Why do we worry about Dioxins?

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 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 by-products in some production processes (see table for some examples). In particular, chemical reactions that involve chlorination can produce dioxins as by-products. This was the source of dioxins in the Italian town of Seveso when a herbicide manufacturing plant exploded in 1976. 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 2013, set out estimates of the relative importance of sources of emissions. A simplified form of this table is shown below.

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Sources of Emissions in the UK 2010

Combustion in Industry, including iron and steel, non-ferrous metals, other industrial combustion	**12%**
Waste including, landfill, small scale waste burning, incineration of municipal waste(<0.1%), other waste disposal	**21%**
Combustion in Commercial and Domestic Use, including commercial , agricultural, institutional and residential fuel use	**12%**
Transport, including cars, commercial vehicles, motorcycles, railways and other mobile sources	**3%**
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	**25%**
Bonfire night	**4%**
Other Combustion Sources including accidental, natural and vehicle fires	**19%**

What risks do they pose?

The physicochemical, bioaccumulative and toxic properties in living organisms, and persistence in the environment vary with the degree and positions of chlorine substitution. It is generally accepted that only a few of the 210 possible dioxin and furan congeners have significant toxicities and that 2,3,7,8-tetrachloro-dibenzo-p-dioxin (2,3,7,8-TCDD) is the most potent 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 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 (the aryl hydrocarbon receptor, AhR) 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...Based on studies of workers exposed to relatively high levels of TCDD, it has been classed as a “known human carcinogen” 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. However, there is no direct evidence that dioxins are affecting the health of the general population at current levels of exposure.

Dioxins probably contribute to problems in wildlife, including poor breeding performance. Other similar organochlorine compounds such as DDT and PCBs are known to cause such effects. 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.

Historically, the main releases to the environment were to the atmosphere, principally from combustion sources. The total releases to the atmosphere decreased by about 85% between 1990 and 2010 (from1316g to 189g 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 ingesting soil on which dioxins have been deposited. 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 believed to outweigh any possible adverse effect from "dioxins".

Inhalation and skin absorption are minor contributors to intake, even in proximity to a point source. Dioxin concentrations in air are very low and we do not breathe enough air for inhalation to be a major exposure route. For heavy smokers, inhalation is more significant (due to dioxins in tobacco smoke) 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). 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, were the only unequivocal effects on human health.

Control measures

These have been greatly strengthened in the UK in recent years in line with the growing awareness of the potential for adverse effects. The Environmental Permitting Regulations apply to many of the combustion processes that were among the biggest sources of releases. As a result, releases from incinerators burning municipal waste have been almost eliminated and from electricity generation have been reduced by over 90%.

Annual releases from road transport peaked in 1993 and have fallen by 97% 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 about 0.1% of all releases.

As a result of tighter controls on most industrial sources of emissions, the principal point sources of emissions to the atmosphere are now those combustion processes that are difficult or impossible to control. The former include the small scale burning of wastes in factories and on farms; the latter include bonfires and accidental fires. For obvious reasons such sources are difficult to quantify and their contribution to total releases are 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 such secondary ("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). 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 of dioxins is a particular problem in colder regions of the world (Arctic and Antarctic) which act as "sinks". Dioxins have been detected in wildlife in these regions, 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. What are the likely exposures?

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 proposed a "Reference Dose" (RfD) of 0.7pg TEQ kg -1 d -1. This is an estimate of the daily exposure that is likely to be without appreciable risk of deleterious effects during a lifetime for health effects that show or can be assumed to have a threshold. The RfD is similar to a TDI although the latter can include cancer outcomes. The EPA excludes cancer risks in deriving the RfD and intends to publish a separate assessment of cancer risk. . This is likely to assume no threshold exists and hence that there is no "safe level of exposure". A "Reference Dose" for cancer therefore is likely to be a level of exposure that equates to an "acceptable level of risk" (e.g. no more than one excess cancer per million of the general population).

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 $kg^{-1}d^{-1}$ (91% reduction) between 1982 and 2001. There is no more recent estimate as regular surveys are no longer carried out. Efforts are now concentrated on surveys of specific types of food in order to eliminate any that are high in dioxins from the food supply and thus continue to reduce exposure and any possible risk. However, a survey of dietary intake of dioxins (and of other contaminants) was conducted in 2010-2012 but the results have not yet been published.

Summary

TCDD has been shown to cause a range of toxic effects in experimental animals and from studies of occupational exposure it has been classed as a known human carcinogen. 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. Skin absorption and inhalation, even close to a source contribute little 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.

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