MECHANISM OF COMMON WATERHEMP RESISTANCE TO PROTOPORPHYRINOGEN OXIDASE (PROTOX)-INHIBITING HERBICIDES. Douglas E. Shoup and Kassim Al-Khatib, Research Assistant, Associate Professor, Department of Agronomy, Kansas State University, Manhattan, KS 66506.

A biotype of common waterhemp (*Amaranthus rudis*) difficult to control with PPO inhibiting herbicides was reported near Sabetha, KS in 2000. Greenhouse experiments confirmed resistance to protox-inhibiting herbicides as well as acetolactate synthase-inhibiting herbicides. The objectives of this study were to determine if absorption, translocation, and metabolism is the basis for protox-resistance in common waterhemp. At 13 to 18 cm tall, susceptible (S) and resistant (R) common waterhemp plants were treated with ^{14}C labled acifluorfen or lactofen. Six, 12, 24, and 72 h after herbicide application, plants were divided into treated leaf, foliage above treated leaf, foliage below treated leaf, and roots. Absorption increased as harvest time increased, however, absorption was similar for both biotypes and herbicides. For both biotypes, 85-95% of acifluorfen and lactofen remained in the treated leaf. An organophosphate insecticide interaction experiment was also conducted to determine if cytochrome P450 was involved in the mechanism for resistance. Resistant plants were treated with a recommended rate of malathon or diazinon followed by a 1x rate of acifluorfen or lactofen. Acifluorfen and lactofen rates required to induce 50% visible injury were not different when herbicides were applied alone and with an insecticide. Because the R and S biotypes had similar acifluorfen and lactofen absorptions and translocations, the mechanism of resistance is not due to insufficient absorption or translocation. Also, results indicate no cytochrome P450 metabolism of acifluorfen or lactofen in the R biotype.

2002 North Central Weed Science Society Abstracts. 57:162.