

IN BRIEF

SAUR19 Links Auxin and Plasma Membrane H⁺-ATPases in Cell Expansion

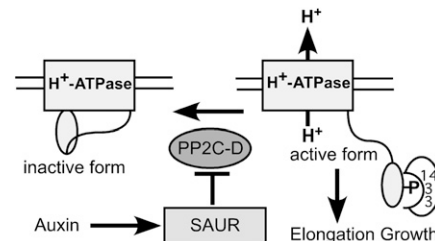
For decades, budding plant scientists have been captivated by accounts of classic experiments in which oat coleoptiles were clamped in equipment that resembled miniature medieval torture devices and stretched to the breaking point. These studies, which measured the extensibility of plant cells under various conditions, showed that hydrogen ions, like auxin, increase the growth and extensibility of cells and gave rise to the acid growth theory of cell elongation (Rayle and Cleland, 1970). According to this theory, auxin stimulates susceptible cells to pump protons into the apoplast, where the decrease in pH activates cell wall-modifying enzymes, including expansins, and hyperpolarizes the plasma membrane, promoting solute and water uptake and generating the turgor needed to drive cell expansion. Plasma membrane H⁺-ATPases are responsible for excreting hydrogen ions into the apoplast, and auxin is thought to boost this process both by activating existing H⁺-ATPases and stimulating the biosynthesis of new ones.

One aspect of the acid growth theory that has puzzled scientists is how auxin activates plasma membrane H⁺-ATPases. Takahashi et al. (2012) provided an important clue with the recent demonstration that auxin promotes phosphorylation of the C-terminal autoinhibitory domain of plasma membrane H⁺-ATPases. However, the underlying mechanism connecting auxin to H⁺ pump phosphorylation remained unclear. In this issue of *The Plant Cell*, Spartz et al. (pages 2129–2142) address this question. Having recently shown that SMALL AUXIN UP-RNA19 (SAUR19) family proteins are positive effectors of cell expansion that are localized to the plasma membrane (Spartz et al., 2012), the authors hypothesized that SAUR19 stimulates cell expansion by modulating the activity of plasma membrane H⁺-ATPases. They found that transgenic *Arabidopsis thaliana* plants that overexpressed stabilized SAUR19 fusion proteins (Pro35S:GFP-SAUR19) were remarkably similar to an *OPEN STOMATA2* mutant (*ost2-2*)

that harbored a constitutively active form of a plasma membrane H⁺-ATPase. Both lines exhibited increased growth and media acidification, as well as reduced apoplastic pH, suggesting that SAUR19 indeed activates plasma membrane H⁺-ATPases.

Given that plasma membrane H⁺-ATPase activation involves phosphorylation of its C-terminal autoinhibitory domain and subsequent binding of 14-3-3 proteins, the authors conducted a far-western gel blot analysis using GST-14-3-3 to examine the phosphorylation status of plasma membrane H⁺-ATPases in various lines. They found that phosphorylation of plasma membrane H⁺-ATPases was increased in transgenic lines overexpressing SAUR19 relative to the wild type, confirming that SAURs play a role in activating these H⁺-ATPases. To decipher the mechanism by which SAUR19 modulates the phosphorylation status of H⁺-ATPases, the researchers searched for SAUR19-interacting proteins using a yeast two-hybrid analysis. They identified three highly related type 2C protein phosphatases (PP2Cs) that interact with SAUR19 and found that these phosphatases also interacted with several additional SAUR proteins. After demonstrating that one of these phosphatases, PP2C-D1, was localized to the plasma membrane, the authors showed that SAUR19 and several other SAUR proteins inhibited the phosphatase activity of this protein. Furthermore, genetic, biochemical, and reconstitution experiments in yeast revealed that PP2C-D1 negatively regulates plasma membrane H⁺-ATPase activity by dephosphorylating the autoinhibitory domain.

Based on these findings, the authors present a revised model of cell expansion, in which SAUR proteins and PP2C-D phosphatases act antagonistically to regulate the activity of plasma membrane H⁺-ATPases (see figure). This work refines our understanding of auxin-induced extensibility and provides molecular evidence for the long-standing acid growth theory.



Model for the role of SAUR19 in promoting cell expansion. Auxin activates SAUR19, which then blocks the phosphatase activity of PP2C-D. Consequently, the proportion of active plasma membrane H⁺-ATPases in the active form increases, resulting in increased hydrogen ion efflux, activation of wall-modifying enzymes, increased turgor pressure, and cell expansion. (Adapted from Spartz et al. (2014), Figure 7D.)

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