# BRICK1/HSPC300 is required for cell elongation of protonema in *Physcomitrella patens*

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In animal cells, the Arp2/3 and SCAR/WAVE complexes are essential for cell motility as well as for a variety of endo-membrane processes. These complexes regulate actin dynamics, i.e. actin nucleation and filament branching. In plants, although neither complexes have been biochemically isolated, individual members of each complex have been associated with actin distribution and epidermal cell morphology. Using a reverse genetic approach, we try here to better understand the role of BRICK1, the smallest subunit of the SCAR/WAVE complex, in apical cell elongation of *Physcomitrella patens* protonemal filaments.

We deleted BRICK1 in *Physcomitrella patens* by targeted gene deletion and observed a striking reduction of filamentous tip growth in these lines (Δ*brk1*). Δ*brk1* was viable, completed, although slowly, the morphogenetic transition from filaments into leafy shoots, and exhibited proper orientation to polarized light. In order to localize BRICK1, we inserted YFP-BRK1 into Δ*brk1*. Transformed regenerants were able to totally rescue the mutant phenotypes and to localize BRICK1 exclusively to the site of polarized growth, at the tip of the apical cell. We were also able to fully restore Δ*brk1* to wild type using the Arabidopsis *Brick1* (*Atbrk1*) homologue. Further molecular analysis of the *Atbrk1* transcript in *Physcomitrella* showed proper intron recognition and splicing of the *Atbrk1* pre-mRNA by the moss splicing machinery.

To further understand the role of BRICK1 in apical tip extension, we asked whether the apical tip localization of two proteins required for apical cell extension were altered in Δ*brk1*. When ARPC4, a member of the Arp2/3 complex, was deleted, apical cell extension was prevented as in Δ*brk1* (1). When the cell wall arabinogalactan proteins (AGPs) were inactivated by the binding of Yariv reagent to the AGPs, apical tip extension is reversibly inhibited (2). Both ARPC4 and AGPs were not only required for apical cell extension, but are exclusively localized at the tip of the apical cell in WT. However, in Δ*brk1*, both were mis-localized and tip extension was prevented. Interestingly, YFP-BRICK1 was not mis-localized from the apical tip in Δ*arpc4*, indicating that the localization of BRICK1 itself is not sufficient for extension. Hence, a possible role of BRICK1 is to stabilize ARPC4 (and perhaps the Arp2/3 complex) at the site of apical cell extension, allowing polar growth.

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References:
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