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The Effects of Polychlorinated Biphenyls on Birds

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POLYCHLORINATED biphenyls (PCBs) are widely used industrial compounds which have been identified at trace levels in a variety of animal species. Due to their apparent ubiquity in the biosphere, PCBs have been under scrutiny to determine their influences on biological systems and their importance relative to food safety and to environmental health. Several instances of gross contamination of commercial poultry feeds by PCBs have focused attention on the toxicology of PCBs in birds.

A. Polychlorinated Biphenyls as Environmental Contaminants

Properties and uses of PCBs. In the United States, PCBs produced have been marketed under the trade name Aroclor ®.[1] They are also produced in France, Japan, West Germany, England, Spain and Italy. The world's largest producer of PCBs, the United States, produced an estimated 43,000 metric tons (4) of these materials in 1970. PCBs are synthesized commercially by the chlorination of biphenyl and the reaction products are fractionated according to specific gravity. Therefore, PCBs are commercially available as mixtures of isomers and homologues of chlorinated biphenyl catalogued according to average chlorine content.

The chemical and physical properties of PCBs suit them for many industrial uses but also make them persistent environmental contaminants. PCBs have low vapour pressures and high dielectric constants, with low solubilities in polar solvents [about 56 ppb in water] (42) and high solubilities in apolar solvents. They are generally chemically inert; they are resistant to acid and alkaline hydrolysis and to most oxidations with good thermal stability. PCBs have been widely used as plasticizers, electrical coolant-insulants and heat transfer agents. They have been introduced into the environment from several sources including vaporization from incinerated refuse, runoff from industrial wastes and dumps, and accidental leakage from heat-exchangers and other PCB-containing units.

Detection of PCBs. PCB residues in biological samples were first reported in 1966 (54) as being the previously unidentified interfering

[1]Monsanto Commercial Products Co., St. Louis, MO.

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peaks in gas-liquid chromatographic (GLC) organochlorine analyses (87). Since then, modified clean-up procedures have been used to separate PCBs from other chlorinated hydrocarbons prior to GLC analysis. Recently, combined gas-liquid chromatography-mass spectrometry (GLC–MS) has become the method of choice for PCB analysis. Only through mass spectrophotometry is it possible to unequivocally identify the various fractions obtained by GLC analysis. In biological materials the analysis of PCB residues is further complicated because known standard compounds have not been readily available. Most investigators have used commercial Aroclor mixtures as standards. The choice of standard is critical, as it can alter quantitative results by more than twofold if only GLC analysis is used (62).

PCBs in the global ecosystem. PCBs are widely distributed in the biosphere. Residues in the parts-per-billion [ppb] range have been detected in both fresh water and marine water throughout the world, apparently resulting from industrial and municipal waste discharges and from diffuse sources, including atmospheric particulates. Residue analyses indicate that animals accumulate PCBs in their tissues. PCBs have been reported in eels, mussels, marine turtles, porpoises, seals, fish and more than 40 avian species (3, 9, 15, 40, 41, 46, 55, 60, 88, 89, 95) in the wild. Fatty tissues such as adipose tissue and egg yolk have had greatest PCBs concentrations (89). Biological accumulation of PCBs has been demonstrated experimentally in several lower species (56, 71, 96), in fish (8, 34, 63, 107) and in the domestic chicken (91,92). Generally, PCBs found in the environment are the more highly chlorinated biphenyls [containing five or more chlorine atoms per biphenyl] (45, 60).

PCB residues in animal feeds. Because low levels of PCBs have been found in several species of fish including menhaden (34), an important source of fish meal for animal feeds, it is not surprising that detectable amounts of PCBs have been found in finished feeds for livestock and poultry. This source, however, did not explain the gross contamination of feeds discovered in the United States in the early 1970's. In 1972, the U.S. Food and Drug Administration (USFDA) analysed 1274 feed samples and found that 4·4% of these contained PCBs (57). This included 16 samples with as much as 578 ppm PCBs, from 515 broiler feeds analysed. One source of these PCB residues was found to be faulty cooling equipment in a North Carolina fish meal plant which produced approximately14,500 metric tons of contaminated [14–30 ppm PCBs] fish meal subsequently distributed to more than 60 feed companies (78).

The economic loss due to PCB contamination of poultry and eggs in the United States was substantial. In Maine, where PCB residues in feeds were very high, an estimated 1,400,000 broilers were destroyed in early 1972 because PCB residues in excess of 5 ppm were found in visceral fat (57). In August 1971, the USFDA seized 75,000 market eggs after 300 eggs were found to contain PCB residues (78). The

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subsequent drop in retail egg sales due to a "PCB scare" was estimated to represent a loss of 8–10 millions dollars to the U.S. egg industry (104).

Fish meals have not been the only sources of PCB contamination of animal feeds. Due to their lipophilic nature, PCBs are generally found in feed ingredients of higher fat content. Also, recycled animal by-products may contain biologically accumulated PCBs. Therefore, ingredients such as feed fats, meat scraps and poultry offal have occasionally been sources of PCBs in finished feeds. Some bakery by-products have also been sources of PCB contamination in the past. PCBs have been found infrequently in samples of ground corn and of corn ensilage, and in farm drinking water.

PCB residues in feed products. The PCBs content of the average American diet was estimated to be approximately 0·5 ppm in 1972 (61). This estimate was based upon results of a USFDA survey which detected PCBs in about 20% of 3500 samples of fish (54% positive), eggs (29% positive), milk (7% positive) and cheese (6% positive). Another USFDA survey of 2189 human adipose biopsy samples from 18 states and the District of Columbia reported that 54·3% contained PCBs, two-thirds of which had one part per million or more (105). The frequency of samples containing more than one ppm PCBs was twice as great for males as for females, and three times as great for Caucasians as for non-Caucasians. From these data one can project that approximately 20% of American males and 10% of American females have detectable PCB residues in their body fat.

In 1971 an *ad hoc* group within the USDA Office of Science and Education (5) was established to examine the PCB problem. It was followed in the same year by an interdepartmental task force on PCBs. One result of the reviews of these committees was the establishment of some PCB tolerances in feeds, foods and food packaging materials (6). These tolerances include 5·0 ppm for a single animal feed component; 0·5 ppm for finished animal feeds; 0·5 ppm for eggs; and 5·0 ppm for the body fat of poultry.

B. Effects of Polychlorinated Biphenyls on Birds

PCB toxicity. PCBs toxicity has been demonstrated in several avian species. Gross pathologies of chickens exposed to high levels of Aroclor ® 1242 in an unhardened epoxy-resin cage paint revealed hydropericardium; hydroperitoneum; enlarged heart, liver and kidney; haemorrhagic internal organs; and marked tubular dilation and numerous casts in kidneys (70). Flick *et al.* (37) noted the similarity of PCBs toxicity to the toxic effects of chick edema factor which also produces hydropericardium and death. It is possible that some toxic properties attributed to PCBs may be due to contamination by the very toxic dioxins (36) or dibenzofurans (30, 90) which have been detected in some commercial PCB mixtures.

Investigations by Rehfeld *et al.* (84–86) have characterized the PCBs toxicity syndrome in chicks: growth depression, decreased blood

haemoglobin levels and haematocrit, depletion of liver vitamin A storage. Vos and Koeman (102) reported porphyria and liver-necrosis in PCB-fed chicks. Briggs (16) showed a dose-dependent depression in spleen and bursa weights in chicks, suggesting immunological effects of PCBs. Iturri *et al.* (51) noted that while Aroclors 1242 and 1254 depressed heart rate [at 100 ppm] and haematocrit and haemoglobin levels [at 50 ppm], Aroclors 1221 and 1260 were reported to exert no toxic effects when fed to chickens at levels up to 150 ppm for nine weeks.

When breeder hens are exposed to PCBs in feed, egg hatchability is severely depressed at levels which do not decrease egg production (16, 19, 23, 24, 64, 65, 91, 92). Scott *et al.* (91, 92) found that hatchability in Single Comb White Leghorn eggs was inversely related to the PCB content of the egg and that 4·5–5·0 ppm was sufficient to completely prevent an embryo from hatching [Fig. 1]. This work has been confirmed (18, 19, 44, 93). The data of Britton and Huston (18) indicate that egg yolk PCB residues of 6–8 ppm are associated with zero hatchability in another commercial Leghorn strain. PCBs may increase embryonic mortality at all stages of development. However, a frequent sign of PCB-induced losses is an increase in late stage [18–21 days] deaths many of which die soon after pipping through the shell. This mortality peak is probably related to yolk absorption which occurs at that time, presenting the bulk of the PCB content of the egg to the embryo in a short period of time. PCB toxicity to chicken embryos is not solely a

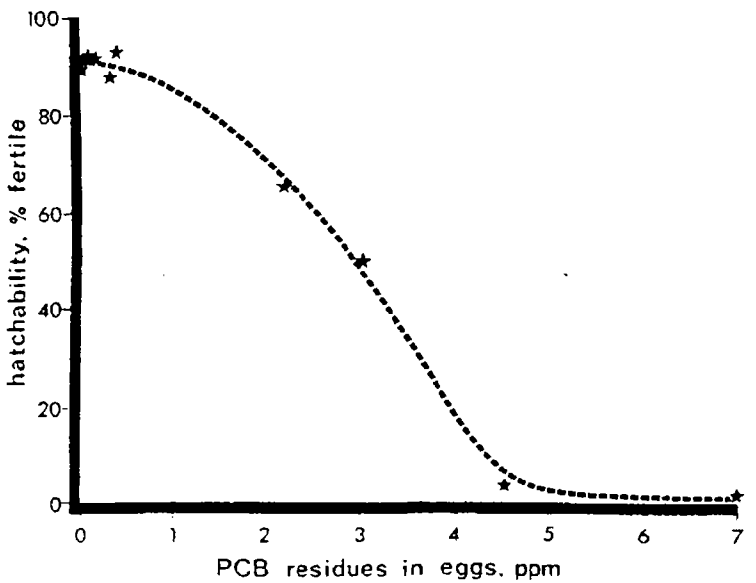

Fig. 1. Influence of PCB deposition in eggs on hatchability.

function of the degree of chlorination of the compounds. Several studies have shown that some more highly chlorinated PCBs are less toxic than less chlorinated analogues in chickens (7, 24, 51) and in Japanese quail (43). Recovery of hatchability occurs only after PCBs are withdrawn from the hen diets (18, 64, 65, 93); and the rate of recovery appears to be a function of the total body PCBs store of the hen. PCB depletion of hens cannot be markedly affected by dietary management. Scott *et al.* (91, 92) were unable to accelerate PCB depletion of hens by feeding a low energy diet designed to mobilize body fat.

Toxic effects of PCBs in other avian species resemble those in the chicken. Dahlgren and Linder (32) showed that Aroclor 1254 decreased hatchability and chick survival between hatching and six weeks of age in pheasants. Embryonic mortality as well as chromosomal alterations were reported in the adult ring dove by Peakall *et al.* (77). PCBs induced hepatic porphyria and a growth depression (103) as well as a 50% depletion of liver vitamin A stores (25) in Japanese quail.

A significant genetic component to PCB-sensitivity is indicated by the fact that ducks and turkeys are more resistant to PCB exposure than are chickens. Friend and Trainer (38) reported that although PCBs increased the lethality of Duck Hepatic Virus, dietary PCBs at a level of 100 ppm stimulated the growth of Mallard ducklings by as much as

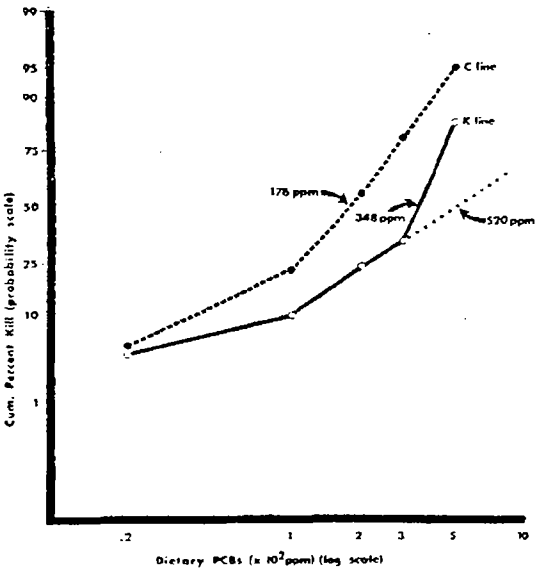

Fig. 2. Strain differences in PCB-sensitivity of Leghorn chicks. Arrows indicate oral LD_{50} (30 day) values; dotted line indicates LD_{50} value for K line chicks corrected for mortality due to inanition at the highest PCB level in that strain.

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20%. The oral LD_{50} for Mallards has been estimated to be greater than 2000 ppm (97). Holeman and Barnett (44) reported no signs of toxicity in turkey poults fed up to 150 ppm PCBs as contaminated fish meal for four weeks. Studies in this laboratory[2] have shown that dietary PCB (Aroclor 1254) levels as great as 500 ppm fed for five weeks depressed the growth of poults but resulted in no mortality. These results are in marked contrast to those obtained with growing chicks of two Cornell White Leghorn lines [Fig. 2]. The oral LD_{50} value for Aroclor® 1254 in 30-day experiments was 176 ppm for Cornell C line chicks. Cornell K line chicks showed an LD_{50} value [348 ppm] at least twice that of the C line when pair-fed to C line controls. However, dietary PCBs caused inanition at the greatest concentration which likely potentiated their lethal effects. Therefore, the true LD_{50} value for K line chicks was projected to be even greater [520 ppm] in the absence of a depression in feed consumption. These values are in the same range as the oral LD_{50} [56-day] value for Aroclor 1254 reported for the Bengalese finch [expressed as 253·6 mg/kg/day] (82).

The metabolic basis for interspecific and interstrain differences in PCB sensitivity may be related to the animal's capacity to degrade and to eliminate ingested PCBs. Studies in this laboratory [Table 1] showed that poults that were not exposed to PCBs had high activities of

TABLE 1: Induction of hepatic microsomal benzopyrene hydroxylase (BPH) in chicks, in young Japanese quail and in turkey poults by dietary PCBs

Source	Dietary PCBs,[1] ppm		
	0	20	100
	BPH specific activity,[1] units		
Chicks, C line	5·3	—	23·0
K line	5·0	—	21·6
Quail	4·3	32·6	—
Poults	21·8	55·2	—

[1]Aroclor 1254 fed in semipurified diets for two weeks.
[2]Specific activity: 1 enzyme unit=1 nmole benzopyrene hydroxylated per mg microsomal protein per 6 min. Assay procedure is described elsewhere (27).

a hepatic microsomal cytochrome P_{450}-dependent enzyme associated with drug metabolism, benzopyrene hydroxylase (BPH). Chicks and young quail, which are more sensitive to dietary PCBs, had lower BPH activities although activities were induced by PCB consumption.

Metabolism of PCBs. PCBs are metabolized in higher animals by microsomal enzymatic oxidations (39). Hutzinger *et al.* (49) found that both rats and pigeons can hydroxylate 4-chlorobiphenyl; the rat produced monohydroxy- and dihydroxy-products, but the pigeon produced only the monohydroxy derivatives of 4,4′-dichlorobiphenyl

[2]G. F. Combs, Jr. and M. L. Scott, unpublished data, 1974

and 2,2′,5,5′-tetrachlorobiphenyl; but no hydroxylated metabolites of 2,2′,4,4′,5,5′-hexachlorobiphenyl were produced by either species. In contrast to the rat, production of tetrachlorobiphenyl diols has been demonstrated in nonhuman primates (47), and the brook trout is apparently unable to produce hydroxylated chlorobiphenyls for excretion (49).

Bailey and Bunyan (10) found greater PCB residues in all tissues of pigeons and Japanese quail when they were fed Aroclor 1254 than when Aroclor 1242 was fed. This was interpreted as indicating an ability of those species to metabolize and to eliminate the PCB isomers of lower chlorine content, a conclusion confirmed by experimentation in rats (10, 14, 49). It has also been found that Aroclors 1254 and 1260 are relatively more toxic to chicken embryos than is Aroclor 1242 (23, 24), and that residues of the higher-chlorine containing isomers accumulate more rapidly in the brains of Japanese quail fed contaminated diets (20).

PCBs or their metabolites are excreted mainly as glucosides, glucuronides and free phenolic compounds in the rabbit (14). Rates of PCB excretion by animals are low. The rat has been reported to excrete a small amount [0·6%] of ingested PCBs via discharge from the small intestine without metabolic modification (106). Price *et al.* (83) found the total excretion of PCBs by preadolescent girls represented only 12% of their daily dietary intake. Platonow *et al.* (81) showed that PCB residues in milk accounted for only 3·8–6·2% of the daily intake of two Jersey cows. The egg is a major route of PCB excretion in the laying bird. Scott *et al.* (91) have shown that egg fat PCB concentrations are generally 6–7% of body fat PCB concentrations in Single Comb White Leghorn hens.

Enzyme induction and possible physiological consequences. PCBs are potent inducers of hepatic microsomal drug-metabolizing enzymes. In rats, the induction of ethylmorphine N-demethylase (2, 68, 69), aminopyrine N-demethylase (52, 68), aniline hydroxylase (52) and pentobarbital hydroxylase (68) has been demonstrated using assays in cell-free systems. Benzopyrene hydroxylase induction by PCBs was shown in rats (2), chicks (27) and young Japanese quail and turkey poults [see Table 1].

Induction of drug-metabolizing enzymes in the liver is associated with the synthesis of microsomal haemoproteins [cytochromes] which function as terminal oxidases in microsomal electron transport reactions. Inducers have been categorized into two groups according to the type of drug-metabolism induced and the effect on cytochrome P_{450} (29). Group I inducers [e.g., phenobarbital] increase the metabolism of a large variety of compounds and also increase the levels of cytochrome P_{450}, so named for its spectral characteristics. Group II inducers [e.g., 3-methylcholanthrene] increase the metabolism of polycyclic hydrocarbon carcinogens, such as 3,4-benzo-α-pyrene and 3-methylcholanthrene, and induce a cytochrome with slightly different spectral

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characteristics, cytochrome P_{448} [also called cytochrome P_{1-450}]. Alvares *et al.* (2) have shown in rats that Aroclor 1254 has properties of both classes of inducers: PCBs induced ethylmorphine N-demethylase [group I] and also induced benzopyrene hydroxylase ten-fold and cytochrome P_{448} three-fold [group II]. Several other reports have demonstrated induction of cytochrome P_{450} by PCBs (2, 52, 69, 99).

PCBs have been shown to affect the metabolism of steroid hormones. Bitman and Cecil (12) demonstrated the estrogenic activity of a series of Aroclors using the 18-hour glycogen response of the rat uterus. Aroclor 1254 has been shown to have anti-androgenic effects in causing severe depression of comb weights of 13-weeks old White Leghorn cockerels (80). Lincer and Peakall (66) found that *in vitro* hepatic microsomal degradation of estradiol increased in a dose-dependent manner with the PCB content of the diets of American kestrels from which the microsomes were prepared. It had earlier been reported that DDT and the cyclodiene chlorinated hydrocarbon insecticide, Dieldrin, induced pigeon hepatic microsomal degradation of testosterone and progesterone *in vitro* (73). Peakall (74), therefore, suggested that chlorinated hydrocarbons may alter steroid hormone levels in a manner which affects the calcium metabolism of the egg-laying bird. Anderson *et al.* (3) proposed that PCBs did this by affecting the vitamin D metabolism of the laying hen. Decreases in eggshell thickness of the pigeon and in the sparrow hawk between 1945 and 1950, and concomitant increases in the use of chlorinated hydrocarbon pesticides have been cited in support of these hypotheses (74).

Controlled studies have not supported these hypotheses. Although Peakall and Lincer (76) reported inhibition of *in vitro* active transport ^{45}Ca across the oviduct of kestrel hens by dietary PCBs, Turk and Heitman (98) demonstrated that Single Comb White Leghorn chicks fed PCBs had increased intestinal absorption of Ca^{++} and Zn^{++}. PCBs were reported to decrease shell thickness in Japanese quail (21), but Scott *et al.* (91, 92) demonstrated that PCBs in the diets of Single Comb White Leghorn hens and in Japanese quail hens did not affect the breaking strength of the eggs laid. Peakall (75) found no effect of PCBs on the shell thickness of ring dove eggs.

Effects on other enzymes. The effects of PCBs on few other enzyme systems have been studied. Vos *et al.* (103) demonstrated induction of a microsomal enzyme δ-aminolevulinic acid synthetase activity in hepatic mitochondria by oral doses of PCBs which caused porphyria in chicks and Japanese quail (102). Dietary PCBs have been shown to inhibit the respiratory components NADH-dehydrogenase and succinic dehydrogenase in rat liver mitochondria (72, 94), and a Na-K-APTase in fish (31, 33, 58, 59). PCB-induced hepatocellular injury in the catfish (22) was indicated by increased serum glutamic-oxalic transaminase activities. PCBs altered the composition of rat liver phospholipids (69). Holub *et al.* (46) showed that PCBs modified *in*

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vitro the activities of three lipid-synthesizing enzymes of the rat, but that this effect did not occur in the intact animal.

Nutritional consequences of PCBs. Recent investigations (27, 28) have demonstrated that PCBs in the diets of chickens can potentiate selenium deficiency. Experiments by Combs *et al.* (27) showed that Single Comb White Leghorn hens fed 10 ppm Aroclor 1254 produced chicks with increased requirements for dietary selenium for prevention of exudative diathesis when chicks were reared on a vitamin E-deficient diet. Although PCBs increased the apparent dietary requirements of the chick for both selenium and vitamin E (27), subsequent studies (28) demonstrated that PCBs specifically interfere with selenium metabolism. Dietary PCBs depressed the activity of the selenium-containing enzyme glutathione peroxidase in plasma [Fig. 3] and increased the dietary requirement for selenium for protection of hepatic microsomal phospholipids from oxidative degradation (28) [Fig. 4].

Predictive functions for estimating effects of dietary PCBs on breeding hens. The data of Scott *et al.* (91, 92) have been useful in developing predictive functions for the assessment of losses due to PCB-contamination of breeding hen diets. In order to assess such a loss, it is necessary to know (a) level of exposure, (b) duration of exposure and (c) level of egg production. If one conservatively assumes no metabolism of ingested PCBs, then the amount of PCB deposited in eggs produced by breeder hens will be largely a function of these three

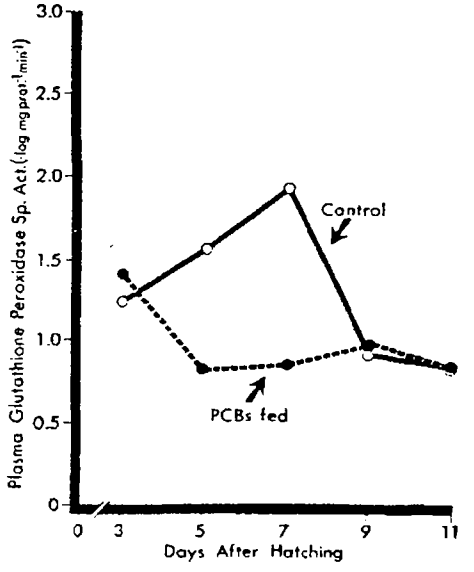

Fig. 3. Early depression of the seleno-enzyme glutathione peroxidase in plasma by PCBs (100 ppm Aroclor 1254) in chcks fed a subadequate level of selenium (0-04 ppm as Na_2SeO_3) [for details, see reference 27].

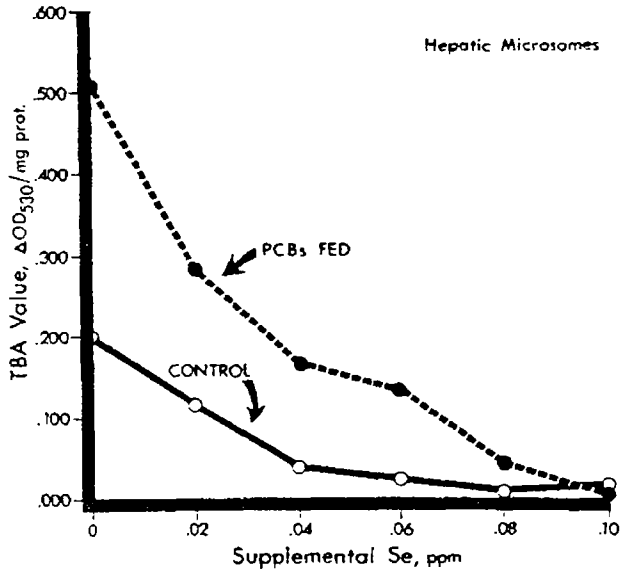

Fig. 4. PCB-induced increased requirement for dietary selenium for protection of membranes from peroxidative degradation in vitamin E-adequate chicks. [for details, see reference 28].

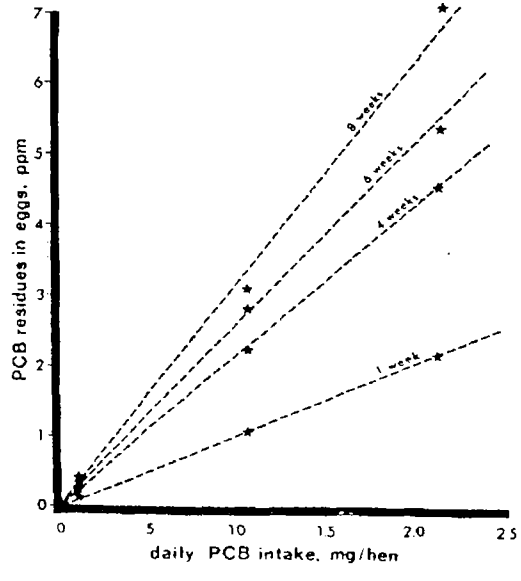

Fig. 5. Influence of daily PCB intake of SCWL hens on PCB deposition in eggs.

factors. At any particular level of egg production, egg PCB residues will be a function only of level and duration of exposure of the breeder hens.

The data of Scott *et al.* (91, 92) have been recalculated to express the relationship of level of exposure [mg Aroclor 1254/hen/day] and egg PCB residues [ppm whole egg, less shell and shell membranes] for four different periods of dietary exposure [Fig. 5]. Because egg hatchability is a function of egg PCB content [see Fig. 1], the effect of PCB exposure on hatchability may be calculated according to the duration of the exposure period. These predictive functions calculated from the data of Scott *et al.* (91, 92) are presented in Figure 6.

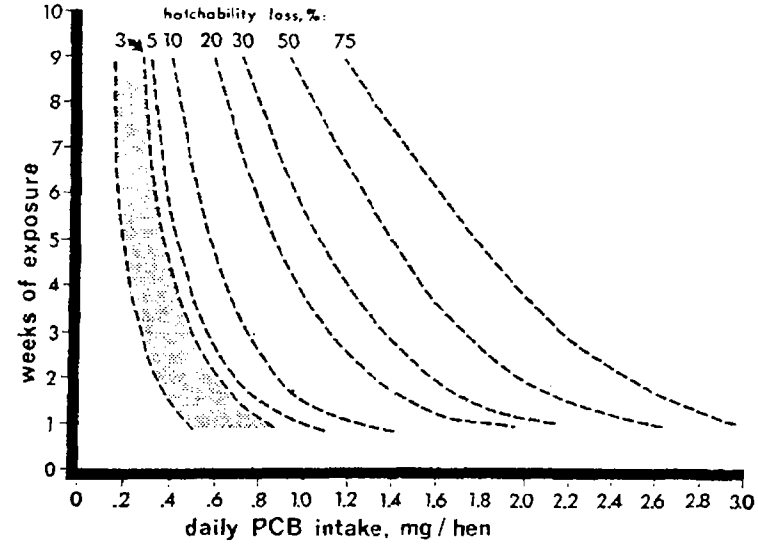

Fig. 6. Predictive functions for assessment of PCB-induced hatchability losses [Stippling indicates area of limit of detectability of hatchability losses.]

Because the Single Comb White Leghorn chicken has been shown to be one of the most PCB-sensitive species, the studies presented in this report may serve as a general guide for determination of maximum safe dietary levels. The results indicate that levels of PCB intake of 0·2 mg/hen/day have no detrimental effects in hens exposed for 12–16 weeks [and probably longer; see Figure 6]. This daily intake corresponds to a permissible level of 2 mg PCBs per kg of breeding diet when the consumption of this diet is approximately 100 g/hen/day. The results indicate that in meat-type breeding hens levels of PCB intake of 0·3 mg/hen/day are safe. Thus, the permissible dietary levels of 2 ppm are satisfactory when these hens consume breeding diet at the rate of approximately 150 g/hen/day. These results indicate in both types of hens that a safe PCB intake is approximately 0·1 mg/kg of body weight

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per day. Short-term exposure to PCBs [two weeks or less] is more likely to be encountered in commercial poultry operations due to feeding accidentally contaminated diets. The curves presented in Figure 6 show that short term levels of 6–9 ppm PCBs may be tolerated without significant losses in egg hatchability.

Thus, while long-term consumption of PCBs at dietary levels in excess of 2 ppm can be definitely considered as unsafe, the results of these studies point out that there is no basis for assigning any hazardous effects to the minute levels of PCBs [ppb] which have been detected in sea water, in drinking water and in various feed materials in many parts of the world.

Summary

Polychlorinated biphenyls [PCBs] are chlorinated hydrocarbons which have been used in a variety of industrial applications ranging from consumer plastics to heat exchangers. More than thirty years of the wide use of these persistent materials have resulted in an apparent ubiquitous distribution of PCBs in the global ecosystem at minute concentrations [parts-per-billion]. The recent development of analytical techniques with greater sensitivities and specificities, and the ensuing recognition of PCBs in the environment have produced a concern for the influences of PCBs on biological systems and for their importance relative to food safety and environmental health. Further problems occasioned by a number of accidental leaks of PCBs into animal feeds have focused attention on animal agriculture, especially commercial poultry production.

Controlled experiments with the chicken have demonstrated that high levels of PCBs can be toxic, causing growth depression, reduced efficiency of feed utilization and mortality with characteristic pathological signs. Studies have shown that the egg-laying chicken is most sensitive to PCBs, lower levels of which can interfere with hatching of the progeny of exposed breeder hens. PCB responses are dose-dependent; controlled experiments indicate that breeding hens can be exposed daily to as much as 0·1 mg per kg body weight without noticeable losses in hatchability. Thus, dietary levels less than 2 ppm are safe for breeder hens; much higher levels can be tolerated if exposure is limited. Experimental evidence shows that the minute concentrations [parts-per-billion] of PCBs detected in environmental materials in various parts of the world represent no threat to the reproductive fitness of birds.

Resume

L'EFFET DES BIPHENYLS POLYCHLORES SUR LES OISEAUX

Les biphényls polychlorés (PCBS) ont été employés dans toute une gamme d'applications industrielles allant des plastiques pour la consommation aux échangeurs de chaleur. La large utilisation de ces matériaux qui dure depuis plus de 30 ans a eu pour conséquence une ubiquité apparente de leur distribution à des concentrations réduites (parties par milliard) dans l'écosystème global. Le récent développement de techniques analytiques plus sensibles et plus spécifiques, et par suite la reconnaissance des PCBS dans l'environnement, a causé des inquiétudes quant à leur influence sur les systèmes biologiques et leur importance dans la sécurité d'emploi des aliments et le caractère sain ou non de l'environnement. Les problèmes causés par des introductions accidentelles de PCBS dans des produits alimentaires d'origine animale ont attiré l'attention sur l'élevage, et spécialement la production avicole commerciale.

Des expériences contrôlées avec le poulet ont démontré que des taux élevés de PCBS peuvent ralentir la croissance, réduire le rendement alimentaire et augmenter la mortalité, avec des signes pathologiques caractéristiques. La poule pondeuse est la plus sensible aux PCBS, dont les taux faibles peuvent interférer avec l'éclosion pour les reproductrices. Les réponses aux PCB dépendent de la dose; les poules reproductrices exposées journellement à des quantités de 0·1 mg par kg de poids vif ne manifestent aucune perte appréciable de taux d'éclosion. Par suite, des taux dans les rations inférieurs à 2 ppm ne sont pas dangereux pour elles: des taux plus élevés peuvent être tolérés pendant de courtes périodes. L'expérience montre que les faibles concentrations (parties par milliard) décelées dans des matériaux de l'environnement dans diverses parties du monde ne représentent pas une menace pour la capacité reproductive des oiseaux.

Zusammenfassung

DIE WIRKUNG VON POLYCHLORINIERTEN BIPHENYLEN AUF VOGELARTEN

Polychlorinierte Biphenyle (PCB) kommen in einer Vielzahl von Industrieprodukten zur Anwendung; von Plastikbehältern zu Wärmeaustauschern. Die inzwischen mehr als 30 Jahre dauernde Verwendung dieser dauerhaften Materialien hat offensichtlich dazu geführt, daß diese Stoffe in geringen Konzentrationen (ppb) überall im globalen Ecosystem vorkommen. Der durch neuentwickelte empfindlichere Methoden mögliche Nachweis von PCB in der Umwelt hat die Befürchtung geweckt, daß sie auf biologische Systeme Einfluß nehmen und für die Gesundheit von Nahrungsmitteln und Umwelt von Bedeutung sind. Probleme, die dadurch entstanden, daß in einigen Fällen durch das Zusammentreffen unglücklicher Umstände PCB in Futtermittel gelangte, haben die Aufmerksamkeit auf die Tierproduktion und speziell auf die kommerzielle Geflügelproduktion gelenkt.

Durch kontrollierte Versuche mit Hühnern wurde gezeigt, daß hohe Dosierungen von PCB das Wachstum behindern, die Futterverwertung nachteilig beeinflussen und zu einer erhöhten Sterblichkeit mit charakteristischen pathologischen Symptomen führen. Legegeflügel ist gegen PCB am empfindlichsten, wobei schon niedrige Gehalte die Schlupffähigkeit von Nachkommen behandelter Hennen beeinflussen können. PCB-Wirkungen sind abhängig von der Dosierung; Zuchthennen, die täglich bis zu 0·1 mg pro kg Körpergewicht erhielten, zeigten keine nennenswerten Einbußen an Schlupffähigkeit. Aus diesem Grunde sind Gehalte im Futter, die 2 ppm unterschreiten für Zuchthennen ungefährlich; höhere Gehalte können für kurze Perioden toleriert werden. Die vorliegenden Versuchsergebnisse zeigen, daß die geringen Konzentrationen (ppb) von PCB, die in Umweltmaterialien in verschiedenen Teilen der Welt festgestellt wurden, für die Vermehrungsfähigkeit der Vogelarten keine Bedrohung bedeuten.

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