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PESTICIDE RESISTANCE: WHAT IT IS AND HOW TO AVOID IT?

This article on pesticide resistance is one of two articles designed to explain the consequences of continually relying on pesticides (in this case, insecticides and miticides) to deal with insect and mite pests of roses, and their potential impact (secondary pest outbreaks and target pest resurgence) on natural enemy (e.g., parasitoid or predator) populations. Well, what is resistance? Resistance is the genetic ability of some individuals in an insect or mite pest population to survive exposure from a pesticide application. In other words, the pesticide no longer kills a sufficient number of individuals to be considered effective. Resistance is an inherited trait and the evolution of resistance in an insect or mite pest population depends on existing genetic variability that allows some individuals to survive exposure of a pesticide application. Any surviving individuals then transfer resistant traits to the next generation thus enriching the gene pool or population with resistant genes. The "selection pressure," or frequency/proportion of the pest population that survives after exposure from a pesticide application, is the main factor that influences resistance. The key is that every time you (as a rose grower) expose an insect or mite pest population to a pesticide, there is potential selection for resistance, which then increases the frequency/proportion of resistant genes within that pest population. The higher the frequency/proportion of resistant genes, the more difficult it is to regulate or control an insect or mite pest population.

Genes for resistance typically occur at a low frequency/proportion in an insect or mite pest population before a pesticide is applied. An individual does not become resistant, but frequent applications of a pesticide over multiple generations remove susceptible individuals, leaving resistant individuals to reproduce. The result is an insect or mite pest population that is more difficult to regulate or control with a given pesticide.

The speed or rate at which resistance develops in a given insect or mite pest population depends primarily on two factors: short generation time (egg to egg) and high female reproduction (ability to produce large numbers of young in a single generation). It is very important to understand how resistance develops and the rate of occurrence when

dealing with the most notorious pest of roses, the twospotted spider mite (*Tetranychus urticae*). Twospotted spider mites have a breeding system that actually accelerates the rate of resistance development because the genes for resistance are fully-expressed or they already pre-exist in the population, and as such it doesn't take a twospotted spider mite population long to develop resistance to a miticide, especially if a miticide with the same mode of action is continually being applied. What is mode of action? Well, this is how or the mechanism by which the pesticide kills the insect or mite pest. If the same mode of action is used continuously then this results in undo "selection pressure" and an increase in the frequency/proportion of resistant individuals in the pest population. How can you overcome or mitigate resistance? The best way is to avoid continually using the same miticide over-and-over again. Always, switch miticides regularly (every 2 to 3 weeks) with different modes of activity. Most of the miticides commonly used on roses are site-specific (attack a very specific target site in the mite) and have discrete modes of action. Table 1 below is a simple listing of the miticides commercially available and their modes of action:

Table 1. Common name, trade name (in parentheses), and mode of action of miticides used for regulation of twospotted spider mite (*Tetranychus urticae*) populations on roses.

Common Name (Trade Name)	**Mode of Action**
Abamectin (Avid/Lucid)	GABA* chloride channel activator
Acequinocyl (Shuttle)	Mitochondria electron transport inhibitor
Bifenazate (Floramite)	GABA-gated antagonist
Etoxazole (TetraSan)	Chitin synthesis inhibitor
Fenpyroximate (Akari)	Mitochondria electron transport inhibitor
Hexythiazox (Hexygon)	Growth and embryogenesis inhibitor
Spiromesifen (Forbid)	Lipid biosynthesis inhibitor
Spirotetramat (Kontos)	Lipid biosynthesis inhibitor

* GABA=Gamma-amino butyric acid

Be sure to take note that Shuttle and Akari, have similar modes of action, as do Forbid and Kontos, so do not use these miticides in succession in order to avoid placing undo "selection pressure" on the twospotted spider mite population and thus enhancing the potential for resistance. Another way to delay the onset of resistance is too use pesticides, with broad modes

of activity that attack multiple target sites in the mite, such as insecticidal soaps (potassium salts of fatty acids), horticultural oils (petroleum or paraffinic oils), or neem oil (clarified hydrophobic extract of neem oil). You should incorporate these pesticides into a spray program with the miticides listed in Table 1.

It is important to understand how resistance develops so you can effectively deal with twospotted spider mite and other pests of roses, and at the same time preserve the longevity and effectiveness of currently existing products. Happy Rose Growing!!

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