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Polychlorinated dibenzodioxins

See also: Dioxin (chemical) and Dioxins and dioxin-like compounds

Polychlorinated dibenzodioxins (**PCDD**s), or simply **dioxins**, are a group of polyhalogenated compounds which are significant because they act as environmental pollutants. They are commonly referred to as dioxins for simplicity in scientific publications because every PCDD molecule contains a dioxin skeletal structure. Typically, the *p*-dioxin skeleton is at the core of a PCDD molecule, giving the molecule a dibenzo-*p*-dioxin ring system. Members of the PCDD family have been shown to bioaccumulate in humans and wildlife due to their lipophilic properties, and are known teratogens, mutagens, and suspected human carcinogens. They are organic compounds.

Dioxins occur as by-products in the manufacture of organochlorides, in the incineration of chlorine-containing substances such as PVC (polyvinyl chloride), in the bleaching of paper, and from natural sources such as volcanoes and forest fires.[1] There have been many incidents of dioxin pollution resulting from industrial emissions and accidents; the earliest such incidents were in the mid 19th century during the Industrial Revolution.[2]

The word "dioxins" may also refer to a similar but unrelated compound, the polychlorinated dibenzofurans (PCDFs) of like environmental importance.

Chemical structure of dibenzo-*p*-dioxins

The structure of dibenzo-*p*-dioxin comprises two benzene rings joined by two oxygen bridges. This makes the compound an aromatic diether. The name dioxin formally refers to the central dioxygenated ring, which is stabilized by the two flanking benzene rings.

In PCDDs, chlorine atoms are attached to this structure at any of 8 different places on the molecule, at positions 1–4 and 6–9. There are 75 different types of PCDD congeners (that is: related dioxin compounds).[3]

The toxicity of PCDDs depends on the number and positions of the chlorine atoms. Congeners that have chlorines in the 2, 3, 7, and 8 positions have been found to be significantly toxic. In fact, 7 congeners have chlorine atoms in the relevant positions which were considered toxic by the NATO *Committee on the Challenges of Modern Society* (NATO/CCMS) international toxic equivalent (I-TEQ) scheme.

Historical perspective

Low concentrations of dioxins existed in nature prior to industrialization due to natural combustion and geological processes.[4][5] Dioxins were first unintentionally produced as by-products from 1848 onwards as Leblanc process plants started operating in Germany.[2] The first intentional synthesis of chlorinated dibenzodioxin was in 1872.

Today, concentrations of dioxins are found in all humans, with higher levels commonly found in persons living in more industrialized countries. The most toxic dioxin, 2,3,7,8-tetrachlorodibenzo-*p*-dioxin (TCDD), became well known as a contaminant of Agent Orange, a herbicide used in the Vietnam War.[6] Later, dioxins were found in Times Beach,

Missouri [7] and Love Canal, New York [8] and Seveso, Italy. [9] More recently, dioxins have been in the news with the poisoning of President Viktor Yushchenko of Ukraine in 2004, [10] the Naples Mozzarella Crisis [11] and the Irish pork crisis of 2008.

Sources of dioxins

The United States Environmental Protection Agency Dioxin Reassessment Report is possibly the most comprehensive review of dioxins, but other countries now have substantial research. Australia, New Zealand and the United Kingdom all have substantial research into body burdens and sources. Tolerable daily, monthly or annual intakes have been set by the World Health Organization and a number of governments. Dioxins enter the general population almost exclusively from ingestion of food, specifically through the consumption of fish, meat, and dairy products since dioxins are fat-soluble and readily climb the food chain. [12]

Occupational exposure is an issue for some in the chemical industry, or in the application of chemicals, notably herbicides. Inhalation has been a problem for people living near substantial point sources where emissions are not adequately controlled. In many developed nations there are now emissions regulations which have alleviated some concerns, although the lack of continuous sampling of dioxin emissions causes concern about the understatement of emissions. In Belgium, through the introduction of a process called AMESA, continuous sampling showed that periodic sampling understated emissions by a factor of 30 to 50 times. Few facilities have continuous sampling.

Most controversial is the United States Environmental Protection Agency (US EPA) assessment's (draft) finding that any reference dose that were to be set would be far below current average intakes.

Children are passed substantial body burdens by their mothers, and breastfeeding increases the child's body burden. [13] Children's body burdens are often many times above the amount implied by tolerable intakes which are based on body weight. Breast fed children usually have substantially higher dioxin body burdens than non breast fed children until they are about 8 to 10 years old. The WHO still recommends breast feeding for its other benefits. [14]

Dioxins are produced in small concentrations when organic material is burned in the presence of chlorine, whether the chlorine is present as chloride ions or as organochlorine compounds, so they are widely produced in many contexts. According to the most recent US EPA data, the major sources of dioxins are broadly in the following types: [15]

- Combustion sources, e.g. municipal waste incinerators [1]
- Metal smelting
- Refining and process sources
- Chemical manufacturing sources
- Natural sources
- Environmental reservoirs

When the original US EPA inventory of dioxin sources was done in 1987, incineration represented over 80% of known dioxin sources. As a result, US EPA implemented new emissions requirements. These regulations have been very successful in reducing dioxin stack emissions from incinerators. Incineration of municipal solid waste, medical waste, sewage sludge, and hazardous waste together now produce less than 3% of all dioxin emissions.

In incineration, dioxins can also reform or form de novo in the atmosphere above the stack as the exhaust gases cool through a temperature window of 600 to 200 °C. The most common method of reducing the quantity of dioxins reforming or forming de novo is through rapid (30 millisecond) quenching of the exhaust gases through that 400 °C window.[16]

Incinerator emissions of dioxins have been reduced by over 90% as a result of new emissions control requirements. Incineration in developed countries is now a very minor contributor to dioxin emissions.
Dioxins are also generated in reactions that do not involve burning — such as bleaching fibers for paper or textiles[17], and in the manufacture of chlorinated phenols, particularly when reaction temperature is not well controlled. Affected compounds include the wood preservative pentachlorophenol, and also herbicides such as 2,4-dichlorophenoxyacetic acid (or 2,4-D) and 2,4,5-trichlorophenoxyacetic acid (2,4,5-T). Higher levels of chlorination require higher reaction temperatures and greater dioxin production. Dioxins may also be formed during the photochemical breakdown of the common antimicrobial compound triclosan.[18]

Dioxins are also in typical cigarette smoke.[19] Dioxin in cigarette smoke was noted as "understudied" by the US EPA in its "Re-Evaluating Dioxin" (1995). In that same document, the US EPA acknowledged that dioxin in cigarettes is "anthropogenic" (man-made, "not likely in nature"). Nevertheless, the use of chlorine-containing tobacco pesticides and chlorine-bleached cigarette papers remains legal.

Toxicity

Dioxins are absorbed primarily through dietary intake of fat, as this is where they accumulate in animals and humans. In humans, the highly chlorinated dioxins are stored in fatty tissues and are neither readily metabolized nor excreted. The estimated elimination half-life for highly chlorinated dioxins (4-8 chlorine atoms) in humans ranges from 7.8 to 132 years.[20]

The persistence of a particular dioxin congener in an animal is thought to be a consequence of its structure. It is believed that dioxins with few chlorines, which thus contain hydrogen atoms on adjacent pairs of carbons, can more readily be oxidized by cytochromes P450. The oxidized dioxins can then be more readily excreted rather than stored for a long time.

2,3,7,8-Tetrachlorodibenzo-*p*-dioxin (TCDD) is the most toxic of the congeners. Other dioxin congeners (or mixtures thereof) are given a toxicity rating from 0 to 1, where TCDD = 1. This toxicity rating is called the Toxic Equivalence Factor, or TEF. TEFs are consensus values and, because of the strong species dependence for toxicity, are listed separately for mammals, fish, and birds. TEFs for mammalian species are generally applicable to human risk calculations. The TEFs have been developed from detailed assessment of literature data to facilitate both risk assessment and regulatory control.[21] Many other compounds may also have dioxin-like properties, particularly non-ortho PCBs, some of which can have TEFs as high as 0.1.

The total dioxin toxic equivalence (TEQ) value expresses the toxicity as if the mixture were pure TCDD. The TEQ approach and current TEFs have been adopted internationally as the most appropriate way to estimate the potential health risks of mixture of dioxins. Recent data suggest that this type of linear scaling factor may not be the most appropriate treatment for complex mixtures of dioxins; further research into non-linear toxicity models is required to substantiate this hypothesis.

Dioxins and other persistent organic pollutants (POPs) are subject to the Stockholm Convention. The treaty obliges signatories to take measures to eliminate where possible, and minimize where not possible to eliminate, all sources of dioxin.

Health effects in humans

Dioxins build up primarily in fatty tissues over time (bioaccumulate), so even small exposures may eventually reach dangerous levels. In 1994, the US EPA reported that dioxins are a probable carcinogen, but noted that non-cancer effects (reproduction and sexual development, immune system) may pose an even greater threat to human health. TCDD, the most toxic of the dibenzodioxins, is classified as a Group 1 carcinogen by the International Agency for Research on Cancer (IARC). TCDD has a half-life of approximately 8 years in humans, although at high concentrations, the elimination rate is enhanced by metabolism.[22] The health effects of dioxins are mediated by their action on a cellular receptor, the aryl hydrocarbon receptor (AhR).[23]

Exposure to high levels of dioxins in humans causes a severe form of persistent acne, known as chloracne.[24] A case-control study has shown an elevated risk of sarcoma (a type of cancer) associated with low-level exposure (4.2 fg/m^3) to dioxins from incineration plants.[25] High levels of exposures to dioxins have been shown by epidemiological studies to lead to an increased risk of tumours at all sites.[25] Other effects in humans may include:

- Developmental abnormalities in the enamel of children's teeth.[26][27]
- Central and peripheral nervous system pathology[28]
- Thyroid disorders[29]
- Damage to the immune systems[30]
- Endometriosis[31]
- Diabetes[32]

Recent studies have shown that exposure to dioxins changes the ratio of male to female births among a population such that more females are born than males.[33]

Dioxins accumulate in food chains in a fashion similar to other chlorinated compounds (bioaccumulation). This means that even small concentrations in contaminated water can be concentrated up a food chain to dangerous levels due to the long biological half life and low water solubility of dioxins.

Health effects in animals

While it has been difficult to establish specific health effects in humans due to the lack of controlled dose experiments, studies in animals have shown that dioxin causes a wide variety of toxic effects. In particular, TCDD has been shown to be teratogenic, mutagenic, carcinogenic, immunotoxic, and hepatotoxic. Furthermore, alterations in multiple endocrine and growth factor systems have been reported. The most sensitive effects, observed in multiple species, appear to be developmental, including effects on the developing immune, nervous, and reproductive systems.[34] These effects are caused at body burdens close to those reported in humans.

Among the animals for which TCDD toxicity has been studied, there is strong evidence for the following effects:

Birth defects (teratogenicity)
In rodents, including rats,[35] mice,[36] hamsters and guinea pigs,[37] birds,[38] and fish.[39]

Cancer (including neoplasms in the mammalian lung, oral/nasal cavities, thyroid and adrenal glands, and liver, squamous cell carcinoma, and various animal hepatocarcinomas)
In rodents[35][40] and fish[41]

Hepatotoxicity (liver toxicity)
In rodents,[40] chickens,[42] and fish[43]

Endocrine disruption
In rodents and fish[44]

Immunosuppression
In rodents[45] and fish.[46]

Studies of dioxins' effects in Vietnam

US veterans' groups and Vietnamese groups, including the Vietnamese government, have convened scientific studies to explore their belief that dioxins were responsible for a host of disorders, including tens of thousands of birth defects in children, that have affected Vietnam veterans as well as an estimated one million Vietnamese, due to their exposure during the Vietnam War to Agent Orange, a defoliant chemical which was widely sprayed over Vietnamese land and which was found to be highly contaminated with TCDD. Several exposure studies showed that some US Vietnam Veterans who were exposed to Agent Orange had serum TCDD levels up to 600 ppt (parts per trillion) many years after they left Vietnam, compared to general population levels of approximately 1 to 2 ppt of TCDD. In

Vietnam, TCDD levels up to 1,000,000 ppt have been found in soil and sediments from Agent Orange contaminated areas, three to four decades after spraying. In addition, elevated levels have been measured in food and wildlife in Vietnam.[47]

The most recent study, paid for by the National Academy of Sciences, was released in an April 2003 report. This report is currently (March 2007) being revised for release again later in 2007.

The Centers for Disease Control and Prevention found that dioxin levels in Vietnam veterans[48] were in no way atypical when compared against the rest of the population.

The only exception existed for those who directly handled Agent Orange. These were members of Operation Ranch Hand. Long-term studies of the members of Ranch Hand have thus far uncovered a possibility of elevated risks of diabetes.

Dioxin exposure incidents

See also: Dioxin controversy

In 1949, in a herbicide production plant for 2,4,5-T in Nitro, West Virginia, 240 people were affected when a relief valve opened.[49]

In 1963, a dioxin cloud escapes after an explosion in a Philips-Duphar plant (now Solvay Group) near Amsterdam.[2] The plant was so polluted with dioxin after the accident that it had to be dismantled, embedded in concrete, and dumped into the ocean.[50]

Between 1965 and 1968 production of 2,4,5-trichlorophenol in Spolana Neratovice plant in Czechoslovakia seriously poisoned about 60 workers with dioxins; after 3 years of investigations of the health problems of workers, Spolana stopped manufacture of 2,4,5-T (most of which was supplied to the US military in Vietnam). Several buildings of the Spolana chemical plant were heavily contaminated by dioxins.[51] Unknown amounts of dioxins were flushed into the Elbe and Mulde rivers during the 2002 European flood, contaminating the soils.[52] Analysis of eggs and ducks found levels of dioxins 15-time higher than EU limit and high concentrations of dioxin-like PCBs in the village of Libiš.[53] In 2004, the state health authority published a study which analysed the level of toxic substances in human blood near Spolana. According to the study, blood dioxin levels in Neratovice, Libiš and Tišice were about twice the level of the control group in Benešov. The quantity of dioxin chemicals near Spolana is significantly higher than the background level in other countries, e.g., USA, Japan or Spain. According to the US EPA, even the background level can pose a risk of cancer from 1:10000 up to 1: 1000, about 100 times

higher than normal.[54] The consumption of local fish, eggs, poultry and some produce was prohibited because of the post-flood contamination.[55]

In 1976, large amounts of dioxins were released in an industrial accident at Seveso, although no immediate human fatalities or birth defects occurred.[56][57][58]

In 1978, dioxins were some of the contaminants that forced the evacuation of the Love Canal neighborhood of Niagara Falls, New York. Dioxins also caused the 1983 evacuation of Times Beach, Missouri.

From 1982 through to 1985, Times Beach, Missouri, was bought out and evacuated under order of the United States Environmental Protection Agency due to high levels of dioxins in the soil.[59] The town eventually disincorporated.[60]

In December 1991, an electrical explosion caused dioxins (created from the oxidation of polychlorinated biphenyl) to spread through four residence halls and two other buildings on the college campus of SUNY New Paltz.

In May 1999, there was a dioxin crisis in Belgium: quantities of dioxins had entered the food chain through contaminated animal feed. 7,000,000 chickens and 60,000 pigs had to be slaughtered. This scandal was followed by a landslide change in government in the elections one month later.[61]

Explosions resulting from the terrorist attacks on the US on September 11, 2001 released massive amounts of dust into the air. The air was measured for dioxins from September 23, 2001, to November 21, 2001, and reported to be "likely the highest ambient concentration that have ever been reported." [in history]. The United States Environmental Protection Agency report dated October 2002 and released in December 2002 titled "Exposure and Human Health Evaluation of Airborne Pollution from the World Trade Center Disaster" authored by the EPA Office of Research and Development in Washington states that dioxin levels recorded at a monitoring station on Park Row near City Hall Park in New York between October 12 and 29, 2001, averaged 5.6 parts per trillion, or nearly six times the highest dioxin level ever recorded in the U.S. Dioxin levels in the rubble of the World Trade Centers were much higher with concentrations ranging from 10 to 170 parts per trillion. The report did no measuring of the toxicity of indoor air.

In a 2001 case study,[24] physicians reported clinical changes in a 30 year old woman who had been exposed to a massive dosage (144,000 pg/g blood fat) of dioxin equal to

16,000 times the normal body level; the highest dose of dioxin ever recorded in a human. She suffered from chloracne, nausea, vomiting, epigastric pain, loss of appetite, leukocytosis, anemia, amenorrhoea and thrombocytopenia. However, other notable laboratory tests, such as immune function tests, were relatively normal. The same study also covered a second subject who had received a dosage equivalent to 2,900 times the normal level, who apparently suffered no notable negative effects other than chloracne. These patients were provided with olestra to accelerate dioxin elimination. [62]

In 2004, a notable individual case of dioxin poisoning, Ukrainian politician Viktor Yushchenko was exposed to the second-largest measured dose of dioxins, according to the reports of the physicians responsible for diagnosing him. This is the first known case of a single high dose of TCDD dioxin poisoning, and was diagnosed only after a toxicologist recognized the symptoms of chloracne while viewing television news coverage of his condition. [10]

In the early 2000s, residents of the city of New Plymouth, New Zealand, report many illnesses of people living around and working at the Dow Chemical plant. This plant ceased production of 2,4,5-T in 1987.

DuPont has been sued by 1,995 people who claim dioxin emissions from DuPont's plant in DeLisle, Mississippi, caused their cancers, illnesses or loved one's death, of these only 850 are pending as of June 2008. In August 2005, Glen Strong, an oyster fisherman with the rare blood cancer multiple myeloma, was awarded $14 million from DuPont – this ruling was overturned June 5, 2008 by a Mississippi jury who found DuPont's plant had no connection to Mr. Strong's disease. [63] In another case, parents claim dioxin from pollution caused the death of their 8 year old daughter; the trial took place in the summer of 2007, and a jury wholly rejected the family's claims as no scientific connection could be proven between DuPont and the family's tragic loss. [64] DuPont's DeLisle plant is one of three titanium dioxide facilities (including Edgemoor, DE, and New Johnsonville, TN) that are the largest producers of dioxin in the country, according to the US EPA's Toxic Release Inventory. DuPont maintains its operations are safe and environmentally responsible.

In 2007 in Italy thousands of tonnes of foul-smelling refuse are piled up in Naples and its surrounding villages, defacing entire neighbourhoods. Polychlorinated dibenzodioxins are found in animals and humans over lethal dose.[65] Sources of Polychlorinated dibenzodioxins was identified in refuse and pvc combustion and industrial refuse disposal in uncontrolled industrial waste disposal. In numbers animals and humans was found lethal dose.

In December 2008 in Ireland dioxin levels in pork were disclosed to have been between 80 and 200 times the legal limit. All Irish pork products were withdrawn from sale both nationally and internationally.

Main article: Irish pork crisis of 2008

According to the last available data,[66] in 2005 the production of dioxin by the steel industry ILVA in Taranto (Italy) accounted for 90.3 per cent of the overall Italian emissions, and 8.8 per cent of the European emissions.

See also

Polychlorinated dibenzofurans (PCDFs) - an unrelated compound with similar environmental concerns

Chemetco - this former copper smelter is cited in an academic study as one of the 10 highest ranking sources of dioxin pollution reaching Nunavut in the Canadian Arctic

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External links

What is Dioxin?

"Dioxin Homepage at Environmental Justice Advocates"

"EPA: Exposure and Human Health Reassessment of 2,3,7,8-Tetrachlorodibenzo-p-Dioxin (TCDD) and Related Compounds National Academy Sciences (NAS) Review Draft"

"Dioxins and Dioxin-like Compounds in the Food Supply: Strategies to Decrease Exposure", a 2003 report by the National Academy of Sciences

National Pollutant Inventory - Dioxin and Furan Fact Sheet

"Rhodes Remediation" Website about remediation of dioxin contaminated Homebush Bay and land in Rhodes, a suburb of Sydney, NSW, Australia. Union Carbide was the polluter.

"Researcher Dispels Myth of Dioxins and Plastic Water Bottles" Johns Hopkins Researcher explains the facts about Dioxins

"Health Risks from Dioxin and Related Compounds: Evaluation of the EPA Reassessment" Includes discussion of methods of evaluating risk of low concentrations, and Toxic Equivalency

"Dioxins in Cigarette Smoke".

Pesticide residues that are legal contaminants of tobacco

Health effects of dioxins

"Assessment of the Health Risks of Dioxins", a 1998 report by the World Health Organisation.

New Zealand Ministry of Health page on dioxins

IARC monograph: "Polychlorinated Dibenzo-*para*-dioxins"