INSECTICIDE CROSS-RESISTANCE IN THE WFT: PHYSIOLOGICAL MECHANISMS AND IPM STRATEGIES

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Summary of Research: Insecticide cross-resistance is an important problem for IPM of western flower thrips in cut flowers. We tested the hypothesis that a cellular detoxification pathway, the efflux transporter system, plays an important role in insecticide cross-resistance in the WFT. We used two insecticides that are labeled for use against thrips in greenhouse production systems (abamectin & tolfenpyrad), one insecticide that is known to be regulated by the efflux transporter system (acetamiprid), and a pyrethroid (lambda-cyhalothrin) to investigate the role of efflux transporters in insecticide resistance in WFT.

RESULTS:

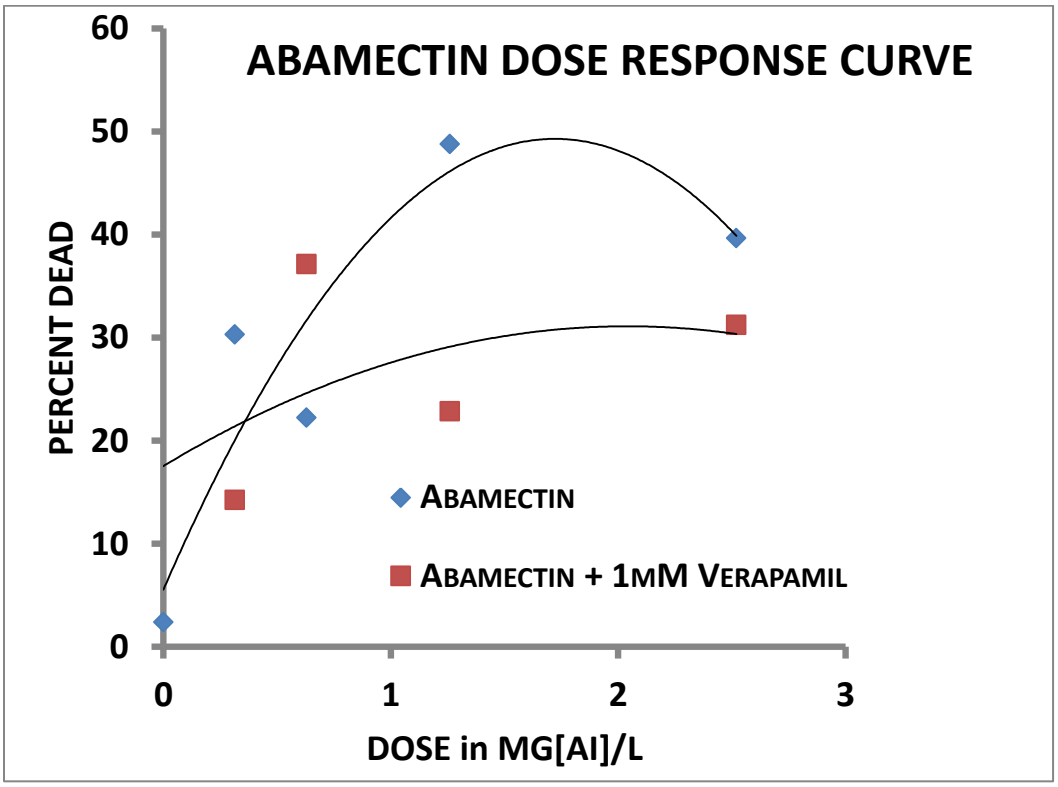

Figure 1. Dose response curves of western flower thrips collected from Washington State University greenhouses and sprayed using a Potter Spray Tower (Burkard Scientific, Ltd.). Mortality was assessed after 24 and 48 hours. Sample sizes ranged from 20-60 individuals per treatment.

Greenhouse WFT showed predictable responses to abamectin (Figure 1) and were unaffected by application of Verapamil. Verapamil is a pharmacological inhibitor of efflux transporters. When Verapamil is present, efflux transporters are unable to act to detoxify cells, rendering the insect susceptible to the toxic chemical. However, this only works on the insecticide if the efflux transporters have an important role in insecticide resistance of a specific insecticide. We clearly show that efflux transporters are not important in WFT resistance to abamectin. The mode of action for abamectin is via the GABA chloride channel and our results suggest that Verapamil has no effect on this mechanism.

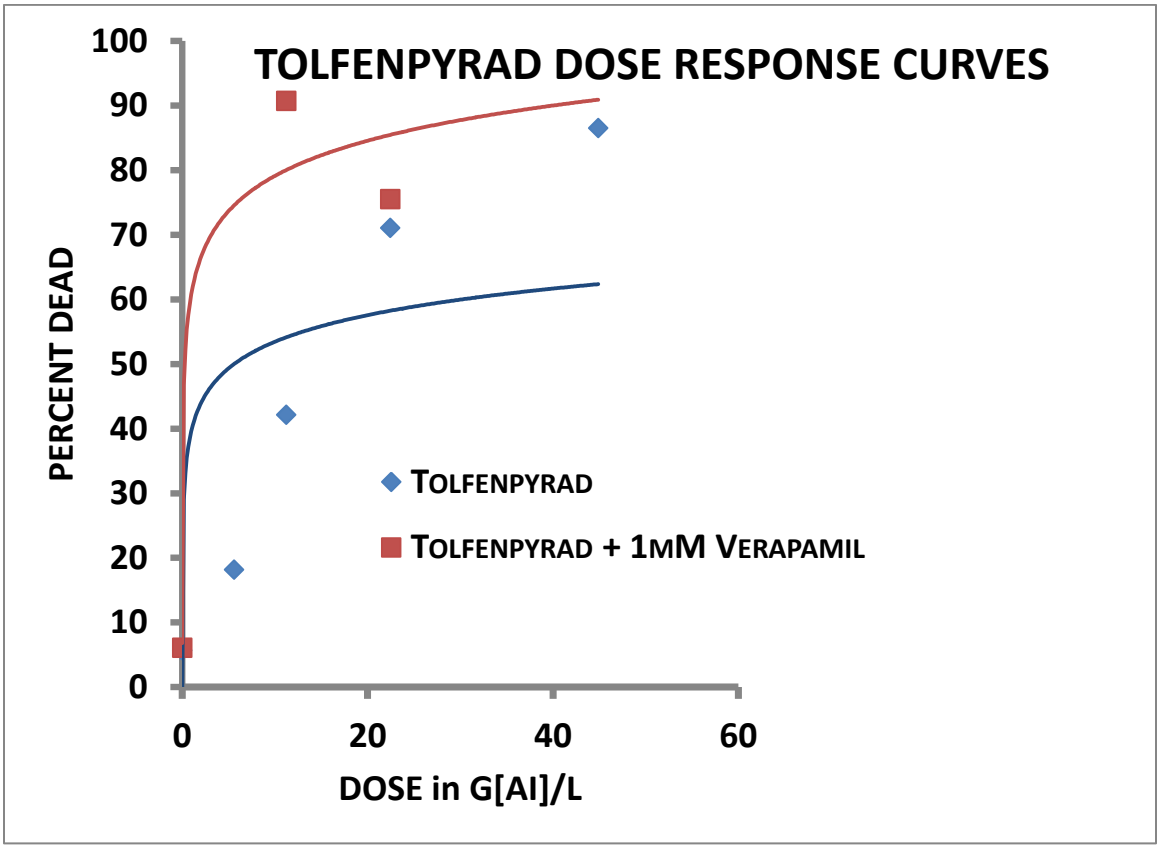

Figure 2. Dose response curves of WFT to tolfenpyrad. Verapamil did increase the number of WFT that died after 48 hours. Only two treatments of 1mM Verapamil were used with the two doses of Tolfenpyrad closest to the LD_{50} concentration.

The response of WFT to tolfenpyrad, a mitochochondrial electron transport inhibitor, is shown in Figure 2. Tolfenpyrad plus 1 mM Verapamil did increase the mortality of WFT at one dose (11.22 g[Ai]/L) but not at the higher dose (22.44 g[Ai]/L). This is an intriguing result as it suggests that efflux transporters are able to detoxify insects exposed to Tolfenpyrad at low doses but once they have received a higher dose, the transporters may not be as efficient at detoxification.

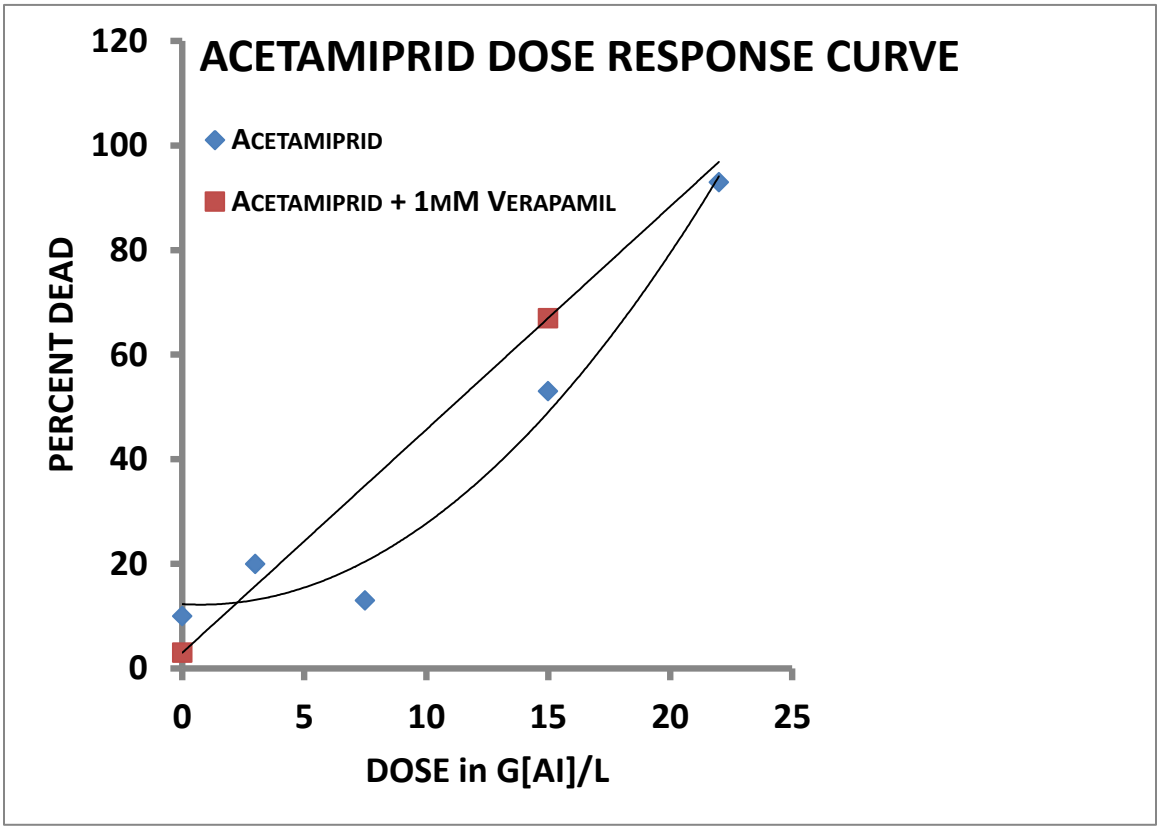

Figure 3. Dose response curves of WFT to acetamiprid. A small increase in mortality was seen when acetamiprid was combined with 1 mM Verapmil at the the LD_{50} concentration.

The number of dead WFT increased slightly in the treatment of acetamiprid plus 1 mM Verapamil at 15 g[Ai]/L. This suggests that the efflux transporters might have a role in susceptibility to acetamiprid in the WFT. The mode of action of acetamiprid is via the acetylcholine receptor and it is a neonicotinoid insecticide.

We specifically chose insecticides with different modes of action to determine the efficacy of the efflux transporters at insecticide detoxification of a diversity of chemistries.

Figure 4 shows that efflux transporters are not acting to detoxify the pyrethroid insecticide lambda-cyhalothrin in the WFT.

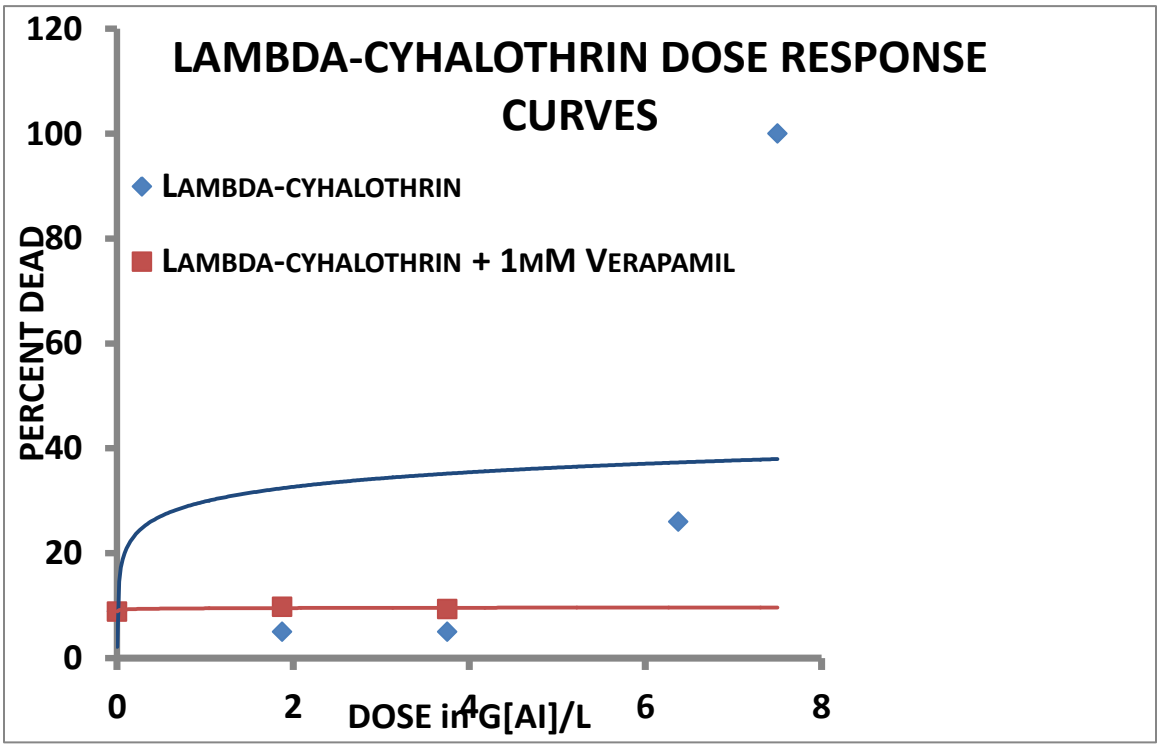

Figure 4. The response of WFT to lambda-cyhalothrin and lambda-cyahlothrin plus 1 mM Verapamil.

DISCUSSION: In our original proposal, we predicted that inhibition of efflux transporters would result in increased efficacy of insecticides commonly used against the western flower thrips. What we found was that efflux transporters may have a role in resistance to tolfenpyrad and acetamiprid but not against abamectin or the pyrethroid lambda-cyhalothrin.

Our results suggest that efflux transporters may indeed have a role in insecticide resistance and that the use of Verapamil or some other efflux transporter inhibitor has potential against target insecticides.