

# Nutrient Sensing and Signaling: NPKS

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## Key Words

phosphorus, nitrogen, potassium, sulfur, nutrient deprivation

## Abstract

Plants often grow in soils that contain very low concentrations of the macronutrients nitrogen, phosphorus, potassium, and sulfur. To adapt and grow in nutrient-deprived environments plants must sense changes in external and internal mineral nutrient concentrations and adjust growth to match resource availability. The sensing and signal transduction networks that control plant responses to nutrient deprivation are not well characterized for nitrogen, potassium, and sulfur deprivation. One branch of the signal transduction cascade related to phosphorus-deprivation response has been defined through the identification of a transcription factor that is regulated by sumoylation. Two different microRNAs play roles in regulating gene expression under phosphorus and sulfur deprivation. Reactive oxygen species increase rapidly after mineral nutrient deprivation and may be one upstream mediator of nutrient signaling. A number of molecular analyses suggest that both short-term and longer-term responses will be important in understanding the progression of signaling events when the external, then internal, supplies of nutrients become depleted.

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## INTRODUCTION

Plants are a diverse group of organisms in part because of the many different environments in which they grow and to which they are adapted. Environmental change occurs due to location, seasonal variation, and daily, weekly, monthly, and yearly changes in climate. Plant diversity in genetic architecture, gene expression, and physiological adaptations is very large (31). Plants are anchored in one place for most of their life cycle and therefore must be able to adapt to a range of abiotic and biotic stresses. Variation in soil nutrient composition is found in agriculture systems and in natural environments and ranges from an extreme lack of nutrients in soils (e.g., 43), to soils containing optimal amounts of fertilizer, and to soils with excess nutrients.

To provide a framework for understanding plant adaptation to the range of nutrients found in soils, one may consider two different modes of plant growth and metabolism. One mode is when plants grow under nutrient-sufficient conditions where mineral resources are not limiting. Growth proceeds at optimal rates and it may be relatively easy for plants to acquire nutrients and water from soils where sunlight is abundant. When nutrients become limiting, growth is reduced and plants alter certain aspects of their acquisition, utilization, and morphology to maximize and acquire scarce resources. Plant species respond differently to nutrient limitations based on the environment in which they evolved (40). Re-

sponses to nutrient limitations also depend on the nutrient, such as whether it is mobile or immobile in the soil (48). Crop plants have been bred over the past 30–40 years under high nutrient conditions and have high intrinsic growth rates and yield. Such characteristics may not be the most suitable for lower input environments. Plant response to the deprivation of essential mineral nutrients is fairly well described, but an understanding of how plants sense and signal changes (at the cellular or whole-plant level) in the availability of nutrients is lacking.

As fertilizer becomes more expensive and as farmers reduce fertilizer usage because of the negative environmental impacts, it will be important to gain a better understanding of how crop plants can be designed to grow more efficiently in environments with lower nutrient inputs (39). In addition, access to fertilizers in developing countries is limited (116), so it could be helpful to create higher-yielding crop plants that are adapted to a lower input environment.

In this review we mainly discuss the events that lead to plant responses to nutrient-deficient conditions. First, we discuss general features of deficiency to the macronutrients. Second, we cover what is known about sensing and signaling phosphorus, nitrogen, potassium, and sulfur deficiency. This field of plant research is still in its infancy and has mainly focused on the downstream responses to nutrient deprivation. As such there is still scope for many fundamental discoveries that will be important to basic and more applied aspects of plant biology.

## TIMING

One of the many open questions in nutrient signaling is the progression of events and the importance of early versus later signals. Plants respond to nutrients by altering their physiology and morphology (48). Responses vary over time and also with the nutrient deficiency encountered. Compared with pathogen response where important signaling events

occur very rapidly after infection (67), important signaling events are likely to occur both very soon (less than six hours) and much later (days or weeks) after nutrient deprivation.

When plants are deprived of nutrients, the root is usually the primary site of perception. Early signals in response to nutrient deprivation may occur when external concentrations of nutrients are reduced and the later signals may be initiated when internal stores of nutrients are reduced below a critical level. At the earliest time points after deprivation, changes in membrane potential may be a dominant effect. During potassium deprivation root cells become hyperpolarized (128), whereas with phosphate- (22), sulfate- and nitrate-deprivation cells become depolarized. It is not yet clear how plant cells decode these changes in membrane potential. Under natural conditions nutrients become depleted slowly in soils due to the activity of roots over time or a lack of soil moisture. Soils may also lack a certain mineral nutrient, roots may encounter a patch of soil that is enriched in nutrients, and other variations in soil fertility may occur. In the laboratory the imposition of nutrient stress can be rapid. This occurs when plants are transferred to conditions lacking or very low in nutrients. As soils become depleted of nutrients or under laboratory conditions, plants will not immediately become deficient because of their ability to remobilize potassium from the vacuole or phosphorus, nitrogen, or sulfur from molecules containing those minerals or the vacuole.

Some clues to how plants respond to nutrient deprivation over time come from several gene expression studies that have used short and longer time courses. Some of the earliest responses to nutrient deprivation come from a study on phosphorus, potassium, and iron starvation of roots (150). After one hour of deprivation the expression of a number of genes changed (150). In that study, genes encoding mitogen activated protein (MAP) kinases, a transcription factor, and a 14-3-3 protein were upregulated, but their role in signaling or adaptation to low nutrients has not

yet been determined. In another study, *Arabidopsis thaliana* plants were deprived of phosphorus for a short time (4 h) and a longer time (28 and 100 h) and changes in gene expression were studied using an 8K GeneChip<sup>TM</sup> (42). The conclusions from that study were that the short-term transcriptional responses in leaves appeared to be more related to general stresses than specifically to nutrient deprivation. Long-term responses were considered to be more specific for phosphorus deprivation. In that study, shoot phosphorus concentrations were significantly lower in the deprived plants between 24 and 72 h after deprivation (42). In published microarrays characterizing sulfur deprivation, the earliest time point studied is 24 h after deprivation, but it is known that sulfate transporters are induced after 6 h of deprivation (156). Based on the sulfate transporter gene induction data, it is likely that important early signaling factors have yet to be identified.

Plant roots in particular will rapidly sense changes in nutrient composition, which will lead to changes in gene expression in 60 min or less. Changes in the tissue concentrations of nutrients do not appear until later. Therefore, a decrease in the tissue concentration of a nutrient acts as a marker for the later events that occur in response to nutrient deprivation. Little is known about the consequences of short- or long-term sensing or signaling in response to nutrient deprivation, so definitive conclusions cannot be drawn regarding the relative importance of short-term and long-term responses.

## CROSS TALK AND SPECIFICITY OF RESPONSE

The interaction between nutrients for uptake and the imbalances caused by the deficiency of one mineral are well-described phenomena (84). For example, sulfur and nitrogen are tightly interrelated on the level of plant metabolism (44). In some plant species a decrease in sulfate disrupts nitrogen metabolism, resulting in high levels of

**ROS:** reactive oxygen species

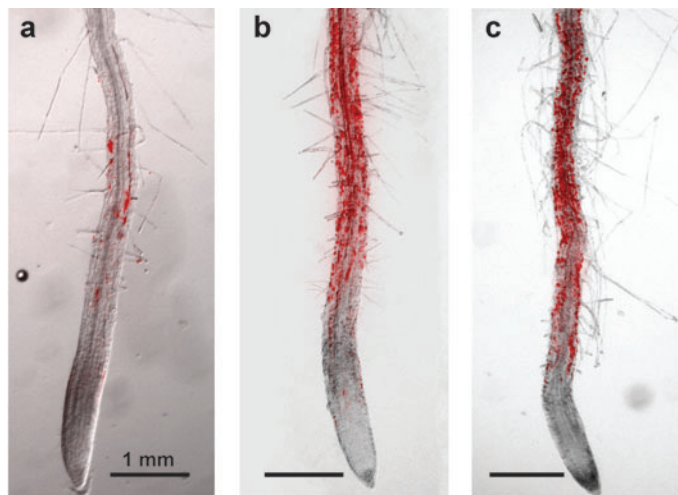
nitrate in leaves (109). Ammonium is known to interact with potassium for uptake (118), and that interaction may be genetically determined (11).

Evidence for interactions are also found in numerous molecular studies. In one study on potassium-deprivation, nitrate transporters were downregulated (4). In another similar study, ammonium transporters were upregulated by potassium deprivation (82). Although the interaction between similar-sized cations ammonium and potassium is expected, the link between nitrate and potassium is less obvious. However, during long-term deprivation of potassium changes in nitrogen assimilation may cause increased glutamine and decreased glutamate concentrations (3). Short-term (6 h) potassium deprivation in corn leads to the rapid upregulation of an *NRT2* homolog in corn (D. Schachtman & K. Huppert, unpublished), suggesting that factors other than changes in nitrogen metabolism may act on nitrate-sensing mechanisms.

Cross talk between potassium and phosphorus deprivation has been demonstrated

in tomato. Genes encoding a MAP kinase, transcription factors, and nutrient transporters were all induced by potassium and phosphorus deprivation (150). Analysis of phosphorus-deprived plants also showed that the upregulation of sulfur transporters and iron transporters occurred over the long term (93). The increased sulfur uptake may be required for sulfolipid synthesis, which to a certain extent replaces phospholipids. A decrease in iron transporter expression was linked to the increase in iron content in plants over time (93). Although one study on shoot transcriptional responses to nitrogen, phosphorus, and potassium noted very few overlapping changes in genes expression (42), a 2.5-fold cutoff was used to select up- and downregulated genes; therefore, important overlapping changes may have been overlooked. Another study showed that the expression of the gene encoding the response regulator AAR6 was upregulated by nitrogen, phosphorus, or potassium deprivation (17). Thus, shared nutrient signaling transduction pathways can be expected. Evidence also suggests that some genes are more specifically regulated in response to the deprivation of a particular nutrient (42, 125).

Although most studies on reactive oxygen species (ROS) have focused on leaves, some work has shown that nonphotosynthetic tissues such as roots or tubers also undergo an oxidative burst due to pathogen challenge (141) or deprivation of nutrients (125). The production of ROS in roots is a common feature in response to nitrogen, phosphorus, and potassium deprivation (125, 126). In this review we present data showing that ROS is also produced when plants are deprived of sulfur (**Figure 1**). Although this response occurs due to the deprivation of several macronutrients, it appears that there are some differences in localization as well as differences in the molecules that produce the ROS (125). One specific NADPH oxidase, AtrbohC, appears to be the main source of ROS produced in response to potassium deprivation (125). When this gene is inactive, ROS is still



**Figure 1**

Reactive oxygen species increases in *Arabidopsis* roots after 24 h of nutrient deficiency. Red fluorescence overlays of brightfield root images are shown for (a) nutrient-sufficient, (b) sulfur-deficient, and (c) potassium-deficient *Arabidopsis* wild-type, Col-0 roots after incubation with 50  $\mu$ M DFFDA.

produced under conditions of nitrogen and phosphorus deprivation.

The amount of ROS visualized after 30 h of sulfur deprivation in roots was greater than in sulfate-sufficient roots (**Figure 1**) and the localization was similar to potassium- or nitrogen-deprived roots (125, 126). The involvement of ROS in sulfur signaling may be more complex than that of potassium deprivation because the ascorbate-glutathione cycle that is downstream of sulfate assimilation is involved in the removal of H<sub>2</sub>O<sub>2</sub>. Changes in the reduced glutathione (GSH) pool depend on the supply of sulfate and correlate inversely with the activity of key components of the sulfate assimilation pathway (68). GSH pools become depleted under sulfur starvation; therefore, the reduction of dehydroascorbate to ascorbate is limited by decreased GSH availability (57). In contrast, GSH is not decreased under nitrogen and phosphorus starvation and ascorbate content is increased (57). Direct evidence showing that ROS is a signaling component in sulfur-starved plants is not yet available. However, in *Bacillus subtilis* several genes involved in sulfur assimilation and synthesis of sulfur-containing amino acids were induced by adding paraquat or by exposure to O<sup>2-</sup> (98). The regulation and interaction between ROS and GSH ascorbate impact the synthesis of plant hormones such as salicylic acid, gibberellins, abscisic acid, and ethylene (18, 107), which may signal plant response to nutrient deficiency. Although ROS plays a role in nutrient signaling in roots (125, 126), the differences in GSH and ascorbate production may provide some specificity to each nutrient deficiency.

The consequences of ROS production in roots are not well understood. Calcium may be part of this signal transduction cascade acting both upstream (141) and downstream (97) of the ROS production. Although calcium is not required for ROS production by NADPH oxidases in plants, the activity of these proteins is modulated by calcium (113), presumably through binding to putative calcium-binding sites (59). One consequence of ROS produc-

tion may be changes in gene expression (125, 142). ROS production may also directly alter the function of proteins through modification of thiols as well as lead to alterations in the redox state of the cell, which would lead to downstream changes in proteins that sense the redox state of the cell. In the longer term, ROS has important consequences that lead to aerenchyma formation in nutrient-deprived roots (60), which may be an important adaptation that lowers the cost of maintaining roots (23).

## PHOSPHORUS

Phosphorus deprivation is probably the most widely studied macronutrient deficiency. Studies on plant response to phosphorus deprivation are ecologically and agriculturally important because in many parts of the world soils are low in available phosphorus due to numerous factors including immobility, low solubility, or lack of the mineral (119). Several recent reviews have focused on aspects of phosphate sensing and transcriptional changes in response to phosphate deprivation (1, 28, 138). Another area of major interest is the change in root development in response to phosphorus deprivation (79), which is a mechanism for increasing phosphorus acquisition. Upstream regulators of changes in root morphology or architecture involve the hormones auxin and ethylene (75, 80, 81, 101, 152). One important factor involved in multiple signal transduction pathways in plants is phospholipase D (PLD) (148). Studies of mutants revealed that PLD is involved in regulating root development under phosphate deprivation (74).

Several studies using microarrays describe the transcriptional responses that occur when plants are deprived of phosphate (42, 93, 147, 150). These microarray experiments confirm what was previously known about plant response to phosphate deficiency and extend the number of known phosphate responsive genes. When plants are deprived of phosphate, scavenging systems are activated to

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**GSH:** reduced glutathione

**PLD:** phospholipase D

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Pi: inorganic phosphate

recover phosphate from lipids and nucleic acids. As a result, lipid composition shifts from phospholipids to more galacto- and sulfolipids, ribonucleases recover phosphate from nucleic acids, acid phosphatase activity increases, and phosphate transporter genes are induced (138). These are well-known downstream responses to phosphorus deprivation. Less is known about phosphate sensing and signaling.

Promoter analysis of genes that are responsive to phosphate deficiency (93) showed the presence of phosphate starvation response 1 (PHR1) binding sites. PHR1 is a Myb transcription factor involved in certain aspects of response to phosphorus deprivation (112) (Figure 2). PHR1 contains a single MYB domain and a predicted coiled-coil domain that may be involved in dimerization that binds to an imperfect palindromic sequence (PIBS element: GNATATNC) in certain promoters. PHR1 was identified through the mutagenesis of lines containing a promoter (*AtIPS1*) driving the expression of  $\beta$ -

glucuronidase (GUS) that is upregulated by phosphate deprivation. The expression of *PHR1* itself is not very responsive to phosphate deprivation and therefore it is thought to be downstream in the phosphate signal transduction pathway. PHR1 also plays a role in phosphorus homeostasis under nutrient-sufficient conditions (Figure 2). This conclusion is based on the finding that *phr1-1* plants have lower phosphorus content than wild types under nutrient-sufficient conditions (112). The promoter element to which PHR1 binds, an 8-nucleotide imperfect palindromic sequence, is significantly enriched in many genes expressed under inorganic phosphate (Pi)-deprived conditions (9, 112). Confirmation of the importance of this promoter region for gene induction due to phosphate starvation was provided by promoter deletion studies of an inducible phosphate transporter from barley (122). Although many phosphate starvation-induced genes contain the GNATATNC motif (28), the expression of some of these genes is attenuated and not

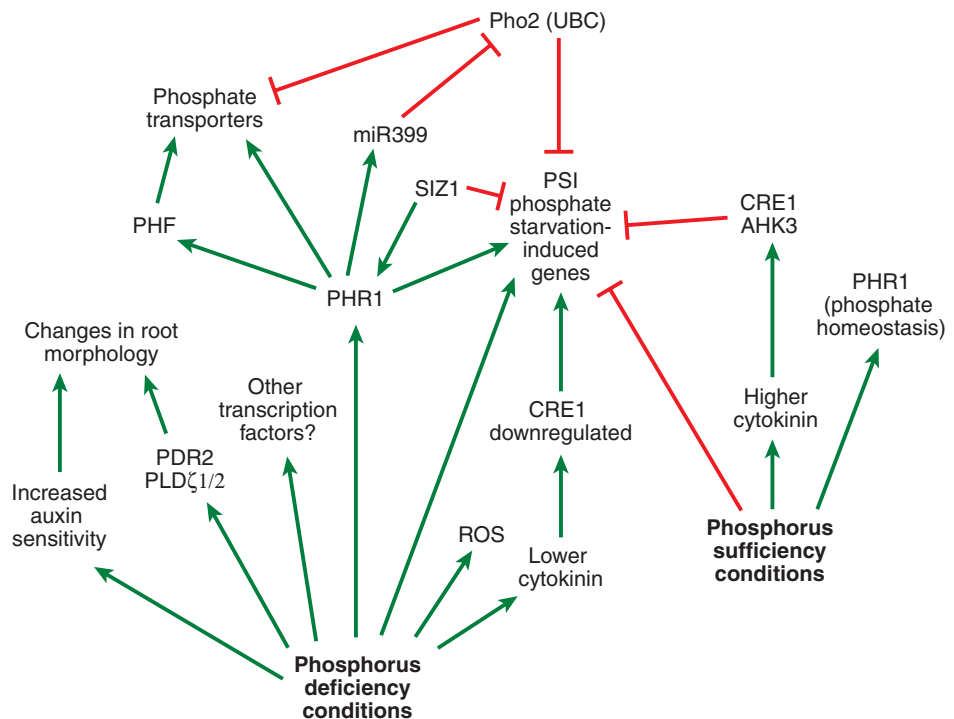


Figure 2

Components involved in plant responses and signaling under phosphorus-deficient and -sufficient conditions as described in the text.

completely abolished in the *phr1* knockout. Therefore, it is likely that other transcription factors may act together with PHR1 in a combinatorial fashion (136), or that other redundant Myb transcription factors are also involved (112).

*PHR1* belongs to a larger gene family that includes 11 homologs in *Arabidopsis*. A second member of this family is rapidly induced by phosphorus deprivation (140). Recent studies using microarray technology and qPCR identified additional genes that are regulated by PHR1 (9). These genes include transcription factors, known phosphate starvation-inducible genes, and genes encoding proteins involved in lipid metabolism. Although these genes may not be directly regulated by PHR1, they do provide an important entry point for further characterization of one branch of the signal transduction pathway in response to phosphorus deprivation (**Figure 2**).

One intermediate link in the signal transduction cascade from *PHR1* to the function of high-affinity phosphate transporters is the phosphate transporter traffic facilitator (*PHF1*) (38). This gene encodes a protein involved in the exiting of phosphate transporters from the endoplasmic reticulum to eventually function in the plasma membrane (38). When plants are deprived of phosphate the genes encoding several different phosphate transport proteins are upregulated. *PHF1* is involved in the localization of these transporters to the plasma membrane (38). This facilitator protein may be specific for phosphate transporters because it is required for localization of one phosphate transporter, but not an aquaporin (38). The gene encoding the facilitator protein contains the PHR binding motif, and the upregulation of *PHF1* is attenuated, but not abolished, in the *phr1* mutant, again suggesting that other transcription factors are involved in the expression of these phosphate responsive genes, which may provide complexity in their regulation.

Although *PHR1* is not regulated at the transcriptional level, the function of this factor is regulated by sumoylation. *SIZ1*

functions as a small ubiquitin-like modifier (SUMO) E3 ligase (94) that contributes to the transfer of a peptide to a substrate. The conjugation of a SUMO peptide influences protein function by a number of different mechanisms (100). Under phosphate-starved conditions, *SIZ1* plays a transient role in the activation of PHR1 (**Figure 2**), which alters the expression of some genes that are activated by this transcription factor (94). *SIZ1* also acts as a repressor of phosphate-deficiency responses because the *siz1* knockout has increased lateral root and root hair growth, increased root-to-shoot ratios, and greater anthocyanin accumulation in response to phosphate deficiency. The hypothesis that phosphate starvation-inducible genes are repressed was previously proposed (99). *siz1* knockout plants exhibit a defect in the signal transduction pathway of phosphate sensing because these responses are observed even though internal phosphate concentrations are maintained at wild-type levels.

Another posttranscriptional mechanism for regulating plant response to phosphate deprivation is a microRNA (miR) miR399 that targets a putative ubiquitin-conjugating enzyme (UBC) whose expression is reduced under deprived conditions (33) (**Figure 2**). Overexpression of miR399 leads to increased phosphate accumulation under nutrient-sufficient conditions. The increased accumulation appears to be due to a defect in the remobilization of phosphate in leaves (16) similar to the phenotype of the phosphate over accumulator mutant *pho2* (20). Studies show that *pho2* (20) contains a mutation in the gene encoding the UBC targeted by miR399, which suggests that the ubiquitination of specific targets is necessary for the repression of certain phosphorus-deficiency responses (6, 9).

To understand and elucidate phosphate signaling pathways in plants, it is important to consider both local and systemic responses to low phosphate. Mineral deficiencies usually first impact the roots and then transmit signals either to leaves [as with chemical

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**SUMO:** small ubiquitin related modifier

**miR:** microRNA

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signaling under drought (151)] or the shoots themselves become nutrient deficient. The *Arabidopsis pdr2* mutant provides one example of local phosphate-sensing responses in plants (139). The *pdr2* mutant alters response to low phosphate in a root-specific manner and does not alter shoot growth. This mutant grows normally under phosphorus-sufficient conditions but is unable to maintain root meristem activity under Pi-deprived conditions even though internal concentrations of phosphate were similar to the wild type. The *pdr2* mutant also supports the concept that plants have different programs for growth in sufficient and deficient conditions.

The allocation of phosphate between roots and shoots is an important systemic response to phosphate limitation. In split-root experiments where the shoot and half the roots maintain sufficient concentrations of Pi, the deprived portion of the roots do not exhibit phosphate starvation responses (8, 12). The sufficient phosphate levels in shoots can suppress some of the deprivation responses in roots. One gene involved in the root-to-shoot allocation of phosphate is *At4* (85), which is induced by phosphate deprivation and expressed primarily in roots. The function of the gene product is unclear. It may produce small peptides or exert control through the RNA itself (124).

In phosphorus deprivation, it appears that the inorganic ion itself is sensed by plants. Evidence for this comes from experiments such as those on *pdr2* and also from experiments with phosphite. Phosphite is an analog of inorganic phosphate (13, 14) that suppresses the phosphate starvation responses such as increased anthocyanin production and the activation of phosphatases, ribonucleases, and root hair growth. Suppression of the starvation responses occurs in the presence of phosphite even though plant growth is greatly reduced; therefore, it appears that phosphite-grown plants do not sense the phosphate deprivation.

Some systemic and local responses to phosphate deprivation may be controlled by

the phytohormone cytokinin. Cytokinin levels decrease when plants are starved of Pi (49) and the reduced flow of cytokinin to leaves likely plays an important role in the resource allocation shift in favor of more root growth. However, it is not known how the low Pi signal is translated into the downregulation of cytokinin synthesis. At the local level in the roots certain genes that are normally induced by Pi deprivation are repressed by exogenous application of cytokinin (85), whereas other responses such as the stimulation of root hair growth are still enhanced even in the presence of this hormone. This highlights the multiple signals and signaling pathways that control plant responses to phosphate deprivation. Using the promoter of a gene induced by phosphate deprivation (*AtIPS1*), changes in repression due to cytokinin were identified using a mutational screening strategy (30). The mutants identified were those that showed *AtIPS1::GUS* expression in the presence of kinetin. A mutation in *CRE1* was responsible for determining the degree of cytokinin sensitivity. *CRE1* is a cytokinin receptor (51) that is involved in repression of phosphate starvation-induced genes (**Figure 2**), providing further evidence for the hypothesis (99) that starvation responses are repressed under nutrient-sufficient conditions. A second cytokinin receptor was also identified as being involved in Pi sensing, demonstrating the redundant roles that these receptors may play in the signal transduction pathway (29).

## NITROGEN

Nitrogen and carbon metabolism are linked and therefore cross talk between the signal transduction pathways that regulate nitrogen assimilation and carbon metabolism is expected. Systems analysis provides new insights that should help to further elucidate these complex networks (106, 129). To date, much work on nitrogen sensing has focused on the metabolic and morphological responses to the addition of nitrate, in particular the induction of nitrogen metabolism (121, 129, 146, 147,

149) and long-distance signaling and transporter responses (26). The root responses to nitrate are well documented, and even though the transporter *NRT2.1* was reported to be involved in nitrate sensing or transduction (77) in roots, a more recent report suggests an alternative role for *NRT2.1* (111). Plant responses and signal transduction pathways for nitrogen limitation are poorly understood because experiments that focus on the switch from nitrogen-sufficient to -deficient conditions have received less attention. However, the importance of nitrogen limitation cannot be overstated as nitrogen-use efficiency is a critical future goal for agriculture because of the need for enhanced stewardship of the environment and for more efficient use of an increasingly expensive input (34).

PII is potentially a key protein involved in the sensing of internal nitrogen supply. PII is a homolog of the well-characterized *Escherichia coli* protein involved in the nitrogen regulatory system (50, 96). The protein PII in *E. coli* is encoded by the *GlnB* gene, which interacts with other proteins to regulate glutamine synthase (GS). In plants, much of carbon and nitrogen metabolism occurs in the chloroplast where PII is localized (50). The plant PII homolog interacts with N-acetyl glutamate kinase, which is a key enzyme specifically involved in arginine biosynthesis (15, 24) and in nitrogen metabolism in general.

Nitrate may be a key molecule that is sensed by plants (as with phosphate) and is involved in controlling the ratio of roots to shoots (120). A highly significant correlation was found between leaf nitrate and shoot:root ratios (120). Another important factor in root-to-shoot allocation that is influenced by nitrogen is the hormone cytokinin (134). Cytokinin levels increase when plants are supplied with nitrogen and decrease when deprived. Cytokinins play a potentially important role in root-to-shoot communication as they are synthesized in roots and are well correlated with observed changes in biomass allocation between roots and shoots (114). Possible response regulators downstream of a

cytokinin receptor have been identified, supporting a role for cytokinin in nitrogen responses (115, 135).

Under nutrient-deprived conditions, phosphorylation also plays a key role in fine-tuning plant responses. In wheat, a gene encoding an *SNF1* kinase is upregulated by nitrogen, phosphorus, and sulfur deficiency (117). This kinase is also induced by cytokinin, even though cytokinin levels would be expected to decline under nitrogen deficiency. Another phosphorylation event and potential signaling cascade regulated by nitrogen deprivation is the phosphorylation and conversion of the nitrate transporter *CHL1* to a high-affinity mode (78). The kinase that phosphorylates *CHL1* has not been identified but will be an important component for understanding the signal transduction cascade in response to nitrogen deprivation. Phosphorylation and dephosphorylation are also known to activate and inactivate nitrate reductase (56). More work is needed to identify the networks of phosphorylated proteins and their downstream targets involved in the nitrogen signal transduction cascade.

In addition to sensing nitrate, plant cells may also sense carbon status, which leads to the regulation of key nitrate transporters *NRT2.1* and *NRT1* (69). A key metabolite involved in nitrogen metabolism is glutamate. The presence of multiple glutamate receptors in plant genomes (66) may indicate the potential importance of this metabolite in initiating signal transduction cascades. However, the role of glutamate receptors or glutamate is only beginning to be elucidated. Antisense lines of *AtGLR1.1* displayed conditional phenotypes in response to changes in carbon to nitrogen ratios. A model was presented that suggests that this receptor regulates abscisic acid (ABA) biosynthesis during seed germination, which is stimulated by nitrate and inhibited by sucrose (58). Glutamate also leads to changes in cation fluxes (21) as well as changes in root growth (25, 144). At this time these putative receptors cannot be placed in signal

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**GS:** glutamine synthase

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transduction cascades. Glutamine is another key metabolite linked to nitrogen metabolism that plays roles in metabolic regulation and possibly signal transduction (130).

Some indications for possible signal transduction networks can be found in studies on global transcriptional changes after two days of nitrogen deprivation and MADS-box transcription factors after 2.5 days of nitrogen deprivation (35, 121). Global transcriptional changes to nitrogen deficiency were mainly described in terms of changes that occurred after reintroduction of nitrate (121), and therefore discussion of this rich data set on the transcriptional changes in response to nitrogen deprivation is beyond the scope of this review. However, extensive changes were found after two days of nitrogen deprivation, including a large number of transcription factors and kinases (see the supplemental table in Reference 121). A previously identified MADS-box transcription factor, *ANR1*, which was identified as being induced by nitrate, is actually upregulated by nitrogen deprivation and not rapidly repressed by nitrate (after 3 h of nitrate) (35). Seven other MADS-box transcription factors are also upregulated by nitrogen deprivation, but to a lesser extent (35).

An Myb transcription factor that is a member of the *PHR* family of transcription factors in plants (112) was found to be upregulated by nitrogen deprivation (140). These Myb factors likely play important roles in the signaling of nitrogen deprivation. Other Mybs in the R2R3 family have also been shown to be responsive to nitrogen deprivation (95), but their targets or physiological roles are unknown. Gene expression data provide potential components of signal transduction pathways, but additional work will be required to link these components into networks.

## POTASSIUM

Potassium is a macronutrient required in large quantities by plants and is the most abun-

dant cation in plant cells. One difference between potassium and the other macronutrients discussed in this review is that potassium is not metabolized or incorporated into other macromolecules. It does, however, play a role in the activation of enzymes. This should simplify how plants respond to low potassium because the scavenging responses are limited to remobilization from intracellular compartments such as the vacuole or from older tissues. As with nitrogen and sulfur response to nutrient deprivation, very little is known about signaling pathways and the sensors of potassium deprivation in plants (3). In bacteria, specific sensors for potassium have been identified (131). In *E. coli*, phosphorylation plays a central role in the sensing of potassium deficiency. KdpD is a sensor kinase that undergoes autophosphorylation and transfers a phosphoryl group to a response regulator KdpE (145). This response regulator controls the expression of an operon coding for a high-affinity potassium uptake system in *E. coli*. The sensor kinase transduces changes in turgor caused by low potassium (55).

Few known potassium transporters are upregulated by potassium deprivation (5, 82), but after 6 h of deprivation *Arabidopsis* plants exhibit a shift to high-affinity potassium uptake (126). This shows that plants rapidly sense the changes in external potassium. At least two transporters are upregulated by potassium deprivation, including a high-affinity potassium uptake transporter (HAK5) (2, 36, 126) and KEA5 (126), which may be involved in remobilization of potassium from the vacuole. In *Arabidopsis*, the high-affinity potassium transporter that is induced by potassium deficiency (2, 36) is also regulated by ROS. Therefore, upstream signal transduction components for plant response to potassium deprivation include ROS (126).

A weak transcriptional response to potassium deprivation, even after 96 h, was observed in a microarray study (36) and this may suggest that posttranslational mechanisms

contribute to the signal transduction networks involved in the response to potassium deprivation. After two weeks of growth on potassium-free medium, *Arabidopsis* plants exhibited a stronger transcriptional response with about 600 genes potentially upregulated and downregulated (4). After two weeks of deprivation plants developed visual symptoms due to reduced tissue concentrations of potassium. Upon changes in tissue potassium concentrations, it is likely that the activity of certain enzymes requiring potassium may be reduced. Pyruvate kinase is one such enzyme that has been suggested to potentially play a key role as an internal potassium sensor (3).

The importance of posttranscriptional regulation was recently demonstrated through the isolation of the gene encoding an SnRK3 kinase (*CIPK23*) that interacts with two calcineurin B-like proteins (CBL1 and CBL9) to regulate a potassium channel by phosphorylation and increase potassium uptake under both controlled and deprived conditions (73, 155). The upstream factors that trigger the phosphorylation of this potassium channel are unknown. However, this new finding implicates calcium in the signal transduction pathway in response to potassium deprivation because the CBLs contain an EF hand for calcium binding (73, 155). Further support for the role of calcium comes from experiments on potassium-deprived roots that showed a number of genes encoding “calcium-related” proteins, including a calcium-dependent protein kinase, a calcium ATPase, and a calmodulin binding protein, were up- and downregulated 2 and 6 h after potassium deprivation (R. Shin and D.P. Schachtman, unpublished). In yeast, many nutrient-regulated kinases have been identified (153). It is likely that more phosphorylation-related events will be found to be involved as part of the signal transduction pathway in response to nutrient deprivation.

ROS are a component of the signal transduction pathway in roots in response to low potassium (125, 126). ROS are necessary for

root hair growth (27) and are involved in root elongation (76) and gene expression under potassium-deprived conditions (125). Using an NADPH oxidase mutant, certain genes involved in response to potassium deprivation were shown to be dependent on ROS production whereas other potassium responsive genes were independent of ROS (126). The upstream factors involved in the initiation of ROS production are unknown, but calcium is one candidate.

The hormonal responses to potassium deprivation include ethylene, jasmonic acid (JA), and auxin. After 6–30 h of potassium deprivation, the expression of genes encoding ethylene biosynthetic enzymes and ethylene production in potassium-deprived roots increased (125). This hormonal response is presumably downstream from unknown factors in the signal transduction network. The consequences of increased levels of ethylene are unknown. Although auxin may play a role in controlling the expression of potassium channels (108), it is not yet clear whether auxin levels change in potassium-deprived *Arabidopsis*. However, the DR5-GUS reporter gene expression, a marker for auxin localization, changes in potassium-deprived roots (143). Changes in auxin localization, concentrations, or sensitivity could also lead to the reduced lateral root growth observed over a longer time course of potassium deprivation (4, 126). Long-term potassium starvation resulted in the conspicuous upregulation of genes linked to JA and defense. This occurred after plants showed visual symptoms of potassium deprivation, which may link this late response to other general stress signaling pathways (4).

## SULFUR

Sulfur metabolism has been extensively studied from many angles (61, 71). This section addresses how sulfur metabolism is regulated under sulfur deficiency and what is known about the upstream signal components of sulfate assimilation. Sulfate is taken up and then assimilated to cysteine and reduced in the

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IAA: indole-3-acetic acid

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chloroplast (72). There are five functional subgroups of sulfate transporters in *Arabidopsis* (127). As with other mineral nutrient transporters, the expression of these high-affinity sulfate transporters is upregulated by sulfate deficiency and they facilitate uptake of sulfate from soil when sulfate is less available (86, 90, 132, 133). Recently, an ethylene insensitive-like (EIL) transcription factor, *SLIM1*, was isolated and shown to be involved in the regulation of a high-affinity sulfate transporter in response to sulfate limitation (87).

One regulatory component of sulfate assimilation is a miRNA. MicroRNAs play a regulatory role during development and in response to environmental stress (83) and were recently implicated in response to nutrient deprivation. The miR395 regulates specific targets under sulfur-deprived conditions. The targets of miR395 are ATP sulfurylase (*APS*) [*APS1* (at3g22890), *APS3* (at4g144680), and *APS4* (at5g43780)], which is the first step in sulfate assimilation. The abundance of *APS* transcripts decreases when the miR395 increases under sulfur-deprived conditions (53). The regulation of the gene encoding ATP sulfurylase may also be upregulated by sulfur deprivation (45, 110). Studies show the upregulation and downregulation of ATP sulfurylase by sulfur deprivation. One factor that may explain the differing results with ATP sulfurylase expression may be that studies with miR395 were performed on plants that had been starved of sulfur for two weeks as compared with shorter time courses in other studies. This further highlights how important the consideration of timing is in the responses to nutrient deprivation.

When plants are starved for sulfur, they activate mechanisms for increasing acquisition from soil. However, when plants cannot acquire enough sulfate, the decreased sulfate uptake leads to reduced assimilation activity (45, 46, 132, 133) and affects many different metabolic processes. Eventually, the limited supplies of sulfur in plants result in decreased plant tissue sulfur content (10, 37, 65,

70, 92, 102, 109). Decreases in sulfur content result in the inhibition of sulfate assimilation; decreased glutathione and cysteine; increased amounts of serine, O-acetylserine, and tryptophan (102); reduced amounts of chlorophyll, RNA, and total protein; increased photorespiration; decreased lipids; and nitrogen imbalance. Overall, these changes lead to a reduced rate of metabolism and growth (103).

Despite the fact that the steps and regulation of sulfate metabolism have been well characterized, the upstream factors that trigger changes in sulfate assimilation and the regulatory components involved in signaling sulfur deficiency have only been partially elucidated. Recent microarray experiments provide some clues as to the possible signaling component; however, data analysis has mainly focused on genes involved in sulfate assimilation (45–47, 54, 86, 102) and the earliest time points considered start 24 h after deprivation. Therefore early signals have not been characterized.

The plant hormones cytokinin, auxin, and JA are signaling components in response to sulfur deficiency. The expression of *APR1* (APS reductase 1) is upregulated by sulfur deficiency (123) and also by exogenous cytokinin (104). Exogenous cytokinin downregulates the expression of the high-affinity transporter *SULTR1;2* (90), which is upregulated by sulfur deprivation. Cytokinin acts through the cytokinin response receptor (CRE1) to regulate sulfate uptake and transporter expression. In the *cre1-1* mutant, application of cytokinin only partly reduces sulfate uptake, suggesting redundancy as noted for the case of phosphate deprivation (91). Auxin is also a signaling component under sulfate limitation (102). The expression of auxin-inducible genes (*LAAL8*, At1g51950, tryptophan synthase beta chain, At5g38530, putative auxin-regulated protein, At2g33830) is upregulated by sulfur starvation (47, 102). The expression of *NIT3* nitrilase, which can convert indole-3-acetonitrile to indole-3-acetic acid (IAA), is strongly increased by sulfur starvation (65, 89). The

increased auxin production may result in an increase in lateral root density in *Arabidopsis* under sulfate-limited conditions (79). JA is also a possible signaling component in leaves. Genes involved in JA biosynthesis are upregulated under sulfur deficiency (45, 54). These genes include 12-oxophytodienoate reductase 1 and lipoxygenase (45, 86, 102). JA may regulate the expression of genes involved in sulfate assimilation and GSH synthesis (54, 154). Furthermore, MeJA is involved in regulating the activity of sulfur assimilation enzymes such as serine acetyltransferase (SAT) and APR (54). Although JA is a regulator of sulfur metabolism, its levels in plants are not well characterized under deficient conditions.

Other known components for signaling under sulfur deprivation include kinases and transcription factors. SAC3 kinase, which is a yeast sucrose nonfermenting-type kinase, was isolated from *Cblamydomonas* and regulates response to sulfur deprivation (19). SAC3 is required for depletion of chloroplast RNAs under sulfate-deficient conditions. In the *sac3* mutant, the chloroplast RNA abundance is less reduced than wild type and accumulation of ARS1 RNA is greater than in wild type under sulfate-deficient conditions. In wild type, the reduction in chloroplast transcripts under sulfur-limiting conditions depends on phosphorylation (52). SAC3 kinase regulates photosynthesis under sulfate-limited conditions and sulfate assimilation through the reduction of chloroplast transcripts. R2R3 Myb transcription factors (Myb16, 56, 69, 75, 90, 93 and 94) have been shown to be upregulated (102) by sulfate limitation in plants. However, the role that these transcription factors play in signaling under sulfate deficiency has not been established.

In contrast to plants, many genes have been identified that are involved in signaling sulfur deficiency in bacteria and yeast. The expression of *Cys3*, a bZIP transcription factor in *Neurospora crassa* (32, 105), is regulated by sulfur deficiency and controls the expression of

some enzymes involved in sulfur metabolism. Mutations in *Scon1* and *Scon2*, which are components of E3 ubiquitin ligase and an F-box protein, lead to constitutive expression of *Cys3* and enzymes involved in sulfur metabolism (32, 105). Under sulfur-sufficient conditions *Scon1* prevents *Cys3* function through the multimerization of *Scon1-Scon2* and binding to *Cys3* (62–64, 105). MET30 (137) is a transcriptional repressor that has similar functions to the *Scon1-Scon2* complex. The expression of sulfate assimilation enzymes in yeast depends on *MET4* and *MET28*, which encode bZIP transcription factors that are homologs of *Cys3* (64). For *E. coli*, the expression of genes that are involved in synthesis of flagella, chemotaxis, and methionine synthesis were all downregulated by sulfur limitation. This may be controlled by RpoS, which is a sigma factor required under sulfur deficiency (41). In plants, a chloroplast sigma factor that controls chloroplast transcriptional activity may be deactivated by the SAC3 kinase (52). Sulfur-containing essential amino acids such as Cys and Met are synthesized through the sulfur assimilation pathway, and changes in the concentrations of these amino acids eventually modulate cell cycle processes and cell viability.

Recently, the 16-bp sulfur-responsive element (SURE) and the 5-bp core sequence were identified from the *Arabidopsis* sulfate transporter, *SULTR1;1* promoter. The SURE is essential for induction under sulfur-deprived conditions of genes, including *SULTR2;1*, *SULTR4;2*, *APR3*, and *NADPH* oxidoreductase (88). Soybean embryo factors (SEFs) 3 and 4 bind to the seed-specific  $\beta$ -conglycinin promoter, which is a sulfur responsive promoter that does not contain SURE elements. However, the function of SEFs in sulfate signaling has not been determined (7). Identification of transcription factor binding sites and the factors that bind to the SURE elements will help in assembling the signal transduction cascade in response to sulfate deficiency and perhaps provide links to other signal networks.

## SUMMARY POINTS

1. Some of the signal transduction pathways in response to nutrient deprivation are beginning to be elucidated and transcriptional responses from microarray studies as well as other approaches are providing insight into possible components of these networks.
2. Interactions between nutrients are commonly observed and suggest that significant cross talk will be identified in signal transduction networks for responses to the deprivation of different nutrients.
3. More work is needed to determine the role of early and late signals in signal transduction networks.
4. ROS production in roots is observed in response to the deprivation of several macronutrients and may be an important component in signaling nutrient deprivation.
5. The PHR1 transcription factor is a central regulator of plant responses to phosphate deprivation.

## FUTURE ISSUES

1. The relative importance of short- and longer-term responses to nutrient deprivation needs to be determined.
2. Cross talk between responses to the deprivation of NPKS and other mineral nutrients is likely, but the extent and importance of these overlapping pathways is not yet known.
3. Nutrient sensors have not yet been identified in plants.
4. Will plant scientists be able to use this basic information on sensing and signaling of nutrient deficiencies to create plants that grow better in low-nutrient environments while maintaining relatively high yields?

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## LITERATURE CITED

1. Abel S, Ticconi CA, Delatorre CA. 2002. Phosphate sensing in higher plants. *Physiol. Plant* 115:1–8
2. Ahn SJ, Shin R, Schachtman DP. 2004. Expression of KT/KUP genes in *Arabidopsis* and the role of root hairs in K<sup>+</sup> uptake. *Plant Physiol.* 134:1135–45
3. Amtmann A, Hammond J, Armengaud P, White PJ. 2006. Nutrient sensing and signaling in plants: potassium and phosphorus. *Adv. Bot. Res.* 43:209–57

4. Armengaud P, Breitling R, Amtmann A. 2004. The potassium-dependent transcriptome of *Arabidopsis* reveals a prominent role of jasmonic acid in nutrient signaling. *Plant Physiol.* 136:2556–76
5. Ashley MK, Grant M, Grabov A. 2006. Plant responses to potassium deficiencies: a role for potassium transport proteins. *J. Exp. Bot.* 57:425–36
6. Aung K, Lin SI, Wu CC, Huang YT, Su CL, Chiou TJ. 2006. *pho2*, a phosphate overaccumulator, is caused by a nonsense mutation in a microRNA399 target gene. *Plant Physiol.* 141:1000–11
7. Awazuhara M, Kima H, Goto DB, Matsui A, Hayashi H, et al. 2002. A 235-bp region from a nutritionally regulated soybean seed-specific gene promoter can confer its sulfur and nitrogen response to a constitutive promoter in aerial tissues of *Arabidopsis thaliana*. *Plant Sci.* 163:75–82
8. Baldwin JC, Karthikeyan AS, Raghothama KG. 2001. LEPS2, a phosphorus starvation-induced novel acid phosphatase from tomato. *Plant Physiol.* 125:728–37
9. Bari R, Datt Pant B, Stitt M, Scheible WR. 2006. PHO2, microRNA399, and PHR1 define a phosphate-signaling pathway in plants. *Plant Physiol.* 141:988–99
10. Blake-Kalff MMA, Harrison KR, Hawkesford MJ, Zhao FJ, McGrath SP. 1998. Distribution of sulfur within oilseed rape leaves in response to sulfur deficiency during vegetative growth. *Plant Physiol.* 118:1337–44
11. Bloom AJ, Finazzo J. 1986. The influence of ammonium and chloride on potassium and nitrate absorption by barley roots depend on time of exposure and cultivar. *Plant Physiol.* 81:67–69
12. Burleigh SH, Harrison MJ. 1999. The down-regulation of Mt4-like genes by phosphate fertilization occurs systematically and involves phosphate translocation to the shoots. *Plant Physiol.* 119:241–48
13. Carswell C, Grant BR, Theodorou ME, Harris J, Niere JO, Plaxton WC. 1996. The fungicide phosphonate disrupts the phosphate-starvation response in *Brassica nigra* seedlings. *Plant Physiol.* 110:105–10
14. Carswell MC, Grant BR, Plaxton WC. 1997. Disruption of the phosphate-starvation response of oilseed rape suspension cells by the fungicide phosphonate. *Planta* 203:67–74
15. Chen YM, Ferrar TS, Lohmeier-Vogel E, Morrice N, Mizuno Y, et al. 2006. The PII signal transduction protein of *Arabidopsis thaliana* forms an arginine-regulated complex with plastid N-acetyl glutamate kinase. *J. Biol. Chem.* 281:5726–33
16. Chiou TJ, Aung K, Lin SI, Wu CC, Chiang SF, Su CL. 2006. Regulation of phosphate homeostasis by microRNA in *Arabidopsis*. *Plant Cell* 18:412–21
17. Coello P, Polacco JC. 1999. ARR6, a response regulator from *Arabidopsis*, is differentially regulated by plant nutritional status. *Plant Sci.* 143:211–20
18. Conklin PL, Barth C. 2004. Ascorbic acid, a familiar small molecule intertwined in the response of plants to ozone, pathogens, and the onset of senescence. *Plant Cell Environ.* 27:959–70
19. Davies JP, Yildiz FH, Grossman AR. 1999. Sac3, an snf1-like serine/threonine kinase that positively and negatively regulates the responses of *Chlamydomonas* to sulfur limitation. *Plant Cell* 11:1179–90
20. Delhaize E, Randall PJ. 1995. Characterization of a phosphate-accumulator mutant of *Arabidopsis thaliana*. *Plant Physiol.* 107:207–13
21. Demidchik V, Essah PA, Tester M. 2004. Glutamate activates cation currents in the plasma membrane of *Arabidopsis* root cells. *Planta* 219:167–75
22. Dunlop J, Gardiner S. 1993. Phosphate uptake, proton extrusion and membrane electropotentials of phosphorus-deficient *Trifolium repens* L. *J. Exp. Bot.* 44:1801–8

---

**30. Provides molecular evidence that cytokinins and two component receptors play important roles in plant response to phosphorus deprivation.**

---

23. Fan M, Zhu J, Richards C, Brown KM, Lynch JP. 2003. Physiological roles for aerenchyma in phosphorus-stressed roots. *Funct. Plant Biol.* 30:493–506
24. Ferrario-Mery S, Besin E, Pichon O, Meyer C, Hodges M. 2006. The regulatory PII protein controls arginine biosynthesis in *Arabidopsis*. *FEBS Lett.* 580:2015–20
25. Filleur S, Walch-Liu P, Gan Y, Forde BG. 2005. Nitrate and glutamate sensing by plant roots. *Biochem. Soc. Trans.* 33:283–86
26. Forde BG. 2002. Local and long-range signaling pathways regulating plant responses to nitrate. *Annu. Rev. Plant Biol.* 53:203–24
27. Foreman J, Demidchik V, Bothwell JH, Mylona P, Miedema H, et al. 2003. Reactive oxygen species produced by NADPH oxidase regulate plant cell growth. *Nature* 422:442–46
28. Franco-Zorrilla JM, Gonzalez E, Bustos R, Linhares F, Leyva A, Paz-Ares J. 2004. The transcriptional control of plant responses to phosphate limitation. *J. Exp. Bot.* 55:285–93
29. Franco-Zorrilla JM, Martin AC, Leyva A, Paz-Ares J. 2005. Interaction between phosphate-starvation, sugar, and cytokinin signaling in *Arabidopsis* and the roles of cytokinin receptors CRE1/AHK4 and AHK3. *Plant Physiol.* 138:847–57
30. Franco-Zorrilla JM, Martin AC, Solano R, Rubio V, Leyva A, Paz-Ares J. 2002. Mutations at CRE1 impair cytokinin-induced repression of phosphate starvation responses in *Arabidopsis*. *Plant J.* 32:353–60
31. Fu H, Dooner HK. 2002. Intraspecific violation of genetic colinearity and its implications in maize. *Proc. Natl. Acad. Sci. USA* 99:9573–78
32. Fu Y, Paietta J, Mannix D, Marzluf G. 1989. Cys-3, the positive-acting sulfur regulatory gene of *Neurospora crassa*, encodes a protein with a putative leucine zipper DNA-binding element. *Mol. Cell. Biol.* 9:1120–27
33. Fujii H, Chiou TJ, Lin SI, Aung K, Zhu JK. 2005. A miRNA involved in phosphate-starvation response in *Arabidopsis*. *Curr. Biol.* 15:2038–43
34. Gallais A, Hirel B. 2004. An approach to the genetics of nitrogen use efficiency in maize. *J. Exp. Bot.* 55:295–306
35. Gan Y, Filleur S, Rahman A, Gotensparre S, Forde BG. 2005. Nutritional regulation of ANR1 and other root-expressed MADS-box genes in *Arabidopsis thaliana*. *Planta* 222:730–42
36. Gierth M, Maser P, Schroeder JI. 2005. The potassium transporter AtHAK5 functions in K<sup>+</sup> deprivation-induced high-affinity K<sup>+</sup> uptake and AKT1 K<sup>+</sup> channel contribution to K<sup>+</sup> uptake kinetics in *Arabidopsis* roots. *Plant Physiol.* 137:1105–14
37. Gilbert SM, Clarkson DT, Cambridge M, Lambers H, Hawkesford MJ. 1997. Sulfate-deprivation has an early effect on the content of ribulose 1,5-bisphosphate carboxylase/oxygenase and photosynthesis in young leaves of wheat. *Plant Physiol.* 115:1231–39
38. Gonzalez E, Solano R, Rubio V, Leyva A, Paz-Ares J. 2005. PHOSPHATE TRANSPORTER TRAFFIC FACILITATOR1 is a plant-specific SEC12-related protein that enables the endoplasmic reticulum exit of a high-affinity phosphate transporter in *Arabidopsis*. *Plant Cell* 17:3500–12
39. Good AG, Shrawat AK, Muench DG. 2004. Can less yield more? Is reducing nutrient input into the environment compatible with maintaining crop production? *Trends Plant Sci.* 9:597–605
40. Grime J. 1977. Evidence for the existence of three primary strategies in plants and its relevance to ecological and evolutionary theory. *Am. Nat.* 111:1169–94
41. Gyaneshwar P, Paliy O, McAuliffe J, Jones A, Jordan M, Kustu S. 2005. Lesson from *Escherichia coli* genes similarly regulated in response to nitrogen and sulfur limitation. *Proc. Natl. Acad. Sci. USA* 102:3453–58

42. Hammond JP, Bennett MJ, Bowen HC, Broadley MR, Eastwood DC, et al. 2003. Changes in gene expression in *Arabidopsis* shoots during phosphate starvation and the potential for developing smart plants. *Plant Physiol.* 132:578–96
43. Handreck K. 1997. Phosphorus requirements of Australian native plants. *Aust. J. Soil Res.* 35:241–89
44. Hesse H, Nikiforova V, Gakiere B, Hoefgen R. 2004. Molecular analysis and control of cysteine biosynthesis: integration of nitrogen and sulphur metabolism. *J. Exp. Bot.* 55:1283–92
45. Hirai MY, Fujiwara T, Awazuhara M, Kimura T, Noji M, Saito K. 2003. Global expression profiling of sulfur-starved *Arabidopsis* by DNA microarray reveals the role of O-acetyl-l-serine as a general regulator of gene expression in response to sulfur nutrition. *Plant J.* 33:651–63
46. Hirai MY, Saito K. 2004. Post-genomics approaches for the elucidation of plant adaptive mechanisms to sulphur deficiency. *J. Exp. Bot.* 55:1871–79
47. Hirai MY, Yano M, Goodenow DB, Kanaya S, Kimura T, et al. 2004. Integration of transcriptomics and metabolomics for understanding of global responses to nutritional stresses in *Arabidopsis thaliana*. *Proc. Natl. Acad. Sci. USA* 101:10205–10
48. Hodge A. 2004. **The plastic plant: root responses to heterogeneous supplies of nutrients.** *New Phytol.* 162:9–24
49. Horgan J, Wareing P. 1980. Cytokinins and the growth responses of seedlings of *Beula pendula* Roth. and *Acer pseudoplatanus* L. to nitrogen and phosphorus deficiency. *J. Exp. Bot.* 31:525–32
50. Hsieh MH, Lam HM, van de Loo FJ, Coruzzi G. 1998. A PII-like protein in *Arabidopsis*: putative role in nitrogen sensing. *Proc. Natl. Acad. Sci. USA* 95:13965–70
51. Hwang I, Sheen J. 2001. Two-component circuitry in *Arabidopsis* cytokinin signal transduction. *Nature* 413:383–89
52. Irihimovitch V, Stern DB. 2006. The sulfur acclimation SAC3 kinase is required for chloroplast transcriptional repression under sulfur limitation in *Chlamydomonas reinhardtii*. *Proc. Natl. Acad. Sci. USA* 103:7911–16
53. Jones-Rhoades MW, Bartel DP. 2004. Computational identification of plant microRNAs and their targets, including a stress-induced miRNA. *Mol. Cell* 14:787–99
54. Jost R, Altschmied L, Bloem E, Bogs J, Gershenzon J, et al. 2005. Expression profiling of metabolic genes in response to methyl jasmonate reveals regulation of genes of primary and secondary sulfur-related pathways in *Arabidopsis thaliana*. *Photosynthesis Res.* 86:491–508
55. Jung K, Veen M, Altendorf K. 2000. K<sup>+</sup> and ionic strength directly influence the autophosphorylation activity of the putative turgor sensor KdpD of *Escherichia coli*. *J. Biol. Chem.* 275:40142–47
56. Kaiser WM, Huber SC. 2001. Post-translational regulation of nitrate reductase: mechanism, physiological relevance and environmental triggers. *J. Exp. Bot.* 52:1981–89
57. Kandlbinder A, Finkemeier I, Wormuth D, Hanitzsch M, Dietz KJ. 2004. The antioxidant status of photosynthesizing leaves under nutrient deficiency: redox regulation, gene expression and antioxidant activity in *Arabidopsis thaliana*. *Physiol. Plant.* 120:63–73
58. Kang JM, Turano FJ. 2003. The putative glutamate receptor 1.1 (AtGLR11) functions as a regulator of carbon and nitrogen metabolism in *Arabidopsis thaliana*. *Proc. Natl. Acad. Sci. USA* 100:6872–77
59. Keller T, Damude HG, Werner D, Doerner P, Dixon RA, Lamb C. 1998. A plant homolog of the neutrophil NADPH oxidase gp91phox subunit gene encodes a plasma membrane protein with Ca<sup>2+</sup> binding motifs. *Plant Cell* 10:255–66

---

48. An up-to-date review that provides an overview of how roots respond to the patchy nutrient supplies found in soils.

---

60. Konings H, Verschuren G. 1980. Formation of aerenchyma in roots of *Zea mays* in aerated solutions, and its relation to nutrient supply. *Physiol. Plant.* 49:265–70
61. **Kopriva S. 2006. Regulation of sulfate assimilation in *Arabidopsis* and beyond. *Annals. Bot.* 97:479–95**
62. Kumar A, Paietta J. 1995. The sulfur controller-2 negative regulatory gene of *Neurospora crassa* encodes a protein with  $\beta$ -transduction repeats. *Proc. Natl. Acad. Sci. USA* 92:3343–47
63. Kumar A, Paietta JV. 1998. An additional role for the F-box motif: gene regulation within the *Neurospora crassa* sulfur control network. *Proc. Natl. Acad. Sci. USA* 95:2417–22
64. Kuras L, Cherest H, Surdin-Kerjan Y, Thomas D. 1996. A heteromeric complex containing the centromere binding factor1 and two basic leucine zipper factors, Met4 and Met28, mediates the transcription activation of yeast sulfur metabolism. *EMBO J.* 15:2519–29
65. Kutz A, Müller A, Hennig P, Kaiser WM, Piotrowski M, Weiler EW. 2002. A role for nitrilase 3 in the regulation of root morphology in sulphur-starving *Arabidopsis thaliana*. *Plant J.* 30:95–106
66. Lacombe B, Becker D, Hedrich R, DeSalle R, Hollmann M, et al. 2001. The identity of plant glutamate receptors. *Science* 292:1486–87
67. Lamb C, Dixon RA. 1997. The oxidative burst in plant disease resistance. *Annu. Rev. Plant Physiol. Plant Mol. Biol.* 48:251–75
68. Lappartient A, Touraine B. 1997. Glutathione-mediated regulation of ATP sulfurylase activity,  $\text{SO}_4^{2-}$  uptake and oxidative stress response in intact canola roots. *Plant Physiol.* 114:177–83
69. Lejay L, Tillard P, Lepetit M, Olive F, Filleur S, et al. 1999. Molecular and functional regulation of two  $\text{NO}_3^-$  uptake systems by N- and C-status of *Arabidopsis* plants. *Plant J.* 18:509–19
70. Lencioni L, Ranieri A, Fergola S, Soldatini G. 1997. Photosynthesis and metabolic changes in leaves of rapeseed grown under long-term sulfate deprivation. *J. Plant Nutr.* 20:405–15
71. Leustek T, Martin MN, Bick JA, Davies JP. 2000. Pathways and regulation of sulfur metabolism revealed through molecular and genetic studies. *Annu. Rev. Plant Physiol. Plant Mol. Biol.* 51:141–65
72. Leustek T, Saito K. 1999. Sulfate transport and assimilation in plants. *Plant Physiol.* 120:637–44
73. Li L, Kim BG, Cheong YH, Pandey GK, Luan S. 2006. A  $\text{Ca}^{2+}$  signaling pathway regulates a  $\text{K}^+$  channel for low-K response in *Arabidopsis*. *Proc. Natl. Acad. Sci. USA* 103:12625–30
74. Li M, Qin C, Welti R, Wang X. 2006. Double knockouts of phospholipases Dz1 and Dz2 in *Arabidopsis* affect root elongation during phosphate-limited growth but do not affect root hair patterning. *Plant Physiol.* 140:761–70
75. Linkohr BI, Williamson LC, Fitter AH, Leyser HM. 2002. Nitrate and phosphate availability and distribution have different effects on root system architecture of *Arabidopsis*. *Plant J.* 29:751–60
76. Liskay A, van der Zalm E, Schopfer P. 2004. Production of reactive oxygen intermediates ( $\text{O}_2^-$ ,  $\text{H}_2\text{O}_2$ , and OH) by maize roots and their role in wall loosening and elongation growth. *Plant Physiol.* 136:3114–23
77. Little DY, Rao H, Oliva S, Daniel-Vedele F, Krapp A, Malamy JE. 2005. The putative high-affinity nitrate transporter NRT2.1 represses lateral root initiation in response to nutritional cues. *Proc. Natl. Acad. Sci. USA* 102:13693–98

78. Liu KH, Tsay YF. 2003. Switching between the two action modes of the dual-affinity nitrate transporter CHL1 by phosphorylation. *EMBO J.* 22:1005–13
79. López-Bucio J, Cruz-Ramirez A, Herrera-Estrella L. 2003. The role of nutrient availability in regulating root architecture. *Curr. Opin. Plant Biol.* 6:280–87
80. López-Bucio J, Hernandez-Abreu E, Sanchez-Calderon L, Nieto-Jacobo MF, Simpson J, Herrera-Estrella L. 2002. Phosphate availability alters architecture and causes changes in hormone sensitivity in the *Arabidopsis* root system. *Plant Physiol.* 129:244–56
81. Ma Z, Bielenberg DG, Brown KM, Lynch JP. 2001. Regulation of root hair density by phosphorus availability in *Arabidopsis thaliana*. *Plant Cell Environ.* 24:459–67
82. Maathuis FJ, Filatov V, Herzyk P, Krijger G, Axelsen K, et al. 2003. Transcriptome analysis of root transporters reveals participation of multiple gene families in the response to cation stress. *Plant J.* 35:675–92
83. Mallory AC, Vaucheret H. 2006. Functions of microRNAs and related small RNAs in plants. *Nat. Genet.* 38 Suppl 1:S31–36
84. Marschner H. 1995. *Mineral Nutrition of Higher Plants*. San Diego: Academic. 889p.
85. Martin AC, del Pozo JC, Iglesias J, Rubio V, Solano R, et al. 2000. Influence of cytokinin on the expression of phosphate starvation responsive genes in *Arabidopsis*. *Plant J.* 24:559–67
86. Maruyama-Nakashita A, Inoue E, Watanabe-Takahashi A, Yamaya T, Takahashi H. 2003. Transcriptome profiling of sulfur-responsive genes in *Arabidopsis* reveals global effects of sulfur nutrition on multiple metabolic pathways. *Plant Physiol.* 132:597–605
87. Maruyama-Nakashita A, Nakamura Y, Tohge T, Saito K, Takahashi H. 2006. *SLIM1/EIL3* transcription factor required for plant growth on low sulfur environment. Presented at Intl. Conf. on *Arabidopsis* Res., 17<sup>th</sup>, Madison, Wis.
88. Maruyama-Nakashita A, Nakamura Y, Watanabe-Takahashi A, Inoue E, Yamaya T, Takahashi H. 2005. Identification of a novel cis-acting element conferring sulfur deficiency response in *Arabidopsis* roots. *Plant J.* 42:305–14
89. Maruyama-Nakashita A, Nakamura Y, Watanabe-Takahashi A, Yamaya T, Takahashi H. 2004. Induction of SULTR1;1 sulfate transporter in *Arabidopsis* roots involves protein phosphorylation/dephosphorylation circuit for transcriptional regulation. *Plant Cell Physiol.* 45:340–45
90. Maruyama-Nakashita A, Nakamura Y, Yamaya T, Takahashi H. 2004. Regulation of high-affinity sulphate transporters in plants: towards systematic analysis of sulphur signaling and regulation. *J. Exp. Bot.* 55:1843–49
91. Maruyama-Nakashita A, Nakamura Y, Yamaya T, Takahashi H. 2004. A novel regulatory pathway of sulfate uptake in *Arabidopsis* roots: implication of CRE1/WOL/AHK4-mediated cytokinin-dependent regulation. *Plant J.* 38:779–89
92. Migge A, Bork C, Hell R, Becker T. 2000. Negative regulation of nitrate reductase gene expression by glutamine or asparagine accumulating in leaves of sulfur-deprived tobacco. *Planta* 211:587–95
93. Misson J, Raghothama KG, Jain A, Jouhet J, Block MA, et al. 2005. A genome-wide transcriptional analysis using *Arabidopsis thaliana* Affymetrix gene chips determined plant responses to phosphate deprivation. *Proc. Natl. Acad. Sci. USA* 102:11934–39
94. Miura K, Rus A, Sharkhuu A, Yokoi S, Karthikeyan AS, et al. 2005. The *Arabidopsis* SUMO E3 ligase SIZ1 controls phosphate deficiency responses. *Proc. Natl. Acad. Sci. USA* 102:7760–65

---

93. A comprehensive study on the many responses in *Arabidopsis* due to phosphorus deprivation.

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94. Shows that sumoylation regulates some phosphorus deprivation responses.

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95. Miyake K, Ito T, Senda M, Ishikawa R, Harada T, et al. 2003. Isolation of a subfamily of genes for R2R3-MYB transcription factors showing up-regulated expression under nitrogen nutrient-limited conditions. *Plant Mol. Biol.* 53:237–45
96. Moorhead GB, Smith CS. 2003. Interpreting the plastid carbon, nitrogen, and energy status. A role for PII? *Plant Physiol.* 133:492–98
97. Mori IC, Schroeder JI. 2004. Reactive oxygen species activation of plant Ca<sup>2+</sup> channels. A signaling mechanism in polar growth, hormone transduction, stress signaling, and hypothetically mechanotransduction. *Plant Physiol.* 135:702–8
98. Mostertz J, Scharf C, Hecker M, Homuth G. 2004. Transcriptome and proteome analysis of *Bacillus subtilis* gene expression in response to superoxide and peroxide stress. *Microbiol.* 150:497–512
99. Mukatira UT, Liu C, Varadarajan DK, Raghothama KG. 2001. Negative regulation of phosphate starvation-induced genes. *Plant Physiol.* 127:1854–62
100. Muller S, Hoege C, Pyrowolakis G, Jentsch S. 2001. SUMO, Ubiquitin's mysterious cousin. *Nat. Rev. Mol. Cell Biol.* 2:202–10
101. Nacry P, Canivenc G, Muller B, Azmi A, Van Onckelen H, et al. 2005. A role for auxin redistribution in the responses of the root system architecture to phosphate starvation in *Arabidopsis*. *Plant Physiol.* 138:2061–74
102. Nikiforova V, Freitag J, Kempa S, Adamik M, Hesse H, Hoefgen R. 2003. Transcriptome analysis of sulfur depletion in *Arabidopsis thaliana*: interacting of biosynthetic pathways provides response specificity. *Plant J.* 33:633–50
103. Nikiforova VJ, Kopka J, Tolstikov V, Fiehn O, Hopkins L, et al. 2005. Systems rebalancing of metabolism in response to sulfur deprivation, as revealed by metabolome analysis of *Arabidopsis* plants. *Plant Physiol.* 138:304–18
104. Ohkama N, Takei K, Sakakibara H, Hayashi H, Yoneyama T, Fujiwara T. 2002. Regulation of sulfur-responsive gene expression by exogenously applied cytokinins in *Arabidopsis thaliana*. *Plant Cell Physiol.* 43:1493–501
105. Paietta J. 1992. Production of the CYS3 regulator, a bZIP DNA-binding protein, is sufficient to induce sulfur gene expression in *Neurospora crassa*. *Mol. Cell. Biol.* 12:1568–77
106. Palenchar PM, Kouranov A, Lejay LV, Coruzzi GM. 2004. Genome-wide patterns of carbon and nitrogen regulation of gene expression validate the combined carbon and nitrogen (CN)-signaling hypothesis in plants. *Genome Biol.* 5:R91
107. Pastori GM, Kiddle G, Antoniw J, Bernard S, Veljovic-Jovanovic S, et al. 2003. Leaf vitamin C contents modulate plant defense transcripts and regulate genes that control development through hormone signaling. *Plant Cell* 15:939–51
108. Philippar K, Fuchs I, Luthen H, Hoth S, Bauer CS, et al. 1999. Auxin-induced K<sup>+</sup> channel expression represents an essential step in coleoptile growth and gravitropism. *Proc. Natl. Acad. Sci. USA* 96:12186–91
109. Prosser I, Purves J, Saker L, Clarkson D. 2001. Rapid disruption of nitrogen metabolism and nitrate transport in spinach plants deprived of sulphate. *J. Exp. Bot.* 52:113–21
110. Ravina CG, Chang CI, Tsakraklides GP, McDermott JP, Vega JM, et al. 2002. The *sac* mutants of *Chlamydomonas reinhardtii* reveal transcriptional and posttranscriptional control of cysteine biosynthesis. *Plant Physiol.* 130:2076–84
111. Remans T, Nacry P, Pervent M, Girin T, Tillard P, et al. 2006. A central role for the nitrate transporter NRT2.1 in the integrated morphological and physiological responses of the root system to nitrogen limitation in *Arabidopsis*. *Plant Physiol.* 140:909–21
112. Rubio V, Linhares F, Solano R, Martin AC, Iglesias J, et al. 2001. A conserved MYB transcription factor involved in phosphate starvation signaling both in vascular plants and in unicellular algae. *Genes Dev.* 15:2122–33

---

**112. Identified a key transcription factor in plants similar in structure to a previously identified transcription factor in *Chlamydomonas reinhardtii* that is involved in regulation of phosphate starvation-induced genes.**

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113. Sagi M, Fluhr R. 2001. Superoxide production by plant homologues of the gp91(phox) NADPH oxidase. Modulation of activity by calcium and by tobacco mosaic virus infection. *Plant Physiol* 126:1281–90
114. Sakakibara H. 2006. Cytokinins: activity, biosynthesis, and translocation. *Annu. Rev. Plant Biol.* 57:431–49
115. Sakakibara H, Suzuki M, Takei K, Deji A, Taniguchi M, Sugiyama T. 1998. A response-regulator homologue possibly involved in nitrogen signal transduction mediated by cytokinin in maize. *Plant J.* 14:337–44
116. Sanchez P. 2002. Soil fertility and hunger in Africa. *Science* 295:2019–20
117. Sano H, Youssefian S. 1994. Light and nutritional regulation of transcripts encoding a wheat protein kinase homolog is mediated by cytokinins. *Proc. Natl. Acad. Sci. USA* 91:2582–86
118. Santa-Maria GE, Danna CH, Czibener C. 2000. High-affinity potassium transport in barley roots. Ammonium-sensitive and -insensitive pathways. *Plant Physiol.* 123:297–306
119. Schachtman DP, Reid RJ, Ayling SM. 1998. Phosphorus uptake by plants: from soil to cell. *Plant Physiol.* 116:447–53
120. Scheible WR, Gonzalez-Fontes A, Lauerer M, Muller-Rober B, Caboche M, Stitt M. 1997. Nitrate acts as a signal to induce organic acid metabolism and repress starch metabolism in tobacco. *Plant Cell* 9:783–98
121. **Scheible WR, Morcuende R, Czechowski T, Fritz C, Osuna D, et al. 2004. Genome-wide reprogramming of primary and secondary metabolism, protein synthesis, cellular growth processes, and the regulatory infrastructure of *Arabidopsis* in response to nitrogen. *Plant Physiol.* 136:2483–99**
122. Schunmann PH, Richardson AE, Vickers CE, Delhaize E. 2004. Promoter analysis of the barley Pht1;1 phosphate transporter gene identifies regions controlling root expression and responsiveness to phosphate deprivation. *Plant Physiol.* 136:4205–14
123. Setya A, Murillo M, Leustek T. 1996. Sulfate reduction in higher plants: molecular evidence for a novel 5-adenylylsulfate reductase. *Proc. Natl. Acad. Sci. USA* 93:13383–88
124. Shin H, Shin HS, Chen R, Harrison MJ. 2006. Loss of At4 function impacts phosphate distribution between the roots and the shoots during phosphate starvation. *Plant J.* 45:712–26
125. Shin R, Berg RH, Schachtman DP. 2005. Reactive oxygen species and root hairs in *Arabidopsis* root response to nitrogen, phosphorus and potassium deficiency. *Plant Cell Physiol.* 46:1350–57
126. **Shin R, Schachtman DP. 2004. Hydrogen peroxide mediates plant root response to nutrient deprivation. *Proc. Natl. Acad. Sci. USA* 101:8827–32**
127. Smith F, Ealing P, Hawkesford M, Clarkson D. 1995. Plant members of a family of sulfate transporters reveal functional subtypes. *Proc. Natl. Acad. Sci. USA* 92:9373–77
128. Spalding EP, Hirsch RE, Lewis DR, Qi Z, Sussman MR, Lewis BD. 1999. Potassium uptake supporting plant growth in the absence of AKT1 channel activity. *J. Gen. Physiol.* 113:909–18
129. Stitt M, Muller C, Matt P, Gibon Y, Carillo P, et al. 2002. Steps towards an integrated view of nitrogen metabolism. *J. Exp. Bot.* 53:959–70
130. Sugiharto B, Suzuki I, Burnell JN, Sugiyama T. 1992. Glutamine induces the N-dependent accumulation of mRNAs encoding phosphoenolpyruvate carboxylase and carbonic anhydrase in detached maize leaf tissue. *Plant Physiol.* 100:2066–70
131. Sugiura A, Hirokawa K, Nakashima K, Mizuno T. 1994. Signal-sensing mechanisms of the putative osmosensor KdpD in *Escherichia coli*. *Mol. Microbiol.* 14:929–38

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121. A rich data set containing information on transcriptional response to nitrogen deprivation and to the introduction of nitrogen; some metabolic data are also provided.

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126. Showed that ROS may be a component in a signal cascade in roots of plants that had been deprived of potassium and that a single NADPH oxidase is important in generating ROS in response to potassium deprivation.

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**139. The culmination of a novel approach to the isolation of phosphate sensing and signaling mutants identified a locus that is involved in sensing phosphate by roots and regulating root meristem activity.**

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132. Takahashi H, Watanabe-Takahashi A, Smith FW, Blake-Kalff M, Hawkesford MJ, Saito K. 2000. The roles of three functional sulphate transporters involved in uptake and translocation of sulphate in *Arabidopsis thaliana*. *Plant J.* 23:171–82
133. Takahashi H, Yamazaki M, Sasakura N, Watanabe A, Leustek T, et al. 1997. Regulation of sulfur assimilation in higher plants: A sulfate transporter induced in sulfate-starved roots plays a central role in *Arabidopsis thaliana*. *Proc. Natl. Acad. Sci. USA* 94:11102–7
134. Takei K, Takahashi T, Sugiyama T, Yamaya T, Sakakibara H. 2002. Multiple routes communicating nitrogen availability from roots to shoots: a signal transduction pathway mediated by cytokinin. *J. Exp. Bot.* 53:971–77
135. Taniguchi M, Kiba T, Sakakibara H, Ueguchi C, Mizuno T, Sugiyama T. 1998. Expression of *Arabidopsis* response regulator homologs is induced by cytokinins and nitrate. *FEBS Lett.* 429:259–62
136. Thanos D, Maniatis T. 1995. Virus induction of human IFN beta gene expression requires the assembly of an enhanceosome. *Cell* 83:1091–100
137. Thomas D, Kuras L, Barbey R, Cherest H, Blaiseau P, Surdin-Kerjan Y. 1995. Met30p, a yeast transcriptional inhibitor that responds to S-adenosylmethionine, is an essential protein with WD40 repeats. *Mol. Cell. Biol.* 15:6526–34
138. Ticconi CA, Abel S. 2004. Short on phosphate: plant surveillance and countermeasures. *Trends Plant Sci.* 9:548–55
139. **Ticconi CA, Delatorre CA, Lahner B, Salt DE, Abel S. 2004. *Arabidopsis pdr2* reveals a phosphate-sensitive checkpoint in root development. *Plant J.* 37:801–14**
140. Todd CD, Zeng P, Huete AM, Hoyos ME, Polacco JC. 2004. Transcripts of MYB-like genes respond to phosphorus and nitrogen deprivation in *Arabidopsis*. *Planta* 219:1003–9
141. Torres MA, Dangl JL. 2005. Functions of the respiratory burst oxidase in biotic interactions, abiotic stress and development. *Curr. Opin. Plant Biol.* 8:397–403
142. Vandenabeele S, Van Der Kelen K, Dat J, Gadjev I, Boonefaes T, et al. 2003. A comprehensive analysis of hydrogen peroxide-induced gene expression in tobacco. *Proc. Natl. Acad. Sci. USA* 100:16113–18
143. Vicente-Agullo F, Rigas S, Desbrosses G, Dolan L, Hatzopoulos P, Grabov A. 2004. Potassium carrier TRH1 is required for auxin transport in *Arabidopsis* roots. *Plant J.* 40:523–35
144. Walch-Liu P, Liu LH, Remans T, Tester M, Forde BG. 2006. Evidence that L-glutamate can act as an exogenous signal to modulate root growth and branching in *Arabidopsis thaliana*. *Plant Cell Physiol.* 47:1045–57
145. Walderhaug MO, Polarek JW, Voelkner P, Daniel JM, Hesse JE, et al. 1992. KdpD and KdpE, proteins that control expression of the kdpABC operon, are members of the two-component sensor-effector class of regulators. *J. Bacteriol.* 174:2152–59
146. Wang R, Guegler K, LaBrie ST, Crawford NM. 2000. Genomic analysis of a nutrient response in *Arabidopsis* reveals diverse expression patterns and novel metabolic and potential regulatory genes induced by nitrate. *Plant Cell* 12:1491–509
147. Wang R, Okamoto M, Xing X, Crawford NM. 2003. Microarray analysis of the nitrate response in *Arabidopsis* roots and shoots reveals over 1,000 rapidly responding genes and new linkages to glucose, trehalose-6-phosphate, iron, and sulfate metabolism. *Plant Physiol.* 132:556–67
148. Wang X. 2005. Regulatory functions of phospholipase D and phosphatidic acid in plant growth, development, and stress responses. *Plant Physiol.* 139:566–73
149. Wang YH, Garvin DF, Kochian LV. 2001. Nitrate-induced genes in tomato roots. Array analysis reveals novel genes that may play a role in nitrogen nutrition. *Plant Physiol.* 127:345–59

150. Wang YH, Garvin DF, Kochian LV. 2002. Rapid induction of regulatory and transporter genes in response to phosphorus, potassium and iron deficiencies in tomato roots. Evidence for cross talk and root/rhizosphere-mediated signals. *Plant Physiol.* 130:1361–70
151. Wilkinson S, Davies WJ. 2002. ABA-based chemical signaling: the co-ordination of responses to stress in plants. *Plant Cell Environ.* 25:195–210
152. Williamson LC, Ribrioux SP, Fitter AH, Leyser HM. 2001. Phosphate availability regulates root system architecture in *Arabidopsis*. *Plant Physiol.* 126:875–82
153. Wilson WA, Roach PJ. 2002. Nutrient-regulated protein kinases in budding yeast. *Cell* 111:155–58
154. Xiang C, Oliver D. 1998. Glutathione metabolic genes coordinately respond to heavy metals and jasmoic acid in *Arabidopsis*. *Plant Cell* 10:1539–50
155. Xu J, Li HD, Chen LQ, Wang Y, Liu LL, et al. 2006. A protein kinase, interacting with two calcineurin B-like proteins, regulates K<sup>+</sup> transporter AKT1 in *Arabidopsis*. *Cell* 125:1347–60
156. Yoshimoto N, Takahashi H, Smith FW, Yamaya T, Saito K. 2002. Two distinct high-affinity sulfate transporters with different inducibilities mediate uptake of sulfate in *Arabidopsis* roots. *Plant J.* 29:465–73



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