Dioxins and polychlorinated biphenyls contamination in poultry liver related to food safety – A review

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ABSTRACT

The present article reviews the most important scientific literature on dioxins and PCBs found in poultry liver and their relation with food safety and consumers' health. Dioxins and polychlorinated biphenyls (PCBs) are persistent organic pollutants with high chemical stability; they are lipophilic compounds and they are not destroyed by microbial, photochemical, chemical or thermal degradation. Dioxins and PCBs are ubiquitous environmental contaminants, which are present in all marine plant and animals, birds, mammals and humans and bioaccumulate through the food chain. In the last years, there has been concern regarding food contamination with different chemical substances and their effect on food safety. More particularly, at the end of the 20th and the beginning of the 21st century, there were a series of incidents related to dioxin and PCBs, which directly affected human or contaminated the soil and accumulated in feed and then in food products, such as poultry liver. It was shown in case of dioxin incident that tetra and penta-chlorinated congeners (the most toxic ones) accumulates selectively in poultry livers. maximal concentrations have been fixed in the European legislation for dioxins and PCBs in food from animal origin, in order to protect the consumer. Data about background poultry liver contamination are scarce and the few available show levels below the legal limit for dioxins, but data are still lacking for DL-PCBs.

1. Introduction

The European Legislation together with the increasing consumer's demands require the slaughterhouses' owners and processors of poultry meat to provide high quality and hygienically
safe products. Thus, careful monitoring should be imposed on all factors of the flow process, which could in any way affect the finished product, by applying the principle “from farm to fork”.

Meat inspection, in particular edible offal inspection, including liver, is one step of that monitoring. Indeed, the liver, a major organ involved in metabolic processes, is considered to be one of the most eloquent witness of any disturbance in the body, as it is the subject to different types of etiologic attacks: infectious, toxic, metabolic, nutritional and traumatic (Doneley, 2004).

For food from animal origin, one of the possible cause of exclusion from consumption because of a risk for public health is the contamination with chemical substances, in particular dioxins, which are lipophilic contaminants tending to accumulate in fatty tissues, but also in liver, an organ rich in proteins able to bind these molecules.

Offal consumption is not negligible in European Union. When analyzing the data extracted from the “Comprehensive European Food Consumption Database; Concise Data Base summary statistics- Total Population”, it can be seen that the consumption of edible offal, including poultry liver, is between 1 g/day (in Ireland) and 26.1 g/day (in Poland), with an average of 7.12 g/day for the European Union, considering the countries that participated to the survey (European Food Safety Authority, 2011).

In particular, poultry liver consumption needs a special attention. Indeed, poultry liver is considered to be an important source of nutrients, such as vitamins, macro elements and microelements. In some countries, it is used in pregnant women diet and in nutritional disorders. Although studies in different countries were conducted regarding dioxins contents of different foods, very few reports which have been published refer to these contaminants level in offal and in particular in poultry liver (Baars et al., 2004; Fernandes, Mortimer, Rose, & Gem, 2010; Hsu et al., 2007; Windal et al., 2010).

The aim of this review was to emphasize the possible contribution of poultry liver to dioxin human exposure through food chain.

2. General data concerning the source and mode of action of dioxins

The term “dioxins” refers to a group of chemically and structurally related halogenated aromatic hydrocarbons including 75 polychlorinated dibenzo-p-dioxins (PCDDs) and 135 polychlorinated dibenzofurans (PCDFs) congeners. The properties, in term of toxicity and bioaccumulation, of individual dioxin and furan congeners differ considerably. From the 210 theoretically possible congeners, only those substituted in each of the 2-, 3-, 7- and 8-positions of the two aromatic rings are of toxicological concern (Safe, 1990).

These 17 congeners exhibit a similar toxicological profile, with 2,3,7,8-tetrachlorodibenzo-p-dioxin (2, 3, 7,8-TCDD) being the most toxic congener (Figs. 1 and 2). Polychlorinated biphenyls (PCBs) are chlorinated aromatic hydrocarbons, which are synthesized by direct chlorination of biphenyls. Depending on the number of chlorine atom substituent (1–10) and their position on the two rings, there are 209 theoretically possible congeners (Fig. 3). PCBs can be divided into different groups according to their biochemical and toxicological properties. Twelve PCBs substituted with at least 4 chlorine (4 non-ortho and 8 mono-ortho) show toxicological properties that are similar to dioxins. They are therefore termed “dioxin-like PCBs” (DL-PCBs). Other PCBs are named non dioxin-like PCBs (NDL-PCBs).

Dioxins are the result of the chlorine-containing manufacturing processes or are produced during incineration (for example, of domestic waste). Weber, Tysklind, and Gaus (2008) pointed out that historic dioxin sources exceed the releases from all contemporary sources. These are stored in soil or sediment and waste reservoirs over decades.

PCBs are anthropogenic chemical compounds, with low electrical resistance, which, in combination with their heat stability, makes them very suitable as cooling liquids in electrical equipment. They were produced between 1930 and 1970 and commercialized in relative large quantities for use as dielectrics, hydraulic fluids, plastics and paints.

PCDDs, PCDFs and PCBs are persistent organic pollutants with high chemical stability; they are lipophilic compounds and they are not destroyed by microbial, photochemical, chemical or thermal degradation. They are ubiquitous environmental contaminants, which are present in all marine plant and animals, birds, mammals and humans and bioaccumulate through the food chain (Erickson, 2001; SCF, 2000; Van den Berg, De Jongh, Poiger, & Olson, 1994).

PCDDs, PCDFs, and to a lesser extent, DL-PCBs, bind to aryl hydrocarbon receptor (AhR) (which are proteins largely present in liver) and induce the transcription of several genes such as metabolizing cytochrome P 450 enzymes (Denison et al., 2002; Whitlock, 1989). International studies have concluded that around 95% of human exposure to dioxin occurs through consumption of food of animal origin, such as meat, dairy products and fish (Ryan & Norstrom, 1991).

The degree of biotransformation or excretion by animals or humans depends on the degree to which they are chlorinated, so PCDD/Fs with few chlorine atoms can be readily oxidized, while highly chlorinated ones can have a half-life in adult humans between 7 and 132 years (7 years for 2,3,7,8-TCDD) (Geyer et al., 2002).

3. Measurement units related to PCDD/Fs and DL-PCBs

A 3-year study conducted by the North Atlantic Treaty Organization Committee on the Challenges of Modern Society (NATO/CCMS) concluded that the TEF (Toxicity Equivalency Factor) approach was the best available interim measure for PCDD/Fs and DL-PCBs risk assessment. On the basis of examination of the available data dealing with exposure, hazard assessment, and analytical methodologies related to dioxins, furans and DL-PCBs, an International Toxicity Equivalency Factor (I-TEF) was proposed.
5. Effects of dioxins in humans

According to the World Health Organization (2010), dioxins are “highly toxic and can cause reproductive and developmental problems, damage the immune system, interfere with hormones and also cause cancer”. Short-term exposure of humans to high levels of dioxins may result in skin lesions, such as chloracne and patchy darkening of the skin and altered liver function. Long-term exposure is linked to impairment of the immune system, the developing nervous system, the endocrine system and reproductive functions (Carpenter, 1998; WHO, 2010).

Based on both animal studies and epidemiologic evidence, 2,3,7,8-TCDD was classified as a “known human carcinogen” (class 1) by the International Agency for Research on Cancer in 1997.

In 2010, Donato and Zani, based on epidemiological studies in Italy, found an association between Non-Hodgkin lymphoma and DL-PCBs serum level concentrations.

Elimination of dioxin in humans depends on dose, age, and quantity of body fat. Aylward et al. (2005) have shown certain variability in individual capacity to eliminate TCDD; elimination was quicker in men and younger people than in women and older people. In addition, clinical studies have shown dose-dependent elimination of TCDD; elimination rate was much greater in higher than in lower levels (Emond, Michalek, Birnbaum, & De Dattes, 2006). These TEF are in use in the current European legislation since the 1st January 2012.

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legislation at 200 ng g\(^{-1}\) fat and 100 ng g\(^{-1}\) fat in milk. This legislation is not valid anymore, since the 1st of January 2012.

Bernard and Fierens (2002) estimated that less than 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. Signs of poisoning were reported only in the poultry, with no reports in other farm animals. There were no cases of acute intoxication in humans that could have been attributed to the consumption of contaminated foods. 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.

Bernard and Fierens (2002) calculated that, in the worst-case scenario, a doubling of the PCB and dioxin body burden could have been reached after consumption of, respectively, 10 and 20 meals of the most contaminated chickens. This conclusion was reached whether the total intake was calculated by assuming a regular consumption of the most contaminated chickens (at least twice a week for 2 months) or on the basis of the average consumption by a standard adult in Belgium of the most contaminated foodstuff.

Another dioxin crisis related with pork meat took place in Ireland in 2008. It was disclosed in early December 2008 that contaminated animal feed was supplied to several pig farms, causing the contamination of the products at levels up to 500 pg TEQ g\(^{-1}\) fat, i.e. between 80 and 200 times the EU’s recommended limit for PCDD/Fs and DL-PCBs. The source of that contamination was burning oil, used for the drying of bakery waste, which was contaminated with PCBs. The contaminated samples were found to contain the same 7 PCBs as in the Belgian crisis, but the ratio between PCBs and PCDD/Fs was lower, and the 7 PCB were not used as indicator (Heres et al., 2010; Hoogenboom et al., 2009).

The last episode in Europe was in January 2011 in Germany, when 47,000 farms were closed as a preventive measure, thousands of chickens killed and hundreds of thousands of eggs destroyed, after a routine testing of eggs had showed dioxin concentrations above the limit for PCDD/Fs and DL-PCBs. The selective accumulation in liver can be explained due to cytoplasmic and microsomal binding within liver tissue, such as the aryl hydrocarbon receptor (AhR) or cytochrome P450 (CYP) metabolizing enzymes (Rose et al., 2010). In rodents, a binding to CYP1A was also shown to explain the hepatic sequestration of these compounds (De Vito et al., 1998). A lower proportion of DL-PCBs, particularly in liver tissue, has been observed and may be related to the ability of PCDD/Fs to bind more strongly than DL-PCBs to AhR, in liver of bovines, sheep, pigs and poultry (Rose et al., 2010). A low carcass fat content may affect the distribution of PCDD/Fs and DL-PCBs between liver and adipose tissues, with increasing concentrations and inducing synthesis of AhR within the liver. The relatively high level expressions of AhR within the liver may also affect the ratio of dioxins to PCBs bound to AhR (Rose et al., 2010).

In an other study concerning the accumulation of PCDD/Fs and PCBs in different species, Huwe et al. (2009), showed that poultry accumulates more PCBs than beef or pork; this is considered to be the result of different dietary regimes or different metabolizing capabilities between these species. In the same way, Zimmerman, Dietrich, Schmid, and Schlatter (1997) remarked that differences in the DL-PCBs pattern are caused more by individual dietary factors than by species differences in biotransformation.

Liver was reported to be different from other tissues because it concentrates preferably high chlorinated congeners (Stephens et al., 1995). This was also shown in the study of Pirard and De Pauw (2005) who conducted a study in which chickens raised according to commercial standards were fed with contaminated feed for 10 weeks. Absorption, distribution and elimination of dioxins, furans and PCBs from feed have been investigated in order to understand the transfer of these compounds from feedstuff to various animal compartments. All organs showed the same congener profile and similar lipid-normalized concentration, except for the liver. Abdominal fat and liver of chickens where the major storage sites. Bioconcentration factors were evaluated, highlighting that the liver preferentially retained highly chlorinated (with six, seven or eight chlorine atoms in the molecule) congeners.

Traqa et al. (2006) conducted a study in which laying hens were fed for seven days with feed from the Belgian dioxin incident, diluted ten times with non-contaminated feed, and after this, for six weeks with non contaminated feed. Samples were collected after the exposure with contaminated feed; the maximal concentration values for dioxins were reached in day nine, and after one, three or six weeks on non contaminated feeding. The concentrations of PCDD/Fs in liver decreased rapidly, being 35, 7, 4 and 3 pg WHO\(_{1998}\)-TEQ g\(^{-1}\) tissue. For indicator PCBs, two samples were collected, one after exposure with contaminated feed, day nine, and the second, after seven weeks. In this case, the concentrations in liver were lower, being 0.77 and 0.14 ng g\(^{-1}\) tissue. Analyzing the data, it can be seen that PCDD/Fs and indicator PCBs are mainly stored in the abdominal fat, and egg fat, and to a lesser extent in the liver. So, livers, weighing around 30 g, contained 1 pg PCDD/Fs WHO\(_{1998}\)-TEQ, 2.7 pg total WHO\(_{1998}\)-TEQ and 23 ng indicator PCBs after feeding with 47 ng PCDD/Fs WHO\(_{1998}\)-TEQ, 155 ng total WHO\(_{1998}\)-TEQ and 2.5 mg non dioxin-like PCBs.

Limited data have shown dioxin half-lives in chickens to be 25–60 days in adipose tissue and eggs (Stephens et al., 1995). Bruggeman et al. (2009) showed that contamination with 2,3,7,8-TCDD of the yolk of chicken eggs is very important for liver protein expression after hatching. After exposure with 2,3,7,8-TCDD, the expression of fibrinogen gamma chain in liver was higher than normal. Also, the NADH ubiquinone oxidoreductase, an enzyme involved in mitochondrial respiratory chain and energy supply, was decreased and could be involved in lower body weight gain of chickens.

Liver has been shown a target organ for the toxic effects of TCDD and concomitant oxidative stress following the results of studies in rats and mice (Bagchi et al., 2002; Hassoun, Wang, Abushaban, & Stobs, 2002). Exposure of birds to TCDD or DL-PCBs during laboratory feeding studies has been associated with a number of
adverse effects including reduced growth, liver enlargement and necrosis, lymphocyte depletion, atrophy of associated organs, enlargement of the thyroid and oxidative stress (Covaci, Ryan, & Schepens, 2002). Although, it is generally accepted that hepatomegaly is one of the key biomarkers of dioxin exposure, Powell, Aulerich, and Meadows (1996) research concluded that hepatomegaly is not observed in birds exposed to non dioxin-like PCBs. PCBs have also been shown to induce embryotoxicity (Ax & Hansen, 1975) and hepatic drug metabolizing enzyme activity in birds (Hansen et al., 1981).

Hilscherova et al. (2003) made a research concerning the mechanisms of toxicity and teratogenicity of 2,3,7,8-TCDD in poultry. One of the mechanisms of teratogenicity is oxidative stress. Eggs were injected simultaneously with TCDD and co-treatment compounds in order to prevent oxidative stress or to block cytochrome P450 activity. In liver, TCDD treatment caused a decrease in glutathione content and glutathione peroxidase activity and an increase in the ratio of oxidized to reduced glutathione. TCDD increased the susceptibility to lipid peroxidation and oxidative DNA damage in liver. Administration of the antioxidants vitamin E and vitamin A provided partial protection against TCDD-induced oxidative stress in liver. Similarly, a study preformed by Jin, Kennedy, Di Muccio, and Moon (2001) on dioxin-contaminated birds demonstrated that TCDD caused a decrease in glutathione content and peroxidase activity in chicken liver after injection exposure of chicken eggs to 3,3',4,4',5-pentachlorobiphenyl (PCB126), while no effects were observed in ducks exposed in the same way.

### 7. Data concerning poultry liver contamination with dioxins and PCBs

According to the Commission Regulation (EC) No 1881/2006, the maximum tolerance level for total PCDD/Fs and DL-PCBs in liver of terrestrial animals including chicken was 12 pg PCDDs/Fs DL-PCBs WHO1998-TEQ g⁻¹ fat and 6 pg PCDD/Fs WHO1998-TEQ g⁻¹ fat, until 31st December 2011.

Starting with the 1st January 2012, a modification of Commission Regulation (EC) No 1881/2006 was done, both to lower the maximal limit and to introduce the use of the WHO-TEF from 2005. The maximum tolerance level for total PCDD/Fs and DL-PCBs in liver of terrestrial animals including chicken was settled at 10 pg PCDD/Fs DL-PCBs WHO2005-TEQ g⁻¹ fat. The maximal value for the sum of 6 non-dioxin like PCBs (PCB 28, PCB 52, PCB 101, PCB 138, PCB 153 and PCB 180) was settled at 40 ng g⁻¹ lipid weight. BERGE et al. (2011) conducted a study in which broiler chickens were fed with similar diet not contaminated (control group) or contaminated with PCDD/Fs and non dioxin-like PCBs. After slaughter, the analysis showed that levels of PCDD/Fs and non dioxin-like PCBs in the liver of the contaminated chickens were higher than in the control group, evidencing their transfer from feed to liver tissue. This study, together with those already mentioned above confirm that a source of liver contamination with dioxin is the feed and there is a direct relation between the quantity of dioxin in feed and respectively in liver. However, although measurable quantities of these contaminants were present in commercial poultry feeds (Iben et al., 2003; Traag et al., 2006), the concentrations were generally very low. The contribution to contaminant uptake from herbage, bedding and drinking water was considered to be minimal. Soil was considered to be the dominant sources of PCBs and PCDD/Fs contamination in chickens reared outdoors, as these birds need a certain amount of dietary soil or grit to aid digestion (Fernandes et al., 2011).

### 7.1. The Belgium crisis and related studies

Samples of chicken feed produced at the end of January 1999 showed the highest levels of contamination, with indicator PCBs and PCDD/Fs concentrations of 450 ng g⁻¹ and 11,140 pg WHO1998-TEQ g⁻¹ respectively.

The indicator PCBs concentrations (expressed as mean and maximal values) in poultry products (including liver) were ranging between 0.84 and 8.2 ng g⁻¹ fat and 1.63–56.8 ng g⁻¹ fat, in the suspected contaminated farms. The mean PCDD/Fs concentration in poultry products ranged from 3.8 to 255 pg WHO1998-TEQ g⁻¹ fat, and the maximal values from 19.6 to 2613 pg WHO1998-TEQ g⁻¹ fat. The highest concentrations of indicator PCBs and PCDD/Fs were found in chicken with signs of intoxication (Bernard et al., 2002).

In conclusion, concentrations decreased rapidly after the exposure period with contaminated feed. Concentrations of indicator PCBs were relatively low after the exposure period and decreased less rapidly during the withdrawal period.

This dioxin crisis had more several direct consequences, as described by Covaci et al. (2008). First, a maximum limit concerning the non dioxin-like PCBs (sum of 7 indicators) was fixed in the Belgian legislation (see section 5). Second, in 2000, a systematic national monitoring program for food of animal origin was created (the CONSUM system), in order to have a permanent control of critical feed ingredients (animal meal and fat, fish meal, kaolinite), a representative screening of feedstuffs and traceability of each production level in the whole food chain. An increasing number of laboratories have refined their analytical methods. This has resulted in more reliable and high-quality results, followed by a significant decrease in the analysis time and cost for PCBs and dioxins tests. The consumption of biologically (organically) grown crops or meat became increasingly popular since the crisis.

### 7.2. Illustrative worldwide levels for dioxins and related compounds in poultry liver

Although consumption of poultry products contributes 5–10% of the estimated daily intake of food in developed countries, the available literature on transfer of these pollutants from feed to chickens and eggs is quite limited (FOCANT et al., 2002).

Table 1 shows the few information found in the literature about PCDD/Fs and DL-PCBs contamination of poultry liver. Normal values for PCDD/Fs in poultry from Europe and United States (1991–1994) were between 1.3 and 2.6 pg WHO1998-TEQ g⁻¹ lipid weight (FERRARIO et al., 2000). Distribution of PCDD/Fs was similar for all analyzed organs (fat, liver, skin and gizzard), in terms of lipid weight.

In the United Kingdom, Total Diet Studies (TDS) conducted by the Food Standards Agency (FSA), showed that oil was the highest PCDD/Fs and DL-PCBs containing food group. This study

<table>
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<tr>
<th>Region/period</th>
<th>WHO1998-TEQ pg⁻¹ fat mean value</th>
<th>Reference</th>
</tr>
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<tbody>
<tr>
<td>UK (2003)</td>
<td>0.2—1.1⁰</td>
<td>FSA (2001)</td>
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<tr>
<td>Spain, 2008</td>
<td>1.7⁰</td>
<td>PARERA et al. (2008)</td>
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ments WHO\textsubscript{1998}-TEQ levels for the sum of PCDD/Fs and DL-PCBs in chicken livers ranging from 0.22 to 1.06 pg g\textsuperscript{-1} fat (FSA, 2003).

Data on liver from The Netherlands corresponding to nation average for summer 1990 showed PCDD/Fs WHO\textsubscript{1998}-TEQ levels ranging from 3.3 pg g\textsuperscript{-1} for chicken liver to 61 pg g\textsuperscript{-1} for horse liver. Levels in pig and sheep liver were 15 and 30 pg WHO\textsubscript{1998}-TEQ g\textsuperscript{-1} respectively, in terms of fresh weight (Liem & Theelen, 1997).

In 2010, the European Food Safety Authority, following Commission Recommendation for the periodic evaluation of dioxin levels in Europe, has reported data on the presence of 17 congeners of PCDD/Fs, and 12 congeners of DL-PCBs in food and feed. An amount of 7270 samples collected in the period 1999–2008 from 19 European Union member states, Norway and Iceland were analyzed. Dioxin and furan congeners represented between 30% and 74% of the total concentrations depending on food or feed group, while DL-PCBs contributed for 15–45%. The highest mean levels of PCDD/Fs and DL-PCBs in food were observed for “Fisher liver and products thereof” (32.6 pg WHO\textsubscript{1998}-TEQ g\textsuperscript{-1}) and “Muscle meat eel” (6.7 pg WHO\textsubscript{1998}-TEQ g\textsuperscript{-1}) expressed on whole weight basis, and for “Liver and products thereof from terrestrial animals” (5.7 pg WHO\textsubscript{1998}-TEQ g\textsuperscript{-1}) expressed on fat basis (FSA, 2010). Low contamination levels were observed in most of the products made using pig and poultry liver such as pâtés, may be due to a number of reasons (Rose et al., 2010). For example, the contaminant loading in the products is likely to be affected by the processing that is carried out during production, such as trimming of the fatty portions or the addition of other non-meat ingredients which generally tend to have lower PCDD/Fs and non-dioxin-like PCBs contents would tend to have a diluting effect on the contaminant content of the product.

8. Conclusions

Although it is known that people ingest dioxins and PCBs because of background levels especially in animal products, this intake is not exceeding the current TWI of 14 pg kg\textsuperscript{-1} body weight per week, and thus it can be considered that there is no risk for human health to develop the specific signs of contamination.

Specifically for chicken liver, the data available in the literature seem to indicate that these background levels are not a concern, being below the European maximal limit, but more information is needed, in particular for DL-PCBs, for a reliable risk assessment. However, in case of incidents, when large amounts of these chemical substances are spread in the environment, dioxins and PCBs contamination represent a real risk for food safety, because of their bioaccumulation thought the food chain. Ideally, at each “dioxin crisis”, a risk assessment should be performed, in order to quantify the exposure of humans to that precise contamination.

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