HERBICIDE DOSE-RESPONSE OF WILD OAT WITH ALTERED ACETYL-COA CARBOXYLASE GENES. Michael J. Christoffers, Shauna N. Pederson, and Aruna V. Kandikonda, Assistant Professor, Research Specialist, and Graduate Research Assistant, Department of Plant Sciences, North Dakota State University, Fargo, ND 58105.

Chemical inhibitors of acetyl-CoA carboxylase (ACCase) are important postemergent herbicides used for selective control of wild oat and other grass weeds. Wild oat biotypes with missense point mutations in the *Acc1;1* gene for plastidic ACCase were evaluated in a greenhouse for whole-plant resistance to ACCase inhibitors. Biotypes AI13R10, 830R34, and VIR35 have Trp_{2027} to Cys, Asp_{2078} to Gly, and Cys_{2088} to Arg substitutions, respectively, with amino acid positions corresponding to blackgrass ACCase. Dose-response was evaluated for the aryloxyphenoxypropionate (APP) ACCase-inhibiting herbicides fenoxaprop-P, clodinafop, and quizalofop; and the cyclohexanedione (CHD) herbicides sethoxydim, tralkoxydim, and clethodim. Treatment was at the two-leaf stage and analysis was based on the dry weight of above-ground tissue three weeks after treatment, with growth reduction compared to that of a susceptible wild oat biotype, KYN119.

Resistance to fenoxaprop-P was confirmed in all three wild oat biotypes including 830R34 and VIR35, but was highest in AI13R10. Resistance to clodinafop was also observed in the three biotypes with altered *Acc1;1*. Biotypes AI13R10 and VIR35 were resistant to quizalofop, while 830R34 treated with quizalofop displayed reduced growth reduction below label rate compared to susceptible KYN119. Reduced growth reduction below label rate was also observed for 830R34 and VIR35 with sethoxydim and clethodim, and all three mutant biotypes with tralkoxydim. These results are consistent with ACCase target-site alterations conferring resistance to ACCase inhibitors, and especially APPs, in AI13R10, 830R34, and VIR35.

2005 North Central Weed Science Society Proceedings 60:35.