

Plasmodesmata permeability unlocks the lateral root

Plants undergo costly developmental modifications when responding to stress. For example, phosphate starvation induces extensive lateral root remodeling to optimize phosphate acquisition from the rhizosphere. One poorly understood mediator of these responses is plasmodesmata. Plasmodesmata are dynamic channels linking adjacent cells by a strand of endoplasmic reticulum, linking most cells of the plant body in a continuous network, the symplast. To close or inhibit movement, callose is a polymer deposited at the channel opening, especially in response to phosphate starvation. However, there are callose-independent mechanisms that are largely not well understood, especially in response to stress. Moreover, the functional differences and interactions between callose-dependent and callose-independent mechanisms have not been characterized as well. This project explores the role that callose-independent mechanisms in regulating lateral root branching, stem cell quiescence, and SHORTROOT (SHR) cell-cell mobility in response to phosphate starvation. Notably, we found that promoted callose-independent mechanisms (using salicylhydroxamic acid, or SHAM) induced lateral root formation similarly in phosphate sufficient or phosphate starvation conditions. Moreover, SHAM recovered stem cell exhaustion under phosphate starvation. Furthermore, we quantified antagonistic effects on SHR cell-cell movement by SHAM in phosphate-starved roots. Together, these data suggest that callose, induced by phosphate starvation, can be bypassed by callose-independent mechanisms to simultaneously modulate lateral root inhibition and primary root stem cell exhaustion. Future work will clarify the key regulatory genes regulating callose-independent mechanisms. This will contribute to a greater understanding of how extensive developmental plasticity is regulated and mediated as it becomes increasingly vital to understand plant stress responses.