DIOXINS AND FURANS IN BREAST MILK IN FRANCE

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Introduction

Dioxins (PCDD) and furans (PCDF), known as "dioxins", are ubiquitous and very stable pollutants. The cancerogenicity and the accumulation of these compounds in living organisms, as well as accidents such as Seveso, have led to an increasing concern in this chemical family from the population, politics and scientists.

In France, the Health Ministry and the Environment and Energy Management Agency (ADEME) have entrusted the National Institute for Public Health Surveillance (InVS) and the Rhônes-Alpes Center of Epidemiology and Public Health (CAREPS) to conduct a study in order to assess the mean concentration of "dioxins" in breast milk in France, to "compare" it within the European Union and to identify the factors which could influence the different levels in the population.

Materials and methods

The study was conducted between 1998 and 1999 in collaboration with the 18 French lactariums where mothers give their breast milk. In each of the eight French territorial zones, about 30 mothers who were willing to cooperate, who were primiparous, healthy, breastfeeding between the 4^{th} and 8^{th} weeks after birth and aged less than 35, were included in the study. Of the women who were asked to participate, less than 10 percent refused to be part of the study.

A total of 244 breast milk samples (350 ml) were collected from the mothers who filled in a standardized questionnaire on their individual characteristics (e.g. age, height and pre- and post-pregnancy weight used to calculate Quételet index, duration of pregnancy, tobacco consumption, education level), the place of residence during the last 10 years (urban/rural, size of the population, proximity to different industries susceptible to release "dioxins", such as metallurgy, incinerator) and occupational exposure. In addition, food habits were assessed through a semi-quantitative questionnaire (frequency and portion of food originating from animals).

The 17 PCDD and PCDF congeners in each individual milk sample were quantified by HPGC/HRMS according to the applied analytical methods by the WHO program[1,2]. The French laboratory was submitted to an interlaboratory comparison with the RIVM, which showed the good quality of our data. Results were essentially expressed using the NATO nomenclature. A statistical analysis was performed to i) describe the study population and the levels of the different congeners and of the I-TEQ PCDD/F and ii) to study the influence of individual characteristics, of food consumption and of environmental exposure using non linear multiple regression models (GAM, S-plus software).

Results and discussion

A. Descriptive results

a) in France

The mean concentration of PCDD/PCDF in milk was 16.5 pg I-TEQ_{NATO} /g lipid, with levels ranging from 6.5 to 34.3 (Table 1). The mean concentration is similar to those from two French studies conducted on small samples of women with heterogeneous characteristics. An exploratory study conducted in 1990 on 15 milk samples from mothers living in Paris or in its suburbs showed a mean concentration of 20.1 pg I-TEQ_{NATO} /g lipid[3]. In 1998, the federal union of consumers (UFC) measured PCDD/F in the milk of 15 mothers from 9 French districts[4]. Seven women were primiparous ; no information was available on age, body mass index, or the time of sampling. PCDD/F levels varied from 14.6 to 35.2 pg I-TEQ/ g lipid.
Our results give a partial figure of the French population's real exposure, since the study only concerned breastfeeding mothers who voluntary gave their milk to the lactariums. Compared to the national statistics on women aged between 20 and 35, the women in the study are more likely to belong to higher socioeconomic classes and to live in rural zones while having a similar food consumption.

Table 1
Levels of the 17 congeners of PCDD and PCDF in pg/ g lipid and I-TEQ (n=244)

	Arithmetic Mean	Standard deviation	Min	Max
2, 3, 7, 8 – Tetrachlorodibenzodioxin	1.72	0.76	0.72	3.33*
1, 2, 3, 7, 8 – Pentachlorodibenzodioxin	6.37	1.98	2.73	14.00
1, 2, 3, 4, 7, 8 – Hexachlorodibenzodioxin	3.20	1.63	1.06	21.51
1, 2, 3, 6, 7, 8 – Hexachlorodibenzodioxin	20.27	7.03	6.18	50.21
1, 2, 3, 7, 8, 9 – Hexachlorodibenzodioxin	4.09	1.59	1.32	12.36
1, 2, 3, 4, 6, 7, 8 – Heptachlorodibenzodioxin	26.68	14.92	5.93	92.84
Octachlorodibenzodioxin	104.93	57.54	22.29	478.84
Total PCDD : I-TEQ PCDD (NATO)	***8.03***	***2.44***	***3.28***	***15.49***
I-TEQ PCDD (WHO)	***11.12***	***3.35***	***4.60***	***21.90***
2, 3, 7, 8 – Tetrachlorodibenzofuran	0.79	0.46	0.00	2.77
1, 2, 3, 7, 8 – Pentachlorodibenzofuran	0.38	0.21	0.00	1.46
2, 3, 4, 7, 8 – Pentachlorodibenzofuran	14.71	5.35	4.74	34.21
1, 2, 3, 4, 7, 8 – Hexachlorodibenzofuran	3.99	1.75	1.51	17.99
1, 2, 3, 6, 7, 8 – Hexachlorodibenzofuran	3.58	1.09	1.36	7.89
2, 3, 4, 6, 7, 8 – Hexachlorodibenzofuran	1.83	0.74	0.55	4.90
1, 2, 3, 7, 8, 9 – Hexachlorodibenzofuran	0.12	0.15	0.00	1.29
1, 2, 3, 4, 6, 7, 8 – Heptachlorodibenzofuran	3.05	1.82	1.06	14.34
1, 2, 3, 4, 7, 8, 9 – Heptachlorodibenzofuran	0.21	0.34	0.00	3.52
Octachlorodibenzofuran	1.10	1.52	0.00	13.30
Total PCDF : I-TEQ PCDF (NATO)	***8.44***	***2.95***	***2.92***	***19.20***
I-TEQ PCDD (WHO)	***8.44***	***2.95***	***2.92***	***19.20***
Total PCDD/F : I-TEQ PCDD/F (NATO)	**16.47**	**5.07**	**6.50**	**34.33**
I-TEQ PCDD (WHO)	**19.56**	**5.96**	**7.82**	**41.10**

* : an extreme value equal to 10.33

b) International comparison

Our results are similar to those observed in the 2d round of the WHO study[1] conducted in different European countries in the nineties. Nevertheless, it is worthy to note that : (i) the current European levels are probably lower than those reported in 1993, since a decrease between 30 and 50% has been observed between 1988 and 1993 ; in Northrhine Westphalia (Germany[5]), levels have also decreased from 1993 to 1997 (about 10% by year), (ii) the selection of women are not very explicit and the sampling can be very different from a country to another, (iii) most countries have used pooled samples and (iv) the demographic and sociologic characteristics of women are not often described. All these factors limit the between-country comparison.

B. Factors influencing levels of PCDD/F

The present study showed that the characteristics of the mother (age, body mass index, tobacco consumption) or of the sample (lipid levels in milk) significantly influenced PCDD/F levels. Omitting to take them into account in the interpretation of the results would lead to false conclusions.

a) Maternal characteristics

PCDD/F levels in milk were strongly influenced by the mother's age, with an increase of 24% for a 5-year increase in age. The highest levels were observed among women above 30 years old. Slowly metabolized and eliminated, these lipophilic and very stable substances gradually accumulated in lipids (half-life in body about 7 years).

The negative association observed between body mass index and the PCDD/F levels, already reported by Cuijpers (1996), may be explained by the dilution of PCDD/F in adipose tissue.

b) Lipid level in maternal milk

The higher the percentage of lipids was in maternal milk, the lower the level of PCDD/F was, probably for the same reason of dilution.

c) Smoking

As reported before[6], we observed that non-smokers had higher levels of many congeners (non significant increase for PCDD/F) than smokers or ex-smokers. An explanation could be that smoking may interfere with the metabolism of congeners, and thereby enhance excretion.

After taking into account the different factors cited above, the study of the respective role of food consumption, urbanization and industries was made possible.

d) Food consumption

Food intake is the main route of human dioxins and furans exposure (more than 90%), particularly through products originating from animals[7]. In the present study, four types of foodstuff were associated with higher levels of congeners : (i) fishing products (fish, shellfish), (ii) pork, (iii) poultry, (iv) eggs. Except for pork, the different associations between foodstuffs and congener levels were observed only with furans. Fishing product intake was associated to the highest increase in congener levels (5 to 6% rise of five furans with an increase in consumption of 300 grams per month). Nevertheless, the congeners implicated were not those which contribute the most to the total I-TEQ of PCDD/F.

Pork intake, although it is known to be a minor contributor to dietary exposure[7], was associated with an increase in the total PCDD/F levels and in 6 furan or dioxin levels.

Egg and poultry intake were also associated with an increase in some furans. Furans involved were not the same for the consumption of eggs or poultry. This result could be explained by the difference of lipids or by a difference in the origin of the products. Furans linked to poultry intake were essentially furans with high degree of chlorination.

e) Residence place

Urbanization was associated with 2,3,7,8 TCDF level, and, to a lesser extent, with 2,3,7,8 TCDD level. Both congeners were found associated with the residence in town. In addition, 2,3,7,8 TCDF was associated with the industrial density and the 2,3,7,8 TCDD with the size of the population.

A striking observation was about total PCDD and in particular 1,2,3,7,8 PeCDD and 1,2,3,6,7,8 HCDD, whose concentrations increased with the time of residence in the rural zone. This result could be explained by the past use of pesticides contaminated with polychlorinated organohalogen compounds or burning operations of products which could lead to dioxins formation.

f) Influence of sources of PCDD/F

The influence of close industries which could release PCDD/F, such as incinerators, metallurgic industries, pesticide plants, paper-pulp production was investigated. A close source of PCDD/F may contaminate locally-produced foodstuffs via the atmosphere. In our study, no association was observed between milk PCDD/F and residence near such a source the last ten years. Nevertheless, recent exposure (the last three years) to an incinerator or to a metallurgic industry was linked to the concentrations of 2,3,4,6,7,8 HCDF, 1,2,3,7,8,9 HCDF, and to a lesser extent, of 2,3,4,7,8 PeCDF. These associations were in fact essentially due to very few subjects. So, these results need caution for the interpretation. Further and specific studies are required on a more important sample of mothers exposed to such sources.

Conclusions

This study gives the first results on PCDD/Fs concentrations in France allowing a comparison with those from the European neighbouring countries. In addition, in considering future policy options, it will constitute a first point to establish a surveillance program on exposures via maternal milk.

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