

The SLIM1 transcription factor regulates arsenic sensitivity in *Arabidopsis thaliana*

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Abstract

The transcriptional regulators of arsenic-induced gene expression remain largely unknown; however, arsenic exposure rapidly depletes cellular glutathione levels increasing demand for thiol compounds from the sulfur assimilation pathway. Thus, sulfur assimilation is tightly linked with arsenic detoxification. To explore the hypothesis that the key transcriptional regulator of sulfur assimilation, SLIM1, is involved in arsenic-induced gene expression, we evaluated the response of *slim1* mutants to arsenic treatments. We found that *slim1* mutants were sensitive to arsenic in root growth assays. Furthermore, arsenic treatment caused high levels of oxidative stress in the *slim1* mutants, and *slim1* mutants were impaired in both thiol and sulfate accumulation. We also found enhanced arsenic accumulation in the roots of *slim1* mutants. Furthermore, microarray analyses identified genes from a highly co-regulated gene cluster (the O-acetylserine gene cluster), as being significantly upregulated in the *slim1-1* mutant background in response to arsenic exposure. The present study identified the SLIM1 transcription factor as an important component in arsenic-induced gene expression and arsenic tolerance. Our results suggest that the severe arsenic sensitivity of the *slim1* mutants is a result of both altered redox status as well as mis-regulation of key genes.

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