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Late lessons from early warnings: the precautionary principle 1896–2000

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6. PCBs and the precautionary principle

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6.1. Introduction

Polychlorinated biphenyls (PCBs) are chlorinated organic compounds that were first synthesised in the laboratory in 1881. By 1899 a pathological condition named chloracne had been identified, a painful disfiguring skin disease that affected people employed in the chlorinated organic industry. Mass production of PCBs for commercial use started in 1929. Thirty-seven years elapsed before PCBs became a major public issue, recognised as environmental pollutants, and a danger to animals and humans. Large-scale production worldwide, and in particular in some eastern European countries, continued until the mid-1980s. PCBs are the first obvious example of a substance that was not intentionally spread into the environment, but nevertheless became widespread and bioaccumulated to high concentrations.

PCBs are mixtures of synthetic organic chemicals with the same basic chemical structure, and similar physical properties, that range in nature from oily liquids to waxy solids. PCBs were well received in the marketplace as they replaced products that were more flammable, less stable and bulkier. This new group of chemicals facilitated the production of smaller, lighter and what were thought to be safer electrical equipment. In the United States the use of PCBs was very important in the Second World War. PCBs were primarily used in electrical equipment, such as capacitors and transformers, because of their insulating properties and resistance to high temperatures. Over the years the number of uses of PCBs increased to include use as heat-transfer fluids in heat exchangers, as hydraulic fluids, as ingredients in PVC plastics, paints, adhesives, lubricants, carbonless copy paper and as immersion oil for microscopes. Sealants containing PCBs were widely used for constructing and renovating buildings. Between 1929 and 1988 the total world production of PCBs (excluding the USSR and China) was 1.5 million tonnes.

By the late 1930s Monsanto, the US producer of PCBs, was certainly aware of adverse health effects in workers exposed to PCBs. For example, in 1936 several workers at the Halowax Corporation in New York City exposed to PCBs (then called chlorinated diphenyls), and related chemicals called chlorinated naphthalenes, were affected by chloracne. Three workers died and autopsies of two revealed severe liver damage. Halowax asked Harvard University researcher Cecil K. Drinker to investigate. Drinker presented his results at a 1937 meeting attended by Monsanto, General Electric, Halowax, the US Public Health Service and state health officials from Massachusetts and Connecticut. Like the Halowax workers, Drinker's test rats had suffered severe liver damage. Sanford Brown, the president of Halowax, concluded the meeting by stressing the 'necessity of not creating mob hysteria on the part of workmen in the plants' (Francis, 1998). The results were published but did not gain the wider attention of policy-makers (Drinker *et al.*, 1937). Drinker's article did however put the occupational medicine community, labour regulators and manufacturers on notice as to the concerns surrounding PCBs.

The first warning that PCBs were becoming ubiquitous in the environment came from Søren Jensen. In 1966 Jensen, while working on DDT (dichlorodiphenyltrichloroethane), fortuitously detected unknown molecules in the muscle of white-tailed sea eagles in Sweden. The levels were appreciably higher in the fish-eating sea eagles than in fish collected from the same areas. So he concluded that the molecules must be persistent in living tissues and not easily broken down. The mystery chemicals were extremely resistant to degradation, being unaffected even when boiled in concentrated sulphuric acid. It took two further years of study for Jensen to be able to demonstrate that they were PCBs. In 1969 Søren Jensen published his findings (Jensen *et al.*, 1969) which showed remarkably high PCB concentrations in a large proportion of the Baltic Sea fauna (See Figure 6.1.). PCBs had entered the environment in large quantities

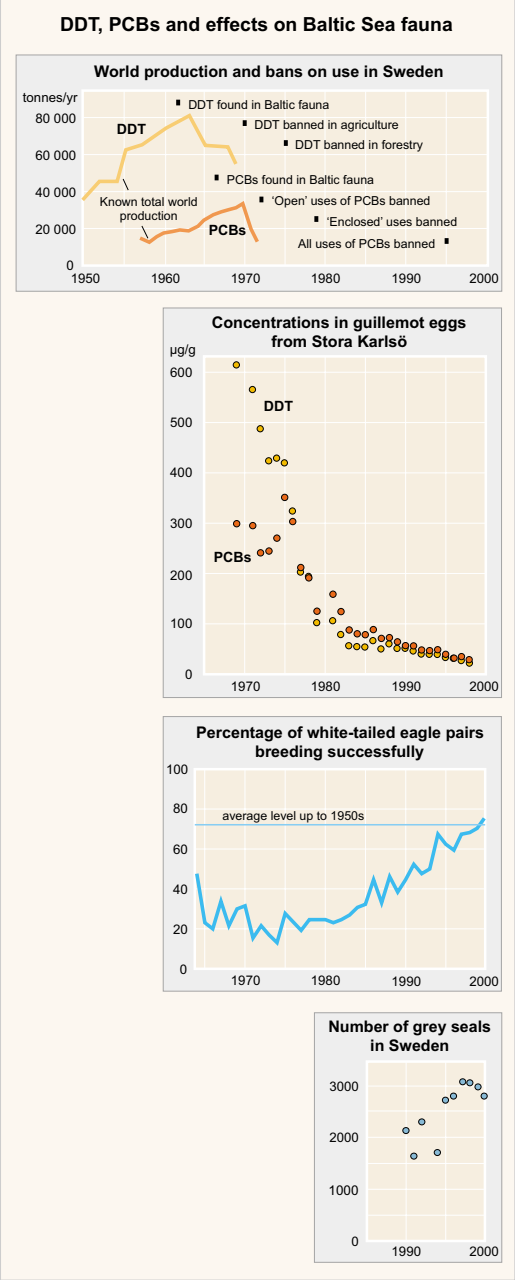
for more than 37 years and were bioaccumulating along the food chain.

In the 1960s it became apparent that the fertility of all three seal species occurring in the Baltic Sea was in decline. By the 1970s nearly 80 % of the females were infertile. Some studies drew a link with the presence of persistent pollutants — high levels of DDT and PCBs had been recorded in all three species. A clear correlation was found between the pathological uterine changes and elevated concentrations of contaminants, particularly PCBs. Further studies appeared to link high PCB levels not only with reproductive disorders in seals but with other symptoms such as damage to skin and claws, intestines, kidneys, adrenal glands and skeleton (Swedish Environmental Protection Agency, 1998).

The first well-publicised warning that PCBs could be harmful to humans came from an accident in 1968 in Japan with a mass poisoning among 1 800 people who ingested contaminated rice oil. The rice oil was found to contain a large amount of Kanechlor 400, a brand of PCB, that was believed to have leaked from a heating pipe in the factory (Kimburgh *et al.*, 1987). This resulted in serious health problems for those who had consumed the contaminated oil (see Box 6.1.). The impact of this incident was such as to give rise to a new word in the Japanese language — Yusho — or Japanese rice oil disease. Many debates ensued as to whether it was the PCBs or their breakdown products that had caused these effects. However, it was agreed that PCBs that had been subjected to heating did have harmful effects on humans and that if PCBs were present in places where food was processed accidents like this one could happen.

In the late 1960s press reports about the presence of PCBs in the environment began to appear and in response Monsanto launched its public defence, denying that the chemicals were PCBs. 'The Swedish and American scientists... imply that polychlorinated biphenyls are 'highly toxic' chemicals,' Monsanto said in a widely distributed statement. It continued: 'This is simply not true. The source of marine-life residue identified as PCB is not yet known. It will take extensive research, on a worldwide basis, to confirm or deny the initial scientific conclusions.' (Francis, 1998)

Figure 6.1.



Source: Bernes, C. (2001) 'Will time heal every wound'

Nevertheless in 1969 Monsanto privately took a different view in its internal 'Pollution abatement plan', which admitted that 'the problem involves the entire United States, Canada and sections of Europe, especially the United Kingdom and Sweden.... other areas of Europe, Asia and Latin America will surely become involved. Evidence of contamination (has) been shown in some of the very remote parts of the world.' The plan also stated that stopping the production of PCBs was not an option as it would cause 'profits to cease and liability to soar because we would be admitting guilt by our actions' (Francis, 1998).

Box 6.1. The Yusho accident
The first clinical sign was a strange skin disease with conjunctivitis, swelling of eyelids and chloracne. The victims' whole bodies, including the extremities, were covered with acne-like pustules. The most common symptoms were pigmentation of nails, skin and mucous membranes; increased sweating of the palms; severe headache; swollen joints and feelings of weakness. About half of the victims coughed persistently with expectoration resulting in a chronic bronchitis. These respiratory symptoms correlated with PCB levels in the blood. It took more than 10 years for these symptoms to decline.
Some of the victims were pregnant when they consumed the PCB-contaminated oil. Of 11 babies born to these mothers, two were stillborn. All were 'Coca-Cola' coloured, growth-retarded, had increased eye discharges and nail pigmentation. Follow-up studies of the children showed their growth to be hampered, their IQs to be low, and their demeanour generally apathetic and dull. There was an increased mortality of the whole group of Yusho patients due to malignancies of the liver and respiratory system. Their body burden of dioxins was about 450 micrograms dioxin toxic equivalent level per kilogram, not including the amounts of phenobarbital-like PCBs (Masuda, 1994).

6.2. Growing evidence of persistence, presence and toxicity

During the 1970s the evidence that PCBs were ubiquitous environmental pollutants continued to build. PCBs were found in remote areas such as the Arctic. In the Netherlands, large amounts of PCBs were found to be entering the environment from the River Rhine. At Lobith, on the German border, inputs measured between 1976 and 1981 ranged from 14 300 kg to 24 000 kg. Most of the PCBs were attached to fine silt in suspension; consequently the levels in the sediments were highest where the flow rates of the rivers were low. The sediments in Rotterdam harbour had the highest concentrations — 12–24 mg/kg. As these sediments were used for reclaimed land, this resulted in the Netherlands being contaminated at the rate of 5 000 kg of PCBs a year. Fatty fish caught off the Dutch coast were highly polluted. Eels from the rivers and lakes, tested in 1977 and 1988, contained 3.0–131 mg/kg of PCBs (CBS, 1980; Greve and Wegman, 1983). PCBs were found in fish, mink, seabirds and in humans; these studies provided further evidence that PCBs bioaccumulate. Evidence of actual or suspected harm caused by their bioaccumulation was also being documented (see the case study on the Great Lakes).

What also became clearer throughout the 1970s was an understanding of the major reason for the disagreement between those who said PCBs were harmful in low quantities and those who said they were not. It was found that different forms — 'congeners' — of PCBs have different numbers and positions of chlorine atoms that determine the molecule's physical and chemical properties. Studies in the late 1970s recognised the significance of these different congeners, although at first the differences were wrongly attributed solely to the level of chlorination. This proved too simplistic, and after confusing the debate for a while it became evident that both the position and numbers of the chlorine atoms had an effect on toxicity — and that different congeners have different effects (see Box 6.2.).

6.3. Action from industry and governments in the 1970s

In 1971 Monsanto realised that its public position was untenable and voluntarily limited the types of PCB mixtures that had the overall group trade name Aroclor to those containing less than 60 % chlorine substitution (by weight). They also reformulated one Aroclor to reduce the percentage of higher-chlorinated PCBs. This move was based on the then current scientific opinion that molecules with fewer chlorine atoms were less toxic. Unfortunately, as described above, this opinion was soon found to be an oversimplification.

In 1972, as a result of the reports concerning the presence and effects of PCBs in the environment, Sweden banned these substances for 'open' uses, such as in sealants, paints and plastics, which resulted in uncontrolled losses to the environment. The first international governmental action was in February 1973, when the Organisation for Economic Co-operation and Development (OECD) made Council Decision C(73) 1 (Final) on protection of the environment by control of polychlorinated biphenyls. The OECD's decision was taken as a result of 'concerns about environmental contamination by PCBs, and their health and environmental effects' (OECD, 1973). The decision required use in new open products to be banned in OECD member states. However, large amounts continued to be used in supposedly 'closed systems' such as transformers, probably as the technical problems and costs of measures needed to replace them were considered prohibitive.

In the United States, Congress also responded to the dangers associated with the use of PCBs and other toxic chemicals. In 1976 the Toxic Substances Control Act was passed. Although the act covered the regulation of all chemical substances, the severity of the threat posed by PCBs resulted in a section (6(e)) devoted solely to PCBs. No other chemicals received such singular attention. During the debate over the Senate version of the act, Senator Nelson, the author of Section 6(e), noted that PCBs were widespread in the environment and that they posed significant potential dangers to human health and to wildlife (United States Court of Appeal, 1980). The section required that, one year after the act came into effect, PCBs could only be manufactured, processed, distributed and used in a 'totally enclosed manner'. Eighteen months later, all manufacture, processing and distribution of PCBs was prohibited. Production in the United Kingdom ended in 1978 and in the United States in 1979. However, elsewhere, and in particular in some eastern European countries, large-scale production continued until the mid-1980s (Boersma *et al.*, 1994).

6.4. Scientific understanding becomes more sophisticated

In 1979 came another costly reminder of how PCBs can cause serious harm to human health. In Taiwan 2 000 people were poisoned by polluted rice oil. The accident, named Yucheng, received more publicity and follow-up than the Yusho accident of the 1960s, demonstrating how awareness of PCBs as an environmental pollutant was changing. Cases with the typical clinical features as seen in the Yusho illness were reported. The children born to poisoned mothers became the subjects of a long-term health study. A quarter of the children died before the age of four years as a result of respiratory infections. At the age of eight years the children still had nail deformities and chronic otitis media (middle ear inflammation) together with bronchitis. Follow up studies of the adult victims of Yucheng showed an increase in skin allergies, chloracne, headache, spine and joint diseases and goitre (Guo, 1999).

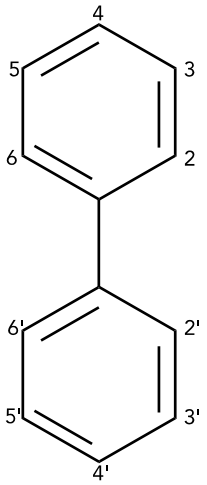
A further development in the understanding of the phenomena occurred in the 1980s, when it was realised that PCBs change during bioaccumulation and biodegradation in the environment. This allowed many of the earlier inconclusive studies to be reinterpreted and the apparently conflicting evidence to be resolved. Bioaccumulation through the food chain tends to concentrate congeners of higher chlorine content, producing residues that are considerably different from the original Aroclor mixtures (Schwartz *et al.*, 1987; Oliver and Niimi, 1988). Bioaccumulated PCBs appear to be more toxic than commercial PCBs because some toxic congeners can be preferentially retained (Aulerich *et al.*, 1986; Hornshaw *et al.*, 1983). Mink that were fed Great Lakes fish contaminated with PCBs showed liver and reproductive toxicity comparable to mink fed three times the quantity of Aroclor 1254 (Hornshaw *et al.*, 1983).

Evidence of PCB contamination of human breast milk also became apparent in the 1980s. Due to the high level of PCBs in the Dutch environment, the levels of these chemicals in the Dutch population remain among the highest in the world, with average PCB content in adipose tissue ranging from 1.6–2.5 mg/kg fat (Greve and Wegman, 1983).

In the 1980s came the first published studies on possible developmental effects in children. Examples of endocrine disruption became apparent. The penises of boys affected by the Yucheng accident were underdeveloped and — another sign of endocrine disruption in the prenatal period — their understanding of spatial relationships was diminished, an abnormality also seen in boys of mothers using phenobarbital in pregnancy (Dessens *et al.*, 1998). Children are more vulnerable to the toxic effects of PCBs than adults because they have developmental periods, so called 'windows of time', when metabolic systems develop (see also the case study on DES). During the intrauterine development of a child and in the postnatal period, many processes take place that fix homeostatic systems to states that then persist throughout life. Examples include a steady body temperature of 37 degrees Celsius; the setpoints for circulating hormone levels; and the functioning of the immune system. This 'fetotoxicity' represented a new paradigm for toxicology, one where both dose and timing is important. A natural example is the exposure to viral rubella infection in the first three months of gestation when the different organs are formed. Depending on the day of development of the organs the heart or a leg or an eye or the brain is malformed.

Figure 6.2. **Biphenyl skeleton structure**

Source: Søren Jensen



Chlorine substitutions in positions 2 and 6 in the biphenyl are known as ortho-position substitutions, in 2 and 4 as metaposition substitutions, and at the four locations as a paraposition substitution.

Figure 6.3. **Dibenzofuran skeleton structure**

Source: Søren Jensen

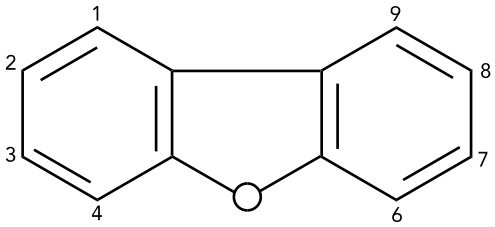
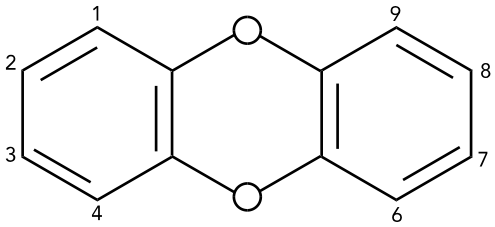


Figure 6.4. **Dibenzo-p-dioxin skeleton structure**

Source: Søren Jensen



Box 6.2. Explanation of the toxicity of PCBs by Søren Jensen

The basic biphenyl chemical form is shown in Figure 2. Theoretically 209 chlorinated biphenyls are possible, but the greater the number of chlorine atoms already present in the molecule, the more difficult it becomes for further substitution of hydrogen with chlorine. Only 135 congeners have been found in technical products or in biota.

In most PCB congeners chlorine atoms are quite evenly distributed between the two phenyl rings. From a toxicological viewpoint positions 2 and 6 are the most important (see Figure 6.2.) — these are called the ortho positions. The chlorine atom is rather bulky, so many chlorine substitutions in position 2 and 6 will force the biphenyl to twist around the axis. Most molecules have two or more chlorine molecules in the ortho position. Less frequent are molecules with only one chlorine atom in an ortho position, and in this case the molecule is more planar (flat). Finally the non-ortho congeners, called the co-planar PCBs, are only present in trace amounts in technical products.

Polychlorinated dibenzofurans (see Figure 6.3.) are created as accidental by-products during the iron-catalysed synthesis of PCB. They are also formed during fires in oxygen-poor environments such as fires in PCB-containing transformers; in products used as heat-transfer agents; and in other types of PCB wastes exposed to heat or fire.

Polychlorinated dibenzo-p-dioxins (see Figure 6.4.) are closely related to the dibenzofurans. The 2,3,7,8-tetra chloro congener was first found as a by-product in the 2,4,5-trichloro-phenoxy-acid herbicides. TCDD (tetrachlorodibenzoparadioxin) is also formed in fires where chlorine is present. TCDD is the most toxic substance of which we know, this being the result of its interaction with a vital cell cytoplasm protein known as the Ah-receptor, which in turn affects enzyme production. This effect, via the Ah-receptor, is often simply referred to as a 'dioxin-like effect'. TCDD is a liver and nerve, and bone-marrow toxin and carcinogenic in human beings.

Different chlorinated dioxins have been given a TEF-value (toxic equivalent factor), where the ability of TCDD to induce aryl-hydrocarbon-hydroxylase activity via the Ah- receptor was given the value 1. The different chlorinated dibenzofurans could be given a TEF-value as they have been proven to possess a dioxin-like effect. Generally the effect is somewhat lower than the dioxin with the same substitution pattern.

The ability to possess a dioxin-like effect is linked both to the planarity of the molecule and the specific chlorine substitution pattern. Some PCB congeners are planar enough to have a small dioxin-like effect — especially the non-ortho congeners. The same is true, at a still lower TEF-value, for those PCB molecules with only one ortho chlorine. The total dioxin-like effect is, however, the TEF-value multiplied by the total amount present. As a result the net dioxin-like effect of the one ortho congeners can be substantial, as they are present in organisms at a relatively high level. The majority of the PCB-congeners in biological samples as well as in technical products have two or more ortho substituents; they are too twisted to bind to the Ah-receptor and their TEF-value is zero. They possess however a phenobarbital-like effect, affecting various enzymes in a different manner from dioxins.

Prenatal exposure to PCBs and furans (polychlorinated dibenzofurans, or PCDFs, see Box 6.2.) was associated with negative behavioural effects amongst the Yusho offspring. The children were apathetic and uninterested in their surroundings (Harada, 1976). Monkeys prenatally exposed to PCBs exhibited hyperactive behaviour during infancy, followed by inactivity at four years of age (Bowman and Heironimus, 1981). The four-year-old children of mothers whose diets had included significant amounts of fish from Lake Michigan also displayed reduced activity in relation to their body burdens of PCBs (Jacobson *et al.*, 1990). Furthermore, at the age of 11 years, children more highly exposed in the prenatal period had lower IQ-test scores, and showed difficulties in verbal comprehension and reduced ability to concentrate. They were also more than twice as likely to be two years behind in reading skills and word comprehension (Jacobson and Jacobson, 1996). Further research has demonstrated negative effects as a result of prenatal exposure to background levels of PCBs in the United States, Canada and western Europe (see Box 6.3.).

6.5. Government action in the 1980s and 1990s

The North Sea ministerial conferences, in conjunction with the Oslo and Paris conventions on the protection of the Northeast Atlantic, have been important international fora that have stimulated action on hazardous substances. At the first North Sea conference in 1984, it was agreed that the phasing out of the use and discharge of PCBs should be intensified. At the second North Sea conference in 1987 targets were agreed to reduce discharges of 'substances that are toxic, persistent and liable to bioaccumulate' by the order of 50 % by 1995 (DoE, 1987).

In 1987, the OECD made a further decision on PCBs, on the basis that 'current controls of polychlorinated biphenyl have not led to a clear and consistent downward trend of environmental levels of PCBs. Previous concerns about environmental contamination by PCBs, and their health and environmental effects remain unabated. New concerns have arisen over the use of PCBs particularly in situations where highly toxic products such as chlorinated dioxins or chlorinated dibenzofurans might be produced by their decomposition in fires.' (OECD, 1987). The decision recommended that member countries should cease the manufacture, import, export and sale of PCBs by 1 January 1989. The decision also called for the acceleration of the withdrawal of PCBs from use.

During the 1980s some national governments issued health advice suggesting the reduction of consumption of PCB-contaminated fish and time limits on breast-feeding. The advice was controversial and heavily debated because of the positive benefits to health from both breast-feeding and the consumption of fish (Fuerst *et al.*, 1992).

In 1990 the third North Sea conference agreed a specific plan to phase out the use of PCBs by 1999 and safely dispose of them by the same date (DoE, 1990). In 1995 the Barcelona Convention for the Protection of the Mediterranean Sea against Pollution agreed to 'reduce, by the year 2005, discharges and emissions which could reach the marine environment, of substances which are toxic, persistent and liable to bioaccumulate, in particular organohalogens, to levels that are not harmful to man or nature with a view to their gradual elimination'. In the same year Sweden prohibited the use of old equipment which contained PCBs.

At the May 1995 meeting of the United Nations Environment Programme (UNEP) Governing Council, Decision 18/32 was adopted on persistent organic pollutants (POPs). This decision resulted in a number of studies and meetings culminating in November 1995 with the Washington Declaration, an agreement to a global programme of action to phase out POPs, including PCBs. The declaration was signed by 100 national governments.

In 1996 Directive EC96/59 of the European Union called for the elimination of PCBs and PCTs (polychlorinated triphenyls) and their phase-out by 2010. However, transformers filled with PCB oil are still in use. As they become older and rust, the chances of leakage into the environment increase.

Some 100 years after the first serious adverse effects had been documented, closure had finally been reached on the seriousness of the threat. However, the toxic legacy will remain for many decades.

Box 6.3. Further research on fetotoxicity
In Rotterdam and Gröningen in the Netherlands, starting in 1990 and 1991, a study was performed to investigate the effects of prenatal exposure to background levels of PCBs and dioxins on growth and development of the child (Huisman, 1996; Koopman-Esseboom, 1995; Patandin, 1999; Lanting, 1999). The total study group consisted of 400 healthy mother-infant pairs, of which half the infants were breast-fed and half bottle-fed. Prenatal PCB exposure was estimated by the PCB-sum (PCB congeners 118, 138, 153 and 180) in maternal blood and cord blood and the total dioxin toxic equivalent level (TEQ) in the mother's breast milk (17 dioxin and a total of 8 dioxin-like PCB congeners — 3 planar, 3 mono-ortho and 2 di-ortho PCBs). Postnatal dioxin exposure was calculated as a product of the total dioxin TEQ level in breast milk multiplied by the weeks of breast-feeding.
Of the measured PCB congeners 118, 138, 153 and 180, the first is dioxin-like, but the last three are phenobarbital-like. In general 63 % of the total amount of PCBs in human breast milk is ortho-substituted non-planar (PCB-22, -52, -138, -153 and -180) — that is phenobarbital-like PCBs. Current body exposure to the PCB congeners 118, 138, 153 and 180 was measured in plasma at 42 months of age (Patandin, 1999).
The study detected hyperactivity and slower mean reaction times in relation to the current PCB levels in the children at 42 months of age. Irritability and hyperactivity are well-known side-effects of the use of phenobarbital in childhood. At the age of 42 months attention during free play behaviour was reduced relative to umbilical cord PCB concentrations and maternal PCB exposure. This persistent effect on behaviour from damage that happened prenatally is similar to that found by Jacobson (Jacobson *et al.*, 1990; Patandin, 1999). Effects of prenatal PCB exposure were also adversely associated with neurological outcome at 18 months of age (Huisman *et al.*, 1995), but this was no longer seen at 42 months of age (Lanting, 1998b). In contrast, prenatal and lactational exposure to dioxin-like PCBs and dioxins was not shown to affect attention and activity at 42 months of age (Patandin, 1999).
Thus, negative effects on cognitive and behavioural development, as demonstrated by Jacobson and the Dutch study, are related to the prenatal or current accumulated exposure to phenobarbital-like PCBs and not to dioxin-like PCBs.
A study by Seegal and Schantz (Seegal and Schantz, 1994) also demonstrated the different effects of phenobarbital-like PCB congeners and dioxin-like PCB congeners. Monkeys exposed to di-ortho PCBs were impaired on simple spatial discrimination and reversal problems. However, those monkeys exposed to TCDD (tetrachlorodibenzoparadioxin) performed better than the control group. The di-ortho-substituted PCB-congeners tested in adult monkeys are dopamine neurotoxicants. They reduce the amount of dopamine by inhibiting tyrosine hydroxylase — the enzyme which controls the rate at which dopamine is synthesised. It is probable that this is a long-term or even permanent effect (Seegal and Schantz, 1994). A particular concern is this may have implications relating to an increased incidence of Parkinson's disease.
Dessens found impaired spatial ability in human adults who had been prenatally exposed to anticonvulsants (mostly phenobarbital) (Dessens *et al.*, 1998). The new disease entity 'late haemorrhagic disease of the new-born', first detected in the late 1970s in Japan and western Europe and originally attributed to vitamin K deficiency, might also be related to the effects of phenobarbital-like PCBs (Koppe *et al.*, 1989; Bouwman, 1994). Prenatal exposure to background levels of dioxin-like PCB congeners has also been shown to affect thyroid hormone metabolism (Pluim *et al.*, 1992; Koopman-Esseboom *et al.*, 1994).

6.6. Routes of environmental exposure

When products containing PCBs decompose, or are destroyed by fire, significant amounts of the PCBs survive, because of their stability. During fires in oxygen-poor environments, and where PCBs are otherwise broken down, highly toxic polychlorinated dibenzofurans can be formed and released (see Box 6.2.). There have also been accidental releases of PCBs and associated contaminants into the environment via leakage from 'sealed' PCB fluid compartments during commercial use of transformers and capacitors, and from the improper disposal of equipment or products.

By the late 1990s, even organisations generally critical of 'over-regulation' of industry had accepted that: 'In the past, discharges of PCB-laden wastes into rivers, streams, and open landfills were considered acceptable, legal, and hazard-free practices. PCBs were also sometimes intentionally released into the environment — for example, to reduce dust emissions from dirt roads, or as extenders in some agricultural pesticide formulations. In retrospect, these practices were inappropriate and potentially harmful.' (ASCH, 1997)

Once released, PCBs can volatise or disperse as aerosols, providing an effective means of transport in the environment. Strong south to north air flows, especially over west Eurasia, selectively favour the accumulation of PCBs and certain pesticides in the Arctic, once regarded as a pristine environment (AMAP, 1997). PCBs, together with the pesticides DDT, HCH and HCB, are detectable at low concentrations (0.01–40 nanograms (one thousand-millionth of a gram) per gram dry weight) in all samples of freshwater surface sediments from Alaska, northern Canada, Greenland, Norway, Finland and Russia (1995 and 1996). Freshwater and marine ecosystems tend to contain higher levels of PCBs than terrestrial ecosystems. Biomagnification of PCBs is especially significant in food webs dominated by organisms with a high fat content. Additionally, species overwintering at lower attitudes deliver POPs and metal burdens to the Arctic during the summer (Holden, 1970; AMAP, 1998).

Humans absorb PCBs from ingestion, inhalation and through the skin, although over 90 % of exposure is via food (Theelen and Lie, 1997). Animal fat is the major

source of PCBs. Once absorbed, PCBs equilibrate among lipid compartments in the whole body due to their high affinity for, notably, triglycerides and cholesterol esters and their lower affinity for phospholipids and cholesterol (Lanting *et al.*, 1998a). In the liver PCBs interfere with enzyme systems important for detoxification (Matthews and Anderson, 1975).

6.7. The most recent PCB accident

Despite the various international and national regulations and legislation, releases, whether accidental or deliberate, are inevitable. In Belgium, in January 1999, chickens were found to have been fed a mixture of animal fats mixed with 8–50 litres of PCBs and furans. Although the source of PCBs has not been proven it was strongly suspected that they derived from the illegal disposal of old transformers. Levels in chickens and eggs were high, varying for 2,3,4,7,8 PCDF from 1 299 picograms (one million-millionth of a gram) per gram in chicken fat to 1 103 pg/g in egg yolk. Total dioxin (toxic equivalent level or TEQ) was respectively 958 and 685 pg/g fat (Hens, 1999). According to WHO guidelines the tolerable daily intake or TDI for people is 1–4 picograms dioxin per kg bodyweight per day (WHO, 1999). The discovery was only made because the levels were so high as to cause chick edema (swelling of tissue due to increased fluid content). It is likely that other food contamination incidents have occurred at lower levels before and will continue to occur in future. This was underlined in 2000, a year later, when in another part of Belgium more animal food was found to be contaminated with PCBs and dioxins.

In 1999 Belgian toxicologists published an analysis of the incident and suggested that it was very unlikely that the isolated episode of contamination in Belgium would have caused health effects in the general population. Without providing data on the background levels of PCBs in the Belgian population, they speculated that a two- to three-fold increase would be comparable with levels in the 1980s of those regularly eating contaminated seafood (Bernard *et al.*, 1999). This fails to take into account Dutch studies showing the effects of background levels of PCBs and dioxins on unborn babies. Since the PCB levels in the Belgian population are comparable to those of the Dutch, their statement is somewhat surprising and appears reminiscent of Sanford Brown of the Halowax Corporation in 1937 when endeavouring 'not to alarm the workforce'. One can legitimately wonder just how much we have learned since then.

6.8. Conclusion

By the 1930s there was already evidence, some at a low level of proof, that PCBs could poison people. This information was largely retained within the industry, and does not appear to have been widely circulated amongst policy-makers or other stakeholders. The application of the precautionary principle at that time would have prevented the toxic legacy that now exists.

Thirty years later, by the end of the 1960s, there was a high level of proof, mainly due to the Yusho accident, that in certain circumstances PCBs, or their breakdown products, such as dibenzofurans, could cause serious harm to human health. The findings of Søren Jensen also offered a high degree of proof that PCBs did bioaccumulate and were present in the Baltic food chain. A lower, but still substantial level of proof of the adverse reproductive effects on marine mammals was also available from the Baltic seal studies. Had precautionary action at a level of proof less than 'beyond reasonable doubt' been acceptable to, and applied by, policy-makers of that era, their action would still have resulted in a more manageable, less costly problem than we are faced with today. Many years of use of PCBs would have been avoided.

The 1960s evidence, with attendant worries of future liabilities, is likely to have been a factor that influenced Monsanto to reformulate some of its PCB products in 1971. Unfortunately this action was based on interpretation of the then current, but incomplete, scientific knowledge of how PCBs could cause harm.

During the 1970s evidence increased both of the environmental transport of PCBs to the remotest parts of the world and of the potential of PCBs to cause harm. PCBs were becoming widespread in the environment even though generally not being intentionally spread. It took until the early 1970s for any government to act and even then these actions only affected new 'open' uses of PCBs. By the late 1970s some governments had accepted that there was a greater risk of harm and enacted legislation to stop new 'closed' uses of PCBs. At this

point there was no action by any government to address the problem of existing uses or the cleaning up of contaminated sites. It is probable that the technical difficulties and costs of such actions were the reasons behind this half measure. By the late 1970s, a few countries had called for all production to end. By this time alternatives to PCBs in closed uses were available.

In the 1980s a deeper understanding developed as to how PCBs caused harm. This did much to increase the level of certainty because some discrepancies between studies could now be explained as due to different PCB congeners. During this time the first evidence that PCBs could affect the unborn child was published. The North Sea states agreed that there was a danger and in 1987 adopted the political aspiration to reduce inputs to the marine environment by 'the order of' 50 %, by 1995.

Also in 1987 the OECD accepted that the level of concern had increased and current legislation had not been effective. At this point all OECD countries agreed to end all new uses of PCBs by 1989. This time the risk posed by existing uses of PCBs was fully acknowledged and the introduction of 'controls' was recommended, as well as the removal of PCB equipment in certain circumstances. Nevertheless, production of PCBs still continued in several countries.

Although nine states within the North Sea catchment agreed in 1990 to phase out the use of all PCBs, it took until the mid-1990s, and the UNEP Washington Declaration, for a significant global response to emerge, acknowledging the problem and agreeing the need to act on PCBs in use.

Even today some of the science is still being debated. Much of this is to do with the different effects of commercial PCB mixes and those found in the environment, especially those that have been bioaccumulated. It could be argued that further scientific study at an earlier time would have allowed an earlier resolution. However, it could also be argued that the call for more science can also be used as a reason to delay justifiable action.

The resistance of PCBs to degradation means they are expensive to destroy. As a result of the many delays, a large percentage of historic PCB production has escaped beyond our control into the environment. Wide dispersal, and bioaccumulative properties, means that in many cases PCBs are in places where their recovery and destruction is not possible. Also, in many countries where they are still in use, the facilities for safe destruction do not exist.

In 1999, during the negotiations for a global convention to phase out all POPs, attention was focused on PCBs still in use, particularly those posing a risk due to accident or leakage. While this focus on prevention of further releases is essential, it is also necessary to pay greater attention to our response to the high levels of PCBs that are already in the environment. Behavioural problems and respiratory diseases affecting children, two of today's most important problems in paediatrics, could be due, in substantial part, to intoxication with PCBs. There is an urgent need to find ways of reducing current body burdens of these chemicals in people. Of course, no equivalent action is possible for other species.

PCBs also highlight issues such as who judges what risks are acceptable, and whether all stakeholders are fairly represented in this debate. Both PCBs and dioxins are fetotoxic. Is it acceptable to tolerate risks for involuntary exposure to unborn babies, when this may affect their future capabilities and those of their offspring?

At almost every stage government action was taken only when there was a high level of scientific proof. The non-application of the precautionary principle has left us with a legacy, the total effects and costs of which can only be guessed at.

PCBs: early warnings and actions Table 6.1.

1899	Chloracne identified in workers in chlorinated organic industry
1929	Mass production of PCBs for commercial use begins
1936	More workers affected by chloracne and liver damage
1937	Chloracne and liver damage observed in experiments with rats. Results did not gain attention from policy-makers but both labour regulators and manufacturers were made aware of the concerns surrounding PCBs
1966	Jensen discovers unknown molecules in sea eagles in Sweden — only in 1969 was he able to demonstrate that they were PCBs
1968	Poisoning of 1 800 people who had ingested PCB-contaminated rice oil in Japan gives rise to a new Japanese word: Yusho — rice oil disease, and to the first well-publicised warning that PCBs are harmful to humans
1970s	High levels of PCBs found in infertile seals of three different species
1972	Sweden bans 'open' uses of PCBs
1976	Toxic Substances Control Act (United States) — PCBs to be used only in a 'totally enclosed manner'
1979	2 000 people again poisoned, in Taiwan, by polluted rice oil. Follow-up research showed that 25 % of children born of poisoned mothers died before the age of four years
1980s	Evidence of PCB contamination of breast milk
1990s	PCBs associated with IQ and brain effects in children exposed *in utero* to mothers' PCB-contaminated diets. Fetotoxicity represents a new paradigm for toxicology
1996	EU directive to eliminate PCBs, with phase-out by 2010
1999	Chicken food contaminated with PCBs is found in Belgium

Source: EEA

6.9. References

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