Insecticide Series: Part IV
How Insecticides Work

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Pesticides are chemical agents that are designed to kill (or otherwise control) various kinds of pests. Insecticides are one group of pesticides designed to control insects. Many of the insecticides that are currently available to turf managers are "broad spectrum" materials, meaning they control many different kinds of insects. They are broad spectrum because they affect most insects in the same way, often by interfering with the functioning of their nervous systems.

While some turf managers might find the concept of broad spectrum attractive at first glance, it often backfires. There are numerous beneficial insects active in the turf environment that can be killed by broad-spectrum insecticides. Some of them are **predators**, actively feeding on harmful insects. Others are **parasites**, which lay eggs on or inside the bodies of other insects. These eggs hatch into small larvae that feed on harmful insects.

The third group is **saprophytes**. They feed on dead organic material (such as thatch) and assist in decomposition as part of the natural process of growth, death, decay, and rebirth.

Routes of Entry

An insect might come in contact with an insecticide in three ways. The first is by **dermal exposure**. Some insecticides are able to penetrate an insect's cuticle, or outer skin that protects the internal organs. These insecticides are often chemically complex because they must be able to pass through several different layers of "skin," each with different chemical properties. Once they get into the body, they often target some function of the nervous system.

An insect might ingest a plant or another insect that has pesticide residue on it. This is a type of **oral exposure**. Entry through the digestive system allows disruption of the digestive process or absorption into the insect's internal tissues, where insecticides can attack the nervous system of the victim.

The third way an insect is exposed to lethal doses of insecticide is through **inhalation**. These insecticides evaporate and give off vapors that the insect breathes. Once inside the insect, most current insecticides interfere in some fashion with the nervous system of the target pest.

People who come in contact with insecticides are subject to the same routes of entry — dermal, oral, and inhalation. Dermal exposure is the most likely avenue for accidental exposure, particularly during the mixing and loading process.

Worker protection standards have been mandated to protect pesticide applicators from exposure while handling insecticides. Oral ingestion accounts for many of the accidental exposures occurring around the home. Inhalation exposure occurs more often when pesticide applications are made indoors without proper ventilation.

Measuring Toxicity

Chemists (both in the private sector and government) have tried to establish procedures for measuring the toxicity of various compounds. Testing for toxicity requires killing test organisms to determine how large a dose and how many doses are necessary to kill the target organism. Scientists have devised a procedure that gives fairly specific toxicity measurement. However, considerable controversy surrounds the interpretation of this measurement.

Acute toxicity occurs when an organism is adversely affected by a single, often relatively large, exposure to a toxic material. **Chronic toxicity** refers to exposure by low doses over an extended period of time or on several successive occasions.

Chemical toxicity is measured in laboratory settings using test animals, such as mice, rats, and rabbits. Initial screening tests are aimed at determining acute toxicity. These tests can be completed quickly and less expensively. Toxicologists determine the LD50 for a compound — the dose that kills 50 percent of a test population of animals.

An example will help to describe this procedure. A laboratory has 600 male white rats, each weighing one pound. The rats are divided into six groups of 100 each. The first group is fed 1/4 teaspoon of the test material, the second group get 1/2 teaspoon, the third group gets 1 teaspoon, the fourth group receives 2 teaspoons, and the fifth group is fed 4 teaspoons of the material. The sixth group is fed a teaspoon of water instead of the test material. All groups are handled identically. They are left in their cages for 24 hours (or 48 or 72 hours, depending on the material being tested and various other factors), after which the researchers record the number of survivors in each group. The results might look something like this:

Group	Dose	Number Alive After 24 Hours
A	0.0	99
B	0.25	94
C	0.5	76
D	1.0	50
E	2.0	27
F	4.0	3

In this test, the dose of one teaspoon per rat killed 50 of the rats. Therefore, the LD50 for the compound would be one teaspoon per one pound of male rat. The LD50 for female rats, mice or rabbits might be quite different. The age, overall health, and treatment of the test animals can also have a significant effect on their response. LD50 values can only be used as guidelines for determining the toxicity of compounds.

Obviously, laboratory tests are not conducted on every conceivable animal and using data generated from rats (mice or rabbits) to estimate how toxic a material might be to humans is risky. The way different animals metabolize or detoxify materials might vary greatly.

LF50s are expressed in amount of material per unit body weight of the particular test organism. LD50s are normally expressed metrically as milligrams (mg) of material per kilogram (kg) of body weight. This is equivalent to part per million. The key point to recognize is that the lower the LD50, the more toxic the material. For example, the lethal dose for an average man (154 pounds) of a chemical with an LD50 of 10 mg/kg would probably be 0.7 g, which is equivalent to less than half the weight of a paper clip.

LD50s are measured for both dermal and oral exposure. There is often a considerable difference in the LD50 between the two exposures. Usually, the oral LD50 is lower than the dermal for a given species of animal, although there are exceptions.

Signal Words

The Environmental Protection Agency (EPA) assigns a signal word to each pesticide formulation to indicate its toxicity and need for special handling. The EPA often uses the LD50 of a compound (either the pure or the formulated material) as one guideline for determining what the signal word should be.

The most restricted materials carry the signal words, "**Danger - Poison**," and prominently display the skull and crossbones on the label. Such materials usually have an oral LD50 of 0 to 200 mg/kg, the equivalent of a taste to a teaspoon for an average man or woman.

Pesticides carrying a "**Warning**" signal word usually have an oral LD50 of 51 to 500 mg/kg or a dermal LD50 of 201 to 2,000 mg/kg. A lethal dose for an average man or woman would probably one to two teaspoons.

Pesticides that carry a "**Caution**" signal word usually have an oral LD50 or 501 to 5,000 mg/kg or a dermal LD50 of 2,001 to 5,000 mg/kg. This is equivalent to a lethal dose of one ounce to one pint for an average man or woman. Some of the newer insecticides carry a caution label even though their LD50s are greater than 5,000 mg/kg.

The measurement of acute toxicity is not the only criteria used for determining signal words. Some materials have LD50s high enough to warrant a

"Caution" label, but instead carry a "Warning" label. Usually, this is because the material is highly irritating to eyes or causes an allergic respiratory response in some sensitive people. A more restrictive label can be assigned because a material is particularly prone to leaching into groundwater or volatilization.

Toxicity to bees, fish, or birds can also result in upgrading the signal words. Sometimes, the signal word on a label will be more restrictive than its toxicity to vertebrates would seem to indicate.

How Insecticides Work

Now that we understand a few things about pesticide toxicity, how do insecticides actually kill insects? Most insecticides currently on the turf market affect the nervous system in some fashion. Nerve cells carry information throughout the body. These messages move extremely quickly. An organism's response to various stimuli, such as wind, light, heat, or touch, is governed by information being sent through a string of nerve cells. Therefore, insecticides affecting the nervous system can disrupt both metabolism and survival response.

Chlorinated Hydrocarbons

Nerve cells are very complex. For the purposes of this discussion, we can consider them a series of cells lined up end-to-end, not quite touching. Membranes of the cells allow certain ions (charged particles) to pass through. When a nerve cell is not receiving or transmitting impulses, its interior is charged negatively and its exterior is more positively charged. When a one cell receives an impulse, it passes positively charged sodium ions over to increase the positive charge on the next cell. The electrical impulse is thus sent from one cell to the next.

Normally, this movement of ions in and across the cell membrane occurs very quickly. As soon as an impulse has passed through, the cell returns to normal, waiting for the next impulse to arrive.

Some insecticides, such as DDT and most of the chlorinated hydrocarbons, interfere with the ability of ions to move across the cell's membrane. As a result, the normal sharp spike of activity is spread out over a much longer period and the cell takes longer to return to its normal position. The cell acts as if it were receiving an impulse for an extended period of time, rather than for an instant. The effect is of constant firing.

The functional effects of these insecticides is very similar on both insects and humans. Poisoning symptoms include muscle twitching, stomach cramps, difficulty breathing, and difficulties with other types of muscle control. Examples of chlorinated hydrocarbons are chlordane, DDT, dicofol (Kelthane) and methoxychlor.

Organophosphates and Carbamates

As discussed earlier, nerve cells occur in "strings" but are not physically connected to each other. There is a space between the cells, called the synaptic gap, which contains different kinds of molecules. One such substance is a neurotransmitter called acetylcholine (ACh). A neurotransmitter transmits nerve impulses from one cell to the next. When an impulse arrives at the end of one cell, acetycholine in the synaptic gap bridges from one cell to the next to deliver the impulse. When the impulse has been transferred to the next cell, the acetycholine bridge breaks to let the nerve cell return to a normal resting position.

For a nervous system to work efficiently, there must be a way to remove the acetycholine from the receiving cell so that the sending cell can return to normal and be ready for the next impulse. This is the job of another molecule, acetycholinesterase. The cholinesterase attaches to the acetycholine on the receiving cell membrane and pulls its off. Once the combined molecule (ACh:ChE) is free of the cell membrane, the two components split apart and are available for the next impulse.

Organophosphate and carbamate insecticides are often called cholinesterase inhibitors because they tie up the cholinesterase in the synaptic gap. Acetycholine remains attached to the receiving cell membrane. The cell never returns to normal and impulses keep firing. Thus, poisoning symptoms

in humans include nausea, headaches, tremors, muscle twitches, and difficulty breathing. Examples of organophosphates are acephate (Orthene), chlorpyrifos (Dursban), diazinon, fonofos (Crusade, Mainstay), isazofos (Triumph), isofenfos (Oftanol), and trichlorfon (Dylox, Proxol). Carbamates include bendiocarb (Turcam) and carbaryl (Sevin).

Antidotes

A couple of antidotes are available for persons who have been poisoned as a result of exposure to organophosphates and carbamates. They must be administered by a physician and are very tricky to use. Dosages are difficult to determine, and incorrect dosages of antidote can result in serious injury or death.

Atrophine works by inactivating the membrane on the receiving cell, which functionally numbs the system and stops the constant onslaught of nerve impulses. Atropine can be used for both organophosphate and carbamate poisoning.

The second antidote, **2-PAM**, can only be used for overexposure to organophosphates. It works by competing with the organophosphate at the receiving cell membrane. This improves the chance that the cholinesterase will remove the acetycholine normally and permit cells to relax.

Cholinesterase Blood Tests

Blood tests can be carried out to determine the base level of cholinesterase for each person. "Normal" values vary among individuals so each pesticide applicator should have a blood test performed to establish his or her base level. Then, if an overexposure to an organophosphate or carbamate insecticide occurs, a physician will be able to determine the amount of reduction in the individual's cholinesterase. There are two different types of blood tests for cholinesterase, so it's important to know which type established the base line.

Insect Growth Regulators

Insect growth regulators (IGRs) are relatively new compounds in the turf and ornamental markets that interfere with the target insect's ability to grow normally. Some IGRs interfere with the molting process. Others interfere with hormones governing development.

Some of the most exciting developments are occurring in the identification of "juvenile hormones." Each insect completes a series of molts during its growth to adulthood. The insect has juvenile hormones in its body throughout most of that process. As long as the juvenile hormone is present, the insect will not make the final molt to the adult stage. When the concentration of this hormone decreases, the insect molts one last time and emerges as an adult. Juvenile hormones tend to be quite specific in regard to species.

Chemists have identified juvenile hormones for several kinds of insects. They can artificially produce them and they can be applied to confuse the insect's life cycle. When the artificial juvenile hormone is applied to an area where a sensitive insect in active, the insect does not make the final molt to adult and remains juvenile. These juveniles can't reproduce like adults.

Because IGRs are so specific, the have little to no measurable effect on humans and other mammals. Examples of IGRs are azadirachtin (Azatin, Bioneem, Turplex), halofenozide (RH 0345) and methoprene (altosid).

Many insecticides have detrimental effects on humans and other mammals, in addition to insects. Therefore, it is imperative that pesticide applicators follow all the safety precautions outlined on the label and avoid any use that can jeopardize themselves, wildlife, or local water safety.

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