Interestingly, mutations in the ARF C-terminal PB1 domain, or in the prion-like domain (PLD) in the MR that reduce multivalent interactions, abrogate condensate formation.[2] While not fully explored experimentally, this raises the possibility that the interaction of the MCTP C2-domain with the ARF7/19 MR reduces ARF multivalent interactions, thereby dissolving or preventing ARF condensation (Figure 1). While interference with multivalent interactions is a plausible mode of action, it remains to be seen if MCTP activities also regulate ARF stability. Notably, it will be of great interest to see how this MCTP-based regulation of ARF condensation connects to the previously identified AFF1-based mechanism, which has a prominent effect on ARF stability and nuclear import.[4]

Thus far, mutants in plant *MCTP* were mainly found to have defects in symplastic transport.[6,7] This raises the intriguing question of whether control of condensate formation could be functionally relevant for symplastic transport.[9] In this scenario, MCTPs would prevent or interfere with ARF condensate formation, not only to increase the pool of nuclear ARFs but also to allow intercellular transport of individual ARFs. The latter effect remains speculative, but is in line with reports on symplastic transport in auxin-regulated processes.[10]

While the principles of auxin signaling are well established, it remains enigmatic how different cells within a tissue can display differential auxin responsiveness. The identification of MCTPs as inhibitors of ARF7/19 condensation[5] represents a major step forward in our understanding of how cellular auxin responsiveness is determined.

DECLARATION OF INTERESTS

The authors declare no competing interests.

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Apical hook opening of plant seedlings: Unfolding the role of auxin and the cell wall

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Apical hook opening is crucial for seedling establishment and is regulated by unequal distribution of the hormone auxin through unknown mechanisms. In this issue of *Developmental Cell*, Walia et al.[4] demonstrate that apical hook opening is an output of tissue-wide forces; auxin and cell wall integrity (CWI) signaling interact to restrict elongation to the concave side of the apical hook.

In dicotyledonous plants, stem cells located in the shoot apical meristem are initially shielded by a structure known as the apical hook. After exposure to light, the apical hook unfolds to allow cotyledon expansion and photosynthesis

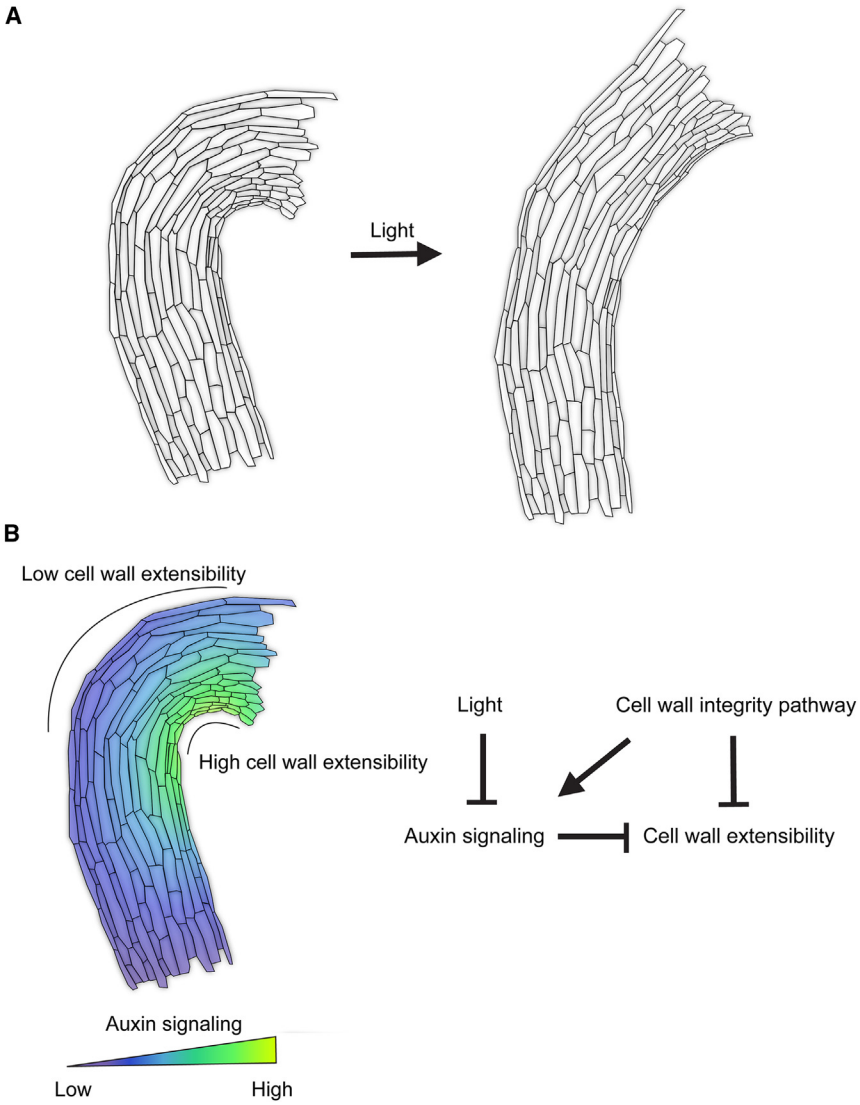

Figure 1. A network of inputs shapes apical hook opening

(A) Diagram of a young *Arabidopsis thaliana* seedling before and after exposure to light. Young seedlings curl as they grow up through soil, forming an apical hook. Light signals to open the apical hook by triggering differential elongation only on the short side.

(B) Higher cell wall extensibility in the short side of the apical hook allows only this side to elongate, following light perception. Cell wall extensibility is shaped by auxin and cell wall integrity signaling.

(Figure 1A). The plant hormone auxin is crucial for both the bending during the formation of the apical hook and its light-triggered opening. Elevated auxin levels on the concave side of the hook inhibit cell elongation and the resultant asymmetric cell expansion creates the hook in this tissue. Light exposure results in decreased auxin levels, allowing for regained cell expansion on the concave surface and hook opening.[1–3] In this issue of *Developmental Cell*, Walia et al.[4] demonstrate that cell walls in the concave side of the hook are mechanically more flexible, driving differential responsiveness to tissue-wide cues. Their results suggest that feedback between auxin and the cell wall integrity (CWI) surveillance pathways controls apical hook responsiveness, guiding growth during this crucial stage of seedling establishment (Figure 1B).[4]

Plant cell expansion is driven by the combination of turgor pressure and cell wall relaxation. Cell wall relaxation is linked to acidification of the apoplast, which consists of the cell wall and extracellular space. Auxin has long been known to regulate cell wall flexibility by adjusting the apoplastic pH, biosynthesis of cell wall polymers, and expression of cell wall structural proteins[3,5,6]; however, the exact auxin-related contributions to cell expansion during hook opening were previously unclear. To address this knowledge gap, Walia et al. used a ratiometric reporter of apoplastic pH to examine light effects on cell elongation during apical hook opening. Consistent with previous thinking on auxin effects on cell expansion, light triggered acidification of the apoplast.[3,6] However, in contrast to the observed differential growth, the authors found that apoplast acidification occurred across the entire apical hook, instead of being restricted to the concave side. Thus, although apoplast acidification is permissive for cell expansion, it is insufficient to explain apical hook opening. In fact, their genetic and chemical experiments demonstrate that the effects of apoplast pH and auxin signaling are uncoupled during apical hook opening. Further, inducible overexpression of the auxin biosynthesis gene *YUCCA6* (*YUC6*) reduced apoplast acidification in the light and inhibited apical hook opening. This effect could not be rescued by stimulation of apoplast acidification by fusicoccin, suggesting that processes beyond apoplast pH control gate cell expansion in apical hook opening.Auxin depletion is key to apical hook opening,[1] which complicates interpretation of the output of auxin signaling in this process. Auxin inhibits elongation of the cells in the apical hook,[1,6] but the concave-side cells with the most auxin signaling elongate the most. Seeking auxin-independent explanations of differential growth in apical hook opening, the authors combined computational modeling, cell ablation, and additional genetic and chemical experiments to probe how the cytoskeleton and cell wall properties are tuned in response to mechanical stresses. Their results suggest that light has a dosage-dependent effect on epidermis expansion by controlling the force exerted on it by subepidermal cells through an unknown mechanism. Epidermal cells are strained by expansion of the subepidermal cells and expand to relieve this stress. Their modeling also supports a role for light in controlling epidermal cell wall properties through auxin-dependent and -independent mechanisms. Cell wall stress and

the integrity of the cell wall are monitored by FERONIA (FER) and related receptor-like kinases.[3,7,8] Walia at al. found that the CWI pathway is required for apical hook opening after cell wall damage (simulated by isoxaben treatment, which inhibits cellulose biosynthesis). Cell wall damage slows apical hook opening, likely due to the reinforcement of the cell wall by the CWI pathway. Cellular cortical microtubules are essential for scaffolding the extracellular cell wall and to support delivery of wall components aligned according to mechanical stress vectors.[7,9] By imaging cytoskeletal dynamics, the authors show that inhibition of growth on the concave side increases the local stress on the epidermis. In concordance with these results, their modeling supports a view in which the force generated by subepidermal cell expansion during hook unfolding predominantly acts on the concave side of the apical hook. Therefore, the mechanical properties of the epidermal cell wall determined by auxin and CWI signaling constrain the extent of tissue elongation in response to this stress.

The tissue expansion driven by subepidermal cell growth predicted by Walia et al. is analogous to the forces driving leaf expansion.[9] However, leaves expand isotropically, and therefore rather than driving elongation, stress caused by subepidermal expansion is minimized by interdigitated "puzzle piece"-shaped leaf epidermal cells.[7,9] In leaf expansion, the auxin and CWI pathways are also closely linked. Auxin has a prominent role in the interdigitation of the leaf epidermis, through the control of Rho of Plants (ROP) GTPases.[10] FER and the CWI pathway are required for ROP6 function in pavement cell interdigitation.[7,8] How much the molecular function of auxin and FER in leaf cell wall modification may translate to apical hook unfolding is unknown, but the modeling of leaf dynamics generates anisotropic growth patterns[9] consistent with the linear expansion of apical hook cells.

Control of apical hook formation is well described, and also involves intimate connections between auxin signaling and the status of the cell wall.[2,3,5] Auxin controls pectin modification in the cell wall during apical hook formation in a feedback loop similar to what is shown by the CWI pathway.[6] FER binds to pectins as part of sensing the status of the cell wall,[8] suggesting that these two processes may share a common mechanism. In the future, this possibility could be explored by testing the involvement of demethylesterified pectin in apical hook opening.[6] Although a role for the peptide ligands of FER, the RAPID ALKALINIZATION FACTORs, remains to be demonstrated in apical hook opening,[5] they have the potential to tune growth during this critical period. In summary, the inhibition of auxin depletion and apical hook opening by the CWI pathway identified by Walia et al. provides a model that allows for continued apical hook maintenance during growth past obstacles in the soil, such as rocks or pebbles, even if light has been perceived, contributing to seedling establishment and navigation of their environment.

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DECLARATION OF INTERESTS

L.C.S. is on the scientific advisory board of Prose Foods.

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