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Insecticide

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1. Introduction

Insecticides are organic or inorganic chemicals substances or mixture of substances intended for preventing, destroying, repelling or mitigating the effect of any insect including crawling and flying insects which may occur naturally or is synthesized (pyrethroids) e.g. organic perfumed and hydrocarbon oil and pyrethrins. There are various forms of insecticides. Most of the synthesized insecticides are by their nature are hazardous on health under the condition in which it is used. Insecticides therefore, range from the extremely hazardous to those unlikely to produce any acute hazard. Most are repellants and or insect growth regulators used in agriculture, public health, horticulture, food storage or other chemical substances used for similar purpose.

It is evident that insecticides have been used to boost food production to a considerable extent and to control vectors of disease. However, these advantages that are of great economic benefits sometimes come with disadvantages when subjected to critical environmental and human health considerations. Many insecticides are newly synthesized whose health and environmental implications are unknown.

Insecticides have been used in various forms from hydrocarbon oils (tar oils), arsenical compounds, organochlorine, organ phosphorous compounds carbonates, dinitrophenols, organic thiocynates, sulfur, sodium fluoride, pyethroids ,rotenone to nicotine, in solid or liquid preparation. Interestingly, most of these have been withdrawn due to the deleterious effect of the substances. Analysis of these formulations, their by- products and residues had in the past aided objective re–evaluation and re-assessment of these substances on a benefit–risk analysis basis and their subsequent withdrawal from use when found to be dangerous to human health and the environment. The quality and sophistication of these analyses have grown and very minute quantities of these insecticides or their residues can be analysed these days with a high degree of specify, precision and accuracy.

1.1 Inorganic and organ metal insecticides

The sequential organomentals and organometalloids insecticides are described in connection with the corresponding inorganic compounds. The highly toxic and recalcitrant compounds e.g. trichloro-bis-chlorophenyl ethane (DDT) and bis-chlorophenyl aqcetic acid (DDA) are formed unintentionally. The organic combination usually changes the absorption and distribution of a toxic metal and thus changes the emphasis of its effects, while the basic mode of action remains the same. The toxic effects of insecticides depend on the elements

that characterize it as inorganic or organmetal insecticides and on the specific properties of one form of the element or one of its components or merely on an inordinately high dosage. Some highly toxic elements such as iron, selenium, arsenic and fluorine are essential to normal development. The organometals and organometalloids are here described in connection with the corresponding inorganic compounds. Organic combination usually changes the absorption and distribution of a toxic metal and as a result changes the emphasis of its effects, but the basic mode of action remains the same.

The distinction between synthetic compounds and those of natural origin somewhat artificial. In practice, related compounds are assigned to one category or the other, depending on whether the particular compound of the group that was first known and used was of synthetic or of natural origin. For example, pyrethrum and later the naturally occurring pyrethrins were well known for years before the first synthetic parathyroid was made; as such, pyrethroid are thought of as variants of natural compounds, even though they have not been found in nature and are unlikely to occur.

1.2 Pyrethrum and related compounds

The insecticidal properties of pyrethrum flowers (chrysanthemum cinerarae- folum) have been recognized as insect powder since the middle of last century (McLaughlin 1973). In addition to their insect-killing activity, an attractive feature of the natural pyrethrins (pyrethrum) as insecticides was their lack of persistence in the environment and their rapid action whereby flying insects very quickly become incapacited and unable to fly. Prior the development of DDT, pyrethrum was a major insecticides for both domestic and agricultural use, despite its poor light stability. Development of synthetic pyrethroid with increased light stability and insecticidal activity allows it to be used as foliar insecticide while the natural pyrethrins are now used mainly as domestic insecticides.(Elliot, 1979).

1.3 Mode of Action

Pyrethrum and the synthetic pyrethroids are sodium channel toxins which, because of their remarkable potency and selection have found application in general pharmacology as well as toxicology (Lazdunski et al., 1985). Pyrethroids have a very high affinity for membrane sodium channels with dissociation constants of the order of $4x10^{-8}$ M (Sodeland,1985), and produce subtle changes in their function. By contrast, inexcitable cells are little affected by pyrethroids. The pyrethroids are thus referred to as open channel blockers.

1.4 Metabolism

The relative resistance of mammals to the pyrethriods is almost wholly attributable to their ability to hydrolyze the pyrethroids rapidly to their inactive acid and alcohol components, since direct injection into the mammalian CNS leads to susceptibility similar to that seen in insects (Lawrence and Casida, 1982). Metabolic disposal of the pyrethroids is very rapid (Gray et al., 1980), which means that toxicity is high by intravenous route, moderate by slower oral absorption, and often immeasurably moderate by slower oral absorption.

1.5 Poisoning syndromes

The pyrethroids are essentially functional toxins, producing their harmful effects largely secondarily, as a consequence of neuronal hyperexitability (Parker et al.1985). Despite this dependence on a relatively well-defined mode of action, the pyrethroids are capable of

generating a bewildering variety of effects in mammals and insects, which although showing some analogies with those produced by other sodium channel toxins (Gray, 1985; Lazdunski et al., 1985) and with DDT (Narahashi, 1986), have many unique characteristics (Ray, 1982b). Thus, toxicity of pyrethroids is divided into two groups Table 1. Type 1 pyrethroids produce the simplest poisoning syndrome and produce sodium tail currents with relatively short time constants (Wright et al., 1988). Poisoning closely resembles that produced by DDT involving a progressive development of fine whole-body tremor, exaggerated startle response, uncoordinated twitching of the dorsal muscles, hyperexcitability, and death (Ray, 1982b). The tremor is associated with a large increase in metabolic rate and leads to hyperthermia which, with metabolic exhaustion, is the usual cause of death. Respiration and blood pressure are well sustained, but plasma noradrenalin, lactate, and adrenaline are greatly increased (Cremer and Servile 1982). Type 1 effects are generated largely by action on the central nervous system, as shown by the good correlation between brain levels of cismethrin and tremor (White et al., 1976). In addition to these central effects, there is evidence for repetitive firing in sensory nerves (Staatz-Benson and Hosko, 1986).

Type I	**Intermediate**	**Type II**
Allethrin	Cyhenothrin	Cyfluthrin
Barthrin	Fenproponate	Cyhalothrin
Bioalethrin	Flucythrinate	Cypermethrin
Cismethrin		Deltamethrin
Fenfluthrin		Fenvalerate
Trans-fluorocyphenothrin		Cis-fluorocyphenothrin
Kadethrin		
Permethrin		
Phenothrin		
Pyrethrin I		
Pyrethrin II		
Resmethrin		
Tetramethrin		

Table 1. I Acute toxicity of pyrethroids (Wright et al., 1988; Forshow and Ray, 1990).

The type 11 pyrethroid produces a more complex poisoning syndrome and act on a wider range of tissues. They give sodium tail currents with relative longterm constants (Wright, et al., 1988). At lower doses more suble repetitive behavior is seen (Brodie and Aldridge, 1982). As with type I pyrethroids, the primary action is on the central nervous system, since symptoms correlate well with brain concentrations (Rickard and Brodie, 1985). As might be expected, both classes of parathyroid produce large increases in brain glucose utilization (Cremer et al. 1983). A final factor distinguishing type 11 pyrethroids is their ability to depress resting chloride conductance, thereby amplifying any sodium or calcium effects (Forshaw and Ray, 1990).

Intermediate signs representing a combination of type I and type 11 are produced by some pyrethroids. These appear to represent a true combination of the type I and 11 classes (Wright et al., 1983) and thus represent a transitional group. Evidence in support of this is given by measurement of the time constants of the sodium after potential produced by the

pyrethroids. Since pyrethroids appear to be essentially functional toxins, they produce few if any specific neuropathological effects.

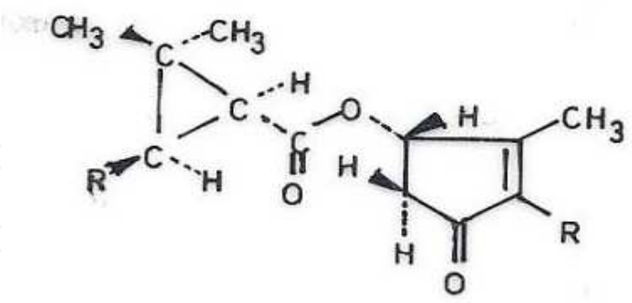

Fig. 1. Pyrethrins of the form.

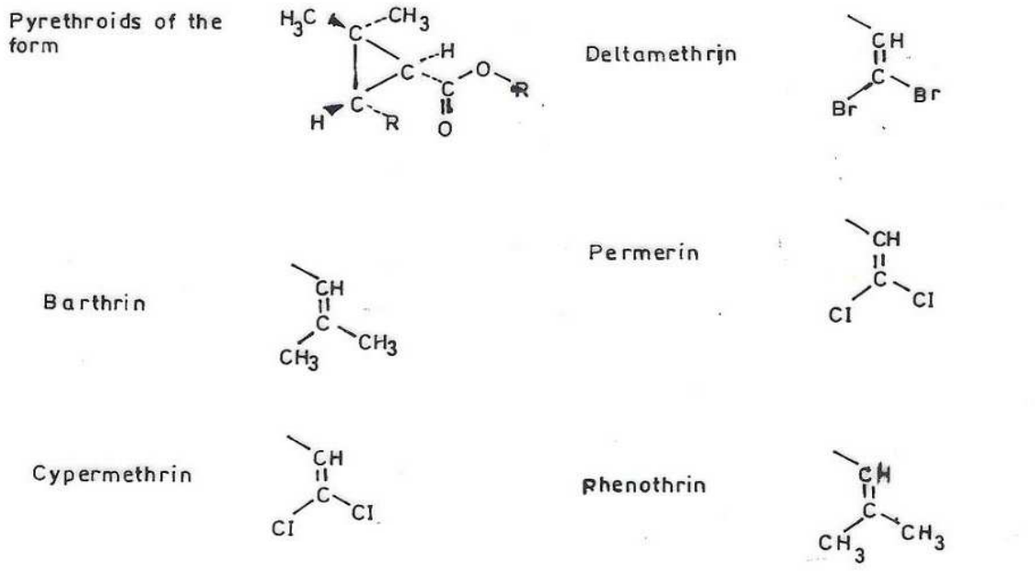

Fig. 2. Pyrethroids of the form.

1.6 Identity, properties and uses

The six known insecticidal active compounds in pyrethrum are esters of two acids and three alcohols. Insect powder made from "Dalmatian insect flower" (*Chrysanthemum cinerariaefolium*) is called pyrethrum powder or simply pyrethrum. The powder itself was formerly used as an insecticide, but now it is usually extracted. The six active ingredients are:

- Pyrethrin I – pyrethrolone ester of chrysanthemic acid
- Pyrethrin II- pyrethrolone ester of pyrethric acid
- Cinerin I- cinerolone ester of chrysanthemic acid
- Cinerin II- cinerolone ester of pyrethric acid
- Jasmolin I- jasmolone ester of chrysanthemic acid
- Jasmolin II- jasmolone ester of pyrethric acid

The six known insecticidally active compounds in pryrethrum are esters of two acids and three alcohols. Specifically, pyrethrins l is the pyrethrolone ester of chrysanthermic acid, -pyrethrin II is the pyrethrolone ester of pyrethria acid, cinerin l is the cinerolone ester of chrysanthemic acid, cinerin II is the cinerolone ester of pyrethric acid, jasmolin I is the jasmolone ester of chrysanthemic acid and jasmolin II is the jasmolone ester of pyrethric acid. There is much evidence indicating that the biological activity of these molecules depends on their configuration (Elliot, 1969, 1971).

The six active ingredients are known collectively as pyrethrins; those based on chrysanthemic acid are called pyrethrin I, and those based on pyrethric acid are called pyrethrins II. Pyrethrins, generally combined with a synergist, are used in sprays and aerosols against a wide range of flying and crawling insects. Usually about 0.5% active pyrethrum principles are formulated. They are equally effective for control of head lice and flea in dogs and cats.

1.7 Raid as insecticide

The insecticide 'Raid' belongs to a group of chemically stable pyrethrin, has widespread use in control of insects. Chemical stability, insecticide and organic phosphorus hydrocarbon have been shown to accumulate rapidly in tissues causing death and have profound effect on growth (Nebeker et al., 1994). Insecticide raid shows no observable effects on mortality and growth at lower test concentrations in rats. At higher concentration of 430 and 961 µg/g, survival decreased as concentration increases. In addition, mean total body weight of animals fed insecticides raid with concentrations of 430 and 961 µg/g were significantly decreased ($P<0.05$) than the controls. Conclusively, the higher the concentration of the insecticide Raid, the more hazardous it has on cell death (Achudume et al., 2008)(Table II).

Bioaccumulation factor of insecticide Raid was observed in lipids, up to three times that of the feed at the first concentration and gradually decreases as the concentrations increase (Table III), whereas accumulation factor in the muscle (0.7), brain (0.5), and liver (0.3) was about the indicated number times that of the feed. At higher concentration of 961 µg/g, bioaccumulation factor decreased in the lipid to 1.2 and 0.6 in the muscle, 0.03 in the brain, and 0.08 in the liver. Using the mean of insecticide in feed, the tissues accumulate the insecticide in the following ascending order: brain < liver < muscle < lipid. Similarly, Table III indicates the estimated detectable levels of toxicity in rat tissues exposed to the insecticide Raid. The brain shows mild decrease in toxicity of the enzymes glucose-6-phosphatase and lactic acid dehydrogenase, whereas significant decreases were noticeable in the muscle and liver (Achudume et al. 2008).

Long-term exposure of insecticide had been reported to result in systemic toxicity such that may impair the function of the nervous system and increase the risk of acute leukemia in children (Menegaux et al., 2006). Also, pesticides including organ phosphorus insecticides used against crawling and flying insects in homes have the potential of being carcinogens (Peter and Cherion, 2000). The adverse effect of insecticide Raid was demonstrated in a study by increase in alkaline phosphates activity in both plasma and liver which is a known measure of hepatic toxicity, and confirms "Raid" as a hepatotoxicant. The significant increase in alkaline phosphates activity (Table IV) may be due to hepatocellular necrosis which causes increase in permeability of cell membrane resulting in the release of this enzyme into the blood stream. The insecticide Raid significantly decreased reduced glutathione levels especially in the liver and this has implications for the ability of the animal to withstand oxidative stress. Studies have shown that GSH deficiency in cells is

associated with markedly decreased survival (Kohlmeier et al., 1997), thus, chemically stable lipid-soluble, organophosphorus insecticides are hazarddous to health through mechanisms including depletion of GSH (Menegaux et al., 2006).

Means SD concentrations of insecticide "Raid" in feed (Mg/g)	Mortality	Means: SD body weight (g)
0.00	Nil	135=5.4
25.0=2.4	Nil	135=21.7
54.0=9.5	Nil	132=2.9
108.2=12.5	Nil	129=3.2
216.2=14.6	Nil	128=19.8
430.0=20.2	1	118=20.5
961.2=70.5	2	116=5.3

Table 1. II mortality and growth of wistar rats exposed to different concentrations of "Raid".

Mean±SD Insecticide "Raid" in Feed (µg/g)	Raid Concentration in Wistar Rats (µg/g)[a] and Bioaccumulation Factor (BAF)			
	Lipid	Muscle	Brain	Liver
00.0	-	-	-	-
25.0±2.4	72.5± (2.9)	17.5(0.7)	12.5(0.5)	7.5(0.3)
54.0±9.2	86.4(1.6)	21.7(0.4)	16.4(0.3)	9.4(0.2)
108.2±12.5	172.8(1.6)	30.4(0.3)	19.5(0.2)	10.8(0.10)
216.2±14.6	280.8(1.3)	45.8(0.2)	22.9(0.1)	19.8(0.09)
430.0±20.6	324.0(0.8)	86.4(0.2)	25.8(0.06)	37.3(0.09)
961.2±70.5	1153.2(1.2)	576.6(0.6)	28.8(0.03)	76.9(0.08)

Table 1. III Tissue total raid concentrations and bioaccumulation factors (BAF) in wistar rats.

Raid concentrations In feed (µg/g)	Tissue	Alk pase activity µgml^{-min-L}	GSH level mg/ml	Glucose level mg/g liver
430±20.2	Control	0.08±0.04	0.18±0.02	0.96±0.04
	Plasma	0.06±0.09	0.15±0.6	0.90±0.04
	Control	0.08±0.04	0.18±0.02	0.94±0.01
	Liver	0.06±0.02*	0.15±0.01	1.05±0.12
961.2±70.5	Control	0.09±0.05	0.19±0.05	0.96±0.52
	Plasma	0.06±0.01	0.11±0.05	1.09±0.52
	Control	0.08±0.08	0.19±0.02	0.96±0.06
	Liver	0.05±0.08*	0.09±0.03*	1.66±0.04

Data values are mean±SD
*Statistically significant $p< 0.05$

Table 1. IV Effect of Raid concentrations in feed on hepatic enzyme activity, reduced glutathione and glucose levels.

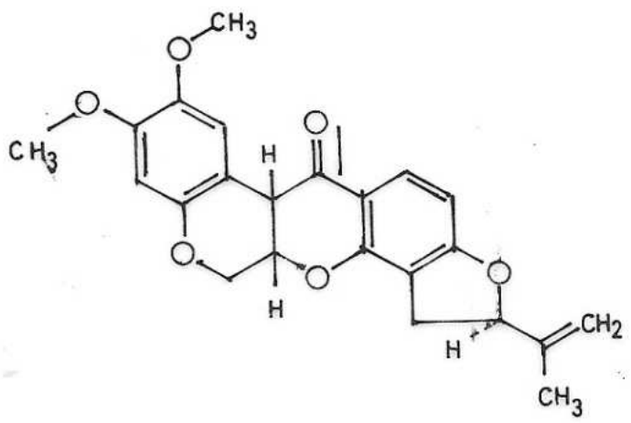

Fig. 3. Structure of rotenone.

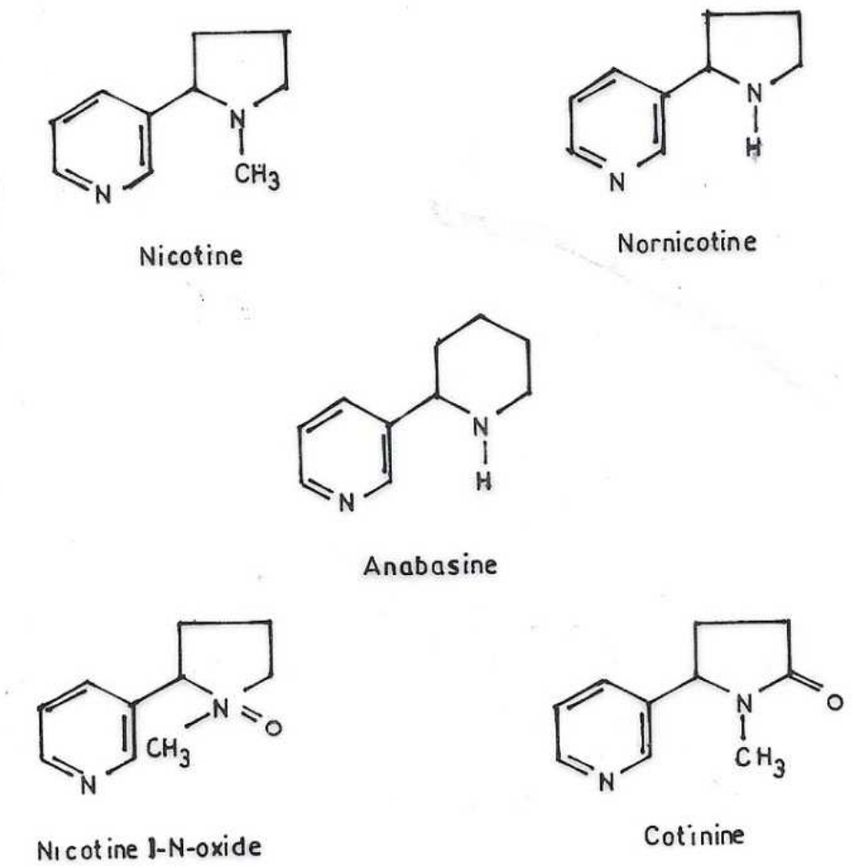

Fig. 4. Nicotine, narnicotine and anabasine with two important metabolites of nicotine.

Some other studies confirm that glutathione deficiency is associated with impaired survival in HIV disease (Herzenbery et al., 1997). Glutathione may be consumed by conjugation reaction, which mainly involve metabolism of zenobiotic agent. However, the principle mechanisms of hepatocyte glutathione turnover are known to be by cellular efflux (Sies et al., 1978). Glutathione reducatase is a known defense against oxidative stress, which in turn needs glutathione as co-factors. Catalase is an antioxidant enzyme which destroys H_2O_2 that can form a highly reactive radical in the presence of iron as catalyst (Gutter ridge, 1995).

Achudume et al., 2008 showed that bioaccumulation factor of insecticides raid was observed in lipid. Lipid peroxidation is a chemical mechanism capable of disrupting the structure and function of the biological membranes that occurs as a result of free radical attack on lipids. Some study confirms that insecticide raid increased lipid peroxidation, oxidative stress and hepatotoxicity due to reduced antioxidant system.

In addition, SOD is family of metalloid enzyme which is considered to be stress protein which decreases in response to oxidative stress (McCord, 1990). It is evident that decrease of SOD in the tissue is a confirmation of its protection from damage caused by insecticide Raid.

2. Classes of insecticides

- The classification of insecticides is done in several different ways (Hayes, 1982),(Heam 1973, Lehman, 1954, Martin and 1977).

- Systemic insecticides are incorporated by treated plants. Insect ingest the insecticide while feeding on the plants.
- Contact insecticides are toxic to insects by direct contact. Efficacy is often related to the quality of pesticide application in aerosols which often improve performance.
- Natural insecticides, such as pincotine, pyrethrum and neem extracts are from plants as defences against insects.
- Inorganic insecticides are manufactured with metals e.g. Heavy metals
- Organic insecticides are synthetic chemicals which comprise the largest numbers of pesticides available.

Insecticides are pesticides used to control insects many of these insecticides are very toxic to insects and many others are relatively harmless to other organism except fish.
Insecticides decompose readily so the residues do not accumulate on crops or in the soil. Insecticides include ovicides and larvicides used against the eggs and larvae of insects respectively.
The use of insecticides is believed to be one of the major factors behind the increase in agricultural productivity (McLaughlin,1973, van Emden and Pealall, 1996). Nearly all insecticides have the potential to significantly alter ecosystems; many are toxic to humans; and others are concentrated in the food chain (WHO 1962, 1972). Selected inorganic metals are discussed in the next section followed by individual insecticides organ metals.

2.1 Barium

Barium is an alkaline earth metal in the same group as magnesium, calcium, strontium and radium. It valence is two. All are water-and acid soluble compounds. They are poisonous. Barium carbonate is a rat poison. It is used in ceramics, paints, enamels, rubber and certain plastics.
Absorption, Distribution, Metabolism Excretion (ADME): Barium carbonate is highly insoluble in water. It is partially solubilized by acid in the stomach. The danger of the insecticide is through ingestion. Various barium compounds can cause pneumoconiosis. It is absorbed from gastrointestinal tracts of rat rapidly and completely. It is stored in bone and in other tissues (Hayes 1982, Castagnou et al 1957, Dencker et al., 1976, 83). Excretion takes place rapidly in urine and feces in 24hr (Bauer et al. 1956).
Mode of action: Barium stimulates striated cardiac and smooth muscle, regardless of the innervation.

2.2 Chromium

Chromium is a metal somewhat like iron and separated in the periodic table by manganese. Only hexavalent chromium compounds (chromates) are important as pesticides. They are also the most toxic. Chromate is absorbed by the lung (Baetjer et al., 1959), gastrointestinal tract and skin. It is widely distributed in the liver, kidney, bone and spleen (Mackenzie et al 1958). Acute poisoning may produce death rapidly through shock or renal tubular damage and uremia (Steffee and Baetjer 1965).

2.3 Mercury

Mercury is toxic no matter what its chemical combination. It is widely distributed in the environment, and traces of it occur in food, water and tissues even in the absence of occupational exposure. Inhaled mercury vapour diffuses across the alveolar regions of the

lung into the blood stream. Mercury vapour is a monatomic gas which is highly diffusible and lipid soluble (Berlin et al 1969a , Hush,1985). Once in the bloodstream mercury vapour enters the blood cells where it is oxidized to divalent inorganic mercury under the influence of catalase (Halbach and Clarkson 1978). Mercury is widely distributed with the highest concentrations in the kidney.

2.4 Thallium

Thallium stands between mercury and lead in the periodic table, and compounds of these metals show marked similarities. All of them may produce immediate local irritation followed by delayed effects in various organs, notable the nervous system. Thallium sulphate has been more widely used as pesticide than any other compound of thallium. It has produced many cases of poisoning and serves as good example of the toxicity of thallium generally (Lund, 1956b).

Thallium is easily absorbed by the skin as well as by the respiratory and the gastrointestinal tracts. Thallium accumulates in hair follicles and much less in those in the resting phase. Excretion is slow and is entirely by urine in humans but in rats via faeces (Barclay et al., 1953, Lund, 1956a)

2.5 Lead arsenate

Lead arsenate includes acid lead arsenate, dibasic lead arsenate, dilead orthoarsenate, diplumbic hydrogen arsenate, lead hydrogen arsenate and standard lead arsenate. Lead arsenate is used as an insecticide. it is used to control moths, leaf rollers and other chewing insects and in soil for the treatment of Japanese and Asian beetles in lawn. Absorption is generally via gastrointestinal. Dermal absorption is extremely small. Lead and arsenate are distributed separately in the body. lead is stored in highest concentration in the bone with much lower concentrations in soft tissues. Arsenic is stored in the liver and in some instances in the kidney at higher concentrations than those for lead (Fairhall and Miller, 1941. Lead is transferred to the fetus of animals humans (Heriuchi et al., 1959).

2.6 Antimony potassium tartrate

This compound serves as a poison in baits to control insects, especially thrips, and as an emetic in bait to control rodents. Ingestion of the compound usually leads to repeated vomiting. Excretion is mainly urinary (Fairhall and Hyslop, 1947).

2.7 Sodium selenate

Sodium selenate is an insecticide used in horticulture for control of mites, aphids and mealybugs. Various compounds of selenium are freely absorbed from the respiratory and gastrointestinal tracts. Dermal absorption is less important. Selenium is stored more in the liver, kidney, spleen, pancreas, heart and lung than in other organs (Underwood, 1977). Selenium is excreted chiefly in the urine but about 3-10% is metabolized and excreted by the lungs and through faecal excretion.

2.8 Sodium fluoride

Sodium fluoride is toxic to all forms of life. It has been used as an insecticide, rodenticide and herbicide and as fungicide for preservation of timber. Its toxicity to plants generally has

restricted its use as an insecticide to bait formulations (who, 1970). Sodium fluoride concentrates more in the plasma and liver and is excreted in urine.

3. Miscellaneous elements

3.1 Boric acid

Boric acid and borax have been used as an insecticide, both mainly for the control of cockroaches. Boric acid also is known as boracic acid and as orthoboric acid. Absorption from the gastrointestinal tract is rapid and virtually complete. Its peak concentration is in brain and less in other tissues. Boric acid is excreted unchanged in the urine (wong et al., 1964).

3.2 Insecticides derived from living organisms and other sources:

Different groups of insecticide; derived from living organisms are entirely unrelated chemically and pharmacologically. They range from relatively simple alkaloids such as nicotine, with a molecular weight of only 162.2, through proteinaceous poisons to virulent living organism. They range in toxicity from harmless and fragile pheromones, which are used as a chemical warfare agent.

The distinction between synthetic compounds and those derived from living organisms is somewhat artificial. In practice, related compounds are assigned to one category or the other, depending on whether the particular compound of the group that was first known and used was of synthetic or of natural origin. For example, pyrethrum and later the naturally occurring pyrethriums were well known for years before the first synthetic pyrethroid was made; as a result, pyrethroids are thought of as various of natural compounds, even though they have not been found in nature and are unlikely to occur. By contrast, synthetic sodium fluoroacetate acquired a reputation as a rodenticids and was explored as a synthetic insecticide before it was realized that the potassium salt is the active principal of a poisonous plant. Thus pyrethroids are discussed extensively.

Perhaps the only unifying feature of the diverse array of poisons derived from living organism is the popular view that "natural" substances are harmless. On this matter of safety, an expert committee of the world health organisation pointed out that "all the most poisonous materials so far know are, in fact, of natural origin" (WHO,1967).

3.3 Pyrethrum and related compounds

The insecticidal properties of pyrethrum flowers (genus chrysanthemum) have been recognized since the middle of 1st century, when commercial sale of "insect powder" from Dalmatian pyrethrum flower heads began (McLaughlin, 1973). In addition to their insect-killing activity, their lack of persistence in the environment and rapid "knock down" activity whereby flying insects become uncoordinated and unable to fly makes it very useful. Pyrethrum used to be a major insecticide for both domestic and agricultural use despite its poor light stability. Its usefulness was extended by introduction of piperonyl butoxide and other compounds as synergists, which greatly reduced the unit cost of crop treatment. Development of synthetic pyrethroids with increased stability and insecticidal activity (Elliot 1977) reduced the use of pyrethrum. However, natural pyrethrins are now used mainly as domestic insecticides, while the synthetic pyrethroids represented 20-25% of the world foliar insecticide market in 1983 (Herve's 85) and the proportion is increasing

steadily. Thousands of new synthetic pyrethroids have been synthesized, some showing complete divergence from the original pyrethrins (casida et al., 1973). Table 2.1 and Table 2.2.

4. Mode of action

Pyrethrum and the synthetic pyrethroid are sodium channel toxins which, because of their remarkable potency and selectivity, have found application in general toxicology (Lazdunski et al 1985). Their actions on the nerve membrane sodium channel are well understood. Pyrethroids have a very high affinity for membrane sodium channel, they have little effect on inactive sodium channels or close channels and produce subtle changes in their functions. After modification by prethroids, sodium channels continue in many of their normal functions, retaining their selectivity for sodium ions and link with membrane potential (Narahashi, 1986). The pyrethroids are thus known as open channel blockers. Detailed studies can be found in Narahishi 1986, Jacques et al., 1980, and Gray 1985.

4.1 Metabolism

The relative resistance of mammals to the pyrethroids is almost wholly attributable to their ability to hydrolyze the pyrethroids rapidly to their inactive acid and alcohol components, since direct injection into the mammalian CNS leads to susceptibility similar to that observed in insects (Lawrence and Casida, 1982). Some additional resistance of homoeothermic organisms can be attributed to the negative temperature coefficient of action of the pyrethroids (Van den Bercken et al., 1973) which are thus less toxic at mammalian body temperature but the major effect is metabolic.

The metabolic pathways for the breakdown of the pyrethroids vary little between mammalian species but vary somewhat with structure. This literature has been ably summarized by Leahy (1985), and further references to the metabolism of specific pyrethroids are given in the sections on individual compounds. Generally pyrethrum and allethrin are broken down mainly by oxidation, whereas for the other pyrethroids ester hydrolysis predominates. These reactions can take place in both liver and plasma and are followed by hydroxylation and conjugation to glucuronides or sulphates, which are then excreted in the urine (Gray 1985).

4.2 Individual insecticides

Other known insecticides pyrethroids under organophosphates are listed below only selective ones are discussed.

Allethrin	Permethrin
Bifenthrin	
Cyhalothrin, Lambda-cyhalothrin	
Cypermethrin	Phenothrin
Cyfluthrin	Prallethrin
Deltamethrin	Resmethrin
Ftofenprox	Tetramethrin
Fenvalerate	Transfluthrin

Table 4. I Other known insecticides.

4.3 Cypermethrin

Cypermethrin (R, S)-∞- cyano-3-pheno-xybenzyl-2, 2-dimethyl. There are eight isomeric forms. It was introduced commercially in 1977 as an emulsifiable concentrate to be used against a wide range of insect pest (Elliot, 1977).

4.4 Deltamethrin

Deltamethrin S-∞- cyano-3-phenoxybenzyl-(IR)-cis-3-(2, 2-dibromovinyl)2,2-dimethcyclo-propane carboxylate. It is a single isomer. It is used against a wide range of insect pests. It produces a typical type II motor symptom in mammals (Barnes and verschoyle, 1974. Metabolism of deltamethrin involves rapid ester cleavages and hydroxylation (Shono eta al; 1977).

4.5 Fenproponate

Fenproponate(∞-cyano-3-phenoxybenzyl-2,2,3,3-tetra-methylcyclopropanecarboxylate).
There are eight isomerism forms. Fenpropathrin is another common name, was first developed by sumitomo and commercialized in 1980 as an emulsifiable concentrate to be used against a wide range of insect pests. Fenproponate produces intermediate or mixed motor symptoms in mammals (Wright et al., 1988).

4.6 Fenvalerate

Fenvalerate (R,S)- ∞- cyano-3-phenoxy-benzyl (IR,IS)-2-(4-chlorophenyl)-3-methyl-1-butyrate. There are four isomeric forms. It should be noted that fenvalerate is not based on a cyclopropane ring structure. It was introduced commercially to be used against a wide range of insect pests fenvalrate produces typical type II motor symptoms in mammals (Verschoyle and Aldrige, 1980).

4.7 Phenothrin

Phenothrin (3-phenoxybenzy-(IR,IS)-cis,trans-3(2-methylprop-1-enyl)-2,2 dimethyicyclo-propane carboxylate). There are four isomeric forms. It is used as a domestic insecticide in a partially resolved mixture rich in the IR isomer (Sumithrin) and for grain protection. Phenothrin produces typical type 1 moto symptoms in mammals (Lawrence and Casida, 1982)

4.8 Rotenone and related materials

Rotenone-bearing plants have longed being used as a fish poison by many ancient different indigenous people, nut their use as an insecticide is probably more than a century old. Plants known to produce rotenone and other rotenoids belong to at least 68 species of the family Leguminosae, the same as that for peas and beans. The genera most exploited so far are Derris, native to southeast Asia, and lonchocarpus to south America (shepard 1951).
Rotenone and other active principles often occur chiefly in the roots of rotenone bearing plants but may be in the leaves (as in *Tephrosia vogeli*), seeds (as in *Milletia pachycarpa*), or bark (as in *Mundulea serica*).
Regardless of the genus or the particular part of the plant involved, the active constituents of rotenone-bearing plants may be extracted with ether or acetone as resin.
Rotenone is (2R,6a 5,12a 5)-1,2,6,6a,12,12a-hexahydro-2-isopropenyl-8,9-dimethoxy-chromeno (3,4-b) furo(2,3-h) chromen-6-one. Its structure is depicted in fig. 3. Although

rotenone generally is considered to be the active ingredient in all resins isolated, the other constituents show considerable insecticidal activity (Metcalf, 1955).
Rotenone is readily oxidized and racemized in the presence of light and the process is accelerated in alkaline solution (Cheng et al., 1972). It is active as a nonsytemic pesticide against a wide variety of insects, arachnids and molluscs. Its rapid photodecomposition means that it is active only for about 1 week on plants or 2-6 days in water and this limits its commercial use though still finds use as a domestic garden insecticide.
Rotenone is a highly potent mitochondrial poison, blocking NADH oxidation, this property dominates its actions in animals (Heikkila et al., 1985).
Rotenone is metabolized rather effectively by the liver in isolated rat liver mitochondria, the aerobic oxidation of pyruvate is almost completely inhibited by rotenone (Haley 1978).

4.9 Nicotine and related compounds

Three closely related compounds (nicotine, nornicotine and anabasine fig4) were commonly used as insecticides, although only the most potent, nicotine, is now used to any extent. Nicotine is usually obtained from the dried leaves of nicotiana tabacum, but it also occurs in *N. rustica* and *Duboisia*, another genus of the solanaceae, and in three other taxonomically diverse general, namely Asclepia (Asclepidaceae), Equisetaceae (Equisetaceae), and Lycopodium (Lycopodiaceae); Nicotine (S-3-(1-methyl pyrrolidin-2-yl) pyridine) is used as nicotine sulphate as a stomach poison for leaf eating insects (Haigh and Haigh 1980). Nicotine is rapidly absorbed from all mucosal surfaces, including those of the mouth, gastro-intestinal tract, and lung. Since nicotine readily forms salts in acid solution, its penetration through biological membranes is strongly pH dependent (Schievelbein, 1982).
The metabolism of nicotine is highly complex and reviewed by Gorrod and Jenner (1975) and schievelbein (1982). Metabolism mainly by cytochrome P.450 linked microsomal oxidative pathways in the liver. Cotinine (Fig4) is major metabolite, which then undergoes further oxidation. Nicotine stimulates the action of acetylcholine at nicotinic receptors in the central nervous system, autonomics ganglia and some pheripheral nerves. It central actions result in tremor and convulsions, stimulation and then depression of ventilation and induction of vomiting by a direct action on the medulla. Ventilation is stimulated by peripheral actions on the aortic and carotid chemoreceptors, and adrenal catecholamine. Secretion is increased at low doses. Heart rate and blood pressure are largely dominated by sympathetic effects and show increases compounded by adrenal catecholamines. The gastrointestinal tract is dominated by parasympathetic effects and shows hypersecretion followed by block as well as increased tone and peristalsis. Death is usually a result of block of neuromuscular transmission in the respiratory muscles or a consequence of seizures. In addition to its action on cholinergic transmission, nicotine can act at noncholinergic sites and also activate receptors on sensory nerve endings and vagal C fibers (Martin, 1986).
The carcinogenic potential of tobacco is well established, but there is debate about the role of nicotine, which, although probably not carcinogenic itself can be converted to carcinogens such as N'-nitrosonornicotine and 4-(methyl-nitrosamino)-1-(3-pyriyl)-1-butanone. The metabolites cotinine and nicotine 1-N-oxide are not carcinogenic although they do produce hyperplasia of the bladder epithelium (Hoffmann et al., 1985).

5. Living organisms as pesticides

The use of biological control agents has many potential advantages over chemical control, not least the possibility of high selectivity for the predators and other beneficial species. Several microorganisms or microbial products have been identified as potential insecticides (Miller et al, 1983). Most successful attempts have been directed against insects, as biological control of vertebrates has met with little success due to cross-infection problems. The world Health Organisation has investigated viruses, bacteria fungi and nematodes as potential insect control agents since all play a part in limiting the growth of natural insect populations.

5.1 Viral insecticides

Viral insecticides are still in the experimental stage but many are under investigation, as reviewed by Miller et al., 1983. Bacterial insecticides represent the largest and widest used group and reviewed by Burges (1982) and Lysenko(1985). All of those used are spore-formers, since the spores can be readily stored in dried form and applied by conventional means as wettable powders or dusts. Many form a crystalline toxin within the spore which enhances their pathogenicity to insects. The most widely used is *Bacillus thuringiensis*. A closely similar bacterium, *Bacillus papilliae* has been used against Japanese beetle. It has the advantage that once spores are introduced into the environment the bacterial population is sustained by reinfection of the insect hosts, but the disadvantage that spore production requires expensive in-vivo production using insect pupae and is now of declining importance. It is highly specific, does not infect vertebrates, and despite production of a crystal toxin is nontoxic to mammals by repeated oral administration (Burges, 1982).

5.2 Fungal insecticides

Fungal insecticides are commercially produced for a variety of specific applications. Their importance in controlling natural insect populations has been recognized since 1834, Aschersonia has been used to control Floridian white fly on citrus since the early 1900s. Fungi have the advantages of forming a stable population in the insect environment and are capable of infection through the insect cuticle, not by ingestion as bacteria. A disadvantage is their susceptibility to widely used fungicides. Examples include *Beauveria basiana* is marketed as Boverin and used against Colorado beetle and corn borer in Russia and China. *Metarhizium anisopliae* was used against a range of insects as metaquino. *Hirsutella thompsoni*, is used to control citrus rust in the united states as myear and *Vecticillium lecani* is used as vertalec or mycotal for aphid control in united kingdom. Some fungi such as *Beauveria bassiana* produce toxins which may be involved in their pathogenicity. *Culicinomyces clavosporus* and *lagenidium giganteum* are mosquito pathogens (Miller et al. 1983).

5.3 Nematate insecticides

Nematate insecticides have been isolated from mosquito larvae at low natural population densities. They are reared in vivo, which is expensive, and there some resistant mosquito population Nametodes are tolerant of many insecticides and insect growth regulators and can be used in combined malaria control programs and are rapidly broken down by human gastric juice (Gajana et. al; 1978).

6. Conclusion

Given the enthusiasm of the proponents of biological insect control and the limited role that these agents play in current pest control may be perhaps surprising. There are however, a number of difficulties in sustaining a usefully large population of the control agent on crops, or in the case of mosquitoes at the water surface, and in agriculture difficulties associated with the very high host specificity of some agents. More fundamental problems are the potential risk from replicating agents which can increase in the environment and the possibility of transfer of toxin encoding genes from invertebrate to vertebrate bacteria or viruses. It is clear, however, that current experience with biological control agents is very encouraging and that they can be expected to play an important part in integrated pest control programs in the future (Laird, 1985).

While animals as well as humans may be adversely affected mainly by ingestion of the active ingredients, the effect of propellant chemical cannot be ignored. Inflammatory activation might be an important mechanism underlying toxicity effects in the tissue (Mense et al., 2006). The role of propellant in the toxicity of insecticide Raid may not be cleared. A comprehensive assessment of the risk associated with environmental use of insecticide Raid was determined in various tissues as it affects the basal biochemical molecules of cells (Achudme et al., 2008).

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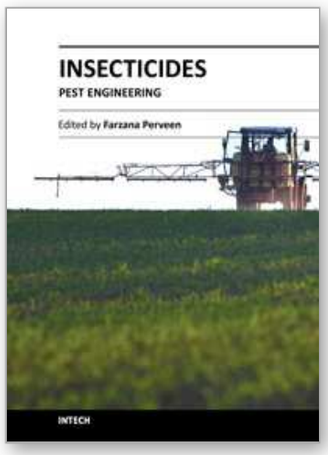

Insecticides - Pest Engineering

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This book is compiled of 24 Chapters divided into 4 Sections. Section A focuses on toxicity of organic and inorganic insecticides, organophosphorus insecticides, toxicity of fenitrothion and permethrin, and dichlorodiphenyltrichloroethane (DDT). Section B is dedicated to vector control using insecticides, biological control of mosquito larvae by Bacillus thuringiensis, metabolism of pyrethroids by mosquito cytochrome P40 susceptibility status of Aedes aegypti, etc. Section C describes bioactive natural products from sapindacea, management of potato pests, flower thrips, mango mealy bug, pear psylla, grapes pests, small fruit production, boll weevil and tsetse fly using insecticides. Section D provides information on insecticide resistance in natural population of malaria vector, role of Anopheles gambiae P450 cytochrome, genetic toxicological profile of carbofuran and pirimicarp carbamic insecticides, etc. The subject matter in this book should attract the reader's concern to support rational decisions regarding the use of pesticides.

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