PCB Problems in the Future: Foresight from Current Knowledge

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ABSTRACT

The present paper overviews the forthcoming PCB problems from current knowledge of their use, environmental contamination and toxicology. From a global point of view, PCB levels in the environmental media and biota are unlikely to decline in the near future due to the greater quantities of PCBs still in use than the quantity that has already escaped into the open environment. Considering all the information on the occurrence, distribution and behaviour of PCBs in the ecosystems, the marine mammals are probably the most vulnerable and possible target organisms in forthcoming long-term PCB toxicity. The recent isomer-specific analyses suggest that the intrinsic toxicity of PCBs principally resulted from the coplanar PCB congeners which may impose a greater toxic threat than chlorinated dioxins and furans to humans and wildlife. The measures necessary to reduce further discharge of PCBs into the environment should be set in motion immediately, otherwise there may be a subsequent deleterious biological impact.

INTRODUCTION

'Polychlorinated biphenyls (PCBs)' is a generic name of many isomers and congeners with different numbers of chlorine atoms substituted in biphenyl rings. This group of chemicals was first synthesised by Schmidt & Schulz (1881) before the turn of the century and has been produced commercially since 1929. From the latter half of the 1950s, PCB production in OECD member countries (contributing substantially to the world production) increased drastically and its peak was at the end of the 1960s (Bletchly, 1984).
After the discovery of their widespread environmental contamination in the 1970s, PCB production decreased, but significant quantities are still in use, primarily in older electrical equipment.

Of the huge number of man-made organic chemicals, PCBs are one of the most widely studied chemicals in terms of environmental contamination and toxicology with a great social concern. Since the first discovery of the environmental occurrence of PCBs by a Swedish scientist, Jensen (1966), a large number of reviews on various aspects of PCBs has been published. Because of their large production and worldwide use, PCBs have already extended their boundaries of distribution over the global environment, as is evidenced by their detection in the Arctic and Antarctic atmosphere, hydrosphere and biosphere (e.g. Atlas et al., 1986; Tanabe & Tatsukawa, 1986). The persistent and bioaccumulative nature of PCBs have been recognised particularly in aquatic ecosystems, where the stepwise accumulation in higher-ranking predators of the food-chain is rather common (e.g. Phillips, 1980; Reijnders, 1980; Tanabe et al., 1984b). Due to the high lipophilicity and low biodegradability, the preferential persistency of PCBs has also been observed in birds and terrestrial mammals as well as humans (e.g. Jensen et al., 1977; Wasserman et al., 1979; Clark, 1981; Kimbrough, 1987; Peakall, 1987; Yakushiji, 1988). Although the environmental toxicity of PCBs is not yet clearly demonstrated, the probable susceptibility of wildlife to these chemicals has already been reported in many cases (e.g. Delong et al., 1973; Helle et al., 1976a,b; Helander et al., 1982; Reijnders, 1986; Martineau et al., 1987).

In 1968, accidental poisoning by cooking rice in bran oil contaminated by PCBs occurred in Japan and over 1000 patients have been suffering from various morbid symptoms and signs (Higuchi, 1976; Kuratsune, 1980). A similar poisoning outbreak occurred in Taiwan in 1979 (Chen et al., 1981; Hsu et al., 1985). The causative agents of these poisonings have been considered to be the co-contaminants of PCBs such as polychlorinated dibenzofurans that were secondarily formed during the heating of PCBs in rice bran oil (Masuda & Yoshimura, 1984). However, recent studies have demonstrated the presence of extremely toxic PCB congeners such as 3,3',4,4'-tetrachlorobiphenyl, 3,3',4,4',5-pentachlorobiphenyl and 3,3',4,4',5,5'-hexachlorobiphenyl in commercial PCB mixtures and biological samples including humans in significant quantities (Kannan et al., 1987, 1988; Tanabe et al., 1987a,b). This has awakened the re-evaluation of PCB toxicity not only to accidental cases but also to occupational exposures (industrial workers) and to environmental animals.

Considering the history of PCB use, its resultant environmental pollution and the toxicological incidents mentioned above, the PCB problem is likely to enter a new phase. The present paper overviews the forthcoming PCB
problems as well as reviewing the current knowledge of PCB contamination in the environment.

HOW FAR IS THE END POINT OF PCB POLLUTION?

Commercial PCB preparations were manufactured in several industrial countries with variable chlorine contents ranging from 20% to 60% by weight of chlorine. The physical and chemical characteristics of PCB preparations such as high stability, inertness and dielectric properties were extremely advantageous for many industrial purposes. Consequently, these versatile properties of PCBs have led to their widespread application not only in industry but also to the various spectra of human life (Hutzinger et al., 1974).

Due to the worldwide use of PCBs in large quantities, they have been identified in a wide variety of environmental media and biota as early as the 1960s (Risebrough et al., 1968; Nisbet & Sarofim, 1972; Peakall, 1975). Serious environmental contaminations of PCBs have been documented in urban and heavily industrialised areas such as the Great Lakes (Mackay et al., 1983), the Baltic Sea (Olsson, 1987) and Tokyo Bay (Tatsukawa, 1976), etc. The widespread contamination of PCBs has also been evidenced by their detection in various environmental samples from polar regions such as air (Tanabe et al., 1983a; Oehme & Mano, 1984), water, including snow and ice (Risebrough et al., 1976; Tanabe et al., 1983a), fish (Giam et al., 1973; Subramanian et al., 1983), birds (Risebrough et al., 1976; Ballschmiter et al., 1981; Norheim et al., 1982; Subramanian et al., 1986) and mammals (Addison & Smith, 1974; Bowes & Jonkel, 1975; Born et al., 1981; Hidaka et al., 1983; Tanabe et al., 1984a, 1986; Norstrom et al., 1988). The findings of the PCBs in the open ocean atmosphere (e.g. Atlas & Giam, 1981; Bidleman et al., 1981; Tanabe et al., 1982a), surface water (e.g. Bidleman & Olney, 1974; Harvey & Steinhauer, 1976; Tanabe & Tatsukawa, 1980; Tanabe et al., 1982a), subsurface water (Harvey & Steinhauer, 1976; Tanabe & Tatsukawa, 1983) and various organisms (Bennington et al., 1975; O'Shea et al., 1980; Gaskin, 1982; Wagemann & Muir, 1984; Tanabe & Tatsukawa, 1986; Tanaka et al., 1986) also ensured their penetration throughout the global environment. The environmental ubiquity of PCBs suggests the importance of atmosphere as a medium of transport to remote areas.

In the open ocean environment, the contamination of PCBs is more prominent in the northern hemisphere than in the southern hemisphere. Such a trend has also been recognised in other environmental components such as air, water, plankton, fish and marine mammals from remote oceans (Tanabe & Tatsukawa, 1986). According to the survey of the western North
and South Pacific including the Bering Sea and the Antarctic Ocean (Tatsukawa & Tanabe, 1984), the surface water was found to be contaminated more in the North than in the South. Interestingly, the maximum PCB contamination was observed in the mid-latitudes of the northern hemisphere (Fig. 1). This geographical distribution can plausibly be explained by the extensive production and use of PCBs in industrialised countries which are mostly located in the mid-latitudes of the northern hemisphere. Moreover, a similar distribution was also reported in marine mammals living in the same areas of water sampling (Fig. 1), indicating that PCB pollution in water also determines the degree of that in biological systems (Tanabe et al., 1983b).

In the early 1970s, Woodwell et al. (1971) and US Ocean Affairs Board (1971) predicted that the open ocean environment might serve as a reservoir of persistent pollutants. This may be true for PCBs also because of the immense volume of water in the ocean, even though the PCB concentration is expected to be generally very low. However, it was difficult to demonstrate it using the technique and knowledge of that time. In the late 1970s, the US
Environmental Studies Board (1979) estimated that about 50% to 80% of the total PCB residues in the US environment had been present in North Atlantic water. This document was the first report stating the importance of open ocean water as the sink of PCBs based on their actual measurement in various environmental samples. Following this, the global estimation had also been attempted (Tanabe, 1985; Tanabe & Tatsukawa, 1986) and a tentative result was reported as shown in Table 1. Although it is necessary to improve further the accuracy of this estimation because of non-availability of PCB data in some parts of the monitored area, important suggestions can be found in this result. PCBs currently present in the global environment were estimated to be about $370 \times 10^3$ t. Of this, almost the entire amount, approximately $360 \times 10^3$ t are retained in coastal sediments and open ocean water. Particularly the oceanic water contains the major portion of PCB residues, over 60% of the world environmental PCB loads, clearly indicating that the open ocean water serves as a vast reservoir and final sink of PCBs.

**TABLE 1**
Estimated PCB Loads in the Global Environment

<table>
<thead>
<tr>
<th>Environment</th>
<th>PCB load (t)</th>
<th>Percentage of PCB load</th>
<th>Percentage of world production</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Terrestrial and coastal</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Air</td>
<td>500</td>
<td>0.13</td>
<td></td>
</tr>
<tr>
<td>River and lake water</td>
<td>3500</td>
<td>0.94</td>
<td></td>
</tr>
<tr>
<td>Seawater</td>
<td>2400</td>
<td>0.64</td>
<td></td>
</tr>
<tr>
<td>Soil</td>
<td>2400</td>
<td>0.64</td>
<td></td>
</tr>
<tr>
<td>Sediment</td>
<td>130000</td>
<td>35</td>
<td></td>
</tr>
<tr>
<td>Biota</td>
<td>4300</td>
<td>1.1</td>
<td></td>
</tr>
<tr>
<td>Total (A)</td>
<td>143000</td>
<td>39</td>
<td></td>
</tr>
<tr>
<td><strong>Open ocean</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Air</td>
<td>790</td>
<td>0.21</td>
<td></td>
</tr>
<tr>
<td>Seawater</td>
<td>230000</td>
<td>61</td>
<td></td>
</tr>
<tr>
<td>Sediment</td>
<td>110</td>
<td>0.43</td>
<td></td>
</tr>
<tr>
<td>Biota</td>
<td>270</td>
<td>0.07</td>
<td></td>
</tr>
<tr>
<td>Total (B)</td>
<td>231000</td>
<td>61</td>
<td></td>
</tr>
<tr>
<td><strong>Total load in the environment (A + B)</strong></td>
<td>374000</td>
<td>100</td>
<td>31</td>
</tr>
<tr>
<td>Degraded and incinerated</td>
<td>43000</td>
<td>4</td>
<td></td>
</tr>
<tr>
<td>Land-stocked*</td>
<td>783000</td>
<td>65</td>
<td></td>
</tr>
<tr>
<td>World production</td>
<td>1200000</td>
<td>100</td>
<td></td>
</tr>
</tbody>
</table>


* Still in use in electrical equipment and other products, and deposited in landfills and dumps.
Considering the above-mentioned point, the behaviour of PCBs in the open ocean water should determine their fate in the global environment, thus emphasising the need to assess the details of PCB movement in the hydrosphere. As stated elsewhere, the open ocean ecosystems, particularly higher animals, are considered to be endangered by the long-term accumulation and toxicity of PCBs due to their specific biological and physiological processes. Thus, particular attention should be paid to the PCB dynamics in surface water because biological activity, and hence the bioaccumulation of PCBs, takes place mainly in the upper 100m of the water column.

Table 1 further points out an important PCB problem for the future. The cumulative world production of PCBs is estimated to be \(1.2 \times 10^6\) t (Bletchly, 1984). Of this, about 31\% (\(370 \times 10^3\) t) is present in the global environment. Although the destruction and confinement of PCBs are carried out presently following the increasing demand for restrictions on their disposal, the degradation and incineration could account only for 4\% or more of the total PCB production. After all, it can be estimated that about \(780 \times 10^3\) t of PCBs are still in use in older electrical equipment and other products and deposited in landfills and dumps or in storage. This quantity is equivalent to more than double the total PCB loads that escaped into the environment, implying that these land-stocked PCBs hold a crucial key to the forthcoming environmental pollution and possible biological effects of PCBs. It has been suggested in recent reports that PCB residue levels in the environment are gradually declining (OECD, 1980; Passivirta & Linko, 1980; Norstrom et al., 1985; Schmitt et al., 1985; Addison et al., 1986). In most cases, a significant downward trend of PCB concentrations was evident in areas where local sources were predominant such as Lake Ontario, the Baltic Sea, etc. This is reasonable to accept because of the effectiveness of restricted use and regulated storage and disposal of PCBs in these areas. However, considering from a global viewpoint, taking account of the land-stocked PCBs in large quantities, PCB levels in the general environment, particularly in remote areas, are unlikely to decline in the near future. In fact, Norstrom et al. (1988) found that PCB concentrations in Polar bears are nearly as high now as they were in the late 1960s. Bletchly (1984) estimated that the disposal of PCBs used in older transformers and capacitors will reach its peak in the next decade. These overviews strongly suggest that the end of PCB pollution is far off, unless a major effort is made to reduce the further discharge of PCBs into the open environment.

**WHAT ORGANISMS ARE VULNERABLE TO PCB TOXICITY?**

The accumulation process of pollutants in biological systems is rather complex and varies with biological, environmental and chemical factors
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(Phillips, 1980; Aguilar, 1985, 1987). In order to understand the biological effects of environmental contaminants, it is necessary to premeditate on the basic behaviour of pollutants in natural ecosystems.

As mentioned earlier, the widespread distribution of PCBs has been documented in various biological systems all over the world. The major concern regarding PCB pollution of the biota has been focused on the aquatic organisms because many factories which produced and used PCBs were located in riverine, estuarine, coastal and lake-side areas (Tatsukawa, 1976; US Environment Studies Board, 1979). The concern for PCB pollution in aquatic organisms has also been emphasised as the primary route of exposure to humans and domestic animals due to the consumption of fish, etc., from contaminated waters (Highland, 1976; Watanabe et al., 1979; Schwartz et al., 1983; Sawhney & Hankin, 1985). Although the concentrations of PCBs in environmental water samples are generally very low (parts per trillion or less) due to their hydrophobicity, detectable residues are found in aquatic organisms at greatly amplified levels because of their lipophilicity and biological stability (Jensen et al., 1969; Ten Berge & Hillebrand, 1974; Phillips, 1980, Reijnders, 1980). A study on the PCB residues in a food chain which includes plankton, fish and marine mammals from western North Pacific clearly illustrates their preferential biomagnification from lower to higher trophic levels in which the highest predator, striped dolphins, revealed the bioconcentration factor (concentration ratio of PCBs in organisms to water) as high as $10^7$ (Tanabe et al., 1984b).

It has been generally accepted by many authors that the concentrations of PCBs in gill-breathing aquatic animals are controlled by the law of equilibrium partitioning which determines the biomagnification of lipophilic pollutants (e.g. Hamelink et al., 1971; Branson et al., 1975; Clayton et al., 1977; Scura & Theilacker, 1977; Schneider, 1982; Tanabe et al., 1984b, 1987d); that is, the uptake and release of PCBs has been considered as a function of their exchange through the gills and across the body surface to equilibrate their levels between the ambient water and body lipids. Concentrations of PCBs in lower trophic levels such as plankton, crustacea, shellfish and fish, etc., therefore depend primarily on their levels in water, and the uptake of additional quantities of PCBs through the ingestion of contaminated food does not greatly influence the total concentrations attained in the organisms. Thus, the increasing concentrations of PCBs (on lipid weight-related data) with age or body-size in these lower trophic animals are a great deal less as long as PCB levels in water are in the steady state.

In contrast, the age-dependent accumulation of PCBs is often found in higher trophic levels. In marine mammals, an increasing concentration of PCBs with increasing age has been clearly recognised in many species of pinnipeds (e.g. Addison et al., 1973; Frank et al., 1973; Addison & Smith,
1974; Helle et al., 1983) and cetaceans (e.g. Gaskin et al., 1983; Tanabe, 1985; Tanabe et al., 1986; Martineau et al., 1987; Subramanian et al., 1988). This is, however, less pronounced in females than in males (Addison & Smith, 1974; Holden, 1978; Born et al., 1981; Gaskin, 1982). In female cetaceans, a sharp decline of PCB concentrations with increasing age is evident in sexually mature animals (Gaskin et al., 1983; Tanabe, 1985; Tanabe et al., 1987c; Subramanian et al., 1988). Such a specific pattern in female marine mammals has been explained by the transfer of PCBs to their offspring in appreciable quantities during lactation (Addison & Brodie, 1977; Reijnders, 1980; Gaskin, 1982; Tanabe et al., 1982b; Wagemann & Muir, 1984). A female grey seal is known to excrete 15% of its PCB body burdens through lactation (Addison & Brodie, 1977). More strikingly, in striped dolphins, over 90% of the total PCB load from the female’s body is transferred to the newborn calf via milk (Tanabe et al., 1981). This particular transfer rate is probably due to the high lipid content in the milk of this animal and the lipophilic nature of PCBs. It is commonly accepted that the lipid content in cetacean milk is as high as 30% in most species (Arvy, 1973–4). Such lipid rich milk, and hence the transfer of PCBs in large quantities, is almost rare in terrestrial mammals. Thus, in the case of cetaceans, the lactational process is of great concern in view of the long-term residues of PCBs and their possible biological impacts to the following generations. PCB contaminations in cetaceans are unlikely to decrease rapidly in the near future, even if pertinent measures to prevent their discharge into the open environment could be attained.

For all the pollutants, the degree of contamination in organisms is usually affected by the distance between their living areas and the major source of pollution. The decreasing level of pollutants with increasing distance from source is rather common. However, a recent study reports an example which is not in agreement with the above rule (Tanabe et al., 1988). As shown in Fig. 2, small cetaceans such as striped dolphins, melon-headed whales and Dall’s porpoises were found to contain much higher levels of PCBs than terrestrial mammals and birds, in spite of living in the pristine oceans far from the land-based PCB pollution source. The detection of PCBs at very high concentrations from oceanic cetaceans has also been reported by many authors (e.g. O’Shea et al., 1980; Gaskin, 1982; Wagemann & Muir, 1984; Tanabe & Tatsukawa, 1986). As examples of highest values in oceanic species, Alzieu & Duguy (1978) reported \(833 \mu g g^{-1}\) of PCBs in a blue-white dolphin from the coast of France and Ono et al. (1987) recently found in killer whale \(410 \mu g g^{-1}\) of PCBs in wet blubber. In the course of the study by Tanabe et al. (1988), it has also been found that PCB isomer and congener compositions are apparently different in different animal species, where large numbers of residual PCB components and enrichment of lower
chlorinated biphenyls (tri-, tetra- and pentachlorobiphenyls) were obviously more in small cetaceans than in other higher animals such as seals, sea birds and terrestrial mammals. Subsequent investigations on the cause of the above observation revealed that these animals have a particular mode of PCB metabolism which also means smaller capacity for their degradation. They have no capacity to metabolise a group of PCB components with adjacent non-chlorinated meta and para carbons in biphenyl rings. The comparative approach of PCB isomer and congener compositions in different species of higher animals further suggests that, as shown in Fig. 3, drug-metabolising enzyme systems in small cetaceans have smaller function of MC (3-methylcholanthrene)-type enzymes but no function of PB (phenobarbital)-type enzymes (Tanabe et al., 1988). Walker (1983) reported that the hepatic microsomal monooxygenase activities are apparently lower in fish-eating birds than in mammals (rats). As seen in Fig. 3, the low enzyme activities were estimated not only in fish-eating birds but also in piscivorous mammals such as small cetaceans, seals and mink. Probably due to this, a remarkable bioaccumulative tendency of PCBs has been found in these animals.

It is well known that the mink is extremely sensitive to the reproductive toxicity of PCBs (Jensen et al., 1977; Aulerich & Ringer, 1977; Bleavins et al., 1980; Hornshaw et al., 1983; Aulerich et al., 1985). In natural biological systems, it seems likely that susceptible toxicological effects of PCBs and related compounds have occurred primarily in pinnipeds (Delong et al., 1973; Helle et al., 1976a,b; Duinker et al., 1979; Reijnders, 1986) and birds (Helander et al., 1982; Newton et al., 1982; Wiemeyer et al., 1984; Hoffman et al., 1986; Peakall & Fox, 1987), especially causing reproductive abnormalities. Interestingly, these animals are estimated to have smaller capacity for PB- or MC-type enzymes (Fig. 3). This may emphasise the possibility that the drug-metabolising enzyme systems are responsible for
the reproductive toxicity of these chemicals—the animals with low enzyme activities are vulnerable to this toxicity. If so, small cetaceans may be considerably susceptible to the reproductive impacts of these chemicals because of deficiency in PB-type enzyme systems and also low activity of MC-type enzymes. Recent reports which documented the possible reproductive effects of PCBs and related compounds to small cetaceans (Martineau et al., 1987; Subramanian et al., 1987) may support the above point.

As stated earlier, cetaceans have been recognised as animal species receiving these pollutants arising out of a worldwide contamination at high concentrations. They can amplify much greater amounts of PCBs through feeding and also pass them in large quantity from one generation to the next through lactation. Unfortunately, these animals have smaller capacity for PCB degradation resulting from lower activity of specific drug-metabolising enzyme systems. Moreover, these particular enzyme systems may be related to the possible reproductive effects of PCBs. Considering all these facts, marine mammals, particularly cetaceans, are probably the most vulnerable and possible target organisms in forthcoming long-term PCB toxicity.

**WHAT ISOMERS ARE INTRINSIC IN PCB TOXICITY?**

PCBs consist of 209 theoretically possible isomers and congeners having different toxic and biologic responses. Although PCB toxicity varies largely
with chlorine numbers and their substituted positions in biphenyl rings, it has been generally known that the acute toxicity (LD$_{50}$) of commercial PCB formulations increases with increasing chlorine contents in the order of Aroclors 1221, 1232, 1242, 1248 and 1254, but highly chlorinated Aroclors 1260, 1262 and 1268 are less toxic than Aroclor 1254 (Kimbrough et al., 1978; Safe, 1984). The long-term toxicity of PCBs has also been pronounced in Aroclor 1254 and Kanechlor 500 as most toxic formulations (Kimbrough et al., 1978). In the light of toxicological investigations, it is believed that the polychlorinated dibenzofurans contained in commercial PCB preparations as co-contaminants are rather responsible for the toxic action of PCBs. Besides this, several earlier studies have indicated that the toxic nature of technical PCB mixtures may be associated with the presence of trace levels of particular toxic PCB congeners having four or more chlorine atoms at both para and meta positions in the biphenyl rings but no chlorine atoms in ortho positions (McKinney et al., 1976; Poland & Glover, 1977; Yoshimura et al., 1978).

Among 209 theoretical PCB isomers and congeners, 20 members attain coplanarity due to non-ortho chlorine substitution in the biphenyl rings. In this group, three coplanar congeners such as 3,3',4,4'-tetrachlorobiphenyl (T$_4$CB), 3,3',4,4',5-pentachlorobiphenyl (P$_5$CB) and 3,3',4,4',5,5'-hexachlorobiphenyl (H$_6$CB) are approximate isostereomers of highly toxic 2,3,7,8-tetrachlorodibenzo-p-dioxin (T$_4$CDD) and 2,3,4,7,8-pentachlorodibenzofuran (P$_5$CDF) (Fig. 4) and hence elicit similar toxic and biologic responses typical of dioxins and furans (Safe, 1984). In the study of acute toxicity for coplanar PCB congeners, McKinney & McConnell (1982) found that 3,3',4,4'-T$_4$CB and 3,3',4,4',5,5'-H$_6$CB were several hundred times less toxic than 2,3,7,8-T$_4$CDD in guinea pigs. Yoshihara et al. (1981) also reported very high toxic potency of 3,3',4,4',5-P$_5$CB that was only 10 times
less toxic than 2,3,4,7,8-P}_{5}\text{CDF} in rats. They further confirmed the highly toxic clinical signs in guinea pigs exposed to 3,3',4,4',5-P}_{5}\text{CB (Yoshimura et al., 1981). The toxic characterisation of these coplanar PCB congeners has also been performed in many other experiments and resulted in serious toxic and biological effects such as body weight loss, thymic atrophy, dermal disorder, hepatic damage, teratogenicity, reproductive toxicity, immunotoxicity, high binding affinity to hepatic cytosolic receptor protein and high induction potency of 3-methylcholanthrene type hepatic microsomal enzymes (e.g. Goldstein et al., 1977; McConnell & Moore, 1979; Yoshimura et al., 1979, 1981, 1985; Marks et al., 1981; Poland & Knutson, 1982; Yoshihara et al., 1982; Harris & Bradshaw, 1984; Safe, 1984; Aulerich et al., 1985; McNulty, 1985). It was also observed in the literature that 3,3',4,4',5-P}_{5}\text{CB was the most toxic of the three coplanar PCBs as well as of other PCB isomers and congeners. A recent study of Quantitative Structure-Activity Relationships (QSARs) indicates that the high biological potency of coplanar PCB congeners in AHH (aryl hydrocarbon hydroxylase) induction and binding for the rat 2,3,7,8-T}_{4}\text{CDD cytosolic receptor protein is likely responsible for their toxic effects such as body weight loss and thymic atrophy (Safe et al., 1985). These facts suggest the significant contribution of coplanar PCB congeners for possible environmental impacts of PCBs.}

In contrast to the many evidences on the extreme toxic potential of coplanar PCB congeners, very limited information is available on their environmental contamination and possible effects in wildlife and humans. Until 1986, only four reports have dealt with the environmental residues of coplanar PCB congeners which were the detection of 3,3',4,4'-T}_{4}\text{CB in fish from Hudson River (Stalling et al., 1980), Ohio River (Huckins et al., 1980) and Green Bay, Lake Michigan (Trotter et al., 1982) and all the three coplanar PCBs in Forster's tern eggs from Green Bay (Harris et al., 1985). The contents of these toxic coplanar PCBs in commercial PCB preparations have also been not fully understood. Only three reports have appeared on 3,3',4,4'-T}_{4}\text{CB in Aroclors (Albro & Parker, 1979; Kamops et al., 1979; Huckins et al., 1980) and no quantitative data are available on the highly toxic 3,3',4,4',5-P}_{5}\text{CB and 3,3',4,4',5,5'-H}_{6}\text{CB. However, a recent development of a sensitive and simple analytical method for coplanar PCB congeners (Tanabe et al., 1987b) has made it possible to determine these toxic residues in commercial PCB preparations and environmental samples. Kannan et al. (1987) identified and quantified all the three toxic coplanar PCB congeners in Aroclors (1242, 1248, 1254 and 1260) and Kanechloris (300, 400, 500 and 600), where 3,3',4,4'-T}_{4}\text{CB was found to be highest, ranging from about 250 to 8500 \mu g g^{-1} and the toxic 3,3',4,4',5-P}_{5}\text{CB and 3,3',4,4',5,5'-H}_{6}\text{CB were also detected in the ranges of 8-3 to 89 \mu g g^{-1} and <0-08 to 1-2 \mu g g^{-1}, respectively. This finding ensured the possible...}
widespread environmental contamination by these toxic congeners as in the case of PCBs in general. In fact, a recent analytical study by Tanabe et al. (1987a) demonstrated the significant residues of three toxic coplanar PCB congeners in a wide variety of environmental animals and clearly indicated that (1) these coplanar PCBs were generally found to be in a range of a few pg g\(^{-1}\) to several tens ng g\(^{-1}\) which were 3 to 5 orders of magnitude lower than total PCBs, but apparently much higher than most toxic 2,3,7,8-T\(_4\)CDD and 2,3,4,7,8-P\(_5\)CDF, (2) the environmental pollution of coplanar PCBs was already widespread all over the world, as evidenced by their detection in many wild animals collected from remote areas such as the North Pacific, (3) the pollution sources of coplanar PCBs are derived principally from commercial PCB preparations and hence the formation of these toxic congeners during combustion of chlorinated aromatics and diverse types of chemical, industrial and municipal wastes, as in the case of dioxins and furans, is a great deal less, and (4) among these three coplanar PCB congeners, 3,3',4,4'-T\(_4\)CB was estimated to be most biodegradable, 3,3',4,4',5-P\(_5\)CB is rather persistent but moderately metabolisable and 3,3',4,4',5,5'-H\(_6\)CB is almost metabolically stable.

They also determined three toxic coplanar PCB congeners in human adipose tissues of the general public in Japan (Kannan et al., 1988; Tanabe et al., 1987a). The analyses of these samples revealed 94 to 860 pg g\(^{-1}\) of 3,3',4,4'-T\(_4\)CB, 120 to 730 pg g\(^{-1}\) of 3,3',4,4',5-P\(_5\)CB and 36 to 200 pg g\(^{-1}\) of 3,3',4,4',5,5'-H\(_6\)CB on a wet weight basis. Interestingly, as in the case of wild animals, these concentrations were significantly higher than 2,3,7,8-T\(_4\)CDD (<1 to 18 pg g\(^{-1}\)) and 2,3,4,7,8-P\(_5\)CDF (4.1 to 71 pg g\(^{-1}\)) detected in the same human samples. Detection of coplanar PCBs at much higher levels than toxic dioxins and furans prompts us to necessitate an evaluation of their toxic contribution on a comparative basis. However, the toxicity data of trace levels of coplanar PCBs, PCDDs and PCDFs on a long-term basis to humans and environmental animals are neither fully understood nor clearly demonstrated. A probable approach to estimate their environmental toxic significance is by comparing their biologic response to induce the hepatic microsomal enzymes. The relative biologic potencies of coplanar PCBs, PCDDs and PCDFs to induce AHH and EROD (ethoxyresorufin \(O\)-deethylase) in the rat hepatoma cell lines have been determined and the direct quantitative correlation between \textit{in vitro} dose–response biologic effect (microsomal enzyme induction) and \textit{in vivo} toxic effects (body weight loss and thymic atrophy) of these chemicals have also been demonstrated in rats (Sawyer & Safe, 1982; Leece \textit{et al.}, 1985; Safe, 1986). The hepatic microsomal enzyme systems are most likely correlated with certain reproductive effects also because they are known to affect steroid hormone levels (Helle \textit{et al.}, 1976a; Reijnders, 1986; Subramanian \textit{et al.}, 1987; Norstrom \textit{et al.}, 1988). In
the absence of any reliable long-term toxicity studies on coplanar PCB congeners, AHH and EROD induction bioassay seems to be the only dependable tool presently available to predict their significant contribution in the possible toxic effects for humans and environmental animals.

Based on the data of AHH induction potencies of coplanar PCBs, PCDDs and PCDFs and their residual concentrations in environmental animals, Harris et al. (1985) estimated '2,3,7,8-T₄CDD toxic equivalents' to assess the relative toxic contribution of these chemicals for the reproductive impairment of Forster's tern in Green Bay. In this study, the relative potency of AHH induction by coplanar PCBs and other toxic halogenated aromatics is calculated in relation to the induction potency of the most potent member of this family; namely 2,3,7,8-T₄CDD as one and then these calculated values are multiplied by their individual concentration as measured in chemical analyses. By this assessment, they suggested greater toxic contribution of coplanar PCB congeners than 2,3,7,8-T₄CDD. Similar assessment has also been attempted by Kannan et al. (1988) and Tanabe et al. (1987a) for the residues of some toxic coplanar PCB, PCDD and PCDF isomers and congeners found in human adipose tissues. As seen in Fig. 5, the higher residual concentration and relative high induction potential of 3,3',4,4',5-P₅CB which is only 2.5-fold less potent as 2,3,7,8-T₄CDD (Sawyer & Safe, 1982) showed the highest value of '2,3,7,8-T₄CDD toxic equivalents' among all the toxic chemicals examined including most toxic 2,3,7,8-T₄CDD and 2,3,4,7,8-P₅CDF. Based on all these observations, it can be concluded that non-ortho chlorine substituted coplanar PCBs, especially

![Fig. 5. '2,3,7,8-T₄CDD toxic equivalents' of PCDDs, PCDFs and coplanar PCB congeners in humans (data from Tanabe et al., 1987a).](image-url)
$3,3',4,4',5$-PsCB, impose a greater toxic threat than dioxins and furans to humans and probably to wildlife also.

Even in taking account of other PCB isomers and congeners, coplanar PCB congeners are most likely responsible for the intrinsic environmental impact of PCB mixtures because their biologic and toxic responses are extremely high, overwhelming the toxic balance resulting from the difference of residue levels between coplanar and other PCBs in the biota. However, some of mono-ortho chlorine substituted coplanar PCBs should be taken into toxic consideration because of their high potencies of AHH and EROD induction similar to coplanar PCBs (Sawyer & Safe, 1982; Safe, 1984). Indeed, Harris et al. (1985) reported the highest value of $'2,3,7,8$-T$_4$CDD toxic equivalents' for $2,3,3',4,4'$-PsCB found in Forster's tern eggs from Green Bay. This warrants a critical evaluation of mono-ortho congeners as well as non-ortho coplanar PCBs in future work. It is also to be understood in future whether there is any additive effect among these toxic contaminants; however, some recent investigations point in that direction (Harris et al., 1985; Sawyer & Safe, 1985).

Similarly, the same concern shown to coplanar PCB congeners should be paid to the accidental PCB poisoning such as the Yusho rice bran oil incident and occupational exposures. In the case of Yusho patients, the reported acute and long-term effects are believed to be caused principally by the most toxic and persistent chlorinated dibenzofurans and quarterphenyls rather than PCBs (Miyata et al., 1978; Kashimoto et al., 1981; Hori et al., 1982; Kunita et al., 1984). However, a recent study found the presence of all the three toxic coplanar PCB congeners in Yusho causal oil and tissue samples of Yusho patients with comparable levels to $2,3,7,8$-T$_4$CDF (Kashimoto et al., 1987). This also insists the need for a reevaluation of PCB toxicity, giving more emphasis to specific toxic isomers and congeners.

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