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EXECUTIVE SUMMARY

Numerous papers have been published on the effects of various pesticides on non-target aquatic organisms at concentrations simulating the contamination of surface waters at field application rates. However, such chemicals are known to undergo biotic and abiotic transformations in the aquatic environment following their use but few studies include the effects of these transformation products on aquatic biota. This paper reviews the scientific literature for information on the effects of pesticide transformation products on non-target aquatic organisms and highlights areas of concern where future research is required. Results indicate that pesticide transformation products can be less, more or similar in toxicity when compared to the parent chemical. As a general trend, the toxicity of these products is reduced for most pesticides, particularly herbicides, although this is somewhat dependent upon the species of organism tested i.e., plant or animal. Several metabolites of the organophosphate and carbamate insecticides e.g., fenitrothion and aminocarb, were found to be similar to or more toxic than the parent compound. Factors which must be considered when evaluating the hazards of transformation products of pesticides in aquatic ecosystems include a) the rate at which the compounds appear and disappear b) concentrations of residues in the field c) time of exposure for aquatic biota and d) compartmentalization of transformation products in the ecosystem. Areas of concern where future research is indicated include the required toxicity testing of transformation products for new chemicals prior to registration and

the interactive effects of the parent compound, its transformation products and any formulation adjuvants on the toxic response of non-target organisms.

PERSPECTIVE ADMINISTRATIVE

De nombreux articles ont été publiés sur les effets exercés par divers pesticides sur les organismes aquatiques non visés, à des concentrations qui simulent la contamination des eaux de surface, aux débits d'application employés sur le terrain. Toutefois, bien qu'il soit reconnu qu'après leur application ces produits chimiques subissent des transformations biotiques et abiotiques dans l'environnement aquatique, peu d'études rapportent les effets de ces produits de transformation sur le biote aquatique. Le présent article passe en revue la documentation scientifique afin 1° d'assembler des renseignements sur les effets des produits de transformation des pesticides sur les organismes aquatiques non visés et 2° de cerner les questions sur lesquelles devra porter la recherche dans l'avenir. Les résultats indiquent que les produits de transformation des pesticides peuvent être moins toxiques, plus toxiques ou aussi toxiques que le produit chimique d'origine. De manière générale, la toxicité de ces produits est réduite dans le cas de la plupart des pesticides, surtout en ce qui concerne les herbicides, bien qu'elle puisse varier quelque peu selon que l'organisme testé est végétal ou animal. Plusieurs métabolites des insecticides organophosphorés et des carbamates, p. ex., le fénitrothion et l'aminocarbe, sont aussi toxiques ou plus toxiques que le composé d'origine. Les facteurs dont il faut tenir compte lorsqu'on évalue les dangers des produits de transformation des pesticides dans les écosystèmes aquatiques comprennent : a) la vitesse d'apparition et de disparition des composés, b) les concentrations de résidus sur le terrain, c) la durée d'exposition du biote aquatique et d) la compartimentation des

produits de transformation dans l'écosystème. Les questions qui devront faire l'objet d'études ultérieures comprennent l'évaluation toxicologique nécessaire des produits de transformation des nouvelles substances chimiques avant leur homologation et les effets interactifs du composé d'origine, de ses produits de transformation et de tout adjuvant de la formulation sur la réponse toxique des organismes non visés.

ABSTRACT

A review of the scientific literature for information on the effects of transformation and/or breakdown products of pesticides on aquatic biota indicates that such compounds can be less, more or similar in toxicity when compared to the parent chemical. In general, the toxicity of transformation products is reduced for most pesticides, particularly herbicides, but this is dependent upon the species of organism tested i.e., plant or animal. In addition, several metabolites of the organophosphate and carbamate insecticides were similar to or more toxic than the parent compound especially to fish. Factors which must be considered when evaluating the hazards of transformation products of pesticides in aquatic ecosystems include a) the rate at which the compounds appear and disappear b) concentrations of residues in the field c) time of exposure for aquatic biota and d) compartmentalization of transformation products in the ecosystem. Areas of concern where future research is indicated include the required toxicity testing of transformation products for new chemicals prior to registration and the interactive effects of the parent compound, its transformation products and any formulation adjuvants on the toxic response of non-target organisms.

RÉSUMÉ

Une revue de la documentation scientifique effectuée dans le but d'assembler des renseignements sur les effets des produits de transformation ou de dégradation des pesticides sur le biote aquatique indique que ces composés peuvent être moins toxiques, plus toxiques ou aussi toxiques que le produit chimique d'origine. De manière générale, la toxicité des produits de transformation est réduite dans le cas de la plupart des pesticides, surtout en ce qui concerne les herbicides, mais elle varie selon que l'organisme testé est végétal ou animal. De plus, plusieurs métabolites des insecticides organophosphorés et des carbamates sont aussi toxiques ou plus toxiques, surtout pour les poissons, que le composé d'origine. Les facteurs dont il faut tenir compte lorsqu'on évalue les dangers des produits de transformation des pesticides sur les écosystèmes aquatiques comprennent : a) la vitesse d'apparition et de disparition des composés, b) les concentrations de résidus sur le terrain, c) la durée d'exposition du biote aquatique et d) la compartimentation des produits de transformation dans l'écosystème. Les questions qui doivent faire l'objet de recherches ultérieures comprennent l'évaluation toxicologique nécessaire des produits de transformation des nouvelles substances chimiques avant leur homologation et les effets interactifs du composé d'origine, de ses produits de transformation et de tout adjuvant de la formulation sur la réponse toxique des organismes non visés.

INTRODUCTION

The direct and indirect contamination of surface waters by pesticides is known to occur via aerial drift, watershed runoff and accidental spillage during the widespread use of these chemicals in both agriculture and forestry. In addition, many pesticides are applied directly to aquatic ecosystems to control noxious biting insects and aquatic weeds. Most pesticides with the exception of very persistent compounds undergo transformations either chemically (e.g., isomerisation, hydrolysis, photolysis, etc.) or biologically (degradation and/or metabolism by organisms) soon after application. The rate of transformation is dependent upon a variety of factors including the physicochemical properties of the pesticide, the temperature, moisture, pH, and light in the surrounding environment, the presence and abundance of organic matter and the micro- and macroflora and fauna of the ecosystem.

Numerous papers have been published on the effects of various pesticides on non-target aquatic organisms at concentrations simulating the contamination of surface waters at field application rates. Most toxicity studies, however, do not take into account the possible transformation of pesticides into compounds of equal or greater toxicity than their precursors but instead presume that transformation of a chemical results in compounds that are less persistent and less toxic. With the exception of a few studies on fish (1,2,3,4), daphnids (5,6), and algae (7,8), most studies have not examined the effects of degradation and/or tranformation products of pesticides on aquatic biota. It is the purpose of this

paper to review the available information on the toxic effects of pesticide transformation products to biota in aquatic ecosystems and to suggest future considerations for research in this area of ecotoxicology.

Toxicity of the Transformation Products of Insecticides to Aquatic Biota

Organochlorines

Although the chorinated hydrocarbons are known to be fairly persistent in the environment, they can be converted under natural conditions to even more stable and sometimes more toxic residues than the parent compounds (9). Amongst insecticides, p,p'-DDT (1,1,1-trichloro-2,2-bis (p-chlorophenyl) ethane) and its metabolites, p,p'-DDE (1,1-dichloro-2,2-bis (p-chlorophenyl) ethylene) and p,p'-TDE (1,1-dichloro-2,2-bis (p-chlorophenyl) ethane) are the most thoroughly studied compounds for their comparative effects on aquatic biota. p,p'-DDT has been shown to be toxic to many aquatic organisms at various concentrations (for a review, see reference 10) but its transformation products can have similar, lower or greater toxicity than the parent compound depending on the species of organism tested and the duration of the bioassay. For example, Sanders and Cope (11) found that p,p'-TDE is 100-fold less toxic to stonefly larvae (*Pteronarcys* spp.) than p,p'-DDT. In contrast, Kouyoumjian and Uglow (12) found that for the planarian worm *Polycelis felina*, p,p'-TDE was the most toxic and p,p'-DDT the least toxic, with p,p'-DDE showing intermediate toxicity in acute studies. At sublethal concentrations, both p,p'-

DDT and p,p'-TDE were shown to reduce the righting time of animals turned onto their backs and this was presumed to be due to an effect on the nervous system.

Studies with fish have shown that the toxicity of p,p'-TDE and p,p'-DDE is 5-10X less than p,p'-DDT in the same test system (13,14,15). In studies on sublethal toxicity, Peterson (16) monitored the selection of temperature by juvenile Atlantic salmon (Salmo salar) previously exposed to p,p'-DDT or its metabolites and found that low concentrations produced no effect on temperature selection but as concentrations of chemicals increased, the temperature selected by the fish also increased. Fish were the most sensitive, in this respect, to p,p'-DDE and showed decreasing sensitivity to o,p'-DDT, p,p'-TDE and p,p'-DDT. Conversely, Gardner (13) found that p,p'-DDE did not produce any temperature preference for the same species of fish and at concentrations similar to p,p'-DDT or any of its analogues (e.g., methoxychlor).

Some data are also available for the effects of p,p'-DDT and its metabolites on algae. Luard (17) studied the effects of p,p'-TDE, p,p'-DDE and p,p'DDT on C^{14} uptake by Scenedesmus quadricauda and found that concentrations of 0.1 to 1000 ppb were generally nontoxic; low concentrations of p,p'-TDE were stimulatory. p,p'-DDT and p,p'-DDE have been shown to have similar toxicity towards other algal species (18,19), although in other studies, this metabolite (p,p'-DDE) was less toxic than the parent compound (20).

Endosulfan (1,4,5,6,7,7-hexachloro-5-norbornene-2,3 dimethanol cyclic sulfite) is one of only a few cyclodiene

organochlorine pesticides which are still registered for use in North America. This product consists of two isomers, α-endosulfan and β-endosulfan, which differ both in their toxicities to aquatic organisms and their persistence (21,22). Transformation products of both isomers of endosulfan include endosulfan diol, endosulfan ether, endosulfan sulfate, endosulfan α-hydroxy ether and endosulfan lactone (Fig. 1) but their presence in aquatic ecosystems depends upon pH and levels of dissolved oxygen in the medium. Residues found in surface waters adjacent to agricultural land have been limited to the oxidation product, endosulfan sulfate, and the hydrolysis product, endosulfan diol (23). Aquatic organisms, particularly fish, are highly sensitive to both endosulfan and endosulfan sulfate. For example, the toxicities of both compounds to the guppy, Lebistes reticulatus, and the goldfish, Carassius auratus, have been shown to be generally within an order of magnitude of each other with the parent compound being the most toxic (0.8-10 μg/L vs. 1.6-17.5 μg/L) (24). However, the endosulfan diol, as with other transformation products not containing a sulfur group, is several thousand times less toxic than endosulfan to these same species of fish e.g., 1-10 mg/kg vs. 0.01-0.001 mg/kg respectively. The conversion of the active substances to more polar compounds with reduced penetration into the animal has been given as the explanation for decreased toxicity (25).

Aquatic algae (e.g., Chlorella vulgaris and Phormidium spp.) are not particularly sensitive to endosulfan or its metabolites.

Knauf and Schulze (24) and Goebels et al (26) observed that continuous 5-day bioassays with endosulfan and its metabolites at concentrations of 1 mg/L had no effect on the physiological activity of the algae. Rates of cell division, photosynthesis and biomass production were not affected at levels below 2 mg/L. Endosulfan sulfate, released into the water during metabolism by green algae, did not impair the physiological properties, reproductive rate or photosynthetic rate of algae. Concentrations of endosulfan in waters from agricultural regions of Canada have been reported to be < 1.0 μg/L (27) and, therefore, aquatic algae will not be at risk in these surface waters. However, there is a limited safety factor with regard to sensitive species of fish and the concentrations of endosulfan and endosulfan sulfate present in these waters. In addition, since endosulfan and its more polar degradation products appear to be concentrated several thousand times by sediments and suspended solids, the impact of these residues on benthic organism and filter feeders may merit particular attention (23).

Batterton et al. (28) studied the effects of several chlorinated hydrocarbons containing an endomethylene bridge and their transformation products, namely, aldrin, photoaldrin, photodieldrin, metabolites F and G of dieldrin and ketoendrin, on the growth responses of the blue-green algae, Anacystis nidulans and Agmenellum quadruplicatum. In nature, aldrin is converted to photoaldrin, epoxidized to dieldrin, and dieldrin transformed to aldrin trans-diol which may be the active toxicant (29). Results

indicated that transformation products of aldrin and dieldrin can be at least as inhibitory to algal growth as the parent compounds but concentrations used in this study were much higher for both the parent and metabolites than concentrations found in nature. Both blue-green algae were more tolerant of ketoendrin than endrin in this study.

Organophosphates and Carbamates

Organophosphate and carbamate insecticides are diverse and extensively used groups of chemicals which have replaced the more persistent and environmentally damaging organochlorines. Organophosphorus insecticides are normally esters, amides, or thiol derivatives of phosphoric, phosphonic, phosphorothioic or phosphonothioic acids and the carbamates are alkyl and aryl esters of carbamic acid. Thus, these compounds can be expected to be subject to hydrolysis and to be chemically reactive. For example, parathion has been shown to form paraoxon and the S-ethyl and S-phenyl isomers under the influence of sunlight and ultraviolet radiation (30). Limited information is available on the effects of transformation products of organophosphates and carbamates to aquatic biota with the exception of the organophosphate, fenitrothion, and the carbamates, aminocarb and aldicarb.

Fenitrothion (O,O-dimethyl-O-(3-methyl-4-nitrophenyl)-phosphorothioate) is a phosphorothioate compound which requires transformation to fenitrooxon, the oxidative desulfuration metabolite of fenitrothion, for toxicity. This activation step

usually occurs under the influence of enzymes known as mixed function oxidases (MFOs) found in animals, plants and certain microorganisms (31). However, the conversion of phosphorothioate insecticides to the more potent phosphoroate analogue can also occur by photo- or chemical oxidation under natural conditions (32). The known transformation products of fenitrothion are aminofenitrothion (microbial), carboxyfenitrothion (photolysis), demethylaminofenitrothion (microbial, anaerobic), demethyl-fenitrothion (microbial) and fenitrooxon (oxidation) (33) (Fig. 2). Miyamoto et al. (1) studied the relative toxicity of fenitrothion and its various degradation products to the killifish, Oryziaas latipes, and found that all compounds were less toxic than the parent fenitrothion with the exception of 3-methyl-4-aminophenol (e.g., LC_{50} values were 3.4 mg/L for fenitrothion, >10.0 mg/L for fenitrooxon, aminofenitrooxon, demethylaminofenitrothion and carboxyfenitrothion, 8.4 mg/L for 3-methy-4-nitrophenol and 0.078 mg/L for 3-methyl-4-aminophenol). In Daphnia, fenitrothion and fenitrooxon were the most toxic among the compounds tested. Hiroaka et al. (4) exposed the same species of fish to several dilutions of a fenitrothion emulsion placed in natural sunlight for 47 days and compared the results to a control group of fish exposed to an untreated fenitrothion emulsion. The effects of the treated solution on the hatching rate of fertilized eggs and the rate of survival of larvae were greater than those of the untreated solution e.g., percent hatch of eggs was 46-64% in the treated solution vs. 90-94% hatching success in the untreated solution.

In acute tests on adult fish, the number of survivors at 24 and 48 h in the untreated solution was 60-70% whereas none survived at a concentration of 4 mg/L. The authors concluded that exposure of fenitrothion to sunlight results in degradation and/or transformation to products which are more toxic than the parent compound; unfortunately, the authors did not identify these products or measure their concentrations.

Moody et al. (34) studied the fate of fenitrothion in stream water following aerial spraying and found traces of demethylamino-fenitrothion, S-methylfenitrothion and aminofenitrothion up to 100 h following application. Ohmae et al. (35) studied the environmental behaviour of fenitrothion and its decomposition products after operational aerial application and found that the initial concentration of fenitrothion in water (38.2 μg/L) immediately after application was rapidly reduced although small amounts of the parent compound (0.02 μg/L) and 3-methyl-4-nitrophenol were detected after 49 days. 3-Methyl-4-nitrophenol has been shown to be an inhibitor of the enzyme, ribonucleotide reductase, which is a key regulatory enzyme in DNA synthesis in mammals (36) and therefore it is possible that sublethal concentrations of this chemical in aquatic ecosystems could have detrimental effects on biota. 3-Methyl-4-aminophenol has not been detected in field studies but low concentrations of this chemical could cause toxic effects based on the results of Miyamoto et al. (1). Technical fenitrothion is also known to contain an impurity, S-methyl fenitrothion, which is significantly more toxic than fenitrothion

(37) but this impurity would have been present in both solutions in the study by Hiraoka et al. (4) and is not likely the cause of the observed toxicity.

Aminocarb (4-dimethylamino-3-methylphenyl N-methylcarbamate) is a broad spectrum carbamate insecticide applied extensively throughout the world but used particularly in forestry in Canada to control the spruce budworm, Choristoneura fumiferana (38). Aminocarb has been studied by many authors and many transformation products have been identified; for example, AA (4-amino-m-tolyl N-methylcarbamate), AC (4-amino-3-methylphenol), FA (4-formamido-m-tolyl N-methylcarbamate), FC (N-(4-hydroxy-2-methylphenyl)-N-methylformamide), MFA (4-methylformamido-m-tolyl N-methylcarbamate), MAA (4-methylamino-m-tolyl N-methylcarbamate), MAC (3-methyl-4-(methylamino)phenyl-N-methylcarbamate) have been identified (Fig. 3) as well as phenol, methylamine and CO_2 (38,39). Szeto et al. (2) determined the toxicity of the oxidative demethylation metabolites (i.e., MFA, MAA, FA and AA) by measuring the inhibition of brain acetylcholinesterase (AChE) in brook trout (Salvelinus fontinalis). They found that the toxicity expressed as the in vitro molar concentrations at which 50% of the enzyme is inhibited (I_{50}'s) could be ranked as AA (3.62×10^{-6}) > MAA (7.92×10^{-6}) > aminocarb (1.01×10^{-6}) > MFA (4.29×10^{-5}) > FA (7.11×10^{-5}). These data correlate very well with the LC_{50}'s of aminocarb (5.7 mg/L) and its metabolites (e.g., 1.7 mg/L for AA, 0.349 mg/L for MAA, >15 mg/L for MFA and 15 mg/L for FA) to rainbow trout (Salmo gairdneri) determined by Lamb and Roney (40). In vivo

recovery of fish brain (brook trout) AChE activity after transfer of fish from water contaminated with aminocarb or MAA was also studied by Szeto et al. (2). Mortality was greater and levels of AChE in living fish were lower in animals exposed to MAA compared to those exposed to similar concentrations of aminocarb. The authors concluded that according to enzyme inhibition, the metabolites AA and MAA were more potent than the parent compound and MFA And FA were less potent.

Monitoring studies suggest that concentrations of aminocarb in natural woodland waters rarely exceed 10 μg/L although considerably higher concentrations have occasionally been cited (i.e., 25-53 μg/L) (41). These residues are within the range of the lowest concentrations of aminocarb and metabolites which have been shown to cause effects; for example, Szeto et al (2) observed reductions in fish brain AChE at concentrations of 25 μg/L. Although residues of MAA have been detected in fish tissues after exposure to various concentrations of aminocarb in the laboratory (42) the metabolite has never been detected in natural water after aerial spray although few field studies have analysed for this tranformation product. Ernst et al. (43) found three metabolites (FA, AC and MAC) to persist for 24 days following spray for spruce budworm control in New Brunswick but FC, MAA and AA were not detected.

Aldicarb (2-methyl-2[methylthio]propionaldehyde O-[methyl carbamoyl]-oxime) is a highly water soluble and widely used carbamate insecticide and nematocide. Due to its toxicity to

mammals as a potent AChE inhibitor, aldicarb is only applied as a granular to the soil where it is mobilized and released by moisture (44). It then undergoes rapid microbial oxidation to the relatively stable aldicarb sulfoxide and then slower oxidation to aldicarb sulfone (Fig. 4). The degradation and transport of this compound in water, especially groundwater, has been studied widely due to recent findings of the parent compound and its metabolites in drinking water in parts of Canada and the United States (45,46). Foran et al. (5) studied the acute toxicity of aldicarb, aldicarb sulfoxide and aldicarb sulfone to the cladoceran, Daphnia laevis, and found that aldicarb sulfoxide was similar in toxicity to the parent compound (e.g., range 43 - 65 μg/L) for both adults and juveniles; however, the aldicarb sulfone was almost an order of magnitude less toxic than aldicarb and aldicarb sulfoxide (369 μg/L vs. 51 for adults; 556 μg/L vs. 65 for juveniles). Similar results were obtained for bluegill sunfish (47); for example, static 72-h LC_{50}'s were approximately 100 μg/L for aldicarb, 400 μg/L for aldicarb sulfoxide and over 1000 μg/L for aldicarb sulfone). These data indicate that aldicarb and the first oxidative metabolite, aldicarb sulfoxide, are equal in toxicity and therefore microbial oxidation does not lessen the toxic impact of aldicarb contamination of surface waters; however, further degradation of aldicarb sulfoxide as well as aldicarb sulfone, to corresponding oximes and nitriles is thought to occur fairly rapidly with additional degradation to aldehydes, acids and alcohols, none of which are toxicologically significant (45).

Synthetic Pyrethroids

The synthetic pyrethroids are a class of lipophilic insecticides which have been marketed for agricultural uses for approximately 10 years. This group of chemicals can be manufactured as a mixture of complex molecules with several optically active centers (e.g., permethrin, cypermethrin and fenvalerate) or as single isomers (e.g., deltamethrin). The environmental fate and effects of these compounds have been described by various authors and they are known as insecticides which are highly toxic under laboratory conditions to fish and other aquatic organisms (48) and are very easily degraded in the natural environment (49). There are three main chemical reactions involved in this degradation i.e., isomerisation, hydrolysis and oxidation. Isomerisation usually involves the cyclopropane ring and is initiated by sunlight but the process may be affected by the presence of pigments or humic substances (50). Hydrolysis occurs at the ester bond and results in a breaking of the parent molecule into two fragments, the acid and alcohol moieties. Oxidation may occur in any part of the molecule.

The stereochemical structure of pyrethroid insecticides greatly influences their toxicity to aquatic organisms (51). Cis 1R, αS-deltamethrin is the only enantiomer present in the product registered for agricultrual use (50). However, this parent compound (isomer 1) has been shown to convert to 3 other isomers in natural water exposed to sunlight (i.e., cis 1S, αS (isomer 2'),

trans 1R, αS (isomer 3), and trans 1S, αR (isomer 4')) (Maguire, J. Agric. Food Chem., in press) (Fig. 5). Four other isomers (1', 2, 3' and 4) are known to exist but have not been found in treated water. Day and Maguire (6) found that of these isomers, only isomers 1,2,3 and 4 were toxic to juvenile Daphnia magna with the parent compound being approximately 10X more toxic. These results indicate therefore that isomerization of isomer-1 to isomer-3 in natural water is only a partial detoxification step as far as some aquatic organisms are concerned although isomer-3 has not been reported in ponds oversprayed with deltamethrin (52,53). The isomer pair (3+3') has however, been found on pasture forage and litter (54) and alfalfa (55).

There have been several studies on the toxicities of the products of ester hydrolysis of pyrethroids to aquatic organisms (6,7,48). The major degradation products of these insecticides are considerably more polar than the parent molecules and all have been shown to have very much lower toxicity to fish and invertebrates than the parent compounds (Table 1).

In contrast, Stratton and Corke (7) found that two to five of the degradation products of permethrin were significantly more toxic towards algae and cyanobacteria than the parent compound. For example, permethrin is relatively nontoxic towards phototrophic microorganisms, with EC_{50} values for growth and photosynthesis being > 10 and > 100 mg/L, respectively; however, 3-phenoxybenzaldehyde (PBald) and 3-phenoxybenzyl alcohol (PBalc), followed by benzoic acid, 3-hydroxybenzoic acid and 3-phenoxybenzoic acid had values

ranging from 2 to 6 mg/L for growth and 30 to 70 mg/L for photosynthesis. The cyanobacteria were more sensitive than the green algae and the authors attributed this to the basic cellular organizational differences between these organisms (procaryotic vs. eucaryotic cells). However, the concentrations used in this study were much higher than concentrations expected in natural waters contaminated with pyrethroids (48). Therefore, it is the toxicity of the parent pyrethroid molecule (or its active isomers) rather than its degradation products which are of potential concern in aquatic ecosystems.

Toxicity of the Transformation Products of Herbicides to Aquatic Biota

There are relatively fewer data available on the effects of transformation products of herbicides on aquatic biota compared to those available for insecticides.

The triazine herbicides are a group of heterocyclic nitrogen compounds which have been largely responsible for the substantial increases in corn yields in North America observed over the last 25 years (56). Of these herbicides, atrazine (2-chloro-4-ethylamino-6-isopropylamino-1,3,5-triazine) is the most heavily applied agricultural pesticide in North America and it is used to control broadleaf and grassy weeds in corn and sorghum (57).

Atrazine has been detected in lakes and streams at levels ranging from 0.1 to 30.3 μg/L with peak concentrations up to 1000 μg/L known to occur in surface runoff from agricultural fields

adjacent to bodies of water during times of application (58). Many studies have been conducted on the effects of atrazine on various species of aquatic flora under controlled conditions and these studies have found that at concentrations of 1 to 5 μg/L and exposure periods of 5 min. to 7 weeks, adverse effects on photosynthesis, growth and oxygen evolution of aquatic plants have occurred. Higher concentrations have altered species composition, reduced carbon uptake and reduced reproduction (57).

The half-life of atrazine in aquatic environments has been shown to range from 3.2 days to 7 to 8 months (57). The major route of degradation is thought to be hydrolysis to hydroxyatrazine (2-hydroxy-4-ethylamino-6-isopropylamino-1,3,5-triazine) although N-dealkylation through removal of the ethylpropyl or the isopropyl group has also been shown to occur (59) (Fig. 6). Few studies have measured the concentrations of these metabolites in the natural environment and only one study to date has examined the effects of these degradation products of atrazine on aquatic biota. Stratton (8) found that atrazine was 4 to 10 times more effective than its transformation products in producing reductions in growth, inhibition of photosynthesis, and acetylene-reducing ability in two species of green algae, Chlorella pyrenoidosa and Scenedesmus quadricauda, and three species of cyanobacteria, Anabaena spp. For example, atrazine reduced growth by 50% at 0.03 to 5.0 mg/L and inhibited photosynthesis by 50% at 0.1 to 0.5 mg/L. Comparable values for deethylated atrazine (2-chloro-4-amino-6-isopropylamino-1,3,5-triazine) were 1.0 to 8.5 mg/L for growth

reduction and 0.7 to 4.8 mg/L for photosynthetic inhibition. For deisopropylated atrazine (2-chloro-4-ethylamino-6-amino-1,3,5-triazine) these values were 2.5 to >10 mg/L and 3.6 to 9.3 for the same physiological functions. Hydroxyatrazine and diaminoatrazine (2-chloro-4,6-diamino,1,3,5-triazine) were nontoxic to most cultures tested. Acetylene reduction with cyanobacteria was found to be insensitive to all of the test compounds with the exception of atrazine which had an EC_{50} of 55 mg/L towards Anabaena inaequalis. This study concludes that atrazine degradation products would not normally be present in the aquatic environment at levels inhibitory to algae and cyanobacteria.

Several other transformation products of herbicides have been studied for their effects on photosynthetic microorganisms. For example, 3,4-dichloroaniline, a major degradation product of the amide herbicide, propanil (3',4'-dichloropropionanilide) has been shown to be up to 10X less inhibitory than the parent compound towards phototrophic organisms (60). In addition, this metabolite had no effect on algal populations in experimental enclosures treated with this chemical (61).

The effects of the carbanilate, chlorpropham (isopropyl m-chlorocarbanilate) and 3-chloroaniline, a metabolite, on populations of the cyanobacterium, Anacyctis midulans and the alga, Chlamydomonas reinhardii were monitored in large scale batch cultures by Maule and Wright (62). 3-Chloroaniline was less inhibitory than the parent herbicide, chlorpropham; for example, 6.1-9.5 μg chlorpropham/mL inhibited growth rates and final yield

of A. nidulans vs. 5.4-19.9 μg 3-chloroaniline/mL for the same parameters. Similar results were obtained for C. reinhardii. In addition, chlorpropham caused marked morphological changes such as increase in cell size, the formation of multi-layered cell envelopes, etc. at concentrations which still permitted substantial growth whereas 3-chloroaniline produced no such changes.

Triclopyr (3,5,6-trichloro-2-pyridinyloxyacetic acid) (Fig. 7) is the active ingredient in several relatively new herbicides which are formulated as either an amine (Garlon 3A) or an ester (Garlon 4). These herbicides are used in selective post-emergent control of woody plants and broadleaf vegetation in forest site preparation and conifer release programs. In such applications, these chemicals can potentially reach surface water through drift or inadvertent overspray of aquatic areas and this raises concerns about the potential hazards of the active ingredient or its transformation products to aquatic life, particularly fish.

Wan et al. (3) evaluated the acute toxicity of both formulated products, technical triclopyr and several transformation products to juvenile Pacific salmonids and found that the order of increasing toxicity was - Garlon 3A (347 ± 44 mg/L), triclopyr (7.9 ± 0.7 mg/L), pyridine (3.7 ± 0.8 mg/L), pyridinol (2.1 ± 0.2 mg/L), Garlon 4 (2.0 ± 0.2 mg/L) and triclopyr ester (0.7 ± 0.2 mg/L). Both triclopyr and the amine formulation, Garlon 3A, were less toxic than both the triclopyr ester and the major transformation products; the metabolic product, pyridinol, was as toxic as the formulated triclopyr product, Garlon 4, which contains the

triclopyr ester.

The triclopyr ester (2-butoxyethyl 3,5,6-trichloro-2-pyridinyloxyacetate) has a short half-life and undergoes hydrolysis in water and soil to the active acid, triclopyr, within 6-24 h (63). The acid is resistant to further hydrolysis but susceptible to photolysis and transformation by microorganisms to 3,5,6-trichloro-2-pyridinol and CO_2. Absorption by fish and metabolism to the acid form of the chemical and excretion of the acid back into the water is also a known route of transformation (3). Under field conditions, the concentration of Garlon 3A in a stream unintentionally oversprayed during an aerial operation would not likely exceed a level greater than 10 mg/L in 15 cm water even at the highest recommended rate of application (i.e., 10 kg active ingredient/ha). Therefore, the potential for this formulation or its transformation products to cause fish kills is small when it is used under prescribed conditions. However, the use of the lower recommended rate of Garlon 4, (i.e., 2.4 kg active ingredient/ha), has some potential to generate toxic concentrations (approximately 4 mg/L in 15 cm water) if the residues of the parent compound or its degradation products are not rapidly diluted and flushed out of the aquatic system.

The phenoxy alkanoic acid herbicides such as 2,4-D (2,4-dichlorophenoxy acetic acid) are also formulated as esters or amine salts to improve their solubility in oil or water respectively. The ester formulations (e.g., butoxyethanol ester (BOEE) and propylene glycol butyl ether ester (PGBEE)) have been shown to

rapidly convert to the acid in water; for example, approximately 21 and 38% hydrolysis of BOEE and PGBEE, respectively, to 2,4-D acid occurred within a 3 h 50% water volume replacement time in continuous flow bioassays with chinook salmon (Oncorhynchus tshawytscha) and steelhead-rainbow trout (Salmo gairdneri) (64).

The acute toxicities of the ester formulations are approximately 100X more toxic than the acid formulation (65) presumably due to the quicker absorption of nonpolar (esters) rather than polar (acid) compounds via passive transport through the gill membranes of fish. Thus the rapid conversion of the ester results in the reduced toxicity of 2,4-D to fish although the toxicity of other transformation products to fish and other aquatic organisms has not been tested.

Other Pesticides

The tributyltin compounds (e.g., bis (tributyltin oxide) and tributyltin fluoride) are biocides used in organotin antifouling paint formulations which are classified as pesticides under the Pest Control Products Act in Canada. They are known to be extremely toxic to aquatic organisms and several countries have restricted their use (66). Tributyltin degrades by successive debutylation to dibutyltin, monobutyltin and finally to inorganic tin. Studies on the acute toxicity of these transformation products to aquatic invertebrates and algae have shown that the tributyltin species is the most toxic chemical with toxicity decreasing with successive degradation (Table 2).

Very little information on the toxicity of other transformation products of pesticides, particularly fungicides, to aquatic biota is available in the open literature.

Summary and Conclusions

The transformation and/or degradation of pesticides in the natural environment results in chemicals which have the potential to be deleterious to non-target organisms. A review of the scientific literature for information on the effects of these transformation products on aquatic biota has indicated that such compounds can be less, more or similar in toxicity when compared to the parent chemical (Table 3). As a general trend, the toxicity of these products is reduced for most pesticides, particularly herbicides, although this is somewhat dependent upon the species of organism tested i.e., plant or animal. In addition, several metabolites of the organophosphate and carbamate insecticides were similar to or more toxic than the parent compound e.g., fenitrooxon, 3-methyl-4-aminophenol; AA and MAA of aminocarb. More information on a wider variety of pesticides, particularly organophosphates, carbamates and synthetic pyrethroids would be necessary to conclude that the transformation products of most pesticides are less toxic than the parent compound.

There are several factors which must be considered when evaluating the hazards of transformation products of pesticides in aquatic ecosystems. The breakdown and/or conversion of pesticides

to their respective metabolites in the natural environment is a continuous process which occurs from the time of application (or before) and beyond. The nature of the compounds formed, the rate at which they appear and disappear, their concentrations and their compartmentalization into various parts of the ecosystem are dependent upon the physical, biological and chemical properties of the system involved. These complicating factors make it difficult to estimate the actual concentrations and the length of time to which organisms are exposed to any given metabolite in the environment. Some metabolites may not be biologically available to organisms or are present for such a limited duration (e.g., minutes to hours) that there is no time for a toxic reaction and response to occur. On the other hand, the uptake, storage and/or eventual metabolism of pesticides by biota and/or the microbial release of pesticides bound to particulate organic matter over time may result in a continuous input of low levels of toxic metabolites into the environment. Few studies have examined the sublethal, long-term effects of persistent transformation products on biota in the aquatic environment.

Much of the available information on the toxicity of transformation products to specific organisms is based on laboratory toxicity data where conditions are very different from those in the field. For example, the presence of natural sunlight produces radiation of various wavelengths which cannot be simulated in the laboratory and which can cause photodegradation and transformation. In addition, most laboratory tests are static and

concentrations of chemicals are not permitted to diminish as rapidly as they would under field conditions especially in the lotic (running water) environment. The concentrations of transformation products used in laboratory toxicity tests often exceed by several orders of magnitude the actual concentrations of chemicals which occur following application. Realistic concentrations of residues of pesticide transformation products may be below the limits of detection. In addition, few field studies monitor for residues of transformation products under field conditions.

An area that requires further research is an evaluation of the interactive effects of the parent compound, its various transformation products and other formulation adjuvants on the toxic response of the non-target organism. No contaminant is ever present alone in the environment but is always in association with other organic and inorganic chemicals including its own transformation products. Stratton (8) found that when atrazine and its dealkylated breakdown products were combined and tested against the blue-green alga, A. inaequalis, synergistic, antagonistic and additive interaction responses were observed depending upon the test system employed. For example, whenever atrazine was present in a mixture with deisopropylated- or deethylated-atrazine, antagonism occurred using the photosynthetic response as a parameter i.e., the apparent inhibitory effects of the individual toxicants were reduced. In contrast, a synergistic interaction was recorded for culture growth i.e., toxicity was enhanced.

Combinations of deethylated- and deisopropylated-atrazine interacted antagonistically towards photosynthesis and additively towards the growth yield. Additive interactions occur when the overall toxicity of the mixture is greater than that of the component compounds. The interactive effects of pesticides, their transformation products, and any other chemicals (toxic or non-toxic) could lead to situations in the natural environment where degradation products of low individual toxicity still pose a serious treat to non-target organisms when in combination.

The determination of the toxicity of pesticide transformation products to aquatic biota is a difficult task due to the uncertainties of concentrations, time of exposure and availability; however, for new pesticides being considered for registration under the Pest Control Products Act in Canada, it has been suggested that toxicity tests be conducted on any transformation product which is present at concentrations > 10% of the applied pesticide or accumulates over the course of laboratory transformation and/or field dissipation studies (Commercial Chemicals Branch, Environment Canada, personal communication). The toxicity of such transformation products would initially be tested at a concentration comparable to that which results from an application at the maximum label-recommended rate to provide a worst-case scenario. If no toxicity is observed, no further testing would be required; however, if toxicity is observed, a number of lower concentrations would be tested to generate a dose-response curve. Such extensive testing of toxic transformation products of new chemicals coming

on the market would ensure that pesticides with very toxic breakdown products or those chemicals which convert to more toxic isomers would be detected.

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TABLE 1.

ACUTE TOXICITY OF PYRETHROID METABOLITES

Chemical	*Daphnia magna* 48 h EC_{50} µg/L	Fish 96 h LC_{50} µg/L	Reference
3-(2,2-dichlorovinyl)-2,3-dimethyl cyclopropane carboxylic acid (DCVA)[a]	130,000	3,000	48
3-(2,2-dibromovinyl)-2,2-dimethylcyclopropane carboxylic acid (DBCA)[b]	> 50	-	6
(1RS)-cis-3-(chloro-3,3,3-trifluoroprop-1-enyl)-2,2-dimethyl cyclopropane carboxylic acid[c]	100,000	> 16,000	48
3-phenoxybenzyl alcohol	10,000	3,000-7,000	48
(PBalc)	> 25	-	6
3-phenoxybenzoic acid	85,000	13,000-36,000	48
(PBacid)	> 25	-	6
3-phenoxybenzaldehyde (PBald)	> 25	-	6
2-(4-chloropheynl)-3-methyl butyric acid	-	> 10,000	48

[a] degradation product of permethrin
[b] degradation product of deltamethrin
[c] degradation product of lambda-cyhalothrin

TABLE 2.

TOXICITY OF TRANSFORMATION PRODUCTS OF TRIBUTYLTIN SPECIES

SPECIES	PARAMETER	COMPOUND	CONCENTRATION		REFERENCE
Daphnia magna	24 h EC_{50}	Tributyltin	0.013	mg/L	67
		Dibutyltin	49.0	"	"
		Monobutyltin	0.9	"	"
Ankistrodesmus falcatus	IC_{50}	Tributyltin	0.02	mg/L	68
		Dibutyltin	6.8	"	"
		Monobutyltin	25.0	"	"
Skeletonema costatum	EC_{50}	Tributyltin	0.36	µg/L	69
		Dibutyltin	40.0	"	"
S. costatum	LC_{50}	Tributyltin	11.5	µg/L	69
		Dibutyltin	>500	"	"

TABLE 3.

RELATIVE TOXICITY OF PESTICIDE TRANSFORMATION PRODUCTS TO THE PARENT COMPOUND

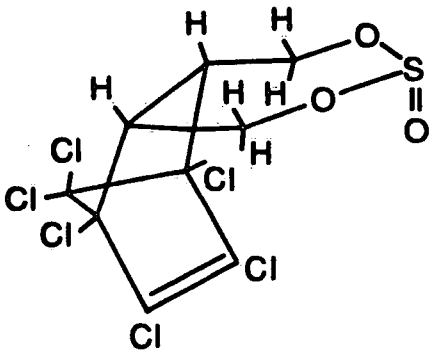
CHEMICAL & METABOLITE	SENSITIVITY		
	LESS	EQUAL	MORE
Insecticides			
DDT			
TDE	ab		ab
DDE	c	ac	a
Endosulfan			
endosulfan sulfate		a	
endosulfan diol	a	c	
Aldrin			
photodieldrin	c		
Endrin			
ketoendrin	c		
Fenitrothion			
fenitrooxon			b
aminofenitrothion	a		
carboxyfenitrothion	a		
demethylfenitrothion	a		
3-methyl-4-nitrophenol	a		
3-methyl-4-aminophenol			a
Aminocarb			
AA			a
MAA			a
MFA	a		
FA	a		
Aldicarb			
aldicarb sulfoxide		ab	
aldicarb sulfone	ab		
Deltamethrin			
isomer 2'	ab		
isomer 3	ab		
isomer 4'	ab		
PBald	ab		c
PBacid	ab		c
PBalc	ab		c

TABLE 3. (cont.)

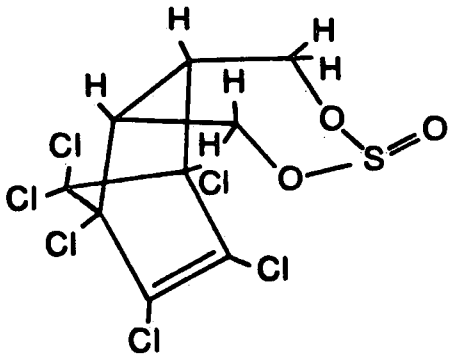
RELATIVE TOXICITY OF PESTICIDE TRANSFORMATION PRODUCTS TO THE PARENT COMPOUND

CHEMICAL & METABOLITE	SENSITIVITY		
	LESS	EQUAL	MORE
Herbicides			
Atrazine			
deethylated	c		
deisopropylated	c		
diaminoatrazine	c		
hydroxyatrazine	c		
Propanil			
3,4-dichloroaniline	c		
Chlorpropham			
3-chloroaniline	c		
Triclopyr			
pyridinol		a	
pyridine	a		
Biocides			
Tributyltin			
dibutyltin	bc		
monobutyltin	bc		

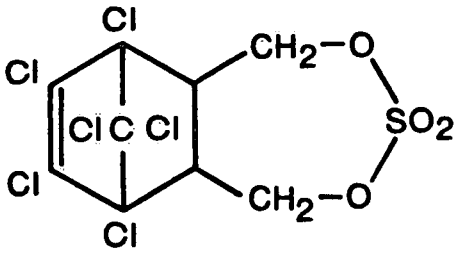
a = fish; b = invertebrates; c = algae



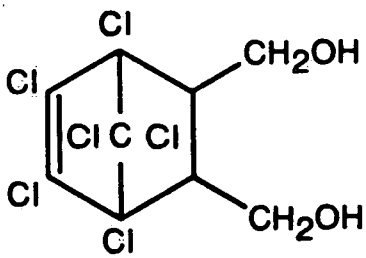
α-endosulfan



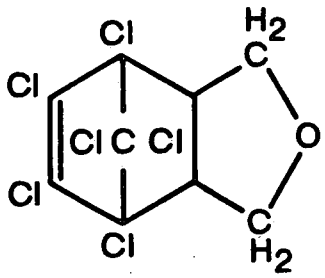
β-endosulfan



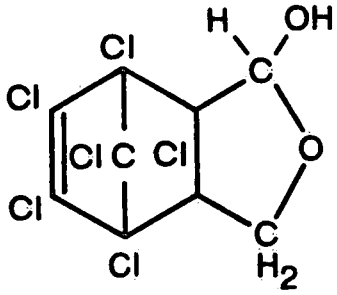
endosulfan sulfate



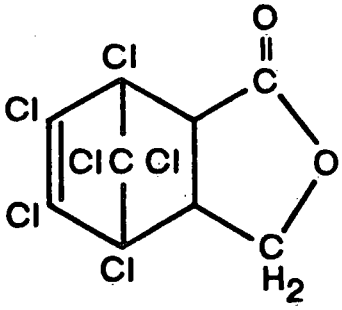
endosulfan diol



endosulfan ether

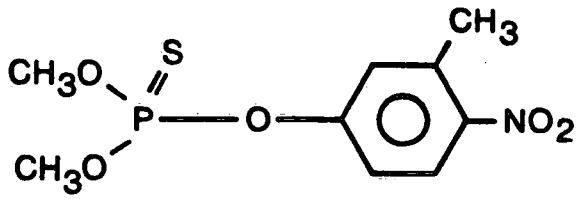


endosulfan α-hydroxy ether

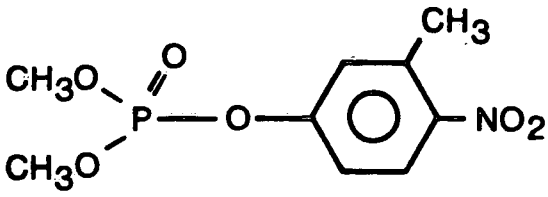


endosulfan lactone

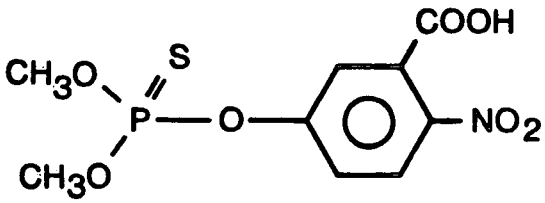
FIGURE 1. Some transformation products of endosulfan.



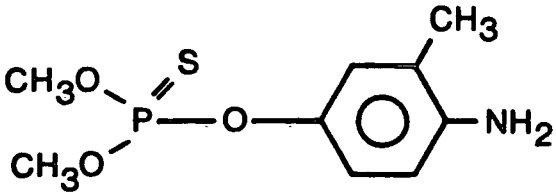
fenitrothion



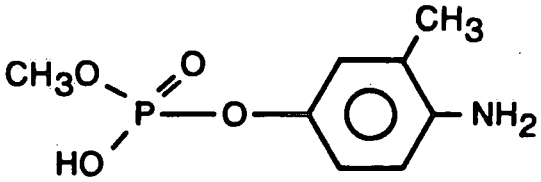
fenitrooxon



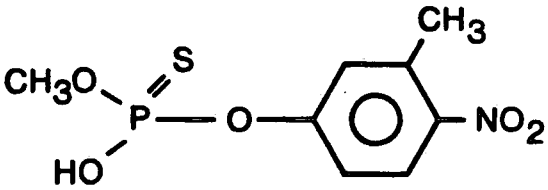
carboxy fenitrothion



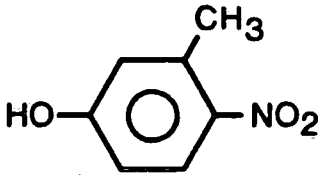
amino fenitrothion



demethyl-amino fenitrothion

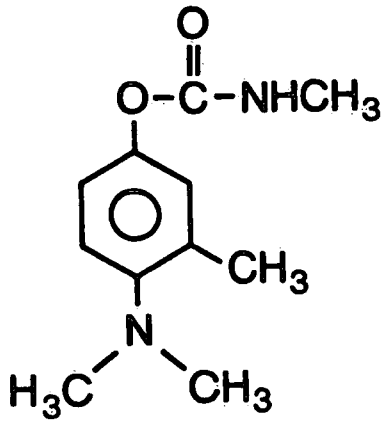


demethyl fenitrothion

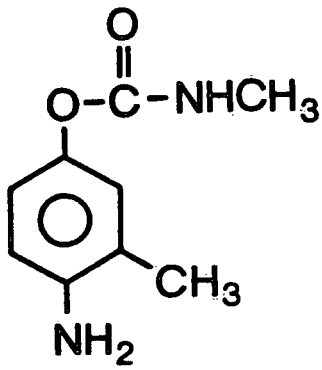


3-methyl-4-nitrophenol

FIGURE 2. Some transformation products of fenitrothion.



aminocarb



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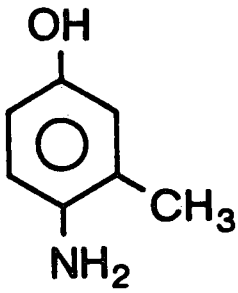

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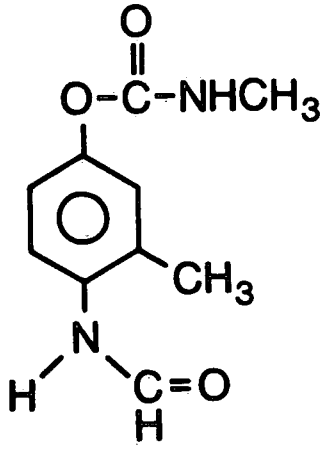

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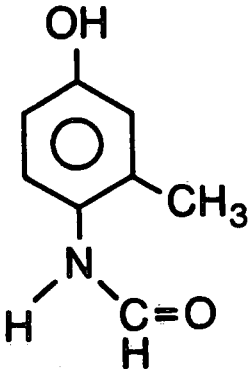

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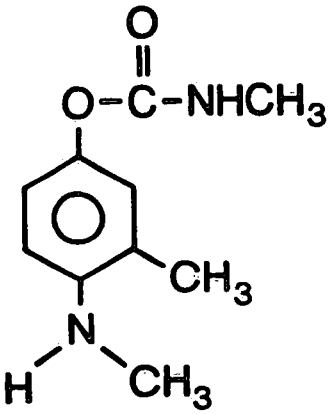

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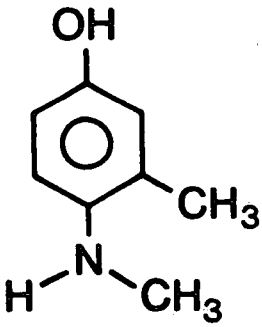

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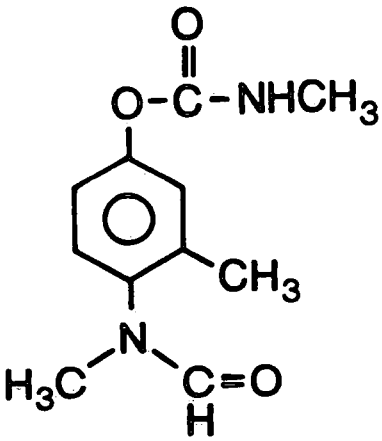


MFA

FIGURE 3. Some transformation products of aminocarb.

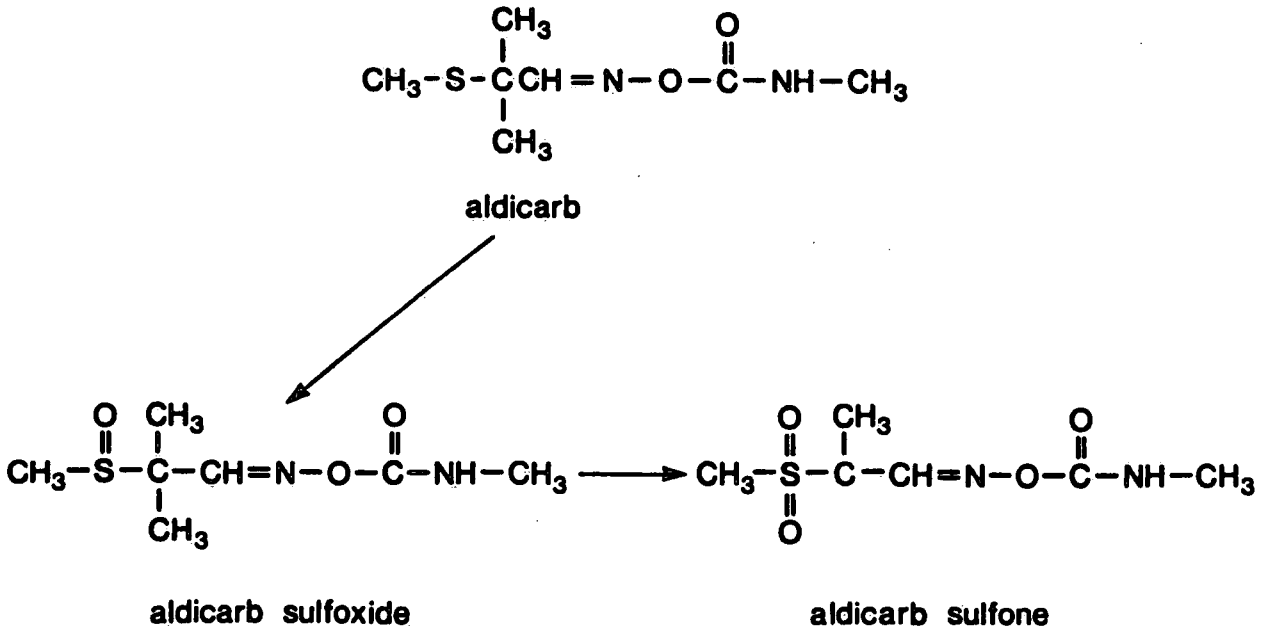


FIGURE 4. Some transformation products of aldicarb.

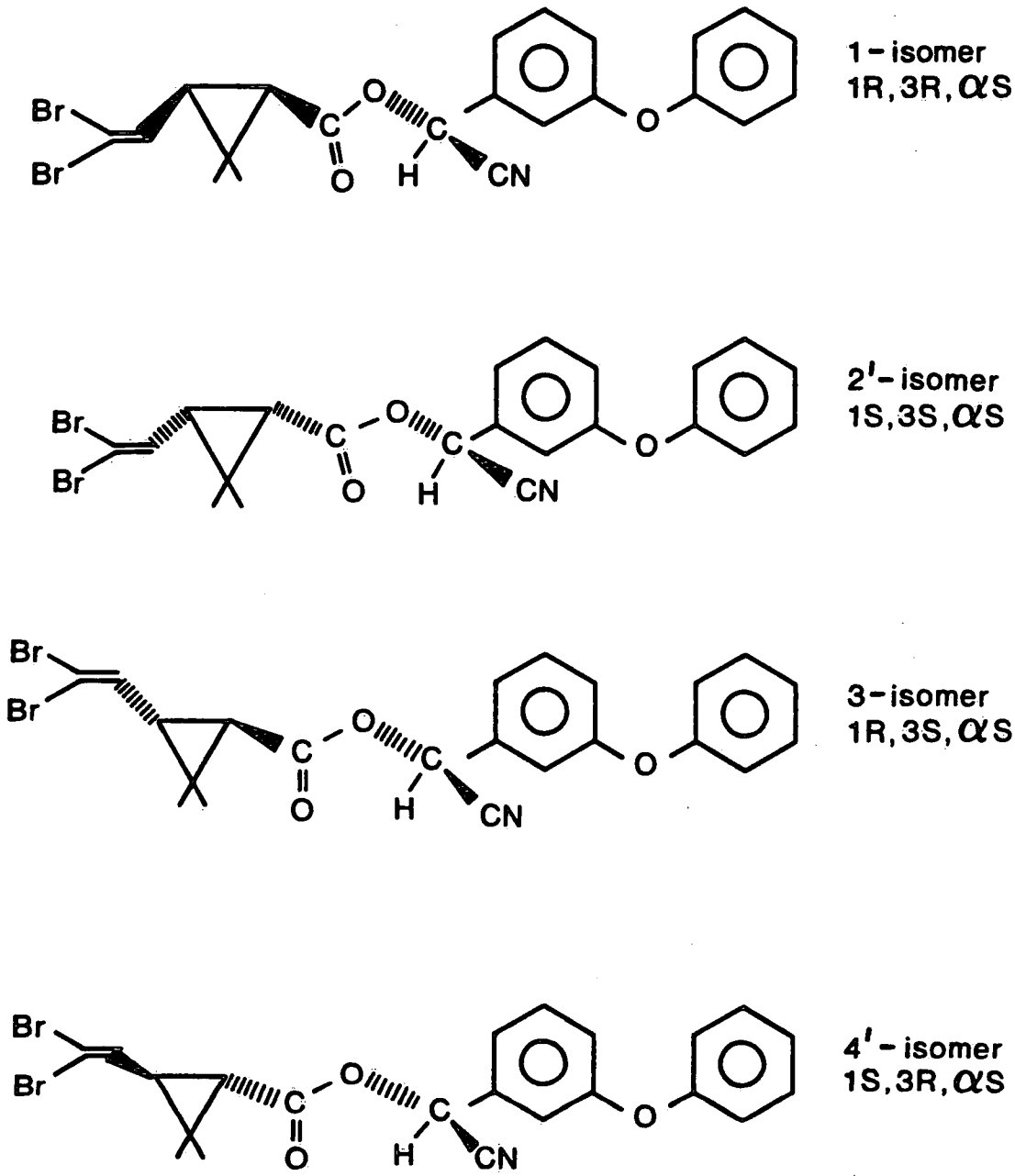


FIGURE 5. Toxic isomers of deltamethrin.

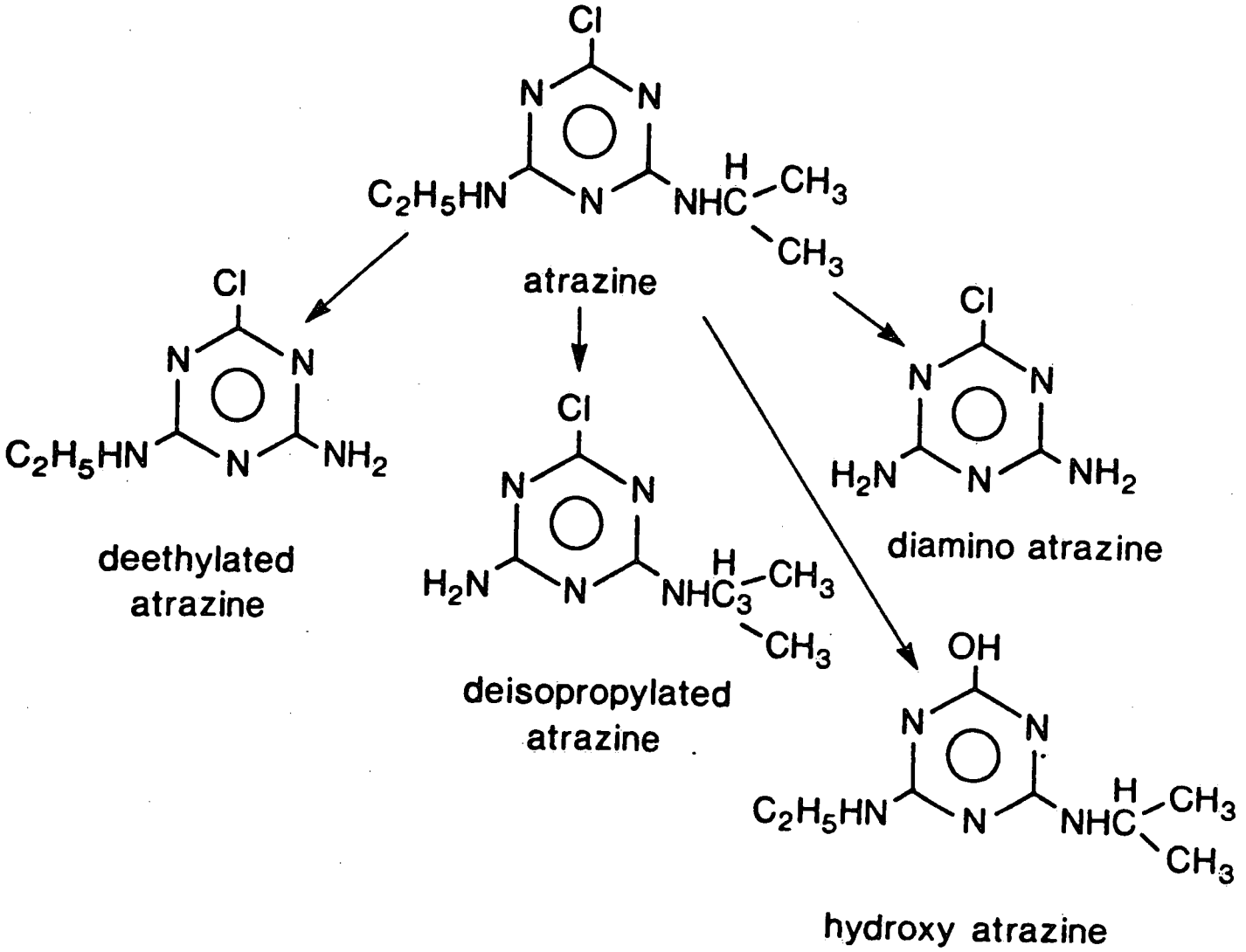
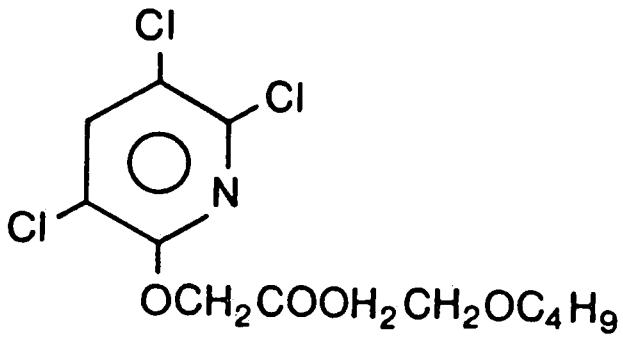
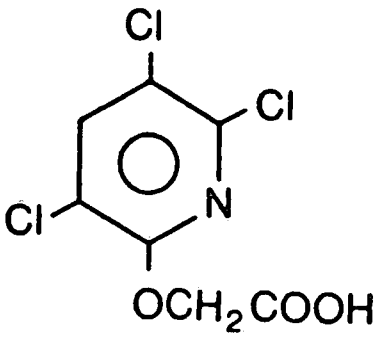


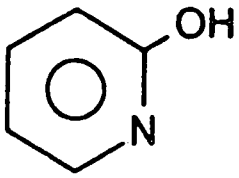
FIGURE 6. Some transformation products of atrazine.



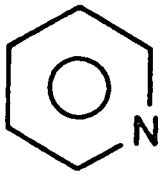
Triclopyr ester



Triclopyr



Pyridinol



Pyridine

FIGURE 7. Some transformation products of triclopyr.

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