Abiotic stress series

Short on phosphate: plant surveillance and countermeasures

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Metabolism depends on inorganic phosphate (Pᵢ) as reactant, allosteric effector and regulatory moiety in covalent protein modification. To cope with Pᵢ shortage (a common situation in many ecosystems), plants activate a set of adaptive responses to enhance Pᵢ recycling and acquisition by reprogramming metabolism and restructuring root system architecture. The physiology of Pᵢ starvation responses has become well understood, and so current research focuses on the initial molecular events that sense, transmit and integrate information about external and internal Pᵢ status. Recent studies have provided evidence for Pᵢ as a signaling molecule and initial insight into the coordination of Pᵢ deficiency responses at the cellular and molecular level.

Phosphorus, the ‘bearer of light’ in Greek mythology, is also the conveyor of light in plant biology. The assimilated and fully oxidized form of the element, inorganic phosphate (Pᵢ), is indispensable for coupling the light and dark reactions in photosynthesis, and thus plays a pivotal structural and regulatory role at the nexus of energy conservation and carbon assimilation. As a consequence, Pᵢ availability affects nitrogen and sulfur assimilation, which both require an adequate carbon supply [1]. The chemical stability and cellular retention of nucleic acids, phospholipids and many metabolites are dependent on Pᵢ as a building block because Pᵢ is the only inorganic anion that forms charged diesters at physiological pH [1]. It is therefore not surprising that Pᵢ availability profoundly affects plant metabolism and crop performance.

Although phosphorus is abundant in the lithosphere, the physicochemical properties and soil chemistry of Pᵢ render it one of the least available nutrients. Soil concentrations of soluble Pᵢ are often up to a 1000 times lower than those of other required ions [2,3]. Phosphorus malnutrition poses a severe threat to agriculture that is typically averted in affluent countries by extensive application of concentrated Pᵢ fertilizers. However, crop yields are severely depressed by Pᵢ deficiency in large parts of the world with poor soils and limited economic resources [2].

To cope with inadequate Pᵢ supply, plants activate a set of adaptive responses that reprioritize internal Pᵢ use and maximize external Pᵢ acquisition. Such countermeasures include adjustment of metabolism to protect intracellular Pᵢ homeostasis and redesign of root system architecture to accelerate soil exploration. Sensory mechanisms that monitor environmental Pᵢ status and transmit the nutritional signal in Pᵢ rescue efforts are largely unknown. However, substantial progress has been made during the past few years, which promises to unravel plant Pᵢ sensing. In this review, we first highlight how plants counteract Pᵢ shortage and then discuss inroads into the underlying surveillance systems.

Biochemical responses

Internal Pᵢ conservation

When Pᵢ is limiting, plant metabolism adjusts to maintain cytosolic Pᵢ concentrations [2–6]. To secure carbon flow through glycolysis, alternative reactions that do not require Pᵢ or adenylates become more prevalent. Such bypasses are catalyzed by a pyrophosphate-dependent phosphofructokinase, a nonphosphorylating glycerol-3-phosphate dehydrogenase that shortcuits flux to glyceraldehyde-3-phosphate, and a suite of enzymes that convert phosphoenolpyruvate to pyruvate without the need for ADP [5]. In some species, Pᵢ deficiency activates alternative oxidase to regenerate NAD⁺ when oxidative phosphorylation is slowed by decreased Pᵢ and ADP levels [2].

Accumulation of starch, anthocyanins and other secondary metabolites are typical Pᵢ starvation responses that recycle substantial amounts of Pᵢ from phosphorylated precursors and condition the nutrient-stressed plant against photoinhibition and other environmental challenges [5]. Although increased anthocyanin production is considered to be a stress response, analysis of the phr1 mutant in Arabidopsis points to the existence of multiple anthocyanin-inducing pathways, including a specific Pᵢ-sensitive route [7].

Phospholipids, nucleic acids and small metabolites contribute about equally to the organic phosphorus content of a leaf [8]. It is therefore not surprising that membrane lipid composition and nucleic acid metabolism are attuned to balance Pᵢ economy. Unlike in other eukaryotes, non-phosphorous lipids are prominent constituents of plant membranes, probably reflecting an evolutionary adaptation to latent Pᵢ deficiency [8]. To conserve Pᵢ in conditions of acute Pᵢ deprivation, the phospholipid content of thylakoid and extraplastidic membranes is further reduced and replaced by galactolipids and...
sulfolipids [8–10]. Digalactosyldiacylglycerol is an example of this and can account for up to 70% of the total plasma membrane lipid content in roots of P<sub>i</sub>-starved oat plants [11]. P<sub>i</sub> deficiency leads to the coordinated induction of various substrate-specific phosphatases and nucleases that recycle P<sub>i</sub> from intracellular metabolites and nucleic acid substrates such as rRNA [2,4,5].

**External P<sub>i</sub> mobilization**

A set of phosphohydrolases is induced and secreted to mobilize P<sub>i</sub> from external organic matter (Figure 1). As shown for tomato cell cultures, the combined action of P<sub>i</sub>-starvation-inducible (PSI) enzymes such as ribonucleases and other phosphoesterases allows the efficient use of RNA in the medium [12]. Likewise, Arabidopsis plants develop normally on a P<sub>i</sub>-free, RNA-containing agar, whereas the conditional pdr1 mutant, which is defective in the induction of multiple PSI genes, requires P<sub>i</sub> supplementation [13]. Additional evidence for the importance of P<sub>i</sub> mobilization from external organic sources is provided by the pup mutants [14] and by transgenic Arabidopsis plants that secrete bacterial phytase for better hydrolysis of external phytic acid [15]. While secreted phosphohydrolases release P<sub>i</sub> from organic substrates, which contain up to 80% of the total soil phosphorus, exudation of organic acids such as citrate and malate facilitates chelation of Fe<sup>3+</sup>, Al<sup>3+</sup> and Ca<sup>2+</sup> cations followed by P<sub>i</sub> mobilization from insoluble salts and other soil complexes [2–4,16]. High-affinity P<sub>i</sub> transporters are induced in response to P<sub>i</sub> limitation and are responsible for uptake of mineralized phosphorus against a steep (up to 10 000-fold) concentration gradient [3,17,18].

**Metabolic networks**

Biochemical adjustments in response to P<sub>i</sub> limitation are instituted by metabolic control and regulation of gene expression [2–4,19–21]. Various PSI genes have been cloned whose steady-state mRNA levels are affected by altered P<sub>i</sub> availability [2–4,20,21]. Genome-wide expression studies confirmed observations for individual genes and allowed a more comprehensive reconstruction of P<sub>i</sub>-responsive metabolic networks [22–27]. For example, 29% of the 6172 Arabidopsis genes analyzed showed altered expression levels in plants experiencing P<sub>i</sub> deprivation [22]. Genes with roles in photosynthesis and nitrogen assimilation were coordinately repressed within 24 h of P<sub>i</sub> withdrawal. The resulting carbon starvation caused repression of genes necessary for the biosynthesis of starch, proteins, fatty acids and lipids. By contrast, genes encoding enzymes for the degradation of proteins, fatty acids, lipids and isoprenoids were induced, as were genes with roles in carbon transport, sucrose synthesis and glycolytic bypass reactions [22]. Similar analyses in Lupinus albus [26] and Arabidopsis [23] also indicated that secondary metabolism had a greater role in P<sub>i</sub> deficiency responses. The Arabidopsis study distinguished between early (4 h) and late (>28 h) responding genes: whereas many late genes were P<sub>i</sub> specific, the early group was enriched with general stress-response genes [23]. However, these first studies monitored only a proportion of the transcriptome after challenging P<sub>i</sub>-sufficient plants with P<sub>i</sub> deprivation. The kinetics of gene expression in response to P<sub>i</sub> withdrawal is confounded by the shielding effect of internal P<sub>i</sub> stores. Profiling experiments that examine the entire transcriptome of P<sub>i</sub>-starved plants after P<sub>i</sub> replenishment should

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**Figure 1.** Arabidopsis genes involved in inorganic phosphate (P<sub>i</sub>) deficiency responses. P<sub>i</sub>-starvation-inducible (PSI) genes are coordinately activated by low P<sub>i</sub> supply [20]. PHR1 (a MYB transcription factor) and CRE1 (a cytokinin receptor) regulate the expression of a subset of PSI genes [7,67]. The pup (phosphatase under-producer) mutants are defective in acid phosphatase production and in mobilizing P<sub>i</sub> from organic phosphorus sources [14]. High-affinity P<sub>i</sub> transporters (Pht1) facilitate P<sub>i</sub> uptake by roots (blue cone). PDR2 (PHOSPHATE DEFICIENCY RESPONSE 2) is hypothesized to regulate the activity of root meristems (red disc) in response to local P<sub>i</sub> availability [49]. The PHT1 protein product (an SPX domain-containing protein) is necessary for loading P<sub>i</sub> into the xylem (brown rectangle) and for its transport from the root to the shoot (green square) [73,74]. The pho2 mutant overaccumulates P<sub>i</sub> in the shoot, and the PHO2 protein is thought to regulate P<sub>i</sub> recycling from the shoot to the root [68]. Additional classes of P<sub>i</sub> transporters (Pht2, Pht3) are responsible for the cellular distribution of P<sub>i</sub> throughout the plant [17]. Solid arrows indicate transport processes or chemical reactions. Broken arrows designate regulatory interactions.
provide important insight into metabolic responses to Pi availability, particularly as wild-type plants and Pi sensing mutants are compared.

**Developmental strategies**

**Remodeling of root system architecture**

Although biochemical adjustments to Pi deprivation enhance Pi recycling and acquisition, restructuring the root system architecture and increasing the root-to-shoot growth ratio improve soil exploration. Pi availability impacts all aspects of root development via changes in root meristem activity. Pi-dependent root growth has been studied in some detail in common bean (Phaseolus vulgaris) [28] and Arabidopsis [29–32]. Pi deprivation favors lateral over primary root growth, a response known as topsoil foraging [28], which is believed to maximize Pi acquisition as the macronutrient becomes more limiting with increasing soil depth [2] (Figure 2).

Although Arabidopsis provides a powerful system for dissecting root development, one of the most dramatic responses to limiting Pi occurs in only a select group of plant species. Clusters of lateral roots, so-called proteoid roots, are principally formed by members of the Proteaceae that are adapted to environments with extremely low soil fertility. Determinate, short-lived clusters of abundant rootlets with dense root hairs arise from the pericycle and provide a considerable increase in absorptive surface area. Cluster roots are short and therefore assist in Pi uptake only from the immediate vicinity of the root. However, they are exquisitely adapted for exploiting the patchiness of Pi availability in the soil. Studies with Lupinus have shown that cluster roots actively secrete organic acids, acid phosphatases and phenolic compounds, which facilitate the mobilization of bound forms of Pi [2,33].

**Extending the root–soil boundary**

The limited mobility of Pi in soil necessitates a larger absorptive surface for Pi acquisition. Root hairs contribute significantly to the root surface area (up to 80%) and are the primary site of Pi uptake in species that do not form mycorrhizal associations [34]. Plants respond to low Pi by increasing both the length and the density of root hairs [28,30,35–37]. The importance of root hairs for Pi acquisition and plant growth has been demonstrated using Arabidopsis mutants with defects in root hair development [38,39]. However, most plants take advantage of specialized fungi to access scarce Pi from soil [40]. Symbiotic associations with mycorrhizal fungi considerably extend the root surface and increase Pi supply to the plant in exchange for carbon. The mechanisms that allow this exchange are beginning to be revealed. Pi transporters have been isolated that are induced in mycorrhizal roots [41–43] and one such transporter, MtPT4, was localized specifically to the periarbuscular membrane [41].

**Surveillance of Pi availability**

**Pi as a signal**

Physiological studies have established a framework of Pi deficiency responses that promote plant survival and reproduction. We are now beginning to understand how Pi or its absence is perceived at the molecular level. The central role of Pi in metabolism complicates deconvolution of its intertwined functions as reactant, allosteric effector and regulatory moiety in covalent protein modification. However, a series of recent reports indicates that Pi acts as a signal rather than a reactant by mass action to regulate the diverse responses to Pi limitation. Such evidence is provided by observations that phosphite (H2PO3−, the reduced but metabolically inert form of Pi) selectively attenuates metabolic and developmental Pi deficiency responses [44–49]. The value of phosphite as an experimental tool for dissecting Pi signaling was first recognized by William Plaxton’s group, which showed that phosphite is readily absorbed by Brassica plants but is not significantly oxidized to Pi or otherwise metabolized. Furthermore, the work in Brassica indicated that phosphite represses typical Pi deficiency responses in spite of its inability to substitute Pi as a nutrient [44,45]. Studies in other plant species including Arabidopsis corroborated and extended these early observations. Adaptive responses that are similarly regulated by Pi and phosphite include anthocyanin accumulation, expression of multiple PSI genes and enzymes, root hair development, proteoid root formation, and root meristem activity [46–49]. The effects of Pi and phosphite on meristem activity are best revealed in the pdr2 mutant, which displays a conditional, Pi-dependent short root phenotype [49] (Figure 2). As indicated by expression of a transcriptional fusion between cyclin B1 and β-glucuronidase (CYCLINB1::GUS), the pdr2 phenotype is caused by selective inhibition of root cell division below 0.1 mM external Pi, followed by meristem death. However, presence of phosphite above this threshold maintains root meristem activity and suppresses cell death in Pi-starved pdr2 root tips, suggesting that a Pi signaling mechanism regulates root meristem activity [49].

The repression of molecular and developmental Pi starvation responses by phosphite is selective [44–46]. Although phosphite does not participate in enzyme-catalyzed phosphoryl group transfer reactions, other Pi-binding proteins apparently fail to discriminate between Pi and phosphite. Such proteins might function as Pi transporters or as Pi signal transduction components [5,50]. The analysis of yeast pho mutants suggests that phosphite targets a high-affinity Pi transporter that is part of a putative Pi-sensor complex [50]. The data in yeast and plants are consistent with the hypothesis that phosphite simulates a state of Pi sufficiency by interfering with early events in Pi signaling. The potential of phosphite as a selective inhibitor of Pi starvation responses remains to be further exploited.

**Local Pi sensing**

The heterogeneous distribution and low mobility of Pi in soil forces plants to monitor Pi fluctuations in the rhizosphere with high spatial resolution. Although most of the well-established adaptations to Pi limitation appear to be controlled by whole plant Pi status, mounting evidence indicates that metabolic and developmental responses can be triggered locally by external Pi availability.

Root hair development provides evidence for cell-autonomous perception of external Pi. Experiments with Arabidopsis have demonstrated that sporadic Pi availability
Figure 2. The impact of inorganic phosphate (P$_i$) availability on root system architecture in Arabidopsis. (a) P$_i$ limitation slows primary root growth (vertical green arrows) by attenuating meristem activity (red discs) and promotes lateral root proliferation, which leads to the development of a shallower, more branched root system [29–31,49]. Although the pdr2 (phosphate deficiency response 2) root phenotype is indistinguishable from the wild type under high P$_i$ conditions, the root system of pdr2 seedlings is hypersensitive to P$_i$ deprivation. Altered root system architecture of pdr2 in low P$_i$ can be explained by an unrestrained inhibition of root cell division followed by meristem death (broken circles), and by a repetitive pattern of development and subsequent abortion of lateral roots (bright-green arrows), which is consistent with a P$_i$-sensitive checkpoint in root development that leads to competency for monitoring environmental P$_i$ availability [49]. The photograph shows CYCLINB1::GUS reporter gene expression (a marker for meristem activity) in root tips of pdr2 seedlings grown for 11 days in P$_i$-deficient medium (photograph reproduced, with permission, from Ref. [49]). (b) Early lateral root development can be divided into discrete stages that are regulated by hormonal signals and nutrient availability (from left to right): initiation, establishment and emergence of lateral root primordia (gray cones), followed by meristem activation and maintenance (red discs) [80]. Experimental evidence supports a role for auxin during most of these processes, as well as for abscisic acid (ABA), which regulates a developmental checkpoint in response to nitrate availability [29,80]. Because auxin and other plant hormones fail to rescue or mimic the pdr2 root phenotype [49], it is likely that a P$_i$-specific nutrient-sensing pathway reports local P$_i$ status and modulates mitotic activity downstream of auxin action in meristem maintenance to fine-tune the root cell division rate.

This page contains a series of diagrams and text discussing the impact of phosphate availability on root system architecture and the role of specific genes and hormones in regulating this process. The text explains that phosphate limitation can alter root system architecture, promoting lateral root proliferation and reducing the growth of individual trichoblast cells. It mentions the role of phosphate-sensing pathways in regulating meristem activity and cell division.

The diagrams illustrate the impact of phosphate limitation on root growth and the role of specific genes and hormones in this process. The text references various studies and experiments to support these findings, including the role of phosphate-sensing pathways in regulating meristem activity and cell division.

The text also mentions the role of phosphate availability in the expression of specific genes and hormones, such as auxin and abscisic acid (ABA), which regulate the development of lateral roots. The diagrams and text together provide a comprehensive overview of the complex interplay between phosphate availability and root system architecture and development.
Pᵢ sensing [53]. Such a mechanism is also supported by genetic studies in yeast, which suggest that the Pᵢ sensor is either intracellular [54,55] or part of a plasma membrane protein complex that includes Pᵢ transporters [50,56,57].

Integration and communication

Regulation of gene expression

Responses to Pᵢ limitation are activated in a synchronized fashion, suggesting that the underlying changes in Pᵢ-responsive gene expression are under coordinated control. The existence of a Pᵢ regulatory system in plants was first proposed by Alan Goldstein et al. [58] by analogy to the PHO regulon of microorganisms [59,60]. However, the paucity of successful complementation of yeast PHO homologs in Arabidopsis indicate that different and more complex regulatory networks control Pᵢ starvation responses in vascular plants.

Studies with transgenic plants expressing various PSI-promoter::reporter gene fusions have demonstrated that at least some Pᵢ-responsive genes are transcriptionally regulated [7,47] and that phosphate interferes with this control [47]. A genetic screen for defective expression of the Pᵢ-specific AIPS1::GUS reporter identified the phr1 mutant, which displayed reduced induction of a subset of PSI genes [7]. PHR1 encodes a transcription factor with similarity to PSR1 from Chlamydomonas reinhardtii [61], containing a MYB domain and a predicted coiled-coil (CC) domain for protein–protein interaction. Pᵢ limitation induces expression of the gene and PHR1 is constitutively localized to the cell nucleus, where it binds to an imperfect palindromic 8-bp DNA sequence present in the promoter of several PSI genes [7,20]. These observations indicate that PHR1 acts as a positive factor downstream of Pᵢ signaling and affects a subset of molecular responses to Pᵢ limitation. Transcription factors of the MYB–CC class are unique to plants and are represented by 15 members in Arabidopsis [20]. Thus, functional redundancy can explain the partial phenotypes observed for the phr1 mutant [7]. The single recessive pdr1 mutation causes diminished induction of a large set of PSI genes by low Pᵢ and can thus affect another positive regulator of Pᵢ deficiency responses [13] (C.A. Delatorre and S. Abel, unpublished). Indirect evidence for a role of transcriptional repressors was obtained by DNA binding studies that showed that promoter fragments of PSI genes specifically interact with nuclear proteins prepared from Pᵢ-sufficient plants, but not from Pᵢ-starved seedlings [62]. Similar studies further suggested a role for HD-ZIP (homeobox-leucine zipper domain) transcription factors in regulating some Pᵢ-responsive genes [63]. The involvement of various additional transcription factors in Pᵢ-responsive gene expression is indicated by large-scale expression studies [22–26]. It is clear that transcriptional regulation of Pᵢ-responsive genes will continue to be a prominent area of research.

Long-distance signaling

The magnitude of Pᵢ starvation responses is regulated both locally by external Pᵢ supply and systemically by plant Pᵢ status [20]. Divided-root studies have demonstrated that PSI genes are repressed by Pᵢ application to only part of the Pᵢ-deficient root system [64]. The nature of the systemic signal is elusive. Cytokinin, or a component of its transducing pathway, is a candidate signal because application of the hormone to Pᵢ-starved plants represses PSI genes [65,66] via the CRE1 receptor [67]. However, in divided-root experiments, external application of cytokinin does not recapitulate the systemic effect of Pᵢ exposure on PSI gene expression [20]. Although Pᵢ itself has not been considered to be a likely systemic signal [20,64], the available data do not rule out this possibility [20]. Altered root development as an adaptive response to Pᵢ limitation is influenced by whole-plant Pᵢ status. Primary root growth of the pho2 mutant, which over-accumulates Pᵢ in the shoot [68], shows an exaggerated response to increasing external Pᵢ [13,30], suggesting shoot-to-root communication. However, the Pᵢ uptake rate of pho2 plants is twice that of the wild type under Pᵢ-sufficient conditions, which is not observed for Pᵢ-starved plants [68]. Thus, accelerated root tip growth of pho2 seedlings in high Pᵢ medium might be the result of increased Pᵢ concentrations in primary root tip cells or of enhanced Pᵢ flux.

Because Pᵢ deficiency causes a dramatic redesign of root system architecture, plant Pᵢ sensing probably intersects with signaling pathways of hormones that govern root development, such as auxin or ethylene. A role for auxin as a mediator of Pᵢ starvation responses is currently unclear. In Arabidopsis, Pᵢ limitation sensitizes roots to the effects of auxin on primary root growth inhibition and lateral root formation [29]; however, several auxin-signaling mutants respond normally to changing Pᵢ [29,30]. Because the auxin sensitivity of primary root growth is not altered in the conditional pdr2 mutant and auxin fails to mimic or rescue its short and branched root phenotype [49], it is unlikely that auxin is directly involved in the response of root meristems to local Pᵢ availability. The data suggest that a specific Pᵢ-sensing pathway reports local Pᵢ status and modulates cell division rate independently of the auxin requirement for default root development. Attenuation of primary root growth rate in response to low Pᵢ probably initiates auxin signaling by an unknown mechanism, perhaps by altering auxin transport [69], to accelerate lateral root formation. In addition to auxin, ethylene has been implicated in Pᵢ-responsive changes in root development but its involvement remains to be established [32,70,71]. Likewise, root hair initiation and elongation are regulated by developmental and stress-sensing pathways that intersect with ethylene and auxin signaling [72]. However, the effect of Pᵢ deficiency on root hair formation appears to be independent of auxin and ethylene [35,36].

System-wide access to Pᵢ is achieved by coordinating uptake and subsequent distribution throughout the plant, which is mediated by several transport systems that move Pᵢ across various plant membranes. High-affinity Pᵢ uptake from the soil solution is facilitated by Pht1 transporters, many of which are inducible by Pᵢ starvation conditions and expressed in roots [3,17,18]. Some Pht1 family members are also expressed in shoot tissues and pollen grains, suggesting that they play a wider role in
P_i transfer [18]. Xylem loading is essential for root-to-shoot P_i transport, and the PHO1 protein of Arabidopsis plays a crucial role in this process [73]. The lack of any proven transport activity of PHO1 and the presence of a SPX domain suggest that PHO1 might be a regulatory factor of P_i transport [74]. Recent work in yeast indicates that low-affinity P_i transporters are involved in PHO gene regulation [57]. Interestingly, these yeast proteins and all members of the PHO1 gene family share the SPX domain, which has been implicated in G-protein-associated signaling [57,74]. This has led to speculation that PHO1-like proteins function in P_i sensing.

Transporters also facilitate uptake of P_i from the vascular system into target cells and remobilization of P_i from senescing tissues, particularly under P_i stress. The \textit{pho2} mutant overaccumulates P_i in the shoot, which suggests a defect in phloem loading and shoot-to-root recycling. It is not clear whether the PHO2 protein functions directly in P_i transport [68]. Because of the importance of P_i in cellular processes, the intracellular P_i pools and interorganellar transport needs to be regulated. Low-affinity P_i transporters appear to control flux between the chloroplasts [Pht2, PTs (phosphate transporters)], mitochondria (Pht3), vacuoles and the cytoplasm according to metabolic demand [17]. Several classes of transporters mediate the exchange of P_i for phosphorylated carbohydrates between the chloroplast and the cytosol [75]. A chloroplast transporter, PHT2;1, was cloned from Arabidopsis, Medicago and Solanum, and shown to function in P_i remobilization from senescing to young leaves [76–79]. Although the vacuole plays a prominent role in buffering cytosolic P_i [6], tonoplast P_i transporters have yet to be identified.

**Future prospects**

The diverse structural and regulatory functions of P_i in metabolism make the dissection of P_i-specific nutrient perception a challenging but exciting task. Although our knowledge of P_i signaling is most advanced for yeast, the sensing mechanisms involved are not known. Plants as multicellular organisms have evolved more complex strategies than yeast to maintain P_i homeostasis and therefore the underlying regulatory networks are likely to differ from those in Saccharomyces. A combination of molecular, biochemical and genetic approaches at the single-gene to genome-wide scales should illuminate initial signaling events in P_i perception. For example, the power of the ‘early gene’ concept, P_i-responsive mutants, phosphite as a selective inhibitor of P_i starvation responses and divided-root as well as grafting experiments remain to be fully exploited in the analysis of P_i-responsive gene expression. Likewise, the rapid post-transcriptional attenuation of PSI gene expression during recovery of P_i-starved plants can provide an experimental system for elucidating P_i perception. Although the first components of plant P_i response pathways have been genetically identified and biochemically characterized, dissection of P_i sensing by forward and reverse genetics is at an early stage and remains to be rigorously developed to uncover additional regulatory factors. Large-scale gene expression profiling of loss- and gain-of-function mutants should provide insight into the architecture of P_i-responsive metabolic networks. Identification of genes that affect P_i-responsive root meristem initiation and maintenance holds the promise to reveal how local P_i availability and perhaps other environmental cues regulate cell cycle progression. Finally, a comprehensive and mechanistic understanding of the involvement of plant hormone action, cross talk between mineral signaling pathways and long-distance transport in whole-plant P_i homeostasis will be important tasks for the future.

**Acknowledgements**

We thank the United States Department of Energy, Energy Biosciences Program, for continued funding of our research (DE-FG03-00ER15057 to S.A.).

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