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METABOLISM OF PESTICIDES
UPDATE II

212

UNITED STATES DEPARTMENT OF THE INTERIOR
FISH AND WILDLIFE SERVICE

Special Scientific Report—Wildlife No. 212

DEPOSITORY

METABOLISM OF PESTICIDES UPDATE II

By Calvin M. Menzie

UNITED STATES DEPARTMENT OF THE INTERIOR
FISH AND WILDLIFE SERVICE

Special Scientific Report—Wildlife No. 212

Washington, D.C. • 1978

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Metabolism of Pesticides
Update II

by

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INTRODUCTION

This publication supplements the preceding Metabolism of Pesticides (1969) and Metabolism of Pesticides -- An Update (1974). Readers are also advised that, during the period from preparation to printing of this volume, a considerable additional literature on this subject matter has been published.

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ALAR [Succinic acid 2,2-dimethylhydrazide]

N-Methyl-^{14}C-labeled alar was applied to four soils under greenhouse conditions. The data indicated that microbial degradation was the major route of alar dissipation from soil. The half-life of alar war 3 to 4 days on all soils and the major degradation product was $^{14}CO_2$. In 14 days, about 84% of the label was recovered as $^{14}CO_2$ and most of the remainder of the ^{14}C was associated with the soil organic matter (Dannals et al., 1974).

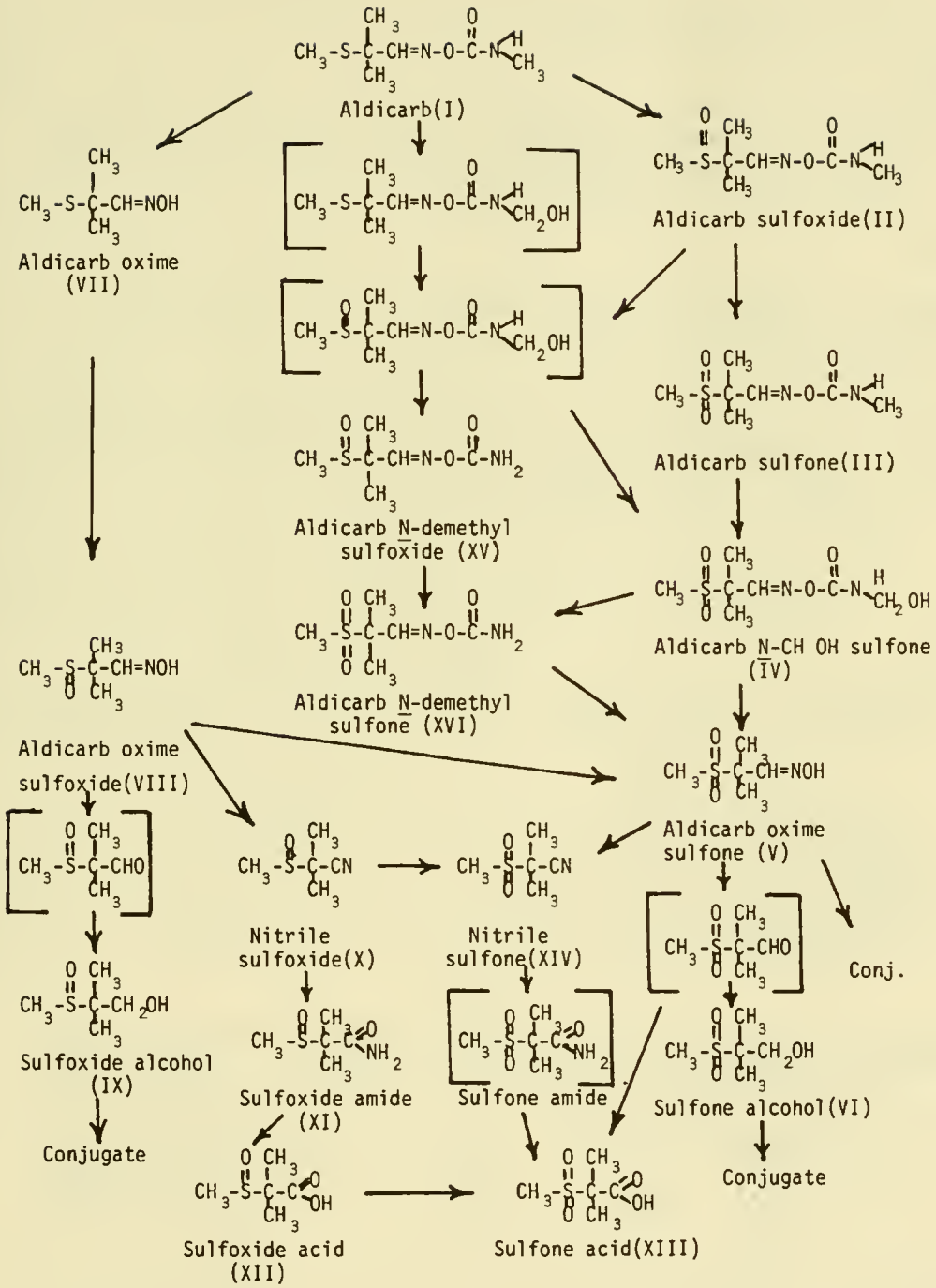
ALDICARB (Temik) [2-Methyl-2-(methylthio)propionaldehyde O-(methylcarbamoyl)oxime]

Five soil fungi were tested in culture media for their ability to degrade aldicarb. The results indicated the following order of effectiveness: Gliocladium catenulatum > Penicillium multicolor = Cunninghamella elegans > Rhizoctonia sp. > Trichoderma harzianum. Metabolites found included aldicarb sulfoxide and sulfone, nitrile sulfoxide and sulfone, and oxime sulfoxide and sulfone. A considerable amount of water-soluble metabolites were also observed, including the alcohol and amide sulfones and sulfoxides. Small amounts of the acid sulfone and sulfoxide were also observed (Jones, 1976).

In soil treated with aldicarb, the sulfoxide and sulfone were observed (Jamet et al., 1974).

After administration of a single dose (0.7 mg/kg) of Temik-S^{35} to hens, about 1% of the radioactivity was observed in eggs over a 10-day period. Analyses of tissues, eggs and feces showed the presence of the sulfoxide and sulfone metabolites (Hicks, 1970).

Boll weevils (Anthonomus grandis Boheman) and houseflies (Musca domestica L.) were treated with carbonyl-^{14}C-aldicarb. Following topical application of the insecticide, aldicarb disappeared rapidly. The sulfoxide was the major non-conjugated metabolite in boll weevils and houseflies. The sulfone formed subsequently in smaller amounts. $^{14}CO_2$ detected was very small. Water soluble products that formed were suspected of being conjugated metabolites but were not identified (Andrawes and Dorough, 1970).


Aldicarb(I)
Aldicarb oxime (VII)
Aldicarb sulfoxide(II)
Aldicarb sulfone(III)
Aldicarb N-demethyl sulfoxide (XV)
Aldicarb N-CH OH sulfone (IV)
Aldicarb N-demethyl sulfone (XVI)
Aldicarb oxime sulfoxide(VIII)
Aldicarb oxime sulfone (V)
Nitrile sulfoxide(X)
Nitrile sulfone(XIV)
Conj.
Sulfoxide alcohol (IX)
Sulfoxide amide (XI)
Sulfone amide
Sulfone alcohol(VI)
Conjugate
Conjugate
Sulfoxide acid (XII)
Sulfone acid(XIII)

ALDRIN, DIELDRIN, ISODRIN, and ENDRIN; HCE and HEOM

Aldrin

1,8,9,10,11,11-Hexachloro-2,3-7,6-endo-2,1-7,8-exo-tetracyclo [6.2.1.1^{3,6}.0^{2,7}]dodec-4,9-diene

Dieldrin

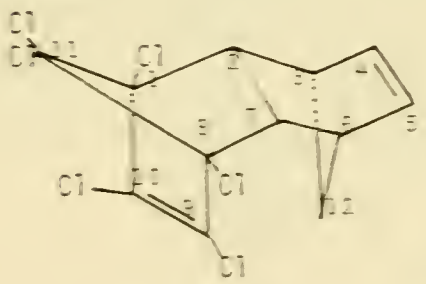
1,8,9,10,11,11-Hexachloro-4,5-exo-epoxy-2,3-7,6-endo-2,1-7,8-exo-tetracyclo[6.2.1.1^{3,6}.0^{2,7}]dodec-9-ene

Isodrin

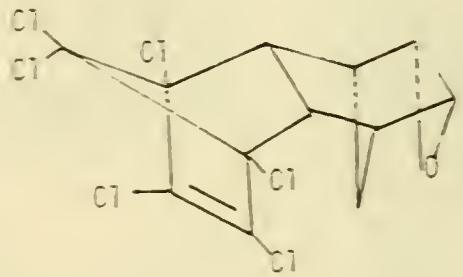
1,8,9,10,11,11-Hexachloro-2,3-7,6-endo-2,1-7,8-endo-tetracyclo [6.2.1.1^{3,6}.0^{2,7}]dodec-4,9-diene

Endrin

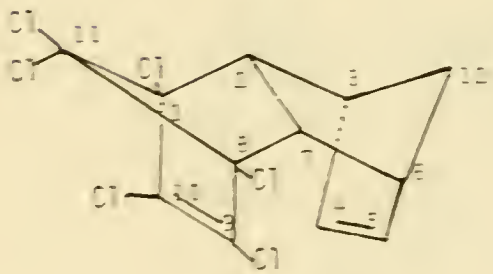
1,8,9,10,11,11-Hexachloro-4,5-exo-epoxy-2,3-7,6-endo-2,1-7,8-endo-tetracyclo[6.2.1.1^{3,6}.0^{2,7}]dodec-9-ene



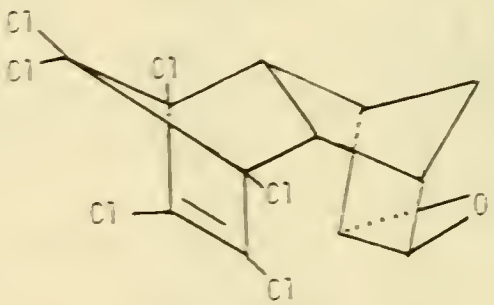
Aldrin



Dieldrin



Isodrin



Endrin

When labeled trans-4,5-aldrindiol was administered to rats, a small portion (6%) of the radioactivity was excreted as a polar metabolite which was identified as the hexachloro-tetrahydroindane-1,3-dicarboxylic acid (dihydrochlordene dicarboxylic acid) by TLC, GLC, IR and mass spectrometry (Oda and Muller, 1972). This compound was administered with labeling intravenously to rats. The radioactivity was rapidly excreted and almost half consisted of metabolites. Of the nine compounds isolated from feces and urine, three have been identified as: dihydrochlordene dicarboxylic acid dimethyl ester; and two isomers of the monodechlorinated dihydrochlordene dicarboxylic acid. Two compounds were very polar and, after hydrolysis with methanol/HCl, could be methylated to form the dimethyl ester of dihydrochlordene dicarboxylic acid. The other four compounds have molecular weights ranging from 244 to 336 and four to six chlorines (Lay et al., 1975).

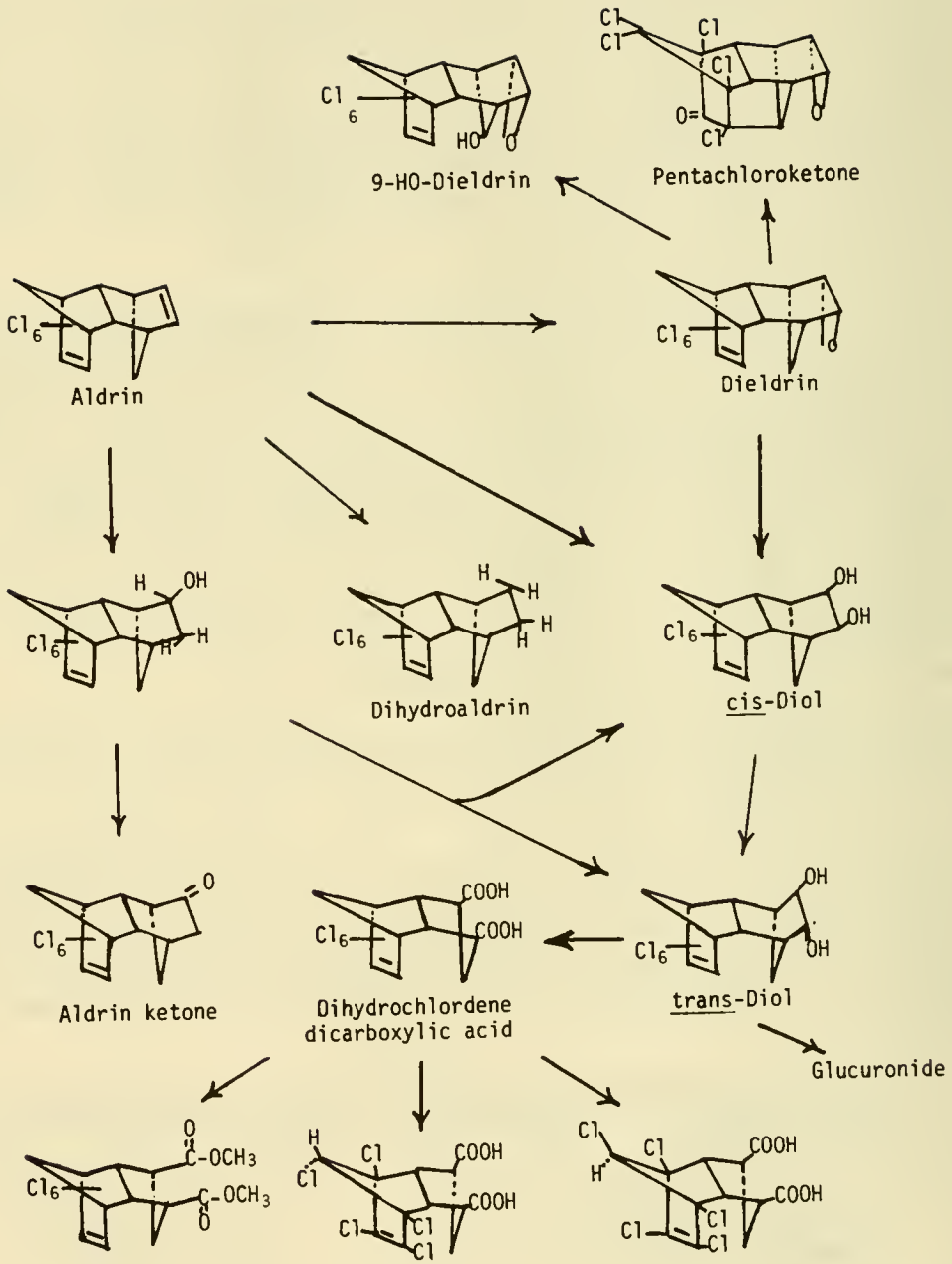
Dieldrin was administered intravenously to rhesus monkeys. Excreta was collected and four metabolites were isolated by TLC. Three were identified: 12-hydroxydieldrin; 4,5-aldrin-trans-dihydrodiol and a glucuronic acid conjugate of the diol (Muller et al., 1975b). The same metabolites were found in urine of Swiss white mice, Sprague-Dawley rats, New Zealand white rabbits, and a female chimpanzee when they were fed dieldrin (Muller et al., 1975a). In other studies, aldrin-trans-dihydrodiol and the di- acid were observed in treated rats and mice. The pentachloroketone was also excreted by rats but not the mice. Rhesus monkeys excreted 9-hydroxydieldrin but no pentachloroketone. Bile contained the glucuronide of 9-hydroxydieldrin (Baldwin, 1971).

Using microsomal preparations, the conversion of aldrin to dieldrin was found to be much slower in trout liver microsomes than in male rat liver microsomes (Chan et al., 1967).

Mixed function oxidase (MFO) activity in liver of bluegill (Lepomis macrochirus) converted aldrin to dieldrin in the presence of NADPH. Optimal activity occurred at pH 8.2-8.4 and 22-26C. Bass (Micropterus dolomeiux) MFO was linear with aldrin for 46 min at pH 7.4 and 25C. Mouse MFO also epoxidized aldrin to dieldrin (Stanton and Khan, 1973).

Hepatic mixed function oxidase with aldrin substrate

Epoxidation	V_{max}	K_m
Bluegill (adult)	0.62	8.58
Bluegull (fry)	1.72	6.98
Bass (fry)	1.72	5.97


9-HO-Dieldrin
Pentachloroketone
Aldrin
Dieldrin
Dihydroaldrin
cis-Diol
Aldrin ketone
Dihydrochlordene dicarboxylic acid
trans-Diol
Glucuronide

V_{max} Hepatix mixed-function oxidase

	Dieldrin	Photodieldrin	Endrin
Bass (fry)	1.19	-	
Bluegill (fry)	1.45	-	0.81
Mouse	3.35	2.15	1.17

(Stanton & Khan, 1973)

Susceptible and resistant mosquitofish (Gambusia affinis) were treated with ^{14}C-aldrin. Resistant fish converted aldrin to dieldrin and other unidentified materials at a greater rate than did susceptible fish (Wells et al., 1973).

When hepatopancreases from male lobsters (Homarus americanus) were incubated with aldrin, dieldrin was formed. The pH optimum was 8.0 and greatest activity was observed in the 105,000xg soluble fraction (Carlson, 1974).

The freshwater ostracod [Chlamydotheca arcuata (Sars.)] metabolized aldrin to dieldrin. No metabolism of dieldrin was observed (Kawatski, 1970).

Resistant and susceptible strains of the house fly (Hylemia antigua) metabolized aldrin to dieldrin at the same rate when aldrin was applied at the rate of 0.02 µg/fly. At 25 µg/fly, metabolism of aldrin by the resistant strain was very low (Temizer, 1970).

Radiolabeled dieldrin was observed in hemolymph of adult male Periplaneta americana and P. brunnea after application of ^{14}C-aldrin to the pronotum. There were indications that dieldrin was bould to a protein of about 18,900 MW and two groups of proteins of MW ≥ 160,000 (Olson, 1973).

Larvae of Heliothis zea and Heliothis virescens metabolized aldrin to dieldrin. Larvae of the latter exhibited a greater rate of conversion. Most of the labeled material used was excreted as metabolites more polar than dieldrin. Treatment with HCl indicated that these polar metabolites were primarily conjugated. None were identified. Within 24 h of treatment with labeled aldrin, H. zea excreted the radioactivity as aldrin (5%), dieldrin (12%), and polar metabolites (83%). With H. virescens, it was 3, 5 and 92%, respectively (Plapp, 1973).

Pea and bean root preparations degraded aldrin to a series of related polar metabolites (in addition to dieldrin, aldrin ketone, cis- and trans-aldrin diols, and exo-aldrin alcohol). Exo-aldrin

alcohol gave rise to traces of aldrin ketone. An unidentified major component was observed and thought to be 12-hydroxydieldrin from GC relative retention times.

An isomer of dieldrin, possibly the endo-epoxide isomer, was also observed (McKinney and Mehendale, 1973). The aldrin epoxidase enzyme from peas is particulate and not soluble. Almost all activity was located in the pellet from centrifugation at 250,000xg. Epoxidation of aldrin was not stimulated by addition of NADPH to high centrifugation fractions. Addition of Mg++ inhibited the reaction but the addition of 10^{-4} M p-aminobenzoic acid increased the activity of dwarf bean root homogenates. The results of the studies suggested the presence of two or more aldrin epoxidizing systems (Mehendale, 1973). In other studies cell-free pea root preparations metabolized aldrin to dieldrin. In 0.02 M phosphate buffer, the optimum pH was 6.5. The reaction increased up to 35C and decreased thereafter. In these studies, a need was not shown for the cofactors $NADPH_2$ and Mg++ (Oloffs, 1970).

In Japanese soils, on which organochlorine compounds has been sprayed for 2-20 years, photodieldrin was observed in 14 of 52 soil samples tested (Suzuki et al., 1974). Ten years after a single application of aldrin to soil, dieldrin and photodieldrin were detected (Lichtenstein et al., 1971).

Maize and wheat were grown in soils treated with aldrin-^{14}C at locations in Europe and the U.S. Aldrin-^{14}C treated wheat seeds were also planted. When the grains were harvested, residues in the grain did not exceed 0.01 ppm. The main labeled products identified in soils and plants from all locations were dieldrin, dihydrochlordene dicarboxylic acid, photodieldrin, aldrin and some unidentified acidic and non-polar compounds (Weisgerber et al., 1974). Sugar beets grown in soil treated with ^{14}C-aldrin, gave similar results. At harvest, dieldrin and a group of hydrophylic compounds comprised more than 95% of the ^{14}C-label recovered from soils. Besides dihydrochlordene dicarboxylic acid, photodieldrin and two minor acidic compounds not identified were observed (Kohli et al., 1973).

In England and Germany, aldrin-^{14}C was applied to soils outdoors and potatoes were sown. Traces of aldrin were found in potato hulm and peeled tubers. Small amounts of aldrin were detected in the peel. The main metabolite in potato samples and upper soil layers from Germany was dieldrin. In the potato hulm from England, but not from Germany, a compound was observed which behaved like photodieldrin. Small amounts of photoaldrin were also observed. In all samples, after aldrin and dieldrin, most of the radioactivity recovered was in the form of hydrophilic material. The main

compound was identified as dihydrochlordene-^{14}C-dicarboxylic acid (1,2,3,4,8,8-hexachloro-1,4,4a,6,7,7a-hexahydro-1,4-endo-methylene-indene-5,7-dicarboxylic acid). Formation of this acid apparently does not take place via dieldrin (Klein et al., 1973).

Vapor phase ultraviolet irradiation of aldrin gave rise to photo-aldrin, dieldrin and photodieldrin. Irradiation of dieldrin produced photodieldrin (Crosby and Moilanen, 1974).

Diquat inhibited microsomal aldrin epoxidation. $I_{50} = 6.6 \times 10^{-6}$M (Krieger et al., 1973). Fenton's reagent, modified by addition of bovine serum albumin, effected epoxidation of aldrin (Marshall, 1972).

Rat liver preparations transformed dieldrin in vitro to the cis- and trans-isomers of dihydroaldrindiol. An epimerase, also present in the same fraction of rat liver homogenate, rapidly epimerized the cis-isomer to the trans-isomer. The epimerase, located in the microsomes, required NADPH and cytochrome P-450 but not molecular oxygen (Matthews and McKinney, 1974).

A major animal metabolite of dieldrin was identified as the syn-9-hydroxy derivative. When photolyzed, it rearranged to form an isomer analogous to that of the photosiomer of HEOD. The syn-9-hydroxydieldrin was oxidized to 9-keto-dieldrin by refluxing for 1 h at 100C in a saturated solution of chromium trioxide in dry pyridine. Reduction of the ketone with sodium borohydride gave a product identified as 9-anti-hydroxydieldrin. Neither by photolysis nor after oral administration to a male CFE rat was the 9-anti-hydroxydieldrin converted to the pentachloroketone metabolite of dieldrin (Baldwin et al., 1973 and 1974).

Dieldrin half-life in lake water was 4.7 days. When freshwater mussels were exposed to the lake water, dieldrin concentration in the mussels increased 1200-fold (Bedford, 1971).

After injection of two female American cockroaches with dieldrin, extraction and analysis of an homogenate of the roaches indicated the presence of at least eight metabolites. The major metabolite was found to be cis-aldrindiol. Trans-aldrindiol, syn-hydroxy-dieldrin, 9-hydroxy analog of photodieldrin, 9-keto analog of photodieldrin and one unidentified compound were also observed. Metabolites from German cockroaches differed only quantitatively. Metabolites isolated from houseflies after exposure to dieldrin differed only in that cis-aldrindiol was not observed (Nelson and Matsumura, 1973).

In houseflies, dieldrin metabolism was very slow. Six metabolites were observed and one was characterized in five to seven chromatographic systems as trans-aldrindiol. Another resembled one metabolite from rats (Sellers, 1971).

Four weeks after application of ^{14}C-trans-aldrindiol to leaves of lettuce heads, the radioactivity was in the form of extractable and non-extractable residues. Less than 1% was in the soil. In addition to unchanged aldrindiol, analyses showed the presence of non-polar, methanol insoluble, and hydrophilic material. The latter consisted of at least 4 radioactive substances, the main product being about one-third of the mixture and identified by GLC/MS as dihydrochlordenedicarboxylic acid (Kilzer et al., 1974).

^{14}C-trans-aldrindiol was applied to soil. Seven weeks later, the soil analyses revealed the presence of 90.7% of the radioactivity as unchanged aldrindiol. In addition to non-polar material which could not be chromatographed, there was hydrophilic material. In this fraction, dihydrochlordenedicarboxylic acid was observed (Kilzer et al., 1974).

Aldrin was stable in demineralized water irradiated with UV at λ>300nm. When sensitizers (acetone and actaldehyde) were added, dieldrin formed (Ross and Crosby, 1975).

When ^{14}C-dieldrin-treated onion-seed was grown, residues found in skins, roots and soil consisted of photodieldrin, hydrophilic products and some non-extractable material (Kohli et al., 1972).

^{14}C-Dieldrin was applied to soil in which kohlrabi was grown. Leach water contained a compound identified by GC and mass spectrometry as dihydrochlordenedicarboxylic acid. Studies indicated that this compound was formed in the plants. Photodieldrin was also observed in soil and plant material. Similar results were obtained with carrots. Some radioactive material was not extractable (Kohli et al., 1973).

Studies on the effect of waste composting on dieldrin indicated little or no further degradation of dieldrin in a 3-week period (Muller and Korte, 1975).

Various species of marine algae differed significantly in ability to take up dieldrin. Two hours after treatment, the percent removal of dieldrin from the medium was:

Skeletonema costatum	42.0
Tetraselmis chuii	16.0
Isochrysis gallana (Chrysophyta)	15.5
Olisthodiscus luteus (Xanthophyta)	13.0
Cyclotella nana (Bacillariophyta)	13.0
Amphidinium carteri (Pyrrophyta)	2.3

(Rice and Sikka, 1973)

After 4-day exposure of Daphnia pulex or 30-day exposure of Ankistrodesmus spiralis to ^{14}C-dieldrin and ^{14}C-photodieldrin at 4 ppb, both compounds were recovered unchanged (Neudorf and Khan, 1975).

Little or no degradation of dieldrin occurred in skim milk containing E. coli, B. subtilis, P. fluorescens or S. aureus (Collins, 1969).

The epoxide ring of dieldrin is unusually stable and does not react with Grignard compounds or $LiAlH_4$ nor in molten KOH/KNO_3 at 230C.

No reaction occurred in alkali at elevated pressure. With acid catalysis, in methanol/benzene at 13K bar and 140C, the epoxide ring of dieldrin reacted to form a series of compounds (Roemer-Mahler, 1973).

In a model ecosystem, the accumulation, metabolism and degradation of dieldrin was studied. About 97% of the dieldrin was recovered unchanged. In addition to about seven unidentified metabolites, the 9-hydroxy and 9-keto derivatives were observed.

		Algae	Clam	Crab	Daphnia	Elodea	Mosquito	Fish	Snail
Dieldrin		+	+	+	+	+		+	+
9-keto				+				+	
9-hydroxy		+						+	+
Unknown	1					+			+
	2								+
	3								
	4								
	5	(In water only)							
	6								
	7								
Polar	1								
	2				+	+		+	+
Dieldrin C.F.		7480	1015	247	2145	1280		6145	114,935

C.F. = Concentration Factor

(Sanborn and Yu, 1973)

^{14}C-Photodieldrin was administered orally and ip to make rabbits. Most of the radioactivity in urine and feces was in the form of water soluble and/or conjugated material. Two of the urinary metabolites were identified as trans-dihydro photodieldrin and photodieldrin ketone (Reddy and Khan, 1975a) or trans-photoaldrin diol and photodieldrin ketone (Reddy and Khan, 1975c).

The in vitro metabolism of photodieldrin was studied with microsomal mixed function oxidase (MFO) of mouse, rat and houseflies. Photodieldrin was converted at very low levels to three metabolites in preparations from male and female mice; two metabolites in male rat and none in the female; one metabolite in female houseflies. Piperonyl butoxide blocked formation of these metabolites. None were identified

(Reddy and Khan, 1974). Photoaldrin was oxidized by mouse MFO to photodieldrin (Stanton and Khan. 1973). In other studies with CFE rats and beagles, there was evidence of some metabolism of photodieldrin to pentachloroketone (Baldwin, 1971).

Photodieldrin was applied to primary leaves of bush red kidney beans (Phaseolus vulgaris). Under laboratory light and sunlight, photodieldrin decreased by 17% during an 8-day period. No metabolites were detected. When freshwater algae (Ankistrodesmus spiralis) were exposed to dieldrin. no metabolites were detected. UV irradiation of photodieldrin on silica gel plates produced two metabolites not identified (Reddy and Khan, 1975b).

Soil was treated with labeled photodieldrin at the rate of 5 ppm on a dry-weight basis. Fifteen months later, the treated soil was removed and analyzed. Most of the radioactivity was not extractable with organic solvents. Three metabolites were isolated and identified:

1. Bridged isomer of dihydrochlordene dicarboxylic acid.
2. Bridged isomer of dihydrochlordene dicarboxylic acid, methoxylated.
3. Bridged isomer of aldrin-trans-diol.

The latter compound, because of the bridged skeleton and the asymmetry from the diol group, should exist in a total of four isomeric forms (Weisgerber et al., 1975).

Endrin in arachis oil was administered by stomach tube to six male and six female rats. In a 6-day period, males excreted in feces 66% of the dose whereas females eliminated only 37%. Excretion in urine was small but females excreted three times that of males. Feces from rats fed endrin was collected and analyzed. Analysis revealed the presence of endrin (11%), anti-12-hydroxyendrin (83%), syn-12-hydroxyendrin (<0.01%), 3-hydroxyendrin (5%), 12-ketoendrin (1%) and Δ-ketoendrin (<0.01%). A polar metabolite was identified as trans-4,5-dihydroisodrin-4,5-diol. Another polar metabolite was not identified. Collected urine from male rats contained endrin, 12-ketoendrin, anti-12-hydroxyendrin and 3-hydroxyendrin (17:19:2:1, respectively). The extracts from female rats did not contain the 12-ketoendrin. The major metabolite in female rat urine was identical to 12-hydroxyendrin O-sulfate by paper and thin-layer chromatography and paper electrophoresis (pH7). In tissues analyzed, 12-ketoendrin was the major compound in fat of males; endrin, in females, but 12-ketoendrin was also present. In liver of males no endrin was detected and the 12-ketoendrin was the major metabolite. Liver of females contained endrin and traces of 12-ketoendrin. Kidneys of males primarily contained 12-ketoendrin; those of females contained endrin. Much of the residue was not identified. Bile contained anti-12-hydroxyendrin, 12-

ketoendrin and 3-hydroxyendrin as glucuronides (Hutson et al., 1975). After exposure of rats to endrin, analyses have also shown the presence of 9-ketoendrin in tissues and urine and the presence of 5- and 9-hydroxyendrin in feces (Baldwin, 1971).

^{14}C-Labeled isodrin was applied to leaves of young white cabbage (Brassica oleracea var. capitata). After 4 weeks, only 2% was unchanged isodrin. After 10-weeks, there were only conversion products as residues. Endrin and Δ-keto-endrin were found (Klein et al., 1972).

In other studies when isodrin-^{14}C was applied to cabbage leaves, six metabolites were isolated and identified or characterized:

I (27%) Endrin
II (27%) Δ-keto-endrin
III (14%) A monohydroxy acid
IV (0.5%) A monohydroxy acid
V (1%) A dicarboxylic acid
VI (1.4%) A dicarboxylic acid

(Weisgerber et al., 1975)

After application of ^{14}C-isodrin to soil, carrots (Daucus carota ssp. sativus) grown in the soil contained 3.1% of the radioactivity after 4 weeks. The soil contained 48% unchanged isodrin after 4 weeks and 41% after 12 weeks. Endrin and Δ-keto-endrin were identified as conversion products of isodrin. Four water-soluble compounds were also found but not identified (Klein et al., 1972).

In vitro studies showed that MFO of bluegill and mouse converted isodrin to endrin (Stanton and Khan, 1973).

After exposure of third instar larvae of tobacco budworm (Heliothis virescens), ^{14}C-labeled endrin was more readily extracted from nerve tissue of the susceptible strains. Susceptible budworms degraded endrin somewhat to the aldehyde and ketone. The same metabolites plus two unidentified decomposition products were recovered from resistant strains (Polles, 1971).

Endrin in hexane was exposed to sunlight. The main photolysis product, identified as the ketone, was also obtained by UV irradiation of endrin in hexane (Fujita et al., 1969).

A dieldrin analog, HEOM, was incubated with enzyme preparations from pupae of blowfly (Calliphora erythrocephala), larvae of the southern armyworm (Prodenia eridania), and adult Madagascar cockroaches (Gromphadorhina portentosa). HEOM was hydrated by the three enzyme systems to the corresponding diol. Maximum activity was associated with the 100,000xg pellet (Slade et al., 1975).

Homogenates and microsomal preparations from livers of rabbits, rats, quail and pigeons were used to study the transformation of HCE, a dieldrin analog. The results are summarized in the following tables.

		% HCE Converted To Diol	% HCE Oxidized	% HCE Metabolized	PERCENTAGE OF TOTAL METABOLITES Trans Diol	HHC	Dihydroxy HCE	U_1	U_2	U_3
Rabbit	M			73	29	33	10	20		4
	S	20	75	95		37	12b	18		3b
Rat	M			27	5	95	a			
	S	13	39	48		55	b			b
Quail	M			17	8	92	a			
	S	2	31	67		67	b			b
Pigeon	M			27	0	90	4		6	
	S	0	95	95		42	34b		11	

a = formed from HCE where more than 70% of substrate had been converted.
b = formed from HHC under oxidative conditions.
M = liver microsomal preparation.
S = 11,000xg supernatant from liver homogenate

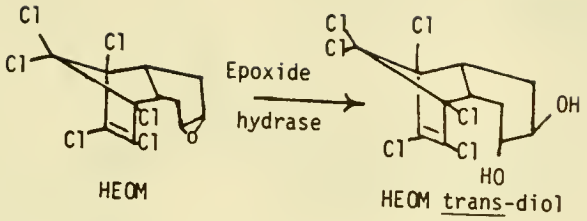
After rabbits were dosed with labeled HCE, only 35-40% of the radioactivity was extractable with ether. After refluxing with acid, most of the remainder became ether extractable. HHC and an unidentified polar compound were released by the acid hydrolysis.

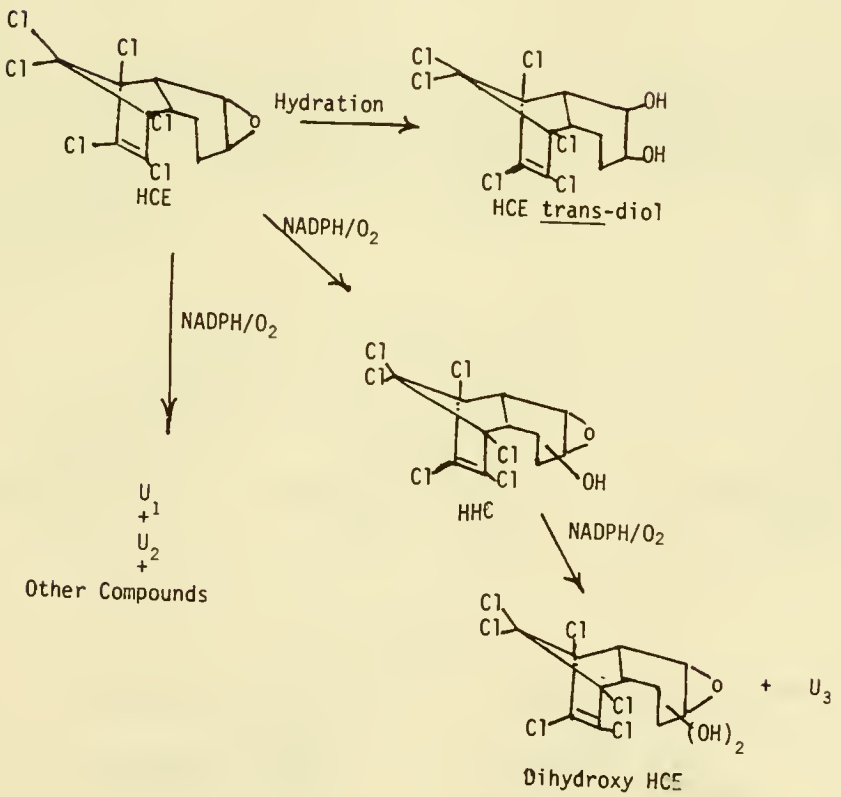
PERCENT OF DOSE

	HHC	HCE Diol	Dihydroxy HCE	U_1	U_3	Others
Rabbit	42.0	2.2	6.3	5.0	1.0	36.0
Rat	29.0	1.9		0.0	0.0	6.7
Pigeon	5.7					3.0
Quail	20.0	<2.0	6.0	0.0	0.0	21.0

(Walker and El Zorgani, 1973 and 1974)

The shag (Phalacrocorax aristotelis) has been found to contain rather high dieldrin levels. A study of shag enzymes indicated that the epoxide hydrase and the hydroxylating capacity of liver preparations was substantially lower in the shag than in the rat. Liver microsomes of the shag exhibited <8% of epoxide hydrase and <14% of the hydroxylating activity of a similar preparation from a rat (Walker et al., 1975).


Epoxide
hydrase
HEOM
HEOM trans-diol

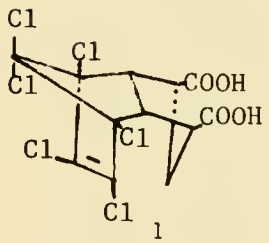

Hydration
HCE
HCE trans-diol
NADPH/O2
NADPH/O2
U1
+
U2
+
Other Compounds
HHC
NADPH/O2
+ U3
Dihydroxy HCE

Dihydrochlordene dicarboxylic acid was irradiated by UV (λ>300 nm). After treatment with diazomethane, the following compounds were observed:

Compound 1 or 2 $\xrightarrow[\text{Acetone}]{\text{UV, } CH_3N_2}$ Compounds 5 to 11

Compound 2 $\xrightarrow{\text{n-Hexane}}$ Compound 3

Compound 2 $\xrightarrow{H_2O/CH_3OH}$ Compound 4 (Gab et al., 1974)



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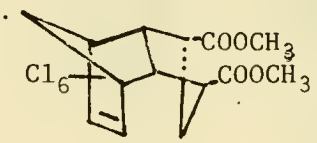

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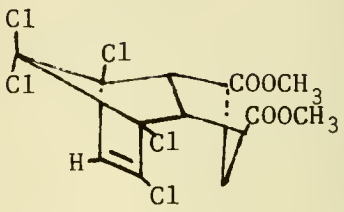

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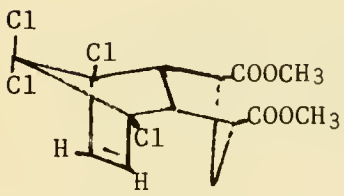

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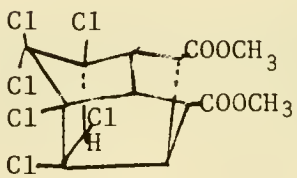

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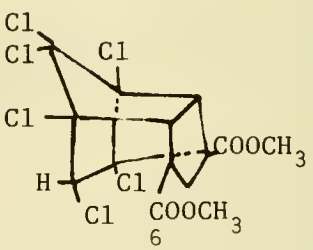

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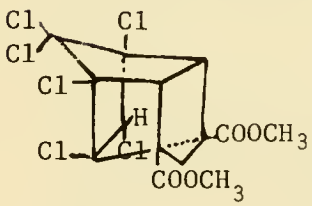

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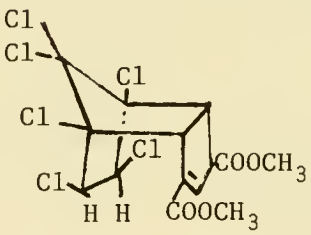

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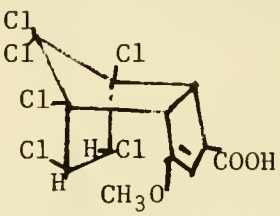

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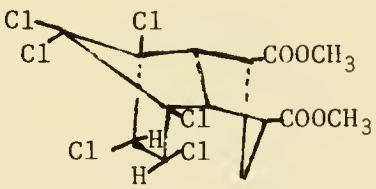

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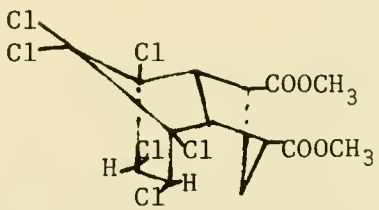

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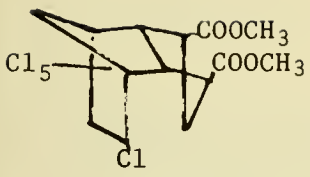

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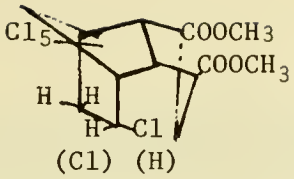

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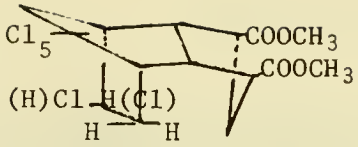

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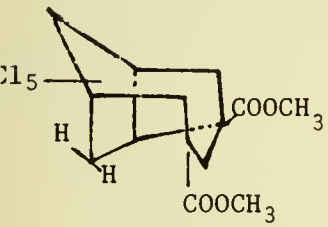

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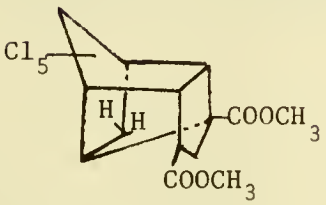

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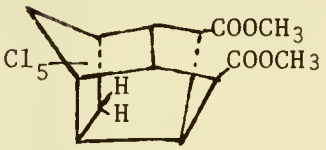

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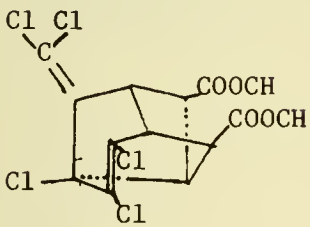

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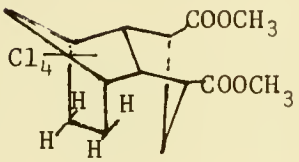

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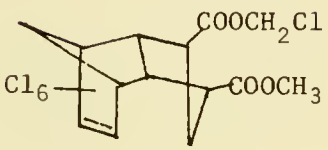

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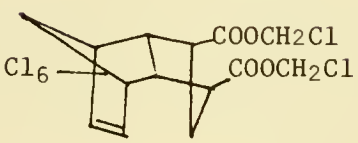

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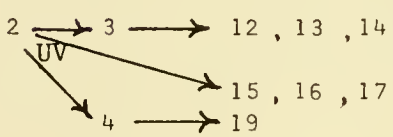
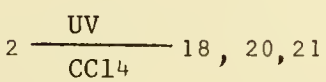




(Gab et al., 1973)

Incubation of compound 1 with rat liver homogenate produced two isomers of the monodechloro analog as the main metabolites (Lay et al., 1974).

Allyl alcohol

When allyl alcohol was added to growing cultures and washed mycelium of *Trichoderma viride*, acetate and acrylate were produced. Additional studies with arsenite-inhibited mycelium indicated that allyl alcohol was metabolized by *T. viride* by a pathway that includes acrylate, lactate, or their acyl-CoA esters, and pyruvate (Jackson, 1973). Acrylic acid appeared to be an intermediate in metabolism of allyl alcohol by *Pseudomonas fluorescens* and *Nocardia corallina* (Jensen, 1961).

AMINOPYRIDINE [4-Aminopyridine]

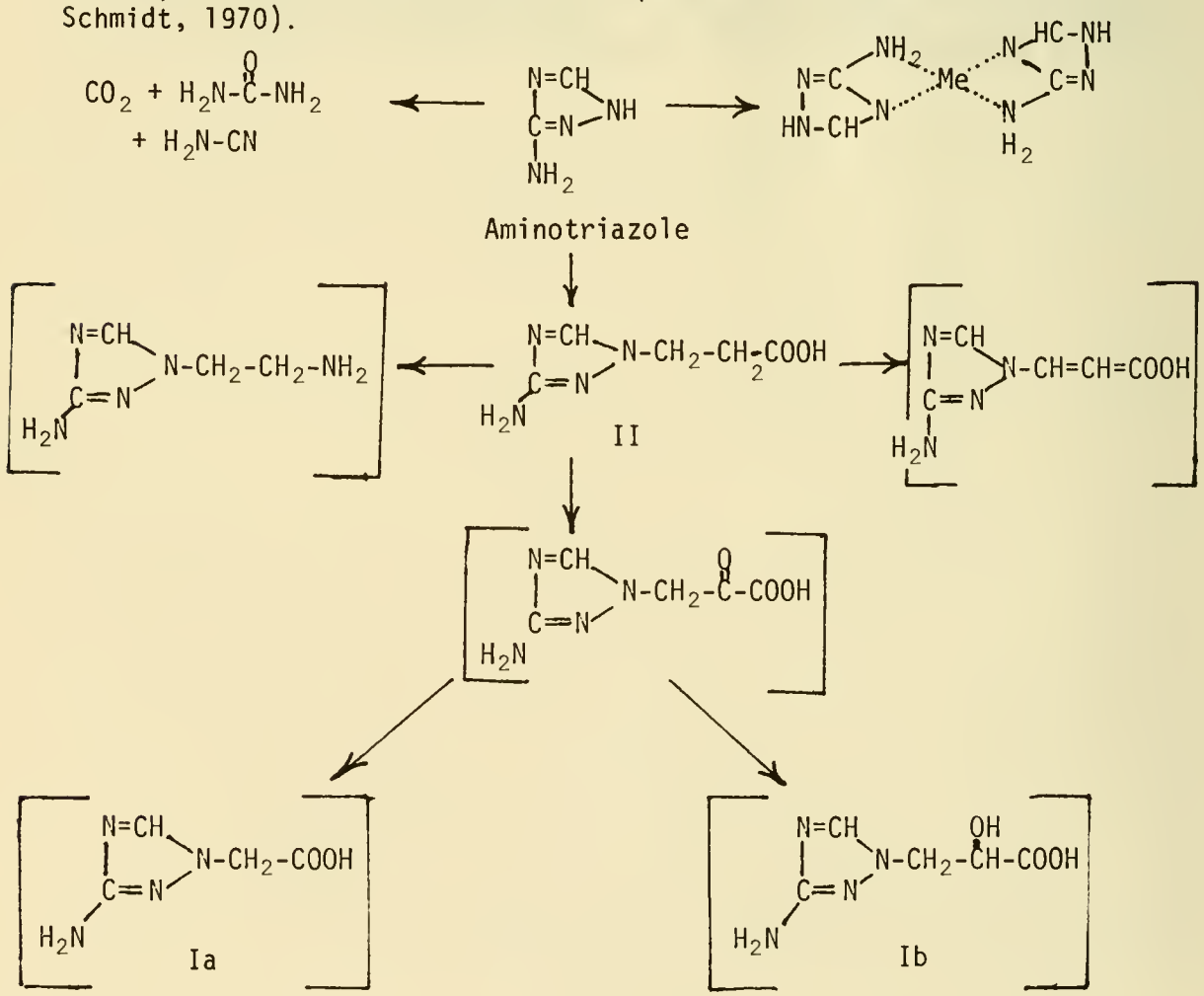
After application of ^{14}C-labeled aminopyridine to corn and sorghum grown in nutrient cultures, studies showed that the aminopyridine was readily absorbed by the roots of both plants and translocated to foliar portions of the plants. There was a general distribution pattern throughout the plants. After one week, no acetone-soluble metabolites were found in corn. In sorghum, however, autoradiograms indicated some degradation within shoots and roots. The presence of acetone-soluble radioactivity suggested that some of the aminopyridine was bound by cellular components (Starr, 1972; Starr and Cunningham, 1974).

Adsorption to soil increased with pH. Soil metabolism studies indicated the need for aerobic conditions for degradation. After a one-week lag period, in soils having optimum temperature, moisture and oxygen requirements, 42 to 60% of the ^{14}C-labeled aminopyridine was degraded to $^{14}CO_2$ within two months. The rate of breakdown decreased with increasing soil pH. Two metabolites were detected in trace amounts (Starr, 1972; Starr and Cunningham, 1975).

AMINOTRIAZOLE [3-Amino-1,2,4-triazole]

When couch grass [Agropyron repens (L.)] was treated with aminotriazole, two main metabolites were observed. These chromatographed identically with two metabolites previously observed in Canada thistle [Cirsium arvense (L.)]. Several other metabolites were also observed but none were identified (Fiveland et al., 1972). In other studies with Canada thistle, three compounds were observed. One was identified as β-(3-amino-1,2.4-triazolyl-1)-α-alanine). An enzyme system from pea (Pisum sativum L. cv. Thompson Laxton) also metabolized aminotriazole. The pea studies indicated that aminotriazole was metabolized via a pathway similar to tryptophan synthesis. The metabolic pathway appeared to be: aminotriazole → alanine derivative (II) → Ib (Smith and Chang, 1973).

After a 39-year-old woman ingested 20 mg/kg of aminotriazole, urine taken some hours later contained unchanged aminotriazole (100 mg/100 ml). No metabolites were found (Geldmacher-v. Mallinckrodt and Schmidt, 1970).



ANILINE

When soil was treated with more than one substituted aniline, asymmetric azobenzenes were produced. Phenylhydroxylamine was identified as a key intermediate which condensed with excess aniline to form the corresponding hydrazobenzene. This was then oxidized to the azobenzene (Bordeleau, 1972).

The fungus Geotrichum candidum produced two extracellular enzymes, an aniline oxidase and a peroxidase, capable of transforming anilines. The apparent K_m (aniline) were 3.1 x 10^{-4}M and 4.4 x 10^{-4}M for peroxidase and aniline oxidase, respectively. Susceptibility to transformation was dependent on electron distribution in the molecule with increased susceptibility to enzymic transformation correlated to increased electron density at the amino group. Anilines substituted at both 2 and 6 positions by electron attracting groups were not transformed. Sequential transformation of propanil to 3,3',4,4'-tetrachloroazobenzene and other complex materials was affected by synergistic interaction of the two common fungi Penicillium piscarm and Geotrichum candidum (Bordeleau, 1972).

Chloroaniline residues of herbicides were immobilized in soil by formation of complexes with humus. In studies with labeled 3,4-dichloroaniline (DCA), it was found that P. frequentans degraded DCA-humic complexes by creating humic oligomers with considerable radioactivity still attached. When the fungus A. versicolor was used, the aniline ring was mineralized (Hsu and Bartha, 1974).

An aryl acylamidase, inducible in Bacillus sphaericus, was obtained and purified. Molecular weight was 75,000 and K_m (linuron) was 2 x 10^{-6}. Substrate specificity of the enzyme was rather low. The enzyme was inducible by linuron, maloran, monalide, propanil, propham, 2-chlorobenzanilide, and 2,5-dimethylfuran-3-carboxanilide (Engelhardt et al., 1973).

A mixture of microorganisms was cultured on propham as the sole carbon source. The microorganisms grew rapidly on nonchlorinated anilides. Ring chlorination depressed respiration and inhibited growth. Acylanilides were hydrolyzed more rapidly than carbanilates and chlorinated rings were degraded more slowly than unchlorinated rings. Ring degradation was affected by chlorination in order: 0 > 2, 4 > 2,4,5 > 3 > 4 > 3,4. Compounds tested were isopropyl N-phenylcarbamate and the 3-, 4-, 3,4-, 2,4-, and 2,4,5-chlorinated analogs; karsil; propanil; swep; propionanilide; fenuron; and phenylurea. The mixture of organisms used was identified as Mycobacterium sp., Arthrobacter sp., Nocardia sp., Fusarium sp., Streptomyces sp., Aspergillus sp., Penicillium sp., and possibly Corynebacterium sp. (McClure, 1974).

The soil fungus Fusarium oxysporum metabolized 4-chloroaniline via oxidation as well as acylation of the amine moiety. The oxidative route was the major metabolic pathway. Products isolated and identified included: 4-chlorophenylhydroxylamine; 4-chloronitrosobenzene; 4-chloronitrobenzene; 4-chloroacetanilide; 4,4'-dichloroazoxybenzene; and 4,4'-dichloroazobenzene. Chloride ion and three phenolic metabolites were also detected. One phenol has been identified tentatively as 2-chloro-4-nitrophenol. The latter implies chlorine migration and requires further identification (Kaufman et al., 1973).

In the presence of a NADPH-generating system, heparinized rat blood, and a liver homogenate, a methemoglobin-forming metabolite was produced with 3,4-DCA (Chow and Murphy, 1975). Other studies have also shown the formation of N-hydroxylated derivatives of p-chloroaniline (Debackere and Uehleke, 1964, from Chow and Murphy, 1975).

ARSENICALS

Sodium arsenate (Na_3AsO_4)

Sodium Arsenite (Na_3AsO_3)

Cows and dogs were fed sodium arsenite and sodium arsenate daily for five days. Urine was collected and analyzed for methylarsenate and inorganic arsenate. In the cow, the levels rose to 0.1 to 0.5 and 1.0 to 4.0 ppm, respectively. When the cows were returned to normal diets, all values returned to control levels (0.02 to 0.10 ppm and 0.1 to 0.2 ppm). In dogs, arsenite feeding produced identical peak values 5.0 to 7.0 ppm for both methylarsenate and inorganic arsenate. Feeding of sodium arsenate to dogs produced a rise to 10 ppm methylarsenate and 5.0 ppm inorganic arsenate. Six days after withdrawal from the arsenic-containing diet, all values reached control levels (Peoples and Lakso, 1973).

Cacodylic acid [Dimethyl arsinic acid]

When applied to soil, cacodylic acid decreased by two routes. The observation of a pungent garlic odor suggested production of alkylarsine and a source of arsenic loss. Degradation to CO_2 and arsenate by microbial action was another route for cacodylic loss (Woolson and Kearney, 1973).

When exposed to DMA and MMA, the three fungi Candida humicola (Dazewska) Diddens and Kodder, Giocladium roseum Bain, and Penicillium sp. produced trimethylarsine. Of these three, Candida humicola only was able to metabolize arsenate and arsenite to trimethylarsine (Cox and Alexander, 1973).

ASULAM (Asulox) [Methyl 4-aminobenzenesulfonylcarbamate]

Asulam exhibits rapid mobility in soil and would be expected to readily leach into subsurface water. Mobility of asulam in soil would be pH dependent, where the undissociated form would leach less rapidly than the associated form (Babiker and Duncan, 1975).

AZINPHOSMETHYL (Methylguthion) [O,O-Dimethyl S-(4-oxo-1,2,3-benzotriazin-3-(4H)-ylmethyl)phosphorodithioate]

The kinetics of azinphosmethyl persistence in soil was studied. Losses of insecticide followed first-order kinetics.

$[A]_o$ = initial concentration

x = amount of A decomposed per unit volume at time t

k_1 = rate constant

$$\log([A]_o - x) = \left[\frac{k_1}{2.303}\right] t + \log [A]$$

plotting of $\log([A]_o - x)$ vs t, y intercept = $\log[A]_o$

and $k_1 = -2.303$ (slope)

$T_{1/2} = t_o + t_{1/2}$ where t_o = lag period
$t_{1/2}$ = time for half of azinphosmethyl to be lost

t_o = experimentally determined

$t_{1/2} = 0.693 k_1$

Moisture and temperature affected the persistence of azinphosmethyl. The half-life varied from 5 days (40C and wet) to 484 days (6C and wet or dry) (Yaron et al., 1974).

The degradation of azinphosmethyl in water was studied. The effect of pH and temperature was determined.

Aqueous solution

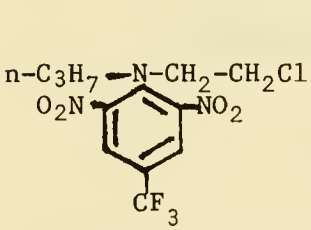
Temperature °C	pH	$t_{1/2}$	K_1
6	8.6	36.4	1.9×10^{-2}
	9.6	4.95	1.4×10^{-1}
	10.7	3.9	1.8×10^{-1}
25	8.6	27.9	2.5×10^{-2}
	9.6	2.4	2.46×10^{-1}
	10.7	2.0	3.4×10^{-1}
40	8.6	7.2	9.5×10^{-2}
	9.6	0.65	1.06
	10.7	0.41	4.1×10^{-1}

(Heuer et al., 1974)

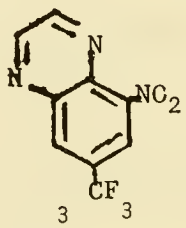
BASALIN (Fluchloralin, BAS-392) [N-(2-Chloroethyl)-N-propyl-2,6-dinitro-4-trifluoromethylaniline]

Irradiation of basalin in methanol-water with a photoreactor yielded nine products. Identification was based mainly on mass spectral data.

When basalin was irradiated in water-methanol in sunlight for 48 days, compounds 3,5,6,7,8 and 11 were observed (Nilles and Zabik, 1974).



Basalin
2



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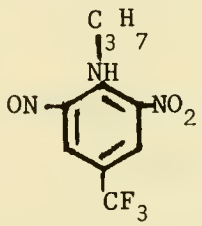

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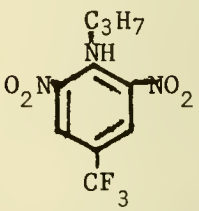

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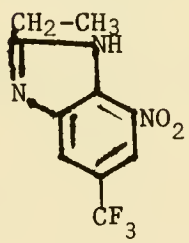

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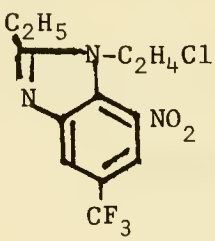

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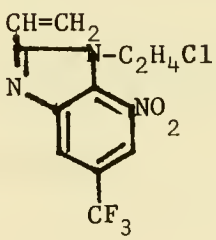

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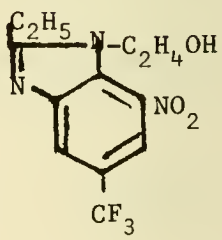

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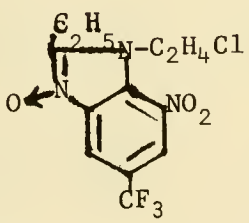

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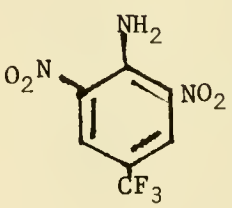

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BENAZOLIN [4-Chloro-2-oxobenzothiazolin-3-ylacetic acid]

The breakdown of ^{14}C-benazolin was studied in wild mustard [Brassica kaber (DC L.C. Wheeler var. pinnatifida (Stokes) L.C. Wheeler], turnip rape (Brassica campestris L. 'Echo'), and rape (Brassica napus L. 'Target'). Negligible amounts of $^{14}CO_2$ were released by the three species after treatment with benazolin. The Brassica species metabolized benazolin to four less toxic derivatives. None of the metabolites were identified but one was characterized as a conjugate of unchanged benazolin (Schafer and Stobbe, 1973).

BENEFIN [N-Butyl-N-ethyl-α,α,α-trifluoro-2,6-dinitro-p-toluidine]

Roots of tobacco seedlings (Nicotiana tobacum L. Kentucky) accumulated benefin from nutrient solutions containing labeled benefin. Two compounds were detected but not identified (Long et al., 1974).

BENOMYL (Benlate, Dupont F - 1991) [Methyl N-(N-butylcarbamoyl)-2-benzimidazolyl carbamate]

EBC [Ethyl N-(2-benzimidazolyl)carbamate]

MBC (Carbendazim) [Methyl N-(2-benzimidazolyl)carbamate]

PARBENDAZOLE [Methyl N-(5(or 6)-butyl-2-benzimidazolyl)carbamate]

THIOPHANATE (TPE) [1,2-Bis(ethoxycarbonylthioureido)benzene]

THIOPHANATE-METHYL (TPM) [1,2-Bis(methoxycarbonylthioureido)benzene]

MCA (NF 48) [2-(3-methoxycarbonylthioureido)aniline]

The fate of benomyl was studied in mice, rabbits, and sheep and with enzyme preparations made from mouse liver, kidney, heart, brain, intestine and blood. Preparations were also made from sheep blood, liver and rumen fluid and from rabbit liver and blood. A summary of the pattern of metabolites is given in Table 1. The pattern of metabolites with mouse kidney, heart and brain was similar to that with liver; but with the intestinal preparation, compounds III and IV were not observed. The butylcarbamoyl side chain was stable at pH 7.5 but became increasingly labile with increased acidity. Optimal pH for hydroxylation was between pH 7.0 and 8.0, and at about pH 8.0 for ester cleavage. After benomyl was administered *per os* to animals, urine and feces were collected at 24-h intervals for 96 h. Results are shown in Table 2 (Douch, 1973).

TABLE 1

In Vitro prep.	Mouse	Rabbit	Sheep
Liver	II - VI	II - VI	II - VI
Blood	II, III, V, VI	II, III, V, VI	II, III, V, VI
Rumen Fluid	---	---	II, III, V, VI

After melon plants were treated with benomyl containing ^{3}H-MBC, most of the label was recovered in the leaves after three weeks. In addition to MBC, 2-AB, conjugates of MBC and 2-AB, benzimidazole, o-aminobenzonitrile, and aniline (Rouchaud et al., 1974). In other studies, benomyl degradation occurred in non-sterilized soil. Cleavage of the benzimidazole ring and production of CO_2 was observed (Siegel, 1975).

TABLE 2

	Metabolite	Urinary Metabolites (%) Free	Conjugated	Fecal Metabolites (%) Free	Conjugated
Mice	VI	12.2		7.8	
	IV	1.6	3.9	1.5	3.8
	II	29.2		15.0	
	III	3.0	5.1	3.3	5.6
Rabbit	VI	11.1		6.5	
	IV	5.4	5.1	3.5	3.2
	II	23.0		9.9	
	III	3.1	8.3	3.0	8.0
Sheep	VI	23.5		12.1	
	IV	1.1	1.9	1.6	2.6
	II	18.6		4.4	
	III	4.1	6.5	3.0	4.7

[2-^{14}C]-Benomyl was applied to soil and turf. After 3 months, the parent compound was not detected in soil. Soil residues consisted of [2-^{14}C]-MBC and [2-^{14}C]-AB. The "half-life" of the total labeled residues was about 6-12 months on bare soil and 3-6 months on turf (Baude et al., 1974).

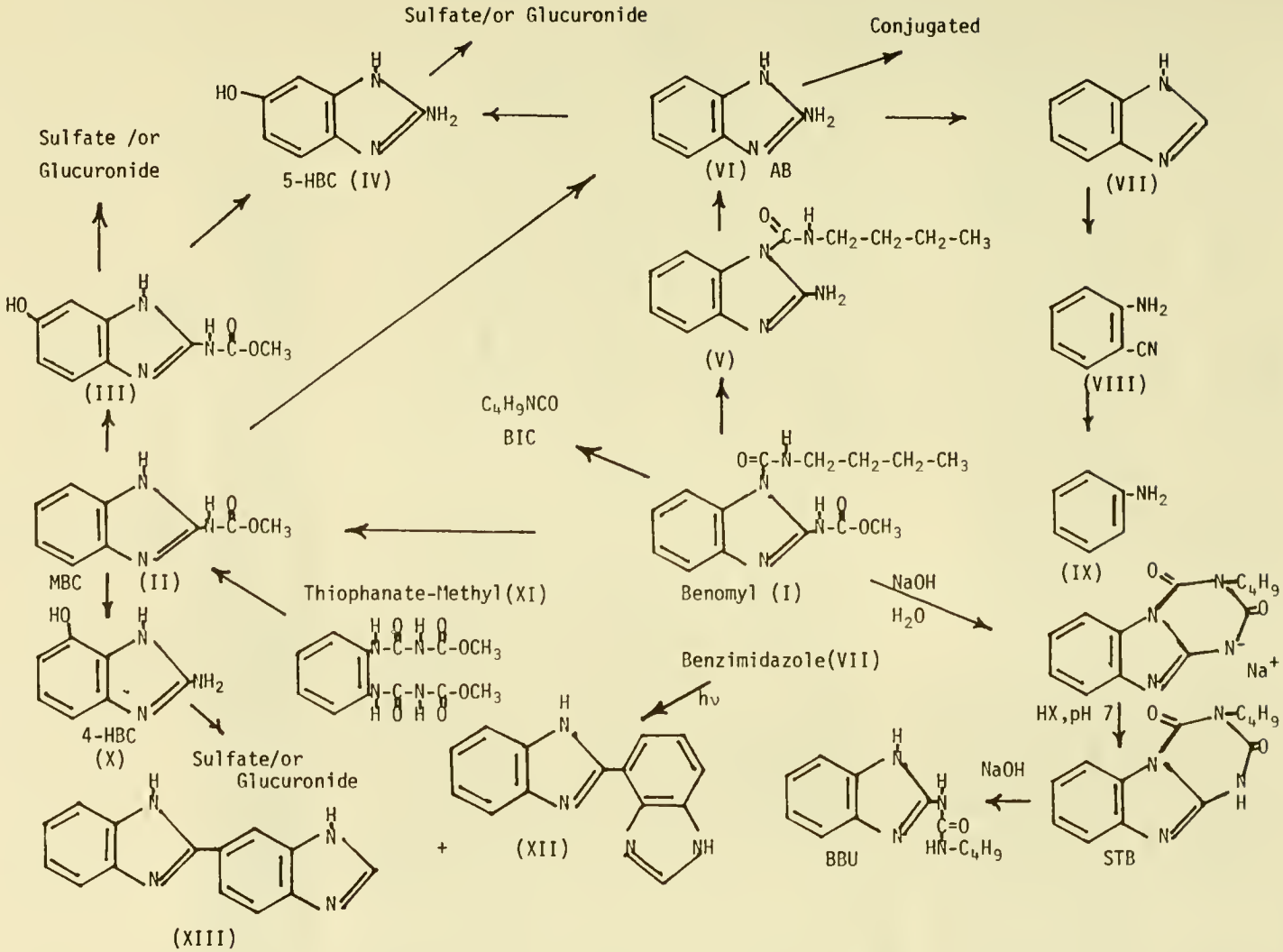
When benomyl was applied to plant foliage, only MBC was found (Baude et al., 1973).

Some studies indicated that benomyl hydrolysis was not rapid in plant tissues nor in aqueous solution. The complete hydrolysis indicated by others may be the result of extraction procedures used (Jhotty and Singh, 1972).

From air over moistened benomyl, a volatile compound was trapped in hexane and identified by GLC on two different columns and by infrared spectroscopy as butyl isocyanate (BIC) (Hammerschlag and Sisler, 1973).

2-^{14}C-Benomyl was fed to a rat. After hydrolysis, the urine contained 5-hydroxy analog (III). Similar results were obtained with 2-^{14}C-MBC. Three conjugates were indicated. When administered to a beagle dog, benomyl was metabolized to 5-HBC. The dairy cow metabolized benomyl to 4- and 5-HBC. In the eggs of chickens fed benomyl, only 5-HBC was observed at the high (25 ppm) feeding level. No residues (<0.02 ppm) were observed in eggs from hens on the low (5 ppm) feeding level (Gardiner et al., 1974).

Dwarf pea plants were grown in nutrient solutions and root-treated with ^{14}C-benomyl. MBC was present in large quantities. Hydrolysis of plant-bound residues with hot NaOH released half the bound label, part of which was 2-aminobenzimidazole (Siegel and Zabbia, 1972).


Sulfate/or Glucuronide
Conjugated
5-HBC (IV)
(VI) AB
(VII)
Sulfate /or Glucuronide
(III)
(V)
(VIII)
C_4H_9NCO
BIC
(IX)
MBC
(II)
Benomyl (I)
NaOH
H_2O
Thiophanate-Methyl(XI)
Benzimidazole(VII)
$h\nu$
4-HBC
(X)
Sulfate/or Glucuronide
HX,pH 7
NaOH
(XII)
BBU
STB
(XIII)

Cells of apple and cucumber leaves were exposed in nutrient media to MBC. Cytoplasmic uptake by apple cells was constant for 26 h, whereas uptake by cucumber cells was negligible. When applied to cucumber leaves, 1.56% of the [ring-^{14}C]-MBC was metabolized to CO_2 (Solel et al., 1973).

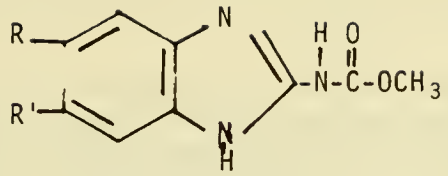
Benomyl decomposes in many solvents to give MBC as a precipitate. Solvents included: benzene, ethyl ether, ethanol, acetone, ethyl acetate, methylene chloride, chloroform (Chiba and Doornbos, 1974).

Benomyl was taken from sprays, waxes, and alkaline peeling solutions and brought to pH 6 with 0.1N HCl. The precipitate was removed and analyzed. After characterization on the basis of evidence derived from infrared, nmr, and mass spectra, verification of the structure as that of STB was made by synthesis and comparison of the preceding physical evidence plus m.p. or decomposition point. Standing alkaline solutions also produced a second precipitate which was identified as BBU (White et al., 1973).

PARBENDAZOLE

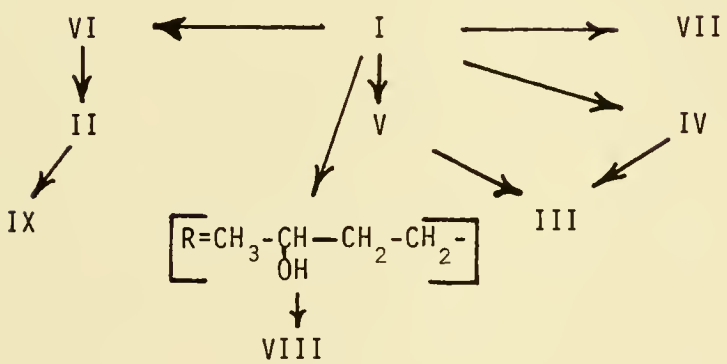
In cattle and sheep, one of the major parbendazole metabolites was identified as compound II by means of UV and mass spectra. The other major metabolite in sheep and cattle was identified as the glycol, compound III. Other metabolites obtained from sheep were the alcohols, compounds IV, V and VI. The phenol VII was obtained from cattle (Dunn et al., 1973). In other studies with sheep, after an oral dose of ^{14}C-parbendazole (labeled at C-2), urine was collected and analyzed. Structures of the metabolites were determined by means of UV, IR, proton magnetic resonance, mass spectrometry and chemical synthesis. Seven metabolites were identified as compounds II, III, IV, V, VI, VII and IX. Incubation of the metabolites with glucuronidase resulted in 38% hydrolysis of the total radioactivity and supported the fact that compounds II, III and IX were excreted primarily unconjugated, whereas compounds IV, V, VI and VII were excreted mainly as glucuronides. Other studies with glusulase indicated little excretion of the metabolites as sulfates (DiCuollo et al., 1974).

The metabolites identified as compounds II and VI were obtained from Cunninghamella bainieri; and the fungus Paecilomyces sp., produced compound VIII (Dunn et al., 1973). Other studies with C. bainieri ATCC 9244 also gave compounds II and VI (Valenta et al., 1974).



Parbendazole (I)

	R	R'
I.	$CH_3-CH_2-CH_2-CH_2-$	H-
II.	$HOOC-CH_2-CH_2-CH_2-$	H-
III.	$CH_3-CH_2-CH(OH)-CH(OH)-$	H-
IV.	$CH_3-CH_2-CH_2-CH(OH)-$	H-
V.	$CH_3-CH_2-CH(OH)-CH_2-$	H-
VI.	$CH_2(OH)-CH_2-CH_2-CH_2-$	H-
VII.	$CH_3-CH_2-CH_2-CH_2-$	HO-
VIII.	$CH_3-C(=O)-CH_2-CH_2-$	H-
IX.	$HOOC-CH_2-CH(OH)-CH_2-$	H



THIOPHANATE

When thiophanate or the methyl analog was irradiated with UV in the solid state, no reactions occurred. When exposed in aqueous solution to UV and sunlight, both fungicides were converted to their respective alkyl benzimidazol-2-yl carbamates (EBC and MBC). Residues of the fungicides on cotton plants, following spray application, were also converted by sunlight to EBC and MBC (Buchenauer et al., 1973).

In soil, thiophanate underwent rapid conversion to MBC. Conversion was reduced by treating the soil with steam or increasing the alkalinity. At pH 7.4 the rate was more than 4 times that at pH 5.6. Very little (less than 1%) of ring-^{14}C-labeled MBC was converted to $^{14}CO_2$ even after 51 days incubation of soil. When ^{14}C-methyl label was used, about 15% of the label appeared as CO_2 after 51 days (Fleeker et al., 1974).

Photoisomerization of benzimidazole gave rise to two compounds identified as the dimers XII and XIII (see diagram on page 31) (Cole et al., 1973).

BENTAZON [3-Isopropyl-1H-2,1,3-benzothiadiazin-4(3H)-one-2,2-dioxide]

After oral administration to rats, bentazon was rapidly absorbed. Excretion, primarily (84%) in urine, was rapid and largely unchanged bentazon. Two metabolites, one of which may be the N-glucuronide of bentazon, were detected. Traces of radioactivity were also found in the bile (BASF, 1973; Chasseaud et al., 1972).

When ^{14}C-bentazon was applied to spring wheat, Opal variety, the active ingredient was taken up via roots from a nutrient solution. At harvest, 173 days after test start, two-thirds of total activity could not be extracted from the straw. The remainder consisted of soluble complexes of the active ingredient and of free bentazon. Fifty days after foliar spraying of soya plants, more than 40% of the methanol extractable residues was in the form of complexes of mono- or oligo-saccharides with hydroxylation of the aromatic ring (BASF, 1973).

Bentazon does not persist in loamy sand soil. Within 15 weeks, bentazon broke down quantitatively at room temperature and 15% soil moisture. Anthranilic acid-isopropylamide was identified. This broke down quickly (BASF, 1973).

N-Benzoyl chloride-N'-(2,4,6-trichlorophenylhydrazide)

The photolytic half-lives of this compound were 12 and 16 h, with and without a filter. Under the experimental conditions, using a combination of TLC, GLC and MS, eight products were identified:

I. Benzoyl 2-(2,4-dichlorophenyl)hydrazide
II. 2,4,6-trichlorobenzophenone
III. N-(2,4,6-trichlorophenyl)benzamide
IV. N-(2,4-dichlorophenyl)benzamide
V. N-(2,6-dichlorophenyl)benzamide
VI. 2,4-dichlorobenzophenone
VII. 1,2-dibenzoyl-1-(2,4,6-trichlorophenyl)hydrazine
VIII. 1,2-dibenzoyl-1-(2,4-dichlorophenyl)hydrazine

(Koshy et al., 1975)

BENZOYLPROP-ETHYL (Suffix) [Ethyl N-benzoyl N-(3,4-dichlorophenyl)-2-aminopropionate]

^{14}C-Benzoylprop-ethyl was applied to foliage of wheat (Triticum aestivum), oat (Avena sativa), and barley (Hordeum vulgare) seedlings. Metabolism of herbicide was similar in all three plant species. Extracts of wheat seedlings sampled up to 15 days after treatment indicated the presence of as many as five metabolites: des-ethyl analog and its ß-glucoside, debenzoylated analog, o-hydroxybenzoic acid as a conjugate, and one unidentified compound (Beynon et al., 1974d). In other studies with labeled herbicide and spring and winter wheat, the crop was sampled at harvest and the following compounds were observed: N-benzoyl 3,4-dichloroaniline; benzoyl prop; several sugar complexes. Other products present were not identified (Beynon et al., 1974a and c).

In soil, benzoylprop-ethyl was de-ethylated. The acid, upon standing, became tightly bound to the soil before undergoing slow debenzoylation to N-(3,4-dichlorophenylalanine) and benzoic acid. 3,4-Dichloraniline which formed was present as humic acid complexes. Polar products observed were shown to arise from the dichloroaniline. No 3,3^{1},4,4^{1}-TCAB was detected. The rate of degradation for various soils varied from 1 to 12 weeks (Beynon et al., 1974a,b,c).

After ^{14}C-benzoylprop-ethyl was applied to the leaves of cereal plants, only 7% of the total applied radioactivity moved from the treated leaf during a 3-day period. Movement of this herbicide occurred in the form of the acid and acid conjugates (Jeffcoat and Harries, 1973).

BHC (HCH) [1,2,3,4,5,6-Hexachlorocyclohexane]

When α, β, γ and δ isomers of BHC were orally administered to rats, some β isomer accumulated in the tissues, presumably from isomerization. The level of accumulation was β>>α>γ>δ (Kamada, 1971).

γ-PCCH was metabolized by rats primarily to 2,4,5-TCP and a trace of 2,3,5-TCP; β-BHC was metabolized to 2,4,6-TCP. When α- and δ-BHC were administered to rats, 2,4,5- and 2,4,6-TCP formed. The γ-isomer was metabolized to 2,4,6-, 2,3,5- and 2,4,5-TCP and 2,3,4,5- and 2,3,4,6-tetrachlorophenol (TTCP) in addition to a configurational isomer of 2,3,4,5,6-pentachlorocyclohex-2-en-1-ol (Freal and Chadwick, 1973). When rats were fed γ-BHC plus DDT, there was significantly more excretion of 2,4,5-TCP and 2,3,4,6- and 2,3,4,5-TTCP than in the absence of DDT (Chadwick and Freal, 1972).

Wistar rats were orally administered α-BHC. Controls and treated rats were sacrificed; the livers were removed, homogenated and then centrifuged for 10 min at 12,000g. The supernatant was centrifuged at an average 100,000g (140,000g maximum) for 90 min. Portions of these fractions were dialyzed or gel-filtered. When these preparations were incubated with labeled (^{14}C, ^{3}H, or ^{36}Cl) α-BHC in the presence of air (2 h, 37C, and pH 7.4), 5 to 10% of the label was converted to water-soluble materials. Longer incubation, more alkaline pH, and preparations from treated rats increased the amount of water-soluble labeled materials. GSH was required for the reaction. These studies indicated that dechlorination was part of the overall reaction and that four atoms of chlorine per molecule HCH were eliminated. Although the main product of the reaction was not established, there was evidence that it was a conjugate of glutathione with the BHC-moiety rendered aromatic, probably S-2,4-dichlorophenylglutathione. Alkaline hydrolysis produced a thiophenol or mixture of thiophenols (Kraus et al., 1973; Noack and Portig, 1973; and Portig et al., 1973).

After adaptation of rats to lindane, ^{14}C-lindane was orally administered. Fat, kidney and musculature were the main sites of deposition. Pituitary and thyroid glands had highest activity. Differences between cortex, stem and cerebellum were marked. The metabolites γ-PCCH, pentachlorobenzene and hexachlorobenzene were observed. Large amounts of conjugates and strongly polar, hexane-soluble metabolites were also present in feces, urine and organs. Glucuronides and other unidentified water-soluble conjugates were observed. Half of the administered lindane was excreted within 3 or 4 days (Seidler et al., 1975).

Studies with mice indicated differences in excretion rates of α, β and γ isomers. The data indicated that metabolism of the γ-isomer was greater than the β-isomer and that the α-isomer was intermediate. Most metabolites from the γ- and β-BHC were conjugated as sulfates and glucuronides. After hydrolysis, chlorophenols were obtained. About 25% of the total metabolites in urine was 2,4,6-trichlorophenol. 2,4-Dichlorophenol was also prominent. From β-HCH, traces of 2,4,5-trichlorophenol were also identified. Free chlorophenols were also observed. One behaved like 2.4-dichlorophenol (Kurihara and Nakajima, 1974).

Uniformly labeled lindane-^{14}C was fed in gelatin capsules to rabbits for 26 weeks. About 54% of the label was excreted in urine and 13% in feces by the end of the feeding period. Of the urinary metabolites, 55% was ether-soluble, in which 14 chlorophenols were observed. Four were identified by infrared: 2,3,5-, 2,4,5- and 2,4,6-trichlorophenol and 2,3,4,6-tetrachlorophenol. Three were identified by gas chromatography and mass spectrometry: 2,3- and 2,4-dichlorophenol and 2,3,4,5-tetrachlorophenol. Seven chlorophenols were tentatively identified by gas chromatography: 2,5-, 2,6- and 3,4-dichlorophenol; 2,3,4-, 2,3,6- and 3,4,5-trichlorophenol; and pentachlorophenol. Six chlorobenzenes were also observed: 1,2-dichlorobenzene; 1,2,4-trichlorobenzene; 1,2,3,4-, 1,2,4,5- and/or 1,2,3,5-tetrachlorobenzene; and pentachlorobenzene (Karapally et al., 1973).

After injection of uniformly ^{14}C-labeled α-BHC into adult rats, urine and feces were collected. In 4 weeks, 65% of the label was excreted in urine and 16% in feces. Most of the urinary metabolites apparently contained chlorine. Nearly all of the fecal ^{14}C was unchanged α-BHC (Noack et al., 1975). Urinary metabolites obtained in other studies indicated that the proportion of free chlorophenols was 5% or less of all urinary BHC metabolites. Both 2,4,5- and 2,4,6-trichlorophenol were identified by UV, IR and cocrystallization with authentic compounds. There were indications that 2,3,5-trichlorophenol and 2,3,4,6-tetrachlorophenol were also present but this could not be confirmed. After alkaline and acid hydrolysis, 2,4,6-trichlorophenol was found. The presence of 2,3,4,6-tetrachlorophenol, 2,4,5- or 2,3,5-trichlorophenol were indicated. The presence of dichlorothiophenols was also observed (Koransky et al., 1975).

Incubation of lindane with rat liver homogenates produced hexachlorocyclohexene (HCCH). When rats were administered HCCH, previously observed lindane phenolic metabolites were observed: 2,4,6-, 2,3,5-, and 2,4,5-trichlorophenol; 2,3,4,5- and 2,3,4,6-tetrachlorophenol. In addition to these, 2,3,4,5,6-pentachloro-2-cyclohexen-1-ol was also found. Similar results were obtained with *in vitro* studies. The enzyme system involved in the initial

dehydrogenation of lindane to HCCH was characterized as a hepatic microsomal MFO and a cytochrome P-450 which requires molecular oxygen and NADPH (Chadwick et al., 1975).

When pentachlorobenzene was orally administered to rabbits, unidentified dechlorinated compounds appeared in feces and tissues. In urine, data indicated the presence of p-chlorophenol, pentachlorophenol and some less chlorinated benzenes. After administration of 1,3,5-trichlorobenzene to rabbits, monochlorobenzene was expired and found in feces and tissues. Urine contained 2,4,6-trichlorophenol, 4-chlorophenol, 4-chlorocatechol and perhaps other monochlorophenols (Parke and Williams, 1960).

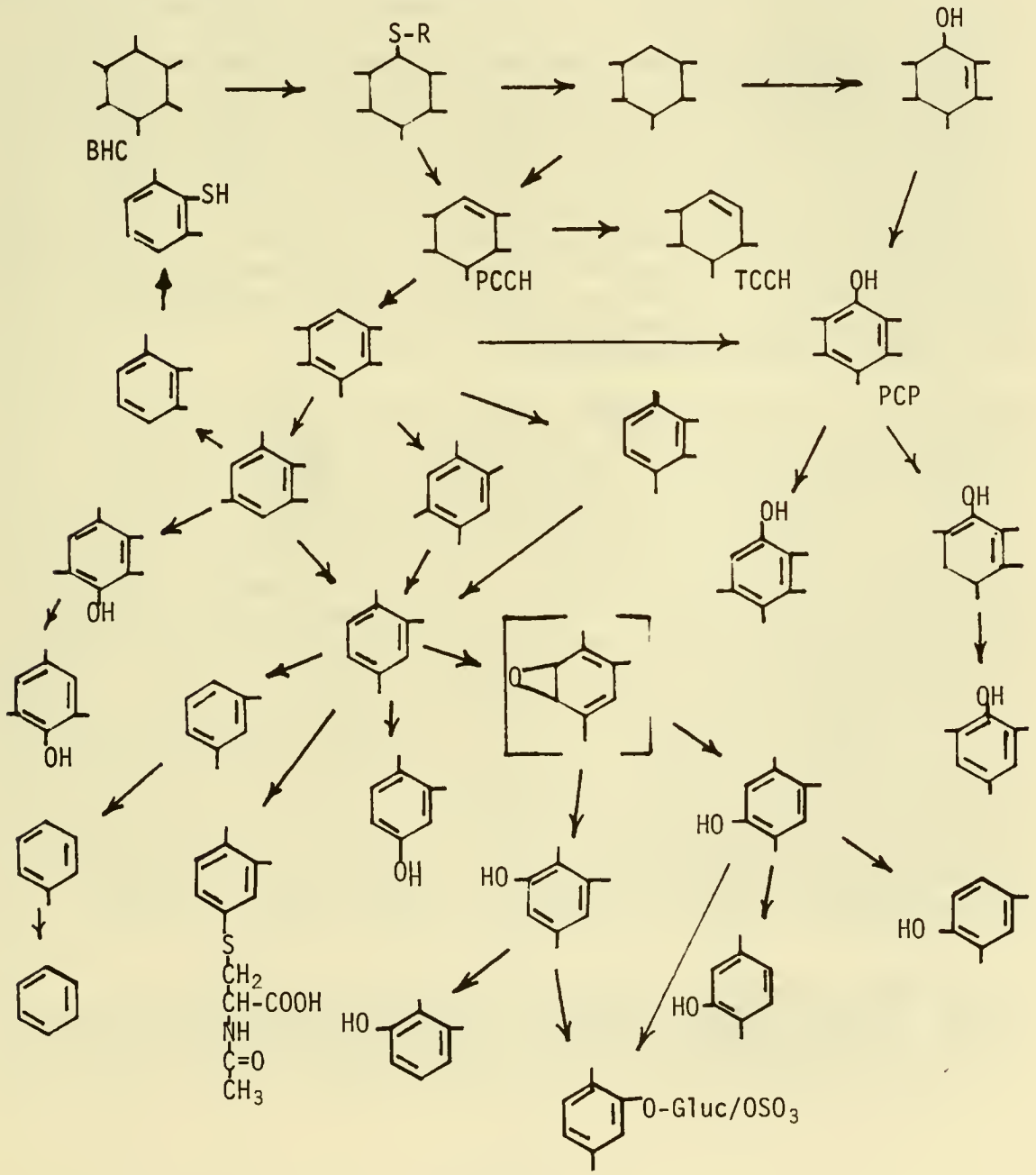
When the three tetrachlorobenzenes were orally administered to rabbits in arachis oil they were partly excreted unchanged in feces. The 1,2,3,4-tetrachlorobenzene was slowly metabolized to 2,3,4,5-tetrachlorophenol which was excreted in urine as such and conjugated. In 6 days, 43% of the 1,2,3,4-tetrachlorobenzene was oxidized. 1,2,3,5-tetrachlorobenzene was oxidized (5% in 6 days) to the 2,3,4,6-tetrachlorophenol. Approximately 2% of the administered 1,2,4,5-tetrachlorobenzene was oxidized to 2,3,5,6-tetrachlorophenol in 6 days. Some dechlorination products were also probably formed. The phenols were excreted as glucuronides and sulfates as well as unconjugated (Jondorf et al., 1958).

	Percent of dose excreted			
Tetrachlorobenzene administered	Glucuronide	Sulfate	Mercapturic	Free
1,2,3,4-	30	3	<1	8
1,2,3,5-	6	2	0	1
1,2,4,6-	4	1	0	1

The mussel Mytilus edulis was exposed to ^{14}C-labeled lindane. Analyses indicated the formation of two highly polar compounds amounting to about 3% of the lindane. Neither metabolite was identified (Ernst, 1975).

^{14}C-Lindane was added to a nutrient solution in which lettuce plants were grown. Radioactivity extracted from the nutrient solution after 4 weeks amounted to 7.8% of the applied material. About 14.1% of the applied radioactivity was recovered from the plants and the remainder was lost, possibly by evaporation. Of the material recovered from the nutrient solution, 82% was unchanged lindane; 15%, polar material; and 3%, nonpolar. The polar material was identified with the aid of GLC/MS as 2,3,4,6-tetrachlorophenol (<1%), pentachlorophenol (ca. 5%) and conjugated pentachlorophenol (1%). An unidentified highly hydrophilic substance (8%) was also present. In the nonpolar fraction, there was 1,2,3-trichlorobenzene, 1,2,3,4-tetrachlorobenzene, pentachlorobenzene, hexachlorobenzene,

γ-pentachlorocyclohexene and an unidentified compound that is probably a hexachlorocyclohexene. From the lettuce plants, the extracted radioactivity consisted of unchanged lindane (77%), polar material (20%), and nonpolar material (about 3%). Again, the polar fraction contained free 2,3,4,6-tetrachlorophenol (ca. 1%), conjugated phenols including a tetrachlorophenol (ca. 3%), pentachlorophenol (ca. 4%) and a highly polar unidentified compound (11 to 12%). The nonpolar material consisted of 1,2,3-trichlorobenzene, 1,2,4-trichlorobenzene, pentachlorobenzene, γ-pentachlorocyclohexene and a hexachlorocyclohexene. There were also indications of a tetrachlorobenzene (Kohli et al., 1976).



Wheat plants were grown from seeds. From the roots, metabolites identified included 1,3,5-trichlorobenzene; 1,2,4-trichlorobenzene; 1,2,3-trichlorobenzene; 1,2,3,4-tetrachlorobenzene; 1,2,4,5- and/or 1,2,3,5-tetrachlorobenzene; γ-PCCH; and pentachlorobenzene. The presence of m- and p-dichlorobenzene were also indicated. Also observed, but lacking confirmation because they appeared in quantities too small, were a number of chlorophenols: pentachlorophenol; 2,3,5,6- and/or 2,3,4,6-tetrachlorophenol; 2,3- and 2,4-dichlorophenol; 2,3,4- and/or 2,4,5-trichlorophenol; and 2,4,6-trichlorophenol (Balba and Saha, 1974).

Use of lindane for protection of stored wheat grain against insects has been suggested. When the fate of lindane residues in wheat flour under normal conditions of bread making was studied with ^{14}C-labeled compound, about 75 to 82% of the radioactivity was retained by the baked bread and 94% of this was present as lindane. Identified in the bread were: γ-PCCH; 1,2,4-trichlorobenzene; 1,2,3,4-tetrachlorobenzene; 1,2,4,5- and/or 1,2,3,5-tetrachlorobenzene (Saha, 1974).

After treatment with ^{14}C-labeled lindane, wheat grains were stored in closed containers at varying temperatures and times, with and without added water. Less than 3% of the lindane was degraded. Small amounts of γ-PCCH were present as residues. No other products were detected (Saha and Lee, 1974).

Houseflies were dosed topically with ^{14}C-γ-BHC. Homogenation and chromatography of the extract indicated the presence of S-2,4-dichlorophenylglutathione. Studies with grass grubs gave similar results. Houseflies and grass grubs also converted γ-PCCH and δ-PCCH into metabolites that had chromatographic properties identical with S-2,4-dichlorophenylglutathione. Inhibitors and colorimetric assays lead to the conclusion that a PCCH is not a major intermediate metabolite of γ-BHC in these insects. These studies tend to support earlier assumptions that a pentachlorocyclohexylglutathione is the initial metabolite of γ-BHC (Clark et al., 1969).

A mold capable of degrading lindane was isolated but not identified. The main metabolite, short-lived, was identified as γ-pentachlorocyclohexene. The following compounds were also found in varying amounts: hexachlorobenzene; pentachlorobenzene; 1,2,3,4-, 1,2,4,5- and 2,3,4,6-tetrachlorobenzene; 1,2,3-, 1,2,4- and 1,3,5-trichlorobenzene; 2,3,4- and 2,4,6-trichlorophenol; 1,2- and 1,4-dichlorobenzene; 2,3,4,5-tetrachlorophenol; and pentachlorophenol (Engst et al., 1974).

In laboratory studies, P. putida normally produced γ-PCCH from γ-BHC. In the presence of NAD, α-HCH was also formed. γ-Tetrachlorocyclohex-1-ene was observed (Benezet and Matsumura, 1973).

In an aerobic artificial lake impoundment, 15% of γ-BHC was converted in 2100 h to the α-isomer. Under anaerobic conditions, 90% of the γ-isomer was converted to α- and δ-isomers in 2100 h (Newland, 1969).

Two-thirds to three-fourths of the material lost from a calcareous soil treated with lindane was lost by volatilization as PCCH (Cliath and Spencer, 1972). Fifteen years after application of HCH to soil, samples were taken and analyzed. Isomeric composition of BHC in the soil samples was considerably different from that of technical BHC. Persistence was of the order β>δ>γ>α (Stewart and Chisholm, 1971).

A bacterium was isolated from rat feces and identified as Escherichia coli. When incubated in trypticase soy broth with lindane, 10% of the lindane was metabolized to γ-PCCH. Structure was confirmed by synthesis, gas chromatography and mass spectra (Francis et al., 1975).

The relationship between concentrations of BHC in the medium and the concentration in bacteria (living and dead) is given by the relationship

$$C_B = KC_M^n$$

C_B = concentration in bacteria (ppm)

C_M = concentration in media (ppm)

α-BHC	$K = 4.2 \times 10^1$	n = 0.7
β-BHC	$K = 3.7 \times 10^2$	n = 0.7
γ-BHC	$K = 2.6 \times 10^1$	n = 1.0

The process is apparently not energy dependent (Sugiura et al., 1975).

An apparent half-life equal to 16 days was calculated for γ-BHC degradation in an anaerobic artificial impoundment. Under aerobic conditions, degradation was considerably slower. Onset of degradation occurred at 264th and 840th hour anaerobically and aerobically, respectively. After 2100 h of incubation of lindane in anaerobic and aerobic sediments, 83.2 and 19.0% respectively, of the added ^{14}C activity was volatilized. Chromatography of the hexane-acetone extract of the aerobic impoundment sediment showed only γ- and α-BHC. With the anaerobic sediment, chromatography of the extract showed α-, γ- and δ-BHC. These studies indicated that γ-BHC could undergo isomerization in natural systems. Thermodynamic stability of the isomers is of the order β>δ>α>γ (Newland et al., 1969).

Lindane was added to a sandy loam soil and incubated for 6 weeks under flooded conditions. The soil and water was extracted and chromatographed. Five peaks were observed and the retention times

corresponded to 1,2,4-trichlorobenzene, 1,2,3,5- and/or 1,2,4,5-tetrachlorobenzene, 1,2,3,4-tetrachlorobenzene, γ-PCCH and γ-3,4,5,6-tetrachlorocyclohexene (γ-BTC). GC/MS was used to confirm the identities of the compounds except 1,2,3,5- and/or 1,2,4,5-tetrachlorobenzene which were not present in sufficient quantity (Mathur and Saha, 1975).

When pentachlorocyclohexene (PCCH) was synthesized by partial additive chlorination of chlorobenzene, combined gas chromatography-mass spectrometry revealed that at least five different isomers of pentachlorocyclohexene had been formed. Dechlorination products of various isomers of BHC or PCCH in NaOH and in pyridine were compared. Results indicated that β-PCCH was the monodechlorination product of α-BHC (Munster et al., 1975).

The isomerization of 1,3,4,5,6-pentachlorocyclohexene-1 (γ-PCCH) was studied in dimethyl sulfoxide. After a long reaction time, starting with any isomer, γ-PCCH became the most abundant component. Three new isomers were also isolated for the first time (Kurihara, et al., 1974b).

Recent studies have also shown that lindane forms a colored complex with montmorillonite clay (Haque and Hansen, 1975).

Diphenyl [Biphenyl]

Adult male Wistar rats were fed a diet containing biphenyl. Urine was collected and analyzed. Five metabolites were isolated and identified by means of melting point depression, chemical test, and infrared spectra as:

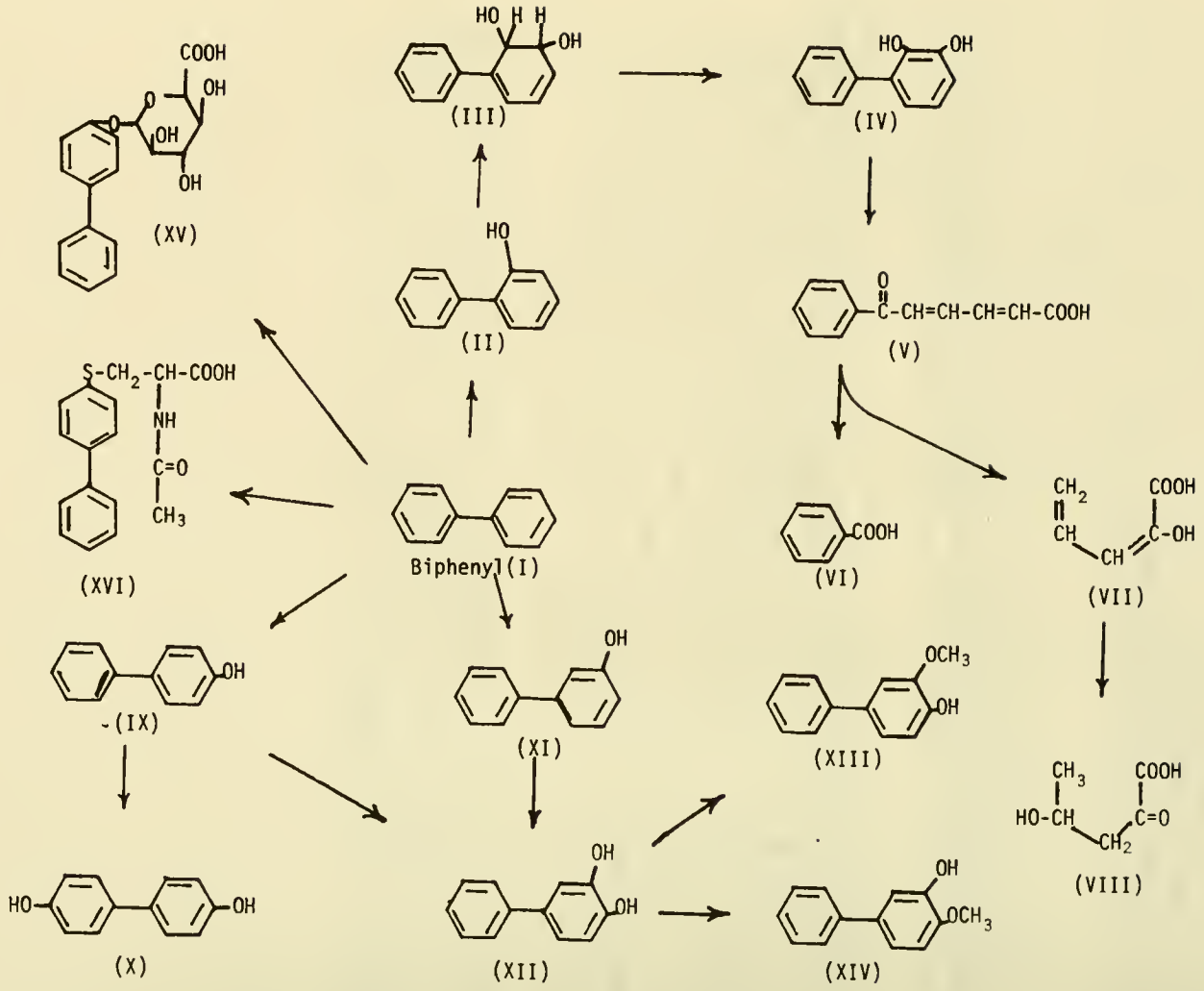
IX. 4-hydroxydiphenyl
X. 4,4-dihydroxydiphenyl
XII. 3,4-dihydroxydiphenyl
XV. p-(β-D-glucuronosidodiphenyl)
XVI. N-acetyl-p-(S-diphenyl)-L-cysteine

(West et al., 1956)

Studies with rabbits fed biphenyl, showed that the 3-hydroxybiphenyl (XI) and a mixture of monomethylated analogs of 3,4-dihydroxybiphenyl (XIII and XIV) were present in urine (Raig and Ammon, 1972).

The ability of several phyla of marine organisms to metabolize biphenyl was investigated with in vitro studies: mature skate (Raja ocellata), mussels (Mytilus edulis), starfish (Asterias vulgaris), rock crab (Cancer irroratus), red crab (Gerydon quinquidens), lobster (Homarus americanus), brook trout (Salvelinus fontinalis), and plankton that consisted mainly of large zooplankton. Tissue homogenates and intact plankton samples were used. Biphenyl was metabolized in vitro by all tissues primarily to 4-hydroxybiphenyl (IX) and to some extent to 2-hydroxybiphenyl (II). Rates of formation of the 4-hydroxy analog ranged from a high of 400 n moles/g skate tissue to a low of 2 n moles/g starfish tissue (Willis and Addison, 1974).

Metabolism of biphenyl by Pseudomonas putida apparently proceeded by way of 2,3-dihydro-2,3-dihydroxybiphenyl (III), 2,3-dihydroxybiphenyl (IV), and 2-hydroxy-6-oxo-6-phenylhexa-2,4-dienoic acid (V) to benzoic acid (VI) (Catelani et al., 1971 and 1974). The presence of either 2-hydroxypenta-2,4-dienoate (VII) or 4-hydroxy-2-oxovalerate (VIII) was also indicated (Catelani et al., 1973).

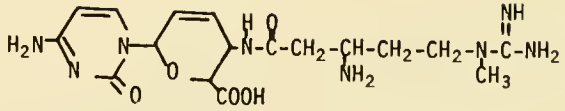

(XV)
(III)
(IV)
(II)
(V)
(XVI)
Biphenyl (I)
(VI)
(VII)
(IX)
(XI)
(XIII)
(VIII)
(X)
(XII)
(XIV)

After application to rice plants via culture solution, Blasticidin S(I) was degraded. A small amount of cytomycin(III) and the deamino-blasticidin S analog II were also observed. When incubated with micro-organisms, Blasticidin S was also degraded: soil bacterium (unident-ified) > Ps. aernginosa > Phytophythora parasitica > Fusarium oxysporum > soil fungus (unidentified) > Ps. ovalis 1002 > Ps. marginalis. The main products after exposure to washed mycelia of a soil fungus were compounds II, III, and IV (Yamaguchi et al., 1972). In other studies, a strain of Aspergillus fumigatus, isolated from soil, converted blasticidin S into four metabolites. One was identified as deaminohydroxyblasticidin S(II). Another was identified as deamino-hydroxycytomycin(IV) (Seto et al., 1966).

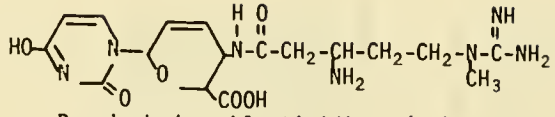
When hydrolyzed with acid, compound III gave rise to uracinine(V) and pseudoblastidone(VII); XI gave uracinine(V) and blastidone(VIII); blasticidin S(I) gave cytosine(IX), cytosinine(VI), blastidic acid(X); cytomycin(III) gave pseudoblastidone(VII) (Seto et al., 1966).

Cytosinine acid hydrolysis gave cytosine, levulinic acid, NH_3, and CO_2. Analysis of this data plus that derived from PtO_2 reduction and ozonolysis of the N,N^1- diacetyl methyl ester permitted assignment of the structure of cytosinine. Similarly, acid hydrolysis of uracinine gave uracil and permitted assignment of a structure (Otake et al., 1966a).

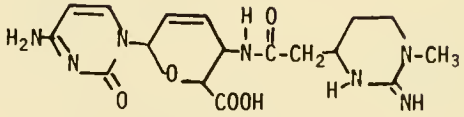
Acid hydrolysis of blasticidin S gave cytosinine and blastidic acid. Products of alkaline hydrolysis and PtO_2 hydrogenolysis permitted structural assignments to blasticidin S and cytomycin (Otake et al., 1966b).



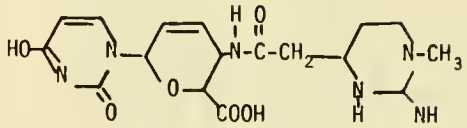
Blasticidin S (I)



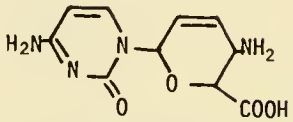
Deaminohydroxyblasticidin S (II)



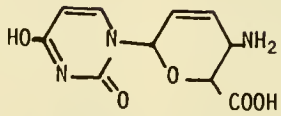
Cytomycin (III)



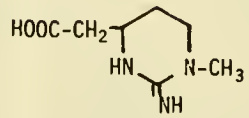
Deaminohydroxycytomycin (IV)



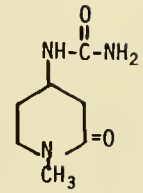
Uracinine (V)



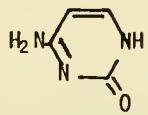
Cytosinine (VI)



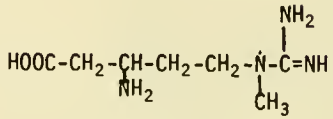
Pseudoblastidone (VII)



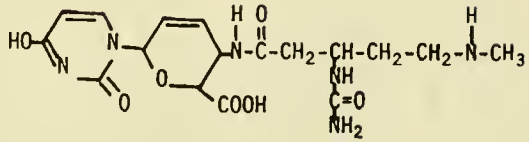
Blastidone (VIII)



Cytosine (IX)



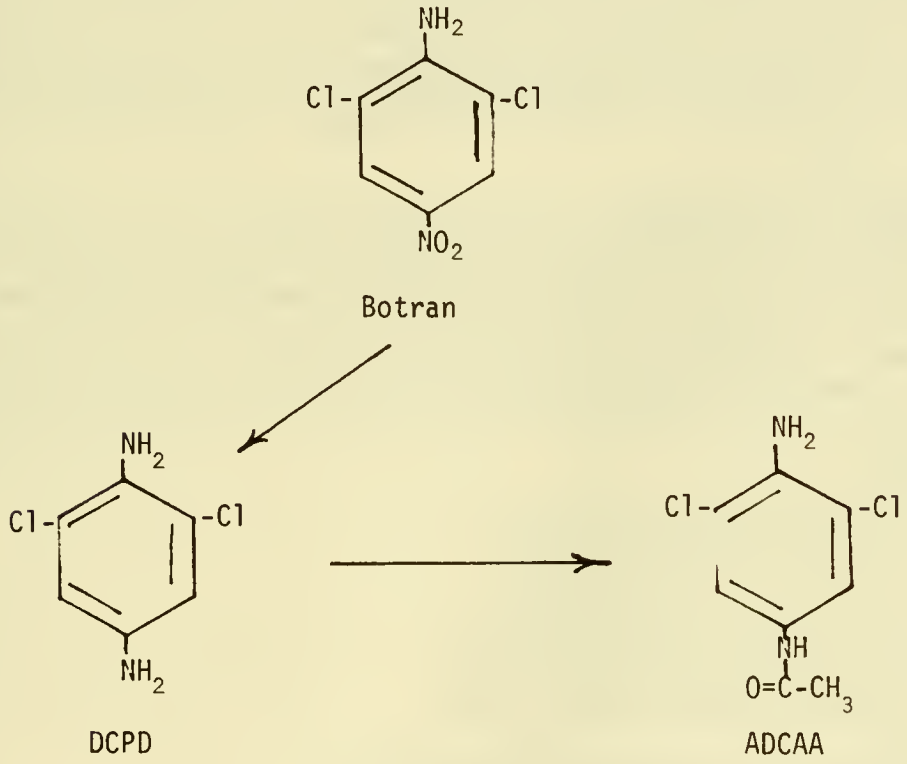
Blastidic Acid (X)



(XI)

BOTRAN (DCNA) [2,6-Dichloro-4-nitroaniline]

Incubation of botran with cultures of bacteria indicated that metabolism proceeded via 2,6-dichloro-p-phenylenediamine (DCPD) to 4-amino-3,5-dichloroacetanilide (ADCAA) (Van Alfen and Kosuge, 1974).



BPBSMC (Chevron RE 11775) [3-(2-Butylphenyl)N-benezenesulfenyl N-methylcarbamate]

BPMC (Chevron RE 5365) [3-(2-Butylphenyl)N-methylcarbamate]

Male albino rats (Sprague-Dawley strain) were orally administered ^{14}C-carbonyl and ^{14}C-butyl-labeled BPBSMC and BPMC in dimethyl sulfoxide. Urine was collected for 48 h and then analyzed. Quantitatively the major urinary products were identified in the order 2-(3-hydroxyphenyl) butan-2-ol (4), the butan-3-ol analog (5), and then the butan-1-ol analog (3). In addition to these, compounds 2 and 6 to 18, inclusive, were also observed and identified using syntheses and infrared, electron impact mass, chemical ionization and NMR spectra and TLC (Cheng and Casida, 1973).

When BPBSMC was exposed to either UV, sunlamp, or sunlight irradiation for one hour, six compounds formed. BPMC degraded only under UV irradiation to form six or more products of which only compounds 2 and 18 were identified. BPBSMC yielded compounds 2, 10, 18, 19, and 22. Under more rigorous conditions, compounds 2, 4, 10, 12, 18, 19, 20 and 21 were observed (Cheng and Casida, 1973).

1. BPBSMC
2. 3-(2-butyl)phenol
3. 2-(3-hydroxyphenyl)butan-1-ol
4. 2-(3-hydroxyphenyl)butan-2-ol
5. 2-(3-hydroxyphenyl)butan-3-ol
6. 2-(3-hydroxyphenyl)butan-4-ol
7. 2-(3-hydroxyphenyl)butan-3-one
8. 2-(3-hydroxyphenyl)butanoic acid
9. 3-(3-hydroxyphenyl)butanoic acid
10. BPMC
11. 3-(2-butan-1-ol)N-methylphenylcarbamate
12. 3-(2-butan-2-ol)N-methylphenylcarbamate
13. 3-(2-butan-3-ol)N-methylphenylcarbamate
14. 3-(2-butan-4-ol)N-methylphenylcarbamate
15. 3-(2-butan-3-one)N-methylphenylcarbamate
16. 2-(3-N-methylcarbamoylphenyl)butanoic acid
17. 3-(3-N-methylcarbamoylphenyl)butanoic acid
18. 3-butyl N-hydroxymethylphenylcarbamate
19. 3-(2-butyl)phenylcarbamate
20. 3-(2-butyl)N-benzenesulfinyl N-methylphenylcarbamate
21. 3-(2-butyl)N-benzenesulfonyl N-methylphenylcarbamate

BROMOPROPYLATE (Acarol, Phenisobromolate, Neoron, Isopropyl 4,4^{1}-dibromobenzilate) [Isopropyl 2-(4,4^{1}-dichbromophenyl)-2-hydroxyacetate]

CHLOROPROPYLATE (Isopropyl 4,4^{1}-dichlorobenzilate) [Isopropyl 2-(4,4^{1}-dichlorophenyl)-2-hydroxyacetate]

When chloropropylate was fed to a cow, the major route of elimination was via urine (>80% of total dose). About 28% of the material was identified as 4,4^{1}-dichlorobenzilic acid and 55% as conjugates, not further identified. Chloropropylate was stable (up to 7 h) in rumen fluid but decomposed in 10,000xg supernatant fraction of beef liver (St. John and Lisk, 1973).

When exposed to bromopropylate, spider mites (Tetranychus urticae Kock) and house flies (Musca domestica L.) metabolized this material to the bromine analogs of benzilic acid, benzhydrol, benzophenone, and benzoic acid (Al-Rubae and Knowles, 1972).

Spider mites and house flies also metabolized chloropropylate to the corresponding chlorine-containing analogs of benzilic acid, benzhydrol, benzophenone, and benzoic acid (Al-Rubae and Knowles, 1972).

BROMOXYNIL [3,5-Dibromo-4-hydroxybenzonitrile]

IOXYNIL [3,5-Diiodo-4-hydroxybenzonitrile]

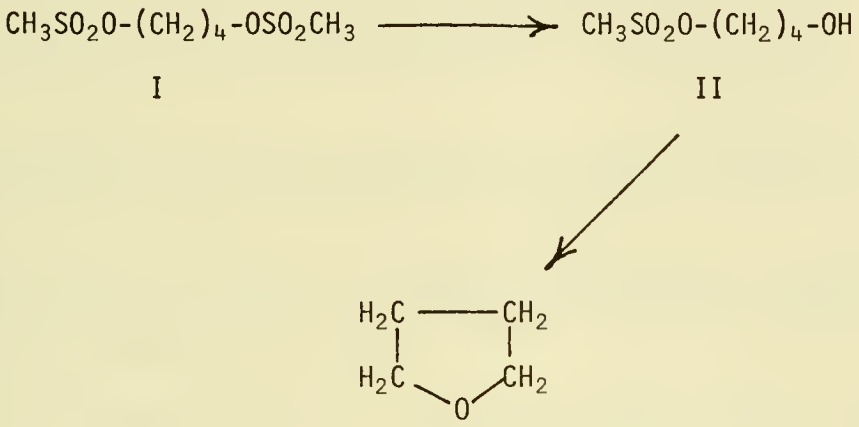
Labeled bromoxynil was applied as the octanoate to wheat (Triticum vulgare var. Kloka). Under outdoor conditions, when ring-labeling was used, 88% of the radioactivity was gone in 28 days. Using ^{14}C-cyano labeling and ^{14}C-ring labeling, the studies indicated elimination of label occurred more rapidly with the former and that metabolic attack occurred on the cyano group (Buckland et al., 1973a). After application to leaves of wheat seedlings, bromoxynil octanoate was initially hydrolyzed. This was followed by hydrolysis of the cyano group to the amide and acid and decarboxylation; replacement of bromine by hydroxy; and replacement of bromine by hydrogen (Buckland et al., 1973b).

When exposed to a flexibacterium, strain BR4, bromoxynil was rapidly degraded. After five weeks, only 5% of the herbicide remained. The benzamide and benzoic acid analogs were identified. A third metabolite was not identified (Smith and Cullimore, 1974). In other studies, when the octanoate ester of bromoxynil was applied to soils, 80% of ^{14}C-label in the cyano group and as much as 63% of ^{14}C-ring label were liberated as carbon dioxide. Small amounts of benzamide and benzoic acid analogs were detected (Collins, 1973). At 25C, 50% of bromoxynil applied to Regina heavy clay was degraded in 2 weeks. The amide and acid were detected (Smith, 1971).

Ioxynil was degraded in a clay loam with high organic matter content. Most of the ^{14}C-label, both cyano and ring, was recovered as $^{14}CO_2$. Mercuric chloride (10^{-5}M) and p-chloromercuribenzoate ($5x10^{-5}$M) inhibited production of $^{14}CO_2$. Ferricyanide was slightly inhibitory at 10^{-4}M. The benzamide and benzoic acid analogs were identified as metabolites (Hsu and Camper, 1975).

BUSULFAN [1,4-Butanediol di(methylsulfonate)]

The hydrolysis of busulfan (I) proceeded through the unstable 4-(methylsulfonate)butanol (II) to the cyclic tetrahydrofuran (III). At pH 3 and 7.4 and 37C, the cyclization reaction was determined to be first order with a half-life of 12 min (Feit and Rastrup-Andersen, 1973).



I II III

C-2307 [O,O-Dimethyl O-3-(N-methoxy-N-methyl-cis-crotonamide) phosphate]

Rats were treated with ^{32}P- and ^{14}C-labeled C-2307. With both labels, N-dealkylation occurred to produce the unsubstituted amide derivative. An intermediate, thought to be the N-hydroxy-N-methyl analog, was detected (Bosik, 1971).

CAPTAN [N-Trichloromethylthio)-cyclohex-4-ene-1,2-dicarboximide]

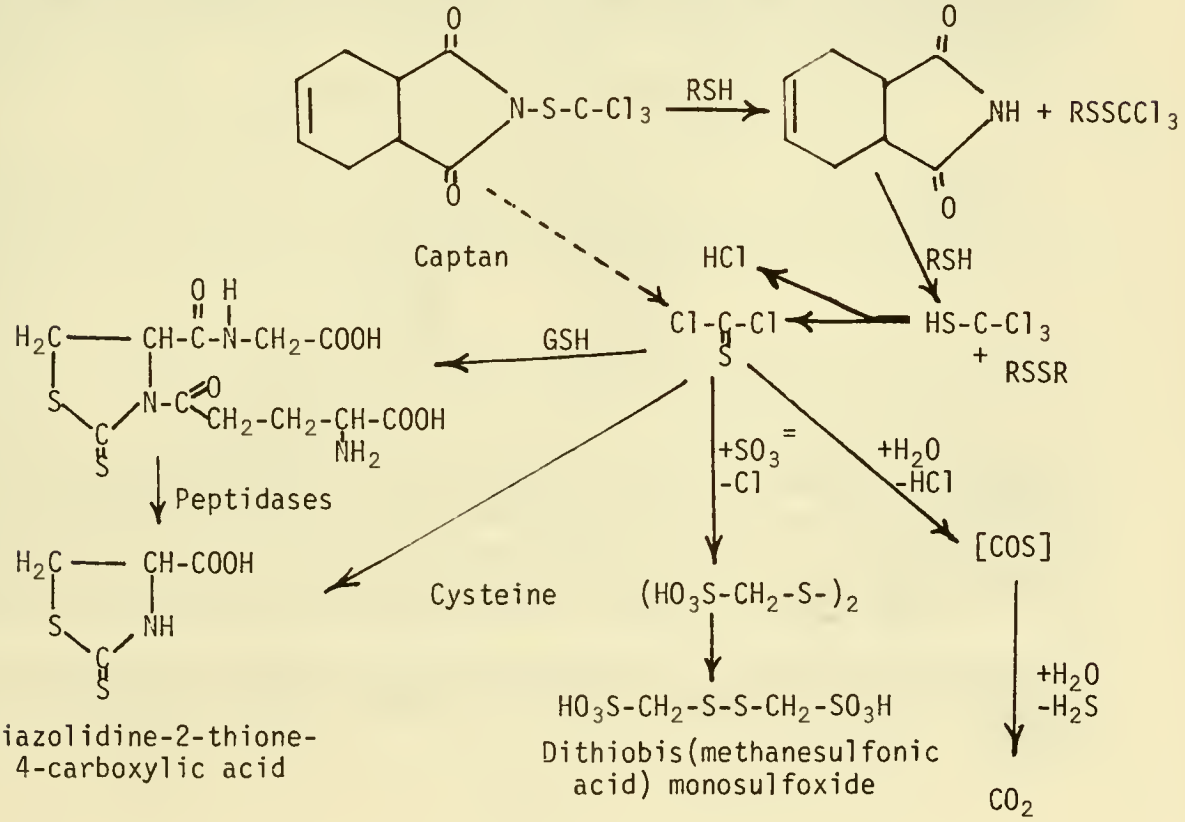
FOLPET (Phaltan) [N-(trichloromethylthio)phthalimide]

^{14}C-Captan, orally administered to rats, was rapidly metabolized. Urinary metabolites four days after oral dosing consisted of thiazolidine-2-thione-4-carboxylic acid (18.6%), dithiobis (methanesulfonic acid) (54%) and its disulfide monoxide (13.8%). After ip administration, the latter two metabolites were not seen. T. viride and R. solani degraded captan by a pathway different than in rats (DeBaun et al., 1974a and b).

Captan was degraded in wort. Tetrahydropthalimide was detected chromatographically and HCl was inferred from the pH change. CS_2 and H_2S were not observed (Davidek et al., 1973).

Hydrolysis of captan in water was pH independent in the range 2-6 and exhibited a half-life of less than one day at 27C. Products of hydrolysis indentified were 4-cyclohexene-1,2-dicarboximide, sulfur and chloride (Wolfe et al., 1974).

Folpet was degraded in wort with formation of phthalimide. HCl was detected but CS_2 and H_2S were not observed (Davidek et al., 1973).



CARBARYL (Sevin) [1-Naphthyl N-methylcarbamate]

Selected human tissues were incubated with carbaryl labeled in the ring or in the N-methyl group. Five of the 11 metabolites were identified.

	Male			Female				
	Li	K	Lu	Li	P	V	U	UL
Dihydro-dihydroxycarbaryl glucuronide	+	+	+	+				
Hydroxycarbaryl glucuronide	+			+		+		
Napthylglucuronide	+	+	+	+		+		tr
Hydroxycarbaryl sulfate	+			+		+		
Naphthyl sulfate	+		+	+	+	+	tr	tr

Li = Liver
K = Kidney
Lu = Lung
P = Placenta
V = Vaginal mucosa
U = Uterus
UL = Uterine Leiomyoma

(Chin et al., 1974)

Discrepancies between toxic level of carbaryl in various studies were the result of varying absorption associated with the mode of administration and vehicle (Pekas and Giles, 1974).

Absorption from perfused swine intestinal loops

	% Absorption in 1 h
Baygon	55-56
Carbaryl	67-69
Carbyne	66-71
Mobam	64-70
Zectran	66

(Pekas, 1974)

Studies have shown that some carbaryl can be absorbed intact from the stomach of a fasted rat (Casper et al., 1973). The glucuronidation of 1-naphthol by intestine was also observed (Bock and Winne, 1975). When injected into cats, 1-naphthol was excreted in urine almost entirely as sulfate conjugates. When injected in pigs, 1-naphthol was excreted as the glucuronide and sulfate in the ratio of 2:1 (Capel et al., 1974).

Treatment of rats with 1-naphthyl-glucoside-^{14}C showed that hydrolysis preceded formation of sulfate and glucuronide formation (Dorough et al., 1974).

After 72 h of incubation with HEL (human embryo lung) cell cultures, carbaryl was almost completely altered but not to CO_2. The major metabolite was 1-naphthol. Other metabolites included 4-hydroxy-, 5-hydroxy-, 1,4-dihydroxy-, and 1,5-dihydroxy-carbaryl and 5,6-dihydro-5,6-dihydroxycarbaryl. Other unidentified more polar metabolites were also observed. After acid hydrolysis of the aqueous extraction phase, three compounds freed from conjugates were identified as 4-hydroxy-carbaryl, 1,4-naphthalenediol, and 5,6-dihydro-5,6-dihydroxycarbaryl. Carbaryl was also incubated with sonicated HEL cells and cofactors for 3 h. About 90% of the carbaryl was converted to ether soluble metabolites. Cochromatography revealed five spots which were identified as 1-naphthol, carbaryl, 4- and 5-hydroxycarbaryl and 5,6-dihydro-5,6-dihydroxycarbaryl. The controls also contained 1-naphthol (Lin et al., 1975).

Incubation of rat enzyme preparations with carbaryl indicated the formation from carbaryl of CO_2 and three unidentified hydroxy derivatives, free and conjugated (Palut et al., 1970).

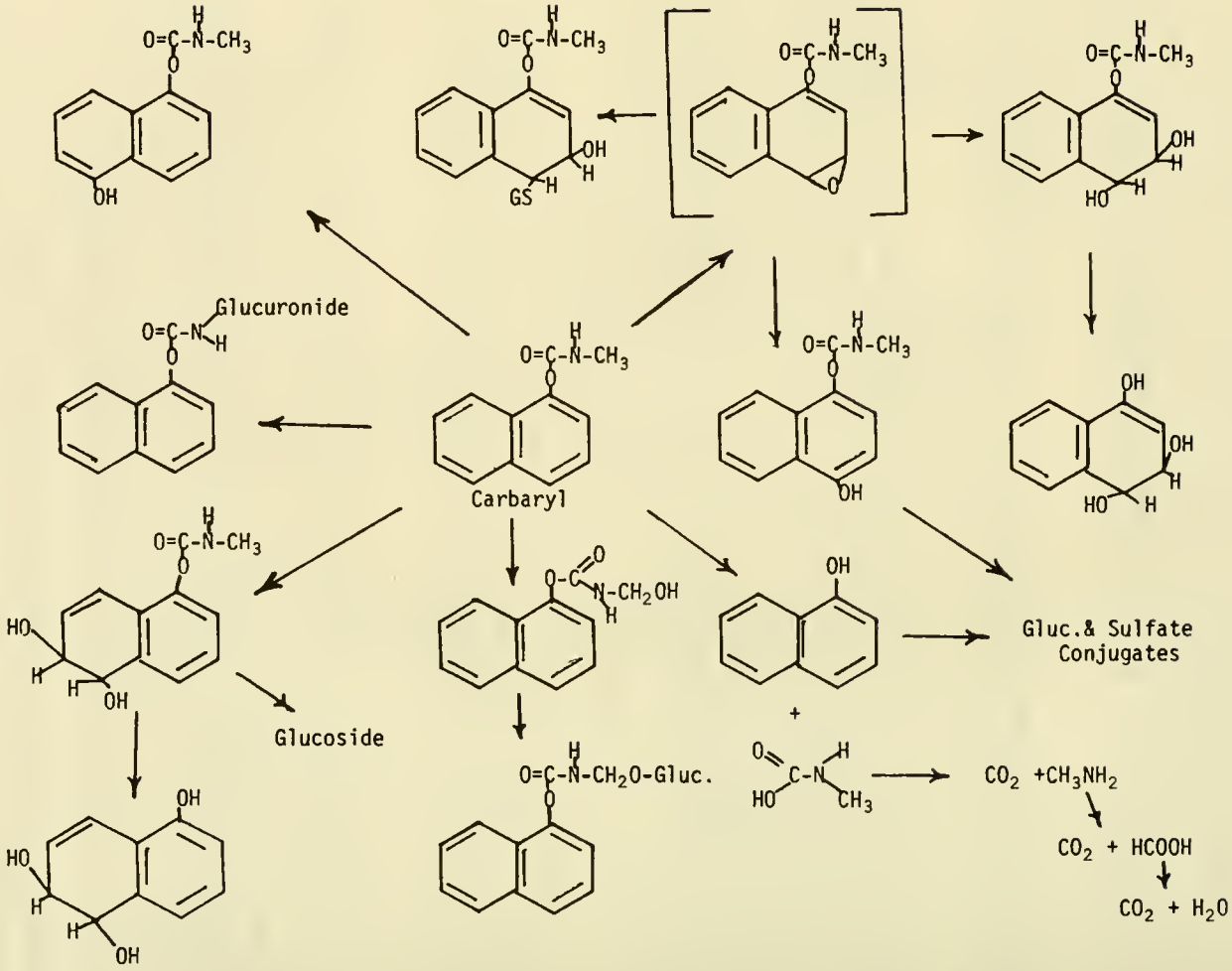
In vivo resistance varied substantially with age and sex in the sarcophagids (*S. bullata* Parker, *S. crassipalpis* Macquart, *S. argyrostoma*) and there was a corresponding variation in *in vitro* ring hydroxylation of carbaryl. In *Phornia regina* (Meigen), carbaryl resistance and ring hydroxylation also increased with age but N-demethylation remained constant. *In vitro* ring hydroxylation was lower and N-demethylation was higher in *Musca autumnalis* than in the sarcophagids or *Phormia*. Optimal incubation was at 30C and pH 7.3 for ring hydroxylation and N-demethylation. The I_{50} values of carbaryl to brain cholinesterase ranged from 1.2×10^{-7} to 6.6×10^{-7} M (Brattsten, 1972).

Houseflies were allowed to feed on 1-naphthol in milk plus sucrose. Ionophoretic studies indicated the presence of a sulfate, glucoside, phosphate and glucoside phosphate conjugate. Blowflies (*Lucilia sericata*) and grass grubs (*Costelytra zealandica*) behaved in a similar manner (Heenan and Smith, 1974).

Alfalfa leafcutting bees (*Megachile pacifica*) were exposed to carbaryl. In the water-soluble fraction, five unidentified metabolites were observed. The organosoluble fraction contained two unidentified metabolites as well as 5,6-dihydro-5,6-dihydroxycarbaryl, N-hydroxymethylcarbaryl, 4-hydroxy- and 5-hydroxy-carbaryl, N-hydroxycarbaryl and 1-naphthol (Guirquis and Brindley, 1975).

Male and female *Periplaneta americana* metabolized injected ^{14}C-carbaryl to about the same extent as measured by $^{14}CO_2$ (Cocks, 1974).

In pond water, carbaryl rapidly hydrolyzed to 1-naphthol. One bacterium, possibly a flavobacterium, rapidly degraded 1-naphthol. Of


Glucuronide
Carbaryl
Glucoside
Gluc.& Sulfate Conjugates
CO_2 +CH_3NH_2
CO_2 + HCOOH
CO_2 + H_2O

three compounds observed, two were identified as a hydroxycinnamic acid and salicylic acid (Hughes, 1971). In other studies, bacterial isolates from river water were used. When ^{14}C-labeled 1-naphthol was used, $^{14}CO_2$ was observed, indicating rupture of the naphthyl ring. Also isolated and identified by IR, NMR and mass spectroscopy was 4-hydroxy-1-tetralone (Bollag et al., 1975).

Carbaryl and 1-naphthol are stable in weakly acid solutions. In basic solutions, 1-naphthol turns yellow and then amber. Photo-oxidation of 1-naphthol gave rise to 2-hydroxy-1,4-naphthoquinone. Hydrolysis studies with carbaryl indicated a difference between sea water and NaOH solution (Wauchope and Haque, 1973).

pH	°C	Obs. $T_{1/2}$	$k_2 \times 10^2$
10.0	12	99 min	0.7
10.0	25	20	3.4
10.0	35	8	9.0
9.8	25	27	4.3
9.5	25	58	3.8
9.2	25	116	3.8
9.0	25	173	4.0

(Wauchope and Haque, 1973)

(Using Sea Water)

8.0	3.5	1 mo.	0.08
8.0	17	4.8 days	1.0
8.0	20	3.5 days	1.4
8.0	28	1.0 day	4.6

(Karinen et al., 1967)

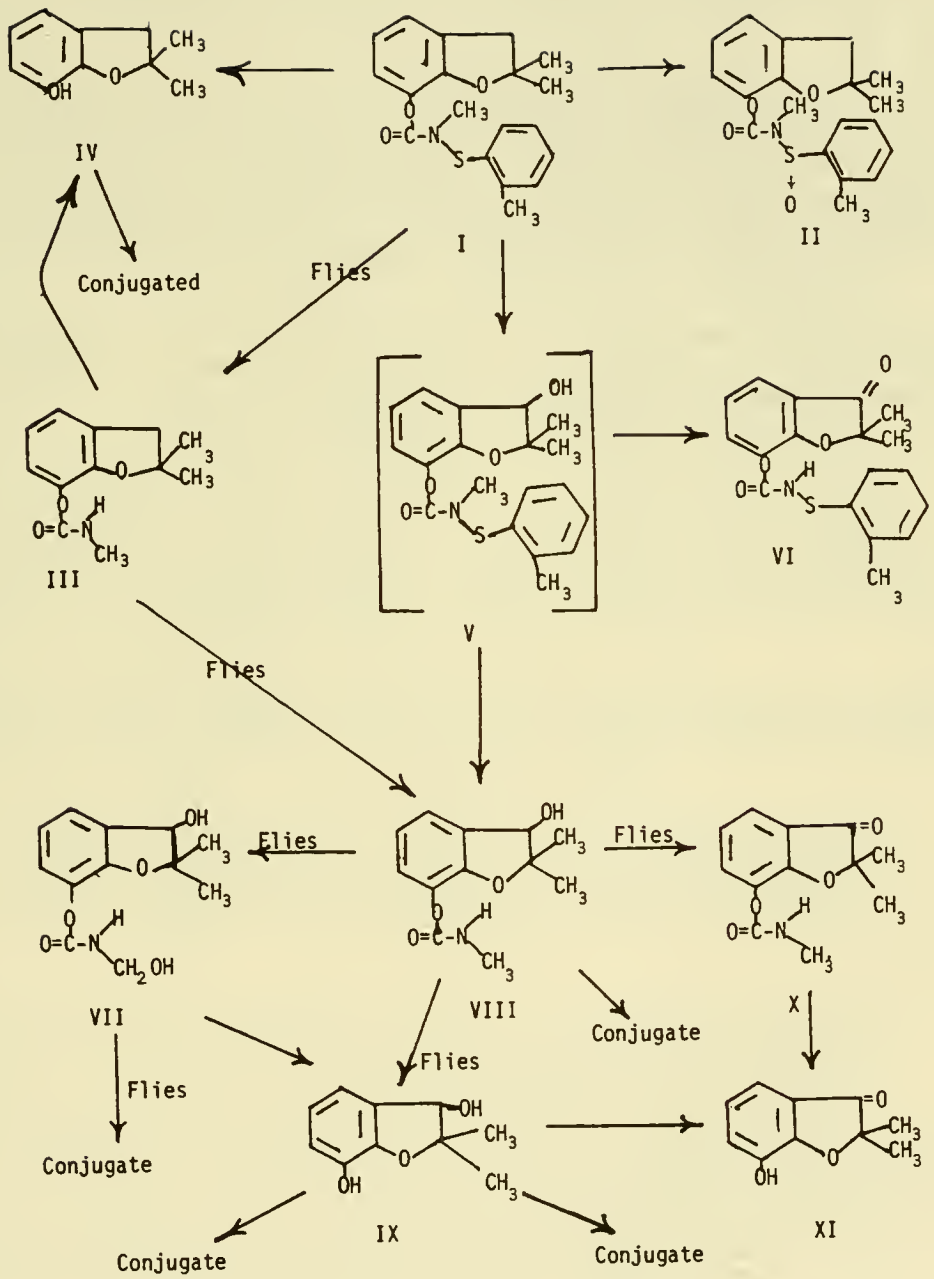
In vitro oxidation and N-demethylation of carbaryl was observed in the Udenfriend chemical hydroxylation system. The N-methyl oxidation product, 1-naphthyl N-hydroxymethylcarbamate, and the N-demethyl product, 1-naphthyl carbamate, were isolated and identified by mass and/or infrared spectrometry (Locke and Mayer, 1974).

CARBOFURAN (Furadan) [2,3-Dihydro-2,2-dimethyl-7-benzofuranyl-N-methyl-carbamate]

The metabolism of carbofuran in laying hens was studied with ring- and carboxyl-^{14}C labeling. Highest residues occurred in livers. About 80% of ring-^{14}C and about 20% of carboxyl-^{14}C appeared in feces. About 70% of the latter appeared as $^{14}CO_2$. The following metabolites were detected in combined and/or conjugated form: 2,3-dihydro-2,2-dimethyl-3-hydroxylbenzofuranyl N-hydroxymethylcarbamate (III); 3-hydroxy carbofuran (IV); 2,2-dihydro-2,2-dimethyl-3,7-dihydroxybenzofuran (VII); 3-ketocarbofuran (VIII); 2,3-dihydro-2,2-dimethyl-3-keto-7-hydroxbenzofuran (IX) (Hicks, 1970).

After injection of labeled carbofuran into earthworms (Lumbricus terrestris), five radioactive materials were excreted. In addition to carbofuran and two unidentified metabolites, 3-hydroxycarbofuran and 3-hydroxycarbofuran phenol were identified (Stenersen et al., 1973).

^{14}C-Carbofuran was applied to solutions in which the roots of 3-4 year old mugho pine were immersed. Radioactivity appeared in needles within 3 days and rose to 3.12% of the total radioactivity after 70 days. In addition to two unidentified compounds, carbofuran phenol and lesser amounts of 3-hydroxyfuran and 3-ketocarbofuran phenol were present. As the content of one compound, tentatively identified as N-hydroxymethyl-carbofuran, leveled off, the content of carbofuran phenol increased. Although 3-ketocarbofuran was not isolated in these studies, its transient occurrence was suggested by the increase of 3-ketocarbofuran phenol as the 3-hydroxycarbofuran content leveled off. All metabolites were found conjugated as well as free (Pree and Saunders, 1974).


IV
Conjugated
Flies
I
II
III
V
VI
Flies
Flies
Flies
VII
VIII
X
Flies
Conjugate
Conjugate
IX
XI
Conjugate
Conjugate

N-(2-Toluenesulfenyl)carbofuran [2,3-Dihydro-2,2-dimethyl-7-benzofuranyl-N-methyl-N-(2-toluenesulfenyl)carbamate]

N-(2-Toluenesulfenyl)carbofuran (I) was rapidly metabolized by white mice. Most of the administered radioactivity appeared in urine within 24 h. Most identities were confirmed by cochromatography in at least four different solvent systems. In the urine were: N-(2-toluenesulfenyl)-3-ketocarbofuran (VI); 3-hydroxycarbofuran (VIII); 3-ketocarbofuran (X); 3-hydroxy-N-hydroxymethylcarbofuran (VII); carbofuran phenol (IV); 3-hydroxycarbofuran phenol (IX); 3-ketocarbofuran phenol (XI). In feces, there was the sulfinyl analog of carbofuran (II); carbofuran (III); and compound VI. $^{14}CO_2$ was also formed (Black et al., 1973).

When compound I was applied to flies, carbofuran and metabolites VII and VIII were observed free. In addition, metabolites VII, VIII and X were found as conjugates (Black et al., 1973).

CARBOXIN (Vitavax) [2,3-Dihydro-5-carboxanilido-6-methyl-1,4-oxathiin]

OXYCARBOXIN (Plantvax) [2,3-Dihydro-5-carboxanilido-6-methyl-1,4-oxathiin-4,4-dioxide]

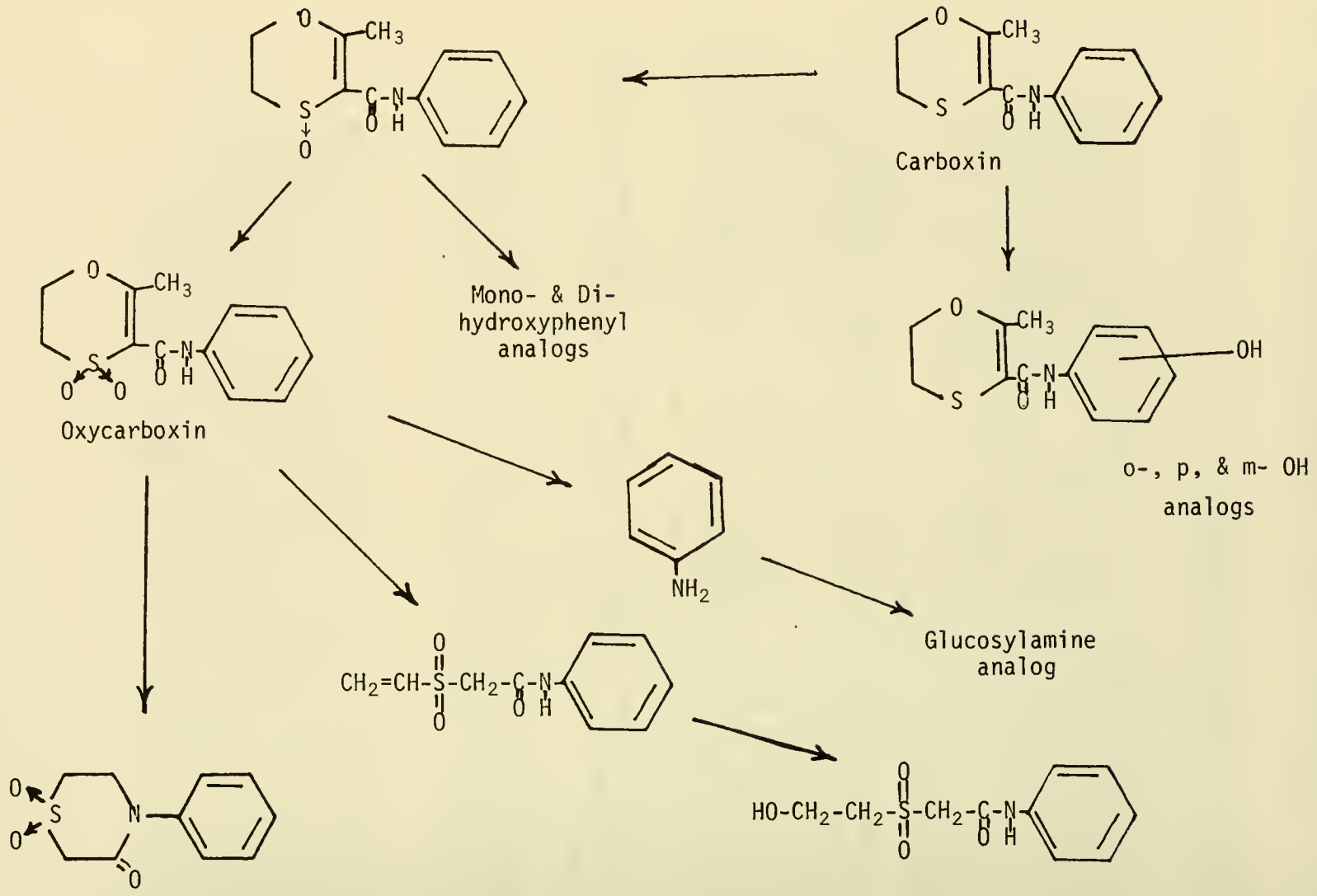
Carboxin was administered to female rabbits (New Zealand White strain) and female rats (Wistar strain) by stomach tube. Urine and feces were collected and analyzed. In addition to unchanged carboxin, p- and o-hydroxy derivatives were observed. Trace amounts of the m-hydroxy analog were also found occasionally. Three other minor metabolites observed appeared to be a hydroxylated sulfoxide, a dihydroxy and a dihydroxy sulfoxide of carboxin. The hydroxy metabolites were excreted largely as glucuronides in both species (Waring, 1973).

When carboxin was applied to bean plants (Phaseolus vulgaris L.) in nutrient solutions, roots readily oxidized this fungicide to the sulfoxide. The 4,4-dioxide, oxycarboxin, was detectable in roots and unifoliate leaves for 21 days after application. Most of the fungicide residue in the roots was in the form of acetone-insoluble material (Snel and Edgington, 1970). Other studies have indicated that carboxin is hydrolyzed with formation of aniline which is bound probably as a glucosylamine (Newby and Tweedy, 1970).

Barley plants formed carboxin-lignin complexes in the leaves. These were liberated by hot dimethyl sulfoxide and identified as carboxin (30%) and its sulfoxide (70%) (Chin et al., 1973).

Rhizopus japonicus, a synthetic glucose medium, converted carboxin into the corresponding sulfoxide and sulfone. Under anaerobic conditions the sulfoxide and a substituted anilide (not further identified) were observed. No sulfone was observed (Wallnofer et al., 1972).

Carboxin-treated barley seed was grown in vermiculite saturated with distilled water. Plants were harvested at intervals over a 21-day period and analyzed. Paper chromatography, GLC and mass spectrometry were used to identify metabolites. Young shoots contained carboxin, p-hydroxyphenyl analog and unidentified dihydroxyl derivatives. Mature plants contained carboxin, p-hydroxyphenyl derivative, polymeric material and traces of the sulfoxide. Hydrolysis of the lignin produced material that gave a chromatographic band coincident with the p-hydroxyphenyl (Briggs et al., 1974).


Carboxin
Mono- & Di-
hydroxyphenyl
analogs
Oxycarboxin
o-, p, & m- OH
analogs
Glucosylamine
analog

CDAA (Randox) [2-Chloro-N,N-diallylacetamide]

CDAA rapidly decomposed in plants. In rumen fluid of cows, it was stable for 24 h. When incubated with beef liver 10,000xg supernatant, CDAA was not detectable after 30 min. No metabolites were identified (St. John and Lisk, 1974).

When ^{14}C-CDAA administered as a single dose to rats, 86% was excreted in the urine during the first 48 h; 16%, in feces. About 89% of the urinary ^{14}C was the mercapturic acid of CDAA. Studies also showed that CDAA reacted non-enzymatically with glutathione (Lamoureux and Davison, 1975).

2-CEPA [2-Chloroethylphosphonic acid]

In leaf and stem tissue of Hevea brasiliensis, 2-CEPA was converted into 13 and 20 compounds, respectively. One of the compounds obtained from stem and leaf was identified by TLC as 2-hydroxyethylphosphonic acid (Archer et al., 1973).

This compound also formed in small amounts when 2-CEPA was incubated for several days in buffer solutions at room temperature, When heated in alkali, ethylene and non-volatile material, identified as 2-hydroxyethylphosphonic acid by autoradiography and TLC, was formed (Audley and Archer, 1973).

CHLORAL HYDRATE

Within a few days after application to soil, chloral hydrate was oxidized to trichloroacetic acid (TCA). TCA degraded with evolution of CO_2. Some formaldehyde was also detected at the beginning of the decomposition (Schutte and Stephan, 1969).

Chlordane and Related Compounds

α - (or cis-) chlordane

1-exo,2-exo,4,5,6,7,8,8-octachloro-2,3,3a,4,7,7a-hexahydro-4,7-methanoindene

γ- (or trans-) chlordane

1-exo,2-endo,4,5,6,7,8,8-octachloro-2,3,3a,4,7,7a-hexahydro-4,7-methanoindene

Chlordene

4,5,6,7,8,8-hexachloro-3a,4,7,7a-tetrahydro-4,7-methanoindene

Chlordene epoxide

4,5,6,7,8,8-hexachloro-exo-(cis)-2,3-epoxy-3a,4,7,7a-tetrahydro-4,7-methanoindene [also an endo-(trans)-2,3-epoxy- isomer].

Oxychlordane

1-exo,2-endo,4,5,6,7,8,8-octachloro-2,3-exo-epoxy-2,3,3a,4,7,7a-hexahydro-4,7-methanoindene

Heptachlor

1,4,5,6,7,8,8-heptachloro-3a,4,7,7a-tetrahydro-4,7-methanoindene

Human adipose tissue specimens routinely collected from postmortem examinations and therapeutic surgery procedures were analyzed for a series of organochlorine pesticide residues. Oxychlordane was found in 21 of 27 specimens and ranged from 0.03 to 0.40 ppm (Biros and Enos, 1973).

HCS-3260-^{14}C (a 3:1 cis-chlordane and trans-chlordane) was administered to rats. After a single oral dose, over 90% was eliminated in 7 days. Feces was the main route of excretion. After feeding HCS-3260-^{14}C to rats for 56 days, cis- and trans-chlordane, oxychlordane, dichlorochlordene and seven unidentified compounds were observed. Oxychlordane was observed after single doses of either cis- or trans-chlordane. After feeding a male rabbit HCS-3260-^{14}C for 2 days, in addition to cis- and trans-chlordane, oxychlordane and dichlorochlordane were observed in liver and kidney. Seven unidentified compounds were also found in urine and feces (Barnett and Dorough, 1974).

Sugar beets were grown in soil treated with HCS-3260 or chlordane. Sugar beet pulp processed from these plants was fed to cows. In milk fat, there were detectable amounts of α- and γ-chlordane and oxychlordane. The major product in the milk fat was identified as oxychlordane by mass spectrum. Similar residues were found in the fat (Dorough and Hemken, 1973).

The hepatic mixed function oxidase (MFO) of several fish was investigated. When chlordene was used as substrate, hydroxylation preceded epoxidation. With preparations from the Kissing Gourami (Helostoma sp.) and pigeon, 1-hydroxy-2,3-epoxychlordene formed after the epoxide. V_{max} and K_m were also determined (Garretto and Khan, 1975; Runnels and Khan, 1973; Stanton and Khan, 1973).

Chlordene substrate	MFO V_{max}	MFO K_m
Epoxidation		
Kissing Gourami	0.26	15.59
Bluegill (Young)	0.18	15.00
Hydroxylation		
Kissing Gourami	0.47	11.06
Bluegill (Young)	1.25	13.00
Epox.-Hydrox.	0.08	10.90

	MFO V_{max}		
	Substrate		
	Chlordene epoxide	Hydroxychlordene	Hydrox.-Epox. Chlordene
Kissing Gourami	0.16	0.32	0.09
Bluegill fry	0.16	1.05	----
Trout	0.33	0.38	----
Bluegill fry	0.13	0.99	----
Pigeon	0.26	0.44	0.10
Mouse	0.23	1.13	----

A 3-day aquatic system was used to evaluate uptake and biotransformation of chlordene, heptachlor and heptachlor epoxide. Results are tabulated. Water samples indicated the rapid formation (~24 h) of heptachlor epoxide and 1-hydroxchlordene and its epoxide.

Compd. Used	Metabolite	Oedogonium (Snail)	Physa (Snail)	Culex (Mosquito)	Gambusia (Fish)
Chlordene	Chlordene epoxide	+	+	+	+
	1-Hydroxychlordene	+	+	+	+
	1-Hydroxychlordene epox.	+	+	+	+
	Unknown I		+		+
	Unknown II		+		+
	Polar	+	+	+	+
Heptachlor	Heptachlor epoxide	+	+	+	+
	1-Hydroxychlordene		+	+	+
	1-Hydroxychlordene epox.		+	+	+
	Unknown I		+	+	
	Unknown II		+	+	
	Unknown III		+	+	
	Unknown IV		+	+	+
	Unknown V		+	+	
	Polar	+	+	+	+
Heptachlor Epox.	1-Hydroxychlordene epox.	+	+	+	+
	Polar	+	+	+	+

Hexachlorocyclopentadiene was also evaluated and found in all phases of the system. In addition, four unidentified compounds and polar material were also found in all phases (Lu et al., 1975).

Other studies with the salt marsh caterpillar, Estigmane acrea, and sheep liver microsomes were summarized in the following table.

Compound Used	Metabolite	Salt Marsh Caterpillar	Sheep Liver Microsomes
Chlordene	Chlordene epoxide	+	+
	1-Hydroxychlordene	+	+
	1-Hydroxychlordene epox.	+	+
	Unknown I	+	
	Unknown II		+
	Unknown III	+	+
	Polar	+	+
Heptachlor	Heptachlor epoxide	+	+
	1-Hydroxychlordene	+	+
	1-Hydroxychlordene epox.	+	+
	Unknown I	+(feces only)	+
	Unknown II	+(feces only)	
	Polar	+	+
Heptachlor Epoxide	Polar	+	+

(Lu et al., 1975)

In less than one day, heptachlor was converted to heptachlor epoxide in skim milk and trypticase soy broth, with or without the presence of the test bacteria (Collins, 1969).

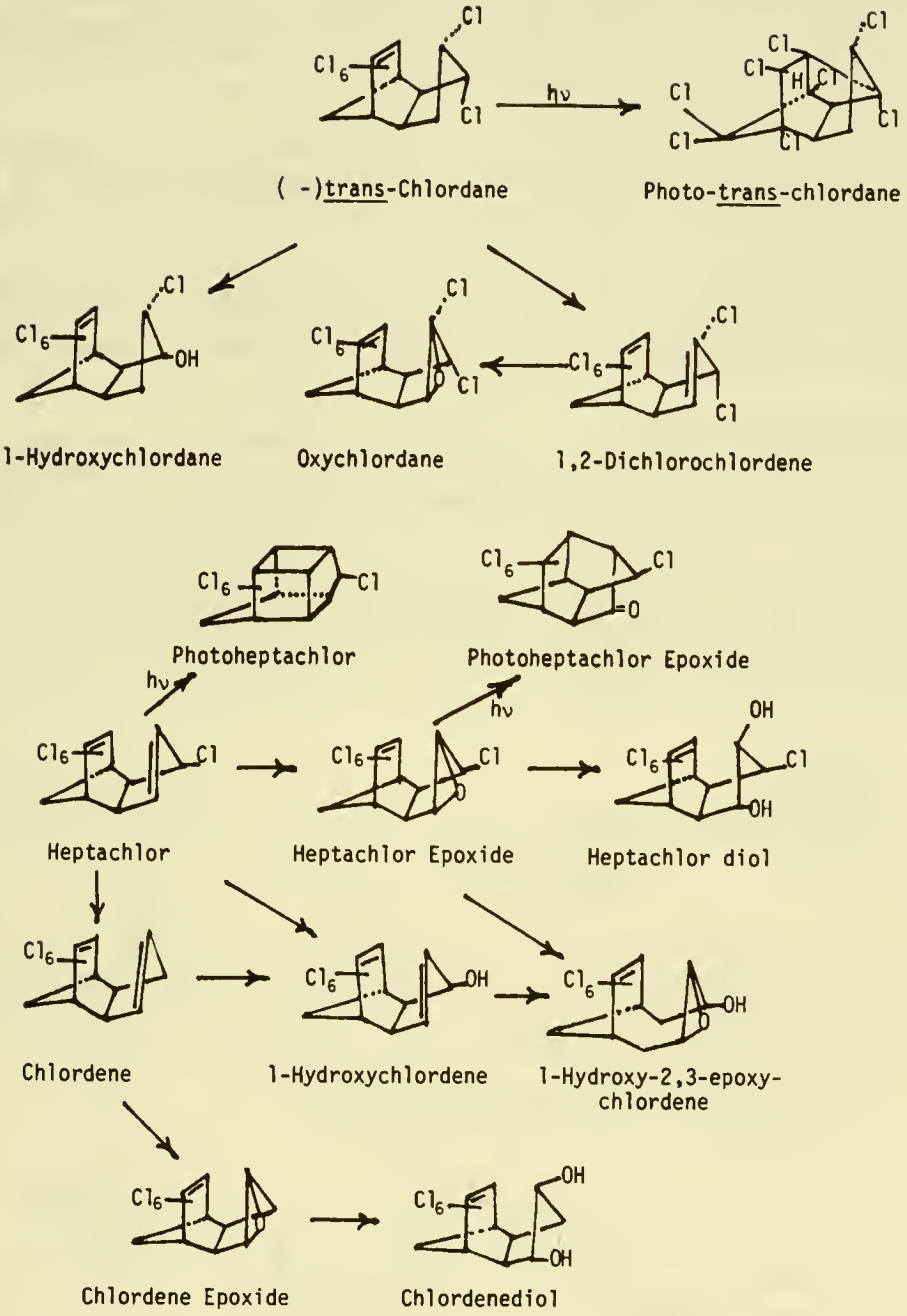
Soil was treated with high purity chlordane ($\geq$95% α plus β chlordane; cis/trans $\sim$ 3:1). Alfalfa was grown on the treated soil. It was sampled 2 months after treatment; cut back at 3 months; and then sampled at 4 months and again at 1 year. Analysis by GLC indicated the presence of α-, γ-, oxy- and photo-cis-chlordane. Oxychlordane comprised 9 to 13% of the total residues in the alfalfa but was not detected in the soils. The intermediate compound, 1,2-dichloro-chlordene, was found at 0.011 ppm in the alfalfa from plots treated at 10 lb a.i./A and in trace quantities at 5 lb a.i./A. Photo-cis-chlordane accounted for 9 to 16% of the total residues in the alfalfa and was also found in the soil (Wilson and Oloffs, 1973).

Ultraviolet irradiation of cis- and trans-chlordane in acetone produced 3 products. The half-caged analog of cis-chlordane, photo-cis-chlordane, was obtained in high yield. Two products were formed from trans-chlordane. One was identified as photo-trans-chlordane. The other was not identified, but a molecular composition of $C_{10}H_6Cl_6$ appears likely (Onsuka and Comba, 1975). Irradiation of β-chlordane in acetone produced II and III. Irradiation of IV gave VI (Parlar and Korte, 1973).

A mixture of oxychlordane and xanthone was streaked on the surface of silica gel chromatoplates and then exposed to sunlight. Two photo-isomers were formed. Several structures were possible for each compound and this has not been resolved (Ivie, 1973).

Chlordene was adsorbed on silica gel and then irradiated with ultraviolet light at $\lambda \geq 290$ and $\lambda \geq 230$nm. Differences observed were mainly quantitative. Chlordene required 1.5 h for 50% conversion of the starting materials. Products included: chlordene epoxide, photochlordene, 1-exo-hydroxy chlordene, ketochlordene, an unidentified compound and polar and polymer material. When quartz was used, instead of pyrex, heptachlor was also obtained (Gab et al., 1975).

The decomposition of heptachlor epoxide in KBr disks with exposure to ultraviolet radiation and sunlight was studied. The products obtained were identical to those obtained by exposure of solid heptachlor epoxide to sunlight and ultraviolet radiation (Graham et al., 1973).


(-)trans-Chlordane
Photo-trans-chlordane
1-Hydroxychlordane
Oxychlordane
1,2-Dichlorochlordene
Photoheptachlor
Photoheptachlor Epoxide
Heptachlor
Heptachlor Epoxide
Heptachlor diol
Chlordene
1-Hydroxychlordene
1-Hydroxy-2,3-epoxy-chlordene
Chlordene Epoxide
Chlordenediol

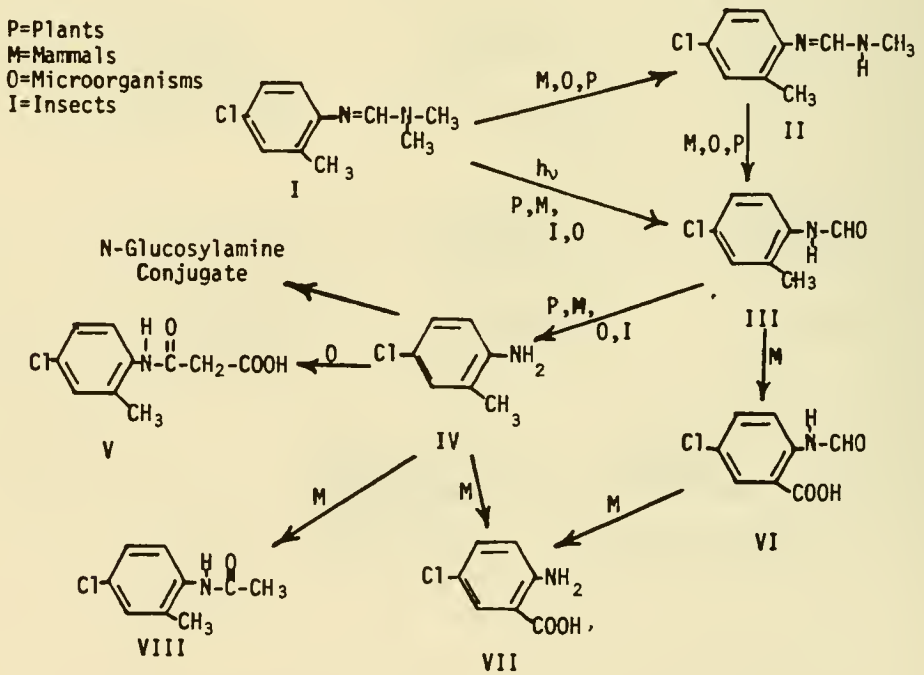
CHLORDIMEFORM (Galecron) [N-(4-Chloro-o-tolyl)-N'-N'-dimethylformamidine]

Chlordimeform (I) was quite susceptible to degradation by cultures of primary human embryonic lung cells. Dimethylation to N-demethyl-chlordimeform (II) preceded cleavage to N-formyl-4-chloro-o-toluidine (III). Three unidentified compounds were also found in addition to 4-chloro-o-toluidine (IV) (Lin et al., 1975b).

Aqueous solutions of ^{14}C-chlordimeform were administered by stomach tube to Sprague-Dawley rats. Thin-layer chromatography was used to separate the metabolites and IR and UV were employed for identification. Half of the dose was excreted in urine in 24 h; 87.1 and 95.4% in 96 h by males and females, respectively. In addition to I, compounds II, III and IV were found in urine. Five unidentified compounds were also present. In vitro studies with liver homogenates produced the same metabolites (Morikawa et al., 1975).

Absorption and metabolism of chlordimeform by the rice stem borer was slow. Analyses, however, indicated the formation of compounds II, III, IV and five unidentified compounds (Morikawa et al., 1975).

From cultures of mixed populations of soil microorganisms to which chlordimeform had been added, a new metabolite of the pesticide was isolated and identified as 4'-chloro-2'-methylmalonanilic acid. Confirmation of structure was obtained by synthesis and mas spectral data (Ross and Tweedy, 1973).



CCC-$^{14}CH_3$ was metabolized to choline in barley, wheat, tobacco and maize. Choline isolated from these plants contained 10-20% of the applied radioactivity. A small part of the radioactivity was also found in the betaine fraction. In Nicotiana rustica L., methyl groups of CCC-$^{14}CH_3$ were incorporated into the alkaloid nicotine; in Hordeum vulgare, into the alkaloid gramine (Stephan and Schutte, 1970). Radioactivity from 1,2-^{14}C-CCC was also found in the choline moiety of phosphatidyl choline in winter barley (Hordeum vulgare L. var. Dover) (Belzile and Willemot, 1972).

When applied to coastal bermudagrass (Cynodon dactylon L. Pers.), 1,2-^{14}C-CCC was metabolized; and, within 24 to 48 h after application, about 25% of the label was found distributed among choline, betaine hydrochloride, serine, ethanolamine, glucose and CO_2 (Ayeke, 1969).

Wheat seedlings were root-treated with 1,2-^{14}C-chlormequat. Translocation was rapid and choline was formed. The latter was metabolized via betaine, which was demethylated, to glycine and serine. These were then incorporated into the plant protein fractions. Some $^{14}CO_2$ was also formed (Dekhuijzen and Vonk, 1974).

CHLORODIOXIN

(See also Irgasan.)

Although these compounds are not pesticides, their presence as contaminants in phenol based pesticides and their potential hazard to the environment places them in a position of great interest. Consequently, these compounds have been included in this compilation.

When fed to rats, 2,3,7,8-tetrachloro-dibenzo-p-dioxin (TCDD) was stored primarily in the liver. Total retention was dose dependent and varied from 5.5 to 10.0 times daily intake between 14 and 42 days. At steady state, analyses indicated that retention would approximate 10.5 times daily intake. After removal of TCDD from the diet, the half-life for elimination was 12 and 15 days for males and females, respectively (Fries and Marrow, 1974 and 1975).

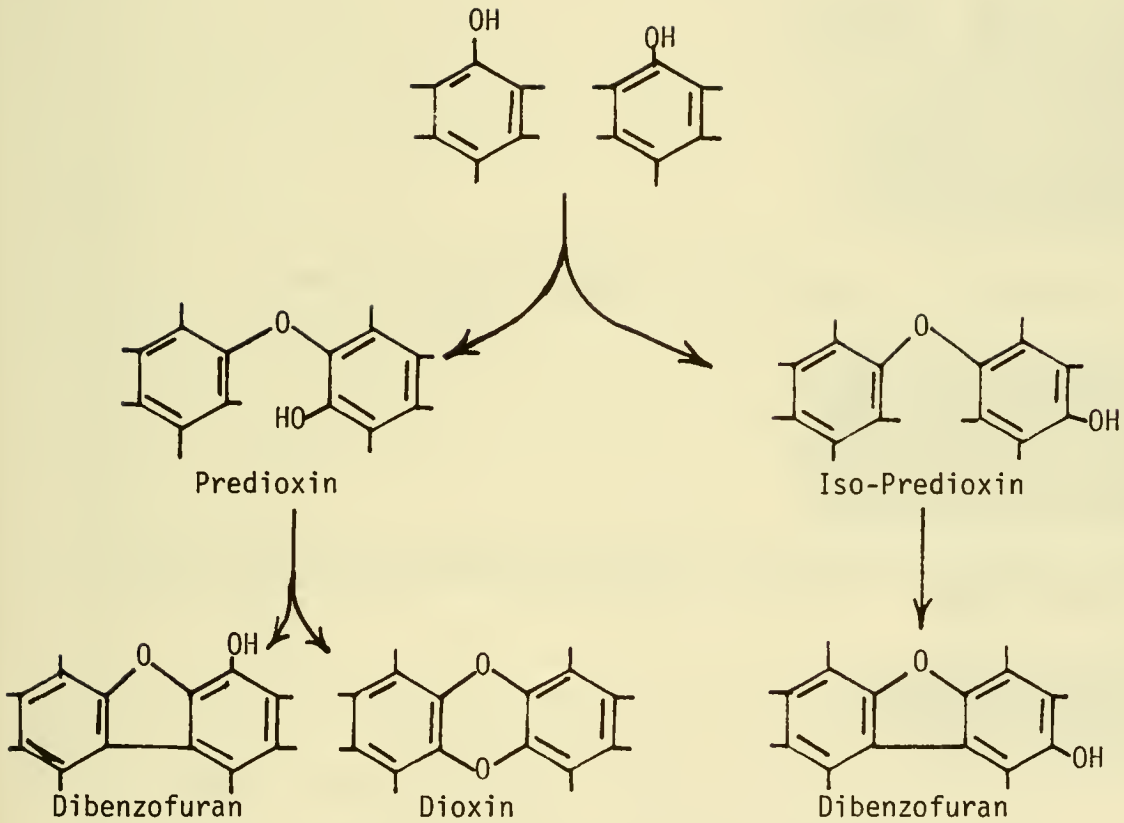
Thermal decomposition of 2,4,5-trichlorophenol gave rise to 2,3,7,8-TCDD (Milnes, 1971).

Irradiation of TCDD in methanol produced 2,3,7-trichloro-dibenzo-p-dioxin and in a dichloro analog. Octachlorodibenzo-p-dioxin yielded a series of dechlorinated dioxins (Plimmer et al., 1971).

Irradiation of photosensitized 2,4-dichlorophenol in water yielded dimeric materials primarily and traces of dechlorinated products. Using mass spectrometry, two tetrachlorophenoxyphenols and two tetrachlorodihydroxybiphenyls were detected. A trace of trichlorophenoxyphenol was also detected but there was no evidence of substituted dibenzo-p-dioxin. The major product was identified as 4,6-dichloro-2-(2,4-dichlorophenoxy)phenol. An unidentified isomer of this compound was also present. The presence of riboflavin and oxygen was necessary (Plimmer and Klingebiel, 1971).

2-Hydroxy nonachlorodiphenyl ether (pre-dioxin) was found as a contaminant in PCP. This material is thermally unstable and undergoes ring closure to form octachlorodioxin. An iso-predioxin was also found in a technical organic salt of PCP (Jensen and Renberg, 1972).

The persistence of 2,3,7,8-tetrachlorodibenzo-p-dioxin (TCDD) in Hagerstown and Lakeland (Md.) soils was found to be 56 and 63%, respectively, after one year. When 2,4-dichlorophenol or 2,4,5-trichlorophenol were added to soil, neither the 2,7-dichloro- nor the tetrachloro-dibenzo-p-dioxin was detected after 70 days. However, a polar metabolite of the dichloro analog was observed but not identified. Neither of these two dioxins was synthesized by microbial actions (Kearney et al., 1972).


OH
OH
O
HO
Predioxin
O
OH
Iso-Predioxin
OH
O
Dibenzofuran
O
O
Dioxin
O
OH
Dibenzofuran

CHLORONEB (Demosan, Tersan SP) [1,4-Dichloro-2,5-dimethoxybenzene]

After exposure to chloroneb, young bean plants absorbed the chloroneb and accumulated it mostly in roots and lower stem. Chloroneb was metabolized to 2,5-dichloro-4-methoxyphenol (DCMP) and then converted to the β-D-glucoside (Thorn, 1973).

Incubation of chloroneb, and DCMP indicated that methylation of the phenol, as well as demethylation of chloroneb, occurred. Twenty-three different microorganisms were studied. Eight organisms demethylated chloroneb and methylated DCMP:

Fusarium solani f. pisi
Fusarium solani f. phaseoli
Mucor ramannianus
Cephalosporium gramineum
Aspergillus fumigatus
Verticillium albo-otrum
Cephalosporium gregatum
Chaetomium globosum

Six organisms were capable only of demethylating chloroneb:

Sclerotinia sclerotiorum
Helminthosporium victoriae
Corynebacterium fascians
Stemphyllium sarcinaeforme
Cladosporium cucumerinum
Helminthosporium sativum

Three organisms were capable of methylating DCMP but did not degrade chloroneb to DCMP:

Trichoderma viride
Penicillium frequentans
Rhizoctonia solani

(Wiese and Vargas, 1973)

In other studies, however, mycelium of R. solani degraded chloroneb to DCMP, which was identified by IR, NMR, and mass spectrographic analyses. N. crassa metabolized chloroneb slowly to a compound thought to be an aglycoside (Hock, 1969).

CHLOROTHALONIL [2,4,5,6-Tetrachloroisophthalonitrile]

In cells of Saccharomyces pastorianus, chlorothalonil acted as an alkylating agent. Initial uptake resulted in the formation of glutathione derivatives. The fungicide also reacted with proteins (Tillman et al., 1973).

CHLORPROPHAM (CIPC, Isopropyl-m-chlorocarbanilate) [Isopropyl N-(3-chlorophenyl)carbamate]

PROPHAM (IPC, Isopropyl carbanilate) [Isopropyl N-phenylcarbamate]

After dosing a goat and chicken with labeled propham, analyses conducted showed the presence of the p-sulfate derivative in goat milk and goat and chicken carcasses. The glucuronide was also found in chicken carcass (Paulson and Jacobsen, 1974).

The main route of excretion of propham by a goat was via the urine. Small amounts appeared in milk and feces. After administration of phenyl-^{14}C-labeled propham to a goat, 13 labeled metabolites were obtained and purified. Identification procedures included derivatization and chromatography, mass spectrum analyses, enzyme hydrolysis, syntheses and IR spectrometry. The metabolites identified were:

1. glucuronic acid conjugate of 4-hydroxypropham
2. sulfate ester of 4-hydroxypropham
3. glucuronic acid conjugate of 4-hydroxyacetanilide
4. sulfate ester of 2-hydroxyaniline
5. conjugate of 2-hydroxyisopropyl 4-hydroxycarbanilate
6. sulfate ester of 2-hydroxypropham

[7. sulfate ester of 4-hydroxyacetanilide--not obtained from goats.]

Compounds 1 and 2 were obtained from dosed rats as well as goats. Compound 7 was obtained from rats only. All others appeared in both animals. Partial identification of four other metabolites from the goat indicated the presence of:

1. a 4-hydroxypropham conjugate
2. a 2-hydroxypropham conjugate
3. a p-aminophenol conjugate
4. a 3,4-dihydroxypropham conjugate

(Paulson et al., 1973)

In rats, the average biological half-life for IPC in internal organs was 5.0 h; in brain, muscle and fat tissue, 13.3 h. Tentative identification was made for the following urinary metabolites: 4-hydroxy-IPC; 1-OH-2-propyl-IPC; 1-carboxy-1-ethyl-IPC; 1,3-$(OH)_2$-2-propyl-IPC; and 4-hydroxy-IPC-sulfate. Several other metabolites were observed but not identified (Fang et al., 1974).

Rats and sheep were fed alfalfa which had been root-treated with propham-^{14}C. In sheep urine, the major labeled metabolite was 4-OH-IPC sulfate ester. Another metabolite was identified as the glucuronic acid conjugate of 4-OH-IPC. Co-chromatography, IR and mass spectral analyses were used for identification (Paulson et al., 1974).

Alfalfa was root-treated with CIPC. Analyses of the root and shoot tissues indicated the presence of two aglycones. After hydrolysis, 2-OH-CIPC was found in root and shoots, 5.1 and 51.3%, respectively. The 4-OH-CIPC was also found, 26.4 and 17.6% in shoots and roots, respectively. The nature of the conjugates was not determined. However, both aglycones were liberated by treatment with hesperidinase, glucuronidase or cellulase (Still and Mansager, 1974).

Alfalfa, which had been root-treated with chlorpropham-phenyl-^{14}C, contained chlorpropham and glycoside conjugates of 2-OH-CIPC and 4-OH-CIPC. This plant material was fed to rats and a sheep. Little difference was observed in metabolite patterns in urine and feces of the sheep and rats. Sulfate and glucuronic acid conjugates were present in urine from both rat and sheep. The 4-OH-CIPC and 2-OH-CIPC conjugates were most abundant. The 2-OH-CIPC sulfate appeared in sheep urine. Glucuronide or sulfate conjugates of 2-OH-5-chloroacetanilide and 4-OH-3-chloroacetanilide were found in rat urine (Still et al., 1974a).

Soybean [Glycine max (L.) Merr. variety Hawkeye] were germinated, grown and root treated with chlorpropham-^{14}C. A polar metabolite was obtained from the roots and identified by enzymatic studies, derivatization and GLC, mass spectrum and NMR as an O-glucoside of 2-hydroxychlorpropham. Similar analyses of shoots showed the presence of the 2- and 4-hydroxychlorprophams as O-glucosides. The latter was found to be unstable to aqueous acid hydrolysis (Still and Mansager, 1973a). In cucumbers, only 4-hydroxychlorpropham conjugates were observed (Still and Mansager, 1973c).

In resistant soybeans, the major metabolite of CIPC was 2-hydroxy-CIPC whereas in susceptible soybeans it was the 4-hydroxy analog. The phenolic metabolites were conjugated in both susceptible and resistant plants (Still et al., 1974b).

Soybeans were grown in the presence of ^{14}C-CIPC. Polar extracts of shoots, when subjected to alkaline hydrolysis, yielded some 3-chloroaniline. Analyses also showed the presence of 1-OH-CIPC. When grown in soil, soybeans metabolized CIPC primarily by alkyl hydroxylation and only small amounts of 2-OH- and 4-OH-CIPC were formed. In hydroponic culture, metabolism was primarily to aryl hydroxylated metabolites (Wiedmann and Ecke, 1975). In oats, the 4-OH-CIPC formed is converted to an S-cysteinyl hydroxychlorpropham (Rusness and Still, 1975).

Soybean plants were root-treated with propham-^{14}C. Analyses of the metabolites indicated that they were glycosides of 2-hydroxypropham and that one of these metabolites was probably β-O-glucoside (Still and Mansager, 1973b).

CLOPIDOL [3,5-Dichloro-2,6-dimethylpyridin-4-ol]

When administered to rabbits, clopidol was rapidly absorbed and excreted mostly via urine. Less than 1% of the dose remained in the tissues at 16 h after dosing. In addition to unchanged clopidol, 3,5-dichloro-2-hydroxymethyl-6-methylpyridin-4-ol and its glucuronide were present in urine. Another metabolite may be the 2,6-dihydroxymethyl analog (Cameron et al., 1975).

CREDAZINE [3-(2-Methylphenoxy)pyridazine]

When this herbicide was irradiated with sunlight in aqueous solution at pH 6.8, about 65% of the credazine was unchanged after 3 weeks of exposure. The photolytic decomposition was greater at pH 9.0 and produced primarily 3-pyridazinone-(2H) and o-cresol. o-Cresol decomposed rapidly but pyridazinone was unaffected. Also identified were salicylic acid, hydroxylated credazine, and 3-(2-methylphenoxy) pyridazine-1-oxide (Nakagawa and Tamari, 1974).

CYANIDE

Opposums were dosed with sodium cyanide by means of a stomach tube. Feces and urine were collected and analyzed.

Analysis indicated that the major route of detoxication of cyanide was via conversion to thiocyanate, which was excreted in the urine. Traces of 2-imino-4-thiazolidine carboxylic acid were observed in the crude concentrated extract of the urine (Turner, 1969).

Studies with a basidiomycete showed that ammonia, HCN, and acetaldehyde condense to form α-aminopropionitrile which can be hydrolyzed by nitrilase to alanine (Strobel, 1966 and 1967). In other studies, HCN was added to cultures of Rhizoctonia solani. α-Aminobutyronitrile was isolated and has been proposed as an intermediate in cyanide fixation by Rhizoctonia solani (Mundy et al., 1973).

Cyanide was administered to C57 Black mice by i.p. injection. When given alone, cyanide was extensively converted to thiocyanate and excreted in the urine. Pretreatment of animals with nitrite and thiosulfate increased thiocyanate excretion. Pretreatment with cobalt compounds alone or in combination with thiosulfate decreased the formation of thiocyanate and gave increased urinary excretion of cobalt ions and strongly bound cyanide complexes (Frankenberg and Sorbo, 1975).

CYOLANE [2-Diethoxyphosphinylimino-1,3-dithiolane]

Cyolane-treated alfalfa hay was fed to a cow one year after treatment of the alfalfa. Cholinesterase was depressed and did not return to normal levels until 3 months after feeding of the cyolane-contaminated hay had been discontinued. Identification of the residues was not made (Tadjer and Egyed, 1974).

2,4-D and RELATED COMPOUNDS

2,4-D [2,4-Dichlorophenoxyacetic acid]

2,4-DB [4-(2,4-Dichlorophenoxy)butyric acid]

Erbon [2-(2,4,5-Trichlorophenoxy)ethyl 2,2-dichloropropionate]

MCPA [4-Chloro-2-methylphenoxyacetic acid]

Silvex [2-(2,4,5-Trichlorophenoxy)propionic acid]

2,4,5-T [2,4,5-Trichlorophenoxyacetic acid]

CPA [Chlorophenoxyacetic acid]

2,4-D [2,4-Dichlorophenoxyacetic acid]

Two h after ingestion by human volunteers, 2,4-D appeared in the urine. More than 75% of the dose was excreted within 96 h. No metabolites were detected.

Pharmacokinetics

$$C_t = \frac{A_o k_f}{v_d(k_f - K_e)}[\exp(-k_e t) - \exp(K_f t)]$$

After a 5 mg/kg dose:

$k_f \times 10^2(h^{-1}) = 27.4 \pm 4.0$

$k_e \times 10^2(h^{-1}) = 2.1 \pm 0.2$

$t_{0.5}(e)(h) = 33.0 \pm 3.1$

$V_d \times 10^2$ (l/kg) = 10.1 ± 0.3

Where:

C_t = Plasma concentration at time t

A_o = dose in mg/kg

k_f = first order rate constant - absorption

k_e = first order rate constant - clearance

V_d = Volume of distribution

(Kohli et al., 1974)

Bovine serum albumin and 2,4-D interact. Binding of 2,4-D is rather extensive but is greatly reduced in the presence of palmitic acid (Kolberg et al., 1973). The properties of the binding site resemble the properties of the amino acid sequence adjoining the tryptophan at the binding site of bovine serum albumin (Mason, 1975).

After feeding 2,4-D to sheep and cattle, analysis of muscle, fat, liver and kidney showed the presence of 2,4-dichlorophenol (Clark et al., 1975).

The butyl ester of 2,4-D is unstable in water and undergoes hydrolysis in 9 to 10 days with formation of 2,4-D. If fish are present, high residue levels may accumulate within a few days and persist for a week or more (Shcherbakov and Poluboyarinova, 1970).

In studies with maize leaf tissues, 2,4-D bonded to some extent with cellular protein (Zemskaya et al., 1971). Oat, wild cucumber, and to a lesser extent cocklebur were able to bind or alter large amounts of 2,4-D (Dexter, 1970). Yellow nutsedge (Cyperus esculentus L.) did not absorb 2,4-D as well as some of the susceptible broadleaf plants. Analysis of methanol-soluble extracts indicated that yellow nutsedge does not appreciably degrade the 2,4-D (Bhan et al., 1970).

Shortly after exposure of Ribes sativum leaves to 1-^{14}C-2,4-D, 37.8% of the label appeared in glycolic acid in the water-soluble metabolites. ^{14}C-Glycine was also observed (Fleeker, 1973).

Soybean (Glycine max L. var. Acme) cotyledon callus stock cultures were grown on an agar medium. After 2,4-D-1-^{14}C was added, 2,4-D-glutamic acid and 2,4-D-aspartic acid were the major amino acid conjugates found. In addition to these, five other conjugates were isolated and observed: alanine, leucine, phenylalanine, tryptophan, and valine. Two other conjugates were not identified. When 2,4-D-glutamic acid was added to the media, 2,4-D and the aspartate complex were formed. The aglycones 4-hydroxy-2,5-D and 4-hydroxy-2,3-D were also formed. These studies also indicated a more rapid conversion of the glutamate complex to the foregoing inactive aglycones than of 2,4-D itself (Feung et al., 1973). Additional studies were conducted with callus tissues of carrot, jackbean (Canavalia ensiformis), sweet corn (Zea mays), tobacco and sunflower (Helianthus annus). After the five plant callus species were exposed to 2,4-D, metabolism produced amino acid conjugates and hydroxylated metabolites as glucosides. After β-glucosidase treatment of water soluble extracts, 2,4-D, 4-OH-2,3-D and 4-OH-2,5-D were found in callus tissue of all five plants. Corn callus tissue alone contained 4-hydroxy-2-chlorophenoxyacetic acid and 3-OH-2,4-D. The glutamate conjugate of 2,4-D was found in all five plants; and the aspartate conjugate, in corn, tobacco and jackbean (Feung et al., 1975).

In studies with labeled 2,4-D and seedlings of wheat, insoluble complexes of protein and 2,4-D formed. Water-soluble compounds also formed (Hallmen and Eliasson, 1972). Other studies have also shown that plants form water-soluble complexes and hydroxylated 2,4-D derivatives (Eidel'nant and Mostovaya, 1972).

After treatment of tomato plants of the variety "Eurocross A," acid hydrolysis of the n-butanol extracted glycosides yielded 4-OH-2,3-D and smaller amounts of 4-OH-2,5-D, 2,4-D and an unidentified metabolite (Muller and Schuphan, 1975).

In potatoes, the differences between total and free residue levels of 2,4-D were small but statistically significant and indicated the presence of conjugated 2,4-D. Residues of 2,4-dichlorophenol were also present (Bristol et al., 1974).

The results of studies in susceptible rape (Brassica napus L. cv. Nilla) and sunflower (Helianthus annus L. var. uniflorus) indicated that 2,4-D existed mainly in the free state and only to a small extent in water-soluble complexes (Hallmen, 1974). Similar results were obtained with wheat (Triticum aestivum L. cv. Starke) and Norway spruce [Picea abies (L.) H. Karst] (Hallmen, 1975).

The influence of four algae on 2,4-D residues in water was studied. Of the four algae used [Chlorella pyrenoidosa Chick., Chlamydomonas reinhardtii Dangeard, Euglena gracilis Krebs. 'urophora', and Scenedesmus quadricanda (Turp.) Breb.], only the latter was effective in removing the herbicide. When ring-labeled 2,4-D was incubated with Scenedesmus, the main metabolite observed was 3-OH-2,4-D (26% of total radioactivity). The 5-hydroxy analog was also identified. Two other metabolites having higher R_f values than 2,4-D were not identified (Valentine and Bingham, 1974).

Molds grown in culture broths containing 2,4-D analogs produced growth-inhibiting principles. Active principles were isolated as crude yellow oils (Naito, 1958; Naito and Kojima, 1957; Naito and Tani, 1955 and 1956a and b). When the molds Gloeosporium olivarum, Gloeosporium kaki and Schizophyllum commune were grown on media containing 2,4-D, colorless needles were isolated and identified by IR spectrum, mass spectrum, elemental analysis and M.P. as the ethanol analog (Nakajima et al., 1973).

Arthrobacter sp. cultures were grown on 2,4-D. An enzyme preparation prepared from these cultures converted cis,cis-2,4-dichloromuconate to chloromaleylacetate. The enzyme that converts dichloromuconate to 2-chloro-4-carboxymethylene but-2-enolide was separated from the enzyme that opens the butenolide lactone (Sharpee et al., 1973).

In other studies with an enzyme preparation from Arthrobacter sp., the ether linkage of 2,4-D was cleaved to produce initially 2,4-dichlorophenol and glyoxylate. Evidence for the latter was indirect with observation of the formation of α-alanine. Neither acetate nor glycolate was metabolized by the bacterial enzyme preparation. Catechols were cleaved to cis,cis-muconic acids. The products of 4-chloro- and 3,5-dichloro-catechol were the β-chloro- and α,γ-dichloromuconic acids, respectively. The latter was proposed on the basis of its UV spectrum and analogous formation of β-chloromuconic acid from 4-chlorocatechol. Acidification caused lactonization to the corresponding butenolides. Some cis,trans-β-chloromuconic acid also formed. α-Chloromaleylacetate, formed from α,γ-dichloromuconic acid, was identified by GLC, UV and mass spectrometry. This compound decarboxylated readily to form the lactol of cis-α-chloro-γ-ketopent-2-enoic acid and was identified by GLC, IR, mass spectrometry and nuclear magnetic resonance. In the presence of NADH, α-chloromaleylacetate was metabolized to yield succinate. Preliminary studies indicated α-chloro-γ-ketoadipate and chlorosuccinate as intermediates (Tiedje, 1969).

In studies with 2,4-D and MCPA, applications of either to soil affected a cross adaptation. After a period of 19 years, repeated applications reduced the time for 50% degradation for 2,4-D and MCPA from 10 and 20 weeks to 4 and 7 weeks, respectively (Torstensson et al., 1975).

The irradiation of aqueous solutions of 2,4-D and several of its esters indicated differences in mode of decay. The free acid undergoes mono- and di-dechlorination, ortho and para hydroxylation, and polymer formation. Intact esters (ethyl, butyl and 2-methylheptyl) undergo monodechlorination. In light stronger than sunlight, further dechlorination and rearrangement occurs (Binkley and Oakes, 1974a and b, Binkley et al., 1974).

Irradiation of 2,4-D aqueous solutions with UV above 280 nm gave little reaction. When a sensitizer such as riboflavin was also used, the products included tetrachlorophenoxyphenols and two isomeric tetrachlorodihydroxybiphenyls. The major product was 4,6-dichloro-2-(2,4-dichlorophenoxy)phenol. A trace of a trichlorophenoxy phenol was also observed (Plimmer and Klingebiel, 1971).

Studies have also shown that photochemical degradation of 2,4-D derivatives follows two paths. In water, the predominant path, except for esters, involves removal of a chlorine and replacement with a hydroxyl group and cleavage of the ether to form the phenol. In methanol, a chlorine is abstracted from the ring (Binkley et al., 1974).

2,4-D was formulated as a urea type polymer and exposed to irradiation at 356 nm. This form of 2,4-D was less resistant to degradation than conventional 2,4-D. UV degradation of the polymerized 2,4-D was eliminated by incorporation of UV absorbers. Resistance to thermal degradation, however, was greater in the polymerized form than in non-polymerized 2,4-D (Baur and Bovey, 1974).

Pyrolysis of amine salts of 2,4-D produced corresponding amides. Above 160C, 2,4-dichlorophenol, imines, lactones, and other compounds also formed. Above 200C, the amides seemed to decompose (Hee and Sutherland, 1974).

Basic and acid hydrolysis of 2,4-D esters yielded 2,4-D and the corresponding alcohol. The hydrolysis half-life of the butoxyethyl ester at 25C increased from 9 h at pH 8 to more than one year at pH 5. The major photoreaction of 2,4-D esters at $\lambda<290$ nm involved cleavage of the ortho C-Cl bond. Calculated sunlight photolysis half-lives of butoxyethyl ester, at latitude 34°N ranged from 59 h in summer to 430 h in winter, and 13 h and 109 h, respectively, in hexadecane (Zepp et al., 1974).

MCPA [4-Chloro-2-methylphenoxyacetic acid]

MCPA was applied to a rice field. About 70% of the material reaching the target area was lost by evaporation and soil percolation. Photolysis of MCPA with an indoor photoreactor or sunlight yielded 4-chloro-2-methylphenol and lesser amounts of o-cresol and 4-chloro-2-formylphenol (Soderquist and Crosby, 1975).

Bovine serum albumin bound 2,4,5-T extensively. Palmitic acid reduced the binding (Kolberg et al., 1973). The binding site properties resembled those the amino acid sequency adjoining the tryptophan residue of human serum albumin. There was evidence, too, of the presence of tryptophan at the high affinity binding site of bovine serum albumin (Mason, 1975).

Human male volunteers ingested a single dose of 5 mg/kg. Excretion of 2,4,5-T was essentially in unchanged form. Clearance from the plasma and excretion both followed first-order kinetics with a half-life of 23.10 and 23.06 h, respectively (Gehring et al., 1973).

[1-^{14}C]2,4,5-T was administered by stomach tube to pregnant and non-pregnant rats. The rate of elimination was the same for both groups. Urinalysis revealed that 90-95% of the label excreted was in the form of unchanged 2,4,5-T. Two non-polar and one water soluble metabolite were observed. Acid hydrolysis of the latter produced 2,4,5-T. The biological half-life in the various organs was essentially the same but differed between adult and newborn rats: 3.4 h vs. 97 h, respectively (Fang et al., 1973). The plasma and elimination half-lives for Sprague-Dawley rats was 4.7 and 13.6 h, respectively, when a 5 mg/kg dose was administered (Piper et al., 1973).

When adult beagle dogs were administered 5 mg/kg doses, the half-life for plasma clearance and elimination was 77.0 and 86.6 h, respectively. Three unidentified metabolites were detected in the urine (Piper et al., 1973).

Photolytic decomposition of 2,4,5-T by sunlight in distilled water at pH 8 was studied. The principal reaction was ether cleavage and replacement of ring chlorines by hydroxyl and hydrogen. The products identified were: 2,4,5-trichlorophenol > lactone of 4,5-dichloro-2-hydroxyphenoxyacetic acid > 2,5-dichlorophenol, 4-chlororesorcinol and 4,6-dichlororesorcinol. Unidentified and polymer material accounted for as much as all metabolites combined except the 2,4,5-trichlorophenol (Crosby and Wong, 1973).

DBNPA (2,2-Dibromo-3-nitrilopropionamide) [2,2-Dibromo-2-cyanoacetamide]

The rate of hydrolysis of DBNPA at various pH levels was determined at 25C and the hydrolysis products determined.

pH	$t_{1/2}$, h
3.9	2140 (23C)
6.0	155
6.7	37
7.3	8.8
7.7	5.8
8.0	2.0
8.9	0.34
9.7	0.11

As DBNPA decreased, dibromoacetonitrile increased. Dibromoacetamide formed by hydrolysis of the latter. Dibromoacetic acid and CO_2 formed under more stringent conditions and hydrolysis of the acid yielded bromide ions and glyoxylic acid. Two unexpected compounds were identified as tribromoacetonitrile and tribromoacetamide. These must have arisen via a bimolecular reaction and hydrolysis. These latter products occurred because of the high (12000-15000 ppm) concentrations used in these studies. At use levels of 1-10 ppm, these should not occur. When a solution of DBNP was mixed with sodium bisulfite, cyanoacetic acid was obtained. DBNPA was also exposed, in water and in a quartz tube, to a G.E. sunlamp for 3 days. A residual oil was obtained which contained cyanoacetic acid, malonic acid amide, malonic acid and oxalic acid. IR and mass spectral analyses were employed for identification (Exner et al., 1973).

DCB [Dichlorobenzene]

When dichlorobenzenes were fed to rabbits, the compounds were slowly metabolized over a 3-6-day period.

o-DCB

The o-isomer yielded glucuronides (48%), sulfates (21%), mercapturic acid (5%) and catechols (4%). The major metabolites were 3,4- and 2,3-dichlorophenol conjugates (2:1, respectively). 4,5-Dichlorocatechol and traces of 3,4-dichlorocatechol comprised the major portion of the catechol fraction and 3,4-dichloro phenylmercapturic acid was the probable mercapturic acid (Azouz et al., 1954).

m-DCB

When fed to rabbits, the m-DCB yielded glucuronides (31%), sulfates (11%), mercapturic acid (9%) and catechols (4%). The major product was 2,4-dichlorophenol. Traces of 3,5-, but no 2,6-, dichlorophenol were observed. 2,4-Dichlorophenylmercapturic acid and 3,5-dichlorocatechol were also observed (Azouz et al., 1954).

p-DCB

Rabbits fed p-dichlorobenzene excreted glucuronides (37%) and sulfates (27%). No mercapturic acid or catechols were observed. 2,5-Dichlorophenol and a quinol, probably 2,5-dichloroquinol, were observed (Azouz et al., 1954).

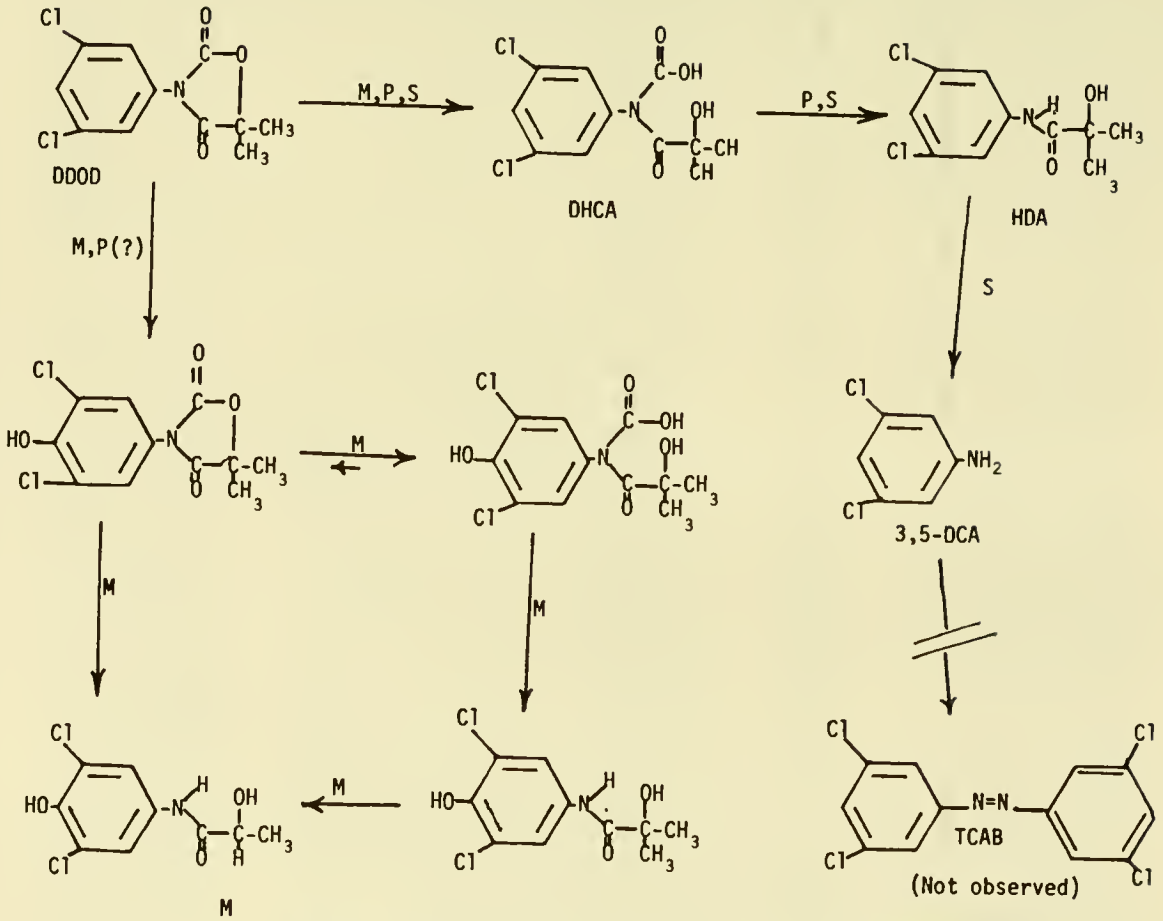
DDOD (Dichlozoline) [3-(3,5-Dichlorophenyl)-5,5-dimethyloxazolidine-2,4-dione]

Labeled DDOD was administered orally to 200g male Wistar rats. Urine and feces were collected and analyzed. Approximately equal amounts of radioactivity appeared in urine and feces. Most of the radioactivity in feces was unchanged DDOD. About 85% of the radioactivity was extractable from urine with ether and contained eight metabolites. Five were identified:

N-(3,5-dichloro-4-hydroxy)-5,5-dimethyloxazolidine-2,4-dione
N-(3,5-dichloro-4-hydroxyphenyl)-α-hydroxyisobutyramide
N-(3,5-dichloro-4-hydroxyphenyl)-N-(α-hydroxyisobutyryl)carbamic acid
N-(3,5-dichlorophenyl)-N-(α-hydroxyisobutyryl)carbamic acid
N-(3,5-dichloro-4-hydroxyphenyl)lactamide

Two fractions treated with glucuronide or sulfatase released compounds, one of which was identified as N-(3,5-dichloro-4-hydroxyphenyl)lactamide (Sumida et al., 1973a).

Bean plants were treated with labeled DDOD. After root treatment, DHCA and HDA were observed. Neither compound was observed when DDOD was injected into the bean plant. Other unidentified metabolites were observed. After injection of DDOD into grape plants, analysis of the leaves showed the presence of DMDOD, HDA and unidentified material. In soil, DDOD degraded to DHCA, HDA, DCA and unidentified material. No TCAB was found (Sumida et al., 1973b).


DDOD
M,P,S
DHCA
P,S
HDA
M,P(?)
S
M
M
3,5-DCA
M
M
TCAB
(Not observed)
M
Glucuronid or Sulfate
Conjugates
M=Mammals
P=Plants
S=Soil

DDT and RELATED COMPOUNDS

DDT [2,2-Bis(p-chlorophenyl)-1,1,1-trichloroethane]

DDD (TDE, Rhothane) [2,2-Bis(p-chlorophenyl)-1,1-dichloroethane]

Kelthane (Dicofol) [2,2-Bis(p-chlorophenyl)-1,1,1-trichloroethanol]

DDE [2,2-Bis(p-chlorophenyl)-1,1-dichloroethylene]

Studies with human serum has shown that p,p'-DDE can be bound to proteins. This binding increased with aged serum (Schoor, 1973). When ring-labeled ^{14}C-DDT was incubated with human embryonic lung cells, reductive dechlorination produced DDD. The only other metabolite found was DDA (North, 1972; North and Menzer, 1972). Binding of p,p'-DDA with human and bovine serum has also been observed (Ross and Biros, 1975).

Syrian golden hamsters were fed p,p'-DDT in their diet for 4 months. Urine and feces were collected. Autoradiography of thin-layer plates on which urine had been spotted indicated the presence of at least 10 radioactive bands. The major metabolite (80%) apparently was DDA, free and as the glucuronide. Other bands were identified as the DDA conjugates of glycine and alanine. DDD was observed in urine and feces (Wallcave et al., 1974).

After Swiss albino mice were fed p,p'-DDT, analysis of urine revealed the presence of DDE and DDA as the glucuronide, alanine and glycine conjugates. DDD was found in the feces (Wallcave et al., 1974).

In other studies with mammals, oral dosing of mice with metabolites of p,p'-DDT indicated that DDE is not an intermediate in DDT metabolism to DDA. Administration of p,p'-DDD produced increased levels of p,p'-DDA. However, DDMU led to a decrease in DDA production in the urine over that found from DDD administration. This would not be expected if DDMU was a metabolite of DDT or DDD leading to production of DDA. When administered intraperitoneally, p,p'-DDT dosing led to a decrease in DDA production and indicated that the intestines may play a major role in DDT degradation to DDA (Apple, 1969). Other studies with hepatic microsomes also indicate that the pathway for DDT in birds may differ from that in mammals. A pathway involving DDMU to a highly active liver inducer may be involved (Bunyan and Page, 1973).

Oral dosing of mammals with p,p'-DDT gave rise to o,p'-DDA. No p,p'-isomer was observed (Apple, 1969).

^{14}C-Labeled o,p'-DDD was administered orally to Sprague-Dawley rats. Urine and feces were collected at 6, 12 and 24 h and then at 24 h intervals. Rats were sacrificed at 15 days. Urine contained: o,p'-DDA and its 3-hydroxy, 4-hydroxy and 3,4-dihydroxy analogs; the serine and glycine conjugates of DDA; monomethyoxy- and dimethoxy-o,p'-DDD (Reif and Sinsheimer, 1975).

After mice were administered DDT, urine and feces were collected and analyzed. In addition to DDE, DDT, DDMS, DDD, DBP, DDA and kelthane, five unidentified metabolites and some conjugates were observed (Kapoor et al., 1972). In other studies, DDT was incubated with ovine (Ovis aries) rumen fluid. DDD, DDE and DDMU were observed (Sink et al., 1972).

After mule deer fawns, Odocoileus hemionus, were fed p,p'-DDT, DDD was found in the serum and in feces. p,p'-DDE was found in fecal matter but not in serum (Watson et al., 1975).

Studies with a worker exposed to DDT indicated that the human liver excretes DDT into the bile (Paschal et al., 1974).

A common loon (Gavia immes), found in a moribund state in a soybean field, was autopsied and tissue was analyzed. In addition to p,p'-DDE, p,p'-DDD, p,p'-DDMU, p,p'-DCBP, p,p'-DDMS, o,p'-DDD, and o,p'-DDE, another compound was observed and identified by synthesis and mass spectrum as 1,1-bis(p-chlorophenyl)-2,2-dichloroethanol (Prouty et al., 1975).

Chickens, modified surgically to facilitate collection of urine and feces were administered single oral doses of ring-^{14}C-labeled o,p'-DDT. Feces contained o,p'-DDT and o,p'-DDD, predominantly. The following metabolites were also observed:

3-hydroxy-2,4'-DDD
4-hydroxy-2,4'-DDD
4-hydroxy-3-methoxy-2,4'-DDD
o,p'-DDE
3-hydroxy-2,4'-DDE
4-hydroxy-2,4'-DDE
4-hydroxy-3-methoxy-2,4'-DDE
3-hydroxy-2,4'-DDT
4-hydroxy-3-methoxy-2,4'-DDT
o,p'-DDA
3-hydroxy-2,4'-DDA
o,p'-DDA methyl ester
Methyl ester of a methoxy o,p'-DDA (probably 3-isomer)
4-hydroxy-3-methoxy-2,4'-DDA

(Feil et al., 1974 and 1975)

12,000xg Liver preparations, to which NADPH and riboflavin had been added, were incubated with DDT. Percentage conversion of DDT to DDD was in order: hamster > mouse > pigeon > Wistar rat > quail > cockerel; after heating and then supplementing: Wistar rat > mouse > quail > hamster > cockerel > pigeon (Hassall, 1975). In other studies, incubation of pigeon liver preparations with DDT and DDD gave rise to analogs showing loss of one chlorine (Hassall and Manning, 1972).

When pooled fat extracts from tissues of guillemots and grey seals were analyzed, two hydroxylated DDE analogs were observed. The 4,4'-dichlorobenzophenone and 2-bis(p-chlorophenyl) acetic acid were also present in some samples (Jansson et al., 1975). The hydroxylated DDE

analogs were identified by mass spectra and synthesis as: 1,1-dichloro-2-(4-chloro-3-hydroxyphenyl)-2-(4-chlorophenyl)ethylene; 1,1-dichloro-2-(3-chloro-4-hydroxyphenyl)-2-(4-chlorophenyl)ethylene. When rats were fed p,p^1-DDE, these two and a third compound identified as the 2-(4-chloro-2-hydroxyphenyl) analog were isolated (Jansson et al., 1975; Sundstrom et al., 1975).

When tadpoles of the common frog (Rana temporaria) were exposed to p,p^1-DDT, no DDE was detected. The only metabolite observed was DDD and post-mortem breakdown was suspected (Cooke, 1970).

In the environment, conversion of DDT to DDD has been observed in a wide variety of biological systems, living and dead. The mechanism has not been clearly established but existing data indicates the involvement of reduced porphyrins. A series of studies demonstrated that the DDT to DDD conversion was affected whenever DDT and reduced porphyrins were brought together in solution (Zoro et al., 1974).

Agricultural loam soils were treated with DDT in 1954 and sampled periodically. Analyses in 1970 showed the presence of DDD, DDE and dicofol (Lichtenstein et al., 1971).

In flooded soil, p,p^1-DDT was dechlorinated to p,p^1-DDD. Some DDE was also observed (Bhulya, 1969). When DDT was added to an Everglades muck, the amount of degradation was related to the changes in redox potential. Under aerobic or flooded anaerobic conditions in substrate-amended muck, where the redox potential dropped to +350 and +180 mV, respectively, little DDT degradation occurred. Where the redox potential dropped to 0 and -250 mV, considerable degradation occurred (Parr and Smith, 1974).

E. coli, B. subtilis and S. aureus degraded DDT to DDD and DDE in trypticase soy broth (Collins, 1969). Anaerobiosis was found to be an environmental factor in the degradation of DDT to DDD. The ability to make this conversion was demonstrated by 27 bacterial species from the following genera: Achromobacteria, Aerobacter, Agrobacterium, Bacillus, Clostridium, Erwinia, Kurtha, Pseudomonas and Xanthomonas (Johnson, 1969). Reductive dechlorination of p,p^1-DDT to DDD has been observed under both aerobic and anaerobic conditions (French, 1969). Bacillus megaterium converted a small amount of DDT to DDD (Hicks, Jr., and Corner, 1973). When DDT was added to raw sewage, DDD, DDE and DBP were produced. Addition of glucose enhanced the rate of DDD formation but reduced DBP formation. Addition of diphenylmethane reduced formation of DDD and DBP (Pfaender and Alexander, 1973).

Membrane-bound enzymes from Hydrogenomonas sp. appeared to mediate initial anaerobic degradation of DDT to DDD. Soluble cell fractions and flavin enzymes stimulated the reaction. Enzyme preparations from Hydrogenomonas sp. were capable of degrading DDT to DDD, DDMS, DBP. Addition of fresh cells to the anaerobically incubated extract and

aerobic incubation of the mixture produced p-chlorophenylacetic acid (PCPA). An isolated Arthrobacter sp., that could use PCPA as a sole carbon source, produced p-chlorophenylglycolaldehyde during growth on PCPA (Pfaender, 1972).

In studies with adult catfish (Heteropneustes fossilis) exposed to DDT, only DDE was observed (Agarwal and Gupta, 1974). The thorny skate (Raja radiata) metabolized ^{14}C-p,p^{1}-DDT to DDD and DDE (Darrow and Addison, 1973).

Ring-labeled p,p^{1}-DDT-^{3}H was force-fed to mature brook trout (Salvelinas fontinalis) one dose each week for five weeks. The eggs were collected, fertilized and incubated. Analyses of the eggs and fry indicated metabolism to p,p^{1}-DDD and p,p^{1}-DDE. Distribution of the three compounds was determined from the day of fertilization until 80 days post-fertilization:

p,p^{1}-DDT	92.4	to	61.7%
p,p^{1}-DDD	0.4	to	5.1%
p,p^{1}-DDE	7.2	to	33.2%

(Atchison and Johnson, 1975)

When ^{14}C-labeled DDT was given orally to common soles [Solea solea (L.)], there was a characteristic distribution pattern of accumulated DDT that was independent of dosage. Brain, liver and gastro-intestinal tract ranked highest. More than 80% of the accumulated DDT remained unchanged and DDE, DDD and a polar compound occurred as metabolites (Ernst and Goerke, 1974).

After oral application of μg-amounts of DDT to the polychaete Nereis diversicolor, analysis indicated that most of the DDT (51 to 67% of the initial dose) was stored unchanged. There was only slight metabolism, probably to DDA (Ernst, 1969).

Studies with the freshwater planarian Phagocata velata indicated that it was capable of metabolizing DDT to DDD and DDE (Phillips et al., 1974).

All algae tested converted DDT to DDE. No other metabolites were observed. Algae used included:

Chlorella vulgaris
Ankistrodesmus braunii
Anacystis nidulans
Nostoc muscorum
Synechococcus elongatus
Synechococcus cedrorum
Anabaena variabilis

Anabaena flos-aquae
Calothrix parieatina
Phormidium luridum var. olivacea
Skeletonema costatum
Cyclotella nana
Isochrysis galbana
Olisthodiscus luteus
Amphidinium carteri
Tetraselmis chuii

(Rice, 1972; Rice and Sikka, 1973)

In other studies with the unicellular freshwater alga (Ankistrodesmus amalloides), after exposure to ^{14}C-DDT for 30 days, both DDE and DDD were recovered. When daphnids (Daphnia pulex) were exposed for 24 h, DDE was recovered (Neudorf and Khan, 1975).

Collembola, Folsomia candida, was fed brewer's yeast spiked with 100,000 ppm DDT. These were then released into fenced plots in a beech-maple forest. Arthropod fauna were sampled at intervals. Reductive dechlorination of DDT gave DDD and dehydrochlorination gave DDE (Klee, 1972).

Vitamin B_{12} has been suggested as having a role in the degradation of DDT. To study this possibility, macrocyclic alkyl-cobalt complexes were synthesized and reacted with DDT. In one of these studies, the Co(II) complex was prepared, reduced to Co(I) species, and allowed to react with DDT. A maroon powder was isolated. Infrared spectra indicated that the complex contained a carboxyl group. After a few days in sunlight, a solution of the material was extracted. Mass spectrum analysis indicated that the white material obtained was bis(p-chlorophenyl) ketone (Prince and Stotter, 1974; Prince et al., 1974).

Gamma-radiation of DDT induced loss of chlorine but DDD and DDE were absent. It seems, therefore, that dechlorination proceeds with simultaneous loss of all three chlorine atoms from the CCl_3-group (Woods and Akhtar, 1974).

In a laboratory model ecosystem, DDE was extremely stable in the tissues of the living organisms. Analysis of body and feces of the salt marsh caterpillar showed over 95% unchanged DDE. The remainder appeared as unidentified polar metabolites (Metcalf et al., 1975b).

Under laboratory conditions, DDT, DDE, DDD and DDA were chromatographed on silica gel G on glass plates. The studies indicated that degradation of DDT occurred when the spotted plates were exposed to UV. In addition to DDD and DDE, there appeared to be four other components (Ernst, 1972).

DDT decomposed when irradiated, whether as a pure solid or in hexane solution, by UV (2537Å). In hexane solution, DDD and HCl were identified. When irradiated in the solid form, DDD, DDE and DBP were formed (Mosier et al., 1969).

When DDT and DDE in pyrex tubes were exposed to UV-irradiation (λ>290nm) as solids in an oxygen stream, mineralization products (CO_2 and HCl) were observed in small amounts after 7 days. DDE yielded dichlorobenzophenone and trichlorobenzophenone. No photoproducts from DDT were detected (Gab et al., 1975).

Kelthane residues on apple pomace were subjected to UV irradiation filtered through pyrex glass (λ>290nm). Analyses indicated that only DBP was formed from Kelthane (Archer, 1974).

After oral administration of Kelthane to rats, analyses of tissues, urine and feces showed the presence of DDE, DBP and 4,4^1-dichlorobenzhydrol (Brown et al., 1969).

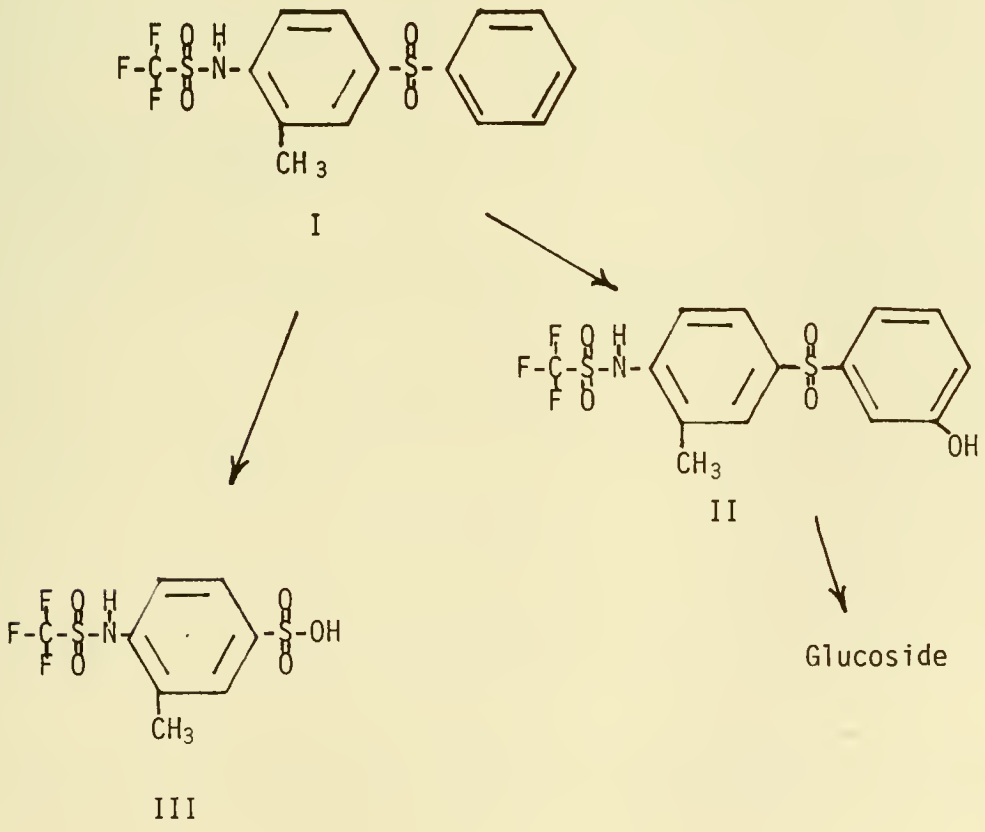
Recent studies have indicated that colored complexes can form between montmorillonite clay and p,p^1-DDT, o,p^1-DDT, p,p^1-DDE, p,p^1-DDD, p,p^1-DDA, dicofol, or 4,4^1-dichlorobenzophenone (Haque and Hansen, 1975).

In other studies, larvae of the bollworm H. zea and tobacco budworm H. virescens were exposed to DDT. Analyses indicated rapid accumulation of DDE and only small amounts of metabolites more polar than DDT in H. zea. In H. virescens, there was only minor accumulation of DDE but rapid accumulation of more polar metabolites (Plapp, 1973).

DESTUN (Perfluoridone) [1,1,1-Trifluoro-N-[2-methyl-4-(phenylsulfonyl) phenyl]methanesulfonamide]

In sandy loam soil, destun was degraded to 3-methyl-4-[[(1,1,1-trifluoromethyl)sulfonyl]amino]benzenesulfonic acid (II) (Bandal et al., 1974).

Peanut seedlings were root treated with ^{14}C-labeled destun. Young developing lateral branches contained the highest concentrations of ^{14}C in foliar tissue. TLC of aqueous extracts of roots gave eight radioactive zones. Hydrolysis of one of the zones yielded destun and a metabolite tentatively identified as 1,1,1-trifluoro-N-[2-methyl-4-(3-hydroxyphenylsulfonyl)phenyl]methanesulfonamide (III). The most abundant metabolite, not identified, yielded 2 moles of glucose and one of compound II upon acid hydrolysis, but one mole of II and no glucose when treated with β-glucosidase (Lamoureux and Stafford, 1974).



DEXON [p-Dimethylaminobenzenediazo sodium sulfonate]

Pseudomonas fragi metabolizes dexon by a co-metabolic process. One of several compounds that is formed was identified as N,N-dimethyl-p-phenylenediamine (DMPDA). The enzyme, a reductase, was found in the soluble fraction and required dithioerythreitol as reductant. When dexon was applied to soil, DMPDA was also found (Karanth et al., 1974).

DIANISYLNEOPENTANE [1,1-Bis(p-methoxyphenyl)-2,2-dimethylpropane]

In an effort to find a more selective and biodegradable replacement for DDT, analogs of DDT were studied. Metabolic studies were conducted with an analog, dianisylneopentane, and the DDT-resistant housefly R_{SP}, the salt marsh caterpillar Estigmene acrea (Drury), female Swiss white mice, and mouse liver microsomes (see Table 1). Additionally a model ecosystem was used to study biodegradation (see Table 2). In the model ecosystem, this compound accumulated in fish to about the same level as has been found in studies with methoxychlor; and the neopentyl group exhibited about the same stability as the trichloromethyl group (Coats et al., 1974).

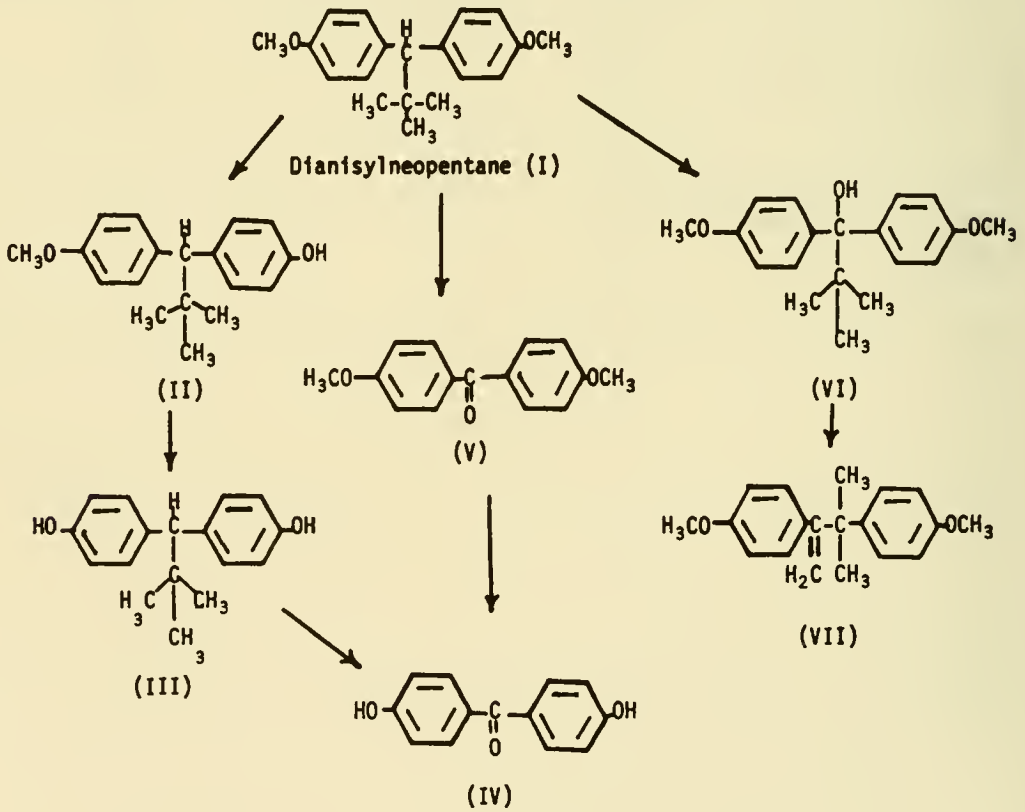
Table 1

Compound	Radioactivity recovered			
	Housefly	Caterpillar	Mouse	Liver homogenate
I	++++	+++++	+++	++
II	+++		+++	+
III	++		++	+++
IV	++		+	+
V	+		+	
VI	+		+	
VII	+	+	+	+
Conjugates & acids	+++	+	+++	+
Unknown A	+			
B	++		+	+

Table 2

Metabolites found in the model ecosystem

Compound	H O	Alga (Oedogonium)	Snail (Physa)	Fish (Gambusia)
I	+	+	+	+
II	+	+		+
III	+	+		+
IV	+			+
V	+			+
VI	+		+	+
VII	+	+	+	+
Conjugates & acids	+	+	+	+
Unknown A	+	+	+	+
B	+	+	+	+



DIAZINON [O,O-Diethyl O-(2-isopropyl-4-methylpyrimidin-6-yl) phosphorothioate]

In the beagle dog, metabolism of diazinon proceeded rapidly. Analyses of urine indicated the presence of two metabolites identified as: 4-hydroxy-2-isopropyl-6-methylpyrimidine (III) and 4-hydroxy-2-isopropanol-6-methylpyrimidine (VIIa) (Iverson et al., 1975).

Three diazinon metabolites were isolated from sheep dosed by stomach tube. Identification was made by mass spectra: from urine, the 2-isopropanol diazinon (VI); 4-methanol diazinon (XI); primarily from fat, the isopropenyl analog (VIII) (Janes et al., 1973; Machin, 1973).

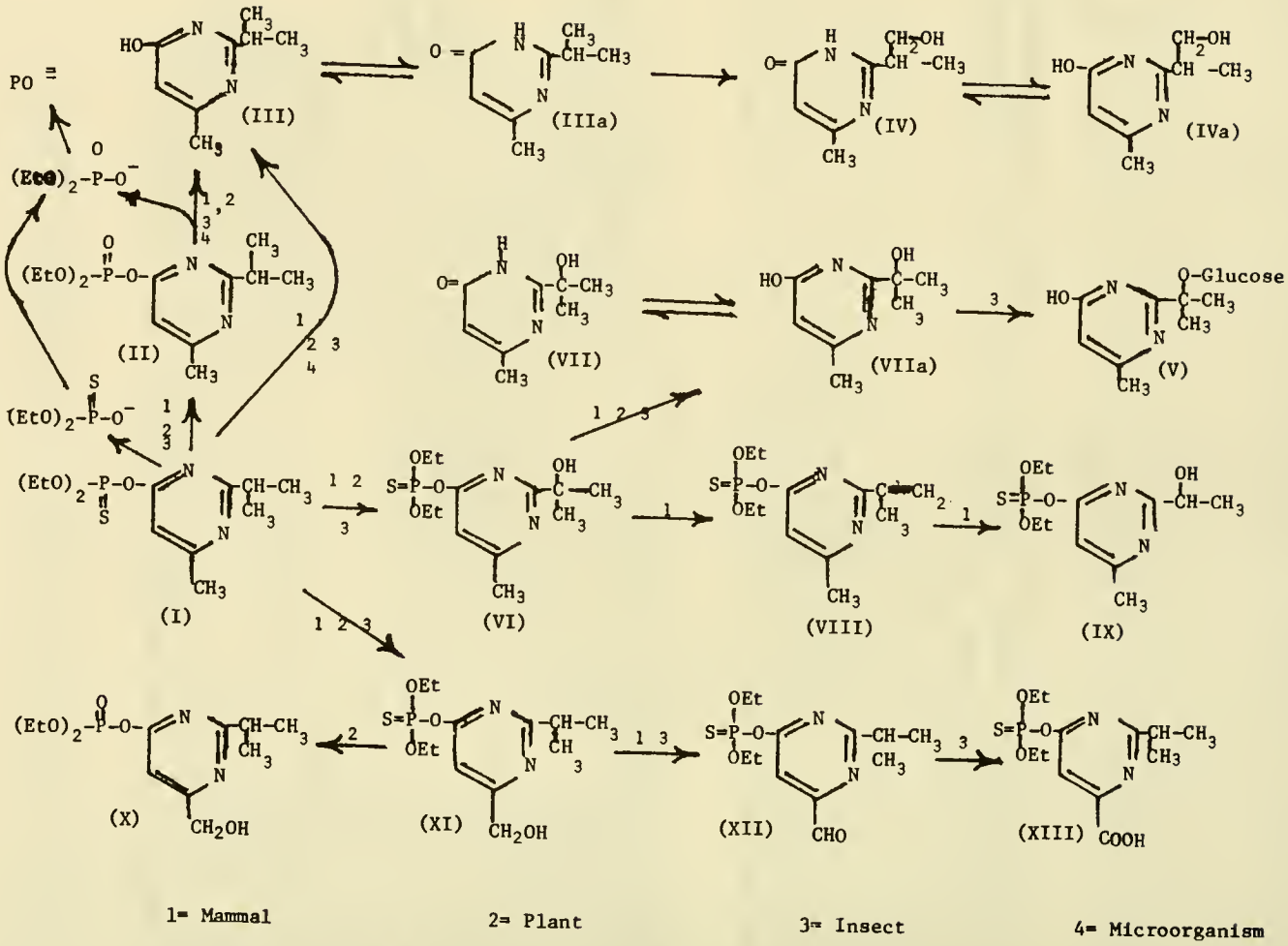
Liver microsomes rapidly degraded diazinon to diethyl phosphorothioic and diethyl phosphoric acids. Diazinon was degraded by hydrolases in mitochondrial, microsomal and soluble fractions of rat liver (Yang et al., 1969; Yang, 1971).

Houseflies, susceptible and resistant, degraded diazinon and diazoxon. The major metabolites were diethyl phosphorothioic and/or diethyl phosphoric acids. In addition to the MFO system which was responsible for the foregoing, there is another soluble system in the housefly which used reduced glutathione. The major metabolites were the same (Yang, 1971).

Degradation of diazoxon by homogenates of Hokota strain (diazinon-resistant) and NAIDM strain (diazinon-susceptible) houseflies produced diethyl phosphate. Optimal pH was ca. 7.0. Resistant flies degraded more diazoxon than susceptible flies. The homogenate supernate degraded diazinon in the presence of reduced glutathione with no significant difference between rates by the supernatant of Hokota and NAIDM strains. The main degradation products were diethyl phosphorothioic acid and S-(2-isopropyl-4-methyl-6-pyrimidinyl) glutathione. Optimal pH was 8.5. Microsomal MFO plus NADPH metabolized diazinon to diazoxon and diethyl phosphorothioic acid (Shono, 1973, 1974 a and b).

Metabolism of diazinon by gypsy moth larvae (Porthetria dispar L.) was qualitatively the same whether after ingestion or topical application. The major products were 2-isopropyl-4-methyl-6-pyrimidinol; diazoxon; hydroxydiazinon; 2-(2-hydroxy-2-propyl)-4-methyl-6-pyrimidinol. Formation of these metabolites was reduced by the synergists 2,6-dichlorobenzyl-2-propynyl ether and piperonyl butoxide (Ahmad and Forgash, 1975).

From submerged soild and rice paddies, microorganisms capable of decomposing diazinon have been obtained. These fall into three categories:


(III)
(IIIa)
(IV)
(IVa)
(II)
(VII)
(VIIa)
(V)
(I)
(VI)
(VIII)
(IX)
(X)
(XI)
(XII)
(XIII)
1= Mammal
2= Plant
3= Insect
4= Microorganism

1. Sole carbon source - *flavobacterium* sp.

2. Synergism - *Arthrobacter* sp. plus *Streptomyces* sp.

3. Co-metabolism
   Arthrobacter sp.
   Corynebacterium sp., *Pseudomonas melophthora*, *Streptomyces* sp.
   Trichoderma viride

Microorganisms accelerated hydrolysis of diazinon and subsequent mineralization of 2-isopropyl-6-methyl-4-pyrimidinol to CO_2 (Sethunathan, 1972).

The inhibition of MFO by pesticide synergists was investigated. Piperonyl butoxide and NIA 16824 (*O*-isobutyl-*O*-propargyl phenylphosphonate) inhibited all oxidative reactions to the same extent. However, 1-(2-isopropyl phenyl)imidazole inhibited conversion of thiophosphate to phosphate and oxidative de-arylation. There was no significant effect on hydroxylation of ring side chain (Smith et al., 1974).

Ultraviolet irradiation of diazinon gives a mixture of products. One has been identified as *O*,*O*-diethyl *O*-(2-acetyl-6-methylpyrimidin-4-yl) phosphorothioate (Machin and Quick, 1971).

DICAMBA (Banvel; 3,6-dichloro-o-anisic acid) [2-Methoxy-3,6-dichlorobenzoic acid]

DISUGRAN (Racuza; methyl 3,6-dichloro-o-anisate) [Methyl 2-methoxy-3,6-dichlorobenzoate]

Breakdown of dicamba in bracken litter occurred rapidly. At 25C, 50% of the initial dose (1.25 lb/acre) was lost in 4 days at pH 4. Raising the pH to 7.4 reduced the loss rate (Parker and Hodgson, 1966).

Dicamba degraded rapidly in prairie soils when applied at rate of 1.1 kg/ha. At 25C over 50% dissipated in 2 weeks. In other studies over half was lost within 4 weeks and only $^{14}CO_2$ and 3,6-dichlorosalicylic acid were detected (Smith 1973a and b, 1974b).

Incubation of disugran with rumen fluid of ewes produced seven metabolites. Four were not identified. However, the three identified metabolites comprised about 95% of the total metabolites. Degradation proceeded primarily via ether cleavage and then ester hydrolysis. The third metabolite was 3,6-dichloro-o-anisic acid (Ivie et al., 1974a).

A radiochemical analytical procedure was used to monitor dicamba breakdown in soil. At -5 ± 1C breakdown was not observed but was observed at 5 ± 1C. At temperatures above 15 ± 1C, over 80% of the dicamba was dissipated in 8 days. When heavy clay and sandy loam were used, 14 days and temperatures above 20 ± 1C were needed to degrade similar amounts of the herbicide (Smith and Cullimore, 1973).

DICHLOBENIL [2,6-Dichlorobenzonitrile]

The fate of dichlobenil in alligator weed and parrot feather (Myriophyllum brasiliense) was investigated. In the latter, the major metabolite was the 3-hydroxy analog. Small amounts of the benzamide and benzoic acid analogs of dichlobenil were also observed as was 3-hydroxy-2,6-dichlorobenzamide. Conjugates of the 3-hydroxy compounds also occurred. There was other material not identified (Sikka et al., 1974).

In soil, degradation of dichlobenil was determined at 6.7 and 26.7C. At 6.7C, the half-life was 28 weeks after a ten-week lag; 19 weeks at 26.7C. Only 2,6-dichlorobenzamide was detected (Montgomery et al., 1972).

In a farm pond treated with dichlobenil at 10 lb a.i. per surface acre, the concentrations in water and hydrosoil had decreased by 85 and 87%, respectively, after 7 weeks (Rice et al., 1974). In other studies with pond water, more than 75% of the added dichlobenil disappeared because of volatilization. Some dichlobenil was metabolized microbiologically to 2,6-dichlorobenzamide and other unidentified metabolites. A cell suspension of Arthrobacter sp. metabolized up to 71% of added dichlobenil in 6 days to dichlorobenzamide and small amounts of other compounds not identified. Evolution of $^{14}CO_2$ from labeled dichlobenil indicated that some of this herbicide could be completely degraded in the environment (Miyazaki et al., 1975).

DICHLOROPROPENE (Component of DD) [Trans- and cis-dichloropropene]

At 15 to 20C in sandy soils, 1,3-dichloropropenes exhibited an average half-life of 24 days and disappeared at the rate of 2 to 3.5% per day with no marked isomer difference. Some chloride was released but this slowed to about 3% after an initial rapid release. Chloroallyl alcohols are assumed to arise from dichloropropenes in soil. The trans-isomer ($t_{1/2}$ = <one day) degrades more rapidly than the cis-isomer ($t_{1/2}$ = ca. 2 days) (Van Dijk, 1974).

DICHLORVOS (DDVP, Vapona, Nuvan, Mafu) [2,2-Dichlorovinyl dimethyl phosphate]

Dichlorvos was rapidly metabolized by mice, hamsters, rats and man. Urine of rat, mouse and hamster contained compounds tentatively identified as hippuric acid, desmethyldichlorvos, urea and dichloroethanol glucuronide. The latter was also found in urine of man (Hutson and Hoadley, 1972).

In pigs, after one dose of dichlorvos-^{14}C in slow release PVC, ^{14}C in the tissues was in the form of C-1 and C-2 fragments from the vinyl moiety of dichlorvos and incorporated into normal tissue constituents such as glycine, serine, creatine, glucose, glycogen, fatty acids, cholesterol, choline, lecithin and ribonucleic acid (Page et al., 1972; Potter et al., 1973a and b).

In vitro studies were conducted with isolated nucleic acids and in cells of E. coli and the human tumor cell line HeLa. DNA and RNA were methylated (Lawley et al., 1974).

Stored soybeans were treated with an aqueous emulsion of dichlorvos at the rate of 20 ppm. Within 24 h post-treatment, 77% of the calculated deposit had disappeared. During processing, most of the residue was removed with the hull. Further processing, including refining of crude oil and toasting the defatted meal, removed the remaining residue. Degradation of dichlorvos in the stored crude soybean oil was slow (0.36 ppm after 12 weeks) but was rapid on the hulls (<0.02 ppm after 12 weeks) (La Hue et al., 1975).

DICROTOPHOS (Bidrin, Carbicron, Ektafos, SD 3562, C709) [Cis-N,N-dimethyl-3-(dimethyl phosphate) crotonamide]

Mouse liver fractions were incubated with [^{14}C] O-methyl dicrotophos. When phenobarbital and dieldrin were present, oxidative as well as hydrolytic metabolism was induced. In the presence of the whole homogenate, different metabolism patterns were noted in contrast to that observed with supernatant. Des-N-methyl dicrotophos and N-hydroxymethyl dicrotophos were detected. Other metabolites were not identified (Tseng and Menzer, 1974).

DICRYL [N-(3,4-Dichlorophenyl)methacrylamide]

Dicryl was incubated in a culture medium with the fungus Rhizopus japonicus. After extraction of the culture medium and purification by TLC, a metabolite was identified as N-(3,4-dichlorophenyl)-2-methyl-2,3-dihydroxypropionamide (Wallnöfer et al., 1973b).

DIMETHOATE [O,O-Dimethyl S-(N-methylcarbamoyl)methyl phosphorodithioate]

An amidase was prepared from sheep liver microsomes. The optimum pH was 9 and it had a molecular weight of 230,000 to 250,000. The amidase was able to hydrolyze the N-alkyl and various O,O-dialkyl analogs of dimethoate (Chen, 1972).

Elution patterns of N-hydroxymethyl analogs of dimethoate and dimethoxon indicated that these compounds were converted to their corresponding de-N-methyl derivatives by heat on the GLC column (Steller and Brand, 1974).

When a surfactant was added to a dimethoate wettable powder, initial penetration of citrus leaves was greater than when no surfactant was used. Without surfactant, there was no significant penetration for 1 day as against 2 h with a surfactant. Residues of dimethoxon in grapefruit pulp, following a dimethoate-wettable powder treatment was less than 0.05 ppm. Residues of dimethoate averaged 0.09 ppm after 2 days and 0.03 ppm after 14 days (Woodham et al., 1974).

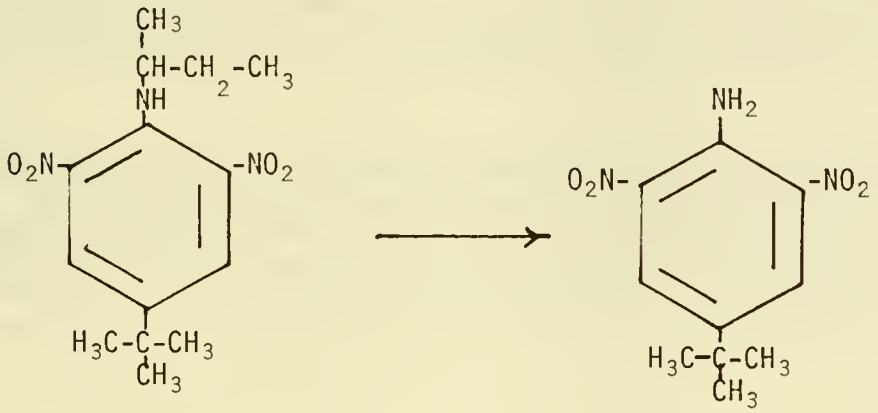
After dimethoate was applied to soils, dimethoate carboxylic acid and dimethoxon were found. Identification was by co-chromatography. Other compounds were observed but could not be identified (Duff and Menzer, 1973).

[^{14}C]Carbonyl-dimethoate was incubated with mouse liver fractions. The oxon analog and other metabolites were observed. When the homogenate was pretreated with phenobarbital or dieldrin, dimethoxon concentration was increased by more than fourfold. Similar results were observed *in vivo* with mice (Tseng and Menzer, 1974).

DINITRO COMPOUNDS

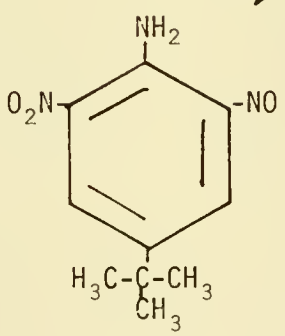
N-sec-Butyl-4-tert-butyl-2,6-dinitroaniline

In methanol, 75% of this compound was decomposed after 8 h exposure to sunlight. A major product of decomposition in methanol or water was the nitrosoaniline (III). Some dinitroaniline (II) was also observed. Mass spectrometric analysis of minor products indicated that two pathways may be simultaneously operative (Plimmer and Klingebiel, 1974).



I

II



III

DINITRO COMPOUNDS

DNBP (Dinoseb) [2-(1-methyl-n-propyl)-4,6-dinitrophenol]

DINOBUTON [2-(1-methyl-n-propyl)-4,6-dinitrophenol isopropyl carbonate]

In rats and rabbits DNBP was metabolized to the 2-amino analog and then converted to the glucuronic acid conjugate. A propionic acid analog and another compound, thought to be hydroxylated on the side chain, were observed (Ernst, 1969).

After application of dinobuton to bean leaves, dinoseb (DNBP) and other unidentified compounds were observed (Matsuo and Casida, 1970).

Dinobuton was slowly hydrolyzed to dinoseb after application to apple trees. Subsequent degradation occurred to produce the 2-amino and butyric acid analogs. Other unidentified polar compounds were also observed (Hawkins and Saggers, 1974).

Twenty-eight days after topical application of dinobuton or dinoseb to apple fruits, about 75 and 72% of these materials, respectively, was lost. Analyses of peel indicated the presence of two polar metabolites which chromatographed on TLC similar to 2-amino-6-sec-butyl-4-nitrophenol and 3-(3,5-dinitro-2-hydroxyphenyl)butyric acid. Other polar material, not identified, was also present. The same compounds were found in the apple flesh also (Hawkins and Saggers, 1974).

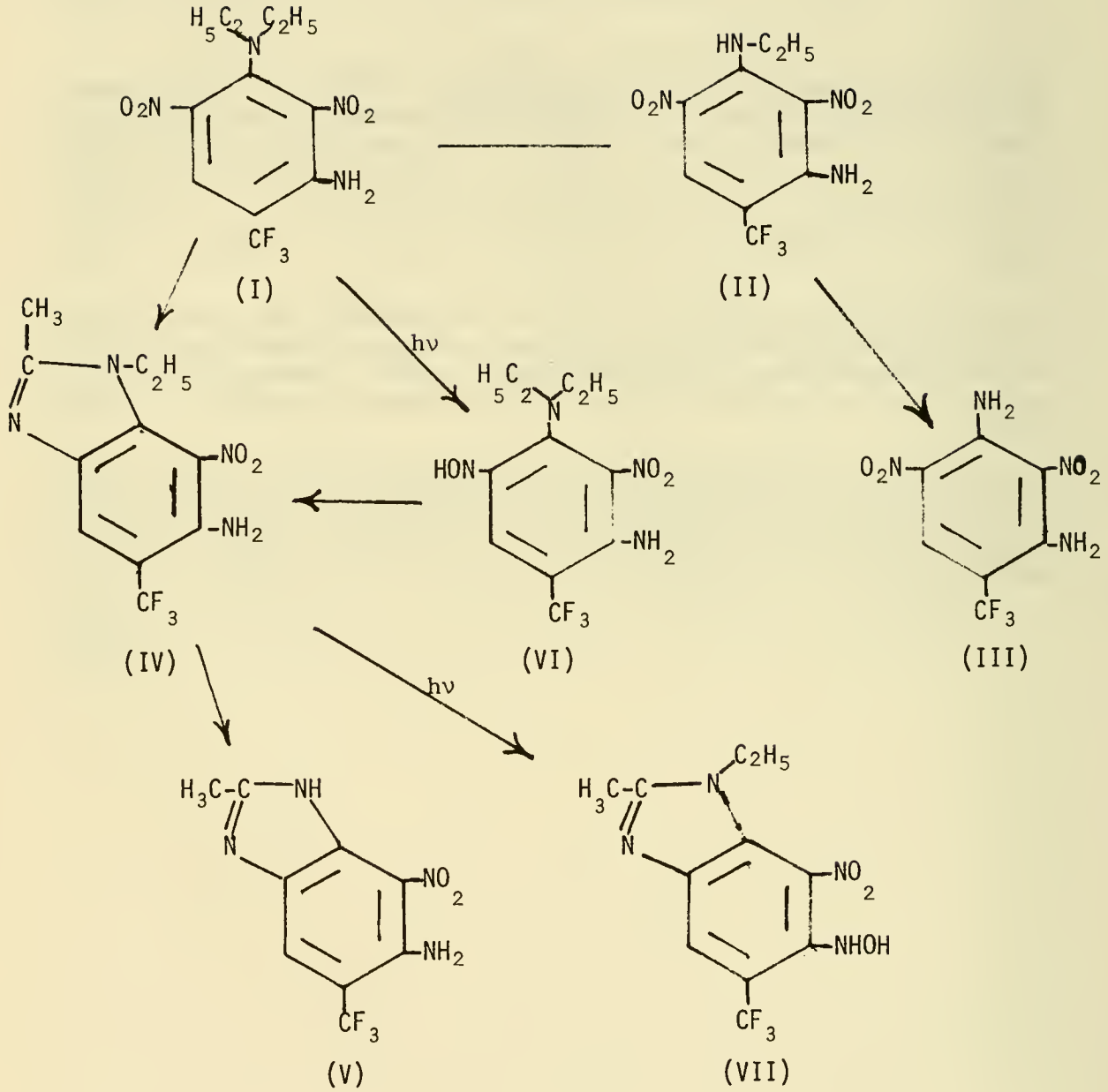
DINITRO COMPOUNDS

DINITRAMINE (Cobex, Cobeko, USB-3584, Diethamine) [N^3,N^3-Diethyl-2,4-dinitro-6-trifluoromethyl-*m*-phenylenediamine]

Soil fungi used were identified as *Aspergillus fumigatus* Fres., *Fusarium oxysporum* Schlecht and *Paecilomyces* sp. When these organisms were exposed in culture to dinitramine, all three degraded the herbicide. Quantitative differences were observed. Four metabolites were identified as the mono- (II) and di- (III) dealkylated dinitramine, the ethylbenzimidazole (IV), and the dealkylated benzimidazole (V). Crude cell extracts of *A. fumigatus* also dealkylated dinitramine (Laanio et al., 1973).

Radioactive dinitramine ($^{14}CF_3$ and ^{14}C-ring-labeled) was incorporated into Anaheim silty loam soil. Analyses over an eight-month period revealed the presence of compounds II, IV and VI. Perhaps as many as ten other unidentified metabolites were also formed (Smith et al., 1973).

Dinitramine underwent rapid photolytic decomposition in methanol and water with a 10 min half-life. Mass spectrum analyses were used to identify the products as compounds IV, V, VI and VII (Newsom and Woods, 1973).


(I)
(II)
(III)
(IV)
(V)
(VI)
(VII)
hν
hν

DINITRO COMPOUNDS

ISOPROPALIN (2,6-Dinitro-N,N-dipropylcumidine) [2,6-Dinitro-N,N-dipropyl-4-isopropylbenzene]

Isopropalin (I) was incorporated in soil, and tomato and pepper seeds were planted in some of the plots. Tobacco transplants were placed in other plots. After all were harvested, wheat was sown and harvested the following year. Analyses of soil and plants involved TLC, column chromatography, GC-MS and thin-layer radioautography. In soil, in addition to bound and unidentified compounds, nine metabolites were identified in addition to unchanged isorpopalin:

II. 2,6-dinitro-N-propylcumidine
III. 2,6-dinitro-cumidine
IV. 2-amino-N,N-dipropylamino-6-nitrocumidine
V. 2-amino-6-nitro-N-propylcumidine
VI. 2-ethyl-5-isopropyl-7-nitro-1-propylbenzimidazole
VII. 2-ethyl-5-isopropyl-7-nitrobenzimidazole
VIII. 4'-isopropyl-2',6'-dinitro-N-propylpropionanilide
IX. 4'-isopropyl-2',6'-dinitropropionanilide
X. α,α-dimethyl-3,5-dinitro-4-dipropylaminobenzyl alcohol

Negligible amounts of isopropalin or its degradation products were found in the plants grown on treated soil (Golab and Althaus, 1975).

DINITRO COMPOUNDS

N-(1-Methyl-n-propyl)-4-tert-butyl-2,6-dinitroaniline

In soil, compound I was dealkylated to produce 4-tert-butyl-2,6-dinitroaniline (II). A soil fungus Paecilomyces sp. produced 3-(4-tert-butyl-2,6-dinitroaniline)-2-butanol (Kearney et al., 1974).

In methanol, 75% of this compound was decomposed after 8 h in sunlight. A major product of decomposition in methanol or water was 4-tert-butyl-2-nitro-6-nitrosoaniline (III). Some 4-tert-butyl-2,6-dinitroaniline (II) was also observed. Mass spectrometric analysis of minor products indicated that two pathways may be simultaneously operative (Plimmer and Klingebiel, 1974).

DIPHENAMID (Dymid, Enide, L-34314) [N,N-Dimethyl-2,2-diphenylacetamide]

Tomato plants (Lycopersicon esculentum Mill., var. Sheyenne), 27 to 35 days old, were fumigated with ozone prior to exposing their roots to ^{14}C-diphenamid in nutrient solution. Some differences were observed:

1. fumigated plants absorbed 70 to 100% as much diphenamid as did controls

2. fumigated plants contained less radioactivity in the $CHCl_3$-soluble fraction than did controls

3. fumigated plants generally contained more radioactivity in the water-soluble and insoluble fractions than did controls.

Fumigated and control plants contained N-methyldiphenylacetamide (MDA), 2,2-diphenylacetamide and an unidentified compound. There were two additional compounds. When hydrolyzed, one gave MDA plus glucose and the other gave MDA plus moieties positive to sugar reagents (Hodgson, 1971).

Diphenamid metabolism in tomato plants was altered by ozone fumigation. There was little effect on root absorption, translocation or conversion to water-soluble conjugates; but the proportions of conjugates were altered. There was a marked shift toward more polar material and increased production of methanol-insoluble residues. The predominant compounds formed, in both fumigated and non-fumigated plants, were the β-glucoside (MDAG) and the β-gentiobioside (MDAGB) of N-hydroxymethyl-N-methyl-2,2-diphenylacetamide (MODA). The latter is a postulated intermediate in the formation of the glucoside and gentiobioside (Hodgson et al., 1973). The primary metabolites of diphenamid metabolism in tomato plants included MODA, N-methyl-2,2-diphenylacetamide and 2,2-diphenylacetamide. In addition to MDAG and MDAGB, soluble polar products were also obtained (Hodgson et al., 1974).

The metabolism of diphenamid in the corn root was studied with carbonyl-^{14}C labeling. The only compound identified was N-methyl-2,2-diphenylacetamide (Yaklich, 1970).

^{14}C-Diphenamid accumulated in foliage of tobacco seedlings (Nicotiana tobacum L. Kentucky) when incubated in nutrient solutions. The amount of radioactivity in the roots remained minimal. The N-methyl-2,2-diphenylacetamide and traces of 2,2-diphenylacetamide were found in the foliage. In the roots, only traces of N-methyl-2,2-diphenylacetamide was observed (Long et al., 1974).

DIQUAT [1,1'-Ethylene-2,2'-bipyridylium dibromide]

PARAQUAT (Gramoxone) [1,1'-Dimethyl-4,4'-bipyridylium dichloride]

Adsorption studies were conducted with diquat and paraquat. Adsorption for both compounds was increased in the order:

$Mg^{+2} > Ca^{+2} > H^{+} > Mn^{+2} > Co^{+2} > Zn^{+2} > Ni^{+2} > Cu^{+2} > Fe^{+3} > Al^{+3}$

(Khan, 1974)

In studies with sandy loam, paraquat was degraded only slightly, if at all, over a seven-year period (Fryer et al., 1975).

DISYSTON [O,O-Diethyl-S-(2-ethylthio ethyl)phosphorodithioate]

Disyston was rapidly converted to its oxidative metabolites in soils. Conversion in soil was predominantly by side chain oxidation of the sulfur. The oxygen analogs form in small amounts only. Oxidation in flooded conditions was at a much faster rate than in upland soils (Takase et al., 1972).

DITHIOCARBAMATES

Under acid conditions, dithiocarbamates may form significant amounts of carcinogenic nitrosamines. In the presence of sodium nitrite and ziram, the carcinogen dimethylnitrosamine formed in small amounts at pH 1.5 to 2.0 (Eisenbrand et al., 1974).

MANEB [Manganese ethylenebisdithiocarbamate]

After treatment of beans and tomatoes with maneb, residues were measured at intervals until 14 days post treatment. Residues on beans were higher than on tomatoes for unchanged maneb as well as ETU, ETM and EDA. Residues of these three metabolites were also found in soil 15 days after treatment (Newsome et al., 1975).

Maneb was suspended in buffer pH=6 and aerated. Degradation products identified by TLC included ETM, ETU, EDA, CS_2 and sulfur (Hylin, 1973).

ZINEB [Zinc ethylenebisdithiocarbamate]

When fruits and vegetables containing zineb were boiled, ETU was formed (Newsome and Laver, 1973).

Maneb and Zineb formulations were studied under laboratory conditions of controlled heat and humidity. At elevated temperatures, zineb was less stable than maneb. The zinc-manganese coordination products, however, were considerably more stable than either zineb or maneb (Bontoyan and Looker, 1973).

ETU [Ethylenethiourea)

Although not a pesticide, there is considerable interest in its occurrence as a breakdown product of a group of ethylenebisdithiocarbamate fungicides very widely used. There is considerable interest too in its fate in the environment.

^{14}C-ETU was injected into corn, lettuce, pepper and tomato seedlings. Some ETU was present after 14 days. Some $^{14}CO_2$ was formed but the major degradation product in all plant tissues was identified as ethyleneurea (EU) (Hoagland and Frear, 1976).

When ETU was administered to rats and guinea pigs, elimination was rapid. About 50% was eliminated within 24 h in urine. Elimination in feces was negligible (Newsome, 1974).

Photolysis of ETU (λ>285 nm) on a solid substrate produced 2-imidazolidone as the major product, bis (imidazolin-2-yl) sulfide and an unidentified product. Aqueous solutions of ETU undergo slow photolysis but are stable to hydrolysis in the pH range 5.0 to 9.0 at 90C (Cruickshank and Jarrow, 1973). Although aqueous solutions of ETU were stable to sunlight, in the presence of dissolved oxygen and sensitizers, ETU was rapidly degraded. When less than 5% of the ETU remained, a compound was indentified as glycine sulfate. When the reaction was stopped while there was more than 50% of the ETU remaining, 2-imidazolidone was obtained (Ross and Crosby, 1973).

DS-15647 (Thiofanox) [3,3-Dimethyl-1-methylthio-2-butanone O-(methylcarbamoyl) oxime]

Fully expanded leaves of cotton plants grown in the field were treated individually with ^{35}S-labeled DS-15647. The sulfone and sulfoxide were formed. Metabolism to the sulfoxide was essentially complete in 4 days. Polar compounds not identified, the oxime and the oxime sulfoxide, also formed. Similar results were obtained with excised cotton leaves. Seedlings grown from treated seed gave the same results as those obtained in mature plants and in soil. The rate of the initial reaction was more rapid than that of the second, particularly in plants (Whitten and Bull, 1974a).

DYFONATE [O-Ethyl S-phenyl ethylphosphonodithioate]

The degradation of dyfonate was studied with a series of fungi: Aspergillus flavus, Aspergillus fumigatus, Fusarium oxysporum, Trichoderma viride, Aspergillus niger, Mucor alternans, Rhizopus arrhizus and Mucor plumbeus. All species degraded dyfonate to some extent. Degradation by P. notatum was slowest; M. alternans, R. arrhizus and M. plumbeus were the most active species in producing water-soluble metabolites. In addition to dyfonate, the extracts contained dyfoxon, thiophenol, diphenyl disulfide, $MPSO_2$ (methyl phenyl sulfone), ethylethoxyphosphonic acid (EOP) and ethylethoxyphosphonothioic acid (ETP) and methyl phenyl sulfoxide (MPSO) (Flashinski and Lichtenstein, 1974a and b).

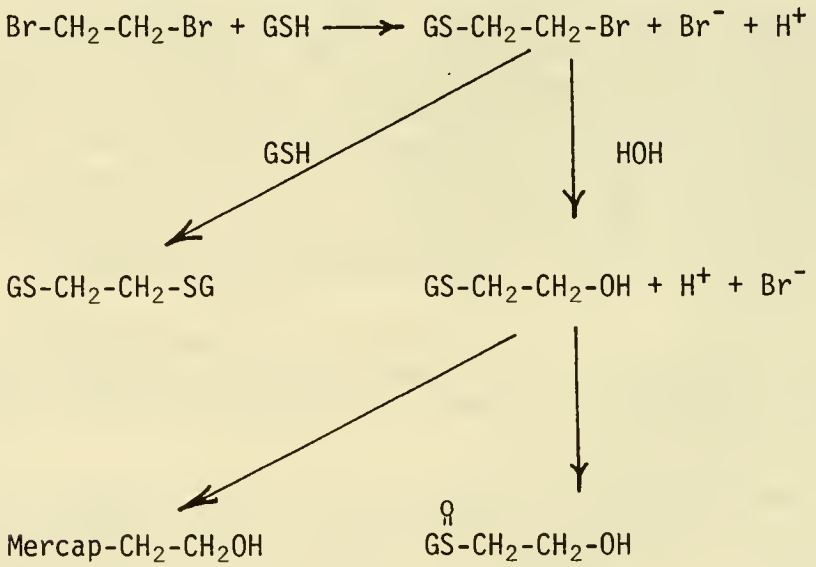
Factors affecting the ability of soil fungi to degrade dyfonate included nturient supply, temperature, pH and incubation time. The studies indicated that the mycelium absorbed dyfonate and that metabolism of dyfonate occurred in the mycelium. Degradation apparently involved formation of the ethyl acetate-soluble metabolites: ethylethoxyphosphonic acid, ethylethoxyphosphonothioic acid, methyl phenyl sulfoxide and methy phenyl sulfone. These were then metabolized to water-soluble compounds. For R. arrhizus, the optimum was at 15-25C and pH 6.0 to 7.0 (Flashinski and Lichtenstein, 1975).

When plants were grown in nitrogen deficient nutrient solutions, concentrations of dyfoxon in greens were reduced due to deficiencies of all elements (potassium, calcium, and magnesium) except nitrogen (Talekar and Lichtenstein, 1973).

Dyfonate was incorporated into soil at the rate of 5.6 and 11.2 kg/ha as granules or emulsifiable concentrate; and its persistence and absorption by plants was observed. Four months after application, 33-35% of the granular application and 38-41% of the emulsifiable concentrate application (both 5.6 kg/ha) remained in the soil. No dyfoxon was detected in soil. Potatoes, beets, and rutabagas had little or no detectable residue at either application rate. Wheat had 0.01-0.07 ppm dyfonate but little or none in the mature plant or in the grain. Carrots grown in soil treated at the lower rate had 0.35 and 0.04 ppm dyfonate and dyfoxon, respectively (Saha et al., 1974).

EDB [Ethylene dibromide]

In vitro studies of the enzyme catalyzed reaction between EDB and glutathione were conducted. Chromatography and comparison with synthetic samples indicated the presence of S-(β-hydroxyethyl) glutathione as the main product (93%) and S,S^1-bis(glutathione) ethylene (7%). In vivo studies indicated that the reaction occurs primarily in the liver with formation of S-(β-hydroxyethyl)glutathione, S-(β-hydroxyethyl)glutathione sulfoxide and S,S^1-bis(glutathione)ethylene. Later degradation occurs primarily in the kidneys to yield S-(β-hydroxyethyl) mercapturic acid and its sulfoxide (Nachtomi, 1970).



ENDOSULFAN (Thiodan) [6,7,8,9,10,10-Hexachloro-1,5,5a,6,9,9a-hexahydro-6,9-methano-2,4,3-benzo[e]dioxathiepin-3-oxide]

Endosulfan residues on alfalfa were exposed to ultraviolet light and to sunlight while drying for 10 days. A third lot was dried in the dark. Residue analyses showed the presence of endosulfan isomers I and II, endosulfan diol (XI), endosulfan ether (VII), endosulfan α-hydroxy ether (XIV) and endosulfan sulfate (IX). No lactone was observed (<0.1%). In all three groups, the sulfate percentage of the total residue increased but the increase was most dramatic in the dark drying lot (Archer, 1973).

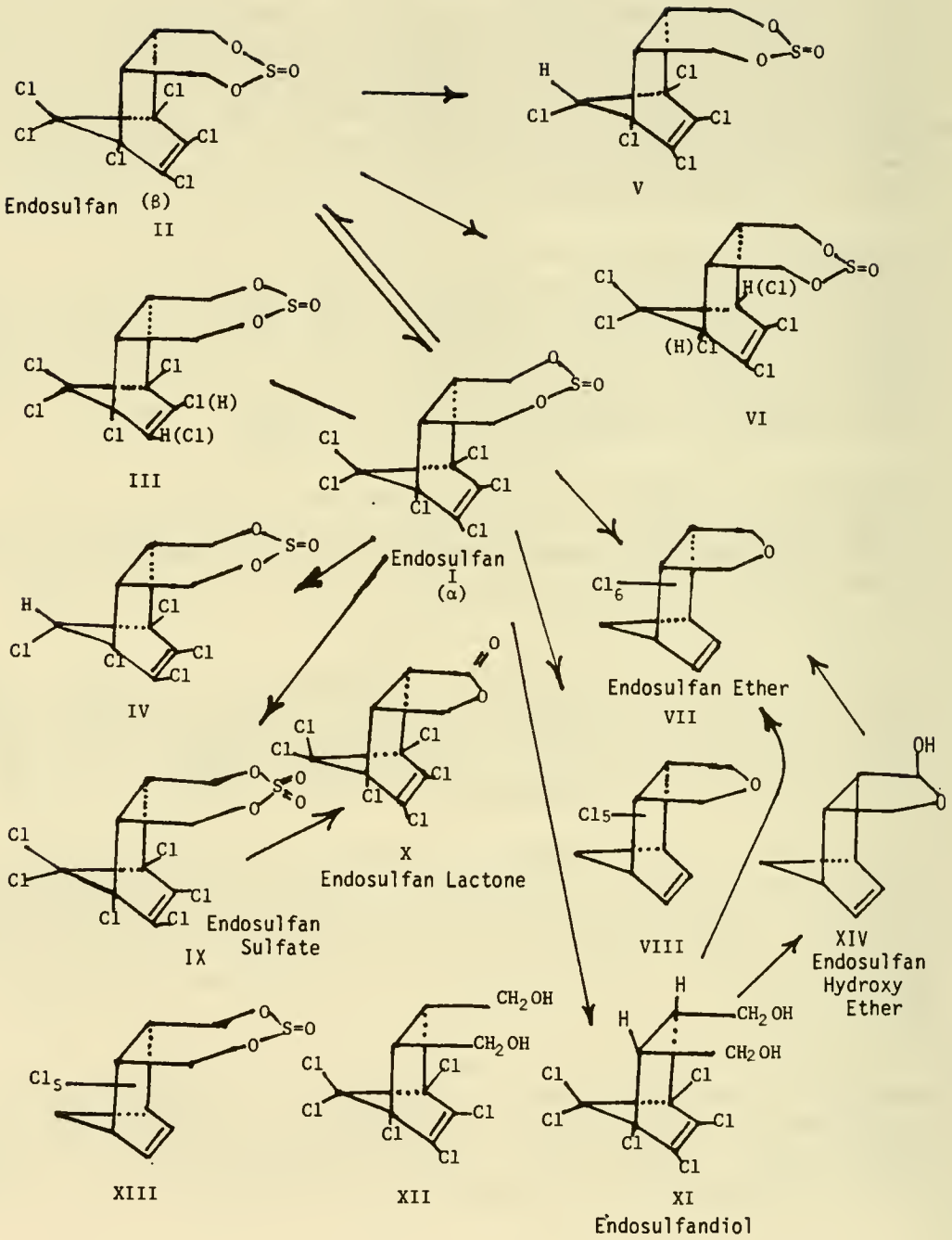
In temperature stressed rats orally dosed with endosulfan I or II, endosulfan sulfate was the metabolite most commonly recovered from tissues, organs and feces regardless of temperature stress. The diol, α-hydroxy ether and lactone (X) were found in most urine and feces samples. Studies with the diol showed this to be converted to the α-hydroxy ether and that both diol and α-hydroxy ether were metabolized to the lactone in small amount (Whitacre, 1970).

Degradation of endosulfan by a soil microorganism of the family Pseudomonad was studied. The alcohol was the main metabolite from either isomer. The β-isomer also yielded small amounts of the endosulfan ether as well as isomerized to the more stable α-isomer (Perscheid et al., 1973).

Endosulfan isomers (α and β) were incubated with Aspergillus niger. Endosulfan alcohol was formed from both isomers (El Zorgani and Omer, 1974). These same authors refer to studies by Domsch and coworkers in which endosulfan sulfate was also observed after exposure of microbes and fungi to endosulfan.

After incorporation of endosulfan into soil, analyses indicated a half-life for the α-isomer of about 60 days and about 800 days for the β-isomer. Endosulfan sulfate formed in amounts equivalent to the endosulfan that decomposed. Potato tubers grown in the treated soil contained residues of both isomers and the sulfate (Stewart and Cairns, 1974).

UV irradiation of endosulfan isomers gave a variety of products, depending on the medium used. α-Endosulfan (I) yielded compound III in n-hexane; compound IV in dioxane-water; and compounds VII to XIII in gas phase. β-Endosulfan (II) gave compound V and VI in dioxane-water and compound V in n-hexane-acetone (Schumacher et al., 1973).


Endosulfan (β)
II
V
VI
III
Endosulfan
I
(α)
IV
Endosulfan Ether
VII
X
Endosulfan Lactone
IX
Endosulfan
Sulfate
VIII
XIV
Endosulfan
Hydroxy
Ether
XIII
XII
XI
Endosulfandiol

ENDOTHALL [7-Oxabicyclo(2.1.1)heptane-2,3-dicarboxylic acid]

Aquatic microorganisms readily degrade endothall. One species of Arthrobacter, isolated from lake water and sediment, utilized endothall as a sole source of carbon for cell growth. When ^{14}C-endothall labeled in the oxabicyclo ring was used, the ^{14}C was found in cellular amino acids, proteins, nucleic acids and lipids. Citric, aspartic, alanine and glutamic acid were labeled as well as CO_2 (Sikka and Saxena, 1973).

When exposed to ^{14}C-endothall in tap water, bluegill absorbed the labeled material but only unchanged herbicide was found in the alcohol extractable fraction of the fish (Sikka et al., 1975).

ETHION (Diethion) [S,S-Methylene bis(O,O-diethyl phosphorodithioate)]

When sediment samples from a saline lagoon were incubated with ethion for 20 days, ethion degradation was slow. Analyses indicated the formation of sulfide from ethion. In these studies, ethion was the sole sulfur source (Sherman et al., 1974).

Ethylene oxide (EO, ETO)

When wheat is fumigated with ethylene oxide, 85% of the bound EO was converted to water soluble compounds. The EO residue concentration in the wheat was between 14 and 37.5 ppm and was distributed among water-soluble and water-insoluble proteins, organic acids, mono-oligo-saccharides, lipids, lipoproteins, starch, and bran (Pfeilsticker and Rasmussen, 1974).

FENAZAFLOR (Lovozal) [Phenyl 5,6-dichloro-2-trifluoromethylbenzimidazole-1-carboxylate]

NC-2983 [5,6-Dichloro-2-trifluoromethylbenzimidazole]

A major degradation product of the acaricide fenazaflor was found to be 5,6-dichloro-2-trifluoromethylbenzimidazole (NC-2983). After application of NC-2983 to field plots, soil analyses indicated no significant disappearance in fall, winter or early spring but a rapid decline during the summer. About 85% of an NC-2983 application in summer disappeared within 135 days. When applied in the autumn, disappearance of a comparable amount required about one year (Ercegovich et al., 1972).

FENSULFOTHION (Dasanit, Terracur P) [O,O-Diethyl O-(p-methylsulfinyl) phenyl phosphorothioate]

After application to a sandy loam soil, fensulfothion degraded rapidly to the corresponding sulfone. Traces of the sulfone were found in rutabagas grown in fensulfothion-treated soil. It was also found in carrots, in which it persisted during a 4-year storage period when the carrots were frozen (Chisholm, 1974).

FLUENETHYL (Fluenetil, Flu, Lambrol) [2-Fluoroethyl 2-(4-biphenylyl) acetate]

FLUOROACETIC ACID (1080) [2-Fluoroacetic acid]

Studies with mice, houseflies and the two spotted spider mite clearly indicate that the toxicity of Flu depends on the release of monofluorethanol by ester cleavage of fluenethyl and subsequent oxidation to the monofluoroacetic acid. The latter is converted to fluorocitrate which inhibits aconitase (Johannsen and Knowles, 1974).

Seeds of Acacia georginae, peanut (Arachis hypogaea L.) and bean (Phaseolus vulgaris L. var. Pinto) were treated with ^{14}C-sodium fluoroacetate. Labeling was found in the lipids and in the water-soluble fractions. $^{14}CO_2$ also evolved (Preuss et al., 1968).

When lettuce plants were incubated with labeled fluoroacetate, a labeled material was obtained and identified as S-carboxymethylglutathione (Ward and Huskisson, 1972).

FLUORODIFEN (C-6989, Preforan) [2,4^1-dinitro-4-trifluoromethyl diphenylether]

Rats, given a single oral dose of $^{14}CF_3$-fluorodifen, excreted 48% of the dose in 48 h in urine; 23% in feces. Most (83%) of the urinary radioactivity in the 24-h sample was 2-nitro-4-trifluoromethylphenyl-mercapturic acid. In vitro studies with rat liver homogenates indicated the formation of S-(2-nitro-4-trifluoromethylphenyl)gluthathione after cleavage of fluorodifen (Lamoureux and Davison, 1974).

From epicotyl tissues of pea seedlings, a soluble glutathione S-transferase was obtained. The enzyme, capable of cleaving the ether, had a pH optimum of 9.3-9.5. With labeled substrates, the cleavage products were separated and identified as p-nitrophenol and S-(2-nitro-4-trifluoro-methylphenyl)gluthathione (Frear and Swanson, 1973). This gluthathione conjugate was also formed by peanut (Arachis hypogaea L.). O-Conjugates of p-nitrophenol and traces of the 2-amino analog of fluorodifen also formed. Other unidentified metabolites were observed (Shimabukuro et al., 1973a). In other studies with peanut seedlings, metabolites identified included 2-amino-4-trifluoromethylphenol, 2-amino-fluorodifen and traces of p-aminofluorodifen and 2,4^1-diaminofluorodifen. The major product was not identified (Eastin, 1971a and c). Cucumber seedlings were also exposed to labeled fluorodifen. Leaves, stem, cotyledons and root were analyzed. Metabolites identified included p-nitrophenol, 2-aminofluorodifen, p-aminophenol, 2,4^1-diaminofluorodifen and p-aminofluorodifen (Eastin, 1971b).

FLURECOL (Flurenol, EMD-IT 3233) [9-Hydroxyfluorene-9-carboxylic acid]

FLURECOL-n-BUTYL ESTER (IT-3233, Aniten, Florencol, Flurenol-n-butyl ester) [n-Butyl 9-hydroxyfluorene-9-carboxylate]

^{14}C-labeled fluorecol-n-butyl ester was applied to leaves of Phaseolus vulgaris. Five metabolites were observed: two isomeric β-glucosides and three amino acid conjugates. The aglycone moieties were identified as 2^1- and 3^1-hydroxy analogs of flurecol-n-butyl ester (Wotschokowsky, 1972).

FORMETANATE [m-{(Dimethylaminomethylene)amino}phenyl N-methylcarbamate]

About 84% of formetanate injected into houseflies was metabolized in 4 h. The major metabolite was 3'-hydroxyformanilide. Microsomes from housefly abdomen plus NADH resulted in only 10% metabolism of formetanate. Incubation of labeled formetanate with alkaline soil resulted in a 50% decomposition in less than two days. The major products were 3-formamidophenyl methylcarbamate, 3'-hydroxyformanilide and m-aminophenol. Irradiation of formetanate with UV (254 mμ) produced 3'-hydroxyformanilide (the main product), 3-formamidophenyl methylcarbamate and m-{(dimethylaminomethylene)amino}phenol. N-Demethylation of formetanate was of minor importance photochemically (Arurkar, 1971).

FRESCON (Trifenmorph, WL 8008) [N-Trityl morpholine]

Rats eliminated 97% of a single dose within 96 h. Within the gut, frescon was probably hydrolyzed prior to absorption. The morpholine was excreted largely unchanged. Triphenylcarbinol was metabolized to a glucuronide in part and also hydroxylated (mainly para). The latter was conjugated with glucuronic acid (Beynon, 1971).

When fish were exposed to frescon at 0.2 ppm, the only residue observed in fish after 30 min was unchanged frescon (Beynon, 1971).

Rice plants grown in treated water contained residues of triphenylcarbinol and hydroxytriphenylcarbinols (o-, m-, and p-) and their glycosides. In water frescon hydrolyzes to triphenylcarbinol. Mud and sediment gradually adsorb frescon and the carbinol. In soil, within a few weeks frescon is aerobically converted to the carbinol which undergoes slow hydroxylation (Beynon, 1971).

Hydrolysis $t_{1/2}$ at 0.05 ppm

pH	time in hours
6.5	3
7.1	10
7.4	28
8.0	ca. 100
9.0	ca. 1000

GARDONA [Dimethyl 2-chloro-1-(2',4',5'-trichlorophenyl)vinyl phosphate]

Exposure of gypsy moth larvae to gardona produced metabolites, both free and bound, identified as 2,4,5-trichloroacetophenone, 1-(2',4',5'-trichlorophenyl)ethan-1-ol, 2,4,5-trichlorophenacyl chloride and 1-(2',4',5'-trichlorophenyl)-2-chloroethan-1-ol (Tomlin, 1972).

GLYPHOSATE [N-(Phosphonomethyl)glycine]

When applied to clay loam or muck soil, 56 kg/ha of glyphosate was rapidly inactivated. This inactivation was probably the result of reversible adsorption to clay and organic matter (Sprankle et al., 1975a). Iron and aluminum clays and organic matter adsorbed more glyphosate than sodium or calcium clays and was readily bound to kaolinite, illite, bentonite, charcoal and muck but not to ethyl cellulose. ^{14}C-Labeled glyphosate was degraded in soil and $^{14}CO_2$ was released (Sprankle et al., 1975b).

GRISEOFULVIN (Fulvicin) [7-Chloro-4,6-dimethoxycoumaran-3-one-2-spiro-1-(2'-methoxy-6-methylcyclohex-2'-en-4'-one)]

Liver homogenates prepared from Charles River male mice and rats were incubated with griseofulvin. Analyses showed the presence of 4-desmethylgriseofulvin and 6-desmethylgriseofulvin (Chang et al., 1973). Pretreatment with phenobarbital increased both 4- and 6-desmethylation of griseofulvin whereas 3-methylcholanthrene increased only 6-demethylation in rats (Lin et al., 1973).

Single doses of 10 ppm of hexachlorobenzene were administered to rats and several Rhesus monkeys. In rat feces and liver, pentachlorobenzene (PCB) and unchanged HCB were identified by GLC-MS. Pentachlorophenol (PCP) and PCB were identified in urine and feces by GLC-MS. Tetrachlorobenzene (TCB) was identified by GLC in rats and monkeys (Rozman et al., 1975).

In other studies, after administration of a single oral dose of ^{14}C-HCB to adult male rats, primary excretion was via feces (16%). No metabolites appeared to be present. In urine, less than 1% of HCB was excreted but analyses indicated the presence of PCB, TCB, PCP and 2,4,5-trichlorophenol. Homogenates of liver, lung, kidney and small intestines were incubated with HCB. Trace amounts of chlorobenzenes were produced. Liver microsomal preparations with added NADPH produced chlorophenols. Pentachlorophenol probably formed a glucuronide or other conjugate. The studies also indicated the formation of glutathione conjugates (Mehendale et al., 1975).

Hexachlorobenzene was slowly decomposed by a mold that was capable of decomposing lindane. The only metabolite detected after 52 days was pentachlorobenzene. When pentachlorobenzene was added to a culture of the mold, degradation produced the following metabolites: pentachlorophenol; 2,3,4,5- and 2,3,4,6-tetrachlorophenol; 1,2,3,4-tetrachlorobenzene; 1,2,4,5- and/or 1,2,3,5-tetrachlorobenzene; 2,3,4-, 2,4,6- and 3,4,5-trichlorophenol; and 1,3,5-trichlorobenzene (Engst et al., 1975).

Irradiation of HCB by UV (λ>290nm) in quartz produced CO_2, HCl and Cl_2 (Gab et al., 1975a).

When sheep were dosed with HCB, residues in omental fat were approximately proportional to dose rates. Although about 1000 times lower in HCB concentration, blood also reflected the residue in fat. Similarly, tissue levels in pigs and chickens reflected feeding levels of HCB. The half-life for HCB in sheep, chickens and pigs was 10 to 18 weeks, 8 to 14 weeks and 10 to 12 weeks, respectively (Avrahami, 1975; Avrahami and Steele, 1972a and b).

HINOSAN (Edifenphos) [O-Ethyl S,S-diphenyl phosphorodithiolate]

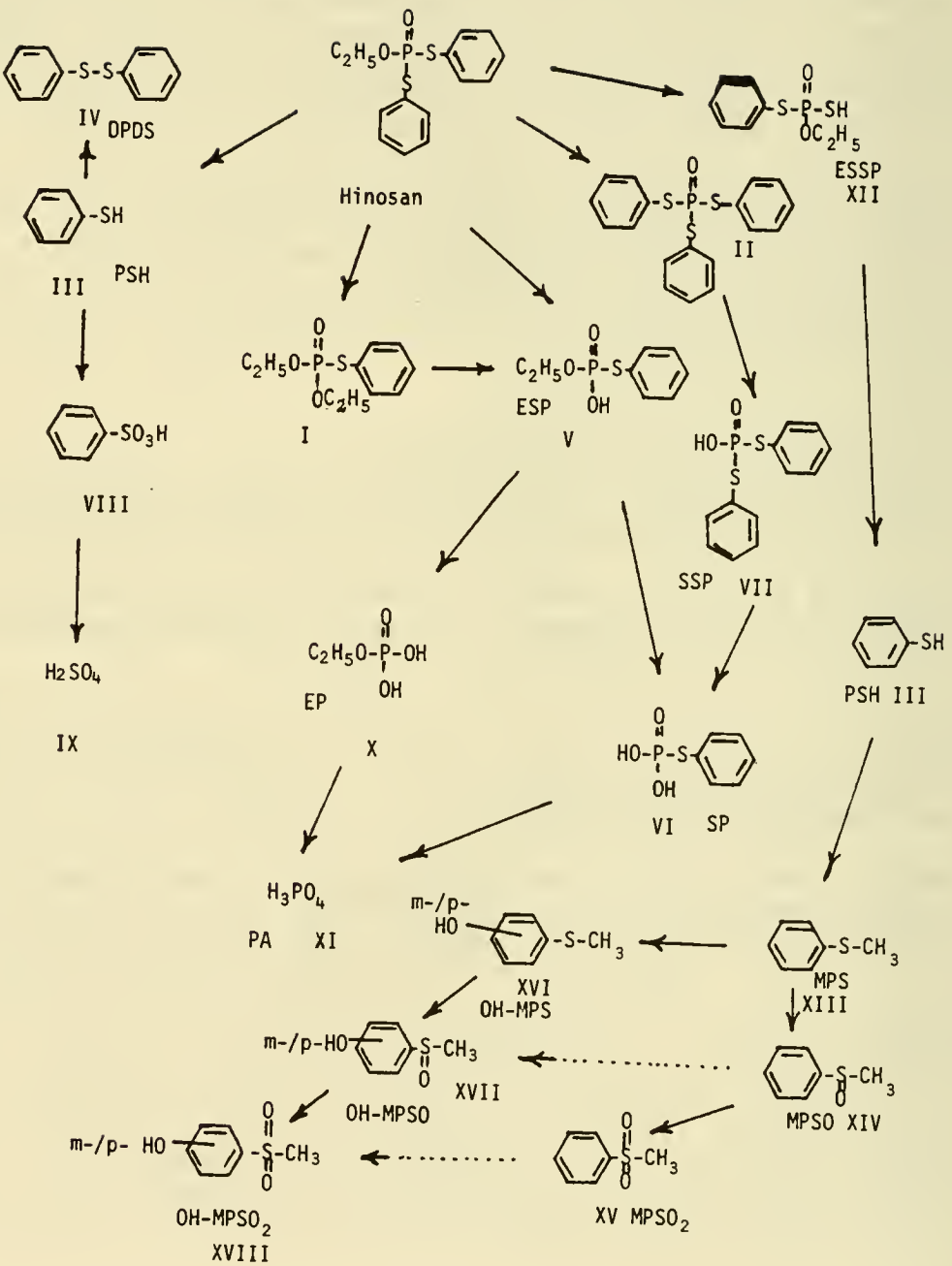
Hinosan was incorporated in fodder and fed to female goats for 10 days. Several goats were also administered hinosan orally via gelatin capsules. Urine, feces and milk were collected for analyses. At a dose level of 1 mg/kg, no hinosan appeared in the milk. At 10 mg/kg, residues appeared at extremely low levels but disappeared rapidly after 3 days. Low residue levels also appeared in tissues after 4 days. Analyses of urine, after administration of 10 mg/kg of hinosan, showed the presence of 13 metabolites but no hinosan was observed. The following metabolites were identified in urine by co-chromatography by TLC and GLC:

O-ethyl S-phenyl hydrogen phosphorothiolate (ESP) (V)
S,S-diphenyl hydrogen phosphorodithiolate (SSP) (VII)
O-ethyl S-phenyl hydrogen phosphorodithiolate (ESSP (XII)
O-ethyl dihydrogen phosphate (EP) (X)
S-phenyl dihydrogen phosphorothiolate (SP) (VI)
phosphoric acid (PA) (XI)
diphenyl disulfide (DPDS) (IV)
methyl phenyl sulfide (MPS) (XIII)
methyl phenyl sulfoxide MPSO) (XIV)
methyl phenyl sulfone ($MPSO_2$) (XV)
m- and p-(hydroxyphenyl)methylsulfoxide(m- and p-(OH)-MPSO) (XVII)
m- and p-(hydroxyphenyl(methylsulfone(m- and p-(OH)-$MPSO_2$) (XVIII)

Since some of the metabolites were not determined without acid hydrolysis, they were probably conjugated. In feces, only $MPSO_2$ and some unchanged hinosan were observed (Ueyama and Takase, 1975).

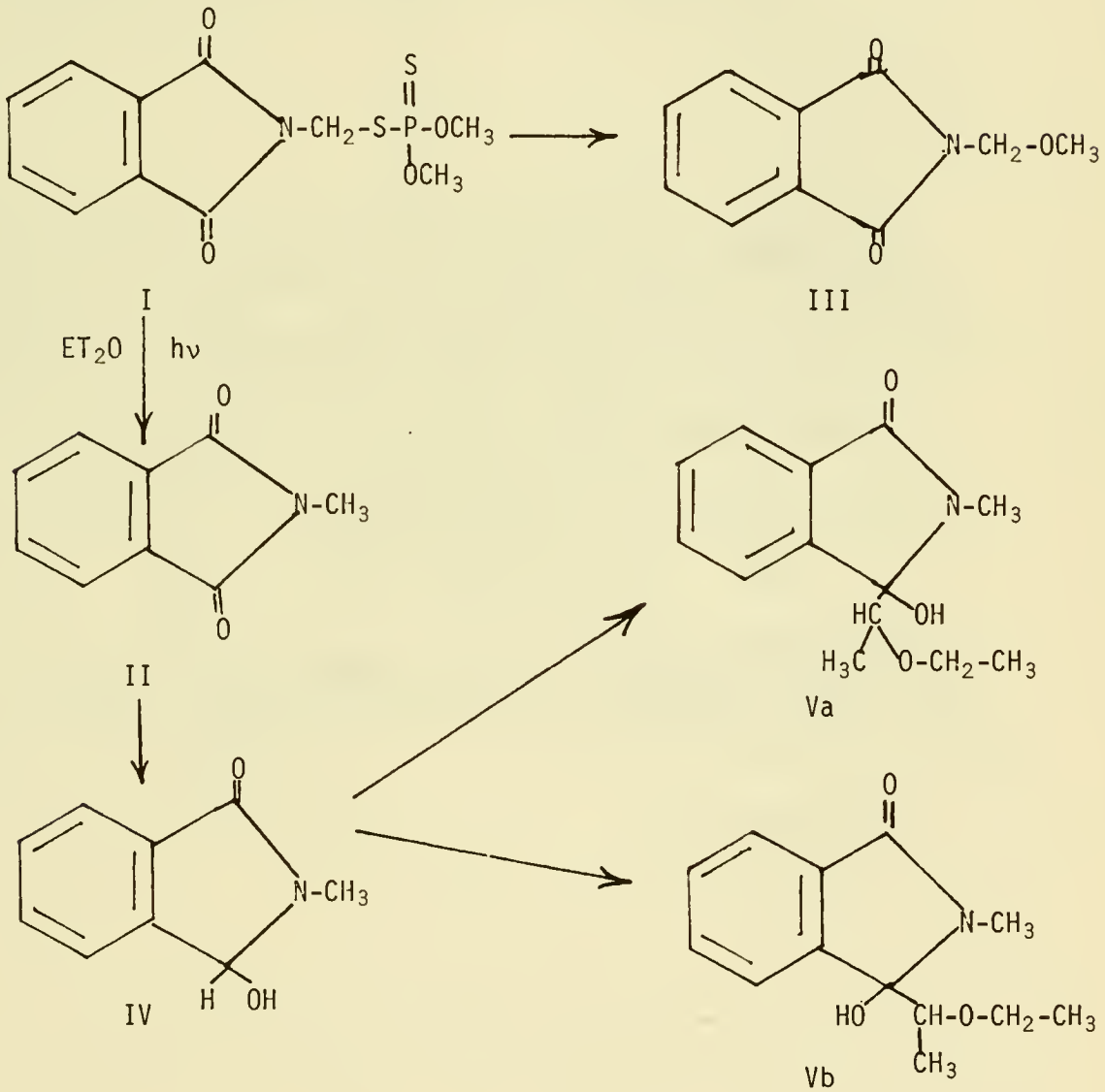
When administered to a rat and dog, hinosan was rapidly metabolized. Diphenyl disulfide was found in urine but benzenethiol was not observed. The O-conjugate of hydroxyphenyl methyl sulfone was also present (Eben and Kimmerle, unpubl., 1972).

When applied to rice plants (Oryza sativa L. v. hatsukinode and v. jukkoku), hinosan persisted somewhat longer than many phosphorus insecticides. The half-life on rice leaves was about 4 days (Ishizuka et al., 1973). Degradation of hinosan was primarily by cleavage of the P-S bond. ^{35}S- and ^{32}P-labeled hinosan was used to elucidate the metabolic pattern. Co-chromatography indicated the presence of O,O-diethyl S-phenyl phosphorothiolate (I), triphenyl phosphorotrithiolate (II), diphenyl disulfide (IV), S-phenyl dihydrogen phosphorothiolate (VI), O-ethyl S-phenyl hydrogen phosphorothiolate (V), S,S-diphenyl hydrogen phosphorodithiolate (VII), benzenethiol (III), benzenesulfonic acid (VIII), sulfuric acid (IX) and phosphate (XI) (Ueyama et al., 1973). In other studies ethyl phosphate (X) was also observed (Takase et al., 1973).


IV DPDS
III PSH
VIII
IX
Hinosan
I
ESP V
II
ESSP XII
SSP VII
EP X
VI SP
PSH III
PA XI
XVI OH-MPS
MPS XIII
XVII OH-MPSO
MPSO XIV
XV MPSO2
OH-MPSO2 XVIII

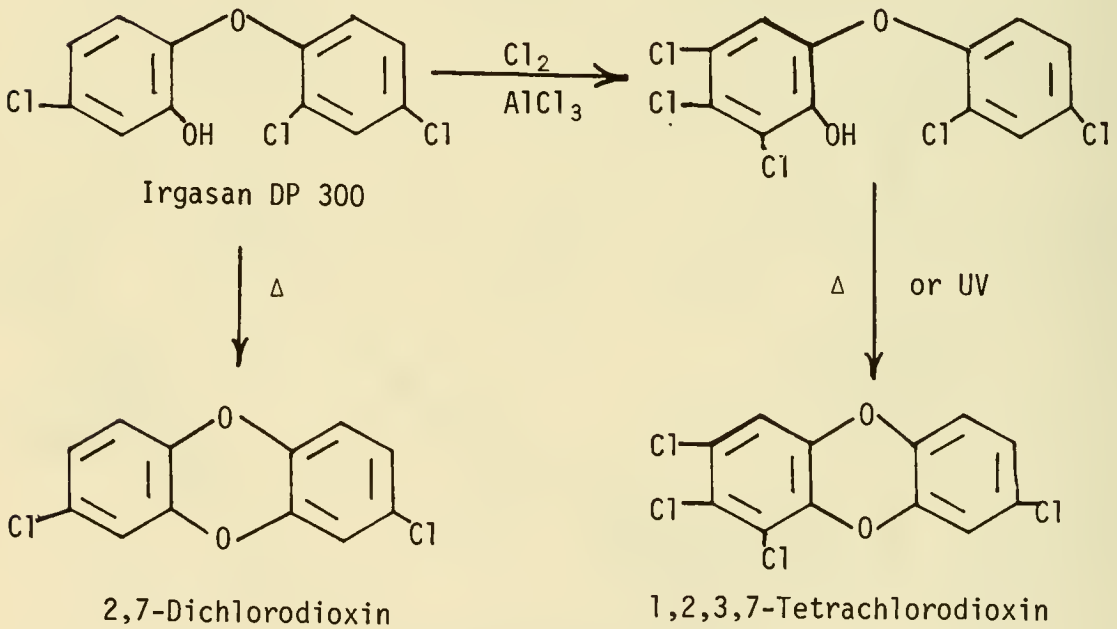
IMIDAN [O,O-Dimethyl S-phthalimidomethyl phosphorodithioate]

In diethyl ether, photolysis of imidan produced N-methylphthalimide (II) and N-methoxy methylphthalimide (III). Further irradiation of N-methylphthalimide produced approximately another six compounds, three of which were identified as 3-hydroxy-2-methylphthalimidine (IV) and the two isomers of 3-(1'-ethoxyethyl)-3-hydroxy-2-methylphthalimidine (V). The latter appear to arise from the reaction of product IV and the ether solvent (Tanabe et al., 1974).



IRGASAN DP 300 [5-Chloro-2-(2,4-Dichlorophenoxy)phenol]

Technical formulations of tri-, tetra-, and penta-chlorophenols contain dimeric impurities. The main constituent of these impurities are 2-phenoxyphenols with 4-9 chlorine atoms. The bactericide known as Irgasan DP 300 contained 2,3,4-trichloro-6-(2,4-dichlorophenoxy)phenol. This compound undergoes ring closure with application of heat or when irradiated with UV (λmax = 290-430 nm). When Irgasan was subjected to heat and irradiation, only heat produced 2,7-dichlorodioxin (Nilsson et al., 1974).



ISOXATHION (Karphos) [O,O-Diethyl O-(5-phenyl-3-isoxazolyl)phosphorothioate]

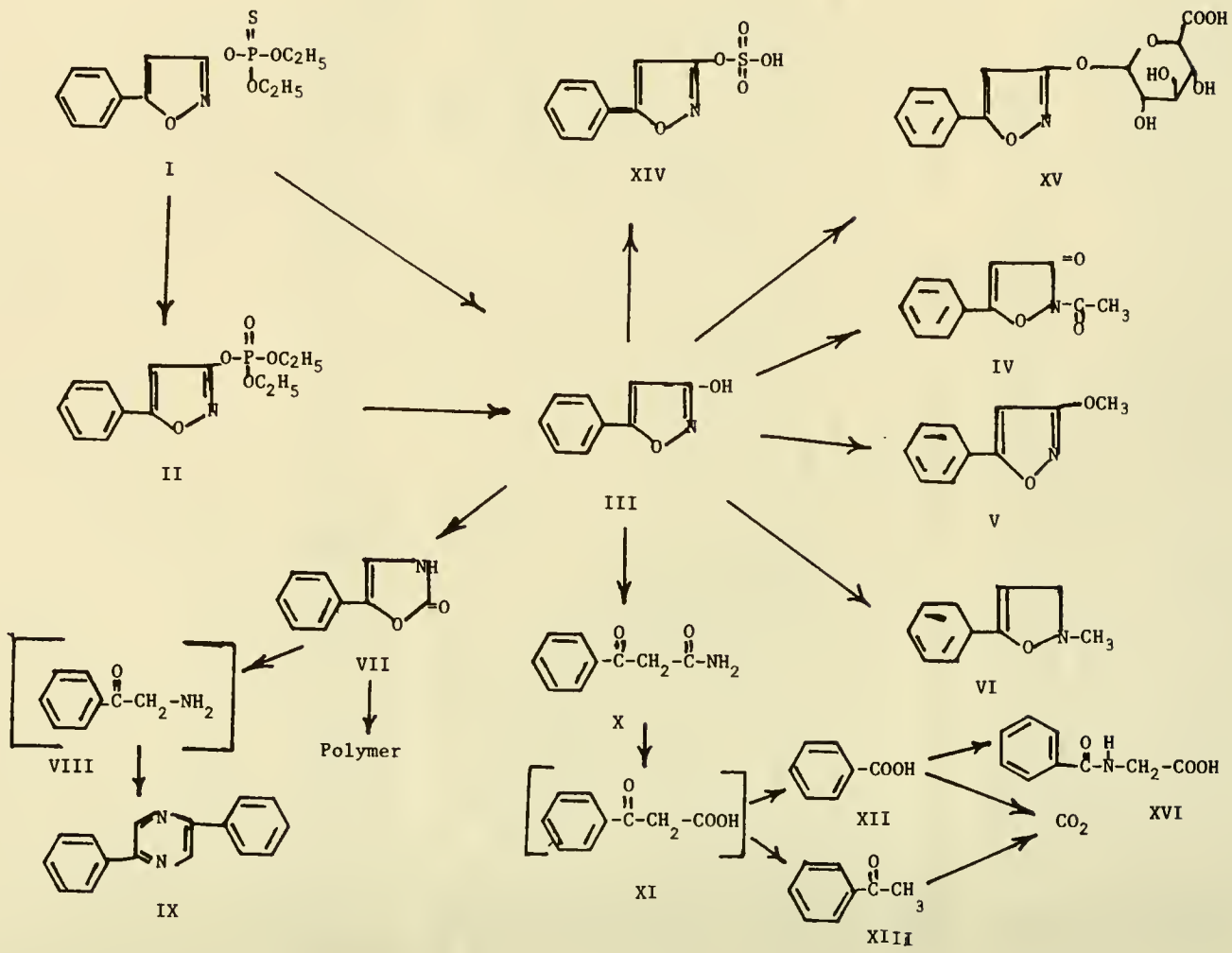
Isoxathion-^{14}C was administered to Wistar strain rats. Radioactivity was eliminated rapidly mainly in the urine. Four major and seven minor metabolies were detected. Because of the small amounts available, the minor products were not identified. The major metabolites were identified as:

(III) 3-hydroxy-5-phenylisoxazole;
(XIV) 5-phenyl-3-isoxazolyl sulfate;
(XV) 3-(β-D-glucopyranuronosyloxy)-5-phenylisoxazole; and
(XVI) hippuric acid.

Compounds III, XIV and XV were also found in the tissues (Ando et al., 1975).

Persistence of isoxathion in soil was influenced by soil type and moisture content with an approximate half-life of 15 to 40 days in nonflooded soils and a much faster disappearance in flooded soil. In addition to CO_2, biochemical degradation produced: 3-hydroxy-5-phenylisoxazole (HPI) (III); the rearrangement product, 5-phenyl-4-oxazolin-2-one (VII); benzoylacetamide (X); and benzoic acid (XII). Six non-persistent metabolites were tentatively identified as: isoxathion oxon (II); 3-methoxy-5-phenylisoxazole (V); 2-methyl-5-phenyl-4-isoxazolin-3-one (VI); 2-acetyl-5-phenyl-4-isoxazolin-3-one (IV); 2,5-diphenylpyrazine (IX); and acetophenone (XI). There were strong indications also of conjugated material. Hydrolysis with boiling 6N HCl for six h released HPI, benzoic acid and α-aminoacetophenone. The latter arises from 5-phenyl-4-oxazolin-2-one and its metabolites or degradation products (Nakagawa et al., 1975).

HPI (III) was stable when exposed to sunlight. When exposed to UV, HPI decomposed to yield primarily 5-phenyl-4-oxazolin-2-one (VII). Benzoic acid (XII) and benzoylacetamide (X) were also produced (Nakagawa et al., 1974).


I
II
III
IV
V
VI
VII
VIII
IX
X
XI
XII
XIII
XIV
XV
XVI
Polymer
CO_2

KEPONE [Decachloropentacyclo[5.3.0.$0^{2,6}$.$0^{3,9}$.$0^{4,8}$]decan-5-one]

MIREX [Dodecachloropentacyclo[5.3.0.$0^{2,6}$.$0^{3,9}$.$0^{4,8}$]decane]

KELEVAN [Decachloropentacyclo[5.3.0.$0^{2,6}$.$0^{3,9}$.$0^{4,8}$]decan-5-ol-5-levulinic acid]

Kepone hydrate was irradiated in cyclohexane with ultraviolet. There were two major products identified as compounds XI and XIa (Alley et al., 1974a).

Mirex was absorbed more rapidly from digestive tract of female quail than of the males and was rapidly excreted. Male quail excreted more via feces than did female quail. No metabolism of mirex was observed (Ivie et al., 1974c). In other studies, after administration of ^{14}C-mirex, the half-life in fat of female and male quail was about 20 and 30 days, respectively, and in whole body of fish, 130 days. In fat of female rats, 10 months after being returned to a "clean" diet, residues of mirex had declined by only 40% (Ivie et al., 1974d). In studies with young leghorn roosters, the amount fed correlated well with amount accumulated over a 20-week period.

If: X = ppm in feed
Y = ppm in fat

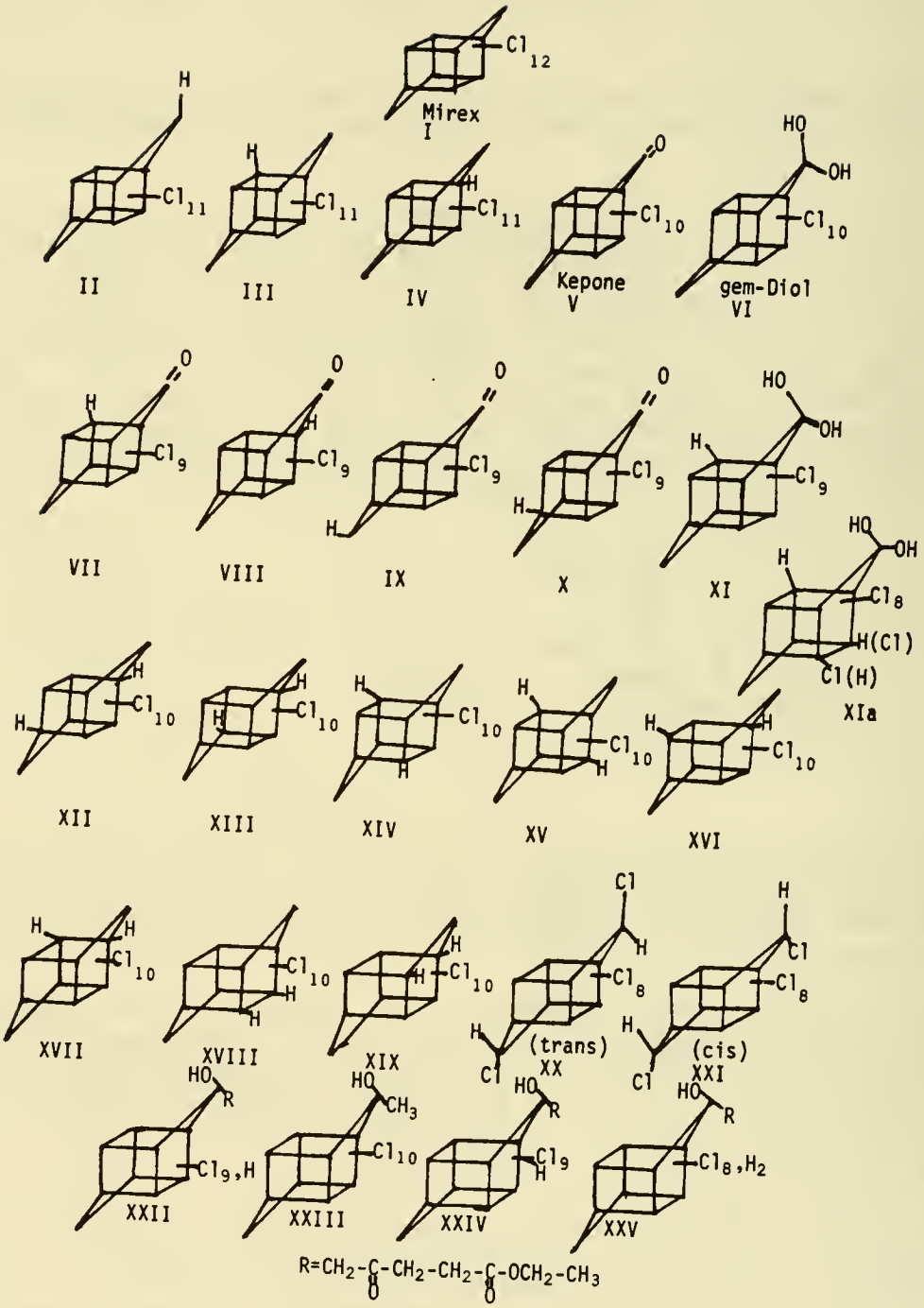
Then: Y = a+bX and
Y intercept a = 1.0508
slope b = +73.7628
correlation coefficient r = +0.9999767

(Medley et al., 1974)

When mallard duck eggs containing mirex were irradiated with UV and γ irradiation, seven and eight products were formed, respectively. Two were identified and tentative identification of two other products was made (Lane et al., 1976).

Cows were fed rations containing mirex. Analyses of fat and milk were then conducted. Residue levels over a 31-week feeding period did not exceed 0.08 ppm in milk and 1.87 in omental fat when mirex was fed at 1.00 ppm (Bond et al., 1975). In eggs of hens fed 1.06 ppm mirex, the residue level reached 2.03 ppm at 28 weeks and then began declining (Woodham et al., 1975).

Anaerobic incubation of sewage sludge with mirex gave indications of degradation. After two months incubation in the dark at 30C, the sludge was centrifuged and the supernatants were extracted. Gas chromatography in three columns, chromatography in two solvent systems


Mirex
I
II
III
IV
Kepone
V
gem-Diol
VI
VII
VIII
IX
X
XI
XIa
XII
XIII
XIV
XV
XVI
XVII
XVIII
XIX
(trans)
XX
(cis)
XXI
XXII
XXIII
XXIV
XXV
R=CH2-C-CH2-CH2-C-OCH2-CH3

on silica gel thin layer plates, and mass spectra were used to identify the metabolite as the 10-monohydro analog of mirex (Andrade and Wheeler, 1974a; Andrade et al., 1975). Other studies with soil microorganisms were conducted with nine aerobic soils and four anaerobic lake sediments. No mirex degradation occurred (Jones and Hodges, 1974).

Photolytic degradation of mirex in cyclohexane or isooctane produced two compounds. The monohydro was narrowed to III or IV; the dihydro is believed to be one of four compounds XII, XIII, XIV or XV (Alley et al., 1973). In other studies, the monohydro photoproduct was identified as compound III (Alley et al., 1974a).

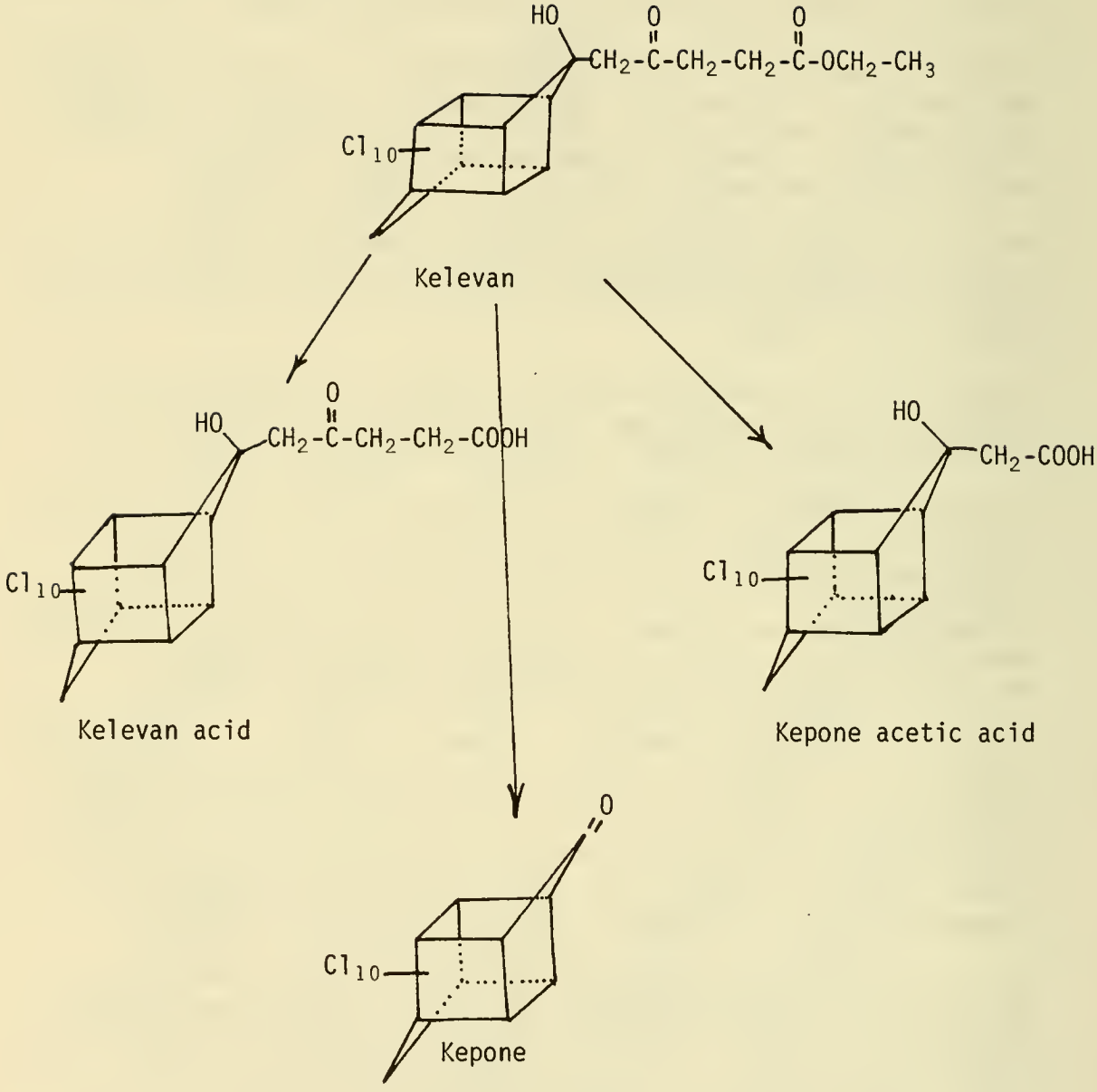
When mirex in triethylamine was irradiated with UV, the major photoproduct was compound II. A second compound was identified as III. A mixture of dihydro photoproducts formed was believed to be XX or XXI (Alley et al., 1974b).

Pyrolysis of mirex produced hexachlorobenzene as the major product and hexachlorocyclopentadiene in small amounts. The vapor phase contained CO, CO_2, HCl, Cl_2, CCl_4 and $COCl_2$ (Holloman et al., 1975).

Mirex was exposed on silica gel thin-layer chromatoplates to sunlight or ultraviolet light. Slow degradation occurred. The major photoproduct was identified as the monohydro derivative III. Another compound more polar than mirex was identified as kepone hydrate (VI). A compound appearing in small amounts was identified as the monohydrokepone hydrate XI. Exposure of compound III to artificial light resulted in conversion to compound VII (Ivie et al., 1974b).

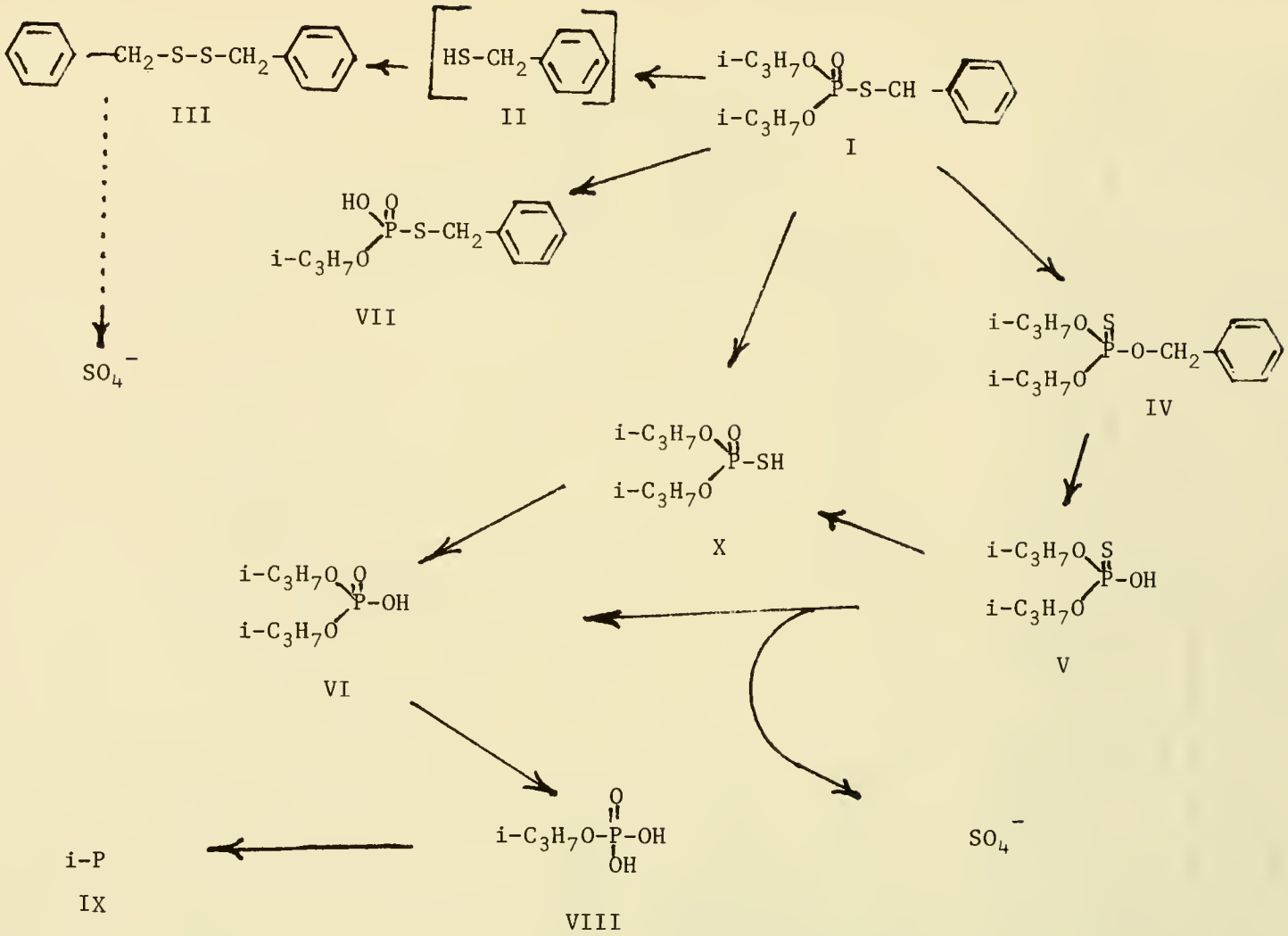
Irradiation (λ>300nm) of kelevan in n-hexane produced compounds XXII, XXIII and V. Identification was made by chromatography, IR and mass spectra. When kelevan was irradiated in methanol, compounds XXIII and the methyl esters of kelevan and XXII were observed. In acetone, XXII was formed. When quartz filter was used, XXIV and XXV were formed. After prolonged (5½ h) irradiation of kelevan, mirex and kepone were found in 15 and 28% yield (Begum et al., 1973).

In other studies, kelevan was applied to potato leaves and to soil. Analysis after 11 weeks indicated the same metabolites were present in both. In addition to unchanged kelevan, kelevan acid, kepone and kepone acetic acid and some unextractable material were present. Similar results were obtained when the soil was analyzed one year later. GC/MS was used for identification (Sandrock et al., 1974).


HO
O
O
CH2-C-CH2-CH2-C-OCH2-CH3
Cl10
Kelevan
HO
O
CH2-C-CH2-CH2-COOH
Cl10
Kelevan acid
HO
CH2-COOH
Cl10
Kepone acetic acid
O
Cl10
Kepone

KITAZIN P [O,O-Diisopropyl S-benzyl phosphorothiolate]

^{32}P and ^{35}S-labeled Kitazin P was applied to rice plants. When applied as a spray, Kitazin P disappeared fairly rapidly. When applied to the water, the disappearance rate increased. The water-soluble metabolites were separated into five fractions. Products identified were: O,O-diisopropyl hydrogen phosphorothioate (V); phosphoric acid (IX); isopropyl dihydrogen phosphate (VIII); diisopropyl hydrogen phosphate (VI); O-isopropyl S-benzyl hydrogen phosphorothiolate (VII); and dimethyl sulfate. When the toluene-soluble fractions were chromatographed, eight metabolites were observed on TLC. O,O-Diisopropyl O-benzyl phosphorothionate (IV) and dibenzyl disulfide (III) were identified by GLC (Yamamoto et al., 1973).


III
II
I
VII
IV
X
VI
V
VIII
IX
i-P
SO_4^-
SO_4^-

LANDRIN [3,4,5- and 2,3,5-Trimethylphenyl N-methylcarbamate]

The persistence of landrin in eight soils was studied. The half-life varied between <4 to >40 days. As pH increased above pH 7, the breakdown rate increased. Although microorganisms played a role in the breakdown, alkaline hydrolysis was a major cause of landrin degradation (Asai et al., 1974).

Photolysis of landrin in ethanol or cyclohexane produced 3,4,5-trimethylphenol (Addison et al., 1974).

N-Lauryl-L-valine

Many organisms could utilize the sodium salt of this compound as carbon and nitrogen sources for growth. *Pseudomonas aeruginosa* AJ 2116 apparently cleaved the N-acyl linkage with release of lauric acid. Gas chromatography also produced two peaks corresponding to caprylic and capric acids. When ^{14}C-labeled N-lauryl-L-valine sodium salt was used, $^{14}CO_2$ was also observed (Shida et al., 1973).

LEPTOPHOS (VCS-506, Phosvel, Abar) [O-(4-Bromo-2,5-dichlorophenyl)-O-methyl phenylphosphonothioate]

After application of leptophos to tomato plants, initially degradation was very slow but accelerated three weeks after treatment. The phenol metabolite increased in fruit and leaves during the initial three weeks but then decreased. The oxon analog was detected in leaves but not fruit. In grapes, results were similar. The phenol increased during the first three weeks and then decreased (Aharonson and Ben-Aziz, 1974).

LUPROSIL [Propionic acid]

Luprosil breaks down completely in the citric acid cycle. Successive reactions involving CoA, methylmalonyl CoA and succinyl CoA bring the luprosil into the cycle (Anon., BASF, 1974).

MALATHION [O,O-Dimethyl S-(1,2-dicarbethoxy)ethyl phosphorodithioate]

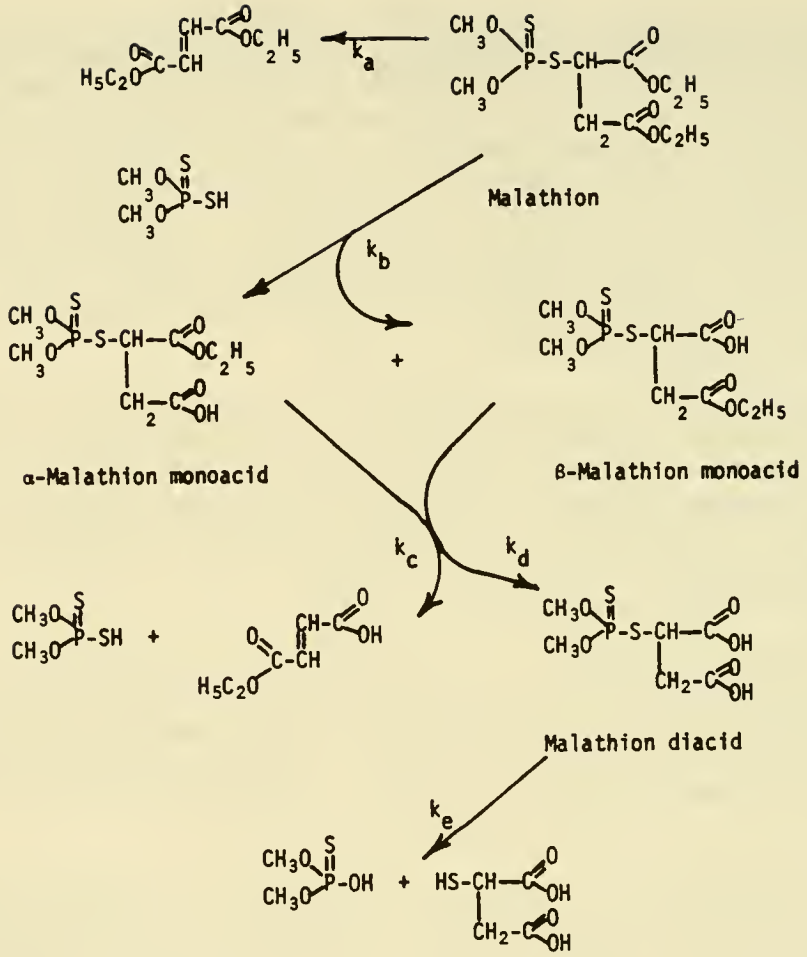
After topical application of ^{32}P-malathion to adult larva of the cotton leaf worm (Spodoptrea littoralis), ^{32}P-activity was distributed between hemolymph, gut and fat. About 23% of the applied dose was metabolized in 24 h. A major site of enzymatic attack occurred at the P-O-alkyl bond and then hydrolysis of the P-S bond to give thiophosphoric acid. Oxidation of P=S to P=O produced malaoxon. Hydrolysis of the esters gave mono- and di- acid derivatives. O,O-Dimethylphosphorodithioic acid was isolated in small amount. Oxidation of this could give rise to the O,O-dimethylphosphorothioic acid found. Dimethyl phosphate was also found. The latter gave rise to monomethyl phosphate and inorganic phosphate (Zayed et al., 1973).

With resistant and non-resistant housefly strains, in vitro studies showed that resistant strains degraded malaoxon oxidatively at a rate 10x higher than that of the susceptible strain. The oxidation product was malaoxon β-monocarboxylic acid when a susceptible strain was used. The resistant strain produced some β-monoacid but the malaoxon α-monoacid was probably the main metabolite. Positive identification, however, was not made (Welling et al., 1974).

Studies with an Arthrobacter sp. showed that this organism was capable of degrading malathion. Laboratory studies identified the metabolites as malathion half ester, the dicarboxylic acid, dimethyl phosphorodithioate and dimethyl phosphorothioate. O-Demethyl malathion was also observed but was non-biological in origin. Identification by TLC was confirmed by infrared spectroscopy (Walker, 1972).

When larval homogenates of a malathion-resistant and malathion-susceptible strain of the Indian meal moth (Plodia interpunctella Hubner) were tested for esterase activity, the resistant strain had greater α-napthyl acetate esterase than the susceptible strain; less carboxylesterase and butyrylcholinesterase; and similar acetylcholinesterase activity (Zettler, 1974).

A heterogeneous bacterial population was isolated from river water and incubated with malathion as a sole carbon source. About 1% of the malathion was converted to the dicarboxylic acid, diethyl maleate, and O,O-dimethyl phosphorothioic acid. The major metabolite was the β-monoacid. The bacteria present were identified as Flavobacterium meningosepticum, Xanthomonas sp., Comamonas terrigeri, and Pseudomonas cepacia (Paris et al., 1975).



			T	pH	$t_{1/2}$
k_b	4.8×10^{-5}	$M^{-1}\ sec^{-1}$	67	4.0	1 yr
k_{a+b}	5.5 ± 0.3		27	8.0	36 h
k_{c+d}	3.1 ± 0.2		27	8.0	24 d
k_e	1.8 ± 0.2		27	8.0	1 yr

(Wolfe et al., 1975)

MALEIC HYDRAZIDE (MH) [1,2-Dihydropyridazine-3,6-dione]

Activated carbon delayed decomposition of maleic hydrazide in soil. Degradation followed first order kinetics. The Freundlich k determined for adsorption on activated carbon was 2300 µg/g (Helweg, 1975).

MATACIL [4-Dimethylamino-3-tolyl-N-methylcarbamate]

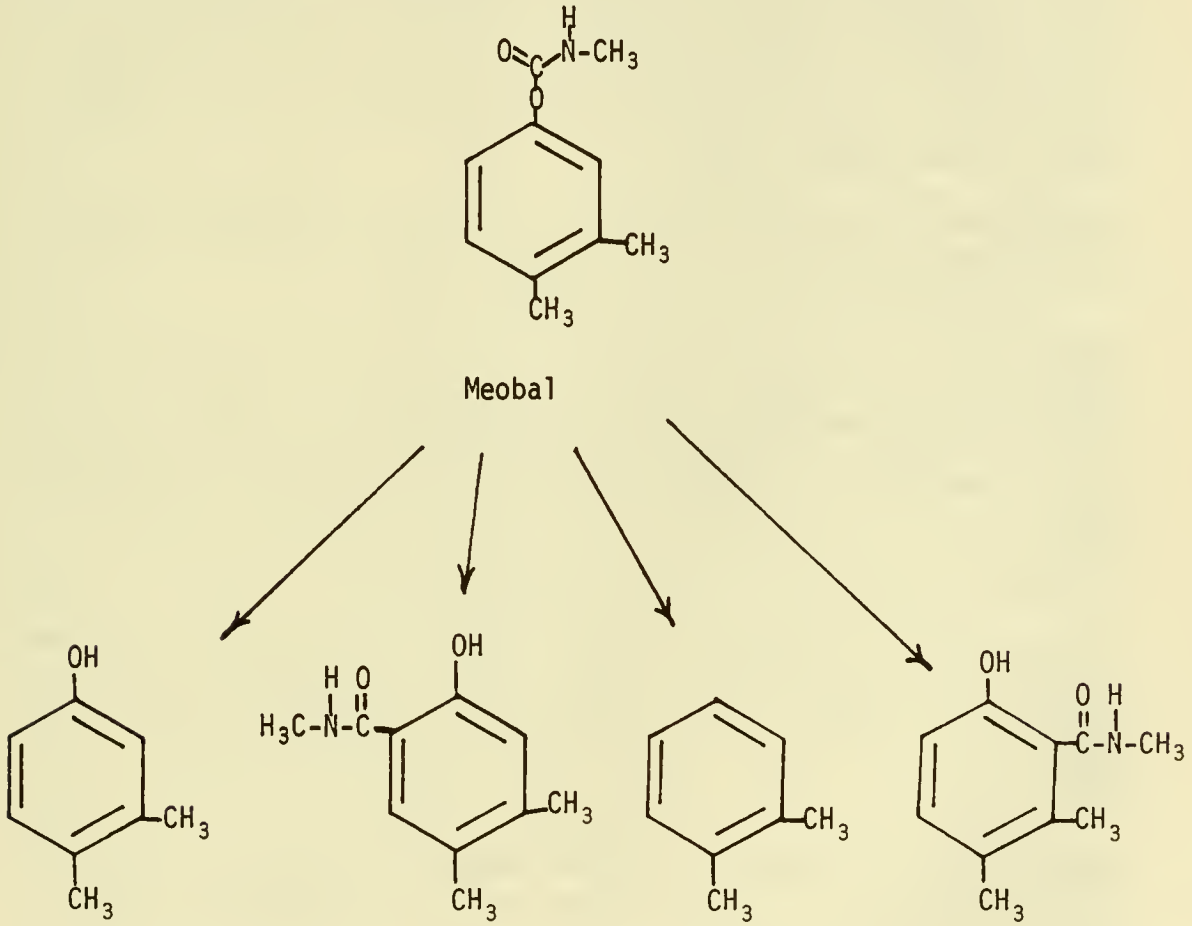
Matacil was added to an ascorbic acid system containing L-ascorbic acid, ferrous ions, EDTA and dissolved oxygen. After 2 h at 37C, the mixture was extracted and analyzed. Isolated compounds were identified by co-chromatography and/or IR and MS. About 12 compounds formed. Seven of 12 products could not be identified. One was identified as 4-amino-3-tolyl-N-methylcarbamate. Tentative identification was made for two compounds: hydroxy matacil and the N-hydroxymethyl matacil. Another compound co-chromatographed with 4-monodemethylamino matacil. IR and mass spectrometry confirmed its identity. Another compound was identified as 4-N-dimethylamino-3-methylphenol (Balba and Saha, 1974).

The major photoproduct from matacil irradiation (λ>300nm) in ethanol or cyclohexane was 4-dimethylamino-3-methylphenol (Addison et al., 1974).

MEOBAL [3,4-Xylyl-N-methylcarbamate]

Photolysis of meobal in cyclohexane produced only phenol and some polymer. In ethanol, in addition to the 3,4-dimethylphenol, three other compounds were identified:

4,5-dimethyl-2-hydroxy-N-methylbenzamide;
2,3-dimethyl-6-hydroxy-N-methylbenzamide; and
o-xylene (Kumar et al., 1974).



In studies with 48 patients who had ingested seeds treated with mercurials, methyl mercury was determined and its biological half-life in man was calculated to vary from 35 to 189 days with an average of 72 days (Al-Shahristani and Shihab, 1974).

In the squirrel monkey, four days after administration of a single dose of methyl mercury chloride, blood and brain concentrations came into equilibrium. The biological half-time was found to be about 49 days and 134 days, respectively, in blood and whole body. Biotransformation of the methyl mercury produced inorganic mercury. In the liver, 20% of the total mercury was inorganic; in the kidney, 50%; in the bile, 30% to 85%; but in the brain less than 5% of the mercury was inorganic (Berlin et al., 1975).

Biotransformation of methyl mercury in the guinea pig produced a high mercuric level in the kidney and a low liver level (50% and 5%, respectively) (Iverson and Hierlihy, 1974).

After exposure of bovine erythrocytes to methyl mercury chloride, rapid and almost complete uptake of mercury occurred. Over 90% of the mercury penetrated the bovine cell membrane and associated with intracellular hemoglobin. Various sulfhydryl agents elicited release from cells. Cysteine alone induced a transient release of methyl mercury from erythrocytes but did not facilitate equilibrium with albumin. Rat red cells released much less methyl mercury to albumin than human red cells (White and Rothstein, 1973).

Single doses of ^{203}Hg-methyl mercury chloride were administered into the rumen of a milk goat and a milk cow. Less than 20% of the dose appeared in the feces within 72 hours and no radioactivity was detected in the cow's milk. The half-time retention of the ^{203}Hg from methyl mercury chloride was 22 days in goats (Sell and Davison, 1975).

Methyl mercury chloride was incubated with intact and ghost erythrocytes and reticulocytes of adult New Zealand white rabbits. Within 5 min these cells accumulated almost all of the available mercury (Garrett and Garrett, 1974).

In cats administered a single oral dose of ^{203}Hg-labeled methyl mercury chloride, the half-period of methyl mercury for whole body was 117.7 $\pm$ 1.4 days including hair and 76.2 $\pm$ 1.6 days excluding the hair (Hollins et al., 1975).

After injection of ^{203}Hg-methyl mercuric chloride into adult Wistar rats, 3% to 6% of the total brain mercury was present in the inorganic form. This was more than from an equal dose of mercuric chloride. Myelin and mitochondrial fractions accumulated more inorganic mercury than other fractions (Syversen, 1974). When $^{14}CH_3{}^{203}HgCl$ was force fed to rats, the amount of C-Hg bond cleavage was calculated to be between 5.1% and 10.6% in blood fractions. Highest concentration of mercury was in the hemoglobin one day after force-feeding (Garcia et al., 1974a). In milk of lactating Sprague-Dawley rats which had been force fed labeled methyl mercury chloride, there was an average of 4.5% bond breakage in the milk; 6.2% in the cerebrum; 6.2% in the liver; 8.0% in the kidney (Garcia et al., 1974b). Brains of Wistar rats, given CH_3HgCl intraperitoneally every second day from 5 to 27 days of age, were analyzed. The myelin fraction contained a larger proportion of inorganic mercury than found in other fractions (Syversen, 1974). In rat bile, the principal methyl mercuric compound observed was methyl mercuric glutathione. This compound also formed in vitro with bile. A small amount of methyl mercuric cysteine also occurred in the bile. This increased with storage of the bile (Refsvik and Norseth, 1975). When N-acetyl-homocysteine was intravenously administered, after methyl mercury chloride, urinary excretion of mercury increased. The corresponding thiolactone turned out to be more effective in removing mercury from the body (Aaseth, 1975). Administration of methyl mercuric chloride in the presence of selenium greatly increased the concentration of the mercury in the brain of rats and reduced uptake by kidneys (Chen et al., 1975).

Methyl mercury-203 was orally administered to Jersey cows. About 59% was absorbed. Tissue concentrations were kidney > liver > skeletal muscles > heart > smooth muscle > spleen > lung > brain > ovaries > pancreas. Of the total mercury body burden, 72% was in muscle and 7% in liver and only 0.17% appeared in milk in 14 days (Neathery et al., 1974).

Aspergillus niger and Penicillum notatum were able to grow and reproduce in limited amounts of methyl mercury chloride. Twenty-five and 20 μg Hg per gm fungal tissue was absorbed (Hardcastle and Mavichakana, 1974).

Bacteria were obtained from river bottom sediments in an area highly polluted with inorganic mercury. When incubated with methyl mercury chloride, mineralization of the mercurial occurred (Billan et al., 1974). Some enteric bacteria were capable of causing volatilization of ^{203}Hg-methyl mercuric chloride (Schottel et al., 1974). When methyl mercuric chloride was anaerobically incubated with human feces, Ch_3Hg^+ disappeared at a constant rate during a 7-day test. Methane was not observed (Edwards and McBride, 1975).

In studies with plasmalogens, methyl mercury chloride was soluble in this phospholipid. The methyl mercuric ion catalyzed rapid hydration and hydrolysis of the vinyl ether linkage to give a mixture of palmitic and stearic aldehydes plus the linolenic monoglyceride product (Segall and Wood, 1974).

Guppies accumulated methylmercury from solution but converted very little to inorganic mercury. When the guppy (Labistes resticulatus) and coontail (Ceratophyllum demersum) were exposed to water containing ^{203}Hg-ethyl-mercuric chloride (EMC), the uptake of EMC was related to exposure time and concentration. Internal organs of the guppy contained the highest concentration of ^{203}Hg and the half-life of ^{203}Hg was about 20 to 23 days. The guppy and coontail were both capable of converting EMC to inorganic mercury (Fang, 1974).

After a single oral dose to rats, methylmercury dicyandiamide was slowly excreted in feces and urine, primarily in organic form. Methylmercury dicyandiamide slowly broke down in kidneys and liver to inorganic mercury. Methylmercury dicyandiamide was rapidly absorbed into circulation and bound by the tissues, particularly by blood cells (Rusiecki and Osicka, 1972).

Methylmercury hydroxide was shown to have a lower affinity than Cd^{++} or Hg^{++} for thionein.

When $Hg(OAc)_2$ was intravenously injected into laying quail, the mercury was bound to lipovitellin and transported into ovarian follicles (Nishimura and Urakawa, 1972). A microorganism, found in activated sludge and identified as Pseudomonas ovalis, tolerated mercury acetate (Tomoyeda et al., 1973). In other studies, R-factor systems in enteric bacteria were able to reduce Hg^{++} from $Hg(OAc)_2$ to elemental mercury (Schottel et al., 1974). When solutions of mercuric acetate were irradiated with a 20 watt black-light having the spectral distribution of sunlight, methylmercuric compound formed. In this reaction mercuric oxide can replace mercuric acetate. The studies indicated that mercuric acetate was hydrolyzed to mercuric oxide and acetic acid in water. The mercuric oxide stimulated the light-induced methylation of inorganic mercury (Akagi and Takabatake, 1973).

A metallic mercury-releasing enzyme (MMR-Enz), which catalyzes the reduction of mercurials to metallic mercury, was induced when Pseudomonas sp. were incubated with PMA, PCMB, merzonin, mercuric chloride, and metallic mercury (Furukawa and Tonomura, 1972).

In a study of organisms capable of degrading methyl mercury, 207 organisms from sediments and fish were screened with methyl mercury bromide. Thirty isolates were capable of degrading methylmercury with volatilization of labeled mercury (Spangler et al., 1973a). In addition to Hg^0, methane also formed (Spangler et al., 1973b).

Liver preparations from rat, mouse, and guinea pig degraded methoxyethylmercury chloride (MEMC) with formation of ethylene. No evidence of Hg^0 formation was observed. Preparations from rat brain and kidneys of rat, guinea pig, ferret, and chicken also degraded MEMC (Lefevre and Daniel, 1973).

When phenylmercuryacetate (PMA) was incubated with liver preparations from rat, mouse, guinea pig, ferret, and chicken, benzene was formed. Brain preparations from rat, guinea pig, and ferret and kidney preparations of rat, guinea pig, ferret, and chicken also degraded PMA (Lefevre and Daniel, 1973). A number of bacterial isolates were tolerant of PMA (Tomoyeda et al., 1973) or capable of volatilizing ^{203}Hg-PMA (Schottel et al., 1974). In addition to Pseudomonas sp., Arthrobacter sp., Citrobacter sp., Enterobacter sp., Vibrio sp., and Flavobacterium sp. also degraded PMA. Elemental mercury vapor and benzene were observed products of degradation (Nelson et al., 1973). When guppies, snails, elodea, and coontail were exposed to ^{203}Hg-PMA in water, PMA was readily taken up and converted mainly to inorganic mercury. Small amounts of ethylmercuric chloride (EMC) were also formed. The biological half-life of ^{203}Hg in guppies, coontail, and elodea was between 43 and 56 days but was dependent on the initial concentration. At higher concentrations the half-life was between 7 and 11 days. In snails, the biological half-life was about 10.8 days (Fang, 1973). When river sediments were incubated with PMA, some methylmercury formed. More was formed under more acidic conditions (Jacobs and Keeney, 1974).

Phenyl mercuric salts were converted to diphenylmercury as the main product. Simultaneously, phenylmercuric chloride was produced in amounts related to the amount of chloride contaminations (Dressman, 1972).

Incubation of liver preparations of rat, mouse, guinea pig, ferret, and chicken with p-chloromercury benzoate (PCMB) produced benzoic acid. Rat brain and rat, guinea pig, and chicken kidney preparations also degraded PCMB (Lefevre and Daniel, 1973).

Photolytic half-lives of phenyl mercurials are summarized:

Compound	$t_{1/2}$, hrs.
Diphenyl mercury	8.5 ± 1.8
PMA	16.0 ± 2.0
PMN	20.0 ± 1.0
Phenyl mercury BO_3	14.0 ± 2.0
Phenyl mercury hydroxide	16.0 ± 2.0

(Zepp et al., 1973).

The mechanism of mercury elimination in waste water was studied. Mercury removal rates were over 99.8% when waste water containing mercuric chloride was treated with acclimated sludge. It was found that the added mercuric chloride was removed rapidly by volatilization after reduction to metallic mercury. Optimum pH and temperature were 8 to 9 and 42 to 43C, respectively (Nakamura et al., 1974a,b). In other studies, the mercury-resistant bacterium Pseudomonas K62 strain was incubated in culture medium for 6 h with various mercurials. Results are summarized in the following table.

Mercury removal by Pseudomonas K62 ($6x10^8$ cells/ml)

Hg-Compound Added	Concentration (ppm)	% Removal added mercury	
		Without Ps. K62	With Ps. K62
$HgCl_2$	30	11	65
$Hg(CN)_2$	30	0	72
$Hg(NO_3)_2$	30	0	47
$Hg(OAc)_2$	30	15	45
$HgSO_4$	30	25	73
$Hg(SCN)_2$	30	9	55
HgI_2	30	14	69
HgO	15	29	69
PMA	100	0	80

Uptake of mercury in these studies was severely inhibited by sodium chloride, sodium nitrate, KH_2PO_4 and K_2HPO_4 (Suzuki et al., 1968).

Mature specimens of dungeness crabs, Cancer magister, were exposed to dissolved inorganic mercury in aquarium water, returned to unpolluted sea water, and then analyzed for total mercury. Experimental data indicated that inorganic mercury has a biological half-life of 20-25 days in the dungeness crab (Sloan et al., 1974). In the mollusc, Tapes decussatus, the half-life was 5-10 days (Unlii et al., 1972).

When elemental mercury was incubated with pure culture of micro-organisms, oxidation and accumulation of mercury occurred. Six cultures were tested:

P. aeruginosa
P. fluorescens
Citrobacter sp.
E. coli
B. subtilis
B. megaterium

Concentration factors calculated for E. coli, P. fluorescens, and Citrobacter sp. were 196, 1202 and 222, respectively. The distribution of mercury in aquatic biota from a stream receiving a continuous input of Hg^{++} was also determined. Dragonfly nymphs (Neurocordulina alabamensis) and damselfly nymphs (Argia sp.) exhibited highest total

mercury levels. Methylmercury was highest in mosquito fish (Gambusia affinis), predaceous diving beetles (Dytiscidae) and water boatmen (Hesperocorixa sp.). No methylmercury was found in algae, fungi and bacteria (Holm and Cox, 1974).

Studies with bovine serum albumin indicated that mercury (II) was bound at sites in addition to the carboxyl and thiol groups (Katz and Samitz, 1973). Within 5 min after exposure to mercuric chloride, intact and ghost erythrocytes and reticulocytes accumulated approximately 30% (intact) and 50% (ghost) of the available mercury (Garrett and Garrett, 1974). Kinetic studies of mercuric chloride indicated that mercury was contained in three compartments of short, medium, and long retention time within the rat. Kidneys were the largest compartment for mercury and kidney retention probably accounted for the long-term compartment. The biological half-life was about 30-33.5 days (Phillips, 1972).

When a goat was given $^{203}HgCl_2$, less than 30% of the dose was absorbed. Excretion of ^{203}Hg in milk accounted for 0.22% of the dose. Half-time of retention by goats of ^{203}Hg given $^{203}HgCl_2$ was 78 days (Sell and Davison, 1975).

Suspensions of rat caecal and small intestinal contents were incubated with $HgCl_2$. Analyses indicated that these materials were able to synthesize methylmercury (Rowland et al., 1975).

The ability of algae to grow in media containing $HgCl_2$ was studied. Lag periods of 3 or more days were observed. The growth rate was then similar to that of controls without Hg. The rate of decrease of mercury content was not dependent on initial Hg concentrations except at the lowest concentration (2 μM) (Ben-Bassat and Mayer, 1975).

In the presence of sublethal amounts of $HgCl_2$, small amounts of methylmercury were produced during 7 days aerobic growth by the following bacteria:

Pseudomonas fluorescens
Mycobacterium phlei
Escherichia coli
Aerobacter aerogenes
Bacillus megaterium

and by mycelium of the fungi:

Aspergillus niger
Scopulariopsis brevicaulis
Saccharomyces cerevisiae

A yeast, isolated from a stream and identified as Cryptococcus sp., was grown in media containing $HgCl_2$. Analyses indicated the presence of high levels of mercury in viable cells. The form of the mercury was not determined but is believed to be elemental (Brunker and Bott, 1974).

When $^{203}HgCl_2$ was incubated anaerobically with human feces, methylmercury was produced in amounts directly related to the amount of Hg^{+2} added. The disappearance of methylmercury occurred at a constant rate during the 7-day test. $^{14}CH_4$ was not observed (Edwards and McBride, 1975).

One mercury resistant strain of E. coli converted 95% of 10^{-5} M Hg^{+2} ($HgCl_2$) to metallic mercury at a rate of 4 to 5 n moles Hg^{+2}/min/10^8 cells. Metallic mercury was eliminated as a vapor (Summers and Silver, 1972). In addition to the E. coli, S. aureua and P. aeruginosa were also capable of carrying out these reactions (Summers and Lewis, 1973).

Methylation activity is higher in tuna liver than in other fishes. Fractionation studies with tuna liver strongly suggested that the factor was methylcobalamin, a known methyl donor in many biological systems (Pan et al., 1973).

Studies have shown that Hg^{+2} may be non-enzymatically reduced to elemental mercury by humic acid (Alberts et al., 1974) or by reducing agents such as ethylene and acetylene (DeFilippis and Pallaghy, 1975).

In the presence of sulfur, inorganic mercury may be alkylated in aquatic environments. Sulfur photooxidation to sulfate couples with reduction of mercuric ions. A basic mercuric sulfate formed and was an effective photosensitizer for the methylation (Akagi et al., 1974).

The possibility that sediment materials might cause symmetrization and conversions of monomethylmercurials into dimethylmercury was investigated. Results of this study indicated that alkylmercuric halides are not symmetrized under the test conditions although arylmercuric halides were. The procedure used consisted of placement of the mercurial halide on a basic Al_2O_3 column and eluting with a hydrocarbon solvent (Cross, 1973).

Studies were conducted to determine the kinetics of microbially mediated methylation of mercury in aerobic and anaerobic aquatic environments. From these studies the following was concluded:

1. Methylation can occur under aerobic or anaerobic conditions.

2. Methylation under both conditions is dependent on growth rate or metabolic activity of the methylating organisms, mercuric ion concentration, and availability of mercuric ions.

3. At neutral pH, monomethylmercury is the main product but dimethylmercury forms in small amounts.

4. The rate of formation of the mono- and dimethylmercury can be described by

$$NSMR = \gamma\beta^{n}(Hg_{total})^{n} \qquad \text{where}$$

NSMR = net specific methylation rate
γ = coefficient of microbial activity
β = coefficient of mercuric ion availability
n = reaction order

5. The average reaction order value (n) is 0.15 and 0.28 for anaerobic and aerobic systems, respectively.

6. Methylation is temperature dependent only to the extent that it affects microbial activity.

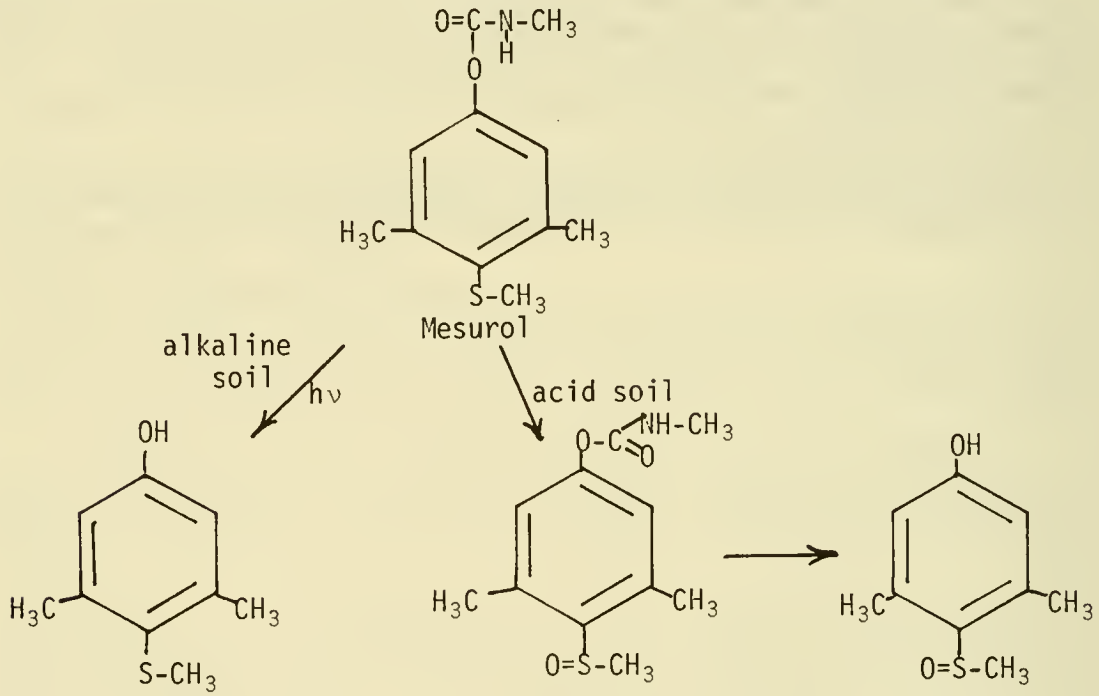
7. Large amounts of Hg^{0} are formed and removed from the aqueous phase when a gas is forced through the system.

(Bisogni and Lawrence, 1975).

MESUROL [4-Methylthio-3,5-xylyl-N-methylcarbamate]

Photodecomposition of mesurol produced only the compound 4-methylthio-3,5-dimethylphenol. This is the same product obtained by basic hydrolysis of mesurol (Kumar et al., 1974).

In alkaline soils, mesurol was rapidly hydrolyzed and CO_2 evolved. Hydrolysis in acid soil was shown. The half-life varied between 4 days at pH 7.6 to more than 56 days at pH 4.1. While hydrolysis was the main route of degradation in alkaline soil, in acid soil the primary route appeared to be oxidation to the sulfoxide prior to hydrolysis to the phenol (Starr and Cunningham, 1974b).



METHAZOLE (Oxydiazol, Probe, VCS-438) [2-(3,4-Dichlorophenyl)-4-methyl-1,2,4-oxadiazolidine-3,5-dione]

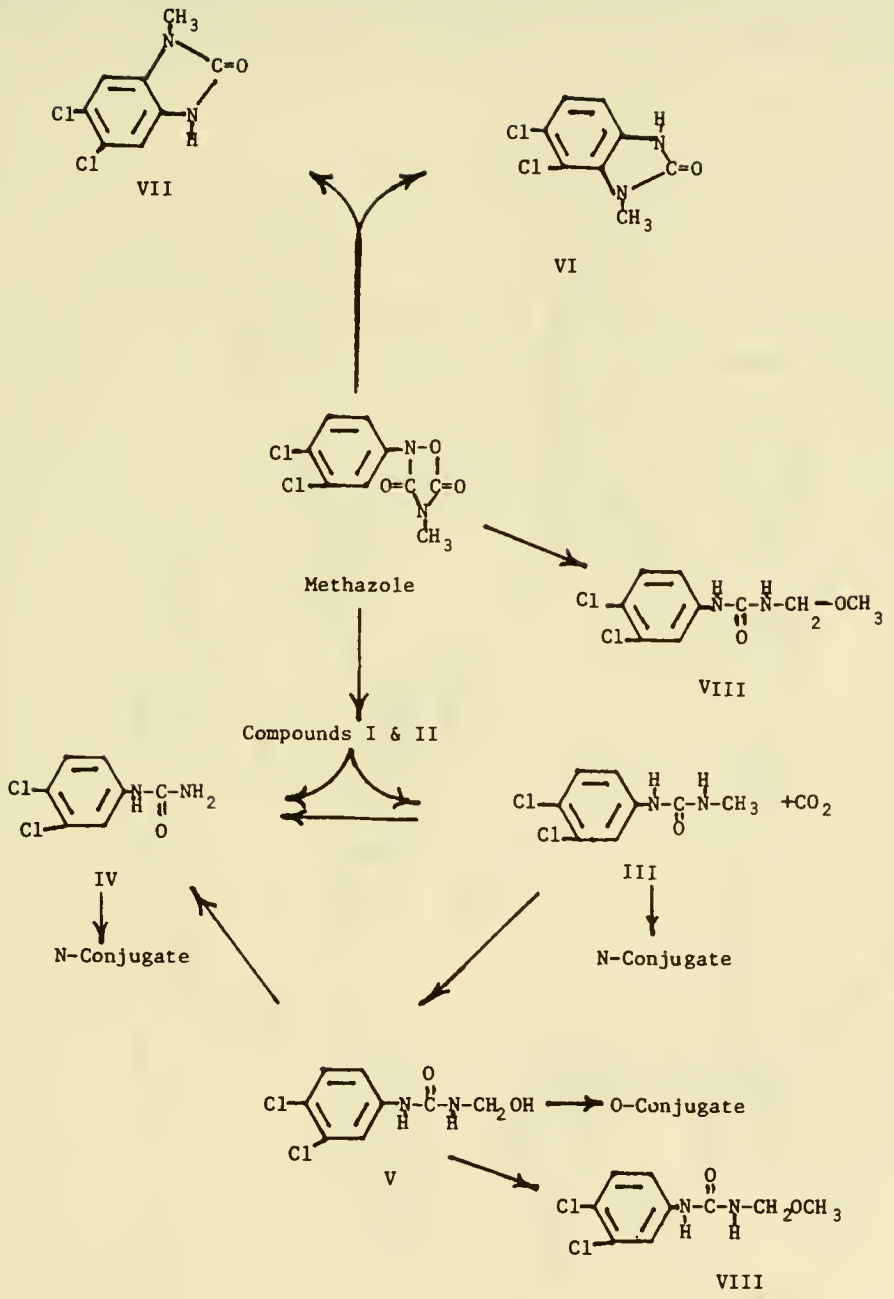
Wheat plants and Bermuda onions were placed in water containing ^{14}C-methazole for 24 h and then removed and analyzed after being washed. Radioautography and ultraviolet light were used to visualize the materials on silica gel. With TLC, about seven compounds were resolved: two metabolites were not identified; however, acid hydrolysis (HCl for 1 h at 90C) released methazole-methylurea (III) and methazole-urea (IV). In onions, metabolite II was in greater concentration than I; in wheat, metabolite I was in greater concentration. Acid hydrolysis of I gave predominantly methazole-urea and small amounts of methazole-methylurea. With metabolite II, the reverse was true. Two metabolites were not identified but did chromatograph similar to 6,7-dichloro-1-methyl-2-benzimidazolinone (VI) and the 5,6-dichloro analog (VII). Other conjugates were present as evidenced by acid treatment of plant solids after methanol treatment and the release of metabolites I, III and IV. In wheat, 50 to 60% of the dose was accounted for by metabolites III and IV. In onions, methazole-methylurea was the predominant metabolite and methazole-urea (IV) was present only at low concentrations. Treatment of wheat seedlings with methazole-3-^{14}C and methazole-phenyl-^{14}C gave similar results. This showed that methazole was metabolized in wheat to form $^{14}CO_2$ and the methylurea metabolite (III). Some methoxymethylurea was also detected in extracts of wheat and onions (Dorough, 1974).

Beans and cotton were treated with labeled methazole. Quantitative rather than qualitative differences were indicated. From cotton, 1-(3,4-dichlorophenyl)-3-methylurea (III) and 3,4-dichlorophenylurea (IV) were obtained. The latter was the major metabolite. In addition to these compounds, the hydroxymethyl derivative and three conjugates were observed (Dorough et al., 1973). In other studies with cotton and prickly sida (Sida spinosa L.), compounds III and IV were also observed (Butts and Foy, 1974).

After application of VCS-438 to cotton (Gossypium hirsutum L. 'Acala 4-42-77'), foliar penetration occurred within 3 h and increased with time. Cotton tissue readily metabolized VCS-438 to 1-(3,4-dichlorophenyl)-3-methylurea (DCPMU) and 1-(3,4-dichlorophenyl)urea (DCPU). When plants were treated through the roots with VCS-438, DCPMU, DCPU and unidentified polar material formed. Digestion of plant residues with the proteolytic enzyme pronase indicated that some of the unextractable ^{14}C may be complexes of DCPMU and DCPU with proteins (Jones, 1972).

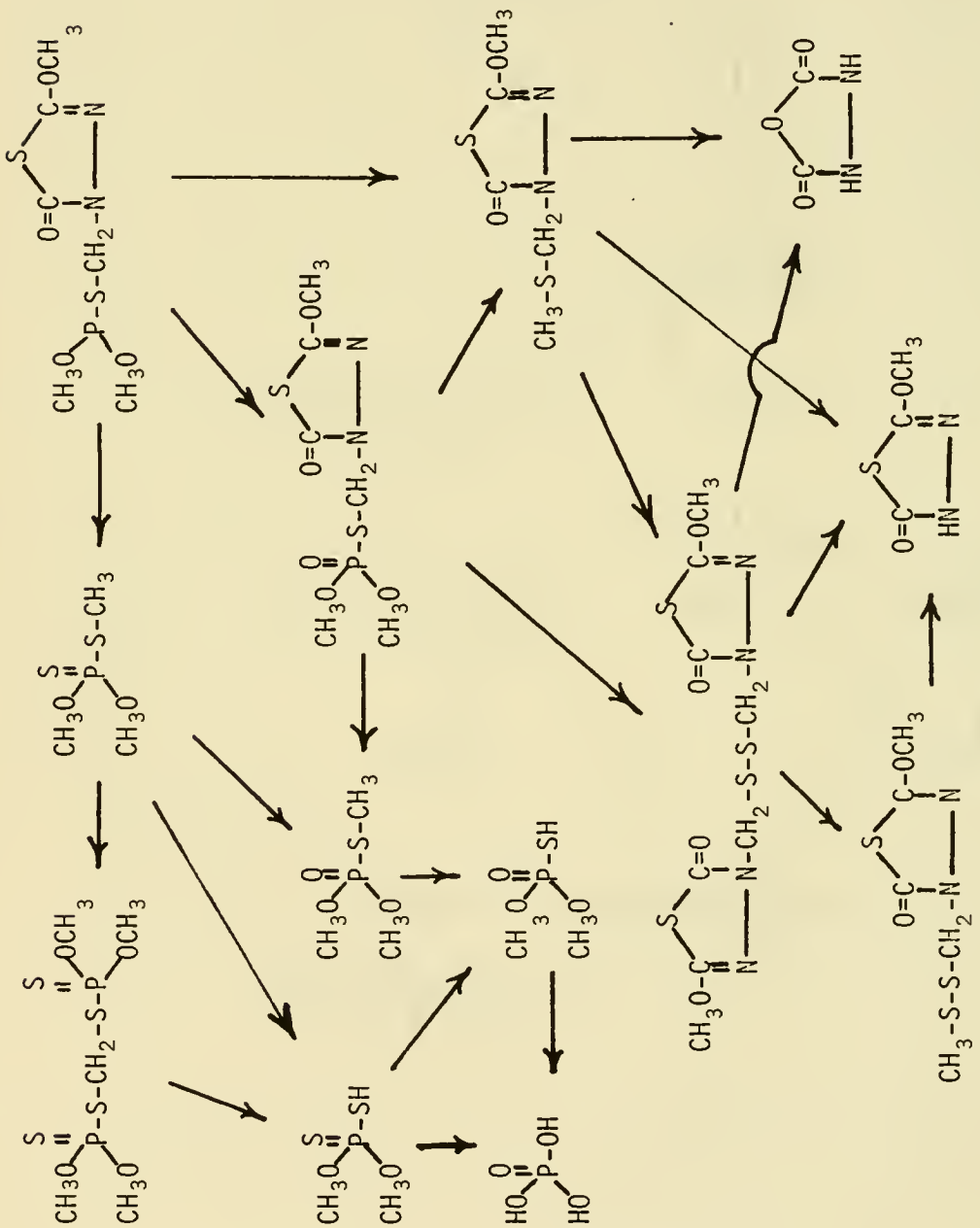
When metabolites I and II were fed to rats, nearly 50% of a single dose was excreted in the urine and 11% in feces within 1 day after treatment; 65 and 21%, respectively, in 6 days (Dorough et al., 1973).

Water solutions of methazole-phenyl-^{14}C were exposed to sunlight for 7 days. Analyses indicated that compounds III, VI and VII were present. In methanol, compounds VI, VII and VIII were observed (Dorough et al., 1973). In other studies, irradiation of methazole in water also formed VI, VII and III and compound VIII in methanol (Ivie et al., 1973).


VII
VI
Methazole
VIII
Compounds I & II
IV
III
+CO2
N-Conjugate
N-Conjugate
V
O-Conjugate
VIII

METHIDATHION (Supracide, GS 13005) [S-(2-Methoxy-5-oxo-Δ^2-1,3,4-thiadiazolin-4-yl)methyl O,O-dimethyl phosphorodithioate]

Methidathion was irradiated with UV of λ = 254 mμ. TLC, IR, mass spectra and synthesis were used to isolate and identify nine products. The proposed breakdown scheme is indicated (Dejonckheere and Kips, 1974).



Methidathion was fed to a cow as a residue in forage. No intact methidathion was found in the milk and was found to be stable in rumen fluid for 24 h. Incubation with beef liver 10,000xg supernatant fraction degraded methidathion 74-86% within 30 min. Urinalyses showed the presence of dimethyl dithiophosphate and dimethyl thiophosphate (St. John, Jr., and Lisk, 1974).

METHOMYL (Lannate, DuPont 1179) [S-Methyl N-(methylcarbamoyloxy) thioacetamidate]

Charles River-CD rats were administered labeled methomyl. $^{14}CO_2$ and acetonitrile were observed. Urine metabolites were not identified (Harvey et al., 1973).

Radiolabeled methomyl was applied to tobacco, corn and cabbage. Rapid degradation occurred to produce CO_2 and acetonitrile with a methomyl half-life of 3 to 6 days. Labeled lipids, Krebs cycle acids, sugars and other materials were also present (Harvey and Reiser, 1973). The half-life on cotton was found to be between 2 and 4 days (Bull, 1974).

Radiolabeled methomyl was injected into 5th-instar cabbage loopers [Trichoplusia ni (Hubner)]. Unidentified water soluble metabolites were formed. Acetonitrile and other volatiles also probably formed (Kuhr, 1973).

In soil, labeled methomyl was degraded to $^{14}CO_2$ and other materials, some of which were reincorporated into normal components of soil organic matter (Harvey and Pease, 1973).

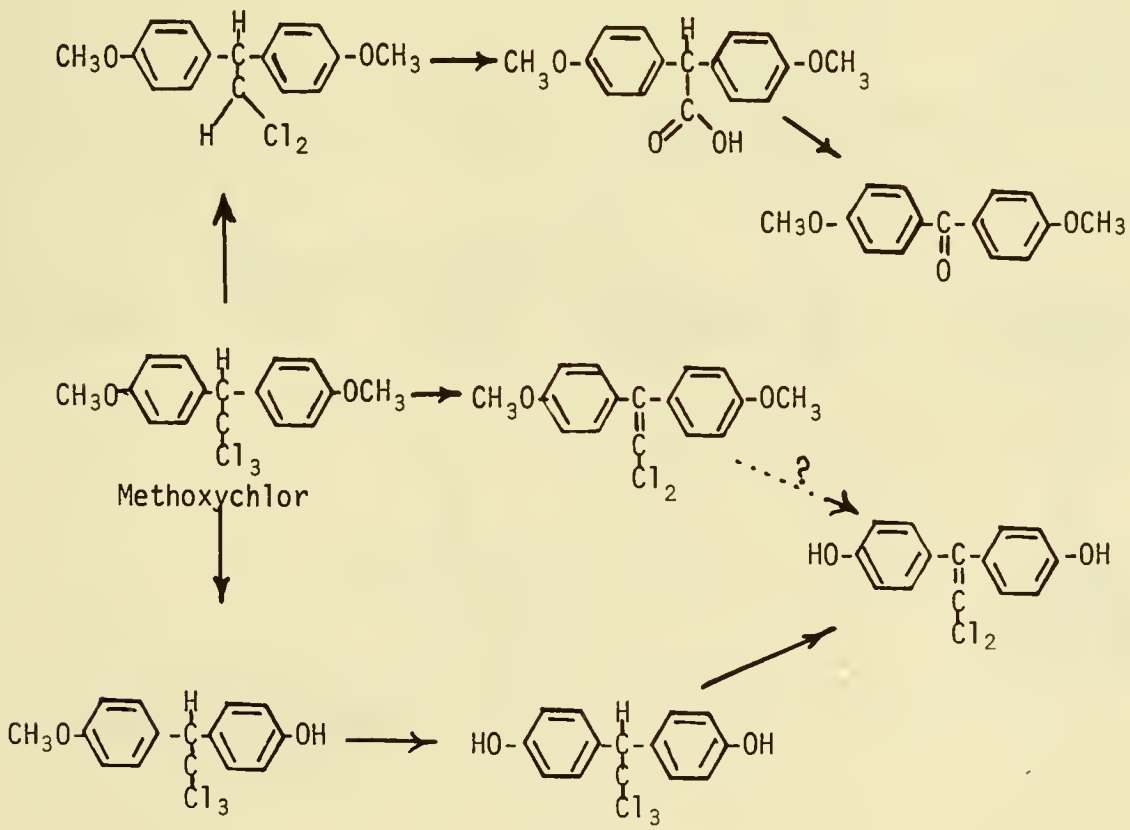
METHOXYCHLOR [2,2-Bis(p-methoxyphenyl)-1,1,1-trichloroethane]

ETHOXYCHLOR [2,2-Bis(p-ethoxyphenyl)-1,1,1-trichloroethane]

When ^{14}C-ring-labeled methoxychlor was incubated with a sheep liver microsomal preparation for 30 min at 39C, two products were identified: 2-(p-hydroxyphenyl)-2-(p-methoxyphenyl)-1,1,1-trichloroethane (8.13%) and 2,2-bis(p-hydroxyphenyl)-1,1,1-trichloroethane (3.44%) (Hirwe et al., 1975).

Chemical decomposition of methoxychlor was slow in water. The half-live at pH 5 to 9 and 27C was 100 days. The photolytic half-life in distilled water was 37 days. In some river waters, methoxychlor photolysis in sunlight was rapid with a half-life of 2 to 5 h. The ethylene analog (DMDE) was formed in each case (Zepp et al., 1975).

Methoxychlor was administered orally to mice in olive oil and topically in acetone to flies. Excrement was collected. Identification of metabolites was by thin-layer chromatography. O-Demethylation was observed with both species and the monohydroxy and dihydroxy metabolites were observed. Similar results were obtained with mouse liver and housefly microsomes (Hansen et al., 1974).



After exposure of the housefly (R_{SP}) and saltmarsh caterpillar (Estigmine acrea) to ethoxychlor, excrement was collected and analyzed. In addition to unidentified conjugates, six metabolites were observed. Studies with microsomal preparations of these two species gave similar results. When mice were used, only conjugates and the O-monodealkylated analog were observed (Table 1) (Kapoor et al., 1972). In another study with mouse liver microsomes, metabolite III was observed (Hansen et al., 1974).

Studies with a model ecosystem are summarized in Table 2. Ethoxychlor was concentrated in the higher trophic levels. In fish, this was 1500 times that in water; and in snails, 98,000 times that in water. The latter compares with a factor of 35,000 for DDT and 120,000 for methoxychlor (Kapoor et al., 1972).

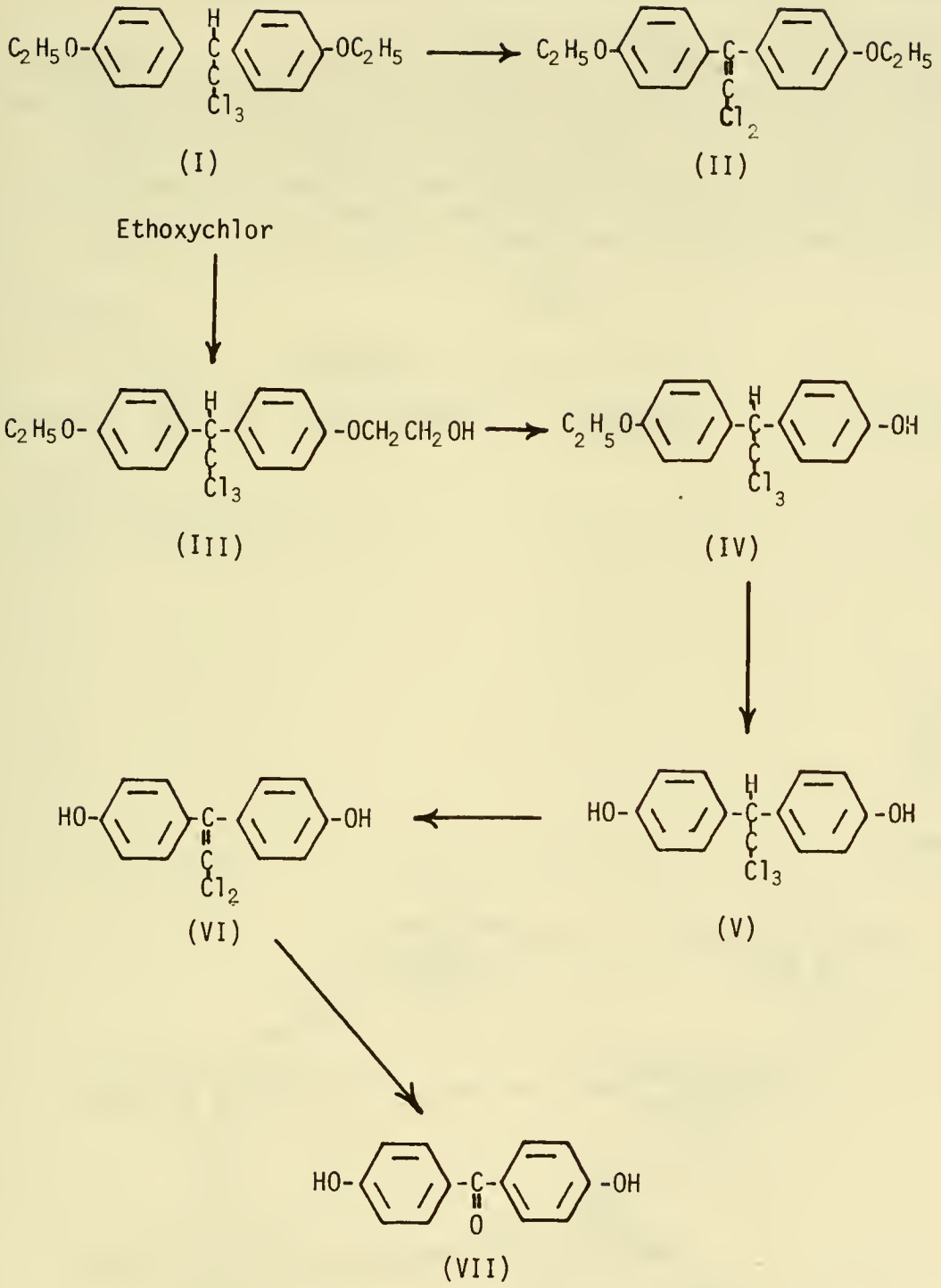
Table 1

	Test Species			
		Saltmarsh	Mice	
Metabolite	Housefly	Caterpillar	In vivo	In vitro
II	+	+	+	
III	+		+	
IV	+	+	+	+
V	+		+	
VI	+		+	
VII	+		+	
Conjugates	+	+	+	+

Table 2

	Ethoxychlor distribution				
		Algae	Snail	Mosquito	Fish
Metabolite	H_2O	(Oedogonium)	(Physa)	(Culex)	(Gambusia)
II	+	+	+	+	+
III	+		+		
IV	+	+	+	+	+
V	+	+	+		+
VI	+		+		+
VII	+		+		+
Unknown I	+			+	
Conjugates	+	+	+	+	+
Polar Cmpds.	+				

(Kapoor et al., 1972)


(I)
(II)
Ethoxychlor
(III)
(IV)
(V)
(VI)
(VII)

METHYLCHLOR [2,2-Bis(p-methylphenyl)-1,1,1-trichloroethane]

In the search for biodegradable DDT analogs, methylchlor was studied. Experiments were conducted with houseflies (R_{SP}), salt marsh caterpillar (Estigmine acrea) and mouse, and microsomal preparations from each (Table 1), as well as with a model ecosystem (Table 2). Methylchlor was found to concentrate in fish 1400 times over that in water and in snails by a factor of 120,000. The latter compares with a factor of 3500 for DDT and 120,000 for methoxychlor (Kapoor et al., 1972).

Table 1

	Specie			
			Mouse	
Metabolite	Housefly	Caterpillar	In vitro	In vivo
II		+		
III	+	+	+	+
IV			+	+
V	+		+	+
VI				
VII	+	+	+	+
VIII				
Unknown I	+		+	+
II			+	+
III				+
IV				+
Conjugates	+	+	+	+

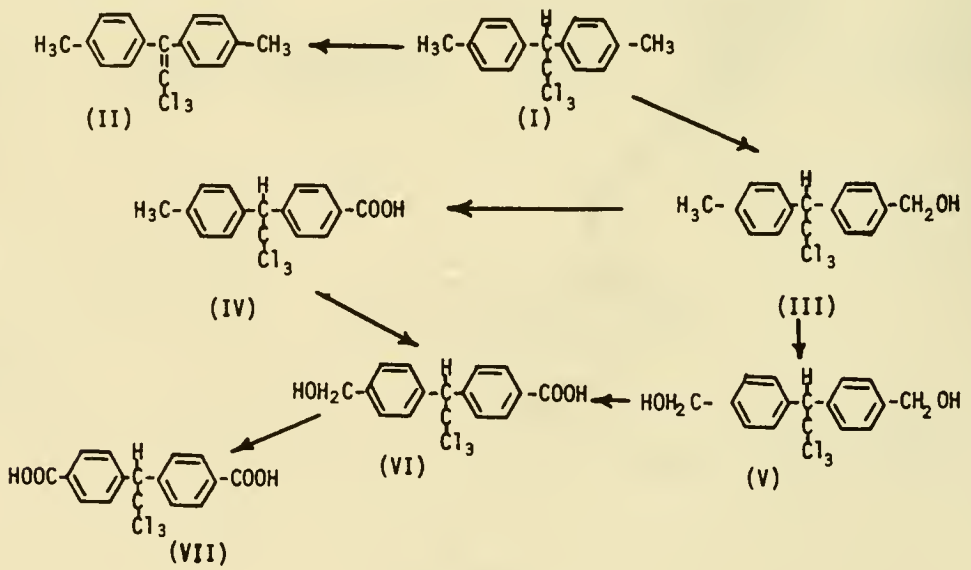


Table 2

Compound	Methylchlor distribution in model ecosystem				
	H_2O	Algae	Snail	Mosquito	Fish
II					
III		+	+	+	
IV	+			+	
V					
VI	+				
VII					
Unknown I			+		
II			+		
III	+		+		
IV	+				
V	+				
Conjugates	+	+	+	+	+
Polar metabolites	+				

METHYLENEDIOXYPHENYL COMPOUNDS

PIPERONYLIC ACID

Pseudomonas fluorescens strain PM 3 was used to prepare a soluble fraction containing oxidative activity. When this was incubated with piperonylic acid, oxidative attack produced protocatechuate and formate. One mole of O_2 was consumed per mole piperonylate and NADH or NADPH was required (Buswell and Cain, 1973).

MOBAM [4-Benzo[b]thienyl N-methylcarbamate]

Resistant and susceptible strains houseflies rapidly metabolized mobam. Differences were quantitative rather than qualitative. Of the five to six metabolites produced, one was identified as 4-hydroxybenzothiophene (Morallo, 1970).

MOCAP (Ethoprop) [O-Ethyl S,S-dipropyl phosphorodithioate]

When mocap was administered to rats, the urine contained despropyl mocap, O-ethyl phosphoric acid, S-propyl phosphorothiolic acid and desethyl mocap. Rat and rabbit liver supernatant enzymes de-ethylated mocap in the presence of glutathione and formed S-ethylglutathione. Despropyl mocap was also isolated from liver microsomes and supernatant and from plants. S-propyl phosphorothiolic acid was also present in plants. Methylene chloride extracts of bean and corn plants, grown in mocap-treated soil, contained ethyl propyl sulfide, ethyl propyl sulfoxide, ethyl propyl sulfone and propyl disulfide. Traces of S-methylation products and subsequent oxidation were observed: methyl propyl sulfide, methyl propyl sulfoxide and methyl propyl sulfone (Iqbal, 1971).

MONITOR (Methamidophos, acephate-met, Tamaron, Ortho 9006) [O,S-Dimethyl phosphoramidothioate]

ORTHENE (Acephate) [O,S-Dimethyl N-acetyl phosphoramidothioate]

Within 1 h, 130 day-old loblolly pine seedlings absorbed and distributed ^{14}C-orthene from nutrient solution. Metabolites were separated by TLC. Cochromatography and GLC were used to identify the main metabolite as O,S-dimethyl phosphoramidothioate (Monitor) and another unidentified compound (Werner, 1974).

In plant tissue, orthene is partially metabolized to O,S-dimethyl phosphoramidothioate, the active ingredient in the insecticide monitor (Leary, 1974).

The alkaline hydrolysis of monitor was investigated. P-O bond cleavage occurred in aqueous potassium hydroxide. In the methanol and acetone solutions, P-S bond cleavage occurred. In the aqueous solution, S-methyl phosphoramidothioate formed; in the less polar methanol and acetone, O-methyl phosphoramidate. The potassium salt of O-ethyl and O-propyl phosphoramidate were the main products formed in ethanolic and propanolic potassium hydroxide. Dimethyl sulfide also formed. The second order rate constants for P-O and P-S cleavage were determined in potassium hydroxide at 27C for monitor and two closely related analogs:

	k, M^{-1} min^{-1}	
	P-O	P-S
O,S-dimethyl phosphoramidate	8.4	0.6
O,S-dimethyl N-methylphosphoramidate	1.0×10^{-3}	4.4×10^{-2}
O,S-dimethyl N,N-dimethylphosphoramidate		1.5×10^{-4}

(Fahmy et al., 1972)

Conjugation of NAA in plant tissue studied. Eleven to 14-day-old cowpea plants (Vigna sinensis, Endl., cultivar Black Eye, Early Ramshaw) were used. Leaves, floating on α-NAA-buffer solution, quickly formed NA-glucose. The formation of NAA-aspartate followed a 2 to 4 h lag (Goren and Bukovac, 1973).

When Kinnow mandarin fruits were dipped in aqueous solutions of NAA, four metabolites were formed. Two of these were in small amounts and were not identified. The other two, in larger amounts, were identified as the α-NAA-aspartate and α-naphthylacetyl-β-D-glucose (NA-glucose) (Shindy et al., 1973).

NEMACUR (Bay 68138) [O-Ethyl O-(4-methylthio-m-tolyl) N-isopropyl-phosphoramidate]

After application to turf grass, nemacur was oxidized to its sulfoxide and sulfone (Bowman, 1972).

NEODECANOIC ACID (NDA) [Mixture of di-α-branched decanoic acids]

^{14}C-Carboxy labeled neodecanoic acid was applied to onions to dry the tops. Some $^{14}CO_2$ was formed during the first 12 days. Thereafter $^{14}CO_2$ formation was negligible. When onion foilage containing ^{14}C-NDA was applied to muck soil, $^{14}CO_2$ was formed over a 30-day period with little change in the rate of evolution. (Gilbert et al., 1974).

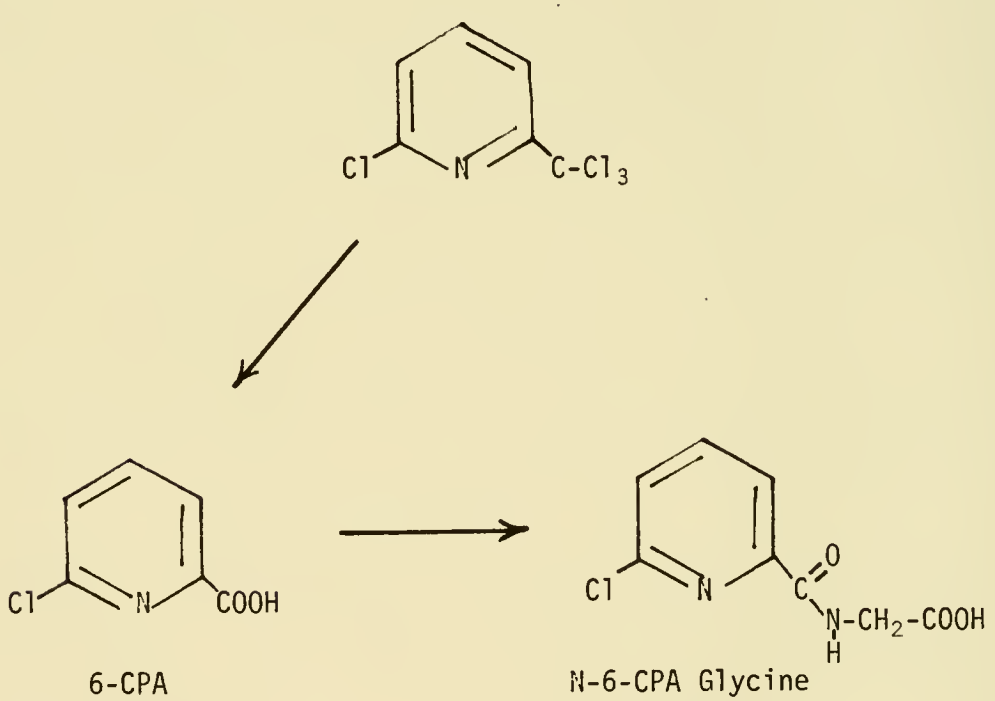
NIAGARA 10637 [Ethyl propylphosphonate]

When pea seedlings were treated with ethyl propylphosphonate, ethylene was apparently produced. In ancillary studies, ethylene and propylene were produced when this phosphonate was exposed to oxygen in combination with a reduced metal. Of the three systems studied, the cuprous system was most effective; ferrous system, least; and metallic copper intermediate (Dollwet and Kumamoto, 1970).

N-SERVE (Nitrapyrin) [2-Chloro-6-trichoromethylpyridine]

N-Serve was incubated at 20C and 10C in three soils. The half-life at 20C for the three soils was 9, 15 and 16 days for coarse sandy loam, loamy sand and loam, respectively; and 43, 77 and 43 days, respectively, at 10C (Herlihy and Quirke, 1975).

Carboxy-labeled 6-chloropicolinic acid(6-CPA), administered to a rat, was rapidly eliminated. Urine collected during the first 8 h showed the presence of 6-CPA and the N-(6-CPA) glycine conjugate. The half-life of 6-CPA in the rat was 2.4 h (Ramsey et al., 1974).

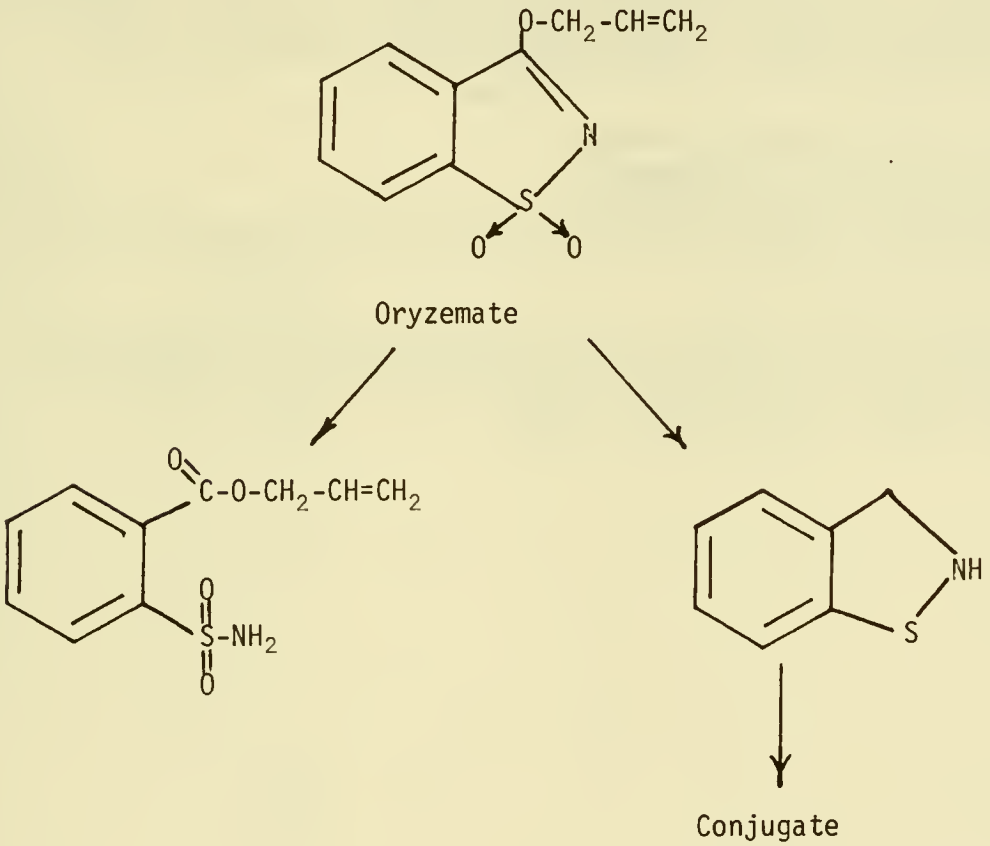


6-CPA

N-6-CPA Glycine

ORYZEMATE [3-Allyloxy-1,2-benzisothiazole-1,1-dioxide]

When applied to rice plants, oryzemate was preferentially accumulated in plant leaves. Acetomitrile extracts of rice shoots showed the formation of products which were identified as: ally o-Sulfamoylbenzoate, saccharin and its N-β-D-glucopyranosyl conjugate (Uchiyame et al., 1973).

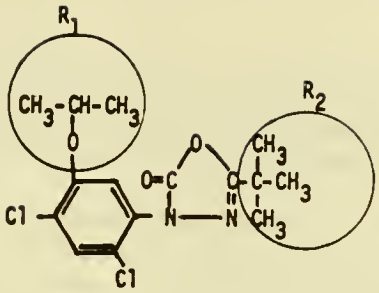


OXADIAZON [2-tert-Butyl-4-(2,4-dichloro-5-isopropoxyphenyl-Δ^2-1,3,4-oxadiazolin-5-one]

^{14}C-Oxadiazon was applied to submerged soil in which rice plants (Oryza sativa L. v. kimmaze or v. nihombare) were grown. Once taken into root tissue, oxadiazon was translocated to shoots and leaves. Metabolism of oxadiazon in plants involved the side chain primarily and produced dealkylated metabolites, an alcholol, and an acid in shoots of seedlings and straws of plants at harvesting. Most of the radioactivity in the roots, shoots or straws, however, was unchanged oxadiazon. The metabolites were identified as the following derivatives:

-4-(2,4-dichloro-5-methoxyphenyl)-	(III)
-4-(2,4-dichloro-5-hydroxyphenyl)-	(IV)
-4-(2,4-dichloro-5-ethoxyphenyl)-	(II)
2-[2-(2-methylpropanoic acid)]-4-(2,4-dichloro-5-hydroxyphenyl)-	(V)
2-(2-methylisopropanol)-	(I)

A sixth metabolite, wherein the nitrogen ring had been opened, was also found and identified as 1-(2,4-dichloro-5-isopropoxyphenyl)-1-methoxycarbonyl-2,2-dimethylpropanoylhydrazine (VI) (Hirata and Ishizuka, 1975; Ishizuka et al., 1975).



I R_2 = $-C(CH_3)_2-CH_2OH$

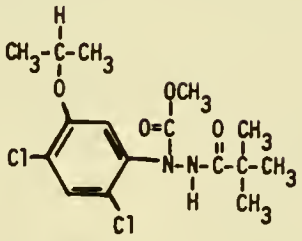
II R_1 = C_2H_5O-

III R_1 = CH_3O

IV R_1 = -OH

V R_1 = -OH

R_2 = $-C(CH_3)_2-COOH$

VI 

PARATHION [O,O-Diethyl O-p-nitrophenyl phosphorothioate]

Mixed function oxidase enzymes metabolized parathion to phosphate and diethyl phosphorothioate. Desethyl parathion was identified (Wolcott, 1971).

When ^{35}S-parathion was incubated with rabbit and rat hepatic microsomes, a good portion of the released sulfur in paraoxon formation became bound to microsomal macromolecules. This was decreased by Cu^{2+} which is known to inhibit MFO (Poore and Neal, 1972).

Over 90% of the radioactivity administered to rats as ^{14}C-ethyl parathion was eliminated within 72 h. In urine, the principal radioactive metabolites of paraoxon were diethyl phosphoric acid and desethyl paraoxon. Some bound p-nitrophenol in urine was indicated. In vitro studies with microsomal enzymes degraded ^{14}C-paraoxon to desethyl paraoxon, diethyl phosphoric acid, monoethylphosphoric acid and an amino acid conjugate of paraoxon. Ethanol and acetaldehyde were also found in small amounts. When rats were pretreated with microsome inducers, the microsomes prepared from these rats produced ethanol from the paraoxon. When NADPH was added to the incubation mixture, paraoxon degrading activity was greater than without NADPH or with NADH (Ku and Dahm, 1973).

In other studies with rat liver cells, data indicated that the soluble fractions obtained at 105000xg and 500000xg were primarily responsible for the degradation of parathion to water-soluble metabolites. Analyses indicated that aminoparathion was the primary metabolite produced by soluble cell fractions, followed by paraoxon. Small amounts of p-nitrophenol were also detected. Parathion was reduced to aminoparathion and oxidized at the same time to paraoxon. The data indicated that paraoxon was more readily degraded than parathion. It appeared that parathion and paraoxon were degraded to water-soluble materials primarily by soluble fractions. Enzymes in the soluble fraction reduced parathion to aminoparathion. Some oxidation to paraoxon was also affected. Paraoxon was degraded mostly by particulate-associated enzymes through hydrolysis to p-nitrophenol. The largest amounts of water-soluble metabolites were produced by the soluble fractions which also reduced paraoxon to aminoparaoxon (Lichtenstein et al., 1973).

The nature of the serum enzyme catalyzing paraoxon hydrolysis was partly characterized. The enzyme, paraoxonase, apparently has a single binding site (Lenz et al., 1973).

Weanling Holtzman rats, male and female, were injected with parathion. Cochromatography was used to identify tissue residues as consisting of diethylphosphorate (DEPA), diethyl phosphorothioate (DEPTA), paraoxon and parathion. Whereas liver, kidney, skeletal muscle and plasma contained residues of all four compounds, only parathion and paraoxon

were found in brain and fat tissues. DEPA and DEPTA occurred as the major metabolites in urine with parathion and paraoxon present as traces only. Higher paraoxon levels were present in plasma and brain of weanlings than of adult rats. In vitro studies with liver homogenates showed that adult male rats possess higher activity than weanlings or adult females for both the oxidative and hydrolytic metabolic pathways of parathion (Gagne and Brodeur, 1972).

The comparative metabolism of parathion to p-nitrophenol in rats and lobsters was measured in vitro. The rate was considerably greater in rats than in lobsters. No paraoxon could be detected (Carlson, 1973).

Oxidative activation of methyl parathion and ethyl parathion in vitro by NADP-glucose-6-phosphate-fortified mouse and liver homogenates were measured. Fish liver homogenates did not differ in their ability to cleave both parathions to p-nitrophenol and the respective dialkyl phosphorothioates. In these studies, enzymatic hydrolysis of the oxygen analogs was negligible. Fish liver homogenates without added cofactors degraded less than 10% of methyl parathion and no ethyl parathion. Addition of GSH increased methyl parathion degradation but did not enhance ethyl parathion degradation (Benke et al., 1974). Dearylation of parathion occurred predominantly via microsomal MFO. In resistant fish, highest MFO levels correlated with highest parathion tolerance (Chambers and Yarbrough, 1973).

Parathion was applied to peach trees. Residue analyses showed the presence of S-ethyl parathion and paraoxon as well as parathion. Degradation of S-ethyl parathion was very rapid with EC, WP and encapsulated parathion formulations (Winterlin et al., 1975).

After application of parathion to cotton, analyses indicated a constant increase in photoalteration products. Products found on the foliage included S-ethyl parathion, S-phenyl parathion, paraoxon and p-nitrophenol (Joiner and Baetcke, 1973).

When parathion was applied to spinach in the field, paraoxon, diethyl phosphate and p-nitrophenol residues increased. Aminoparathion, S-ethyl parathion and S-phenyl parathion did not increase in percentage of harvest residues or were undetectable (Archer, 1974). In other studies, parathion was incubated with spinach homogenate. TLC and gas chromatography were used to demonstrate the presence of parathion, aminoparathion, hydroxylaminoparathion, and nitrosoparathion. Non-enzymatic reduction of nitrosoparathion by NADPH to hydroxylaminoparathion was also demonstrated (Suzuki and Uchiyama, 1975).

In submerged soils, parathion degraded to aminoparathion. In upland conditions, aminoparathion was not detected. Addition of Flavobacterium sp. to the soil accelerated decomposition of parathion to p-nitrophenol (Sethunathan and Yoshida, 1973). A bacterium obtained from

flooded alluvial soil was identified as a Pseudomonas sp. When this organism was incubated with parathion, hydrolysis to p-nitrophenol occurred within 3 h. Release of nitrite from p-nitrophenol occurred within 24 h. A Bacillus sp., also obtained from flooded alluvial soil, liberated nitrite from p-nitrophenol but not from parathion (Sethunathan, 1973; Siddaramappa et al., 1973).

When Rhizobium japonicum and R. meliloti were incubated with parathion, 95% of the parathion was not detectable after 50 h. Degradation proceeded primarily via aminoparathion which accounted for about 85% of the initial parathion. Diethyl phosphorothioic acid (DEPTA) accounted for 10%. The remaining 5% was unreacted parathion. No paraoxon was detected (Mick, 1969).

Microorganisms from Lake Tomahawk were incubated with parathion. Degradation to aminoparathion occurred under aerobic and anaerobic conditions. Aminoparathion underwent further degradation aerobically but not under anaerobic conditions (Graetz, 1970). In some studies, after addition of methyl parathion to soil, the oxon was identified (Baker and Applegate, 1970).

The persistence of parathion was partially dependent on soil type. In some soils degradation was rapid and probably through a combination of hydrolysis and strong microbial activity. In other soils, parathion loss was slow and attributable to hydrolysis. In Madera sandy loam, after parathion was added at 200 ppm, aminoparathion was not observed although parathion residues declined after 10 days. However, with parathion at 20 ppm in this soil and submerged under water, 3 ppm aminoparathion was recovered after 7 days (Iwata et al., 1973). At exaggerated levels (30000 to 95000 ppm), parathion was applied to field soil plots as an emulsifiable concentrate or wettable powder. Degradation was slower than anticipated and persisted at relatively high levels for 5 years after gross topical contamination with parathion (Wolfe et al., 1973).

Degradation of parathion proceeded via hydrolysis to p-nitrophenol and diethyl phosphorothioate when adsorbed on kaolinites. Ca-kaolinite was most active (Saltzman et al., 1974).

After exposure of parathion for 35 days to ultraviolet irradiation, 12 products were identified:

paraoxon
O,S-diethyl O-p-nitrophenylphosphate
O,O-diethyl S-p-nitrophenyl phosphate
p-nitrophenol
p-aminophenol
diethyl phenyl phosphate

O,O-diethyl O-phenyl phosphorothioate
O-ethyl O,O-bis(p-nitrophenyl)phosphorothioate
ethyl bis(p-nitrophenyl phosphate)
diethyl phosphate, free acid
monoethyl phosphate, free acid
phosphate

(Joiner and Baetcke, 1974).

Parathion was dissolved in 80% aqueous ethanol or 80% aqueous tetrahydrofuran and irradiated at 2537Å. Photolysis in either solvent produced O,O,S-triethylphosphorothioate as the major product and lesser amounts of triethylphosphate, paraoxon, ethanethiol, and p-nitrophenol. Photolysis of paraoxon under identical conditions yielded triethylphosphate (Grunwell and Erickson, 1973). In other studies, photolysis of parathion produced 15 products of which 12 were identified and confirmed by infrared spectroscopy: ethyl paraoxon, S-ethyl parathion, S-phenyl parathion, p-aminophenol, diethyl phenyl phosphate, diethyl phenyl phosphorothioate, ethyl bis(p-nitrophenyl)phosphate, ethyl bis(p-nitrophenyl)phosphorothioate, diethyl phosphate and monoethyl phosphate. After application of ^{14}C-parathion to cotton plants exposed under different environmental conditions, identification of photo-alteration products after extraction was by thin-layer chromatography and liquid scintillation spectroscopy: paraoxon, p-nitrophenol, S-ethyl parathion and S-phenyl parathion. As much as 15.4% of the applied ^{14}C-parathion remained after 28 days (Joiner, 1972; Joiner and Baetcke, 1973).

Sampling of air downwind from a parathion-treated prune orchard revealed the presence of parathion and paraoxon as well as some p-nitrophenol (Woodrow et al., 1975).

Methyl parathion was incubated with isolated bacteria strains from water from the Vistula River (Poland) and from municipal sewage. These studies indicated that degradation of this pesticide was due to Bacillus cereus and to Bacillus sp. 8. The presence of other organic material apparently accelerated the biodegradation. Most rapid degradation was observed when serine, threonine, asparagine, and alanine were present (Maleszewska, 1974).

In other studies, p-nitrophenol, a metabolite of parathion, in milk was fed to houseflies. An observed conjugated metabolite behaved like the glucose-6-phosphate analog of p-nitrophenol (Heenan and Smith, 1965).

PCB (Aroclor, Clophen, KC, Kanechlor, Phenoclor) [Polychlorinated biphenyl]

Following intravenous administration to rats of 4-chloro, 4,4^1-dichloro, 2,2^1,4,5,5^1-pentachloro- and 2,2^1,4,4^1,5,5^1-hexachlorobiphenyl, these PCBs were initially rapidly removed from blood and stored in liver and muscle primarily. Redistribution to skin and adipose tissue followed. Half-lives were calculated for each compound:

	Half-life, h					
Biphenyl	Blood	Liver	Muscle	Skin	Adipose	Feces
4-chloro-	4.7 120.0	4.9 85.6	1.26 68.2	4.78 74.0	0.79	15.7
4,4^1-dichloro-	5.25 39.6	6.1 99.2	0.131 35.2	11.4 87.4	5.46 50.6	22.2
2,2^1,4,5,5^1-pentachloro-	0.53 27.7 256.7	0.25 2.8 40.1 193.0	2.23 613.4	43.0 613.0	51.3 389.0	39.2 211.0
2,2^1,4,4^1,5,5^1-hexachloro-	3.77 27.1 1359.0	2.4 18.3 1308.0				49.0 642.0

Following administration of the PCBs, there was an initial very rapid phase when part of the dose was removed from the tissue. This was followed by a much slower rate of removal. The respective decay rates are summarized (Matthews and Anderson, 1975b).

After intraperitoneal injection of 4-chlorobiphenyl in young male rats, urine and feces were collected. A mono- and dihydroxychlorobiphenyl was observed (Hutzinger et al., 1972a).

When 4-chlorobiphenyl was fed to rats, 4-chloro-4^1-hydroxybiphenyl was obtained (Safe et al., 1974). In subsequent studies, this metabolite was administered intraperitoneally to rats. Urine and feces were collected and analyzed. Mass and NMR spectroscopy identified the major urinary compound as 4^1-chloro-3,4-dihydroxybiphenyl. Two other dihydroxy compounds were observed but could not be separated by chromatographic procedures. However, demethylation of urinary chloromethoxy analogs gave only one product, 4^1-chloro-3,4-dihydroxybiphenyl. This indicated that the two components were 4^1-chloro-3-methoxy-4-hydroxybiphenyl and 4^1-chloro-4-methoxy-3-hydroxybiphenyl. A fourth urinary compound was identified as 4^1-chloro-4-methoxy-3,5-dihydroxybiphenyl. No fecal metabolites were observed (Safe et al., 1975a).

When 4-chlorobiphenyl was administered to rabbits, the compound was metabolized to 4^1-chloro-4-hydroxybiphenyl and 4^1-chloro-3,4-dihydroxybiphenyl. The 4-hydroxy compound was metabolized by rabbits to the 3,4-dihydroxy analog and small amounts of 4^1-chloro-4-hydroxy-3-methoxybiphenyl and 4^1-chloro-3-hydroxy-4-methoxybiphenyl. The results of this study were consistent with the formation of an arene oxide intermediate (Safe et al., 1975c).

After intravenous injection of 4-chloro- and 4,4^1-dichlorobiphenyl to goats and a cow, urine was collected. After extraction and cleanup of the urine samples, mass spectrometry was used to identify the metabolites of 4-chlorobiphenyl as 4^1-chloro-4-hydroxybiphenyl and 4^1-chloro-3,4-dihydroxybiphenyl. The 4-chlorobiphenyl was also identified in urine of cow. Confirmation of metabolite structure identity was made by NMR spectrometry and by comparison with samples of metabolites identified in the rat and confirmed by synthesis (Safe et al., 1975b).

4-Chlorobiphenyl was fed to male albino rabbits. Urine was collected and analyzed. About 50% of the ingested dose was excreted as a glucosiduronic acid derivative. Free phenol accounted for 3% and sulfate for 11%. No mercapturic acid derivatives were observed (Block and Cornish, 1959).

When pigeons were fed 4-chlorobiphenyl, a monohydroxylated chlorobiphenyl was found. Trout excreted no detectable metabolites into the water when exposed to 4-chlorobiphenyl (Hutzinger et al., 1972b and c).

Thorny skate (Raja radiata) and winter skate (Raja ocellata) were intravenously administered 2-, 3-, and 4-chlorobiphenyl. The 3-isomer cleared more readily from plasma than did the other two. Accumulation varied considerably from fish to fish but was highest in muscle and liver (Zinck and Addison, 1974).

^{14}C-Labeled 2,4^1-dichlorobiphenyl was administered intravenously to 4 female rhesus monkeys. Biological $t_{1/2}$=1.1 to 2.9 days. An average of 73.5% of the administered dose was recovered within 14 days. About 70% of the recovered material appeared in urine and 30% in feces. Blood levels were negligible after 14 days but about 7% of the dose was in the fatty tissue (Greb et al., 1973). Metabolites in urine and feces were identical. About 17% of the metabolites were conjugated as the sulfate or glucuronide. Three (of six) monohydroxy and three dihydroxy dichlorobiphenyls formed (Greb et al., 1975b).

When fed to rats, 4,4^1-dichlorobiphenyl yielded the 3-hydroxy analog (Safe et al., 1974). After intraperitoneal injection of an oil solution of 4,4^1-dichlorobiphenyl into young male rats, a monohydroxylated chlorobiphenyl was observed. Pigeons, when fed the 4,4^1-compound,

gave similar results. Trout did not excrete any metabolite into the water when fed this material (Hutzinger et al., 1972b and c). When this 4,4^1-dichloro compound was intravenously injected into a goat and a cow, the 3-hydroxy analog was found in the urine (Safe et al., 1975b).

Adult female rats (Swiss Webster strain) were treated with phenobarbital for 3 days to increase MFO activity. On the fourth day, the animals were killed and the livers removed and homogenized. The supernatant from 10,000g centrifugation was centrifuged at 100,000g. The resultant pellet was used to assay conversion of PCBs. When 2,2^1-dichlorobiphenyl was incubated with the liver enzyme system, four monohydroxy and four dihydroxy dichlorobiphenyls were found. When 2,4^1-dichlorobiphenyl was used, only 2 of 6 monohydroxy derivatives were found. Two dihydroxy-dichlorobiphenyls also formed. When 2,2^1,5-trichlorobiphenyl was incubated with the liver enzyme system, 3 monohydroxy (of seven possible) and 2-dihydroxy trichlorobiphenyls were produced (Greb et al., 1975a).

Within 14 days after 2,2^1,5-trichlorobiphenyl was intravenously administered to a rhesus monkey, about 82% of the dose was excreted. Urine and feces contained about equal amounts of radioactivity. The $t_{\frac{1}{2}}$ was 2.1 days (Greb et al., 1973). Three monohydroxy, two dihydroxy and one trihydroxy derivative was formed (Greb et al., 1975b).

2,2^1,5,5^1-Tetrachlorobiphenyl in oil was injected intraperitoneally into young male rats. A monohydroxylated chlorobiphenyl was observed. A similar observation was made when the compound was fed to fish. Trout did not metabolize this material (Hutzinger et al., 1972b and c).

A 1% suspension of 2,4,3^1,4^1-tetrachlorobiphenyl in Tween 80-saline (1:3) was intravenously injected into adult male Wistar King rats. About 0.6% of the dose was excreted unchanged daily into the gastrointestinal tract through the wall of the small intestine (Yoshimura and Yamamoto, 1975). After oral administration of this 2,4,3^1,4^1-isomer to adult male Wistar strain rats, urine and feces were collected. At least four phenolic compounds were excreted into feces in addition to unchanged material. No evidence of conjugated metabolites was obtained. About 43% of the administered dose was excreted within 12 days after treatment. Synthesis and spectrophotometric analyses identified the major metabolite as the 5-hydroxy derivative and the minor metabolite as the 3-hydroxy derivative (Yamamoto and Yoshimura, 1973; Yoshimura et al., 1973).

Tritiated 2,5,2^1,5^1-tetrachlorobiphenyl was administered to male infant rhesus monkeys by gastric intubation. At 72 h, the animals were sacrificed. Urine was collected and analyzed. Identification of metabolites involved IR, GLC-MS and derivatization. Four metabolites were isolated and identified as a monohydroxytetrachlorobiphenyl, a dihydroxytetrachlorobiphenyl, trans-3,4-dihydro-3,4-dihydroxy-2,5,2^1,5^1-tetrachlorobiphenyl, and a hydroxylated derivative of the latter with the additional hydroxy group on the aromatic ring (Hsu et al., 1975a and b).

Sprague-Dawley rats were administered a single dose of ^{3}H-2,5,2^{1},5^{1}-tetrachlorobiphenyl by gastric intubation. Of six metabolites observed, one from feces was identified as the 3-hydroxy analog by mass and IR spectra. Three other compounds had mass spectra indicative of monohydroxy analogs also but were not further identified (Van Miller et al., 1975).

When rabbits were fed 2,5,2^{1},5^{1}-tetrachlorobiphenyl, three hydroxylated compounds were identified by gas chromatography, mass spectra and IR spectra. Two compounds were identified as 3- and 4-hydroxy-2,5,2^{1},5^{1}-tetrachlorobiphenyl. The third compound was identified as trans-3,4-dihydro-3,4-dihydroxy-2,5,2^{1},5^{1}-tetrachlorobiphenyl (Gardner et al., 1973).

3,4,3^{1},4^{1}-Tetrachlorobiphenyl was administered orally to male Wistar rats every third day for 9 days. The fecal extract contained three metabolites. One of these was isolated and identified as either 2- or 5-hydroxy-3,4,3^{1},4^{1}-tetrachlorobiphenyl (Yoshimura and Yamamoto, 1973).

Rats, mice and quail were administered 2,2^{1},3,5^{1},6-pentachlorobiphenyl. Analyses of collected feces indicated differences in metabolism of this compound between mammals and birds. Quail excreted the 4^{1}-hydroxy analog as the major monophenol whereas with rats and mice, the major monophenol excreted was the 5-hydroxy analog. In rat feces, 17 hydroxylated derivatives were observed. Of these, eight were identified as the following hydroxylated analogs of 2,2^{1},3,5^{1},6-pentachlorobiphenyl:

3^{1}-hydroxy-	3^{1},4-dihydroxy-
4^{1}-hydroxy-	3^{1},5-dihydroxy-
4-hydroxy-	4,4^{1}-dihydroxy-
5-hydroxy-	4^{1},5-dihydroxy-

A resume of the metabolites found in rat feces in this study follows:

Description of biphenyl	No. derivatives
Monohydroxytetrachloro-	3
Monohydroxypentachloro-	7
Dihydroxytetrachloro-	1
Dihydroxypentachloro-	6

(Sundstrom and Jansson, 1975)

A phenolic metabolite of 2,2^{1},4,5,5^{1}-pentachlorobiphenyl was isolated from feces of rats given this compound. Mass spectrum, synthesis, elaborate chemical studies and chromatography showed that the metabolite was the 3^{1}-hydroxy analog (Sundstrom and Wachmeister, 1975).

In other studies with ^{14}C-labeling, single doses were administered intravenously to rats. Most of the material was excreted in the form of glucuronides, primarily a 3^1-hydroxy or 3^1,4^1-dihydroxy derivative. More than 90% of the total dose was removed from the blood within 10 min. Prior to translocation to skin and adipose tissues, the material was initially deposited in the liver and muscle. Most of the radioactivity was excreted in the bile and feces. Excretion in the urine accounted for less than 7% of the dose and ceased after 8-9 days (Matthews and Anderson, 1975).

Uniformly ^{14}C-labeled 2,4,5,2^1,5^1-pentachlorobiphenyl was administered intravenously and orally to mice. Autoradiography and scintillation counting after intravenous administration showed that most radioactivity left the circulation within 1 h. Peak concentrations were highest in brown fat. Excretion of radioactivity occurred mainly via bile with a half-time of six days. During the first 20 min after injection, in addition to appearing in the fat, radioactivity also appeared mainly in liver and kidneys. Analyses after oral administration indicated that the major metabolite was an unidentified monohydroxylated compound, which occurred free and conjugated (Berlin et al., 1975).

For five weeks 50 μg of 2,4,6,2^1,4^1-pentachlorobiphenyl was administered daily to four male and female rats. Urine and feces were collected daily. Two compounds were isolated but not identified. Mass spectra indicated a hydroxy and a methoxy derivative (Lay et al., 1975). In other studies when labeled material was fed to rats, most of the radioactivity was found in the feces. The metabolites found in the urine were conjugated. Monohydroxylation predominated and all three meta-hydroxy compounds formed. No 2- or 4-hydroxybiphenyl was observed (Goto et al., 1975).

When labeled 2,4,6,2^1,6^1-pentachlorobiphenyl was fed to rats, most of the label appeared in the feces. Metabolites found in the urine were conjugated. Two m-hydroxy derivatives were found and identified by mass spectra and gas chromatography. No 2- or 4-hydroxy analogs were observed. A dihydroxy compound was identified as the 3^1,4^1-dihydroxy derivative. The main conjugate was identified as the β-glucuronide by incubation with β-glucuronidase (Goto et al., 1975).

Feeding of 2,4,6,3^1,4^1-pentachlorobiphenyl to rats yielded the 3-hydroxy derivative. The 5^1-analog was not definitely known to be present (Goto et al., 1975).

When 2,4,6,3^1,5^1-pentachlorobiphenyl was fed to rats, a trace of the 3-hydroxy derivative was observed (Goto et al., 1975).

No excreted hydroxylated metabolites were observed after intraperitoneal injection of 2,2^1,4,4^1,5,5^1-hexachlorobiphenyl in rats, feeding of pigeons or feeding of trout (Hutzinger et al., 1972b and c). When this

compound was fed to rats, a hydroxy metabolite was found in the feces but not in urine. Mass spectra data favored the 3-hydroxy structure (Jensen and Sundstrom, 1974). After feeding this hexachloro compound to rabbits for 7 days, collected urine was analyzed. Three metabolites were observed and corresponded to hydroxylation, hydroxylation and monodechlorination, and a monodechlorinated analog that contained a hydroxy and a methoxy group (Hutzinger et al., 1974).

Feeding of labeled 2,4,6,2^1,4^1,6^1-hexachlorobiphenyl to rats yielded the meta-hydroxy derivative (Goto et al., 1975).

When fed to rats, decachlorobiphenyl remained unchanged (Goto et al., 1975).

Studies of extracts of human fat and animal material indicated that many PCBs were metabolized (indicated by + in the following table). Many of the individual PCBs have been identified. In this study, the products were not identified (Schulte and Acker, 1974).

2,5,2^1,5^1-	+
2,3,4,2^1,5^1-	+
2,3,6,2^1,5^1-	+
2,4,5,2^1,5^1-	+
2,4,5,2^1,3^1-	+
2,3,4,2^1,3^1,4^1-	+
2,3,4,2^1,4^1,5^1-	+
2,3,4,5,2^1,5^1-	+
2,3,5,6,2^1,5^1-	+
2,3,6,2^1,3^1,6^1-	+
2,3,6,2^1,4^1,5^1-	+
2,4,5,2^1,4^1,5^1-	Neg.
2,3,4,5,2^1,3^1,4^1-	Neg.
2,3,4,5,2^1,3^1,6^1-	(+)
2,3,4,5,2^1,4^1,5^1-	Neg.
2,3,4,5,6,2^1,5^1-	(+)
2,3,5,6,2^1,4^1,5^1-	Neg.
2,3,4,5,2^1,3^1,4^1,5^1-	Neg.
2,3,5,6,2^1,3^1,5^1,6^1-	Neg.
2,3,4,5,2^1,3^1,5^1,6^1-	Neg.

When rats were fed Aroclor 1016 and 1242, measurable residues were still present five and six months, respectively, after exposure was discontinued (Burse et al., 1974). Studies also indicated that components of Aroclor 1254 were metabolized at different rates (Grant et al., 1971). Twenty-seven PCBs in Aroclors 1221, 1242, and 1254 were separated and identified by GLC and IR comparison with known prepared compounds (Webb and McCall, 1972).

The main components of Kanechlor-400 (KC-400) were identified as 2,4,3^1,4^1-, 2,5,3^1,4^1-, 2,3,4,4^1-, and 3,4,3^1,4^1-tetrachlorobiphenyl and 2,3,4,3^1,4^1-pentachlorobiphenyl (Saeki et al., 1971). Single doses of KC-400 were orally administered to female DDD strain mice. The results indicated that each component was almost equally absorbed and distributed in tissues. Skin concentration of chlorobiphenyls one day after injection was about twice as high as that of liver and kidneys; and chlorobiphenyls were retained longer in the skin than in other tissues. While tetrachlorobiphenyls were almost completely eliminated from liver and kidney in 3 to 4 weeks, the minor components penta- and hexachlorobiphenyls were still retained in small amounts after 9 to 10 weeks (Yoshimura and Oshima, 1971). In other studies using rats and ^{3}H-labeled KC-400, distribution and excretion radioactivity was measured at 3, 28 and 56 days. Levels were higher in skin, adipose tissue, liver, adrenal gland and GI tract than in plasma. Skin and adipose tissues were highest. Although most of the radioactivity was eliminated from the tissues after four weeks, a significant amount was still present after eight weeks. During four weeks of observation, 2% of the dose was excreted via urine and 70% via feces (Yoshimura et al., 1971).

Cows were fed Aroclor 1254 for 60 days. PCB concentrations in milk fat approached equilibrium after 40 days. After feeding stopped, the PCB concentration in milk fat declined 50% within 15 days. The average rate constant was 0.010 day^{-1} and varied from 0.005 to 0.016 day^{-1}. The decline in body fat concentration of PCB paralleled that in milk (Fries et al., 1973).

$$C = 30.6e^{-0.32t} + 32.3e^{0.010t}$$

Studies with sheep and pigs yielded the following with Aroclor 1254: (Blood Concentration) $C_A = Ae^{-\alpha t} + Be^{-\beta t}$

	Sheep	Pig
A	0.616 ppm	1.213 ppm
α	0.465	.537
B	0.115 ppm	.252 ppm
β	0.047	.046
V_A	284.54	65.60

(Borchard et al., 1974)

Aroclor 1254 was fed to bobwhite quail for 14 days. Absorption of all components occurred at the same rate. Two peaks containing six chlorines showed a distinct increase but a third peak declined. Some

dechlorination occurred but no products were identified (Bagley and Cromartie, 1973).

Leghorn hens and a rooster were given 50 µg/ml of the PCB mixture Aroclor 1254. Elimination of PCB isomers was studied. It was found that 3,4,2^1,3^1,6^1-pentachlorobiphenyl was eliminated more rapidly than 3,4,2^1,4^1,5^1-pentachlorobiphenyl and 2,3,4,2^1,4^1,5^1-hexachlorobiphenyl. Chlorination in the 4-position in penta- and hexachlorobiphenyls was associated with slow clearance of PCB isomers from hen, embryo, and chick (Bush et al., 1974).

Fish, exposed to Clophen A50, accumulated residues up to 70 ppm. When transferred to fresh water, half of the PCB residues was eliminated in 20 days (Hattula and Karlog, 1973).

Baltic herring (Clupea harengus) were shown to carry high PCB residue levels. Since the guillemot (Uria algae) and the grey seal (Halichoerus grypus) feed heavily on the Baltic herring, studies were undertaken to determine PCB residues in droppings of the guillemot and seal. These studies showed a difference in the metabolism of PCB and are summarized in the following table. Compounds were identified by GC-MS (Jansson et al., 1975).

Compounds found in the phenolic fraction from feces after methylation.

Compound	Number of Isomers	
	Seal	Guillemot
Methoxytrichlorobiphenyl	2	-
Methoxytetrachlorobiphenyl	5	6
Methoxypentachlorobiphenyl	9	8
Methoxyhexachlorobiphenyl	7	7
Methoxyheptachlorobiphenyl	2	3
Dimethoxypentachlorobiphenyl	-	1
Dimethoxyhexachlorobiphenyl	1	1

Grass shrimp (Palaemonetes pugio), exposed for 3 months to sediments contaminated with Aroclor 1254, concentrated the Aroclor to about the level observed when exposed to 0.09 µg/l in water for 2 weeks in the laboratory (Nimmo et al., 1974).

A laboratory ecosystem used to study the fate of ^{14}C-labeled PCBs consisted of water, alga (Oedogonium cardiacum), snail (Physa), plankton, water flea (Daphnia magna), mosquito (Culex pipiens quinquefasciatus) and fish (Gambusia affinis). After introduction of 2,5,2^1-trichlorobiphenyl into the system, six compounds were observed: in water, compounds I, II, IV, V, VI; in alga, compounds I, II, VI;

in snail, I through VI; in mosquito, I; and in fish, I and II. When 2,5,2^1,5^1-tetrachlorobiphenyl was introduced, two compounds were observed in all phases except fish, where only one was found. After introduction of 2,5,2^1,4^1,5^1-pentachlorobiphenyl, five compounds were observed in the system. Compounds II, III, IV, and V in water; I, II, III and V in alga; I, II and III in mosquito; I, II, III and V in fish; and all five in snail. All three compounds were biomagnified in all components of the system. All three compounds were also degraded by the salt marsh caterpillar larva: five compounds from the trichloro-; three compounds from the tetrachloro-; and three compounds from pentachlorobiphenyl. None of the metabolites observed were identified (Metcalf et al., 1975b).

Two plants, Ranunculus fluitans and Callitriche sp., which grow submerged in water, were exposed to 2,2^1-dichlorobiphenyl. In the water where the plants had been cultured; one dihydroxy compound free and conjugated, and a dechlorinated product (Moza et al., 1974). ^{14}C-2,2^1-Dichlorobiphenyl was also applied directly to leaves of Veronica beccabunga. After 6 weeks, the plants were homogenized with methanol and extracted for 48 h in a Soxhlet. Thin-layer chromatography indicated four compounds, one of which was conjugated. Mass spectra indicated monomethylation of a phenol when it was reacted with diazomethane. A second compound gave similar results, indicating a second monophenol. These phenols were also observed after acid hydrolysis of the conjugated fraction (Moza et al., 1973).

The soil fungus Rhizopus japonicus was incubated in a medium containing 4-chlorobiphenyl, 56 µg/30 ml culture medium. A compound having a melting point of 145C was isolated. NMR and mass spectra were identical with that of 4-chloro-4-hydroxybiphenyl (Wallnofer et al., 1973).

4,4^1-Dichlorobiphenyl was hydroxylated by Rhizopus japonicus. The metabolite was not identified (Wallnofer et al., 1973).

Two strains of Achromobacter were isolated from sewage effluents using biphenyl and p-chlorobiphenyl as sole carbon sources. Achromobacter BP grew only on biphenyl but did co-metabolize the mono- and dichlorobiphenyls. Achromobacter pCB, grown on p-chlorobiphenyl, grew better on biphenyl than on p-chlorobiphenyl and co-metabolized meta- and ortho-chlorobiphenyl and dichlorobiphenyls. Achromobacter pCB metabolized p-chlorobiphenyl to benzoic acid and p-chlorobenzoic acids. The latter probably arises also from p,p^1-dichlorobiphenyl. The two acids were also produced by Achromobacter BP. Chloride was not produced by either strain during degradation of the chlorobiphenyls. Washed cell suspensions of both isolates oxidized biphenyl, o-phenyl phenol, phenylpyruvate, catechol, p-chlorobiphenyl, m-chlorobiphenyl, o-chlorobiphenyl, o,o^1- and p,p^1-dichlorobiphenyl (Ahmed and Focht, 1973b). Using resting cell suspensions of Achromobacter pCB, they were first grown on p-chlorobiphenyl. Except for 2,5,3^1,4^1-tetrachlorobiphenyl which was not oxidized at all,

and 3,3^1-dichlorobiphenyl which was oxidized after a brief lag, all other PCBs tested were oxidized without lag: 2,3-, 2,4-, 3,4- and 3,5-dichlorobiphenyl; 3,4,2^1-trichlorobiphenyl; 2,3,2^1,3^1-tetrachlorobiphenyl; 2,3,4,5,6-pentachlorobiphenyl. Products were not identified (Ahmed and Focht, 1973a).

In studies with microorganisms, various PCBs were incubated with Nocardia spp. (NCIB 10603) and Pseudomonas spp. (NCIB 10643) for varying periods of 7 to 73 days. Nocardia spp. degraded at least 50% of the following PCBs:

2,4^1-dichloro-	2,3,2^1-trichloro-
2,3-dichloro-	2,3,4^1-trichloro-
3,4-dichloro-	3,4,3^1-trichloro-

When biphenyl was added to the incubation mixture, 2,5,4^1-trichlorobiphenyl was also metabolized. Similarly, a mixture of 2,3,4,5,2^1,3^1-hexachloro-, 2,3,2^1-trichloro-, and 2,3^1,4^1-trichlorobiphenyl plus biphenyl was metabolized. Not metabolized with or without added biphenyl were 2,4,6-trichloro-, 2,4,2^1,4^1-tetrachloro- and 2,4,6,2^1-tetrachlorobiphenyl. Pseudomonas spp. metabolized only 2,4^1-dichloro-, 4,4^1-dichloro, and 2,5,4^1-trichlorobiphenyl. Both strains also metabolized 85% or more of Aroclors 1016 and 1242 in 100 days (Baxter et al., 1975).

Lake bacteria used Aroclor 1221 and 1242, but not 1254, as sole carbon and energy sources for growth. Aroclor 1221 was completely degraded into several low molecular weight compounds after one month. Of the seven bacterial isolates capable of degrading Aroclors, five were Achromobacter sp. and two were Pseudomonas sp. (Wong and Kaiser, 1975). In other similar studies with bacteria from Hamilton Harbour, Ontario, a solution containing 0.1% Aroclor 1242 was incubated at 20C for two months. After extraction, the metabolites were isolated and identified by gas chromatography and mass spectrometry. No phenols or chlorine-containing metabolites were observed. Metabolites identified included: iso-hexane, iso-octane, ethyl benzene, isobutyl benzene, n-butyl benzene and iso-nonane (Kaiser and Wong, 1974).

Aroclor 1254 was added to a loam soil at the rate of 10 ppm. After one year, 95% was recovered. In other soils, 25-50% of some of the peaks were lost (Iwata et al., 1973).

Unsymmetrical PCBs were irradiated in quartz with UV above 287 nm. Mono- and di-dechlorination was observed.

Starting biphenyl	Biphenyl product
2,4,5-trichloro-	4-chloro- 3,4-dichloro-
2,4,6-trichloro-	4-chloro- 2,4-dichloro-
2^{1},3,4-trichloro-	3,4-dichloro-
2,3,4,5-tetrachloro-	3,4-dichloro- 3,4,5-trichloro-
2,3,5,6-tetrachloro-	3,5-dichloro- 2,3,5-trichloro-

(Ruzo et al., 1975)

With irradiation of PCBs at 300 nm in cyclohexane, stepwise, dechlorination occurred. Where ortho-chlorines were present, PCBs yielded products arising from loss of the chlorines. The two main products accounted for more than 98% of PCB reacted. Less than 1% of the products arose from the loss of a meta-chlorine in the presence of ortho-chlorine. Para-chlorines were not cleaved after 20 h of irradiation. After more than 50 h of irradiation, 3,3^{1},4,4^{1}-tetrachlorobiphenyl yielded some 3,3^{1},4-trichlorobiphenyl in addition to 3,4,4^{1}-trichlorobiphenyl. In methanol solution, dechlorination was the major reaction; but some methoxylated products were also observed. In all cases, these comprised less than 3% of the reacted PCB. Results have been summarized in the following table (Ruzo et al., 1972, 1974a and b).

	Biphenyl Products	
Biphenyl used	Dechlorinated	Methoxylated
4,4^{1}-Cl_2	4-Cl-	
2,2^{1},5,5^{1}-Cl_4	2,3^{1},5-Cl_3- 3,3^{1}-Cl_2-(<1%) 3-Cl	Cl_3O Me- Cl_2(O Me)$_2$-(<1%)
2,2^{1},4,4^{1}-Cl_4-	2,4,4^{1}-Cl_3- 4,4^{1}-Cl_2- 4-Cl- (<1%)	Cl_3O Me- (*) Cl_2 (O Me)$_2$-(<1%)
2,2^{1},3,3^{1}-Cl_4-	2,2^{1},3-Cl_3-(<1%) 2,3,3^{1}-Cl_3- 3,3^{1}-Cl_2	Cl_3O Me- Cl_2(O Me)$_2$-(<1%)

*Main product was 2,4,4^{1}-Cl_3-2^{1}-OCH_3-biphenyl.

3,3^1,4,4^1-Cl_4-	3,4,4^1-Cl_3- 3,3^1,4-Cl_3 4,4^1-Cl_2-	Cl_3O Me-(<1%)
3,3^1,5,5^1-Cl_4-	3,3^1,5-Cl_3-(<1%)	--------
2,2^1,6,6^1-Cl_4	2,2^1,6-Cl_3- 2,2^1-Cl_2-(<1%)	Cl_3O Me-

Photolysis of 4,4^1-dichlorobiphenyl with 3100Å light in degased 2-propanol and methanol yielded HCl and 4-chlorobiphenyl. In other solvents and in the presence of oxygen, a complex mixture containing 4-chlorobiphenyl was obtained (Nordblom and Miller, 1974).

After photolysis (λ>286 nm) of 2,2^1,4,4^1,5,5^1-hexachlorobiphenyl in methanol in sealed borosilicate tubes for 1 h, about 10% of the biphenyl reacted to give 3,3^1,4,4^1-tetrachlorobiphenyl (70%) and 2,3^1,4,4^1,5-pentachlorobiphenyl (30%) (Ruzo and Zabik, 1975).

In other studies the 2,2^1,4,4^1,5,5^1-hexachloro- and 2,2^1,5,5^1-tetrachlorobiphenyls were adsorbed on silica gel and then exposed to UV irradiation at λ>290 nm. When quartz was used, mineralization occurred with the hexachlorobiphenyl but not with the tetrachlorobiphenyl (Gab et al., 1975a).

A sample of 2,2^1,4,4^1,6,6^1-hexachlorobiphenyl in hexane was irradiated at max 3100Å for 100 min. Thin-layer chromatography showed two bands and gas liquid chromatography revealed 11 major peaks. Mass spectrum analysis of one band indicated the presence of di-, tri-, tetra-, penta- and hexa-chlorobiphenyls. Irradiation in methanol produced similar results but also yielded some more polar material containing oxygenated polychlorinated compounds (Safe and Hutzinger, 1971).

Chlorinated biphenyls in a small volume of methanol were suspended in distilled water. These were then irradiated for 15-26 days in sunlight or 1-2 weeks in an aerated photoreactor (F40 BL lamp). GC/MS analyses indicated the formation (about 0.2%) of 2-chlorodibenzofuran from 2,5-dichloro- and 2,2^1,5,5^1-tetrachlorobiphenyl (Crosby and Moilanen, 1973).

	Product	
Test biphenyl	Reduced	Hydroxylated
2,4-Cl_2	-	+
2,5-Cl_2	+	+
3,4-Cl_2	-	-
4,4^1-Cl_2	+	+
2,4,5-Cl_3	-	-
2,2^1,3,3^1-Cl_4	+	+
2,2^1,4,4^1-Cl_4	-	-

PCB (not identified) in 2-propanol and sodium hydroxide was irradiated with 100 W-high pressure mercury lamp under nitrogen and at about 30C. PCB was dechlorinated about 80, 95 and 100% after 5, 10 and 15 min, respectively. Biphenyl and sodium chloride were identified (Nishiwaki et al., 1972).

The photolysis of chlorobiphenyls was studied under laboratory conditions and in sunlight. Progressive dechlorination and polymerization occurred in hexane. In hydroxylic solvents and at pH 9, irradiation of Aroclor 1254 produced compounds corresponding to the addition of water as well as more polar carboxylic material. When thin films of Aroclors were photolyzed in the presence of water, the major products were those resulting from dechlorination and/or polymerization. Polar compounds having little or no chlorine were also produced. 4,4^1-di-, 2,2^1,5,5^1-tetra-, 3,3^1,4,4^1-tetra, 2,2^1,4,4^1,5,5^1-hexa-, 2,2^1,3,3^1,4,4^1,5,5^1-octa- and decachlorobiphenyl were studied (Hutzinger et al., 1972a and d).

Aroclor 1254 was irradiated in hexane, water and benzene. Products were not identified; but the increase in size of some peaks indicated an increase in PCBs with lower molecular weights and shorter retention times (Herring et al., 1972).

UV irradiation of hexachlorobiphenyls in n-hexane, acetone, methanol, or methanol-water produced photolytic products which had lost one to six chlorine atoms (Hustert and Korte, 1972).

PCBs (KC 300, 400, 600) were chlorinated to decachlorobiphenyl by reaction with $AlCl_3$, SO_2Cl_2 and S_2Cl_2 at 65-70C for an hour (Nose, 1972). At 165C and 3 h and in the presence of antimony pentachloride, decachlorobiphenyl was produced. If bromine is present, a monobromo-nonachlorobiphenyl is produced (Huckins et al., 1974).

Solubility of PCB isomers in water was determined after allowing the solution to come to equilibrium. After 3 months, monthly measurements produced reproducable values (Haque and Schmedding, 1975).

Biphenyl	Solubility, ppb
2,4^1-Cl_2	637 ± 7
2,2^1,5-Cl_3	248 ± 4
2,2^1,5,5^1-Cl_4	26.5 ± 0.8
2,2^1,4,5,5^1-Cl_5	10.3 ± 0.2
2,2^1,4,4^1,5,5^1-Cl_6	0.95 ± 0.01

Recent studies have also shown that PCBs form colored complexes with montmorillonite clay (Haque and Hansen, 1974 and 1975).

1-Chloronaphthalene in corn oil was administered by retrocarotid injection in pigs. Analysis of urine samples showed the presence of a monohydroxy compound, identified as 4-chloro-1-naphthol, and a trace of a dihydroxy derivative (Ruzo et al., 1975).

2-Chloronaphthalene in corn oil was administered by retrocarotid injection in pigs. Analysis of urine samples showed the presence of monohydroxy compound, identified as 3-chloro-2-naphthol (Ruzo et al., 1975).

PCNB (Quintozene, Terraclor) [Pentachloronitrobenzene]

Soil samples were collected from greenhouses in which PCNB had been regularly used. Analyses indicated the presence of:

Pentachloronitrobenzene	(PCNB)
Pentachloroaniline	(PCA)
Pentachlorothioanisole	(PCTA)
Tetrachloronitrobenzene	(TCNB)
Tetrachloroaniline	(TCA)
Tetrachlorothioanisole	(TCTA)
Hexachlorobenzene	(HCB)
Pentachlorobenzene	(QCB)

Tetrachloronitrobenzene and penta- and hexachlorobenzene occurred as impurities in the technical PCNB. Analyses indicated the presence of 2,3,5,6-TCNB. Others have indicated that it is the 2,3,4,5-TCNB isomer that is present as an impurity. However, no pure 2,3,4,5-TCNB was available to check which isomer was actually present. Analyses were conducted with GLC, EC-GLC and MS-GLC (de Vos et al., 1974).

PCP [Pentachlorophenol]

Sprague-Dawley rats and NMRI mice were administered PCP in olive oil or propylene glycol. Most of the PCP was excreted unchanged. One metabolite was identified as tetrachlorohydroquinone (TCH). Both PCP and TCH were present in small amounts as conjugates (Ahlborg et al., 1974).

The amount of PCP accumulated by goldfish (Carassius auratus) increased with time. At 0.1 ppm, the concentration factor at 120 h was about 1000; at 0.2 ppm, about 580. Excretion was rapid with active elimination with half eliminated after 10 h in PCP-free water. Most of the PCP in the fish had not undergone decomposition. It appeared that most of PCP transferred to the hepatopancreas was detoxified by sulfate conjugation or by decomposition. Excretion of PCP was in the form of a conjugate identified as pentachlorophenylsulfate (Akitake and Kobayashi, 1975; Kobayashi and Akitake, 1975a and b).

Photolysis of PCP produced octachlorodibenzo-p-dioxin and a smaller amount of the heptachloro analog (Plimmer and Klingebiel, 1973). In other studies, photolysis of solid PCP in an oxygen stream produced some CO_2 and HCl (Gab et al., 1975a).

PHENMEDIPHAM (Betanal) [Methyl 3-(m-tolylcarbamoyloxy)phenylcarbamate]

In slightly acid soil of low humus content, phenmedipham decomposed with a half-life of 28 to 55 days (Kossmann, 1970).

PHOSPHAMIDON (Dimecron) [2-Chloro-N,N-diethyl-3-(dimethyl phosphate) crotonamide]

[^{14}C] Vinyl-carbonyl-phosphamidon was incubated with mouse liver fractions. Microsomes and homogenate plus supernatant metabolized phosphamidon into organosoluble metabolites. When there was pretreatment with dieldrin or phenobarbital, increased conversion of phosphamidon and its organosoluble metabolites into more polar substances was observed (Tseng and Menzer, 1974).

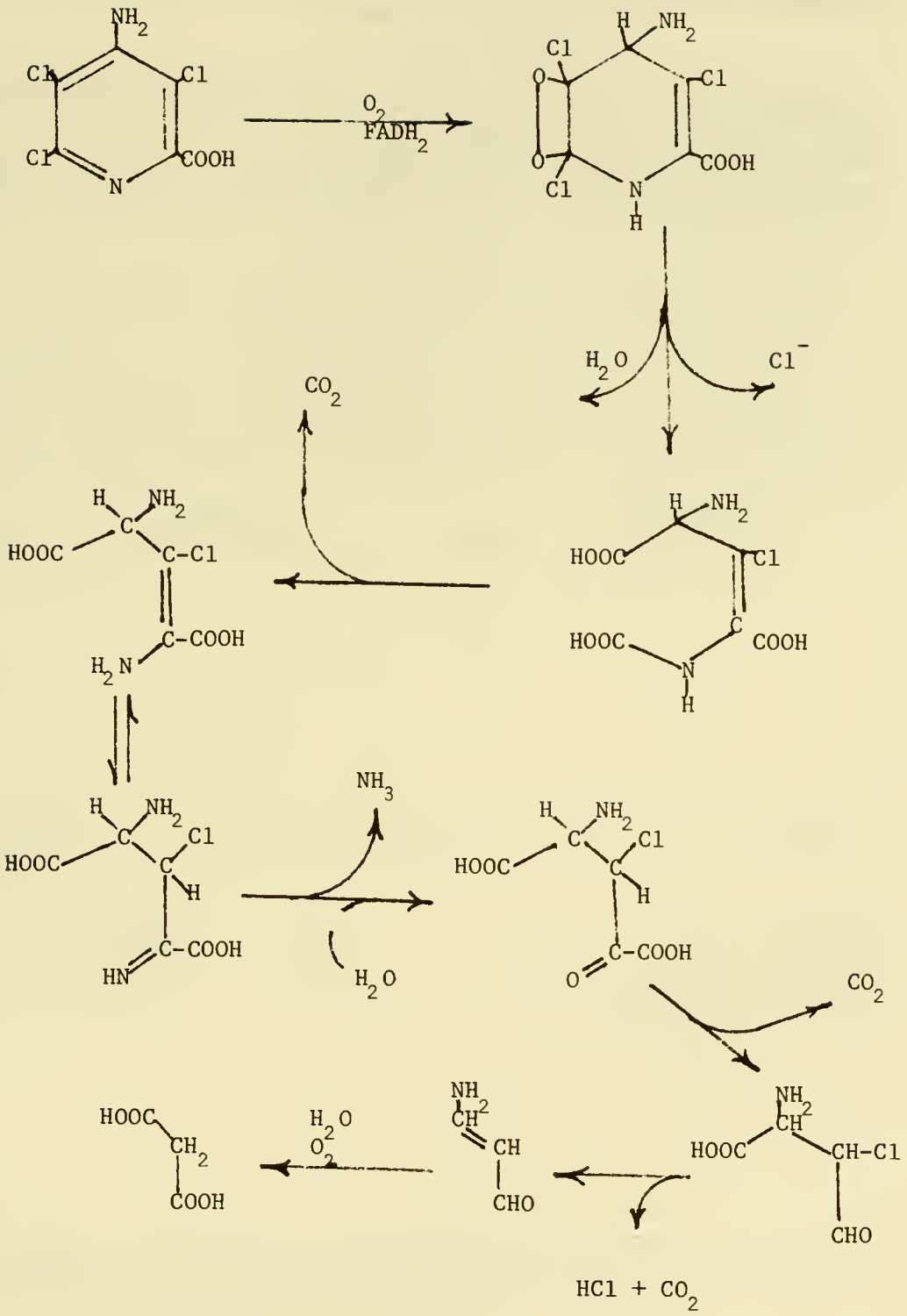
PICLORAM [4-Amino-3,5,6-trichloropicolinic acid]

When wheat seedlings (Trichicum aestivum L.) were treated with picloram, water-soluble conjugates not further identified were formed. Similar results were obtained with rape (Brassica napus L. cv. Nilla) (Hallmen, 1974 and 1975; Hallmen and Eliasson, 1972).

In soil, microbial degradation of picloram underwent a series of reactions that lead to CO_2 and chloride ion. Only the 6-hydroxy analog of picloram has been observed but it probably is not on the main degradative pathway. Apparently decarboxylation of picloram is not involved in this degradation either. A mechanism involving oxidation of the ring was proposed (Meikle et al., 1974).

The yeast Rhodotorula glutinis (Fres.) Harrison decarboxylated more than 19% of added picloram in less than 28 days when some dextrose was present. Aspergillus tamarii Kita and Trichoderma sp. also cometabolized picloram (Rieck, 1970).

Photolysis of picloram followed pseudo first-order kinetics. The half-life exhibited a straight line relationship vs solution depth (Hedlund and Youngson, 1972). During photolysis, two chloride ions were produced per molecule of picloram (Mosier and Guenzi, 1973).


NH2
Cl
Cl
Cl
COOH
N
O2
FADH2
H
NH2
Cl
O
Cl
O
Cl
COOH
N
H
H2O
Cl−
CO2
H
NH2
HOOC
C
C-Cl
C-COOH
H2N
H
NH2
HOOC
Cl
C
COOH
HOOC
N
H
NH3
H2O
H
NH2
HOOC
C
Cl
C
H
HN
C-COOH
H
NH2
HOOC
C
Cl
C
H
C-COOH
O
CO2
NH2
CH2
CH
CHO
NH2
CH
HOOC
CH-Cl
CHO
HOOC
CH2
COOH
H2O
O2
HCl + CO2

PIRIMIPHOS-METHYL [O,O-Dimethyl O-(2-dimethylamino-4-methylpyrimidin-6-yl)phosphorothionate]

Pirmiphos-methyl was applied to a clean wooden plank upon which a new cloth-wrapped cheese was placed. Very little pesticide was absorbed by the cheese and TLC analyses indicated only traces of the oxon and pyrimidinol derivatives in the wrapping (Thomas and Rowlands, 1975).

PROLAN [1,1-Bis(p-chlorophenyl)-2-nitropropane]

^{14}C-Prolan was administered in olive oil orally to mice. Urine and feces were collected and analyzed. Results of these analyses are tabulated.

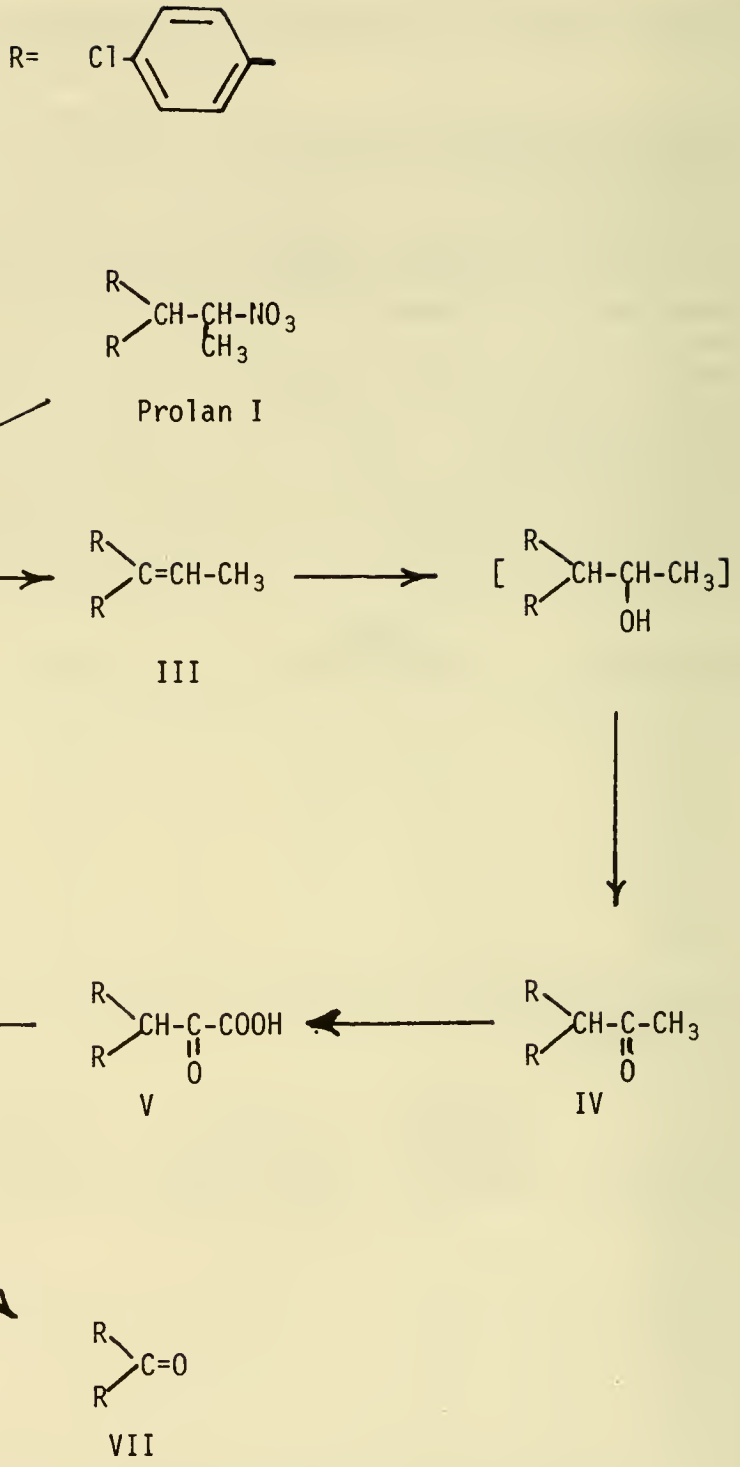
^{14}C-Labeled prolan was incubated with a preparation of sheep liver microsomes. After 30 min incubation at 39C, 99% intact prolan was recovered. In a comparative study with methoxychlor, about 88% remained intact. When larva of the salt marsh caterpillar (Estigmene acrea) ingested ^{14}C-prolan, 9% degraded to the 2-propanone and acetic acid analogs. After topical application to the S_{NAIDM} female housefly (Musca domestica), ^{14}C-prolan was degraded to polar compounds which comprised 83% of the label in excreta and 75% in body homogenates. In addition to prolan, the acetic acid and benzophenone analogs were indicated. Conjugation was also indicated by the release of the 2-amino analog after hydrochloric acid treatment of the polar material.

Metabolite	Mouse		Fly	Salt Marsh Caterpillar
	Urine	Feces		
I	+	+	+	+
II	+	+	+	
III	+	+	+	
IV	+	+	+	+
V	+	+	+	
VI	+	+	+	+
VII	+	+	+	

The degradation and accumulation of prolan in a model ecosystem was also studied.

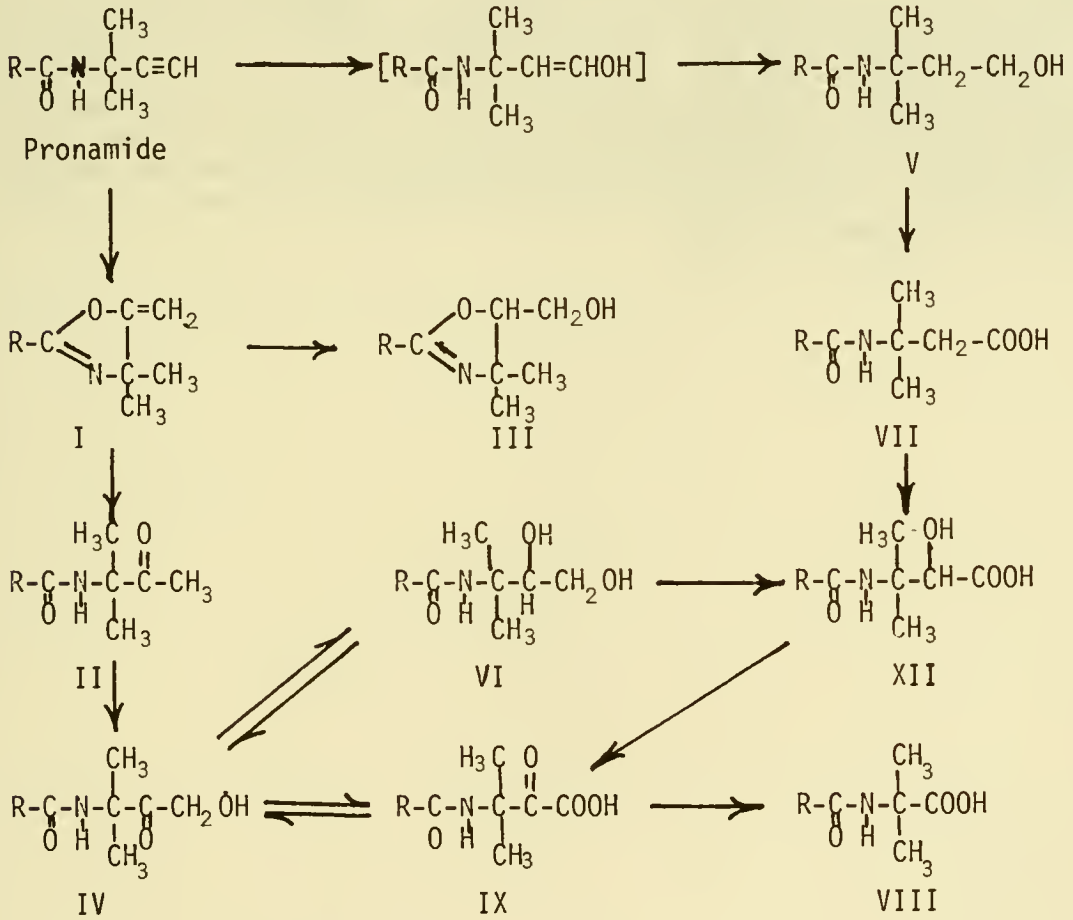
Compound	Oedogonium (alga)	Daphnia (Waterflea)	Physa (Snail)	Culex (Mosquito)	Gambusia (Fish)
I	+	+	+	+	+
II	+	+	+		+
III	+		+	+	
IV	+	+	+		+
V	+		+		
VI	+		+		+
VII	+				

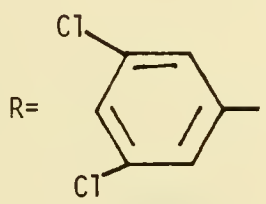
In all studies, polar conjugated materials were also observed as well as several unidentified compounds (Hirwe et al., 1975).


R=
Cl
Prolan I
II
III
IV
V
VI
VII

PRONAMIDE (Kerb) [3,5-Dichloro-N-(1,1-dimethyl-2-propynyl)benzamide]

The metabolism of pronamide in soil was studied with ^{14}C-carbonyl labeling. It was shown that formation of $^{14}CO_2$ was biologically mediated. The treated soils were extracted after 33 days and the extracts were chromatographed. No 3,5-dichlorobenzoic acid was detected. In addition to pronamide and two unidentified metabolites, five metabolites indentified were compounds I, II, III, VII, and VIII (Fisher, 1974).





PROPANIL [3,4-Dichloropropionanilide]

In studies with propanil and 3,4-DCA (3,4-dichloroaniline), it was observed that in different soils there were differences in the amount of tetrachloroazobenzene (TCAB) formed. Formation of TCAB did not correlate with soil pH. With five soils of pH 4.5 to 5.5, TCAB was formed from both compounds; pH 5.8 and 7.4, TCAB formed from 3,4-DCA only; pH 3.4 and 6.4, no TCAB from either substrate (Hughes and Corke, 1974). In soil, the conversion of 3,4-DCA to TCAB increased with an increase in peroxidase activity (Lay and Ilnicki, 1974).

Propanil was hydrolyzed by Corynebacterium pseudodiphtheriticum NCIB 10803 (Grant and Wilson, 1973). A strain of Fusarium solani used propanil as a sole source of carbon and energy for growths and the primary product of degradation was 3,4-DCA. This acylamidase did not catalyze hydrolysis of dicryl, karsil, fenuron, monuron or IPC. It seems to be different than acylamidases isolated from rice, rat liver and chick kidney (Lanzilotta, 1969). Studies with rice have shown that diazinon and carbaryl, absorbed from soil and translocated, could inhibit propanil hydrolysis (El-Refai and Mowafy, 1973).

PROXIMPHAM [O-(N-Phenylcarbamoyl)isopropyloxime]

Hydrolysis of proximpham in acid produced diphenylurea, aniline and isopropoxime. In alkaline solution, the salt of carbanilic acid and isopropoxime formed. Model tests of proximpham degradation in soil indicated a half-life of 7-10 days. In soil, aniline was degraded much more rapidly than proximpham and was not detected (Spengler and Jumar, 1969).

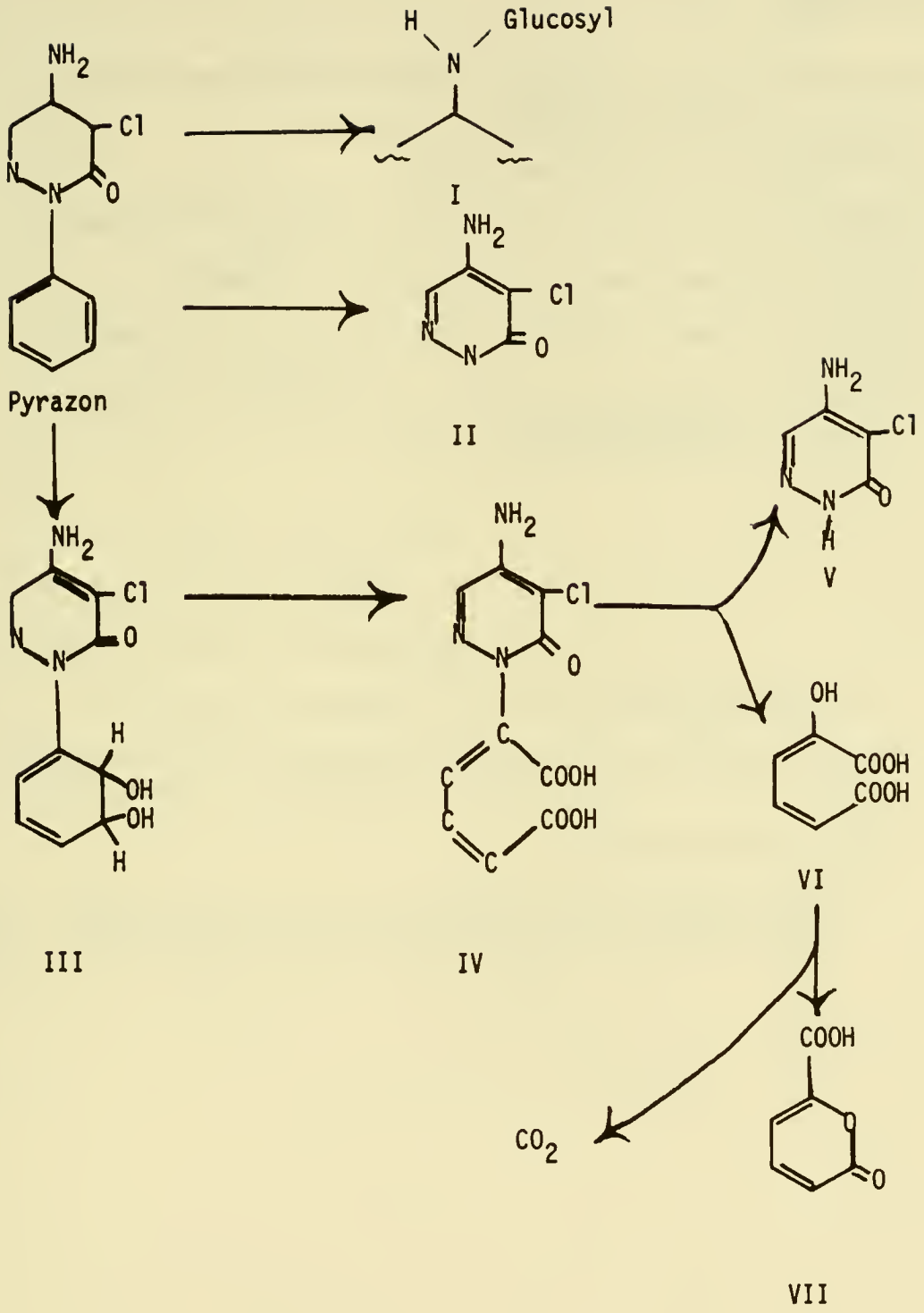
PYRAZON (Pyramin) [5-Amino-4-chloro-2-phenyl-3(2H)-pyridazinone]

A photochemically controlled process was involved in the degradation of pyrazon in the leaves of beets. No degradation occurs in the dark. In sugar beets and red beets, pyrazon formed N-glucosylpyrazone (I). In addition 5-amino-4-chloro-3(2H)-pyridazinone (II) and a strongly hydrophilic compound were formed (Anon., BASF 1973; Stephenson et al., 1971).

In soil pyrazone is degraded by bacteria with formation of compound II and is not detectable in soil after 20 weeks (Anon., BASF 1973).

A strain of Azotobacter sp., capable of using pyrazon as a sole source of carbon, was used to study bacterial decomposition of pyrazon. Incubation of the bacteria with pyrazon produced 5-amino-4-chloro-2-(2,3-cis-dihydroxycyclohexa-4,6-diene-1-yl)-3(2H)-pyridazinone (III); 2-(5-amino-4-chloro-3-oxo-2,3-dihydro-2-pyridazino-cis,cis-muconic acid (IV); 2-pyrone-6-carboxylic acid (VII); and 5-amino-4-chloro-3(2H)-pyridazinone (V) (de Frenne et al., 1973).

Analogs of pyrazon were co-metabolized by Azotobacter sp. When o-methylpyrazon was used, the 2-hydroxymethylphenyl derivative was formed and with m-methylparazon, the 3-hydroxymethylphenyl derivative. The p-methylpyrazon was not metabolized (de Frenne et al., 1974).


H
Glucosyl
N
NH2
Cl
N
N
O
I
NH2
Cl
N
N
O
Pyrazon
II
NH2
Cl
N
N
O
H
OH
OH
H
III
NH2
Cl
N
N
O
C
COOH
C
C
COOH
C
IV
NH2
Cl
N
N
H
V
OH
COOH
COOH
VI
COOH
O
O
CO2
VII

PYRETHRINS

BIO-ALLETHRIN [3-Allyl-2-methyl-4-oxocyclopent-2-enyl (±)-trans-chrysanthemate]

BIORESMETHRIN [(±)-trans-resmethrin]

FURAMETHRIN [5-Propargyl-2-furylmethyl (+)-trans-chrysanthemate]

PHENOTHRIN [3-Phenoxybenzyl (+)-trans-chrysanthemate]

RESMETHRIN [5-Benzyl-3-furylmethyl (±)-cis,trans-crysanthemate]

TETRAMETHRIN [3,4,5,6-Tetrahydrophthalimidomethyl (±)-trans-chrysanthemate]

Mouse liver microsomal preparations were capable of hydrolyzing the ester link of pyrethroids made with primary alcohols. The enzyme system did not hydrolyze the esters formed with secondary alcohols. Several relationships were observed.

1. Cleavage of (+)-trans chrysanthemate esters: rate decreased in order of:

   5-propargyl-2-furylmethyl

   5-benzyl-3-furylmethyl

   3-phenoxybenzyl

   tetrahydrophthalimidomethyl

2. Hydrolysis rate of benzylfurylmethyl esters decreased in order of:

   (+)- or (-)-trans chrysanthemate

   (+)-trans-ethanochrysanthemate

   tetramethylcyclopropanecarboxylate

   (+)- or (-)-cis-chrysanthemate or (+)-cis-ethanochrysanthemate

3. Trans-isomers of the above were hydrolyzed up to 50-fold more rapidly than the cis-isomers.

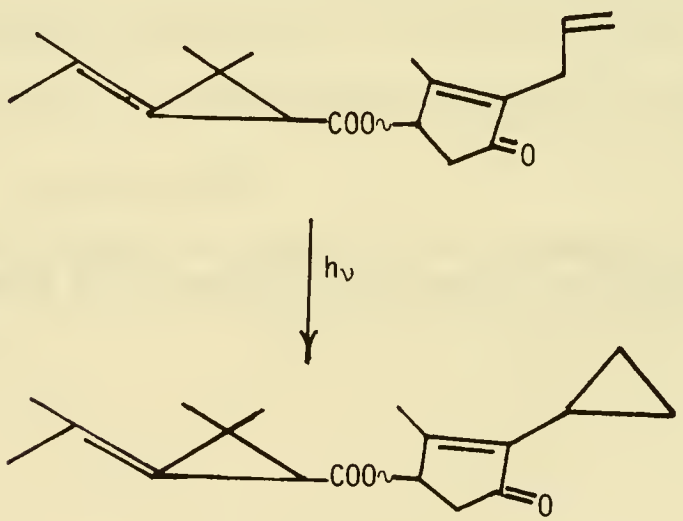
(Abernathy et al., 1973)

In other studies, pyrethroids were decomposed in solution by tabu powder. Decomposition was caused by the prophyrin ring in the pheophytin in solution (Iguchi et al., 1974).

BIOALLETHRIN

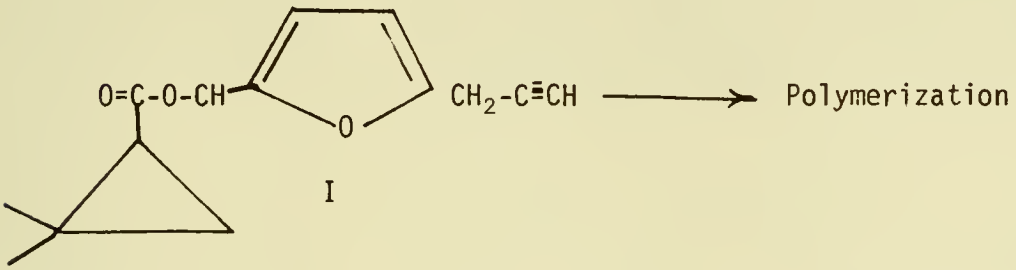
Irradiation of a 0.15% solution of a diastereoisomeric mixture of commercial bioallethrin in n-hexane produced a single photoproduct in up to 90% yield. Mass, NMR and IR spectral data were used to identify the product as corresponding to a rearrangement in the prop-2-enyl side chain of the alcohol moiety (Bullivant and Pattenden, 1973).

Bioallethrin was not cleaved by esterases in acetone powder preparation from milkweed bugs, cockroaches (Blattella germanica L.), houseflies, cabbage loopers (Trichoplusia ni Hubner), yellow meal worms (Tenebrio molitor L.) or mouse liver (Jao and Casida, 1974).

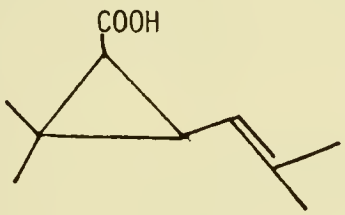


FURAMETHRIN

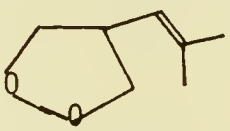
Purified furamethrin (I) was heated at 120-140C for 10 h. Chyrsanthemic acid (II) and an unidentified compound were formed in very small amounts. At 150C for 8 h, about 0.5% pyrocin (III) and cis-dihydrochrysanthemo-δ-lactone (IV), chrysanthemic acid and an unidentified compound were formed. At 200C for 7 h, in addition to four unidentified compounds, chrysanthemic acid and two aldehydes (V and VI) were formed (Abe et al., 1974).



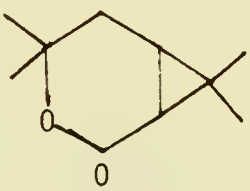
I



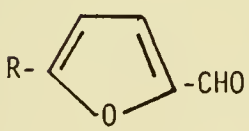
II



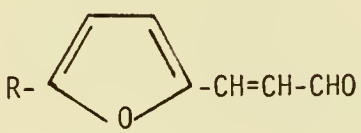
III



IV



V



VI

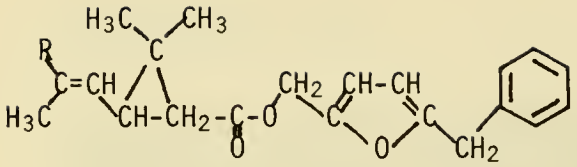
PHENOTHRIN (S 2539)

^{14}C-Phenothrin, labeled at the hydroxymethyl group of the alcohol moiety, was orally administered at the rate of 200 mg/kg to male Sprague-Dawley rats. Absorption and elimination was rapid. About 60% of the radioactivity was eliminated in urine and 40% in feces in 3 days. In addition to phenothrin, 3-phenoxybenzyl alcohol and 3-phenoxybenzoic acid were found in brain, liver, kidney and blood. Unidentified water and ether solubles were also present. Urine contained low levels of 3-phenoxybenzoic acid and its glycine conjugate and some ether and water soluble material. In addition to these, 3-(4'-hydroxyphenoxy)benzoic acid was present and accounted for 42.3% of the radioactivity originally applied. This compound was also the major metabolite in feces but accounted for only 11.9% of the initially applied radioactivity. In addition to unchanged phenothrin and unidentified water and ether solubles, feces contained 3-phenoxybenzoic acid and the glycine conjugate. 3-Phenoxybenzyl alcohol was not observed in urine or feces (Miyamoto et al., 1974).

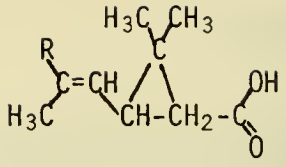
Mouse hepatic microsomal esterases cleaved the (+)-trans ester more rapidly than the (+)-cis-isomer (Abernathy and Casida, 1973). Acetone powder preparations of milkweed bugs, cockroaches (Blatella germanica L.), houseflies, cabbage loopers (Trichoplusia ni Hubner) and yellow mealworms (Tenebrio molitor L.) also hydrolyzed both (+)-trans- and (+)-cis-isomers. Of these, the (+)-trans-isomer was cleaved more rapidly than the (+)-cis-isomer (Jao and Casida, 1974). Other studies indicated that microsomal esterases were important in hydrolyzing trans- but not cis-isomers (Ueda et al., 1975b).

Resmethrin isomers were metabolized in microsome-NADPH systems to the extent of 95 to 98%. The extent to which trans- and cis-isomers were metabolized differed. In the presence of NADPH, ester cleavage was much greater with TEPP-treated microsomes. An oxidative ester cleavage seemed to be most important with (-)-cis-resmethrin. In the latter case, alcohol moieties released include unstable compounds and protein-bound metabolites. Seventeen percent of the initial radiocarbon appeared in 11 ester metabolites (not identified) of (+)-trans-resmethrin. These were recovered only with TEPP-treated microsomes fortified with NADPH. Oxidized chrysanthemic acid derivatives (VIII to XXII and XXVI) were comparatively stable. The metabolites IV and VII were major products only in the presence of NADPH and the supernatant fraction. Compounds II, III, V and VI were not isolated (Ueda et al., 1975b).

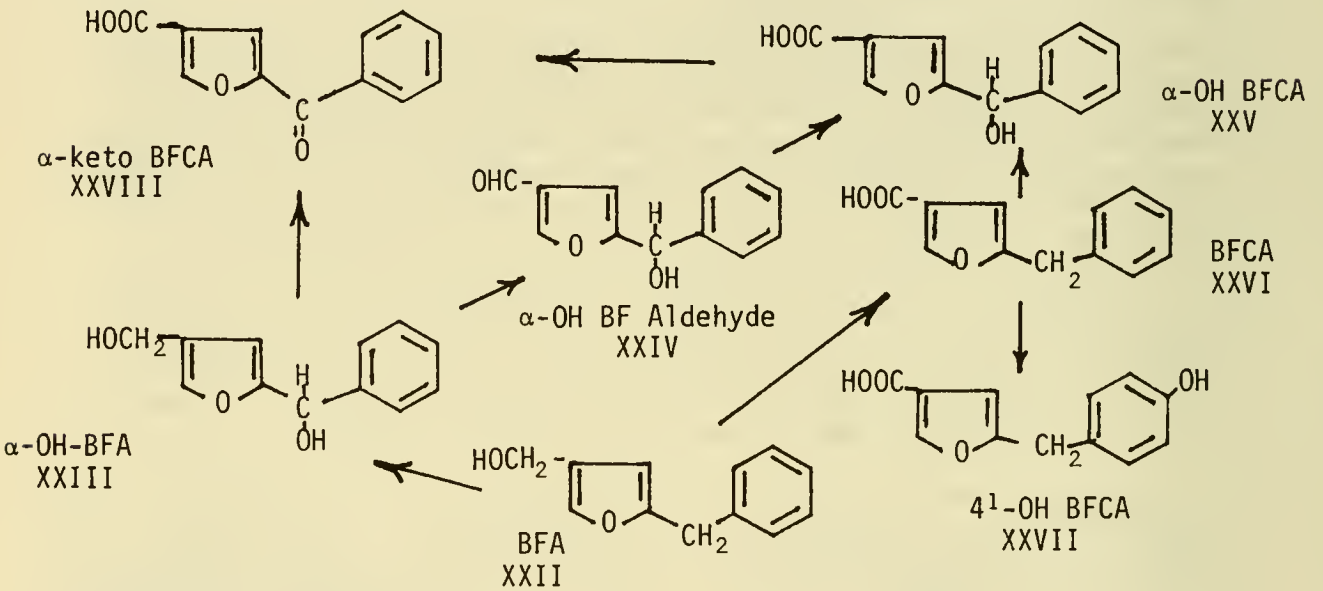
Six days after administration of ^{14}C-resmethrin to rats at a dose of 1 mg/kg, 53-73% was accounted for in urine and feces. Low residue levels were observed in tissues. In urine, there were almost equal parts of free and conjugated metabolites. Much of the conjugated material was released after incubation at pH 5, in buffer, with or without glusulase. When the aqueous phase was acidified to pH 2, more of the metabolites were recovered. Differences were observed between the metabolism of (+)-trans- and (+)-cis-resmethrin. When the alcohol was labeled, compounds BFCA, α-OH-BFCA and 4'-OH-BFCA were found in urine after administration of (+)-trans-isomer whereas only BFCA was observed after the (+)-cis-isomer. With the latter, all three compounds were released by incubation with glusulase but only BFCA and 4'-OH-BFCA were released when the (+)-trans-isomer was used. After administration of acid labeled (+)-trans-resmethrin, tE-CDA and t-CA were found in urine. The t-CHA found probably consisted of t-CHA from both (+)-trans- and (+)-cis-resmethrin. The (+)-cis-isomer yielded c-CA, cE-CHA, cE-CDA and cZ-CDA when the acid moiety was labeled (Ueda et al., 1975a).

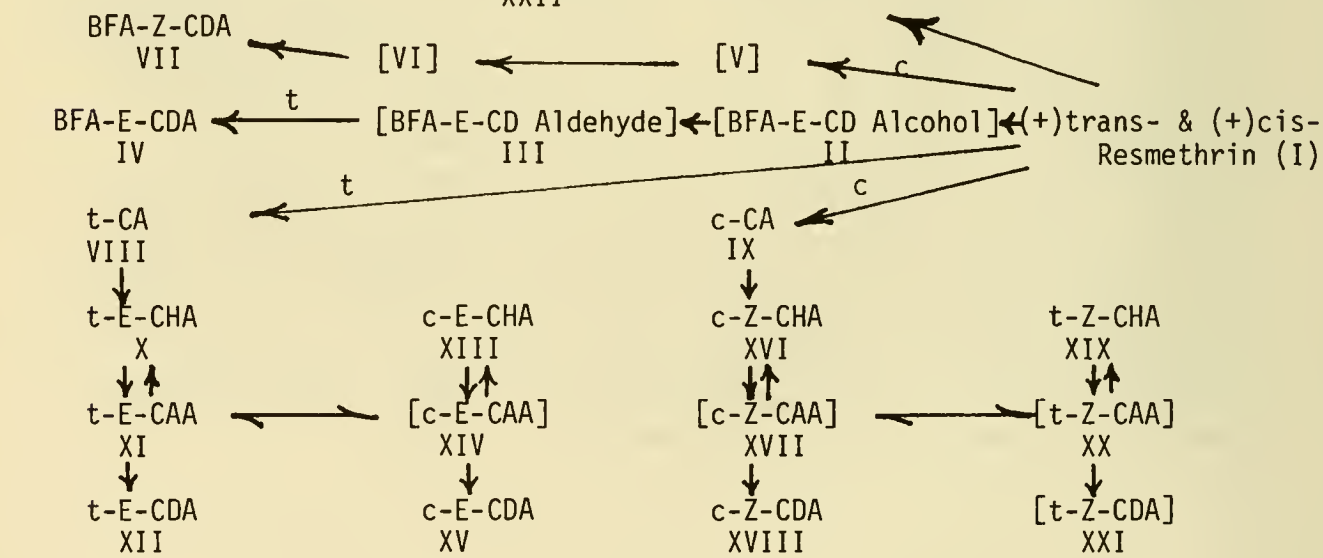


R_1	$-CH_3$	Resmethrin
R_2	$-CH_2OH$	BFA- -CD Alcohol
R_3	$-CHO$	BFA- -CD Aldehyde
R_4	$-COOH$	BFA- -CD Acid



R_1	$-CH_3$	CA
R_2	$-CH_2OH$	CHA
R_3	$-CHO$	CAA
R_4	$-COOH$	CDA

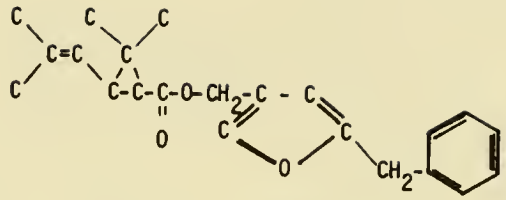




Acid derivatives	Observed	
	In Vitro	In Vivo
t-CA	+	+
c-CA	+	+
tE-CHA	+	+
tE-CAA	+	+
tE-CDA	+	+
cE-CHA	+	+
cE-CAA	+	
cE-CDA	+	+
cZ-CHA	+	+
cZ-CAA	+	
cZ-CDA	+	+
tZ-CHA	+	
tZ-CAA	+	±
tZ-CDA	+	±
Alcohol derivatives		
BFA-E-CDA	+	
BFA-Z-CDA	+	
BFA	+	
α-OH-BFA	+	
BFCA	+	+
α-OH-BFCA		+
4'-OH-BFCA		+

Photodecomposition of (+)-cis-resmethrin produced cis-chrysanthemic acid; benzaldehyde; phenylacetic acid; 5-benzyl-5-hydroxy-2-oxo-2,5-dihydro-3-furylmethyl cis-chrysanthemate and 4-benzyl-5-hydroxy-3-oxo-cyclopent-1,2-enylmethyl cis-chrysanthemate (Ueda et al., 1974).

Irradiation of the (+)-trans-isomer produced 11 photoproducts. The major component was trans-chrysanthemic acid. Other compounds observed included benzaldehyde (VIII); 2-benzyloxy-5-oxo-2,5-dihydro-3-furylmethyl trans-chrysanthemate (IV); compound III, compound V, benzyl alcohol, benzoic acid, phenylacetic acid and two epoxyresmethrin isomers (see following figure) (Ueda et al., 1974).



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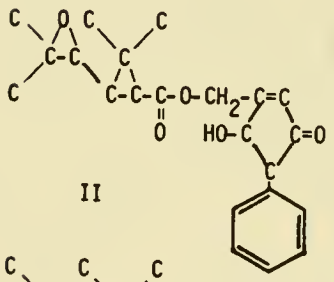
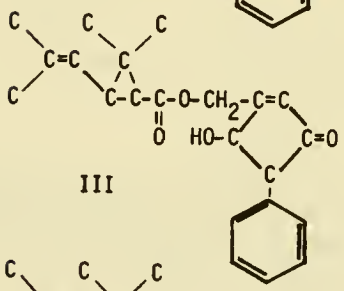


II



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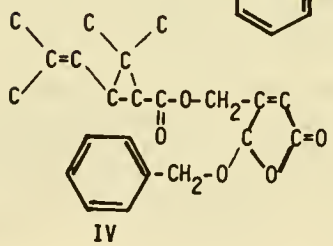

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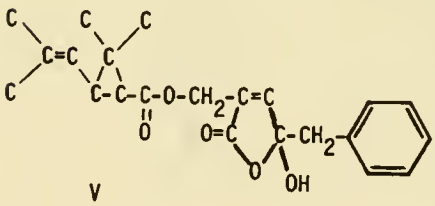
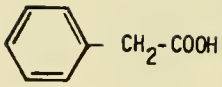
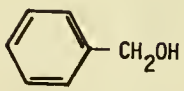


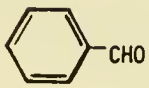
V



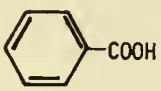
VI



VII



VIII

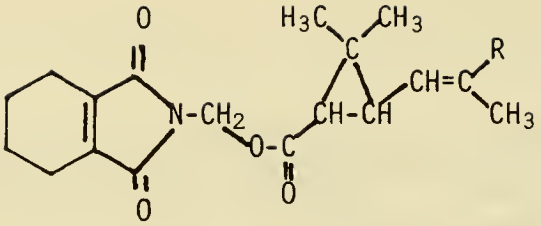


IX

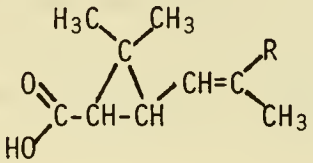
The microsomal fraction of rat liver homogenates was incubated with tetramethrin (I). The major metabolites were chrysanthemumic acid (V) and tetradydrophthalimide (X). Addition of $NADPH_2$ decreased formation of compound (X), increased tetramethrin oxidation and produced primarily the propenol derivative II and the hydroxychrysanthemumic acid VI. Only a small amount of the hydrolysis product N-hydroxymethylphthalimide (IX) was observed. This product went non-enzymatically to compound X. Minor amounts of compounds III, IV, VII and VIII were also observed (Suzuki and Miyamoto, 1973).

Studies conducted with homogenates of three strains of houseflies showed that the major microsomal metabolites were compounds V and X. Addition of $NADPH_2$ did not affect formation of compound X. Small amounts of compounds III, IV, VII and VIII were observed. Similar results were obtained when tetramethrin was applied to flies by injection (Miyamoto and Suzuki, 1973; Suzuki and Miyamoto, 1974).

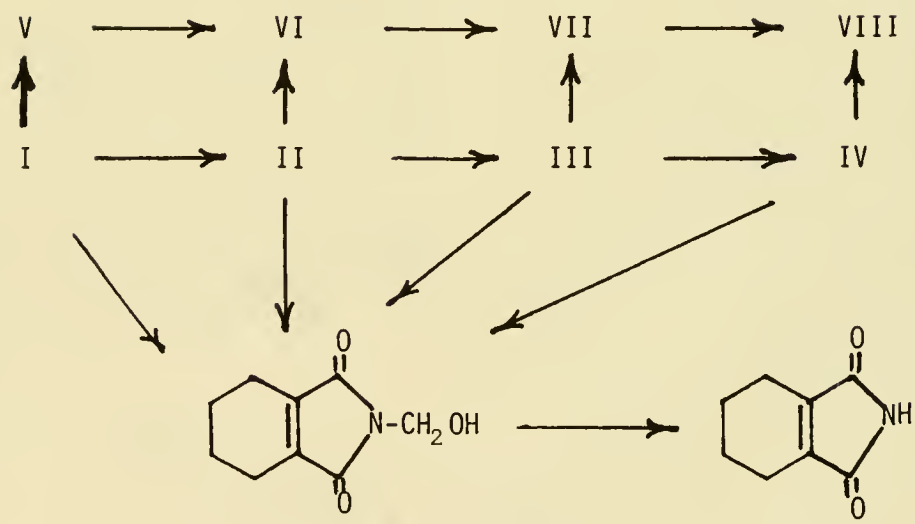
Acetone powder preparations of milkweed bugs, cockroaches (Blattella germanica L.), houseflies, cabbage loopers (Trichoplusia ni Hubner) and yellow mealworms (Tenebrio molitor L.) hydrolyzed both (+)-trans- and (+)-cis-isomers of tetramethrin. Of these two isomers, the (+)-trans-isomer was cleaved more rapidly (Jao and Casida, 1974).



I	R=	$-CH_3$
II		$-CH_2OH$
III		-CHO
IV		-COOH



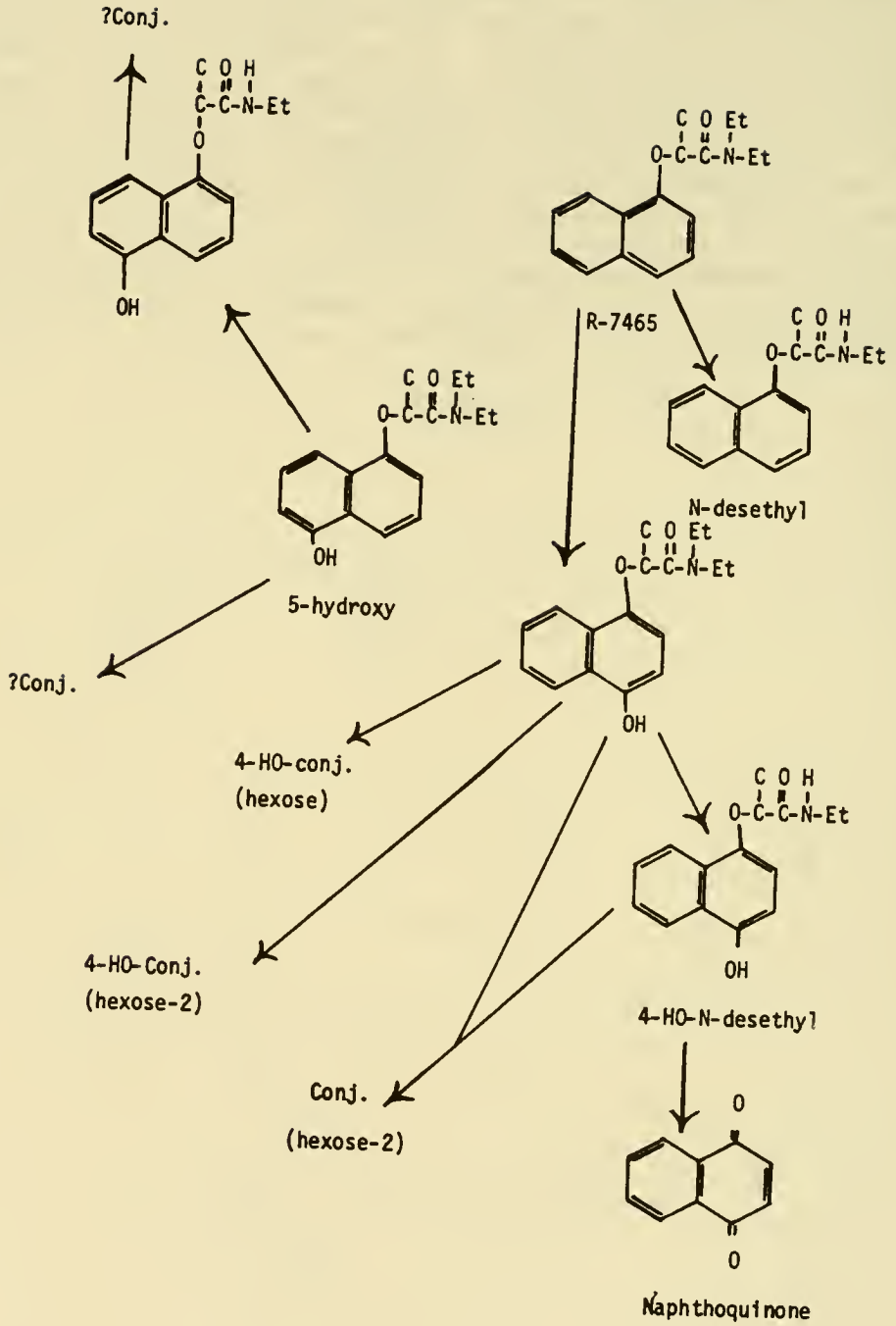
V	$-CH_3$
VI	$-CH_2OH$
VII	-CHO
VIII	-COOH



IX X

R-7465 [2-(α-naphthoxy)-N,N-diethyl propionamide]

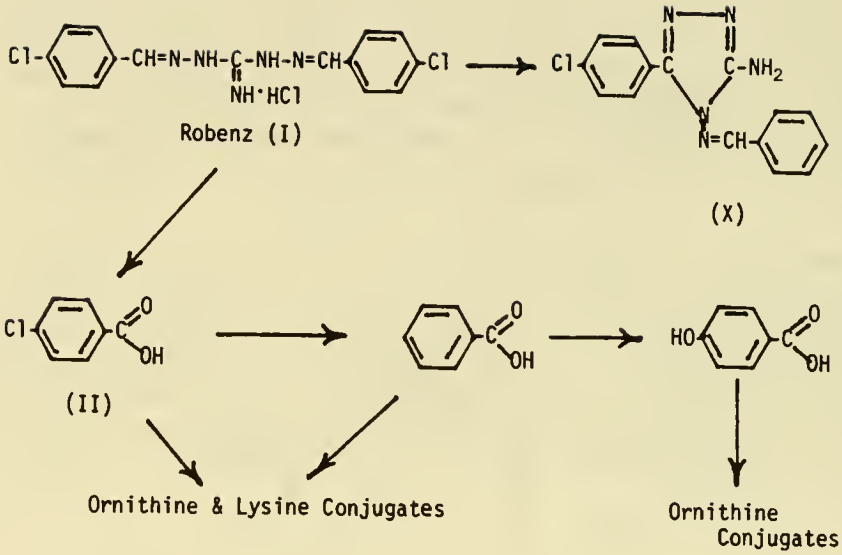
Labeled R-7465 was applied to roots of tomato plants. Translocation of the material throughout the leaves occurred within 8 h. Extraction of the plants and analyses revealed the presence in the organic fraction of three metabolites identified as: 2-(α-naphthoxy)-N-ethyl propionamide; 2-(5-hydroxy-1-naphthoxy)-N-ethyl propionamide; 2-(5-hydroxy-1-naphthoxy)-N,N-diethyl propionamide; and 1,4-naphthoquinone. The aqueous fraction contained a conjugate of 4-hydroxy R-7465 identified as a hexose conjugate. Two other compounds, which gave positive glycoside tests, were identified as different hexose conjugates of 4-hydroxy R-7465 and a hexose conjugate of the monodesethyl 4-hydroxy R-7465. The 5-hydroxy R-7465 and its desethyl analog were also present in small amounts after enzymatic hydrolysis of the conjugates. However, the sources of these two compounds after hydrolysis were unresolved (Murphy et al., 1973).

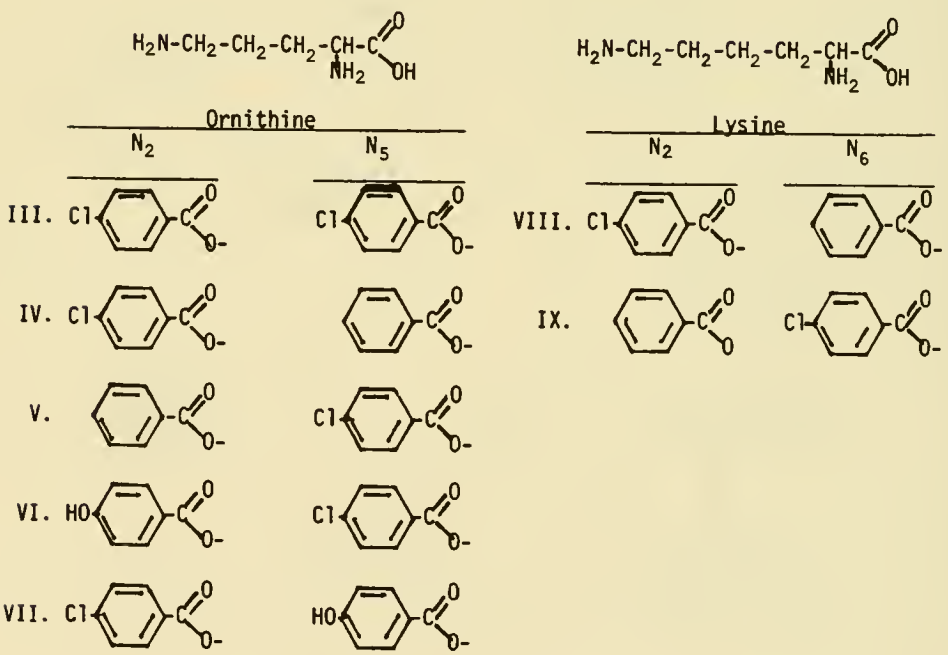

?Conj.
OH
R-7465
N-desethyl
OH
5-hydroxy
?Conj.
4-HO-conj.
(hexose)
OH
4-HO-Conj.
(hexose-2)
4-HO-N-desethyl
Conj.
(hexose-2)
O
O
Naphthoquinone

ROBENZ (Robenidine hydrochloride) [1,3-Bis(p-chlorobenzylideneamino) guanidine hydrochloride]

Carbonyl-^{14}C-labeled robenz was administered as a single dose to rats. A small amount was converted to CO_2. The major urinary metabolite (88%) was identified as p-chlorohippuric acid. A minor metabolite, about 2% of urinary radioactivity, was identified as p-chlorobenzoic acid. About 12 other unidentified materials were also present. More than 60% of the radioactivity in feces was unreacted robenz. Liver, kidney and muscle contained p-chlorohippuric acid, p-chlorobenzoic acid and robenz (Zulalian and Gatterdam, 1973).

Chickens were administered robenidine (I) ^{14}C-labeled in the α-carbon of p-chlorobenzylidene or in the aminoguanidine carbon. Within 24 h, 82% of the label had been excreted, mostly as unchanged robenidine. After solvent extraction, column chromatography and mass spectral analyses, the metabolites were identified as p-chlorobenzoic acid (II), 3-amino-4-(p-chlorobenzylideneamino)-5-(p-chlorophenyl)-(4H)-1,2,4-triazole (X), and compounds III to IX (Zulalian et al., 1975).


Robenz (I)
(X)
(II)
Ornithine & Lysine Conjugates
Ornithine Conjugates


Ornithine
N2
N5
Lysine
N2
N6
III.
IV.
V.
VI.
VII.
VIII.
IX.

ROTENONE

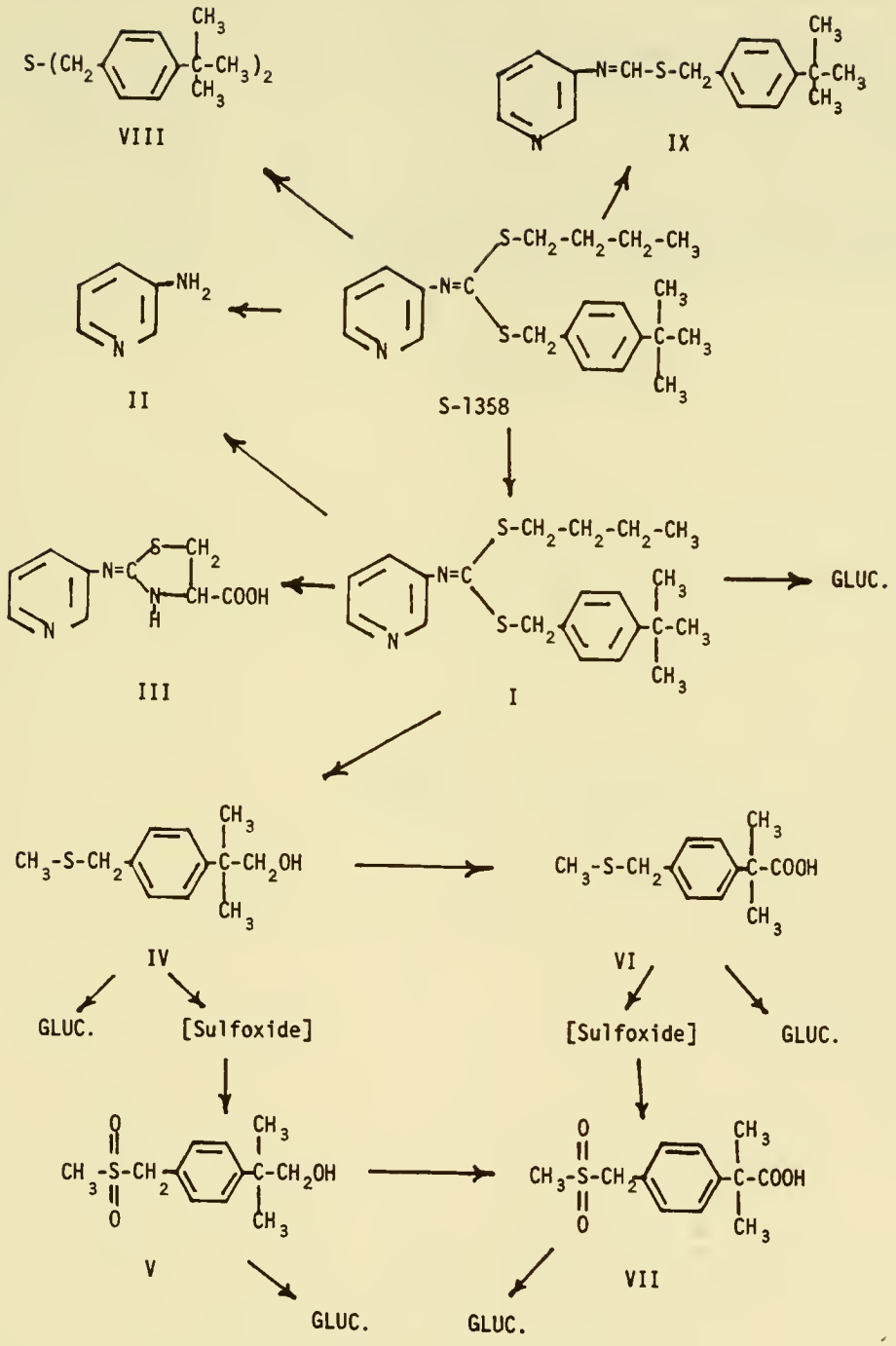
Rotenone is stable in the solid state but degradation is accelerated by the presence of organic solvents. Air and light are required (Cahn et al., 1945). The rate of decomposition varies with solvents, temperature and access of air. Decomposition produced the yellow crystalline dehydrorotenone and rotenononе and a complex mixture of other oxidation products of rotenone (Jones and Haller, 1931).

S-1358 [S-n-Butyl S'-(p-tert-butylbenzyl)-N-3-pyridyldithiocarbonimidate]

Male Sprague-Dawley rats were intubated with an aqueous suspension of labeled S-1358 in 10% Tween 80. Excretion of radioact-vity was almost complete within 4 days. Depending on the dose level, 36 to 43% was excreted in urine and 54 to 57% in feces. After separation of metabolites by TLC, identification procedures included the use of NMR, IR, MS and derivatization:

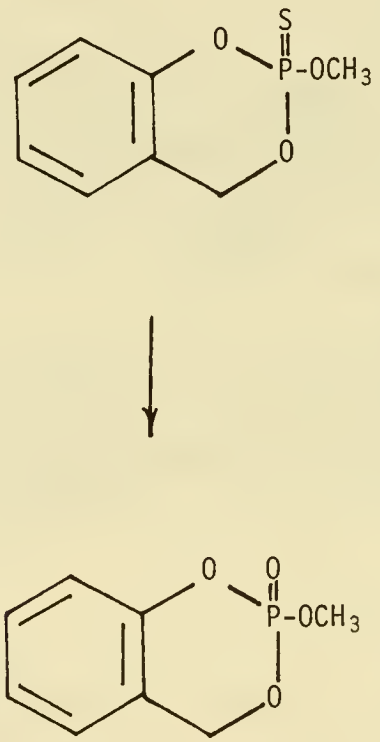
I. S-n-butyl S'-(p-2-methylisopropanol)benzyl N-3-pyridyldithiocarbonimidate
II. 3-aminopyridine
III. 2-(3'-pyridylimino)-4-carboxythiazolidine
IV. 4-(2-methylisopropanol)benzyl methylsulfide
V. 4-(2-methylisopropanol)benzyl methylsulfone
VI. 4-[2-(2-methylpropanoic acid)]benzyl methylsulfide
VII. 4-[2-(2-methylpropanoic acid)]benzyl methylsulfone
VIII. bis(p-tert-butylbenzyl disulfide)
IX. S-(p-tert-butylbenzyl)N-3-pyridyldithiocarbamate

Urine contained metabolites I, IV, V, VI and VII. The latter four were also present probably as glucuronide conjugates. Feces also contained metabolites I, II, IV, V, VI and VII. Analysis of bile indicated the presence of compounds IV, V, VI and VII both free and as glucuronides, metabolite I free and conjugated, metabolites VIII and IX. In each case, there was also some unidentified material (Ohkawa et al., 1975).


VIII
IX
II
S-1358
III
I
GLUC.
IV
VI
GLUC.
[Sulfoxide]
[Sulfoxide]
GLUC.
V
VII
GLUC.
GLUC.

SALITHION [2-Methoxy-(4H)-1,3,2-benzodioxaphosphorin-2-sulfide]

After oral administration of ^{32}P-salithion to mice, analyses indicated that this material was rapidly degraded and excreted. In houseflies, salithion persisted for a comparatively long time. Analysis of the chloroform-soluble fraction of houseflies indicated the presence only of the oxo analog (Ohkawa et al., 1970).



SAN-6706 [4-Chloro-5-dimethylamino-2-(α,α,α-trifluoro-m-tolyl)-3(2H)-pyridazinone]

SAN-9789 (Norflurazone) [4-Chloro-5-methylamino-2-(α,α,α-trifluoro-m-tolyl-3(2H)-pyridazinone]

Both compounds were readily absorbed from nutrient solution by cotton (Gossypium hirsutum L. "Coker 203"), corn (Zea mays L. "WF9") and soybean (Glycine max (L.) Merr. "Lee") plants. In corn and soybean plants, these compounds were translocated more rapidly and in greater amount than in cotton. SAN-6706 was not degraded to any great extent in cotton. In corn and soybean, significant amounts of the mono- and des-methyl derivatives were seen after 24 h. Metabolism of SAN-9789 also was more rapid in corn and soybean than in cotton (Strang and Rogers, 1974).

SUMITHION (Fenitrothion, Accothion) [O,O-Dimethyl O-(3-methyl-4-nitrophenyl)phosphorothioate]

Labeled sumithion was applied to apple tree. After 21 days, nearly 86% of the residue on the apple surface was sumithion. In the tissues degradation was more rapid, and approximately 45% of the radioactive carbon was in the form of degradation products. In addition to sumioxon, the S-methyl isomer of sumithion, p-nitrocresol and its β-glucoside, and desmethylsumithion were also found. The latter product was detected only in fruit harvested on the 21st day (Hosokawa and Miyamoto, 1974).

Irradiation of sumithion in various solvents produced considerable photodecomposition rate variations.

Product	Solvent/Surface				
	Water	Acetone	1*	2*	Bean leaves + sunlight
Carboxysumithion	+	+	+	+	+
Methylparathion		+			
Sumioxon	+	+	+	+	+
Carboxysumioxon		+	+	+	
3-methyl-4-nitrophenol	+	+	+	+	+
3-carboxy-4-nitrophenol	+	+		+	+
Sumithion S-isomer	+				
CO_2		+			
Unknowns	+	+	+	+	+

(Ohkawa et al., 1974b)

1* Silica gel chromatoplates + UV
2* Silica gel chromatoplates + sunlight

Solvent	Half-life (min)	
	Air	Nitrogen
Water	<5	<5
50% Aq. CH_3OH	20	60
Acetone	50	100
CH_3OH	100	>240
Benzene	>360	>360

(Ohkawa et al., 1974b)

Sumithion can isomerize to the S-methyl analog. The latter is a more potent cholinesterase inhibitor. The I_{50} varied from 2.34×10^{-5} (human blood serum) to 1.47×10^{-6} for fly head for sumithion and 9.04×10^{-8} to 5.26×10^{-9}, respectively, for S-methylsumithion. The hydrolysis rate was also determined at pH 10.99 and 25C: $K_{Hyd}(min^{-1})$ for sumithion = 3.54×10^{-4} and for S-methylsumithion = 2.96×10^{-1} (Kovacicova et al., 1973).

[Methoxy-^{14}C]sumithion was used to assay glutathione-dependent demethylation. Results of these studies indicated the presence of a glutathione-dependent enzymatic breakdown of sumithion in both Heliothis zea and Heliothis virescens but was lower in the latter (Plapp, 1973).

Sumithion has been used over a period of years for spruce budworm control. Some studies have shown that, although 70-85% of the initial dose deposited on trees was lost within two weeks after spraying, about 10% persists for at least 10 months. In view of these findings, a survey was made to check residue accumulations in areas of N.B., Canada, which had been treated for up to 5 consecutive years. No measurable amounts of sumithion or known breakdown products were found in any tested soils. Balsam fir foliage contained measurable year-end residues but no major breakdown products. Total residues appeared to have accumulated in foliage in relation to dosage and number of applications. Maximum residue observed was 1 ppm sumithion in Spring 1973 in fresh balsam fir foliage (Yule, 1973 and 1974; Yule and Duffy, 1972).

TERBUFOS (Counter) [O,O-Diethyl S-(tert-butylthiomethyl)phosphorodithioate]

In soil, terbufos exhibited a half-life of 4 to 5 days. The sulfoxide formed by oxidation reached a maximum at about 14 days. The sulfone appeared one week after the start of incubation of soil with terbufos. Some other compounds observed in less than 1% amounts were the thiolophosphate, thiolophosphate sulfoxide and the thiolophosphate sulfone (Laveglia and Dahm, 1975).

When rainbow trout were exposed *in vivo* to ^{14}C-labeled TFM, only the glucuronide conjugate was detected and was excreted primarily in bile. The highest glucuronide levels were found in liver (Lech, 1972 and 1973; Lech and Costrini, 1972). Coho salmon exposed to TFM excreted about 35 times more conjugated than free TFM in a 24-h study period (Hunn and Allen, 1975a).

When rainbow trout were exposed to 5 mg/l solution of TFM, the concentration of free TFM in gallbladder bile rose to 4.12 µg/ml after 2 h and the TFM glucuronide rose to 196 µg/ml. Plasma concentration of TFM was 2.73 µg/ml and of TFM glucuronide, 0.87 µg/l (Hunn and Allen, 1974). Within 24 h after transfer to fresh water, TFM disappearance from the fish plasma was complete. Concentration of free TFM in the bile remains stable during 12 h of withdrawal but the conjugated TFM concentrations rise. Then, between 12 and 24 h of withdrawal, the levels of both declined (Hunn and Allen, 1975b).

Chironomid larvae (*Chironomus tentans* Fabricius) were exposed to TFM. TFM accumulation from water was dependent on concentration and water hardness. The half-life varied from 3.6 to 15.3 h. Analyses of homogenates of exposed larvae indicated the presence of TFM glucuronide and sulfate as well as another conjugate which was hydrolyzed by 0.25N HCl and 0.25N NaOH but not by β-glucuronidase or by sulfatase. RTFM was also observed (Kawatski and Bittner, 1975). In other *in vitro* studies, all fish tissue homogenates (except trout brain) produced metabolites. Except bluegill liver, all homogenates produced TFM glucuronide (Kawatski and McDonald, 1974).

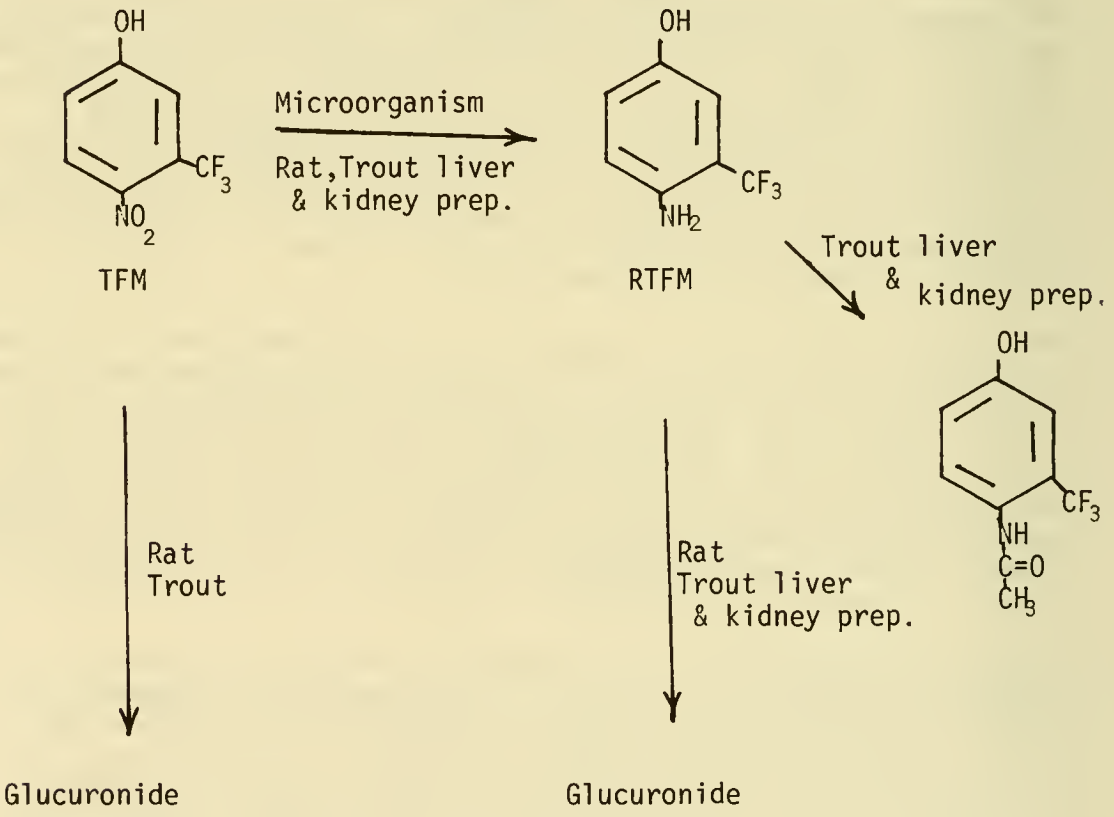
When rainbow trout were pre-exposed to salicylamide, TFM toxicity was increased; blood levels of TFM increased and TFM glucuronide decreased. The half-life of i.p. administered TFM was increased from 1.59 to 4.13 h (Lech, 1974). Administration of novobiocin produced similar effects (Lech et al., 1973). Administration of salicylamide to the sea lamprey did not increase TFM toxicity. *In vitro* studies indicated lower glucuronyl transferase than in rainbow trout and *in vivo* studies showed a higher free to conjugated TFM ratio in sea lamprey than in trout (Lech and Statham, 1975).

Studies were conducted with ^{14}C-TFM in a model stream community. Uptake and accumulation by several plant and animal components was basically an adsorption phenomenon consisting of a rapid initial uptake and then a reduced linear uptake phase. Elimination of TFM accumulations was rapid: in riffle dwelling species, an average half-life of 17.8 h; in pool-dwelling species, 140 h (Maki, 1974).

Scud (Gammarus pseudolimnaeus) concentrated TFM by a factor of about 58 in 7 days. After being transferred to TFM-free water, they eliminated half of the accumulated TFM in 3.5 days and 98% after 14 days (Sanders and Walsh, 1975).

When TFM was incubated with isolates from river muds, some fluoride was released. The organisms seemed to be Pseudomonas sp. Although cultures degraded TFM somewhat, isolates were unable to do so (Kempe, 1973).

TFM was exposed to sunlight and to UV lamp. TFM half-life in aqueous solution exposed to sunlgith was about 100 h; on silica gel plates, 71 h. A number of degradation products were observed. One chromatographed the same as RTFM. None were identified (Dawson, 1973).



THIABENDAZOLE (TBZ) [2-(4'-thiazolyl)benzimidazole]

^{14}C- and ^{35}S-labeled thiabendazole was orally administered to sheep. Within 96 h, sheep excreted 75% of the dose via urine and 14% in feces. Chromatography indicated that nearly all of the radioactivity was in the form of metabolites. Residues were distributed throughout most body tissues initially. ^{35}S was detectable in only a few tissues at less than 0.2 ppm by the 16th day and 0.06 ppm or less after 30 days. The metabolites were identified as the 5-hydroxy analog and the sulfate and glucuronide esters (Tocco et al., 1964).

In pepper plants, thiabendazole (TBZ) accumulated only in the leaves. Disappearance of TBZ from the leaves exceeded that of methyl-2-benzimidazolecarbamate (MBC) by three to fourfold. 2-Aminobenzimidazole, a degradation product of MBC, was also present at levels up to 2% of the parent compound (Ben-Aziz and Aharonson, 1974).

^{14}C-Thiabendazole was sprayed on sugar beet leaves. The plants were grown for 34 days under incandescent and fluorescent illumination. Analyses accounted for 97-98% of the radioactivity as unchanged thiabendazole. When treated beet leaves were exposed to sunlight for the equivalent of 14 8-hr days, only 78% of the radioactivity was present as unchanged thiabendazole. The remainder appeared to be photoproducts. In addition to benzimidazole-2-carboxamide, benzimidazole and polar and polymer products were formed. When photolysis was conducted on glass plates, benzimidazole-2-carboxamide and benzimidazole were observed (Jacob et al., 1975).

Thiabendazole was not metabolized by potatoes or cotton (Tisdale and Lord, 1973).

THIOLCARBAMATES

BENTHIOCARB [S-(4-Chlorobenzyl)-N,N-diethylthiolcarbamate]

BUTYLATE [S-Ethyl-N,N-(di-2-methylpropyl)thiolcarbamate]

CYCLOATE [S-Ethyl-N-ethyl-N-cyclohexylthiolcarbamate]

DIALLATE [S-(2,3-Dichloroallyl)-N,N-diisopropylthiolcarbamate]

EPTC [S-Ethyl-N,N-dipropylthiolcarbamate]

MOLINATE [S-Ethyl-N,N-hexamethylenethiolcarbamate]

PEBULATE [S-Propyl-N-butyl-N-ethylthiolcarbamate]

TRIALLATE [S-(2,3,3-Trichloroallyl-N,N-diisopropylthiolcarbamate]

VERNOLATE [S-Propyl-N,N-dipropylthiolcarbamate]

Thiocarbamates are detected in the liver of mice 20 min after i.p. treatment with EPTC, molinate, pebulate, and vernolate at 1 m mole/kg but not after administration of benthiocarb, butylate, or cycloate (Casida et al., 1974).

	DEGRADATION PRODUCTS*				
	Sulfoxide	Sulfone	CO_2	$EtSO_2$-CH_3	N-depropyl
EPTC (Eptam)	1,2,3,4	2	2	2	1
Pebulate (Tillam)	1,2,3	2	2		
Benthiocarb (Saturn)	1				
Butylate (Sutan)	1,5				
Cycloate (Ro-Neet)	1				
Molinate (Ordram)	1,2				
Vernolate (Vernam)	1,2				

*Formed with

1. Mouse liver microsome - NADPH system
2. Living mice
3. Photolysis
4. Corn
5. Soil

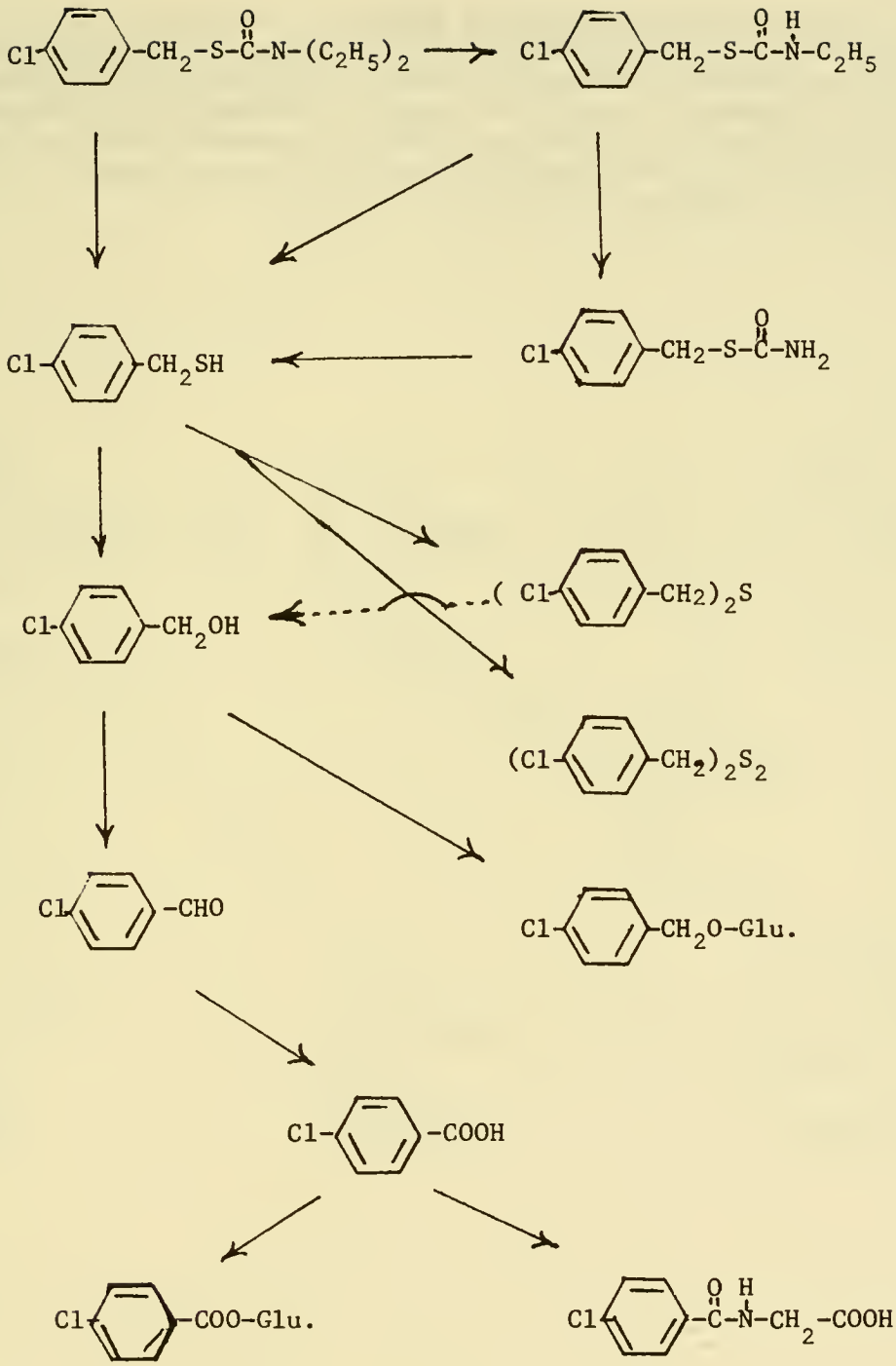
After oxidation to the sulfoxide, cleavage by the GSH-S-transferase system occurred with EPTC and pebulate. Mercaptans were released.

(Casida et al., 1974 and 1975)

BENTHIOCARB (Saturn) [4-Chlorobenzyl N,N-diethylthiolcarbamate]

The fate of ^{14}C-labeled benthiocarb was studied with white mice *in vivo* and *in vitro*. After oral administration, benthiocarb was rapidly translocated into organs. There was rapid urinary excretion of labeled material; slight excretion in feces; and only a little expired. The major metabolites identified were: 4-chlorohippuric acid, 4-chlorobenzoic acid, glucuronide of 4-chlorobenzoic acid, and 4-chlorobenzyl alcohol. In liver homogenates, the microsomal fraction exhibited highest activity and NADP accelerated the degradation. *In vitro* metabolites were identified as: N-desethylbenthiocarb, bis(4-chlorobenzyl)mono- and di-sulfides, and 4-chlorobenzoic acid (Ishikawa et al., 1973).

^{14}C-Benzyl methylene labeled benthiocarb was taken up through the roots and translocated into whole plants by rice, barnyard grass, wild amaranth, smartweed, and lambsquarters plants. It was translocated from a leaf into other leaves also. When applied to seeds, it was rapidly absorbed and accumulated mostly in the embryo. The plants degraded benthiocarb rapidly. No metabolites were identified (Nakamura et al., 1974).

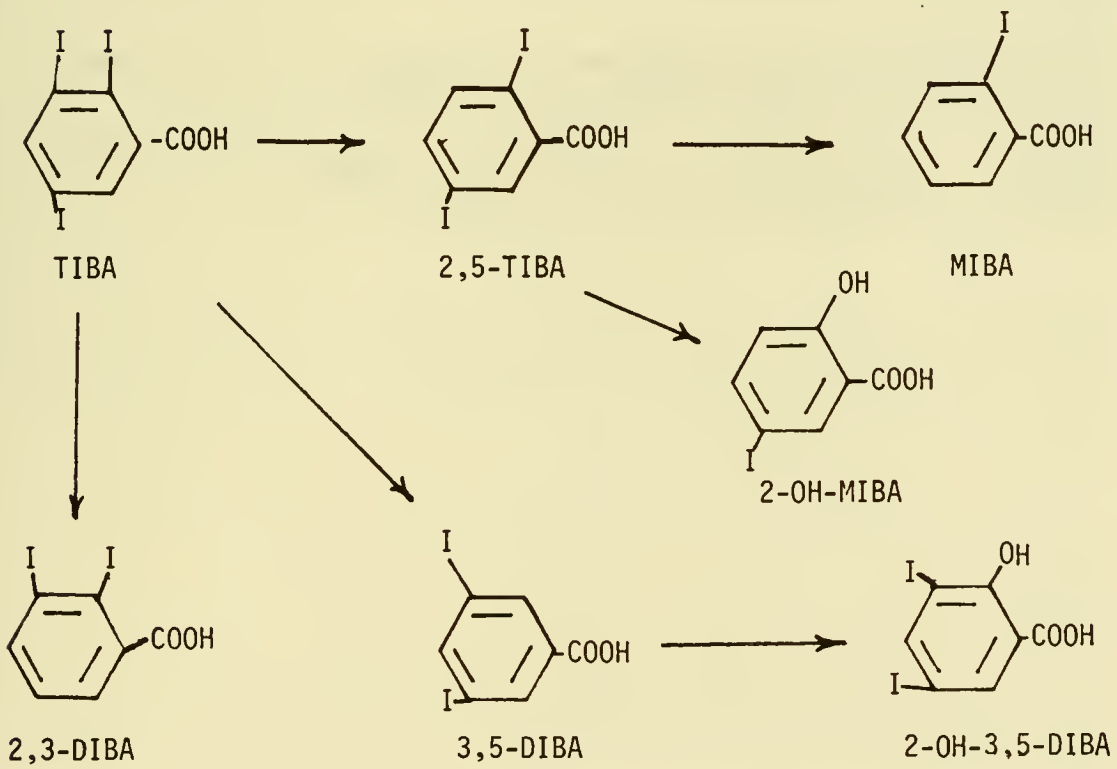

Cl-CH2-S-C(=O)-N-(C2H5)2
Cl-CH2-S-C(=O)-NH-C2H5
Cl-CH2SH
Cl-CH2-S-C(=O)-NH2
Cl-CH2OH
(Cl-CH2)2S
(Cl-CH2)2S2
Cl-CHO
Cl-CH2O-Glu.
Cl-COOH
Cl-COO-Glu.
Cl-C(=O)-NH-CH2-COOH

PEBULATE (Tillam) [S-Propyl butylethylthiocarbamate]

When tobacco seedlings (Nicotiana tobacum L. Kentucky 14) were incubated in nutrient solution with pebulate-^{14}C, in the roots the concentration of pebulate reached a maximum 1 day after treatment and decreased after 5 days of treatment. Very little pebulate accumulated in the shoots. The data indicated very rapid metabolism of pebulate after it was within the plant and incorporation into plant constituents. No metabolites were identified (Long et al., 1974b).

TIBA [2,3,5-Triiodobenzoic acid]

^{14}C-Carboxy-labeled TIBA was applied to soybeans at the beginning of the flowering stage. Residue analyses indicated the presence of conjugates in addition to 2,5-diiodobenzoic acid (2,5-DIBA) and 3,5-diiodobenzoic acid (3,5-DIBA). Seeda also contained conjugates of TIBA and 3,5-DIBA (Spitznagle, 1970).



FENTIN ACETATE [Triphenyltin acetate]

When fentin was added to soil, the amount that could be extracted with methanol decreased with time. Some fentin acetate was adsorbed to the soil. When [^{14}C]triphenyltin acetate was incubated with soil, 50% of the label evolved as $^{14}CO_2$ in 140 days. In sterile soil, only 0.47% evolved in 60 days. Several Aspergillus sp. were able to degrade fentin in liquid culture with release of $^{14}CO_2$. A Gram-negative bacteria was also able to metabolize fentin (Barnes et al., 1973).

Photochemical degradation of fentin at λ>250 produced diphenyltin, monophenyltin, and inorganic tin (Chapman and Price, 1972; Barnes et al., 1973).

In distilled water, fentin underwent rapid hydrolysis with formation of triphenyltin hydroxide (Barnes et al., 1973).

When rats were exposed p.o. to labeled tin, it was found that 50% of absorbed tin was excreted within 48 h (Hiles, 1974).

TIRPATE (Ent 27696) [2,4-Dimethyl-1,3-ditholane-2-carboxaldehyde O-(methylcarbamoyl)oxime]

The half-life of tirpate, when administered to young tobacco plants (Nicotiana tobacum) in hydroponic culture, was about 8-9 h. ^{14}C-Tirpate was readily taken up and translocated throughout the shoot but was not rapidly retranslocated to new young leaves. The initial metabolite was the sulfoxide. The sulfoxide nitrile was also found. Identification was by GC-MS. The conjugated materials were not identified. Some were released by sulfatase or glucosidase. One material released by sulfatase exhibited an R_f similar to that of the sulfoxide (Hill and Krieger, 1975).

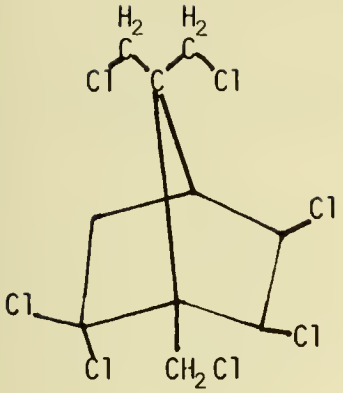
TOXAPHENE (Camphene chlorinated to 67-69% chlorine by weight and an average $C_{10}H_8Cl_{10}$ composition)

Technical toxaphene is a complex mixture consisting of at least 175 C_{10} polychloro compounds (Casida et al., 1974b; Khalifa et al., 1974). Male rats were orally administered ^{36}Cl-toxaphene. Within 9 days after dosing, the rats excreted over 50% of the administered dose. About 30% of this was in the urine and 70% in the feces. Although excretion in feces plateaued early, excretion in urine continued upward. Ionic chloride was excreted in large amount (Crowder and Dindal, 1974). Other studies confirm the rapid elimination of chloride with a half-time of 2 to 3 days. When ^{14}C-toxaphene was used, only low ^{14}C-levels were observed in tissues several days after administration. Similar observations were made after administration of toxicant B from toxaphene. Urinary and fecal metabolites included CO_2, dechlorinated materials and acidic compounds (Ohsawa et al., 1975). Most of the ^{36}Cl-toxaphene was metabolized before excretion. Less than 1% of the ^{36}Cl in the urine and less than 3% in feces appeared as unmetabolized toxaphene. Subfractions of toxaphene did not differ greatly (Casida et al., 1974a). The only urinary metabolite identified when ^{36}Cl-toxaphene was administered to rats was chloride ion. Tissue residues were very low when ^{14}C-toxaphene was used (Casida et al., 1974c).

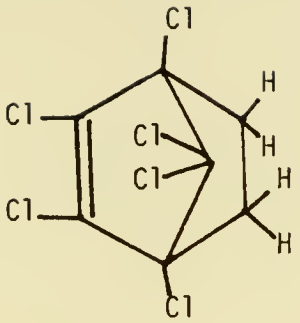
The closely related polychlorinated norbornenes were also studied. Hexachloronorbornene (II) was added to a culture of Clostridum butyricum. Analyses of the medium showed the presence of compounds III, IV and V. The pentachloronorbornene VI gave rise to compounds III and IV. Tetrachloronorbornene (III) gave rise to compound IV (Schuphan and Ballschmitter, 1972).

Toxaphene was applied to alfalfa, range grass and winter wheat. Analyses gave no evidence of toxaphene metabolism (Carlin et al., 1976).

Toxaphene reacted rapidly with hematin in aqueous media to produce dechlorinated toxaphene derivatives. About half of the carbon-chlorine bonds were broken. Toxicant fractions A and B underwent dechlorination, dehydrochlorination and a combination of both (Holmstead et al., 1975).



2,2,5-endo-6-exo-8,9,10-heptachloronorbornane
(Toxicant B)
I



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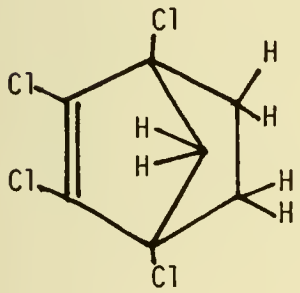

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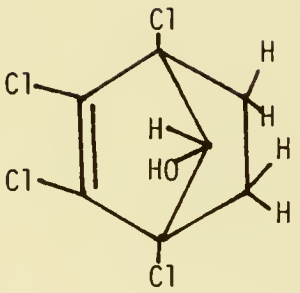
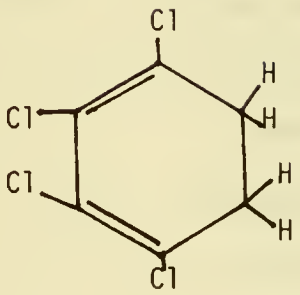
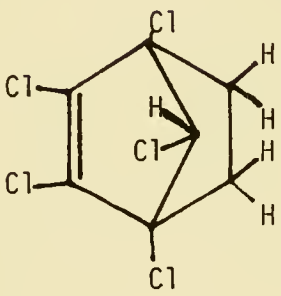


IV



V



VI

TRIAZINES

Ametryne

Atrazine

Bromosimazine

Cyanazine

Cyprazine

Fluorosimazine

Iodoatrazine

Iodopropazine

Iodosimazine

Metribuzin

Prometone

Prometryne

Propazine

Simazine

Simetryne

WL 9385

Photodecomposition of symmetrical triazines was found to be dependent on the nature of the substituent and the solvent. The rate constant k decreased in the order: I>Br>Cl>F; ethyl > propyl; and methanol > n-butanol. Decomposition exhibited zero-order rate constants. Materials tested included:

Ametryne
Atrazine
Bromosimazine
Fluorosimazine
Iodoatrazine
Iodopropazine
Iodosimazine
Prometryne
Propazine
Simazine
Simetryne

(Ruzo et al., 1973)

AMETRYNE [2-Ethylamino-4-isopropylamino-6-methylthio-s-triazine]

In nutrient solution-sugarcane system, ametryne degraded rapidly (90% in 30 days) to more polar materials. Dealkylated ametryne was the major product at 20 days. 2-Methylthio-4,6-diamino-s-triazine and ammeline formed in substantial amounts by 30 days. When ring-^{14}C-ametryne was applied to sugarcane, some $^{14}CO_2$ was formed. Ametryne was not metabolized to hydroxyametryne in sugarcane. In addition to CO_2, another volatile metabolite was observed but not identified. In soil, ametryne was degraded through N-dealkylation and 2-hydroxylation (Goswami, 1972; Goswami and Green, 1974).

ATRAZINE [2-Chloro-4-ethylamino-6-isopropylamino-s-triazine]

Canada thistle (Crisium arvense L.) plants were treated with labeled atrazine. Analyses indicated that over 90% of the material was present in the shoot where the atrazine was applied. TLC showed that most (64%) of the chloroform extracted material was unaltered atrazine; 6%, 2-amino-4-chloro-6-isopropylamino-s-triazine; 5%, 2-amino-4-chloro-6-ethylamino-s-triazine; and 25% unidentified. The water-soluble fraction contained 42% of the ^{14}C in the form of 2-ethylamino-4-hydroxy-6-isopropylamino-s-triazine; 2-amino-4-hydroxy-6-isopropylamino-s-triazine; and 2-amino-4-ethylamino-6-hydroxy-s-triazine. About 58% was unidentified (Burt, 1974).

The degradation of atrazine in submerged soil was measured. When ring-^{14}C-atrazine was used, some $^{14}CO_2$ evolved. A metabolite, hydroxyatrazine, was degraded more rapidly than atrazine by microorganisms. Products of microbial degradation of atrazine and hydroxyatrazine, an atrazine metabolite, included the product 2-amino-4-chloro-6-isopropylamino-s-triazine. These studies indicated that two pathways may be simultaneously operative: (1) biological, with dealkylation; and (2) chemical, with hydrolysis of the halide followed by microbial degradation (Goswami and Green, 1971).

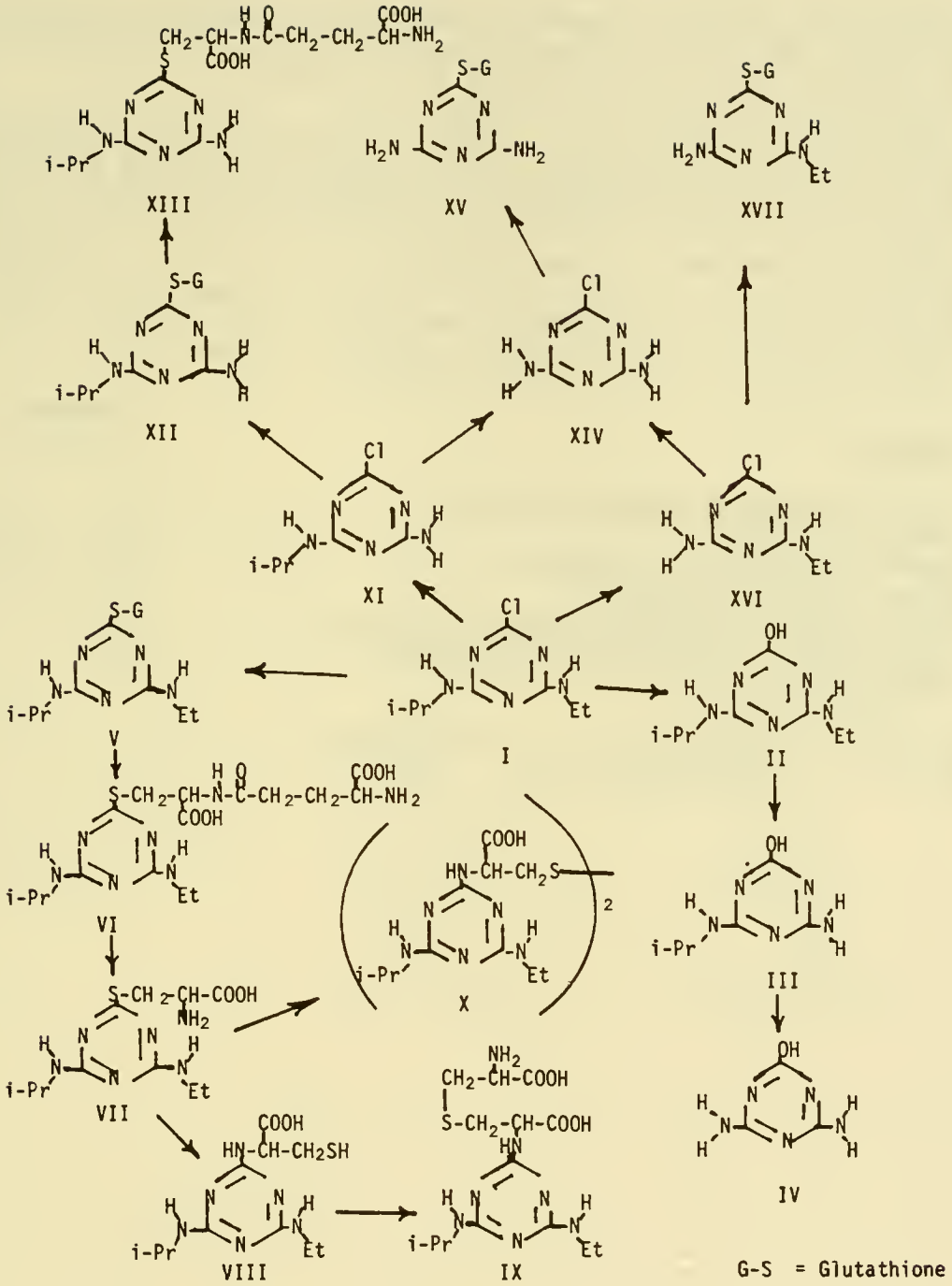
Atrazine was applied to shoots of fall panicum, Texas panicum (P. texanum Buckl.), witchgrass (P. capillare L.), giant foxtail (Setaria faberii Herrm.), yellow foxtail (S. glauca L. Beauv.), green foxtail (S. viridis L. Beauv.), giant green foxtail (S. viridis var. major Gaud Posp.), robust white foxtail (S. viridis var. robusta-alba Schreiber) and robust purple foxtail (S. viridis var. robusta-purpurea Schreiber). Analyses of plant extracts indicated the presence of hydroxyatrazine, monodealkylated hydroxyatrazine, and amino acid or peptide conjugates (Thompson, 1972).

Further studies were conducted to elucidate the metabolism of atrazine in sorghum (Sorghum vulgare Pers., N.D. 104). The major pathway was shown to involve the glutathion conjugate. Subsequent reactions removed the glycine moiety and then glutamic acid in that order. From the S-cysteine derivative, rearrangement produced the N-analog N-(4-ethylamino-6-isopropyl-s-triazinyl-2)cysteine (VIII). Addition of alanine produced the N-lanthionine derivative (IX). Two other compounds were observed in sorghum for the first time: 2-amino-4-hydroxy-6-isopropylamino-s-triazine (III) and 2,4-diamino-6-hydroxy-s-triazine (ammeline) (IV). The two mono-N-dealkylated products and ammeline (IV) were observed and one more new metabolite identified as N,N'-bis(4-ethylamino-6-isopropylamino-s-triazinyl)cystine (X) (Lamoureux et al., 1973; Shimabukuro et al., 1973b).

The degradation of atrazine by rat liver preparations was investigated. Cochromatography with standards was used for identification. These studies indicated that dealkylation was the predominant reaction and preceded conjugation with glutathione. Removal of the isopropyl group apparently occurred more easily than removal of the ethyl group. However, addition of NADPH to 10800xg supernatant and the microsomal fraction brought about removal of the second alkyl group to produce 2-chloro-4,6-diamino-s-triazine (XIV). When GSH as well as NADPH was added to the mixture, GSH-conjugates were formed with atrazine and the two mono-N-dealkylated derivatives but not with the diamino analog. However, when the desethyl or diamino derivatives were used as substrates, some GSH-conjugate of the diamino derivative was formed (Dauterman and Muecke, 1974).

In soil, atrazine hydrolysis proceeded with a second-order kinetics under non-sterile conditions and first-order kinetics under sterile conditions. Laboratory studies indicated that about half to two-thirds of applied atrazine was hydrolyzed non-biologically by soil samples (Agnihotri, 1971). After application to soil, atrazine degradation produced both mono-N-dealkyl derivatives and the hydroxy-atrazine (II) (Sirons et al., 1973). The major metabolite, hydroxy-atrazine, probably occurred through non-biological processes. Degradation was greater at pH 5.5 than at pH 7.5 (Best and Weber, 1974).

In the presence of nitrite and acid, atrazine reacts to form an N-nitrosamine (NNA). Maximum NNA formation occurs at pH 1. Thermal decomposition on GLC produces atrazine (Wolfe et al., 1975).


XIII
XV
XVII
XII
XIV
XI
XVI
I
II
V
VI
VII
X
III
VIII
IX
IV
G-S = Glutathione

CYANAZINE [2-Chloro-4-(1-cyano-1-methylethylamino)-6-ethylamino-s-triazine]

When applied to soil, cyanazine degraded primarily to des-isopropyl atrazine. The anticipated amide, the initial hydrolysis product of the nitrile, was also seen (Sirons et al., 1973).

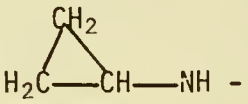
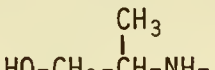
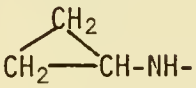
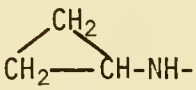
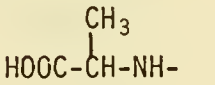
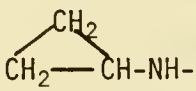
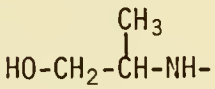
Using a system developed by R. Metcalf, ^{14}C-ring-labeled cyanazine was introduced into an aquatic model ecosystem. After 35 days, analyses of the components were conducted. In addition to unchanged cyanazine (I), N-deethylcyanazine (II), cyanazine amide (III), N-deethylcyanazine amide (IV), and three unknowns were found in the water. Radioactivity did not increase in the food chain of algae to mosquitoes to fish (a decrease from 1.3 to 0.05 ppm was observed), indicating that this compound does not concentrate through the food chain (Yu et al., 1975a).

Organism	Compound Found
Algae [Oedogonium cardiacum (Huss.)]	U_e
Crab (Uca Minax)	II, U_b, U_c, U_e
Daphnia (Daphnia magna Strauss.)	U_e
Elodea (Elodea canadensis)	U_c, U_e
Mosquito (Culex pipiens quinquefasciatus Say)	U_e
Fish (Gambusia affinis Baird and Girard)	U_e
Snail (not identified)	

U_a, U_b, U_c = Three unidentified metabolites
U_e = Unextractable ^{14}C

CYPRAZINE [2-Chloro-4-cyclopropylamino-6-isopropylamino-s-triazine]

^{14}C-Ring-labeled cyprazine (I) was administered by stomach tube to rats which were sacrificed after 3 days. Feces and urine were collected daily and pooled. About 98% of the radioactivity was excreted within 72 h. Very little (<0.1%) appeared as $^{14}CO_2$ and carcass and hide contained 7.5% of the ^{14}C. Paper, thin-layer and gas chromatography, derivatization and mass spectral analyses were used to separate and identify the metabolites. Compounds II to IV were identified and VI to IX were characterized (Larsen and Bakke, 1975).

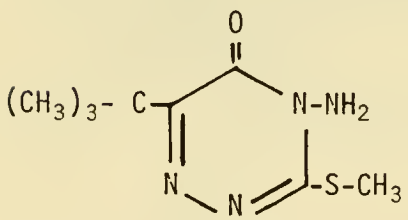
	R_1	R_2	R_3
I.	Cl-		i-C_3H_7NH-
II.	HO-	-NH_2	-NH_2
III.	Cl-	-NH_2	-NH_2
IV.	HO-	-NH_2	i-C_3H_7NH-
V.	Cl-	-NH_2	i-C_3H_7NH-
VI.	Cl-	-NH_2	
VII.	Cl-		-NH_2
VIII.	Cl-		
IX.	Cl-		

METRIBUZIN (BAY-94337, SENCOR) [4-Amino-6-tert-butyl-3-methylthio-as-triazin-5(4H)-one]

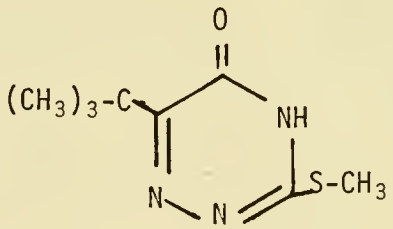
Metribuzin was added to nutrient solutions in which sugarcane cuttings of cultivar H50-7209 had been established. After one week, very little parent material was present in the nutrient solution or plant tissue. The deaminated product II, the hydrolysis product III, and the diketo product IV were detected in small amounts. Most of the metribuzin metabolites present in sugarcane were not identified (Hilton et al., 1974).

Preliminary greenhouse studies with metribuzin indicated that soybean cultivars exhibited different responses to exposure through nutrient solutions. The major portion of the metabolites was water-soluble. The "Semmes" and "Coker" cultivars produced primarily 6-tert-butyl-as-triazin-3,5-(2H,4H)-dione (IV). The major metabolite from "Bragg" roots and stems was an N-glucoside conjugate (Smith and Wilkinson, 1974).

The rates of degradation of metribuzin and two analogs were studied. All three herbicides exhibited pseudo first-order kinetics.



I



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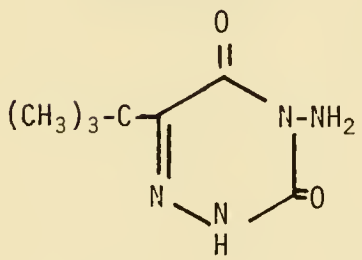
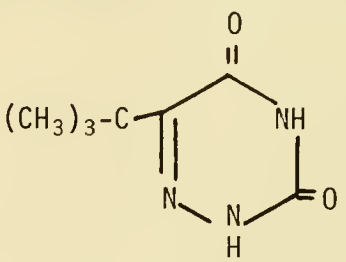


III



IV

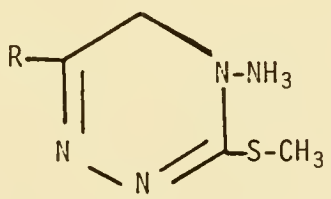
R	°C	k x 10^{-2} day^{-1}	$t_{1/2}$ (days)	ΔE_a (kcal/mole)
t-butyl	5	1.84	377	
	20	1.52	46	12.7
	35	4.40	16	
i-propyl	5	2.07	335	
	20	1.50	46	13.3
	35	4.56	15	
cyclohexyl	5	2.53	274	
	20	1.75	40	10.5
	35	4.21	17	

(Hyzak and Zimdahl, 1974)

A number of asymmetric triazin-5(4H)-ones were irradiated in carbon tetrachloride, methanol, benzene and water. Photolysis yielded essentially identical results in each case. The major product was identified as the 5-hydroxy triazine. Chromatography and mass spectra indicated minor products resulting from oxidation and desulfurization. Compounds tested were the 6-t-butyl, 6-isopropyl and 6-cyclohexyl analogs. Photolysis produced the following derivatives of these three compounds:

4-amino-3,5-diketo-
3-hydroxy-5-keto-
5-hydroxy-3-keto-
4-amino-5-keto-
4-azo-5-keto- and
5-hydroxy-as-1,2,4-triazine.

(Pape and Zabik, 1972)



PROMETONE [2,4-Bis(isopropylamino)-6-methoxy-s-triazine]

Rats were administered ^{14}C-ring-prometone. Urine was collected and analyzed and eight metabolites were identified or characterized by mass spectrometry or trimethylsilyl derivatives. The major metabolites identified were ammeline (31.5%) and 2,4-diamino-6-methoxy-s-triazine (10-14%). Other metabolites included: 2-amino-4-(1-carboxyethylamino)-6-methoxy-s-triazine; 2-amino-4-(1-carboxyethylamino)-6-hydroxy-s-triazine; 2-amino-4-methoxy-6-(2-propan-1-ol)amino-s-triazine; 2-amino-4-hydroxy-6-(2-propan-1-ol)amino-s-triazine; and 2-amino-4-hydroxy-6-isopropylamino-s-triazine (Bakke and Price, 1973).

PROPAZINE [2,4-Bis(ethylamino)-6-chloro-s-triazine]

Propazine was applied to six species and varieties of Setaria and three species of Panicum. In all cases, the hydroxypropazine was formed (Thompson, 1972).

SIMAZINE [2-Chloro-4,6-bis(ethylamino)-s-triazine]

Six species and varieties of Setaria and three Panicum species were exposed to ^{14}C-simazine. After absorption, simazine was metabolized to water-soluble compounds. Hydroxy derivatives were detected but peptide conjugates apparently were the only major metabolites formed by each species or variety. Hydroxysimazine was detected by chromatographic analysis (Thompson, 1972).

Simazine was added to a nutrient solution in the presence of citrus tree roots. Hydroxysimazine was not observed. Monodealkylation was followed by di-dealkylation of the simazine (Jordan and Jolliffe, 1973).

Simazine was applied three times at the rate of 2.8 kg/ha and five times at the rate of 5.6 kg/ha. After 12 and 18 months, respectively, the total simazine residue was less than 10% of the annual dose (Clay and Stott, 1973).

Black walnut (Juglans nigra L.) and yellowpoplar (Liriodendron tulipifera L.) 1-year-old seedlings were placed in nutrient solutions to which simazine had been added. Analyses after 3 days exposure showed the presence of simazine and monodealkylated simazine in leaves, stems and roots. The concentration of the monodealkylated simazine was greater in the yellowpoplar than in black walnut. Other degradation products found included 2-chloro-4,6-diamino-s-triazine, found in higher concentrations in yellowpoplar than black walnut, and hydroxysimazine which was found in yellowpoplar but not in black walnut. Two other compounds found in both plants were not identified (Wichman and Byrnes, 1975).

WL 9385 [2-Azido-4-*t*-butylamino-6-ethylamino-*s*-triazine]

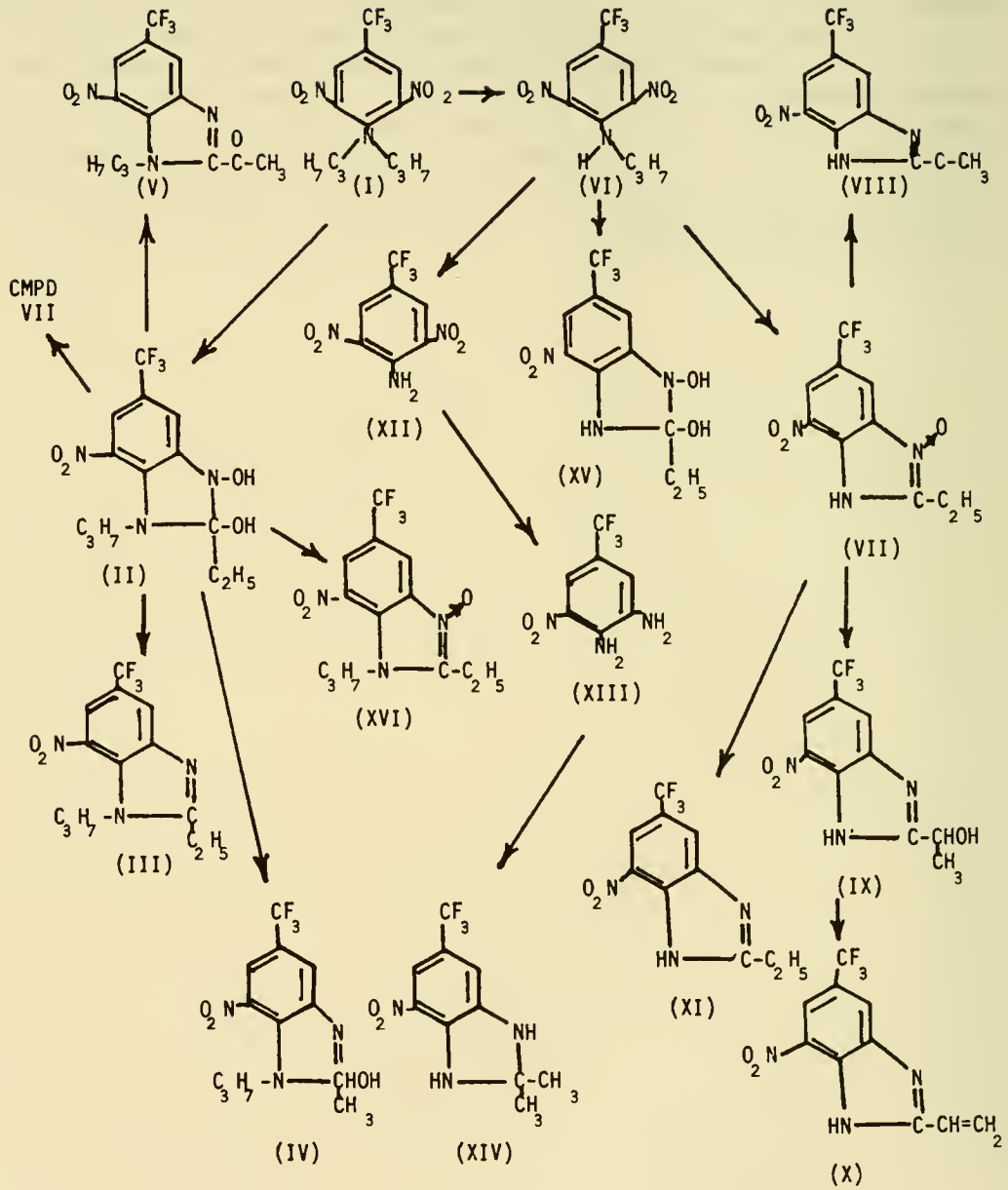
In the presence of moisture, in all soils tested, WL 9385 decomposed with formation of the corresponding 2-amino derivative. The rate constant is about 1.5 to 2.0 mg/g soil/day with some dependence on soil pH indicated. The reaction is not of a biological nature. In the solid state, this herbicide changes from white to yellow-brown when exposed to daylight or ultraviolet light. A half-life of about 240 h was observed. Decomposition followed first order kinetics (Barnsley and Gabbott, 1966).

TRIDEMORPH (Calixin) [2,6-Dimethyl N-tridecylmorpholine]

After the use of tridemorph on cereals, residues in grain were not detectable (less than 0.05 ppm) after 48 days. The half-life is about 5 or 6 days. Tridemorph is almost completely adsorbed from aqueous solution by soil at pH 6.7 and 7.5. Within 30 days, 80% of tridemorph added to Limburgerhof soil was degraded. The degradation was initiated by formation of tridemorph-N-oxide. Carbon dioxide and 2,6-dimethylmorpholine formed subsequently. Tridemorph half-life in loamy sand soil was about 8 weeks (Anon., BASF 1974).

TRIFLURALIN [2,6-Dinitro-N,N-dipropyl-α,α,α-trifluoro-p-toluidine]

Trifluralin readily decomposed when aqueous solutions were exposed to sunlight. Under acidic conditions, the main product was 2-amino-6-nitro-α,α,α-trifluoro-p-toluidine (XIII). Under alkaline pH, 2-ethyl-7-nitro-5-trifluoromethylbenzimidazole (XI) was the main compound (80%) of the photolytic products. Two other compounds, which were present under all conditions were identified as 2,3-dihydroxy-2-ethyl-7-nitro-1-propyl-5-trifluoromethylbenzimidazole (II) and 2-ethyl-7-nitro-5-trifluoromethylbenzimidazole-3-oxide (VII). These latter two degraded readily by heat or irradiation. Further irradiation of compounds II and VII helped elucidate the photolytic degradation of trifluralin. About 25 compounds were detected; 14 were identified (Crosby and Leitis, 1973; Leitis and Crosby, 1974). Photolysis of trifluralin vapor produced II, III, VI, VII, XI, XII, XV and XVI (Soderquist et al., 1975).


CMPD VII
(V)
(I)
(VI)
(VIII)
(XII)
(XV)
(II)
(VII)
(XVI)
(XIII)
(III)
(IX)
(XI)
(IV)
(XIV)
(X)

TRIFORINE (Cela W524) [N,N'-Bis(1-formamido-2,2,2-trichloroethyl) piperazine]

A soil drench application of triforine was applied to barley plants in pots. The biological half-life was 9 to 10 days when applied at the rate of 5 mg/plant but rose to 25 to 26 days with 50 mg. In the shoots four metabolites were observed. One was identified as piperazine and another was tentatively identified as N-monoglyoxyl-piperazine (Bruchhausen and Stiasni, 1973; Fuchs et al., 1972).

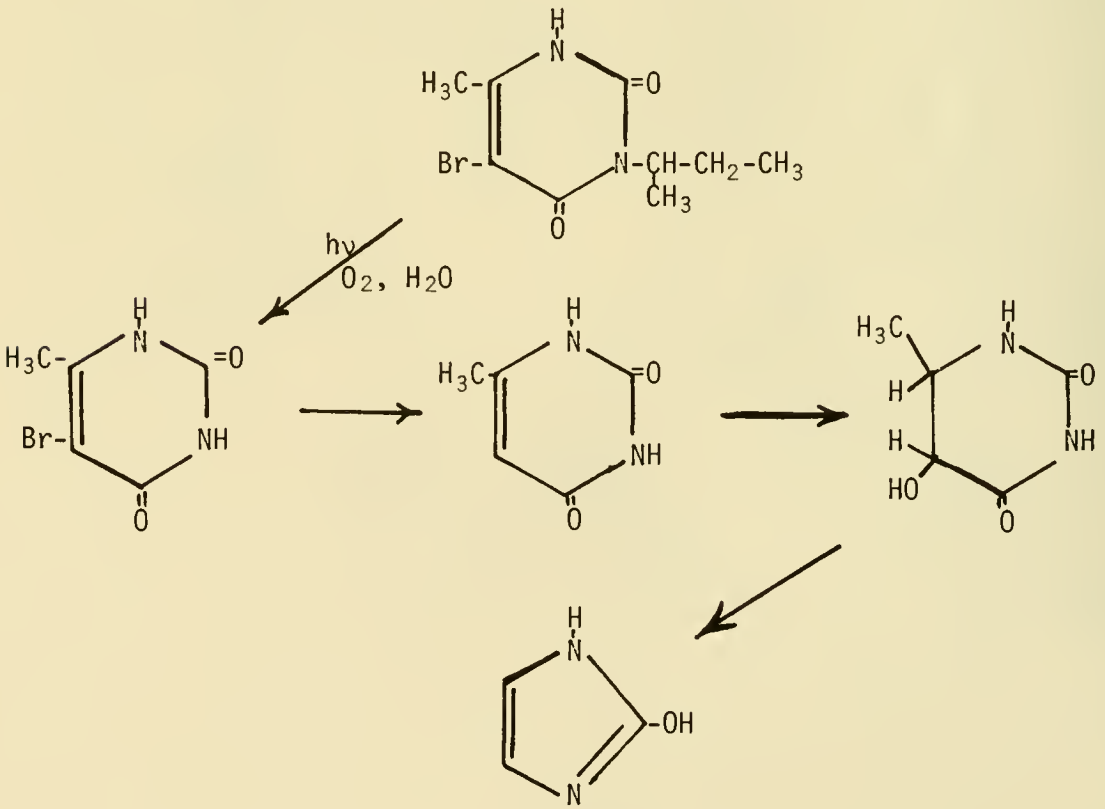
URACILS

BROMACIL [5-Bromo-3-sec-butyl-6-methyluracil]

Aqueous solutions of bromacil were irradiated 6 days in the laboratory and for 4 summer months outdoors. In sunlight, a low yield of 5-bromo-6-methyluracil was obtained. Laboratory photolysis yielded this compound also. Other studies indicated the formation of four volatile substances after irradiation of a 10 ppm aqueous bromacil solution for 6 days. The major product (37%) was 6-methyluracil. The mass spectrum of a second compound was identical to that of 5-bromo-6-methyluracil. The mass spectra of the two other products were consistent with the addition of a water molecule to 6-methyluracil to form 5-hydroxy-6-methyl-5,6-dihydrouracil and photooxidation of the latter to form 2-hydroxyimidazole (Moilanen and Crosby, 1974).

Of 55 fungal and 73 bacterial cultures isolated from soil, only four fungi were capable of degrading bromacil. One culture was identified as Penicillium paraherquei Abe. (Torgeson and Mee, 1967).

Bromacil was incubated in various soils to determine its persistence. The half-life in flooded soil was 155 days; in flooded soil plus bean straw, 198 days (Wolf and Martin, 1974).



UREAS

Buturon

Chlorbromuron

Dimilin

Fluometuron

Linuron

Methbenzthiazuron

Monolinuron

Monuron

BUTURON [N-(4-Chlorophenyl)-N'-methyl-N'-(1-methylprop-2-ynyl)urea]

After application of buturon to leaves, no p-chloroaniline was observed. Other metabolites less polar than buturon were not demonstrated and the unextractable material was not characterized (Haque et al., 1974).

Ultraviolet irradiation of buturon in methanol, methanol/water and benzene produced dechlorobuturon and N-phenyl-N'-methylurea. Highly polymerized material was also present (Kotzias et al., 1973).

When buturon was incubated with Rhizopus japonicus, the methylpropynyl group was lost to form the 1-(4-chlorophenyl)-3-methylurea (Wallnofer et al., 1973a).

CHLORBROMURON [3-(3-Chloro-4-bromophenyl)-1-methoxy-1-methylurea]

After application of chlorbromuron to corn and pigweed, the desmethyl and phenylurea metabolites were present in greater amount than the desmethoxy and unidentified metabolites (Palm, 1971).

DIMILIN (Diflubenzuron, TH 60-40, OMS 1804, PH-6040)
[1-(4-Chlorophenyl)-3-(2,6-difluorobenzoyl)urea]

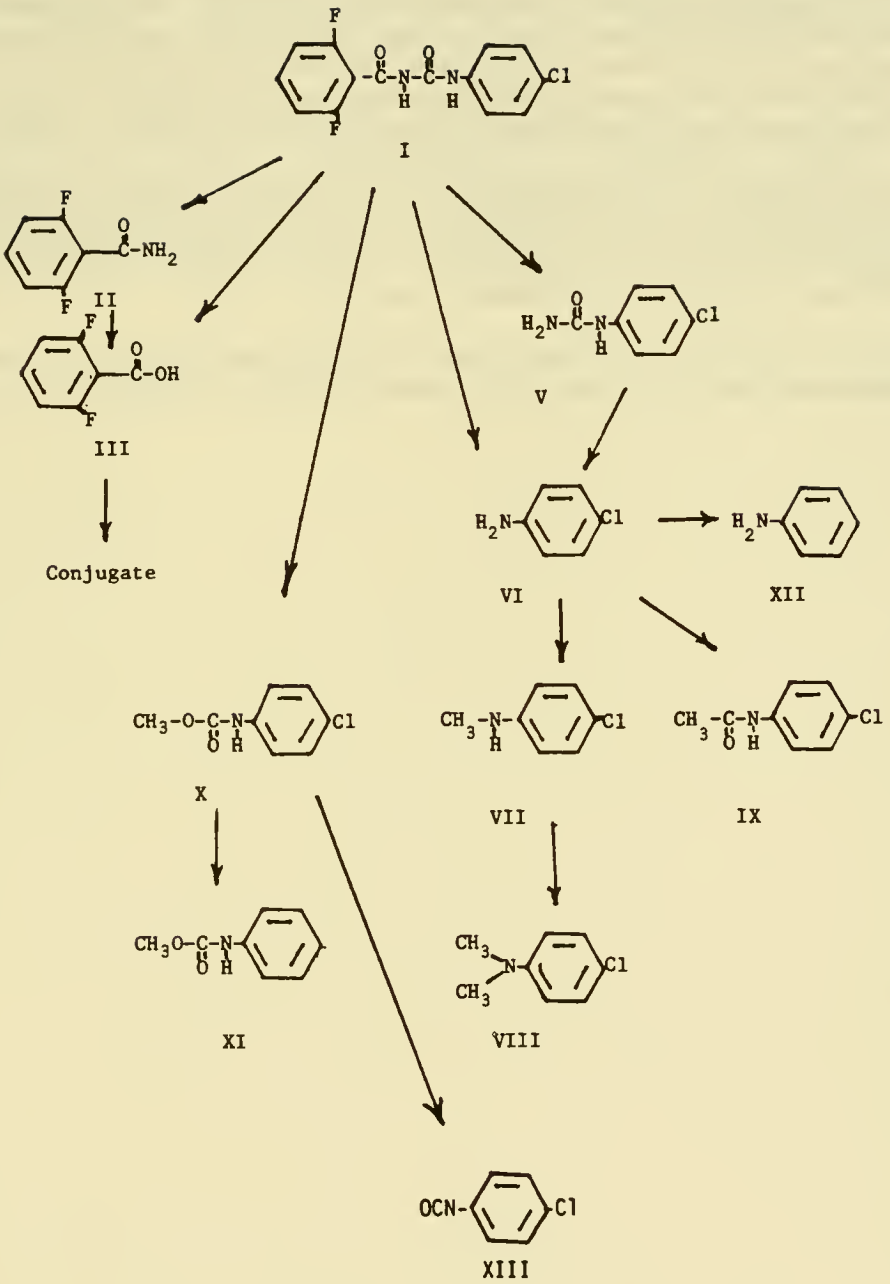
Dimilin was incubated for 1 h with sheep microsomes. Slightly more than 99% of the parent compound was unchanged. Metabolites identified included compounds II, III, V, VI, VII and IX. When the salt marsh caterpillar, Estigmene acrea, was fed ^{14}C-labeled dimilin, 99+% of the parent material was recovered unchanged in both feces and body homogenates. Incubation of dimilin with the soil microorganism (Pseudomonas putida) produced no evidence of degradation. When incubated with soil, dimilin degradation was very low. Traces of products cochromatographed with 4-chlorophenylurea (V) and 4-chloroaniline (VI) (Metcalf et al., 1975a).

A model ecosystem was constructed and contained water, alga (Oedogonium cardiacum), snail (Physa sp.), mosquito larva (Culex pipiens quinquefasciatus), fish (Gambusia affinis), plankton (Daphnia magna), sorghum (Sorghum vulgare) and the salt marsh caterpillar (Estigmene acrea). Fourth instar caterpillars fed on dimilin treated sorghum leaves and dispersed the material into the aquatic portion of the system. In addition to those metabolites identified, there were nine unidentified compounds. No azobenzenes were observed (Metcalf et al., 1975a).

Compound Found	SYSTEM						
	Sheep Microsomes	Soil	Found in Ecosystem				
			Water	Alga	Snail	Mosquito	Fish
I	+		+	+	+	+	+
II	+		+				+
III	+		+			+	+
IV							
V	+	+	+	+		+	+
VI	+	+	+	+			+
VII	+						
VIII			+	+			
IX	+		+				+

(Metcalf et al., 1975a)

Dimilin was irradiated in methanol for 9 h at 254 nm. Some colored material was produced. TLC analysis showed the presence of 2-difluorobenzamide (II), methyl phenylcarbamate (XI), and methyl 4-chlorophenylcarbamate (X). In aqueous dioxane, a dark brown solution formed after 4-h irradiation. In addition to compound II, 4-chloroaniline (VI) and aniline (XII) were found. Cochromatography, IR and mass spectrometry were used in the identification of these compounds (Metcalf et al., 1975a). In other studies, irradiation in methanol produced compounds II, X, XI and XIII (Ruzo et al., 1974c).


I
II
III
Conjugate
V
VI
XII
X
VII
IX
XI
VIII
XIII

FLUOMETURON [1,1-Dimethyl-3-(m-trifluoromethylphenyl)urea]

The metabolism of fluometuron in corn (Zea mays var. Dixie 18) and wheat (Triticum aestivum var. Wakeland) was studied. In both species, metabolism involved two-step demethylation and then hydrolysis to the aniline. Ring hydroxylation was indicated. Further metabolism of the aniline derivative into numerous metabolites occurred with an indication of formation of the hydroxyaniline derivative (Neptune, 1970).

Sour orange (Citrus auranthium L.) or sweet lime (C. limetioides Tanaka) were exposed to ^{14}C-fluometuron. After 4 days, analyses showed the presence of desmethylfluometuron, 3-trifluoromethylphenyl urea, and 3-trifluoromethylaniline (Menashe and Goren, 1973).

After incubation of fluometuron with Rhizopus japonicus, desmethyl-fluometuron was observed (Wallnofer et al., 1973c).

When linuron was applied to corn or pigweed, the desmethyl and phenylurea metabolites were present in larger amounts than the desmethoxy and unidentified metabolites (Palm, 1971).

Data derived from studies of adsorption of linuron on humic acid, saturated with Fe^{3+}, Al^{3+}, Cu^{2+}, Zn^{2+}, Ni^{2+}, Ca^{2+}, H^{+} was found to be consistent with a physical type of adsorption. Infrared spectroscopy showed no coordination of linuron to the cations on humic acid (Khan and Mazurkewich, 1974).

METHBENZTHIAZURON (MBT, Tribunil) [1-(2-Benzothiazolyl)-1,3-dimethylurea]

MBT was applied to hydroponic cultures of Triticum vulgare and allium cepa. Analyses indicated that MBT was metabolized to the 1-hydroxymethyl analog initially and subsequent loss of the hydroxymethyl group (Pont et al., 1974). In other studies with sensitive and resistant plants, MBT passed rapidly from roots towards leaves where active metabolism seemed to occur. Metabolites identified include the 3-hydroxymethyl analog and its glucoside and the 3-demethylated analog. The 1-OH-methyl analog was suggested as another metabolite. One other metabolite was characterized and the structure most compatible with the information was that of a 1,2-bis(MBT) ethane (Collet and Pont, 1974).

MONOLINURON (Aresin) [3-(4-chlorophenyl)-1-methoxy-1-methylurea]

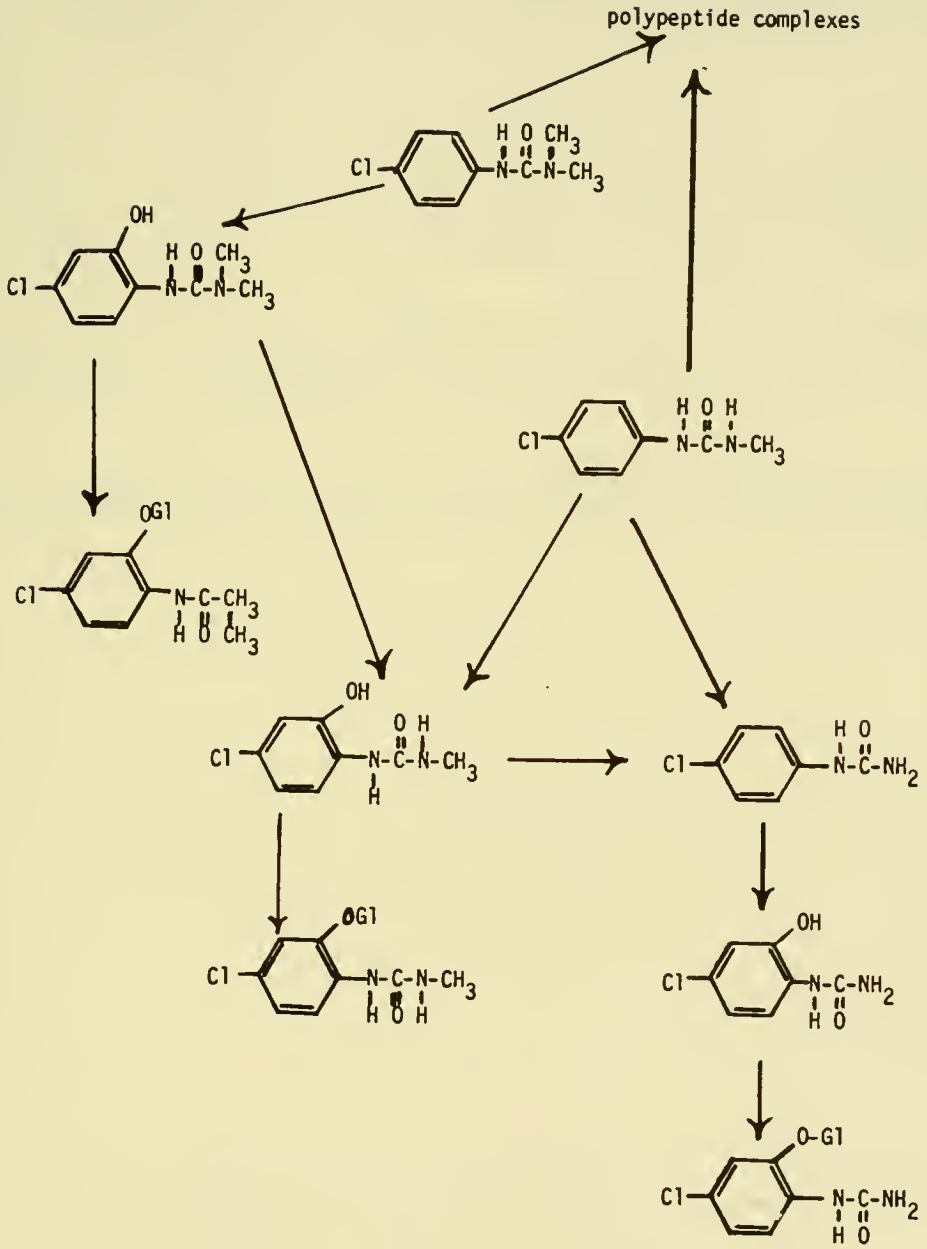
Monolinuron was sprayed on ground waste. After 3 weeks of composting, the material was extracted and analyzed. Most of the residue was unreacted monolinuron. About 0.4% was demethyl monolinuron (Muller and Korte, 1975).

Incubation of monolinuron with Rhizopus japonicus produced desmethyl monolinuron (Wallnofer et al., 1973c).

MONURON [3-(4-Chlorophenyl)-1,1-dimethylurea]

Young leaves were cut from 2-week-old plants of bean (Phaseolus vulgaris L. var. Black Valentine) and corn (Zea mays L. var. Batam Cross) and exposed to carbonyl-^{14}C-labeled monuron in water. After monuron uptake by the leaves, analyses showed the presence in both plants of: 1-(4-chlorophenyl)-3-methylurea; 4-chlorophenylurea; an unidentified conjugate: and 1,1-dimethyl-3-(2-hydroxy-4-chlorophenyl) urea. Although the conjugates were not identified, these studies indicated the presence of a monuron-polypeptide larger than 5000 and three glucose conjugates. The latter were identified as mono-β-D-glucose conjugates of 2-hydroxy-4-chlorophenylurea; 1-(2-hydroxy-4-chlorophenyl)-3-methylurea; and 1,1-dimethyl-3-(2-hydroxy-4-chlorophenyl) urea (Lee and Fang, 1973; Lee et al., 1973).

Incubation of monuron with Rhizopus japonicus produced 1-(4-chlorophenyl)-3-methylurea (Wallnofer et al., 1973c).


polypeptide complexes
Cl
OH
OGl
O-Gl
NH2

WARFARIN [3-(α-Acetonylbenzyl)-4-hydroxycoumarin]

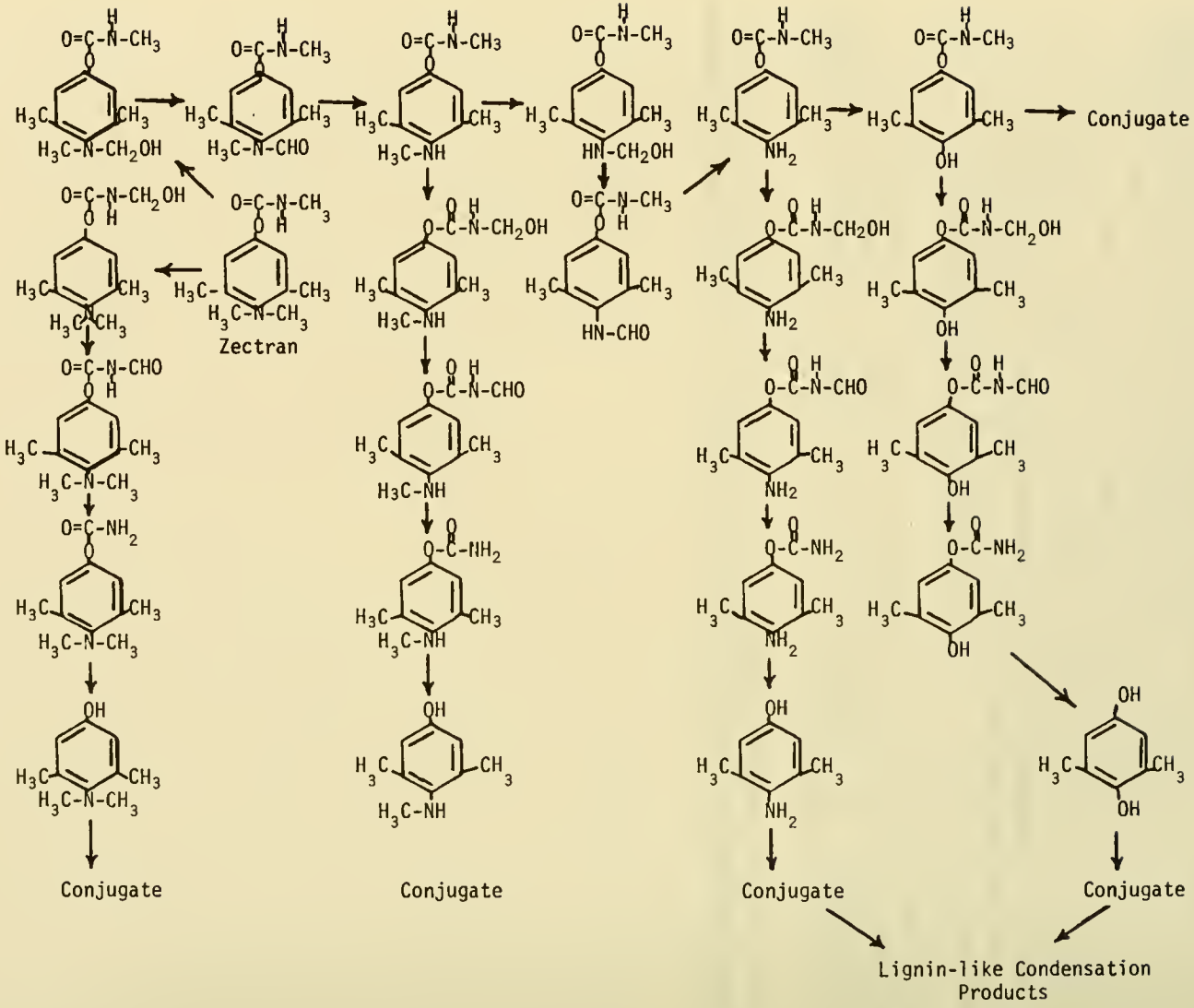
In vivo studies with Wistar-derived warfarin susceptible strain (TAS) rats indicated that warfarin metabolism was the limiting step with regard to toxicity. Using *in vitro* studies, 6-, 7-, 8-, and 4'-hydroxy-warfarins were separated by means of thin-layer and paper chromatography and identified by mass spectrometry and UV spectra (Townsend et al., 1975).

ZECTRAN (Mexacarbate) [4-Dimethylamino-3,5-Xylyl N-methylcarbamate]

Bacteria, mold and fungi were screened for their ability to degrade zectran. All organisms tested were able to metabolize this pesticide. A bacteria (HF-3) and a fungus (Trichoderma viride) were further investigated. The major T. viride metabolite was 4-dimethylamino-3,5-xylenol (DMAX). Methylamino zectran (MAZ) and methylformamido zectran (MFZ) were formed by HF-3. Other degradation products identified by TLC were amino zectran (AZ) and formamido zectran (FZ). Addition of cofactors and carbon sources altered the metabolic patterns. Decarbamylation predominated in the presence of ATP, $NADP^+$ and NADPH; demethylation, in the presence of NAD^+ and FAD. Thus, T. viride could be induced to degrade zectran via demethylation of the N-dimethyl group whereas metabolism of zectran normally proceeds principally via decarbamylation in this organism (Benezet and Matsumura, 1974).

Irradiation of zectran in cyclohexane or ethanol by a high-pressure xenon-mercury lamp produced a number of compounds. Three major products were produced: MAZ, DMAX and 2,6-dimethyl-4-hydroxy-N-methyl benzamide (HMB) (Silk and Unger, 1973).

A scheme was proposed by Meikle (1973) to explain the gradual flow of zectran metabolites to water-soluble compounds and other plant substances.


Zectran
Conjugate
Conjugate
Conjugate
Conjugate
Conjugate
Lignin-like Condensation Products

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Appendix I

Effect of Temperature on Carbamate Insecticides

Compound	C	Molarity of NaOH	K_1 (Min^{-1})	K_2 $l\text{-}min^{-1}md^{-1}$	Q_{10}	Heat of Activation (kCal mol^{-1})
Carbaryl	3	0.009	2.18×10^{-2}	2.42×10		
	13		6.22×10^{-2}	6.91×10		
	23		1.84×10^{-1}	2.04×10^{2}	2.9	16.9
	33		4.84×10^{-1}	5.37×10^{2}		
Baygon	5	0.01	7.30×10^{-2}	7.37		
	10		1.12×10^{-1}	1.12×10		
	20		3.04×10^{-1}	3.04×10	2.49	15.8
	30		7.59×10^{-1}	7.59×10		
	40		1.16	1.16×10^{2}		
Pyrolan	5	0.1	2.00×10^{-3}	2.00×10^{-2}		
	10		3.20×10^{-3}	3.20×10^{-2}		
	20		7.00×10^{-3}	7.00×10^{-1}	222	13.7
	30		1.49×10^{-2}	1.49×10^{-1}		
	40		2.99×10^{-2}	2.99×10^{-1}		
Dimetilan	10	0.5	1.65×10^{-4}	3.40×10^{-3}		
	20		1.65×10^{-3}	3.40×10^{-3}	1.91	14.0
	30		3.25×10^{-3}	6.50×10^{-3}		
	40		7.14×10^{-3}	1.43×10		

(Aly and El-Dib, 1971)

Appendix II

In Vivo Inhibition of Liver Arylamidase

Compound	Dose (mg/kg i.p.)	Percent Inhibition Male	Female
Parathion	0.05	9.2 ± 7.58	22.6 ± 1.69
	0.2	19.0 ± 6.57	33.1 ± 7.42
	0.4	64.9 ± 4.40	81.5 ± 2.01
	0.8	97.9 ± 0.87	98.9 ± 0.52
Paraoxon	0.05		39.3 ± 7.05
	0.2	26.5 ± 8.51	67.8 ± 3.78
	0.4	67.7 ± 4.07	86.0 ± 1.90
	0.8	97.0 ± 0.69	98.8 ± 0.48
EPN	0.4	20.3 ± 2.27	22.5 ± 6.70
	1.0	53.2 ± 2.77	57.3 ± 5.95
	4.0	95.9 ± 1.12	98.1 ± 0.32
Folex	1	41.8 ± 7.61	50.2 ± 3.82
	5	83.3 ± 1.04	88.6 ± 0.96
	10	96.0 ± 0.89	96.0 ± 0.45
	20	98.5 ± 0.22	98.2 ± 0.50
Sumithion	0.5	28.0 ± 0.78	36.8 ± 3.55
	2	42.8 ± 2.98	50.6 ± 5.95
	10	59.5 ± 5.17	66.0 ± 2.75
	50	61.7 ± 1.59	68.8 ± 2.62
	100	67.7 ± 1.26	81.4 ± 1.75
Malathion	2	22.4 ± 2.71	0 ± 7.72
	10	50.1 ± 4.35	49.4 ± 5.65
	50	70.2 ± 1.71	73.6 ± 7.44
	100	84.4 ± 1.78	84.2 ± 1.29
TOTP	1	36.8 ± 5.09	31.0 ± 2.20
	5	83.5 ± 4.18	57.5 ± 1.90
	10	98.3 ± 0.39	77.1 ± 1.42
	20	100.0 ± 0.00	98.0 ± 0.43

(Satch, 1973)

Appendix III

Effect of Substitution in Parathion Analogs

Substituent	Hydrolysis (k_{OH} min^{-1}) (pH 8.5)	Inhibition (k_i $M^{-1}min^{-1}$) Fly ACh E	Bovine ACh E
H	6.14×10^{-4}	1.14×10^{6}	2.44×10^{5}
3-F	1.34×10^{-3}	2.88×10^{6}	2.32×10^{5}
3-Cl	1.69×10^{-3}	7.17×10^{6}	1.90×10^{5}
3-Br	1.74×10^{-3}	2.02×10^{7}	2.53×10^{5}
3-I	1.49×10^{-3}	4.51×10^{7}	6.61×10^{5}
3-CF_3	3.12×10^{-3}	2.69×10^{7}	5.94×10^{4}
3-CH_3	4.15×10^{-4}	1.26×10^{6}	3.79×10^{4}
2-F	2.27×10^{-3}	1.21×10^{7}	3.36×10^{5}
2-Cl	2.67×10^{-3}	7.82×10^{6}	1.87×10^{5}
2-CF_3	3.84×10^{-3}	3.56×10^{5}	1.27×10^{3}
2-CH_3	4.61×10^{-4}	1.78×10^{5}	1.43×10^{3}
2,5-Cl_2	3.42×10^{-3}	3.22×10^{6}	7.94×10^{3}
3,5-Cl_2	2.26×10^{-3}	8.59×10^{6}	8.34×10^{4}
2,5-$(CH_3)_2$	2.56×10^{-4}	1.48×10^{5}	2.8×10^{2}
3,5-$(CH_3)_2$	2.20×10^{-4}	1.49×10^{5}	6.2×10^{2}

(Metcalf and Metcalf, 1973)

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