



## Scientific Facts on **Dioxins**

**Source document:**  
IPCS - WHO (1998)

**Summary & Details:**  
GreenFacts

**Context** - Dioxins are mainly released by human activities such as incineration and fuel combustion. Some dioxins and some "dioxin-like" PCBs are known to be harmful.

In 2008, the facts presented here are still considered a valid reference. Since 1998, there have been no big changes in our scientific understanding of dioxins - other than to strengthen the conclusions that dioxins are bad actors. General dioxin levels have since continued to drop both in the environment and in people.

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This Digest is a faithful summary of the leading scientific consensus report produced in 1998 by the International Programme on Chemical Safety (IPCS) of the World Health Organization (WHO):  
*"Executive Summary of the Assessment of the health risk of dioxins"*

The full Digest is available at: <https://www.greenfacts.org/en/dioxins/>

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- Each question is answered in Level 1 with a short summary.
- These answers are developed in more detail in Level 2.
- Level 3 consists of the Source document, the internationally recognised scientific consensus report which is faithfully summarised in Level 2 and further in Level 1.

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## 1. What are dioxins?

1.1 "Dioxins" refers to a group of chlorinated organic chemicals with similar chemical structures. Some have harmful properties, depending on the number and position of chlorine atoms in their chemical structure. One of the most harmful dioxins is known as TCDD. Some PCBs, which have similar properties, are considered "dioxin-like".



1.2 Unlike PCBs which were used in several industrial applications, dioxins have no uses. They are formed unintentionally and predominantly released as byproducts of human activities such as incineration and fuel combustion. They are also formed in minor quantities by natural processes such as forest fires and volcanoes.

1.3 Dioxins travel through the air and deposit on water or land. In water, dioxins initially bind to small particles or plankton. On land, dioxins deposit on plants or bind to the soil, most often without contaminating groundwater. Animals accumulate dioxins in fat through their food; concentrations increase at each step in the food chain.

## 2. How are humans exposed to dioxins?

2.1 Over 90% of the human intake of dioxins is through food, mainly from animal origin. The intake [see Annex 1, p. 5] is ten to hundred times higher for breast fed babies than for adults with respect to their body weight. In most industrialized countries, dioxin exposure has been reduced by almost 50% compared to the early 90's.

2.2 Local populations have been accidentally exposed to high dioxin levels, e.g. in Seveso (Italy) after an explosion at a chemical factory, or in Japan and Taiwan with people eating rice oil accidentally contaminated with PCBs and dioxins. In the past, some workers have also been highly exposed to dioxins in waste incineration or chemical plants.

2.3 Dioxins are slowly bio-transformed in the body and are not easily eliminated. They tend to accumulate in fat and in the liver. By interacting with a cellular receptor, dioxins can trigger biological effects such as hormonal disturbances and alterations in cell functions. The mechanism of dioxin toxicity is similar in man and other vertebrates.

## 3. What are the effects of dioxins in laboratory animals?

3.1 Toxic dioxins may cause non-cancer effects to animals, affecting development, reproduction, the immune system and the uterus. Human background exposures in industrialized countries have sometimes reached levels at which these effects were seen in animals.

3.2 In laboratory testing, TCDD and some other types of dioxins increase the number of cancers in several animal species, in both sexes. They do not initiate cancers but promote the growth of existing precancerous cell.

## 4. What are the effects of dioxins on human health?

4.1 For workers accidentally exposed to the highest doses of dioxins, studies estimate that the risk of cancer increases by about 40%. However, the average exposure of the general population is much lower.

4.2 Some delay in nervous system development as well as changes of behavior were seen in children of mothers who had been highly exposed to dioxins and PCBs. In some cases these effects occurred even at current background levels. The effects were likely due to exposure through the placenta rather than through breast milk. However, at least in one case high levels of PCBs and dioxins in breast milk were shown to affect young children's neurobehavioural test results.

4.3 Other non-cancer effects observed on adults accidentally exposed to high levels of toxic dioxins include: diabetes, liver and heart diseases, skin problems (e.g. chloracne), conjunctivitis, fatigue, malaise and slowed nervous reactions.

## 5. How can dioxin exposure be linked to health effects?

5.1 The International Agency for Research on Cancer (IARC) of the World Health Organization (WHO) has proposed a model linking TCDD exposure to cancer in humans. However, studies on rodents show a wide variety of dose-effect relationships.

5.2 Models can not yet predict adequately non-cancer effects in humans but may be used to help understand the effects observed.

5.3 The evaluation of risk posed by mixtures of various dioxin types is more complicated. A Toxic Equivalency Factor (TEF) [[see Annex 3, p. 6](#)] value has been determined for each toxic dioxin. Using these values, a total Toxic Equivalent (TEQ) [[see Annex 3, p. 6](#)] value can be calculated for any dioxin mixture.

## 6. Evaluation and conclusions

6.1 Dioxin levels in food, environmental samples and breast milk have decreased over the 1990s. In most industrialized countries, the daily dioxin intake [[see Annex 1, p. 5](#)] is currently in the order of 1 to 3 pg I-TEQ/kg body weight per day [[see Annex 1, p. 5](#)].

6.2 At very high dioxin exposure, the risk for all cancers combined appears to increase. Non-cancer effects include cardiovascular diseases, diabetes and changes in blood composition. Infants of accidentally highly exposed mothers showed severe developmental and neurological effects.

6.3 A Tolerable Daily Intake (TDI) of 1 to 4 pg I-TEQ per kg body weight per day [[see Annex 1, p. 5](#)] has been established for dioxins by the World Health Organization (WHO). The upper limit of 4 is provisional: the ultimate goal is to reduce human intake [[see Annex 1, p. 5](#)] levels below 1 pg I-TEQ per kg body weight per day [[see Annex 1, p. 5](#)]. This value was derived from the lowest doses causing adverse effects in experimental animals, divided by a safety factor of 10. This Tolerable Daily Intake (TDI) should be seen as an average over a life-time, implying that this value may be exceeded occasionally for short periods without expected health consequences.

6.4 Although breast-fed infants are more exposed to dioxins, under normal conditions the many beneficial effects of human milk generally outweigh the risks. Dioxin levels in human milk have been reduced since the early 90's.

*Note from the editor: In 2008, these conclusions are still considered valid. Since 1998, there have been no big changes in our scientific understanding of dioxins - other than to strengthen the conclusions that dioxins are bad actors. General dioxin levels have since continued to drop both in the environment and in people.*

## Annex

### Annex 1:

#### Daily Dioxin Intakes

The long-term intake of dioxins can be expressed in:

- picogram I-TEQ [see Annex 3, p. 6] **per kilogram of body weight** per day (pg I-TEQ/kg body weight per day, pg I-TEQ/kg bw.day)
- picogram I-TEQ [see Annex 3, p. 6] **per person** per day (pg I-TEQ/person/day); to convert from one unit to the other, it is generally assumed that an adult person weighs 60 kg.

In these units, I-TEQ stands for International Toxicity Equivalent (and is sometimes omitted in writing).More... [see Annex 3, p. 6]

The uptake of dioxins by living organisms leads to accumulation in the body, resulting in a body burden [see Annex 2, p. 5] .More... [see Annex 2, p. 5]

#### Some average daily intakes for long term exposures to dioxins:

	Daily intake (pg I-TEQ per kg body weight per day)	See question
Adults in industrialized countries	1-3	2.1.2 [see <a href="https://www.greenfacts.org/en/dioxins/l-3/dioxins-2.htm#1p2">https://www.greenfacts.org/en/dioxins/l-3/dioxins-2.htm#1p2</a> ]& 6.1.1 [see <a href="https://www.greenfacts.org/en/dioxins/l-3/dioxins-99.htm#1p1">https://www.greenfacts.org/en/dioxins/l-3/dioxins-99.htm#1p1</a> ]
Breast fed babies	Tens to hundreds of times more than adults	2.1.4 [see <a href="https://www.greenfacts.org/en/dioxins/l-3/dioxins-2.htm#1p4">https://www.greenfacts.org/en/dioxins/l-3/dioxins-2.htm#1p4</a> ]
Rice oil contamination with PCBs in Japan (over 1 month)	154 000	2.2.1 [see <a href="https://www.greenfacts.org/en/dioxins/l-3/dioxins-2.htm#2p1">https://www.greenfacts.org/en/dioxins/l-3/dioxins-2.htm#2p1</a> ]
Tolerable Daily Intake,TDI (over a life time)	1-4	6.3.1 [see <a href="https://www.greenfacts.org/en/dioxins/l-3/dioxins-99.htm#3p1">https://www.greenfacts.org/en/dioxins/l-3/dioxins-99.htm#3p1</a> ]
Calculated LOAEL for long term exposure	14-37	6.3.1 [see <a href="https://www.greenfacts.org/en/dioxins/l-3/dioxins-99.htm#3p1">https://www.greenfacts.org/en/dioxins/l-3/dioxins-99.htm#3p1</a> ]

a This is the estimated human long term intake corresponding to the "Least Observed Adverse Effect Level" for the most sensitive adverse responses reported in experimental animals.

### Annex 2:

#### Dioxin Body Burden

Dioxins taken-in by living organisms (see Daily Dioxin Intakes) [see Annex 1, p. 5] and absorbed into the body (uptake), are able to accumulate in the body, resulting in a body burden, which is the total amount of dioxin uptake present in the body at any one time.

For dioxins, the body burden is usually expressed in:

- Mass (weight) Units I-TEQ [see Annex 3, p. 6] **per kilogram of body weight**(pg I-TEQ/kg bw )
- Alternatively, as dioxins accumulate in fat, the body burden can be expressed in picogram I-TEQ [see Annex 3, p. 6] **per gram of serum lipid**, i.e. per gram of fatty substance in the blood (pg I-TEQ/g lipid).

Instead of the weight unit picogram (pg), nanogram (ng) can be used, 1000 pg being 1 ng.

In these units, I-TEQ [see Annex 3, p. 6] stands for International Toxicity Equivalent (and is sometimes omitted in writing).More [see Annex 3, p. 6]

Some Body Burdens at the time of exposure:			
	in pg I-TEQ per kg body weight	in pg I-TEQ per g serum lipid	See question
Adults in industrialized countries	2 000 to 6 000	10 to 30 **	2.1.2 & 7.1.1
Moderately exposed workers	3 000 to 13 000	15 to 650 **	5.1
Highly exposed workers	28 000 to 400 000	140 to 2 400 **	2.2.2 & 7.2
SEVESO incident (median values)	Zone A: 90 000 * Zone B: 25 000 * Max : 10 000 000 *		2.2.1
US Air Force Ranch hands in Vietnam	About 10 000	About 50 **	4.3
* Derived by dividing the body burden in lipids by a factor of 5.			
** Derived by multiplying the body burden per body weight by a factor of 5.			

See also "Estimated tissue concentrations in human populations exposed to dioxin and dioxin-like compounds" in Table 2 question 4.3.

## Annex 3:

### Toxic Equivalent Scheme (TEFs & TEQs)

Dioxins are generally found in mixtures containing several kinds of dioxins and dioxin-like compounds, each having its own degree of toxicity. To express the overall toxicity of such a mixture as a single number, the concept of "International Toxic Equivalent" (TEQ) has been developed.

The "**Toxic Equivalent**" (TEQ) scheme weighs the toxicity of the less toxic compounds as fractions of the toxicity of the most toxic TCDD. Each compound is attributed a specific "**Toxic Equivalency Factor**" (TEF). This factor indicates the degree of toxicity compared to 2,3,7,8-TCDD, which is given a reference value of 1.

To calculate the total TCDD toxic equivalent (TEQ) of a dioxin mixture, the amounts of each toxic compound are multiplied with their Toxic Equivalency Factor (TEF) and then added together.

The TEQ scheme refers **only** to adverse effects (e.g. cancer) following interactions with the cellular Ah receptors. Other toxic effects of dioxins and dioxin-like compounds are not quantified by this method. Toxic Equivalency Factor (TEF) values vary for different animal species.

#### Two schemes:

1) **I-TEF and I-TEQ**: The older International Toxic Equivalent (I-TEQ) scheme by the North Atlantic Treaty Organisation (NATO) initially set up in 1989 and later extended and updated.

2) **WHO-TEF and WHO-TEQ** (also referred to as TEF or TEQ): More recently, the World Health Organization (WHO) suggested modified Toxic Equivalency Factor (TEF) values (see level 3, question 5.3.1, table 3 WHO-TEFs for human risk assessment).

On average, the result of TEQ-calculations is about 10% higher when I-TEFs are used compared to when WHO-TEFs are used.

References to I-TEQ or TEQ are sometimes omitted when figures are given in a text, which makes it impossible to know which TEFs have been used.

See also question 5.3. How are dioxin mixtures accounted for?