A SECOND TRIAZINE RESISTANCE MECHANISM IN WATERHEMP. Patrick J. Tranel, William L. Patzoldt, Bradley S. Dixon, and Dean E. Riechers, Assistant Professor, Graduate Research Assistant, Undergraduate Research Assistant, and Assistant Professor, Department of Crop Sciences, University of Illinois, Urbana, IL 61801.

Resistance to triazine herbicides is well documented, and is usually due to an altered herbicide site of action (D1 protein). Because the gene encoding the D1 protein is encoded in chloroplasts, target-site triazine resistance is maternally inherited. Recently, we identified waterhemp (*Amaranthus rudis* and *A. tuberculatus*) populations with atrazine resistance that did not appear to be maternally inherited. Specifically, atrazine resistance was observed to segregate among half-sib families that each had a common female parent. Therefore, research was conducted to verify the novel atrazine resistance in waterhemp and to begin charactering it.

Three waterhemp populations where included in the study: SegR, a population segregating for atrazine resistance and suspected of having the new resistance mechanism; UniR, a population uniformly resistant to atrazine and suspected of having target-site resistance; and UniS, a uniformly atrazine-sensitive population. Sequencing of a fragment of the gene encoding the D1 protein revealed that UniR plants contained the point mutation typically found in triazine-resistant plants. No sequence differences were identified when the gene was compared among atrazine-resistant and atrazine-sensitive plants of the SegR population and plants from the UniS population. Results from chlorophyll fluorescence assays provided further confirmation that atrazine resistance was target-site mediated in UniR plants, but not in SegR plants. SegR plants exhibited modest recovery of electron transport perturbation over a period of four days after atrazine treatment, suggesting resistance was due to atrazine metabolism.

Crossing experiments were conducted to begin to understand the inheritance of the new resistance mechanism. Progeny from a SegR by UniS cross, and from the reciprocal cross, could be classified as having one of three atrazine-response phenotypes: sensitive, resistant, or intermediate. Progeny from a SegR by SegR cross also grouped within these classifications. Because sensitive plants were recovered from the SegR by SegR cross, resistance must not have been fixed in the parents, even though only resistant plants were used for the parents. Heterozygosity in the SegR parents complicated analysis of the SegR by UniS crosses; however, resistance must have been nuclear transmitted, since resistant progeny were recovered from both of the two reciprocal crosses.

Our current hypothesis is that the newfound triazine resistance is mediated by enhanced herbicide metabolism, and is controlled by two or more genes. Although preliminary, results from experiments using tridiphane to overcome atrazine resistance indicated that resistance is not primarily mediated by glutathione-S-transferases.

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