CHAPTER-3

3.0 BIOCIDAL ACTIVITY OF INSECTICIDES

More than 70% population of world depends upon agriculture sector for their survival but unfortunately agricultural crops are facing huge reduction in their growth, production, and quality on regular basis (Oerke, 2006). In order to overcome the problem of yield reduction farmers started to use insecticides as one of the best control strategy (Martins et al., 2014). Protection of crops from injurious insect pests heavily relies on the use of various insecticides since many years (Hussain et al., 2012). Use of insecticides is dominant among all the pest management tactics due to their efficacy and quick action against different insect pests. Insecticide use also enhances the yield and quality of produce which give high returns to producer (Karar et al., 2012). Despite controlling crop pests, insecticides are also tool to control various vector borne diseases by killing the insect vectors and carriers such as houseflies, mosquitoes, sand flies and bedbugs (Kaur & Sandhu, 2008). There are two main categories of insecticides; natural and synthetic. Synthetic insecticides replaced the natural insecticides due to their quick killing action since World War II (Karar et al., 2013). The first used synthetic insecticide was DDT from organochlorins group (Abbas et al., 2014b). Later, the phenomenon of biomagnification and highly persistence character of organochlorins necessitated the development and launching of other insecticide groups including organophosphates (1960s), carbamates (1970s), and Pyrethroids (1980s) (Aktar et al., 2009). After the discovery of these conventional insecticides, new chemistry insecticides with novel mode of action were also discovered in 1990s (Ferré & Van Rie, 2002). The insecticide use in insects brings various morphological, physiological; biochemical and molecular changes in insect body that result in mortality in most of the cases (Ferré & Van Rie, 2002). The focus of this chapter is to highlight the various physiological, physical, biochemical and molecular pathways in insects that are responsible for the toxic effects of the insecticides.

3.1 Conventional Insecticides

3.1.1 Organochlorins

Organochlorins (OCs) are chlorinated hydrocarbons and contain the elements carbon and chlorine (Baird C, 2004). They have been used extensively from the 1940s through the 1960s. Some of the most widely used insecticides in this group are dichloro-diphenyl-trichloroethane (DDT),dieldrin, aldrin, heptachlor, chlordane, lindane, endosulfan, and toxaphene (Coats et al., 1990; Calle et al., 2002). DDT was first developed and most famous pesticide from this group and was used during World War II for control of lice and mosquitoes to eliminate typhus and malaria, respectively (Snedeker, 2001). Insecticides of this group target the peripheral nervous system. OCs are the axonic poisons, interrupt normal nerve impulse transmission along axon. These poisons after reaching the axon act on the sodium gates and keep them open for longer period of time. This results in constant leakage of sodium (Na) ions through the nerve membrane, creating a state of action potential continuously without any resting potential. This causes triggers (repetitive discharges) and insects show the symptoms of hyper excitability, paralysis, convulsions and finally death occurs (Hardman et al., 2007) (Figure 3.1). Death occurs only if doses are lethal. The use of OCs is banned now in majority of countries because of significant residual effects, high persistence in the environment and also due to phenomenon of biomagnification (decompose slowly in their environment and become part of animal's fatty tissues; they also stay longer in environment and food web after their application (Jayaraj et al., 2016).

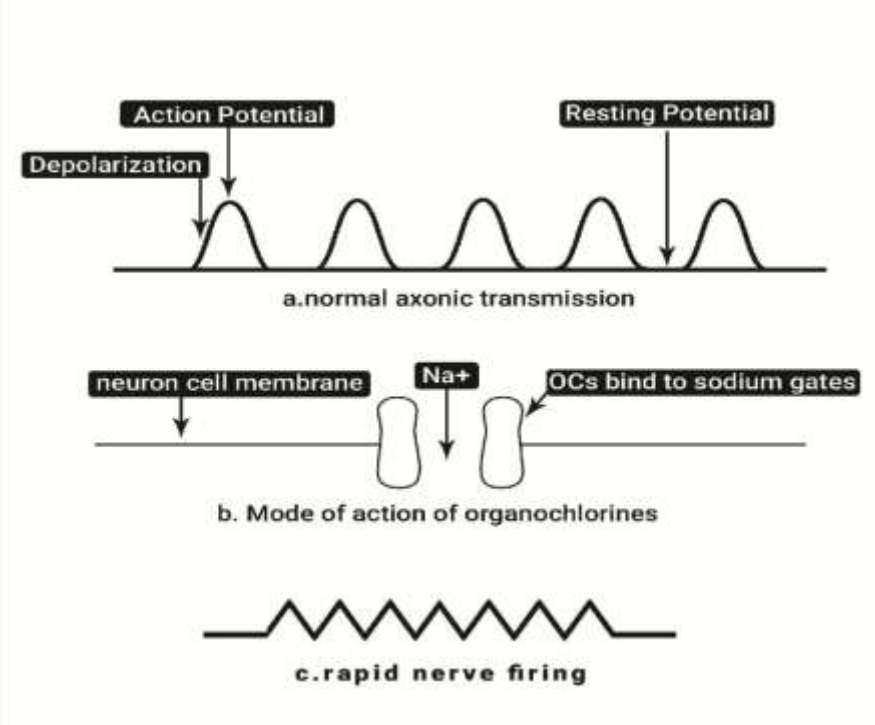

Figure 3.1 Interruption of axonic transmission by organochlorines

3.1.2 Organophosphates

Insecticides of this group are used most widely throughout the world. Above 100 compounds of this group are used commercially for the control of various insect pests although over one lac compounds have been tested for insecticidal properties. Some of the main insecticides of this group are triazophos, profenofos, chlorpyrifos, fenitrothion, quinalphos and malathion (Greene & Pohanish, 2005). Organophosphates (OPs) have a wide range of pest control applications as contact, systemic, stomach and fumigant insecticides. These insecticides show their toxic effect at insect synapse. The biocidal effects of the OPs are almost entirely due to the inhibition of acetylcholinesterase (AchE) in the nervous system at post-synaptic neuron, inhibiting the breakdown of neurotransmitter acetylcholine (Ach) resulting in respiratory, myocardial and neuromuscular transmission impairment (Smegal, 2000). Inhibition of AchE by OPs is irreversible and the process is called phosphorylation (Hwang, 2014) (Figure 3.2). The poisoned insect shows the symptoms of restlessness, hyper excitability, tremors, convulsion, paralysis and ultimately death occur due to rapid nerve firing (Costa et al., 2006).

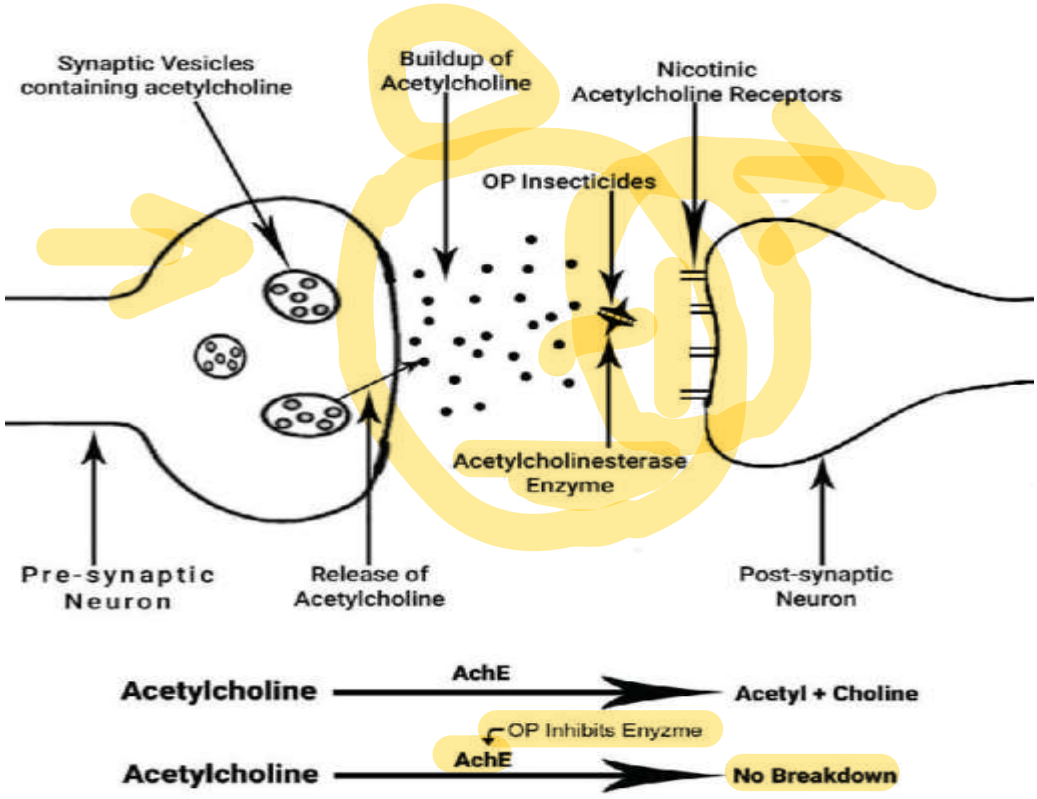

Figure 3.2 OP insecticides interrupt synaptic transmission

3.1.3 Carbamates

The derivatives of carbamic acid (NH_2COOH) are called carbamates. These are organic in nature. Chemicals from this group find applications in crop protection as insecticides, herbicides and fungicides. Some important representatives of carbamates are carbofuran, carbaryl, methomyl, oxamyl and propoxur. They act as stomach as well as contact poisons. Some carbamates are also used as fumigants. Insecticidal activity of carbamates is due to hydrogen (H) and methyl group (-CH3) in the place of R_2 and R_1, respectively (Figure 3.3). They are also called synaptic poisons. Their insect killing action is based on reversible AChE inactivation, the process is called carbamylation. Insect suffer same kind of symptoms as discussed for organophosphates (Colovic et al., 2013).

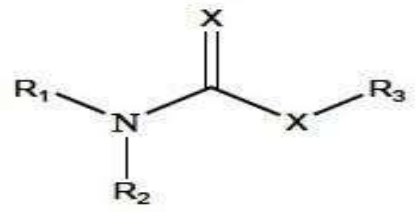

Figure 3.3 General chemical structure of biologically active carbamates

3.1.4 Pyrethroids

Pyrethroid insecticides interact with the insect voltage sensitive sodium channel (VSSC), causing death. Synthetic pyrethroids are derivatives of Pyrethrins. There are two types of

pyrethroids; Type I and Type II that differ in their structure and biocidal activity in insects. Allethrin, resmethrin, tetramethrin, bioresmethrin, and permethrin are Type I pyrethroids while deltamethrin, cypermethrin, fenvalerate, cyfluthrin, and lambda-cyhalothrin are Type II pyrethroids (Sumita & Aichi, 2016). Pyrethroids gain entry into insect body through contact and act as knockdown agents. Pyrethroids which are used as aerosols gain entry into insect body through spiracles and provide rapid knockdown action by acting on the nervous system. Similar to organochlorins, pyrethroids are also known as axonic poisons. In neurons, these insecticides bind to the voltage gated sodium channels (proteins) in nerve cell membrane, and delay the closing of sodium ion channels. It results in continuous nerve stimulation and multiple action potentials. Bursts of repetitive discharges and action potentials with increased amplitude are the characteristic effects of Type 1 pyrethroids. However, action potentials with lower amplitude are due to Type II pyrethroids. This significant depolarization of neuron membrane leads to total blocking of neural activity (Söderlund & Ihre, 1985) (Figure 3.4). Poisoned insects lose control over their nervous system, display lack of coordinated movement. Hyper-excited insects display the symptoms of frequent tremors, convulsions, paralysis and ultimately die. Many pyrethroid insecticides find applications as household products in the world due to their rapid knockdown effect and low toxicity profile to mammals.

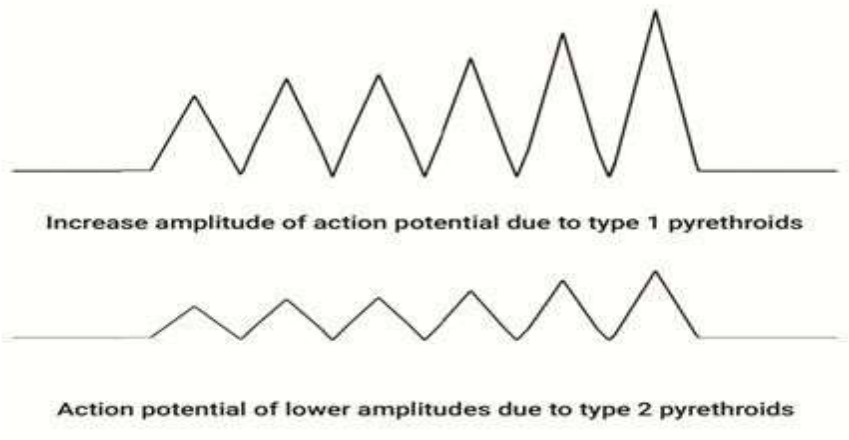

Figure 3.4 Effect of pyrethroid insecticides in insects

3.2 New Chemical Insecticides

3.2.1 Neonicotinoids

Efforts were made to synthesize the insecticides which have high binding affinity for insect nicotinic acetylcholine receptors (nAChR), and this ultimately resulted in the development of nicotinyl insecticides as one of a new group (Elbert et al., 1998). This is the group of insecticides with novel chemistries. Other names of this group are nicotinoids, neonicotinyls and chloronicotines. Insecticides of this group are imidacloprid, acetamiprid, thiamethoxam, clothianidin, nitenpyram, dinotefuran, thiacloprid, and nithiazine (Elbert et al., 2008). These insecticides are highly recommended to be used against various sucking insect pests such as thrips, jassids, mealybugs, dusky cotton bugs, aphids, planthoppers; and well fitted in multiple IPM programs due to their mild effect on some bio-control agents and honeybees (Ishaaya et al., 2007). These insecticides are systemic in nature and translocate throughout the plant tissues. These properties make these insecticides effective for many pests for longer period of time. These are also used in small quantities for seed treatment (Jeschke et al., 2011). These insecticides act in the insect central nervous system (CNS), bind to nicotinic acetylcholine receptors (nAChRs), agonizes their action and hinder nerve impulse transmission as a result of a depolarizing effect (Elbert et al., 2008). Irreversible blocking of nicotinergic acetylcholine receptors may also occur due to these insecticides (Figure 3.5).

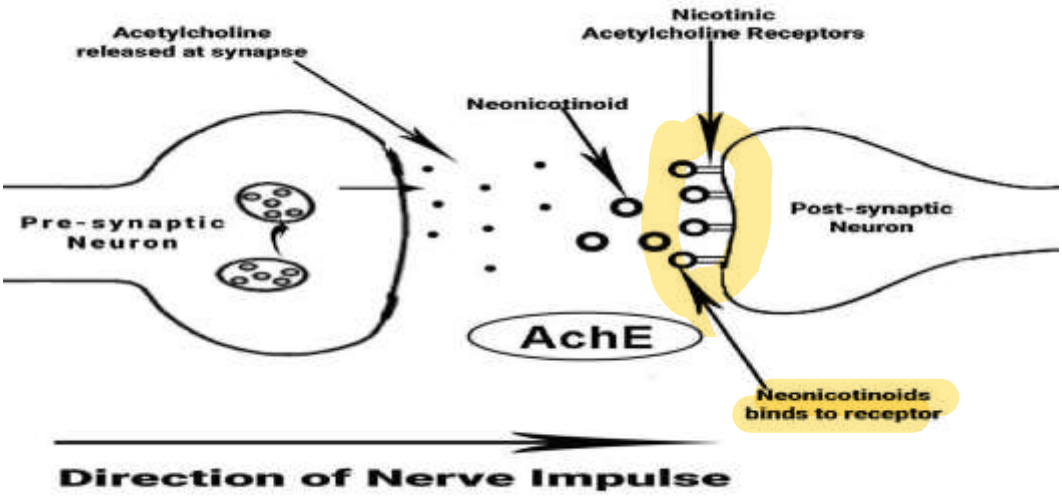

Figure 3.5 Biocidal mechanism of neonicotinoids

3.2.2 Oxadiazines

Oxadiazine contain single insecticide, indoxacarb that was discovered by E.I. DuPont Co. and is exploited for crop protection (McCann & Johnston, 1992). This insecticide gives promising control of different insects belonging to order Lepidoptera, Homoptera and Coleoptera and also possess low mammalian toxicity with reduced pesticide risk (Jeffery et al., 2000). Indoxacarb reaches the insect target site either through cuticle or via ingestion. It is a pro-insecticide and biologically activated during metabolism. The biocidal activity of indoxacarb in various insects is due to phenomenon of bio-activation. In this process, the indoxacarb act as pro-insecticides but after entering into insect body undergoes metabolism by esterase and amidase enzymes which ultimately produces an N-decarbomethoxylated metabolite. This active metabolite binds to the voltage gated sodium ions channels; blocks them and preventing sodium ions movement in to nerve cells, finally insect paralysis and death occur (Wing et al., 2000; Wing et al., 2010) (Figure 3.6). Potency of N-decarbomethoxylated metabolite is forty times higher as compared to pure indoxacarb in blocking the sodium ions channels (Silver et al., 2010; Wing et al., 2010). Feeding cessation, loss of nerve function, paralysis and death are the outcomes of indoxacarb poisoning. Feeding cessation occurs almost immediately after ingestion or absorption of indoxacarb even though it may take several days for insects to die (Wing et al., 2010). Indoxacarb was evaluated against the European earwig (*Forficula auricularia* Linnaeus), and was found to be an effective contact toxicant with residual activity on substrates commonly encountered in urban environments. Within 16 h of being directly sprayed with indoxacarb, ≥90% of earwigs were either ataxic, moribund, or dead, and 100% displayed these symptoms of severe intoxication at 1day. Brief exposure (5 min or 1 h) to dried residues on either a porous (pine wood) or non-porous (ceramic tile) substrate also was sufficient to cause severe intoxication of earwigs within 1 day. In all bioassays, indoxacarb-treated earwigs showed no signs of recovery during the 21 day observation period. In outdoor urban habitats, intoxicated earwigs would be more vulnerable to desiccation, predation, or pathogens leading to higher mortality than in a laboratory setting (Jones & Bryant, 2012).

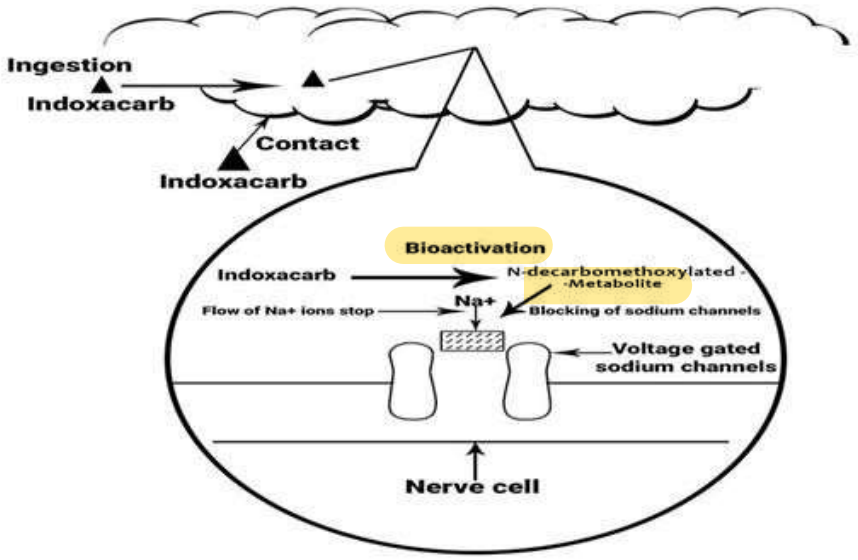

Figure 3.6 Mode of action of indoxacarb

3.2.3 Spinosyns

Spinosyns family contain two important insecticides spinosad and spinetoram with broad activity spectrum such as against crop pests that cause extensive damage and also kill many important external insect parasites of humans, livestock and companion animals. Spinosyns are macrocyclic lactones and were derived from actinomycete soil bacterium *Saccharopolyspora spinosa* Mertz & Yao (Sparks et al., 1998). Spinosad contains spinosyn A and D, with spinosyn A as a major component. Spinosad is toxic to insects both by contact and ingestion. Spinosad acts on insect nervous system and kills the insects in quite unique and different way when compared to other insecticides (Sparks et al., 1998), It has two different target sites, first one is the nicotinic acetylcholine receptor while gamma amino butyric acid (GABA) receptor the secondary site of attack (Salgado, 1997). Insects treated with spinosad result in excitation of the insect nervous system, which further causes involuntary muscle contractions, tremors, and paralysis and finally death (Duke et al., 2010). In the United States, spinosad has been registered for more than 180 crops and in over 35 countries for the control of leafminers, beetles, lepidopteran insects, and thrips (Zhao et al., 2002). The effects of spinosad on tobacco beetles at different life stages were investigated by feeding tobacco beetles with tobacco leaves and flours containing different doses of spinosad and the larvae of tobacco beetles were treated with spinosad solution of different concentrations. The results indicated that the contact and stomach toxicities of spinosad to 5-instar larvae were weak, however at the concentrations of 5 and 10 mg/kg, spinosad could completely inhibit the hatching of tobacco beetle eggs and reproduction of adult tobacco beetle (Mei & Ling, 2009).

3.2.4 Avermectins

Avermectins are compounds of biological origin and are obtained during fermentation process by soil actinomycete bacterium, *Streptomyces avermitilis* (Burg et al., 1979). The important representatives of this class are abamectin and emamectin benzoate and are known for insecticidal and miticidal activity, and are known as macrocyclic lactones (Dybas, 1989). These compounds act on the GABA and/or glutamate receptors as chloride channel agonist at neuromuscular junction and cause the permanent activation (opening) of chloride channels. These actions stimulate an influx of chloride ions into neurons which results in disruption of nerve impulses and complete loss of cell function (Figure 3.7). Insects are paralyzed and stop their feeding activity, which leads to death. These compounds cause maximum mortality in mites and insects in approximately 4 days. Although, avermectins are not known for quick knock down effect but shortly after their ingestion feeding is ceased due to rapid paralysis and feeding damage to crops is reduced significantly.

Arthropods intake avermectins both by ingestion and contact, however, primary route of uptake is ingestion whereby arthropods accumulate a lethal dose (Turner & Schaeffer, 1989). Avermectins also possess translaminar activity thus providing a relatively prolonged residual activity (MacConnell et al., 1989). Abamectin has biocidal activity against some mite species such as eriophyid mites (citrus rust mite) and tetranychid mites (two spotted spider mite) (Dybas, 1989). Among insects, the abamectin has been proved highly toxic to diamondback moth *Plutella xylostella* (Linnaeus), tobacco budworm *Heliothis virescens* (Fabricius), tomato pinworm *Keiferia lycopersicella* (Walsingham), tobacco hornworm *Manduca sexta* (Linnaeus), serpentine Leafminer *Liriomyza trifolii* (Burgess) and Colorado potato beetle *Leptinotarsa decemlineata* Say (Dybas, 1989). It is also effective in controlling the vegetable Leafminer *Liriomyza sativae* Blanchard (Reitz, 2013). Insecticidal activity spectrum of emamectin benzoate is much broader as compared to abamectin. Emamectin benzoate is recommended at low use rates in field crops and vegetables for the control of different lepidopteran pests for instance *H. virescens*, beet armyworm *Spodoptera exigua* (Hübner), *P. xylostella*, and cabbage looper *Trichoplusia ni* (Hübner) (Ishaaya et al., 2002). It has been studied that spray of emamectin with concentration of 25 mg a. i /liter in a cotton field resulted more than 90% suppression in larval population of *H. armigera* up to 28 days after treatment. It is also effective insecticide against the western flower thrips, *Frankliniella occidentalis* Pergande both under laboratory and under both field and laboratory conditions. Its activity for adult insect stages was greater than 10-fold as compared to abamectin. Emamectin also exhibits a significant activity against whitefly *Bemisia tabaci* (Gennadius) under laboratory conditions (Ishaaya et al., 2002).

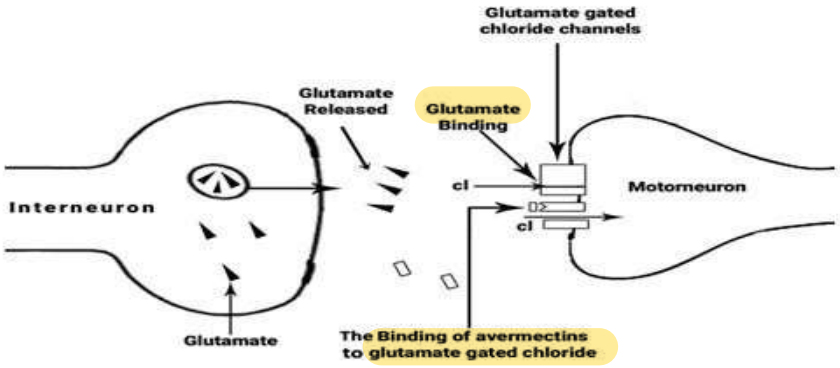

Figure 3.7 Mode of action of avermectins

3.2.5 Anthranilic diamides

Anthranilic diamides are insecticides with unique modes of action from IRAC group 28 and contain chlorantraniliprole and cyantraniliprole as major active ingredients (Lai & Su, 2011; Wang & Wu, 2012). These insecticides bind to the ryanodine receptors (RyRs) at insect muscles and cause the continuous opening of calcium ion (Ca^{2+}) channels, releasing stored calcium from the sarcoendoplasmic reticulum (Figure 3.8). The uncontrolled release and depletion of calcium from muscle cells stops further muscle contraction and insect dies in 72 hours due to feeding cessation, regurgitation, general lethargy, and muscle paralysis (Wang & Wu, 2012) Chlorantraniliprole (®) has excellent insecticidal activity against many pests of Hemiptera, Isoptera, Coleoptera, Diptera and Lepidoptera and it is the first generation ryanodine receptor insecticide from this novel class (Lahm et al., 2005; Sattelle et al., 2008). Cyantraniliprole is a second-generation insecticide of this class with systemic and translaminar action against wide range of chewing and sucking insect pests (Sattelle et al., 2008). Chlorantraniliprole has been shown to cease feeding in *S. exigua*, corn earworm, *Helicoverpa zea* (Boddie), *T. ni* and *P. xylostella* within 25.3, 20.3, 23.4 and 15.4 min, respectively, after larval exposure to chlorantraniliprole treated at the rate of 167 mg a.i/L (Hannig et al., 2009). Exposure of tobacco thrips, *Frankliniella fusca* (Hinds) and *F. occidentalis* to cyantraniliprole treated plants at the rate of 4.41 mg a.i per plant has also reduced feeding (Asner et al., 2011). Up to a 50% reduction in Asian citrus psyllids *Diaphorina citri* Kuwayama feeding was also recorded following cyantraniliprole treatment (Tiwari & Stelinski, 2013).

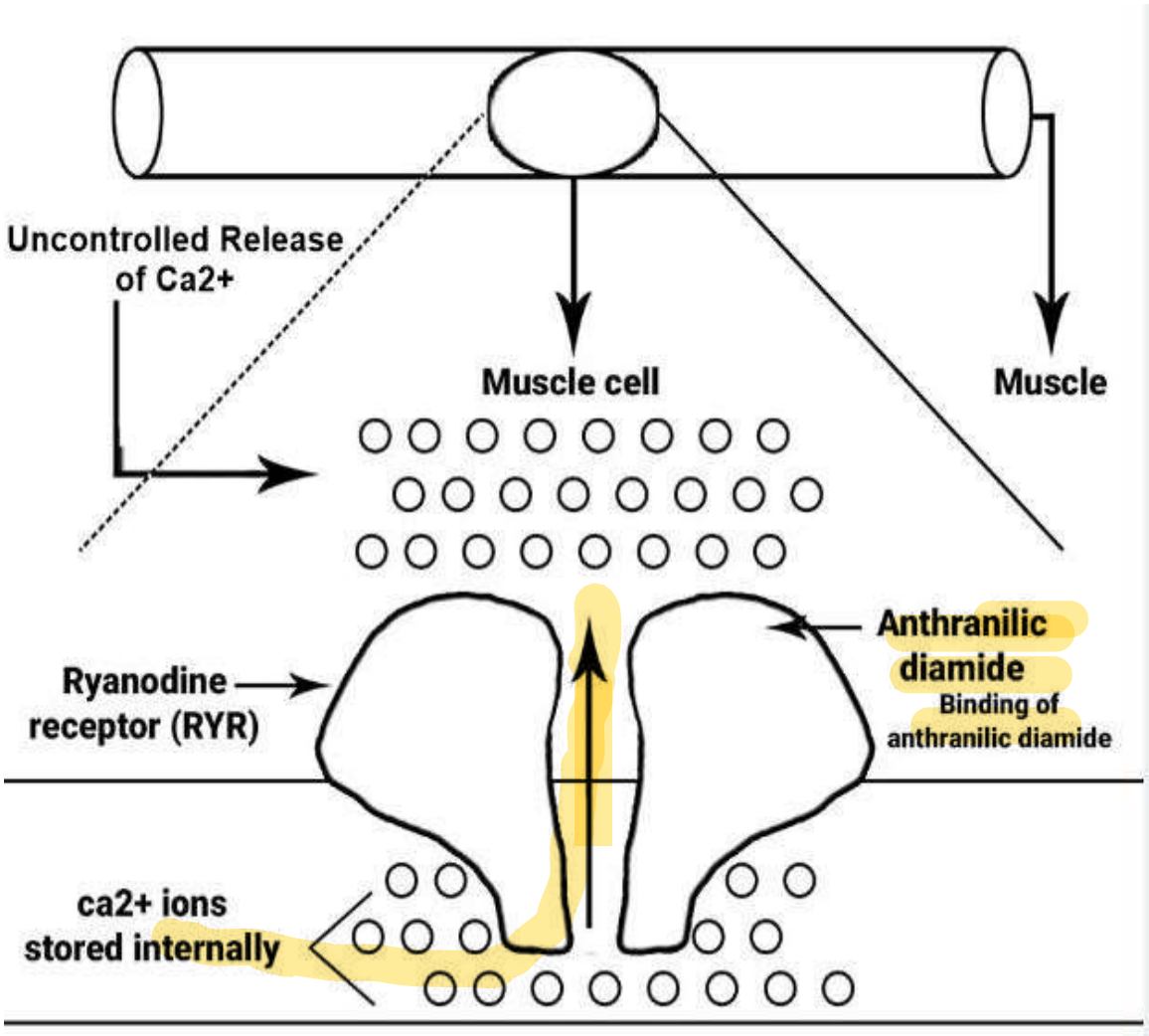

Figure 3.8 Biocidal mechanism of chlorantraniliprole (Rynaxypyr®)

3.2.6 Phenylpyrazoles

Phenylpyrazoles have been developed to manage insect pests of field crops and public health importance. This group contains two insecticides Ethiprol and Fipronil, the latter is widely used. Fipronil has biocidal activity against insects of the order Hemiptera, Lepidoptera, Thysanoptera, Coleoptera, Diptera and other pests especially rice borers (Abbas et al., 2014a; Abbas et al., 2014b). For toxicity, the main function of this product is to cause stomach poisoning as well as contact and systemic poisoning. Fipronil is noncompetitive blocker and inhibits the chloride ion flow by targeting the gamma-amino butyric acid receptor (GABAR), primary site of action, in the central nervous system. It is antagonistic in action and prevents the inhibitory effect of neurotransmitter, the GABA, and ultimately causes the hyper excitation of central nervous system (CNS) (Cole & Engeström, 1993). In cockroach neurons, recently fipronil has been found to potently block glutamate-activated chloride channels (Zhao et al., 2004). Being systemic, fipronil translocate throughout all plant tissues making it toxic to any insects (and potentially other organisms) that feed upon the plant. This protects the plant from direct damage by herbivorous (mainly sap feeding) insects and indirectly from damage by plant viruses that are transmitted by insects (Simon-Delso et al., 2015).

3.2.7 Pyrroles

Pyrroles are broad-spectrum insecticides, which show contact and stomach toxicity (N'Guessan et al., 2007). Pyrroles are pro-insecticides that are transformed into the toxic form by mixed function oxidases (MFOs) within the body of an insect. The activated Pyrroles after reaching mitochondria express their biochemical action by uncoupling oxidative phosphorylation. These insecticides impair the mitochondrial ability to produce ATP by disrupting the proton gradient across mitochondrial membranes. This causes the disruption of respiratory pathways, cell death and ultimately affected pest dies (Hunt & Treacy, 1998) (Figure 3.9). The important active ingredients of this group are cyanopyrrole and chlorfenapyr. Laboratory assays have shown that cyanopyrrole and chlorfenapyr possess potent, broad-spectrum biocidal activity against many species of Lepidoptera, Coleoptera, Thysanoptera and Acarina. Field trials conducted with cyanopyrrole at the dose 60-400 g/ha have been proved very effective for the control of more than 70 species of phytophagous insects and mites in different crops (Miller & Borden, 1990).

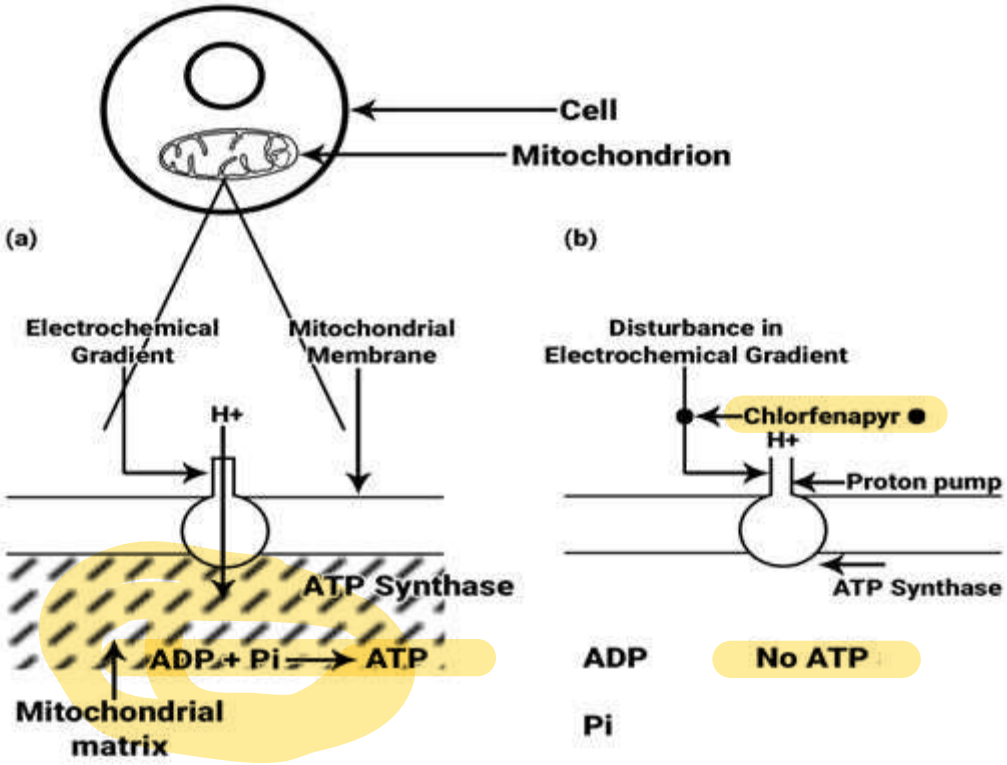

Figure 3.9 (a) Normal ATP synthesis in insects (b) ATP synthesis inhibited due to chlorfenapyr poisoning in insects

3.2.8 Thiourea insecticides

This group has single active ingredient called diafenthiuron. Diafenthiuron is broad-spectrum insecticide and is reported to be effective against all sucking pests in cotton against whiteflies, aphids and thrips in tomato, whiteflies and leaf hoppers in brinjal, and is also effective against diamondback moth (DBM) in vegetables. It also acts as acaricide. Contact or stomach action of this insecticide kills the larvae, nymphs and adults. It also possesses ovicidal properties. Pests hidden in the plant canopy or below the leaves surfaces are also controlled by this insecticide due to its trans-laminar action. Its vapor phase action enables the farmers to apply this insecticide in larger fields as well as in dense cropping system. Diafenthiuron is pro-insecticide and after entering the insect body or by sunlight converted to more toxic form called carbodiimide (Farlow et al., 1991). Hence biocidal activity of thiourea insecticides is due to carbodiimide. This active compound targets the insect mitochondria and inhibits the process of oxidative phosphorylation and mitochondrial ATP synthesis by binding with ATPase, which ultimately disturbs the insect

respiratory system (von Ruden & Neher, 1993) (Figure 3.10). Insects become immobile and paralyzed, death occurs in 3-4 days after initial exposure.

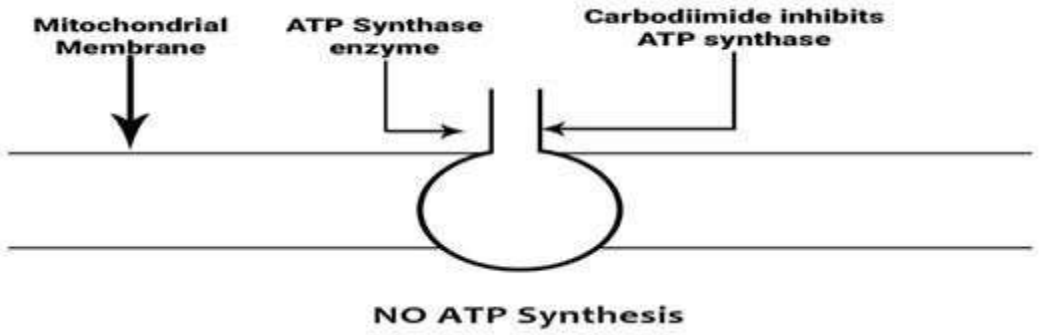

Figure 3.10 Biocidal activity of thiourea insecticide

3.2.9 Ketoenoles

This is the novel group of insecticides. It has two important active ingredients spiromesifen and spirotetramat. Spiromesifen is a derivative of spirocyclic tetronic acid, acts as non-systemic insecticide/ acaricide. It is mainly active against whiteflies following foliar applications, and as an acaricide possess highly active against *Tetranychus spp*. in many cropping systems such as cotton, vegetables, and ornamentals (Nauen & Lauder, 2002). This insecticide enters the arthropod body by direct feeding or as a result of contact and inhibits lipid biosynthesis in insects and mites by preventing the formation of fatty acids and their derivatives. Ultimately, it inhibits development in egg and immature stages and also reduces fecundity of adult female (Nauen & Lauder, 2002). Spirotetramat is a derivative of spirocyclictetramic acid. This is also lipid biosynthesis inhibitor. It is chiefly effective against immature stages of sucking pests such as aphids, scales, whiteflies, psyllids, and mealy bugs. The compound reduces the fecundity and fertility in adult female significantly and in consequence reduces insect populations. It is a fully systemic insecticide with distinguishing translocation properties. Following foliar application and uptake the insecticidal activity is trans-located throughout the vascular system, i.e. through its translocation in the xylem and phloem it moves upwards and downwards, respectively. Hence it is called two-way systemic (ambimobile) insecticide. These unique characteristics of spirotetramat allow the control of concealed pests for instance root aphids and the protection of new shoots or leaves appearing after foliar application (Nauen et al., 2008).

3.2.10 Bt toxins

Bacillus thuringiensis Berliner (*Bt*) is rod-shaped, gram-positive, and spore forming bacterium which has been isolated worldwide from different sources such as dead insects, soil, water, silos and insectivorous mammals (Palma et al., 2014). Bt strains are sources of different insecticidal proteins (crystal proteins) that are useful in killing the larvae of different insects. Toxins produced by *Bt* strains are called endotoxins which are actually inactive proteins. For biocidal activity, insect (larvae) must ingest these toxins. After ingestion, these toxins reach the insect midgut where they are dissolved in midgut lumen having alkaline pH. These toxins are activated in midgut due to proteases (Proteolytic enzymes includetrypsin-like serin-proteases, elastase-like and chymotrypsin-like proteases). In the midgut, these active toxins bind to specific receptors of midgut epithelial cells. At this stage insect stops feeding. The active toxins cause perforation and lyses of midgut epithelial cells and enter to insect haemolymph where they germinate and spread. Finally, insect dies due to septicemia (Palma et al., 2014).

3.2.11 Insect growth regulators (IGRs)

These are the novel insecticides, also called third generation insecticides; kill the insects by interfering with the normal process of growth and development in insects (Keeley, 1990).

3.2.11.1 Juvenile hormone agonists or analogs (JHAs)

Active ingredients of this group are fenoxycarb, hydroprene, kinoprene, methoprene, and pyriproxyfen. In immature stages of insects, these toxic compounds mimic juvenile hormone (JH), which is important in controlling development and growth in insects and also helps in normal maturation process. The high quantity of JH in haemolymph blood of immatures prevents them from becoming adult. During the course of development, the level of JH reduces in the immature insects due to degradation of this hormone by enzymes.(DeFur, 1999). The insect can proceed naturally toward adulthood with less JH. Reproductive maturation such as spermatogenesis and oogensis in adult males and females respectively is also considered to be due to JH. Differentiation of casts in social insects for example termites also depends upon presence of JH for instance; worker termites with high JH levels develop into soldiers.

Studies suggest that Biocidal activity of Juvenile hormone analogs (JAHs) is due to binding of these chemicals with JH-degrading enzymes of JH receptors. JHAs help in maintaining JH at high levels within the insect body at a time when it should not be present naturally. This condition effects the reproduction and survival in insects significantly, severely altering its reproductive physiology and/or disrupting the insect's development. Exposure to JHAs often results in death or sterilization. For example, egg production in queens of fire ants was stopped after exposure to JHA-based baits (Nijhout et al., 2014).

Physiological processes related to development in immature fleas and mosquitoes are negatively affected when exposed to methoprene, which results in severe developmental abnormalities that finally lead to death. In a study, exposure of German cockroaches to JHAs converted the last instar adult males or females with physical inability to mate and deformed ovaries, respectively. This resulted in the production of sterile adults in the population of cockroaches. This ultimately resulted in the decline of population due to death of sterile adults. Interestingly, sterile adults have curled, crinkled or twisted wings, which is the only apparent sign of JHA exposure.

3.2.11.2 Ecdysone agonists

The compounds halofenozide, tebufenozide, and methoxyfenozide have been developed as insecticides and act as ecdysone agonists. These compounds are called diacylhydrazines. All these compounds are safe for mammals and environment but are very toxic to insects. These insecticides are selective in action. Methoxyfenozide and tebufenozide are effective against larvae of Lepidoptera that ingest this material but weakly active or inactive on Diptera and Coleoptera. However, halofenozide is effective on Coleoptera but mildly active on Lepidoptera. These compounds mimic the action of 20-hydroxyecdysone (20E), the insect molting hormone. They bind directly to the binding sites of 20-hydroxyecdysone and act as complete agonist at that site. This binding induces premature apolysis in the larvae and they stop feeding. Intoxicated larvae after 24 h start abnormal molting process characterized by premature removal of old head capsule, abnormal cuticle deposition and lack of sclerotization and tanning of the new cuticle, loss of hemolymph and molting fluid. This incomplete molting results in desiccation and death of larva (Smagghe & Degheele, 1998).

3.2.11.3 Chitin synthesis inhibitors:

This group includes the compounds like buprofezin, cyromazine, diflubenzuron, leufenuron, novaluron and noviflumuron. Like the JHAs, these insecticides have no action on nervous system of insects. They interrupt the normal biochemical pathway essential for chitin synthesis. Chitin is dominating and most critical component of insect exoskeleton. During molting process chitin is synthesized and deposited into insect newly generated exoskeleton. Scientific studies reveal that biocidal action of chitin synthesis inhibitors is due to blocking of enzyme designated as chitin synthase responsible for its synthesis. In the absence of this enzyme, chitin cannot be synthesized. The prevention of chitin synthesis is fatal for the affected insect.

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