

What are they and how are they formed?

Dioxins are mixtures of compounds formed by chlorine substitution of the dibenzo-*p*-dioxin molecule. Mixtures of dioxins are more properly designated as polychlorinated dibenzo-*p*-dioxins (PCDDs) and there are 75 possible forms (known as congeners), differing in the number (mono- to octa-) and the positions of chlorine substituents. A group of polychlorinated dibenzo-furans (PCDFs), of which there are 135 congeners, can be produced under similar circumstances and can be considered in the same way as PCDDs. In this note, "dioxins" is used as a general term for mixtures of PCDDs and PCDFs.

There are no beneficial uses of "dioxins" and they are not deliberately manufactured except on a very small scale for use in research. Combustion processes are the major sources of releases to the environment. "Dioxins" are produced as minor components of flue gases during combustion when a source of chlorine is present in the fuel. The production of "dioxins" is favoured by temperatures in the range 200°C to 450°C and some metals e.g. copper and fine particulates can act as catalysts. Such conditions are most likely to occur when the combustion conditions are poorly controlled e.g. bonfires or uncontrolled e.g. forest fires. Dioxins can be produced as minor by-products in some chemical reactions that involve chlorination and this was the source of dioxins in Seveso when a herbicide manufacturing plant exploded. Modern processes ensure that dioxins are no longer formed in significant amounts during normal manufacture of this type of compound.

The UK Dioxin National Action Plan, published in 2006, set out the relative importance of the sources in 2004. A simplified form of this table is shown below.

Sources of Emissions in the UK in 2004

Combustion in Industry , including iron and steel, non-ferrous metals, other industrial combustion	33%
Waste including, landfill, small scale waste burning, incineration of municipal waste(0.1%), other waste disposal	27%
Combustion in Commercial and Domestic Use , including commercial , agricultural, institutional and residential fuel use	6%
Transport , including cars, commercial vehicles, motorcycles, railways and other mobile sources	4%
Combustion in Energy production , including electricity production, oil refining, manufacture of solid fuels, other energy industries	4%
Production Processes , including glass, asphalt, chemical, metal and other products	3%
Bonfire night	2%
Other Combustion Sources including accidental, natural and vehicle fires	21%

What risks do they pose?

The physicochemical, bioaccumulative and toxic properties and persistence in living organisms and in the environment vary with the degree and position of chlorine substitution. It is generally accepted that only a few of the 210 possible dioxin and furan congeners have significant toxicities and 2,3,7,8-tetrachloro-dibenzo-*p*-dioxin (2,3,7,8-TCDD) is considered as the most toxic of these.

A system of "toxic equivalents" (TEQs) has been developed and adopted internationally for relating the toxicities of other dioxin and furan congeners to

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2,3,7,8-TCDD. This allows the toxicities of the “dioxin” congeners in a mixture to be aggregated. The system is based on the ability of each congener to bind to a specific cell receptor involved in the mechanism of toxicity.

In experimental animals, 2,3,7,8-TCDD can cause a wide range of toxic effects including carcinogenicity, endocrine disruption and developmental toxicity. Its toxicity varies widely between test species and the relevance of such studies to adverse effects in man, at realistic exposure levels, is not clear. However, based on studies of workers exposed to high levels, it has been classed as a “known human carcinogen” by government agencies in the USA and by the World Health Organisation. Chloracne is a well-characterised effect arising from occupational exposure to “dioxins” and it can leave sufferers sensitive to dioxins. There is no direct evidence that “dioxins” are affecting the health of the general population.

“Dioxins” probably contribute to problems in wildlife, including poor breeding performance that are known to be caused by other organochlorine compounds such as DDT and PCBs. Because “dioxins” bioaccumulate, species at the top of food chains (e.g. fish, fish-eating birds and mammals) are particularly at risk.

Exposure routes

Occupational exposure can arise where organochlorine compounds are manufactured or handled. Intake can occur through skin absorption, inhalation or ingestion.

The main releases to the environment are to the atmosphere, principally from combustion sources. The total releases to the atmosphere decreased by over 70% between 1990 and 2004 (from 1316g to 357g TEQ). Levels in ambient air are extremely low and, in the general population, over 90% of intake (some estimates are as high as 99%) is via diet, particularly through the consumption of dairy products. Grazing animals ingest “dioxins” deposited on vegetation and, through soil ingestion, the “dioxins” deposited on the soil surface. The ingested “dioxins” concentrate in fatty tissues and part of this intake is then excreted in milk.

In humans, the exposure of breast-fed babies is a particularly sensitive concern. Their exposure depends on the body burden of the mother, which is determined by what she has absorbed from her diet. Because much of a mother’s body burden of “dioxins” is mobilised during lactation, young babies can, for a brief period, receive doses from breast milk that exceed recommended maximum daily intakes. However, such recommended levels are based on intakes over a lifetime and, on balance, the benefits of breast-feeding are thought to outweigh any possible adverse effect from “dioxins”. Dietary intakes are on a downward trend and a recent study indicated that levels in breast milk may have fallen by about 50% since 1993/94.

Inhalation and skin absorption are minor contributors to intake, even in proximity to a point source. “Dioxin” concentrations in air are low and we do not breathe enough air for inhalation to be a major exposure route. For heavy smokers, inhalation is more significant but diet is still the predominant route of exposure.

There are several well-documented cases where “dioxins” have been released into the surrounding area as a result of industrial accidents (e.g. at Seveso in Italy). Monitoring the effects of such accidents has shown increased contamination of plants and soil in the immediate neighbourhood and increased levels in ambient air for a short time. However, studies of those exposed as the result of the accident at Seveso have shown that chloracne, and less severe skin lesions, are the only unequivocal effects on human health.

Control measures

These have been strengthened in the UK in recent years in line with the growing awareness of the potential for adverse effects. Environmental Permitting Regulations (which came into force on 6 April 2008) revoke the Pollution Prevention Control Regulations (2000) and apply to many of the combustion processes that were among the biggest sources of releases. As a result, releases from incinerators burning municipal waste and from electricity generation have been reduced by 99.9% and 95% respectively between 1990 and 2004. Incineration of municipal waste represents only about 0.1% of total emissions.

Annual releases from road transport in the same period fell by 90%, despite the greater number of vehicles on the roads. The fitting of catalysts, stricter emission standards for new vehicles and greater fuel efficiency have contributed to this improvement.

Some chemical products can contain “dioxins” as by-products of the production processes e.g. some pesticides and wood preservatives. In the EU, product standards and controls on the marketing and use of such products have reduced or eliminated these sources. Overall, chemical production contributes only a fraction of 1% of all releases.

As a result of tighter controls on most historical sources of emissions, the principal point sources are now poorly controlled and uncontrolled combustion processes. The former include the small scale burning of wastes in factories and on farms; the latter include bonfires and accidental fires. Not only are such sources difficult to control, they are also very difficult to quantify. Their contribution to total releases are therefore subject to considerable uncertainty.

Dioxins have small but measurable vapour pressures and can be re-mobilised into the atmosphere from soil, sediments, landfills and sites contaminated as a result of former industrial activity. Tighter controls on emissions have highlighted the contributions coming from diffuse sources. Diffuse sources also include “dioxins” reaching the UK as a result of long-range transport from abroad (conversely, remobilisation and long-range transport can remove “dioxins” from the UK environment and deposit them elsewhere). There is indirect evidence that diffuse sources may now be the dominant contribution to emissions in the UK.

Models have been developed to study the remobilisation, transport and deposition of “dioxins” and, while they are subject to considerable uncertainty, they indicate that perhaps 20% of airborne “dioxins” in the UK originate abroad. Remobilisation and long-range transport is a particular problem in colder regions of the world (Arctic and Antarctic) and “dioxins” have been detected in wildlife, far from any known source. “Dioxins” are among the substances subject to international controls under the International Convention on the Long-range Transport of Persistent Pollutants (Stockholm Convention). The UK Dioxin National Action Plan is intended to meet our national obligations under this Convention.

Levels of exposure in the general population

The UK and most European countries work to a tolerable daily intake (TDI) of PCDDs/PCDFs from all sources, of 2pg TEQ kg⁻¹ d⁻¹. (The TDI is the amount of a substance that can be consumed each day for an entire life-time without adverse effect). The US Environmental Protection Agency (EPA) has estimated “a virtually safe dose” 0.006pg TEQ kg⁻¹ d⁻¹. This is not a TDI: it is the level that would result in no more than one excess cancer per million of the general population. (Nearly all the population of the USA are believed to exceed this level). Some of the assumptions used in deriving this value have been much criticised and it is currently being reviewed.

The TDI provides a benchmark against which estimates of current intakes can be evaluated and compared. Diet is the predominant source of human exposure and measuring the small contributions from other exposure routes is difficult, hence estimates of dietary intakes can be used as a close approximation to total daily intake. Estimates of the average dietary intake of adults in the general population in the UK fell from 4.6 to 0.4 pg TEQ kg⁻¹d⁻¹ (91% reduction) between 1982 and 2001. There is no more recent estimate as efforts are now concentrated on surveys of specific types of food in order to eliminate foods that are high in “dioxins” from the food chain and thereby continue to reduce exposure and any possible risk.

Summary

TCDD has been shown to cause a range of toxic effects in experimental animals. However, there is no clear evidence that the health of the general population is affected by past or current levels of “dioxins” in the environment.

For the general population, food, particularly dairy products, is the principal route of exposure. Lactation, inhalation, and skin absorption, even close to a source, are lesser contributors to intake.

Releases to the environment probably peaked in the 1970/80s and have declined sharply since then. Exposure of the general population, as measured in dietary studies, has declined by over 90% since 1982. These trends are likely to continue.

Further reading

Casarett and Doull's Toxicology. The basic science of poisons, 6th Ed., C. D. Klaassen (ed.), McGraw-Hill. 2001.

The Food Standards Agency: <http://www.foodstandards.gov.uk/>

National Implementation Plan for the Stockholm Convention on Persistent Organic Pollutants; DEFRA, December 2006 (This includes the National Action Plan for dioxins).

Dioxin Source Inventory: DEFRA, April 2006.

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Timbrell J., Principles of Biochemical Toxicology, 3rd Ed., Taylor and Francis. 1999.

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