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Metabolic changes sustain the plant life in low-sulfur environments

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Plants assimilate inorganic sulfate into various organic sulfur (S) compounds, which contributes to the global sulfur cycle in the environment as well as the nutritional supply of this essential element to animals. Plants, to sustain their lives, adapt the flow of their S metabolism to respond to external S status by activating S assimilation and catabolism of stored S compounds, and by repressing the synthesis of secondary S metabolites like glucosinolates. The molecular mechanism of this response has been gradually revealed, including the discovery of several regulatory proteins and enzymes involved in S deficiency responses. Recent progress in this research area and the remaining issues are reviewed here.

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Introduction

Sulfur (S) is an essential macronutrient for all organisms. Plants use inorganic sulfate as an S source and assimilate it into a variety of organic S compounds [1–4]. Animals are unable to assimilate sulfate and consume S-containing amino acids and proteins as dietary S sources, which suggests the importance of plants in the global S cycle in nature [3,4].

Plants take up sulfate through the activity of sulfate transporters (SULTRs) [3,5]. Sulfate absorbed into root cells is transported to the plastids, activated by ATP sulfurylase (ATPS) to form 5′-adenylylsulfate (APS), and reduced to sulfide by a two-step-reaction catalyzed by APS reductase (APR) and sulfite reductase [1–4,6]. Cysteine, the first organic form of S, is then synthesized from sulfide and *O*-acetyl-L-serine (OAS) and further converted into methionine and glutathione (GSH) [1–4,6]. In addition to these, a number of plant metabolites containing sulfur act as redox controllers, vitamins, coenzymes, lipids, flavors, and defense compounds [3,4,7–9]. Glucosinolates (GSL) are the major secondary S metabolites in defenses against pathogens and herbivores as well as possibly in S storage in Brassicaceae species, including *Arabidopsis*, for example [7,8,10,11].

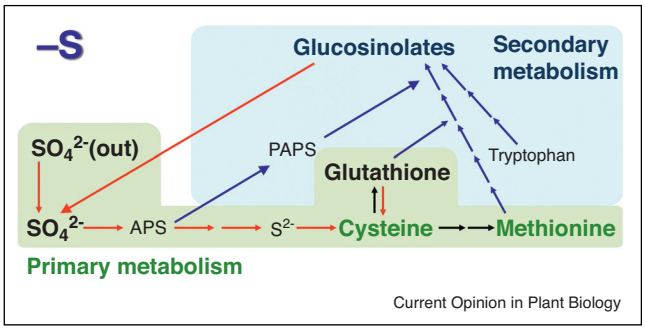
Sulfate concentration in soil is not always high enough to provide adequate S for plant growth, in which case S deficiency (–S) causes growth retardation [4,6]. In general, an –S condition decreases the quantity of S-containing compounds such as sulfate, cysteine, GSH, and GSL, and increases the OAS contents. Along with S metabolism, many metabolic pathways such as photosynthesis and nitrogen assimilation are also affected by –S. These influences of –S sometimes result in the reduction of crop yield and quality. To overcome such influences, plants have evolved systems to adapt to low-S environment (Figure 1). Here, I review recent advances in our understanding of the molecular mechanisms of plant adaptations to low-S environments.

Sulfate uptake is activated by low S conditions via a myriad of activation processes

Sulfate is the starting metabolite for S assimilation, so sulfate acquisition is critical for plant life. In –S environments, plants activate sulfate uptake by inducing the expression of high-affinity sulfate transporters that facilitate uptake of sulfate from the rhizosphere; in *Arabidopsis*, these are *SULTR1;1* and *SULTR1;2* (Figure 2) [12–14]. Retrieval of sulfate stored in vacuoles is also activated, by inducing the expression of *SULTR4;1* and *SULTR4;2* [15]. The –S-induced expression of the 4 SULTRs depends on the promoter activities of their 5′-regions [15–17]. The sulfur-responsive *cis*-acting element SURE11, which contributes to the enhanced expression of *SULTR1;1* under –S, exists in the 5′-region of *SULTR1;1* [18]. SURE11 has been found in the 5′-region of many –S-induced genes including *SULTR4;2*, but not in that of *SULTR1;2*, which is consistent with previous reports that suggest the different regulatory mechanisms underlying the expression of *SULTR1;1* and *SULTR1;2* [16,19].

Recently, epigenetic regulation of sulfate uptake and assimilation has been suggested based on the analysis of the *more sulfur accumulation1* (*msa1-1*) mutant [20••]. In this mutant, both the expression of –S induced genes such as *SULTR1;1*, *SULTR1;2*, and *APR* family genes and the

Figure 1



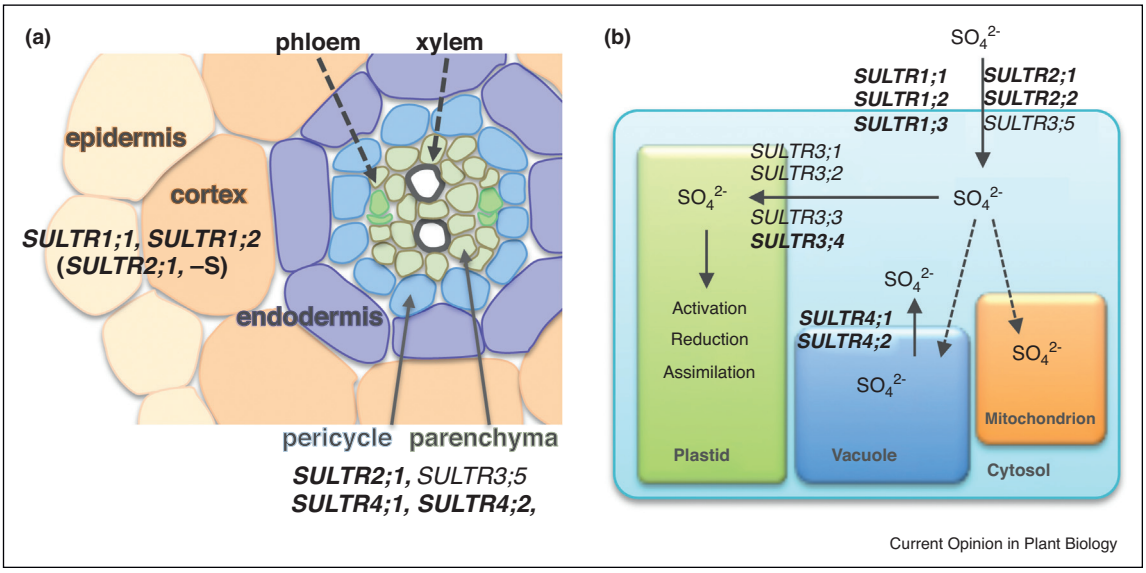
Major metabolic processes of sulfur (S) and the responses to sulfur deficiency (–S) in *Arabidopsis*. The S assimilatory process from sulfate to cysteine and the synthesis of methionine and glutathione (GSH) are defined as primary S metabolism. The majority of glucosinolates (GSLs) are synthesized from methionine or tryptophan, which requires 3′-phosphoadenosine-5′-phosphosulfate (PAPS) and GSH. GSL metabolism is defined as secondary S metabolism. Plants activate the S assimilatory process and the degradation of GSH and GSLs, and repress GSL biosynthesis.

levels of total S, sulfate, sulfite, cysteine, GSH and methionine are highly induced under S-sufficient conditions. MSA1 is the nuclear protein required for both S-adenosylmethionine (SAM) biosynthesis and DNA methylation, and its transcript levels are highly prompted by –S (Figure 2). SAM production and genome-wide DNA methylation, including DNA methylation of SURE11, *SULTR1;1*, *SULTR1;2*, and *APR3*, are affected in the *msa1-1* mutant. Elevated S accumulation in the mutant disappears due to the disruption of *SULTR1;1* and *SULTR1;2*, suggesting that MSA1 is a key regulator of maintaining S homeostasis, at least by regulating sulfate uptake capacity.

Vascular transport of sulfate is also activated under –S conditions [15,21,22]. Root-to-shoot sulfate transport is stimulated by the SULTR2;1 and SULTR3;5 expressed in the xylem parenchyma and pericycle cells of roots, which bring the apoplasmic sulfate to these cells [22–24,25••]. Co-expression of SULTR2;1 with SULTR3;5 increases sulfate uptake activity in yeast, which suggests that the induced expression of *SULTR2;1* under –S in roots might be a key for the increased root-to-shoot sulfate transport [22]. The –S-induced expression of *SULTR2;1* in roots is mediated by the *cis*-acting element SURE21, located in the 3′-non-transcribed region [25••]. SURE21, which consists of the two regions SURE21A and SURE21B, is found only in the 3′-region of *SULTR2;1*. The T-DNA insertion in the 3′-region of *SULTR2;1* (tKO) decreases root-to-shoot translocation of sulfate specifically under –S conditions. Another contribution of SULTR2;1 to the uptake of sulfate under –S is suggested from the induction of *SULTR2;1* expression in the cortex and the reduction of sulfate uptake activity in tKO lines [25••].

Several post-transcriptional regulations of sulfate transport and assimilation have been reported. One is the unknown post-transcriptional mechanisms that maintains the SULTR1;1 and SULTR1;2 protein levels under –S [14]. A second is the regulation mediated by microRNA395 (miR395) expression under –S (Figure 3) [26]. By targeting and reducing the transcripts of ATPS1,

Figure 2



Distribution of sulfate transporters (SULTRs) in (a) root tissues and (b) plant cell. *SULTRs* induced by –S are presented in bold.