# DIOXINS AND DIOXIN-LIKE PCBs IN FOODSTUFFS. LEVELS AND TRENDS

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## Introduction

Human exposure to polychlorinated dibenzo-p-dioxins (PCDDs), dibenzofurans (PCDFs) and biphenyls (PCBs) may occur through background (environmental), accidental and occupational contamination.

For the general human population, the major pathway of exposure to dioxins and PCBs is food. Over 90 percent of human exposure is estimated to occur through the diet, with foods of animal origin usually being the predominant sources (Table 1). Contamination of food is primarily caused by deposition of emissions of various sources (e.g. waste incineration, production of chemicals, metal industry) on farmland and a subsequent accumulation in the food chain in which they are particularly associated with fat. Other sources may include contaminated feed for cattle, chicken and farmed fish, improper application of sewage sludge, flooding of pastures, waste effluents and certain types of food processing [1-4].

**Table 1**
Estimated contributions of ambient, food and non-food sources in total daily exposures to PCDDs and PCDFs for the general population of Canada [5-6], Germany [7] and The Netherlands [8].

| Route of exposure | Canada | | Germany | | Netherlands | |
|---|---|---|---|---|---|---|
| | (pg/day) | (%) | (pg/day) | (%) | (pg/day) | (%) |
| Air and soil | 5.1 | (3.5) | 4.5 | (3.2) | 3.2 | (2.5) |
| Food | 139.7 | (96.1) | 131.2 | (93.7) | 110.2 | (86.6) |
| Non-food | 0.3 | (0.2) | 5.0 | (3.6) | 9.1 | (7.1) |
| Total | 145 | | 140 | | 127 | |

Within the general population, some subpopulations may be exposed to higher amounts of dioxins and PCBs as a result of particular consumption habits (e.g. nursing infants, high fish consumers in contaminated areas). The high exposure of nursing infants during periods of breastfeeding has led to a worldwide concern and to many studies to evaluate the presence of dioxin related compounds in human milk in different countries[9].

In the last few decades, several accidents have been reported causing excess exposure of humans to dioxin related compounds. Well-known examples are exposure of the local population at Seveso, and from fires in PCB filled electrical equipment. High exposure may also be caused by food items accidentally contaminated. Known examples are the contamination of edible oil, such as the Yusho (Japan) and Yu-Cheng (Taiwan) food poisoning. Occupational activities in which 2,3,7,8-TCDD and related compounds are unintentionally produced, such as waste incineration or production of certain pesticides or chemicals may also result in an additional human exposure.

In this paper, the available information on concentrations of PCDDs, PCDFs and (dioxin-like) PCBs in foods is briefly summarized. A more detailed overview will be presented at the symposium.

**Table 2**
Range of concentrations of PCDDs, PCDFs and non-ortho PCBs (in pg TEQ/g fat) in selected food groups as reported by various authors (adapted from [10])

| Food group | PCDDs and PCDFs (pg TEQ/g fat) | Non-ortho PCBs (pg TEQ/g fat) |
|---|---|---|
| Dairy products | 0.2 - 4.3 | 0.5 - 1.8 |
| Farm milk (a) | 0.6 - 70 | |
| Beef fat | 0.3 - 7.2 | 0.9 & 2.4 |
| Mutton fat | 1.2 - 1.8 | 2.0 |
| Pork fat | 0.2 - 0.6 | 0.2 |
| Chicken/poultry fat | 0.2 - 2.3 | (<) 1.6 |
| Horse fat | 14 | 25 |
| Meat products | 0.6 - 2.5 | 0.5 & 1.3 |
| Nuts | 0.2 | 0.05 |
| Eggs | 0.2 - 2.0 | 0.9 & 1.8 |
| Cereals | 0.3 - 0.4 | 0.2 - 1.2 |
| Lean sea fish | <21 - 49 | 20 - 160 |
| Freshwater fish | 0.8 - 46 | 2.9 - 540 |
| Fatty sea fish | 6.8 - 34 | 11 - 160 |
| Game | 17 | 17 |
| Vegetables | 0.1 (b) | 0.015 - 0.4 (b) |
| Vegetable fats and oils | 0.02 & 0.03 | 0.01 |
| Fish oils (raw) | 0.1 - 80 | 0.5 - 12 |
| Fish oils (refined) | 0.1 - 1.8 | 0.3 - 3.4 |

(a): Milk collected in urban and industrial areas in the period 1990-1993
(b): Levels on wet weight basis.

**Occurrence of dioxins and dioxin-like PCBs in foodstuffs**

The range of concentrations as reported until the midth of 1997 is presented in Table 2 (38 publications and reviews). The TEQ values have been calculated using the International TEFs for the PCDDs and PCDFs (NATO/CCMS, 1988) [11], and the WHO-TEFs for dioxin-like PCBs according to Ahlborg et al. [12].

If expressed on fat basis, most foodstuffs contain dioxins and non-ortho PCBs in the range of less than one up to 20 pg TEQ/g fat*. If expressed on whole weight basis, total TEQ levels are generally below 5 pg TEQ/g wet weight (not shown). Higher levels have been found for certain animal fats, in particular for fish (on fat basis) and horse. As can be expected, geographical variations have been found to occur in the concentration of dioxins and dioxin-like PCBs in certain foods. In the vicinity of point sources, concentrations may reach levels up to a factor of ten higher than found at sites remote from industrial activity.

**Temporal trends**

In the last few years, evidence has grown that at least in some industrial nations the exposure of the general population has decreased. In Canada, most food composites did not appear to have changed their contaminant level significantly between 1988 and 1993, except for whole milk (76-87% lower than in 1988). This decrease was attributed to bleaching techniques which have minimized the formation of dioxins and furans[13].

At the DIOXIN'97 symposium in Indianapolis, Fürst and Wilmers[14] reported a strong decline in the average dietary intake due to a lower exposure through the consumption of fish. While in 1991 fish and fish products (like dairy products and meat and meat products, respectively), contributed approximately 30% to the daily PCDD/PCDF intake via food, its corresponding share amounted to only 10% in 1995.

For the Netherlands, a total decrease of 55% in the mean dietary intake of the general population could be attributed to changes in food consumption (about 15%) and to a decrease in levels of dioxin related compounds in the period 1990-1996[15]. A decline in the total daily TEQ-intake from 11.0 to 1.45 pg/kg body weight † in the period 1978-1994 has been found for an adult subpopulation based on recent analyses of duplicate diet samples collected in 1978, 1984/85 and 1994[16].

In 1996, results became available from analysis of Total Diet Study (TDS) samples of about 20 food groups collected in 1982 and 1992 in the UK. Based on seven-day consumption records for over 2000 adults, mean intake estimates were 250 pg (i)-TEQ/day in 1982 and 88 pg (i)-TEQ/day in 1992, and 2.7 and 0.9 pg (WHO)-TEQ/kg.day for the mean intake of dioxin-like PCBs. In the period 1982-1992, there was a decrease in the concentrations of the majority of PCDD, PCDF and PCB congeners quantified in the majority of TDS samples. Small apparent increases were found for concentrations in bread, cereals and milk, which could partly be attributed to the application of upper bound estimates for the concentrations in the respective samples[17].

**Conclusions**

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* Summed TEQ contributions of PCDDs, PCDFs, and of 3 non-ortho PCBs 77, 126 and 169.

† TEQ contributions of PCDDs, PCDFs and dioxin-like PCBs (all congeners except PCB 77).

The database of levels of PCDDs and PCDFs has grown extensively in the past ten years. Recent reviews[19] show that these compounds may be present in all kinds of animal fats included in our normal diet, whereas concentrations are usually negligible in vegetables and fruits. Unfortunately, the present database of dioxin-like PCBs is far less complete than for the PCDD/Fs.
Recent studies from countries which started to implement measures to reduce dioxin emissions in the late 80s, such as Canada, Germany, The Netherlands and the United Kingdom, clearly show decreasing PCDD/PCDF and PCB levels in food and human milk (not discussed here) and, consequently, a significantly lower dietary intake of these compounds by almost a factor of 2 within the past 7 years.

**References**


1. Fürst P, Beck H, Theelen RMC; *Toxic Subst. J.*, **1992**, 12, 133
2. McLachlan MS, and Riechter,W; *J. Agric. Food Chem*., **1988**, 46, 1166
3. Rappe C., Bergek S., Fiedler H., Cooper K.R.; Chemosphere, **1998**, 36, 2705-2720
4. Malisch R; *Organohalogen Compounds*, **1998**, 38, 65
5. Birmingham B, Gilman A, Grant D, Salminen J, Boddington M, Thorpe B, Wile I, Toft P., Armstrong V (1989a); *Chemosphere*, **1989**, 19, 637
6. Birmingham B, Thorpe B, Frank R, Clement R, Tosine H, Fleming G, Ashman J, Wheeler J, Ripley BD, Ryan JJ; *Chemosphere*, **1989**, 19, 507
7. Beck H, Dross A, and Mathar W; *Chemosphere*, **1992**, 25, 1539
8. Theelen RMC; in *Banbury Report 35, Biological basis for risk assessment of dioxins and related compounds*, Eds. Gallo MA, Scheuplein RJ and van der Heijden KA, Cold Spring Harbor Laboratory Press, **1991**, 277
9. WHO/ECEH, Environmental Health in Europe 3, World Health Organization, European Centre for Environment and Health, Bilthoven-Nancy-Rome, **1996**
10. Liem AKD, Theelen RMC *(Thesis),* Dioxins: Chemical Analysis, Exposure and Risk Assessment, Utrecht University, **1997,** ISBN 90-393-2012-8
11. NATO/CCMS; International toxicity equivalency factors (I-TEF) method of risk assessment for complex mixtures of dioxins and related compounds. North Atlantic Treaty Organization, Brussels, report no. 176, **1988**
12. Ahlborg UG, Becking GC, Birnbaum LS, Brouwer A, Derks HJGM, Feeley M, Golor G, Hanberg A, Larsen JC, Liem AKD, Safe SH, Schlatter C, Wærn F, Younes M, Yrjänheikki E; *Chemosphere*, **1994**, 28, 1049
13. Ryan J.J., Beaudoin N., Mills P., Patry B.; *Organohalogen Compounds*, **1997**, 32, 229-232
14. Fürst P, Wilmers K; *Organohalogen Compounds,* **1997**, 33, 116
15. Van Dooren-Flipsen MMH, van Kleveren JD, Liem AKD; RIKILT-DLO report 97.36, **1997**, Wageningen
16. Liem AKD, Hoogerbrugge R, Cuijpers CEJ, Den Hartog RS, Hijman WC, Linders SHMA, Marsman JA, Van der Velde EG, Zomer B; *Organohalogen Compounds*, **1997**, 33, 112
17. Wearne SJ, Harrison N, de M Gem MG, Startin JR, Wright C, Kelly M, Robinson C, White S, Hardy D, Edinburgh V; *Organohalogen Compounds*, **1996,** 30, 1
18. IARC Monographs on the Evaluation of Carcinogenic Risks to Humans, Vol. 69, Lyon, **1997**, 666 pp