Dietary intake of organic pollutants in children from Catalonia, Spain

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Introduction

Potential human toxicity of persistent organic pollutants (POPs) is well known¹⁻³. Moreover, it is also well established that dietary intake is the major route of human exposure for most POPs. In recent years, concern on dietary intake of POPs and other organic environmental contaminants has notably increased. Dietary intake of these pollutants is of special interest in children populations, who in relation to their body weights consume greater quantities of food than adults. Consequently, they are more exposed to potentially toxic pollutants. In 2000, a wide survey on dietary intake of a number of organic contaminants by the general population of Catalonia Spain was carried out⁴⁻¹⁰. The levels of the following pollutants were determined in an important number of food samples belonging to various food groups: polychlorinated dibenzo-p-dioxins and polychlorinated dibenzofurans (PCDD/PCDFs), polychlorinated biphenyls (PCBs), polybrominated diphenyl ethers (PBDEs), polychlorinated diphenyl ethers (PCDEs), polychlorinated naphthalenes (PCNs), hexachlorobenzene (HCB) and polycyclic aromatic hydrocarbons (PAHs).

The present study was undertaken to estimate the dietary intake of the above pollutants by children between 4 and 9 years old, as well as to assess the potential health risks derived from this intake.

Materials and Methods

Sample collection: In June-August 2000, food samples were randomly acquired in local markets, big supermarkets, and grocery stores from seven different cities of Catalonia with populations between 150,000 and 1,800,000 inhabitants. A total of 108 samples were analyzed. For collection of food samples, two groups were made up. The first group included meats of beef (steak, hamburger), pork (loin, sausage), chicken (breast) and lamb (steak); fish (hake, sardine) and shellfish (mussel); vegetables (lettuce, tomato, potato, green beans, cauliflower); fresh fruits (apple, orange, pear), and eggs. The second group included cow milk (whole, semiskimmed) and dairy products (yogurt, cheese); cereals (bread, pasta, rice); pulses (lentils, beans); fats (margarine) and oils (olive, sunflower); tinned fish (tuna, sardine), and meat products (ham, hot dogs, salami). Because in the first group most products are usually retailed, their origins could be very diversified in the different cities. Therefore, in that group 4 composite samples were analyzed for each food item. Each composite was made up by 10 individual samples. In contrast, most food items included in the second group corresponded to brands/trademarks that could be acquired in many places.

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Consequently, in this group only 2 composite samples were analyzed for each food item. Each composite was made up by 8 individual samples.

Analytical procedure: Analyses were performed in series of 5 samples and 1 blank. Analytical methods, including preparation of samples for analysis and clean-up, were recently reported⁴⁻¹⁰. Measurement and quantification of PCDD/PCDFs, PCBs, HCB, PCNs, PBDEs and PCDEs were performed by high-resolution gas chromatography/high-resolution mass spectrometry (HRGC/HRMS), model Fisons 8000 GC coupled with a VG Autospec Ultim system (EI and multiple ion determination mode resolution 10000). Analyses of PAHs were done by high performance liquid chromatography (HPLC) on a reverse phase column (C18) with a water/acetonitrile gradient program. Fluorescence detection (multiple wavelengths, programmed) was generally used as the detection method. Quantitative determinations were carried out using internal standards.

Calculations: For PCDD/Fs and PCBs, TEQ calculations were carried out using the WHO-98 toxicity equivalent factors (TEF). For PAHs, TEF proposed by the EPA were used. It was assumed that non-detected isomer concentrations would be equal to one half of the respective limit of detection (ND = $\frac{1}{2}$ LOD). Average daily food consumption data were obtained from a recent diet study carried out in Catalonia¹¹. Population was divided in groups according to different age and sex. Food daily consumption for children was calculated as the mean value for boys and girls between 4 and 9 years old. Daily intake of contaminants was calculated by multiplying the respective concentration in each food by the weight of that food group consumed by an average child from Catalonia.

Results and Discussion

PCDD/PCDF/PCBs: Estimated intakes of PCDD/Fs were 80.1 and 77.6 pg WHO-TEQ/day, for boys and girls, respectively. Taking into account the sum of PCDD/PCDF and PCBs "dioxin-like", intake for boys and girls were 193.5 for 186.4 pg WHO-TEQ/day, respectively. For risk evaluation, an average body weight of 24 kg was assumed. It means a mean daily intake of 7.9 pg WHO-TEQ/kg body weight. PCBs "dioxin-like" contributed to this intake in a 58%, being PCB#126 the most important contributor. The percentages of contribution from each food group to the total dietary intake of PCDD/PCDFs and PCBs "dioxin-like" by children from Catalonia are shown in Figure 1.

In 1998, the WHO proposed a Tolerable Daily Intake for dioxins and "dioxin-like" compounds in the range 1-4 pg WHO-TEQ/kg body weight/day. The non-carcinogenic risks derived from exposure to PCDD/Fs and PCBs "dioxin-like" trough the diet was calculated by dividing the total daily intake by the WHO-TDI. Risk for children resulted in a range of 2.0-7.9, a range significantly higher than that obtained for adults⁴ (0.3-1.4), which in turn is under the WHO-TDI.

For potential carcinogenic effects, risk is expressed as the probability of suffering cancer over the lifetime. Using as a cancer slope factor, that proposed by the US EPA in 2000 (1 x 10^{-3} per pg TEQ/kg/day), the risk level due to PCDD/PCDF exposure trough the diet for children from Catalonia would be about 8000 excess cancer over a lifetime of 70 years. However, it is important to note that in relation to body weight, daily intake decreases significantly after 10 years old to 1.5 pg WHO-TEQ/kg b.w.. Consequently, this exposure would not be so high over lifetime and it might be taken into account for an estimation of the carcinogenic and non-carcinogenic risks.

PBDEs: Total intake of PBDEs through the diet by children from Catalonia was estimated to be 74.6 ng/day. It means 3.1 ng/kg body weight/day, assuming an average body weight of 24 kg. The highest contribution to this intake corresponded to fats and oils (26%), followed by fish and seafood (23%) and meat (21%) (Figure 2).

On the basis of the most sensitive endpoints for toxic effects of PBDEs, a LOAEL (lowest observed adverse effect level) of 1 mg/kg/day was recently suggested as reasonable for compounds or mixtures belonging to the PBDEs group. For the evaluation of the risk derived from the intake of these compounds by children of Catalonia, the comparison of the current dietary intake (3.1 ng/kg body weight/day) with the suggested LOAEL value was made. A safety factor over 5 orders of magnitude in relation to PBDE exposure from food was obtained.

PCDEs: PCDEs could be only detected in samples of fish and seafood. Therefore, the total estimated daily intake of these compounds would be only due to this food group. For children, the estimated daily intake through fish and seafood was 21.5 ng (0.9 ng/kg body weight).

A toxicity similar to that of PCBs has been suggested for PCDEs¹². Although TEFs for the calculations of TEQ for PCDEs are not available, for individuals consuming daily notable amounts of fish and shellfish, contribution of these pollutants to total TEQ could be of concern.

PCNs: In the present survey, total intake of PCNs by children was 39.5 ng/day. TetraCNs were the main contributor, representing 62% of the intake. Fats and oils was the most important contributor group to the total dietary intake of PCNs (39%), followed by cereals (37%) (Figure 3). Based on kg of body weight, children intake of PCNs was 1.65 ng/day. This value is notably higher than that found for adults $(0.54 \text{ ng/kg/day})^8$.

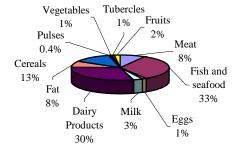
HCB: Hexachlorobenzene intakes were 154.5 and 151.6 ng/day for boys and girls, respectively. The main food groups contributing to these intakes were dairy products, 64% (for boys) and 67% (for girls), and meat with 16% and 17%, for boys and girls, respectively. When intakes of HCB were calculated in relation to body weight, a value of 6.4 ng/kg/day was obtained. This value was remarkably higher than that reported for adults: 2.37 ng/kg/day¹⁰. However, dietary intake of HCB by children is still considerable lower than the WHO tolerable daily intake (TDI), which is 0.17 and 0.16 µg/kg for non-carcinogenic and neoplastic effects, respectively.

PAHs: Total intakes of PAHs were 7.43 and 7.28 μ g/day, for boys and girls respectively. According to the food groups, the main contribution to these intakes was due to cereals (38%), meat (26%), and dairy products (10%) (Figure 4). Assuming a body weight of 24 kg, daily intake by children was 0.3 μ g/kg for total PAHs, which is again higher than the daily intake found for adult men and women (0.12 and 0.11 μ g/kg, respectively)⁹.

Benzo(a)pyrene intake was 0.11 μ g/day for both boys and girls. The sum of carcinogenic PAHs: benz(a)anthracene, benzo(a)pyrene, benzo(b)fluoranthene, benzo(k)fluoranthene, chrysene, dibenz(a,h)antracene and indeno(1,2,3-c,d)pyrene was 1.37 μ g/day for boys and 1.35 μ g/day for girls. In order to estimate the carcinogenic risk, and using the TEF proposed by the US EPA, the total daily intake of PAHs would be associated with a 65/10⁶ increase in the risk for the development of cancer in children. This value is higher than that estimated for adults (5/10⁶).

A summary of the results of the present study concerning the different pollutants analyzed is shown in Table 1. In general terms, in relation to the average body weight, dietary intake by children of all analyzed organic pollutants is higher than those found for the adult population. It indicates that children would be a group of population of special susceptibility to the potential adverse effects of POPs and other organic contaminants. In order to assess the health risks derived from dietary exposure to these compounds, to establish new and clear tolerance levels for children could be of great interest.

BODY BURDENS AND DIETARY INTAKE



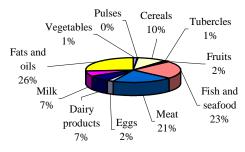


Figure 1. Percentage of contribution of each food group to the total dietary intake of PCDD/PCDFs and PCBs "dioxin-like" by children from Catalonia.

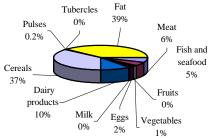
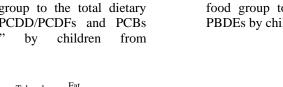


Figure 3. Percentage of contribution of each food group to the total dietary intake of PCNs by children from Catalonia.

Figure 2. Percentage of contribution of each food group to the total dietary intake of PBDEs by children from Catalonia.



Figure 4. Percentage of contribution of each food group to the total dietary intake of PAHs by children from Catalonia.



| Table 1. Dietary intake of some organic ponduants by enhance of Catalonia, Spanis | | | | | | |
|---|----------|----------|----------|----------|----------|----------|
| PCDD/F | PCBs | PCNs | PBDEs | PCDEs | HCB | PAHs |
| (pg WHO- | (pg WHO- | (ng/day) | (ng/day) | (ng/day) | (ng/day) | (µg/day) |
| TEQ/day) | TEQ/day) | | | | | |
| 78.9 | 111.1 | 39.5 | 74.6 | 21.5 | 153.0 | 7.4 |

Table 1. Dietary intake of some organic pollutants by children of Catalonia, Spain.

Acknowledgement

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References

1 De Boer J., Denneman M. (1998) Rev. Environ. Contam. Toxicol. 157, 131.

2 Kogevinas M. (2001) Hum. Reprod. Update 7, 331.

3 Gilbert M.E. (2003) Neurotoxicology 24, 851.

4 Llobet J.M., Domingo J.L., Bocio A., Casas C., Teixido A., Müller L. (2003a) Chemosphere 50, 1193.

5 Llobet J.M., Bocio A., Domingo J.L., Teixido A., Casas C., Müller L. (2003b) J. Food Prot. 66, 479.

6 Bocio A., Llobet J.M., Domingo J.L., Corbella J., Teixido A., Casas C. (2003) J. Agric. Food Chem. 51, 3191.

7 Bocio A., Llobet J.M., Domingo J.L. (2004) J. Agric. Food Chem., in press.

8 Domingo J.L., Falcó G., Llobet J.M., Casas C., Teixidó A., Müller L. (2003) Environ. Sci. Technol. 37, 2332.

9 Falcó G., Domingo J.L., Llobet J.M., Teixidó A., Casas C., Müller L. (2003) J. Food Prot. 66, 2325.

10 Falcó G., Bocio A., Llobet J.M., Domingo J.L., Casas C., Teixidó A. (2004) Sci. Total Environ., *in press*.

11 Capdevila F., Llop D, Guillén N, Luque V, Pérez S, Sellés V, Fernández-Ballart J, Martí-Henneberg C. (2000) Med. Clin. (Barc) 115, 7.

12 Koistinen J., Stenman O., Haahti H., Suonperä M., Paasivirta J. (1997) Chemosphere 35, 1249.