Chapter 19
Pesticide-Mediated Toxicity in Modern Agricultural Practices

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Abstract Pesticides are, at the moment, man's main weapon against insect pests. Pesticides have been used heavily in almost all parts of the world. The average consumption of pesticides in India was only 3.2 g/ha in 1954–55. In 1075–76, this had increased to 4.4 kg/ha, and it keeps on increasing day by day. Proper use of pesticides increases farmers' incomes. Yields of most crops increase by 10–20% when pesticides are used. Most experts agree that removal of pesticides from crop protection would result in an immediate drop in food production. Discontinuation of all pesticide use would reduce the production of crops and livestock by 30% and would increase the prices of farm products by 50–70%. Phenomenal progress has been made in the development of insecticides. But their detrimental effects are numerous, including several acute and chronic illnesses in humans and worsening quality of the environment. Two types of contamination of the ecosystem are recognized: point- and nonpoint-sourcepollution. Pesticides are transported into the aquatic environment and the agricultural field as a nonpoint source most of the time and thereby reach all organisms and interfere in the food chain. So we need to supplement natural modern agricultural practices.

19.1 Introduction

Pesticides are chemicals used to control pests, destroy weeds, control microbes and harmful organisms that spoil agricultural commodities, and control parasites and vectors causing dangerous plant diseases. Pesticide toxicology is the qualitative and quantitative study of the adverse effects of pesticides on organisms.

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The adverse effects may include both lethality (i.e., mass mortality of organisms) and sublethal effects such as changes in growth, development, reproduction, survival, physiology, biochemistry, and behavior of the organisms. Pesticide toxicology is also concerned with the concentrations or quantities of pesticides that can be expected to occur in the environment, i.e., water, air, soil, and biota (Headley and Lewis 2015). Therefore, pesticide toxicology is the study of the transport, distribution, transformation, and ultimate fate of pesticides in the environment.

The average utilization of pesticides in India was only 3.2 g/ha in 1954–55. In 1975–76, this had increased to 4.4 kg/ha. Proper use of pesticide increases farmers' incomes and increases yields of most crops by 10–20%. Discontinuation of all pesticide use would reduce the production of crops and livestock by 30% and would increase the prices of farm products by 50–70%. Extensive use of pesticides leaves residues in the environment, and these residues are responsible for disturbance of the ecological balance and health hazards. Damage to the environment by pesticide residue results through two types of interaction: interaction with biotic components and interaction with abiotic components.

The first type of interaction is responsible for causing imbalance in nature and serious health hazards to various forms of animal life (i.e., creation of new pests and establishment of resistant populations). In addition, pesticide residues also cause damage to the natural environment. Persistent insecticides are known to destroy, directly or indirectly, various phytoplankton and zooplankton, affecting the entire interdependent evolutionary ladder because the lower animals are involved in energy transfer to high trophic levels. Once the pesticide enters into the soil, the pesticide affects the microbial population in the soil and affects nutrient availability to plants (Thompson 2017).

Some organochlorine pesticides get deposited in adipose tissues of animals and are concentrated in higher animals through the biomagnification process. The second kind of interaction is a direct physical or chemical reaction with nonbiological components of the environment, such as soil and water. Such a direct reaction may directly or indirectly modify the utility of these components. This type of problem is common with soil pesticides, which change the soil pH, thus affecting microbial growth and micronutrient levels (Sun et al. 2017).

Insecticides were in use before man learned to write, and early stone tablets are said to have referred to red squall as a rat poison. By 1960, tobacco was being used for control of lace bug on pear trees. The modern use of insecticides dates from 1867, when Paris green was first used for Colorado potato beetle control. The discovery of the insecticidal properties of DDT (dichlorodiphenyltrichloroethane) in 1939 revolutionized our concept of insecticides and of insect control. Other families of chemicals are being investigated for possible insecticidal activity. Many pesticides used for agricultural purposes drain into water, causing hazardous effects on aquatic ecosystems. Pesticides travel from agricultural areas to potential catchment areas by two methods: horizontal travel during rain through runoff water and underground travel during rain or at other times (Majid et al. 2016).

On the other hand, two types of contamination of aquatic ecosystems are recognized as point- and nonpoint-source pollution (Murthy 1986). Point sources refer to

a single source of contamination, such as effluent from a pesticide-manufacturing or formulation plant. Nonpoint sources refer to contamination of a widespread and diffuse nature, as in the case of agricultural runoff (Ouyang et al. 2016).

19.2 Sources of Pesticides

19.2.1 Nonpoint Sources

19.2.1.1 Agricultural Land Sources

Sediment resulting from soil erosion is regarded as the largest pollutant that affects water quality. More than land use, land form characteristics such as soil texture, soil type (mineral or organic), surface geology, slope, and drainage density—and also soil chemistry—influence the degree of nonpoint-source pollution. Pesticides adsorbed to clay particles are transported considerable distances. When land is exposed to erosive forces such as rain and surface runoff, the hazard of aquatic pollution is much greater (Yang et al. 2016).

Pesticide Mobility

Volatilization Pesticide volatilization can be defined as the movement of pesticide vapors through the air. People such as farm workers and bystanders can be exposed to pesticides by breathing these vapors after an application has occurred. Volatilization is considered different from pesticide movement by spray drift, erosion, or windblown dust/soil particles (Astoviza et al. 2016). Exposure to pesticide vapors due to postapplication volatilization generally occurs from three main sources. Temperature increases the rate of volatilization. The rate of evaporation of pesticides from the soils is related to the vapor pressure, water content of the soil, adsorption of the chemicals by the soil, and soil characteristics. Wind and water carry pesticide residues in the gaseous phase, in solution, or as a soil–pesticide complexes.

Adsorption Soil moisture and organic matter content determine the extent of adsorption. The amount of insecticide leaching is inversely related to the organic matter content.

19.2.1.2 Urban Land Sources

Urban areas that have construction sites with exposed soil contribute the highest amounts of nonpoint pollution loads.

Sewage

Urban sewage is another nonpoint source of pesticide residues in the aquatic environment.

Used Pesticide Containers

Careless handling of spraying equipment and used containers transfers pesticide residues to the aquatic environment, as in the case of washing and cleaning of spraying equipment in various water sources.

19.2.2 Point Sources

19.2.2.1 Effluent from Pesticide-Manufacturing Plants

In the aquatic environment the effluent from pesticide-manufacturing plants contains between a few and 100 lb/day of pesticides.

19.2.2.2 Joint Action of Pesticide Mixtures

In nature, organisms are exposed not to a single toxicant but often to numerous chemicals present either in considerable amounts or in traces. For discussion we take the interaction between any two or a group of toxicants. It was realized that interactions between chemicals may either increase or decrease the overall effect, i.e., the resulting action is more or less than the simple combined effect, or there may not be any interaction at all between the two. Further, the situation is complicated when one of the compounds is not toxic by itself but, in its presence, the other compound may act as an enhancer.

The joint action is defined as additive, more than additive, or antagonistic. If half of the concentration of toxicant A needed to produce a given response and half of the concentration of toxicant B needed to produce the same response together produce that response, the action of toxicants A and B is additive. If this combination causes more than that response, the resultant action is more than additive, and if it causes less than that response, then it is less than additive, antagonistic, or without interaction.

Sprague (1970) explained that supposing that one toxic unit of toxicant A or B is needed to produce a particular response, then if the same response is produced by combinations of toxicants A and B at concentrations that meet within the square, the toxicants are helping each other and the resultant action is called “joint action.” Akobundu et al. (1975) used the term *synergism* when both components are active and the combined effects are more than the sum of the two individual effects.

19.2.2.3 Enhancement

Enhancement occurs if one of the components is inactive but somehow increases the effect of the other. More recently, models have been proposed that take into account the underlying components of joint action, including the effect of one substance on another with respect to the site of action, elimination, metabolism, competition for receptor sites, speed of action, and interaction.

19.3 Observations in Fish

Various reports on the combined toxicity of pesticides to fish may be examined from the above discussion.

19.3.1 Pre-exposure

It is not clear whether pre-exposure (also called preconditioning) confers any protection for the organism. Exposure of spot to 0.01 and OL.ug/L toxaphene for 5 months made the fish more susceptible to the action of the same compound, as evidenced by the increased acute toxicity of toxaphene (lowered LC50 values) to pre-exposed fish in comparison with those that were not previously exposed. Previous exposure of goldfish to alkyl benzene sulfonate (ABS) made them more susceptible to the toxic effects of dieldrin and DDT. Higher mortality was recorded in the group that was pre-exposed to ABS.

19.3.2 Observations on the Joint Toxicity of Pesticides in Fish

The possible interaction between methyl parathion and the cotton defoliant DEF (S,S,S-tributyl phosphorotrithioate) was tested. Gambusias were exposed to 0.5 mg/L DEF or 5 mg/L methyl parathion, or both. There was 8% mortality of the fish exposed to ethyl parathion, 89% mortality of the fish exposed to both the toxicant and no mortality of the fish exposed to both the toxicant, and no mortality of fish exposed to DEF. It was concluded that there was potentiation of the toxicity of DEF by methyl parathion.

19.4 Pesticide-Induced Morpho-anatomical Changes

19.4.1 Morphological Changes

Many morphological changes have been reported following the exposure of fish either to high concentrations for a short time or to sublethal concentrations for longer periods. Some of these changes may apparently change the body coloration or may be an erosion of something, distortion of vision, etc. Exposure of *Oreochromis mossambicus* to any pesticides for 15 days resulted in darkening of the skin, formation of a brown short on the forehead, swelling of the eyes, and erosion of the fin margins.

Many pesticides, irrespective of the group to which they belong, have been reported to induce vertebral damage and skeletal deformities. Mehrle et al. (1982) suggested that a decrease in vertebral mechanical properties is an early indicator of contaminant stress. They also suggested that containment-induced competition for vitamin C between collagen metabolism in bone and microsomal mixed function oxidase would cause vertebral damage. The competition for vitamin C would decrease the water bodies. Commonly, fish are subjected to long-term stress arising from exposure to sublethal concentrations.

In the long run, these sublethal concentrations may prove more deleterious than lethal concentrations because subtle and small effects on aquatic organisms, especially fish, may alter their behavior, vitamin C, and collagen content of the bone with a concomitant increase in the ratio of bone minerals to collagen, resulting in increased fragility of the bone.

19.5 Pesticide-Induced Behavioral Changes

19.5.1 General Behavioral Changes

The behavior or activities of an organism represent the final integrated result of a diversity of biochemical and physiological processes. Thus, single behavioral parameters are generally more comprehensive than a physiological or biochemical parameter. In addition, behavior patterns are known to be highly sensitive to changes in the steady state of an organism. Hence, an alteration in the behavior of the organism due to stress is a diagnostic tool for identifying the ecological effects of the release of a toxicant into the environment.

Sherer (1975) considered that behavioral tests may sometimes ascertain lower thresholds than physiological techniques because the response results from an intact, integrated, functional system. Bull and McInerney (1974) studied behavioral changes in salmon exposed to several concentrations of fenitrothion. Various behavioral changes occurred with zhrobl exposure. Marked nipping, etc., was noticed. On the other hand, comfort behaviors such as thrusts, coughs, etc., increased with

increasing concentrations of the toxicant. Some exposed individuals were unable to maintain position and were swept downstream. Some freshwater fish exposed to phosphate esters became hypersensitive to disturbance, fed leers, and showed impaired swimming ability (Pyle and Ford 2017).

19.5.2 Avoidance Responses

Fish can sometimes sense the presence of xenobiotic chemicals in water and tend to avoid them. In an experiment to test the avoidance reactions of *O. mossambicus* to pesticides, the fish avoided DDT, endrin, and malathion. Most of the fish preferred on with mediate lethal concentration of 0.1 mg/L which is a sort of a sensory trap for the high.

19.5.3 Swimming and Hypersensitivity

Swimming activity of fish in sublethal concentrations of pesticides may be greater than that of fish in pesticide-free water. When the high was exposed to lethal concentrations of pesticides, the fish exhibited erratic movements and sometimes lost balance. The number of opercula beats increased with increasing concentrations of pesticides (Banaee et al. 2014).

19.5.4 Schooling Behaviors

Weis and Weis (1982) reported that pesticides had a marked effect on the schooling behavior of fish. Increased uptake of the toxicant resulted in disruption of schooling behavior. This disrupted schooling resulted in increased predation of the fish. Apart from disruption of schooling behavior, pesticides affect the spawning migration of fish.

19.5.5 Coughing

Coughing in fish has been described as an interruption of the normal ventilating cycle, with more rapid expansion and contraction of the filament and opercular cavities, and serves the purposes of clearing the gills of accumulated debris and coughing up concentrations of pesticides. The frequency of cough was proportional to the sublethal toxicant concentration and predicted chronic effects at levels near the maximum acceptable toxicant concentration. Thus, the cough response is a useful and sensitive tool for evaluation of the quantity of toxicant in the riotous medium.

19.6 Pesticide-Induced Biochemical Changes

Many pesticides have been reported to produce a number of biochemical changers in fish at both lethal and sublethal levels. Changes in ionic concentrations, organic constituents, enough the activity, and osmoregulation in fish have been attributed to pesticides (Melo et al. 2015).

19.6.1 Effects of Pesticides on Osmotic and Ionic Regulation

Osmoregulation is the process by which the total electrolyte content and water volume in an organism are held relatively constant. It is well known that any chemical that affects osmoregulation in marine or freshwater fish will alter the percentage of water in tissue. Changes in osmoregulation in fish exposed to a toxicant are generally determined by measuring the blood sodium chloride or total osmolality.

Researchers have studied the effects of pesticides on ion balances in freshwater and marine fish. In seawater-adapted fish, exposure to a wide variety of different pesticides induces osmoregulation dysfunction, which is reflected in a rise in plasma NaCl and osmolality, while the opposite occurs in freshwater-adapted fish. Evidently a large majority of pollutants affect osmoregulation or ionic regulation (Vernberg and Vernberg 2013).

19.7 Effects of Pesticides on Feeding Energetics

Intake of food is an important factor governing various physiological functions such as growth and reproduction. Growth represents a net outcome of a series of processes such as digestion, assimilation, metabolism, and excretion. In this context, proposed a hypothetical model for average partitioning of dietary energy in carnivorous fish. In this hypothetical partitioning of dietary energy, in a fish with an intake of 100 calories, 20 calories are lost as feces and 7 calories as metro groups waste. The cost of digestion and assimilation of the food is 14 calories for splenetic dynamic fountain, leaving net energy of 59 calories to be used by the organism. This net energy is partitioned between maintenance activity, metabolism, and growth. Energy metabolism and growth compete for the net energy. Thus, if metabolism is elevated, growth will be limited unless the intake of food is increased. Certain environmental pollutants reduce the feeding rate, growth, and food conversion efficiency of fish (Narra 2016; Zheng et al. 2016 and Hoseini et al. 2016). Effects of pesticides on feeding energetics have been studied in certain commercially freshwater fish such as *O. mossambicus*, *Channa punctata*, *Mystus vittatus*, *Cyprinus carpio*, *Lepidocephalichthys thermalis*, etc.

Many pesticides, irrespective of the group to which they belong, have been reported to reduce the feeding rate, absorption rate, growth rate, metabolic rate, absorption efficiency, and food conversion efficiency. Intake of food may be affected by the effect of pesticides on the appetite of the fish. Reduction of the growth rate and conversion efficiency may be due to utilization of body organic reserve substances for the purpose of energy requirements during stress conditions (Giaquinto et al. 2017).

19.8 Effects of Pesticides on Liver Function

The liver is of key importance when considering the effects of polluting chemicals on fish. It is the primary organ for biotransformation of organic xenobiotics and probably also for excretion of household chemicals. Since many of these organic chemicals tend to accumulate to high concentrations in the liver, liver cells are exposed to higher levels of harmful chemicals than may be present in the environment or in other organs of the fish. The liver serves a number of functions related to other physiological activities such as endocrine control of reproduction and interconversion of foodstuffs.

Therefore, the potential effects of xenobiotics on the liver are numerous but often seen only by industry. Chemical laboratories have long used measurement of blood plasma as a diagnostic tool for assessing liver damage in humans. Alkaline phosphatase, glutamic oxaloacetic transaminase, and glutamic pyruvic transaminase are normally found in low concentrations in blood, but their concentrations may be increased by leakage from damaged liver cells, increasing their catalytic activity. Liver cells are particularly rich in transaminase because this organic is the major one for interconversion of foodstuffs.

When a hepatotoxic pesticide is administered to either freshwater or marine fish, increased plasma glutamic oxaloacetic transaminase and glutamic pyruvic transaminase from samies is glutamic usually observed. (Sabra and Mehana 2015).

19.8.1 The Liver Has a Wide Range of Functions

Some pesticides affect liver function, which is usually reflected in a change in blood chemistry. For example, DDT and enduring caused a 50% elevation in serum cholesterol when mullet were exposed to insecticides for 96th. This elevation in cholesterol could be explained by either enhanced production by the liver or inhibition of cholesterol excretion. Regarding the first possibility, harmful cortisol inhibits cholesterol synthesis and this hormone becomes elevated in fish under stress.

It is well known that the liver is responsible for elimination, in bile, of breakdown products, primarily bilirubin. If this process is impaired, the condition of jaundice may develop and be reflected in a rise in the bilirubin concentration in

plasma. Exposure of fish to organic pesticides produced a marked elevation in plasma concentrations. Concomitant inhibition of the enzyme uridine diphosphate (UDP) glucuronosyltransferase (UGT) in liver tissue was also noted. Since this enzyme is required for the normal configation reactions by which bilirubin is prepared for exertion, its inhibition may be the primary cause of jaundice.

Ascorbic acid is better known as vitamin C. It plays a number of known biochemical roles. It is essential for normal synthesis of collagen. Other less well-studied functions include involvement in the metabolism of adrenal steroids by the liver, oxidation of tyrosine and, in conjunction with superoxide dismutase and catalase, helping to prevent build-up of free radicals in cells. Mayer et al. (1977) exposed catfish to various phenols in the diet for 150 days. They noted dose-dependent depression of backbone collagen and fin deformities.

However, when ascorbate was added to the diet at several different doses, the extent of collagen depression and the incidence of deformities in the fish exposed to toxaphene were decreased roughly in proportion to the ascorbate dose. Toxaphene was shown to cause a depletion of vitamin C in the spine but not in the liver. Vitamin C supplementation also reduced the whole-body residues of toxaphene (Singh and Singh 2017). This suggested that this molecule is used by the liver in detoxifying toxaphene but at the expense of collagen. The depletion of tissue ascorbic acid during chronic exposure to pollutants may depend on the ability of the species of fish to synthesize this vitamin. For example, a week of exposure to pesticide (monocrotophos) caused a 45% loss of liver ascorbic acid in *Oreochromis mossambicus*.

It was also observed that liver ascorbic acid was not affected much by natural stressors such as temperature and salinity. Thus, measurements of this trace nutrient in the liver may have potential for specifically indicating chemical pollution. From the stoned point of the fish, depletion of ascorbic acid can result in pathological conditions including delayed wound healing, anemia, skin lesions, fin necrosis, etc. (Thomas et al. 1990; Hilton et al. 1977).

19.9 Development of Resistance

When an insecticide is used to control a pest, not all members of the population against which it is used are killed by the toxic material. Some survive. To control the surviving members, a higher concentration of insecticide would be required, and a stage may be reached when the insecticide is totally ineffective against the pest.

According to Georghion and Taylor (1977), the number of species of insects and acarines in which resistant strains have been reported has increased from in 1908 to 364 in 1975. Agriculturists are compelled to use higher doses and more frequent applications to kill the same numbers of pests. This leads to disruption of the ecosystem. Some strains of insects and acarines have developed resistance to arsenic, DDT, and other chlorinated hydrocarbons, followed by organophosphates (OPs), carbamates and, more recently, pyrethroids and all compounds available commercially for pest control (Naqqash et al. 2016).

The development of resistance has been termed the most serious problem for modern pest control. According to the World Health Organization (1976), resistance is probably the biggest single obstacle in the struggle against vector-borne disease and is mainly responsible for preventing malaria eradication in many countries. Resistance has been reported not only to the most recent insecticides but also to insect growth regulators, chemosterilants, and even biological agents.

Reports in the literature show that an insect's development of resistance to an insecticide does not occur until after the insect has been exposed to the chemical for several generations. It took 25 generations of intensive selection for insects to develop resistance to IC compounds. DDT resistance develops after an initial latent period of several generations before it increases steeply, whereas cyclodiene resistance develops without delay (Elhag 2016). The resistance to carbamate takes the same time as the resistance but develops a little faster when it builds on a basis of OP resistance.

The rate of development of resistance in an unexposed population is initially very low. But as the frequency of major genes for resistance in the surviving population is gradually increased, the insect becomes better organized genetically to exist in the contaminated environment. The more intense the selection pressure, the more rapid the development of resistance, provided that the number of survivors is large enough to maintain genetic variability. The persistence of the resistance genes prevents introduction of insecticides against populations that have apparently reverted to full susceptibility as a result of release of pressure from insecticides.

Several ecological factors may influence the development of resistance to insecticides in a population. It may depend on the relative isolation of populations from each other or the degree of exchange of genetic material, and variations of ecological tissue factors such as the season, size, growth, and generations per year. The reproductive potential of the population may influence the development of resistance. Behavioral factors also influence the development of resistance. Several species of insects are known to have inherited ability to detect the pressure of specific insecticides and escape before taking up lethal quantities.

19.9.1 Types of Resistance

There are three types of resistance:

1. Cross-resistance
2. Multiple resistance
3. Multiplicate resistance

The term *cross-resistance* refers to resistance of a strain of insects to compounds other than the selecting agent, due to the same biochemical mechanism.

Multiple resistance occurs when two or more distinct mechanisms are present together, each protecting against different possessing.

When two or more mechanisms coexist in the same organism and protect it against the same position, the animal is considered to possess *multiplicate resistance*. Such resistance usually results from simultaneous or consecutive use of several insecticides. In countries where many different insecticides have been used organist houses, resistance may be both multiple and multiplicate. Insects with a high level of resistance to chlorinated hydrocarbons may exhibit a high level of cross-resistance to carbamates. Where resistance to an OP is already present, exposure to an alternative OP may result in development of resistance to the new compound.

19.9.2 Mechanisms of Resistance

Considerable information is available regarding the biochemistry, physiology, and genetics of resistance of some species of arthropods. Sawicki (1979) classifies the resistance mechanism into three types:

1. Delayed entry of the toxicant due to factors such as decreased permeability of the cuticle
2. Decreased sensitivity of the site of action
3. Increased detoxification of the insecticide or decreased activation by metabolic processes

19.9.3 Mechanisms of Resistance to Organochlorine Compounds

It is a well-known fact that chlorinated insecticides are highly toxic because of their effects on the nervous system and they may also interfere with various metabolic processes in animals. One mechanism for DDT resistance was found to be metabolic conversion to DDE (dichlorodiphenyldichloroethylene) of DDT dehychlorinase. A gene responsible for this process is situated on chromosome II in the housefly. Some resistant strains of insects have increased detoxification of DDT while others exhibit absorption of the compound. Lindone is metabolized in insect tissues, but the mechanism is not clear.

Dehydrochlorination is involved in these processes. In the house fully agency responsible for slower penetration of DDT is located on chromosome III, which also carries the gene responsible for resistance to knockdown. The gene responsible for conversion of both DDT and DDE to the more easily excreted polar metabolic sites is situated on the fifth chromosome in the housefly.

The housefly also possesses other types of resistance against DDT. Knockdown resistance in the housefly is believed to be result of decreased nerve sensitivity,

which causes resistance to DDT and pyrethroids. Changes in the ability to metabolize glucose may be a factor in some cases of insect resistance to chlorinated insecticides (Zhu et al. 2016).

The mechanism of cyclodiene resistance is not well studied. Some researchers agree that resistance to insecticides does not involve reduced penetration, increased metabolism, or enhanced excretion of insecticides when compound with the same processing die—susceptible insects (Schaefer and Sun 1967) believe that they did in resistance in house—these may be due to insensitivity at the neuromuscular receptor site.

19.9.4 Organophosphate Compounds

The biochemical mechanism for the toxicity of OP compounds was first established in mammals. Now it is known that metabolism of these compounds in mammals and insects is the same. The mammalian liver and the insect's body fat resemble one another biochemically in several aspects. Though insects possess a variety of mechanisms resisting the toxic effects of insecticides, an important mechanism is enhanced detoxification. OP poisoning in insects is also known to occur through inhibition of acetylcholine esterase. In a few cases the property of resistance is due to the possession of acetylcholinesterase, which is insensitive to OP and carbamate insecticides. Slower penetration and increased metabolism of the chemical are responsible for resistance to OP compounds in larvae of the mosquito *Culex* spp. and not the insensitivity of acetylcholinesterase to OP (Wu et al. 2016).

19.9.4.1 Three Factors Control or Modify Resistance to Organophosphate Compounds

These factors are:

1. Decreased penetration through the cuticle
2. Detoxification mechanisms
3. Decrease in the sensitivity of cholinesterase

Among these factors, increased detoxification is believed to be the main cause of insect resistance to OPs. Genetic studies show that resistance to OP compounds is high by complicated though it may be caused by a single gene. In *Drosophila* spp., the main OP resistance gene is called "a" because of its association with reduced aliesterase activity located on chromosome IV. Three different detoxifying mechanisms in the parathion resistant house this are controlled by different genes on chromosome II. Delayed penetration is many resistant strains of house these is controlled by the gene "Pen" on chromosome III.

19.9.5 Carbamates

These are active anticholinesterases, which cause characteristic span atoms of cholinergic stimulation similar to those caused by OP insecticides. In house by the OP oxidative mechanisms may be responsible for resistance. The occurrence of high levels of oxidases in the microsomes of carbonate resistant house this supports this theory. Resistance to carbamate insecticides in the housefly has sometimes been attributed to a single gene and sometimes to polygenic systems. Plapp (1970) concluded that in the housefly this resistance to carbamate insecticides is due to the interaction of genes located on chromosomes II, III, and IV.

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