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Mini-Review

Updated Mini-Review on Polychlorinated Diphenyl Ethers (PCDEs) in Food: Levels and Dietary Intake

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ABSTRACT

Polychlorinated diphenyl ethers (PCDEs) are a class of chlorinated aromatic compounds with structural similarities to polychlorinated biphenyls (PCBs) and polychlorinated dibenzo-p-dioxins/dibenzofurans (PCDD/Fs). Due to their physicochemical properties, PCDEs are highly resistant to degradation and tend to accumulate in soils, sediments, and aquatic organisms, making them capable of entering and persisting in the food chain. As with other persistent organic pollutants (POPs), diet represents the primary route of human exposure to PCDEs. This mini-review focuses on recent studies evaluating the concentrations of PCDEs in foodstuffs. The most recent available dietary intake, estimated in 2008, was 51.6 ng/day, showing an increase from 41 ng/day (reported in 2004). In both cases, the highest concentrations of ΣPCDEs were observed in fish and seafood (1,094.7 ng/kg wet weight in 2008). Notably, studies indicate that ΣPCDE levels are lower in cooked food samples compared to their raw counterparts. However, the potential health risks associated with dietary exposure to PCDEs remain uncertain, as no tolerable daily intake (TDI) values have been established for these compounds. This gap is directly linked to the absence of assigned toxic equivalency factors (TEFs) for PCDE congeners. Establishing TDIs and TEFs for PCDEs is critical to comprehensively assess their health risks and to inform regulatory and public health interventions.

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Polychlorinated diphenyl ethers (PCDEs) are a group of chlorinated aromatic compounds, whose chemical structure (Fig. 1) is between that of polychlorinated biphenyls (PCBs) and that of polychlorinated dibenzo-p-dioxins (PCDDs) and dibenzofurans (PCDFs) (Domingo, 2006). As PCBs, PCDD/PCDFs and PCDEs, polybrominated diphenyl ethers (PBDEs), are another group of persistent organic pollutants (POPs), with a similar structure to PCDEs. PBDEs are used in similar industrial applications, such as flame retardants in plastics and textiles, and have been widely studied due to their environmental persistence and toxicity (Linares et al., 2015). According to its chemical structure,

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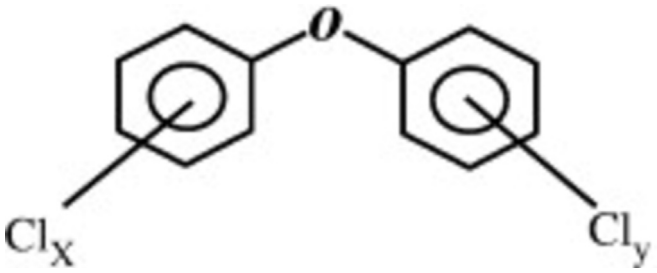

Figure 1. Chemical structure of polychlorinated diphenyl ethers ("x" and "y" represent the number of chlorine (Cl) atoms that can be substituted on each of the benzene rings).

there can be up to 209 possible congeners of this family of POPs. PCDEs are lipophilic and with a low water solubility and they have been used as hydraulic fluids, electric insulators, plasticizers, flame retardants, lubricants, as well as fungicides, slimicides, and as wood preservatives (Rappe et al., 1979; Tulp et al., 1979; Koistinen, 2000). With respect to the physicochemical properties of PCDEs, which in general terms are like those of PCBs and PCDD/Fs, for those that were not experimentally determined, in recent years, the quantitative structure–property relationships models have been established and verified (Huang et al., 2004; Sun et al., 2007; Hui-Ying et al., 2010; Zhang et al., 2018).

Sources and environmental occurrence of PCDEs

The main sources of PCDEs are the technical production of chlorinated phenols, as well as all processes of incomplete combustion of organic materials in the presence of chlorine. It has been reported that PCDEs are formed by combustion from a small home waste incinerator (Nakao et al., 2006), from condensation of chlorophenols with chlorobenzenes (Liu et al., 2008), on a simulated fly ash surface (Liu et al., 2011, 2013), from municipal solid waste incinerators during the start-up operations (Yang et al., 2015; Cheruiyot et al., 2020), and in hazardous waste thermal treatment system (Lin et al., 2024). PCDEs have been also found as common impurities in chlorophenol formulations (Nilsson and Renberg, 1974; Koistinen et al., 2007).

Despite the persistence and potential bioaccumulation of PCDEs, studies on the environmental occurrence and risks of these contaminants are rather scarce (Domingo, 2006; Wu et al., 2023). The production volumes of PCDEs have been significantly lower than those of PCBs and PBDEs. While PCDEs are mainly unintentional by-products, PCBs and PBDEs were produced in substantial quantities for specific industrial purposes. For that reason, in the scientific literature, studies on PCDEs are much more limited than those on PCBs and PBDEs. It is well known that PCDEs are quite resistant to degradation and can bioaccumulate in aquatic environments, which has been shown in a number of studies (Hornung et al., 1996; de Boer and Denneman, 1998; Koistinen et al., 2010; Ross et al., 2013; Aznar-Alemany et al., 2021; Kunisue et al., 2021; Zhang et al., 2023). PCDEs possess biomagnification potential, increasing their concentrations in various species at higher trophic levels (Koistinen, 2000; Domingo, 2006). PCDEs have been found in air (Chao et al., 2014), oceanic deep-water deposited particulates (Lee et al., 2023), river and lake waters (Qin et al., 2015; Zhang et al., 2018), suspended particulate matter in fresh surface waters (Zhang et al., 2018), soils (Frankki et al., 2006; Persson et al., 2007; Zhang et al., 2015), and sediments (Koistinen et al., 1997; Guo et al., 2014; Li et al., 2018).

Toxicity of PCDEs

Various studies showing toxic effects of PCDEs in rats and mice were already published in the 80s and 90s of the 20th century (Iverson et al., 1987; Howie et al., 1990; Kodavanti et al., 1996; Koistinen et al., 1996; Rosiak et al. 1997a, 1997b). In general, the results of those studies showed that some PCDE congeners could cause toxic effects like those also caused by some PCBs, effects that in both cases would be mediated by the Ah-receptor (aryl hydrocarbon). Some PBDEs and analogs can also interact with the Ah-receptor, while PBDEs and their metabolites can interfere with some endocrine nuclear receptors, influencing a variety of physiological processes (Zhang et al., 2022a). Howie et al. (1990) reported that the potencies of some PCDE congeners, as inducers of hepatic microsomal Ah hydroxylase (AHH) and ethoxyresorufin O-deethylase (EROD) in mice, were comparable to those of toxic PCB congeners. However, PCDEs did not cause EROD activity in rat H411E hepatoma cell line (Koistinen et al., 1996). Moreover, it was also found that PCDEs could bind to thyroid hormone receptor, altering thyroid function (Rosiak et al. 1997a, 1997b). Since the publication of the "in vivo" mentioned studies on the potential toxicity of PCDEs, until recently, there was a complete absence of new studies on that topic. However, in recent years, the toxicity of PCDEs has again been the subject of research interest, with a couple of studies conducted in aquatic organisms. Thus, Yang et al. (2022) evaluated the acute toxicity of 12 PCDE congeners in three model aquatic organisms: green algae, water flea, and zebrafish. It was found that most PCDE congeners, but particularly the congener 3,3',4,4'-tetraCDE, were highly toxic to the three species. Interestingly, it was concluded that the concentrations of 3,3',4,4'-tetra-CDE -even at environmental levels- might induce serious oxidative damages in the three aquatic organisms. In turn, Ye et al. (2022) examined in zebrafish the biomarker responses, gene expression alterations, and histological changes following exposure to PCDEs. The results showed dose-dependent changes in the antioxidant enzyme activities, malondialdehyde contents, and vitellogenin levels in the liver of zebrafish. In addition, 4,4′-diCDE exposure caused severe liver and ovary tissue damage after 14 days of exposure.

Although studies on the potential toxicity of PCDEs are certainly limited, the available results suggest that some congeners can alter the hormonal system, can cause developmental effects, and can induce hepatic monooxygenase activity, among other potential toxic effects (Iverson et al., 1987; Kodavanti et al., 1996; Koistinen et al., 1996; Rosiak et al. 1997a, 1997b; Yang et al., 2022; Ye et al., 2022). Despite this, PCDEs are not included in the list of POPs established by the Stockholm Convention, in contrast to other POPs such as PCDD/Fs and PCBs, which were already included in the 12 initial POPs, or also PCNs, and some PBDEs and per- and polyfluoroalkyl substances (PFASs), for example, which have been included more recently (Stockholm Convention, 2024).

Levels of PCDEs in foodstuffs

While general scientific information on PCDEs is rather limited, research aimed at determining PCDE concentrations in foods and assessing human dietary exposure is even scarcer, with only a few studies. This is especially relevant considering the considerable volume of data regarding other groups of polyhalogenated aromatic compounds such as PCDD/Fs and PCBs, polybrominated diphenyl ethers (PBDEs), polybrominated biphenyls (PBBs), polychlorinated naphthalenes (PCNs), and in the current century, also perfluorinated compounds (PFCs). While in recent years, the number of studies on human daily dietary intake to these compounds is certainly important (Zhao et al., 2009; De Filippis et al., 2014; Linares et al., 2015; Domingo and Nadal, 2017; Trabalón et al., 2017; Ceci et al., 2022; Marquès et al., 2022; Stadion et al., 2024), in a previous review on that topic (Domingo, 2006), only a study was found in which the concentrations of several groups of foodstuffs had been determined (Bocio et al., 2004). What are the reasons why these compounds have been much less studied than other similar families of POPs? The response

is not easy considering that -as above indicated- the toxicity of PCDEs is not at all irrelevant.

Taking the above into account, the current paper was aimed at updating my previous review (Domingo, 2006). It has been focused on the concentrations of PCDEs in foodstuffs, as well as the human exposure to these environmental contaminants through the diet. The scientific literature has been reviewed using the PubMed (https://pubmed.ncbi.nlm.nih.gov/) and Scopus (https://www.scopus.com/) databases. The following keywords -and their combinations- were used for the search: "polychlorinated diphenyl ethers", "PCDEs", "food", "dietary intake", and "human exposure". Details on the studies found in these databases are next summarized.

Dietary exposure to PCDEs

In 2004, we reported the results of the first investigation focused on quantitatively examining the concentrations of PCDEs in several food items, belonging to various food groups (Bocio et al., 2004). Until that date, in the scientific literature, there were no available studies on that subject. Only some data corresponding to the levels of PCDEs in certain animal species -considered edible- were found. However, the objectives of those studies were not directly related with the human dietary exposure to PCDEs. With respect to fish species (marine and freshwater), there were some results regarding the levels of PCDEs in salmon, pike, bream, walleye, mussel, clam, lobster and cod (Lake et al., 1981; Koistinen et al., 1993a, Koistinen et al., 1993b, Koistinen et al., 1995a; Niimi et al., 1994; de Boer and Denneman, 1998). Specific details about these studies can be found in the previous review by Domingo (2006), and in the recent one by Wu et al. (2023).

During the period 2000–2002, we conducted in our laboratory (located in Reus, Catalonia, Spain), a wide study aimed at determining the human dietary exposure to toxic metals (As, Cd, Hg and Pb) (Llobet et al. 2003a), as well as various environmental organic pollutants: PCDD/Fs (Llobet et al., 2003b), PCBs (Llobet et al., 2003c), PBDEs (Bocio et al., 2003), PCNs (Domingo et al., 2003), hexachlorobenzene (Falcó et al., 2004), and polycyclic aromatic hydrocarbons (PAHs) (Falcó et al., 2003). For the very first time, PCDEs were also included in a project of these characteristics. As for the rest of the pollutants examined, the diet was also expected to be the main route of exposure to PCDEs. A total of 54 composite samples -composed by 8 or 10 individual subsamples- were analyzed for PCDE concentrations. Samples of the following food groups were included in the study: meat and meat products, fish (fresh and tinned) and shellfish, vegetables and tubers, fresh fruits, eggs, cow's milk, dairy products, cereals, pulses, and fats and oils. Analyses of PCDEs were carried out in accordance with the US EPA 1625 method (semivolatile organic compounds by isotope dilution GC–MS). The mean recovery ratios varied from 80% for the sum of tetraCDEs to 88% for the sum of heptaCDEs. The detection limits (LOD) ranged between 5 and 40 ng/kg (fresh weigh, fw), depending on the specific food item and the respective PCDE homologues. PCDEs were only detected in the samples of fish and shellfish (hake, mussel and sardine, as well as in tuna and sardine in vegetable oil). The PCDE concentrations in the rest of food groups (meat and meat products, vegetables and tubers, fruits, etc.) were under the detection limits (for all homologues: tetra- to octaCDEs). The ∑PCDEs varied between 3.3 ng/kg fw in tuna in vegetable oil, and 400–2,707 ng/kg fw in the samples of fresh sardine. The total dietary intake of PCDEs was estimated to be 41 ng/day for a standard male adult of 70 kg body weight, aged 20–65 years. That intake was calculated assuming that if a congener was below the detection limit, its concentration would be equal to one-half of the respective LOD (ND = ½LOD). Although a potential toxicity like that of PCBs was suggested for PCDEs (Safe, 1990; Hornung et al., 1996), when that study was performed, TEFs for TEQ calculations for PCDEs were not available (Bocio et al., 2004). Interestingly, these TEFs have not been yet established. Therefore, in that study, we could not determine the TEQ values corresponding to the dietary intake of PCDEs. However, it was concluded that the PCDE contribution to total TEQ should not be underrated. Considering that some families of POPs may act through similar mechanisms of toxicity, this could be particularly concerning for individuals whose diets include significant amounts of fish and seafood.

One of the most important limitations of the survey by Bocio et al. (2004) was the very scarce number of fish and shellfish species that were analyzed. Being that food group the only one in which PCDEs were detected, in 2005, we conducted a new study in which the number of species of fish and seafood was extended to 14 (sardine, tuna, anchovy, mackerel, swordfish, salmon, hake, red mullet, sole, cuttlefish, squid, clam, mussel, and shrimp) (Domingo et al., 2006). A total of 42 composite samples (3 for each marine species) were analyzed. The detection limits for PCDEs ranged between 0.2 and 0.4 ng/kg fw). The highest mean PCDE (tetra- to octa-) concentrations corresponded to red mullet (7,088 ng/kg fw), followed by sardine (1,829 ng/kg fw), and anchovy (1,606 ng/kg fw). In contrast, shrimp (27 ng/kg fw), clam (48 ng/kg fw) and cuttlefish (50 ng/kg fw) were the species with the lowest ∑PCDEs. In shrimp and cuttlefish samples, some PCDE congeners were even under the analytical LOD. Contrary to bioaccumulation principles, smaller species like sardines and anchovies contained higher concentrations of total PCDEs than larger predatory fish such as tuna and swordfish. In general, there was a predominance of the hexaCDEs, with the exceptions of clam and mussel, for which tetraCDEs were the predominant group. An important increase was found when the mean of total PCDEs (1,095 ng/kg) was compared with that found in the previous survey, 41.8 ng/kg (Bocio et al., 2004). Nevertheless, when the comparison was carried out with only the three common species (sardine, mussel, and hake) that were analyzed in both studies, the differences were rather scarce, with just some small increases. In relation to the total intake of PCDEs through the consumption of fish and seafood, it was estimated to be 38.4 ng/day, with the highest contributions corresponding to tuna (13.1 ng/day) and hake (7.3 ng/day). Clam (0.01 ng/day) and shrimp (0.10 ng/day) were the lowest contributors to the intake of PCDEs. In our previous survey (Bocio et al., 2004), the dietary intake of PCDEs by a standard male adult of a 70 kg body weight was 41.8 ng/day. Although the difference between both intakes of PCDEs is negligible, it should be highlighted that in the second study, fish and seafood was the only food group assessed. A limitation of that wide study, including 14 edible marine species, was the impossibility of assessing the human health risks of PCDEs, as no data on the tolerable daily/weekly/monthly intakes for these pollutants have been reported. This could be relevant considering that PCDEs might act as dioxin-like compounds, adding their potential risks to those of PCDD/Fs and dioxin-like PCBs (Domingo, 2006; Domingo et al., 2007).

In order to establish the temporal trend in the dietary intake of PCDEs by the population of Catalonia, in 2006, a new survey was conducted in our laboratory (Martí-Cid et al., 2008). In that study, samples of food items, belonging to the same groups that those included in our initial survey (Bocio et al., 2004) were analyzed. Since the concentrations of PCDEs in fish and seafood had been recently determined (Domingo et al., 2006), samples of this food group were not included in the new study. Composite samples of food items belonging to the following food groups were analyzed: meat and meat products, vegetables and tubers, fruits, eggs, cow's milk and dairy products, cereals pulses, oils and fats, and bakery products. The concentrations of PCDEs were determined based on the US EPA Method 1625. The limits of detection ranged between 0.2 and 0.4 ng/kg fw. The highest ΣPCDEs (average level of 1,094.7 ng/kg fw) corresponded to the samples of fish and shellfish, which had been recently analyzed (Domingo et al., 2006). In comparison to that average level, the ΣPCDEs found in the remaining food groups were, in fact, practically negligible

Table 1
Daily Consumption of Various Food Groups and Dietary Intake of PCDEs by Adults in Catalonia, Spain[a]

Food group	Daily consumption[b] (g)		PCDE intake[c] (ng/day)	
	2000	2006	2000	2006
Meat and meat products	185 (12.8)	172 (14.0)	0.34	0.18
Fish and seafood	92 (6.4)	68 (5.5)	38.4	50.24
Vegetables	226 (15.7)	160 (13.0)	0.12	0.13
Tubers	74 (5.1)	73 (5.9)	0.08	0.05
Fruits	239 (16.6)	194 (15.8)	0.19	0.08
Eggs	34 (2.4)	31 (2.5)	0.04	0.05
Milk	217 (15.0)	128 (10.5)	0.13	0.11
Dairy products	106 (7.3)	76 (6.2)	0.23	0.06
Cereals	206 (14.3)	224 (18.3)	1.01	0.43
Pulses	24 (1.7)	30 (2.5)	0.03	0.02
Oils and fats	41 (2.8)	27 (2.2)	0.45	0.24
Bakery products	NA	45 (3.7)		0.09
Total	1,444 (100)	1,228 (100)	41.8	51.68
			0.59[d]	0.74[d]

[a] Data of 2000 were taken from Bocio et al. (2004), while those of 2006 were taken from Martí-Cid et al. (2008). In both cases, results are given for a standard male adult of 70 kg body weight.
[b] In parentheses are the percentages of total consumption. NA: not available at that time.
[c] Data were calculated assuming that when a congener was below the detection limit, the concentration was equal to one-half the respective limit of detection (ND = ½LOD).
[d] Total intake expressed in ng/kg body weight/day.

ranging between 0.4 ng/kg fw in fruits, and 8.3 ng/kg fw in the group of oils and fats. There were important differences in the contribution of each homologue group to the ΣPCDEs. With respect to the dietary intake of PCDEs, it showed an increase of 26% between the 2000 and the 2006 surveys, with values of 41.04 and 51.68 ng/day (for a standard adult male of 70 kg body weight), respectively (Table 1). Based on the concentrations found in the analyzed samples of the different food groups, that increase was doubtless due to the high contribution to the total daily intake of PCDEs of the fish and seafood group. It is worth to highlight that while the consumption of marine species from this group by an adult male was in 2006 lower than that in 2000, its contribution to the total dietary intake of PCDEs increased by 31%: 38.43 vs 50.24 ng/day. However, it should be also remarked that in the 2000 study, samples of only three fresh edible marine species had been analyzed (Bocio et al., 2004). The absence of data from other regions/countries on the daily dietary intake of PCDEs did not allow to establish if the PCDE intakes by the population of Catalonia might be considered as "normal" values. It, together with the lack of TEFs for the different PCDE congeners, prevented drawing conclusions about the human health risks of the dietary intake of PCDEs.

In our laboratory, we also investigated the influence of various common cooking processes (fried, grilled, roasted and boiled) on the levels of several heavy metals and POPs in foods. PCDEs were one of the families of environmental pollutants also evaluated (Perelló et al., 2010). In 2007, food samples were randomly acquired in various local markets, big supermarkets, and grocery stores of Catalonia. Sixty-eight (22 raw and 46 cooked) composite samples -consisting of a minimum of 10 individual units- were analyzed. These were the selected foodstuffs: meat (veal steak, loin of pork, breast and thigh of chicken, and steak and rib of lamb), fish (sardine, hake and tuna), rice, string bean, potato, and olive oil. For each food item, two composite samples were prepared for analyses. Chemical analyses of PCDEs were based on the US EPA method 1625. The limit of detection was in the range 0.2–5 ng/kg fw. When a concentration was under the LOD, results were estimated assuming that the respective values would be equal to one-half of that LOD (ND = ½LOD). With respect to the ΣPCDEs (tetra- to octa-), the highest values corresponded to sardine (263.1, 157.8 and 306.1 ng/kg fresh weight, for raw, fried and grilled samples, respectively), followed by oil (raw and cooked, fried at two different temperatures: 100.7 (raw), 43.1 and 39.0 ng/kg (cooked), respectively). All cooking processes (except frying for sardine) increased the PCDE levels in fish and meat (except roasting for chicken) samples. The increases might be attributed to sample dehydration, resulting in more concentrated PCDEs, or potentially incomplete extraction efficiency during sample processing. However, ΣPCDE levels were lower in cooked than in the remaining raw foods analyzed (Perelló et al., 2010). When that study was carried out, there were no available reports or studies focused on assessing the effects of cooking on the levels of PCDEs in food. To the best of my knowledge, this is still the only one available in the scientific literature. Consequently, it has not been possible to provide a context for those PCDE data. Anyhow, as it has been also found for other environmental pollutants in foods (Perelló et al., 2009; Sun et al., 2023; Meschede et al., 2024), the influence of cooking on the levels of PCDEs depends not only on the specific cooking process but also even more on the specific food item (Domingo, 2011).

Human exposure to PCDEs and tissue concentrations

As it happens with human exposure to PCDEs through the dietary intake, the number of studies on the concentrations of PCDEs in human tissues is also very limited. In addition, all the available data were published in the last years of the 20th century. This is a summary of that information. As part of a program to assess human exposure to toxic chemicals, Williams and LeBel (1988) developed an analytical method for the analysis of seven chlorinated diphenyl ethers in samples of human adipose tissue, which were obtained in five Ontario municipalities located in the Canadian Great Lakes Basin. None of the analyzed chlorinated diphenyl ethers could be detected in any of the samples (detection limit: 20 ng/g). In a subsequent study conducted by the same research group (Williams et al., 1991), nonachlorodiphenylether and decachlorodiphenyl ether were detected at mean values of 1.53 and 0.38 ng/g fat, respectively. These concentrations were of the same order than those found in samples of human adipose tissue collected in USA, which were in a range between 0.020 and 2.0 ng/g fat (Stanley et al., 1991).

In Finland, Koistinen et al., 1995b analyzed the concentrations of PCDEs in human (from the Helsinki area) samples of liver, testis and adipose tissue. While some PCDE congeners were detected in adipose tissue (range of concentrations of PCDEs 99, 147 + 153 (coeluting)

and 206: 2–8 ng/g fat), PCDEs were not detectable in the samples of liver and testis. The levels of PCDEs were also measured in samples of cod (Koistinen et al., 1995a). Taking all these data together, the authors concluded that although the origin of PCDEs in human adipose tissue could be due to fish consumption, most likely, the main sources of PCDEs would be the pollution in the Finnish environment, which at that time, were the use of wood preservatives (Koistinen et al., 1995a). The fact that PCDEs were not detected in samples of human liver suggested that PCDEs were probably metabolized in the liver (Koistinen et al., 1995b). To the best of my knowledge, no other new studies regarding the concentrations of PCDEs in human tissues have been published in the scientific literature. This contrasts markedly with the notable number of articles on the levels of other POPs (PCDD/Fs, PCBs, PBDEs or PFCs, among others) in different human tissues (mainly adipose tissue, considering the lyophilic characteristics of all these POPs), which have been published in recent years (Kärrman et al., 2010; Ploteau et al., 2017; Marquès and Domingo, 2019; García et al., 2021; Sontag et al., 2021; Moriceau et al., 2022; Sousa et al., 2022; Zhang et al., 2022b; Onat et al., 2024; Palaniswamy et al., 2024).

Summary and conclusions

The main objective of this article has been to update (Domingo, 2006) the scientific information on the concentrations of PCDEs in food, and the associated human dietary exposure to these POPs, as initially reviewed by Domingo (2006). Since the publication of that article, new data on these topics have remained scarce, reflecting a broader trend observed for other critical aspects of PCDE research, including environmental levels and potential toxic effects. These additional issues are also briefly reviewed and updated in this paper.

Regarding the main focus of the current review, no new studies have been identified apart from those conducted in our laboratory (Bocio et al., 2004; Domingo et al., 2006; Martí-Cid et al., 2008; Perelló et al., 2010). When assessing the temporal trends of human exposure to PCDEs through dietary intake in Catalonia (Spain), we observed that fish and seafood consistently showed the highest ∑PCDE concentrations (1,094.7 ng/kg wet weight). This value was significantly higher than those observed in other food groups included in the dietary study. For a standard adult male weighing 70 kg, the estimated dietary intake of PCDEs was 51.6 ng/day (Martí-Cid et al., 2008), an increase from the previously reported value of 41 ng/day (Bocio et al., 2004). This increase was primarily attributed to fish and seafood consumption. This finding contrasts with trends observed for other POPs, such as polychlorinated dibenzo-p-dioxins and dibenzofurans (PCDD/Fs), polychlorinated biphenyls (PCBs), and polybrominated diphenyl ethers (PBDEs), where decreases in human dietary exposure were reported in our laboratory (González et al., 2018; Marquès et al., 2022).

A critical gap in assessing the potential health risks of dietary PCDE exposure is the absence of established tolerable daily intakes (TDIs) for these compounds. This limitation stems from the lack of assigned toxic equivalency factors (TEFs) for PCDE congeners. However, it is important to note that some studies have shown comparable structure–activity relationships between PCDEs and PCBs. Therefore, although toxic equivalency (TEQ) values for PCDEs cannot currently be calculated, their contribution to total TEQ values should not be overlooked. This consideration is particularly relevant given that diets typically consist of diverse foodstuffs, many of which may be contaminated by multiple POP subfamilies that share similar mechanisms of toxicity. This concern is especially pertinent for individuals with diets high in fish and shellfish, as these food groups generally contain the highest levels of environmental pollutants (Domingo, 2016). To enable a comprehensive assessment of the human health risks associated with PCDE exposure, it is essential to establish TDIs for these compounds. Furthermore, research groups are encouraged to conduct regular assessments of environmental contaminants in food to include PCDEs in their studies. Such inclusion would provide critical insights into the prevalence and risks of these often-overlooked POPs.

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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