IN BRIEF

The WKRY6 Transcription Factor Is a Key Player in a Multifaceted Defense against Arsenate

Arsenic (As) is a highly toxic, nonessential metalloid that may have played a large role in the evolution of life (Oremland et al., 2009). Arsenate [As(V)] is taken up and moved through the plant by phosphate (Pi) transporters (reviewed in Mendoza-Cózatl et al., 2011). Previously, it was reported that exposure of Arabidopsis thaliana to As(V) led to specific repression of genes responsible for Pi uptake, including that for the high-affinity phosphate transporter PHT1;1 (Catarecha et al., 2007). New work from Castrillo et al. (pages 2944–2957) reveals that this repression is just one aspect of a rapid and sensitive As(V)-specific defense response.

Castrillo et al. sought to elucidate the mechanism responsible for As(V) repression of PHT1;1. PHT1;1 promoter-driven expression of the luciferase (LUC) reporter showed that Pi starvation induced LUC expression and that treatment with As(V), even at very low concentrations, reduced LUC expression even more than did treatment with Pi, consistent with the previous results that PHT1;1 is sensitive to As(V). Analysis of PHT1;1-GFP (for green fluorescent protein) fusion lines revealed that As(V) exposure led to PHT1;1 removal from the plasma membrane via endocytosis (see figure). The timing of the relocalization of the transporter to the vacuole lumen corresponded with that of a decrease in the cellular uptake of As(V), supporting the idea that this relocation represents a method to terminate As(V) uptake.

The authors then treated Pi-starved plants with As(V) or Pi and analyzed the promoter regions of those genes that were more responsive to As(V) than to Pi (similar to PHT1;1). They discovered a cis As(V) repression element (ARE), which is similar to the WK-box motif bound by WRKY transcription factors in plants. Indeed, WRKY6 gene expression was induced by As(V), and overexpression of WRKY6 caused a reduction of PHT1;1 expression. Furthermore, WRKY6 interacted with the ARE in the PHT1;1 promoter, and a wrky6 null mutant showed no As(V)-induced repression of PHT1;1. Together, these results suggest that WRKY6 acts as a transcriptional repressor of PHT1;1 in response to As(V).

Transcriptomic analysis also revealed that 869 transposon genes were induced by As(V). This transposon burst was repressed by WRKY6 overexpression, and many of the As(V)-induced transposons had WRKY binding sites in their promoters. The promoters of at least six of these transposon genes could be directly bound by WRKY6, suggesting that in addition to repressing PHT1;1, WRKY6 also represses transposon activation in response to As(V) stress.

Together, this work demonstrates that plants have evolved an As(V)-specific response wherein the high-affinity Pi transporter PHT1;1, which also transports As(V), is removed from the plasma membrane and is also transcriptionally repressed upon exposure to As(V). This dual regulation allows the plants to halt As(V) uptake quickly. WRKY6 mediates the transcriptional repression of PHT1;1 and further protects the plant by suppressing transposon activation in response to As(V) stress via directly interacting with transposon promoters. This work thus establishes WRKY6 as a key mediator of arsenate defenses in plants.

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REFERENCES


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