TARGET-SITE RESISTANCE TO ALS INHIBITORS IN HORSEWEED. Danman Zheng, Patrick J. Tranel, Vince M. Davis, Greg R. Kruger, and William G. Johnson, Graduate Research Assistant and Associate Professor, University of Illinois, Urbana, IL 61801, and Graduate Research Assistants and Associate Professor, Purdue University, West Lafayette, IN 47907.

The mechanisms of resistance to ALS-inhibiting herbicides were investigated in four horseweed populations designated 13R, 40R, 525R and 116R. Results from acetolactate synthase (ALS) activity assays indicated that altered target sites caused herbicide resistance in 13R, 40R and 525R. In addition, cross-resistance patterns were compared between protein extracts derived from 40R and 525R in response to cloransulam and chlorimuron. The R/S ratios calculated based on estimated I_{50} values were 400 and 222-fold for cloransulam and 789 and 947-fold for chlorimuron in 40R and 525R respectively. Southern blot analysis showed that there was only one *ALS* gene locus in the diploid horseweed genome. Three overlapping *ALS* gene regions were amplified in three selected plants from each biotype. Amino acid sequences were inferred and compared with each other to identify the resistance-conferring mutations. Amino acid substitutions conferring ALS inhibitor resistance in horseweed plants were population specific. Substitution of Ser for Pro at position 197 was identified in both 13R and 40R. However, substitution of Ala for Pro at position 197 and Glu for Asp at position 376 were identified in 525R and 116R, respectively. Based on mechanisms identified from the above studies, PCR/*BsaJ*I and PCR/*Mae*II molecular markers were developed to differentiate between wild type and resistant codons at position 197 and 376 of horseweed *ALS*, respectively.

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