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BOTANICAL BRIEFING

Plant KT/KUP/HAK Potassium Transporters: Single Family – Multiple Functions

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• *Background and Aims* Potassium transporters belonging to the KT/KUP/HAK family are important for various aspects of plant life including mineral nutrition and the regulation of development. Genes encoding these transporters are present in the genomes of all plants, but have not been found in the genomes of Protista or Animalia. The aim of this Botanical Briefing is to analyse the function of KT/KUP/HAK transporters from evolutionary, molecular and physiological perspectives.
• *Scope* This Briefing covers the phylogeny and evolution of KT/KUP/HAK transporters, the role of transporters in plant mineral nutrition and potassium homeostasis, and the role of KT/KUP/HAK transporters in plant development.

Key words: Potassium transporters, mineral nutrition, K^+ transport, plant development, homeostasis, salt stress, root-hairs, cell expansion.

INTRODUCTION

Potassium is an essential macronutrient, which is important for various aspects of plant life. In contrast to other metals, the concentration of potassium in living cells is very high and in plants, particularly, may reach up to 8 % of the dry weight (Evans and Sorger, 1966). The high abundance of this cation in the cell implies its function in maintenance of cellular osmolarity and compensation of negative electrical charges associated with organic molecules. As a major cellular solute, K^+ is important for turgor-dependent cell expansion, e.g. in plant tropisms and the regulation of stomatal aperture. At the biochemical level, the potassium-rich environment is essential for activity of various cytosolic enzymes (Evans and Sorger, 1966).

Although potassium is one of the most abundant elements in the Earth's crust, the bulk of it is not readily available to plants because they acquire this mineral from the soil solution only in ionic form. Owing to the fast uptake of the nutrient by plants, the actual concentrations of K^+ at the surface of a root are even lower than in bulk soil solution and often fall down to the μM range (for a recent review, see Ashley *et al.*, 2006). In order to maintain an adequate potassium status under conditions of potassium deficiency, plants have developed a sophisticated mechanism of potassium acquisition, which enables them to transport the cation against >1000-fold concentration gradients.

In classical experiments on potassium acquisition in barley, Epstein demonstrated for the first time that potassium uptake in plants is mediated by at least two systems, characterized by low and high affinity for K^+ (Epstein *et al.*, 1961). Because K^+ concentrations in soil are quite variable (Ashley *et al.*, 2006), the existence of multiple K^+ uptake systems in roots is vital for the efficient mineral nutrition in a dynamic environment.

Channels and carrier-type transporters, which in the recent Transporter Classification (TC) System (Busch, 2002) are attributed to the Class 1 (channels/porins) and Class 2 (electrochemical potential-driven transporters), respectively, constitute two major pathways for K^+ acquisition in plants. Porters (TC: 2·A) are the largest group of the Class 2 transporters and incorporate uniporters, symporters and antiporters. An important feature of antiporter- and symporter-mediated processes is that they can be energized by the proton- or sodium-motive force and therefore some Class 2 transporters are capable of transporting a substrate against steep concentration gradients. This property of the Class 2 transporters implies their role in K^+ acquisition at low external $[K^+]$. Indeed it has been demonstrated that K^+ uptake in *Arabidopsis thaliana* roots must be energized if external $[K^+]$ are <1 mM (Maathuis and Sanders, 1993). The required extra energy for K^+ uptake can be provided either through coupled transport mediated by a carrier-type transporter or through a hypothetical K^+-motive ATPase (Kochian and Lucas, 1988). The existence of K^+ ATPase in the plant plasma membrane has not been confirmed yet and therefore carrier-type (Class 2) transporters have been suggested to facilitate K^+ uptake in the high-affinity range of concentrations. Low-affinity K^+ transport is conventionally attributed to channels (Maathuis and Sanders, 1997). Recent findings suggest, however, that channels also contribute to high-affinity K^+ uptake. Disruption of AKT1 potassium channels in the *akt1-1 Arabidopsis* mutant, for instance, is associated with reduced ability of plants to grow in low (10 μM) external $[K^+]$ (Hirsch *et al.*, 1998). This potassium-dependent growth defect was also accompanied by reduced permeability of the plasma membrane to K^+. Effects of the *akt1-1* mutation on plant growth, however, were observed only in the presence of NH_4^+ in the nutrient medium (Hirsch *et al.*, 1998). The conditional redundancy of the AKT1 channel strongly indicates that its function in high-affinity K^+ uptake can be complemented by a different system, which is sensitive to NH_4^+. It has also been demonstrated that the K^+ transport capacity of this second system is

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stimulated by increased concentrations of H^+ and Na^+ in the medium (Spalding *et al.*, 1999). Because of this, it is quite likely that this system comprises the carrier-type (Class 2) transporters, in which the energization of K^+ uptake is achieved through coupling with H^+ or Na^+ transport.

In plants, there are four multi-gene families of Class 2 transporters (Mäser *et al.*, 2001) which may contribute to K^+ transport: *Trk/HKT* (TC: 2·A·38), *KEA* (K^+/H^+ antiporters, TC: 2·A·37), *CHX* (cation/H^+ antiporters, TC: 2·A·37) and *KT/KUP/HAK* (TC: 2·A·72). The HKT1 transporter from wheat (*Triticum aestivum*), belonging to the Trk/HKT family, has been shown to mediate K^+/Na^+ co-transport in heterologous expression systems (Rubio *et al.*, 1995). In arabidopsis, however, the *HKT1* orthologue does not contribute to potassium nutrition and is solely involved in the long-distance transport of sodium (Berthomieu *et al.*, 2003). HKT transporters from barley (*Hordeum vulgare*) and rice (*Oryza sativa*) have also been shown to function just as sodium uniporters (Garciadeblas *et al.*, 2003; Haro *et al.*, 2005). The *CHX* gene family in arabidopsis consists of 28 members, which are homologous to mammalian and bacterial *C*ation/H^+ e*X*changers. Most of these transporters facilitate Na^+ transport, but one member of the family, *AtCHX17*, is involved in K^+ acquisition and homeostasis in arabidopsis (Cellier *et al.*, 2004). *KEA* transporters have been identified in the arabidopsis genome through their homology to bacterial K^+/H^+ transporters. So far, there is no physiological evidence that KEA transporters play a role in potassium transport in plants, but activation of *KEA5* by potassium deficiency (Shin and Schachtman, 2004) may imply its involvement in K^+ homeostasis.

Plant transporters homologous to bacterial *kup* (K^+ *up*take) were first identified and cloned from arabidopsis and barley (Quintero and Blatt, 1997; Santa-Maria *et al.*, 1997). Different research groups have used different acronyms for *kup* orthologues in plants (Quintero and Blatt, 1997; Santa-Maria *et al.*, 1997; Fu and Luan, 1998; Kim *et al.*, 1998) and these transporters are commonly referred as the *KT/KUP/HAK* gene family. This review is focused primarily on this family of potassium transporters because of their importance for a variety of essential physiological processes in plants including nutrient acquisition and regulation of plant development.

PHYLOGENY AND EVOLUTION OF KT/KUP/HAK TRANSPORTERS

Bacterial Kup transporters

The Kup (TrkD) potassium uptake system in bacteria was identified by Epstein and Kim (1971) through mutagenesis of K^+-dependent (Kdp) *Escherichia coli* strains carrying the *kdpABC5* mutation. Introduction of the *trkD1* mutation into a strain with mutated *trkA* and *kdp* K^+ transport systems further exacerbated growth in low [K^+] media (Epstein and Kim, 1971). Half-maximal growth rate was observed at 16 mM K^+ in the strain TK401 (*kdpABC5 trkA401 trkD1*), in which all three K^+ uptake systems, Kdp, TrkA and TrkD, were mutated as compared with 2·3 mM in the strain TK133 (*kdpABC5 trkA133*) with functional TrkD (Rhoads *et al.*, 1976). An ability to grow in low [K^+] was only moderately affected by the *trkD1* mutation in a *kdpABC5* background (Rhoads *et al.*, 1976). The latter fact suggests that the Kup (TrkD) transporters may be functionally redundant in *E. coli* at standard experimental conditions and their role in K^+ uptake is masked by the more active TrkA system. K^+ uptake through the Kup (TrkD) was approx. 2-fold stimulated when pH was reduced from 7·0 to 5·6, while the TrkA system was inhibited under these conditions by 50 % (Rhoads *et al.*, 1976). In agreement with these data it was subsequently demonstrated that the bacterial Kup transporter is crucial for K^+ uptake by cells under stress conditions such as hyper-osmolarity at low pH (Trchounian and Kobayashi, 1999). Stimulation of K^+ uptake by low external pH suggests that this transporter may function as a K^+/H^+ symporter.

Occurrence of KT/KUP/HAK *transporter genes in prokaryotic and eukaryotic genomes*

Although KT/KUP/HAK transporters were first identified in bacterial cells, their genes are not ubiquitously present in prokaryotic genomes. As based on the analysis of the Integrated Microbial Genomes (IMG) database (http://img.jgi.doe.gov) only 38 % of species in Bacteria contain these transporters (Fig. 1). This percentage is even lower in Archaea (IMG, http://img.jgi.doe.gov), where only two species out of the 24 analysed contain the *kup* gene (Fig. 1). The results of genomic analysis are consistent with physiological data, indicating that Kup transporters may play a crucial role in some habitats (Trchounian and Kobayashi, 1999), but are functionally redundant in others (Epstein and Kim, 1971; Rhoads *et al.*, 1976). Analysis of the Gene Indices database at the Institute for Genomic Research (TIGR; http://www.tigr.org/tdb/tgi/index.shtml) and the eukaryotic database at the DOE Joint Genome Institute (JGI; http://genome.jgi-psf.org/euk_home.html) shows that among eukaryotes, Kup orthologues are found in Plantae, Fungi and probably Amoebozoa. Interestingly, all plant genomes analysed contain genes encoding KT/KUP/HAK transporters, while in Fungi these genes are found only in 12 species out of 21 [plant indices containing <5000 unique sequences (*Cocoa* and *Petunia*) were excluded from the analysis]. Some ESTs homologous to the 5′ end of *KT/KUP/HAK* transporter genes were generated from *Dictyostelium discoideum* transcripts (http://www.tigr.org/tdb/tgi/protist.shtml).

Correlation between the genomic occurrence of KT/KUP/HAK transporters and the predominant mode of nutrition in a taxonomic group

As shown in Fig. 1, *KT/KUP/HAK* transporter genes are found primarily in organisms that acquire nutrients through absorption. The concentration of nutrients like K^+ is often very low in surrounding media and therefore an absorptive mode of nutrition requires a more sophisticated system of membrane transporters as compared with ingestion. Organisms obtaining nutrients through ingestion forage on potassium-rich organic matter and probably do not require

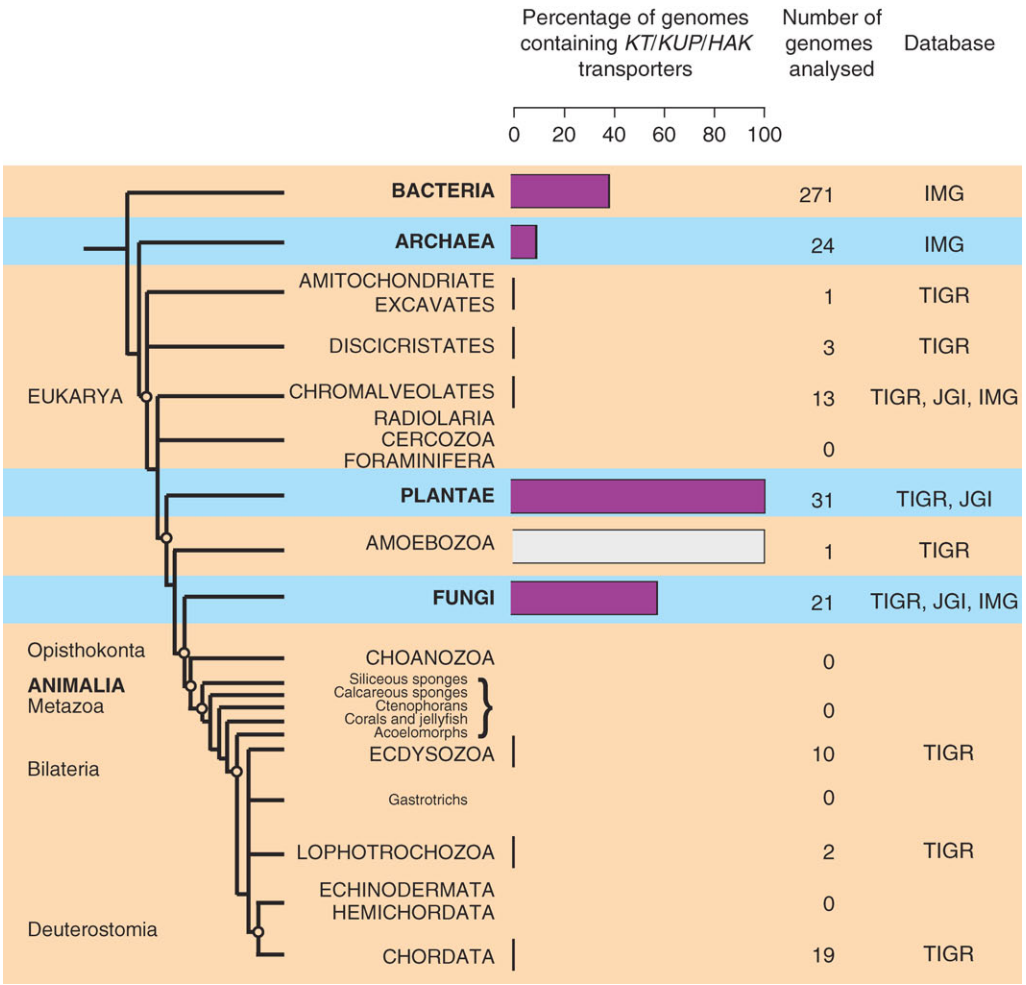

FIG. 1. Genomic distribution of *KT/KUP/HAK* transporters. *KT/KUP/HAK* transporter genes are present in all plant genomes studied so far (see text for details), but were not found in Protista and Animalia. The presence of these transporters in Amoebozoa requires further confirmation. The evolutionary tree is adapted from Pennisi (2003).

KT/KUP/HAK transporters for their potassium homeostasis. Apart from *D. discoideum*, which in the feeding stage obtains nutrients through ingestion (phagocytosis), no *KT/KUP/HAK* transporters have been found in the genomes of such organisms.

In plants, *KT/KUP/HAK* transporter genes have been found in evolutionarily diverse organisms ranging from green algae to angiosperms (Fig. 2). The ubiquitous presence of these genes in plants implies that they play an important role in nutrient acquisition and in the ability of plants to survive in potassium-poor environments. The evolution of such an ability was a prerequisite for the colonization of land by plants, because the concentration of potassium in soil may be up to 100-fold lower than in ocean water (Adams, 1971; Garciadeblas *et al.*, 2002). The presence of *KT/KUP/HAK* transporter genes in ancestral plant genomes was probably crucial for development of terrestrial plants.

The Fungi is another major Kingdom in the Eukaryota, in which *KT/KUP/HAK* transporter genes are found. Although fungi, as well as plants, are sessile and obtain nutrients by absorption, they are chemoheterotrophs and feed on either living organisms or dead organic matter. These sources are rich in minerals and adequate potassium supply in some fungi can probably be achieved without KT/KUP/HAK transporters. This may explain why only 57 % of fungal genomes analysed contain these genes.

ROLE OF KT/KUP/HAK TRANSPORTERS IN MINERAL NUTRITION AND POTASSIUM HOMEOSTASIS IN PLANTS AND FUNGI

HAK1 transporter from Schwanniomyces occidentalis *is crucial for high affinity potassium uptake*

Although *KT/KUP/HAK* transporter genes are not ubiquitous in fungi, they are crucial for fungal nutrition in some habitats. It is not surprising therefore that the first eukaryotic *KT/KUP/HAK* transporter gene, *HAK1* (*H*igh *A*ffinity K^+ transporter), was cloned from *Schwanniomyces occidentalis*, an ascomycete yeast which is able to grow in a nutrient-poor environment (Banuelos *et al.*, 1995). Particularly remarkable is the ability of *S. occidentalis* to

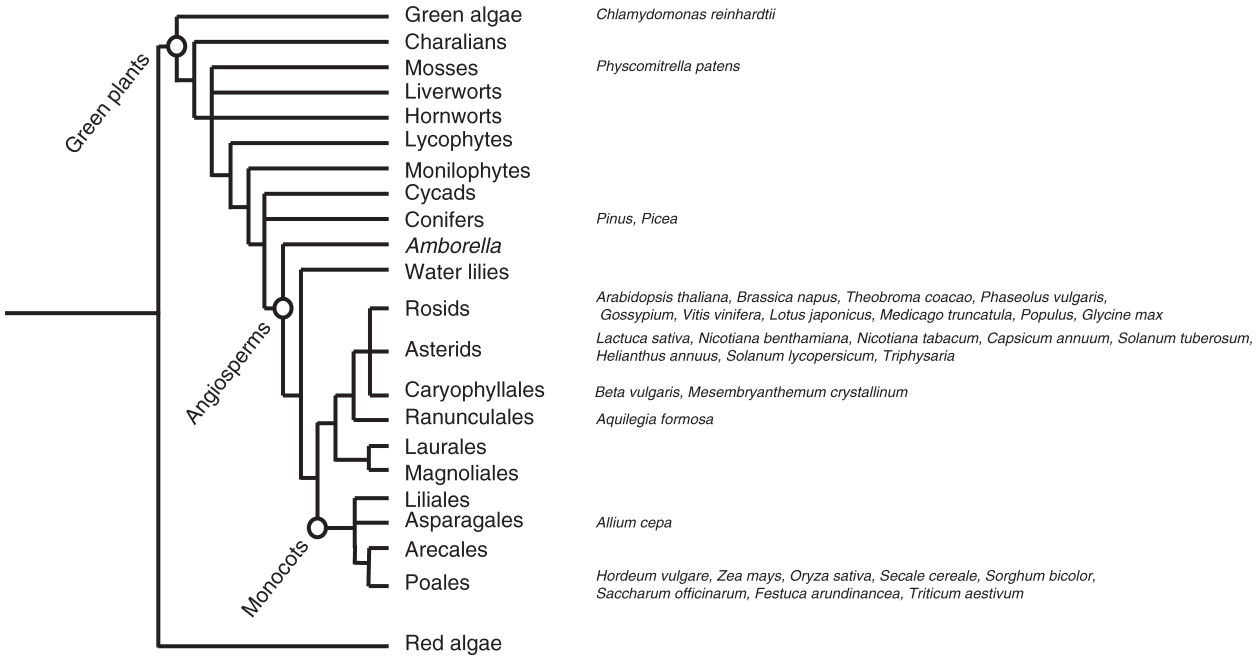

FIG. 2. *KT/KUP/HAK* transporter genes in plant genomes. Data were obtained through the mining of TIGR and JGI genomic databases. The evolutionary tree is adapted from Pennisi (2003).

accumulate K^+ from solutions in which the concentration of this nutrient is as low as 0·03 μM. If the cytosol contains around 100 mM K^+, this organism is able to transport the cation against a >3000 000-fold concentration gradient! The extraordinarily high K^+ concentrative capacity of *S. occidentalis* is largely due to the activity of the HAK1 potassium transporter (Banuelos *et al.*, 1995). This remarkable finding provided the first experimental evidence that KT/KUP/HAK transporters might constitute a major high-affinity potassium acquisition system in eukaryotes.

Phylogeny of plant KT/KUP/HAK transporters

In plants the transporters are present as multi-gene families and in arabidopsis, for instance, there are 13 *KT/KUP/HAK* genes, while the rice genome contains at least 27 members of the family. As proposed recently (Rubio *et al.*, 2000; Banuelos *et al.*, 2002) all KT/KUP/HAK transporters can be grouped into four distinct clusters. This idea is confirmed by the phylogenetic analysis of the *KT/KUP/HAK* transporter genes for which full-length sequences are available. The most studied members of the *KT/KUP/HAK* family belong to the two largest groups, which were named Clusters I and II (Banuelos *et al.*, 2002). Current analysis indicates that all plants have transporters homologous to members of Cluster I or Cluster II. To date, genes belonging to Cluster III are found only in arabidopsis and rice. The smallest cluster is number IV, which comprises only four rice genes.

The role of Cluster I transporters in high affinity K^+ uptake

All transporters of Cluster I characterized so far display high affinity for the substrate and therefore they are likely to play a key role in potassium acquisition, particularly when K^+ availability is low (Banuelos *et al.*, 2002; Rodriguez-Navarro and Rubio, 2006). The HvHAK1 transporter from barley is likely to represent Epstein's high-affinity uptake system because its K_m (27 μM; Santa-Maria *et al.*, 1997) is remarkably close to the K_m of System I (21 μM; Epstein *et al.*, 1961). In agreement with the proposed function of HvHAK1 as the main high-affinity potassium uptake system in barley roots, its expression is induced by potassium starvation (Santa-Maria *et al.*, 1997). Similarly, expression of *HvHAK1* orthologues in tomato (*Solanum lycopersicum* syn. *Lycopersicon esculentum*; *LeHAK5*) and arabidopsis (*AtHAK5*) is activated by low external [K^+] (Wang *et al.*, 2002; Ahn *et al.*, 2004; Hampton *et al.*, 2004; Gierth *et al.*, 2005). Genetic mapping of natural variations in [K^+] in arabidopsis also indicated that *AtHAK5* is associated with potassium accumulation (Harada and Leigh, 2006). In line with its function in potassium acquisition, *AtHAK5* is expressed in the epidermis of main and lateral roots (Gierth *et al.*, 2005). The role of AtHAK5 in K^+ uptake was further verified through analysis of $^{86}Rb^+$ fluxes in *athak5* mutant plants (Gierth *et al.*, 2005).

How do plants sense potassium deficiency?

Induction of *AtHAK5* expression by K^+ deficiency raises an important question: how is this response triggered? Do plants sense external K^+ or are conditions of mineral deficiency recognized through internal signalling associated with low plant potassium status?

There are several lines of evidence indicating that internal signals regulate *AtHAK5* expression (Gierth *et al.*,

2005) and K^+ uptake in general (Siddiqi and Glass, 1987). This idea is also supported by the finding that expression of *AtHAK5* is enhanced in mutants in which the K^+ channel *AtAKT1* is disrupted (Hampton, 2005). Disruption of *AtAKT1* obviously affects potassium supply (Hirsch *et al.*, 1998) and alterations in plant potassium status may trigger *AtHAK5* expression. The signal transduction cascade that regulates responses to low [K^+] has yet to be identified, but there is strong evidence that jasmonate, ethylene and reactive oxygen species pathways may be involved (for recent reviews, see Amtmann *et al.*, 2005; Ashley *et al.*, 2006).

Some studies suggest, however, that the sensing of external [K^+] may also be involved in the regulation of potassium homeostasis (Maathuis and Sanders, 1997) and K^+ transporters are prime candidates for this function. Gating of the GORK channel, for instance, is regulated by external [K^+] and therefore this potassium channel may serve as a sensor of the cation (Ivashikina *et al.*, 2001). The AtKUP4/TRH1 potassium transporter may also be involved in the sensing of external [K^+] and regulation of potassium-dependent root development (Vicente-Agullo *et al.*, 2004; Ashley *et al.*, 2006).

K^+/H^+ co-transport energizes high-affinity K^+ uptake

In plant cells, the negative electrical potential across the plasma membrane is generated primarily through activity of the H^+-ATPase, which transports H^+ from the cell to the external medium. This potential constitutes a component of the driving force for the uptake of cations. At high- and mid-ranges of external concentrations, K^+ transport can be driven entirely by transmembrane electrical potential, but K^+ acquisition from K^+-deprived soil requires an additional source of energy (Banuelos *et al.*, 1995). The proton-electrochemical gradient, which normally is inwardly directed, may energize potassium uptake, and indeed it is utilized by some KT/KUP/HAK transporters, which co-transport K^+ and H^+ (Rodriguez-Navarro, 2000).

The role of Cluster II transporters in ionic homeostasis

The physiological functions of Cluster II transporters are probably quite diverse and their role in potassium nutrition is not well defined. Most of these transporters are likely to facilitate the low-affinity K^+ transport complementing potassium channels (Senn *et al.*, 2001; Garciadeblas *et al.*, 2002). Some transporters of Cluster II are probably localized to the tonoplast similarly to OsHAK10 (Garciadeblas *et al.*, 2002). The putative function of the tonoplast KT/KUP/HAK transporters is to facilitate K^+ efflux from the vacuole. The efflux process is particularly important for the maintenance of K^+ homeostasis in K^+-deprived plants. Under such conditions, the K^+ content of vacuoles is quite low and transport of the cation from the organelle to the cytosol must be energized (Walker *et al.*, 1996; Garciadeblas *et al.*, 2002). In contrast to K^+ channels, the KT/KUP/HAK transporters can facilitate such transport by utilizing the energy of the trans-tonoplast H^+ gradient. It has been shown that under conditions of K^+ deprivation, export of K^+ from the vacuole can be mediated by a K^+/H^+ symporter with a 1 : 1 stoichiometry (Walker *et al.*, 1996).

In arabidopsis, the Cluster II genes *AtHAK6* and *AtHAK2*, alongside *AtHAK11* of Cluster III, may be involved in plant responses to salinity because their expression is affected by increased salt concentrations (Maathuis, 2006). *AtHAK6* and *AtHAK11*, however, are up-regulated while the amount of the *AtKUP2* transcript is reduced under the stress conditions. Owing to physical and chemical similarities between the two alkali cations, sodium interference under conditions of salt stress affects K^+ acquisition and the activity of potassium-dependent enzymes, including transporters of Cluster II (Banuelos *et al.*, 1995). Higher expression of *AtHAK6* and *ATHAK11* may help to achieve the required rate of potassium transport and to restore Na^+/K^+ balance in plants at high salt concentrations. AtKUP2 is known to regulate cell size (Elumalai *et al.*, 2002; see below) and therefore the reduced expression of this transporter could be important for developmental rather than physiological responses to salt stress.

Comparative studies of KT/HAK/KUP transporters from barley and the sea grass *Cymodocea nodosa* indicate a remarkable correlation between their characteristics and plant habitat. The transport activity of the HvHAK2 is extremely sensitive to the presence of Na^+, whereas CnHAK1 from *C. nodosa* is practically unaffected by salt (Garciadeblas *et al.*, 2002).

POTASSIUM TRANSPORTERS AND PLANT DEVELOPMENT

Regulation of the cell size

Some KT/KUP/HAK transporters have been found to play important roles in plant development. The mutation *shy3-1* in the *AtKUP2* gene, for instance, causes a reduction in the size of arabidopsis shoot cells (Elumalai *et al.*, 2002). Because K^+ is a major cellular solute, impairment in potassium homeostasis may weaken cell turgor and thus restrict rates of cell expansion. However, [K^+] in the *shy3-1* mutant is only marginally lower than in wild-type plants. Puzzlingly, this mutation had little effect on the ability of the protein to transport K^+ when produced in a heterologous expression system, and the *atkup2* null mutant did not display a morphological phenotype (Elumalai *et al.*, 2002). Currently, no clear explanation of the observed effects is available, but partial dominance of *shy3-1* alongside other observations suggests that the mutation may cause gain of function and/or alteration of protein properties (Elumalai *et al.*, 2002). It remains obscure, however, whether the affected function is the K^+ transport. Alternatively it has been suggested that the *shy3-1* mutation may affect activities of other proteins, which normally interact with AtKUP2 and are involved in regulation of the cell size (Elumalai *et al.*, 2002).

The role of the KT/KUP/HAK transporters in turgor-dependent growth has been demonstrated in rapidly expanding cotton fibres (*Gossypium hirsutum*), where expression

of the *GhKT1* member of the gene family correlates positively with build-up of turgor pressure (Ruan *et al.*, 2001). In growing grapevine fruits (*Vitis vinifera*) expression of *VvKUP1* and *VvKUP2* potassium transporter genes is also strongly dependent on developmental stage. It is likely that these transporters are required for the potassium-driven cell expansion in young grape berries (Davies *et al.*, 2006).

Root-hair development and gravitropic responses

The *trh1* (*t*iny *r*oot-*h*air *1*) mutant, in which the *AtKUP4/TRH1* gene is disrupted, was identified through phenotyping of a T-DNA-tagged arabidopsis population (Rigas *et al.*, 2001). Root-hairs are initiated in this mutant, but they fail to elongate and appear as small bulges on the surface of epidermal cells (Rigas *et al.*, 2001). Apart from this defect, *trh1* roots display agravitropic behaviour when grown on vertical agar plates (Vicente-Agullo *et al.*, 2004; Grabov *et al.*, 2005). Surprisingly, none of the morphological defects observed in *trh1* is rescued by increased external [K^+]. Therefore, similarly to *shy3-1*, *trh1* phenotypes are unlikely to be due to a K^+ deficit *per se*. This idea was further supported by the observation that the highest level of expression of the *TRH1* gene was observed in the root-cap and not in epidermal cells, the development of which is affected by the *trh1* mutation (Vicente-Agullo *et al.*, 2004). The fact that both root-hair elongation and gravitropic responses in *trh1* are complemented by exogenous auxin points to a role for the TRH1 potassium transporter in auxin signalling and transport. Indeed, experiments with the *trh1* mutant expressing an auxin-sensitive *DR5–GUS* construct demonstrate that disruption of the *TRH1* transporter is associated with distortion of auxin profiling in the arabidopsis root (Vicente-Agullo *et al.*, 2004). The DR5–GUS expression pattern in the *trh1* root provides evidence that TRH1 is required for the facilitation of polar auxin transport in the root-cap. Figure 3 illustrates auxin distribution in wild-type and *trh1* plants. According to this scheme, disruption of the TRH1 transporter partially blocks auxin flow through the root-cap, resulting in sub-optimal concentrations of the phytohormone in the cortex and epidermis. In accordance with the scheme in Fig. 3, blockage of auxin transport in the root-cap should also cause build-up of the phytohormone in the central cylinder, which indeed was observed in experiments with *trh1* plants expressing the *DR5–GUS* construct.

Although root-hair growth is independent of external [K^+], the *trh1* phenotype is rescued by phosphate deficiency (Muller and Schmidt, 2004). This fact again supports the idea that the *trh1* phenotype is due to a defect in signalling rather than in potassium supply for turgor-dependent growth.

It is not clear yet how the K^+ transporter facilitates auxin transport. TRH1 could either transport auxin directly or create the electrochemical gradients that drive phytohormone transport across the plasma membrane. A role for TRH1 in the regulation of trans-plasma membrane H^+ gradients, for instance, is quite plausible because many KT/KUP/HAK transporters are known to co-transport K^+ and H^+ (Rodriguez-Navarro, 2000). Alterations in H^+ transport may affect auxin transport in the *trh1* mutant, but this hypothesis has yet to be tested experimentally.

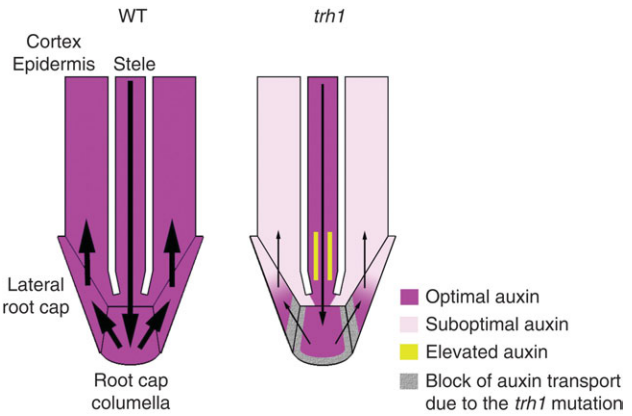

FIG. 3. Auxin fluxes in the root-tip of wild-type (WT) and *trh1* plants. The *trh1* mutation partially blocks auxin transport through the root-cap. This blockage causes a reduction in overall acropetal auxin transport through the stele and decreases in the auxin concentration in the cortex and epidermis. Defects in root-hair development and gravitropic behaviour in the *trh1* mutant are due to sub-optimal concentrations of auxin in the epidermis. Reproduced from Vicente-Agullo *et al.* (2004) with permission.

CONCLUDING REMARKS

The various functions of plant *KT/KUP/HAK* transporter genes range from mineral nutrition to the regulation of cell growth and development. Although all the transporters studied so far do transport potassium and some of them are important for potassium uptake, some morphological defects in *kt/kup/hak* knock-outs are not directly linked to impairment in potassium acquisition and/or homeostasis.

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