SP
Aroclor

FEBRUARY [illegible], VOLUME [illegible] No. 1

Response to
Scott Tucker
This gives a good capsule
picture of the PCB problem
in Britain
REK

INFORMATION BULLETIN B.I.B.R.A.

CURRENT DEVELOPMENTS IN
FOOD ADDITIVE LEGISLATION
AND TECHNOLOGY

BRITISH INDUSTRIAL
BIOLOGICAL RESEARCH
ASSOCIATION

DSW 229575

STLCOPCB4053703

in deciding where priorities lie. It is surely no more important to study the possibility of poisoning by food additives than to keep a watch on fried potatoes.

THE PCB STORY UNFOLDS

It has only recently been recognized that polychlorinated biphenyls (PCBs), widely used as plasticizers, lubricants and fire retardants and as vapour suppressors to prolong the residual life of insecticides, are extremely stable compounds which resemble the organochlorine insecticides in their ability to persist in the environment. The similarity does not end there, for their retention times on the gas chromatograph are close to those of some structurally-related organochlorine pesticides such as DDT, and their unsuspected presence interfered in the past with analyses of DDT in biological material (BIBRA Bull. 1970, 9, 363). The realization of their persistence in the environment was reflected last October in an order from the US Department of Agriculture's Pesticides Regulation Division that the use of PCBs and polychlorinated terphenyls as active or inactive ingredients in pesticide formulations should be phased out within 6 months, since any benefits derived from this application were "outweighed by the resulting environmental contamination and the possible long range adverse effects on fish and wildlife" (Food Chemical News 1970, 12 (35), 20). A few weeks later the FDA asked its 17 District Offices to survey foodstuffs for contamination with PCBs, giving particular attention to liquid milk (ibid 1970, 12 (35), 5).

The FDA announcement noted that studies on the effects of PCBs on growth, mortality and reproduction were in progress and referred to indications that these chemicals might increase the toxicity of pesticides (BIBRA Bull. 1970, 9, 90). Comparatively little is known about PCB toxicity and a recent study has shown that its assessment is considerably confused by other compounds commonly present in commercial PCBs (ibid 1970, 9, 377; Vos et al. Fd Cosmet. Toxicol. 1970, 8, 625). Earlier reports have associated ingestion of PCBs with liver damage in rats and guinea-pigs and lesions resembling 'chick-oedema' in the chick and quail, but Vos et al. (loc.cit.), comparing the oral toxicities and analyses of three commercial samples, found that high mortality, subcutaneous and abdominal oedema and centrilobular liver necrosis were produced in chicks by only two of the samples, and hydropericardium, a common effect of these two samples, was only occasionally associated with the third. The analytical results and other toxicological evidence indicated that these toxic effects were in fact due not to the PCB itself, but to chlorinated dibenzofurans, principally the tetra- and pentachloro compounds, present as impurities in only two of the commercial PCB products.

Just how widespread are PCB residues in wildlife? At the Nature Conservancy's Monks Wood Experimental Station, wild birds and their eggs have for some years been analysed for PCB as well as pesticide residues (BIBRA Bull. 1970, 9, 470) and Prestt et al. (Envir. Pollut. 1970, 1, 3) have now presented the results obtained so far. Analyses have been completed for a total of 196 livers from 33 species and 363 eggs from 28 species, obtained between April 1966 and August 1968. PCBs have been detected in terrestrial species from most regions of Britain, in seabirds and all seabird eggs from one west coast and two east coast colonies, and in most of the freshwater species, which were collected principally from

STLCOPCB4053704

the Midlands and eastern and southern England. Liver residues were highest in freshwater fish-eating birds, levels of 300 and 900 ppm having been found in two herons from Derbyshire and Yorkshire, and in bird-eating raptors, in which levels ranged from 0-70 ppm. Birds that fed on mammals generally contained somewhat lower levels (0-50 ppm), while those on a mixed diet of carrion, birds and mammals and those feeding on insects had maximum levels of 15 and 1 ppm respectively. The same pattern was observed in the egg residue figures, the highest level detected being 80 ppm in a heron's egg. The eggs of this species also contained a maximum of 26.5 ppm p,p´-DDE and 6.5 ppm dieldrin, but there were more eggs with <1 ppm PCBs than with <1 ppm p,p´-DDE or dieldrin, suggesting that of the three, PCBs were generally present in the smallest amount. Residue levels of the three compounds in kestrel livers followed the same distribution, except that PCBs occurred at <2 ppm less frequently than did dieldrin.

Could the higher residues detected have caused the death of the birds concerned, without any contribution from other factors? Prestt et al. (loc.cit.) obtained an indication of the avian toxicity of PCB by feeding Aroclor 1254 to Bengalese finches at dose levels ranging from 26-1940 µg/day for 56 days. No dose level produced 100% mortality over this period, but the estimated dose causing 50% mortality was equivalent to 254 mg/kg/day, although one bird died on less than 12 mg/kg/day. In contrast, the level of p,p´-DDT required to cause 50% mortality over the same period was only about 19 mg/kg/day, but the mortality curve was much steeper than with PCB, suggesting that the latter may be the more toxic at low dose levels. Some birds dying from PCB poisoning displayed apparent leg paralysis or body and wing trembling, and on autopsy all had enlarged kidneys and two showed considerable amounts of fluid in the pericardial sac. The liver concentration of PCB increased with increasing dose levels, although great variation was observed at each level; at the dose causing 50% mortality the mean liver content was 345 ppm with a range of 39-3070 ppm, while one bird survived with a liver concentration of 634 ppm. Birds starved at the end of the feeding period had a significantly increased liver concentration of PCB. In normally fed birds, however, PCB was stored mainly in the depot fat, as was shown in one bird which was found to have accumulated 9% of the total dose by the end of the experiment.

The relationship between liver PCB concentration and death was so vague that no definite conclusions could be drawn as to the high levels found in certain predatory birds. A high ratio of brain-to-liver concentrations was much more closely correlated with mortality in the finch, but unfortunately brain levels were not measured in any of the wild birds studied. Assuming a similar sensitivity to the finch, roughly 3% of birds would have died with liver residues of 5-360 ppm, 34% with 22-1780 ppm and 50% with 39-3070 ppm. On this basis it was thought unlikely that PCBs could have caused widespread lethal toxicity in wild predatory birds in Britain, but no reference was made to the mass deaths of seabirds in the autumn of 1969 (BIBRA Bull. 1969, 8, 554; Bourne & Mead, B.T.O. News 1969, no.36). It was considered, however, that PCBs could be a component cause of the present breeding failure reported in several species.

Work by Lincer & Peakall (Nature, Lond. 1970, 228, 783) confirmed that PCBs affect hormone metabolism in birds at very low dose levels. Kestrels were fed for 5 months on dead cockerels which had been injected with 0.5 or 5.0 ppm Aroclor 1254 or Aroclor 1262 dissolved in sesame oil, the higher dose being roughly equivalent to a dose for the kestrel of 2 mg PCB/kg. When at the end of this period the microsomal fraction from the kestrel liver was incubated with oestradiol in vitro, a dose-dependent

STLCOPCB4053705

breakdown of the oestradiol to a more polar metabolite took place. There was no apparent difference between the effects of the two PCBs, and no such conversion occurred with the livers of control birds fed cockerels injected with sesame oil alone. In a further study on kestrels given 5 ppm Aroclor 1254, this increase in hepatic-enzyme activity was correlated with an increase in cytoplasmic RNA, suggesting that the action of PCB was similar to that of DDT and its metabolites.

PCB residues in seabirds appear commonplace (Prestt et al. loc.cit.) and high residues have been reported in birds from the Irish Sea (BIBRA Bull. 1969, 8, 554) and the Baltic (ibid 1970, 9, 120). The source of such residues in the marine environment has been something of a mystery, for which Holden (Nature, Lond. 1970, 228, 1220) now offers a probable solution. High PCB concentrations have been detected in water from the Firth of Clyde in recent years, and levels of up to 2.6 ppm have been found in clupeoid fish from that area. In the autumn of 1969 analysis of samples of surface and sub-surface water and of trunk-sewer effluents between the estuary mouth and Glasgow failed to reveal any local discharges of PCB. A close resemblance was found, however, between the gas-liquid chromatograms of extracts from crude sewage sludge, normally dumped in deep water 6 miles south of the estuary mouth at the rate of about 1 million wet tons/year, and those of some commercial PCBs. Estimated PCB concentrations were in the range <0.1-14 ppm (wet weight) or 1-185 ppm (dry weight). A simple calculation revealed that the mean PCB level of 1 ppm (found in 15 samples of wet sludge weighted according to the proportions dumped) would lead to an annual PCB discharge into the Clyde estuary of about 1 ton. Crude sludge from Manchester and London contained respectively about 2 and 0.2 ppm PCB, suggesting that about 1 ton of PCB isomers is similarly discharged into both the northern Irish Sea and the Thames estuary every year.

The PCBs in this sludge come from industrial waste which is normally combined with domestic sewage for treatment. When it is considered that some coastal towns discharge untreated wastes directly into the sea, while in inland areas treated sludge is commonly spread on the land, the ease with which PCBs can enter both the marine and terrestrial ecosystems becomes obvious.

That PCBs may also enter foodstuffs directly as a result of contamination from packaging is indicated by the findings of Bailey et al. (Chemy Ind. 1970, 30, 705). One sample of ground cashew nuts, analysed at the Pest Infestation Control Laboratory as part of a routine survey for pesticide residues in imported foodstuffs, was found to contain about 10 ppm of a PCB containing 40% chlorine. Unlike other samples from the same shipment which were free from PCB, this sample had been received in a lacquered cardboard drum, which on analysis proved to contain 200 ppm of the same PCB. Similar drums were used in the laboratory to store empty gelatine capsules intended for toxicological feeding, but fortunately no PCB could be detected in the capsules. PCBs were found, however, in most other samples of cardboard analysed, and the ease with which contamination from this source could occur is emphasized.

The gradual slope of the avian mortality curve for PCBs presented by Prestt et al. (loc.cit.), indicating that these compounds may cause deaths at low dose levels (albeit in only a small proportion of birds), and the ability of PCBs to induce microsomal enzymes at a dietary level of only 0.5 ppm (Lincer & Peakall, loc.cit.) suggests that even low residues of PCBs in the environment could present some threat to wildlife. Whether a

STLCOPCB4053706

ban like that imposed on certain organochlorine pesticides will be necessary is another matter. In the USA, Monsanto Chemical Corp., one of the major PCB manufacturers, is reported to have discontinued sale of PCB products for uses other than in 'closed systems' such as electrical transformers (Food Chemical News 1970, 12 (35), 6) and in the UK a similar move is afoot to introduce a voluntary ban on uncontrolled use of these compounds in products like paints and varnishes (Sunday Times, 3 January 1971). If this public-spirited move succeeds the need for legislative measures will be obviated.

AFLATOXIN—A HARDY ANNUAL

Reports on aflatoxin (AF) have continued to be published at a considerable rate since our last review (BIBRA Bull. 1970, 9, 273) but progress in our understanding of the health hazard presented by this toxin remains fairly slow. Many strains of the Aspergillus flavus group can produce AF, which has been shown to be, for some animals, one of the most potent carcinogens known. In a study of some 28 of these strains, Hesseltine et al. (Mycologia 1970, 62, 123) were unable to distinguish between them on grounds of gross morphology or biochemistry and found that the presence and ratios of the various types of AF appeared to be the best basis for classification.

One of the more notable of the recent contributions to the ever-growing AF literature is a topical survey of the problem given by Dr. J.M. Barnes as the Second Stenhouse-Williams Memorial Lecture to the Society for Applied Bacteriology (Barnes, J. appl. Bact. 1970, 33, 285). Dr. Barnes points out that there is little point in trying to analyse food eaten at any particular time in order to find out whether it contains AF because chance plays too great a part in determining the degree to which any particular harvests or batches of stored food may be contaminated. Information must be obtained at a much earlier stage, with adequate surveys of food harvesting and storage, so that the populations that may be most heavily exposed to food regularly or intermittently contaminated with fungi may be identified.

However, many reports on the levels of AF contamination in food products are still compiled and remain of some passing interest. Cheese has always seemed a likely candidate to play host to AF and, indeed, AF production by Aspergilli has been demonstrated in Cheddar cheese by Lie & Marth (J. Dairy Sci. 1967, 50, 1708). However, more recently, samples of Roquefort, Camembert and domestic blue cheese were all found to be free from AF (Shih & Marth, ibid 1969, 52, 1681). Similarly in a German study (Kiermeier & Groll, Z. Lebensmittelunters. u. -Forsch. 1970, 143, 81; idem, ibid 1970, 142, 120), no AF was found in 169 samples of 19 cheese varieties, but artificial infection with A. flavus resulted in large amounts of AF B_1 on Tilsit cheese under favourable environmental conditions, although on Camembert and Romadur the growth of the fungus was inhibited. Clearly the right conditions for AF contamination are present in some hot and humid cheese factories. A further German paper (Spicher, Brot Gebäck 1969, 23, 149) has shown that AF may occasionally be formed in some wholemeal and mixed breads. However, the state of deterioration of the bread was such that it was unlikely to have been eaten.

The potential formation of AF during the storage of meat in display

STLCOPCB4053707