

IN BRIEF

Redox Regulation of Auxin Signaling and Plant Development

Redox signaling plays a critical role in relaying accurate information about a myriad of environmental conditions (including light, pathogens, herbivores, water status, and various abiotic stresses) toward optimizing plant cell metabolism and maintaining metabolic homeostasis. There is also indirect evidence that redox status affects hormonal signaling pathways controlling fundamental developmental processes, such as meristem maintenance and organogenesis (reviewed in Gapper and Dolan, 2006; Beveridge et al., 2007).

Now, **Bashandy et al. (pages 376–391)** provide evidence of a direct link between cellular redox status and auxin signaling, through their characterization of a triple mutant of *Arabidopsis* that is disrupted in key components of thioredoxin and glutaredoxin signaling. The mutant *ntra ntrb cad2*

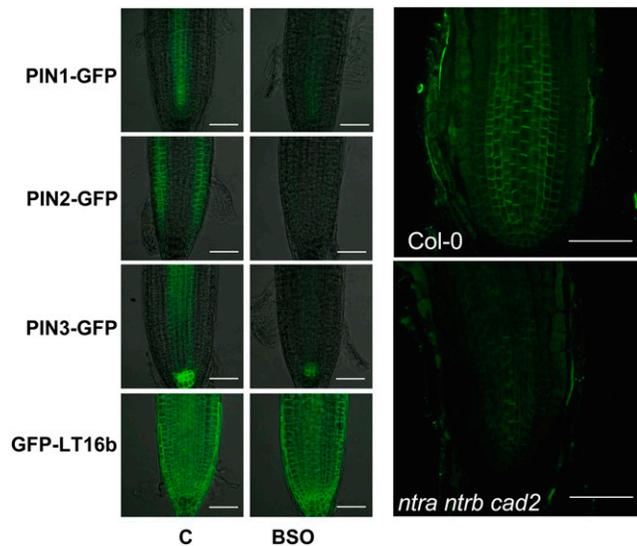
lacks both cytosolic NADPH thioredoxin reductases (*NTRA* and *NTRB*) and has severely reduced capacity for glutathione (GSH) biosynthesis (*cad2* represents a weak allele of *GSH1*, encoding the first enzyme in GSH biosynthesis). The authors found that the *ntra ntrb cad2* mutant grew normally at the rosette stage but failed to generate lateral organs from the inflorescence meristem, producing almost naked stems that were reminiscent of mutants affected in auxin transport or biosynthesis. The triple mutant exhibited other defects in processes regulated by auxin, including a loss of apical dominance, vasculature defects, and reduced secondary root production.

The authors go on to show that the capacity for auxin polar transport and the steady state levels of auxin are reduced dramatically in the triple mutant and that

exogenous auxin treatment rescues organogenesis and root development. In addition, the expression of PIN proteins involved in auxin transport is downregulated in the triple mutant and in wild-type *Arabidopsis* treated with BSO, a specific inhibitor of glutathione synthesis (see figure). Furthermore, it was found that exogenous application of GSH conferred partial rescue of flower development in the triple mutant. GSH was applied via root uptake through irrigation water, indicating that it could be transported from roots to the shoot to trigger flower development. The reduction in expression of auxin transport and response genes in the triple mutant relative to the wild type offered further evidence of a defect in auxin transport in the mutant inflorescence.

This work opens up an exciting area for future research into the role of GSH and redox signaling in regulating auxin transport, homeostasis, and auxin responses.

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GSH and redox status affect auxin transport gene expression. Left panels: wild-type plantlets expressing PIN-GFP fusion proteins treated (BSO) or untreated (C) with BSO. GFP fused to a plasma membrane protein (GFP-LT16b) was used as a control. Right panels: PIN1 immunolocalization in the wild type (Col-0) and triple mutant. Bars = 50 μ m. (Figure adapted from Figure 5 of Bashandy et al. [2010].)

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Plant Cell 2010;22;295; originally published online February 17, 2010;
DOI 10.1105/tpc.110.220212

This information is current as of April 10, 2021

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