http://cfpub.epa.gov/ncea/cfm/recordisplay.cfm?deid=159286).

*"Dioxin" here is defined as the totality of 7 dioxins and 10 furans. "TEQ" denotes "toxic equivalent," a quantitative measure of the combined toxicity of a mixture of dioxin-like chemicals.

Figure 2 demonstrates that, overall, industrial, municipal and transportation dioxin emissions have declined dramatically as a result of regulation, whereas emissions from backyard barrel burning of rubbish and residential wood burning have remained essentially constant since 1987, and as of 2000 have overtaken industrial/municipal/transportation sources as more significant emitters of dioxin (#DioxinFacts).

Ways to Reduce Exposure

- Trimming fat from meat
- Consuming low fat dairy products
- Balanced diet (including adequate amounts of fruits, vegetables and cereals);it will help to avoid excessive exposure from a single source (#WHO).

Source : http://www.toxipedia.org/display/toxipedia/Dioxin