Phosphate-Deprived Roots Are Hypersensitive to Auxin

Low concentrations of phosphate (Pi) in soil trigger alterations to the architecture of the root system that increase a plant’s capacity to absorb Pi. Many plants stimulate the uptake of this essential nutrient by promoting lateral root (LR) formation, increasing root hair length and density (see figure), and suppressing primary root growth.

Auxin is a key player in LR development: exogenous auxins enhance LR formation (Blakely et al., 1988), and auxin transport inhibitors reduce it (Reed et al., 1998). At low concentrations of auxin, AUX/IAA repressors block the activity of AUXIN RESPONSE FACTOR (ARF) transcription factors. When auxin concentrations exceed a certain threshold, TIR1/AFB1-3 receptors trigger the destruction of AUX/IAA proteins. ARFs are then free to regulate the expression of auxin-responsive genes (reviewed in Lau et al., 2008), such as those involved in LR formation. These results suggest that Pi deprivation heightens the root system’s sensitivity to auxin.

Since the TIR1 auxin receptor is a central player in auxin perception, the group tested whether TIR1 is responsible for the increased auxin sensitivity of Pi-deprived seedlings. Mutant tir1-1 seedlings, which are unable to mediate AUX/IAA degradation, failed to increase LR production in response to Pi deprivation. A GUS fluorometric assay (see figure) and quantitative RT-PCR analysis revealed that TIR1 is specifically induced in response to Pi deprivation. By examining a series of mutants, other members of the TIR1/AFB1-3 family were shown to have partially redundant roles in LR formation in response to Pi deprivation, and ARF19 was found to be essential for increased LR formation in Pi-deprived seedlings. An experiment using transgenic Arabidopsis seedlings expressing a heat-inducible AUX/IAA protein demonstrated that Pi deprivation accelerates the degradation of the AUX/IAA protein.

Thus, auxin sensitivity appears to be enhanced in Pi-deprived seedlings due to the increased expression of TIR1. This is proposed to cause the degradation of AUX/IAA proteins, which liberates ARF transcription factors and modulates the expression of genes involved in LR formation.

REFERENCES


