ORIGINAL ARTICLE

Considerations for the regulation of polychlorinated dibenzodioxins, furans (PCDD/Fs) and biphenyls (PCBs) in liver

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Abstract

Introduction There is increasing evidence that liver produced from farm animals such as cows, pigs and sheep may breach European Union regulatory limits for dioxin concentrations even when the livestock are given compliant animal feed and are exposed only to normal background levels of dioxin contamination in the environment. The dioxin concentrations in other commonly consumed tissues such as muscle, fat, kidney, etc., from the same animals will typically be well within regulatory limits. When dioxin limits were initially proposed and eventually established by the European Union, almost a decade ago, they were based on a small data set available at the time, and were expressed on the basis of concentrations in fat in common with limits for other animal products (except for fish which were set on a whole weight basis because of seasonal and other variations in the lipid content of this species). Data generated for liver since the regulations came into force, suggest that dioxin concentrations in liver from certain types of animal are higher than were anticipated. *Objectives* We set out to establish whether liver on retail sale produced using normal farming practices was likely to contain dioxins and polychlorinated biphenyls above regulatory limits and to consider whether or not the regulations were appropriate. *Method* We did this by analysing samples of liver obtained from retail outlets. *Results and Conclusion* Nine of the 22 ovine (sheep) liver analysed and reported here exceeded the European Union limit for liver and all 10 of the venison liver exceeded this value, although venison liver is not covered by the regulation. The high cost of compliance monitoring, together with the likely proportion of non-compliances, could amount to a *de facto* ban on the sale of ovine (sheep) liver even in the absence of a risk to consumer health. The preferred solution would be to revise the existing dioxin and total TEQ limits set out in Commission Regulation (European Commission) No. 1881/2006 to reflect the levels that are normally found, with the addition of precautionary advice to consumers if this is considered necessary.

Introduction

Offal refers to any edible part of an animal, other than the skeletal muscle or bone. Thus offal can potentially include the liver, heart, kidneys, blood, sweetbreads (thymus glands and pancreas), fries (testes), lungs, intestines, brains, tripe or stomach lining, feet, tongue, tail, scrotum, head and eyeballs. In the United Kingdom, offal is widely sold on its own as liver, kidney, tongue, heart, etc, or processed into products such as pates, pies and sausages including black pudding. The animal species most commonly used are sheep, pigs, chickens, oxen and calves. Between 1982 and

1996 the annual consumption of offal from all species by the UK human population declined from 2.2 to 1.7 kg per person (Meat and Livestock Commission, 1997) and the decline in consumption has probably continued further since then. These figures are averages across the whole population. Consumption by high-consuming individuals is likely to be significantly higher.

Some offals, particularly the liver, have been reported to contain relatively high levels of dioxins and polychlorinated biphenyls (PCBs) (Liem & Theelen, 1997; Rose *et al.*, 2005). Animal studies have shown that, following oral exposure, dioxins were distributed via the blood to organs such as the liver and adipose tissue (Olson, 2003). When rodents were exposed to increasing doses of 2,3,7,8-tetrachlorodibnzo-*p*-dioxin, preferential accumulation occurred in the microsomal fraction of the liver, such that the concentration exceeded that in adipose tissue (Diliberto *et al.*, 1996). This 'hepatic sequestration' is observed for rodents under test conditions, but even in practice, comparisons of dioxin levels in tissues of food animals, such as sheep and pigs show higher levels in the liver (Liem & Theelen, 1997; Rose *et al.*, 2005). There is very little information on dioxin and PCB levels in other commonly consumed offals, but the occurrence of the contaminants in these organs is likely to be associated with the fat content of the tissues. The few studies where concentrations in liver and kidney have been measured showed relatively lower levels in kidneys. This accords well with organ functionality as the liver processes dietary fats, which are likely to be relatively high level sources of lipophilic contaminants compared with kidneys which separate urea, mineral salts, toxins and other waste products from the blood before excretion and have mineral balance and endocrine functions.

In 2002, the European Commission introduced regulatory limits for dioxins in a range of food types, including carcass meat and liver (European Commission, 2001). Limits were established taking into account available data and, for both meat and liver, the limits were expressed on a fat basis. Limits for fish were set on a whole weight basis because of seasonal and other effects on changes of lipid concentration in fish species. The limit for animal liver was established on the basis of the results for a small number of samples that were available at the time, covering all animals. Action limits were set at a lower value than maximum limits, and these should trigger investigation of the source of contamination. Target levels are lower still and are set on the basis of toxicological information along with a safety margin to ensure exposure from the diet would be within an acceptable risk tolerance.

Although a number of studies in different countries on the dioxin and PCB contents of different foods have been published (Hallikainen & Vartiainen, 1997; Domingo *et al.*, 1999; Liem, 1999; Tsutsumi *et al.*, 2001; Bocio *et al.*, 2002; Cuervo *et al.*, 2002; Baars *et al.*, 2004; Fernandes *et al.*, 2004a; Kiviranta *et al.*, 2004), very few reported levels in offal. The UK total diet studies carried out over the last two decades (Fernandes *et al.*, 2004a), showed that offal has consistently been the highest dioxin and PCB containing food group. During the 2001 foot and mouth disease outbreak in the United Kingdom, samples of food produced around the pyres used to incinerate culled livestock were analysed to see if the relatively uncontrolled combustion had increased concentrations of dioxins (Rose *et al.*, 2005). There was no evidence for such contamination but it was observed that dioxins in some samples of animal liver exceeded the regulatory limits. A subsequent research project on farm animals, kept under controlled but standard farm conditions, investigated the relative contribution of different sources of exposure to dioxins and PCBs. The results showed that, even when provided with animal feed well within regulatory limits, concentrations of these compounds in liver can exceed the regulatory limits (Foxall *et al.*, 2004).

In order to investigate these anomalies further, a survey for dioxins and PCBs in offal was carried out in the United Kingdom during 2005 and 2006, which included liver of cows, lambs, pigs, chickens and venison (mainly red deer), as well as kidney, heart and other offal-based food products (Fernandes *et al.*, 2010).

Materials and methods

Samples of offal, including 22 livers of lambs and two of venison, were obtained from randomly selected retail outlets throughout the United Kingdom between April and June 2005. An additional eight samples of venison liver from different geographical locations were obtained from a Scottish retail outlet in March 2006.

The analytical methodology used was based on that reported previously (Fernandes *et al.*, 2004b). Briefly, the sample was freeze-dried and ground, and thoroughly homogenized. An aliquot was fortified with known amounts of surrogate ($^{13}C_{12}$-labelled) analogues of target analytes and was exhaustively extracted using mixed organic solvents. The extract was purified by acid hydrolysis followed by adsorption chromatography. *Ortho*-PCBs, non-*ortho*-PCBs and polychlorinated dibenzodioxins and furans (PCDD/Fs) were segregated into three separate fractions. Each fraction

was concentrated and further cleaned up before the inclusion of additional $^{13}C_{12}$-labelled internal sensitivity standards. Final determination was by high-resolution gas chromatography with either low-resolution mass spectrometric detection (*ortho*-PCBs) or high-resolution mass spectrometric detection (non-*ortho*-PCBs and PCDD/Fs). All 29 PCDD/Fs and PCBs assigned a WHO-TEF, the ICES seven PCBs and some additional PCBs were measured. All analytical data were assessed for compliance with published acceptance criteria, (Ambidge *et al.*, 1990), the analytical quality assurance criteria prescribed in European Union (EU) legislation for control of these contaminants in food (European Commission, 2002), and the demands of ISO 17025 accreditation. Further validation and quality assessments were made by participation in interlaboratory comparison exercises including those run by the Folkehelsa Institute for Public Health in Norway (e.g. National Institute of Public Health, 2001).

Results and discussion

Because of the very large volume of data on individual congeners produced in the studies, only summarized versions based on upper-bound WHO-TEQ are presented here (Table 1). Notably, high results were reported for the dioxin levels in 11 out of 22 samples of sheep/lambs' liver. These included nine that were above the existing EU limit of 6.0 pg WHO-TEQ g^{-1} fat. A further two samples were above the EU action level of 4.0 pg WHO-TEQ g^{-1} fat. In the case of venison, to which the regulations do not apply, both of the samples tested were significantly above 6.0 pg WHO-TEQ g^{-1} fat. In the light of the results for venison liver, a further eight samples were obtained.

More detailed results, including fat content, are shown in Tables 2a and b. From an inspection of the data for lamb, there are several important points of note. Firstly, about half of the lamb liver samples contained very low levels of dioxin

Table 1 Summary of results for liver samples tested

Sample	Total number tested	Number over European Union action level	Number over European Union limit
Calf/veal	5	1	0
Chicken/poultry	14	0	0
Lamb	22	2	9
Ox	7	1	0
Pig	21	2	0
Venison*	10	0	(10)
Total	79	6	9 (19)

*Not covered by Commission Regulation 466/2001.

with levels in the range 0.24–1.5 and 0.03–0.12 pg WHO-TEQ g^{-1} on a fat and a fresh weight basis, respectively. The dioxin contents of the remainder ranged from 2.8 to 25 pg WHO-TEQ g^{-1} (fat) and 0.28 to 1.2 pg WHO-TEQ g^{-1} (whole weight). The fat content of the samples ranged from 3.6% to 11.1%. There was no clear relationship between the fat levels and the whole-weight dioxin concentrations. The samples were selected at random and information is very limited but it is possible that the data reflect different ages or classes (husbandry systems) of livestock.

The analytical method involves extracting the dioxins and PCBs together with the fat present in the sample into lipid solvents and expressing the result as dioxins and PCBs per mass of fat. As a result, a liver sample containing a lower level of fat will appear to have a higher fat-based concentration of dioxins and PCBs than a liver containing a similar amount of dioxins and PCBs but with a higher fat content. For muscle (meat), strong correlation is expected between the fat content of the sample and the whole weight dioxin concentration because dioxin is associated with the fat present in the sample but in this survey there was no correlation between fat content and dioxin content of livers. This was the case whether the dioxin concentration was expressed on a fat or a fresh weight basis. The results suggest that, as for fish, dioxins limits should be related to the weight of the food product rather than to fat content, which would be more meaningful when estimating intakes and assessing risk to consumers.

For carcass meat of most common food animals, the dioxin to dioxin-like PCB ratio is normally between 1 and 2. In this survey, the dioxin to dioxin-like PCB ratio in all of the lamb liver samples was very high, ranging from 1.2 to 11.8, with an average of 4.0. Similar observations can be made for venison liver, which was largely from red deer, although the dioxin and PCB levels were generally higher. This higher ratio may be an indicator of differences in source contamination ratios or differences in husbandry or lipid metabolism for lambs and deer. The possibility that high results were due to high levels of localized contamination was ruled out because not only were the samples obtained from a wide range of UK sources but also there were significant differences in the individual congener profiles.

Age, diet and environment may all affect dioxins and PCB accumulation in liver. The liver is a target organ for dioxins and PCBs. Dioxins and dioxin-like compounds bind to aryl hydrocarbon receptor (AHR) sites, which are largely present in the liver (Gasiewcz & Park, 2003). Increased exposure to dioxins and PCBs within the liver would be expected to induce the generation of greater amounts of these proteins

Table 2 Results for liver (*n* = 32): (a) lamb liver and (b) venison liver

	pg WHO-TEQ g⁻¹, fat		pg WHO-TEQ g⁻¹, whole weight			pg WHO-TEQ g⁻¹, fat		pg WHO-TEQ g⁻¹, whole weight			pg WHO-TEQ g⁻¹, fat		pg WHO-TEQ g⁻¹, whole weight	
Fat (%)	Dioxin	PCB	Dioxin	PCB	Fat (%)	Dioxin	PCB	Dioxin	PCB	Fat (%)	Dioxin	PCB	Dioxin	PCB
(a) Lamb liver (n = 22)										*(b) Venison liver (n = 10)*				
11.10	0.24	0.12	0.03	0.01	7.10	4.0	0.75	0.28	0.05	2.6	109	50	2.8	1.3
9.00	0.35	0.26	0.03	0.02	7.50	5.2	1.0	0.39	0.07	3.8	98	22	3.7	0.83
6.30	0.54	0.46	0.03	0.03	5.70	7.0	1.8	0.40	0.10	3.7	92	19	3.4	0.71
6.40	0.65	0.42	0.04	0.03	5.20	7.5	1.8	0.39	0.09	4.0	64	14	2.6	0.56
7.40	0.66	0.38	0.05	0.03	4.60	9.1	2.0	0.42	0.09	4.1	45	17	1.9	0.69
7.80	0.92	0.50	0.07	0.04	8.30	9.8	2.3	0.81	0.19	3.3	32	17	1.0	0.54
4.30	0.93	0.22	0.04	0.01	5.70	10	2.2	0.60	0.12	3.4	24	5.9	0.8	0.20
7.90	0.95	0.41	0.08	0.03	6.40	13	2.3	0.82	0.16	4.5	24	7.2	1.1	0.32
7.90	1.5	0.59	0.12	0.04	5.80	14	2.9	0.78	0.17	3.7	20	5.4	0.74	0.19
3.60	1.5	0.34	0.05	0.01	4.40	20	1.7	0.88	0.07	4.1	13	3.7	0.53	0.15
4.10	2.8	1.4	0.11	0.06	4.90	25	3.2	1.2	0.16					

PCB, polychlorinated biphenyls.

(Gasiewcz & Park, 2003) and consequently to increase the dioxins and PCBs concentrations bound in liver. Dioxins have a higher affinity for AHRs than do PCBs (as reflected in their Toxic Equivalency Factors), but this is likely to be similar in all ruminant species. Therefore the difference in dioxins:PCB ratios in lambs and deer livers may be a result of the lipid content or lipid metabolism in these animals. Cattle, sheep and deer are all ruminants with similar lipid metabolism but the amounts of carcass fat are significantly different in different classes of livestock. In the United Kingdom, the typical ages at which meat animals are slaughtered are broiler poultry at around 6 weeks, pigs at around 5–6 months, beef cattle (apart from veal calves) and deer typically at between 1 and 3 years and lambs from about 3 months of age (Easter/spring lambs) up to about a year (hoggets). Culled ewes (mostly disposed of as mutton or halal meat) and culled cows are likely to be at least 3 years old and may be much older. Broiler chickens, Easter lambs and pigs are relatively lean because they are young and immature. Fat accumulation increases dramatically in all species after bone and muscle growth have developed (Halley & Soffe, 1988). Venison is a very lean meat and deer carcasses are likely to have relatively low fat content compared with cattle and sheep. A low carcase fat content may affect the partitioning of dioxins and PCBs between liver AHR and adipose tissues, effectively increasing exposure within the liver and possibly inducing synthesis of AHR and increasing the dioxins and PCB concentrations in livers. The relatively high level exposure of AHR within the liver may also affect the ratio of dioxins to PCBs bound to AHR. As the livers of poultry and pigs contained relatively low concentrations of dioxins and PCBs the maturity at slaugh-

ter was not the critical risk factor, although it may have contributed together with other factors especially diet and environment.

Most pigs and poultry are fed on cereals supplemented with soya bean, but free-range systems may cause significant exposure to the environment and to soil. Deer would be expected to be mostly or entirely grass fed, depending on whether they were free living or farmed. Most beef is produced using a mixture of grass, conserved forages and concentrates but the most expensive beef is produced from milk grass and conserved forage only. Most breeding ewes rely on grass or conserved forage for most of the year with concentrates only fed in late pregnancy and early lactation. Spring lambs have been fed predominantly on their mother's milk, probably with a supplement of concentrate feed and a relatively small proportion of grass. Older lambs and hoggets will have relied increasingly on grass and other compounded ('concentrate') feeds based on commodities such as cereals and soya. Cull dairy cows will have probably been fed approximately half their diet as concentrates and half as grass or other forage. Human breast milk is known to be a very significant source of dioxin to human infants because humans accumulate dioxins in depot fat over many years and release this during lactation. There is no representative data for dioxins in sheep or cows' milk, although regular milking would be expected to lead to significant reductions, as is reflected in data for retail samples (Food Standards Agency, 2007). Soil ingestion is a potential pathway for dioxins and PCBs. Involuntary soil intakes of up to 18% of total dietary dry matter in cattle and 30% in sheep have been recorded (Thornton & Abrahams, 1983) and grazing animals are expected to ingest at least 10% of their

diet dry matter as soil. Pasture conditions, especially sward density and sward height and husbandry practices, especially stocking rates and housed period, all affect the ingestion of soil. The range of dioxin and PCB contamination in soils is very large but some soils can be expected to contain significant amounts (Creaser *et al.*, 1990) and will be affected by the geographical area, proximity of industry and terrain (e.g. upland or river valley). The results suggested that the high liver concentrations were not caused directly by dioxins and PCBs hotspots but classes of livestock ingesting relatively high soil intakes of soils with levels of dioxin/PCB contamination within expected background that will have relatively high exposures and may accumulate relatively high concentrations.

While exceedances of existing regulatory limits was of concern, the most important considerations were, firstly, to assess the risk to consumers and, secondly, to determine whether the limit had been set in an appropriate manner in view of the proportion of lamb's liver samples close to or above that limit. In addition, it was essential to consider the implication for consumers of venison liver, for which results as high as 160 pg WHO-TEQ g^{-1} of fat had been reported. Intake estimates based on the samples containing the highest concentrations of dioxin and dioxin-like PCBs are shown in Table 3. The data show the effect that the consumption of one or two 100 g portions of liver weekly has on the average daily intake for an adult when taking into account exposure from the rest of the diet. These figures indicate that consumption of lambs' liver is likely to have very little impact on overall dietary exposure to dioxins. In the case of venison liver, for which the highest whole weight total TEQ concentrations are similar to those of oily fish, frequent consumption could lead to an exceedance of the tolerable daily intake of 2.0 pg WHO-TEQ kg^{-1} bodyweight but dietary intakes of deer liver are very unlikely to be high for most of the population.

In light of these results, it is apparent that the existing limits of 6.0 pg WHO-TEQ g^{-1} fat for dioxins and 12.0 pg g^{-1} fat for dioxins plus dioxin-like PCBs may need to be revised. On the basis of available data, it should be possible to set limits for lambs' liver expressed on a whole weight basis that continues to provide an adequate level of consumer protection. In the case of venison liver, it might be necessary to provide advice to consumers to limit consumption. However, it is of note that UK consumer advice already recommends that all liver consumption should be limited in order to avoid the excessive intake of vitamin A, which has been associated with possible foetal damage and also brittle bones in the elderly. This is also the view of the European Food Safety Authority.

The European Commission is currently reviewing limits for dioxins to take into account the new TEFs recommended by the WHO. It is likely that the limits for dioxins in liver will be updated and limits for venison liver may be established at the same time.

Conclusion

High results and high dioxins:PCB ratios were reported for a significant proportion of lamb and venison liver. Further investigations suggested that this was not due to poor husbandry practices or high localized contamination but was much more likely to be associated with the physiology of the animals. Subsequent data reported on dioxins in liver from the Irish Republic supports this view (Tlustos *et al.*, 2005). Targeted surveillance that takes account of age, class of livestock, husbandry systems and environments would assist the investigation of which of the several risk factors may be responsible for the high concentrations and unexpected dioxins:PCB ratios recorded for lambs and deer livers. Although concentrations in liver from venison were high, this is not widely consumed and is unlikely to pose a health risk for most consumers. Consideration should be given to include venison liver in the existing list of foods regulated for dioxins and PCBs, and whether or not these limits should be set on a whole weight basis. The latter approach may better reflect animal physiology with respect to disposition of dioxins in the liver, and would be in line with the policy of setting limits to remove only the most contaminated products from the food supply.

Acknowledgements

Table 3 Intake estimates (pg WHO-TEQ kg^{-1} bw day^{-1})

	Lamb					Venison		
Concentration in liver (pg WHO-TEQ g^{-1} whole weight)	1.4	1.00	0.98	0.95	0.72	4.56	4.14	3.12
Average daily intake from 1 weekly 100 g portion of liver	0.3	0.24	0.2	0.2	0.17	1.1	1.0	0.7
Total intake from typical diet plus one portion of this liver	1.2	1.1	1.1	1.1	1.0	1.9	1.8	1.6
Average daily intake from 2 weekly portions of liver	0.7	0.48	0.5	0.5	0.34	2.2	2.0	1.5
Total intake from typical diet plus two portions of this liver	1.5	1.3	1.3	1.3	1.2	3.0	2.8	2.3

Intake from non-offal part of the diet = 0.8.

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References

Ambidge P., Cox E.A., Creaser C.S., Greenberg M., Gem M.G., Gilbert J., Jones P.W., Kibblewhite M.G., Levy J., Lisseter S.G., Meredith T.J., Smith L., Smith P., Startin J.R., Stenhouse I., Whitworth M. (1990) Acceptance criteria for analytical data on polychlorinated dibenzo-*p*-dioxins and polychlorinated dibenzofurans. *Chemosphere*, **21**, 999–1006.

Baars A.J., Bakker M.I., Baumann R.A., Boon P.E., Freijer J.I., Hoogenboom L.A.P., Hoogerbrugge R., van Klaveren J.D., Liem A.K.D., Traag W.A., de Vries J. (2004) Dioxins, dioxin-like PCBs and non-dioxin-like PCBs in foodstuffs: occurrence and dietary intake in the Netherlands. *Toxicology Letters*, **151**, 51–61.

Bocio A., Llobet J.M., Domingo J.L., Casas C., Teixidó A., Müller L. (2002) Dietary intakes of dioxin and dioxin-like PCBs by the population of Catalonia, Spain. *Organohalogen Compounds*, **55**, 211–214.

Creaser C.S., Fernandes A.R., Harrad S.J., Cox E.A. (1990) Levels and sources of PCDDs and PCDFs in urban British soils. *Chemosphere*, **21**, 931–938.

Cuervo L., Jalon M., Rose M., Fernandes A., White S., Gonzalez de Galdeano L. (2002) Dietary Intakes of PCDDs, PCDFs and PCBs from total diet samples from the Basque Country (Spain). *Organohalogen Compounds*, **55**, 219–222.

Diliberto J.J., Jackson J.A., Birnbaum L.S. (1996) Comparison of 2,3,7,8-tetrachlorodibnzo-p-dioxin (TCDD) disposition following pulmonary, oral, dermal and parenteral exposures to rats. *Toxicology Applied Pharmacology*, **138**, 158–168.

Domingo J., Schuhmacher M., Granero S., Llobet J. (1999) PCDDs and PCDFs in food samples from Catalonia, Spain. An estimate of dietary intake. *Chemosphere*, **38**, 3517–3528.

European Commission. (2001) Council Regulation (EC) no. 2375/2001 of 29/11/2001 amending Commission Regulation no. 466/2001 setting maximum levels for certain contaminants in foodstuffs, *Official Journal of the European Communities*; **L 321**, 6 December 2001, 1–5. As amended.

European Commission. (2002) Council Directive 2002/70/EC of 26 July 2002 establishing requirements for the determination of levels of dioxins and dioxin-like PCBs in feedingstuffs. *Official Journal of the European Communities*, **L 209/15**, 2002, 15–21.

Fernandes A., Gallani B., Gem M., White S., Rose M. (2004a) Trends in the dioxin and PCB content of the UK diet. *Organohalogen Compounds*, **66**, 2053–2060.

Fernandes A., Mortimer D., Gem M., Rose M. (2010) The occurrence of dioxins and dioxin-like PCBs in offal and offal products (in preparation).

Fernandes A., White S., D'Silva K., Rose M. (2004b) Simultaneous determination of PCDDs, PCDFs, PCBs and PBDEs in food. *Talanta*, **63**, 1147–1155.

Food Standards Agency. (2007) Food Surveillance Information Sheet 04/07 Dioxins and dioxin-like PCBs in foods – EU monitoring 2006. Available at http://www.food.gov.uk/science/surveillance/fsisbranch2007/fsis0407 (accessed 05 January 2010).

Foxall C.D., Lovett A.A., Fernandes A.R., White S., Rose M., Donkin R., Dunning P., Farley A., Morton D., Nix C. (2004) Transfer and uptake of organic contaminants into meat and eggs of chickens, sheep and pigs. Report to the Food Standards Agency, UK.

Gasiewcz T., Park S. (2003) Ah receptor: involvement in toxic responses. Chapter 12. In: *Dioxins and Health, ISBN 0-471-43355-1*, 2nd edition, eds Schecter A., Gasiewcz T. Wiley, New York. pp. 491–532.

Halley R.J., Soffe R.J. (1988) *Primrose McConnell's The Agricultural Notebook, ISBN 0-408-03060-7*, 18th edition. Butterworths and Co, London.

Hallikainen A., Vartiainen T. (1997) Food control surveys of PCDDs and PCDFs and dietary estimates. *Food Additives and Contaminants*, **14**, 355–366.

Kiviranta H., Ovaskainen M., Vartiainen T. (2004) Market basket study on dietary intake of PCDD/Fs, PCBs and PBDEs in Finland. *Environment International*, **01209**, 1–10.

Liem A.K.D. (1999) Dioxins and dioxin-like PCBs in Foodstuffs. Levels and trends. *Organohalogen Compounds*, **44**, 1–4.

Liem D., Theelen R. (1997). *Dioxins: Chemical Analysis, Exposure and Risk Assessment, ISBN 90-393-2012-8*. Utrecht University, Utrecht, the Netherlands.

Meat and Livestock Commission. (1997). *Meat and Livestock Commission Beef Yearbook*. MLC, Milton Keynes, UK.

National Institute of Public Health. (2001). *Interlaboratory Comparison on Dioxins in Food – Second Round of an International Study, ISBN 82-7364-178-3*. Oslo, Norway.

Olson J. (2003). In: *Dioxins and Health, ISBN 0-471-43355-1* eds Schecter A., Gasiewicz T. Wiley Interscience, Hoboken, NJ, USA. pp. 191–246.

Rose M., Harrison N., Gem M., Fernandes A., White S., Duff M., Costley C., Leon I., Petch R.S., Holland J., Chapman A. (2005) Dioxins and polychlorinated biphenyls (PCDD/Fs and PCBs) in food from farms close to foot and mouth disease animal pyres. *Journal of Environment Monitoring*, **7**, 378–383.

Thornton I., Abrahams P. (1983) Soil ingestion – a major pathway of heavy metals into livestock grazing contaminated land. *The Science of the Total Environment*, **28**, 287–294.

Tlustos C., Pratt I., White S., Fernandes A., Rose M. (2005) Investigation into levels of PCDD/Fs, PCBs and PBDEs in Irish produce. *Organohalogen Compounds*, **67**, 1474–1477.

Tsutsumi T., Yanagi T., Nakamura M., Kono Y., Uchibe H., Iida T., Hori T., Nakagawa R., Tobiishi K., Matsuda R., Sasaki K., Toyoda M. (2001) Update of daily intake of of PCDDs, PCDFs and dioxin-like PCBs from food in Japan. *Chemosphere*, **45**, 1129–1137.