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Root architecture remodeling induced by phosphate starvation

Aiko Sato and Kenji Miura*

Graduate School of Life and Environmental Sciences; University of Tsukuba; Tsukuba Japan

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Plants have evolved efficient strategies for utilizing nutrients in the soil in order to survive, grow and reproduce. Inorganic phosphate (Pi) is a major macroelement source for plant growth; however, the availability and distribution of Pi are varying widely across locations. Thus, plants in many areas experience Pi deficiency. To maintain cellular Pi homeostasis, plants have developed a series of adaptive responses to facilitate external Pi acquisition, limit Pi consumption and adjust Pi recycling internally under Pi starvation conditions. This review focuses on the molecular regulators that modulate Pi starvation-induced root architectural changes.

Introduction

Although a sufficient supply of inorganic phosphate (Pi) is vital to plants, plants often experience fluctuations in Pi availability or even chronic Pi limitation.[1,2] Pi is important for the production of nucleic acids, phospholipids, proteins and small molecules.[3] Because plants are sessile organisms, they have evolved an array of adaptive responses to adjust their growth, development and metabolic activities. These adjustments include the following: (1) enhanced top soil foraging by modulating root architecture components, such as increasing the growth of lateral roots and number of root hairs to increase the absorption surface area, and by establishing a symbiotic association with mycorrhizal fungi; (2) improved Pi uptake by activating Pi transporters and increased Pi recycling and scavenging for phosphate from intra- and extracellular organic sources by secreting organic acid, ribonuclease and acid phosphatases; and (3) optimization of Pi use by a wide range of metabolic alterations.[4] It is presumed that the primary root tip senses low Pi availability and initiates a signaling cascade.

The application of phosphorus (P) fertilizer can compensate for low Pi availability, but the global resource of P from rock is not renewable and may be depleted in 50–100 years.[5] Although P is exogenously fertilized, most P is integrated with Ca, Fe or Al salts and formed as organic molecules, whereas only orthophosphate, mainly $H_2PO_4^-$ and HPO_4^{2-}, is available for plants. For a typical plant, Pi are accumulated in concentration of about 1 μM in the soil, 10,000 μM in the cells and 400 μM in the xylem. To understand the strategies used by plants to acquire and utilize Pi efficiently is essential for the rational breeding and engineering of crop plants with greater capacity to acquire, store and recycle soil Pi. This review is focused on recent advances in our understanding of the mechanism of root architecture remodeling under Pi deficiency.

*Correspondence to: Kenji Miura; Email: kmiura@gene.tsukuba.ac.jp
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Regulators Involved in Pi Starvation-Induced Root System Architecture Modulation

When a primary or lateral root encounters low Pi levels, its growth is restrained. This inhibition results from reduced cell elongation[6] and meristem activity.[7-11] This response requires physical contact between the root tip and the low-Pi medium.[9,12] *CycB1;1::GUS* seedlings, which identifies dividing cells at the G_2/M transition, demonstrates that cells in the root tips ceased dividing when the root tips encountered the low-Pi medium.[9] Thus, it is presumed that a local Pi sensing mechanism exists in the roots, whereas alterations in the development of lateral roots are regulated by systemic Pi status.[13] Auxin is believed to play a central role in root architecture modulation during Pi starvation because a change in auxin distribution was observed during Pi starvation,[11,14,15] auxin-accumulating *YUCCA1*-overexpressing plants[16] exhibited hyperresponsivenesss to Pi starvation,[11] and both TIR1 (transport inhibitor response 1)- and ARF19 (auxin response factor 19)-dependent auxin signaling are implicated in the regulation of lateral root development under Pi deficiency.[17] Exogenous auxin treatment in plants grown in low Pi conditions drastically prevents the growth of primary roots and induces the formation of lateral roots.[11,18] Furthermore, mutations in *BIG*, which encodes a large calossin-like protein that is required for normal polar auxin transport,[19] reduced lateral root formation in low Pi conditions.[15] Auxin transport and BIG function have fundamental roles in pericycle cell activation, which stimulates lateral root primordia formation and promotes root hair elongation.[15]

Several mutants have been isolated and studied to understand Pi starvation-induced root architecture modulation. The EMS-induced Arabidopsis *pdr2* (*phosphate deficiency response 2*) mutant, which has impaired ER-localized P_5-type ATPase function,[20] exhibited reduced primary root growth and root cell division and elongation under Pi deficiency.[21,22] The *pdr2* mutant was not affected by the P concentration within the root tip or by Pi uptake rates, excluding a defect in high-affinity Pi acquisition,

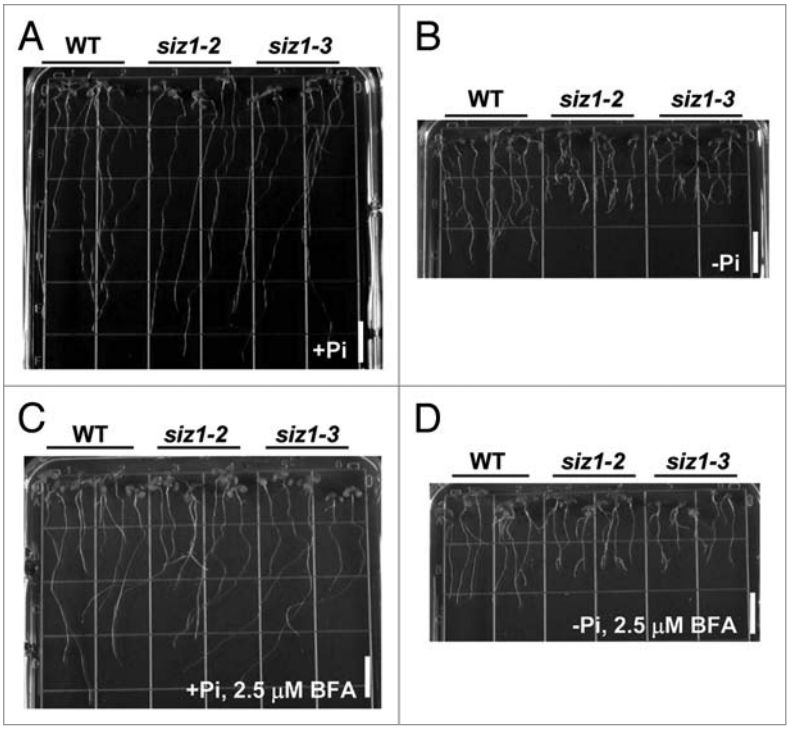

Figure 1. The *siz1* mutants exhibited exaggerated prototypical Pi starvation responses, including cessation of primary root elongation and extensive lateral root development (B), although no obvious difference between wild-type and *siz1* seedlings under Pi sufficient condition (A). Treatment with brefeldin A (BFA) suppressed lateral root formation in low Pi (D) of wild-type and *siz1* seedlings, compared to that in high Pi condition (C). Bars indicate 1 cm length.

suggesting that the *pdr2* mutant phenotype is caused by a defect in sensing the local external Pi concentration.[22] PDR2 regulates stem cell differentiation and meristem activity through SCR and SHR,[20] which are GRAS family members and key regulators of radial root patterning.[23,24]

Genetic data suggest an interaction between *PDR2* and *LPR1/LPR2* (*low phosphate root1/2*), which encode multicopper oxidase,[9] in an ER-resident pathway;[20] however, LPR1 and PDR2 could have opposing functions in regulating the root growth response to Pi starvation.[25] Quantitative trait loci analyses revealed that *LPR* loci were detected in a recombinant inbred line population derived from a cross between the Pi starvation-insensitive accession Bay-0 (Bayreuth) and the Pi starvation-sensitive accession Sha (Shahdara) in Arabidopsis.[9] *LPR1* is expressed in the root tip, including the meristem and root cap. Mutations in *LPR1* and *LPR2* strongly reduced the Pi-induced inhibition of primary root growth,[9,25] suggesting that LPRs modify or activate a compound in the root tip that inhibits meristematic growth. The function of *LPR1* is genetically independent on *SIZ1* (*SAP and Miz1*) in response to Pi starvation.[25]

SIZ1 is a SUMO (small ubiquitin-related modifier) E3 ligase that is involved in Pi starvation responses[26] as well as several stress responses[27-33] and flowering.[34] The *siz1* mutation exaggerates prototypical Pi starvation responses; including cessation of primary root growth, extensive lateral root development and root hair development.[26] Root architecture modulation is likely caused by the regulation of auxin patterning in response to Pi limitation.[11] Application of 1-*N*-naphthylphthalamic acid, an inhibitor of auxin efflux activity, reduced the Pi starvation-induced lateral root elongation effects of *siz1*. Treatment with brefeldin A, an inhibitor of vesicle trafficking, also reduced the lateral root formation induced by *siz1* in response to Pi limitation (**Fig. 1**). Brefeldin A disrupts the polarization of the auxin transporter PIN1, resulting in PIN1 being equally distributed along the entire cell surface and reduced auxin efflux activity.[35] *SIZ1* negatively regulates root architecture modulation under low Pi by controlling auxin patterning.

Microarray analyses demonstrated that genes encoding EXP17 (expansin 17), GH1 (glycosyl hydrolase 1), embryo-abundant, dehydrin xero 2, UGT73B4 (UDP-glycosyltransferase73B4) and PGIP (polygalacturonase inhibiting protein) as well as GST (glutathione transferase) were induced by phosphate deficiency, auxin treatment and the *siz1* mutation.[11] UGT73B4, PGIP and GST are known to be involved in disease resistance. Cell wall remodeling enzymes, such as pectate lyase, polygalacturonase, xyloglucan:xyloglucosyl transferases, expansins and GHs, are expressed in root cells adjacent to new lateral root primordia, presumably to promote the emergence of LR elongation.[17,36] For cell growth, the cell wall must be loosened. Expansins are plant cell wall-loosening proteins involved in cell enlargement and cell modification.[37] GHs have large families and diverse functions.[38] GH family 1 includes β-mannosidase, β-glucosidase, thioglucosidase and glycosyltransferases. β-Glucosidase hydrolyses the exo-cellulase product into individual monosaccharides. The precise biological function of these proteins is unknown, but it is possible that some of these products are involved in cell wall loosening, resulting in elongation of the lateral root.

Three transcription factors, WRKY75, Cys2/His2 zinc-finger transcription factor ZAT6 and MYB62, have been identified[39-41] on the basis of microarray data.[42] *WRKY75* RNAi-lines exhibit increased growth of lateral roots and the number of root hairs. *WRKY75* expression was induced by Pi limitation. Because WRKY75 is also involved in disease resistance,[43] a relationship may exist between nutrient deficiency and defense mechanisms. *ZAT6* was induced during Pi starvation and became localized to the nucleus.[40] Because the RNAi suppression of *ZAT6* was lethal, the gene is vital for plants. Overexpression of *ZAT6* resulted in a constitutive exaggerated Pi starvation-induced root architecture modulation with a significant decrease of primary root length and the number of lateral roots, but with a strong increased lateral root length.[40] The target genes for ZAT6 are not yet known, but ZAT6 may function as a transcriptional repressor to repress primary root growth during Pi deficiency. Pi starvation also induced *MYB62* in the leaves.[41,42] *MYB62*-overexpressing plants exhibited shorter lateral roots, diminished shoot growth rate, decreased Pi accumulation in the shoot and attenuated

regulation of Pi starvation-induced genes.[41] Interestingly, the growth retardation induced by *MYB62* overexpression was reversed by the application of gibberellic acid, suggesting that MYB62 also regulates GA biosynthesis. Other microarray data demonstrated that the expression of *bHLH32* (basic helix-loop-helix) transcription factor was induced after 48 h of Pi starvation.[44] The *bhlh32* mutant exhibited increased Pi accumulation, higher anthocyanin levels and increased numbers of root hairs in Pi-sufficient conditions and also higher activity of PPCK, a protein kinase that activates PEP carboxylase,[45] suggesting that bHLH32 is a repressor of the Pi deficiency response. Because *WRKY75*, *bHLH32* and *MYB62*, which appear to be negative regulators of the Pi starvation response, were induced by Pi deficiency, these factors may be involved in feedback mechanisms that attenuate the response in certain tissues.

The mutation in *RPD* (phosphate root development), which encodes an AINTEGUMENTA-like protein, repressed the development of primary and lateral roots under Pi starvation.[46] Because Pi influx and Pi starvation-inducible gene expression were similar in wild-type and the *prd* plants in response to Pi starvation, it is suggested that *PRD* is not a checkpoint gene for Pi starvation responses but acts as a regulator of root architectural responses to Pi starvation.[46]

The nuclear actin-related protein ARP6 is required for the SWR1 chromatin-remodeling complex, which regulates transcription via deposition of the H2A.Z histone variant into chromatin. Mutation of *ARP6* decreased H2A.Z abundance in a number of Pi starvation-responsive genes, resulting in increases in gene transcription and correlating with Pi starvation-related phenotypes, such as shortened primary roots and increases in the number and length of root hairs.[47] These results suggest that SWR1-dependent H2A.Z deposition is required for the modulation of root system architecture as well as the regulation of Pi starvation-induced gene expression.

Several mutant genes remain unidentified. The *lpi* (low phosphorus-insensitive) mutants, representing four different genetic loci, were isolated from an EMS-mutagenized population due to their ability to maintain primary root growth, leading to a long primary root, during Pi deficiency.[7] These mutants exhibited reduced induction in the expression of several Pi starvation-induced genes. Gene expression profiling with the *lpi4* mutant demonstrated that the large number of genes related to oxidative stress were downregulated in the root tip of *lpi4* plants.[48] Dramatic decrease in H_2O_2 levels occur during the meristem exhaustion process in the primary root tip of the wild-type plants grown in low Pi conditions, whereas in *lpi4* plants, H_2O_2 accumulation was observed even under Pi deficiency, suggesting that Pi starvation triggers alterations in redox status, leading to the loss of root elongation and meristem maintenance.[48] Thus, an appropriate redox status may be required for primary root meristem maintenance.

Several lines of evidence suggest that signaling mechanisms for Pi accumulation in the shoot and root architecture modulation may not be linked because the *pho1* and *pho2* mutants, which accumulate less and more Pi, respectively,[49,50] exhibited a similar root phenotype as wild-type seedlings. miRNA399 overexpression, which targets *PHO2* encoding the ubiquitin E2 conjugate enzyme UBC24 and results in increased accumulation of Pi,[51,52] did not result in a hyperresponsive root architecture phenotype in plants.[51] miRNA399-PHO2 signaling is regulated by the MYB-type transcription factor PHR1.[53] The *phr1* mutant accumulated less Pi but exhibited a similar phenotype of root architecture as wild-type plants.[7,54] Thus, it is plausible that the PHR1-miRNA399-PHO2 pathway controls Pi accumulation in shoots but is not involved in the regulation of root architecture modulation.

Phytohormones and Modulation of Root Architecture in Pi Deficiency

Auxin and the associated polar auxin transport mechanism are known to be essential for lateral root formation[55,56] and play an important role in modulation of root architecture in response to Pi starvation as described above.

Additional phytohormones are also involved in the regulation of root architecture modulation. In addition to the decrease in cytokinin levels upon Pi starvation,[57] the exogenous application of cytokinin reduced the expression of several Pi starvation-responsive genes,[58] as well as inhibited lateral root formation,[59] suggesting a role for cytokinins in the negative regulation of Pi starvation responses. Cytokinin modulated the level of meristem cell cycle, which influences the expression of Pi starvation-responsive genes.[10]

Ethylene is also involved in primary root elongation and root hair formation in seedlings grown in Pi-limited medium.[60,61] However, analysis of the root architecture of ethylene-signaling mutants and plants treated with the ethylene precursor 1-aminocyclopropane-1-carboxylic acid demonstrated that ethylene did not promote the formation of lateral roots under Pi starvation.[18] Ethylene, in combination with jasmonic acid, may be involved in the meristem exhaustion process triggered by Pi starvation.[48]

Gibberellin plays an important role in regulating plant growth and development.[62] The binding of bioactive gibberellin to the receptors GID1 (gibberellin-insensitive dwarf 1) proteins promotes interaction between these gibberellin receptors and DELLAs.[63] The DELLAs are subsequently polyubiquitylated by the $SCF^{SLY1/SLY2}$ E3 ubiquitin ligase and thus degraded by 26S proteasome.[64,65] The gibberellin-DELLA growth regulatory system contributes to plant growth responses to Pi starvation.[66] Exogenous gibberellin application overcomes several characteristic growth responses to Pi starvation, including the recovery of primary root inhibition and decrease in lateral root density. DELLA-deficient mutants did not exhibit Pi starvation growth responses, whereas the mutations that enhance DELLA function promote Pi starvation responses.[66] Pi starvation promoted the accumulation of DELLA in root cell nuclei.[66] These results suggest that gibberellin-DELLA-dependent signaling contributes to root architecture modulation in response to Pi starvation but not to Pi uptake and the regulation of Pi starvation-responsive gene expression. Bioactive gibberellin levels were also reduced in low Pi conditions.[66]

Conclusion and Perspectives

Much progress has been made in research on Pi deficiency-induced root architecture remodeling and several reports suggest that the root tip is a site for locally sensing the status of Pi deficiency. The functional analyses of the different root tissues of the root tip are required to identify the early steps of Pi starvation responses. Several phytohormones, particularly auxin, are involved in the modulation of root architecture adaptation.

The roots of more than 80% of the vascular flowering plants, excluding Arabidopsis, can be colonized by arbuscular mycorrhizal fungi. Symbiosis improves plant mineral, essentially Pi, acquisition. Lateral root branching is stimulated by arbuscular mycorrhizal fungi symbiosis.[67] Precise mechanisms of root branching and fungi symbiosis may lead to a better understanding and may help to improve crop growth under Pi starvation.

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