BINDING OF VIP3A TOXIN TO RESISTANT AND SUSCEPTIBLE *HELICOVERPA ZEA* BRUSH BORDER MEMBRANE VESICLES

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Abstract

Corn and cotton that has been genetically modified to produce the insecticidal proteins derived from the bacterium, *Bacillus thuringiensis*, are one of the most important contributions of biotechnology to agriculture. The Cry and Vip proteins expressed in these crops both target midgut receptors and form pores in the midgut epithelium following receptor binding. The Cry and Vip proteins differ in that they target different receptors, and thus have proven to be valuable tools for the management of Lepidopteran pests when used in conjunction. However, bollworm, *Helicoverpa zea*, has been difficult to manage with Bt crops and widespread resistance to the Cry proteins (Cry1A and Cry2A) expressed in Bt crops has been reported in bollworm field populations. Vip3A remains as the last effective insecticidal protein available in Bt crops for the management of bollworm. Therefore, it will be increasingly important to determine the mechanism of resistance associated with cases of Vip3A resistance as Bt crops capable of expressing Vip3A become more widely adopted. A *Helicoverpa zea* strain with >588-fold higher levels of Vip3A resistance compared to a susceptible strain was established from F_2 screening of Texas field populations in 2019. Brush border membrane vesicles (BBMV) were prepared from midguts dissected from the Vip3A-RR strain and Vip3A susceptible strains. Binding assays using Vip3Aa39 toxin labeled with either biotin or Iodine-125 were performed to determine if reduced binding of Vip3A to midgut receptors was associated with resistance. Results from western blot binding assays using biotinylated Vip3Aa39 toxin suggested reduced binding in the Vip3A-RR strain compared with susceptible strains. Preliminary data from binding assays using ^{125}I-Vip3Aa39 suggest a reduction in binding as well. However, more assays need to be conducted to generate full saturation curves and confirm that binding of radiolabeled Vip3Aa is reduced in the Vip3A-RR strain. The data presented in this presentation provide evidence of reduced binding as a mechanism of Vip3Aa resistance in a field-derived strain of *Helicoverpa zea*.