

Current dietary exposure to polychlorodibenzo-*p*-dioxins, polychlorodibenzofurans, and dioxin-like polychlorobiphenyls in Italy

Elena Fattore¹, Roberto Fanelli¹, Aida Turrini² and Alessandro di Domenico³

¹ Department of Environmental Health Sciences, “Mario Negri” Institute for Pharmacological Research, Milan, Italy

² Italian National Institute of Research on Food and Nutrition, Rome, Italy

³ Department of the Environment and Primary Prevention, Italian National Institute for Health, Rome, Italy

This study deals with an assessment of dietary exposure to polychlorodibenzo-*p*-dioxins (PCDDs), polychlorodibenzofurans (PCDFs), and dioxin-like polychlorobiphenyls (DL-PCBs) for the Italian general population, obtained by combining data from a national food consumption survey with contamination concentrations of European foodstuffs available on the market. The distribution of PCDD, PCDF, and DL-PCB dietary intake(s) in the Italian population was investigated to assess to what extent the variability in dietary habits may cause higher exposures to the previously mentioned contaminants. Results indicate that the main contributions to total PCDD, PCDF, and DL-PCB intake are due to fish and fish products (44%) and to milk and dairy products (27%). The mean PCDD, PCDF, and DL-PCB intake (total toxic equivalents) via food was estimated 5.34, 3.37, and 2.28 pg World Health Organization (WHO)-TE/kg of body weight (kg-bw) per day for the three age groups 0–6 (breastfeeding excluded), 7–12, and 13–94 years old, respectively. The highest exposures due to variation in dietary habits are in general within a factor of 2–3. From the mean exposure estimated for the general population (adults), it can be inferred that a consistent part of it would exceed the tolerable daily intake of 2 pg WHO-TE/kg-bw adopted by the Scientific Committee on Food of the European Commission in 2001.

Keywords: Dietary intake / Dioxin-like PCBs / General population / PCDDs / PCDFs

Received: October 19, 2005; revised: January 12, 2006; accepted: February 8, 2006

1 Introduction

Polychlorinated dibenzo-*p*-dioxins (PCDDs), dibenzofurans (PCDFs), and biphenyls (PCBs) are chemical families containing 75, 135, and 209 compounds (generally referred to as congeners), respectively. They exhibit chemical, phy-

sical, and toxicological analogies. Their presence in the environment is unintentional for PCDDs and PCDFs, whereas PCBs were extensively produced by industry in the past and worldwide utilized [1, 2]. Industrial and natural combustion processes, the massive industrial use of chlorine, and a large production of chlorine-containing materials are the main sources of PCDDs and PCDFs, whereas PCBs are now primarily released by environmental reservoirs (soil and sediments), during illegal disposal of contaminated material, and in accidental events.

The previously mentioned contaminants are ubiquitous in the environment and show a strong tendency to bioaccumulation/biomagnification in animal and human tissues. However, as a consequence of the banning of PCBs and the implementation of PCDD and PCDF risk-reducing policies since the 1980s, a decrease in their environmental levels has been observed during the last two decades [3].

The 17 PCDD and PCDF congeners chlorosubstituted at substratum positions 2, 3, 7, and 8, and 12 PCB congeners, which are non- or mono-ortho chlorosubstituted, are usually

Correspondence: Dr. Elena Fattore, Department of Environmental Health Sciences, “Mario Negri” Institute for Pharmacological Research, Via Eritrea 62, 20157 Milan, Italy
E-mail: Fattore@marionegri.it
Fax: +39-02-3900-1916

Abbreviations: **2,3,7,8-TCDD**, 2,3,7,8-tetrachlorodibenzo-*p*-dioxin; **Dioxins**, trivial term to indicate the 17 2,3,7,8-chlorosubstituted toxic congeners of the tetra- to octachlorinated PCDD and PCDF homologues; **DL-PCBs**, dioxin-like polychlorobiphenyls; **EU**, European Union; **kg-bw**, kilogram of body weight; **PCBs**, polychlorobiphenyls; **PCDDs**, polychlorodibenzo-*p*-dioxins; **PCDFs**, polychlorodibenzofurans; **SCF**, Scientific Committee on Food; **TDI**, tolerable daily intake; **TE**, **TEQ**, indication that the analytical values of dioxin and DL-PCB congeners have been converted to toxicity equivalents of 2,3,7,8-TCDD and summed up into a single figure; **TWI**, tolerable weekly intake; **WHO**, World Health Organization

referred to as “dioxins” and “dioxin-like” PCBs (DL-PCBs), respectively. These congeners are highly toxic to mammals and many other animal species and seem to exert their biological activity through the same mechanism of action that involves the binding to cytoplasmatic protein aryl hydrocarbon (*Ah*) receptor [4].

Food consumption represents by far the main intake route of dioxins and DL-PCBs for the general population. In spite of the declining trend, such an intake appears to be close to the exposure doses causing neurobehavioral changes and disturbances to the reproductive system in experimental animals, in particular when exposure occurs during the critical windows of the prenatal and postnatal development [5–11]. In 2000, the Scientific Cooperation of the European Union (EU SCOOP) carried out an assessment of dietary daily intakes in a number of European countries [12], that on average resulted in an estimation of 0.4–1.5 pg international (I)-TE per kg of body weight (bw) for dioxins and 0.8–1.8 pg PCB-TE/kg-bw for DL-PCBs {I-TE and PCB-TE indicate that the analytical values of dioxin and DL-PCB congeners were converted to toxicity equivalents of 2,3,7,8-tetrachlorodibenzo-*p*-dioxin (2,3,7,8-TCDD) according to the I- and PCB-TEF systems [13, 14], not congruent with each other}. In 2001, the Scientific Committee on Food (SCF) of the European Commission adopted a tolerable weekly intake (TWI) for dioxins and DL-PCB together (total TEQ) of 14 pg World Health Organization (WHO)-TE/kg-bw per week, corresponding to a tolerable daily intake (TDI) of 2 pg WHO-TE/kg-bw per day [15, 16]. For conversion to toxicity equivalents of 2,3,7,8-TCDD the late-developed WHO-TEF system was used [17]. According to the EU Scientific Cooperation results, it became evident that a consistent part of the European population exceeded the TWI (or TDI) for dioxins and DL-PCB. Since then, several investigations in different European countries were undertaken to quantify in more detail the dietary intake of these persistent pollutants [18–27].

This paper deals with an assessment of exposure to dioxins and DL-PCBs through diet for the Italian general population, obtained by combining data from a national food consumption survey with contamination concentration data of European foodstuffs available on the market.

The purpose was to describe the distributions of the dietary intakes of dioxins and DL-PCB in the Italian population as part of the European food system, and to investigate to what extent the variability in dietary habits may cause higher exposures to the previously mentioned contaminants.

2 Materials and methods

National food consumption data were provided by the second nation-wide food consumption survey INN-CA 1994–

1996, carried out by the Istituto Nazionale di Ricerca per gli Alimenti e la Nutrizione (INRAN) [28, 29]. Food intake at individual level was recorded for a week on a self-compiled diary over 3 to 7 consecutive days, by people randomly selected to represent four main Italian geographical areas (northwest, northeast, center and south plus islands). Consumption figures in g/person/day were normalized on bw. At the end, the dataset utilized for the present assessment contained the mean consumption rates for 64 food categories based on 3–7-day recordings and 1940 persons 0–94 years old.

By far, most dioxin and DL-PCB concentration data were obtained from an original database available from the European Commission [30]. Where necessary, the database was integrated with pertinent results from international and national surveys. Concentration values – expressed as upper-bound WHO-TEQ – concerned the following food groups: Cereals and cereal products; fruit and vegetables; eggs; fats and oils (butter excluded); fish and fishery products; meat and meat products, comprising subgroups such as poultry, ruminant, and pork meat, and liver (offal); milk and dairy products, also comprising a variety of milk derivatives. For cereals and cereal products, fruit and vegetables, and fish and fishery products, concentrations were expressed on a whole weight, so that dioxin and DL-PCB intakes could be calculated by simply multiplying concentration values for the corresponding consumption rates. Concentrations were expressed on a lipid base for the remaining food groups. Thus they were disaggregated into “specific” components (subgroups and/or items) and then transformed on a whole weight by multiplying for the pertinent average lipid fraction (Table 1), according to INRAN food composition tables [31]. For composite food items (*e.g.* animal fats, beef, ham, salami, sausages, various dairy products), an average weighted lipid fraction was used (Table 1). Once the specific intakes for dioxins and DL-PCB were calculated, data were aggregated again to obtain the intakes associated with the original aforementioned food groups.

The dietary daily intakes were evaluated for the entire sample of subjects and separately for three main age groups 0–6, 7–12, and 13–94 years old. Intake data were tested for normality and log-normality distribution (Shapiro-Wilks and Kolmogorov-Smirnov tests, $p > 0.05$). Pearson's coefficient was utilized to test the existence of a correlation ($p < 0.05$) between dioxin and DL-PCB intake (total TEQ) and age. Finally, the *t*-test to compare two independent samples ($p < 0.05$) was used to test the difference in dioxin and DL-PCB intakes in adult males and females. The statistical analysis was carried out with software Statistica 6.1 (StatSoft Italia).

3 Results

The estimated mean dietary intakes of dioxins and DL-PCBs associated with the main food groups are shown in Fig. 1 for the entire sample of subjects (0–94 years old).

On average, for both dioxins and DL-PCBs the main contributions come from fish and fishery products and, second next, milk and dairy products. However, with reference to the chemicals, the relative weight of these food macrocomponents on total intake differs visibly (Fig. 2). The mean

Table 1. Food breakdown, lipid indicative levels, and mean TEQ concentrations utilized in this Country-related exposure assessment. Based on European representative mean dioxin and dioxin-like PCB occurrence data as reported in text.

Food groups, subgroups, and specific items			Lipids Contents (%)	PCDDs + PCDFs	DL-PCBs
Groups	Subgroups	Items and notes		Concentrations (pg WHO-TE/g) ^{a)}	
<i>Cereals and cereal products</i>				0.027 ww	0.0039 ww
<i>Fruit and vegetables</i>				0.028 ww	0.0039 ww
<i>Eggs</i>				8	1.20 fat
<i>Fats and oils (butter excluded)</i>	Vegetable oil	Olive oil	100	0.21 fat	0.24 fat
		Seeds oil	100	same	same
		Margarine	90	same	same
				0.49 ww	1.30 ww
<i>Fish and fishery products</i>				0.65 fat	0.80 fat
<i>Meat and meat products</i>	Poultry		6	0.46 fat	1.22 fat
	Ruminants	Beef	5	same	same
		Sheep	9	same	same
		Horse	3	same	same
		Rabbit ^{b)}	4	same	same
	Pork	Fresh meat	7	0.21 fat	0.23 fat
		Ham, salami, sausages, etc.	23	same	same
	Liver	Offal ^{c)}	5	5.33 fat	0.64 fat
		Milk	2.6	0.71 fat	2.42 fat
	<i>Milk and dairy products</i>				
		Yogurt	3.0	same	same
		Cream	35	same	same
		Butter	83	same	same
		Cheese	23.5	same	same

a) ww: whole weight; rounding off to a maximum of three figures.

b) And other “white” meat.

c) And miscellaneous meat.

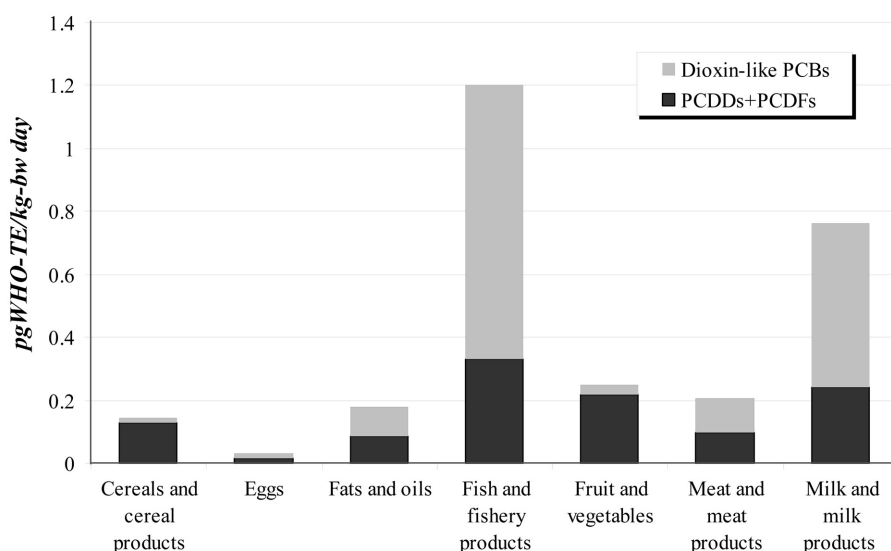


Figure 1. Mean dietary PCDD + PCDF and DL-PCB intakes (pg WHO-TE/kg-bw per day), associated with consumption of food macrocomponents, estimated for the Italian general population.

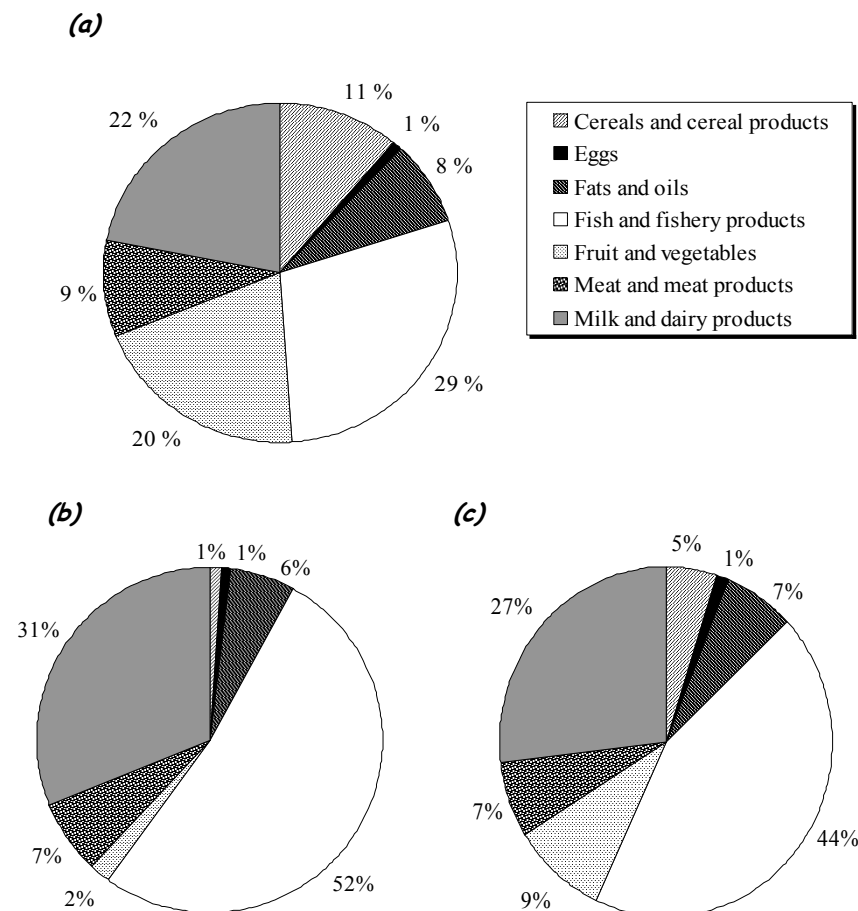


Figure 2. Relative contributions of food macrocomponents to total dietary intakes (pg WHO-TE/kg-bw per day) of PCDDs + PCDFs (a), DL-PCBs (b), and total TEQ (c), estimated for the Italian general population.

intake of dioxins seems to be slightly less unevenly distributed between the different food groups, whereas more than 80% of DL-PCB intake is due to fish and fishery products and milk and dairy products. In addition, the contributions of cereals and cereal products and fruit and vegetables are quite high for dioxins (11 and 20%, respectively) but very low for DL-PCBs (1 and 2%, respectively) (Fig. 2). This distinction reflects the difference in the mean concentrations of dioxins and DL-PCBs in the latter two food groups (Table 1). The contribution due to meat and meat products (Fig. 2) to total intake appears to be similar for dioxins (9%) and DL-PCBs (7%). Within this food group, poultry, ruminant, and pork meats show a similar relative contribution of approximately 2% (breakdown not shown).

The mean intake of dioxins and DL-PCBs in the entire sample of subjects (0–94 years old) was respectively estimated at 1.02 and 1.38 pg WHO-TE/kg-bw per day, indicating that dioxins and DL-PCBs contribute by approximately 42 and 58% to total dietary TEQ intake, respectively.

Even if similar, the mean daily intakes (total TEQ) in adults (13–94 years old) resulted slightly but significantly higher in women than in men. In general, women show higher intakes associated with fruit and vegetables, eggs, fats and oils, fish and fishery products, and milk and dairy products, and lower intakes through cereals and cereal products and meat and meat products.

For the entire sample of subjects (0–94 years old), dietary intake (total TEQ) appears to be inversely correlated with age (Fig. 3). This is likely due to decreasing food consumption (bw-normalized) with increasing age. However, the statistical significance of the correlation was lost when only adults (18–94 years old) were considered. The highest intake value exhibited in the Figure (23.5 pg WHO-TE/kg-bw per day) corresponds to a 1-year-old toddler, who consumed a large amount of fish during the survey.

Table 2 summarizes the estimates of dioxin and DL-PCB intakes via food consumption for the three groups of age considered. Intake data resulted to fit log-normal distribu-

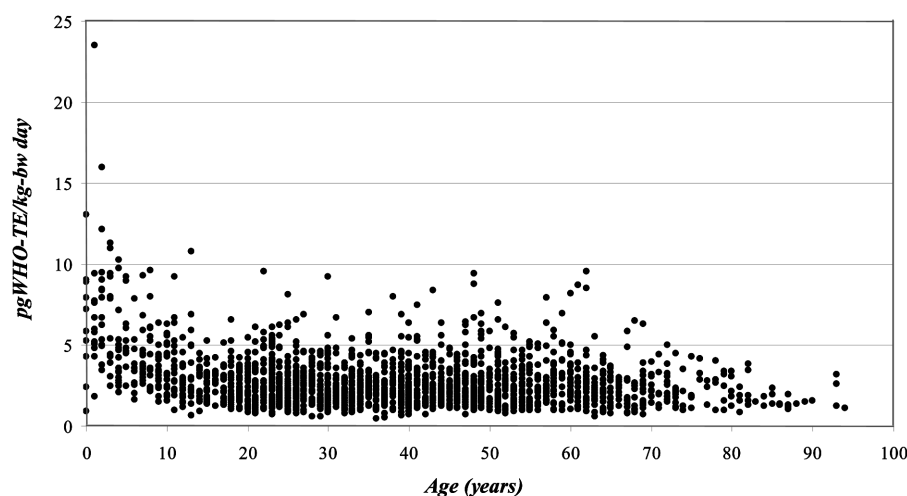


Figure 3. Mean dietary PCDD + PCDF and DL-PCB intake (pg WHO-TE/kg-bw per day) estimated for the Italian general population vs. age.

Table 2. Statistical descriptors of estimated dietary dioxin and dioxin-like PCB intakes (pg WHO-TE/kg-bw per day) for three age groups of the general Italian population

Descriptors ^{a)}	Results by age group ^{b)}		
	Group 0–6 years ^{c)} (N = 88)	Group 7–12 years (N = 105)	Group 13–94 years (N = 1747)
PCDDs + PCDFs			
X _{MIN}	0.34	0.45	0.27
Q ₁₀	1.14	0.85	0.59
X _{MEAN} ^{d)}	2.07	1.40	0.96
CI	1.88–2.28	1.30–1.51	0.84–0.99
Q ₉₀	3.75	2.32	1.56
Q ₉₅	4.44	2.68	2.31
X _{MAX}	7.42	3.44	3.75
DL-PCBs			
X _{MIN}	0.56	0.27	0.19
Q ₁₀	1.40	1.00	0.62
X _{MEAN} ^{d)}	3.11	1.95	1.30
CI	2.72–3.54	1.76–2.15	1.26–1.33
Q ₉₀	6.87	3.80	2.71
Q ₉₅	8.61	4.59	3.34
X _{MAX}	16.1	6.37	7.04
PCDDs + PCDFs and DL-PCBs			
X _{MIN}	1.65	0.95	0.49
Q ₁₀	2.59	1.87	1.23
X _{MEAN} ^{d)}	5.34	3.37	2.28
CI	4.79–5.95	3.08–3.68	2.23–2.33
Q ₉₀	10.5	6.07	4.21
Q ₉₅	12.8	7.16	5.00
X _{MAX}	23.5	9.61	10.8

- a) Q, percentile; CI, confidence interval of the mean ($p = 95\%$).
b) Rounding off to a maximum of three figures and two decimals.
c) Breastfeeding excluded.
d) Undistinguishable from median (Q₅₀).

tions, so that they were log-transformed to derive the statistical descriptive parameters. The derived theoretical curves were used to calculate the high percentile values. Toddlers and young children (0–6 years old, breastfeeding excluded) exhibit the highest estimated mean intake (total TEQ) of 5.34 pg WHO-TE/kg-bw per day, followed in descending intake magnitude by the 7–12-year-old children (3.37 pg WHO-TE/kg-bw per day) and the 13–94-year-old adults (2.28 pg WHO-TE/kg-bw per day).

In all the three age groups, most of the intake values appear to vary within a factor of two around the mean values, whereas maximum values are approximately within a factor of four-to-five. The dioxin and DL-PCB intakes corresponding to the high percentiles and maximum values are generally associated with a high consumption of fish. In this case, fish contribution accounts for 60–70% of total TEQ dietary intake.

4 Discussion

Estimates of dietary intakes in other industrial countries are reported in Table 3. Even if the studies shown are not completely comparable – different methodologies employed, different congeners analyzed, upper-bound vs. lower-bound approximation approach, different statistical treatments, etc. – intakes (total TEQ or contaminant-related) span on average rather narrow ranges, approximately comprised within a factor of three. A recent investigation carried out in the Netherlands [24] reported the lowest dioxin and DL-PCB intakes. This result may be due to a different methodology used in the previously mentioned study to estimate

Table 3. Mean dietary dioxin and dioxin-like PCB intake estimates (pg WHO-TE/kg-bw per day) for adults in different countries^{a)}

Country	PCDD + PCDF	DL-PCB	Total TEQ	References
Belgium ^{b, c)}	1.00	1.04	2.04	[22]
Finland ^{d)}	0.79	0.74	1.53	[25]
Italy ^{d)}	0.96	1.30	2.28	this study
Japan ^{c)}	0.89	1.36	2.25	[20]
Norway ^{d, e)}	1.21	1.52	1.73	[18]
Spain (Catalonia) ^{f, g)}	1.36			[23]
The Netherlands ^{c)}	0.6	0.5	1.1	[24]
USA ^{f)}	1.67	0.65	2.32	[21]

a) Rounding off to a maximum of three figures.

b) Only non-*ortho* dioxin-like PCB included.

c) Lower bound.

d) Upper bound.

e) I-TEQ.

f) Medium bound.

g) Dioxin-like PCB not included.

the life-long average intake and/or to the treatment of analytical concentrations below the limit of determination (upper bound *vs.* lower-bound approach). Nevertheless, different country-related dietary habits may also account to some extent for the diverse exposure estimates in Table 3. For example in the Dutch study, dairy products represent the main contributor (27%) to total dietary intake of dioxins and DL-PCBs, followed by meat products (23%), oils and fats (17%) and fish (16%). In the present evaluation dealing with the Italian population, the contribution of fish and fishery products is the most important, in particular for DL-PCBs, accounting for 44% of the mean total TEQ intake (Fig. 2). Another difference is related to the intake contribution of fruit and vegetables (9%), that appears to be high in this study (Fig. 2) relative to other investigations [23, 24, 26]. Our outcome should be taken with some precaution due to the sparseness of available data above limit of determination for the previously mentioned food group and the upper-bound approach adopted. Nevertheless, the outcome may be quite relevant when the high consumption of fruit and vegetables in the Italian diet is taken into account. Furthermore, according to our data, the impact on dietary intake of dioxins is quite greater than that of DL-PCBs, a plausible consequence of dioxin-contaminated atmospheric depositions on vegetation, not occurring for DL-PCBs.

In this study as in others (Table 3), the average daily intakes estimated are within the range of TDI recommended by the WHO (1–4 pg WHO-TE/kg-bw per day) [32] and slightly above the more recent TDI of 2 pg WHO-TE/kg-bw per day adopted by the EU/EC SCF [15, 16]. In particular, according to the results of the present study, 96% of toddlers and young children (0–6 years old), 87% of children (7–

12 years old), and 61% of adults (13–94 years old) exceed the previously mentioned TDI.

The TDI is clearly exceeded by toddlers and small children (0–6 years old) (Table 2). In this study, breastfeeding was not taken into consideration; however, in other investigations the dioxin and DL-PCB intake via mother milk of a nursing baby was estimated to be about 20 times the TDI and possibly up to two orders of magnitude higher than that of adults [33–37]. Nevertheless, the TDI derived by the EU/EC SCF is not directly comparable to intakes in children, as it is based on the body burden built up following a chronic exposure of 20 or more years and on the adverse effects observed in the offspring of exposed mothers. Even if compared to adults children experience higher exposures, this is for short periods when related to average human lifespan. Moreover, during infancy weight increases rapidly determining a potential dilution of contaminant body burden, whereas contaminant accumulation rate is probably lower due to a faster metabolism and a higher excretion rate: both phenomena tend to hinder contaminant accrual. In general, exposures well above the TDI can be more relevant in increasing body burden when they take place over prolonged lifetime periods, as may be the case of a regular consumption of highly contaminated food components.

The dietary exposure of the Italian population estimated in this study (Table 2) indicates a mean dioxin and DL-PCB intake (total TEQ) for adults slightly above the TDI for 50% of the population, whereas 5% of adults would exceed the TDI by a factor of 2.5 (5.00 pg WHO-TE/kg-bw per day). The highest exposures (total TEQ or contaminant-related), associated with marked variations of diet habits, are in most cases within a factor of two-to-three of the pertinent mean estimates (Table 2). In any case, the intake variability due to dietary habits seems to be relatively low, suggesting the existence of a sort of internal dietary compensation, as-by-and-large expected: for example, if a subject were a high consumer of fish, he would probably cut down on consumption of other nutrient- and energy-rich meat or dairy products. Thus, a substantial lowering of intake below the TDI for a large sector of the population would likely be more efficiently obtained through reducing dioxin and DL-PCB levels in foodstuffs rather than through modification of dietary habits.

5 References

- [1] International Programme on Chemical Safety, World Health Organization (Geneva). Polychlorinated dibenzo-*para*-dioxins and dibenzofurans. *Environ Health Crit.* 1989, 88.
- [2] International Programme on Chemical Safety, World Health Organization (Geneva). Polychlorinated biphenyls and terphenyls (2nd edition). *Environ. Health Crit.* 1993, 140.

- [3] Hays, S. M., Aylward, L. L., *Regul. Toxicol. Pharmacol.* 2003, 37, 202–217.
- [4] Hankinson, O., *Annu. Rev. Pharmacol. Toxicol.* 1995, 35, 307–340.
- [5] Schantz, S. L., Seo, B.-W., Moshtaghian, J., Peterson, R. E., Moore, R. W., *Neurotoxicol. Teratol.* 1996, 18, 305–313.
- [6] Rier, S. H., Martin, D. C., Bowman, R. E., Dmowski, W. P., Becker, J. L., *Fundam. Appl. Toxicol.* 1993, 21, 433–441.
- [7] Gray, L. E., Ostby, J. S., Kelce, W. R., *Toxicol. Appl. Pharmacol.* 1997, 146, 11–20.
- [8] Mably, T. A., Moore, R. W., Peterson, R. E., *Toxicol. Appl. Pharmacol.* 1992, 114, 97–107.
- [9] Mably, T. A., Moore, R. W., Goy, R. W., Peterson, R. E., *Toxicol. Appl. Pharmacol.* 1992, 114, 108–117.
- [10] Mably, T. A., Bjerke, D. L., Moore, R. W., *Toxicol. Appl. Pharmacol.* 1992, 114, 118–126.
- [11] Faqi, A. S., Dalsenter, P. R., Merker, H.-J., Chahoud, I., *Toxicol. Appl. Pharmacol.* 1998, 150, 383–392.
- [12] Health and Consumer Protection Directorate-General, European Commission (Brussels). *Reports on tasks for scientific cooperation, Task 3. 2. 5, Assessment of dietary intake of dioxins and related PCBs by the population of EU Member States.* Released on June 7, 2000.
- [13] Committee on the Challenges of Modern Society, North Atlantic Treaty Organization. *International toxicity equivalency factor (I-TEF) method of risk assessment for complex mixtures of dioxins and related compounds.* NATO/CCMS report 1988, 176.
- [14] Ahlborg, U. G., Becking, G. C., Birnbaum, L. S., Brouwer, A., *et al.*, *Chemosphere* 1994, 28, 1049–1067.
- [15] Scientific Committee on Food, Health and Consumer Protection Directorate-General, European Commission (Brussels). *Opinion of the Scientific Committee on Food on the Risk Assessment of Dioxins and Dioxin-Like PCBs in Food.* Adopted on November 22, 2000.
- [16] Scientific Committee on Food, Health and Consumer Protection Directorate-General, European Commission (Brussels). *Opinion of the Scientific Committee on Food on the Risk Assessment of Dioxins and Dioxin-Like PCBs in Food. Update based on new scientific information available since the adoption of SCF opinion of November 22, 2000.* Adopted on May 30, 2001.
- [17] van den Berg, M., Birnbaum, L., Bosveld, A. T., Brunström, B., *et al.*, *Environ. Health Perspect.* 1998, 106, 775–792.
- [18] Becher, G., Erikseb, G. S., Lund-Larsen, K., Skaare, J. U., *et al.*, *Organohal. Compd.* 1998, 38, 79–82.
- [19] Zuccato, E., Calvarese, S., Mariani, G., Mangiapan, S., *et al.*, *Chemosphere* 1999, 38, 2753–2765.
- [20] Tsutsumi, T., Yanagi, T., Nakamura, M., Kono, Y., *et al.*, *Chemosphere* 2001, 45, 1129–1137.
- [21] Schecter, A., Cramer, P., Boggess, K., Stanley, J., *et al.*, *J. Toxicol. Environ. Health A* 2001, 63, 1–18.
- [22] Focant, J.-F., Eppe, G., Pirard, C., Massart, A.-C., *et al.*, *Chemosphere* 2002, 48, 167–179.
- [23] Llobet, J. M., Domingo, J. L., Bocio, A., Casas, C., *et al.*, *Chemosphere* 2003, 50, 1193–1200.
- [24] Baars, A. J., Bakker, M. I., Baumann, R. A., Boon, P. E., *et al.*, *Toxicol. Lett.* 2004, 151, 51–61.
- [25] Kiviranta, H., Ovaskainen, M.-L., Vartiainen, T., *Environ. Int.* 2004, 30, 923–932.
- [26] Bocio, A., Domingo, L., *Environ. Res.* 2005, 97, 1–9.
- [27] Charnley, G., Doull, J., *Food Chem. Toxicol.* 2005, 43, 671–679.
- [28] Turrini, A., Lombardi-Boccia, G., *Nutr. Res.* 2002, 22, 1151–1162.
- [29] Turrini, A., Saba, A., Perrone, D., Cialfa, E., D'Amicis, A., *Eur. J. Clin. Nutr.* 2001, 55, 571–788.
- [30] Gallani, B., Verstraete, F., Boix, A., Von Holst, C., Anklaam, E., *Organohal. Compd.* 2004, 66, 1917–1924.
- [31] van Leeuwen, F. X. R., Younes, M. M., *Food Addit. Contam.* 2000, 17, 223–240.
- [32] Beck, H., Dross, A., Mathar, W., *Environ. Health Perspect.* 1994, 102 (Suppl. 1), 173–185.
- [33] Schecter, A., Startin, J., Wright, C., Kelly, M., *et al.*, *Chemosphere* 1994, 29, 2261–2265.
- [34] Harrison, N., Wearne, S., de M. Gem, M. G., Gleadle, A., *et al.*, *Chemosphere* 1998, 37, 1657–1670.
- [35] van Leeuwen, F. X. R., Feeley, M., Schrenk, D., Larsen, J. C., *et al.*, *Chemosphere* 2000, 40, 1095–1101.
- [36] Focant, J.-F., Pirard, C., Thielen, C., De Pauw, E., *Chemosphere* 2002, 48, 763–770.