PERSPECTIVES

The Belgian PCB/Dioxin Incident: A Critical Review of Health Risks Evaluations

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The Belgian PCB/dioxin incident is a food contamination that occurred in Belgium in January 1999 when a tank of recycled fats used to produce animal feeds was accidentally contaminated by approximately 100 L of polychlorinated biphenyl (PCB) oil containing 50 kg PCBs expressed as the sum of the seven markers, 1 g (TEQ) dioxins (PCDD/Fs) and 2 g toxic equivalent (TEQ) dioxin-like PCBs. The incident was discovered when a poultry poisoning resembling the classic chick edema disease broke out in several farms that had received contaminated feeds. The delay in making public this incident resulted in a major political and food crisis and caused much concern in the population. We review here the health risk evaluations that were made after this incident and we assess the likelihood of the different scenarios by taking into account recent data on the real scale of the contamination and on the dioxin body burden of the general population in Belgium. These new data confirm that the incident was too limited in time and in scale to have increased the PCB/dioxin body burden of the general population at large, a conclusion supported by a survey of dioxin levels in blood conducted at the end of 1999. Only farmers in poultry farms affected by the incident (about 30 farms) and having regularly consumed their own products could have increased their PCB/dioxin body burden. It is unlikely, however, that these farmers could have increased their PCB/dioxin body burden above levels prevailing in the 1980s or now found in communities regularly consuming seafood.

Keywords Chick Edema Disease, Dioxin, Food Safety, MSWI, PCB, Risk Assessment

In January 1999, a food contamination incident occurred in Belgium when a mixture of polychlorinated biphenyls (PCBs) was inadvertently mixed with recycled fats used in the production of animal feeds. Although signs of poultry poisoning resembling those of the classic chick edema disease were already noticed in February, the contamination was identified and made public much later in May, triggering a major political and food crisis. Because dioxins (polychlorodibenzodioxins [PCDD/Fs]) were the first contaminants identified, this crisis was referred to in the media as a dioxin crisis but the incident was primarily due to a PCB contamination, the dioxins being present only as secondary contaminants arising from the thermal degradation of the PCBs (Bernard et al. 1999; Belgian Parliament 2000).

When the incident was discovered, the stock (60 tons) of contaminated fats had already been sold to nine manufacturers of animal feeds, who in turn had delivered potentially contaminated feeds to more than 2500 farms. At the exception of the poultry farms where signs of chick edema disease were discovered, it was thus impossible to identify the farms having received contaminated feeds and even more so to trace back the contaminated foodstuffs that had been sold and people having consumed them. This uncertainty about the real extent of the contamination resulted in an embargo on all Belgian food products of animal origin. This led the Belgian authorities to implement what was probably the largest screening program for contaminated foods ever conducted over such a short period of time.

Signs of poisoning were reported only in the poultry, and more precisely in the reproduction animals such as hens and chicks. These signs consisted in a marked drop in egg production observed early February and, a few weeks later, in a reduced hatchability of eggs and increased mortality of chicks. No signs of intoxication were found in other farm animals. There were also no cases of acute intoxication in humans that could have been attributed to the consumption of contaminated foods. However, because of the very high levels of dioxins and PCBs and the well-documented toxicity of these chemicals, a controversy rapidly arose in the media and then later in the scientific community about the possible health consequences of this incident. At the end of 1999, several publications and meetings reports were released, concluding that this incident was unlikely to have had

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an impact on public health (World Health Organization [WHO] 1999; Conseil Supérieur d'Hygiene Publique de France 1999; Tuomisto, Vartiainen, and Tuomisto 1999; Van Leeuwen 2000; Bernard 2000a). More recently, however, a paper published by Van Larebeke et al. (2001) in *Environmental Health Perspectives* reopened the debate by predicting that the PCB incident might cause up to about 8000 additional cancer cases in Belgium during the next decades. So far, the Belgian PCB/dioxin incident has thus been subjected to separate risk analyses, leading in some cases to contrasted conclusions despite the use of the same database. No study has attempted to put together all these analyses and to compare the risk assessment approaches that were adopted and the assumptions on which risk estimations were based. To fill this gap, we review here the different health risk evaluations that have been made about this incident and we assess the likelihood of the different scenarios envisaged.

REVIEW OF HEALTH RISK EVALUATIONS

In June 1999 the Bilthoven Division of the WHO European Center for Environment and Health released a provisional risk assessment based on the first analyses on poisoned poultry, which were available at the end of May and indicated dioxin (PCDD/Fs) values up 700 pg toxic equivalent (TEQ)/g fat (WHO 1999). This report estimated that the consumption of such contaminated meats could lead to an additional intake in one meal of approximately 8000 to 10,000 pg TEQ, i.e., approximately 30 times the higher end of the range of the tolerable daily intake (TDI, 1 to 4 pg TEQ/kg body weight). Because the TDI is based on a lifetime exposure, WHO experts considered that an occasional short-term excursion above the TDI like the one that might have occurred in Belgium between January and June 1999 could not necessarily have serious consequences. The WHO experts recognized, however, that such an intake might considerably contribute to the current body burden of the general population in Belgium.

On September 17, 1999, a brief communication was published in *Nature* based on a more extended database (Bernard et al. 1999). At that time, the source of the contamination had been identified and most contaminated samples of chicken, eggs, and feeds retrieved in farms where the chick edema diseases broke out had been analyzed for both dioxins and PCBs. This paper concluded that the Belgian PCB incident was unlikely to have affected the health of the general population. It was estimated that in the worst case scenario the consumption of the most contaminated foods during the period of the incident would lead to a doubling of the body burden. This extreme scenario would mean that the body burden would go back to those prevailing in the 1980s. This paper also provided an estimate of the total amount of dioxins (1 g TEQ PCDD/Fs) and PCBs (50 kg for the seven PCB markers) introduced in the food chain in Belgium. In September, Tuomisto, Vartiainen, and Tuomisto (1999) published a booklet in which they made an evaluation very similar to that published in *Nature*. These authors calculated that an increase of the dioxin body burden to levels similar to the Seveso accident would require the regular consumption (twice a week) of the most contaminated foods for a period of 30 to 50 years. They also concluded that such a scenario was conceivable only for farmers who might have consumed their own products. Although recognizing that the risks of PCBs were more difficult to assess, they considered, however, that no measures should be taken for persons who might have consumed contaminated foods. In October 1999, an international meeting was organized by the Royal Academy of Medicine of Belgium to evaluate the possible public health implications of the incident. At that meeting, Dr Van Leeuwen (2000), head of the Bilthoven Division of the WHO European Center for Environment and Health, presented an updated evaluation of the health risks of the incident that basically confirmed the conclusions of the June report (WHO 1999). Dr Van Leeuwen explained that if the Belgian incident could have led to a dioxin intake of 150 pg TEQ/kg/day, i.e., largely above the current TDI, this intake remained however 1000 times lower than the average intake of the Yusho patients. Proceeding with the analysis, Dr Van Leeuwen concluded that, at this daily intake, the health effects were not likely to occur before 1 year of regular consumption of the most contaminated foods. At the Brussels meeting, Dr Willems of the Health Council of Belgium (Willems 2000) presented an assessment that he qualified as intermediate between that in published in *Nature* and the recent publication in *Environmental Health Perspectives*. According to his calculations, the average daily intake of dioxins in Belgium could have been increased by a factor of 5 and, for 5% of the Belgian population, even by a factor of 10. This intake could have doubled the dioxin concentrations in the blood of children aged 2 to 12 years and for persons with special habits, in a worst-case scenario, it could have brought their dioxin blood concentrations to levels observed in occupational settings.

The most contrasted risk evaluation was made more recently by Van Larebeke et al. (2001) who estimated that the Belgian PCB incident has had a significant impact on the dioxin body burden of the Belgian population, having probably doubled or tripled the body burden of selected subgroups. These authors based their risk assessment on the figures of 50 kg PCBs (seven markers) and 1 g TEQ dioxins (PCDD/Fs) published in *Nature* (Bernard et al. 1999). From these figures, they estimated that approximately 300 mg TEQ of dioxins and about 900 mg TEQ PCBs have been really consumed by the Belgian population. Then, assuming that these amounts have been uniformly distributed to the 10 millions Belgian citizens, they calculated the dose received by each citizen that they divided by 25,550 to estimate the corresponding daily dose over a period of 70 years. Assuming also that the whole Belgian population had been similarly exposed to the equivalent of this lifetime daily dose, they estimated that the incident will cause between approximately 40 and 8000 additional cancer cases over a period of 70 years by extrapolation from the lifetime unit cancer risks derived from animal or human data. They also concluded that this incident had

probably caused hormonal and immunological effects on the babies of women having consumed large amounts of contaminated foods. Before analyzing the assumptions at the basis of these three scenarios, we first summarize all data available to date about this incident and its possible impact on the PCB/dioxin body burden of the Belgian population.

DATA AVAILABLE FOR RISK ASSESSMENT

The Nature of the Contamination

The Belgian food contamination is primarily a PCB contamination. It was wrongly presented in the media as a dioxin contamination, which probably greatly contributed to exaggerate the risks perception. From the PCB fingerprints in contaminated feeds retrieved in poultry farms affected by the incident, it was possible to determine that the PCB oil at the origin of the incident was composed of a mixture of Aroclor 1260 and 1254 (or of other commercial PCBs with similar compositions) in the proportions of 75/25 (*N.B.* a different ratio is found in contaminated animals because of the differential elimination of PCB congeners). This PCB oil was highly contaminated with dioxins (90% of which were contributed by polychlorodibenzofurans [PCDFs]) originating from the thermal degradation of PCBs. The patterns of PCBs and dioxins as well as the PCB:dioxin ratios (around 50,000) were fairly constant in all feed samples, suggesting the existence of a single source of contamination. By extrapolating the concentrations of PCBs in the originally contaminated animal feeds to the total volume of the contaminated tank, it was possible to estimate the total amount of PCBs introduced in the food chain. This amount was estimated to 50 kg expressed as the sum of the seven PCB markers, which corresponds to about 150 kg total PCBs and 100 L of PCB oil/wax. This PCB mixture was contaminated by 1 g TEQ dioxins (PCDD/Fs) and 2 g TEQ dioxin-like PCBs (Bernard et al. 1999, 2002).

The Transmissibility of the Contamination in the Food Chain

The contamination was due to higher chlorinated PCBs, which are known to be very persistent in living animals and hence transmissible along the food chain to man. In poultry products, the dioxin and PCB patterns as well as the PCB:dioxin ratios were almost unaltered because of the low capability of birds to metabolize polyhalogenated hydrocarbons. By contrast, in pigs, the patterns of dioxins and PCBs as well as the PCB:dioxin ratios were markedly altered as a result of the preferential elimination of dioxins by these animals (Bernard et al. 2002).

The Levels of Contamination

The highest concentrations of PCBs and dioxins were observed in poultry with symptoms of chick edema disease. The PCB concentrations in most contaminated samples reached 50 μg/g fat and the corresponding dioxin (PCDD/Fs) concentrations about 1000 pg TEQ/g fat (total TEQ activity was 3000 pg TEQ/g fat by including the TEQ values of dioxin-like PCBs). The PCB concentrations in pigs were nearly as high as in poultry reaching values of 20 to 30 μg/g fat but these animals had very low values of dioxins (the highest value was 36.3 pg TEQ/g fat), most likely as a result of the faster elimination of dioxins than PCBs in that species. The bovine livestock was almost spared with very few values above the tolerance levels. The highest values that were found in some nonlactating cows did not exceed a 10th of the maximal values found in pigs and chickens (Bernard et al. 2002).

The Duration of Contamination

The duration of the incident could be determined with a certain accuracy on the basis of contaminated animal feeds retrieved in affected farms and in animal feed manufacturers. As shown in Figure 1, the PCB contamination of feeds peaked at the end of January, then very rapidly declined to almost normal values in April. The peak of PCBs in feeds manufactured in January coincided with the outbreak of poultry poisoning, which occurred a few days later in several poultry farms. The duration of food contamination and hence of human exposure is however much more difficult to assess but can be logically inferred from the lifecycles of farm animals and food products. Because of the relatively short life cycle of chickens, the peak of contamination in most poultry products probably coincided relatively well with that in feeds with a contamination period not exceeding 2 months. One can thus assume that most contaminated poultry products had been consumed when the incident was made public at the end of May. Only some products derived from eggs could still be on the market by that time but mixed with other ingredients. For animals with a longer life cycle such as pigs, the contamination was delayed and probably lasted much longer. In July to August 1999, high concentrations of PCBs were indeed still found in young pigs, which presumably had been contaminated via their mother in utero and during lactation. These animals, however, had eliminated most of their dioxin body burden together with lower chlorinated PCBs and those who had still abnormal PCB levels were destroyed. These two factors, metabolism and sacrifice after the detection of the incident, have probably contributed to reduce the amounts of PCBs/dioxins that entered the food chain via swine products. The bovine livestock was almost unaffected by the incident because of their lessen dependence on manufactured feeds and also their very long life cycle compared to the period of contamination (Bernard 2000b; Bernard et al. 2002).

The Proportion of the Food Chain Affected

For assessing the risks at the population level, this parameter is probably one of the most critical, determining not only the number of individuals possibly exposed but also the likelihood that these individuals could have had sufficiently consumed

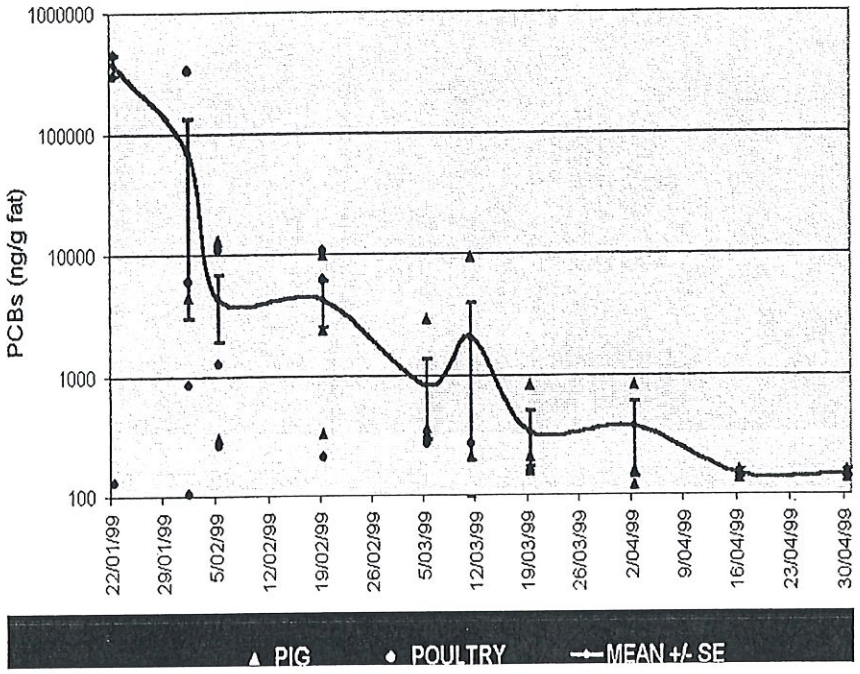

FIGURE 1

Time course of PCB (sum of seven markers, PCB no. 28, 52, 101, 118, 138, 153, 180) concentrations in poultry and pig feeds retrieved in farms affected by the Belgian PCB incident. The mean value of January 22 has been calculated by excluding the negative sample (the sample containing about 0.1 μg PCB/g fat). Each point represents one to three values.

contaminated foodstuffs to increase their PCB/dioxin body burden. At the heart of the crisis, a theoretical estimate was made on the basis of the volume of PCBs/dioxins introduced in the food chain. Because about 80% contaminated feeds were delivered to poultry farms, this meant that a maximum of one million chickens could have been contaminated with PCBs and dioxins at levels sufficiently high (50 μg and 1000 pg TEQ/g fat, respectively) to lead to a doubling of the dioxin or PCB body burden after a period of 2 months of regular consumption. This number represented less than 2% percent of the total number of chickens consumed during the incident by the Belgian population (20 million chickens sacrificed per month). The rest of contaminated feeds (20%) being mostly delivered to pig farms, it can be calculated that a maximum of 20,000 pigs were contaminated at sufficiently high levels to rise the PCB/dioxin body burden of consumers during the incident. This number again represents just a few percents of the total number of pigs sacrificed during that period (10 million pigs are produced yearly in Belgium). These estimates are in accordance with those from the PCB monitoring program implemented in June which showed that less 2% of poultry farms (30 out of 1517), 1.5% of pig farms (167 out of 13,389), and 0.5% of all farms (223 out of 67,152) in Belgium had been contaminated by this incident. Of these estimates, one should however subtract the numbers of animals that were destroyed during the crisis (2 million chickens already in February according to Dr. Destikkere, communication during the hearings at the Belgian parliament) (Belgian Parliament 2000), which means that the numbers of highly contaminated animals introduced in the food chain were probably much less (Bernard et al. 2002).

The Size of the Population Affected

The major uncertainty about the Belgian PCB incident concerns the number and identity of exposed individuals. These individuals could not be traced back from the sales of foods because the incident was discovered several months after the products had been marketed. There were also no cases of human intoxication that could have helped to localize certain areas more at risk.

The Critical Adverse Effects

The debate about the health impact of the Belgian incident largely stemmed from the use of two different approaches to assess the risks of dioxins and PCBs. In their study, Van Larebeke et al. (2001) have considered that cancer risks were the most critical effects to be feared from this incident. In order to calculate the number of additional cancer cases expected, they have used the Environmental Protection Agency (EPA) approach, treating cancer risk as nonthreshold stochastic effects. By contrast, other evaluators considered that carcinogenic effects were unlikely at the exposure levels attainable during the Belgian incident and that the health risk evaluation should be focused on developmental and hormonal effects, which on the contrary are nonstochastic

effects that are likely to occur during pregnancy or in breastfed babies. Thus, depending on the end point considered as critical, the time scale relevant for risk assessment and the size of the population at risk are completely different. Developmental effects are related to intakes during pregnancy and thus concern women who were pregnant during the incident. The cancer risks evaluation by contrast necessitates an extrapolation over lifetime and concerns the whole Belgian population. Both approaches, however, rely on the body burden concept, which assumes that once allowance is made for toxicokinetic differences between species, risks can be extrapolated from animal to man and from acute/subacute exposures to chronic exposures (Larsen et al. 2000).

The PCB and Dioxin Body Burden Before and After the Incident

Before the incident, the only data concerning the dioxin and PCB body burden of the Belgian population were those on pooled human milk samples obtained during the two exposure studies coordinated by WHO, the first in 1986/87 and the second 1992/93 (WHO 1996). These samples were obtained from primiparae breastfeeding mothers. In both rounds, dioxin concentrations in human milk observed in Belgium were amongst the highest of industrialized countries with mean values in the three studied areas (Brabant Wallon, Liège, and Brussels) ranging from 33.7 to 40.2 in 1986/87 and from 20.8 to 27.1 pg TEQ/g fat in 1992/93. The mean concentrations of PCBs (seven markers) in human milk were also amongst the highest of industrialized countries with values ranging from 558 to 609 μg/g fat in 1986/87 and from 261 to 306 μg/g fat in 1992/93. Between the two rounds, i.e., over a period of 5 years, the mean dioxin concentrations had decreased on average by 34.2% and that of the seven PCB markers by 51%. Between 1993 and early 1999 when the dioxin crisis broke out, there were thus no dioxin measurements done on the general population in Belgium. The most recent data before the incident were thus the values on pooled milk samples collected in 1992/93. These dioxin values (20.8 to 27.1 pg TEQ/g fat) were obtained from breastfeeding mothers with an average age of 26.4 (Brabant Wallon, $n = 8$), 24.3 (Liège, $n = 20$), and 28 years (Brussels, $n = 6$). During the Winter of 1999–2000, we collected blood samples from 112 subjects living in the center and South of Belgium. These samples were obtained in the framework of an epidemiological study designed at the end of 1998 to identify environmental factors likely to influence the dioxin body burden in Belgium (Bernard et al. 2001). The fact that this study started a few months after the PCB incident was thus a pure coincidence. Subjects were recruited in three areas of Belgium: a rural area in the South of Belgium with no known source of pollution by dioxins and PCBs (area 1, $n = 27$), a rural area in the center of the country in the vicinity of a municipal solid waste incinerator (MSWI, area 2, $n = 52$), and an industrialized area with a variety of potential sources of dioxin emissions (steel industry, MSWI, urban pollution; area 3, $n = 33$). The recruited population consisted of 57 women and 55 men aged 21 to 80 years (mean, 50.6 years). All subjects participated to the study on a volunteer basis after having given their informed consent. Information about their medical history, lifestyle, and food habits was obtained from a self-administered questionnaire. Between 200 and 300 ml of whole blood was taken from each subject in the morning under fasting conditions, which allowed to evaluate their dioxin body burden. Dioxins (17 PCDD/Fs congeners) were determined on serum lipids by high-resolution mass spectrometry and results were expressed per gram fat using the WHO toxic equivalent factor (TEF) factors. 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. Residents of the area 2 (rural area in vicinity of a MSWI) had a significantly higher dioxin body burden than those in the two other areas (pg TEQ/g fat, 37.7 vs. 27.2 in area 1 and 24.3 in area 3), a difference that persisted after adjustment for age. As expected, the dioxin body burden was significantly correlated with age. For the 60 subjects living in areas 1 and 3 whose dioxin body burden was not increased by MSWI emissions, the correlation coefficient between dioxin body burden and age amounted to $r = .35$ ($p < .006$) and the equation of the regression line was: log PCDD/Fs in serum (pg TEQ/g fat) = 1.057 + 0.0072 age (years). The adjustment of the dioxin body burden of these subjects for the mean ages (24 to 28 years) of the breastfeeding mothers examined in 1992/93 gives dioxin concentrations in the range from 17 to 18 pg TEQ/g fat. Even lower values (9.5 to 17.5 pg TEQ/g fat) are obtained if the adjustment for age is based on the yearly increment of dioxin body burden estimated by Papke (1998) (0.4 to 0.8 pg TEQ/g fat per year). Because under steady-state conditions, concentrations of dioxins expressed in TEQ values are similar in serum and milk fat (Schecter, Kassis, and Papke 1998), these data suggest that there has not been a generalized increase of the dioxin body burden in Belgium after the PCB incident and in fact that the dioxin body burden of the general population has slightly decreased if one refers to the values reported in human milk in 1992/93. Furthermore, the patterns of dioxins in these subjects did not reveal any increase of the congeners characterizing the PCB incident pentachlorodibenzofuran (PeCDF) and hexachlorodibenzofurans (HxCDFs). These congeners were present in very small concentrations, which were indistinguishable from that reported in other countries or in the WHO studies on human milk (Figure 2). These data thus do not support the hypothesis of a generalized increase of dioxin body burden of the Belgian population (Bernard et al. 2001).

Estimation of the Likelihood of Different Scenarios

Scenario 1: Adverse Effects Are Improbable

This conclusion is based on the estimation of the likelihood that during the incident some individuals could have had consumed a sufficiently number of times the most contaminated foods to increase their body burden at levels that might cause adverse effects (Bernard et al. 1999; WHO 1999; Tuomisto,

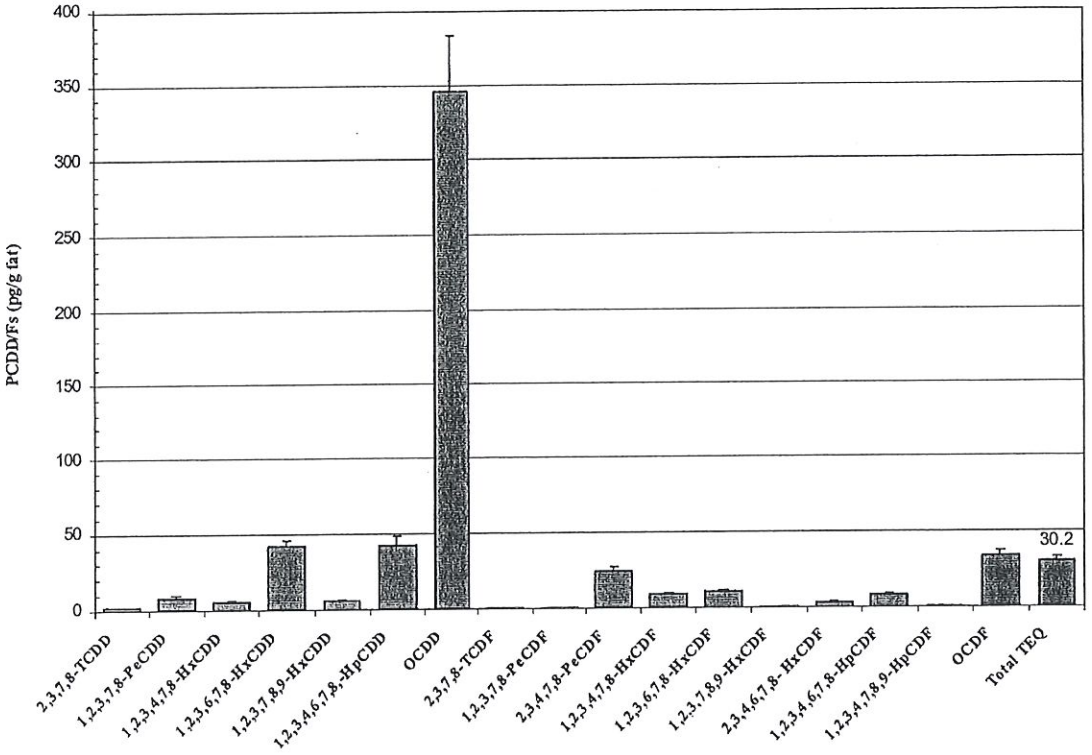

FIGURE 2

Patterns of dioxin congeners (PCDD/Fs) in serum lipids from 113 subjects examined at the end of 1999 and early 2000 in Belgium (from Bernard et al. 2001). Data are geometric mean + *SE*.

Vartiainen, and Tuomisto 1999; Bernard 2000a, 2000b; Bernard et al. 2001). This risk assessment thus makes no assumption about the nature of the possible health effects nor on the size of the affected population. It simply relies on the fact that PCBs and dioxins are toxins that are readily absorbed and accumulate in the body fat in a predictable way according to the kinetics of one-compartment model. Two factors together have contributed to make improbable the risk of an increase of the PCB/dioxin body burden of the general population during this incident. The first is the time factor, which limits this risk to the consumption of the most contaminated food products such as those found in farms where signs of poultry poisoning were found. The second factor is the very limited number of poultry farms really contaminated (less than 2% of all poultry farms in the country). This factor obviously has considerably reduced the probability that some individuals could have regularly consumed the most contaminated foods especially as a significant proportion diseased animals were destroyed. The only subjects who might have been regularly exposed to such foods are farmers in contaminated farms consuming their own products. Although the epidemiological study recently conducted in Belgium might not have caught individuals having consumed contaminated meals, the results of this study tend to corroborate this first scenario as no trace of the PCB incident was found in the dioxin concentrations and the dioxin patterns in the studied population.

Scenario 2: An Increase of Cancers Deaths

This scenario developed by Van Larebeke et al. (2001) rests on three assumptions concerning the exposure of the population and the extrapolation of cancer risks. First, by dividing the total doses of dioxins and PCBs by the number of inhabitants in Belgium, the authors assume that the contamination has been rather uniformly distributed all over the food chain. Such a premise would be quite conceivable in the case of a community who would have consumed almost exclusively foods from affected farms but it appears unlikely when less than 2% of the poultry farms of Belgium have been affected by the incident. The second assumption is that the cancers risks due to a peak exposure to dioxins can be estimated by extrapolating the ingested dose over lifetime without any adjustment for the elimination of these compounds nor for the future trend in dioxin exposure. The authors recognize that their calculation is based on a simplified model that does not take into account several variables, including the subsequent elimination of dioxins and PCBs from the body, which is a justifiable approximation for TCCD and other dioxin congeners with half lives around or higher than 10 years. However, observations after the Yusho and Yu-Chen rice poisoning as well as after the Binghamton building fire incident indicate that PeCDFs and HxCDFs, the predominant dioxin congeners in the Belgian incident, are eliminated with much shorter half-lives, between 2 to 5 years (Olson 1994). This means that the

period during which dioxins from the PCB incident could be active as cancer promoters is much shorter than the lifetime period on which Van Larebeke et al. (2001) have based their extrapolation. The third assumption is that the cancer risks can be linearly extrapolated from high to low doses, which is a debatable premise especially for nongenotoxic chemicals such as dioxins and PCBs. But even if one accepts that these three assumptions are founded, one must then wonder about the real public health significance of a lifetime excess of 80 to 8000 cancers cases for a country of 10 millions inhabitants such as Belgium. The lowest estimate is certainly insignificant whereas the highest estimate does not appear really impressive compared to the total number of cancers expected in the next 70 years in Belgium (more than 2 millions).

Another important factor overlooked in this prediction is the foreseeable evolution of the dioxin body burden of the Belgian population during the next decades. The dioxin body burden of the general population in Belgium, like that in most industrialized countries, has been decreasing for more than 15 years, as shown by the WHO studies and confirmed by our data. There is little doubt that this decrease will continue in the coming decades as a result of the measures that were taken by the Belgian Authorities after this incident (e.g., banning of animal fat recycling, implementation of a PCB monitoring program for animal feeds) and also with the proposal at the European level of increasingly stringent dioxin standards for food products. All standards presently adopted for dioxins in biological and environmental media are indeed set at levels that are intended to bring the dioxin intake in European countries below the lowest range of the WHO recommended TDI (1 pg TEQ/kg day). Actually, with all these measures, if dioxins and PCBs represent a cancer risk for the general population at environmental exposure levels, the most likely scenario for the coming decades is a decrease of cancer cases attributable to these pollutants, in parallel with the declining body burden of the general population that logically should result from the decreasing levels of these pollutants in human foods.

Scenario 3: Risks of Developmental Effects

The most likely adverse effects that could have occurred during the Belgian PCB incident are developmental anomalies in babies of women who were pregnant or were breast feeding during the incident (Willems 2000; Van Larebeke et al. 2001). In the worst-case scenario, one can calculate that the consumption of the most contaminated chickens (200 g of chicken meat with a dioxin level of 1000 pg TEQ/g fat, 5% fat after cooking) could lead in one meal to a total dioxin intake up to 10,000 pg TEQ. If one adds to this value the activity of dioxin-like PCBs, this results in a total intake of 30,000 pg TEQ in one meal. This value (about 500 pg TEQ per kg) exceeds the upper limit of the WHO TDI by a factor of 100. Although very high, such an intake remains however still at least 2 orders of magnitude below those that have produced clinical manifestations of toxicity in adults or newborns in industrial accidents (Seveso) or in previous episodes of food poisoning (Yusho). It is also unlikely that such an acute exposure causes subclinical adverse effects in breast-fed children, because a similar exceedance of the TDI occurs normally during breast-feeding with so far no evidence of clinically significant adverse effects on the newborns. The only situation for which an acute exposure of that magnitude could lead to developmental effects is during pregnancy. Such a possibility is suggested by epidemiological studies associating subtle developmental effects to the in utero exposure to backgrounds levels of these pollutants (Van Leeuwen and Younes 1998). However, it is difficult to evaluate to what extent these effects observed in the context of chronic exposures can be extrapolated to the peaks of exposure that the Belgian population might have experienced. Animal data are not very helpful in that respect. Acute developmental effects have been induced in the offspring of rats by a single administration of TCDD (Gray, Ostby, and Kelee 1997) but at doses (64,000 pg TEQ/Kg) at least 100 times higher than that attainable during the Belgian incident in a single meal (see above). A learning deficit has also been described in the offspring of monkeys given a daily TCDD dose in the range of the Belgian incident (160 pg TCDD/kg), but these effects were reported after a continuous administration of 4 years, leading to a body burden about 10 times higher than that of the young adult population (Schantz and Bowman 1989). Theoretically, the risk of subclinical developmental effects cannot be excluded after the Belgian incident but the detection of these effects would have required conducting an epidemiological study at the end of 1999 on women who were pregnant between February and April 1999 and had consumed contaminated foods. These women could have been found among farmers consuming their own products, but, in view of the number of poultry farms really affected by the contamination (2% of poultry farms, i.e., about 30 farms), the number of possible cases appears at the very least limited.

REFERENCES

Belgian Parliament. 2000. *Report on the organization and production of meat, dairy and eggs in Belgium and on the political responsibilities in the dioxin crisis.* Parliament of Belgium, P. Vanhoutte and L. Pâque, authors. Doc 50 0018/007. March 3 (in Dutch/French).

Bernard, A. 2000a. Study of the levels and sources of PCBs/dioxins in the food chain and general population of Belgium. Proceedings of the workshop on possible health implications of the dioxin crisis. *Verhandelingen van de Koninklijke Academie voor Geneeskunde van Belgie* 2:95–97.

Bernard, A. 2000b. La contamination alimentaire par les PCBs/dioxines en Belgique: Analyse d'un accident aux conséquences sanitaires improbables. *Bulletin et Mémoires de l'Académie royale de Médecine de Belgique* 153:195–204 (abstract in English).

Bernard, A., F. Broeckaert, G. De Poorter, A. De Cock, C. Hermans, C. Saegerman, and G. Houins. 2002. The Belgian PCB/dioxin incident: Analysis of the food chain contamination and health risk evaluation. *Environ. Res.* 88:1–18.

Bernard, A., S. Fierens, H. Mairesse, C. Hermans, F. Broeckaert, J. F. Focant, and E. Depauw. 2001. Incinérateurs, crise dioxine et risques sanitaires pour la population belge. *Bulletin de la Classe des Sciences. Académie Royale de Belgique*. Tome XII, 1-6, 1041-103-117.

Bernard, A., C. Hermans, F. Broeckaert, G. De Poorter, A. De Cock, and G. Houins. 1999. Food contamination by PCBs and dioxins. *Nature* 401:231–232.

Conseil Supérieur d'Hygiene Publique de France. 1999. *Rapport de la section de l'alimentation et de la nutrition.* [http://www.sante.gouv.fr/htm/dossiers/dioxins/accueil.htm]

Gray, L. E., J. S. Ostby, and R. J. Kelee. 1997. A dose-response analysis of the reproductive effects of a single gestational exposure dose of 2,3,7,8-tetrachlorodibenzo-*p*-dioxin (TCDD) in male Long Evans hooded rat offspring. *Toxicol. Appl. Pharmacol.* 141:11–20.

Larsen, J. C., W. Farland, and D. Winters. 2000. Current risk assessment approaches in different countries. *Food Addit. Contam.* 17:359–369.

Olson, J. R. 1994. Pharmacokinetics of dioxins and related chemicals. In *Dioxins and health*, ed. A. Schecter, 163–189. New York: Plenum Press.

Papke, O. 1998. PCDD/PCDF: human background data for Germany, a 10-year experience. *Environ. Health Perspect.* 106:723–731.

Schantz, S., and R. E. Bowman. 1989. Learning in monkeys exposed perinatally to 2,3,7,8-tetrachlorodibenzo-*p*-dioxin (TCDD). *Neurotoxicol. Teratol.* 11:13–19.

Schecter, A., I. Kassis, and O. Papke. 1998. Partitioning of dioxins, dibenzofurans, and coplanar PCBs in blood, milk, adipose tissue, placenta and cord blood from five American women. *Chemosphere* 37:1817–1823.

Tuomisto, J., T. Vartiainen, and J. T. Tuomisto. 1999. *Synopsis on dioxins and PCBs.* Kuopio, Finland: Publication of the National Public Health Institute. [http://www.ktl.fi/dioxin/]

Van Larebeke, N., L. Hens, P. Schepens, A. Covaci, J. Baeyens, K. J. L. Everaert, R. Bernheim, R. Vlietinck, and G. De Poorter. 2001. The Belgian PCB and dioxin incident of January–June 1999: Exposure data and potential impact on health. *Environ. Health Perspect.* 109:265–273.

Van Leeuwen, F. X. R. 2000. Risk assessment of dioxins and PCBs. Proceedings of the workshop on possible health implications of the dioxin crisis. *Verhandelingen van de Koninklijke Academie voor Geneeskunde van Belgie* 2:81–102.

Van Leeuwen, F. X. R., and M. Younes. 1998. WHO revises the tolerable daily intake (TDI) for dioxins. *Organohalogen Compounds* 38:295–298.

Willems, J. 2000. The risks of the dioxin crisis. Proceedings of the workshop on possible health implications of the dioxin crisis. *Verhandelingen van de Koninklijke Academie voor Geneeskunde van Belgie* 2:90–95.

World Health Organization. 1996. Levels of PCBs, PCDDs and PCDFs in human milk. Environmental Health in Europe no. 3. Bilthoven: WHO European Centre for Environment and health.

World Health Organization. 1999. Contamination with dioxin of some Belgian food products. Bilthoven: WHO European Centre for Environment and Health. [http://www.who.dk/envhlth/dioxin/dioxin.htm]