Progress Report

Investigation of Genetic Basis of 2,4-D and Dicamba Resistance in Palmer amaranth

Objective: ***Understand the genetic basis of 2,4-D and dicamba resistance in Palmer amaranth by the classical breeding approach.***

Four F_1 families were generated by individually crossing two parental female and male plants that were found resistant to 2,4-D (AHR; Auxin Herbicide Resistant) with known 2,4-D-susceptible male and female plants (AHS; Auxinic Herbicide Susceptible). Progenies of each F_1 family, along with parental plants were treated with varying doses of 2,4-D (*i.e.*, 0, 0.5X, 1X, 2X, and 4X; where 1X = 560 g ae ha^{-1}) in dose-response pattern, and the experiments were repeated. Additionally, female and male F_1 plants of Palmer amaranth from three F_1 families that survived 2,4-D (1120 g ae ha^{-1}) in the dose-response experiments were crossed to generate four pseudo-F_2 families. Segregation of resistance or susceptibility was assessed by treating progenies of F_2 families (n=623) with 2,4-D (560 g ae ha^{-1}). F_1 dose-response analysis suggested that 2,4-D resistance in AHR is governed by incompletely dominant nuclear allele(s). Progenies of all four F_1 families had an intermediate level of 2,4-D resistance compared to parental AHR or AHS Palmer amaranth. Cumulative biomass distribution of the F_1 progenies also represented incomplete dominance of 2,4-D resistance trait in AHR. Chi-square analyses of F_2 data suggested that more than one gene (polygenic) mediated 2,4-D resistance in AHR. Interestingly, varying phenotypes were observed in progenies surviving 2,4-D in F_2 families supporting polygenic inheritance. Overall, the results of this study suggest that incompletely dominant genes govern 2,4-D resistance in AHR Palmer amaranth. Herbicide resistance if governed by a single dominant allele spreads faster than an incompletely dominant multiple alleles. Regardless, adopting integrated pest management techniques can diminish selection pressure and help manage the spread of 2,4-D resistance.

Additionally, the rate of [^{14}C] 2,4-D metabolism in 2,4-D resistant Palmer amaranth was compared to two susceptible populations as well as wheat (naturally tolerant to 2,4-D). Our data suggest that the resistant Palmer amaranth rapidly metabolizes 2,4-D compared to susceptible plants. Further, based on the [^{14}C] 2,4-D retention time, both 2,4-D resistant Palmer amaranth and wheat generated similar polar [^{14}C] 2,4-D metabolites. Nonetheless, ~70% of [^{14}C] 2,4-D was metabolized in wheat, compared to only 30% in 2,4-D resistant Palmer amaranth. Application of cytochrome P450

inhibitor malathion caused a 60% reduction in GR_{50} (the amount of herbicide required to reduce dry biomass by 50%) of AHR, indicating the involvement of P450s in metabolizing 2,4-D in AHR. However, application of another P450-inhibitor, piperonyl butoxide (PBO) did not cause any reduction in 2,4-D resistance in AHR indicating selectivity of P450-inhibitors in inhibiting P450 enzymes. Overall, the data from this research suggest that enhanced metabolism, potentially mediated by P450 enzyme activity, is the primary mechanism imparting 2,4-D resistance in Palmer amaranth.