A BRIEF OVERVIEW OF THE HEALTH AND ENVIRONMENTAL EFFECTS OF PCBs

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INTRODUCTION

The PCBs (polychlorinated biphenyls) are a group of some 209 different compounds of which approximately 100 may be found in the technical mixes marketed. Manufacture began in 1929 in the United States and in 1930 in Germany. After 1945 manufacture spread to other countries including the UK and the USSR. Jensen (1966) first described PCBs from environmental samples due to their interference with the routine analysis of DDT, which by this time had been recognised as a contaminant of global significance. Following this discovery extensive evaluation of the problem took place and the UK and US manufacturing operations were voluntarily closed down in 1977. Manufacture continued in some European countries until at least late 1987 and possibly continues still in Spain.

PCBs were employed in a wide variety of applications where thermal and chemical stability made them substances of choice. Applications included transformer and capacitor dielectrics, heat transfer fluids, hydraulic fluids, lubricating and cutting oils, and plasticisers in the printing and paint industries. Waste arisings may therefore take the form of liquids (transformer fluids), solids (old capacitors and contaminated soils) or small components in larger equipment (chokes from fluorescent light fittings). This makes the problem of determining precise figures for PCB wastes difficult. OECD (1987) estimate that the UK has some 12,000 tonnes. This is a minimum estimate derived by assuming, for example, that transformer fluids are pure PCB. To take account of the chlorobenzene solvent used in these applications, the 3000 tonnes of pure PCBs needs to be multiplied by a factor of about 1.6 to give the volume of PCB waste involved. Contamination of soils in particular can lead to high volumes of material classified as special waste.

OECD (1987) give a comprehensive account of the PCB waste and management problem in member countries. This document notes that in the UK, PCBs are dealt with under the legislation on waste and special waste comprising the Control of Pollution Act 1974, The Control of Pollution (Special Waste) Regulations 1980, and the Control of Pollution (Licensing of Waste Disposal Regulations) 1976. The UK Department of the Environment has published a Code of Practice for the disposal of PCBs and PCB contaminated wastes (DoE 1984).

Within the European Community, PCBs are regarded as "blacklist substances"(Gardiner & Mance 1984) although no regulatory directive has yet been proposed. PCBs are also included in the proposed UK "red list" (Jones et al. 1988).

In addition to sources involving PCBs as finished products certain industries also appear to be associated with de novo synthesis as by-product. These include the PVC industry, where waste EDC tars are contaminated, together with aqueous effluents, by dichlorobiphenyls. Pulp and paper industries are also suspected of PCB production due to the use of chlorine bleaching. It is not clear however, to what degree this is due to the recycling of PCB containing carbonless copy paper. Generally, by-production results

in effluent levels of 0.01-0.1 ppb although levels of between 5 and 50 ppb have been found in effluents from the rubber industry and marine engine refurbishment plants.

GLOBAL CYCLING

An idea of the magnitude of the global problem may be gained from the figures presented by Tanabe (1988). He estimates that of a total global production of 1.2 million tonnes, some 31% is free in the wider environment. 4% is accounted for by degradation and incineration, leaving the balance of 780 kilotonnes still in use, or present in stores, dumps and landfills. It is further estimated that the 370 kilotons dispersed into the wider environment are largely retained in coastal sediments and open ocean water. The environmental ubiquity of PCBs may be largely explained by the importance of the atmosphere in the cycling process, although it is generally accepted that PCBs have not yet reached a global equilibrium despite being found in the tissues of organisms in the Antarctic and other remote areas. Larsson & Okla (1989) showed that the atmospheric deposition of PCBs in Sweden has not decreased in recent years despite the restrictions placed on open use of these chemicals. Decline in levels of DDT was observed leading these workers to suggest that the inappropriate storage and disposal of PCBs was responsible for the continued atmospheric input. They conclude that "the impact of local atmospheric PCB contamination on ecosystems in Sweden is greater today than it was over a decade ago when the PCB restrictions were first introduced." Chevreuil et al. (1989) remark that levels of PCB deposition in the Paris area in 1986 were higher than previously recorded. They also note that a large proportion of the PCBs contained in small capacitors and contaminated oils will enter the atmosphere through dumping and incineration of municipal waste.

An indication of the relative importance of atmospheric deposition of PCBs to the overall contaminant budget of the Great Lakes is found in the work of Arimoto (1989). He cites figures showing that this comprises 7% of the input to Lake Ontario, rising to 90% for Lake Superior. These values also indicate the relative importance of non atmospheric inputs which vary according to industrial point sources on each lake. Even in remote oceanic areas, appreciable deposition takes place as evidenced by the work of Knap et al. (1986) who measured PCBs deposited in sediment traps in the Sargasso Sea.

ENVIRONMENTAL EFFECTS

The extensive body of information concerning the global cycling of PCBs has been accumulated in response to concerns about the environmental impact of these chemicals. PCBs are highly persistent. Although there is evidence of biodegradation in contaminated sediments (see Brown & Wagner 1989) and some marine mammals appear to be able to selectively degrade some of the lower chlorinated congeners (Boon et al. 1987) the detoxification potential of these processes would appear to be rather limited. Indeed, Cummins (1988) has suggested that unless further escape of PCBs is prevented then the eventual extinction of marine mammals is a very

real possibility.

Acute toxic effects of PCBs in aquatic systems will occur at concentrations above 2ppb (parts per billion=ug/l) (Jones et al. 1988. In marine systems, acute toxicity will be evident at levels greater than 10ppb. At levels of 0.1-1ug/l growth of marine organisms is severely reduced. Train (1979) suggests a water quality criterion of 0.001ug/l for the protection of marine and freshwater organisms. Michaels et al. (1982) demonstrated photosynthetic suppression in marine diatoms at PCB levels of 50 ppb. Cosper et al.(1987 & 1988) consider that levels of this order of magnitude may result in the evolution of resistant strains of diatoms. Such extremely low levels require sophisticated analytical procedures, a factor which contributes to the difficulties of analysis of biological material also (see eg Allchin et al. 1989). There are, as a result of these analytical difficulties, significant problems relating to interlaboratory calibration.

Levels of PCBs in biological material may be several orders of magnitude higher than ambient. PCBs are bioconcentrated to a factor of 6000 for fish and 47000 for invertebrates (Jones et al. 1988). Train (1979) reports bioconcentration factors of between 2500 and 100,000. PCBs are among the chemicals known to be present in human adipose tissue, a result of a USEPA study which has now been discontinued (USEPA 1986). In the UK there are few reports in the open scientific literature but data presented by MAFF (1983) showed a range of 0.1-10ppm in male adipose tissue and a range of 0.1-1.5 ppm in female fat. The mean values were 0.7 and 0.6ppm respectively. It is thought that much of this body burden is accumulated through the diet, although uptake from the atmosphere is also thought to be possible. The widespread occurrence of these compounds in human adipose tissue is yet further evidence of their global ubiquity.

TOXICOLOGY

Interest in the toxicology of PCBs in mammalian systems was largely generated by two acute poisoning incidents. (Kashimoto & Miyata 1987; Chen & Hsu, 1987). In the first, in 1968, a case of mass food poisoning, the "Yusho" incident, occurred in Japan affecting 1600 people. The second, the "Yu-Cheng" incident in central Taiwan in 1979, affected 2000 people. The cause was identified as the consumption of cooking oil contaminated with commercial PCB mixtures leaked from a heat exchanger. The symptoms included dermal abnormalities (acne, patches of dark pigment, skin swelling and thickening); eye problems (including discharges and pigmentation change); respiratory problems (chronic bronchitis); neurological disorders (headache and visual impairment) and other effects such as fatigue and anorexia. Symptoms persisted for 10 years after the initial event. That these effects were caused by PCBs alone is, however, questioned (Kashimoto and Miyata 1987). It was subsequently found that the oil responsible for the Yusho incident also contained comparatively high levels of polychlorinated dibenzofurans (PCDF) and polychlorinated quaterphenyls (PCQ). Recently, these contaminants have been identified as common contaminants of transformer fluids and are thought to be a significant contributor to

the acute toxic effects of PCB mixtures. This may be a particular problem where high operating temperatures have caused partial pyrolysis of the oil (Narang et al. 1988 & 1989; Hardin et al. 1989). Brown et al.(1988) report dibenzofuran contamination in a variety of Aroclor mixes. They consider that these arise from trace contaminants in the benzene used in the original synthesis of the PCBs. They note that this contamination is unlikely to arise in electrical equipment in normal use. Lack of resolution concerning this point means at worst that toxicity must be considered as a combinative function. This simply adds further difficulties to the already difficult problem of evaluating the toxicology of the 209 congeners and 100 possible constituents of technical PCB mixtures. There is little doubt that PCBs in their own right can exert significant toxicological effects. For example, Heinzow & Tinneberg (1989) describe changes to an in vitro index of immuno-modulation due to PCBs and discuss similar evidence in the literature. Recently, moreover, the non-ortho substituted (co-planar) PCBs, of which there are twenty, have come under scrutiny as a highly toxic component of technical PCB mixtures (See eg Tanabe 1987 & 1988).

Safe (1984) in a review of PCB toxicity, lists sub-lethal effects shown in laboratory animals including dermal lesions, liver damage and carcinogenesis, immmunotoxicity (impairment of immune response and including measurable damage to parts of the immune system), neurotoxicity, behavioural effects, reproductive impairment and hormonal abnormalities. In a later paper (Safe 1987) he concludes that based upon the severity and duration of the toxic symptoms observed in workers exposed to high levels of PCBs it is unlikely that environmental exposure to these chemicals leads to any adverse human effects.

That this view is an extreme oversimplification of the problem may be inferred from the growing literature on sub-lethal effects of PCBs upon humans and animals. Indeed, Safe (1987) concludes "the best indicators of the adverse health effects of PCBs and their role in carcinogenicicty will be derived from the continuing epidemiological and retrospective studies on occupationally exposed workers", thereby acknowledging the widescale uncertainties in this field of toxicology. It should be remembered that lack of success in detecting an effect does not necessarily imply that one does not exist. This is echoed by Smith & Brown (1987) who review the equivocal evidence furnished by study of electrical equipment manufacture. They state: "with time....a more definite conclusion regarding cancer mortality and PCB exposure may become evident." Certainly, in rats fed PCB dietary supplements, changes in vitamin A,C & E metabolism have been described which could not be explained in terms of lipid peroxidation mechanisms (Oda et al. 1987). Where lipid peroxidation is induced by activation of the liver mixed function oxidases (MFOs), a wide variety of effects may be observed at the cellular level (see: Rifkind & Muschik 1983). MFOs mediate free radical disruption of cell membranes causing the further liberation of free radicals. Eventually these may give rise to a carcinogenic effect.

Carcinogenicity, moreover is only one of a spectrum of possible effects. The well known reproductive suppression reported from

seals fed on contaminated North Sea fish (Reijnders 1986) may be paralleled by reproductive effects in humans. Pines et al. (1987) compared organochlorine levels in 29 male patients with decreased fertility against 14 randomly selected males. These two groups were matched in that they were of a similar age range, with similar smoking habits and no history of exposure to PCB. The males with fertility problems generally exhibited higher levels of organochlorine blood residues than did the control group. This was statistically significant for DDT and DDE, lindane and certain PCB congeners (tetra- and penta-), although blood levels of heptachlorepoxide, hexa-PCBs and DDD (another derivative of DDT) were actually lower in the infertile males. This investigation is at an early stage and the need for further research is clear.

Similarly, but from a much larger sample, a correlation has been observed between human miscarriage and the presence of organochlorine compounds. Leoni et al. (1989) compared the blood levels of contaminants in 120 women hospitalised for miscarriage and 120 full-term pregnancy controls. The average PCB level was higher in the miscarriage group. No differences were detected in hexachlorobenzene (another persistent pesticide) and DDT levels between the groups. Curiously, diet was not indicated as the source of PCBs based upon a study of food intake in the subjects. The authors state that the sources of PCBs in the experimental subjects was not clear.

Maternal exposure to PCBS has been correlated with congenital effects. Infants whose mothers had been involved in the Yusho incident were evaluated in 1985 and were found to be shorter and lighter than the children of unexposed mothers (Rogan et al., 1988). They also more frequently had skin abnormalities and some behavioural and developmental problems. The Japanese poisoning also served to illustrate that PCBs and similar compounds could be transferred from mother to foetus via the placenta and also to suckling babies in mother's milk (Kashimoto and Miyata, 1987). Such effects have been reported in epidemiological studies on fish consumers around the Canadian Great Lakes which are subject to appreciable PCB contamination. (Thomann et al. 1987; Fein et al. 1984). Direct correlations have been observed between amounts of fish consumed and PCB blood and breast milk levels. Thomann et al. (1987) concluded that whilst no acute disease had been identified, "...the real concern is exposure to infants" and they found intrauterine exposure to be clearly associated with reduced size at birth and shorter pregnancy. The subtle infant behavioural differences which they also observed have largely unknown consequences for later development. However, poor performances at 7 months of age in visual recognition memory tests are thought likely to be reliably predictive for age groups at 4 and 7 years. A low IQ is reported for these youngsters (Fein et al., 1984).

Swain (1988) reviews the literature and notes that, in the highly exposed category (i.e. those which ate the most fish) maternal health effects included tendencies towards increases in anaemia, oedema and susceptibility to infectious disease. Infants born to mothers in more exposed categories were not only small but had

reduced head circumferences and their response to novel stimuli decreased in direct proportion to maternal exposure to contaminants in fish. That none of the effects seen in the infants could be attributed to 37 potential confounding variables (e.g. consumption by mothers of alcohol, or caffeine or their social status), strongly suggests that contaminants, in particular the PCBs, are implicated.

People in the higher fish consumption categories were consuming some 7-23 pounds of fish/year or more (Swain, 1988). 1 kg fish/year would be roughly equivalent to 1 fish/week. Concentrations of PCBs in Lake Michigan fish were in the region of 1-20 ppm (wet weight) (Thomann et al., 1987) and probably a little lower in other of the Great Lakes.

PCBs IN BREASTMILK

The transfer of PCBs from the maternal body fat via breastmilk to the suckling infant is attracting increasing attention. MAFF (1983) reported that levels up to 0.11 mg/kg in whole human milk were found, with a mean level of 0.018 mg/kg over the 102 samples analysed. Levels in milk fat were found to be 0.5 mg/kg. At the time this was lower than the 1.1mg/kg reported for milk fat from the USA. Recently a new dimension has been added to the argument.The possibility that PCBs, dioxins and furans in breast milk lead to vitamin K deficiency in breastfed infants has recently been postulated (Koppe and Olie, 1989). This leads to "late haemorrhagic disease" which is characterised by intracranial bleeding in babies. This seems to be a new disease which has only become apparent in the 1980's and which is limited, exclusively, to breastfed babies.

CONCLUSIONS

It may be concluded that much concerning the ecological and health effects of PCBs remains unknown, but sufficient evidence exists to warrant extreme caution in the handling of these compounds. It seems likely that high risks will be associated with production and disposal operations. UK production of PCBs was located in South Wales until 1977, and emissions still exist into the River Severn.
Historically, these compounds have been disposed of to landfill from where escape is likely. Incineration is another system used for disposal and South Wales has a PCB disposal operation of this kind at Pontypool. The incineration process has become subject in recent years to considerable scrutiny. Possible emissions of PCBs which have escaped thermal destruction, together with polychlorinated dibenzo-p-dioxins formed as combustion by-products are of increasing concern. This latter group of compounds appear toxicologically similar to the PCBs but exert their effects at much lower levels (in the part per trillion per kg body weight range). Surprisingly, very few data have been published in the open scientific literature concerning PCB and dioxin emissions from this facility although studies have been carried out of PCB levels in the soil of the surrounding area (Creaser & Fernades 1986; Eduljee et al. 1987) as part of a RECHEM research programme. Emissions of

dioxins have been reported by Mundy et al. (1988) presumably under conditions of stable operation. These figures would not necessarily accurately reflect emissions during periods of non-optimal operation. Moreover, this leaves a crucial gap in the published results: there are apparently no data for PCB emissions from the plant nor of ambient air levels in the vicinity. This may be of significance given the observation of Chevreuil et al. (1989) that 93% of atmospheric PCBs in their study were present in the vapour phase. In this case, it might be expected that equilibrium processes due to volatilisation might be active (Eduljee 1987) and that soil levels might not be an adequate reflector of ambient environmental levels. Given the potential adverse health effects of PCB emissions, this situation should be evaluated by independent means without delay.

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