RESISTANCE TO ALS INHIBITORS IN FOXTAIL SPECIES. Danman Zheng, Dean S. Volenberg, Aaron G. Hager, and Patrick J. Tranel, Graduate Research Assistant, Postdoctoral Research Associate, Assistant Professor and Associate Professor, Department of Crop Sciences, University of Illinois, Urbana, IL 61801.

The foxtails (*Setaria spp.*) are among the world's most successful colonizing weeds. Green foxtail, giant foxtail, and yellow foxtail biotypes from the North Central states have been reported to have evolved resistance to certain acetolactate synthase (ALS)-inhibiting herbicides. However, there is currently inadequate knowledge of these foxtail biotypes' mechanisms of resistance. Our objectives were to: 1) characterize resistance to imidazolinone (IMI) and sulfonylurea (SU) herbicides at the whole-plant and enzyme levels, 2) identify the molecular mechanism(s) of resistance and, 3) quantify the genome size and *ALS* gene copy number in the foxtail species.

ALS-resistant green and yellow foxtail biotypes were from Wisconsin (WI) and Minnesota (MN), respectively. Three ALS-resistant giant foxtail biotypes were obtained, one each from WI, MN and Illinois (IL). An ALS susceptible biotype of each foxtail species was obtained from each respective state.

Whole-plant dose-response experiments were conducted on three- to four leaf-stage foxtail plants. Compared to susceptible plants, the ALS herbicide resistant green foxtail plants were 9 and >411-fold resistant to nicosulfuron and imazamox, respectively. ALS herbicide resistant giant foxtail plants from WI were >3,600 and >600-fold resistant to nicosulfuron and imazamox, respectively. Similarly, ALS herbicide resistant giant foxtail plants from MN were >3,600 and >600-fold resistant to nicosulfuron and imazamox, respectively. In contrast, ALS herbicide resistant giant foxtail plants from IL were 1.6 and 81.2-fold resistant to nicosulfuron and imazamox, respectively. The ALS herbicide resistant yellow foxtail was 1.6 and 178-fold resistant to nicosulfuron and imazamox, respectively. Results of in vitro ALS enzyme activity assays corresponded with whole-plant dose-responses; however, the resistance ratio (resistant I_{50} / susceptible I_{50}) was an order of magnitude lower compared to the whole plant resistance ratio. Results from the in vitro ALS activity assays indicated that altered target sites were responsible for herbicide resistance in these foxtail biotypes.

Regions of the ALS gene were sequenced to determine what mutations were responsible for resistance. Specific primers were designed based on green foxtail *ALS* cDNA sequence and were used to amplify region A and B from each foxtail biotype. Sequence data showed that a polymorphism at position 653 correlated with the IMI-resistant phenotype in green and yellow foxtail, and in the giant foxtail biotype from IL. A leucine-for-tryptophan substitution at position 574 (L574W) of ALS was the mechanism of herbicide resistance in the giant foxtail biotypes from MN and WI.

Southern blot analysis indicated that green foxtail has one copy of *ALS*, however, both giant and yellow foxtail have two copies of *ALS*. Consistent with this, chromosome counts indicated that green foxtail is diploid (2X=18) whereas giant and yellow foxtail are tetraploid (4X=36).