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Project Title: Examining issues associated with the efficacy of two neonicotinoid insecticides (CruiserMaxx[®] and Admire Pro[®]) against thrips on peanut in comparison with Thimet[®]

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Summary

Recent increases in spotted wilt incidence is strongly correlated to increase in thrips populations and feeding injuries that accompany thrips infestations in early stages of peanut seedling growth. The shift from using broad spectrum insecticides such as aldicarb (Temik) and phorate (Thimet) to neonicotinoid insecticides such as imidacloprid and thiamethoxam could have influenced this outcome. Imidacloprid is being used in place of phorate more frequently. There has been an increased concern of resistance development towards neonicotinoids by thrips (tobacco thrips, *Frankliniella fusca*). This prompted us to examine various aspects of imidacloprid effects on thrips, including assessing plant residue levels and associated thrips mortality, thrips susceptibility to imidacloprid in the lab and in the field, developing an imidacloprid-resistant thrips population, and identifying the molecular factors that determine resistance to neonicotinoids in thrips using transcriptomic and genomic approaches. Preliminary results indicate that the thrips susceptibility to imidacloprid seems to be more closely correlated to residue levels of the insecticide in the plant, as opposed to resistance to imidacloprid. Field sample testing for the last three years have indicated that the thrips are still susceptible to imidacloprid at pretty much the same level as that of the lab population. Numerous rigorous attempts to develop a resistant population by challenging our lab population with sub-lethal levels of imidacloprid has not yielded a stable resistant population yet, and efforts are underway to develop one such population. In addition, molecular factors such as detoxifying (pesticide) enzymes that influence susceptibility to imidacloprid and others have been identified and phylogenetically compared to other insects. However, the issues have been to narrow down the putative gene homologs. Numerous homologs have been identified for each target, and the fact that these homologs belong to multigene families has further complicated the issue. A more stringent approach would be to develop a genome for thrips to narrow down the targets. The plan is to undertake a genome development approach in the next one to two years.

Susceptibility to neonicotinoid insecticide imidacloprid:

This is a multi-year continuation project. The work so far has examined residual toxicity of neonicotinoid insecticides and thimet in peanut foliar tissue and correlated that to thrips mortality over time. Results revealed that the concentration of insecticide metabolites declined rather quickly in plants following application. The residue levels post two weeks of application were not significant enough to affect thrips populations substantially. This partially explained the lack of thrips control weeks after application of insecticides. The next question of whether thrips have developed resistance to neonicotinoids is being worked upon for the last 2 years. We initially established baseline data with LC50 values for these insecticides using our laboratory population. This allowed us to scout for field populations and examine for reduction in susceptibility to imidacloprid in comparison with the lab colony. We accomplished this by evaluating resistance ratios. For instance, if the resistance ratio is substantially higher than 1, that indicates reduction in susceptibility in field thrips populations in comparison with the lab population. In the last two to three years, we have regularly compared LC50 values of lab populations to field populations. Thrips populations were collected from fields in at least 5 counties routinely and assayed for reduction in susceptibility to imidacloprid. Preliminary screening has not provided substantial evidence for increased resistance in selected field populations in Georgia, and more need to be evaluated. These results have been provided to the NPB in the last few quarterly reports.

Imidacloprid-resistant thrips colony: Numerous attempts have been undertaken to develop a imidacloprid resistant thrips colony by selecting the insects against sub-lethal doses of the insecticide over several generations. This approach so far has shown no increase in resistance or decrease in susceptibility to imidacloprid, and moderate changes have reverted to their original levels following lack of selection. This has further complicated our efforts to examine resistance in thrips, and efforts are continuing to build one such resistant population.

Transcriptome mining: with funding from NPB we have been developing transcriptomes for *F. fusca* (thrips), this effort was undertaken to assess how TSWV affects thrips. Consequently, we could use the same transcriptomes and identify homologs of neonicotinoid detoxifying enzymes such as P450 monooxygenases, esterase, and transferases. Phylogenetic relationships based on existing databases have led to identification of numerous putative homologs of genes that could code for the detoxification enzymes stated above. The phylogenetic correlation of multiple transcripts for each target, and the fact that some of these enzyme genes are from multiple-gene families have at this point precluded us from developing targeted assays that would prove the function of these enzymes in thrips. Even for development of such assays, it is vital to develop a resistant population. We hope to address this issue either by developing one such population, or by collecting insects from cotton where resistance levels reported seem to be much greater than in peanut. Below is a diagram showing multiple homologs from the transcriptome shown for P450 in *F. fusca* in relation to other insects.

