



## DETECTION OF POLYCHLORINATED BIPHENYLS AND DIOXINS IN BELGIAN CATTLE AND ESTIMATION OF THE MAXIMAL POTENTIAL EXPOSURE IN HUMANS THROUGH DIETS OF BOVINE ORIGIN

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## **DETECTION OF POLYCHLORINATED BIPHENYLS AND DIOXINS IN BELGIAN CATTLE AND ESTIMATION OF THE MAXIMAL POTENTIAL EXPOSURE IN HUMANS THROUGH DIETS OF BOVINE ORIGIN**

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*The methodology used to detect a polychlorinated biphenyl (PCB)/dioxin contamination in a Belgian cattle population that was not exposed to the PCB/dioxin incident in 1999 is presented. This population is directly or indirectly destined for human consumption. The methodology consisted in the systematic sampling of all calf-fattening stations and groups of cattle destined for export, and in the random sampling of slaughter cattle. This approach is compared to the method described in directive 96/23/CE from the European Council. When PCB concentrations exceeded the tolerance level of 0.2 µg/g body fat (seven congeners with numbers 28, 52, 101, 118, 138, 153, and 180), dioxins (seventeen 2,3,7,8-substituted congeners of PCDD and PCDF) were also determined. The prevalence of Belgian slaughter cattle with PCB concentrations above this cutoff was 0.3% (95% confidence interval: 0.01–1.50%). Results indicate that the incidence of contamination was minimal, with environmental origin and common in all industrial countries. The maximal potential exposure of an adult human consumer to dioxins through diet of bovine origin is estimated in two worst-case scenarios. The first one corresponds to the consumption of contaminated food products by a small number of consumers during a long period (local consumption) and the second simulates the consumption of contaminated products by a large number of consumers during a short period (supermarket purchase). The theoretical maximum daily intake of dioxins in adults was respectively 374 and 123 pg TEQ/d. The estimated maximum increase of dioxin body burden corresponds to 7 pg TEQ/g fat in the local consumption scheme and 0.07 pg TEQ/g fat in the supermarket consumption scheme.*

In Belgium, ingestion is responsible for 90% (Ansay, 1999; Maghin-Rogister et al., 1999) to 95% (Cox, 1999) of the dioxin exposure in humans. Dioxin accumulation, based on consumption patterns in 1990 (Ministry of Public Health, Consumers Protection and Environment, 1990) and prevalence in Dutch food products is estimated to be 179 pg of 2,3,7,8-tetra-

chlorodibenzo-*p*-dioxin equivalent (toxic equivalent quantity of TCDD, TEQ) per day (Cox, 1999). For an adult consumer of 60 kg this estimate corresponds to an average of 3 pg TEQ/kg body weight/d with 43% coming from beef and milk products (average of 77 pg TEQ/d). Foodstuffs of animal origin normally contribute about 80% of the overall exposure (European Commission, 2000a, 2000b), although no single food group emerges as principal contributor (Guo et al., 2001). Contamination of food is primarily caused by the release of dioxins from various sources and subsequent accumulation in the food chain is due to the high affinity of dioxins for lipid-rich tissues (Matthews & Dedrick, 1984). Various effects have been reported in animals exposed to polychlorinated dibenzodioxins (PCDDs), polychlorinated dibenzofurans (PCDFs) and polychlorinated biphenyls (PCBs) (Weber & Greim, 1997). Many of the toxic effects of dioxins were high dose effects (European Commission, 2000b). The most commonly reported pathologies are endometriosis, developmental neurobehavioral effects, developmental reproductive effects and immunotoxic effects (World Health Organization, 1998a; Kavlock et al., 1996; Weber & Greim, 1997). Dioxins are carcinogenic in several animal species (Mann, 1997) and humans, and increased risk of cancer has been demonstrated at exposure levels more than 100 times the normal intake of the general population (Steenland et al., 1999). Dioxins are classed as human carcinogenic by the International Agency for Research in Cancer (McGregor et al., 1998).

The half-life of 2,3,7,8-TCDD in adults has been estimated as 7 yr (Flesch-Janys et al., 1996; Pirkle et al., 1989), 7.6 yr during a 15-yr follow-up (Michalek et al., 1996), and 8.7 yr during a 10-yr follow-up (Michalek & Tripathi, 1999) and 4 mo (Ansay, 1999; Kreuzer et al., 1997) to 6 mo (Maghin-Rogister et al., 1999) for infants. In cattle, a half-life of 41 d has been found for dairy cows (Jensen & Hummel, 1982) and 16.5 wk for beef (Jensen et al., 1981). Other studies have demonstrated that PCB elimination in milk in cattle and humans is proportional to the level of exposure, and in the case of cattle amounted to 11–12% of the daily dose consumed (Willet, 1975). Half-life increases higher up in the food chain (Delaunoy, 1998). Knowing that a period equivalent to 7 half-lives is required for elimination of 99% of a contaminant (Maghin-Rogister et al., 1999), average periods of respectively 10 mo for cows, 27 mo for bulls, and 50 yr for humans are necessary.

During two exposure studies coordinated by the World Health Organization (WHO), dioxin concentrations in human milk observed in Belgium were among the highest in industrialized countries with mean values in the 3 areas studied ranging from 33.7 to 40.2 in 1986/1987 and from 20.8 to 27.1 pg TEQ/g fat in 1992/1993 (World Health Organization, 1996). In 1999–2000, another study was conducted in rural and industrialized areas involving 112 subjects (average age of 50 yr) living in the central and southern parts of Belgium (Bernard and Fierens, personal communication). In this study, the dioxin body burden for the whole population averaged 30.2 pg TEQ/g fat (geometric mean) and ranged from 5.5 to 114.8 pg TEQ/g

fat. The current Belgian results correspond to other European observations for subjects with the same average age: in Finland, 33 pg TEQ/g fat (Tuomista et al., 1999), and in Germany, 21.9 pg TEQ/g fat in the general population (Päpke, 1998) and 40.8 pg TEQ/g fat in rural areas (Ewers et al., 1996). Taking into account the age differences, the results of dioxin measurements indicate that the dioxin body burden continued to decrease. According to the correlation between age and dioxins concentration in serum lipids (Bernard et al., 2001), the decrease is estimated to average 10–15 pg TEQ/g fat over a period of 20 yr. Using the available toxicological database on dioxins in 1998, the WHO established a tolerated daily intake (TDI) in humans between 1 and 4 pg TEQ/kg body weight/d (Van Leeuwen & Younes, 2000). This TDI is based on the range of lowest observed adverse effect level (LOAEL) of 14–37 pg TCDD/kg body weight/d and applying an uncertainty factor of 10 (World Health Organization, 1998a). The upper limit of the TDI should be considered as the maximum tolerable intake on a provisional basis. The mean intake of dioxins and related compounds from food was estimated between 2.2 and 2.4 pg TEQ/kg body weight/d for adults in the United States (Schechter et al., 2001).

In February 1999, a case of poisoning came to light in several industrial poultry farms in Belgium (Bernard et al., 1999, 2002). The source of the contamination was recycled fat delivered to several feed producers between January 19 and 31, 1999. This article presents the methodology used to detect possible PCB/dioxin contamination in that part of the Belgian cattle population, directly or indirectly destined for consumption and not exposed in the PCB/dioxin incident in 1999. It consisted of a systematic sampling of all calf fattening centers, systematic sampling of all lots of exported bovines, and a random sampling of slaughter cattle. This methodology is compared to the approach described in directive 96/23/CE of the European Council (European Commission, 1996). The maximum potential consumer exposure to dioxins through beef and dairy products is also estimated in two scenarios: local consumption and supermarket consumption.

## **MATERIALS AND METHODS**

### **Analysis of PCBs**

This includes the determination of a set of 7 “indicator” PCBs (International Union of Pure and Applied Chemistry; IUPAC numbers 28, 52, 101, 118, 138, 153, and 180) and is expressed as the sum of these 7 marker congeners (in  $\mu\text{g/g}$  fat). For the sum of these congeners a provisional cutoff was pegged at 0.1  $\mu\text{g/g}$  milk fat and 0.2  $\mu\text{g/g}$  body fat (European Commission, 1999c). The procedures for the PCB analyses used by the different laboratories were described previously and an interlaboratory comparison program was organized to ensure the uniformity of the results obtained from the different participating laboratories. The between-laboratory coefficient of variation was estimated at 30–35% (Beernaert & De Poorter,

1999). All methods applied were based on gas chromatography (e.g., GC-ECD). The advantages of the dosage of PCBs compared to dioxins were a rapid test protocol and the fact that a larger number of laboratories could participate.

### **Analysis of PCDDs and PCDFs**

When PCB concentrations exceeded the above cut-off values, dioxins were also determined. The procedures for PDDD and PCDF analyses used by a limited number of laboratories consisted in gas chromatography—high-resolution mass spectrometry (GC-HRMS) after extraction of fat, purification and concentration of samples. The standard method included the determination of the 17 2,3,7,8-substituted congeners. Determination of these congeners provides diagnostic information to evaluate the concentrations of the most toxic dioxins. The concentration in TEQ was obtained by multiplying the concentration for each congener (usually expressed in pg/g fat) by a toxic equivalency factor (I-TEF or WHO-TEF in this case). These procedures are described elsewhere (Van Den Berg et al., 1998).

### **Systematic Sampling of All Calf Fattening Centers**

All calf fattening centers were sampled (the sampling unit was the animal, the primary sampling cluster was defined as a group of animals with a common feed supplier, and the secondary cluster was the center). Three samples of 40 g fat, taken randomly from three animals per primary sampling unit, were pooled. All samples were analyzed for PCBs by SGS-Agri-lab (accredited by Beltest; norm EN-45004 type A). The sum of the seven congeners was determined.

### **Systematic Sampling of Bovines Before Export**

Between August 2 and October 3, 1999, samples of all lots of bovines were tested at most 1 mo before export, according to decision 99/449/EC as modified by decision 99/551/EC of the European Commission (European Commission, 1999a, 1999b). The epidemiological unit for this sampling is an exported lot of animals. When the number of animals in the lot was below 8, all animals were submitted to a sampling of 100 g fat. When the number of animals in a lot was between 8 and 500, a random sample of seven animals was tested. This sampling frame was designed to have a probability of 95% to detect contamination of 25% or more of the animals in a lot (the so-called quality level of 0.25 as demanded by Ministry of Public Health of Consumers Protection and Environment). In total, 3409 samples were submitted by the Provincial Veterinary Laboratories (PVL) to PCB analysis in different private laboratories that were accredited by the Ministry of Agriculture. During this period the percentage of bovines exported for slaughter was 62% (August) and 39% (September). In case of a PCB concentration above the cutoff level, the lot in question was not exported, additional samples were collected and tested for dioxins, and an

epidemiological, food and environmental inquiry was performed to determine the source of contamination.

### **Random Sampling of Slaughter Cattle**

On a random day during the week of August 23 to 27, 1999, the resident veterinary officials in all cattle slaughterhouses sampled one bovine per herd presented for slaughter. Each sample consisted in 100 g fat. A total of 1235 samples were collected. All samples were dispatched to and stocked at the PVL of Ciney. During the period in question about 17,000 bovines were slaughtered weekly in Belgium. A sensitivity and specificity of PCBs analysis of 100% was assumed, in the absence of actual values. The actual number of samples submitted to PCB analysis was calculated according to the modified formula from Cannon and Roe (1982) and Martins et al. (1987), using a confidence level of 95% and a 1% minimum detectable prevalence of PCB concentrations above cutoff. The required sample size was calculated as 299 and these 299 samples were taken randomly from the 1235 samples in stock, proportionally stratified by province and age group of the animals. All samples were analyzed for PCBs by SGS-Agrilab (accredited by Beltest, norm EN-45004 type A). The sum of the seven congeners was determined.

### **Collection of Epidemiological, Feed-Related, and Environmental Data**

A relational database, containing the following information, was used: sample identification, registration of herds and animals, listing of restricted herds, risk analysis, results of PCB or dioxin analysis, and reports of visits. For all positive PCB results epidemiological, feed and environmental investigations were performed in the herds of origin. These investigations were based on a checklist and a standard report of visit

### **Statistical Analysis**

The statistical analysis were carried out in Stata version 6 (StataCorp, 1999).

### **Estimation of the Potential Consumer Exposure to Dioxin of Through Beef and Dairy Products**

Two scenarios were investigated. The first scenario simulated consumption at local level (either the producer or some local consumer). In this situation, the exposure to contamination is high, because the contaminated foodstuffs are consumed during a very long period by few people (i.e., high cumulative exposure level). Consumption of whole-milk products prevails in this situation (higher percentage of fat). The second scenario describes the situation when products are marketed through supermarkets and are consumed by a large number of consumers during a short period (i.e., low cumulative exposure level). In this case, half-skimmed milk and half-skimmed milk products are the rule. The raw data concerning consumption

came from the available current reports (Van Hecke, 2000; Confédération Belge de l'Industrie Laitière, 1999; World Health Organization, 1998b). The concentrations of dioxins in fat from milk and meat were obtained respectively from the data of the General Food Inspection (58 tank truck samples from all provinces) and from results of the systematic sampling of all lots of bovines before export (30 positive individual samples). Calculation of the theoretical maximum daily intake (TMDI) of dioxins (pg TEQ/d) from single products of bovine origin in adult consumers of 60 kg are based on the following formula:

$$\text{TMDI} = \frac{\text{CCBO (g/day)} \cdot \% \text{ fat} \cdot 97.5 \text{ PDC (pg TEQ/g fat)}}{\text{body weight (kg)}}$$

where CCBO is consumption of commodities of bovine origin and PDC is percentile of dioxin concentration.

The evolution in time of the body burden are based on the following formula:

$$\text{BB}_t = 30.5e^{-\lambda t} + \sum_{i=0}^{\min(t,C)} (de^{-\lambda(t-i)})$$

where BB is the body burden,  $\lambda$  the exponential decay constant = 0.000049,  $t$  the time since start of exposure (days),  $C$  the length of exposure period, and  $d$  the daily intake.

## RESULTS

### Bovine Herds Exposed to the PCB/Dioxin Incident in 1999

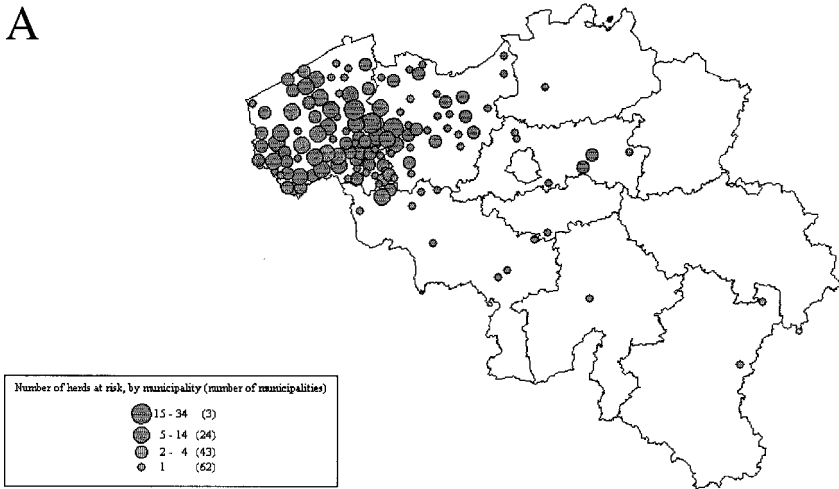
In the cattle sector, 409 herds were exposed to the risk of contaminated feed (0.81% of the Belgian cattle herds) (Figure 1a). These herds were temporarily blocked from all commercial transactions. This measure was lifted on the basis of the results of feed and epidemiological investigations, on the basis of PCB/dioxin analyses carried out on a representative sample of animal products originating from the concerned cattle herds (cutoff: 0.1  $\mu\text{g}$  PCB/g milk fat, 0.2  $\mu\text{g}$  PCB/g body fat, and/or 5 pg TEQ dioxin/g fat), or on the basis of stamping out and incineration of all cattle originating from the 6 herds with PCB/dioxin analyses results above the agreed cutoff. A significant correlation was observed between PCB and dioxin concentration in bovine fat ( $r = .842$ ;  $p = .0006$ , Figure 2).

### Systematic Sampling of All Calf-Fattening Centers

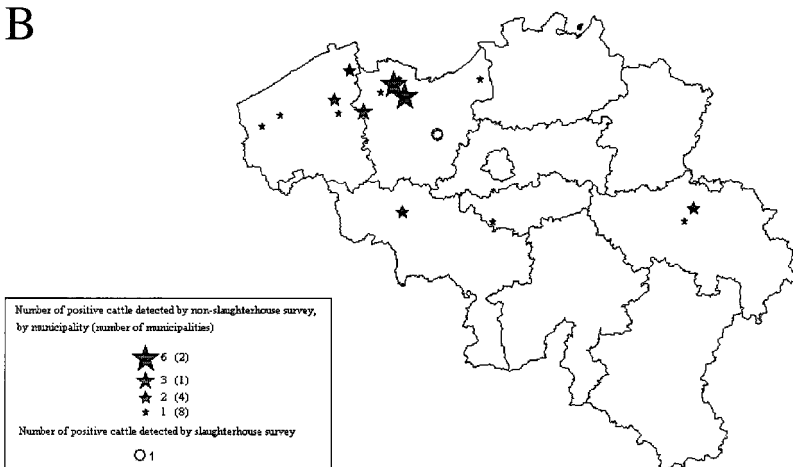
At the time of sampling, 386 calf-fattening centers were active with an average of 450 calves per center. All samples tested negative (range: 0.01–0.04  $\mu\text{g}$  PCB/g body fat), with the exception of one center that had an outlier of 0.277  $\mu\text{g}$  PCB/g fat. Retesting of the same sample in another laboratory



A



B

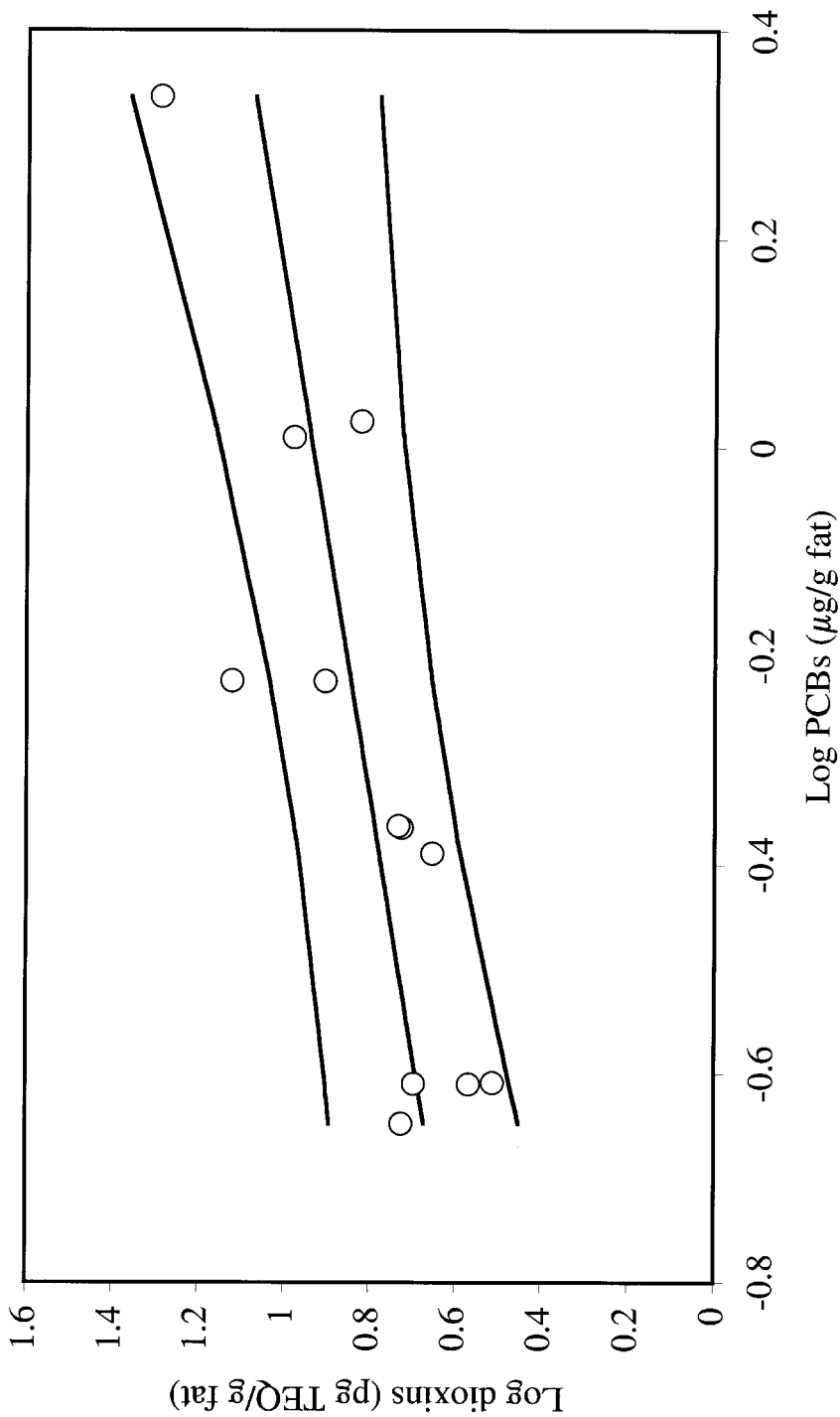


**FIGURE 1.** Geographical location of cattle exposed to the PCB/dioxin incident in 1999 (A) and exposed to environmental contamination of feed (B).

gave a result of 0.166  $\mu\text{g/g}$  fat. An epidemiological, food, and environmental inquiry was immediately carried out in that center and no risk factor could be identified. The center was supplied by two feed producers, one of which did receive fat from the specialized firm but not during the period of risk of contamination. A feed monitoring program performed at this producer yielded only negative results. In addition, extra samples from the center concerned as well as from the three herds where the calves were born tested negative.

### Systematic Sampling of All Lots of Bovines Before Export

Only 31 results with levels over 0.2  $\mu\text{g PCB/g}$  body fat were observed (0.91%; 95% confidence interval: 0.62–1.28): 20 samples with levels be-



**FIGURE 2.** Correlation between concentration of PCBs and dioxins in cattle exposed to PCBs/dioxins during the incident in 1999. Dots, observed values; lines, upper limit 95% confidence interval of the prediction, predicted value, and lower limit of the 95% confidence interval of the prediction.

tween 0.2 and 0.5  $\mu\text{g/g}$  fat (0.59%), 8 with levels between 0.5 and 1  $\mu\text{g/g}$  fat (0.23%), and 3 with results equal to or above 1  $\mu\text{g/g}$  fat (0.09%) (Figure 1b). These positive results were found in animals originating from 15 different herds. Three of the herds showed 9 bovines with results above 0.5  $\mu\text{g/g}$  fat (including all results  $\geq 1$   $\mu\text{g}$  PCBs/g fat, where the results of dioxin analysis were also above 5 pg TEQ/g fat). For these herds a purely environmental source of contamination was identified: ironworks and high-voltage transformers (Debondie et al., 2000). These herds were cleared by destruction of the animals and/or products (with fair compensation for the losses) followed by rehabilitation of the production site.

### Random Sampling of Slaughter Cattle

Only 1 out of 299 samples of body fat was found positive for PCBs: 0.837  $\mu\text{g/g}$  body fat (Figure 1b). Thus, the estimated prevalence of PCBs above 0.2  $\mu\text{g}$  PCBs/g body fat in Belgian slaughter cattle was 0.33% with an Enhanced Wald 95% confidence interval of 0.01% to 1.50%. The farm where the animal in question had originated received feed products from a feed producer that was not supplied with fat by the specialized firm. Feed quality monitoring of the feed producer yielded negative results for PCBs. The positive animal was a calf 4 mo of age, chronically infected with bovine viral diarrhea (BVD—debilitating infection that reduces body weight and thus increases concentration of PCBs in remaining fat). In the herd of origin of this calf an epidemiological, digestive, and environmental inquiry was immediately carried out and four additional samples of fat were analyzed for PCBs; results were negative. No source of contamination was found. The calf's mother, which was 6 yr old, had been culled (BVD eradication program) and might have contaminated the calf. This cow was born in another farm, but this primary farm had been closed for 3 yr. In this farm a potential industrial source of contamination was identified.

### Estimation of the Potential Exposure of Consumers to Dioxins Through Products of Bovine Origin

*Local consumption* An exposure period of 1 yr for meat products (3 frozen contaminated beef meals per week) and a period of 6 mo for whole milk products (farm to consumer) were assumed. For bovine products, in view of the rapid clearance by lactating cows, it was assumed that the highest values found in culled cow meat were representative of the maximal levels attainable in dairy cows (8.41 pg TEQ/g fat), and thus in dairy products such as meat, milk, butter, and cheese. Under these conditions, the TMDI of dioxins from products of bovine origin in adult humans is 374 pg TEQ/d (Table 1). With this level of exposure, an adult consumer of 60 kg experiences a maximum cumulative intake of 63,335 pg TEQ observed after 6 mo. According to the current Belgian data (Bernard & Fierens, personal communication), the increase dioxin body burden is respectively 7

**TABLE 1.** Simulated Total Dioxin Intake in the Case of Local Consumption (Loc) and Supermarket Distribution (Sup)

	Consumption		Percent fat <sup>a</sup>		Fat intake (g/d)		Dioxin intake (pg TEQ/d) <sup>k</sup>	
	ml/d	g/d	Loc <sup>b</sup>	Sup <sup>c</sup>	Loc <sup>b</sup>	Sup <sup>c</sup>	Loc <sup>b</sup>	Sup <sup>c</sup>
Cattle milk	187.7 <sup>d</sup>	—	4.1 <sup>g</sup>	2.1	7.8	3.9	65.2	10.3
Cattle butter milk	5.2 <sup>d</sup>	—	0.4 <sup>h</sup>	0.4 <sup>h</sup>	0.0	0.0	0.2	0.1
Cattle chocolate milk	4.9 <sup>d</sup>	—	4.1 <sup>g</sup>	2.1	0.2	0.1	1.7	0.3
Yogurt of cow milk	25.2 <sup>d</sup>	—	4.1 <sup>g</sup>	2.1	1.0	0.5	8.8	1.4
Cow milk cream, fresh	6.0 <sup>d</sup>	—	40.0	20.0	2.4	1.2	20.3	3.2
Cow milk butter	—	9.6 <sup>d</sup>	85.0	85.0	8.2	8.2	68.5	21.6
Cow milk cheese	—	50.7 <sup>d</sup>	40.0	40.0	20.3	20.3	170.5	53.8
Cow milk cheese, melted	—	3.0 <sup>d</sup>	35.0	35.0	1.1	1.1	8.9	2.8
Subtotal					40.9	35.2	344.1	93.4
Cattle and veal meat	—	58.4 <sup>e</sup>	5.0 <sup>i</sup>	5.0	2.9	2.9	24.5	24.5
Edible cattle offal	—	6.0 <sup>f</sup>	10.0 <sup>i</sup>	10.0	0.6	0.6	5.05	5.1
Subtotal					3.5	3.5	29.5	29.6
Total					44.4	38.7	373.6	123.0

<sup>a</sup>Based on necessary liters of whole milk with 3.91% fat for fabricate those products (*Moniteur Belge*, 1996).

<sup>b</sup>Assuming whole milk and milk products.

<sup>c</sup>Assuming half-skimmed milk and products.

<sup>d</sup>Belgian consumption in 1998 (Confédération Belge de l'Industrie Laitière, 1999).

<sup>e</sup>Belgian consumption in 1998 (Van Hecke, 2000).

<sup>f</sup>Regional diets (World Health Organization, 1998b).

<sup>g</sup>Percent fat is weighted mean of whole milk in year 1999.

<sup>h</sup>Assuming 0.4% fat (Walstra & Jenness, 1984).

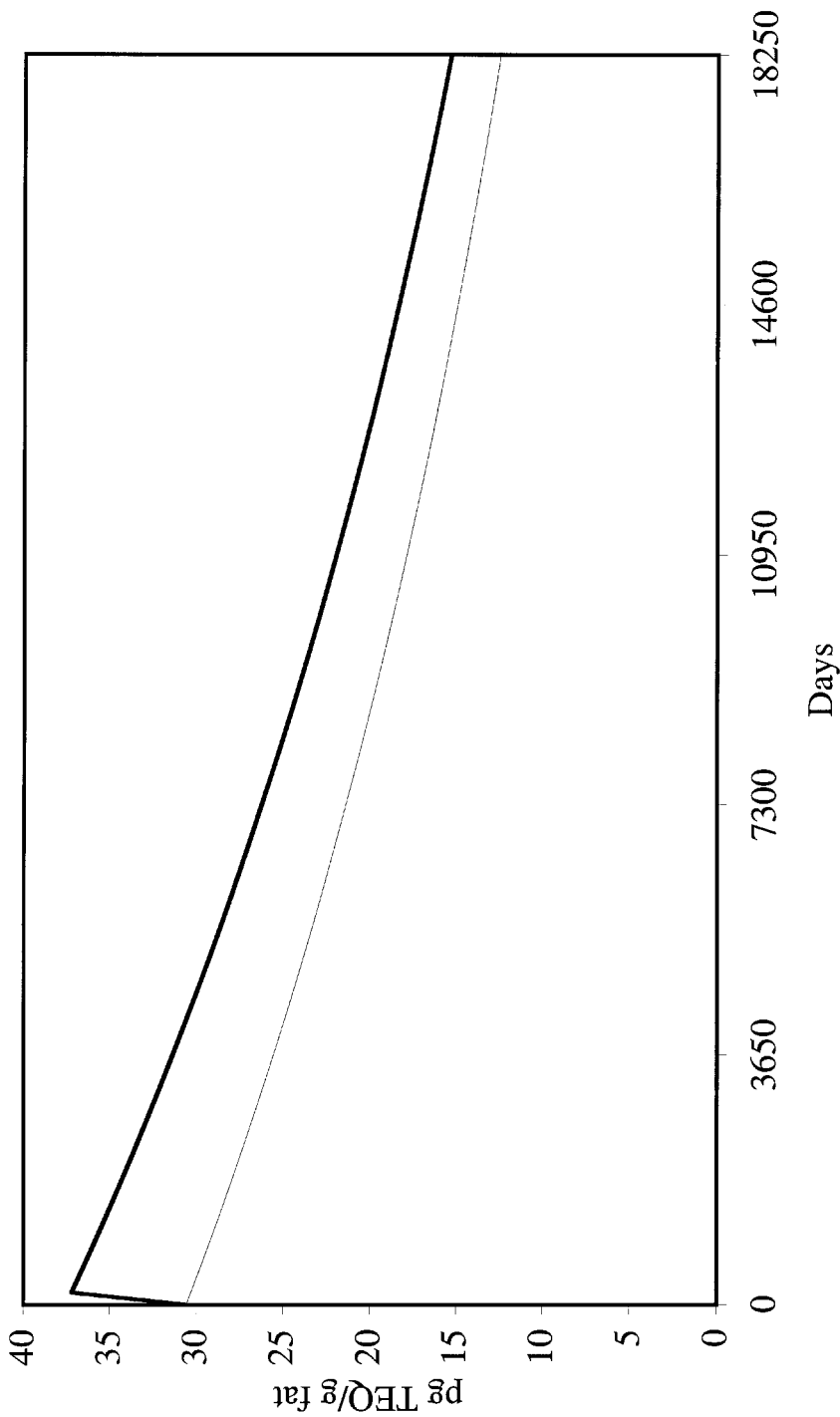
<sup>i</sup>Assuming a 50% lost during cooking (Schechter et al., 1998) and 5% fat in raw American filet (Absolonne, 1995).

<sup>j</sup>Assuming 10% fat in offal; range 3 to 17% fat (Absolonne, 1995).

<sup>k</sup>Assuming a 97.5th percentile dioxin exposure level of 8.41 and 2.19 pg TEQ/g fat for local and supermarket milk product consumption respectively and 8.41 pg for meat products and by-products.

pg TEQ/g fat after 6 mo (maximum increase), 6 pg TEQ/g fat after 10 yr, and 5 pg TEQ/g fat after 20 yr (Figure 3).

**Supermarket consumption** Assuming an exposure period of 1 wk for meat (3 beef meals per week) and half-skimmed milk products and that the highest values recorded in milk tanks is representative of the contamination, the TMDI of dioxins from single commodities of bovine origin in an adult human is 123 pg TEQ/g body fat (Table 1). After 1 wk of exposure to milk and meat products the maximum cumulative intake is 644 pg TEQ. According to the current Belgian data (Bernard & Fierens, personal communication), the increase in dioxin body burden is respectively 0.07 pg TEQ/g fat after 1 wk (maximum increase), 0.067 pg TEQ/g fat after 10 yr, and 0.056 pg TEQ/g fat after 20 yr (Figure 3).



**FIGURE 3.** Evolution in time of body burden (pg TEQ/g fat) after dioxin contamination in two worst-case scenarios (local consumption = heavy line, and supermarket purchase = fine line).

## DISCUSSION

The prevalence of PCB contamination (levels of 0.2  $\mu\text{g/g}$  body fat and higher) in Belgian slaughter cattle was estimated at 0.33%. For comparison, the level for action recommended by the Food and Drug Administration for bovine and poultry meat is 3  $\mu\text{g/g}$  fat for total PCBs (Boyer et al., 1991), which corresponds to about 1  $\mu\text{g/g}$  fat for the 7 PCB markers, a value 5 times higher than that adopted in Belgium (Bernard et al., 2002). This study demonstrated that surveying the national slaughter cattle population within a short time is technically feasible under a joint multidisciplinary collaborative effort. The results of the systematic sampling surveys confirmed that the incidence of the PCB/dioxin contamination in cattle was low. This contamination resulted from environmental sources that are common in all industrialized countries (Debondie et al., 2000). The search for the source of contamination requires a multidisciplinary collaborative effort. The relevance of the current choice of seven congeners merits new consideration for the future.

The present results are objective data and were reported to the Standing Veterinary Committee to the European Commission. With this report the bovine sector was relieved from the application of decision 99/449/CE (European Commission, 1999a). The approach developed in the directive 96/23/CE (European Commission, 1996) is based mainly on a target sampling after a risk analysis of potential source of contamination in each member state. However, random sampling of slaughter cattle is indicated to estimate the PCB prevalence in an individual country, as this approach allows a comparison of prevalence levels in different countries. The validation of new tests in live animals, such as dosage of activity of Ah-receptor by enzyme-linked immunosorbent assay (ELISA), may be considered for a detection system and may offer an opportunity for future population studies (Johnson & Van Emon, 1994); for example, in humans, plasmatic lipid PCB/dioxin levels were found to reflect body lipid levels (Ewers et al., 1996).

Two scenarios were developed to estimate maximal potential exposure of consumers to dioxins through beef and dairy products: local consumption and supermarket consumption, both using worst-case assumptions. In the case of local consumption, the TMDI of dioxins is 374 pg TEQ/d for an adult. According to current Belgian dioxin data (Bernard et al., 2001; Bernard & Fierens, personal communication), a simulation with this level of TMDI results in a maximum increase in body burden of 7 pg TEQ/g fat (total body burden: 37 pg TEQ/g fat). In a 40- to 60-yr-old person the plasmatic lipid concentration of dioxins is in steady state (Kreuzer et al., 1997). The body burden of dioxins ranged from 7 to 20 pg TEQ/g serum lipids in the general population and attained 60 pg TEQ/g serum lipids in a population with high fish intake (Ansay, 1999), and no clear evidence of an increased risk of cancer was found in the latter (Kiviranta et al., 2000). It must also be noted that fish usually does not constitute a principal food item (Guo

et al., 2001). In our estimation, a selection bias is introduced, because for the meat products the 17 dioxins congeners were analyzed only if the PCB result was positive. This in fact constitutes the major drawback for using PCB monitoring as a surrogate for dioxin monitoring. Furthermore, a half-life of 7 yr for dioxins was assumed, but this will vary from 3 to 19.6 yr as a function of the congener considered, and this information is not available for every congener (Flesch-Janys et al., 1996; Michalek & Tripathi, 1999). According to the analysis by Cox (1999) in a normal situation, the TMDI obtained in the simulation corresponds to a fivefold increase of the daily intake originating from foodstuffs of bovine origin. Assuming that other possible ingestion sources of dioxins were constant, the total cumulative daily intake during this contamination in the worst-case scenario is 476 pg TEQ (i.e., 8 pg TEQ/kg/d), which is still below the uncorrected value of 14 pg TEQ/kg/d (lower limit of TDI, World Health Organization, 1998a). Therefore a possible health effect in pregnant women, schoolchildren, and infants cannot be excluded. However, in the last subpopulation the half-life is 4 mo (Ansay, 1999; Kreuzer et al., 1997) to 6 mo (Maghin-Rogister et al., 1999). In the case of supermarket consumption, the TMDI of dioxins from single commodities of bovine origin in adult humans was 123 pg TEQ/g body fat. According to the current Belgian dioxin data (Bernard & Fierens, personal communication), this results in a maximum increase in body burden of 0.07 pg TEQ/g fat after 1 wk (total body burden: 30.3 pg TEQ/g fat). This increase is insignificant for adverse health effects. Taking into account Cox (1999) data (dioxin accumulation of 179 pg TEQ/d with 42% coming from beef and milk products), the total cumulative daily intake during this contamination was 225 pg TEQ. With this result the upper limit of WHO-TDI was reached. A cattle fat concentration of 10% (cooking meat) was assumed for the 2 scenarios with a loss of 50% during cooking (Bernard et al., 2002; Schecter et al., 1998), together with a frequent consumption of raw minced meat with a fat concentration of 5% (Absolonne, 1995). It was also assumed that foodstuffs were of Belgian origin. This assumption was justified because the rate of provisioning was higher than 100% for all milk and meat products with the exception of cheese (for which the principal countries of origin were France and Netherlands).

Several problems were observed when estimating the exposure to PCB/dioxins. Firstly, there was the limit of determination (LOD) of PCBs or dioxins. For PCB/dioxins, the interpretation of a result above the cutoff depends on the individual country. For example, Belgium, France, and the Netherlands apply the "lower bound principle" (if the dosage of a congener is lower than the LOD the result is negative); the United Kingdom applies the "high bound principle" (if the dosage of a congener is lower than the LOD, the result is equal to the LOD of this congener); and Italy applies the "half weight bound principle" (if the dosage of a congener is lower than the LOD the result is equal to half the LOD of this congener). Because of the different interpretations, the estimated mean exposure is not the same in the dif-

ferent countries. The European Commission has proposed the UK method for all its member states in accordance to the principle of maximum precaution (Ministry of Small Enterprises, Trade, and Agriculture, 1999). The choice of the 97.5th percentile of the contamination level, used in the current study, is another possibility to standardize the level of critical exposure. By analogy, the study for pesticide residues recommends the use of the recorded 97.5th percentile consumption (Ministry of Agriculture, Fisheries, and Food, 1995), but these data were not available for the 1999 incident. A second problem lies in the necessity to obtain recent data on quantities of each foodstuff consumed by adults, schoolchildren, and infants. It is therefore proposed to carry out a longitudinal study to determine the consumption and its distribution in the population. The third problem is to obtain representative data of PCB/dioxin contamination in food and food products. This problem is more acute because the primary origin of these food products is not always known. The solution to this problem lies in better tracing of food products and better import control.

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