INSECT GROWTH REGULATORS

These third generation insecticides need to be understood and used wisely

Dr. Michael Pfeiffer

INTRODUCTION

There are many compounds involved in regulating the growth and development of insects. Two compounds which play principal roles in regulating insect growth are ecdysone (commonly called the molting hormone MH) and juvenile hormone (JH). Ecdysone regulates when insects molt and juvenile hormone regulates the body form of the insect after molting. Relative levels of these two hormones MH and JH direct insect development from egg hatch to adulthood. Juvenile hormone also plays a role in development of ovaries and egg yolk production in females and behavior, communication and caste differentiation activities. Some of our most unique insecticides termed insect growth regulators (IGRs) interfere with the action of these hormones and when applied properly result in death of the insect pest. To understand how IGRs function, physiological processes in insect development need to be understood.

MOLTING PROCESS

Insects possess an outer covering called an exoskeleton or cuticle.The primary functions of the exoskeleton are to allow for muscle attachment, protect internal organs and prevent desiccation. The exoskeleton is rigid except during molting and one of the structural components which lends rigidity to the exoskeleton is chitin. Since exoskeletons are rigid and as such are of finite size, juvenile insects must at some point shed the old exoskeleton (cuticle) which is limiting in size and produce a new and larger exoskeleton to accommodate an increase in growth. Exoskeletons in insects are analogous to suits of armor worn by Medieval knights. This shedding of the old armor and building a new suit of armor is called molting. Several events must occur for insect to molt all of which are controlled by hormones. When juveniles reach a certain size the exoskeleton becomes limiting for growth. A "stretch" signal is sent to the insect's brain, which results in the production of prothoracicotropic hormone (PTTH) by neurosecretory cells within the brain (Fig 1). PTTH is translocated and released from the corpus allatum glands into the hemolymph (insect equivalent of blood stream). Release of PTTH stimulates the prothoracic glands to release ecdysone into the hemolymph which is hydroxylated to 20 hydroxyecdysone (20E). A rise in levels of 20E triggers the start of the molting cycle. Shortly after the 20E rise that initiates molting, levels of 20E drop dramatically which initiates other events. Molting fluid rich in enzymes flows between the epidermis and old cuticle and dissolves the old cuticle (Fig 2). Chitin in the old cuticle is recycled to build the new cuticle which is constructed below the old cuticle (Fig 2). After the new cuticle (exoskeleton) is formed, the old cuticle cracks, the insect exits the old cuticle and the new cuticle hardens and pigments.

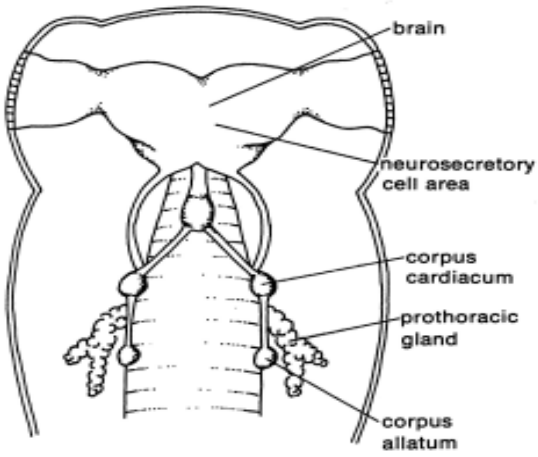

Figure 1. Major endocrine structures in insects.

Horn: 1978

Consider the consequences to insects if materials are applied which disrupt this molting process!

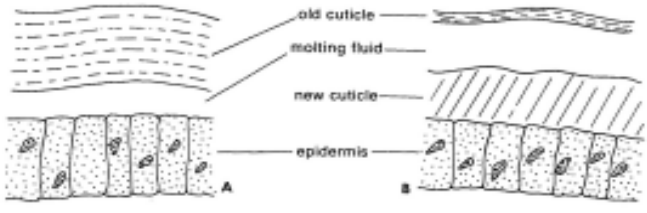

Figure 2. Cross section of insect integument.
A. Before deposition of new cuticle
B. After deposition of new cuticle and before shedding of old cuticle

Horn: 1978

© Copyrighted Pesticide Training Resources 040907

The body form (juvenile, pupae or adult) which insects possess after a molt is controlled by the juvenile hormone (JH). With each molt, the level of JH decreases. With decreasing levels of JH, each subsequent molt results in insect development advancing more toward adulthood. Shortly after the molt which results in adulthood, levels of juvenile hormone often spike again which triggers such events as development of ovaries and egg yolk. Once insects reach sexual maturity, the level of JH is often zero.

Consider the consequences to insects if materials are applied which disrupt the orderly developmental processes regulated by juvenile hormone !

With the discovery of the role that these two hormones play in insect development and the discovery that upsetting the balance of these two hormones could disrupt insect development, thoughts turned to development of compounds that would interfere with these hormonal mediated processes. Compounds have been discovered which interfere with the levels of ecdysone and juvenile hormone in insects and have been termed insect growth regulators (IGRs). Insect growth regulators currently marketed for insect control fall within one of four broad categories depending on mode of action: juvenile hormone agonists, ecdysone agonists, ecdysone antagonists and chitin synthesis inhibitors.

Juvenile Hormone Agonists

Juvenile hormone (JH) agonists mimic the effects of naturally occurring juvenile hormone. If levels of JH or mimics of JH remain high, every molt results in insects emerging as juveniles. If you prevent insect from becoming adults, there can be no reproduction. No reproduction — eventually no insect problem. After exposure to JH agonists, insect death usually results when insect molt from the last instar to the adult. Other potential effects of JH agonists on insects include: sterilization of adults, inhibition of egg hatch and laying of nonviable eggs. Early JH agonists such as hydroprene and methoprene do not have good stability when used in outdoor settings and uses are confined primarily to indoor applications such as roach and flea control and control of pest affecting stored products. Newer JH agonists such as fenoxycarb and pyriproxyfen have good stability and are used in exterior applications. Methoprene also has some "pass through" applications in animals such as cattle. Methoprene is administered to cattle, pass though the cattle into dung and affect developing maggots. Maggots and dung are a good thing. Adult flies and dung are not. JH mimics are also incorporated in flea collars and treated ear tags for cattle. Insects controlled by particular products varies but as a group, they have activity against a wide range of insects.

Ecdysone Agonists

These materials mimic ecdysone and force insects to molt prematurely which typically results in stoppage of feeding and ultimately in insect death. Other effects of these compounds on insects include increased egg mortality and reduced rates of reproduction. Tebufenozide is registered for control of Lepidoptera insects and halofenozide has activity against Lepidoptera, Coleoptera and some Homoptera.

Ecdysone Antagonists

Ecdysone antagonists inhibit the effects of ecdysone. The ecdysone antagonist materials currently on the market come from *Azadiractin indica*, the Neem tree. The active ingredient from the Neem tree is azadiractin and is extracted primarily from seeds of the tree. Azadiractin is composed of at least 25 different compounds which have activity. There are several effects which may become evident after insects are exposed to azadiractin. One effect of Azadiractin on insects is to inhibit PTTH production. PTTH is the hormone which stimulates ecdysone production that ultimately triggers the molting cycle to begin. At some point unless molting occurs in insects, the exoskeleton is going to become like the suit or dress you owned when you were 18: a bit too tight. Other effects of azadiractin on insects included deformities after molts, reduced thriftiness and antifeeding activity although the antifeeding activity is usually short lived.

Chitin Synthesis Inhibitors

The effects of these compound on insects varies but the effects can be grouped into two broad categories: inhibition of chitin synthesis or interference with the organization of exoskeletons. Diflubenzuron, lufenuron and noviflumuron inhibit chitin synthesis. If chitin can not be synthesized, insects have problems when they molt. Even though much of the chitin in the old exoskeleton is recycled into the new cuticle during molting, the larger exoskeleton needed for the next instar necessitates production of new chitin. Buprofezin operates differently than do the compounds which inhibit chitin synthesis. Buprofezin appears to keep levels of 20 hydroxyecdysone (20E) from decreasing during the first stages of molting. If levels of 20E are kept at high levels throughout the molting period, the old cuticle does not get digested and new cuticle does not form properly. Cyromazine is another compound categorized as a chitin synthesis inhibitor however the exact mode of action of cyromazine is unknown. Insects exposed to cyromazine possess cuticles which are abnormally hard. This excess hardening of the cuticle

© Copyrighted Pesticide Training Resources 040907

prevents molting, insects stop feeding and ultimately die. Another effect on insects exposed to chitin synthesis inhibitor type IGRs is an increased in egg mortality. Activity of chitin synthesis inhibitor IGRs as a group is widespread however specific compounds are targeted at specific groups of insects. As with some of the juvenile hormone mimics, some of the chitin synthesis inhibitors are used as "pass though" treatments. Cyromazine is administered to chickens through feed or applied to bedding for control of Diptera larvae. Lufenuron is given orally to dogs and cats for flea control.

Summary

Insect growth regulators have proven extremely effective as components in IPM programs for control of insects which have become resistant to standard insecticides. Typically, IGRs have much lower mammalian toxicities than do most standard insecticides. Usually, IGRs are easy on beneficial insects, have few nontarget problems, do not appear to cause spontaneous genetic mutation, cause cancer or cause birth defects. These material are relatively short lived in the environment which reduces the potential for contamination. Due to the many beneficial aspects of IGRs, some have termed these IGRs as third generation insecticides or biorational insecticides. So why have we not abandoned all other forms of insect control in favor of Insect Growth Regulators? As with most good things, there are negative aspects as well. The assumption that insects will not develop resistance to IGRs is **absolutely false**. Insects have and will continue to develop resistance to current IGRs. IGRs are not without problems associated with nontarget organisms. Certain JH agonists and Chitin Synthesis Inhibitor IGRs have the potential to cause damage if they get into aquatic systems. IGRs do not normally provide instantaneous control and thus are not good materials to use for "rescue" type insect control. Applications of standard insecticides prior to or simultaneously with application of IGRs is a standard practice in many cases: knock down the populations, then rely on the IGR for extended control. When IGRs are incorporated into IPM programs, consideration needs to be given to when is the most advantageous time to apply these materials and which material(s) should be used based on the pest. Would it make sense to uses JH mimics as your sole source of control for juveniles of Lepidoptera on lettuce? You may prevent juveniles from becoming adults and thus prevent future generations but your lettuce patch is gone. ***It's the juveniles that are the damaging stage not the adults in this instance!*** If IGRs are to be and remain useful in IPM programs, we need to get away from the mindset of gauging insect control based solely on a body count after application of insecticides. We need to rethink strategies when incorporating IGRs into IPM programs!

List of common names of IGRs based on mode of activity

Juvenile Hormone Agonists	Ecdysone Agonists	Ecdysone Antagonists	Chitin Synthesis Inhibitors
fenoxycarb	halofenozide	azadiractin	buprofezin
hydroprene	tebufenozide	dihydroazadiractin	cyromazine
kinoprene			diflubenzuron
methoprene			lufenuron
pyriproxyfen			novaluron
			noviflumuron

Pesticide Training Resources

2525 East Seneca Tucson, Arizona
85716-3018
520-323-3135
www.ptrpest.com

© Copyrighted Pesticide Training Resources 040907