Effects of Feeding Polychlorinated Biphenyls to Broiler Cockerels in Three Dietary Regimes[1]

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(Received for publication September 22, 1975)

ABSTRACT Polychlorinated biphenyls (Aroclor 1242 and 1254) were fed at 20 p.p.m. to hybrid broiler cockerels between one and nine weeks of age in three dietary variations. The Aroclors did not affect growth or feed efficiency and no consistent histopathological lesions could be attributed to them. There was a low incidence of hydropericardium in birds receiving Aroclor 1254. Fatty liver was observed in some birds receiving Aroclor 1242 and a single bird receiving this PCB exhibited liver necrosis. Moderate increases in liver weight and mild decreases in the relative weight of the spleen were attributable to PCB.

POULTRY SCI. 55: 1084–1091, 1976

INTRODUCTION

ONE of the early episodes signaling alarm about polychlorinated biphenyls (PCB) in the environment resulted from industrial contamination of 70,000 tons of fish meal with PCB from a heat exchange unit. Distribution of this contaminated fish meal to the nations largest poultry producer eventually resulted in the voluntary slaughter of 77,000 broilers with unsafe tissue levels of PCB's, as high as 40 p.p.m. (Pichirallo, 1971).

Previous investigations of PCB effects have focused on laying hens or pullets (Britton and Huston, 1973; Lillie *et al.*, 1974; Teske *et al.*, 1974) or on cockerels of a layer breed (Rehfeld *et al.*, 1971; Platonow and Funnell, 1972; Platonow *et al.*, 1973). The effects of PCB on avian reproduction are well documented, but the effects of moderate doses on adult chickens and Leghorn cockerels are minimal. At high doses decreased testicle, spleen and comb weights and enlarged liver, kidney and adrenal as well as hydropericardium have been observed (Flick *et al.*, 1965; Rehfeld *et al.*, 1971; Platonow and Funnell, 1972). Various histological changes have been described which could have been caused by toxic impurities (Vos, 1972).

In previous studies from this laboratory we have evaluated the effects of diets containing Aroclor 1242 (42% chlorine) and Aroclor 1254 (54% chlorine) on swine, sheep, channel catfish and laying chickens (Hansen *et al.*, 1975a, b, c; Ax and Hansen, 1975). In this companion study we report the effects of feeding diets containing 20 p.p.m. of these two Aroclors on hybrid broiler chickens for periods of 8 weeks.

MATERIALS AND METHODS

Day old hybrid broiler cockerels were obtained from a commercial source, divided into 9 groups of 40 and reared for four weeks in a Peter-Sime brooder unit. Chicks were then transferred to larger cages and reared for 8 additional weeks. Bi-weekly weight and feed consumption records were maintained. Diets were formulated from ingredients determined to be free from detectable organochlorine contamination as normal (23% protein, 4.5% fat), low in protein (17% protein, 4.9% fat), and high in fat (23% protein, 8.3% fat). Each diet was subdivided into control, 20 p.p.m. Aroclor 1242 and 20 p.p.m. Aroclor 1254. Chickens were fed each treated diet from 1 through 8 weeks of age and were then fed untreated diets from 9 to 12 weeks

1. Supported by Department of Health, Education and Welfare, Food and Drug Administration, Bureau of Veterinary Medicine, Research Contract No. FDA 72-116.

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STLCOPCB4015475

of age to examine elimination of residues. Every two weeks, three birds from each group were sacrificed. Organ weights and gross tissue changes were determined and recorded. Brain, spleen, kidney, liver, heart, intestine, lung, testes, proventriculus and gizzard were examined histopathologically at eight weeks.

RESULTS AND DISCUSSION

A summary of weight gains for the eight week period is presented in Table 1. Cockerels receiving the low protein diet had the lowest weight gains at all levels of treatment PCB, however, did not adversely affect weight gain, either within a dietary group or if all diets are considered collectively (Table 1). Feed consumption was not affected by diet or treatment; feed efficiency was slightly lower on the low protein diet, but the difference was not significant.

Relative organ weights were calculated for groups of 3 chickens at 3, 5 and 7 weeks; at 8 weeks, groups of 6 chickens were compared for each diet and treatment group (Table 2). There were no significant changes in relative kidney, adrenal gland, testicle,

TABLE 1.—*Performance of broiler cockerels receiving dietary variations and PCB for 8 weeks*

Diet / treatment	Mean weights (g./bird) ± S.D. Starting	8 Weeks	% weight gain
Normal			
Control	60.5 ± 11.7	1551.3 ± 267.5	2564
1242	60.8 ± 10.7	1741.7 ± 262.0	2867
1254	61.8 ± 8.4	1707.5 ± 99.4	2765
Low protein			
Control	65.7 ± 5.7	1620.0 ± 108.5	2366
1242	58.5 ± 10.3	1445.8 ± 130.8	2372
1254	65.9 ± 6.6	1690.8 ± 108.9	2467
High fat			
Control	61.6 ± 8.1	1903.4 ± 109.6	2990
1242	62.7 ± 8.7	1730.8 ± 126.2	2663
1254	63.4 ± 10.7	1929.2 ± 125.8	2945
All treatments			
Normal	61.0	1666.8	2732 ± 154
Low protein	63.4	1585.5	2402 ± 57
High fat	62.6	1854.5	2866 ± 177
All diets			
Control	62.6	1691.6	2640 ± 319
1242	60.7	1639.4	2634 ± 249
1254	63.7	1775.8	2726 ± 241

TABLE 2. *Relative organ weights of broiler cockerels receiving dietary variations and PCB for 9 weeks*

Diet / treatment	Mean relative organ weight (% body) ± S.D. Kidney	Adrenals	Testes	Heart	Brain
Normal					
Control	.805 ± .093	.012 ± .004	.028 ± .011	.578 ± .070	.167 ± .028
1242	.664 ± .028	.018 ± .018	.022 ± .006	.428 ± .044	.171 ± .029
1254	.617 ± .049	.011 ± .002	.025 ± .008	.435 ± .041	.174 ± .016
Low protein					
Control	.660 ± .243	.012 ± .002	.025 ± .007	.431 ± .051	.173 ± .023
1242	.660 ± .050	.013 ± .006	.022 ± .006	.434 ± .056	.210 ± .089
1254	.546 ± .139	.012 ± .003	.024 ± .006	.472 ± .053	.177 ± .011
High fat					
Control	.671 ± .052	.011 ± .002	.043 ± .016	.493 ± .081	.158 ± .011
1242	.679 ± .042	.010 ± .001	.020 ± .006	.398 ± .051	.177 ± .010
1254	.620 ± .077	.008 ± .002	.029 ± .013	.478 ± .043	.161 ± .013

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TABLE 3.—*Relative liver weights of broiler cockerels receiving dietary variations and PCB for 3 to 8 weeks*

Diet / treatment	Mean relative liver weight (% body) ± S.D.			
	3 Week	5 Week	7 Week	8 Week
Normal				
Control	2.97 ± .39	2.62 ± .51	2.33 ± .35	2.41 ± .27
1242	2.98 ± .16	2.78 ± .10	2.93 ± .21	2.47 ± .12
1254	3.18 ± .20	3.04 ± .62	3.37 ± .40	2.76 ± .33
Low protein				
Control	2.48 ± .05	3.63 ± .38	2.33 ± .35	1.74 ± .24
1242	2.88 ± .21	3.08 ± .21	2.43 ± .67	2.95 ± .39[a]
1254	3.12 ± .31	3.66 ± .27	3.17 ± .64[a]	2.51 ± .13[a]
High fat				
Control	2.83 ± .52	2.54 ± .25	2.27 ± .35	2.18 ± .35
1242	2.59 ± .29	3.23 ± .54	2.63 ± .12	2.78 ± .30[a]
1254	2.75 ± .25	3.32 ± .43	2.57 ± .32	2.49 ± .26
All diets				
Control	2.76 ± .39	2.98 ± .61	2.31 ± .31	2.09 ± .39
1242	2.82 ± .26	3.03 ± .35	2.67 ± .41	2.73 ± .31
1254	3.02 ± .30	3.34 ± .48	3.03 ± .55	2.60 ± .33[a]

[a]Significant at $p < .05$.
[b]Significant at $p < .001$.

heart or brain weights at 8 weeks. The adrenal glands tended to exhibit slight weight increase at 3 to 5 weeks and subsequent decrease or return to control values at 7 to 8 weeks; however, the individual weights were quite variable at given time periods and no significance could be established. Relative testicle weights increased during the 8 weeks and tended to be higher in control cockerels. Again, it appeared that the lack of synchrony in development masked any differences because of limited sampling. It was determined by microscopic examination that spermatogenesis had not been initiated in any groups.

The only consistent trend in organ weight changes was moderate increase in liver weight in the cockerels receiving PCB (Table 3). After eight weeks of the low protein diet,

TABLE 4.—*Relative spleen weights of broiler cockerels receiving dietary variations and PCB for 3 to 8 weeks*

Diet / treatment	Mean relative spleen weight (% body) ± S.D.			
	3 Week	5 Week	7 Week	8 Week
Normal				
Control	.100 ± .022	.152 ± .056	.195 ± .043	.178 ± .048
1242	.079 ± .003	.116 ± .020	.174 ± .015	.174 ± .033
1254	.072 ± .014	.088 ± .016	.169 ± .011	.180 ± .037
Low protein				
Control	.092 ± .010	.120 ± .030	.197 ± .050	.128 ± .046
1242	.088 ± .023	.149 ± .090	.166 ± .039	.171 ± .072
1254	.073 ± .006	.089 ± .036	.153 ± .030	.150 ± .027
High fat				
Control	.088 ± .041	.144 ± .034	.252 ± .082	.202 ± .024
1242	.107 ± .000	.133 ± .082	.177 ± .013	.179 ± .057
1254	.065 ± .019	.158 ± .029	.161 ± .020	.137 ± .041
All diets				
Control	.093 ± .024	.139 ± .039	.216 ± .061	.169 ± .049
1242	.089 ± .017	.133 ± .063	.172 ± .024	.158 ± .057
1254	.070 ± .013	.115 ± .044	.161 ± .019	.156 ± .038

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the mean relative liver weight of the control birds was considerably lower than all other control and treated groups; thus, the apparent hypertrophy in response to PCB was relatively magnified in this dietary group. Aroclor 1254 caused the greatest liver response, but at 8 weeks the low protein and high fat 1242 groups weighed slightly less than control and 1254 groups (Table 1), thereby increasing the relative liver weight.

The relative weight of the spleen tended to decrease in response to PCB up to 5 weeks and then increase in groups receiving normal and low protein diets. The response in the high fat group was delayed so that reduced spleen size was not apparent until weeks 7 and 8 (Table 4).

Three birds receiving Aroclor 1254 exhibited hydropericardium after 8 weeks and 3 to 5 ml. of fluid was aspirated from the pericardial sac. Gross observations were made on 6 birds per group, 3 being reserved for residue determination; thus, the incidence of hydropericardium was: low protein—1254, 17%; high fat—1254, 33%; other groups 0%. Extensive fatty liver was observed in 3/3 low protein—1242 cockerels at 5 weeks and 2/6 of the same group at 8 weeks. The weight gain of birds showing these gross changes was not affected.

Histological examinations of liver, kidney, heart, brain, spleen, lung, testes, proventriculus, gizzard, intestine and bone marrow were conducted for 3 birds from each treatment group at 8 weeks. All groups, including controls, exhibited changes associated with subacute lymphomatosis (Marek's disease). In addition to lymphocyte infiltration in nearly all tissues, liver necrosis was observed in a single cockerel receiving Aroclor 1242 in the high fat diet.

At 8 weeks, PCB concentrations in the fat of these cockerels averaged 50 p.p.m. Aroclor 1242 and 88 p.p.m. Aroclor 1254 (Hansen *et al.*, 1975d). The Aroclors had no effect on the performance of hybrid broilers when administered in the feed at 20 p.p.m. during the period of growth to market size. The incidence and severity of liver hypertrophy, fatty liver, hydropericardium, splenic atrophy and testicular atrophy were much lower than those previously reported in Leghorn cockerels, primarily because of the differences in dose. The generalized edema observed in layers at doses of 30–500 p.p.m. in the feed (Flick *et al.*, 1965; Rehfeld *et al.*, 1971; Platonow *et al.*, 1973), however, was not observed in the broilers and histopathological lesions were absent. Thus, concentrations many-fold higher than the tolerance limit could be accumulated with no adverse effects observed in the flock.

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Periodicity of *Leucocytozoon smithi* Gametocytes in the Peripheral Blood of Domestic Turkeys[1]

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(Received for publication September 22, 1975)

ABSTRACT Gametocytes of *Leucocytozoon smithi* exhibited a cyclic activity both in cardiac and peripheral blood of domestic turkeys during a given 24-hr. period. Peak parasitemias occurred from 0800 to 2000 hr., and mean low parasitemias from 2200 to 0400. The most prominent feeding period of the vector *Simulium slossonae* coincided with the period of elevated parasitemia in the turkey host.

POULTRY SCIENCE 55: 1088-1093, 1976

INTRODUCTION

NUMEROUS studies have indicated the presence of diurnal fluctuations of not only protozoan parasites but also parasitic worms in their vertebrate hosts. Periodicity of cell division has been demonstrated for several species of *Plasmodium* (Garnham, 1966) which divide synchronously by schizogony at approximately the same time of day every second, third or fourth day depending upon the species. Densities of trypanosomes in the peripheral circulation of rats infected with *Trypanosoma rhodesiense* and bullfrogs infected with *T. rotatorium* undergo cyclical variations (Wijers, 1959, Southworth *et al.*, 1968; Bardsley and Harmsen, 1969). Periodicity is also present among coccodia, the oocysts of *Isospora* appearing in the feces of house sparrows only in the afternoon and evening according to a regular cycle (Boughton, 1938).

Periodic cycles among parasitic worms, as evidenced by rhythmic migrations, are well known. Under specified experimental conditions the rat tapeworm *Hymenolepis diminuta* exhibits a longitudinal circadian migration in the small intestine of the host (Read and Kilejian, 1969; Chappell *et al.*, 1970). One of the most striking examples of rhythmic behavior is the periodicity of microfilariae in the peripheral blood of their vertebrate hosts. The larvae of many filarial worms are numerous in the peripheral blood during one

1. Technical contribution number 1253, Department of Entomology and Economic Zoology, South Carolina Agricultural Experiment Station, Clemson. Published by permission of the Director.

DSW 031517

STLCOPCB4015479