Western Flower Thrips & Resistance

**Question:** How do Western flower thrips populations develop resistance to insecticides?

Western flower thrips (Frankliniella occidentalis) is one of the most destructive insect pests of greenhouse-grown horticultural crops including vegetables and ornamentals. Western flower thrips cause both direct and indirect damage. Direct damage is a consequence of their feeding. Western flower thrips have piercing-sucking mouthparts that are used to obtain nutrients from plant cells; resulting in cell death and leaf or flower deformation that can negatively impact marketability. Indirect damage is related to adult transmission of the tospoviruses: Impatiens Necrotic Spot and Tomato Spotted Wilt virus, which subsequently lowers the tolerance for this insect pest. Therefore, due to the direct and indirect damage associated with Western flower thrips, insecticides are the primary means of suppressing western flower thrips populations.

Western flower thrips feed on a wide-range of greenhouse-grown horticultural crops, which enhances exposure to insecticide applications, even those targeting other insect or mite pests, resulting in a high propensity for resistance developing to different insecticides. Moreover, multiple generations per cropping cycle in greenhouses exacerbates the potential for western flower thrips populations to develop resistance to insecticides. Due to the frequency of applying insecticides during the growing season, Western flower thrips populations have developed resistance to insecticides in a number of chemical classes, including organophosphate (e.g., acephate), carbamate (e.g., methiocarb), pyrethroid (e.g., bifenthrin), neonicotinoid (e.g., imidacloprid), spinosyn (e.g., spinosad), macrocyclic lactone (e.g., abamectin) and pyrazole (e.g., fipronil). So, how do Western flower thrips populations develop resistance to insecticides?

This is a very complicated topic; however, in general, the ability of and rate of resistance development in Western flower thrips populations is associated with three biological parameters: 1) rapid (short) generation time, 2) high reproductive capacity (fecundity), and 3) haplo-diploid breeding system. A rapid generation time allows for multiple generations to be present simultaneously during a crop production cycle, with generations overlapping continuously. As a result, females are able to mate with resistant offspring (young) thus increasing the frequency of resistant individuals in a population. Individuals that survive exposure to an insecticide may pass on resistant traits to the next generation, which enriches the gene pool with resistant genes. A haplo-diploid breeding system involves resistant genes in males (that are haploid with one set of chromosomes), which are directly exposed to selection after an insecticide application; thus accelerating the rate of resistance development. In addition, the intensive use of insecticides removes susceptible individuals from the population, consequently increasing the proportion or frequency of resistance individuals.

Another factor that enhances the ability of Western flower thrips populations to develop insecticide resistance is affiliated with resistance mechanisms such as enhanced metabolic detoxification and target site modification (target site insensitivity). In regards to metabolic resistance, there are three primary enzyme systems involved, including: esterases, glutathione S-transferases and cytochrome P-450 mono-oxygenases. The function of these enzyme systems is to convert hydrophobic (water-hating) compounds, such as insecticides, into less biologically active compounds that are hydrophilic (water-loving), and subsequently removed during excretion. Since Western flower thrips feed on a multitude of horticultural and ornamental plant types, populations of Western flower thrips may have inherently evolved the ability to encode for the three enzyme systems; thus promoting resistance development. However, resistance to spinosad is not based on metabolic detoxification but has been reported to be associated with reduced sensitivity of the target site (e.g., nicotinic acetylcholine receptor).

Therefore, the primary means of delaying or mitigating resistance is by rotating insecticides with different modes of action, with insecticides typically applied every two to three weeks or within a generation. The ideal situation is to utilize a multitude of insecticides with different modes of action by using one mode of action within a generation, and then switching to a different mode of action in the next generation. However, the rotation of insecticides with distinct modes of action assumes that the frequency or proportion of individuals in a Western flower thrips population resistant to one insecticide will be reduced when an alternative insecticide, with a different mode of action, is applied. Consequently, the initial insecticide will provide effective suppression of a Western flower thrips population when re-introduced into the rotation program. Rotating insecticides with different modes of action will reduce selection pressure associated with one insecticide, so that individual western flower thrips will only be exposed to one mode of action during their lifetime. The rotation of insecticides with different modes of action is an important strategy that will help preserve and prolong the effectiveness of existing insecticides, which will then allow greenhouse producers to effectively manage western flower thrips populations.

In addition, greenhouse producers need to make sure that spray applications result in thorough coverage of all plant parts including leaf undersides and flowers, and that insecticide applications are properly timed when the most susceptible life stages (e.g. larvae and adults) are present. However, in addition to exercising caution and judiciously using insecticides, greenhouse producers need to consider implementing alternative plant protection strategies against western flower thrips populations, such as cultural control, sanitation, and biological control.