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Review

# Polychlorinated naphthalenes in animal aquatic species and human exposure through the diet: a review

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**Abstract**

Polychlorinated naphthalenes (PCNs) are a group of environmental pollutants, which contain one to eight chlorine atoms per naphthalene molecule, forming a total of 75 possible congeners. Several of the PCN congeners display toxicity similar to that of 2,3,7,8-tetrachlorodibenzo-*p*-dioxin (TCDD) through AhR-mediated mechanisms. There are toxicological similarities between PCNs and other well known environmental contaminants such as polychlorinated dibenzo-*p*-dioxins (PCDDs), dibenzofurans (PCDFs) and biphenyls (PCBs). However, in contrast to these compounds, information on exposure to PCNs for non-occupationally exposed populations is rather scarce. In this article, information on human exposure to PCNs through dietary intake is reviewed. Because this information is very limited and taking into account that most data on PCN levels in potential foods concern to aquatic species, these data are also reviewed. It is concluded that further investigations on dietary intake and potential human health effects of PCNs are clearly necessary.


*Keywords:* Reviews; Aquatic species; Human exposure; Dietary intake; Food analysis; Polychlorinated naphthalenes

**Contents**



## 1. Introduction

### *1.1. Chemical structure, occurrence and uses*

Polychlorinated naphthalenes (PCNs), a family of two-ringed aromatic compounds, are ubiquitous environmental pollutants [1,2]. PCNs form a complex mixture theoretically up to 75 congeners containing from one to eight chlorine atoms per naphthalene molecule (Fig. 1). Although PCNs are structurally similar to other polychlorinated diaromatic hydrocarbons such as polychlorinated dibenzo-*p*-dioxins (PCDDs), dibenzofurans (PCDFs), and biphenyls (PCBs) [3], PCNs have not been studied as well as these known environmental contaminants. A first review on data concerning environmental pollution, chemistry,

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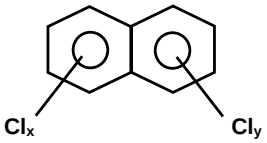


Fig. 1. Chemical structure of polychlorinated naphthalenes.

analysis, sources, formation, persistence, toxicity and behavior of PCNs was published in 1998 [2].

PCNs are hydrophobic, possess a high chemical and thermal stability, good weather resistance, electrical insulating properties, low flammability, and are compatible with other materials [2]. Although the synthesis of PCNs was known since 1833, their use as flame retardants and as a good dielectric began around 1910 [4]. The technical mixtures of PCNs are known under trade names such as Halowaxes, Nibren waxes, Seekay waxes, etc. [2]. Since PCNs have physical and chemical properties largely similar to those of PCBs, they have been used in similar industrial applications: dielectric fluids, engine oil additive, cable insulation, wood preservation, lubricants, heat exchange fluids, dye carriers, etc. [2–4]. However, because of their potential toxicity, persistence and bioaccumulation, PCNs are also substances of concern. Consequently, in spite of the numerous applications, the production of PCNs decreased in the late 1970s. Although countries such as USA voluntarily ceased PCNs production in 1980, these substances are not prohibited in a number of countries.

In the environment, the main sources of PCNs are technical formulations, and thermal and other processes in the presence of chlorine (e.g. chlor-alkali industries) [2]. In addition, PCNs are also present as byproducts in PCB formulations at levels up to 870 $\mu g\,g^{-1}$ [3,5,6]. In recent years, de novo synthesis mechanisms for PCN formulation and emission from municipal waste incinerators have been reported [7], while landfills are also potentially notable sources of PCNs because of the historical use pattern [2].

During the preparation of the United Nations-Economic Commission for Europe Convention on Long-Range Transboundary Air Pollution Protocol on Persistent Organic Pollutants (UN-ECE LRTAP-POPs Protocol) of 1998, many of the substances suggested by the member states (e.g. PCNs) were not included because of a lack of adequate information [8]. However, due to the toxicological profile and the fact that PCNs are long-ranged-transported chemicals in air, recently these compounds were also selected as a candidate to be included in the list of banned or restricted chemicals according to the UN-ECE LRTAP POPs Protocol [8].

### 1.2. Environmental levels

In recent years, it has been shown that PCNs are sufficiently persistent to reach remote regions via long-range transport. PCNs have been detected in Arctic air [9] and marine mammals [10,11]. It has also been shown that PCNs bioaccumulate in the Baltic Sea food web [12,13], as well as in the Arctic and Antarctic marine food webs [14].

Prior to 1975, there were very few reports on levels of PCNs in environmental samples [15]. However, such reports increased with the development of sensitive, congener-specific analytical methods, and increasing availability of standards. In recent years, PCNs have been detected in air [16–20], water [21–23], sediments [24–30], soils [31–33] and biota [12,25,34].

On the other hand, as other persistent lipophilic organohalogenated environmental pollutants, PCNs accumulate in the human body. Although the occurrence of PCN congeners in biological tissues has not been investigated to the same extent of other organochlorine contaminants such as PCDDs, PCDFs or PCBs, PCNs have been found in human blood plasma, adipose and liver tissue [35–37]. PCNs have been also identified in human milk, in which a notable decrease in the concentration of these pollutants was noted between 1972 and 1992 [36,38].

### 1.3. Toxicity

With regard to the toxicity of individual PCNs, experimental data are rather limited [2,4]. As with other polychlorinated diaromatic hydrocarbons, the major mechanism of action for the toxicity of PCNs in rat hepatoma cells is related to their ability to bind to and activate the aryl hydrocarbon receptor (AhR), which is a cytosolic, ligand-activated transcription factor [3]. As for some of the highly toxic planar PCBs, adverse effects include induction of aryl hydrocarbon hydroxylase and ethoxyresorufin *O*-deethylase (EROD). As a result, *in vitro* bioassays that measure AhR-dependent reporter gene activation or enzyme induction were thought to be potential useful tools for characterizing the relative potencies (REPs) of individual PCN congeners and PCN mixtures [3,39]. Among the PCNs tested in fish and rat hepatoma cells, penta-, hexa-, and heptachlorinated congeners were the most potent. In general, they were three to six orders of magnitude less potent than 2,3,7,8-tetrachlorodibenzo-*p*-dioxin (TCDD) [39], being similar to the relative potency of many PCB congeners [40]. It has been found that hexaCN congeners 63, 66/67, and 69 are the most potent. Each of these congeners was assigned a toxic equivalency factor (TEF) value of 0.002. In eggs of double-crested cormorants and herring gulls other TEF values of PCNs were 0.00017, 0.000049, 0.0015, and 0.00015 for the congeners 54, 56, 68 and 70, respectively [41]. The TEF values permit the estimation of TEQs (TCDD toxic equivalents) for PCNs by summing the product of concentrations and their corresponding TEF values. However, it is important to note that TEFs (or relative potencies) for all PCN congeners are not available due to the lack of sufficient quantities of pure, well-characterized individual congeners [39,41]. In fact, only 22 of all 75 congeners have been tested for dioxin-like toxicity [13].

To date, the environmental distribution, bioaccumulation, toxicity and other relevant aspects about PCNs have not been extensively reviewed. A search in the scientific literature indicates that since 1976 only four reviews have been published [2,4,42,43]. Moreover, data on human exposure to PCNs through the probably most important route for the general population, the diet, are only given in a recent review by Falandysz [43]. The objective of the present article was to review the data concerning potential exposure to PCNs for non-occupationally exposed populations. According to the literature, PCNs have been mainly measured in aquatic species. Therefore, most information reviewed here is related with fish and seafood.

### 1.4. Analysis

Although PCNs have been known for decades, until recently special attention to environmental analytical chemistry was not paid [44]. Most scientific research concerning environmental concentrations of PCNs has been conducted by a rather reduced number of international groups. These groups used similar methods of gas chromatography with electron capture or mass spectrometry detection [45–52]. Chromatographic features of technical PCN mixtures and PCN congeners synthesized individually have been previously discussed [39,41,44,47,52]. Moreover, some specific details have been recently reviewed by Falandysz [2,43].

The bulk of the literature data for PCNs is based on quantification performed using calibrated commercial PCN mixtures as standards. In recent years, some researchers have revised their methodologies to incorporate new standards, while others continue using the calibrated commercial mixtures [44]. To assess the comparability of data acquired using these two methods, a first interlaboratory comparison involving nine laboratories belonging to seven countries was recently carried out [44]. These laboratories were responsible for a large portion of the literature data on PCNs. The means of the reported sum of PCN values were less than 15% of the known concentrations of the two test solutions, while the relative standard deviation among laboratories was 11%. However, the variability among laboratories was in the range 20–40% for individual PCN congeners [44].

## 2. PCNs in animal aquatic species

### 2.1. Biomagnification studies

In the scientific literature, only a few studies of biomagnification of PCNs in food chain are reported: a pelagic food chain (plankton, herring, and harbor porpoise) [12], black cormorant in relation to fish [53], fish in relation to mussel [54], salmon in relation to food [55], and a marine benthic food chain [13].

Falandysz and Rappe [12] determined the concentrations of PCNs in a pelagic food chain in the southern part of the Baltic proper. Samples of mixed phyto- and zooplankton, herring (feeding exclusively on plankton), and blubber from harbor porpoise (feeding mainly on herring) were collected in 1991–1993 and analyzed for PCN concentrations. The analytical method used for the determination of these concentrations was part of a multiresidue procedure performed in parallel analysis of a number of organochlorines and polynuclear aromatic hydrocarbons (PAHs). A gas cromatograph coupled to a mass spectrometer was used to determine PCN congeners. Since the standards of individual native mono- through pentaCNs or their $^{13}C_{12}$-labelled analogues were not available during the course of the analyses, the MR (SIM) factors of hexaCNs were used [12]. When expressed on a lipid weight basis, herring showed the highest concentration (29 ng $g^{-1}$) of the total PCNs, while a spatially different gradient from 7.5 to 20 ng $g^{-1}$ was observed for a subsurface plankton. For harbor porpoise, the levels were lower and ranged between 1.7 and 2.8 pg $g^{-1}$. The profile of tetra-, penta-, hexa- and heptaCNs found in examined subsurface plankton was very similar in spite of geographically distant sampling sites. TetraCNs was the most contributing group to total PCNs in plankton, herring and harbor porpoise.

Taking into account that fish-eating birds inhabiting polluted ecosystems are among the most vulnerable species in the bioaccumulation of toxic and persistent organohalogenated compounds accumulated in fish, Falandysz et al. [53] evaluated concentrations, patterns and bioaccumulation features of PCNs in a food chain including fishes and black cormorants from the Gulf of Gdansk, Baltic Sea. Previous available data on PCN concentrations in fish from this Gulf [34] were pooled to estimate the concentration of these xenobiotics in food items of the cormorants. Species such as round goby, eelpout, herring, lesser sand eel, sand eel and lamprey were selected. It was found that related to potential food items, black cormorants biomagnified in their bodies many PCNs, with the congeners 42 and 66/67 showing the highest biomagnification factor (BMF) values.

In a study performed to evaluate the response in juvenile Baltic salmon during long-term oral exposure to a mixture of technical PCNs (0.1, 1, 2, or 10 μg $g^{-1}$ food), it was concluded that the PCN levels found in the low-dose group (0.1 μg $g^{-1}$ food) of that study were comparable with the levels found in the environment [55]. After 8 weeks of exposure, fish from the low-dose group contained 304 ng tetra- to heptaCNs per gram fat. Similar levels were previously reported by Falandysz et al. [34] in round goby of the Gdansk Basin, 260 ng $g^{-1}$ lipid weight, and by Järnberg et al. [25] in samples of pike muscle and liver, 380 and 290 ng $g^{-1}$ lipid weight, respectively, collected in Swedish lakes.

Falandysz et al. [54] investigated the bioaccumulation of PCNs in mussel, fishes and lamprey from the western part of the Gulf of Gdansk, Baltic Sea. Samples of blue mussel and lamprey, perch and flounder were collected. Young specimens of blue mussel are a main food item of flounder. For mussel, PCN concentrations ranged between 80 and 110 ng $g^{-1}$ lipid weight, while they varied between 6.3 and

8.9 ng g$^{-1}$ lipid weight, 19 and 69 ng g$^{-1}$ lipid weight, and 36 and 83 ng g$^{-1}$ lipid weight for lamprey, perch and flounder, respectively. Among the PCN congeners analyzed, most of the hexaCNs, both heptaCNs, and some tetra- and pentaCNs showed a high potency for bioaccumulation in flounder when related to mussel as their food.

Recently, Lundgren et al. [13] measured concentrations of PCNs (tetra-to-hepta congeners) in a marine benthic food chain comprising amphipods, isopods, and fourhorned sculpins. Samples were collected from five locations in the Gulf of Bothnia, northern Baltic Sea. A multiresidue non-destructive analytical procedure was applied to all the samples. Quantification of the PCNs was done by high resolution gas chromatography-high resolution mass spectrometry and based on PCN and $^{13}$C-PCB chromatographic peak areas. A $^{13}$C-labelled non-*ortho* PCB was previously added to the respective samples as an internal standard. A decrease in the sum of PCN concentrations from the lowest to the highest trophic level was demonstrated (amphipods: 10–69 ng g$^{-1}$ lipid weight; isopods: 3.9–16 ng g$^{-1}$ lipid weight; fourhorned sculpins: 0.54–1.50 ng g$^{-1}$ lipid weight). There was a similar spatial variation in the sum of PCN levels between the amphipods and the sediments (also analyzed for PCN levels). The sums of PCN concentrations in the fourhorned sculpins were very similar in all the samples analyzed.

### 2.2. PCNs in marine species

Samples of subsurface plankton, mussel, crab, lamprey, and 10 species of fish were collected in 1992 from the area of the Gdansk Basin, Baltic Sea and analyzed for PCN concentrations [34]. A gas chromatograph coupled to a mass spectrometer was used for the determination of PCN congeners. The technical mixture Halowax 1014 was used to determine elution order and pattern of PCNs in the sample chromatogram. PCNs were detected in all samples. Species such as crab, round goby, mussel and stickleback showed the highest levels of the total PCNs, which ranged between 110 (mussel) and 320 (crab) ng g$^{-1}$ lipid weight. The total PCN concentration in the remaining biological samples ranked from 6.3 (lamprey) to 89 (pikeperch) ng g$^{-1}$ lipid weight, being 36, 29 and 26 ng g$^{-1}$ lipid weight intermediate levels found in flounder, herring and lesser sand eel, respectively. The results of this study [34] concurrently with that by Falandysz and Rappe [12] suggest different absorption/retention rates and/or marked structure-dependent metabolism of some PCN congeners by marine species.

PCNs were also analyzed in blubber, nuchal fat, liver, muscle, kidney and brain of three male harbor porpoises from the west coast of Sweden [56]. The identification of PCNs was based on literature data using Halowax 1014 and $^{13}$C-labelled non-*ortho* PCB mixture run parallel to gas chromatography/mass spectrometry. The sum of total PCNs ranged between 22 (brain) and 682 (nuchal fat) pg g$^{-1}$ wet weight. The contribution of tetraCNs was largest in kidney, muscle and brain (46, 56 and 33%, respectively), while the hexaCNs accumulated mainly in lipid rich tissues and liver (66 and 67%, respectively). This contribution was different from that found in samples of harbor porpoises of the southern part of the Baltic Sea [12], in which tetraCNs (56–65%), followed by hexaCNs (22–30%) and pentaCNs (11–20%), were the main contributors to total PCNs. This distribution pattern might possibly be a result of the exposure pattern found in the major food source of the harbor porpoise, i.e. herring, which mainly contained tetra- and pentaCNs (45 and 50%, respectively) [34].

Kannan et al. [57] measured concentrations of various organochlorine compounds, including PCNs, in bluefin tuna and swordfish collected in the Mediterranean Sea near the Italian coast. Although PCNs were found in all the samples analyzed, concentrations in tuna and swordfish were lower than the PCN levels reported in flounder caught in the Gulf of Gdansk, 36–83 ng g$^{-1}$ lipid weight [54]. However, they were in a similar range than PCN levels in fourhorned sculpin of the Gulf of Bothnia [13]. The observed concentrations of PCNs in livers of tuna from the Italian coast were also less (1.36 ng g$^{-1}$ lipid weight) than those found in the livers of cod from the Baltic Sea (9.8 ng g$^{-1}$ lipid weight) [58]. PCNs were found in swordfish tissues at concentrations of 15 pg g$^{-1}$ wet weight in muscle and 63 pg g$^{-1}$ wet weight in liver.

In a recent report, Lundgren et al. [59] showed concentrations and patterns obtained in perch, herring, whitefish, whitefish roe, and sea-trout caught in the Gulf of Bothnia. The highest average concentration of the sum of PCNs was found in the sea-trout (3.0 ng g$^{-1}$ lipid weight), while the lowest average level corresponded to perch (0.22–1.20 ng g$^{-1}$ lipid weight). In herring, PCN concentrations varied between 0.41 and 0.58 ng g$^{-1}$ lipid weight. Differences in PCN patterns were noted, which might reflect a congener-specific rapid excretion, intestinal absorption, and/or metabolic transformations in the marine species. The PCN congener patterns in the whitefish and roe were similar, demonstrating a non-congener-specific transfer from fish (average concentration: 0.66 ng g$^{-1}$ lipid weight) to roe (average concentration: 2.90 ng g$^{-1}$ lipid weight).

### 2.3. PCNs in freshwater species

Kannan et al. [60] measured PCN congeners in whole body and fillet of various species of fish (largemouth bass, carp, pike, trout, walleye, whitefish and salmon) collected from Michigan waters, including the Great Lakes, during 1996–1997. Identification and quantification of individual congeners was carried out by high resolution gas chromatography/high resolution mass spectrometry. A mixture of Halowaxes 1001, 1014 and 1051 containing all the tri- through octaCNs was used as a standard. Concentrations of PCNs in fishes ranged from 0.019 to 31.4 pg g$^{-1}$ wet weight, depending on sampling location and species.

Samples of walleye and carp from the Detroit River contained the greatest PCN concentrations: 31.4 and 26.4 ng $g^{-1}$ wet weight, respectively. In turn, the lowest concentration (0.019 pg $g^{-1}$ wet weight) was observed in a pike fillet from a wetland. Whole fish contained greater PCN concentrations than did fillets and were correlated with the lipid content of fish tissues. PentaCNs were the most predominant homologues in all fishes except for some samples of whole lake trout, which had greater proportions of hexaCNs. With respect to the toxic potential, contribution of PCNs to sum TEQs in fishes from the Detroit River was similar to or greater than contribution by coplanar PCBs, whose concentrations were also determined in the same study [60].

### 2.4. PCNs in other aquatic species

Recently, Corsolini et al. [14] reported the results corresponding to the first study to document the occurrence of PCNs in Arctic and Antarctic organisms. PCNs were measured in tissues of polar bear from Alaska Arctic and krill, sharp-spined notothen, crocodile icefish, Antarctic silverfish, Adélie pinguin, South polar skua, and Weddell seal from the Ross Sea, Antarctic. PCNs were analyzed following the method reported by Kannan et al. [60]. PCNs could be detected in most analyzed samples. PCN concentrations varied between 1.3 pg $g^{-1}$ wet weight in notothen and 2550 pg $g^{-1}$ wet weight found in skua liver. For PCNs, as well as for other organohalogenated compounds (PCDDs, PCDFs, PCBs, etc.) also measured in the same study, the authors remarked the importance of intake via diet as migration habits.

## 3. PCNs in foodstuffs

Recently, Falandysz [43] reviewed the scientific literature on chloronaphthalenes as food chain contaminants covering their origin, physicochemical properties, toxicity, environmental concentrations and persistency, and homologue group and congener composition in various matrices. The review also covered distribution in environmental compartments and subsequent fate and migration to food sources [43]. According to the literature, only one study has examined quantitatively the levels of PCNs in various food groups and evaluated the dietary intake of PCNs by the general population [61]. In that study, the concentrations of PCNs were measured in 108 samples of foodstuffs (vegetables, tubers, fruits, cereals, pulses, fish and shellfish, meat and meat products, eggs, milk, dairy products, and oils and fats) randomly acquired in several cities of Catalonia, Spain, during June-August 2000. Composite samples were liophilized previously to analyses of PCNs, which were performed in accordance to the US EPA 1625 method (semivolatile organic compounds by isotope dilution GC/MS). The cleanup procedure and fractionation of the sample aliquot was carried out as a multiple cleanup, using adsorption chromatography, a multilayer silica column (from top to bottom: sodium sulfate, silica, silica-sulfuric acid, silica, silica-potassium hydroxide, silica), alumina columns, and gel permeation columns (BioBeads SX3). The final step involved the reduction of the PCN-containing fractions to the volume necessary for the analysis. The cleaned extract was analyzed by high-resolution gas chromatography/high-resolution mass spectrometry. Recovery rates were calculated against external reference standards. Mean recovery rates ranged from 80% (58–116%) for octaCN to 85% (55–113%) for tetraCNs.

A summary of the mean concentrations corresponding to each of the 11 food groups analyzed is shown in Table 1. The highest concentration (wet weight) of total PCNs was found in oils and fats (447 pg $g^{-1}$), followed at a notable distance by cereals (71 pg $g^{-1}$), fish and shellfish (39 pg $g^{-1}$), and dairy products (36 pg $g^{-1}$). In contrast, milk (0.4 pg $g^{-1}$) and fruits (0.7 pg $g^{-1}$) were the groups showing the lowest concentrations of total PCNs. In general terms, tetraCNs was the predominant homologue in all food groups except for fruit and pulses, which had greater proportions of hexaCNs.

Table 1
Mean concentrations (pg $g^{-1}$ wet weight) of polychlorinated naphthalenes (PCNs) in foodstuffs acquired in various cities from Catalonia, Spain[a]

| Food[b] | Sum tetraCNs | Sum pentaCNs | Sum hexaCNs | Sum heptaCNs | OCN | Sum total PCNs |
|---|---|---|---|---|---|---|
| Vegetables ($n = 16$) | 2 | 0.9 | 0.6 | 0.1 | 0.1 | 4 |
| Tubers ($n = 4$) | 1 | 0.9 | 0.6 | 0.2 | 0.2 | 3 |
| Fruits ($n = 12$) | 0.1 | 0.2 | 0.2 | 0.1 | 0.1 | 0.7 |
| Pulses ($n = 4$) | 0.6 | 0.9 | 1 | 0.2 | 0.2 | 3 |
| Cereals ($n = 8$) | 27 | 20 | 21 | 2 | 0.9 | 71 |
| Fish and shellfish ($n = 16$) | 15 | 16 | 7 | 0.9 | 0.3 | 39 |
| Meat and meat products ($n = 30$) | 10 | 5 | 2 | 0.4 | 0.3 | 18 |
| Eggs ($n = 4$) | 13 | 6 | 3 | 0.4 | 0.2 | 23 |
| Milk ($n = 4$) | 0.05 | 0.05 | 0.05 | 0.1 | 0.1 | 0.4 |
| Dairy products ($n = 4$) | 29 | 5 | 1 | 0.7 | 0.4 | 36 |
| Fats and oils ($n = 6$) | 377 | 58 | 7 | 4 | 1 | 447 |

[a] Data from Domingo et al. [61]. For calculations, undetectable concentrations were assumed to be equal to one-half of the respective detection levels.
[b] In parentheses, number of samples analyzed. Details on identification and quantification of PCNs are given in the text.

Table 2
Polychlorinated naphthalenes (PCNs) in aquatic species (a summary of recent results)

| Species | Collection site | Total PCNs (pg $g^{-1}$) | | Reference |
|---|---|---|---|---|
| | | Wet weight | Lipid weight | |
| Herring, porpoises | Baltic Sea (southern part) | – | 29, 1.7–2.8 | Falandysz and Rappe [12] |
| Fourhorned, sculpin | Gulf of Bothnia (Baltic Sea) | – | 0.54–1.50 | Lundgren et al. [13] |
| Mussel/crab, fish, various species | Gulf of Gdansk (Baltic Sea) | – | 110/320, 6.3–260 | Falandysz et al. [34] |
| Herring gulls | Michigan waters | 0.083–1.3 | – | Kannan et al. [41] |
| Mussel/lamprey, perch/flounder | Gulf of Gdansk | – | 80–110/6.3–8.9, 1.9–6.9/36–83 | Falandysz et al. [54] |
| Salmon, porpoises (tissues) | Baltic Sea, Swedish waters | 0.022–0.682 | 346–20880 | Akerblom et al. [55], Ishaq et al. [56] |
| Tuna (liver), swordfish (muscle/liver) | Italian coast of the Mediterranean Sea | 15.5/63 | 1.36 | Kannan et al. [57] |
| Cod (liver) | Baltic Sea | – | 9.8 | Järnberg et al. [58] |
| Sea-trout/perch, herring | Gulf of Bothnia | – | 3.0/0.22–1.20, 0.41–0.58 | Lundgren et al. [59] |
| Fish, various species | Michigan waters | 0.019–31.4 | – | Kannan et al. [60] |
| Fish, various species | Raisin River, USA | 0.041–22.61 | – | Hanari et al. [62] |

On the other hand, in all groups the lowest contribution to total PCNs corresponded to OCN.

In fish and shellfish, the proportion of tetra- and pentaCNs was similar (approximately 39%). Despite the differences between species analyzed, as well as geographically very distant origin and unrelated collection sites, the profiles in fish were similar to those previously reported by Falandysz and Rappe [12], Falandysz et al. [34], Kannan et al. [60], and Hanari et al. [62]. However, hexaCNs was the most contributing group to total PCNs found by Akerblom et al. [55] and Ishaq et al. [56] in juvenile Baltic salmon and tissues of porpoises, respectively. Moreover, when expressed on a wet weight basis, the concentration of total PCNs in fish and shellfish found in the study by Domingo et al. [51] was in the low part of the ranges reported in a number of studies performed in different sites in recent years (Table 2).

## 4. Human exposure to PCNs through the diet

Table 3 summarizes data on food intake and dietary intake of PCNs for a standard male adult of 70 kg body weight, living in Catalonia (Spain) [61]. Total dietary intake was estimated in 45.78 ng per day (assuming for non-detected values in foodstuffs (ND) = 1/2 limit of detection). This value is equivalent to 0.65 ng $kg^{-1}$ body weight per day. The highest contribution to this intake corresponded to oils and fats with 40% of the total intake, followed by cereals with 32%. The lowest contributions in percentage corresponded to milk and pulses, while fish and shellfish contributed with approximately 8%, a similar percentage than those of meat and meat products, and dairy products. Taking into account that TEFs are not available for all PCN congeners, the contribution of these contaminants to the total TEQs could not be determined.

Table 3
Dietary intake of PCNs by a male adult of 70 kg body weight from Catalonia, Spain[a]

| Food group | Daily consumption[b] (g) | PCN intake[c] (ng per day) |
|---|---|---|
| Vegetables | 226 (15.7) | 0.76 |
| Tubers | 74 (5.1) | 0.21 |
| Fruits | 239 (16.6) | 0.17 |
| Pulses | 24 (1.7) | 0.08 |
| Cereals | 206 (14.3) | 14.64 |
| Fish and shellfish | 92 (6.4) | 3.63 |
| Meat and meat products | 185 (12.8) | 3.25 |
| Eggs | 34 (2.4) | 0.80 |
| Milk | 217 (15.0) | 0.08 |
| Dairy products | 106 (7.3) | 3.82 |
| Fats and oils | 41 (2.8) | 18.33 |
| Total intake | 1444 (100) | 45.78 |

[a] Data from Domingo et al. [61].
[b] In parentheses, percentages of total consumption per day.
[c] PCN intake was estimated assuming that when a congener was below the limit of detection (LOD), the concentration was equal to one-half of the respective LOD.

## 5. Summary and research directions

As other polychlorinated diaromatic hydrocarbons such as the well known PCDDs, PCDFs and PCBs, PCNs are also a group of environmental toxins. As with PCDDs, PCDFs and PCBs, the major mechanism of action for the toxicity of PCNs is related to their ability to bind and activate the aryl receptor (AhR). However, at present it should be noted that TEFs for all PCN congeners are not available, and consequently, the possibility of establishing the contribution of PCNs to TEQs is rather limited. Notwithstanding, a recent investigation on PCN levels in fishes from the Detroit River indicated that the contribution of PCNs to sum TEQs was similar to or greater than that contributed by coplanar PCBs [60], suggesting that in some industrialized locations,

contribution of PCNs to TEQs might be greater enough to be of concern. Moreover, the results of another study suggested that the toxic impact of PCNs might be relatively more localized than those of PCDDs, PCDFs and PCBs [41].

Although in recent years, some investigations have determined the concentrations of PCNs in a number of aquatic species, information concerning human exposure to PCNs through fish and seafood consumption is practically non-existent [43,63]. Very scarce is also the information concerning human exposure to PCNs through dietary intake, which is in fact limited to an only study [61]. Exposure to pollutants through the diet is today of great concern, as food safety is essential in daily life of many countries. Therefore, further investigations on the role and potential human health effects of environmental contaminants such as PCNs, which are persistent, high lipophilic, and tend to bioaccumulate are clearly necessary.

Finally, and with respect to PCN analysis, the results from the first phase of a recent survey that examined the variability associated with different quantification methodologies, instrumentation and standards, suggested the need for additional interlaboratory studies [44]. The next phase of the intercalibration exercise should incorporate both a control material and natural-matrix test sample. While ongoing (2003 and 2004) the Second International Exercise for PCNs, certified reference standards are needed in order to improve analytical accuracy and comparability of data published on international scale [43].

## References


[1] J. Falandysz, C. Rappe, Chemosphere 35 (1997) 1746.
[2] J. Falandysz, Environ. Pollut. 101 (1998) 77.
[3] A.L. Blankenship, K. Kannan, S.A. Villalobos, D.L. Villeneuve, J. Falandysz, T. Imagawa, E. Jakobsson, J.P. Giesy, Environ. Sci. Technol. 34 (2000) 3153.
[4] D. Hayward, Environ. Res. 76 (1998) 1.
[5] J.G. Vos, J.H. Koeman, H.L. van der Maas, M.C. ten Noever de Brauw, R.H. de Vos, Food Cosmet. Toxicol. 8 (1970) 625.
[6] P. Haglund, E. Jakobsson, L. Asplund, M. Athanasiadou, A. Bergman, J. Chromatogr. 634 (1993) 79.
[7] F. Iino, T. Imagawa, M. Takeuchi, M. Sadakata, Environ. Sci. Technol. 33 (1999) 1038.
[8] E. van de Plassche, A. Schwegler, W. Iestra, Organohalogen Compd. 58 (2002) 89.
[9] T. Harner, H. Klyin, T.F. Bidleman, C.J. Halsall, W.M.J. Strachan, L.A. Barrie, P. Fellin, Environ. Sci. Technol. 32 (1998) 3257.
[10] B. Jansson, R. Andersson, L. Asplund, K. Litzén, K. Nylund, U. Sellström, U.B. Uvemo, C. Walhberg, U. Wideqvist, T. Osdjö, M. Olsson, Environ. Toxicol. Chem. 12 (1993) 1163.
[11] P.A. Helm, T.F. Bidleman, G.A. Stern, K. Koczanski, Environ. Pollut. 119 (2002) 69.
[12] J. Falandysz, C. Rappe, Environ. Sci. Technol. 30 (1996) 3362.
[13] K. Lundgren, R. Ishaq, B. van Bavel, D. Broman, M. Tysklind, Organohalogen Compd. 58 (2002) 109.
[14] S. Corsolini, K. Kannan, T. Imagawa, S. Focardi, J.P. Giesy, Environ. Sci. Technol. 36 (2002) 3490.
[15] F. Kover, Environmental hazard assessment report: chlorinated naphthalenes, EPA 560/8-75-001, US Environmental Protection Agency, Washington, DC, 1975.
[16] G. Dorr, M. Hippelein, O. Hutzinger, Chemosphere 33 (1996) 1563.
[17] T. Harner, T.F. Bidleman, Atmos. Environ. 31 (1997) 4009.
[18] T. Harner, R.G. Lee, K.C. Jones, Environ. Sci. Technol. 34 (2000) 3137.
[19] P.A. Helm, T.F. Bidleman, Environ. Sci. Technol. 37 (2003) 1075.
[20] R. Ishaq, C. Näf, Y. Zebühr, D. Broman, U. Järnberg, Chemosphere 50 (2003) 1131.
[21] M.J. Crookes, P.D. Howe, Environmental hazard assessment: halogenated naphthalenes, Report TSD/13, Department of the Environment, London, UK, 1993.
[22] I. Espadaler, E. Eljarrat, J. Caixach, J. Rivera, I. Martí, F. Ventura, Rapid Commun. Mass Spectrom. 11 (1997) 410.
[23] I. Martí, F. Ventura, J. Chromatogr. A 786 (1997) 135.
[24] E.T. Furlong, D.S. Carter, R.A. Hites, J. Great Lakes Res. 14 (1988) 489.
[25] U. Järnberg, L. Asplund, C. de Wit, A.K. Grafstrom, P. Haglund, B. Jansson, K. Lexen, M. Strandell, M. Olsson, B. Jonsson, Environ. Sci. Technol. 27 (1993) 1364.
[26] K. Kannan, T. Imagawa, A. Blankenship, J.P. Giesy, Environ. Sci. Technol. 32 (1998) 2507.
[27] N. Yamashita, K. Kannan, T. Imagawa, D.L. Villeneuve, S. Hashimoto, A. Miyazaki, J.P. Giesy, Environ. Sci. Technol. 34 (2000) 3560.
[28] Y. Horii, M. Okada, N. Yamashita, Organohalogen Compd. 57 (2002) 29.
[29] W. Brack, T. Kind, S. Schrader, M. Möder, G. Schüürmann, Environ. Pollut. 121 (2003) 81.
[30] K. Lundgren, M. Tysklind, R. Ishaq, D. Broman, B. van Bavel, Environ. Pollut. 126 (2003) 93.
[31] S.A. Meijer, T. Harner, P.A. Helm, C.J. Halsall, A.E. Johnston, K.C. Jones, Environ. Sci. Technol. 35 (2001) 4205.
[32] M. Krauss, W. Wilcke, Environ. Pollut. 122 (2003) 75.
[33] M. Schuhmacher, M. Nadal, J.L. Domingo, Environ. Sci. Technol. 38 (2004) 1960.
[34] J. Falandysz, L. Strandberg, P.A. Bergqvist, S.E. Kulp, B. Strandberg, C. Rappe, Environ. Sci. Technol. 30 (1996) 3266.
[35] D.T. Williams, B. Kennedy, G.L. LeBel, Chemosphere 27 (1993) 795.
[36] A. Lundén, K. Norén, Arch. Environ. Contam. Toxicol. 34 (1998) 414.
[37] C. Weistrand, K. Norén, J. Toxicol. Environ. Health 53 (1998) 293.
[38] K. Norén, D. Meironyté, Chemosphere 40 (2000) 1111.
[39] D.L. Villeneuve, K. Kannan, J.S. Khim, J. Falandysz, V.A. Nikiforov, A.L. Blankenship, J.P. Giesy, Arch. Environ. Contam. Toxicol. 39 (2000) 273.
[40] M. van den Berg, R.E. Peterson, D. Schrenk, Food Addit. Contam. 17 (2000) 347.
[41] K. Kannan, K. Hilscherova, T. Imagawa, N. Yamashita, L.L. Williams, J.P. Giesy, Environ. Sci. Technol. 35 (2001) 441.
[42] U.A. Brinkman, H.G. Reymer, J. Chromatogr. 127 (1976) 203.
[43] J. Falandysz, Food Addit. Contam. 20 (2003) 995.
[44] T. Harner, J. Kucklick, Chemosphere 51 (2003) 555.
[45] U. Järnberg, L. Asplund, E. Jakobsson, J. Chromatogr. A 683 (1994) 385.
[46] T. Harner, T.F. Bidleman, Atmos. Environ. 31 (1997) 4009.
[47] T. Harner, T.F. Bidleman, J. Chem. Eng. Data 43 (1998) 40.
[48] T. Imagawa, N. Yamashita, Chemosphere 35 (1997) 1195.
[49] P.A. Helm, L.M. Jantunen, T.F. Bidleman, F.L. Dorman, J. High Resolut. Chromatogr. 22 (1999) 639.
[50] P.A. Helm, T.F. Bidleman, G.A. Stern, K. Koczanski, Environ. Pollut. 119 (2002) 69.
[51] P.A. Helm, L.M. Jantunen, J. Ridal, T.F. Bidleman, Environ. Toxicol. Chem. 22 (2003) 1937.
[52] Y. Horii, K. Kaminaka, K. Ono, M. Okada, T. Imagawa, N. Yamashita, Oganohalogen Compd. 50 (2001) 75.
[53] J. Falandysz, B. Strandberg, L. Strandberg, P.A. Berqvist, C. Rappe, Sci. Total Environ. 204 (1997) 97.

[54] J. Falandysz, L. Strandberg, P.A. Berqvist, B. Strandberg, C. Rappe, Sci Total Environ. 203 (1997) 93.
[55] N. Akerblom, K. Olsson, A.H. Berg, P.L. Andersson, M. Tysklind, L. Förlin, L. Norrgren, Arch. Environ. Contam. Toxicol. 38 (2000) 225.
[56] R. Ishaq, K. Karlson, C. Näf, Chemosphere 41 (2000) 1913.
[57] K. Kannan, S. Corsolini, T. Imagawa, S. Focardi, J.P. Giesy, Ambio 31 (2002) 207.
[58] U. Järnberg, L. Asplund, C. de Wit, A. Egebäck, U. Wideqvist, E. Jakobsson, Arch. Environ. Contam. Toxicol. 32 (1997) 232.
[59] K. Lundgren, R. Ishaq, B. van Bavel, D. Broman, M. Tysklind, Organohalogen Compd. 62 (2003) 17.
[60] K. Kannan, N. Yamashita, T. Imagawa, W. Decoen, J.S. Khim, R.M. Day, C.L. Summer, J.P. Giesy, Environ. Sci. Technol. 34 (2000) 566.
[61] J.L. Domingo, G. Falcó, J.M. Llobet, C. Casas, A. Teixidó, L. Müller, Environ. Sci. Technol. 37 (2003) 2332.
[62] N. Hanari, K. Kannan, Y. Horii, S. Taniyasu, N. Yamashita, D.J. Jude, J.P. Giesy, Organohalogen Compd. 58 (2002) 117.
[63] B.E. Erickson, Environ. Sci. Technol. 37 (2003) 204A.