Kentucky Division of Forestry

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FOREST HEALTH BULLETIN

IMIDACLOPRID

MODE OF ACTION

OVERVIEW

Two of Kentucky's most notable forest insect pests are the hemlock woolly adelgid (HWA) and the emerald ash borer (EAB). When people become aware that these pests may affect or are affecting their trees, naturally they want to know how to stop them. Although slowing the rate of spread of the insects and the development of biological controls are important pursuits, the only known effective control method is the use of insecticides. The active ingredient imidacloprid (found in Merit, Touchstone and Imicide just to name a few) is one of the most popular chemicals used to combat insect pests worldwide. Imidacloprid is also one of the most effective insecticides used against HWA and EAB. Even though imidacloprid is frequently used, the way it works against a pest is not well understood by most applicators. This article details the way imidacloprid works, which is also called its "mode of action."

HOW DOES IT WORK?

Imidacloprid is often used as a spray, seed treatment and systemic. When used against borers such as the EAB and piercing-sucking pests such as adelgids and aphids, systemic formulations are used. The term systemic (moves through a plants vascular system) is often incorrectly described as a mode of action. To clarify, a pesticide's mode of action is how the chemical works against an insect after exposure and not how it is delivered to an insect (e.g. through the xylem or phloem) or how an insect is exposed (e.g. ingestion).

Imidacloprid is a nicotinic acetylcholine agonist, which means that it activates specific neuroreceptors to cause a response. This receptor is called nicotinic because it is readily activated by nicotine. Imidacloprid is a synthetic chemical (developed by scientists), and it is based on nicotine. Its activity occurs in the gap found between nerve cells (Fig. 1); also known as the synaptic cleft or synapse. Normally, nerve impulses work on a continuous chemical-electrical-chemical-electrical pattern until it reaches the central nervous system. A chemical neurotransmitter is delivered into the gap from one side and then binds to a receptor on the other side. Upon contact with the receptor, an electrical impulse is stimulated, which upon reaching another gap, stimulates the release of more chemical neurotransmitters. This cycle is repeated throughout the course of a nerve impulse. Another chemical is also released into the gap which causes the neurotransmitter to break down. Imidacloprid binds to the receptor in place of the natural neurotransmitter and is not broken down. Because the neurotransmitter isn't broken down, nerve stimulation is continuous and the insect dies.

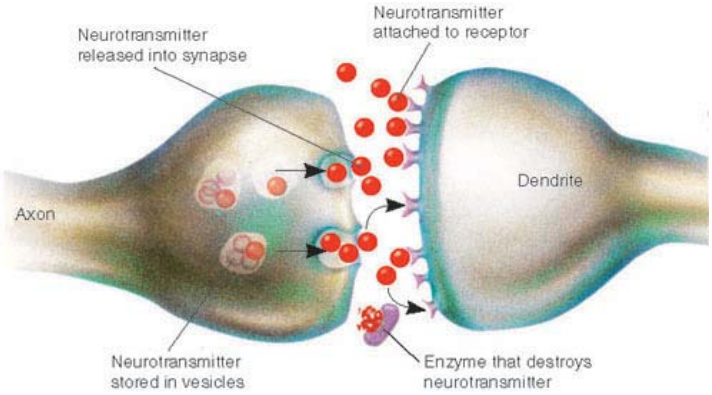

Figure 1. Synaptic cleft between nerves.

INSECT VS. MAMMAL TOXICITY

Two acetylcholine receptors are known in animals, nicotinic and muscarinic. In the animal kingdom, nicotinic acetylcholine receptors are found in the highest concentrations in insects. Imidacloprid affects the nicotinic receptors. Because these receptors are concentrated so highly in insects relative to other animals, toxicity to mammals is relatively low compared to more traditionally-used insecticides. ***Note:*** *This by no means indicates that it isn't harmful to mammals, but that relative to many other insecticides, it is less toxic.*

REFERENCES

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PHOTO CREDITS

Figure 1— *http://designmatrix.wordpress.com/2010/02/20/front-loading-and-the-nervous-system-2/*