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How do plants respond to nutrient shortage by biomass allocation?

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Plants constantly sense the changes in their environment; when mineral elements are scarce, they often allocate a greater proportion of their biomass to the root system. This acclimatory response is a consequence of metabolic changes in the shoot and an adjustment of carbohydrate transport to the root. It has long been known that deficiencies of essential macronutrients (nitrogen, phosphorus, potassium and magnesium) result in an accumulation of carbohydrates in leaves and roots, and modify the shoot-to-root biomass ratio. Here, we present an update on the effects of mineral deficiencies on the expression of genes involved in primary metabolism in the shoot, the evidence for increased carbohydrate concentrations and altered biomass allocation between shoot and root, and the consequences of these changes on the growth and morphology of the plant root system.

Responses of plants to mineral deficiencies

Plant growth and development ultimately depend upon environmental variables, such as temperature, light intensity and the availability of water and essential minerals. One of the mechanisms by which plants adjust to an imbalance of exogenous resources is by allocating new biomass to the organs that are involved in acquiring the resources that are scarcest [1]. Studies examining the relationships between mineral nutrition and plant growth and development have been undertaken, but most work has focussed on elucidating ion transport mechanisms and the biochemical pathways affected by mineral deficiencies [2–4]. Many reviews provide a comprehensive picture of the nature of mineral acquisition from the soil, transport within the plant and homeostasis in the plant cell [5–10]. However, progress is slow in understanding the molecular and physiological events responsible for sensing and signalling mineral resource limitation and their ultimate effects on plant development and biomass allocation. Now, with the emergence of microarray technologies to monitor gene expression, plant physiologists have begun to investigate the rapid transcriptional changes associated with mineral imbalance [11–23]. There is also considerable interest in the functional connection between the genome and the complement of ions in the cell (the ionome) [24].

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Deficiencies of nitrogen (N) [20,25–31] and phosphorus (P) [7,32–37] result in accumulation of carbohydrate in leaves, higher levels of carbon allocated to the root and an increase in root-to-shoot (R:S) biomass ratio. N and P deficiencies therefore affect, to various extents, primary photosynthesis, sugar metabolism and/or carbohydrate partitioning between source and sink tissues. By contrast, although leaves of potassium (K)-deficient [32,33,38,39] and magnesium (Mg)-deficient [32,33,40–43] plants accumulate sugars, they rarely increase their root biomass. This is likely to be a consequence of impaired sucrose export from leaves of K- and Mg-deficient plants, rather than a change in photosynthesis because the withdrawal of K and Mg from the growing medium does not alter photochemical reactions or photosynthetic rate within the timescale of the experiments [33,41–43], unless associated with a lower chlorophyll concentration [39].

Here, we propose a hypothesis to explain how both N and P deficiencies alter carbohydrate metabolism in shoots and thereby increase R:S biomass ratio and alter root morphology. This hypothesis incorporates roles for sucrose as an energy substrate, a carbon source and a signalling molecule. We also suggest the reason why such phenomena are not observed during K and Mg starvation: plants lacking K and Mg are less able to translocate sucrose to the root via the phloem.

Alteration of carbohydrate metabolism and partitioning by nitrogen and phosphorus deficiencies

Nitrogen deficiency results in the accumulation of sugars and starch in leaves (Figure 1) [20,26–28,30,31,36]. Nitrate content in leaves or in the xylem does not correlate with shoot growth [44], but nitrate content in leaves is negatively correlated with the proportion of carbon allocated to the root [27,36]. It is not clear what causes the accumulation of sugars in response to N deficiency. However, some insight can be gained from the transcriptional changes that occur when plants are starved of this element. An exhaustive examination of *Arabidopsis* microarray data suggests that N deficiency initiates transcriptional changes that can be integrated in a pathway directing the accumulation of sugars and starch in shoots and the increased translocation of sucrose to the root. This might account for the increase in plant R:S ratio. Analysis of the microarray data suggests that genes

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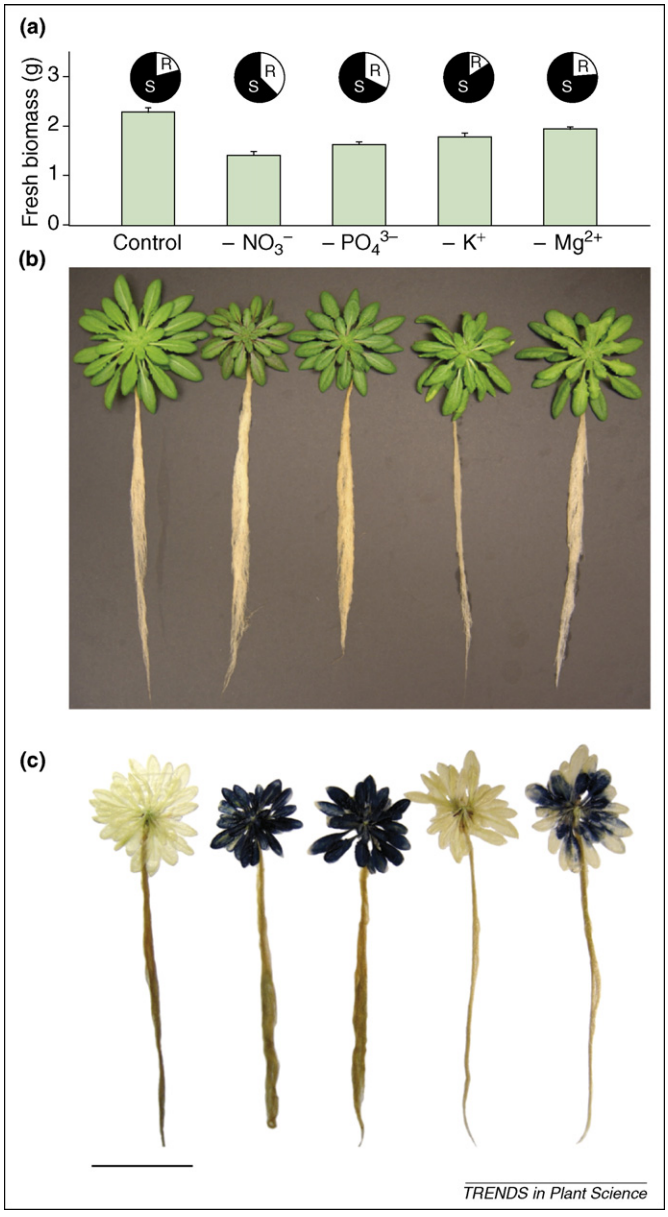

Figure 1. The effect of mineral supply on the morphology of *Arabidopsis thaliana*. **(a)** Individual fresh biomass (histograms) of plants grown in short days for 6 weeks in hydroponic systems containing a complete nutrient solution, then for 12 days in the same solution or one lacking the mineral element indicated. Biomass partitioning (pie charts) between root (R) and shoot (S) (mean of six values) is shown. Salt substitutions from the complete nutrient solution [43] were as follows: N deficiency, 0.2 mM $CaNO_3$, 0.8 mM $CaCl_2$; P deficiency, 0.25 mM KCl; K deficiency, 0.88 mM Na_2SO_4, 0.25 NaH_2PO_4; Mg deficiency, 1.00 mM Na_2SO_4. **(b)** Colour photographs of the plants in (a). **(c)** Iodine staining of the plants in (a). To visualize the differences in the distribution of starch (dark blue) following a dark period, iodine staining of whole plants was performed as a qualitative approach. Scale bar = 10 cm.

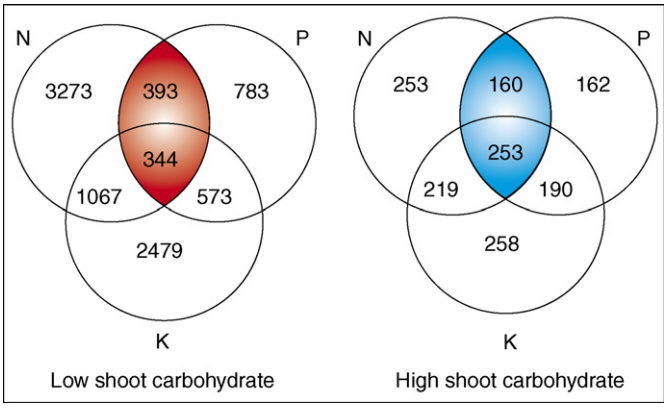

Figure 2. Transcriptional profiles of genes differentially expressed in shoots of *Arabidopsis* plants subjected to N, P or K starvation. The number of genes responding to N, P or K deficiencies against a background of low and high shoot carbohydrate concentrations. Genes responding to high shoot carbohydrate concentrations were defined as genes differentially expressed in shoots of the *pho3* mutant, which has constitutively high shoot carbohydrate concentrations, compared with wild-type plants [56]. Genes from segments highlighted in red and blue were classified in terms of their Gene Ontology (GO) categories.

assigned to the Gene Ontology (GO) category of primary metabolism and the sub-categories of carbohydrate metabolism, including starch metabolism (starch phosphorylase, several amylases and isoamylases), glycolysis and disaccharide metabolism are significantly ($P < 0.005$) over-represented among the differentially regulated genes in shoots of N-deficient plants (Figures 2 and 3) [20]. These could be among the first adjustments (within half an hour) to variations in N supply. In addition, genes associated with the metabolism of N-containing compounds and assigned to the GO categories amino acid, amine and glutamate metabolism and N compound catabolism were significantly ($P < 0.005$) over-represented among the differentially regulated genes in shoots of N-deficient plants (Figure 3) [20]. A repression of sets of genes required for photosynthesis and export of photosynthates also occurs [20,31]. This might re-establish the balance between photosynthesis and carbon use [26].

The reduction of photosynthesis in N-deficient plants is probably a direct consequence of sugar accumulation because sugars exert metabolite feedback regulation and affect many of the genes involved in photosynthesis [45–47]. However, some of the effects of N deficiency on plant growth and gene expression seem to be related to the C:N ratio in the tissue rather than carbohydrate status alone [19,28]. Carbon metabolites and plant C:N status both regulate the expression of several genes involved in N acquisition and metabolism [28,48,49], and nitrate regulates many genes assigned to sugar metabolism [13,31].

There is a tacit assumption that gene expression relates directly to protein abundance, enzyme activity and metabolite levels. The few existing proteomics and metabolomics studies on long-term nitrate deficiency corroborate the transcriptomic observations [31].

Phosphorus deficiency increases the concentrations of sugars and starch in leaves (Figure 1) [32–34,36,50] but not always in roots, depending on the species [33,34,51]. Changes in the level of gene expression and proteins involved in photosynthesis and sucrose synthesis occur when plants become P-deficient [7,11,16,34,52–55]. Low cytosolic inorganic phosphate (P_i) concentrations might restrict ATP synthesis, causing the deactivation of Ribulose-1,5-bisphosphate carboxylase/oxygenase (RuBisCo), or inhibit the activity of RuBisCo directly, resulting in the accumulation of ribulose-1,5-bisphosphate [53]. Genes encoding many photosystem subunits and assigned to the GO category of photosynthesis are downregulated in P-deficient plants and genes encoding sucrose synthases, fructose-1,6-bisphosphatases and UDP-glucose pyrophosphorylases are upregulated, with the GO category of carbohydrate metabolism and several of its sub-categories

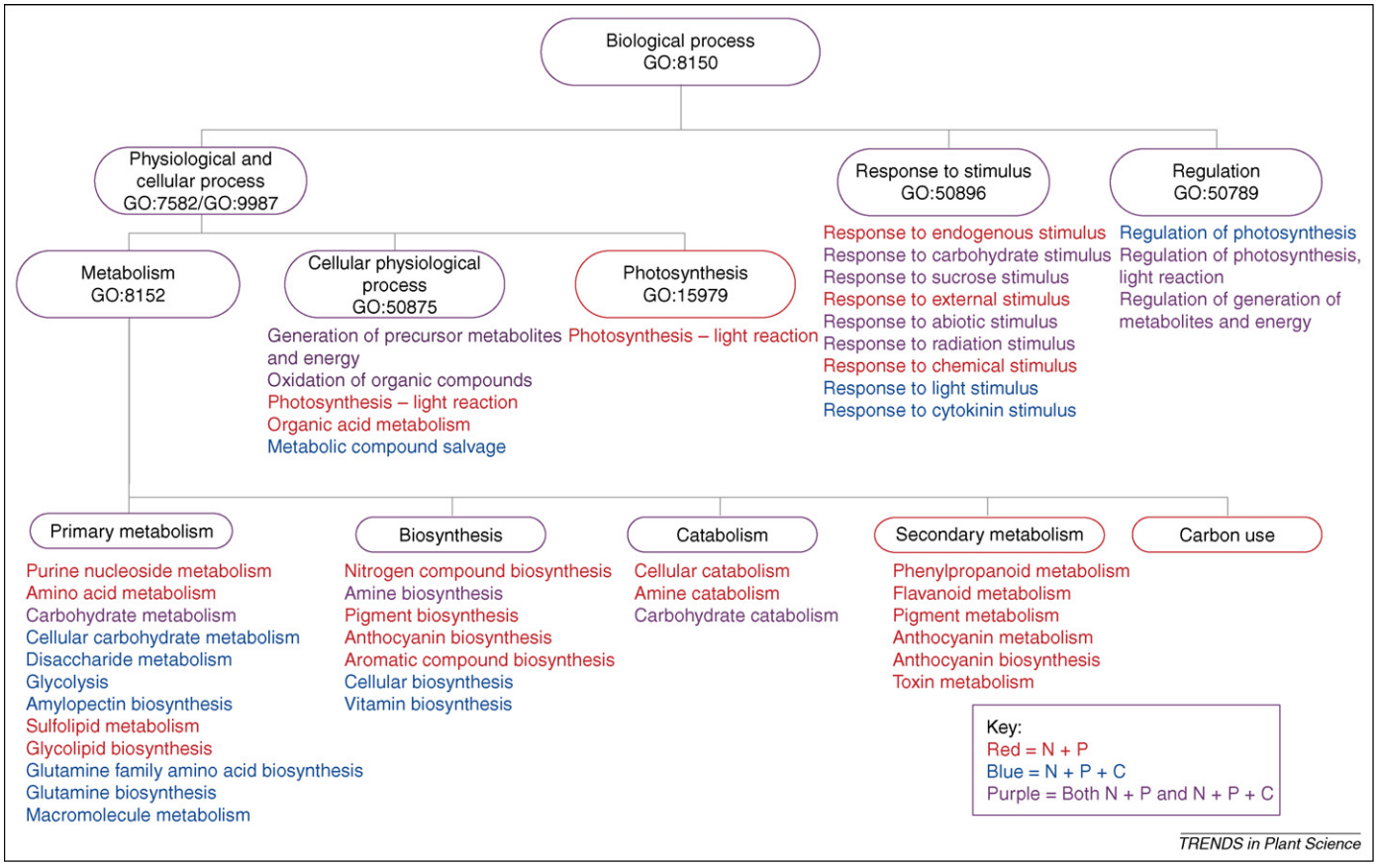

Figure 3. Gene Ontology (GO) categories significantly ($P < 0.005$) over-represented in differentially regulated genes in shoots of N-, P- or K-deficient plants. Identification of significantly over-represented GO categories was performed using the GO Ontology Browser function in GeneSpring GX 7.3 (Aglient Technologies, Santa Clara, CA, USA). Genes significantly ($P < 0.05$) differentially regulated by N deficiency were abstracted from Ref. [20]. Genes significantly ($P < 0.05$) differentially regulated by K deficiency were abstracted from Ref. [76]. A list of genes significantly ($P < 0.05$) differentially regulated by P deficiency was abstracted from Refs [11,17,23] and data on the *pho1* mutant (NASC, Nottingham University, UK), which has constitutively low shoot P concentrations. Genes differentially expressed in shoots of the *pho3* mutant compared with shoots of wild-type plants were abstracted from Ref. [56]. Text and shapes in red correspond to common significantly differentially regulated genes from N-deficient and P-deficient plants under low shoot carbohydrate conditions; text and shapes in blue correspond to common significantly differentially regulated genes from N-deficient and P-deficient plants under high shoot carbohydrate conditions; and text and shapes in purple correspond to overlaps between differentially regulated genes from N-deficient and P-deficient plants under low shoot carbohydrate conditions (red) and differentially regulated genes from N-deficient and P-deficient plants under high shoot carbohydrate conditions (blue).

significantly ($P < 0.005$) over-represented among the differentially regulated genes in shoots of P-deficient plants (Figures 2 and 3) [16,34]. Changes in transcript levels in P-deficient plants also affect other aspects of carbon and nitrogen metabolism, and genes encoding enzymes that remobilize P from cellular sources, such as phosphatases, nucleases and phosphodiesterases, are upregulated in P-deficient plants (Figures 2 and 3) [7,11,12,14,16,17,23]. It is assumed that these transcriptional changes optimize the use of P within the plant. Other GO categories significantly ($P < 0.01$) over-represented among the differentially regulated genes in shoots of P-deficient plants include sulfolipid and galactolipid biosynthesis, the defence response, the response to cytokinin, oxygen and reactive oxygen species metabolism and anthocyanin biosynthesis (Figures 2 and 3) [11,17,23].

The potential commonalities in shoot carbohydrate metabolism between N-deficient and P-deficient plants are observed in the GO categories significantly ($P < 0.005$) over-represented among the common differentially regulated genes in shoots of N-deficient and P-deficient plants (Figures 2 and 3). There are also large overlaps between the GO categories highlighted under N and P deficiency and those plants with high shoot carbohydrate concentrations (Figures 2 and 3). These include carbohydrate metabolism and catabolism, responses to carbohydrate and sucrose stimulus, generation of precursor metabolites and energy and oxidation of organic compounds. Thus, at the transcript level, common processes are induced by N and P deficiencies and high concentrations of carbohydrate in the shoots, which might reflect common components of the responses to these perturbations, with integration from other stress-specific signalling cascades defining the response.

Involvement of sugars and other molecules in signalling nitrogen and phosphorus deficiencies

Sugars are known to perform important regulatory functions in the plant life cycle, including photosynthesis [32,56–59] and carbohydrate partitioning [46,60]. However, the mechanisms by which sugars act to influence gene expression and ultimately plant development (formation of leaves and roots) are just beginning to be deciphered [61,62]. It is possible that many of the prospective metabolic changes that occur in shoots of N-deficient and P-deficient plants are regulated through transcriptional

changes elicited by increased leaf sugar concentrations (Figure 4), in addition to the well-known allosteric regulation of biochemical pathways (e.g. allosteric inhibition of ADP-glucose pyrophosphorylase (AGPase) by P_i in starch synthesis). For instance, AGPase is subject to transcriptional regulation, with expression being increased by sugars [63] and decreased by nitrate [64] and P_i [65]. Other experimental results in different plant systems show that sugars are crucial for signal transduction during N and P deficiency. For example, interruption of phloem supply results in a rapid decline of transcript accumulation of *LaPT1* (a P-deficiency induced phosphate transporter gene) and *LaSAP1* (a secreted acid phosphatase gene) in P-deficient *Lupinus* roots [66].

The hypothesis that sugar concentrations affect gene expression is also consistent with the identification of a significant number of *Arabidopsis* genes whose expression is regulated by both mineral deficiency and increased shoot sucrose concentrations (Figures 2 and 3). About 7% of the genes responding to N deficiency and 22% of the genes responding to P deficiency in shoots of *Arabidopsis* were also differentially regulated in shoots of a mutant plant (*pho3*, also known as *suc2*) with elevated leaf sugar concentrations compared with wild-type plants [56]. Sugar-regulated genes are also responsive to other signalling cascades induced by developmental and environmental signals, as part of cross-talk between signalling pathways sharing common components. The *cis*-regulatory elements

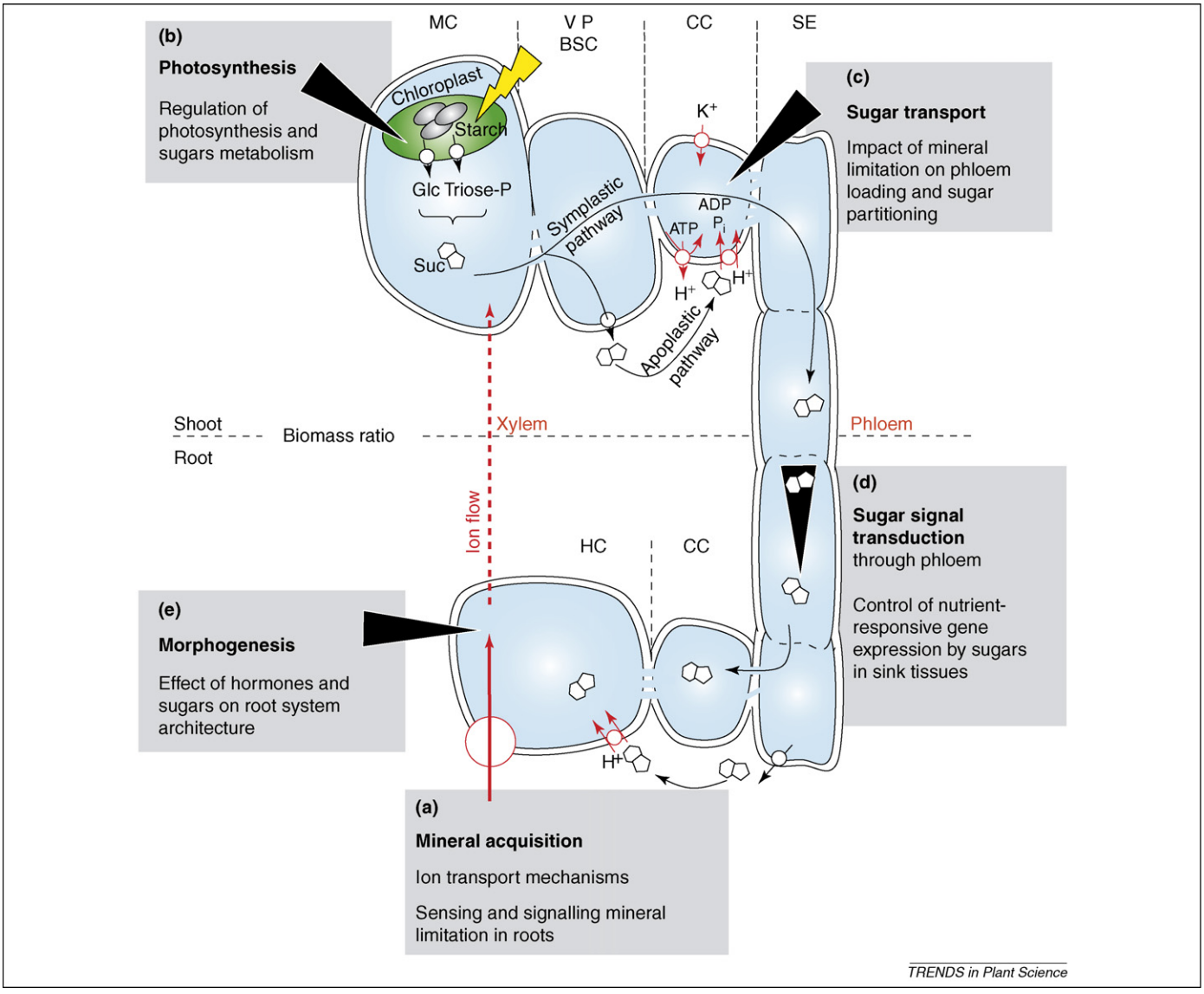

Figure 4. Model of plant response to nutrient shortage by allocating biomass to specific organs. Abbreviations: MC, mesophyll cell; VP, vascular parenchyma; BSC, bundle sheath cells; CC, companion cell; SE, sieve element; HC, heterotrophic cell. **(a)** The root is devoted to mineral nutrient acquisition [5–10] and is the first organ to sense and signal mineral starvation [18,71,79,80]. Crosstalk between root and shoot is established through xylem and phloem vascular tissues. **(b)** N, P, K and Mg deficiencies induce sugar accumulation in MC, affecting photosynthesis and sugar metabolism [7,11,13,16,20,28,30,34,43,52,65,68,70,75]. **(c)** From the point of synthesis in MC to HC, sucrose is loaded into the SE–CC complex either through plasmodesmata by the symplastic route or by the apoplastic route, depending on the mode of phloem loading. The apoplastic pathway requires sucrose export from MC or BSC and re-entry into the SE–CC complex, mediated by sucrose–H^+ symporters, energized by plasma membrane H^+-ATPase and facilitated by K^+ channels. Mg deficiency can affect sucrose phloem loading (probably though H^+-ATPase activity) [33,42,43] and K deficiency can affect K^+ phloem loading [78], limiting sucrose export to sink organs. By contrast upon N and P deficiencies, carbon partitioning favours root growth at the expense of the shoot. **(d,e)** Phloem unloading in sink cells can occur symplastically or through efflux carriers. It is postulated that the flux of sugars delivered to sink organs is crucial for responses to mineral deficiencies. Sugars regulate nutrient-responsive genes in sinks [66] and modify root morphology in tandem with changes in the concentrations of hormones [7,11,16,30,34,52,68,70–72,75].

involved in the transduction of sugar signals could also play a role in the transduction of ozone, peroxide, abscisic acid (ABA) or ethylene signals [67]. Supplying *Arabidopsis* seedlings with exogenous sugars has also revealed, in addition to glucose-responsive genes, large numbers of genes involved in other abiotic stresses [19].

In addition, multiple lines of evidence indicate that phytohormones participate in sugar signalling [46], in signalling between shoot and root and in dry-mass partitioning, both in general and in response to soil mineral imbalances [68]. ABA has been implicated in sugar signalling [46]; cytokinin-mediated signalling seems to control plant development under N deficiency [44,69]; and coordinated changes in cytokinin, auxin and ethylene concentrations might reprogram development under P deficiency [11,52,68].

Nitrogen and phosphorus deficiencies alter root architecture

In addition to increasing R:S ratios, N and P deficiencies alter root system morphology substantially [7,11,37,51,54,55,70]. It is possible that an increased sugar supply to the root affects root morphology through sugar signals. Sucrose is thought to promote cell differentiation and maturation, whereas hexoses favour cell division and expansion [45,65]. Furthermore, changes in hormonal balance in the root tissue might orchestrate changes in root morphology (Figure 4).

At the whole plant level, two types of response are activated. The first depends on external ion concentration and involves local signals. The second depends on whole plant mineral status and involves long-distance signalling. When plants are N-deficient, root growth accelerates and augmented lateral root (LR) branching further increases the foraging capacity of the root system [30]. Interestingly, when roots of N-deficient plants contact nitrate, lateral rooting is stimulated further. Several sensing and signalling pathways are thought to be involved in these local responses. Perception of nitrate availability occurs at least in part through the nitrate transporter NRT2.1, which seems to be directly involved in LR development, independently of its function in nitrate uptake by roots [25,71]. Signal transduction is apparently operated through the MADS-box transcription factor nitrate-regulated1 (*ANR1*) [72,73], and/or a systemic signal (possibly glutamine), through two transcription factors, one with a basic leucine zipper (bZIP) and one with a LIM domain [30]. High concentrations of nitrate in the tissue have a systemic inhibitory effect on LR development (prevention of meristematic activation after emergence) – this is possibly mediated in part by ABA [74].

Long-distance signals mediating the shoot response to nitrate perception in roots seem to involve cytokinins. It is possible that the reduction in cytokinins observed during N deficiency [30] relieves a general inhibition of root growth by this hormone, and that an increase in auxin stimulates cell division and LR development. This process seems to be promoted by increased sucrose concentrations in the roots [72], suggesting that sucrose signals from the shoot could set the magnitude of morphological responses to N deficiency.

Phosphorus deficiency results in the development of a highly branched root system located near the soil surface [6,54]. Deficient plants show reduced primary root (PR) elongation but increased LR formation and elongation and a proliferation of root hairs [7,11,37,51]. These morphological alterations are in part orchestrated by coordinated changes in the concentrations of plant hormones [7,11,34,52,54,55,68,70,75]. Root branching seems to be under the control of auxin, but other aspects of root architecture, such as root hair development and reduced PR growth, seem to be independent of auxin action [75]. The hormonal changes are consistent with both the alterations in root morphology and the relative expression of genes known to be regulated by, or involved in the regulation of, ethylene, auxin and cytokinins in roots of P-deficient plants [11,16,52].

Carbohydrate accumulation in leaves but not roots after potassium and magnesium deficiency

Potassium deficiency results in the accumulation of carbohydrates in leaves as replacement osmotic molecules [3,32,33]. However, in contrast to N deficiency and P deficiency, K deficiency rarely results in the accumulation of starch (Figure 1). The decline in photosynthesis observed in K-deficient plants could be a consequence of sucrose accumulation [32,33]; this hypothesis is consistent with the transcriptional profiles of leaves from K-deficient plants [76]. However, the increased leaf sucrose concentrations in K-deficient plants do not promote accelerated root growth. Roots of K-deficient plants have lower concentrations of sucrose and starch than their K-replete counterparts [32,33]. One reason for this is that sucrose export to the root is reduced in K-deficient plants [33], which can be attributed to a requirement for K^+ for loading sucrose into the phloem (Figure 4) [77,78]. An increase in the volume of soil exploited by roots is not an acclimatory response to K deficiency, and the R:S ratio of plants can even decrease during K deficiency. Indeed, given that K is extremely mobile in the soil solution, an increase in the soil volume explored by plant roots would have only marginal benefits. However, the plant does increase the expression of genes encoding high-affinity K^+-uptake systems when it becomes K deficient [12,18,22,55].

Putative components of the early perception and signalling of K deficiency include reactive oxygen species and the ethylene and jasmonic acid signaling pathways [10,18,54,55,79,80]. Proteomic studies show that the abundance of a negative regulator of the ABA signalling pathway (ATHB6), and of indole-3-glycerole phosphate synthase (IGS), are increased after long-term K^+ deprivation [81].

Magnesium deficiency increases the concentrations of sugars and starch in leaves (Figure 1), [32,33,40–43] and a clear inverse relationship between leaf Mg concentrations and sugar content has been demonstrated [42,43]. At present, no transcriptomic data on Mg deficiency are available. The early accumulation of sugars together with low Mg levels in the leaves seems to result in a downregulation of genes involved in photosynthesis, such as that encoding the chlorophyll *a/b* binding protein (*Cab2*) [43] and, consequently, account in part for the delayed decline in

chlorophyll content and photochemical performance [33,42,43]. Self-imposed heterotrophic conditions in source leaf tissues, rather than a reduction in the amount of Mg available for chlorophyll biosynthesis, could be at the origin of the decrease in chlorophyll content [43]. In contrast to N and P deficiencies, Mg deficiency impairs both sugar metabolism and sucrose export from source leaves [33,42,43]. Given that carbon allocation to the youngest leaves is likely to be affected more than carbon allocation to the root [42,43], an increase in R:S ratio is observed in certain species (e.g. Figure 1) [41–43], which is generally attributed to Mg deficiency reducing the growth of young leaves more than the growth of roots. It has been suggested that reduced sucrose export (Figure 4) can be explained by either (i) a decrease in the metabolic activity of sink organs [40], or (ii) impaired phloem loading, because this process requires Mg (Mg–ATP is a substrate for H^+ pumps) [33]. The second hypothesis is supported by most recent studies [42,43]. Interestingly, a sucrose–H^+ symporter gene is induced in the uppermost expanded leaves of Mg-deficient beet, but this does not increase sucrose loading into the phloem [42].

Conclusions

Plants deficient in N and P improve their ability to acquire these mineral elements by altering their carbon partitioning to favour root growth and by optimizing root morphology. Interestingly, one of the early physiological effects of N and P deficiencies is a rerouting of primary metabolism and the accumulation of sugars in leaves. We propose that this rerouting increases the transport of sugars to the root, which serves to increase the R:S biomass ratio and, in tandem with changes in hormone concentrations, modifies root morphology. This enables plants to respond appropriately to N and P deficiencies (Figure 4) and to forage more effectively for minerals with low availability in the rhizosphere. Transcriptional analysis of plants responding to N and P starvation suggests that they share common signal transduction pathways. However, whether sugar accumulation is a downstream response or part of a systemic signalling system is not yet clear. Given the scarcity of knowledge, we are unable to provide uncontested links between the increase in sugars and nutrient deprivation signalling. Nevertheless, one likely effect of increasing leaf sugar concentrations is the regulation of genes involved in photosynthesis and an acceleration of sucrose export from the leaf. By contrast, although deficiencies of Mg and K lead to the accumulation of sugars in young source leaves, it is unavailable for root growth because phloem transport is impaired in K- and Mg-deficient plants because of biochemical or biophysical limitations (Figure 4). This could explain the contrasting phenotypes of plants responding to N or P and K or Mg deficiency.

Although some of the genetic, biochemical and physiological consequences of mineral deficiencies have been glimpsed, little is known about how mineral deficiencies are perceived by plants. Recent studies have begun to focus on the sites of perception of mineral deficiencies and signalling cascades in *Arabidopsis* [25,55,71,79,80]. Future work must endeavour to link these aspects with consequent genetic, biochemical and physiological events. In particular, it will be important to dissect the interactions between sugar, metabolite and hormonal signals in the context of metabolic optimization, resource partitioning and plant development. This will be facilitated by advances in high-throughput profiling of the transcriptome, proteome, metabolome and ionome [24]. Knowledge of molecular responses to mineral deficiencies in crops such as Brassica [21], rice [82], tomato [12] and lupin [16,66] are a step towards the creation of varieties that have improved mineral acquisition and make more efficient use of minerals, and the development of novel strategies for sustainable agriculture [54,83].

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