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Plant responses to potassium deficiencies: a role for potassium transport proteins

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Abstract

The availability of potassium to the plant is highly variable, due to complex soil dynamics, which are strongly influenced by root–soil interactions. A low plant potassium status triggers expression of high affinity K^+ transporters, up-regulates some K^+ channels, and activates signalling cascades, some of which are similar to those involved in wounding and other stress responses. The molecules that signal low K^+ status in plants include reactive oxygen species and phytohormones, such as auxin, ethylene and jasmonic acid. Apart from up-regulation of transport proteins and adjustment of metabolic processes, potassium deprivation triggers developmental responses in roots. All these acclimation strategies enable plants to survive and compete for nutrients in a dynamic environment with a variable availability of potassium.

Key words: Acclimation, mineral nutrition, plant plasticity, potassium, potassium deficiencies.

Potassium availability and nutrient dynamics in the rhizosphere

Potassium is one of the major nutrients, essential for plant growth and development. Although concentrations of K^+ in soil solution ($[K^+]_o$) are in the range of only 0.1–6 mM (Adams, 1971), plants accumulate large quantities of this element, which constitutes between 2% and 10% of plant dry weight (Leigh and Wyn Jones, 1984; Tisdale *et al.*, 1993). Concentrations of K^+ in the cytosol are maintained in a narrow range, around 100 mM, which is optimal for the function of cytosolic enzymes. Vacuolar content is more variable, depending on potassium availability and tissue type, and is commonly found to be in the range of 20–200 mM (Leigh and Wyn Jones, 1984; Walker *et al.*, 1996).

Potassium is the fourth most abundant mineral, constituting about 2.5% of the lithosphere. However, actual soil concentrations of this mineral vary widely, ranging from 0.04 to 3% (Sparks and Huang, 1985). In accordance with its availability to plants, soil potassium is ascribed to four different pools: (i) soil solution, (ii) exchangeable K, (iii) fixed K, and (iv) lattice K (Syers, 1998). As plants can only acquire K^+ from solution, its availability is dependent upon the nutrient dynamics as well as on total K content. The exchange of potassium between different pools in soil is strongly dependent upon the concentration of other macronutrients in the soil solution, for example, nitrate (Yanai *et al.*, 1996). The release of exchangeable K is often slower than the rate of K^+ acquisition by plants (Sparks and Huang, 1985) and, consequently, K^+ content in some soils is very low (Pretty and Stangel, 1985; Johnston, 2005). Plant potassium status may further deteriorate in the presence of high levels of other monovalent cations such as Na^+ and NH_4^+ that interfere with potassium uptake (Spalding *et al.*, 1999; Qi and Spalding, 2004; Rus *et al.*, 2004).

Apart from long-term deprivation, plant roots can experience transient shortages of potassium because of spatial heterogeneity and temporal variations in the availability of this nutrient. The main sources of soil heterogeneity are often plant roots themselves, the K^+ transport activity of which creates zones with elevated or reduced nutrient content.

Contact between a root and nutrient may occur because of (i) root growth into the area where a nutrient is located, and (ii) transport of a nutrient to the root surface through the soil (Jungk and Claassen, 1997). The first process, termed 'root interception', constitutes less than 1–2% of total K^+ uptake because of rapid removal of K^+ at the root surface (Barber, 1985; Rosolem *et al.*, 2003). The second process, K^+ translocation through the soil to the root surface, is facilitated by diffusion and mass flow (Barber, 1962). Diffusion is the dominant mechanism of K^+ delivery to the

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root surface (Seiffert *et al.*, 1995) and constitutes up to 96% of total soil K^+ transport (Oliveira *et al.*, 2004). Therefore, K^+ depletion around the root is the most frequently observed phenomenon associated with plant-evoked soil potassium perturbations. If nutrient delivery by diffusion is always associated with the reduction of K^+ content in the areas adjacent to the root surface, mass flow may, conversely, result in K^+ accumulation around the root if transpiration is high (Vetterlein and Jahn, 2004).

Experimentally, development of a depletion profile around individual maize root segments has been demonstrated using ^{86}Rb as a potassium tracer (Jungk and Claassen, 1997). These data are consistent with results obtained by Yamaguchi and Tanaka (1990), who demonstrated that roots compete for potassium if half the distance between them is less than 4 mm. Similar results were obtained with flat mats of maize (*Zea mays* L.), rape (*Brassica napus* L.), and rice (*Oryza sativa* L.) roots (Jungk and Claassen, 1997; Hylander *et al.*, 1999; Vetterlein and Jahn, 2004).

Variations in soil density may also affect potassium availability. Soil compaction is associated with higher volumetric water content and therefore tends to facilitate K^+ transport to the root surface (Kuchenbuch *et al.*, 1986). However, the dense soil may also cause a reduction in the root length and so the higher bulk density does not necessarily result in increased K^+ accumulation (Seiffert *et al.*, 1995).

The spatial heterogeneities in K^+ distribution encountered by a root are often superimposed with temporal variations in K^+ availability, caused by continuously changing soil moisture content. In dry soils, bulk K^+ content is normally higher, but mass flow and diffusion are restricted (Seiffert *et al.*, 1995; Vetterlein and Jahn, 2004; Kuchenbuch *et al.*, 1986). The negative effects of drought on K^+ transport in soil are likely to be more significant than increases in $[K^+]_o$ and therefore these environmental conditions lead to reduced availability of the nutrient (Kuchenbuch *et al.*, 1986; Seiffert *et al.*, 1995; Liebersbach *et al.*, 2004).

Mechanisms of potassium acquisition

Following Epstein's pioneering work, the potassium transport system in plants is considered to consist of low- and high-affinity components (Epstein *et al.*, 1961). At the molecular level, these components are conventionally attributed to the activities of channels and transporters, respectively (Maathuis and Sanders, 1994, 1997). Recent findings indicate that functions of the K^+ transport proteins are quite diverse and, at least in terms of affinity to K^+, the boundaries between transporters and channels are not clearly defined (Fu and Luan, 1998; Hirsch *et al.*, 1998; Spalding *et al.*, 1999). Channel-mediated transport has been studied in great detail because of the availability of advanced electrophysiological techniques, and the relative ease of expression in heterologous systems. Less information is available on transporters, which are characterized by a lower rate of K^+ transport.

Channels

Shaker-type channels: The Shaker-type K^+ channels KAT1 (Anderson *et al.*, 1992) and AKT1 (Sentenac *et al.*, 1992) were the first K^+ transporting proteins cloned from plants. Both AKT1 and KAT1 are activated by a more negative membrane potential and are highly selective for potassium. Of these two channels, only AKT1 is expressed in roots and involved directly in arabidopsis (*Arabidopsis thaliana* (L.) Heynh.) mineral nutrition (Lagarde *et al.*, 1996; Hirsch *et al.*, 1998). Several *AKT1* orthologues have been identified in other plant species, for example, *SKT1* in potato (*Solanum tuberosum* L.; Zimmermann *et al.*, 1998), *LKT1* in tomato (*Lycopersicon esculentum* Mill.; Hartje *et al.*, 2000), *MKT1* in common ice plant (*Mesembryanthemum crystallinum* L.; Su *et al.*, 2001), *TaAKT1* in wheat (*Triticum aestivum* L.; Buschmann *et al.*, 2000), and *OsAKT1* in rice (*Oryza sativa*; Golldack *et al.*, 2003).

Physiological characterization of the *akt1* knockout (KO) indicated that AKT1 mediates an NH_4^+-insensitive component of the K^+ uptake system in arabidopsis roots. Surprisingly, this channel facilitates transport within a very broad range of $[K^+]_o$ including μM concentrations (Hirsch *et al.*, 1998; Spalding *et al.*, 1999). KAT1 is a guard cell-specific channel and is likely to mediate K^+ fluxes for turgor-dependent regulation of the stomatal aperture (Nakamura *et al.*, 1995). This KAT1 function is probably redundant (Szyroki *et al.*, 2001).

The product of another member of the Shaker gene family, *ATKC1*, does not form functional ion channels when expressed in a heterologous system, but disruption of this protein affects the biophysical characteristics of the AKT1-mediated inward current in the root hairs (Dreyer *et al.*, 1997; Reintanz *et al.*, 2002). Based on this observation, it has been proposed that ATKC1 and AKT1 are parts of a heteromeric functional channel protein (Reintanz *et al.*, 2002). This hypothesis is supported by two-hybrid testing, confirming a physical interaction between ATKC1 and AKT1 (Pilot *et al.*, 2003).

The GORK member of the Shaker family in arabidopsis is activated by depolarization and is likely to be responsible for a K^+ efflux during stomatal closure (Ache *et al.*, 2000). The inhibition of GORK by an acidic pH is similar to the earlier reported effect of pH on the guard cell outward rectifier (Blatt and Armstrong, 1993; Grabov and Blatt, 1997). In root hairs, GORK facilitates membrane depolarization and K^+ release in response to external stimuli (Ivashikina *et al.*, 2001). Similarly to the outward rectifier from broad bean (*Vicia faba* L.) guard cells (Blatt and Gradmann, 1997), gating of the GORK channel is K^+-sensitive. Because of this property of the channel, it may

function as a potassium sensor (Ache *et al*., 2000; Ivashikina *et al*., 2001).

Another Shaker-type channel in arabidopsis, SKOR, is expressed in the root pericycle and stelar parenchyma cells, and is likely to be involved in xylem loading because the homozygous KO *skor-1* displays a lower rate of K^+ translocation from roots to shoots. In accordance with this function, SKOR is activated by membrane depolarization and provides a pathway for K^+ efflux (Gaymard *et al*., 1998). It has been demonstrated that SKOR and GORK physically interact and form a functional, heteromeric, outwardly rectifying channel (Dreyer *et al*., 2004).

AKT2 and *AKT3* are differentially initiated transcripts from a single gene (At4g22200). As based on the promoter reporter experiments (*AKT2*) and *in situ* hybridization (*AKT3*) this gene is predominantly expressed in phloem and xylem parenchyma (Marten *et al*., 1999; Lacombe *et al*., 2000). Low-level expression of *AKT2* was also detected in the leaf lamina (Lacombe *et al*., 2000). The products of *AKT2/AKT3* expression in *Xenopus* (*Xenopus laevis*) oocytes and COS cells are characterized as weakly rectifying potassium channels. These properties of AKT2/AKT3 enable bi-directional K^+ transport, which may be involved in phloem loading and/or unloading (Marten *et al*., 1999; Lacombe *et al*., 2000; Deeken *et al*., 2002). AKT2 has been shown to interact physically with AKT1 and AtKC1 (Pilot *et al*., 2003).

Two pore channels: Two-pore channels were cloned first from arabidopsis (*AtKCO1* for K^+ channel, Ca^{2+}-activated, outward rectifying 1; Czempinski *et al*., 1997) and more recently from the rain tree (*Samanea saman* (Jacq.) Merr.; *SPOCK1*;, Moshelion *et al*., 2002) and potato (*StKCO1*α; *StKCO1*β; Czempinski *et al*., 2002). It has recently been demonstrated that some members of the family, for example, AtKCO4 do not function as outward rectifiers and, therefore the family was renamed *TPK* (Tandem-Pore K^+, Becker *et al*., 2004). Among the members of the *KCO/TPK* family in arabidopsis, *AtKCO1* and *AtKCO6* demonstrated the strongest expression in roots and leaves (Schonknecht *et al*., 2002), while *AtKCO1-GUS* (β-glucuronidase) expression was detected in mitotically active tissues (Czempinski *et al*., 2002). The recently characterized member of the family, *AtTPK4* (*AtKCO4*), is expressed predominantly in pollen (Becker *et al*., 2004). At the subcellular level, AtKCO1 is localized to the vacuolar membrane (Czempinski *et al*., 2002). By contrast, AtTPK4 functions in the plasma membrane and it is likely that it plays a role in potassium homeostasis and membrane potential regulation in the growing pollen tube (Becker *et al*., 2004).

Cyclic nucleotide-gated channels: The structure of cyclic nucleotide-gated channels (CNGC) is similar to that of Shaker-type channels. In contrast to animal CNGC, domains binding cyclic nucleotide (CN) and calmodulin (CaM) overlap in plants (Köhler *et al*., 1999; Arazi *et al*., 2000; Köhler and Neuhaus, 2000), enabling cross-talk between CaM and CN signalling (Arazi *et al*., 2000). Arabidosis AtCNGC1 and AtCNGC4 (HLM1) display equal permeability for Na^+ and K^+ (Hua *et al*., 2003; Balague *et al*., 2003; Bridges *et al*., 2005). Remarkably, another member of the family, AtCNGC2, characterized by a unique Ala-Asn-Asp selectivity filter was highly selective for K^+ over Na^+ (Leng *et al*., 2002; Hua *et al*., 2003). Because of its high K^+ permeability and the appreciable expression in roots (Talke *et al*., 2003), AtCNGC2 may be directly involved in K^+ uptake.

Transporters

HKT family: Wheat HKT1, which facilitates K^+/Na^+ symport, was the first K^+ transporter to be cloned from plants (Schachtman and Schroeder, 1994; Rubio *et al*., 1995). Because wheat plants expressing antisense *HKT1* DNA were more salt-tolerant than the wild type, it has been concluded that the HKT1 transporter is primarily involved in Na^+ transport (Laurie *et al*., 2002). *HKT1* orthologues have been isolated from a variety of species (see Horie and Schroeder, 2004, for a recent review). In contrast to the rice genome, which has 9 *HKT1* orthologues (Garciadeblas *et al*., 2003), only one gene related to this transporter has been found in arabidopsis (Maser *et al*., 2001). Interestingly, the *hkt1* mutation in arabidopsis suppresses NaCl hypersensitivity in *sos1-1*, *sos2-1*, and *sos3-1* mutants (Rus *et al*., 2001, 2004). Overexpression of AtHKT1 in *sos3-1* plants augmented both Na^+ sensitivity and K^+ deficiency phenotypes when plants were grown at low $[K^+]_o$ (Rus *et al*., 2004). Although it was suggested that AtHKT1 is involved in Na^+ uptake (Rus *et al*., 2001), the expression pattern, as well as analysis of salt tolerance and Na^+ accumulation, in *athkt1* mutants indicate that this transporter facilitates Na^+ recirculation from shoots to roots via the phloem (Maser *et al*., 2002; Berthomieu *et al*., 2003).

KT/KUP/HAK family: The genes of this family are homologous to bacterial KUP (TrkD) potassium transporters. The KUP transporter from *E. coli* is characterized by a mid-range (0.37 mM) K_M for K^+ and a similar affinity for Rb^+ and Cs^+ (Bossemeyer *et al*., 1989). There are indications that KUP-mediated transport of potassium in bacteria is coupled to transport of H^+ (Zakharyan and Trchounian, 2001). Plant KUP transporters were first identified and cloned from arabidopsis (Quintero and Blatt, 1997; Fu and Luan, 1998; Kim *et al*., 1998) and barley (*Hordeum vulgare* L.; Santa-Maria *et al*., 1997). As different groups designated different acronyms such as KT, KUP and HAK to these transporters, they are commonly known as KT/KUP/HAK family.

Barley *HvHAK1* is characterized by a K_M=27 μM (Santa-Maria *et al*., 1997) and probably represents a component of the high affinity K^+ transport system observed

by Epstein *et al*. (1961). This transporter is assigned to the so-called cluster I, along with, *AtHAK5* and *OsHAK1*, the closest related HvHAK1 orthologues in arabidopsis and rice, respectively (Banuelos *et al.*, 2002; Rubio *et al.*, 2000). Notably, *AtHAK5* has been demonstrated to mediate high affinity K^+ transport in arabidopsis (Gierth *et al.*, 2005). Other arabidopsis genes in cluster I include *AtHAK7*, *AtKT5*, and *AtKUP12*.

The barley transporter HvHAK2 belongs to cluster II and facilitates low affinity transport with a K_M~5 mM (Rubio *et al*., 2000; Senn *et al.*, 2001). Arabidopsis HvHAK2 orthologues include *AtKT1/KUP1*, *AtKT2/KUP2*, *AtKT3/KUP4*, *AtKT4/KUP3*, *AtHAK6*, and *AtHAK8*. The affinity of arabidopsis transporters to K^+ is less clearly defined. Depending on the expression system, AtKUP1 was referred to as a dual- (Fu and Luan, 1998) or high (Kim *et al*., 1998) affinity transporter. AtKUP2 was more effective than AtKUP1 in rescuing the growth of *E. coli* (TK2463) and yeast (WΔ3) strains defective in K uptake transporters (Quintero and Blatt, 1997; Kim *et al*., 1998).

Transformation with *AtKT3/AtKUP4* cDNA enabled growth of the M398 strain of yeast (*Saccharomyces cerevisiae*) carrying the *trk1*Δ mutation on media containing as little as 0.2 mM K^+, but failed to rescue CY162 (*trk1*Δ/*trk2*Δ) lethality at low [K^+] (Rigas *et al.*, 2001). Disruption of this gene in the *trh1* (tiny root hair 1) mutant arrested root hair elongation, caused a reduction in the rate of Rb^+ uptake (Rigas *et al*., 2001) and affected root gravitropic behaviour (Vicente-Agullo *et al.*, 2004; Grabov *et al.*, 2005). It was unlikely that the *trh1* developmental phenotypes were due to cell potassium shortage, because defects in root gravitropic behaviour and root hair development were observed in external $[K^+]_o$ as high as 20 mM, while defects in transport were observed across μM ranges of $[K^+]_o$. Lower external potassium did not affect root hair growth, but surprisingly, attenuated the root bending phenotype in *trh1* and rendered WT roots weakly agravitropic (Vicente-Agullo *et al*., 2004). Although the root hair phenotype and agravitropic root bending are both due to the defects in epidermal cell development, no promoter–reporter p*TRH1-GUS* expression was detected in the root epidermis. Instead, the highest level of the promoter–reporter construct expression was observed in the root cap. The latter findings indicated that there were non-cell autonomous effects of the *trh1* mutation on epidermal cell development (Vicente-Agullo *et al*., 2004). Expression patterns of the construct containing a *GUS* reporter gene cloned under the synthetic auxin responsive element *DR5* (*DR5-GUS*; Ulmasov *et al.*, 1997) and experiments with radiolabelled auxin transport in the *trh1* root segments as well as in *TRH1* expressing yeast suggested that this transporter is also required for auxin transport (Vicente-Agullo *et al*., 2004). Therefore, the defects in epidermal development in the *trh1* mutant are most likely due to the alteration of auxin fluxes and not because of a reduction in K^+ supply.

A point mutation in the *AtKT2/KUP2* gene also resulted in developmental defects. The cognate mutant was designated *shy3-1* (short hypocotyl 3-1) and is characterized by reduced cell size (Elumalai *et al.*, 2002). Although *AtKT2/KUP2* does transport potassium (Quintero and Blatt, 1997; Kim *et al*., 1998), the *shy* phenotype was probably not due to the potassium deficiency because the single base *shy3-1* mutation had no effect on the K^+ transport properties of the protein, as assessed in *E. coli* T2463 cells. Moreover, the rate of $^{86}Rb^+$ uptake by *shy3-1* tissues was not significantly different from the wild type and the total K^+ accumulation in the *shy3-1* seedlings was only marginally lower (Elumalai *et al*., 2002).

KEA transporters: *KEA* is the least studied class of plant transporters. The gene family consists of six members in arabidopsis and was identified through homology to bacterial K^+/H^+ antiporters (Maser *et al*., 2001).

CHX transporters: In arabidopsis, 38 genes encode proteins homologous to mammalian and bacterial Na^+/H^+ exchangers (Maser *et al*., 2001). 28 of these genes form the Monovalent Cation:Proton Antiporter-2 (*CPA2*) family, which is also known as *CHX* (Cation/H^+ eXchanger). Surprisingly, a member of the family AtCHX17 is involved in K^+ acquisition and homeostasis rather than Na^+ transport (Cellier *et al.*, 2004). In accordance with this function, AtCHX17 is expressed in the cortex and epidermis of the mature root.

Plant responses to low potassium status

Physiological plasticity and regulation of K^+ transport proteins in response to K^+ deficiencies

Regulation of transporters: Potassium starvation is known to activate K^+ uptake in plants (Siddiqi and Glass, 1987; Fernando *et al.*, 1990; Shin and Schachtman, 2004). This activation has been conventionally associated with induction of expression of high affinity transporters, and was considered a major mechanism of adaptation to K^+ starvation.

Indeed, in several independent studies it has been demonstrated that transcription of the *AtHAK5* transporter is activated in arabidopsis in response to K^+ deprivation (Ahn *et al.*, 2004; Armengaud *et al.*, 2004; Hampton *et al.*, 2004; Shin and Schachtman, 2004; Gierth *et al*., 2005). In some experiments, *AtHAK5* was rapidly and transiently activated by potassium starvation at 6 h after onset of the K^+ deprivation (Shin and Schachtman, 2004), while in others the transcript abundance was significantly increased only after 48 h growth in K^+-free media, and then remained high over at least the next 5 d (Gierth *et al*., 2005). Elevated transcription of *AtHAK5* was down-regulated by K^+ re-supply (Armengaud *et al*., 2004; Gierth *et al*., 2005). Interestingly, in contrast to the above-cited works, Rubio and coauthors observed a decrease of

AtHAK5 transcripts in K^+-depleted roots (Rubio *et al*., 2000). One important difference in experimental conditions in which these contrasting outcomes were obtained is the composition of nutrient media. The decrease in *AtHAK5* transcript abundance was observed in NH_4^+-rich MS media, when K^+ was substituted with NH_4^+ (Rubio *et al*., 2000), while activation of the *AtHAK5* transcription in roots was found in NH_4^+-free media (Ahn *et al*., 2004; Armengaud *et al*., 2004; Gierth *et al*., 2005). The discrepancy between the results obtained in different media raises an interesting question about the selectivity of a K^+ sensing system and possible interference of NH_4^+ in signalling of K^+ status.

Analysis of Rb^+ uptake in K^+-starved wild type and *Athak5* knockouts indicated that the cognate AtHAK5 transporter operates within a high affinity range of concentrations with K_M =14 μM (Gierth *et al*., 2005). This agrees closely with the K^+ affinity of the transporter expressed in yeast (K_M=13 μM; Rubio *et al*., 2000). In accordance with its function in K^+ uptake, *AtHAK5* expression in plants is localized to the epidermis of main and lateral roots, and the stele of main roots (Gierth *et al*., 2005). Activation of this high affinity transporter in response to K^+ deprivation is probably a common feature shared between plant families. To date it has been demonstrated that *AtHAK5* orthologues are induced by low external potassium in barley (*HvHAK1*; Santa-Maria *et al*., 1997), tomato (*LeHAK5*; Wang *et al*., 2002), and rice (*OsHAK1*; Banuelos *et al*., 2002). However, the significance of *AtHAK5* activation for plant mineral nutrition at low $[K^+]$ has yet to be demonstrated. It is likely that some other KT/KUP/HAK transporters may complement AtHAK5 function, at least under some experimental conditions. Activation of *AtKUP3* expression, for instance, has been shown in roots of plants grown for 2–3 weeks on solidified K^+-depleted media (Kim *et al*., 1998). Interestingly, these growth conditions lead to a decrease in *AtKUP2* transcript abundance (Kim *et al*., 1998). *AtKUP12*, another member of the KT/KUP/HAK family, may also play role in acclimation to mineral deficiencies as it was down-regulated in shoots after K^+ re-supply (Armengaud *et al*., 2004).

Among the members of the KEA potassium transporter family, *KEA5* has been shown to be transiently induced after 6 h of K^+ deprivation (Shin and Schachtman, 2004). As localization of this transporter and its role in K^+ transport is not known, it is difficult to assess the contribution of KEA5 in plant acclimation to potassium deficiencies.

Transcription of AtCHX17, the member of the *CHX* family involved in K^+ acquisition and homeostasis was induced by K^+ starvation in arabidopsis (Cellier *et al*., 2004).

Regulation of channels: Electrophysiological experiments indicated activation of inwardly rectifying 5 pS K^+ channels in the arabidopsis root plasma membrane in response to low (100 μM) potassium (Maathuis and Sanders, 1995). These changes in channel activity were parallelled by an increased rate of Rb^+ uptake in K^+-starved plants. Hirsch *et al.* (1998) also demonstrated that the inwardly rectifying AKT1 channel provides a major pathway for potassium acquisition, even if the nutrient is available in the μM range of concentrations. At low $[K^+]_o$ however, disruption of the *AKT1* gene affected plant growth only in media containing NH_4^+ (Hirsch *et al*., 1998; Spalding *et al*., 1999). The contribution of AKT1 to K^+ transport from media with 25 and 50 μM concentrations of this cation has been shown in Rb^+-uptake experiments with K^+-starved *akt1-1* and wild-type plants, but due to the complex uptake kinetics, these results could not be unequivocally extended to the whole μM range of K^+ concentrations (Gierth *et al*., 2005).

In arabidopsis, activation of AKT1 by low $[K^+]_o$ probably occurs post-transcriptionally because neither RNA blot nor microarray experiments revealed an alteration in *AKT1* transcription in K^+-starved plants (Pilot *et al*., 2003; Maathuis *et al*., 2003; Hampton *et al*., 2004). Similarly, no transcriptional activation of *AKT1* orthologues was detected in rape (Lagarde *et al*., 1996). It has been demonstrated that AKT1 can form heteromeric complexes with AtKC1 (Reintanz *et al*., 2002; Pilot *et al*., 2003), and the recent discovery that *AtKC1* is transiently activated by K^+ starvation in arabidopsis (Shin and Schachtman, 2004) indicates an intriguing possibility that AKT1 may be activated through an interaction with AtKC1.

K^+ starvation has also been shown to enhance an inwardly rectifying current across the root cell plasma membrane in wheat. In contrast to arabidopsis, the induced current was associated with increased transcription of *TaAKT1* (Buschmann *et al*., 2000).

Potassium starvation down-regulated transcription of *SKOR* and *AKT2* in arabidopsis (Maathuis *et al*., 2003; Pilot *et al*., 2003). As these channels are involved in long-distance transport, it has been suggested that their reduced expression at low $[K^+]_o$ is required to restrict recirculation of K^+ between the tissues and organs. Alterations in the long-distance transport of K^+ may also be important for the communication of potassium status between shoots and roots (Pilot *et al*., 2003).

The major K^+-transport proteins involved in K^+ acquisition, homeostasis, and responses to K^+ deficiencies in arabidopsis are listed in Table 1.

Signalling cascades regulating responses to potassium deficiencies

Signalling of K^+ deficiencies in bacteria: Although plant responses to potassium deficiencies are well documented at the physiological and transcriptional levels, the regulatory mechanisms underlying these changes are still obscure. The K^+-dependent signalling cascades have been studied

Table 1. *The major K^+ transport proteins putatively involved in responses to potassium deficiencies in arabidopsis roots*

Gene/protein	Localization in root	Putative function	Interacting K^+ transport proteins	Transcriptional responses to potassium deficiencies or potassium re-supply	References
AKT1	Root cap, epidermis, cortex, endodermis, stele[a]	K^+ uptake	ATKC1, AKT2/AKT3	No	Sentenac *et al.*, 1992; Lagarde *et al.*, 1996; Hirsch *et al.*, 1998; Spalding *et al.*, 1999
ATKC1	Meristem, epidermis, cortex, endodermis	K^+ uptake	AKT1	Up	Dreyer *et al.*, 1997; Reintanz *et al.*, 2002; Pilot *et al.*, 2003; Shin and Schachtman, 2004
GORK	Epidermis	K^+ efflux, membrane repolarization, signalling, K^+ sensing	SKOR		Ache *et al.*, 2000; Ivashikina *et al.*, 2001
SKOR	Pericycle and stellar parenchyma	Xylem loading	GORK	Down	Gaymard *et al.*, 1998; Pilot *et al.*, 2003; Maathuis *et al.*, 2003; Dreyer *et al.*, 2004
AKT2/AKT3	Phloem	Phloem loading and unloading	AKT1	Down	Marten *et al.*, 1999; Lacombe *et al.*, 2000; Deeken *et al.*, 2002; Pilot *et al.*, 2003
AtHAK5	Epidermis of main and lateral roots, stele of main roots	High affinity K^+ uptake		Up[b]	Wang *et al.*, 2002; Ahn *et al.*, 2004; Shin and Schachtman, 2004; Armengaud *et al.*, 2004; Hampton *et al.*, 2004; Gierth *et al.*, 2005
TRH1 (AtKT3/AtKUP4)	Root cap	K^+ transport, root hair development, gravitropic responses		No	Rigas *et al.*, 2001; Desbrosses *et al.*, 2003; Vicente-Agullo *et al.*, 2004
AtKUP1[c]		K^+ transport			Quintero and Blatt, 1997; Kim *et al.*, 1998; Fu and Luan, 1998
AtKUP2	Root tip, root–hypocotyl junction	K^+ transport, regulation of cell elongation		Down	Kim *et al.*, 1998; Elumalai *et al.*, 2002
AtKUP3				Up	Kim *et al.*, 1998
AtKUP12				Up	Armengaud *et al.*, 2004
KEA5				Up	Shin and Schachtman, 2004
AtCHX17	Cortex and epidermis	K^+ uptake		Up	Cellier *et al.*, 2004

[a] Very low expression in the stele.
[b] Increased level of transcript was found in high $[K^+]_o$ when media contained NH_4^+ (Rubio *et al.*, 2000)
[c] Different patterns of expression and transport properties were observed in literature cited.

in detail in bacterial cells, where it has been demonstrated that K^+-limiting conditions trigger autophosphorylation of the KdpD sensor kinase and subsequent transfer of a phosphoryl group to the KdpE cytosolic response regulator. Binding of phosphorylated KdpE to the promoter of the *kdpFABC* operon, triggers expression of the KdpFABC high-affinity transport system (Heermann *et al.*, 2003). No similar signalling cascade has so far been identified in plants, although it is known that histidine kinases regulate a variety of responses in plant cells.

AAA-ATPase-related proteins: Screening of a mouse macrophage cDNA library for suppression of the *trk1*Δ phenotype in the potassium uptake-deficient yeast strain CY162, identified the *SKD1* (Suppressor of K^+ Transport Growth Defect) gene (Perier *et al.*, 1994). SKD1 belongs to the AAA-ATPase family and is involved in membrane transport through endosomes and lysosomes (Fujita *et al.*, 2004). Expression of the ice plant *SKD1* orthologue (*mcSKD1*) has been shown to be induced by salt stress, while in high $[K^+]_o$ medium transcription of this gene was reduced (Jou *et al.*, 2004). As with its mammalian counterpart, mcSKD1 was able to complement potassium transport deficiency in yeast Trk^- mutants. Elevated expression of the *SKD1* orthologue was also observed in tomato plants in response to K or Fe deprivation (Wang *et al.*, 2002). Another gene belonging to the AAA-ATPase class, At1g43910, was up-regulated 6-fold in arabidopsis plants subjected to K^+ starvation for 7 d (Hampton *et al.*, 2004). The role of the SKD1 protein in membrane trafficking in mammals and yeast implies that its function in plants might be associated with protein sorting/targeting in response to salt stress and K^+ deficiencies (Jou *et al.*, 2004). This hypothesis, albeit plausible, requires further experimental confirmation.

Jasmonic acid (JA) and related signalling cacades: Some components of signalling cascades regulating responses to K^+ deficiencies are similar to those involved in stress responses to wounding and insect and pathogen attacks, in which JA and derivatives have been demonstrated to play a prominent role (Howe, 2004; Pozo *et al.*, 2004; Rayko and Ian, 2004). Iterative group analyses of the arabidopsis transcriptome in K^+-deprived plants and K^+-starved plants resupplied with potassium, revealed that genes related to JA metabolism and signalling form the largest group affected by these conditions (Armengaud *et al.*, 2004). A subgroup of genes involved in polyamine metabolism was also included in the group of JA-related genes (Armengaud *et al.*, 2004). Transcripts for Arg decarboxylase, *AtADC2*, involved in putrescine biosynthesis, were the most strongly induced in this subgroup. In agreement with earlier data (Watson and Malmberg, 1996), no changes in the abundance of the *AtADC1* transcript were detected. These results suggest the increases in putrescine content demonstrated in K^+-starved plants (Watson and Malmberg, 1996) are probably due to the activity of the AtADC2 Arg decarboxylase isoform. Polyamines efficiently block a variety of ion channels (Bruggemann *et al.*, 1998; Dobrovinskaya *et al.*, 1999; Guo and Lu, 2000), including inwardly rectifying *AtKAT1* (Liu *et al.*, 2000), but it is not yet clear if these physiological effects of polyamines are important for plant acclimation to stress conditions. One can speculate, however, that reduced permeability of ion channels during potassium deprivation may be required for the reallocation of K^+ between the different storage pools.

Reactive oxygen species (ROS): Shin and Schachtman (2004) recently demonstrated that ROS are of primary significance for the regulation of plant responses to K^+ deprivation. An induction of the high affinity K^+ transport component by K^+ deprivation was accompanied by 2-fold increases in H_2O_2 production. The ROS signal is non-redundant and enables activation of the high affinity K^+ uptake component even in the K^+-sufficient plants. Expression of the *RHD2* gene was up-regulated in K^+-deficient plants, indicating involvement of this NADPH oxidase (Foreman *et al.*, 2003) in production of H_2O_2 in response to K^+ deprivation. This has been confirmed in experiments with an *rhd2* knockout line, where no induction of *HAK5* and *KEA5* potassium transporters was observed in K^+-deprived plants (Shin and Schachtman, 2004).

Ethylene: Both activation of JA-related genes and ROS production indicate some overlap between the cascades that signal conditions of K^+-deficiencies, and wounding. These parallels are enhanced by an observation that K^+ deprivation of arabidopsis plants induces strong expression of genes related to ethylene biosynthesis and signalling and, in addition, an orthologue to a tomato wound-inducible gene, At4g10270 (Shin and Schachtman, 2004). Ethylene, alongside JA is known to play an important role in wounding responses (Devoto and Turner, 2003). The role of ethylene signalling in K^+ deficiency stress was confirmed by direct measurements of the amount of ethylene released into the atmosphere by K^+-deficient and K^+-sufficient plants. In these experiments, K^+ starvation caused an almost 2-fold increase in production of this phytohormone (Shin and Schachtman, 2004).

Auxin: Auxin has been shown to control expression of the inwardly rectifying *ZMK1* channel in maize (Philippar *et al.*, 1999). The increased abundance of the *ZMK1* transcripts after addition of auxin was paralleled by increases in K^+ channel density, detected electrophysiologically in maize coleoptile protoplasts (Philippar *et al.*, 1999; Thiel and Weise, 1999). The *ZMK1* channel, which is highly homologous to arabidopsis *AKT1*, may play a role in maize mineral nutrition, but this hypothesis has yet to be confirmed experimentally.

Further evidence for a role of auxin-dependent processes in acclimation to K^+ deficiencies was provided by the

demonstration that the CYP79B2 and CYP79B3 genes involved in the Trp-dependent auxin biosynthesis were down-regulated upon K^+ resupply to K^+-starved roots (Armengaud *et al*., 2004).

Misexpression of the auxin-dependent DR5-GUS construct (Ulmasov *et al*., 1997) indicated auxin accumulation in the central cylinder of the roots of K^+-deficient plants (Vicente-Agullo *et al*., 2004). The perturbations in the auxin profile in these experiments are probably due to alterations in auxin transport, resulting from potassium deprivation. The auxin profile of the K^+-starved plants was reminiscent of those found in *trh1* K^+-sufficient plants in which the *TRH1*(*AtKUP4*/*AtKT3*) potassium transporter was disrupted.

Although the *trh1* mutation does affect K^+ transport under some experimental conditions (Rigas *et al*., 2001), it has been shown that TRH1 is also required for auxin efflux in root cap cells (Vicente-Agullo *et al*., 2004). Three putative mechanisms may be associated with TRH1 involvement in the transport of auxin. (i) TRH1 can generate ionic and electrical gradients that favour phytohormone efflux. (ii) Transport of potassium can be directly coupled to transport of phytohormones, similarly to K^+/Na^+-coupled transport of neurotransmitters (Kanner *et al.*, 2001). (iii) TRH1 may transport auxin independently of potassium. The plausibility of the hypothesis that a cation transporter may facilitate uncoupled transport of anion is supported by recent data by Kuroda *et al*. (2004). These authors found that major yeast Trk potassium transporters also function as anion channels (Kuroda *et al.*, 2004).

Morphological plasticity and K^+ availability

Apart from induction of physiological responses, the variations in mineral nutrient availability often evoke some alterations in root architecture. In barley and many other plants, low phosphorus, nitrogen or potassium supply resulted in a reduction of total lateral root length (Drew, 1975). In soils with non-uniform distribution of mineral nutrients, localized extension of lateral roots in barley was triggered in patches rich in phosphate and nitrogen (Drew, 1975). By contrast, K^+-rich patches in these experiments induced a global response and accelerated the growth of laterals even in those root segments that were exposed to low potassium concentrations. These experimental data indicate that the localized $[K^+]_o$ increases evoke systemic signals responsible for global lateral root proliferation. The observed pattern of morphological responses may also be associated with the high mobility of K^+ in the plant body (Hodge, 2004, and references therein).

Similarly to barley (Drew, 1975), potassium deficiencies arrest lateral root development in arabidopsis (Shin and Schachtman, 2004; Armengaud *et al*., 2004). Increased levels of ethylene may be responsible for the inhibition of lateral root development observed in the K^+-starved plants (Shin and Schachtman, 2004), but this developmental response was not necessarily accompanied by the activation of genes related to ethylene metabolism and signalling (Armengaud *et al*., 2004). The fact that auxin is important for the development of lateral roots indicates that this phytohormone may also be involved in signalling of nutrient availability. Experimental data on the role of this phytohormone in nitrogen- and phosphate-dependent root plasticity are, however, rather contradictory (Bates and Lynch, 1996; Zhang *et al.*, 1999; Linkohr *et al.*, 2002).

Growth and development of root hairs is controlled by the availability of phosphate, nitrate, and iron (see Forde and Lorenzo, 2001, for a review), but potassium in physiologically relevant concentrations has no effect on these developmental processes in the epidermis of arabidopsis (Desbrosses *et al.*, 2003).

One important mechanism of acclimation to phosphate deficiencies in common bean (*Phaseolus vulgaris* L.), soybean (*Glycine max* (L.) Merr.), and pea (*Pisum sativum* L.) is associated with reduction of the gravitropic set-point angle (GSA) in basal roots (Bonser *et al.*, 1996). As a result, at low phosphorus availability the root system is more shallow and enables more efficient exploration of upper soil layers, normally containing higher levels of this nutrient. Changes in root gravitropic behaviour were also observed in low potassium media in arabidopsis (Vicente-Agullo *et al*., 2004; Fig. 1). In these experiments, wild-type arabidopsis plants (Fig. 1A, C) and *trh1* mutants (Fig. 1B, D) grown on standard Murashige–Skoog medium for 3 d, were transferred for a further 3 d growth on media

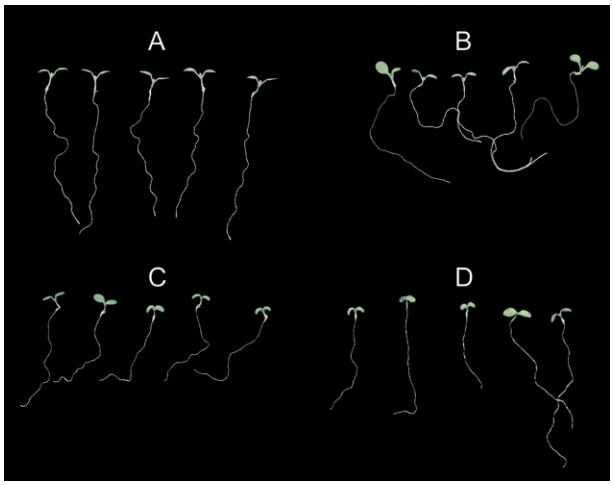

Fig. 1. Gravitropic behaviour of roots of wild-type and *trh1* plants grown on vertical agarose plates containing different concentrations of K^+. Wild-type *Arabidopsis* plants (A, C) and *trh1* mutants (B, D) were grown on the standard Murashige–Skoog medium for 3 d, after which they were transferred for a further 3 d growth on media containing either 20 (A, B) or 0.1 mM (C, D) of potassium as described in Vicente-Agullo *et al.* (2004). The deviation from the gravitropic growth in low $[K^+]$ medium may enable the root to escape from low nutrient patches in soil. The TRH1 transporter is pivotal for the K^+-dependent gravitropic behaviour because the *trh1* mutant displays a different pattern of responses.

containing either 20 (Fig. 1A, B) or 0.1 mM (Fig. 1C, D) of potassium. As the section on the KUP/HAK/KT family indicated, disruption of the *TRH1* (*AtKUP4*/*AtKT3*) potassium transporter affected root gravitropic behaviour in high potassium concentrations (Fig. 1B). Surprisingly, reduced potassium availability triggered agravitropic root growth in the WT plants. This reaction to the low K^+ status may be important for mineral acquisition in soils where K^+ distribution is heterogeneous. The deviation from gravitropic growth, probably enables the root to escape from low $[K^+]_o$ soil patches and potentiates exploration of areas with higher nutrient content (Vicente-Agullo *et al*., 2004). In stark contrast to WT, in *trh1* mutants, the reduction of external $[K^+]_o$ attenuated root agravitropic behaviour. This difference in the effects of K^+ deprivation on the gravitropic behaviour of WT and *trh1* roots suggest that the TRH1 potassium transporter plays a pivotal role in these responses and regulates them through K^+-dependent phytohormone distribution in the root tip (Vicente-Agullo *et al*., 2004).

In conclusion, growing roots continuously experience variations in potassium availability, to which they have to adjust their physiology and growth pattern. In order to optimize their performance as nutrient uptake organs, and to compete for K^+ uptake in the dynamic and heterogeneous environment, plant roots developed mechanisms of acclimation to the current K^+ status in the rhizosphere. Moreover, emerging evidence showing changes in expression of transcripts encoding K^+ transporters and channels in response to ROS and phytohormones, provide the intriguing possibility that K^+ may play a more specific regulatory role in plant stress responses.

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