THE BELGIAN PCB/DIOXIN INCIDENT : ANALYSIS OF THE FOOD CHAIN CONTAMINATION AND HEALTH RISK EVALUATION

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INTRODUCTION

In January 1999, Belgium was the theatre of an unprecedented food crisis caused by the contamination of animal feeds with polychlorobiphenyls (PCBs) and dioxins (PCDD/Fs). The first signs of contamination were observed early February in several poultry farms. The first symptom was a sudden drop in eggs production, followed a few weeks later, by a marked reduction in egg hatchability, a reduced weight gain and an increased mortality of chicks. These birds presented ascites, subcutaneous edema of the neck and neurological disturbances (ataxia). Histology revealed degenerative changes of the skeletal and cardiac muscles. Such lesions resembled the classical manifestations of the " chick-edema disease " which was described in the 1950s-70s in outbreaks of poultry poisoning by polyhalogenated hydrocarbons (1).

These observations led to suspect dioxins as the causative agent, an hypothesis which was confirmed in April when exceptionally high levels of dioxins (more than 1,000 pg TEQ/g fat) were found in feed, meat and eggs of affected poultry. In June, additional analyses revealed that these dioxins, as suggested by their patterns, originated from a contamination by PCB oil accidentally introduced in a tank of recycled fats used in the manufacture of animal feeds. When it appeared that the contaminated fat could have been sold to nine manufacturers of animal feeds which in turn supplied a total 2,500 farms, it became impossible to trace the contamination in the Belgian food chain. This prompted the Belgian authorities to implement a large PCB monitoring program to identify contaminated foods. Initially this program was intended to detect contaminated products from suspected farms but rapidly it was extended to all farms over the country as a result of the embargo imposed on all Belgian food products. At the end of 99, the database contained the results of more than 50,000 PCB and 500 dioxin analyses performed on virtually all types of food products.

In the present study, we analyzed the levels and the patterns of PCBs and dioxins in animal feeds and in food products which were effectively contaminated (poultry, pigs) or suspected of having been contaminated by the incident (bovines). Using the PCB/dioxin fingerprints and the PCBs:dioxins ratio to trace the contamination in the food chain, we determined the time course of the incident, the source and amount of PCB oil as well as the transfer of the contamination into farms animals and their products. By estimating the fraction of the food chain really affected by the contamination, we then assessed the likelihood this incident may have increased the body burden of the general population.

RESULTS

Levels of PCBs and PCDD/Fs in the contaminated food chain

Table 1 gives the geometric means and ranges of PCB and dioxin concentrations in the most contaminated samples discovered after the PCB incident. Samples of chicken feed produced end of January showed the highest levels of contamination, with PCB and dioxin concentrations reaching 450 µg/g fat and 11,140 pg TEQ/g fat, respectively. The PCB concentrations in poultry products were at least one order magnitude lower with means ranging from 0.84 to 8.2 µg/g fat and maximal values between 1.63 and 56.8 µg/g fat. The mean dioxin concentrations in poultry products ranged from 3.8 to 255 pg TEQ/g fat and the maximal values from 19.6 to 2,613 pg TEQ/g fat. The highest concentrations of PCBs (> 50 µg/g fat) and dioxins (> 1,000 pg TEQ/g fat) were found in birds with signs of intoxication (reduced fertility and deformed chicks). In pig farms no sample of the originally contaminated feed manufactured in January could be retrieved, explaining why in Table 1 concentrations of PCBs appear lower in pig

than in poultry feed. Pig farms however have presumably received feeds as contaminated as feeds delivered to chicken farms as evidenced by the PCB concentrations in pig and poultry animals. Surprisingly, despite similar levels of PCBs, pigs showed much lower dioxin concentrations than chickens, the highest values not exceeding 40 pg TEQ/g. No abnormality which could be related a PCB contamination was observed in pig farms. In the bovine livestock, the few samples positive for the PCB test corresponded to meat of dairy and culled cows. The concentrations of PCBs and dioxins in these samples were however much lower than in contaminated pig and poultry animals. In milk, PCB and dioxin concentrations were still lower and were within the range of normal background levels in Belgium and Europe. These observations indicate thus that dairy cows and dairy products were spared by the incident. The concentrations of dioxin-like PCBs were determined in five contaminated samples, two of eggs (dioxin values : 53 and 18 pg TEQ/g fat) and three of chicken feed (dioxin values : 301, 288 and 315 pg TEQ/g fat). The addition of dioxin-like PCBs increases the TEQ values of the egg samples on average by 5.3 and that of feeds by 2.9.

*PCBs and PCDD/Fs in animal feeds***.** The PCB contamination peaked in feeds produced end of January attaining a mean PCB level of about 300 µg/g fat. Between February and March 1999, PCB level in feed rapidly dropped and mid-April, no sample of feed exceeded the PCB tolerance level of 1 µg/g fat. During this period, PCB and dioxin profiles in all contaminated feeds delivered to chicken or pig farms were remarkably similar. It is also interesting to note that the proportions of the PCB 52 and 101 congeners were fairly constant. Since these two congeners are labile in the livestock, this allowed us to rule out the possibility of secondary contamination due to the recycling of fats from contaminated animals. The concentrations of dioxins and PCBs in feeds were very closely correlated, with an average PCBs:dioxins ratio of about 50,000. No difference was found between poultry and pig feeds in their mean PCBs:dioxins ratio. The PCB profiles in poultry and pig feeds were matched to a mixture of Aroclors 1260/1254 (or of commercial PCBs with similar compositions) in the proportion of 75/25 %. Because of its high content in penta- and hexa-chlorinated congeners (more than 60 %), this mixture should have a resinous/waxy appearance, similar to that of a frying oil.

Amounts of PCBs and dioxins at the origin of the contamination. The total amounts of PCBs (sum of 7 markers), dioxins (17 PCDD/Fs congeners) and of dioxin-like PCBs accidentally mixed with the recycled fat in January were estimated at about 50 Kg, 1 g TEQ and 2 g TEQ, respectively. These figures were obtained by extrapolating the mean concentration of PCBs and dioxins in the fat of the most contaminated poultry feeds (end of January) to the total volume of contaminated fat (60 tons), assuming a two-fold dilution of the original PCB and dioxin concentrations with the vegetable fat used in the production of feeds. For estimating the amount of dioxin-like PCBs, we used the ratio of three as derived from the analysis of three contaminated feed samples (see above). Since the sum of the seven PCB markers represents theoretically 30 % of the total weight of all PCB congeners in a mixture 75/25 % of Aroclors 1260/1254 (or of commercial PCBs with similar compositions), we estimated the total amount of PCBs at the origin of the incident at approximately 150 kg (sum of all congeners), which corresponds to a volume of about 100 liters of PCB oil (density 1.60 g/cm3).

Poultry. The patterns of PCBs were very consistent in all poultry products, the fingerprint being transferred almost unchanged from hens to eggs and from eggs to chicks. Compared to the original pattern in feed, the patterns in poultry were characterized by the disappearance of the PCB 52 and 101 congeners as a result of the preferential biotransformation of these lower chlorinated congeners. The PCDD/Fs patterns in poultry feed, chickens, hens, chicks and brooded eggs were almost indistinguishable, indicating a very limited biotransformation of dioxins in these birds. As for PCBs, the dioxin patterns were transferred almost intact from the hens to the eggs and then to the chicks. Laying hens and consumption eggs presented by contrast quite different dioxin profiles with a predominance of OCDD and OCDF. These profiles, especially those in consumption eggs, exhibited a typically environmental fingerprint which was unaltered by the PCB incident. The concentrations of dioxins and PCBs in poultry meat and in eggs were highly correlated, with slopes or PCBs:dioxins ratios almost identical to that in feeds. These correlations allowed to validate the PCB test used to trace the contamination in the food chain.

Pig products. By comparison with the pattern in the contaminated feed, the metabolic alteration of PCB patterns in pigs was still more pronounced than in poultry, leading to the disappearance of PCB 118 in addition to the PCB 52 and PCB 101 congeners. Of the seven PCB markers measured, only the three most chlorinated congeners were still present in significant proportions in pig meat. The dioxin patterns in pigs were very different from that in feeds, containing only a few persistent congeners (OCDD and 1,2,3,4, 7, 8- HxCDF). If one compares these profiles with

those reported in the literature in pigs exposed to the background pollution, the only difference reminiscence of a PCB contamination was the presence of HxCDF in a relatively high proportions. The concentrations of dioxins and PCBs in pigs were significantly correlated but through a non-linear relationship contrasting sharply with the perfectly linear relationships observed in contaminated feeds and in poultry products. The concentration of dioxins in pig meat increased with that of PCB up to value PCB level of 1 µg/g fat. Above this value, the dioxin concentrations leveled off despite very high concentrations of PCBs. As a corollary, the PCBs:dioxins ratio in pigs showed extremely variable values extending from 50,000 up to 10,000,000.

Bovine products. The patterns of PCBs in the contaminated bovine meat (dairy or culled cows) from suspected farms were similar to found in PCB-positive samples of bovine meat (dairy cows) originating from unsuspected farms. Dioxin patterns in dairy cow meat were not different between suspected and unsuspected farms and they exhibited fingerprints corresponding to the background environmental contamination. The dioxin profile characterizing the PCB incident was found only in a few meat samples from culled cows. These cows also had the highest levels of PCBs presumably because of the lack of milk production. The PCB patterns in the few milk samples exceeding the tolerance level were indistinguishable from the patterns in samples with normal PCB levels and also from the patterns reported previously for human and cow's milk in Belgium. The proportion of PCDFs in these samples was increased but this change cannot be interpreted as a reflection of PCB contamination since the same profile was found also in about 60 % of PCB-negative milk samples. At the exception of culled cow meat, the bovine livestock appears thus to have been largely spared by the PCB incident since the levels and patterns of PCBs in bovine products from suspected farms were not different from those associated with the background environmental pollution by these substances.

PCBs:dioxins ratio in the Belgian food chain. The PCBs:dioxins ratio in the PCB oil at the origin of the Belgian incident has been estimated at 50,000. This is a relatively low value reflecting an important thermal degradation of the oil. This ratio was found in all contaminated feeds delivered to pig or poultry farms, suggesting an unique source of contamination since of course biotransformation is unlikely to occur in recycled fat. By contrast, the PCBs:dioxins ratio presented substantial variations between animal species. The ratio was fairly constant in most contaminated poultry products, varying between 47,848 in chicks and 71,563 in brooded eggs. A somewhat higher ratio was observed in consumption eggs (182,238) probably because these products were unaffected by the incident. In all pig animals, by contrast, the PCBs:dioxins ratio was dramatically increased, reaching values more than to 100 times higher than that found in the feed.

Risk assessment. PCBs and dioxins are cumulative toxins which may produce toxic effects when critical concentrations are reached in target tissues. To assess the potential health risks, we have thus calculated the increase of body burden that might be achieved from the consumption of the most contaminated products observed during the incident. Of the whole database, the highest PCB concentrations in foodstuff were found in samples of poultry, and in particular in chicken meat (56.9 µg/g fat) and in chicks with edema disease (47.1 µg/g fat) (Table 1). The worst case scenario would thus consist to regularly consume foods with such levels of contamination. For a reliable estimate, we used the arithmetic means of the three highest concentrations of dioxins and PCBs found in chicken meat (50 µg/g fat and 1,000 pg TEQ/g fat, respectively). If one considers the case of a person eating 200 g of chicken (5 % fat taking into account the loss of fat during cooking), this leads to an intake of about 500 µg PCBs and 10,000 pg TEQ dioxins. Assuming that the average body burden of PCBs and dioxins by the young adult population in Belgium is respectively around 5 mg and 200,000 pg TEQ, this intake would result in a doubling of the PCB and dioxin body burden after 10 and 20 contaminated meals, respectively. Such a doubling of the body burden would mean going back to the levels of PCBs and dioxins which were those in the 80s and a further increase by a factor 3 to 4 would bring the body burden to the levels probably prevailing in the 70s or of populations eating regularly contaminated fish from polluted seas.

The probability that some individuals could have experienced this worst case scenario depends both on the duration of the contamination episode and on the proportion of the food chain really contaminated during that period. As shown by the time course of PCB levels in animal feeds, the contamination has been limited to the period of January 20 to March 15. After that time, the most contaminated part of the food chain (chickens) had probably been consumed and only pigs or their offspring could still contain elevated levels PCBs (but not of dioxins which had been largely eliminated). According to the veterinary inspector (Dr Destickere), about 40,000 reproduction hens and one million chickens were contaminated in farms affected by the incident. This estimate is in good accordance with

the maximal number of chickens (also around one million) which theoretically can be contaminated to a PCB level of 50 µg/g fat and a dioxin level of 1,000 pg TEQ/g fat with a total of 50 kg PCBs (seven markers) and 1 g dioxins (of which 80% were delivered to chicken farms). These numbers represent 2 % of the total number of chickens produced in Belgium in February-March, an estimate which fits well with the proportion of chicken farms where samples positive for the PCB test were discovered (1.98 %). Under these conditions, it appears extremely improbable that a person could have consumed these most contaminated chickens a sufficient number of time to significantly increase his body burden. The only situation in which such a scenario could be conceivable is that of individuals who would have consumed contaminated products from a single farm but even in that case, the time factor allows to exclude any serious health effects even in pregnant women who are the subjects the most at risk.

DISCUSSION

The Belgian PCB incident was almost an exact replica of the poultry poisoning episodes which repeatedly occurred in the 50s and 60s in the USA and Japan (1). Like in these earlier incidents, the alert was given by laying hens which showed a sudden drop in egg production and a few weeks later by the chicks which developed the clinical manifestations of the chick edema disease. The incident most probably would have never been detected if the contaminated fat had been used only in the production of feeds for pigs or bovines. These animals indeed did not show any sign of intoxication and the levels of dioxin in cow's milk (the only foodstuff regularly monitored for dioxins in Belgium at that time) were not increased by the incident. The possibility of a dioxin contamination was envisaged only in March after having tested unsuccessfully a series of other hypotheses. In April, when high concentrations of dioxins were discovered in dead birds and their feed, most of the contaminated poultry had already been destroyed or consumed. End of May, when the incident was revealed to the public triggering a major political and food crisis, saws and their offspring were the only animals to contain still elevated levels of PCBs. These animals, however, had already eliminated most of their dioxin body burden. Paradoxically thus, the dioxin contamination was almost over when the dioxin-contaminated food scare broke out in Belgium and spread all over Europe and even the world.

The PCB oil at the origin of the accident was inadvertently introduced in the food chain via the recycling of oils and fats collected in public containers parks, a practice which was forbidden in Belgium in June 1999. The total volume and the characteristics of this PCB oil could be determined with a certain precision thanks to the analysis of originally contaminated feed samples which could be retrieved in chickens farms. By extrapolating the PCB and dioxin concentrations in these samples to the volume of the contaminated tank, we estimated the total amount of PCBs mixed with animals feeds to about 50 kg PCBs (seven markers) or about 150 kg total PCBs, which corresponds to about 100 liters of PCB oil. The pattern of PCB congeners in the contaminated fat was matched to a mixture of Aroclors 1260/1254 (or of similar commercial PCBs) in the proportions of 75/25. In view of the widespread use in the past of a few commercial PCB mixtures, one can conceive that different sources of PCB oil could by chance present the same profiles of PCBs but it is extremely unlikely that these sources could also present exactly the same extent of thermal degradation as reflected by the PCBs:dioxins ratio. The consistency of both the PCB profiles and the PCBs:dioxins ratio in all contaminated feeds allowed us to conclude that the incident was caused by an unique source of PCB oil which peaked at the end of January and progressively resolved Mid-March 1999. The relatively slow disappearance of PCBs from animal feeds most likely results from the well-known phenomenon of carry-over and memory effect in the transportation (trucks) and production (tanks) facilities. The possibility that the contamination could be have been perpetuated by the recycling of fats from contaminated animals can be formally ruled out on the basis of the PCBs patterns in feeds which showed fairly constant proportions of the labile PCB 52 and 101 congeners during the whole contamination period.

The study of PCB and dioxins patterns revealed interesting differences in the metabolism and elimination of these compounds by farm animals. The patterns of both contaminants were remarkably preserved all along the poultry chain from the hens to the eggs and from the eggs to the chicks. The incapacity of birds to metabolize these polyhalogenated hydrocarbons probably explains the great sensitivity of their embryos which during brooding are exposed to increasing concentrations of these lipophilic pollutants. The most interesting observation however was made when comparing the fates of PCBs and dioxins in pigs. These animals which were killed more than six months after the incident had still elevated levels of PCBs but surprisingly they were almost free of dioxins. Since the PCB fingerprints and the PCBs:dioxins ratios in pig feeds were identical to that in poultry feeds, the only logical way to

explain this discrepancy was to postulate a faster elimination of dioxins by the pigs, which could manifest itself more easily as these animals or their offspring were sacrificed several months after the peak of exposure. Several observations support this interpretation : (i) the different shapes of the relationships between PCBs and dioxins between the two species. In pigs, the PCBs/dioxins relationship was not linear but evoked a saturation kinetics highly suggestive of a faster elimination of dioxins than PCBs. Since PCB mixtures (e.g. Aroclors 1254 and 1260) are classical inducers of the xenobiotics biotransformation and in particular of the glucuroconjugaison, one attractive hypothesis would be that the biotransformation of dioxins in pigs has been progressively induced by the rise of the PCB body burden, explaining the shape of the relationship between both contaminants (20); (ii) as reported by Liem and Theelen (9), the liver to fat ratio of dioxins is about one order of magnitude higher in pigs than in chickens, suggesting also a more efficient hepatic metabolism of dioxins in pigs compared to chickens ; (iii) the alterations of patterns of PCBs and dioxins were much more pronounced in pigs than in chickens, confirming again the greater metabolic potential of mammals compared to birds.

The health risk has been a matter of a hot debate in Belgium during the crisis and even after when several research teams proposed to the authorities to undertake large scale epidemiological studies on the possible health outcomes of the incident. The concern was particularly focused on pregnant women in view of the poorly characterized developmental effects of dioxin-like PCBs. The present analysis indicates that the contamination has not only been limited in time but also has affected a very small fraction of the food chain. This dispersion of the contamination in the whole food chain has made almost impossible the identification of individuals who have consumed contaminated foods. The risk assessment is also further complicated by the lack of information on the PCB and dioxin body burden of the general population of Belgium before the incident. In view of these uncertainties, we have limited the risk assessment to the estimate of the likelihood that some individuals could have increased their PCB and dioxin body by consuming the most contaminated products. We calculated that, in the worst case scenario, a doubling of the PCB and dioxin body burden could have been reached after having consumed respectively 10 and 20 meals of the most contaminated chickens. In the case of PCBs, this estimate is valid whether PCBs are expressed as total PCBs, the seven PCB markers or as dioxin-like PCBs. In proceeding with the analysis, we realize now that this scenario, which was envisaged in the heart of the dioxin crisis, was quite improbable at the population level in view of the proportion of chickens on the food market which could really be contaminated (around 2 %). Such a scenario was conceivable only for individuals like farmers who would have consumed contaminated products from a single farm. But even in this case, a doubling of their PCB or dioxin body burden would have brought them at contamination levels similar to that of fish consumers or of people who were living in the 80s. This conclusion is in accordance with other evaluations and also with the view shared by most scientists and international bodies that for cumulative toxins like dioxins or PCBs, a short excursion above the TDI like that observed in Belgium is not consequential provided the integrated dose remains largely below the critical body burden.

After the development of the bovine spongiform encephalopathy in the UK, the PCB/dioxin contamination of poultry and pig products in Belgium is the second major food crisis in Europe due to recycling practices upstream the food chains. If these practices are beneficial for the environment and the energetic yield of the agriculture, they may accidentally or chronically contaminate the food chains by a variety of polyhalogenated hydrocarbons and other persistent lipophilic contaminants. There is thus an urgent need for food safety and public health to carefully evaluate the risks inherent to such practices and to implement monitoring programs for animal feeds. Another important lesson learned from the Belgian incident is the need to carefully analyze the fingerprints of dioxins and PCBs when a case of contamination is discovered. This analysis may indeed provide crucial information to identify the source of the contamination and also to predict its transmission along the food chain and its possible impact on public health.

REFERENCES AND NOTES

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

Table 1 Concentrations of PCBs and dioxins (PCDD/Fs) in PCB-positive samples originating from farms suspected of having received contaminated feeds during the Belgian PCB incident.

Type of sample	N (PCBs)	PCBs (ng/g fat) [range]	N (PCDD/Fs)	PCDD/Fs (pg TEQ/g fat) [range]
Pig feed	11	4,258 [809-14,154]	3	180.2 [73-301.4]
Poultry feed	20	14,996 [832-452,836]	12	231.5 [20.1-11,143]
Chicken	15	3,409 [1,010-56,856]	4	255.4 [15.8-2,613]
Laying hen	14	889 [234-3,868]	3	3.7 [2.6-6.95]
Chick	5	8,160 [2,721-47,101]	5	170.5 [54.4-965.4]
Hen	13	5,434 [2,549-22,637]	1	463.3
Brooded eggs	23	2,852 [510-38,890]	9	44.9 [1.0-713.1]
Whole Eggs	23	839 [515-1,631]	17	3.8 [1.2-19.6]
Pig	94	2,928 [1,188-15,080]	48	0.9 [0.03-36.25]
Young pig	9	2,957 [1,040-25,472]	4	0.8 [0.38-2.7]
Saw	60	6,688 [654-17,271]	40	2.1 [0.08-23.82]
Bovine [1]	12	487 [246-1,060]	9	5.81 [3.6-13.2]
Milk	55	25 [6-160]	54	2.07 [1.09-6.0]
TOTAL	355		209	

[1] includes dairy and culled cows.