for a serum inhibitory substance in the altered phagocytic activity after surgery.

This work was supported by the American Cancer Society.

THOMAS M. SABA

Department of Physiology,
University of Illinois College of Medicine,
Chicago, Illinois 60680.

Received November 3, 1969; revised July 13, 1970.

1 El Rifai, K., Bacon, B., Mehigan, J., Hoppe, E., and Cole, W. H., *Arch. Surg.*, **91**, 625 (1965).
2 Lewis, M. E., and Cole, W. H., *Arch. Surg.*, **77**, 621 (1958).
3 Cole, W. H., McDonald, G. O., Roberts, S. S., and Southwick, H. W., in *Dissemination of Cancer* (Appleton-Century-Crofts, New York, 1961).
4 Saba, T. M., and Di Luzio, N. R., *Surgery*, **66**, 802 (1969).
5 Donovan, A. J., *Arch. Surg.*, **94**, 247 (1967).
6 Saba, T. M., and Di Luzio, N. R., *Amer. J. Physiol.*, **216**, 197 (1969).
7 Saba, T. M., and Di Luzio, N. R., *Proc. Soc. Exp. Biol. Med.*, **128**, 869 (1968).
8 Jenkin, C. R., and Rowley, D., *J. Exp. Med.*, **114**, 365 (1961).
9 Jenkin, C. R., and Rowley, D., *Nature*, **184**, 474 (1959).
10 Saba, T. M., Filkins, J. P., and Di Luzio, N. R., *J. Reticuloendothelial Soc.*, **5**, 398 (1968).
11 Di Luzio, N. R., and Rigal, S. J., *J. Reticuloendothelial Soc.*, **1**, 136 (1964).
12 Benacerraf, B., Biozzi, G., Halpern, B. N., and Stiffel, C., in *Physiopathology of the Reticuloendothelial System* (edit. by Halpern, B. N.), 52 (Blackwell, Oxford, 1957).
13 Filkins, J. P., and Di Luzio, N. R., *J. Reticuloendothelial Soc.*, **6**, 287 (1969).

Metabolic Effects of Polychlorinated Biphenyls in the American Kestrel

POLYCHLORINATED biphenyls (PCB) have been widely found in wildlife samples[1-3]. PCB are used as plasticizers in plastics, resin and chlorinated rubber[3]. They have been recommended for improving lindane residues[4] and have been shown to increase the insecticidal properties of DDT and dieldrin[5]. PCB are highly stable, insoluble in water and soluble in lipids; so that they can persist and undergo biological magnification in the environment.

Sax[6] reported the toxicity of chlorinated biphenyls as moderate to high. One PCB has been shown to produce chick hydropericardial oedema and growth depression proportional to dietary levels (200 and 400 p.p.m.) of the toxicant[7]. These effects were accompanied by such pathological changes as: cream-coloured pancreas, enlarged haemorrhagic kidney, enlarged adrenal gland, small spleen, dermatitis, defeathering and transient changes in blood glucose, haemoglobin and haematocrit levels. Intramuscular injection of PCB has been shown to increase the rate of steroid metabolism in bird liver homogenates[8] as has been shown for organochlorine pesticides[9]. Although a cause and effect relationship has not been demonstrated, PCB residues have been associated with decreasing populations of fish-eating birds and birds of prey[1].

American kestrels were trapped, allowed to acclimatize for a week, paired (one male and one female per cage), acclimatized for another week and then placed on *ad libitum* experimental diets. The kestrels' diet consisted of one day old dead cockerels. The dead cockerels were injected in the breast region at either 0·5 p.p.m. or 5·0 p.p.m. (w/w) of either 'Aroclor 1254' or 'Aroclor 1262' (Monsanto trade names for PCB containing 54 per cent chlorine and 62 per cent chlorine respectively) dissolved in sesame oil. The higher dose is roughly equivalent to 2 mg/kg PCB to each kestrel. Cockerels for control kestrels were injected with sesame oil only. It is assumed that the kestrels consumed essentially all of the dietary 'Aroclor', for the only parts of the cockerel not eaten were the beaks and feet, and the pellets regurgitated by the kestrels consisted of cockerel down. Each pair was kept in a burlap-covered 6×6×8 foot wire cage and fed experimental diet once a day for 5 months. At the end of the test period, the birds were killed, livers removed, and the microsomal fraction was prepared and incubated as described by Conney and Klutch[9]. Radioactive oestradiol and its metabolites were extracted with dichloromethane and separated by paper chromatography[10] with changes in procedure as reported previously[11,12].

Histological examination was carried out on kestrels fed 5 p.p.m. 'Aroclor 1254' for 14 days. A central portion of the liver was removed immediately after death and fixed in Carnoy's fluid for 45 min. Sections were cut at 5 μm. All sections were treated with deoxyribonuclease (0·1 mg/ml. in 0·1 M phosphate buffer (*p*H 7·5)–0·1 per cent gelatine–0·03 M $MgSO_4$) for 3 h at room temperature to remove DNA which would otherwise stain with azure B. All sections were then stained in azure B (0·1 per cent in 0·1 M citrate buffer, *p*H 4·0) for 2 h at 40° C, washed briefly with water and differentiated overnight with *t*-butyl alcohol[13]. The density of the staining with azure B was measured cytophotometrically at 545 nm (ref. 12). Ten readings of the per cent transmission of a standard area of cytoplasm were made for each of five control and five experimental livers. The mean for control birds was 39·7 ± 3·3 whereas for the experimental birds it was 26·8 ± 3·6. An analysis of variance of both the individual values and the means gave calculated *F* values which exceeded the 0·005 tabular *F*; we therefore conclude that the decrease in percentage transmission is a consequence of the presence of more cytoplasmic RNA in the 'Aroclor'-treated kestrels.

Table 1. *In vitro* MICROSOMAL BREAKDOWN OF OESTRADIOL
Amount of polar metabolites formed in nmoles/g microsomal fraction (sample size, 6 for each group)

Diet	Mean	s.d.
Control	0·00	0·00
0·5 p.p.m. 'Aroclor 1254'	13·66	7·35
5·0 p.p.m. 'Aroclor 1254'	60·50	21·20
0·5 p.p.m. 'Aroclor 1262'	19·05	11·50
5·0 p.p.m. 'Aroclor 1262'	47·96	25·45

A dose-dependent *in vitro* breakdown of oestradiol to a more polar metabolite occurred in livers from kestrels fed either 'Aroclor 1254' or 'Aroclor 1262' (Table 1). There was no apparent difference between the effects of the two 'Aroclors' nor was there any difference between sexes. No such conversion took place in the livers from the control birds.

The increase of hepatic enzyme activity can thus be correlated with an increase of cytoplasmic RNA. Proliferation of the endoplasmic reticulum of rat liver cells following the administration of chlordane[12] and DDT[14] has been noted although these changes were not quantified. Our findings suggest the physiological actions of PCB are similar to those of DDT and its metabolites. This industrial contaminant should be considered as an additional burden to the ecosystem comparable with that imposed by the organochlorine pesticides.

We thank Dr T. J. Cade for help in providing the kestrels and *Monsanto* for supplying 'Aroclors 1254 and 1262' and technical bulletins. This work was supported by the National Institutes of Health, and carried out while one of us (D. B. P.) was an established investigator, American Heart Association.

JEFFREY L. LINCER
DAVID B. PEAKALL

Langmuir Laboratory,
Section of Ecology and Systematics,
Cornell University,
Ithaca, New York 14850.

Received March 31; revised July 9, 1970.

1 Risebrough, R. W., Riecke, F., Peakall, D. B., Herman, S. G., and Kirven, M. N., *Nature*, **220**, 1098 (1968).
2 Jensen, S., Johnels, A. G., Olsson, M., and Otterlind, G., *Nature*, **224**, 247 (1969).
3 *Monsanto Tech. Bull.*, No. O/PL-311 A, 20.
4 Hornstein, I., and Sullivan, W. N., *J. Econ. Entomol.*, **46**, 937 (1953).
5 Lichtenstein, E. P., Schulz, K. R., Fahreman, T. W., and Liang, T. T., *J. Econ. Entomol.*, **63**, 761 (1969).