Occurrence of Polychlorinated Terphenyls (PCTs) in Indoor Particulate Matter

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In the course of a routine investigation concerned with polychlorinated biphenyl (PCB) contamination of dust collected in classrooms of a junior high school, a group of electron capture detector (ECD)-sensitive compounds with high boiling points were found in addition to PCBs. Using gas chromatographic–mass spectrometric techniques, these compounds were identified as polychlorinated terphenyls (PCTs). Additional measurements indicated that the PCTs were present only in particulate matter collected from the tops of fluorescent light frames but not in air samples obtained concomitantly in the classrooms. Attempts to identify the PCT emission source were unsuccessful. A survey of the literature revealed that PCTs are ubiquitously distributed environmental contaminants, although no data on their indoor occurrence have been reported to date. In view of the toxic effects of PCTs, which seem to be as important as those of PCBs, further attention should be given to the possible presence of PCTs in indoor environments. *Key words*: environmental contamination, indoor environment, particulate matter, polyhalogenated aromatic hydrocarbons, polychlorinated terphenyls. *Environ Health Perspect* 104:1172–1179 (1996)

Polychlorinated terphenyls (PCTs; structures shown in Fig. 1) were introduced as plasticizers and flame retardants in the early 1930s and have been used since then in practically the same areas in which their two-ring counterparts, the polychlorinated biphenyls (PCBs), have been employed (*1*). Like PCBs, PCTs are long-lasting environmental contaminants and have been found in water, aquatic and terrestrial organisms, food packaging materials, and also in human tissues (*2–33*). No reports on the occurrence of PCTs in indoor environments, however, have been published to date.

In the course of a routine investigation involved with the PCB contamination of dust samples collected in classrooms of a junior high school located in Southern Germany, a large number of compounds with high boiling points, previously not found in such samples, were detected when the samples were analyzed by gas chromatography (GC) and electron capture detection (ECD). Using GC–mass spectrometric (GC-MS) techniques, these compounds with high boiling points were eventually identified as PCT congeners.

In the present paper, we describe the identification of PCTs in the dust samples and discuss the consequences of this finding for routine monitoring of polychlorinated aromatic hydrocarbons in indoor environments. In addition, we briefly review the former use, environmental contamination, and biological effects of PCTs.

Materials and Methods

Aroclor 5442 (chlorine content 42%) and Aroclor 5460 (chlorine content 60%), standard mixtures (100 µg/ml in *n*-hexane), were purchased from Promochem (Wesel, Germany). PCB-Mix 1, a standard mixture containing 2,4,4′-trichlorobiphenyl; 2,2′,5,5′-tetrachlorobiphenyl; 2,2′,4,5,5′-pentachlorobiphenyl; 2,2′,3,4,4′,5′-hexachlorobiphenyl; 2,2′,4,4′,5,5′-hexachlorobiphenyl; and 2,2′,3,4,4′,5,5′-heptachlorobiphenyl (10 ng of each compound in 1 µl iso-octane), was obtained from Ehrenstorfer (Augsburg, Germany). *n*-Hexane, toluene, and acetone (SupraSolv) used for analytical work was purchased from Merck (Darmstadt, Germany).

Sample collection. Eight dust samples (approximately 100 mg each) taken at four different locations in two classrooms were collected separately in snap-on-lid vials (20 ml) with the aid of a paint brush, and each were thoroughly mixed. The sampling sites chosen were located at least 2 m above the floor, preferably the tops of fluorescent light frames, wooden window frames, wooden bookcases, and cabinets.

In addition, two air samples were taken for analysis of chloroorganic compounds in each of the two classrooms over two 6-hr periods, during which time lectures were being held in the air-conditioned rooms. The sampling device consisted of a funnel fitted with polyurethane-foam (Ziemer, Mannheim, Bermany) through which air was drawn at a flow-rate of 26 l/min. Prior to sampling, the polymer was cleaned with toluene, *n*-hexane, and acetone. The device was installed 1.2 m above the floor and 2 m away from the side walls. The total air volume drawn was 18.500 liters.

Sample preparation. Five milligrams of each particulate sample were taken up in 2 ml of *n*-hexane and treated with ultrasonic vibration for 30 minutes. The resulting suspension was centrifuged at 4000 rpm for 10 min. One-microliter aliquots of the supernatants were injected into the GC system for analysis. Each dust sample was analyzed in duplicate.

For the analysis of chloroorganic compounds in air, the polyurethane-bound compounds were desorbed from the polymer with *n*-hexane. The eluate was cleaned by solid-phase chromatography on a small silica gel column impregnated with sulfuric acid. The samples were then concentrated using a rotavap and subsequently by evaporation under a gentle stream of dry nitrogen to a final volume of exactly 1 ml.

Gas chromatographic analysis. GC analysis of the samples was carried out using a Hewlett-Packard model 5890A instrument (Hewlett-Packard, Waldbronn, Germany), equipped with a fused silica capillary column (30 m × 0.32 mm diameter, 0.25 µm film thickness) coated with the bonded stationary phase DB-17 (J & W Scientific, Folsom, CA). Helium (head pressure 85 kPa) was employed as carrier gas and argon/methane (95:5 v:v) as detector make-up gas. Components eluting from the column were detected with a ^{63}Ni electron capture detector (maintained at 320°C) and recorded using a Hewlett-Packard model 3396A reporting integrator (Hewlett-Packard). Samples were injected automatically (Hewlett-Packard 7673 autosampler; Hewlett-Packard) using the splitless mode of operation (injector block temperature, 250°C) and cold-trapped on the column at 50°C. The column oven temperature was raised rapidly (40°C/min) to 190°C and held at this temperature for 2 min. The temperature was then programmed to increase linearly at 1.5°C/min to 230°C and at 5°C/min to 280°C, where it was maintained for 30 min.

Gas chromatographic–mass spectrometric analysis. Mass spectra were recorded using a Finnigan 4000 quadrupole mass spectrometer (Bremen, Germany) equipped

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The authors would like to thank H. Schlegel for assistance with manuscript preparation.

Received 29 March 1996; accepted 5 August 1996.

with a Carlo Erba model HRGC 5160 gas chromatograph (Mainz, Germany) and coupled to a Maspec PC-based data system (MasCom, Bremen, Germany). Analyses were performed in the EI mode with an electron energy of 70 eV, an emission current of 200 µA, and an accelerating potential of 2 kV. The ion source and GC interface temperatures were maintained at 250°C and 280°C, respectively, and full scans (600–50 daltons) were taken repetitively at a scan rate of 1 sec/decade. Samples were introduced automatically (Carlo Erba model CTC A200SE autosampler) via the GC inlet, through which the capillary column was interfaced directly to the ion source of the mass spectrometer. Chromatographic conditions for GC-MS analyses were identical to those noted above for GC work.

Quantitative analysis. Total PCB concentrations in dust and air samples were determined by GC with the aid of reference compounds and in accordance with the calculation procedure proposed by Länderarbeitsgemeinschaft "Abfall" (LAGA) (*34*). The detection limits for the individual PCB congeners were approximately 2 µg/g in dust samples and 1 ng/m^3 in air samples. The corresponding recovery rates were 90–110% in dust samples and 94–99% in air samples.

Because of the chemical similarity of PCBs and PCTs, detection limits and recovery rates for the PCTs were probably in the same range as those of PCBs. The exact values of these analytical parameters, however, as well as total PCT concentrations in the samples, could not be determined due to the lack of appropriate reference material.

Results

Dust samples. When aliquots of the organic extracts of the dust samples were analyzed by GC, a large number of high-boiling point compounds appeared in the chromatograms (Fig. 2). One group of substances eluting from approximately 18 to 40 min after injection were identified as PCB congeners. Their concentration ranged from 215 to 250 µg/g. A highly abundant component with a retention time of 40 min proved to be the PVC plasticizer di(2-ethylhexyl)phthalate (DEHP). While these chemicals had been shown to be frequent constituents of indoor particulate matter (*35*), a third group of compounds eluting from about 45 to 65 min had not been detected previously in such samples.

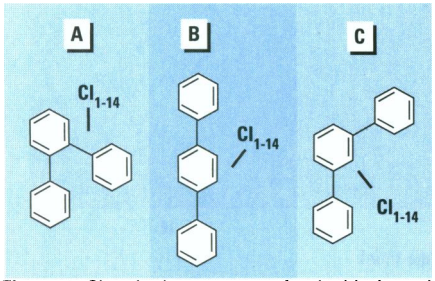

Figure 1. Chemical structures of polychlorinated terphenyls (PCTs). (A) *ortho*-PCTs; (B) *para*-PCTs; (C) *meta*-PCTs.

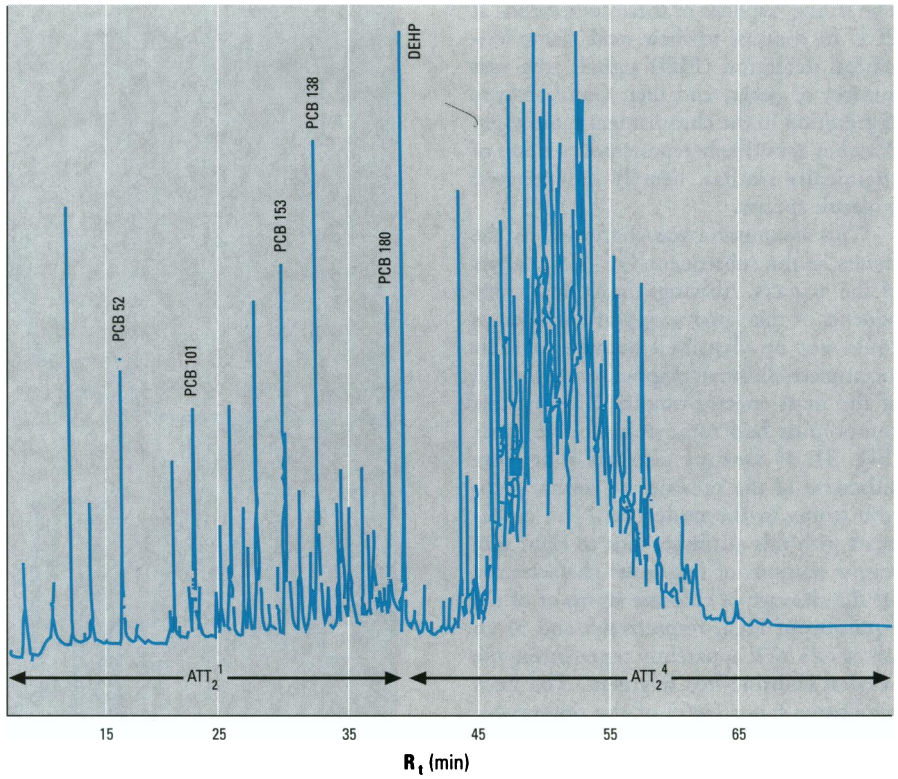

Figure 2. Gas chromatogram (electron capture detector) obtained from the organic extract of a dust sample collected in the classroom of a junior high school. The attenuation of the recorder was increased after the elution of PCBs (40.0 min after injection of the sample). For experimental details see text. DEHP, di (2-ethylhexyl)phthalate; ATT, attenuation.

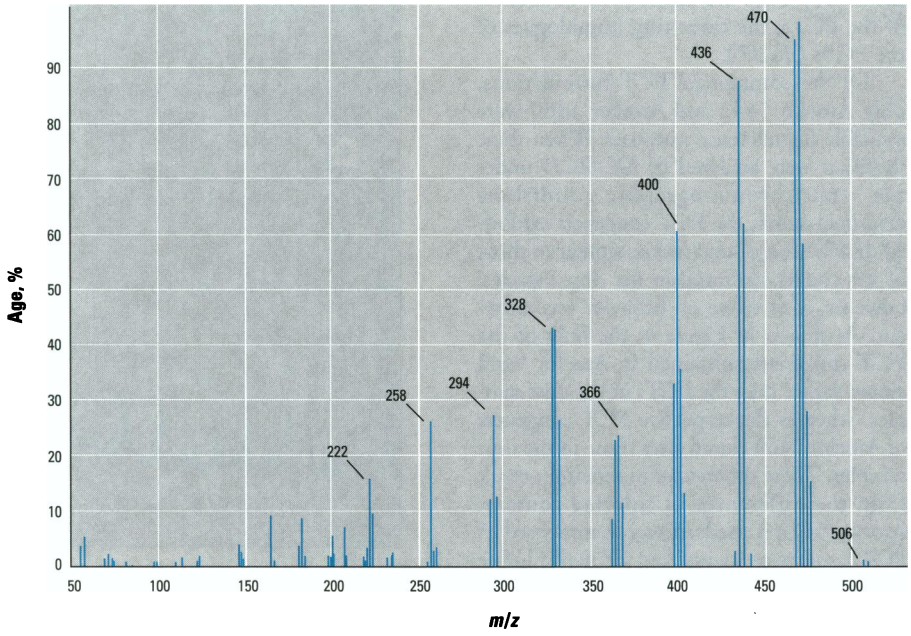

Figure 3. Electron impact (EI)-mass spectrum recorded 51 min after injection of the extract of a dust sample. See text for experimental details.

This group of compounds, the abundance of which exceeded that of PCBs by several orders of magnitude, was found in the four samples collected from the top of the fluorescent light frames but in none of the specimens from the other sampling sites. The strong response of these compounds at ECD in contrast to their weak flame ionization detection (FID) signals, the vast number of peaks, and their Gaussian-type distribution in the chromatogram indicated that they most likely represented a group of chemically similar, heavily halogenated molecule species.

This assumption was confirmed by the results of the subsequent GC-MS analysis of the extracts. Although individual components of this latter group of compounds could not be identified because of their incomplete chromatographic resolution, all of the mass spectra obtained from these compounds had some common features (Fig. 3): 1) isotope clusters that were indicative of the presence of several chlorine atoms in the molecule; 2) in agreement with this attribute, four to eight successive scissions of fragments characteristic for the cleavage of chlorine atoms or of the expulsion of HCl, respectively; and 3) an ion at *m/z* 222, apparently representing the heaviest chlorine-free fragment. The mass per charge ratios (*m/z*) of the presumable molecular ions ranged from 366 to 502 (^{35}Cl isotopes), with most components having a heaviest fragment with *m/z* 468. These mass spectrometric features are consistent with a set of isomers composed of three connected phenyl rings and four to eight chlorine substituents. Molecules with these properties are among the congeners of the PCTs, the three-ring homologues of the PCBs (*36,37*).

Of the commercial PCT formulations, only Aroclor 5442 and Aroclor 5460 were available for reference purposes. When these materials were analyzed by GC-ECD under the same chromatographic conditions described above, the PCT congeners exhibited distribution characteristics similar to those of the congeners found in the dust samples; however, their retention behavior was different. As shown in Figure 4, the bulk of the PCT congeners contained in Aroclor 5442 eluted earlier than the PCTs in the dust samples, whereas the respective PCT congeners of Aroclor 5460 eluted after those in the dust samples. These differences in retention result from the differences in chlorine content. Aroclor 5442 is predominantly composed of PCT congeners having four or five chlorine atoms (i.e., less than the congeners in the dust samples) and most of the PCT congeners of Aroclor 5460 have eight or nine chlorine atoms, (i.e., more than the congeners in the dust samples). Most of the Aroclor 5442 and Aroclor 5460 congeners, which coeluted with the PCT congeners in the dust samples, seem to have seven or eight chlorine atoms in the molecule. Though the makeup of PCT mixtures might change with time, e.g., by evaporation of the more volatile congeners, it is obvious from Figure 4 that, in the case of the Aroclor formulations, such alterations would not have pro-

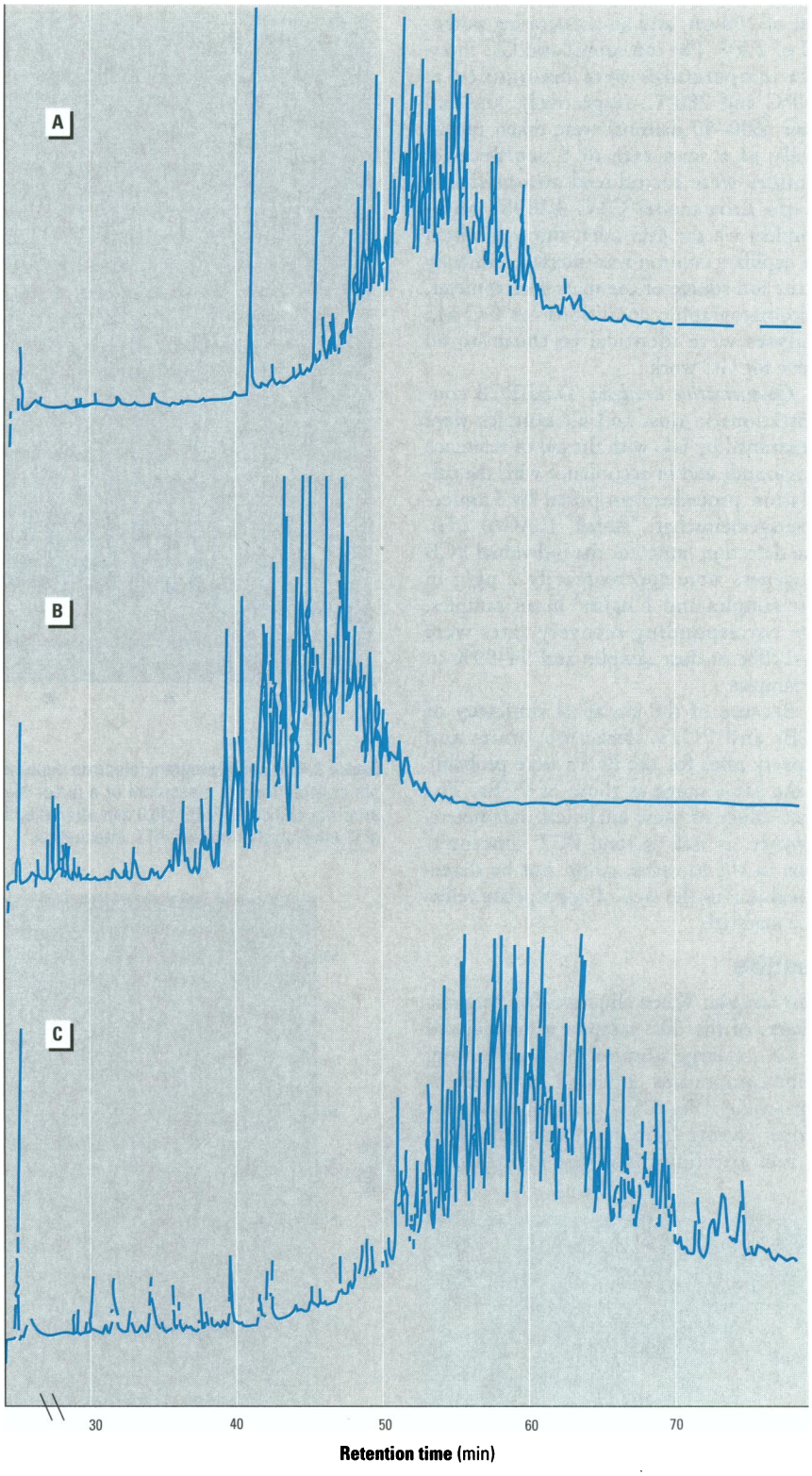

Figure 4. Gas chromatograms (electron capture detector) obtained from (A) organic extracts of particulate matter collected in a classroom; (B) Arochlor 5442; and (C) Arochlor 5460. See text for experimental details.

duced the PCT distribution pattern found in the dust samples. Thus, the identified PCTs must have arisen from a PCT formulation different than the above Aroclors. PCT materials with a chlorination degree similar to that of the PCTs in the dust samples were produced and marketed by Bayer AG in Germany until the 1970s under the trade names of Leromoll and Clophen-Harz W, (*32,38*). Unfortunately, reference samples of these formulations were not available for analysis.

An exhaustive inquiry was undertaken to identify the source of the PCT contamination without success. Although PCBs were found to contaminate fluid and paper of the condenser and also the series resistor, PCTs were not detectable in any of these electrical parts.

Air samples. In contrast to the particulate samples, PCBs, but not PCTs, were present in the collected air samples. The PCB concentration in air amounted to about 500 ng/m^3.

Discussion

As far as we could determine, this is the first report on the detection of PCTs in samples collected in an indoor environment. Of particular concern is the fact that PCTs were found in an area in which teachers and young people spend extended periods of time during which they could be exposed to these potentially hazardous chemicals. Though sealants and electrical appliances in the classrooms were scrutinized for PCT contamination, the PCT emission source could not be identified. One assumption is that the initial PCT-containing material may have been removed from the classrooms after its initial use, presumably unknowingly, whereas PCT-contaminated particulate matter remained until the time of the investigation.

Suspended particulate matter is usually mobile and should be equally distributed within a room. Thus, one would have expected that all collected dust samples were contaminated with PCTs. The analysis of the samples showed, however, that this assumption was incorrect. Only particulate matter deposited on top of the fluorescent light frames, but not the samples collected from other sites of the classroom, contained PCTs. The cause of this unequal distribution remains unclear. One possible explanation is that a specific mechanism, e.g., an electrostatic charge, might have prevented the mobility of particulate matter deposited at special sites. An alternative explanation could be that, in contrast to the other collection sites, particulate matter deposited on top of the fluorescent light frames was not removed by routine room cleaning in recent years due to the difficult access to these sites. The positive aspect of this particular finding is that PCT-contaminated particulate matter, which occurs only in remote places and which is not distributed within the entire indoor environment, is of less toxicological significance.

Regardless of the fact that PCT contamination was restricted to special indoor locations in this case, further attention should be given to the possible occurrence of PCTs in other indoor environments. Inquiries into the PCT contamination of various environments are important, primarily because our knowledge of the biological effects of PCTs is very limited. Because PCT analysis can be carried out basically in the same way as that of PCBs, except that the elution time in the chromatography has to be extended (*39*), the inclusion of a PCT screening step into the analytical protocol established for routine determinations of polychlorinated aromatic compounds requires little additional analytical effort.

Production, chemistry, and use of PCTs. Commercial production of PCTs was initially started by Monsanto Industrial Chemical Co. (St. Louis, MO) in 1929 and continued until the 1970s when it was gradually phased out (*1,38*). Outside the United States, PCTs were mainly produced in France, Italy, Japan, and Germany. The PCT production in the United States reached its maximum in 1971 at approximately 20 million pounds, equivalent to 15% of the PCB production in that year. In the same time period, the average world production of PCTs was in the range of 10,000–12,000 metric tons. Ten years later, it had dropped to approximately 600 metric tons.

In most countries, PCT production was discontinued voluntarily by the manufacturers (*1,40*). In Germany, state regulations introduced in 1978 restricted the use of PCBs and PCTs to closed systems (*41*) in accordance with a corresponding directive of the Council of the European Communities, which was adopted in 1976 (*42*). In 1989, production, marketing, and use of PCBs and PCTs were banned completely (*43*). Trade names of PCT products and the major manufacturers are listed in Table 1.

Commercial PCT formulations—generally obtained by controlled chlorination of aromatic hydrocarbons—consist of a mixture of *ortho-*, *meta-* and *para-*terphenyl derivatives (Fig. 1), among which the *meta-*substituted species seem to predominate (*44*). The exact composition of the formulations, i.e., the fraction of the individual PCT congeners, is unknown, a consequence of the vast number of possible molecular arrangements. All commercial PCT products are contaminated with PCBs and may also contain residues of chlorinated dibenzofurans (*45*).

The physicochemical properties of PCTs are almost identical to those of highly chlorinated PCBs (*1*). PCTs are heat stable, nonflammable, soluble in a number of organic solvents, insoluble in water, and resistant to aggressive and corrosive chemicals such as alkalies and strong acids. Therefore, they were applied in practically the same areas as PCBs, often in combination with them. For a period of time, PCTs were even considered as viable alternatives for their two-ring homologues. In the earlier years of PCT production, most of the PCT formulations were used as plasticizers, hydraulic fluids, or lubricants. PCTs were also used as constituents in hot-melt adhesives, for the preparation of fire-resistant or self-extinguishing materials, as stabilizers for polymers, and to extend the activity of systemic insecticides. In later years, the most important application was in waxes (PCT content up to 50%) for the investment casting industry, which produces high precision metal parts for aircraft, nuclear installations, and jewelry (*1*).

Environmental contamination. Alhough PCTs were not as important as PCBs with respect to amount of production and use, traces of PCTs have been detected in a wide variety of environmental samples. The extent of environmental contamination, as well as the routes of PCT entry into the environment, are nevertheless unknown. The observation that PCTs were present in the atmosphere over Lake Huron (United States)—albeit in small concentrations (approximately

Table 1. Manufacturers and trade names of polychlorinated terphenyl products

Country	Manufacturer	Trade name
United States	Monsanto Industrial Chemical Co.	Aroclor 5460, 5442, and 5432
France	Produits Chimique Ugine Kuhlmann Prodelec	Electrophenyl T-60, Phenoclor Terphenyl Chlore T-60
Italy	Caffaro	Cloresil A, B, and 100
Germany	Bayer AG	Clophen Harz W, Leromoll 112-90, Leromoll 141
Japan	Kanegafuchi Chemical Co. Mitsubishi-Monsanto Chemical Co.	Kanechlor-C Aroclor

2 ng/m^3)—could indicate that long-range atmospheric transport is one source of the ubiquitous contamination (*46*).

PCTs were found in soil around investment casting facilities in Illinois and Michigan (*17*), in sediments and aquatic organisms (oysters, crabs, shellfish, mummichogs, eels, and gulls) of the Great Lakes (*26*), the Chesapeake Bay (*27,30,33*), the Bay of Fundy–Gulf of Maine (*3*), the Ebro River Delta (*32*), and the Bay of Biscay (*29*); in samples of sludge from the coast off Tokyo Bay (*18*); in marine sediments of the Languedocian continental shelf (*12*); in sediments of a lake in Berlin (*23*); in water samples of the Rhine (*5*); and even in sediments from remote places, such as Winter Quarters Bay in Antarctica (*28*). In most cases, PCTs were found along with PCBs, polychlorinated naphthalenes, and other major classes of anthropogenic organic contaminants (polycyclic aromatic hydrocarbons and chlorinated pesticides) (*12,25,26, 28,29*). The concentrations of PCTs in sediments and biota were generally lower than those of PCBs, but usually in the same order of magnitude (*5,28*). PCTs with a low degree of chlorination (e.g., Aroclor 5432) accumulated to higher concentrations than more heavily chlorinated terphenyls (*30*). The highest PCT concentrations measured in sediments (found near shipyards) were approximately 250 ppm on a dry weight basis (*27*).

With respect to aquatic organisms, the highest mean PCT concentrations were measured in oysters (*33*). In tributaries of the Chesapeake Bay, PCT levels in some oysters reached 35 ppm (dry weight), which is equivalent to 6.3 ppm wet weight. This value exceeded the U.S. limit for PCBs in shellfish intended for consumption by a factor of more than 3 (*27,30*). In samples of shellfish from the Ebro Delta, PCT concentrations ranged from 3 to 790 ppb (dry weight) (*32*). Bioaccumulation through food chains was demonstrated in a study on PCT contamination of aquatic animals from the Baltic Sea area; PCT levels in white-tailed eagles (2.8–17.2 ppm) were higher than those of grey seals (0.5–1.0 ppm), which exceeded those of eels (0.08 ppm) (*24*).

Birds, snakes, dogs, and cats are the terrestrial animals in which PCT levels have been determined. Fat tissue of pigeons caught in the Tokyo urban area contained 0.05–0.7 ppm PCT (PCB levels, 0.2–1.9 ppm), with an average value of 0.1 ppm (PCB, 0.8 ppm). PCT concentrations in livers of crows caught in the same area were considerably higher than corresponding values in livers of crows caught in the Tochigi mountainside area (0.17 vs. 0.06 ppm) (*22*). PCT residue levels in the livers of kestrels, barn owls, sparrow hawks, herons, a long-eared owl, and kingfishers found dead in England ranged from <0.05 to 1.2 ppm and were 1–2 orders of magnitude smaller than the corresponding PCB levels (*20*). The PCT concentration measured in a gull egg was 23 ppm, considerably higher than that of PCBs and some halogenated pesticides (*12*). PCT levels measured in domestic cats and dogs in Japan (up to 8.9 ppm) were higher than those obtained in wild animals. This discrepancy was explained by the contamination of a commercial animal feed product with PCT (*18*).

PCTs in food and food packaging materials. Food and food packaging materials are one source of human exposure to PCTs. In a survey carried out in Canada to determine the PCB and PCT content of food wrappers, 84.5% of the samples contained <1.0 ppm, 11.4% contained 1–10 ppm, and 4.1% contained >10 ppm PCT. None of 73 foods analyzed for PCT contained >0.05 ppm of these chemicals (*11*). According to a study by Thomas and Reynolds (*9*), PCT levels in paperboard food packaging material amounted to up to 163 ppm and were considerably higher than the corresponding PCB levels (0–20 ppm). The contamination of paper products by PCT was explained by the use of PCTs in printing ink.

PCT residues found in samples of wall scrapings, silage, and milk from certain farms were attributed to the use of PCTs as a sealant for concrete stave silos (*6*). PCTs were detected in one out of three food samples examined in Japan. The PCT contamination, however, was confined to vegetable products. Daily uptake of PCTs from the diet was estimated to be around 0.05 µg, or about 1/100 of the total daily PCB dose (*18*).

Absorption, distribution, and metabolic fate of PCTs. Accumulation and distribution of PCTs were investigated in mice, rats, fish, and hens.

Appreciable amounts of Aroclor 5460 were found in all tissues of cod fed with Aroclor in hexane (*2*). The highest PCT concentrations were measured in the liver, but PCTs were also detected in brain and gonadal tissue. The excretory efficiency seemed poor since considerable amounts of PCTs were still found in the tissues 70 days after exposure. Differences in the chromatograms obtained from tissue extracts and a standard sample were indicative of some selectivity in absorption, metabolism, and excretion of PCT congeners.

PCT residues were also found in all analyzed tissues when Aroclor 5460 was fed to rats at 10, 100, or 1000 ppm in the diet for 7 days (*47*). The greatest PCT concentrations were found in liver and fat and the lowest concentration in blood. PCT components with a lower chlorine content were metabolized to a higher degree.

In male mice fed a diet containing 20 or 100 ppm of PCTs, PCTs tended to accumulate more in liver than in adipose tissue (unlike polychlorinated biphenyls and other organochlorine pesticides), but this tendency was reversed after 18 months (*48*). The amount of PCTs that accumulated in mouse liver increased continuously throughout the feeding period with the higher PCT diet regimen, but decreased significantly at the later period feeding at 20 ppm. PCT concentrations in adipose tissue correlated well with those in liver, kidney, skin, and brain.

When a single dose of 10 mg of PCTs was administered intraperitoneally to mice, PCT concentrations in adipose tissue increased gradually during the experimental period of 56 days, whereas the levels of PCT in liver peaked on the first day after administration and receded rapidly thereafter (*49*). The composition of accumulated PCTs in the liver was different from that in adipose tissue and changed remarkably with time. *para*-PCTs were more rapidly eliminated from liver than *ortho*- and *meta*-PCTs, and the biological half-lives of *para*-PCTs in both rapid and slow phases were shorter than those of *ortho*- and *meta*-PCTs.

In hens fed Aroclor 5460 orally for 7 days at a dose of 2 mg/day, uptake, distribution, and metabolism of the three terphenyl isomers were also different (*44*). In this animal species, the *para*-isomers accumulated most due to a higher uptake and a slower metabolism. The highest levels of total PCTs were found in egg yolk, followed by liver, kidney, and fat.

PCTs in human tissues. PCT residues found in human tissues—blood, liver, fat, and milk—indicate that PCTs accumulate in the human body. The highest PCT concentrations were found in fat tissue. PCT levels measured in fat tissue samples obtained from various Japanese populations averaged 0.03–1.9 ppm (*18*), 1.11 ppm (*19*), 0.37 ppm (*14*), and 0.13 ppm (*8*). They were comparable to or slightly lower than those of PCBs (*5,14*). The *para*-congeners seem to be more persistent than the other PCT species because their fraction in fat was relatively higher than in the commercial PCT product used in Japan (*19*).

Average PCT levels measured in blood from Japanese people were 3–6 ppb and higher than mean PCB levels (3.2 ppb) (*16,50*). These findings are consistent with results from animal experiments which showed that PCTs accumulate more in liver and blood than PCBs. PCT levels in blood collected from the same donors over a period of 2 years were almost identical

except in one subject, indicating a very long half-life of PCTs in humans (*19*).

Mothers' milk collected from 10 Japanese women in 1972 contained up to 7 ppb PCT, corresponding to 220 ppb fat weight (*8*). Similar levels were found by Minagawa et al. (*14*) in 1974 and Doguchi (*18*) in 1977. In contrast to PCBs, the PCT levels in milk fat were considerably lower than the corresponding levels in adipose tissue (*50*).

Biological effects. Because of the close structural resemblance of PCTs to PCBs, PCTs are assumed to exhibit biological effects similar to those of their two-ring homologues. In contrast to PCBs, however, little information is available on the biological effects of PCTs. In addition, it is not always clear whether the observed effects in some studies are caused exclusively by PCTs or by PCBs or other toxic compounds contained in the PCT products as contaminants. The real scope of PCT biological activity, therefore, is largely unknown and awaits further elucidation.

The acute toxicity of PCTs seems to be lower than that of PCBs, possibly because of the lower solubility and the lower rate of absorption in the body. In rats, the oral mean lethal doses (LD_{50}) of 10,600 mg/kg for Aroclor 5442 and 19,200 mg/kg for Aroclor 5460 have been reported; corresponding values for skin minimum lethal dose were 1,260 mg/kg and 7,940 mg kg, respectively (*51*).

Following subchronic or chronic administration of PCTs, liver injury (including tumors), kidney damage, deleterious effects on the gastric mucosa, and disturbance of the reproductive system were observed. Other adverse effects, e.g., immune alterations, are likely but have not yet been demonstrated.

Animals exposed to PCTs in their daily diet have been shown to gain less weight than control animals (*52,53*). Loss of hair from the head, neck, and back and a progressive generalized subcutanous edema, particularly beginning in the face, was seen in Rhesus monkeys given Aroclor 5460 (5000 ppm) in their diet (*54*). In addition, acneform lesions occurred on skin areas devoid of hair. Kidneys were affected primarily in the proximal convoluted tubules. The earliest change was disruption and distortion of mitochondria, followed by necrosis (*55*). PCT ingestion by monkeys for 3 months produced hyperplasia and dysplasia of the gastric mucosa (*54*). The main target organ for chronic PCT toxicity, nevertheless, was the liver. Aroclor 5460 caused a dose-dependent increase in relative liver weight and proliferation of the smooth endoplasmatic reticulum in rats (*47,52*) and monkeys (*54*). The affected liver microsomes had a higher content of proteins, and the structures of the membranes were less stable. In addition, Toftgaard et al. (*56*) observed the frequent occurrence of lysosomes containing partially degraded lipid material as well as an increased number and size of cytoplasmic lipid droplets.

Proliferation of the endoplasmatic reticulum was related to the induction of microsomal enzyme systems by high doses of PCTs; this has been demonstrated both in laboratory animals and *in vitro* systems. Following exposure to PCTs, an increased turnover rate has been demonstrated for cytochrome P450-dependent reactions such as aromatic hydroxylation and *N*-hydroxylation, *N*- and *O*-dealkylation, aromatic nitro reduction, and glucuronidation. Interestingly, both phenobarbital- and 3-methylcholanthrene-inducible forms of cytochrome P450 were affected (*47,52,56–60*). The activities of epoxide hydrolase and glutathione *S*-transferases were not changed (*60*).

Generally, the observed increases in enzyme activities were dose dependent, and PCTs with a low degree of chlorination were more potent than highly chlorinated PCT mixtures. As shown in immature male Wistar rats, only a portion of the investigated PCT congeners acted as inducers of 4,4′-dimethylamino antipyrine *N*-demethylase (2,4-dichloro-*p*-terphenyl; 2,4,6-trichloro-*p*-terphenyl; and 2,3,4,5-tetrachloro-*o*-terphenyl) or ethoxyresorufin-*O*-deethylase (2,3,4,5-tetrachloro-*p*-terphenyl) (*61*). Since neither 2,3,4,5-tetrachloro-*p*-terphenyl (median effective concentration = 6.6×10^{-6} M) nor the 2,3,4,5-tetrachloro-*ortho*- and *meta*-terphenyls (median effective concentration values $>10^{-5}$ M) exhibit a high affinity for the tetrachlorodibenzodioxin (TCDD) receptor protein (*62*), PCT congeners and commercial mixtures are unlikely to elicit significant TCDD-like biological or toxic effects in target species (*61*).

Carcinogenicity of PCTs has only been tested in a single study with male ICR mice that received Kanechlor-C (250 ppm and 500 ppm) containing 5% PCB as an impurity in their diet for 24 weeks (*53*). The PCT-exposed mice had a significant dose-related higher incidence of nodular hyperplasia of the liver. In addition, the high dose group had a significantly higher incidence of hepatocellular carcinoma. Animals coexposed to Kanechlor-C and hexachlorbenzene (HCB) showed a severalfold increase in prevalence of both nodules and carcinoma, indicating that HCB enhanced the effect considerably. A further indication of PCT neoplastic acitivity is the hyperplasia and dysplasia of gastric mucosa found in monkeys after ingestion of PCT for 3 months (*54*). Finally, the observation that Aroclor 5442 was eight times more estrogenically active in rats than some low chlorinated PCB products (*58*) might deserve special attention in view of the ongoing discussion concerning the role of xenoestrogens in the development of breast cancer (*63*).

Effects on reproduction and development, described particularly from PCBs, have only been investigated in laboratory animals in the case of PCTs. When white leghorn chickens were fed 20 ppm Aroclor 5460 in their diets, a greater number of dead or abnormal embryos were noted, compared with control animals. Hatchability, however, was not affected significantly (*64*). This applied also to the hatchability of fertile eggs when chickens were fed 20 ppm Aroclor 5442 for 9 weeks (*65*). In contrast, in a more recent study, PCTs adversely affected *in vitro* fertilization and increased the incidence of abnormal embryos and oocyte degeneration in $B6D2F_1$ mice (*66*).

Conclusion

In the light of the various adverse effects of PCTs and their persistent and cumulative properties in living organisms, it seems advisable to look more closely at these compounds in the human environment. PCTs have been used in the past in a variety of building materials, and considerable amounts of PCT residues may still be present in interior areas where these materials were applied. Because PCTs are predominantly bound to airborne particulate matter, analysis of dust samples appears to be a simple and suitable approach to screen for PCT contamination in such environments. This might also be an appropriate way to estimate the average exposure to PCTs during the last few years.

References

1. Jensen AA, Jorgensen KF. Polychlorinated terphenyls (PCTs) use, levels and biological effects. Sci Total Environ 27:231–250 (1983).
2. Addison RF, Fletcher GL, Ray S, Doane J. Analysis of a chlorinated terphenyl (Aroclor 5460) and its deposition in tissues of cod (*Gadus morhua*). Bull Environ Contam Toxicol 8:52–60 (1972).
3. Zitko V, Hutzinger O, Choi PMK. Contamination of the Bay of Fundy–Gulf of Maine area with polychlorinated biphenyls, polychlorinated terphenyls, chlorinated dibenzodioxins, and dibenzofurans. Environ Health Perspect 1:47–50 (1972).
4. Zitko V, Hutzinger O, Jamieson WD, Choi PM. Polychlorinated terphenyls in the environment. Bull Environ Contam Toxicol 7:200–201 (1972).

5. Freudenthal J, Greve PA. Polychlorinated terphenyls in the environment. Bull Environ Contam Toxicol 10:108–111 (1973).
6. Fries GF, Marrow GS. Polychlorinated terphenyls as potential contaminants of animal products. J Assoc Off Anal Chem 56:1002–1007 (1973).
7. Mestres R, Illes S. Demonstration of a new environmental pollutant. Accumulation of polychlorotriphenyls by birds. Trav Soc Pharm Montp 33:201–208 (1973).
8. Nishimoto T, Ueda M, Taue S, Chikazawa H, Nishiyama T. PCT (polychlorotriphenyl) in human adipose tissue and mother milk. Igaku No Ayumi 87:264–265 (1973).
9. Thomas GH, Reynolds LM. Polychlorinated terphenyls in paperboard samples. Bull Environ Contam Toxicol 10:37–41 (1973).
10. Villeneuve DC, Reynolds LM, Phillips WE. Residues of PCB's and PCT's in Canadian and imported European cheeses, Canada—1972. Pestic Monit J 7:95–96 (1973).
11. Villeneuve DC, Reynolds LM, Thomas GH, Phillips WE. Polychlorinated biphenyls and polychlorinated terphenyls in Canadian food packaging materials. J Assoc Off Anal Chem 56:999–1001 (1973).
12. Mestres R, Pagnon M, Duboul-Razavet C. Pesticide residues and polychlorinated biphenyls in the sediments of the continental shelf in the Mediterranean Sea. Trav Soc Pharm Montp 35:181–194 (1975).
13. Fukano S, Ushio F, Doguchi M. PCBs, PCTs, and pesticide residues in fish collected from the Tama river. Tokyo Toritsu Eisei Kenkyusho Kenkyu Nempo 25:297–305 (1974).
14. Minagawa K, Takigawa Y, Sakai H, Sasagawa I. Polychlorinated terphenyls in human fat and mother's milk. Nippon Eiseigaku Zasshi 28:543–547 (1974).
15. Shirai F. Detection of polychlorinated terphenyls on food wrappers by reversed-phase partition thin-layer chromatography. Eisei Kagaku 20:282–286 (1974).
16. Doguchi M, Fukano S. Residue levels of polychlorinated terphenyls, polychlorinated biphenyls, and DDT in human blood. Bull Environ Contam Toxicol 13:57–63 (1975).
17. Stratton CL, Sosebee JB Jr. PCB and PCT contamination of the environment near sites of manufacture and use. Environ Sci Technol 10: 1229–1234 (1976).
18. Doguchi M. Polychlorinated terphenyls as an environmental pollutant in Japan. Ecotoxicol Environ Saf 1:239–248 (1977).
19. Fukano S, Doguchi M. PCT, PCB and pesticide residues in human fat and blood. Bull Environ Contam Toxicol 17:613–617 (1977).
20. Hassell KD, Holmes DC. Polychlorinated terphenyls (PCT) in some British birds. Bull Environ Contam Toxicol 17:618–621 (1977).
21. Falandysz J, Stangret I. Use of concentrated sulfuric acid and alcoholic potassium hydroxide for the determination of residues of polychlorinated pesticides and polychlorinated bi- and terphenyls in fish oils and cod-liver oils. Farm Pol 35:465–472 (1979).
22. Ushio F, Doguchi M, Fukano S, Abe M. PCT (polychlorinated terphenyls), PCB, and organochlorine pesticide residues in wild crows and pigeons. Tokyo-toritsu Eisei Kenkyusho Kenkyu Nempo 31:209–211 (1980).
23. Buchert H, Bihler S, Schott P, Röper HP, Pachur HJ, Ballschmiter K. Organochlorine pollutant analysis of contaminated and uncontaminated lake sediments by high resolution gas chromatography. Chemosphere 10:945–956 (1981).
24. Renberg L, Sundström G, Reuthergaardh L. Polychlorinated terphenyls (PCT) in Swedish white-tailed eagles and in grey seals. A preliminary study. Chemosphere 6:477–482 (1981).
25. Haga T, Ozaki K, Tominaga Y. Composition of river sediment downstream from waste paper recycling plant. Niigata Rikagaku 10:43–45 (1984).
26. Furlong ET, Carter DS, Hites RA. Organic contaminants in sediments from the Trenton Channel of the Detroit River, Michigan. J Gt Lakes Res 14:489–501 (1988).
27. Hale RC, Greaves J, Gallagher K, Vadas GG. Novel chlorinated terphenyls in sediments and shellfish of an estuarine environment. Environ Sci Technol 24:1727–1731 (1990).
28. Risebrough RW, De Lappe BW, Younghans-Haug C. PCB and PCT contamination in Winter Quarters Bay, Antarctica. Mar Pollut Bull 21:523–529 (1990).
29. Canton L, Grimalt JO. Distribution of river-transported halogenated biphenyls and terphenyls in coastal environments. Chemosphere 23:327–341 (1991).
30. Hale RC, Greaves J, Vadas GG, Harvey E, Gallagher K. Distribution of polychlorinated terphenyls in tributaries of the southern Chesapeake Bay. Aquat Toxicol Risk Assess 14:305–312 (1991).
31. Galceran MT, Santos FJ, Caixach J, Rivera J. PCBs and chlorinated pesticides in shellfish of a deltaic environment. Chemosphere 27: 1183–1200 (1993).
32. Galceran MT, Santos FJ, Caixach J, Ventura F, Rivera J. Environmental analysis of polychlorinated terphenyls: distribution in shellfish from the Ebro Delta (Mediterranean). J Chromatogr 643:399–408 (1993).
33. Gallagher K, Hale RC, Greaves J, Bush EO, Stilwell DA. Accumulation of polychlorinated terphenyls in aquatic biota of an estuarine creek. Ecotoxicol Environ Saf 26:302–312 (1993).
34. Germany: State Study Group "Waste" [Länderarbeitsgemeinschaft "Abfall" (LAGA)]. German industrial norm (DIN) 51527: determination of polychlorinated biphenyls (PCB). Berlin:Beuth Verlag, 1987.
35. Ullrich D. Results of an intercomparison exercise to determine PCB in indoor air. In: Proceedings of the international conference on indoor air quality and climate (6), vol 2 (Jaakola JJK, Illmarine R. Seppänen O, eds), 4–8 July 1993, Helsinki, Finland. Helsinki:Helsinki University of Technology 1993;363–368.
36. Hutzinger O, Safe S, Zitko V. The chemistry of PCBs. Cleveland, OH:CRC Press, 1974.
37. Rappe Ch, Buser HR. Chemical and physical properties, analytical methods, sources and environmental levels of halogenated dibenzodioxins and dibenzofurans. In: Halogenated biphenyls, terphenyls, naphthalenes, dibenzodioxins and related products. 2nd ed. (Kimbrough RD, Jensen AA, eds). Amsterdam, New York, Oxford:Elsevier, 1989;71–102.
38. De Kok A, Geerdink RB, de Vries G, Brinkman UAT. An evaluation of chromatographic methods for the analysis of polychlorinated terphenyls in environmental samples. Int J Environ Anal Chem 12:99–122 (1982).
39. Ballschmiter K, Rappe Ch, Buser HR. Chemical properties, analytical methods and environmental levels of PCBs, PCTs, PCNs and PBBs. In: Halogenated biphenyls, terphenyls, naphthalenes, dibenzodioxins and related products. 2nd ed. (Kimbrough RD, Jensen AA, eds). Amsterdam, New York, Oxford:Elsevier, 1989;47–69.
40. De Voogt P, Brinkman UAT. Production, properties and usage of polychlorinated biphenyls. In: Halogenated biphenyls, terphenyls, naphthalenes, dibenzodioxins and related products. 2nd ed. (Kimbrough RD, Jensen AA, eds). Amsterdam, New York, Oxford:Elsevier, 1989;3–45.
41. [10. Verordnung zur durchführung des bundesimmissionsschutzgesetzes (beschränkungen von PCB, PCT and VC)- 10. BImSchV, vom 26. (BGBl. I S.1138. Bonn, Germany:Bundesministerium der Justiz, 1978.
42. European Community: directive of the council concerning restrictions on the marketing and use of certain dangerous substances and preparations (76/769), adopted on July 27, 1976, Brussels, Belgium.
43. Verordnung zum verbot von polychlorierten biphenylen, polychlorierten terphenylen und zur Beschränkung von vinylchlorid (PCB-, PCT-, VC-Verbotsverordnung), vom 18. BGBl. I S. 1482. Bonn, Germany:Bundesministerium der Justiz, 1989.
44. Jan J, Josipovic D. Polychlorinated terphenyls in hens. The behavior of *ortho-*, *meta-*, and *para-*isomers. Chemosphere 7:863–866 (1978).
45. Madge DS. Action of dibenzofurans and naphthalenes on intestinal solute transport and fluid transfer in mice. Gen Pharmacol 9:361–367 (1978).
46. Wingender RJ, Williams RM. Evidence for the long-distance atmospheric transport of polychlorinated terphenyl. Environ Sci Technol 18:625–628 (1984).
47. Sosa-Lucero JC, De la Iglesia FA, Thomas GH. Distribution of a polychlorinated terphenyl (PCT) (Aroclor 5460) in rat tissues and effect on hepatic microsomal mixed function oxidases. Bull Environ Contam Toxicol 10:248–256 (1973).
48. Sekita H, Takeda M, Uchiyama M, Kaneko T. Accumulation of polychlorinated terphenyls (PCT) in mouse tissues after long-term feeding of PCT-added diet. Eisei Kagaku 28:18–22 (1982).
49. Ushio F, Oishi S, Funayama K, Doguchi M. Accumulation and metabolism of polychlorinated terphenyls (PCTs). I. Relative amounts of three types of PCT isomers in adipose tissue and liver of mice. Eisei Kagaku 28:325–329 (1982).
50. Watanabe I, Yakushiji T, Kunita N. Distribution differences between polychlorinated terphenyls and polychlorinated biphenyls in human tissues. Bull Environ Contam Toxicol 25:810–815 (1980).
51. Fishbein L. Toxicity of chlorinated biphenyls. Ann Rev Pharmacol 14:139–156 (1974).
52. Norback DH, Allen JR, Lalich JJ. Chlorinated triphenyl-induced extensions of the hepatic endoplasmic reticulum. Proc Soc Exp Biol Med 139:1127–1131 (1972).
53. Shirai T, Miyata Y, Nakanishi K, Murasaki G, Ito N. Hepatocarcinogenicity of polychlorinated terphenyl (PCT) in ICR mice and its enhancement by hexachlorobenzene (HCB). Cancer Lett 4:271–275 (1978).
54. Allen SR, Norback PH. Polychlorinated biphenyl and triphenyl-induced gastric mucosal hyperplasia in primates. Science 179:498–499 (1973).
55. Adamson IYR, Weeks JL. The LD_{50} and chronic toxicity of reactor terphenyls. Arch Environ Health 27:69–73 (1973).

56. Toftgaard R, Nilsen OG, Carlstedt-Duke J, Glaumann H. Polychlorinated terphenyls: alterations in liver morphology and induction of cytochrome P-450. Toxicology 41:131–144 (1986).
57. Nilsen OG, Tolfgard R. Effect of polychlorinated terphenyls and paraffins on rat liver microsomal cytochrome P-450 and *in vitro* metabolic activities. Arch Toxicol 47:1–11 (1981).
58. Bitman J, Cecil HC, Harris SS. Biological effects of polychlorinated biphenyls in rats and quail. Environ Health Perspect 1:145–149 (1972).
59. Cecil HC, Harris SJ, Bitman J. Effect of polychlorinated biphenyls and terphenyls and polybrominated biphenyls on pentobarbital sleeping times of Japanese quail. Arch Environ Contam Toxicol 3:183–192 (1975).
60. Ahotupa M, Aitio A. Effect of chlorinated naphthalenes and terphenyls on the activities of drug metabolizing enzymes in rat liver. Biochem Biophys Res Commun 93:250–257 (1980).
61. Leece BD, Denomme MA, Li SMA, Towner RA, Gyorkos JW, Chittim BG, Safe S. Effects of individual terphenyls and polychlorinated terphenyls on rat hepatic microsomal cytochrome P-450-dependent monooxygenases: structure–activity relationships. Arch Toxicol 59:186–189 (1986).
62. Bandiera S, Sawyer TW, Campbell MA, Fujita T, Safe S. Competitive binding to the cytosolic 2,3,7,8-tetrachlorodibenzo-*p*-dioxin receptor. Effects of structure on the affinities of substituted halogenated biphenyls—a QSAR analysis. Biochem Pharmacol 32:3803–3815 (1983).
63. Davis DL, Bradlow HL, Wolff M, Woodruff T, Hoel DG, Anton-Culver H. Medical hypothesis: xenoestrogens as preventable causes of breast cancer. Environ Health Perspect 101:372–377 (1993).
64. Cecil HC, Bitman J, Lillie RJ, Verret J. Embryotoxic and teratogenic effects in unhatched fertile eggs from hens fed polychlorinated biphenyls (PCBs). Bull Environ Contam Toxicol 11:489–495 (1974).
65. Lillie RJ, Cecil HC, Bitman J, Fries GF. Differences in response of caged white leghorn layers to various polychlorinated biphenyls (PCBs) in the diet. Poult Sci 53:726–732 (1974).
66. Kholkute SD, Rodriguez J, Dukelow WR. The effects of polybrominated biphenyls and perchlorinated terphenyls on *in vitro* fertilization in the mouse. Arch Environ Contam Toxicol 26:208-211 (1994).