The Belgian PCB and Dioxin Incident of January–June 1999: Exposure Data and Potential Impact on Health

Nik van Larebeke,[1] Luc Hens,[2] Paul Schepens,[3] Adrian Covaci,[3] Jan Baeyens,[4] Kim Everaert,[4] Jan L. Bernheim,[2] Robert Vlietinck,[5] and Geert De Poorter[6]

[1]Department of Radiotherapy, Nuclear Medicine and Experimental Cancerology, Ghent University, Belgium; [2]Human Ecology Department, Free University Brussels, Belgium; [3]Toxicological Center, Antwerp University, Antwerp, Belgium; [4]Department of Chemical Engineering, and [5]Department of Human Genetics, Catholic University, Leuven, Belgium; [6]Federal Ministry of Agriculture, Brussels, Belgium

In January 1999, 500 tons of feed contaminated with approximately 50 kg of polychlorinated biphenyls (PCBs) and 1 g of dioxins were distributed to animal farms in Belgium, and to a lesser extent in the Netherlands, France, and Germany. This study was based on 20,491 samples collected in the database of the Belgian federal ministries from animal feed, cattle, pork, poultry, eggs, milk, and various fat-containing food items analyzed for their PCB and/or dioxin content. Dioxin measurements showed a clear predominance of polychlorinated dibenzofuran over polychlorinated dibenzodioxin congeners, a dioxin/PCB ratio of approximately 1:50,000 and a PCB fingerprint resembling that of an Aroclor mixture, thus confirming contamination by transformer oil rather than by other environmental sources. In this case the PCBs contribute significantly more to toxic equivalents (TEQ) than dioxins. The respective means ± SDs and the maximum concentrations of dioxin (expressed in TEQ) and PCB observed per gram of fat in contaminated food were 170.3 ± 487.7 pg, 2613.4 pg, 240.7 ± 2036.9 ng, and 51059.0 ng in chicken; 1.9 ± 0.8 pg, 4.3 pg, 34.2 ± 30.5 ng, and 314.0 ng in milk; and 32.0 ± 104.4 pg, 713.3 pg, 392.7 ± 2883.5 ng, and 46000.0 ng in eggs. Assuming that as a consequence of this incident between 10 and 15 kg PCBs and from 200 to 300 mg dioxins were ingested by 10 million Belgians, the mean intake per kilogram of body weight is calculated to maximally 25,000 ng PCBs and 500 pg international TEQ dioxins. Estimates of the total number of cancers resulting from this incident range between 40 and 8,000. Neurotoxic and behavioral effects in neonates are also to be expected but cannot be quantified. Because food items differed widely (more than 50-fold) in the ratio of PCBs to dioxins, other significant sources of contamination and a high background contamination are likely to contribute substantially to the exposure of the Belgian population. *Key words*: Belgium, cancer, dioxin, food chain, polychlorinated biphenyls, risk assessment. *Environ Health Perspect* 109:265–273 (2001). [Online 1 March 2001]
http://ehpnet1.niehs.nih.gov/docs/2001/109p265-273vanlarebeke/abstract.html

In Belgium, approximately 20 companies collect animal fat from slaughterhouses and melt it into a homogenous substance, which is sold to animal-food producers. It is a common practice to include household waste fat collected at community waste recycling centers in this product. In January 1999, at the Flemish fat-melting company Verkest, 40–50 kg of mineral oil containing polychlorinated biphenyls (PCBs; most likely oil from discarded transformers originating from a waste recycling center) was admixed to the fat delivered to 10 animal-feed producers. Between 15 and 31 January, the resulting 500 tons of contaminated animal feed, containing approximately 60–80 tons of fat contaminated with 40–50 kg of PCBs and almost 1 g of dioxins, were distributed to poultry farms and to a lesser extent also to rabbit, calf, cow, and pig breeding and raising farms, mostly in Belgium. Small quantities were exported to the Netherlands, France, and Germany. In Belgium, 445 poultry farms, 393 bovine farms, 746 pig farms (or a quarter of this type of farm in Belgium) and 237 dairy farms (representing 1.5% of the total number of dairy farms in Belgium) used animal feed from the 10 contaminated animal-feed producers. The 500 tons of contaminated feed represent a limited percentage of the total amount of feed produced and used in Belgium, which is estimated to exceed 28,000 tons/week.

Pathologic conditions were first recorded on 4 February on Belgian chicken farms. They included a decrease in egg production and hatching and an epidemic of chicken edema disease. After excluding other causes of the epidemic, samples of animal feed, chicken carcasses and eggs were sent for toxicologic analysis on 18 March. The authorities were informed of the presence of increased concentrations of dioxins in animal feed, chicken, and eggs on 26 April. The first measures to protect public health were taken, but the public was not informed. On 27 May the Flemish television broke the news; the incident became public and resulted in a political, communication, and economic crisis. The authorities tried to identify the extent of the crisis by identifying the companies involved in the contamination. All available laboratories in Belgium and neighboring countries were mobilized for analytical work. The analyses revealed that the dioxins were part of a PCB contamination, and average PCB/dioxin ratios were determined. During the first days of June, the Ministry of Public Health ordered removal of poultry, derived products (meat, eggs, mayonnaise, custards, cakes, etc.), and all meat products with a fat content > 25% from the market. A widespread product sampling and analysis was organized, resulting in the data used in the present study. Only products with a concentration of < 200 ng PCBs/g fat were released for human consumption. Products with excessive levels were destroyed, including some 2 million chickens. The duration of exposure of the population can hence be estimated as 4 months (February–May).

Dioxin Exposure in Belgium before the Incident

Atmospheric pollution by identified and disperse emission sources of dioxins in Flanders (northern Belgium) has been covered in several studies (*1*,*2*). These studies show a gradual reduction in emissions from > 600 g international toxic equivalents (I-TEQ)/year in 1994 to approximately 100 g I-TEQ/year in 1999. Whereas municipal solid waste incineration accounted for nearly 60% of dioxin emissions in 1993, its contribution is now limited to a low percentage. The nonferrous, ferrous, and steel industries remain major sources, together with household heating and traffic. More stringent regulations and continuous monitoring will further reduce the emissions, although the past levels will contribute to background soil pollution and body burdens for a long time.

In Belgium in 1995, 1,151 g I-TEQ were emitted (*3*). As shown in Table 1, 60% of this value is due to emissions to air. In this figure emissions to water are likely to be underestimated, as the emission inventory identifies only a limited number of sources. The polychlorinated dibenzodioxin/polychlorinated dibenzofuran (PCDD/PCDF) flux in Belgium is high. A comparison of inventories for 15 countries (*4*) shows that

Address correspondence to L. Hens, Human Ecology Department, Free University Brussels - VUB, Laarbeeklaan 103, B-1090 Brussels, Belgium. Telephone: +32-2-477.42.81. Fax: +32-2-477.49.64. E-mail: gronsse@meko.vub.ac.be

Received 18 August 2000; accepted 19 October 2000.

the per capita emission in Belgium is the second highest after Japan.

Background air immission measurements are scarce, but existing information shows values ranging between 100 and 255 fg TEQ/m^3 (*5*).

More information is available on deposition, which is measured systematically at 20 sites. These include a few rural and urban reference areas but are concentrated around specific emission sources such as municipal waste incinerators and steel mills. During 1993–1997, the values ranged between 0.26 to 374 ng I-TEQ/m^2 per year (*5*).

Limited data on the food load of dioxins and PCBs are available. Cox (*6*) reviewed the existing data. Incomplete data were completed by information on Dutch measurements of dioxins in the food basket. Cox concluded that the average total daily intake was 179 pg I-TEQ/day. These data are a rough estimate because the dioxin concentrations in food are only partially known, and because the official food basket composition as used by the government (*7*) is outdated.

Of particular interest are the data on dioxin concentrations in milk. The prevailing mean concentrations are 2.1 pg I-TEQ/g milkfat (range: 1.3–2.5 pg I-TEQ/g milkfat). However, in cows grazing close to municipal waste incinerators, values up to 21 pg I-TEQ/g milkfat have been recorded.

Dioxins and furans in milk of lactating women were studied in 1988 and 1993 by the World Health Organization (WHO) (*8*). Belgian samples in this international study originated from women living in Brussels, the rural area south of Brussels, and Liège, capital of the eastern Belgian province. In these samples an average value of 34.4 pg I-TEQ/g milkfat (range 27.3–43.2 pg I-TEQ/g milkfat) was found. The dioxin concentrations in the Belgian samples are approximately twice as high as those measured in Austria, Denmark, Croatia, or Canada. Levels were stationary between 1988 and 1993.

Table 1 summarizes the data mentioned above and compares them with the available reference values. This comparison illustrates that dioxin background values in Belgium often exceed the reference values. The situation is most pronounced for babies. Assuming the 34.4 pg I-TEQ/g fat in mother's milk is representative of the country as a whole, babies are fed an average of 43 pg I-TEQ/kg per day. This is substantially more than the 1–4 pg/kg per day that has been proposed by the WHO. It is also 20 times higher than the average intake by an adult and indicates that during 3 months of breast-feeding, Belgian babies take in 6% of their lifetime dioxin dose.

The mean preincident concentrations in breast milk samples of mothers nursing their firstborn baby (*9*) also allow one to estimate the body burden. Assuming that fat amounts to 20% of body weight, 34.4 pg I-TEQ/g fat corresponds to a body burden of 6.88 ng I-TEQ/kg body weight in these young women. The global picture which emerges is that Belgians are exposed to high background concentrations of dioxins and furans.

Objectives of This Study

During the initial period (January to end of May) of the PCB–dioxin contamination incident in Belgium, only a few dioxin measurements were performed as the incident was treated in a confidential manner. Large-scale sampling and laboratory testing happened after the crisis went public on 27 May. We assessed the results concerning the samples taken from the end of May until August 1999. The data were used to estimate the overall population exposure and the exposure that might have resulted from selected diets. On this basis the potential impact on public health was estimated. More than 40,000 additional samples were analyzed in the period after August 1999, and monitoring is still ongoing. These results will be dealt with in a separate follow-up paper.

Materials and Methods

Dioxin and PCB Measurements in Animals and Food Products

Samples and measurements. The data assessed here are based on the analyses ordered by the Belgian Ministries of Health and Agriculture. They were presented to the Belgian Parliament in September 1999. As shown in Table 2, 20,491 samples from cattle, pigs, poultry, eggs, milk, and various fat-containing food items (ranging from mayonnaise to Belgian chocolates) were collected by different control departments of the Ministries of Public Health and Agriculture. The sample record form included information on the commercial origin of animal feed for meat or the dairy products, or of dairy ingredients in food.

In this study, samples were divided into three classes:

- "Incident-related samples" for which the origin could be traced back to farms that used feed from one of the 10 animal-feed producers who bought contaminated fat from the Flemish fat-melting company Verkest; such samples are not necessarily contaminated by the incident, but might be so

Table 1. Summary of background exposure data to dioxins in Belgium before the PCB/dioxin incident.

Type of measurement	Area	Mean	Range	Threshold value	Reference
Emission	Belgium				(*4*)
Air		662 g I-TEQ[a]			
Water		4 g I-TEQ[a]			
Soil		485 g I-TEQ[a]			
Immission air	Flanders		100–255 fg I-TEQ/m^3		(*5*)
Deposition	Flanders		0.26–374 ng I-TEQ m^2/year	5.5 ng I-TEQ/m^2/year (German value)	(*5*)
Cow milk	Flanders	2.1 pg I-TEQ/g fat	1.3–2.5 pg I-TEQ/g fat to 21.8 pg in polluted areas	5 pg I-TEQ/g fat (Belgian value)	(*5*)
Human milk	Wallonia and Brussels	34 pg I-TEQ/g fat	27.3–43.2 pg I-TEQ/g fat		(*8*)

[a]Total quantities.

Table 2. Number of samples analyzed by the Belgian authorities during May–August 1999.

	Bovine meat or fat	Milk	Butter	Poultry meat or fat	Eggs	Pig fat or pork meat[a]	Other food products[b]	Unspecified animal fat[c]	Unspecified fat[d]	Waste[e]	Animal feed	Other	Total	Above thresh-hold	Under thresh-hold
PCBs	683	818	46	1,890	718	7,759	3,700	2,288	764	381	1,040	203	20,290	5,687	14,603
Dioxins	19	63	23	62	55	137	43	11	20	2	10	1	446	408	38
PCBs + dioxins	6	58	23	20	30	76	2	5	16	2	6	1	245		
Total													20,491		

[a]Includes PCB measurements on 13 pig milk samples and dioxin measurements on 7 pig milk samples. [b]Processed food not specified as being bovine meat, pork, or chicken. [c]Animal fat not specified as being taken from cattle, pigs, or poultry. [d]Diverse fatty materials that are sometimes incorporated in animal feed, including samples of animal fat that were, however, not labeled as being "animal fat." [e]Waste oils that are sometimes incorporated in animal feed.

- "Samples unrelated to the incident," for which the origin could be traced back to farms that did not use feed from one of these 10 feed producers
- "Samples without a track record leading to the incident"; these samples could not be traced back to farms that did use feed from one of these 10 feed producers, or to farms that did not use feed from one of these 10 feed producers.

Results of 20,290 PCB measurements and 446 dioxin measurements are available; for 245 of the samples, both PCB and dioxin measurements are available. PCB measurements included seven marker congeners—namely, PCBs 28, 52, 101, 118, 138, 153, and 180—and are expressed as the sum of these congeners in nanograms per gram fat. For PCDD/PCDF the "dirty 17" congeners with chlorine substitution of at least the 2, 3, 7, and 8 positions were measured, and the total dioxin content of the sample was expressed in picogram TEQ per gram fat, using the WHO-toxic equivalence factor (TEF) values (*10*).

Quality control. Dioxins were measured using mass spectrometry. PCBs were quantified using gas chromatographic techniques followed by electron capture or mass spectrometry.

All but 18 of the PCB measurements were performed by a pool of 23 laboratories accepted by the Belgian authorities after a technical assessment; dioxin measurements were performed by 18 accredited laboratories, 5 of which accounted for 398 of all 446 measurements. Twenty-two of the laboratories that participated in PCB measurements were also included in a quality-ring test of PCB measurements, organized by the dioxin–PCB unit of the Belgian Ministries of Public Health and Agriculture (*11*).

The results showed that for contaminated feedstuff, egg yolk, and pork fat, coefficients of variation for reproducibility ranging between 16 and 35% were found. However, for slightly contaminated lyophilized milk, unacceptably high coefficients of variation were recorded.

Table 2 reviews the number of PCB and PCDD/PCDF samples analyzed: 71% of the samples tested for PCBs showed values below the detection threshold, which was specific for each laboratory. For dioxins only 10% of the samples had concentrations lower than the detection limits of the individual congeners.

Estimation of Cancer Risk

We estimated the incremental risk of cancer associated with the calculated incremental levels of exposure of the Belgian population to PCBs and/or to dioxins during the 1999 incident. First, we calculated the body burden incurred by a given population, resulting from a defined exposure, whether through a certain diet or by assuming that a certain amount of PCBs and/or dioxins was shared between a defined number of persons. Because the available quantitative risk estimates are based on daily intake values of these persistent chemicals, and since their half-life time in humans is on the order of several years, probably about 7 years for dioxins (*12,13*) and between 4.1 and 34.2 years for PCBs (*14*), we divided the incident-associated body burden by 25,550 (the number of days in 70 years), yielding a corresponding averaged daily intake. This approach assumes that no dioxin is eliminated during the exposure period of 4 months. This daily intake value was then compared to the risk estimates for dioxins as provided by Becher et al. (*15*), and for PCBs as provided by Cogliano (*16*). For academic reasons, an assessment based on a cancer risk estimate of 1 in 1 million for a life-time exposure of 0.006 pg I-TEQ/kg body weight per day, is also added for dioxin-related effects.

Results

Nature of the Contamination and Impact on the Food Chain

The contamination chain is illustrated in Figure 1. It shows how 40–50 kg of PCBs and almost 1 g of dioxins were distributed over an estimated 500 tons of animal feed and a still undefined amount of animals and derived animal food products. During the initial response to the incident, a limited number of mostly heavily contaminated samples were analyzed for both dioxins and PCBs.

The PCDD/PCDF congener distributions in four samples of animal feed heavily contaminated by the incident are shown in Figure 2. The congener profile shows a clear predominance of PCDF over PCDD congeners. This is compatible with a PCB contamination by a substance (such as transformer oil) containing PCBs rather than by dioxins originating from thermal processes such as waste incineration. The fingerprint of PCDD/PCDF-emissions by municipal waste incinerators is substantially different from the one found in animal feed (Figure 2). The furans-to-dioxins ratio is 1.7 for the incinerators and 16 for the animal food.

To further identify the nature of the PCB oil, different commercial PCB formulations were analyzed under the same conditions as the contaminated feed samples. The results for the 7 marker PCBs in 47 contaminated animal feed samples (containing > 1,000 ng PCB/g fat) are given in Figure 3, which also includes the profiles for a 50/50 mixture of Aroclors 1254 and 1260. There is a good agreement between the contamination pattern found in animal feed and the profile of the mixture of both Aroclors. The contamination pattern shown by these 47

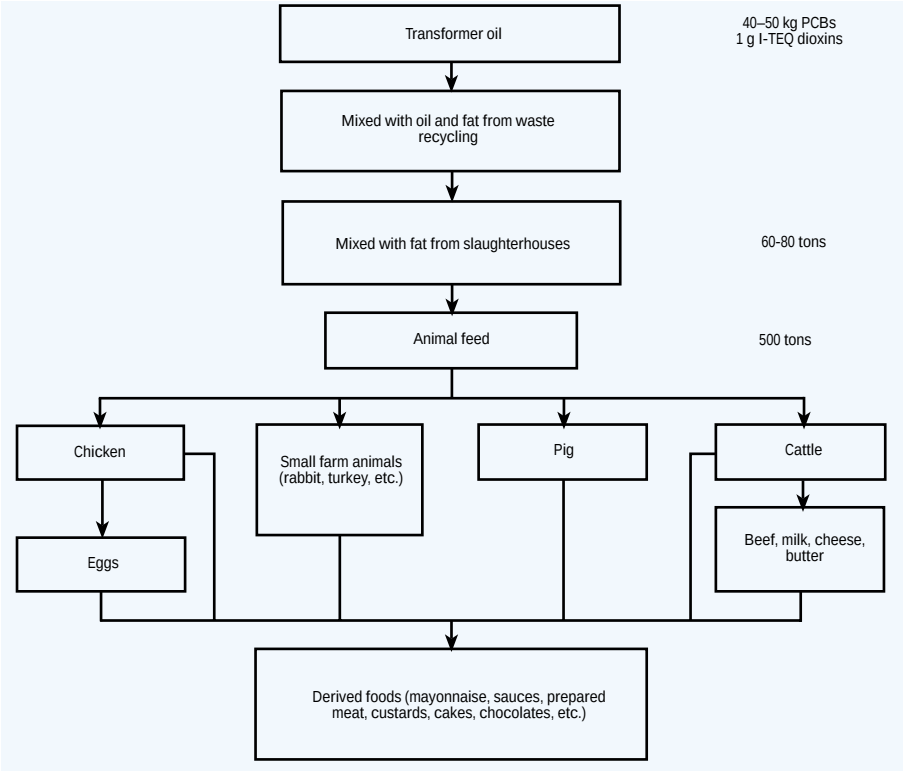

Figure 1. Fate of dioxin and PCBs in the food chain during the contamination incident (*34*).

samples is called the "incident-related PCB profile of animal feed."

The congener distribution in 11 egg samples contaminated with > 1,000 ng PCB/g fat is shown in Figure 4. The data for eggs, reflecting the result of metabolic conversion, are compared with the profiles found in the 47 samples of animal feed. The comparison shows that the higher PCBs (PCB 118, 138, 153, and 180) are the most persistent ones. The lower chlorinated PCBs (PCB 52 and 101) are more easily metabolized or excreted. The contamination pattern shown by these 11 samples is called the "incident-related PCB profile of eggs."

Figure 5 shows the concentration of PCBs plotted against the ratio between the sum of the seven marker PCBs over the dioxins (in I-TEQ) found in the respective samples, for all samples for which both PCB and dioxin data are available. The figure reveals that for most (12 out of 16) samples with concentrations of PCBs > 5,000 ng/g fat, about 50,000 times more PCBs than dioxins are found. This coincides with the ratio found in transformer oil. Numerous less-contaminated samples show substantially different ratios.

The PCB congener distribution in many samples differs from the typical incident-related PCB profile. Figure 6 compares the incident-related PCB profile for animal feed with the PCB profile in four animal-feed samples unrelated to the incident. The figure shows that these five profiles differ. Five egg samples unrelated to the incident but showing PCB concentrations ranging between 1,111 and 1,405 ng/g fat likewise showed a PCB profile that differed substantially from the incident-related PCB profile for eggs.

Table 3 provides concentrations of PCBs and dioxins in a series of food items sampled between the end of May and August 1999. The data show that the highest dioxin concentrations were found in poultry fat and eggs. Pork, beef, and milk were also contaminated, but to a much lower extent. It should be realized that contamination levels in the period February–May might have been higher. Also 4.1% of poultry, 3.94% of egg, and 1.03% of pork samples without a track record leading to the incident contained > 200 ng PCBs/g fat. Samples unrelated to the incident also showed dioxin levels > 2 pg TEQ/g fat in 60% of the beef samples, 31.9% of the milk samples, 16.7% of the poultry meat samples, 58.3% of the eggs, 9.38% of the pork samples, and 27.9% of the processed food samples.

To obtain an indication of the contribution of non-*ortho* and mono-*ortho* PCBs to the toxic potency of contaminated samples, we analyzed two animal feed samples, two egg samples, three chicken samples, and two pork samples for both marker PCBs and dioxin-like PCBs. Table 4 lists the details from seven of these analyses. Taking the WHO-TEF value into account, the TEQ values for the different PCBs were calculated. The ratio between PCB TEQ and the sum of marker PCBs amounted to 1:15,622 and 1:11,373 for the two feed samples; 1:11,702 and 1:11,750 for the two egg samples; 1:17,618, 1:16,722 and 1:17,735 for the chicken samples; and 1:42,892 and 1:44,651 for the pork samples. Taking into account a median value of 16,722, this would imply that the incident brought, with an exposure to 15 kg of marker PCBs, also an exposure to 0.9 g TEQ from PCBs.

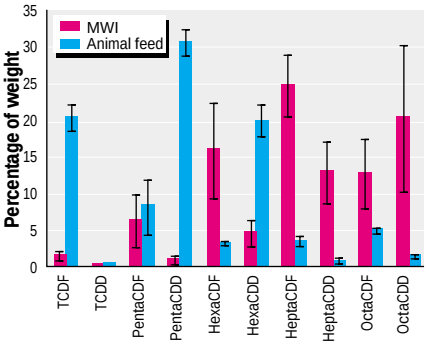

Figure 2. PCDD/PCDF congener distributions (in percentage of weight) measured in four samples of contaminated feed (mean ± SD). The results are compared with the congener distribution as found in emissions of municipal waste incinerators (MWI).

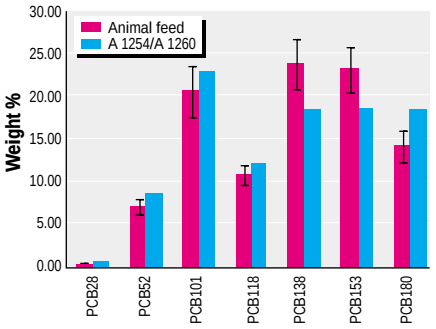

Figure 3. Weight percent distribution of seven PCB marker congeners in 47 contaminated animal feed samples (mean ± SD) and a 50/50 mixture of Aroclors (A) 1254 and 1260.

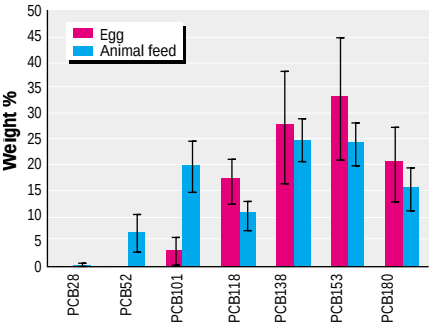

Figure 4. PCB congener distribution (weight %) in contaminated eggs, compared to distribution in contaminated animal feed (mean ± SD).

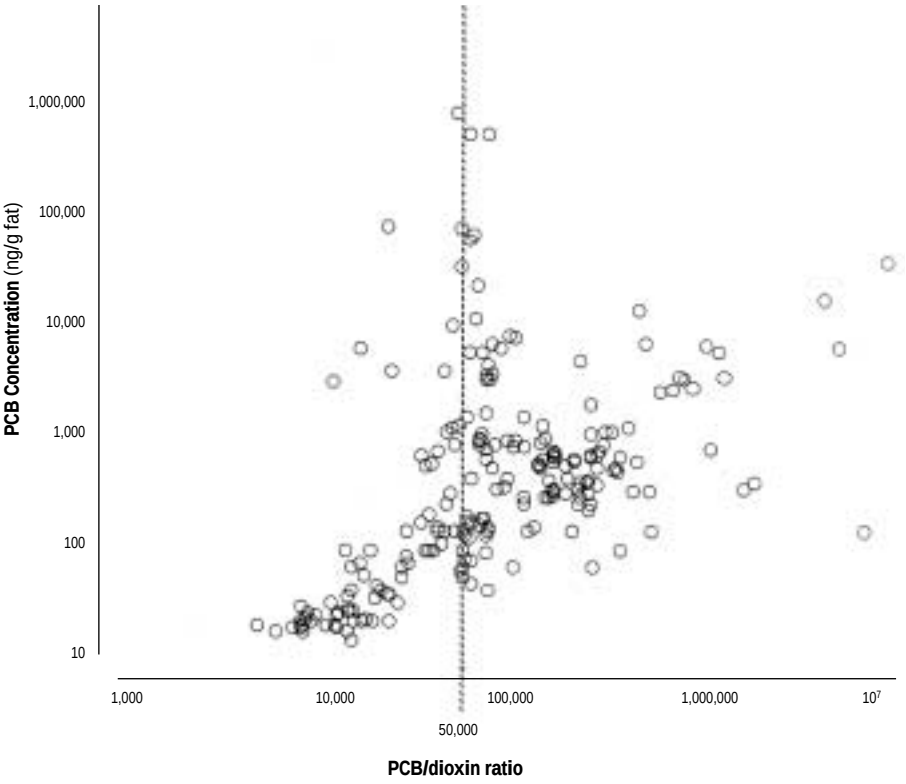

Figure 5. PCB/dioxin ratio in 246 samples for which both dioxin and PCB concentrations were measured.

Individual Exposure

The individual exposure that might have resulted from the incident was assessed using two exposure models. The first model assumes an even distribution of the pollution over the Belgian population. The second model is based on selected diets.

Model based on the total amount of PCBs and dioxins released during the incident. Fifty kilograms of PCBs were introduced in the food chain through this incident. It is assumed that 30% of this amount (i.e., 15 kg) was ingested by 10 million Belgians. With a mean body weight of 60 kg for the modal citizen (children included), this represents an average intake of 25,000 ng/kg body weight.

As the body burden for PCBs of the Belgian population before the incident was about 300 ng/g fat (*8*), corresponding to 60 µg/kg body weight, the incident, responsible for an additional estimated mean of 25 µg/kg body weight, would have increased the average PCB body burden of the Belgian citizen by about 42%. It is assumed that this contribution to the body burden would correspond to a daily intake of 1 ng/kg per day for 70 years (= 25,550 days).

Using similar assumptions, the 1 g I-TEQ of dioxins would have resulted in an increase in body burden with 500 pg I-TEQ/kg body weight. This represents an increase of 7%, assuming a mean baseline dioxin body burden of 6.88 ng I-TEQ/kg. This value also corresponds to an intake corresponding to 0.02 pg/kg per day over 70 years.

Model based on selected dietary patterns. Belgian adults consume an average of 75 g animal fat per day (*6*). As shown in Table 3, 6.5% of the poultry meat, 8.1% of the eggs, and 16.5% of the pork samples contained > 200 ng PCBs/g fat. Two percent of the chicken and 7% of the pork samples analyzed were shown to contain > 1,000 ng PCBs/g fat. The maximum dioxin values of 2,613 and 713 pg I-TEQ/g, respectively, were measured in poultry and eggs.

Levels of contamination up to hundreds of picograms TEQ per gram of fat probably prevailed in a much higher percentage of food items from February until the end of May than from the end of May until August, when measurements took place. Therefore, a certain percentage of the Belgian population could have incurred exposures as those calculated below.

An adult (70 kg body weight) who consumed 15 g of animal fat per day, contaminated with 200 pg I-TEQ dioxins/g, during the 4-month period (120 days) in which the contaminated food was distributed would increase his or her body burden by 360 ng I-TEQ, or 5.14 ng I-TEQ/kg body weight. This corresponds to an increase of 75%, assuming a mean baseline dioxin body burden of 6.88 ng I-TEQ/kg. This intake of 15 g contaminated animal fat per day corresponds to a daily intake of 42.8 pg/kg body weight, 10–40 times the total daily intake (1–4 pg/kg body weight per day) accepted by the WHO.

An adult who consumed 150 g chicken meat contaminated with 700 pg TEQ/g fat

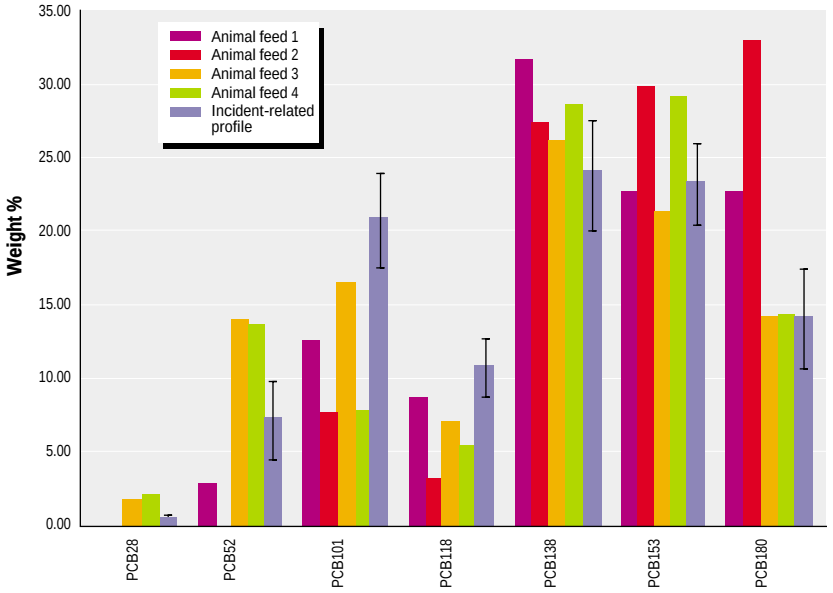

Figure 6. PCB congener distribution of animal feed samples. The results of four contaminated animal feed samples, unrelated to the incident, are compared to the incident-related PCB profile (mean ± SD). Contamination levels are 1,869 ng PCBs/g fat for sample 1; 11,251 ng PCBs/g fat for sample 2; 809 ng PCBs/g fat for sample 3; and 407 ng PCBs/g fat for sample 4. These samples are considered unrelated to the incident.

Table 3. Dioxins and PCBs in Belgian food and in animal feed constituents.

	PCBs					Dioxins				
	Concentration (ng/g fat)		Maximum level	Percent of samples (ng/g fat)		Concentration (pg I-TEQ/g fat)		Maximum level	Percent of samples (pg/g fat)	
	AM ± SD	GM	(ng/g fat)	≥ 200	≥ 1,000	AM ± SD	GM	(pg/g fat)	≥ 2	≥ 5
Beef	64.7 ± 64.8	48.5	1023.9	1.2	0.1	3.9 ± 5.2	2.4	23.0	68.4	21.1
Cattle milk	34.2 ± 30.5	25.5	314.0	0.1	None	1.9 ± 0.8	1.8	4.3	42.9	None
Butter	38.3 ± 14.1	35.2	50.0	None	None	1.7 ± 1.3	1.4	4.0	21.7	None
Poultry	240.7 ± 2036.9	73.2	51059.0	6.5	1.9	170.3 ± 487.7	4.2	2613.4	41.9	30.6
Eggs	392.7 ± 2883.5	71.2	46000.0	8.1	2.7	32.0 ± 104.4	5.1	713.1	63.6	40
Pork[a]	292.9 ± 955.6	79.8	25472.0	16.5	7.1	2.6 ± 6.0	1.0	64.0	39.4	8.8
Other food products[b]	71.6 ± 46.9	59.6	1378.0	0.3	0.1	1.6 ± 1.9	0.8	7.9	27.9	2.3
Animal fat of unspecified origin[c]	67.2 ± 124.0	50.2	4091.5	0.8	0.2	2.8 ± 2.9	1.3	7.6	36.4	27.3
Fat of unspecified[d] origin	101.2 ± 271.5	53.2	3900.0	5.0	1.1	15.9 ± 18.6	4.1	61.7	60	55
Waste[e]	264.9 ± 2814.7	66.7	54909.0	2.9	1.8	0.007 ± 0.01	0.0	0.0	None	None
Animal feed	1658.4 ± 23584.4	60.7	519000.0	6.7	1.4	2319.8 ± 3851.9	97.7	11163.0	70.0	70.0

Abbreviations: AM, arithmetic mean; GM, geometric mean. This table presents an overview of all measurements done in the period May–August 1999. The number of samples for each category is indicated in Table 2.

[a]Includes PCB measurements on 13 pig milk samples and dioxin measurements on 7 pig milk samples.[b]Processed food not specified as being bovine meat, pork, or chicken. [c]Animal fat not specified as being taken from cattle, pigs, or poultry. [d]Diverse fatty materials that are sometimes incorporated in animal feed, including samples of animal fat that were, however, not labeled as being "animal fat." [e]Waste oils that are sometimes incorporated in animal feed.

three times a week during the same 4-month period would increase his or her body burden by 201.6 ng I-TEQ, or 2.88 ng I-TEQ/kg body weight, which corresponds to an increase of 42%.

An average adult with a body weight of 70 kg would consume the following quantities weekly during a 4-month period: seven servings of meat (150 g/serving with 10% fat), contaminated with 70 pg I-TEQ/g; two eggs, contaminated with 200 pg I-TEQ/g; and one serving of chicken (150 g/serving), contaminated with 700 pg I-TEQ/g.

This diet would increase the total dioxin load by 232 ng I-TEQ (i.e., 3.31 ng/kg body weight). The estimated body burden before the incident of 6.88 ng I-TEQ/kg is increased by 48% in this scenario (*9*). Some persons might have incurred even higher exposures because consumption of milk and derived food products such as sauces and pastry were not included in the above estimations.

Cancer Risk Assessment

Cancer risk assessment for dioxins. Based on the cancer risk estimate of 1 in 1 million for a lifetime exposure of 0.006 pg I-TEQ/kg body weight per day, the incident-specific incremental exposure, estimated above to be equivalent to an intake of 0.02 pg I-TEQ/kg per day during 70 years, would entail a risk of 32 additional cancer deaths in the total Belgian population of 10 million.

Becher et al. (*15*) published a considerably higher risk estimate based on a cohort of workers who were primarily exposed to TCDD. They found that a lifetime exposure of 1 pg TCDD/kg body weight per day entails an incremental lifetime cancer mortality risk of between 1.3 and 7.7 per 1,000. This risk estimate can be applied to an exposure quantified in terms of TCDD-like I-TEQ, as is implicit in the TEF and TEQ concepts. Applying the risk estimates of Becher et al. (*15*) to the dioxin I-TEQ load due to the incident and assuming a linear dose–risk model, between 260 and 1,540 additional cancer deaths would be expected in the total Belgian population of 10 million.

Cancer risk assessment for PCBs. PCB mixtures have tumorigenic effects that do not necessarily depend on their dioxin-like TEQ value. Cogliano (*16*) developed equations based on animal experiments allowing extrapolation to calculate the risk for humans. Again, assuming a linear dose–risk relationship based on the central slope of the dose-effect curve for Aroclor 1254 (indicating a relative risk of 1.2 for 1 mg/kg body weight per day), the intake of PCBs of 1 ng/kg body weight per day due to the incident would cause an estimated 12 additional cancer deaths in the Belgian population of 10 million individuals. Taking the upper bound slope for Aroclor 1260 (indicating a relative risk of 2.2 for 1 mg/kg body weight per day), this risk amounts to 22 cancer deaths in a population of 10 million.

Weathered PCB mixtures, after chemical modifications caused by the use of the oil, have an increased toxic activity, usually by a factor of 3 (*16*). In the Belgian incident, the PCB mixtures, before being admixed to animal feed, were repeatedly exposed to heat during their industrial use and storage, and afterward during the fat melting process. As a result, I-TEQ values of 1:11,373 and 1:15,622 of its content in marker PCBs were found for animal feed (Table 4); the egg samples, in which the PCB mixtures underwent metabolic reactions in the chicken, contained PCB-derived I-TEQs corresponding to respectively 1:11,702 and 1:11,750 of their content in marker PCBs. For chicken, ratios were 1:17,618, 1:16,722, and 1:17,735, and for pork ratios were 1:42,892 and 1:44,651. Taking the I-TEQ content of the contaminating PCBs into account leads to a considerably higher estimate of cancer risk. Using the cancer risk estimate of 1 death in 1 million for a lifetime exposure of 0.006 pg/kg body weight, the PCB exposure of 1 ng/kg per day, equivalent to between 0.088 and 0.022 pg I-TEQ/kg per day leads to a risk of 37–147 cancer deaths in a population of 10 million. However, if the risk estimation of Becher et al. (*15*) for TCDD is applied to the PCB-dependent I-TEQ load, between 286 and 6,776 additional cancer deaths can be expected for the total Belgian population of 10 million.

A combined risk resulting from the combination of both dioxins and PCBs would range between 44 and 8,316 cancer deaths. As the risk figures are based on cancer deaths, they do not include the increasing number of curable cases.

Discussion

Population Exposure

Uncertainties. Although more than 20,000 PCBs and hundreds of dioxin measurements

Table 4. Determination of PCBs and TEQ from PCBs in samples of contaminated feed, chicken, pork, and egg.

		Feed sample A[a]		Feed sample B		Egg sample C[a]		Egg sample D[a]		Chicken sample E		Chicken sample F		Pork sample G	
Congener	WHO-TEF mammals (1998)	ng/g	I-TEQ pg/g	ng/g	I-TEQ pg/g	ng/g	I-TEQ pg/g	ng/g	I-TEQ pg/g	ng/g	I-TEQ pg/g	ng/g	I-TEQ pg/g	ng/g	I-TEQ pg/g
28	—	45	—	441.4	—	12	—	< 5	—	19.0	—	7.6	—	0.2	—
52	—	890	—	7588.1	—	< 5	—	< 5	—	4.0	—	1.6	—	2.5	—
101	—	2,900	—	19973.1	—	8	—	6	—	23.0	—	9.2	—	5.0	—
118	0.0001	1,200	120	12243.6	1224.4	710	71	150	15	634.0	63.4	252.8	25.3	9.2	0.9
138	—	3,300	—	33933.2	—	1,500	—	270	—	1691.0	—	674.2	—	254.3	—
153	—	2,500	—	24750.4	—	1,400	—	280	—	1273.0	—	507.6	—	197.8	—
180	—	2,200	—	15533.4	—	1,000	—	150	—	743.0	—	296.2	—	127.2	—
77	0.0001	[a]	—	317	31.7	[a]	—	[a]	—	—	0.0	—	0.0	—	0.0
105	0.0001	570	57	6,521	652.1	280	28	61	6.1	—	24.1	96.1	9.6	0.8	0.1
114	0.0005	220	110	302	151.0	32	16	5	2.5	—	3.0	2.4	1.2	0.2	0.1
123	0.0001	74	7.4	1,485	148.5	29	2.9	6	0.6	—	1.5	6	0.6	0.1	0.0
126	0.1	3.4[b]	340	56.8	5683.2	1.7[b]	171	0.3[b]	31	—	61.8	0.3	30.4	0.0	0.8
156	0.0005	350	175	3,676	1838.0	190	95	33	16.5	—	78.5	62.6	31.3	19.7	9.8
157	0.0005	37	18.5	554	277.0	18	9	3	1.5	—	13.0	10.4	5.2	3.3	1.7
167	0.00001	150	1.5	1,620	16.2	80	0.8	14	0.1	—	1.6	12.3	0.1	4.6	0.1
169	0.01	[a]	—	3,170	31.7	[a]	—	[a]	—	—	0.3	—	0.2	—	0.1
189	0.0001	50	5	106	10.6	24	2.4	4	0.4	—	1.8	7.2	0.7	—	0.3
Sum 7 marker PCBs (ng/g)		13,035	—	114463.2	—	4,635	—	866	—	4387.0	—	1,749.2	—	596.2	—
PCB–TEQ I-TEQ (pg/g)		—	834.4	—	10064.4	—	396.1	—	73.7	—	249.0	—	104.6	—	13.9
Ratio PCB-TEQ: sum marker PCBs		1:15,622		1:11,373		1:11,702		1:11,750		1:17,618		1:16,722		1:42,892	

PCBs 28, 52, 101, 118, 138, 153, 180 are the marker PCBs.
[a]For these samples, congeners 77 and 169 were not measured; inclusion of these congeners would, however, increase TEQ values for these samples by only a few percent. [b]PCB 126 was estimated through extrapolation.

on animal feed, animal fat, and different food items have been performed, there is still an important uncertainty about the extent to which the Belgian population has been exposed to these toxicants. These uncertainties derive essentially from the fact that almost no measurements of body burdens before the crisis are available and that until now no measurements of body burdens during or after the crisis were performed.

There is also uncertainty about the extent to which consumed food was contaminated, as sampling of animal fat and food items was not performed in a systematic way, but evolved rather haphazardly during the crisis in response to many different needs and pressures, some from national or European regulatory authorities, and others were commercial in nature. Some of these demands have biased sampling to the more suspect items, others to less suspect products.

Further uncertainty about the extent to which the consumed food was contaminated originates from the period of sampling. The results presented in this paper relate to the food available from end of May until August 1999. The contamination episode started, however, in January 1999. During January until the end of May, no systematic sampling of the food chain was performed. Food items may have been contaminated more often and at higher levels than is evident from the data presented in this paper.

Comparison with other contamination episodes. In the Yusho (Japan 1968) and Yucheng (Taiwan 1979) incidents, 1,700 and 2,000 victims, respectively, ate contaminated rice oil and ingested respectively, 600 mg PCBs (equivalent to 10 mg/kg body weight) plus 3.5 mg PCDFs, and 1,000 mg PCBs (equivalent to 16.6 mg/kg body weight) plus 3.8 mg PCDFs. In the Belgian crisis we estimated that a modal Belgian ingested 1.5 mg PCBs (equivalent to 0.025 mg/kg body weight). In the Seveso accident (Italy), in which the main toxicant involved was TCDD, individuals studied in the different areas were exposed to between 16 and 78 ng TCDD/kg body weight. The Ranch Hand study on U.S. Air Force veterans concerned individuals with an average exposure of 10 ng TCDD/kg body weight. The mean exposure due to the Belgian incident is estimated at approximately 0.5 ng TCDD I-TEQ/kg body weight. In conclusion, exposure during the Belgian incident amounts to only a fraction of that during other episodes, but far more people were involved.

Incident-related and background exposure. The more than 20,000 measurements reported in this paper provide information not only on contamination as a result of the incident but also on background contamination. This structural contamination is probably similar to the situation in other countries of the European Union and might also be similar in other industrialized countries.

Figure 5 shows a peak of measurements with increased PCB concentrations and a PCB:dioxin ratio of approximately 50,000:1 in samples that could be traced back to the feed producers who used ingredients from the incriminated fat-melting company. Figure 5 also shows samples with increased concentrations of PCBs, but with a PCB/dioxin ratio which is clearly different from the 50,000:1 ratio. Because of the stability of the measured dioxin and PCB congeners, these most variable PCB/dioxin ratios point to the existence of other contamination sources, different from the transformer oil. The wide range of dioxin/PCB ratios in contaminated products suggests that many smaller unidentified contamination events occurred, some of which resulted in high levels of contamination. This hypothesis is equally supported by the congener pattern found in the heavily contaminated animal feed samples shown in Figure 6. These PCB profiles are clearly different from the incident-related PCB profile of animal feed.

Analogously, four egg samples, with a PCB content between 1,111 and 1,405 ng/g fat, with no known link to the incident, also showed a PCB pattern that is different from the incident-related PCB profile of eggs. This indicates that in human food, too, high contamination levels are observed independently from the incident.

The joint occurrence of PCBs and dioxins is commonly encountered in thermal processes where precursors and *de novo* synthesis generate PCDDs and PCDFs. Hence one would expect that the PCB incident resulting from the use of discarded transformer oil in recycled fat would be characterized by a nearly constant ratio of PCBs to PCDD/PCDFs. This is observed for the data in Figure 5, which cluster around the PCB/dioxin ratio of 50,000:1. The other points do not coincide with the expected pattern and at higher ratios, PCB levels exceed by far what is expected. In fact, when measurements corresponding to the PCB/dioxin ratios between 25,000 and 100,000 are omitted, an obvious relationship can be observed between the logarithm of the PCB concentration and the logarithm of the PCB/dioxin ratio. This linear regression can be described as $\log_{10}$ PCB = $-0.964 + 0.638 \times \log_{10}$ PCB/dioxin ($r^2 = 0.368$). These findings point toward a latent contamination by PCB mixtures with a very low dioxin content, clearly different from the thermally degraded transformer oil. This contamination comes on top of the background contamination with environmental dioxins and can be responsible for high PCB levels.

Milk and beef are less contaminated with PCBs and dioxins than pork, chicken, and eggs (Table 3). Although during the incident the European Union used 200 ng PCB/g fat as a guideline for contaminated food, it is reasonable to consider that food for human consumption should contain < 75 ng marker PCBs and < 1 pg dioxins/g fat. The percentage of samples with dioxin levels above 2 pg/g fat is high. This applies to all samples, as well as to samples unrelated to the contamination incident. These percentages are also higher than the percentages of samples with > 200 ng PCBs/g fat. Dioxins stemming from environmental contamination are probably responsible for background levels up to about 5 pg/g fat. Environmental PCBs probably only rarely lead to contamination levels above 100 ng PCBs/g fat (*17*). These data provide strong support for the hypothesis that, independent from the incident, the use of recycled fats, oils, and animal waste can lead to high PCB levels in human food.

Measures are urgently needed to reduce the overall PCB and dioxin burden for the population. Known sources independent from the incident are fish, imported vegetable products from countries where DDT is still in use (*17*), recycled animal fat, and all kinds of waste fat that might contain mineral oils. Especially the recycled animal fat and the waste containing mineral oils appear to account for PCB levels above 200 ng/g fat in a few percentages of the meat samples, as shown by measurements on export meat that originated from farms that did not obtain animal feed from producers contaminated by the transformer oil (*17*).

Individual Exposure

Significant numbers of Belgians who consumed contaminated products have temporarily increased their intake of PCDDs/PCDFs up to a factor 100 over the WHO guidelines [1–4 pg I-TEQ/person per day, with 1 pg as the limit in the future (*18*)].

In interpreting the chronic toxicity of persistent chemicals, the body burden is important (*19*). Here we estimated that the average incident-related increase in body burden in Belgium was 7% for PCDD/PCDF and 42% for PCBs. However, because of geographic and dietary reasons, it is likely that the PCB/dioxin burden of the crisis was unevenly distributed among the Belgian population. The U.S. Environmental Protection Agency has estimated that dioxin body burdens might be 3–4 times above the average in about 10% of the population. It is therefore likely that for some subpopulations the increase in body burdens and the associated risk has been substantial. It is important to identify these subpopulations to study the long-term effects associated with these levels of body burdens.

Effects

Acute clinical health effects have not been reported during the Belgian incident. In view of the type of contamination, this is not expected for acute effects as ischemic heart disease (*20*), chloracne, or conjunctivitis (*21*). However, it is most likely that this contamination episode will have delayed effects on the health of exposed individuals.

We estimated the stochastic incremental cancer risk associated with the incident to vary between 44 and 8,316 cancer deaths. This is a first estimation based on a simple model in which a sudden increase in body burden was transformed in a lifelong daily exposure. More complex models relate exposure to age, energy intake, sex, the acute nature of the body burden increase, and the subsequent elimination of PCBs and dioxins from the body. Cancer risk estimates based on these exposure parameters and the unit cancer figures proposed by Becher et al. (*15*) result in figures of the same order of magnitude as the ones presented here (*22*). Moreover, huge uncertainties exist because of the wide variation in the reference estimates of cancer risk and a manifest lack of precision in estimating the individual dose received. At present there is no indication of how the toxic load is distributed over different groups in the Belgian population, but the main uncertainty probably concerns the pathogenic potency of the chemicals involved. Different risk factors are reported in the literature, accounting for differences in risk by a factor 250 for PCBs (*23*) and 100 for dioxins (*15,24,25*).

Moreover, noncancer effects in neonates, infants, and children are important. Four groups of effects are known to be influenced at relatively low-dose exposure to PCBs, dioxins, and/or co-contaminants. First, several studies point to a lack of optimal neurologic function (*26–28*). Second, changes in thyroid hormone levels include increased plasma thyroid-stimulating hormone (TSH) levels, lower plasma TSH levels, and lower plasma tetraiodothyronine (T_4) levels. In particular, the higher TSH levels are indicative for hypothyroidism (*29*). Third, changes in T-cell subpopulations and lower monocyte and granulocyte counts in the blood (*30*) have been reported. Fourth, these contaminants may interfere with the vitamin K metabolism associated with a late form of hemorrhagic disease of the newborn (*31*). This late form is due to vitamin K deficiency at the age of 1 month to 1 year (*32*).

Table 5 relates these health effects in babies with concentrations of PCBs and dioxins in breast milk. It is important to compare these concentrations to the levels found in the milk of lactating women in Belgium (mean for dioxins only: 34.4 pg I-TEQ/g milk fat; range: 27.3–43.2 pg I-TEQ/g). These concentrations are higher than the threshold value at which thyroid changes occur. Therefore, if these mothers double or triple their body burden, thyroid hormone and immunologic changes in their babies should be expected. Hemorrhagic disease of the newborn also occurs at concentrations that probably have been reached as a result of the Belgian dioxin incident.

Conclusion

We assessed data until August 1999 (i.e., during the last phase and immediately after the incident). Subsequent data became available meanwhile. These will be assessed and compared with the current conclusions in a follow-up paper, which will also address issues of latent PCB contamination.

When dioxin-like PCBs are included, Dutch breast milk samples collected in 1990–1991 contained a mean concentration of 72.3 pg I-TEQ/g milk fat (sum of dioxins and dioxin-like PCBs). This corresponds to a body burden of about 14.5 ng I-TEQ/kg body weight (*18*). These levels are of the same order as those causing morbidity in animals (*10*). Furthermore, prenatal exposure or postnatal exposure through breast milk to background concentrations of PCBs and dioxins, as present in a sizeable portion of the Dutch population, was associated with adverse health effects in the children of healthy Dutch women with a normal pregnancy outcome (*33*). Therefore, it is most likely that dioxin and PCB concentrations as they occur in industrialized countries such as Belgium are an important issue in public health.

The Belgian "dioxin crisis," which probably entailed a higher exposure to TEQ through its PCB content (estimated value equivalent to 0.9 g I-TEQ) than through its PCDDs/PCDFs (estimated value 300 mg I-TEQ), should be considered a potentially important public health event. According to our estimations, the incident had a significant impact on the body burden of the modal citizen and has probably doubled or tripled the body burdens of selected subpopulations who were intensely exposed to contaminated food. Dioxin-like compounds are among the reactive or hormone-disturbing substances whose long-term effects may be insidious and particularly hard to detect because of the high background levels. These high background levels should go down. Individuals exposed during the incident to high PCB and dioxin amounts should be traced, and their health status should be monitored. As a precautionary measure, the exposure to these PCBs and dioxins should be decreased by the promotion of chemical and physical hygiene. This form of hygiene is necessary for the primary prevention of cancer and other health problems related to pollution of the environment and the food chain.

Table 5. Breast milk levels of dioxin or PCB associated with health effects in infants.

Health effects	Compound	Breast milk levels	Reference
Neurologic delay (poor short-term memory)	PCB	835.9 ± 388.4 ng/mL	(*26*)
Changes in thyroid hormone status (↑ TSH and ↓T_4)	Dioxin	30.75–76.43 pg TEQ/g fat	(*29*)
Changes in immune system (↑ T cells and ↓ monocyte and granulocyte counts)	CDD-PCB	66.59 pg TEQ/g fat	(*30*)
Late form of HND	CDD-CDF	29.85–92.88 pg TEQ/g fat	(*33*)

CDD, chlorinated dibenzodioxin; CDF, chlorinated dibenzofuran; HND, hemorrhagic disease of the newborn.

References and Notes

1. Baeyens J. Dioxin Emissions for MWI. Aminal report. Brussels, Belgium:Aminal , 1993.
2. Mira T. Milieu- en natuurrapport Vlaanderen: thema's (Verbruggen A, ed). Leuven, Belgium:Garant, 1998.
3. De Fré R, Wevers M. Stofdossier Dioxines–Eindrapport. MEI-DI-9459. Erembodegem, Belgium:Vlaamse Milieumaatschappij, 1995.
4. UNEP. Dioxin and Furan Inventories: National and Regional Emission of PCDD/PCDF. Geneva, Switzerland:United Nations Environmental Program, 1999.
5. Mira T. Milieu- en Natuurrapport Vlaanderen: thema's (Verbruggen A, ed). Leuven, Belgium:Garant, 1999.
6. Cox D. Achtergronden puntblootstelling van de Belgische bevolking aan dioxines [M.S. Thesis]. Brussels, Belgium:Vrije Universiteit Brussel, 1999.
7. Ministerie voor Volksgezondheid en Milieu. Belgisch Staatsblad, 18/07/1990. Appendix 2 van het Koninklijk Decreet van 25/04/1990. Brussels, 1990.
8. WHO. WHO-Coordinated Exposure Study: Levels of PCBs, PCDDs and PCDFs in Human Milk. Environmental Health in Europe No. 3. Bilthoven:World Health Organization, European Center for Environment and Health, 1996.
9. Van Cleuvenbergen R, Wevers M, Schoeters J, De Fré R. Dioxins (PCDDs and PCDFs) in human milk from Flanders, Belgium: concentration levels and congener profile. Organohalogen Compounds 20:215–220 (1994).
10. Van den Berg M, Birnbaum L, Bosveld ATC, Brunström B, Cook P, Feeley M, Giesy JP, Hanberg A, Hasegawa R, Kennedy SW, et al. Toxic equivalency factors (TEFs) for PCBs, PCDDs, PCDFs for humans and wildlife. Environ Health Perspect 106:775–792 (1998).
11. Beernaert S, De Poorter G. Intercomparison Study of PCBs in Feedstuffs, Animal Fat and Foodstuffs. Brussels, Belgium:Federal Ministry of Public Health and Federal Ministry of Agriculture, 1999.
12. Michalek JE, Tripathi RC. Pharmacokinetics of TCDD in veterans of Operation Ranch Hand: 15-year follow-up. J Toxicol Environ Health 57(6):369–378 (1999).
13. van der Molen GW, Kooijman SA, Michalek JE, Slob W. The estimation of elimination rates of persistent compounds: a re-analysis of 2,3,7,8-tetrachlorodibenzo-*p*-dioxin levels in Vietnam veterans. Chemosphere 37(9–12):1833–1844 (1998).
14. Masuda Y, Haraguchi K, Kuroki H, Ryan JJ. Change of PCDF and PCB concentrations in the blood of Yucheng and Yusho patients for 25 years. Fukuoka Igaku Zasshi 86(5):178–183 (1995).
15. Becher H, Steindorf K, Flesch-Janys D. Quantitative cancer risk assessment for dioxins using an occupational cohort. Environ Health Perspect 106(suppl 2):663–670 (1998).
16. Cogliano VJ. Assessing the cancer risk from environmental PCBs. Environ Health Perspect 106:317–323 (1998).
17. Schepens PJC, Covaci A, Jorens PG, Hens L, Sharpé S,

van Larebeke N. Surprising findings following a Belgian food contamination with polychlorobiphenyls and dioxins. Environ Health Perspect 109:101–103 (2001).
18. WHO. Executive Summary. Assessment of the Health Risk of Dioxins: Re-evaluation of the Tolerable Daily Intake (TDI). WHO Consultation, 25–29 May 1998. Geneva, Switzerland:World Health Organization, European Center for Environment and Health, 1998.
19. DeVito MJ, Birnbaum LS, Farland WH, Gasiewicz TA. Comparisons of estimated human body burdens of dioxin-like chemicals and TCDD body burdens in experimentally exposed animals. Environ Health Perspect 103:820–831 (1995).
20. Kogevinas M. Cohort studies of occupationally and environmentally exposed populations. Organohalogen Compounds 44:353–356 (1999).
21. World Health Organization European Center for Environment and Health. Concern for Europe's Tomorrow. Health and Environment in the WHO European Region. Stuttgart, Germany:Wissenschaftliche Verlagsgesellschaft mbH, 1995.
22. Vlietinck R, Van Larebeke N. Unpublished data.
23. Finley BL, Trowbridge KR, Burton S, Proctor DM, Panko JM, Paustenbach DJ. Preliminary assessment of PCB risks to human and ecological health in the lower Passaic River. Toxicol Environ Health 52(2):95–118 (1997).
24. Schecter A, Olson JR. Cancer risk assessment using blood dioxin levels and daily dietary TEQ intake in general populations of industrial and non-industrial countries. Chemosphere 34(5–7):1569–1577 (1997).
25. Portier C. Risk ranges for various endpoints following exposure to 2,3,7,8-TCDD. Food Addit Contam 17(4):335–346 (2000).
26. Jacobson JL, Jacobson S, Humphrey HEB. Effects of in utero exposure to polychlorinated biphenyls and related contaminants on cognitive functioning in young children. J Pediatr 116:38–45 (1990).
27. Jacobson JL, Jacobson S. Intellectual impairment in children exposed to polychlorinated biphenyls in utero. N Engl J Med 335:783–789 (1996).
28. Brucker-Davis F. Effects of environmental synthetic chemicals on thyroid function. Thyroid 8:827–856 (1998).
29. Koopman-Esseboom C, Morse DC, Weisglas-Kuperus N, Lutkeschipholt IJ, Van der Pauw CG, Tuinstra LGMT, Brouwer A, Sauer PJJ. Effects of dioxins and polychlorinated biphenyls on thyroid hormone status of pregnant women and their infants. Pediatr Res 36 (4):468–473 (1994).
30. Weisglas-Kuperus N, Sas TCJ, Koopman-Esseboom C, Van der Zwan CW, De Ridder MAJ, Beishuizen A, Hooijkaas H, Sauer PJJ. Immunologic effects of background prenatal and postnatal exposure to dioxins and polychlorinated biphenyls in Dutch infants. Pediatr Res 38:404–410 (1995).
31. Koppe JG. Nutrition and breast-feeding. Eur J Obstet Gynecol Reprod Biol 61:73–78 (1995).
32. Lane PA, Hathaway WE. Vitamin K in infancy. J Pediatr 106(3):351–359 (1985).
33. Koppe JG, Pluim HJ, Olie K, van Wijnen J. Breast milk, dioxins and the possible effects on the health of newborn infants. Sci Total Environ 106:33–41 (1991).
34. Hens L. Dioxines en PCBs in Belgische eieren en kippen. Milieu 14:220–224 (1999).