

The genetic architecture and genomic context of glyphosate resistance

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Abstract

Although much of what we know about the genetic basis of herbicide resistance has come from detailed investigations of monogenic adaptation at known target-sites, the importance of polygenic resistance has been increasingly recognized. Despite this, little work has been done to characterize the genomic basis of herbicide resistance, including the number and distribution of involved genes, their effect sizes, allele frequencies, and signatures of selection. Here we implement genome-wide association (GWA) and population genomic approaches to examine the genetic architecture of glyphosate resistance in the problematic agricultural weed, *Amaranthus tuberculatus*. GWA correctly identifies the gene targeted by glyphosate, and additionally finds more than 100 genes across all 16 chromosomes associated with resistance. The encoded proteins have relevant non-target-site resistance and stress-related functions, with potential for pleiotropic roles in resistance to other herbicides and diverse life history traits. Resistance-related alleles are enriched for large effects and intermediate frequencies, implying that strong selection has shaped the genetic architecture of resistance despite potential pleiotropic costs. The range of common and rare allele involvement implies a partially shared genetic basis of non-target-site resistance across populations, complemented by population-specific alleles. Resistance-related alleles show evidence of balancing selection, and suggest a long-term maintenance of standing variation at stress-response loci that have implications for plant performance under herbicide pressure. By our estimates, genome-wide SNPs explain a near comparable amount of the total variation in glyphosate resistance to monogenic mechanisms, indicating the potential for an underappreciated polygenic contribution to the evolution of herbicide resistance in weed populations.

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