ACETOHYDROXYACID SYNTHASE (AHAS) GENES AND HERBICIDE TOLERANCE IN SUNFLOWER: A REVIEW AND CONSIDERATIONS FOR GENE FLOW. Mark L. Dahmer, Siyuan Tan, and Richard R. Evans, BASF Corporation, 26 Davis Drive, Research Triangle Park, NC 27709-3528

AHAS is a critical enzyme for the biosynthesis of branched chain amino acids in plants. Common sunflower (*Helianthus annuus*) has three AHAS genes: AHAS1, AHAS2, and AHAS3. AHAS is the target enzyme for AHAS-inhibiting herbicides such as imidazolinones and sulfonylureas. AHAS resistance occurs widely in weedy common (*H. annuus*) and prairie (*H. petiolaris*) sunflowers and has been reported in several surveys in Colorado, Kansas, Missouri, Nebraska, North Dakota, and South Dakota. Two resistant AHAS gene variants have been introduced from wild *H. annuus* populations to domesticated sunflower lines for developing herbicide tolerant sunflower. One of the variants had a point mutation at codon 205 (in reference to *Arabidopsis thaliana*) of AHAS1 gene with a codon substitution from GCG to GTG and an amino acid substitution from alanine to valine of the encoded AHAS protein. The A205V AHAS1 gene mutation confers a high tolerance to imidazolinone herbicides. It has been used to develop imidazolinone-tolerant (CLEARFIELD®) sunflower. The other AHAS gene variant had a point mutation at codon 197 of AHAS1 gene with a codon substitution from CCC to CTC and an amino acid substitution from proline to leucine of the encoded AHAS protein. The P197L AHAS1 mutation confers a high tolerance to several sulfonylurea herbicides and has been also introduced into domesticated sunflower lines. Besides the resistant mutants originated from the wild common sunflower, a sulfonylurea-tolerant sunflower line named M7 has also been created by using induced mutagenesis and artificial selection. The A205V AHAS1 gene was reported to be able to flow from a domesticated sunflower to wild common and prairie sunflowers, but the outcrossing decreased quickly with the increase of distance from pollen source. The resistant hybrids were found to be able to backcross with wild common or prairie sunflower parent. In the absence of imidazolinone herbicides, the resistant gene does not reduce or increase the competitive ability of either common or prairie sunflower. To prevent development of resistance in wild sunflower from either outcrossing or spontaneous mutation, growers are strongly encouraged to rotate CLEARFIELD® sunflower with other crops and to mix and rotate herbicides with a different mode of action. If CLEARFIELD® sunflower is planted, growers should spray the field with an imidazolinone herbicide at label rate. Growers should also employ other weed management practices that can control resistant weeds such as physical control. Effective control of weedy sunflower adjacent to CLEARFIELD® sunflower field and control of CLEARFIELD®sunflower volunteers in the following season are also good measures to minimize gene flow.

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