MOLECULAR GENETICS OF GLYPHOSATE RESISTANCE IN PALMER AMARANTH. Todd A. Gaines, Philip Westra, Jan E. Leach, Sarah M. Ward, Bekir Bukun, Stephen T. Chisholm, Dale L. Shaner, Christopher Preston, A. Stanley Culpepper, Timothy L. Grey, Ted M. Webster, William K. Vencill, and Patrick J. Tranel, Graduate Student, Professor, Professor, Associate Professor, Visiting Scientist, and Assistant Professor, Colorado State University, Fort Collins, CO 80523, Plant Physiologist, USDA-ARS, Fort Collins, CO 80526, Lecturer, University of Adelaide, Australia, Associate Professor and Assistant Professor, University of Georgia, Tifton, GA 31794, Research Agronomist, USDA-ARS, Tifton, GA 31794, Associate Professor, University of Georgia, Athens, GA 30602, and Associate Professor, University of Illinois, Urbana, IL 61801.

Glyphosate resistant Palmer amaranth populations were identified in Georgia. The molecular basis of resistance is unknown. No target site mutations known to confer resistance were identified in resistant alleles of the 5-enolpyruvylshikimate-3-phosphate synthase (EPSPS) gene, the target of glyphosate. Glyphosate selection in cell culture results in EPSPS gene amplification, so copy number and expression level were compared. DNA blot analysis of Palmer amaranth gDNA suggested more copies of EPSPS in resistant than susceptible plants. Estimation of gene copy numbers of EPSPS relative to acetolactate synthase (ALS) in gDNA by quantitative PCR (qPCR) revealed the same threshold cycle (Ct) for ALS and EPSPS in gDNA from susceptible plants and the same Ct for ALS in both resistant and susceptible plant gDNA. In contrast, the Ct for EPSPS in gDNA from resistant plants was six to seven cycles earlier than the Ct for ALS, suggesting that resistant plant genomes contain 64 to 128 times more copies of EPSPS than ALS. qPCR on cDNA revealed that EPSPS was expressed at 30 to 40 times higher levels in resistant plants. Elevated EPSPS copy number is heritable and correlates with expression level and resistance in F_2 populations. The molecular basis of resistance is likely due to increased production of EPSPS due to gene amplification. The possibility exists that one or a few genomic copies have higher expression due to promoter changes, or have a target-site mutation that has not been detected. This is the first documented occurrence of EPSPS gene amplification in a weed population under glyphosate selection pressure.

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