PCB NEWSLETTER

5 August 1974

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R. C. J. Sampson, Head, Organic Analysis Laboratory, Canada Centre for Inland Waters, Water Quality Laboratory & Network, Ontario Region, P. O. Box 5050, 867 Lakeshore Road, Burlington, Ontario, L7R 4A6

An informal survey was conducted randomly among some twenty-four agencies and/or laboratories analysing for PCB. Our objective was to assess the various interpretive approaches utilizing packed column electron capture chromatograms to determine which, in our view, best fulfilled the following generalized criteria - simplicity, speed, precision, accuracy.

Not surprisingly, the variability we encountered between methods was considerable. As a rigorous comparison of these proved impractical, the scheme used for contrasting their relative merits was necessarily a compromise. Our findings, based on mixtures of Aroclors led us to adopt the method of Webb and McCall. Subsequent work with fish extracts has substantiated the conclusion we drew from the above work. While the "true" PCB content of these was not known, the reduction in chromatogram ambiquity realized by the logic of this approach greatly simplified the task of quantitation.

Our laboratory is currently investigating various means of quantitatively removing PCB from other interfering electron capturing substances without altering the isomer ratios. At present, the most attractive route appears to be high speed liquid chromatography, but a considerable amount of development remains to be carried out if this technique is to be used routinely in our analyses.

Lastly, we are experimenting with the various approaches to perchlorination as a confirmatory test for PCB. We have had little difficulty with the method of Armour and plan to incorporate this into our analytical scheme. Our current concerns are minimizing interferences and ensuring that the PCB results reflect exclusively and quantitatively the PCB content of a given sample.

V. Zitko, Ph.D., Section Leader, Toxicology Section, Fisheries Research Board of Canada, Biological Station, St. Andrews, New Brunswick

Chlorobiphenylols are metabolites of chlorobiphenyls and as such are receiving increased attention. O. Hutzinger, S. Safe and V. Zitko prepared a number of chlorobiphenylols and chlorobiphenyldiols, and determined their GC properties, UV, Fluorescence, PMR, and mass spectra. The paper will be published in the September issue of J.Ass. Off. Anal. Chem.

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Some of the synthetic chlorobiphenylols were used to study the possibilities of their determination in biological samples. A mixture of isopropanol and hexane (1:5) was used for extraction. Chlorobiphenylols were then partitioned into 0.1M sodium hydroxide, after acidification back-extracted into hexane, acetylated, and determined by gas chromatography (V. Zitko, O. Hutzinger, and P.M.K. Choi, Bull. Environ. Contam. Toxicol. 12,No.6). The sensitivity of the determination is in the μg/g wet weight range.

Fish take up PCB (Aroclor 1254), but very little, if any, high-molecular-weight chlorinated paraffins from suspended solids (V. Zitko, Bull. Environ. Contam. Toxicol. 12, No. 5). In the case of PCB, components with shorter retention times are taken up relatively faster than those with longer retention times.

PCB and chlorinated hydrocarbon pesticides were determined in herring (Clupea harengus) and in double-crested cormorant (Phalacrocorax auritas) and herring gull (Larus argentatus) eggs. The analyses of herring were carried out in two, those of eggs in three, consecutive years. PCB levels in herring indicate a steady state or slight buildup, whereas the levels of DDT and metabolites are decreasing. In the case of eggs, PCB levels are either constant or decreasing and the levels of DDT and metabolites are decreasing. The variations between individual specimens are very high not only in the case of organochlorines, but also in weight and lipid (V. Zitko, International Conference on Transport of Persistent Chemicals in Aquatic Ecosystems, Ottawa, May 1-3, 1974).

This monitoring is a continuing program. The herring analyses are included in the OECD Wildlife Sampling and Analysis Program. Ms. Iola Price, Canadian Wildlife Service, Dept. of Environment, Ottawa, K1A OH3 is the program-coordinator for Canada, U.S.A., and Japan.

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Thesis Abstract

The Acute Toxicity of a Polychlorinated Biphenyl, Aroclor 1254, To The Early Life Stages of Coho Salmon And Steelhead Trout

Some acute effects of Aroclor 1254 (PCB) to the early life stages of coho salmon (Onchorhynchus kisutch (Walbaum)) and steelhead trout (Salmo gairdneri irideus Gibbons) were investigated using continous flow bioassay techniques.

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PCB did not influence the acute toxicity of DDT to 5-10 week old coho salmon of Michigan or Oregon parent stock. In 14-day bioassays, fish were exposed to 45.0 to 3.0 µg/l PCB or 3.3 to 0.2 µg/l PCB + DDT. PCB-DDT combinations produced no greater mortalities than could be expected from exposure to the DDT concentration alone. The rapidity of DDT toxicity with respect to that of PCB was suggested as the reason for lack of interaction. Michigan salmon were more resistant to DDT than Oregon salmon. Symptoms of PCB and DDT poisoning were described.

Coho salmon exposed to 56.40 to 4.35 µg/l Aroclor 1254 from the last two weeks of egg stage until four weeks beyond hatching were adversely affected at all exposure levels. Salmon exposed to the same PCB levels during the last two weeks of the egg stage only were adversely affected at levels above 15 µg/l. Egg hatchability, mean incubation time, fry survival, and fry growth were measured. All egg groups exposed to PCB hatched prematurely.

Two month old steelhead trout accumulated PCB at 0.189, 1.044, 1.832, and 3.106 µg/g per hour when exposed to 3.25, 10.45, 27.80, and 51.30 µg/l Aroclor 1254 for 24 days. Uptake rates could be predicted given one uptake rate from a known exposure level. The fish concentrated PCB 32,000 to 38,000 times over the exposure concentration but plateaus were not reached. Bioconcentration factors in fish at 100 hour time intervals through the test were found to be inversely related to the exposure concentration. Fish brains accumulated PCB at a slower rate than the whole body beyond 200 hours exposure or after accumulating about 400 µg/g PCB.

In a thirty-day toxicity test the median survival times of 100 day old steelhead trout exposed to 39.40 and 20.40 µg/l Aroclor 1254 were 293 and 400 hours, respectively. The growth of fish exposed to more than 4.70 µg/l PCB was reduced.

Joan Budd, D.V.M., Fish Pathology Laboratory, University of Guelph, Guelph, Ontario, Canada N1G 2W1

Oral exposure of rainbow trout (Salmo gairdneri Richardson) to a chlorinated biphenyl (Aroclor 1254): Pathological effects

by H. Nestel and Joan Budd

(Paper in preparation)

Rainbow trout were fed Aroclor 1254 daily at the rate of 0, 1, 10, and 100 ppm for 330 days. Foci of tubular degeneration (nephrosis) with cellular or granular casts were seen in the kidneys, especially in fish on the 10 ppm PCB diet, after 258 days. Fish with nephrosis had reduced splenic activity and lowered white cell counts.



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Toxicity, Pharmacokinetics and Residues

Moderate levels of PCB have been administered to food-producing animals in an attempt to assess subclinical effects and residue accumulation and depletion patterns. Analytical procedures have been developed to enable quantitation of individual components (1). Swine and sheep, administered an iv dose of 3 mg/Kg Aroclor 1254, exhibited different elimination rates for each component (2).

Similar oral studies have demonstrated the same relative order of persistance, and one component of Aroclor 1254 is consistently eliminated very rapidly; this peak corresponds with the pentachlorobiphenyl previously shown to be eliminated in rats (3).

In swine and sheep subacute toxicity studies (20 ppm Aroclors for 91-115 days), blood levels of certain components declined during the later weeks of dosing. Many PCB components have been shown to be reasonably good substrates for microsomal mixed-function oxidases, and significant induction of these oxidases was demonstrated in these animals (4).

Twenty ppm Aroclor 1242 in feed has been shown to adversely affect reproduction efficiency in swine (5) and the same level of Aroclors 1242 and 1254 affected performance and/or feed efficiency in swine, sheep and broiler chickens.

Three purified ^{14}C-PCB analogs have shown different qualitative and quantitative metabolism patterns in liver microsomes and a model ecosystem. Pharmacokinetic studies and limited toxicity studies are currently under way with other purified PCB analogs.

1) Welborn, et al. Extraction and analysis of Aroclor 1254 components in blood. J.A.O.A.C., in press.

2) Borchard, et al. Pharmacokinetics of Aroclor 1254 components after intravenous administration to swine and sheep. Arch. Environ. Contam. Toxicol., 2, 179 (1974).

3) Yip and Link, PCB Metabolism, PCB Newsletter, March, p. 20 (1972).

4) Wilson and Hansen. Changes in kinetic characteristics of sheep liver microsomes following induction and dietary stress. Toxicol. Appl. Pharmacol., in press.

5) Hansen, et al. Effect of a polychlorinated biphenyl on swine reproduction and resulting tissue residues. J. Vet. Res., in press.

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Transfer of a polychlorinated biphenyl mixture (Clophen A 60) into the egg

Material and methods

Laying hens (Shaver Starcross), 30 weeks of age, were housed as pairs in cages, and divided into three test groups of ten animals and a control group of five animals. In a preliminary experiment the hens were fed on PCB-free diet for five days. In the main experiment the test animals were maintained 20 days on a dietary dose of 17, 43, and 114 ppm of Clophen A 60. After that five hens of each group were killed and the remaining animals were fed on PCB-free diet for a 35-day recovery period.

Careful records were kept of feed consumption, body weight, and egg production. All eggs laid were broken, the yolk and albumen separated, and equal quantities of the yolks from hens within a PCB-group were thoroughly mixed and samples were taken for PCB analysis. Samples of albumen have been analyzed for PCB's on the first day of the preliminary experiment, on day 4, 8 and 20 of feeding PCB diets, and on day 14 and 35 of the recovery period.

The PCB analysis was carried out on extracts of egg yolk and albumen using gas chromatography. The extraction was done with hot hexane, the clean-up procedure with chromic sulphuric acid and sodium suphate for neutralization.

The PCB content of the sample was determined with a gas chromatograph equipped with ^{63}Ni electron capture detector following separation on a 4 mm x 1.3 m glass column packed with 10% DC-200 on 100/120 Chromosorb W AW-DMCS. The height of ten major peaks was determined and quantitation was accomplished using Clophen A 60 as a standard.

The limit of detection was 0.2 ppm Clophen A 60 for a 1 g sample of yolk. The following PCB recoveries were obtained with Clophen A 60 fortified samples ($\bar{x} \pm s_x$):

yolk	$76.0 \pm 3.0\%$
albumen	$88.9 \pm 2.1\%$

Results

PCB's did not affect feed consumption, body weight and egg production. PCB's could not be detected in the albumen of the eggs throughout the whole experiment as well as in the yolk of eggs

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during the preliminary experiment. The PCB content of the yolk of eggs is shown in Table 1.

Table 1. Clophen A 60 content of the yolk of eggs during the PCB-feeding and the recovery period

Experimental period	t	Dietary PCB's (ppm)			
		0	17	43	114
PCB-feeding	1		0.17	0.13	0.27
	2		0.5	0.8	2.5
	3		2.9	4.9	11.2
	4	0,11	7.7	15.3	34.5
	5		9.3	30.7	64.8
	6		14.4	35.4	103.3
	7		18.0	52.3	130,6
	8	0,15	24.8	66.8	145,1
	9		25.5	65.3	153.1
	10		28.9	77.5	197.8
	11		28.2	77.4	160.5
	12		26.9	84.5	189.0
	13		26.9	69.7	224.6
	14		33.0	88.8	138.9
	15		36.1	92.3	228.9
	16		28.3	92.6	243.8
	17		38.7	94.7	216.3
	18		35.7	80.9	233.2
	19		41.1	97.8	251.5
	20	0.12	38.3	105.4	286.3
Recovery	1		32.3	87.2	221.7
	2		38.4	89.9	287.0
	3		41.1	94.4	261.2
	4		34.5	85.6	230.5
	5		28.3	56.4	200.9
	6		24.5	51.4	154.3
	7		17.4	46.5	162.9
	8		15.4	22.1	97.1
	9		11.7	34.4	113.3
	10		17.3	31.2	99.2
	11		11.3	31.0	92.9
	12		15.4	28.8	95.0
	13		16.8	28.3	87.7
	14	0.18	14.0	29.9	95.8
	15		15.0	30.0	91.2
	16		11.7	25.6	71.4
	17		14.3	28.4	88.9
	18		15.0	25.4	81.1
	19		15.0	27.5	87.6
	20		14.9	27.6	70.9

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Table 1 (Continued)

Experimental period	t	Dietary PCB's (ppm) 0	17	43	114
	21		12.3	23.9	77.0
	22		13.7	26.6	79.9
	23		14.1	25.0	76.6
	24		11.5	23.9	75.0
	25		12.2	21.5	80.2
	26		10.5	24.8	71.3
	27		13.9	22.5	67.5
Recovery	28		10.2	19.9	68.2
	29		10.9	21.6	74.4
	30		10.8	19.7	64.1
	31		9.4	20.0	68.4
	32		12.1	18.3	62.2
	33		8.7	21.8	64.6
	34		9.7	18.1	66.3
	35	0.14	9.0	17.6	64.1

t: day of experimental period

During the PCB-feeding period the yolk content of PCB's increased according to the following equation:

$$C = C_p(1-e^{-k(t-3)}) \quad \text{(ppm)}$$

with
C: yolk content of PCB's at the time $t \geq 3$ (ppm)
C_p: yolk content of PCB's for $t \rightarrow \infty$ (ppm)
k: velocity constant
t: day of feeding PCB's
and reached equilibrium concentrations which were 2.1 times the PCB concentration in the diet (R = 0.9999).

The following constants have been calculated:

Dietary PCB's (ppm)	C_p (ppm)	k
17.4	37.0	0.169
43.0	93.9	0.174
113.9	243.2	0.192

During the period of recovery the yolk content of PCB's decreased at the beginning with a half-life of 5 days (phase I) later on with a half-life of 31-38 days (phase II). The two phases can be described by the following equation:

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$$C = C_o e^{-k(t-a)} \quad \text{(ppm)}$$

with

a: day of the recovery period on which the calculation of phase I or II was started
C: yolk content of PCB's at the time $t \geq a$ (ppm)
C_o: yolk content of PCB's at the beginning of Phase I or II (ppm)
k: velocity constant
t: day of recovery period

The following constants have been calculated:

Phase	Dietary PCB's (ppm)	a	C_o	k	H	R
I	17.4	2	39.8	0.128	5.4	0.94
	43.0	2	91.5	0.136	5.1	0.94
	113.9	2	290.0	0.137	5.1	0.98
II	17.4	12	16.0	0.023	30.1	0.84
	43.0	9	32.7	0.022	31.5	0.95
	113.9	8	100.2	0.018	38.5	0.91

H: half-life (days)
R: correlation coefficient

The gas chromatographic peaks of yolk extracts showed partly differences compared with Clophen A 60 standard.

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We have begun to study the effects of acute and chronic exposure of toxic agents (particularly chlorinated polycyclic hydrocarbons, CPH) on cell structure and function in several cell types. The protozoan systems, Crithidia fasciculata, a flagellated kinetoplastid and the euglenoid, Euglena gracilis, of the Sarcomastigophora have served as model cell types. In addition, preliminary studies have begun in vitro on primary rat hepatocyte cultures and in vivo on AFRRI rats. Cell population growth, energy metabolism, protein and nucleic acid precursor transport and biosynthesis, and ultrastructure are being used initially as parameter for studying toxic agent-induced effects during time-course dose-response studies.

We are particularly interested in the CPH-membrane interaction as the possible site of a primary lesion leading to disease at low level acute and chronic exposures. The hypothesis that p,p'-DDT

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interacts physically rather than chemically with components of neural membranes (1,2) which by some mechanism disrupts ionic balance (3,4), possibly by the inhibition of ATPases (5,6), is of great interest. Accordingly, Spaulding, et al. (7) have proposed that ionic balance disruption and subsequent inhibition of transport processes may be responsible for cell population growth inhibition observed in vitro. As a consequence, many metabolic disturbances observed in vitro and in vivo may be due to indirect effects.

A particular problem common to all experiments involving lipid soluble chemicals, especially CPH compounds, in well defined but nonetheless artificial environment, is working with concentrations greater than the CPH solubility in aqueous solutions. The use of vehicle solvents, miscible in polar and non-polar solutions (e.g. ethanol, ethanol-glycerol, dimethylsulfoxide, acetone, etc.) is required. There are at least two immediate concerns related to this common problem. 1.) The effects of a solvent, which may potentiate CPH-induced effects relative to solvent free controls, must be adequately defined. Two control systems now seem mandatory for in vitro toxicity testing: solvent-free and solvent (minus CPH) controls. Preliminary studies concerning solvent and solvent-xenobiotic interaction should be performed. 2.) In our in vitro studies, we have consistently noted that there is an apparent critical concentration range above which observed effects decrease relative to those in the lower concentration range tested. This implies that the CPH partitions out, consequently lowering the effective dose because it does not migrate through the aqueous solution compartment to the membrane compartment. Routinely, we have used 0.5, 5, 50 and 500 ppm as initial concentrations in toxicity testing. With DDT and PCB in DMSO (0.1% v/v) the critical concentration is usually between 50 and 500 ppm. It is our opinion that pertinent facts concerning these points should be included in reports concerning in vitro toxicity testing. Indeed, such phenomena may also occur in in vivo toxicity tests.

Initial studies have used C. fasciculata as a model cell system which is sensitive to low doses of Aroclor® 1254 (ID_{50} =10.5 ppm/24h). This organism also showed decreasing toxicity (% cell population growth inhibition) with increasing % chlorination of the PCB. Uptake (acid soluble fraction) and incorporation (acid insoluble fraction) of [^{3}H] thymidine was inhibited after 6 h exposure to 5 and 50 ppm, while [^{3}H] uridine was still inhibited after 6 and 12 hr exposure.

[^{3}H] thymidine uptake and incorporation increased after 12 and 24 h exposure 2-3X that of control (DMSO 0.1% v/v) while [^{3}H] uridine increased only after 24 h exposure to 5 and 50 ppm. [^{3}H] L-leucine uptake and incorporation increased after 12 and 24 h exposure at these levels and was not inhibited after any exposure period. [^{14}C] carbon-dioxide evolution from [U-^{14}C] D-glucose was inhibited at 6 h and increased at 24 h, relative to control (DMSO 0.1% v/v). This was probably due to indirect effects and no dose response was observed.

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In summary, the sensitivity of C. fasciculata to PCB makes it a suitable model for PCB-cell interaction studies. The significance of the transient inhibition of thymidine and uridine uptake and transport during early exponential growth and subsequent dramatic increase at later intervals of the cell growth cycle is still unclear. The effects are not due to simple dilution of the PCB in relation to the increase in cell population growth, but possibly mean a loss of cell regulatory capacity for uptake and/or incorporation in the cellular attempts to accommodate to the initial transient inhibition.

We would be pleased to receive comments, advice and questions concerning our research efforts in this area.

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1) Matsumura, F. and R. D. O'Brien, J. Agri. Food Chem. 14 (1966) 36-39.

2) Matsumura, F. and R. D. O'Brien, ibid, 14 (1966) 39-42.

3) Narahashi, T. and H. G. Haas, J. Gen. Physiol. 51 (1968) 177-198.

4) Hille, B., ibid. 51 (1968) 199-219.

5) Yap, H. H. et al., Nature 233(1971) 61.

6) Desaiah, D. et al., Bioch. Pharmacol. 21 (1972) 857-865.

7) Spaulding, J. et al., ibid. 20 (1971) 3185-3196.

David L. DeFoe, U.S. EPA, National Water Quality Laboratory, 6201 Congdon Blvd, Duluth, MN 55804

Two fathead minnow (Pimephales promelas Rafinesque) chronic toxicity tests with Aroclors 1248 and 1260 were conducted and the manuscript is nearing completion.

Total mortality was observed when fathead larvae, less than eight hours old, were exposed for 30 days to 8.5 µg/l 1248 and 9.0 µg/l 1260. The calculated LC_{50} was 4.7 µg/l for 1248 and 3.3 µg/l for 1260. Some effects were noted on survival and growth at and above 3.0 µg/l of 1248 and 2.1 µg/l of 1260. Good reproduction and egg hatchability occurred at and below 3.0 µg/l of 1248 and 2.1 µg/l of 1260.

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Virginia M. Snarski and Frank A. Puglisi, U.S. EPA, National Water Quality Laboratory, 6201 Congdon Blvd., Duluth, MN 55804

Brook trout, Salvelinus fontinalis (Mitchill), were exposed in a continuous-flow bioassay to Aroclor® 1254 concentrations of 0.012 to 0.935 µg/l for 71 weeks. An injector-equipped proportional diluter (1) was used to deliver the five toxicant concentrations and the control to duplicate tanks. Acetone was used as a carrier for the PCBs. The high Aroclor exposure contained the highest concentration of acetone, 0.004 ml/l (approximately 4 ppm), and acetone was added to the controls at this level.

No adverse effects were observed on survival, growth, and reproduction at any PCB concentration tested. Survival and growth to 90 days of fry produced by the chronically exposed parents were also unaffected. Periodic sampling revealed no appreciable change in PCB tissue concentrations after the first samples taken after 14 weeks of exposure. Considerable biological variability was noted in PCB residue levels of fish exposed to a given water concentration. A linear regression analysis showed the PCB concentration in the tissue to be directly proportional to the PCB concentration in the water. Exposure to 0.012 to 0.935 µg/l resulted in PCB residues from less than detectable (0.2) to 2 µg/g in fillets and from 0.5 to 9 µg/g in "whole bodies" (minus one fillet above the lateral line and the gonads), based on the wet weight of the tissue. The difference in the PCB concentrations between fillet and "whole body" samples at a given water concentration was directly related to their fat content.

Throughout the experiment, PCB water concentrations in the exposure tanks were measured weekly by gas chromatographic techniques. Because our exposure levels were so low, it was necessary to extract large volumes of water to obtain sufficient PCB residue for the GC analysis. Polyurethane foam plugs were used to extract the PCBs from the water (2). Each day one liter of water was run through a glass column packed tightly with four "plugs" and each week the composited PCB residue was extracted from the foam plugs with aliquots of acetone and hexane. As the large volume of water required for the detection of the PCBs also increased the interferences in the analysis, the extract was cleaned up on a one gram Florisil column before injection into the GC.

Results of this experiment are being prepared for printing in the EPA Water Pollution Control Series.

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1) Mount, D. I., and W. A. Brungs. 1967. A simplified dosing apparatus for fish toxicity studies. Water Res., 1: 21-29.

2) Gesser, H. D., et al. 1971. The extraction and recovery of polychlorinated biphenyls (PCB) using porous polyurethane foam. Analytical Letters, 4 (12): 883-886.

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O. Hutzinger and S. Safe, Atlantic Regional Laboratory, National Research Council of Canada, Halifax, and V. Zitko, Environment Canada, Fisheries Service, St. Andrews, New Brunswick, Canada

Newly published book on PCBs: Chemistry of PCB's

The aim of the book is to provide workers involved in various phases of research and routine analysis a comprehensive survey of the chemistry of polychlorinated biphenyls. The bulk of the book will deal with four topics: chemistry, spectroscopic properties, metabolism and breakdown, and analytical procedures.

In the chapter on chemistry, synthesis of chlorobiphenyls including labeled materials will be discussed as will be reactions such as oxidations and a variety of substitution reactions.

Mass spectra, nuclear magnetic resonance spectra, infrared-, ultraviolet-, and luminescence spectra will be dealt with in the second main chapters: spectroscopic properties. Both theororetical aspects and analytical potential will be covered.

The chapter on metabolism and breakdown will deal with the behavior of chlorobiphenyls in a number of animal systems, micro-organisms and in the environment. Enzymatic as well as photo-chemical factors will be considered.

In the last main chapter, the background and practical workings of analytical methods will be discussed. Coverage will be given to data on levels of PCB in living organisms and the environment.

Available from: CRC PRESS, INC., 18901 Cranwood Parkway, Cleveland, Ohio 44128. Cat. No. 5040/305 (Each $35.00, add 50 cents for Canada, $1.00 outside Canada and U.S.A. Payment is U.S. Currency)

G. Fred Lee, Institute for Environmental Sciences, The University of Texas at Dallas, Box 688, Richardson, Texas 75080

Newly published paper:

Dube, D. J., Veith, G. D. and Lee, G. F., "Polychlorinated biphenyls in treatment plant effluents." Journal of Water Pollution Control Federation, 46, 966-972, 1974.

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P C B in GERMANY:

not in water - but in a computer

We just completed a one-year program concerning Clophen A-30 Bayer and water.

I. Common fields of interest were:
   1. Analytical procedures - extraction by polyurethane foam, clean-up by H_2SO_4 and Florisil/Al_2O_3, recovery during concentration steps, exhaustive chlorination to decachlorobiphenyl, combined TLC and GLC (packed columns) in samples of water, algae, fish, mud and paper.

II. Special investigations affected the possibilities of eliminating Clophen A-30 by water treatment processes like:
   1. Oxidation by $KMnO_4$, chlorine and ozone (no effect!)
   2. Flocculation by Al^{3+} and Fe^{3+} in combination with suspended materials in river water
   3. Absorption in polyurethane
   4. Adsorption by means of activated carbon powder
   5. Adsorption and desorption by organic matter (algae, bacteria, etc.) in slow sand filtration. Groundwater in a pilot plant was contaminated by di- and trichlorobiphenyls in a few days.

This research (I,II) is to be continued using 9 available PCB isomers (di- to pentachlorobiphenyls).

III. Storage of PCB-literature in a computer

Since 1970 we prepared PCB-literature for storage in terms of keywords in a computer (IBM 370/3145). A special thesaurus was established including keywords of the environment (water, soil, air, food, men, animals), analytical procedures, applications, metabolism, toxicology, isomers, etc.

Any scientist intending to test this system is asked to write and point out his literature request on PCB's. No costs when referring to this announcement!

To complete our "collection" of PCB-publications we ask everybody to send in copies of all new (since 1974), unpublished or unavailable (meetings, communications, etc.) PCB publications.

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