Review

Transfer of persistent organic pollutants in food of animal origin — Meta-analysis of published data

Farida Amutova [a, b, c], Matthieu Delannoy [a], Almagul Baubekova [d], Gaukhar Konuspayeva [c, d], Stefan Jurjanz [a, *]

[a] URAFPA, University of Lorraine-INRAe, 54500, Vandoeuvre, France
[b] Faculty of Geography and Environmental Sciences, Al-Farabi Kazakh National University, 050040, Almaty, Kazakhstan
[c] Antigen LLP, Scientific and Production Enterprise, 040905, Almaty Region, Kazakhstan
[d] Faculty of Biology and Biotechnology, Al-Farabi Kazakh National University, 050040, Almaty, Kazakhstan

HIGHLIGHTS

- High transfer and bioconcentration of the most toxic PCDD/Fs and PCBs.
- PBDEs are less bioconcentrated than chlorinated POPs.
- DDT congeners bioconcentrate easily but β-HCH does it more than the other isomers.
- Transfer of chlorinated POPs in poultry seemed higher than in mammalians.

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ABSTRACT

The transfer of POPs in food of animal origin has been studied by a meta-analysis of 28 peer-reviewed articles using transfer rate (TR) for milk and eggs and bioconcentration factors (BCF) for eligible tissues after establishing an adapted methodology. TRs of the most toxic PCDD/Fs into milk were generally elevated and even higher into eggs. BCFs in excreting adult animals varied widely between studies complicating to hierarchize tissues or congeners, even if liver and fat seemed to bioconcentrate more than lean tissues. Short time studies have clearly shown low BCFs contrarily to field studies showing the highest BCFs. The BCFs of PCDD/Fs in growing animals were higher in liver than in fat or muscle. In contrast to easily bioconcentrating hexachlorinated congeners, octa- and heptachlorinated congeners barely bioconcentrate. PCB transfer into milk and eggs was systematically high for very lipophilic congeners. Highly *ortho*-chlorinated PCBs were transferred >50% into milk and eggs and even >70% for congeners 123 and 167 into eggs. BCFs of the most toxic PCBs 126 and 169 were significantly higher than for less toxic congeners. BCFs seem generally low in PBDEs except congeners 47, 153 and 154. DDT and its metabolites showed high bioconcentration. Differences between tissues appeared but were masked by a study effect. In addition to some methodologic recommendations, this analysis showed the high transfer of POPs into eggs, milk and liver when animals were exposed justifying a strong monitoring in areas with POP exposure.

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* Corresponding author.
  E-mail address: stefan.jurjanz@univ-lorraine.fr (S. Jurjanz).

1. Introduction

Free-range agricultural animals ingest various environmental matrices as vegetation frequently used as feed, but also water or even soil. Several studies have reported that ruminants would consume in normal grazing conditions up to 10% of soil in daily dry matter intake (Healy, 1968; Jurjanz et al., 2012; Collas et al., 2019) and even up to 30% in the worst conditions as winter grazing (Abrahams and Steigmajer, 2003). Poultry also ingest soil along pecking contaminated feed from the ground. In unbalanced feeding conditions, the soil intake in laying hens may reach up to 23% (Jondreville et al., 2010).

During such soil intake, persistent organic pollutants (POPs) deposited on the upper soil can be ingested by livestock (Fries et al., 1982; Jones et al., 1989; Stevens and Gerbec, 1988) and transferred into their subsequent food products. Previous studies have demonstrated that contaminated soil is the main source of exposure to pollutants for livestock, especially when soil is contaminated by lipophilic POPs, such us polychlorinated biphenyls (PCBs), dioxins and furans (PCDD/Fs) (Diletti et al., 2014), or organochlorinated pesticides (OCPs) (Tao et al., 2009). Some studies have reported transfers of OCPs and PCBs to livestock after atmospheric deposition leading to contamination of water, soil and vegetation (Pan et al., 2014) by regular floods (Abrahams and Steigmajer, 2003; Rose et al., 2012a) and others. Subsequently, POPs can be transferred to food of animal origin, such as milk, meat and eggs. As a result, consumption of such contaminated food would be a possible route of POP exposure for humans (McLachlan et al., 1990), and the correct quantification of the POP transfer from contaminated environmental matter to food products is essential for a reliable risk assessment.

Nevertheless, considerable variations can be found between reported transfers among different studies as they are based on different contamination matrices, doses and duration of exposure. However, such data are required to realize a risk assessment of food production systems. The aim of this paper was to derive and summarize current literature data on the transfer of different classes of POPs to edible tissues and food of animal origin. A consolidated methodology was used to discriminate the current knowledge about distribution and accumulation of PCDD/Fs, PCBs, PBDEs and OCPs in animal liver, fat and muscle tissue and their excretion into milk and eggs. At the end, this synthesis aims to improve monitoring system for livestock farming in contaminated areas.

2. Materials and methods

A database was created using peer-reviewed research articles reporting the transfer of POPs from ingested environmental matrices to edible tissues in different animal species (Table 1). Then, the transfer was evaluated by bioconcentration factors and transfer rates to characterize respectively the distribution of pollutants in tissues or the excretion of POPs *via* milk or eggs.

2.1. Building-up the database

In total, forty-one peer-reviewed articles reporting transfer of different POPs were browsed by Google Scholar through queries comprising the following keywords:

Excretion OR mass-balance OR carry-over rate OR transfer rate OR biotransfer AND (PCDD/F OR PCB OR OCP OR PBDE) AND ((milk AND ((cow OR cattle) OR (Goat OR Caprine))) OR (hens OR pig).

Transfer was quantified in two different concepts: as transfer rate (**TR**) when the target was excreted milk or eggs or as a bioconcentration factor (**BCF**) when transfer was measured *via* the enrichment in body tissues such as liver, muscles or body fat. Nevertheless, we did not integrate data of biotransfer factors (**BTF**), i.e. a ratio between tissue concentration and ingested amount of the studied POP. Although this approach would have several advantages, as the ingested amounts of all contaminated materials over the whole exposure period are required. These data are often missing due to field conditions and therefore not available for a risk assessment. As the concepts of TR and BCF were not used homogeneously in the literature, we firstly defined them.

The TR corresponds to the ratio between ingested and excreted amounts of the pollutant (equation (1)) which can be calculated after reaching steady state (Richter and McLachlan, 2001) and expressed as a percentage:

$$TR_{product} = \frac{[pollutant]_{Fat\ of\ product}\ Daily\ fat\ excretion}{[pollutant]_{Diet}*Diet\ Intake}*100\%$$

(1)

The $[pollutant]_{fat\ of\ product}$ corresponds to the concentration of pollutant in the fat of the food product (pg/g fat) and $[pollutant]_{Diet}$ to its concentration in the intake matrix feed or in soil (pg/g dry

Table 1
Studies extracted from the literature, validation of the methodologic criteria and the decision of their integration in the data set.

N°	Reference	Studied compounds	Exposure media	Category of animals	Exposure (days)	SS[a]	Parameters[b]	Intake C[c]	Intake < LQ[d]	Intake Q[c]	edible tissues C[c]	edible tissues < LQ[d]	edible tissues Q[c]	Integration[e]
1	**Aulakh et al. (2006)**	OCPs	Feed	Laying hens	Field exposure	(✓)	(BCF) (TR)	✓	—	(✓)	✓	—	✓	✓ ✓
2	Brambilla et al. (2008)	PCDD/Fs	Minerals	Dairy cows	28	(✗)	TR	✗	—	✓	✗	—	✓	✗ (exposure < half-live)
3	**Costera et al. (2006)**	PCDD/Fs, PCBs	Hay	Dairy goats	70	✓	TR	✓	—	✓	✓	—	✓	✓
4	**Diletti et al. (2014)**	PCDD/Fs, PCBs	Feed	dairy buffaloes	100	✓	TR	✓	—	✓	✓	2 PCDD/Fs congeners out of 17	✗	✓
5	Feil et al., 2000	PCDD/Fs	Feed	Cattle (calves)	120	✓	BCF	✓	—	✓	✓	8 congeners out of 17	—	✗ (C wet basis, 8 congeners < LQs)
6	Fernandes et al. (2011)	PCDD/Fs, dl-PCBs	Feed	Pigs	81–179	(✓)	BTF	✗	—	✗	✗	—	✗	✗ (only BTF, no reported C)
				Sheep (non lact. ewes)	48–153	(✓)	BTF	✗	—	✗	✗	—	✗	
				Broilers, Laying hens	28–250	(✓)	BTF	✗	—	✗	✗	—	✗	
7	Fournier et al. (2013)	iPCBs	Corn silage	goats	39	✓	MISS	✓	—	✓	✗	—	✓	✗ (no reported C in milk)
8	**Fries et al. (1999)**	PCDD/Fs	PCP-treated Wood and feed	dairy cows	58	✓	TR	✓	—	✓	✓	—	✓	✓
9	**Fries et al. (2002)**	PCDD/Fs	Feed	dairy cows	58	✓	TR	✗	3 congeners out of 17	✓	✗	3 congeners out of 17	✗	✓
10	**Hoogenboom et al. (2004)**	PCDD/Fs PCBs	Feed	Broilers	7	✗	BCF	✓	—	✓	✓	3 PCDD/Fs congeners out of 17	✗	✓
		PCDD/Fs PCBs	Feed	Pigs	7	✗	BCF	✓	—	✓	✓	3 PCDD/Fs congeners out of 17	✗	✓
11	**Hoogenboom et al. (2006)**	PCDD/Fs	Feed	Laying hens	56	✓	TR	✓	—	✓	✓	—	✓	✓
12	**Hoogenboom et al., 2015 (a)**	PCDD/Fs	Maize silage or sugar beet	dairy cows	33	(✗)	TR	✓	—	✓	✓	9 congeners out of 17	✓	✗ (exposure < half-live)
		PCBs	Maize silage or sugar beet	dairy cows	33	(✓)	TR	✓	7 congeners out of 12	✓	✓	4 congeners out of 12	✓	✓
13	**Hoogenboom et al., 2015 (b)**	PCDD/Fs, PCBs	Grass	Lamb	113	(✓)	(BCF)	✓	1 congener out of 17	✓	✓	2 congeners out of 17	✗	✓
14	**Huwe and Smith, 2005**	PCDD/Fs dl-PCBs	Mineral Mix	dairy cows	Averaged between 17 and 38	✓	TR BCF	✓	3 congeners out of 17	✓	✓	—	✓	✗ (exposure < half-live) ✓
15	**Kerst et al. (2004)**	dl-PCBs	Grass	dairy cows	Field exposure	(✓)	TR	✓	—	✓	✓	—	✓	✓
16	**Kierkegaard et al., 2009**	PBDE	Feed	dairy cows	90	✓	(BCF)	(✓)	—	✓	(✓)	—	✓	✓
17	**Lorenzi et al. (2020)**	PCDD/Fs, dl and iPCBs	Feed	dairy cows	49	✓	TR	✓	—	✓	✓	—	✓	✓
18	McLachlan et al. (1990)	PCDD/Fs	Feed	dairy cows	35	✗	TR	✗	—	✓	✗	—	✓	✗ (exposure < half-live)
19	McLachlan and Richter, 1998	PCDD/Fs	Feed	dairy cows	84	(✓)	TR	✓	—	✓	✗	4 congeners out of 17	✓	✗ (exposure < half-live)
		PCDD/Fs	Feed	dairy cows	23	(✗)	TR	✓	—	✓	✗	2 congeners out of 17	✓	✗ (exposure < half-live)
20	**Malisch, 2000**	PCDD/Fs	Citrus pulp	dairy cows	180	(✓)	TR	✗	—	✓	✓	1 congener out of 17	✗	✓
21	Olling et al. (1991)	PCDD/Fs	Intraruminally dose in oil	dairy cows	1 (single dose)	✗	TR	✗	—	✓	✓	—	✓	✗ (Single dose exposure)
22	**Ounnas et al. (2010)**	PCBs (dl & mono-ortho), PBDE	Soil	goats	80	✓	TR BCF	✓	—	✓	✓	—	✓	✓
23	**Pan et al. (2014)**	OCPs iPCBs	Grass	yak	Field exposure	(✓)	(BCF)	✓	—	✗	✓	—	✗	✓
24	**Parera et al. (2008)**	PCDD/Fs	Feed	Broilers	39	—	(BCF)	✓	—	✓	✓	2 congeners out of 17	—	✓
25	**Petreas et al. (1991)**	PCDD/Fs	Soil	Laying hens	178	✓	BTF (TR)	✓	2 congeners out of 17	(✓)	✓	—	(✓)	✗ (BTF) ✓
26	**Pirard and De Pauw (2005)**	PCDD/Fs, dl-PCBs	Feed	Laying hens	54	✓	BCF (TR)	✓	1 PCDD/Fs congener out of 17	(✓)	✓	1 PCDD/Fs congener out of 17	(✓)	✓ ✓
27	**Pirard and De Pauw (2006)**	PCDD/Fs,	Feed	Laying hens	98	✓	BCF (TR)	✓	—	(✓)	✓	2 congeners out of 17	(✓)	✓ ✓
28	**Piskorska-Pliszczynska et al. (2014)**	PCDD/Fs	Soil	Laying hens	Field exposure	(✓)	(BCF)	✓	—	(✓)	✓	—	✓	✓
29	Rose et al. (2012b)	PCDD/Fs dl-PCBs,	Feed	pigs	Field exposure	(✓)	(BCF)	✓	10 PCDD/Fs out of 17 congeners	✗	✓	10 PCDD/Fs congeners out of 17	✓	✗ (Most compounds < LQ)

Table 1 (*continued*)

Nº Reference	Studied compounds	Exposure media	Category of animals	Exposure (days)	SS[a]	Parameters[b]	Intake C[c]	< LQ[d]	Q[c]	edible tissues C[c]	< LQ[d]	Q[c]	Integration[e]
	iPCBs, Mono-*ortho*					MISS	✓	—			—		✗ (Missing of intake and its C)
30 **Schuler et al. (1997)**	PCDD/Fs	Grass	dairy cows	Field exposure	(✓)	TR	✓	—	✗	✓	—	✓	✓
31 Shen et al. (2012)	PCDD/Fs, dl-PCB	Soy-bean oil	pigs	91	(✗)	MISS	✗	—	✗	✓	—	—	✗ (no reported intake C
32 **Shih et al.2009**	PCDD/Fs	Feed	ducks	41	✓	BCF (TR)	✓	—	(✓)	✓	—	(✓)	✓ ✓
33 Slob et al. (1995)	PCDD/Fs	Fly ash	dairy cows	1	✗	BA	✓	—	✓	✓	2 PCDD/Fs congeners out of 17	✓	✗ Single dose exposure
	PCDD/Fs, dl-PCB	Grass	dairy cows	30	✓	TR	✓	—	✓	✓	2 PCDD/Fs congeners out of 17	✓	✗ (exposure < half-live)
34 **Spitaler et al. (2005)**	PCDD/Fs	Feed	pigs	126	(✓)	(BCF)	✓	—	✓	✓	5 congeners out of 17	✓	✓
35 **Tao et al. (2009)**	OCPs	Feed	hens	Field exposure	(✓)	(BCF)	✓	—	✗	✓	—	✗	✓
36 **Thomas et al. (1999)**	iPCBs and Mono-*ortho*	Feed	dairy cows	128	✓	TR	✗	—	✓	✗	—	✓	✓
37 Thorpe et al. (2001)	PCDD/Fs iPCBs, and mono-*ortho*	Gelatin capsule	Cattle (heifers)	28	(✗)	MISS	(✗)	—	✗	✓	—	✗	✗ (no reported C in feed)
38 **Traag et al. (2006)**	PCDD/Fs PCBs	Feed	Laying hens	7	✗	(TR)	✓	—	✓	✓	2 PCDD/Fs congeners out of 17	✓	✗ (no SS)
	PCDD/Fs PCBs	Feed	Laying hens	7	✗	(BCF)	✓	—	✓	✓	4 PCDD/Fs congeners out of 17	✓	✓
39 Tuinstra et al., 1992	PCDD/Fs	Feed	dairy cows	105—119	(✓)	MISS	✓	—	✓	✗	—	✗	✗ (no reported C in milk)
40 Watanabe et al. (2010)	PCDD/Fs, PCBs	Dust & soil	pigs	Field exposure	✗	MISS	✗	—	✗	✓	3 PCDD/Fs congeners out of 17	✗	✗ (no reported C in feed)
41 Wittsiepe et al. (2007)	PCDD/Fs	Soil	Minipigs,	28	✗	MISS	✓	—	✓	✓	4 congeners out of 17	—	✗ (no reported C in feed)
	PCDD/Fs	feed	56—78 days		✗	MISS	✓	—	✓	✓	1 congener out of 17;	—	✗ (no reported C in feed)

Bold mean integrated article in our dataset.

[a] Statement of steady state (SS) given by the authors or in brackets when statement was made by us.

[b] TR-transfer rate, BCF — bioconcentration factor, BA-bioavailability, BTF- biotransfer factor. MISS — missing data. Values of the parameters taken from the authors or in brackets when re-calculated by us.

[c] C — concentrations, Q-quantities where ✓ = given in the article; (✓) = Recalculated; ✗ = not available.

[d] Compounds that below limit of quantification (LQ).

[e] Decision of integration (✓) or not (✗) of the given study in our dataset.

matter). Due to their lipophilicity, POP concentrations are commonly expressed in relation to fat content needing therefore information about fat mass of milk or eggs. Pollutants can also be accumulated in body tissues and reach concentrations higher than in the exposure matrix. The extent of this accumulation is expressed as the ratio between the concentrations in intake (i.e. feed or dry soil) and the studied body tissue (equation (2)).

$$BCF = \frac{[\text{pollutant}]_{\text{tissue}}}{[\text{pollutant}]_{\text{intake}}} \qquad (2)$$

In this equation, [pollutant] $_{\text{tissue}}$ corresponds to the pollutant concentration in the tissues (pg/g fat) and [pollutant]$_{\text{feed}}$ to its concentration in feed (pg/g feed at 15% wet content) or in soil (pg/g dry matter).

2.2. Selection criterion of articles in the dataset

A calculation of transfer rate was conditioned by the duration of exposure which would allow the considered compound to reach in the tissue of the exposed animal a stable concentration of steady state (Huwe and Smith, 2005; Ounnas et al., 2010). Casarett and Doull (2008) defined the steady state as the physiological state of an animal during a continuous exposure to a fixed daily dose, leading to a concentration remaining stable in time. Therefore, the compound specific exposure duration is the key parameter to achieve a steady state plateau.

We considered that steady state conditions have been reached when authors reported it clearly in their article. If not, we evaluated the exposure duration in comparison to the half-life of the considered compound in the given animal type (mammalians *versus* birds) in respect to its life expectancy. For example, half-life of PCDD/Fs in hen eggs is 30 days (Petreas et al., 1991), but would be more than 40 days in cow milk (Olling et al., 1991; Huwe and Smith, 2005) and goat milk (Costera et al., 2006). Contrarily, half-life of PCBs in hen eggs and goat milk of only 30 days has been confirmed by averaged results of Hoogenboom et al. (2015a) and Costera et al. (2006) respectively. Indeed, half-life of the target compound would allow to predict the necessary exposure duration to reach steady state (Holford, 2012): 50% of the steady state concentration would be reached after one half-life and 87,6% after three half-lives. We fixed an acceptable level of steady state at 90% of the plateau corresponding to 3.3 half-lives of the considered compound. The study of Hoogenboom et al. (2006) on laying hens reported the transfer of different POPs in eggs after exposure durations of 56 days, declared as "close to steady state plateau". By acceptation, this study has been integrated in the dataset. Furthermore, some studies have reported exposure due to a field contamination (i.e. long-time exposure of several weeks, often several months) for dairy cows (Kerst et al., 2004; Schuler et al., 1997) or laying hens (Aulakh et al., 2006; Piskorska-Pliszczynska et al., 2014). These studies were also integrated in our dataset considering that these animals had probably be exposed long enough to the studied compounds that makes the achievement of a steady state plateau likely.

In contrast to transfer studies into milk or eggs, the achievement of a steady state was not a selection criterion to integrate studies reporting BCFs. Indeed, the target animals were generally meat-producing categories such as pigs, broilers or, lambs. Their rapid increase in body mass will constantly redistribute target substances in tissues that makes the achievement of a stable concentration nearly impossible. In other words, the steady state could only be achieved in adults with a stable body weight. Therefore, we compared BCFs between two groups of animals: firstly, excreting adults (lactating mammalians or laying birds) or secondly, rapidly growing young animals, generally raised for meat production. Then

comparisons were carried out on BCFs issued from the same study: either the different congeners within the same tissue or the different tissues for given congener. Finally, the interpretation of BCFs will distinguish short time exposure ($\leq$7 days) but no single dose and chronic exposure (clearly more than 7 days).

Except some minor adaptions of the expression (i.e. equations (1) and (2)), we used generally TRs and BCFs as they were indicated in the article. In absence of reported TR or BCFs we calculated them ourselves using the data reported in the article. However, some studies needed some zootechnical hypothesis to calculate TRs or BCFs:

1) Some studies have reported only concentrations in feed and tissues (Traag et al., 2006, Huwe and Smith, 2005; Parera et al., 2008, Tao et al., 2009 and Kierkegaard et al., 2009; Hoogenboom et al., 2015b) that we used to calculate BCFs.

2) In absence of any specification in the article, we evaluated the daily feed intake of laying hens as 110g (studies of Petreas et al., 1991; Pirard and De Pauw, 2005). If missing, the output of very lipophilic POPs by laying hens has been evaluated on the basis of a standard egg (55g containing 27% lipids in the yolk) enhancing a fat excretion of 5.4g per egg. The laying productivity in intensive free-range rearing conditions of 6 eggs per week was supposed leading to an average daily fat excretion of 4.6 g/day *via* egg laying. In addition, Petreas et al. (1991) reported TRs of PCDD/Fs in eggs in a study where 10% of contaminated soil was integrated in the feed. Therefore, we considered a global intake of 99g of feed and 11g of (contaminated) soil to establish the global intake concentration of the contaminants.

3) Shih et al. (2009) investigated the transfer of PCDD/Fs into eggs of laying ducks. The contaminants were issued from dust of which 0,6% was integrated in their feed. According to Bley and Bessei (2008), a daily feed intake of 200 g was supposed and then the daily dust intake would represent 1.2 g. The fat excretion in duck eggs has been supposed to be 7.5g daily (laying productivity of 90% for eggs of 66g containing 8.3g of yolk fat; Kaewmanee et al., 2009).

4) The field study of Piskorska-Pliszczynska et al. (2014) considered soil as the only source of PCDD/Fs. Aiming to obtain a reliable concentration in the global intake for BCF calculations, we divided the ingested amounts of contaminants by an estimated amount of ingested matter, i.e. 90 g of feed and 20 g of soil in free range laying hens as reported by Jondreville et al. (2010).

5) The study on yaks (Pan et al., 2014) allowed calculations of BCFs between grass and the tissues muscle and liver considering grass as the only exposure source. Nevertheless, notable differences in POP concentrations had been shown between summer and winter samples. Therefore, the average concentration in tissues has been used. In absence of consolidated data about feed intake or milk yields of these yaks, we renounced to calculate TRs in milk.

6) Aulakh et al. (2006) presented an original field study reporting concentration of different OCPs in feed, eggs and muscles of hens. TR to eggs was calculated using the same feed intake and egg composition than previously presented. The calculation of BCFs in hen muscle was based on a fat content of 10% in the fresh matter according to Kaewkot et al. (2020).

Thus, Table 1 shows the complete list of extracted articles, the studied compounds, species, exposure duration and the integration choices. Thirteen research papers out of 41 have not been integrated and 2 only partly, mainly due to a too short exposure duration of the animals (in case of TRs), which not allowed to achieve steady state conditions (Brambilla et al., 2008; Olling et al., 1991; Huwe and Smith, 2005; Slob et al., 1995; Traag et al., 2006;

Table 2
Transfer of PCDD/Fs in milk and eggs expressed as TR (mean and SD) and transfer level, depending on the congener, its chemical characteristics (number of chlorines (Cl_n), Log of the partition coefficient octanol/water (log K_{ow}), molecular weight (MW)) and Toxic Equivalency Factors (TEF).

Compound	TEF (WHO05)	Chemical characteristics			Transfer to milk		Transfer to hen eggs		Transfer to duck eggs
		Cl_n	Log K_{ow}[a]	MW	TR[b], %(n = 8)	Level[c]	TR, %(n = 4)	Level	TR[d], %(n = 1)
2,3,7,8-TCDD	1	4	6.6	322	**34.0 ± 6.3**	**High**	**39.1 ± 12.6**	**High**	2.0
1,2,3,7,8 -PeCDD	1	5	7.2	340	**26.7 ± 7.1**	**High**	**35.8 ± 12.2**	**High**	3.4
1,2,3,4,7,8-HxCDD	0.1	6	7.6	391	17.8 ± 8.0	Medium	**43.3 ± 16.5**	**High**	2.3
1,2,3,6,7,8-HxCDD	0.1	6	7.6	391	22.7 ± 7.1	Medium	**40.6 ± 14.4**	**High**	3.0
1,2,3,7,8,9-HxCDD	0.1	6	7.6	391	13.2 ± 3.4	Medium	**29.1 ± 12.4**	**High**	1.3
1,2,3,4,6,7,8-HpCDD	0.01	7	8.0	425	4.1 ± 1.3	Low	16.2 ± 6.2	Medium	1.1
OCDD	0.0003	8	8.4	460	1.2 ± 0.8	Low	6.8 ± 4.8	Low	1.0
2,3,7,8-TCDF	0.1	4	6.5	306	3.4 ± 2.9	Low	**39.1 ± 16.8**	**High**	6.4
1,2,3,7,8-PeCDF	0.03	5	7.0	340	4.9 ± 4.5	Low	**38.0 ± 7.4**	**High**	4.5
2,3,4,7,8-PeCDF	0.3	5	7.1	340	**35.6 ± 14.8**	**High**	**40.0 ± 10.1**	**High**	4.8
1,2,3,4,7,8-HxCDF	0.1	6	7.5	375	19.3 ± 8.9	Medium	**39.8 ± 13.0**	**High**	2.5
1,2,3,6,7,8-HxCDF	0.1	6	7.6	375	17.7 ± 6.0	Medium	**37.3 ± 16.1**	**High**	2.4
1,2,3,7,8,9-HxCDF	0.1	6	7.7	375	10.7 ± 7.0	Medium	**25.6 ± 13.0**	**High**	1.9
2,3,4,6,7,8-HxCDF	0.1	6	7.6	375	11.6 ± 8.7	Medium	23.0 ± 16.5	Medium	0.8
1,2,3,4,6,7,8-HpCDF	0.01	7	8.0	409	3.1 ± 1.1	Low	16.6 ± 10.7	Medium	0.7
1,2,3,4,7,8,9-HpCDF	0.01	7	8.2	409	4.6 ± 1.3	Low	17.8 ± 8.7	Medium	1.1
OCDF	0.0003	8	8.6	443	1.0 ± 1.3	Low	4.0 ± 2.5	Low	0.1

Bold values are highly transferred.
[a] log K_{ow} for PCDDs and PCDFs were taken from Chen et al. (2001) and Govers and Krop, 1998 respectively.
[b] Means and SD of TRs to milk were calculated using the data reported in studies McLachlan and Richter 1998; Malisch, 2000; Fries et al., (1999); Fries et al. (2002); Costera et al., (2006), Lorenzi et al., (2020), Diletti et al., (2014); Schuler et al. (1997).
[c] Means and SD of TRs to eggs were calculated using the data of the 2 treatments reported by Hoogenboom et al., (2006); Petreas et al., 1991; Pirard and De Pauw (2005); Pirard and De Pauw (2006).
[d] Transfer to duck eggs based on Shih et al. (2009), level of transfer is low for all congeners.

McLachlan et al., 1990). In addition, there are missing intake and/or tissue concentrations for calculation of BCFs in the studies of Fernandes et al. (2011), Fournier et al. (2013), Shen et al. (2012), Thorpe et al. (2001), Tuinstra et al. (1992), Watanabe et al. (2010) and Wittsiepe et al. (2007). Finally, the studies of Feil et al. (2000) and Rose et al. (2012b) reported transfer of PCDD/Fs and PCBs from feeds but the concentrations of the studied POPs are generally under the limit of quantification.

2.3. Data analysis

2.3.1. Transfer rates

After extraction of TRs values from the relevant studies, a mean and a SD were calculated for each congener in a given product (i.e. milk or egg). Based on these values, the transfer was classified in four levels depending on the TR:

- high transfer when TR>25%;
- medium transfer for TRs between 10 and 25%;
- low transfer when TR is between 0.5 and up to 10% and
- no significant transfer was considered when TRs were <0.5%.

The comparison between the different congeners of PCDD/Fs (Table 2) and PCBs (Table 3) was based on the one hand on the differences between mean TRs weighed by the associated SDs and on the other hand on the attributed transfer levels.

Additionally, two separate principal component analysis (PCA) were carried out respectively for milk and eggs using the software XLSTAT 2020 (Addinsoft Corp., New York, USA) to illustrate the link between TRs and some chemical characteristics of the PCB congeners (Log K_{OW}, MW, number of chlorines Cl_n, *ortho*-substitution). The variable TR being the variable to be explained was added as a supplementary variable (Jolliffe and Penny, 2002).

2.4. Bioconcentration factors

The BCFs derived from the relevant papers were summarized in tables depending on the chemical family (i.e. PCDD/PCDFs, PCBs, PBDEs and OCPs). Within each table, the BCFs per congener and tissue were listed. Tissues were inventoried in the tables in a decreasing order of mean BCFs over all integrated studies.

Then, BCFs for **PCDD/Fs** were divided in two animal categories and presented separately:

-Adult animals with depuration excretion (i.e. lactating mammalians or laying birds) (Table 4 grouping the data issued from the studies of Huwe and Smith, 2005; Pirard and De Pauw, 2005; Pirard and De Pauw, 2006; Traag et al., 2006; Shih et al., 2009; and Piskorska-Pliszczynska et al., 2014).

- Rapidly growing young animals, generally raised for meat purposes (broilers, pigs and lambs) (Table 5 grouping data from the studies of Hoogenboom et al., 2004; Spitaler et al., 2005; Parera et al., 2008; and Hoogenboom et al., 2015b).

The size of the dataset for PCBs, PBDEs or OCPs did not allow such a distinction for the presentation of their data. BCFs for **PCBs** were grouped in one synthetic table (Table 6) for both groups of animals. Nevertheless, the mention of the animal type and the duration allowed the comparison of BCFs between tissues or between congeners. Finally, sparser data of **PBDEs** (2 studies) and **OCPs** (3 studies) were simply listed by tissues and by congeners in a synthetic table (respectively 7 and 8). By the way, the analysis of PBDEs was restricted by numerous tissue concentrations under the LQ.

A statistical comparison of BCFs appeared irrelevant due to the differences between the grouped studies, especially on exposure duration and doses. Therefore, the comparisons were built on the hierarchy of the distribution within a given study on the one hand between the different congeners and on the other hand between the different tissues to generalize pathways of bioconcentration mechanisms.

Table 3
Transfer of PCBs in milk and eggs expressed as TR (mean and SD) and transfer level, depending on the congener, its Toxic Equivalency Factors (TEF) and its chemical characteristics number of chlorines (Cl_n), Log of the partition coefficient octanol/water (log K_{ow}), molecular weight (MW) and the substitution.

PCB No	TEF (WHO05)	Chemical characteristics				Transfer to milk			Transfer to hen eggs		
		Cl_n	Log Kow[a]	MW	Substitution type	TR[b], %	level	n	TR[c], %	level	n
28	Nd	3	5.6	257	mono-*ortho*	9.4 ± 12.8	medium	7	**40.5 ± 3.5**	high	2
52	Nd	3	5.8	292	di-*ortho*	3.8 ± 5.4	low	6	4.5 ± 0.7	low	2
77	0.0001	4	6.4	292	non-*ortho*	5.5 ± 4.3	low	7	**32.6 ± 25.9**	high	3
81	0.0003	4	6.4	292	non-*ortho*	12.5 ± 6.0	medium	6	**30.2 ± 17.8**	high	3
101	nd	5	6.3	326	di-*ortho*	4.8 ± 1.3	low	6	5.0	low	2
105	0.00003	5	6.6	326	mono-*ortho*	**50.0 ± 33.4**	high	4	**49.0 ± 4.2**	high	2
114	0.00003	5	6.6	326	mono-*ortho*	**51.8 ± 16.2**	high	6	**51.5 ± 0.7**	high	2
118	0.00003	5	6.7	326	mono-*ortho*	**77.8 ± 45.1**	high	5	**50.0 ± 2.8**	high	2
123	0.00003	5	6.7	326	mono-*ortho*	22.2 ± 10.6	medium	6	**74.0 ± 5.7**	high	2
126	0.1	5	7.0	326	non-*ortho*	**40.2 ± 11.4**	high	5	**37.7 ± 19.6**	high	3
138	nd	6	6.7	361	di-*ortho*	**46.1 ± 19.0**	high	6	**52.5 ± 2.1**	high	2
153	nd	6	6.8	361	di-*ortho*	**54.1 ± 25.1**	high	4	**59.0 ± 2.8**	high	2
156	0.00003	6	7.1	361	mono-*ortho*	**64.1 ± 26.3**	high	3	**56.5 ± 2.1**	high	2
157	0.00003	6	7.1	361	mono-*ortho*	**50.5 ± 21.5**	high	3	**58.0 ± 9.9**	high	2
167	0.00003	6	7.2	361	mono-*ortho*	**58.1 ± 25.3**	high	3	**80.0 ± 5.7**	high	2
169	0.03	6	7.5	361	non-*ortho*	**40.3 ± 8.0**	high	4	**39.5 ± 19.9**	high	3
180	nd	7	7.2	395	di-*ortho*	**51.8 ± 17.7**	high	3	**50.0 ± 12.7**	high	2
189	0.00003	7	7.6	395	mono-*ortho*	**38.0 ± 23.2**	high	3	**61.0**	high	2

Bold values are highly transferred nd: not determined.
[a] Log K_{ow} taken from Zhou et al., (2005).
[b] Means and SD of TRs to milk were calculated using the data reported in studies Costera et al., 2006; Thomas et al., 1999; Ounnas et al. (2010); Lorenzi et al. (2020); Kerst et al. (2004); Diletti et al., 2014; Hoogenboom et al., 2015 (a).
[c] Means and SD of TRs to eggs were calculated using the data of the 2 treatments reported by Hoogenboom et al., (2006); and Pirard and De Pauw (2005).

Table 4
BCFs of PCDD/Fs of different tissues in excreting adult animals derived from literature. Colors signification: dark grey — high level of BCF (>25), medium grey — medium level of BCF (10−25), light grey — low level of BCF (>1 and < 10), white — no bioconcentration (BCF<1).

Tissue	Study[1]	Exposure (days)	PCDDs							PCDFs									
			2,3,7,8-TCDD	1,2,3,7,8-PeCDD	1,2,3,4,7,8-HxCDD	1,2,3,6,7,8-HxCDD	1,2,3,7,8,9-HxCDD	1,2,3,4,6,7,8-HpCDD	OCDD	2,3,7,8-TCDF	1,2,3,7,8-PeCDF	2,3,4,7,8-PeCDF	1,2,3,4,7,8-HxCDF	1,2,3,6,7,8-HxCDF	1,2,3,7,8,9-HxCDF	2,3,4,6,7,8-HxCDF	1,2,3,4,6,7,8-HpCDF	1,2,3,4,7,8,9-HpCDF	OCDF
Liver	A (hens)	7 (short)	LQ	1	2	10	9	8	33	0.3	0.2	0.4	1	1	LQ	LQ	2	2	20
	B (cows)	27	11	20	29	20	9	11	3	1	1	103	43	49	LQ	43	19	21	3
	C (hens)	54	43	36	72	85	41	52	36	27	25	35	80	57	54	40	29	32	LQ
	D (hens)	98	LQ	9	6	6	4	1	0.3	13	10	9	5	4	2	4	1	1	LQ
	E (hens)	Field	20	45	74	106	113	134	34	32	36	87	14	18	0.3	90	21	10	28
Ovary	E (hens)	Field	6	23	56	82	78	48	10	19	13	29	21	25	21	25	9	LQ	LQ
Serum	C (hens)	54	24	36	26	29	18	7	73	6	12	38	8	14	0.7	36	13	5	10
Abdominal fat	A (hens)	7 (short)	2	1	1	LQ	LQ	0.1	0.2	2	1	1	1	1	LQ	0.5	0.1	0.2	0.1
	C (hens)	54	20	19	17	16	8	4	LQ	15	10	32	15	15	13	10	3	5	LQ
	D (hens)	98	n.d.	9	7	7	4	1	n.d.	13	10	9	5	5	2	3	0.4	1	n.d.
	E (hens)	Field	8	27	28	43	38	13	2	7	11	38	4	7	1	16	4	2	2
Kidney	B (cows)	27	2	2	2	2	0.4	0.2	0.04	0.1	0.1	2	1	1	LQ	0.4	0.1	0.1	0.01
	C (hens)	54	24	25	22	16	11	7	11	20	11	21	19	18	16	12	6	6	LQ
Heart	C (hens)	54	21	18	16	14	8	2	LQ	13	8	16	10	11	11	8	2	4	LQ
Muscles	F (ducks)	41	19	12	5	6	1	0.4	0.4	30	7	11	2	2	1	1	LQ	1	LQ
	C (hens)	54	18	17	15	11	6	2	LQ	13	9	14	10	11	9	8	2	3	LQ
	D (hens)	98	LQ	9	7	6	4	3	3	11	8	7	4	3	1	2	1	1	1
	E (hens)	Field	5	14	11	40	28	12	3	6	15	33	4	6	1	11	4	2	4
Meat	B (cows)	27	2	2	1	1	LQ	0.1	0.03	LQ	LQ	1	0.5	0.4	LQ	0.3	0.1	LQ	LQ

[1] studies: **A**- Traag et al., 2006, **B**- Huwe et al., 2005, **C** - Pirard and De Pauw 2005, **D** - Pirard and De Pauw 2006, **E** - Piskorska-Pliszczynska et al., 2014, **F** - Shih et al., 2009
LQ – values under the limit of quantification did not allow to calculate a BCF.
All BCFs in tissues on lipid weight basis

Table 5
BCFs of PCDD/Fs of different tissues in meat producing animals derived from literature and classified in hierarchical order. Colors signification: dark grey — high level of BCF (over 25), grey — medium level of BCF (over 10 to 25), light grey — low level of BCF (overpass 1 and lower 10), white — no bioconcentration (BCF<1).

Tissue	Study[1]	Exposure (days)	2,3,7,8-TCDD	1,2,3,7,8-PeCDD	1,2,3,4,7,8-HxCDD	1,2,3,6,7,8-HxCDD	1,2,3,7,8,9-HxCDD	1,2,3,4,6,7,8-HpCDD	OCDD	2,3,7,8-TCDF	1,2,3,7,8-PeCDF	2,3,4,7,8-PeCDF	1,2,3,4,7,8-HxCDF	1,2,3,6,7,8-HxCDF	1,2,3,7,8,9-HxCDF	2,3,4,6,7,8-HxCDF	1,2,3,4,6,7,8-HpCDF	1,2,3,4,7,8,9-HpCDF	OCDF
Liver	A (broilers)	39	21	14	16	14	16	13	5	17	15	13	18	16	14	16	7	10	2
	B (lambs)	113	11	20	29	20	9	11	3	1	1	103	43	49	LQ	43	19	21	3
Abdominal fat	C (broilers)	7 (short)	3	2	2	3	1	LQ	1	2	2	2	1	1	LQ	1	0.3	0.4	LQ
	C (pigs)	7 (short)	1	1	1	1	0.3	1	1	0.1	LQ	1	1	1	LQ	0.3	0.4	0.2	LQ
Kidney fat	B (lambs)	113	2	2	2	2	0.4	0.2	0.04	0.1	0.1	2	1	1	LQ	0.4	0.1	0.1	0.01
Muscle	B (lambs)	113	2	2	1	1	LQ	0.14	0.03	LQ	LQ	1	0.5	0.4	LQ	0.3	0.1	LQ	LQ
Belly	D (pigs)	126	0.1	0.1	0.2	0.1	0.03	0.2	0.2	0.01	0.005	0.1	0.2	0.1	n.d.	0.1	0.1	0.1	0.01
Fore-end	D (pigs)	126	0.03	0.02	0.05	0.03	n.d.	0.06	0.07	n.d.	0.03	0.04	0.04	0.02	0.03	n.d.	0.03	n.d.	0.01
Loin	D (pigs)	125	0.03	0.03	0.05	0.03	0.01	0.06	0.01	LQ	LQ	0.03	0.05	0.02	n.d.	0.02	0.04	0.02	0.001

[1] Studies: **A** - Parera et al., 2008, **B** - Hoogenboom et al., 2015, **C** - Hoogenboom et al., 2004, **D**- Spitaler et al., 2005
LQ – values under the limit of quantification did not allow to calculate a BCF.
All BCFs in tissues on lipid weight basis

Table 6
BCFs of PCBs of different tissues in food producing animals derived from literature and classified in hierarchical order. Colors signification: dark grey — high level of BCF (over 25), grey — medium level of BCF (over 10 to 25), light grey — low level of BCF (overpass 1 and lower 10), white — no bioconcentration (BCF<1).

Tissue	Study[1]	Exposure (days)	28	52	77	81	101	105	114	118	123	126	138	153	156	157	167	169	180	189
Liver	A (hens)	7 (short)		0	0.2	0.2	0	0	0	0		0.4	0	0	0	0	0	1	0	0
	B (dairy cows)	17-38										21						5		
	C (hens)	54			2	3						8						7		
	D (goats)	80			1			3		3	3	23	10		4	5	5			
	E (lambs)	113	1	LQ	0.1	5	0.4	6	LQ	4	LQ	46	27	62	9	15	4	26	25	LQ
Serum	C (hens)	54			2	8						15						16		
Abdominal fat	A (hens)	7 (short)	0.5	0.2	2	2	0.4	1	1	1		2	2	2	1	1	1	1	2	1
	F (broilers)	7 (short)	1	1	2	3	1	3	2	2		4	2	2	2	3	3	3	2	2
	F (pigs)	7 (short)	0.2	1	0.1		1	1	1	1		1	1	1	1	1	1	1	1	1
	C (hens)	54			1	4						11						10		
	D (goats)	80			0.4			3		6	1	4	4		6	6	7			
	E (lambs)	113	1	LQ	0.1	1	0.3	5	LQ	4	LQ	2	3	16	14	18	4	13	14	LQ
Muscle	C (hens)	54			6	10						10						11		
	E (lambs)	113	1	LQ	0.2	1	LQ	3	LQ	3	LQ	2	2	8	8	11	3	8	7	LQ
Heart	C (hens)	54			2	4						6						7		
Kidney	C (hens)	54			3	4						8						8		

[1] Studies: **A** - Traag et al.,2006, **B** - Huwe et al.,2005, **C** - Pirard and De Pauw 2005, **D**- Ounnas et al., 2010, **E** – Hoogenboom et al., 2015b, **F** - Hoogenboom et al.,2004.
LQ – values under the limit of quantification did not allow to calculate a BCF.
All BCFs in tissues on lipid weight basis

3. Results

3.1. Transfer into milk and eggs (TRs)

3.1.1. PCDD/Fs

The TRs of PCDD/F congeners (means and standard deviations) into milk and eggs were presented together with the associated transfer levels, their toxicity (TEF) as well as their chemical characteristics Cl$_n$, log K$_{ow}$ and MW in Table 2.

Generally, all studied PCDD/Fs congeners were transferred to milk or eggs at a very variable degree. Unfortunately, the most toxic compounds (i.e. TCDD and 1,2,3,7,8-PeCDD) showed the highest transfer to these food products with over 26% and over 35% for milk and eggs respectively (Table 2). Hexachlorinated dioxins had a medium transfer level to milk (13−23%) but were highly transferred to eggs (29−43%). Hepta- and octachlorinated dioxins were generally transferred at a low level, but always less in milk (<4%) than in eggs (7−16%) (Table 2). Also for PCDFs, transfer to eggs is generally higher than to milk. Tetra- and pentachlorinated furans had highest TR levels in eggs (i.e. 38%−40%) but not in milk where only 2,3,4,7,8-PeCDF had a TRs of over 35%. Nevertheless, this pentachlorinated furan was transferred very differently to milk depending on studies resulting in an important SD (Table 2). As for dioxins, hexachlorinated furans showed medium TRs to milk

(11–19%) in contrast to systematic high transfer levels to egg (23–40%).

3.1.2. Transfer of PCBs

The transfer of PCBs into milk and eggs were also presented as TRs (means and SD) and transfer levels (high, medium and low) in Table 3 together with their characteristics: TEF (WHO$_{05}$), Cl$_n$, log K$_{ow}$, substitution type and MW.

The transfer levels to eggs were classified generally as high except the di-*ortho* congeners 52 and 101. Except these latest, all congeners were transferred at over 30% to eggs and reached the highest TRs of 74 or 80% respectively for the PCBs 123 and 167. The analysis of transfer to milk was built on a quite solid dataset of, depending on the congener, three to six studies (Table 3) even if some TRs to milk were associated to high SD. This generally reflect a study effect and therefore warrants a cautious generalization (for ex. congeners 28, 52, 77,105 and 118). Except PCB 123, the most lipophilic congeners (i.e. log K$_{OW}$ > 6.5) presented high TRs to milk of over 40% (Table 3). By the way, low or medium transferred congeners were characterised by lower MW and number of chlorination, again except PCB 123.

Nine out of 12 dioxin-like PCBs were classified as highly transferred in both food products. A special attention should be paid to the congeners presenting the highest TEFs: 126 and 169, which appeared to be highly transferred to milk and eggs with average TRs of respectively 39% and 40%. Two dioxin-like, non-*ortho* congeners (77 and 81) showed high transfer levels in the egg study (>30%) but much lower transfer to milk (<13%) (Table 3). Only two non-dioxin-like congeners (52 and 101) presented generally low transfer levels to milk and eggs (around 5%).

The PCA of the transfer of PCBs in milk (Fig. 1A) and eggs (Fig. 1B) confirmed the relationship of the transfer degree to the chemical characteristics of the congener. The first axis (F1) was mainly composed by MW, number of chlorination and lipohilicity (i.e. log K$_{OW}$) explained 57.3% and 70.7% of the variation of TRs respectively to milk and eggs. The second axis (F2) was represented by the substitution type and explained 20.1% and 24.7% of TR variations respectively for milk and eggs. Indeed, the first two axes of PCA explained much better the variability of the transfer into eggs (95%) than this of the transfer into milk (77%). This analysis fitted generally well for 13 out of 18 congeners except PCB 28, PCB 52, PCB 77, PCB 81 and PCB 169. Pearson's correlation coefficient of MW (0.640 and 0.520), log K$_{ow}$ (0.618 and 0.578) and number of chlorine atoms (0.660 and 0.586) were significantly ($P < 0.05$) correlated with TRs into milk and eggs, respectively. Contrarily, the correlation between the TRs and the position of the *ortho* substitutions was not significant with Pearson's coefficient of only 0.058 and −0.048 respectively for milk and eggs (Fig. 1A and B).

3.2. Transfer to tissues (BCFs)

3.2.1. PCDD/Fs

BCFs of PCDD/Fs in different tissues were presented separately for excreting adult animals (Table 4, six studies) and rapidly growing animals (Table 5, four studies) and indicated by the shades of grey the four bioconcentration degrees (as described previously).

Despite the difficulties to compare the data of both tables (i.e. differences between the types of animals and their metabolism), bioconcentration in adult animals (Table 4) seemed higher than in rapidly growing animals (Table 5). This tendency appeared especially when BCFs in liver, fat and muscles were compared. The short time study on adults (Traag et al., 2006 in Table 4) showed clearly lower BCFs contrarily to the short time study on rapidly growing animals (Hoogenboom et al., 2004 in Table 5) whose BCFs did not really stand out in comparison to the chronic exposure studies. In

addition, the highest BCFs of PCDD/Fs were extracted from studies with a field exposure. As previously observed for TRs to excretion products, BCFs in liver and kidney of birds (i.e. hens) seemed higher than in (dairy) cows, even if few data issued from very different studies incite to compare these data very carefully.

Generally, PCDDs were bioconcentrated in all studied tissues of excreting adult animals but to a very variable extent. Therefore, it seemed difficult to establish a clear hierarchy between tissues, even if BCFs in liver seemed slightly higher (except the short time study) and muscles had a slight tendency of lower BCFs (Table 4). These large variations seemed to reflect a study effect. The bioconcentration of furans in the studied tissues looks similar. The comparison of the BCFs of the different congeners does not indicate clear differences. Indeed, the hierarchical order of congeners seemed to change from one study to another, and the lower TRs reported for hepta- and octachlorinated PCDD/Fs were not clearly confirmed for the BCFs.

In rapidly growing animals (Table 5) notable BCFs were reported only in liver and did hardly exceed 1 in the other tissues (different fats and muscles). Unfortunately, several tissues were confounded with an effect of studies and even of species but bioconcentration in fat (abdominal and kidney) seemed to vary around 1 and dropped under 1 (i.e. no clear bioconcentration) in different muscles (Table 5). In these animals, BCFs of hepta- and octachlorinated congeners were lower than in less chlorinated ones, especially hexachlorinated PCDD/Fs (Table 5).

3.2.2. Polychlorinated biphenyls

Dataset for the bioconcentration of PCBs in different tissues were derived from six studies which differed widely by the type of exposed animals and by the exposure durations (Table 6). Therefore, comparisons and generalizations were complicated despite the interesting size of the dataset.

Mainly low BCFs (i.e. <10) have been shown in short time studies (Hoogenboom et al., 2004; Traag et al., 2006) or studies on excreting adult animals (Pirard and De Pauw, 2005; Huwe and Smith, 2005; Ounnas et al., 2010). Only chronic exposure study on growing sheep (Hoogenboom et al., 2015b) reported medium or high BCFs in liver and in abdominal fat (Table 6).

The hierarchy between PCB congeners within a same tissue of a given study seemed clearer: The strongest bioconcentration was shown for congener 126, but also for some higher congeners (i.e. more chlorinated and more lipophilic) 138, 153, 156, 157, 169 and 180. Low but significant bioconcentration (i.e. BCFs up to 10) was revealed for a second group of congeners (77, 81, 105, 118 and probably 123). Finally, no or only low bioconcentrations (BCFs ≤1) were revealed for PCBs 28, 52, 101, 114, and 189. The chronic exposure study on growing lambs (Hoogenboom et al., 2015b) reported another interesting result: no (<LOQ) or very low bioconcentration were observed for PCBs 28, 52, 77, 81, 101, 114, and 123 in liver and fat, whereas medium to high BCFs were found for congeners 126 and 138 only in liver, but for PCBs 153, 156, 157, 167, 169 and 180 in both tissues.

3.2.3. Polybrominated diphenyl ethers (PBDEs)

Bioconcentration factors of PBDEs were derived from studies on lactating cows (Kierkegaard et al., 2009) or lactating goats (Ounnas et al., 2010) and presented in Table 7. Generally, bioconcentration of PBDEs seemed limited and never exceed 10. Moreover, several congeners (BDE 28, 49, 66 and 85) were not bioconcentrated and only BDEs 47, 153 and 154 reached a notable but low level of BCFs of 6–7 (Table 7). As previously noted for PCBs, the BCFs of PBDEs also increased with the lipophilicity (i.e. log k$_{ow}$) of the congeners.
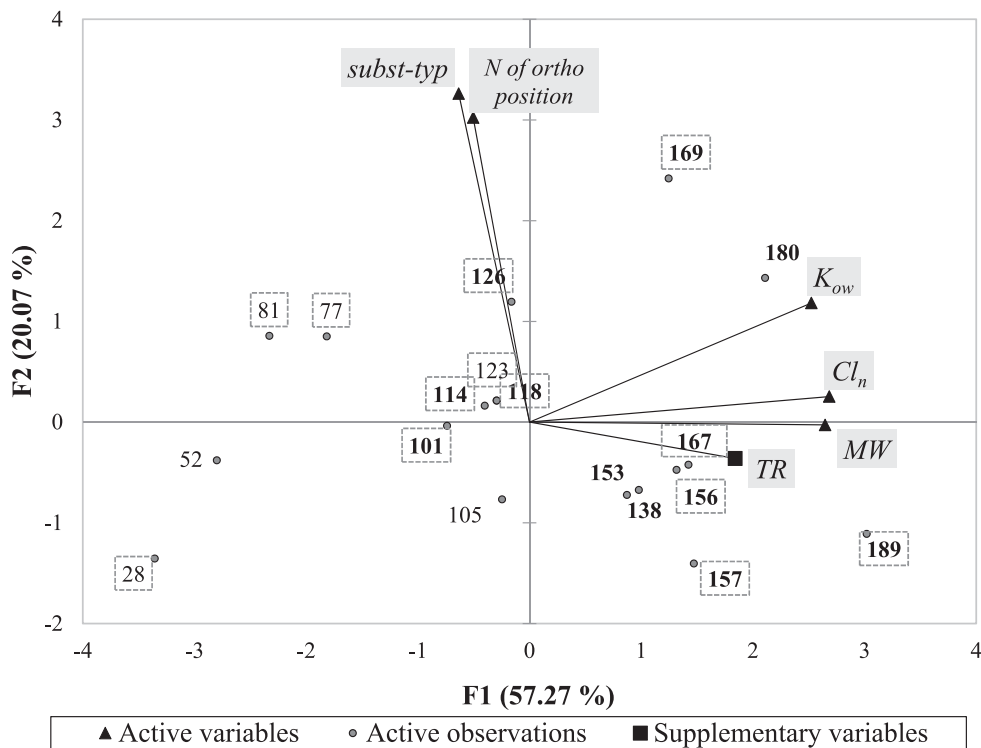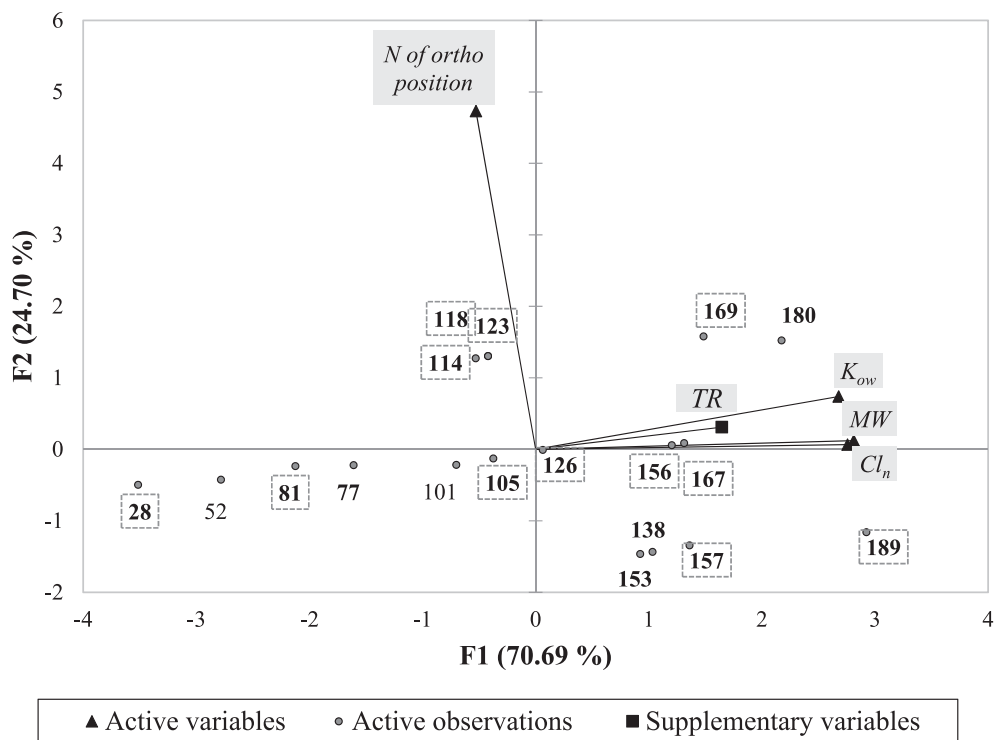
Fig. 1. Factorial plan (F1, F2) of PCA applied on mean TRs of PCBs into milk (1A) and eggs (1B), position of chlorine substitution, number of chlorines (Cl_n), molecular weight (MW), partition coefficient octanol/water (K_{ow}). Indications: Numbers correspond to the PCB congeners. Framed congeners are dioxin-like PCBs. Bold numbers were congeners transferred at a high level ranking from 38 to 78% and from 30 to 80% respectively for milk and eggs.

Table 7

Comparison of bromination (Br$_n$), molecular weight (MW), Log of partition coefficient octanol-water (Log K$_{ow}$) and bioconcentration factors of PBDEs of different tissues in cows (A) and goats (B). Colors signification: dark grey — high level of BCF (over 25), grey — medium level of BCF (over 10 to 25), light grey — low level of BCF (overpass 1 and lower 10), white — no bioconcentration (BCF<1).

Study[1]	Chemical characteristics			Tissues					
	MW	Br$_n$	Log k$_{ow}$[2]	Liver		Heart	Fat		Kidney
				A	B	A	A	B	A
BDE 28	407	3	5.9	1		1	1		0.4
BDE 47	486	4	6.8	9	5	6	5	2	2
BDE 49	486	4	6.3	LQ		LQ	0.4		LQ
BDE 66	486	4	6.3	LQ		LQ	1		LQ
BDE 85	565	5	7.4	LQ		LQ	2		LQ
BDE 99	565	5	7.3	4	3	4	4	2	LQ
BDE 100	565	5	7.2	LQ		LQ	6		LQ
BDE 153	644	6	7.9	5		6	10		3
BDE 154	644	6	7.8	9		6	7		5

[1] A - Kierkegaard et al. 2009, B - Ounnas et al. 2010

[2] Log k$_{ow}$ derived from Braekevelt et al. 2003 except for BDEs 49 and 66 which were derived from Kierkegaard et al. 2007.

LQ – values under the limit of quantification did not allow to calculate a BCF.

[3] All BCFs in tissues on lipid weight basis

3.2.4. Organochlorinated pesticides

Bioconcentrations of dichlorodiphenyltrichloroethane (DDT) and its metabolites as well as hexachlorocyclohexane (HCH) isomers in edible chicken tissues (Aulakh et al., 2006; Tao et al., 2009) and in yak tissues (Pan et al., 2014) were presented in Table 8.

BCFs of DDT ranked up to 50. Indeed, one of the most persistent organochlorine pesticide *p,p'*-DDT demonstrated high BCFs in all studied chicken tissues: stomach (50), skin (36), liver (18) and muscle (17). However, even the DDT metabolites DDD and DDE presented low to medium levels of bioconcentration (Table 8). Values from the study on lactating yaks were very low (<1). It is not

clear if this study effect can be attributed to this specie or the experimental conditions.

Similar results were obtained for HCHs which bioconcentration ranked from low to high depending on the congener and the considered tissue. Bioconcentration was highest for β −HCH (between 3 and 34). In contrast, accumulation of γ −HCH (i.e. lindane) seemed limited with BCFs, which did not overpass 3 in all studied tissues. Except γ-HCH, BCFs in liver were clearly higher than in the gizzard, in which the three other isomers were more bioconcentrated than in skin and muscle. Contrarily, bioconcentration of γ-HCH varied few between the studied tissues (Table 8).

Table 8

Bioconcentration factors of DDT and HCH isomers in different tissues of chicken (study 1: Tao et al., 2009; study 2:Aulakh et al., 2006) or yak (Pan et al., 2014). Colors signification: dark grey — high level of BCF (over 25), grey — medium level of BCF (over 10 to 25), light grey — low level of BCF (overpass 1 and lower 10), white — no bioconcentration (BCF<1).

Compound	stomach (gizzard) Chicken 1	liver Chicken 1	yak	skin Chicken 1	Chicken 1	muscle Chicken 2	yak	eggs TR Chicken 2
DDT and its metabolites								
p,p'-DDT	50	18	0.3	36	17	1	0.5	14
o,p'-DDT	10	24	na	6	6	nd	na	15
p,p'-DDE	14	5	0.5	4	6	7	0.4	58
o,p'-DDE	5	20	na	6	5	3	na	35
p,p'-DDD	14	9	nd	5	4	2	nd	54
o,p'-DDD	10	11	na	13	5	2	na	19
HCH isomers								
α -HCH	5	21	0.3	5	2	0.3	0.3	6
β -HCH	21	34	28	12	12	3	2	32
γ- HCH	3	2	0.5	3	2	1	0.9	22
δ -HCH	2	8	na	1	1	nd	na	

na: not analysed

nd: not detected

All BCFs in tissues on lipid weight basis

Recalculated TRs of DDT to eggs seemed to indicate high transfer of both OCPs as previously stated for PCDD/Fs and PCBs.

4. Discussion

4.1. Methodological considerations of the concepts of TR and BCF

TRs and BCFs are both valuable concepts to describe contaminant transfer into food products. Nevertheless, their meaningfulness is conditioned by several parameters. The main limit in transfer studies of POPs *in vivo* consists in the completion of steady state. Firstly, this commonly accepted condition (see also Material and Methods) was difficult to calculate accurately as the preciseness of modelized curves depends on the number and spacing of the experimental points. Besides the aspect of costs of numerous analysis, any approximation in the measurement of very low concentrations of POPs in the studied tissue would considerably increase the confidence interval around the asymptote of the logarithmic enrichment curve. Therefore, numerous authors calculated the necessary time of exposure *via* the half-life of the target compound but experiments were generally based on a group of congeners whereas the half-live can vary considerably between the different congeners of the same family. Indeed, half-lives have been estimated for PCDD/Fs at several weeks (7 weeks) in laying hens (Traag et al., 2004) and cows (50 days) (Firestone et al., 1979). Olling et al. (1991) estimated half-life for PCDD/Fs ranging from 40 ± 7.7 days for 2,3,7,8-TCDD to 34 ± 13 days for 1,2,3,4,6,7,8-HpCD. PCBs presented also different half-lives according to their chlorination: half-life of PCB 101 in lactating goats were reported at <4 days whereas PCB 180 showed half-life greater than 8 days (Fournier et al., 2013). Sum of PBDEs presented half-lives of 58 days in dairy cows (Fries and Marrow, 1975). Half-life of DDT in egg yolk was estimated to be of 7.6 days (Furusawa and Morita, 2001). Finally, the half-life time should be compatible with the life expectancy of the studied animal: easy to respect for animals such as laying hens (around 1 year), goats and cows (approximately 5 years), but very complicated in chicken which are generally farmed only 2 months for producing meat. Thus, the determination of the minimal exposure time will be always a subjective compromise but should target to approach as good as possible the steady state conditions in the studied animal. This motivated our initial condition to require an exposure time of around 50 days when PCDD/Fs were studied in long life cattle but accepted quite shorter exposure times for PCBs. The achievement of steady state seems impossible when tissue enrichment was studied, i.e. BCF approaches. In rapidly growing animals, the tissue enrichment by the compound intake would be permanently diluted by the body mass gain. In adults with more or less stable body weight, excretions like milk or eggs would at least slow down the achievement of steady state. Therefore, the comparison of bioconcentration between different studies, using different animals with different metabolic status, seems too difficult. Bioconcentration hierarchy can be drawn out between congeners or tissues within the same study (same exposure dose and duration, same animals) and then, these hierarchic orders between congeners or tissues can be compared. Another aspect, especially for BCFs, consisted in the comparison to the concentration in the intake matrix, generally feed. Nevertheless, it is possible that other ingested matrices as soil, water or contaminated vegetable cover would also be contaminated and leading to an underestimation of the real exposure concentration. Finally, the statistical power depends on the number of available data. Indeed, the analysis of the PCDD/Fs transfer to milk built on 8 studies seems quite solid contrarily to the OCP transfer where the small number of data makes it rather a first exploratory approach.

Accepting these minimal requirements, the concepts of TRs and

BCFs are very useful to hierarchize the extent of transfer between different tissues or also between different compounds. Such a ranking is very useful to give recommendations in risk assessments. Indeed, full interdiction of any food produced in contaminated areas makes local people completely dependent from importations while targeting only the most hazardous tissues of the contaminated animals would allow reducing significantly the consumer's exposure but maintaining local food supply.

4.2. Transfer mechanisms

Transfer of POPs to organs and excreted product results from a succession of physiological steps: Absorption, Metabolization, Distribution and Excretion.

Absorption is driven by (i) the physiological characteristics *i.e.* the animal species and (ii) the chemicals properties of the molecule. Absorption of POPs is the first key process determining the transfer of persistent compounds to livestock. Studied compounds were mainly absorbed in the intestine by crossing the epithelium wall, most of the studied compounds by a transcellular route (entering the intestinal epithelial cell by crossing the plasma membrane) (Kelly et al., 2004; Sanford, 1992). From a mechanistically point of view, lipophilic compounds are mostly transported in micelles formed by excreted bile salts and lipids present in the chyme (Kelly et al., 2004). These micelles containing lipophilic POPs, crossed an aqueous thin layer covering the intestinal wall, conceptualized and referred as the Unstirred Water Layer, before crossing intestinal epithelium cells (Kelly et al., 2004) and subsequently reaching blood or lymph streams. As these mechanisms involved aqueous phase and amphiphilic micelles, lipophilicity appears as a key characteristic explaining this absorption efficiency (McLachlan, 1993; Drouillard and Norstrom, 2003; Kelly et al., 2004). For different species like dairy cows (McLachlan, 1993) and birds (Drouillard and Norstrom, 2003), this efficiency is higher for medium lipophilic molecules and decrease after a threshold value of the partition coefficient Kow. This threshold and the extent of the reduction appears to be species-dependent (Kelly et al., 2004). In birds, models illustrate a very high absorption efficiency (close to 100%) of ingested dose until a log of Kow of 8.0, then a slow reduction of efficiency appears for higher Kow mainly explained by the ultimate Unstirred Water Layer. Indeed, this aqueous layer constitutes a physical limitation to solubilization of the most lipophilic compounds. Concerning dairy cow, and due to its particular polygastric digestive system, absorption efficiency of lipids is lower (80%) and the efficiency threshold is attained for lower Kow (7.0) (McLachlan, 1993). Then a drastic reduction of absorption efficiency occurs for more lipophilic compounds: absorption efficiency is lower than 35% for molecules displaying Kow of 8.0 (McLachlan, 1993). These elements are in line with the present results for highly chlorinated dioxins: TRs and BCFs displaying lower values than less lipophilic compounds of the same chemical family. In addition, a greater transfer reduction was obtained in milk compared to eggs, highlighting the potential difference of absorption between species for the highest lipophilic compound, especially for PCDD/Fs.

Metabolization of molecules can take place in distinct phases: in the intestinal lumen, in the liver, in the endothelial cells of blood vessels and in the deposition organs. The hepatic metabolism was the most investigated path of biotransformation of the studied compounds. The BCFs in liver were globally higher than in other organs which may result from a high first pass effect (i.e. xenobiotic metabolism occurring after absorption and before the reach of systemic circulation) for these molecules or a specific sites of binding notably enzyme binding (Ohtake et al., 2007). If few data were obtained for reared animals such as dairy cows, laying hens or

chickens, some studies highlighted the specific role of mono-oxygenase enzymes cytochrome (like CYP1A2 and CYP1A1) and AhR (aryl hydrocarbon receptor) which could explain these high concentrations in the liver (Kuroki et al., 1986; Casarett and Doull, 2008). It should also be noticed that the metabolization rate was found or suspected low for the majority of studied compounds: as shown for PCDD/Fs (Adolphs et al., 2013), PBDEs (Kohli et al., 1978), PCBs (Lutz et al., 1977; Matthews and Dedrick, 1984), DDTs (Casarett and Doull, 2008). Congeners within each POP family present distinct metabolization rates as proven for PCBs for which substitution appeared as a main factor of variation (McLachlan, 1993). However, HCH metabolization appears extensive (ATSDR, 2005) which seems in line with the reduced levels of BCFs found in chicken muscles compared to hepatic tissues. If few data are available concerning PBDE metabolization, debromination of PBDEs was suspected in dairy cows (Kierkegaard et al., 2007) as it was shown elsewhere in fish (Stapelton et al., 2004) and rats (Morck et al., 2003), without a proper demonstration of its existence, extent nor specific mechanism.

The **distribution** of POPs to organs is realized through blood. Different blood constituents such as lipoproteins (HDL, VLDL, IDL, LDL) and albumins are known to vehicles POPs (Casarett and Doull, 2008; Soine et al., 1982, Delannoy et al., *in press*). Distribution of these lipophilic POPs result in preferential accumulation of lipophilic POPs in lipid-rich organs. Found results illustrated this fact for the most lipophilic congeners (i.e. log Kow >7.2) as levels present in muscle tissue, kidneys or heart are generally lower than in adipose tissue, and ovary follicles. Concerning milk and eggs, it is already known that lactating and laying involve a remobilization of POPs from adipose tissue stock to milk and eggs (Gobas et al., 2003; Kierkegaard et al., 2007; Fournier et al., 2011).

Excretion of PCDD/Fs, PCBs, PBDEs, HCH and DDT isomers may be realized through several routes: fecal egestion, bile and urinary excretion represent secondary routes whereas milk and egg excretion constitute the major ones. The importance of excreted food product in the excretion of lipophilic compounds is related to the co-excretion of fat along egg or milk. This explains that most of Quantitative Structure Activity Relationships model to predict milk and egg excretion implements log of Kow (Gobas et al., 2003). These elements are in line with these data and could illustrate the differences found in terms of BCFs: highest BCF were found for PCDD/Fs, DDT and HCH (highest Kow), then PCB (medium Kow) and the lowest for PBDEs (low Kow).

4.3. Toxicity and implications in food safety

Lipophilic POPs are not only highly transferred to edible products such as milk, eggs, meat and offal, but even more, these food products present higher POPs exposure sources than the animal feed carrying them. In this context, PCDD/Fs appeared as the most problematic compound family regarding, notably, their reprotoxicity and carcinogenic potential along their high potency to transfer in animal food products. Indeed, BCFs higher than 10 and TRs over 33% were found for all congeners except the highest chlorinated ones, also known to be less toxic. HCH isomers present, notably, neurotoxicity, reprotoxicity and hepatotoxicity. Similarly, the most toxic congeners (γ- HCH and β −HCH) appeared also to be the most transferred to food of animal origin (Table 8, ATSDR, 2005). Concerning DDT and its *ortho* and *para* metabolites DDE and DDD similar patterns of toxicity were reported by ATSDR (2020), notably neurodevelopmental toxicity, reprotoxicity and hepatotoxicity. BCFs over 10 illustrated their potency to bioconcentrate. Finally, neurotoxicity, reprotoxicity and endocrine disruption were also reported for PBDEs (ATSDR, 2017), with higher toxicity for less brominated compounds. BDE 47 appears to be the most

problematic congener in terms of food safety as it is one of the most transferred to food product and presents high toxicity. Health effects associated to PCB exposure in humans and/or animals include hepatic, thyroid, dermal and ocular changes, immunological and neurodevelopmental alterations, reduced birth weight, reproductive toxicity, and cancer (ATSDR, 2000). They present also the highest TRs emphasizing their high potency to be transferred into milk and eggs.

5. Conclusions

The understanding of POP transfer into food of animal origin is a huge field of investigation necessary to ensure food safety and therefore the sustainability of livestock productions. Through the present meta-analysis of many but very different experimental or case studies, a better understanding of the transfer of POPs was attempted. Thus, TRs and BCFs are shown to be valuable tools to describe and assess contaminant's transfer to animals and their subsequent food-products. They allow to predict maximum POP levels when the exposure context (ingest dose of POPs) is accurately known, and play by consequence a major role in risk assessments dealing with food contaminations. However, to derive them, a careful attention must be paid to the duration of animals exposure in order to reach conditions as close as possible to steady state. For lipophilic elements such as POPs the duration could be challenging and numerous valuable articles could not reach this appropriate length. Nevertheless, selected literature provides helpful insight concerning POPs behavior and to expected levels in edible products. Lipophilicity tend to these POPs to be excreted or accumulated in fatty tissues. Unfortunately, the most toxic POPs (tetra-to hexachlorinated dioxins and furans, highly chlorinated PCBs, especially congener 126, as well as DDT) are highly transferred to the most consumed food products such as eggs, milk, and to a lesser extent also meat. The bioconcentration of these pollutants could threaten the animal rearing system. This risk must be considered in free-range rearing systems and especially when the surrounding environment of the system is contaminated.

Declaration of competing interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

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