Journal of Plant Physiology 166 (2009) 447—466

Available online at www.sciencedirect.com

ScienceDirect

JOURNAL OF PLANT PHYSIOLOGY

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REVIEW

K^+ transport in plants: Physiology and molecular biology

Mark W. Szczerba[b,*], Dev T. Britto[a], Herbert J. Kronzucker[a]

[a]Department of Biological Sciences, University of Toronto, 1265 Military Trail, Toronto, Ontario, Canada M1C 1A4
[b]Department of Plant Sciences, University of California, Davis, 1 Shields Ave., Davis, CA 95616, USA

Received 25 August 2008; received in revised form 10 November 2008; accepted 10 December 2008

KEYWORDS
Efflux;
HATS;
Influx;
LATS;
Potassium

Summary

Potassium (K^+) is an essential nutrient and the most abundant cation in plant cells. Plants have a wide variety of transport systems for K^+ acquisition, catalyzing K^+ uptake across a wide spectrum of external concentrations, and mediating K^+ movement within the plant as well as its efflux into the environment. K^+ transport responds to variations in external K^+ supply, to the presence of other ions in the root environment, and to a range of plant stresses, via Ca^{2+} signaling cascades and regulatory proteins. This review will summarize the molecular identities of known K^+ transporters, and examine how this information supports physiological investigations of K^+ transport and studies of plant stress responses in a changing environment.

Contents

*Corresponding author. Tel.: +1 530 754 7322.
E-mail address: mwszczerba@ucdavis.edu (M.W. Szczerba).

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doi:10.1016/j.jplph.2008.12.009

Introduction

Potassium (K^+) is an essential nutrient for plant growth and development. It is the most abundant cation in plant cells and can comprise as much as 10% of plant dry weight (Leigh and Wyn Jones, 1984; Véry and Sentenac, 2003). Plant roots take up K^+ from a wide range of external concentrations ($[K^+]_{ext}$), which typically vary from 0.1 to 10 mM (Reisenauer, 1966; Hawkesford and Miller, 2004). Occasionally, much higher $[K^+]$ are observed (Ramadan, 1998), while in some intensively cultivated areas such as rice fields of Southeast Asia, the depletion of soil K^+ threatens to reduce crop yields (Dobermann and Cassman, 2002; Yang et al., 2004). Other environmental stresses, such as metal toxicity, salinity, and drought, are known to adversely affect K^+ uptake and transport by plants (Schroeder et al., 1994; Amtmann et al., 2006; Shabala and Cuin, 2008), and such stresses can often be ameliorated by increased K^+ supply (Cakmak, 2005). The link between K^+ and crop production has been highlighted in two recent reviews: one on the role of K^+ in reducing the effects of pests and disease on plants (Amtmann et al., 2008) and the other on the importance of K^+ in the onset of sodium (Na^+) toxicity (Shabala and Cuin, 2008).

The extraction of K^+ from soil and its distribution within the plant require the presence of membrane-bound transport proteins. A large number of such transporters have now been identified at the molecular level, demonstrating the complex nature of K^+ transport. The physiological roles of these proteins in primary K^+ influx, efflux, compartmentation, and transport within the plant have been partially characterized (Gierth and Mäser, 2007; Lebaudy et al., 2007), while many putative K^+ transporters and transport regulators are currently under investigation. The present review will begin with a synopsis of the functions of K^+, then discuss the known classes of K^+ transporters and their regulation, with attention to special topics such as K^+-use efficiency and root zonation. Throughout, we shall assess some of the latest investigations into K^+ transport at cellular and whole-plant levels. It is our hope to generate new discussion for K^+ transport research by bringing together important advances in plant molecular biology and physiology.

Functions of K^+

Potassium plays major biochemical and biophysical roles in plants. General maintenance of the photosynthetic apparatus demands K^+, and K^+ deficiency reduces photosynthetic activity, chlorophyll content, and translocation of fixed carbon (Hartt, 1969; Pier and Berkowitz, 1987; Zhao et al., 2001). Plant movements such as closing and opening of stomata, leaf movements, and other plant tropisms are driven by K^+-generated turgor pressure (Maathuis and Sanders, 1996a; Philippar et al., 1999). The osmotic pressure brought about by K^+ accumulation within cells is also used to drive cellular and leaf expansion (Maathuis and Sanders, 1996a; Elumalai et al., 2002). K^+ is highly mobile within plants, exhibiting long-distance cycling between roots and shoots in the xylem and phloem. This is most evident in the cotransport of K^+ with nitrate (NO_3^-) to shoots and its subsequent retranslocation to roots with malate when plants are supplied with NO_3^-, and is also seen in the cotransport of K^+ with amino acids in the xylem (Ben Zioni et al., 1971; Jeschke et al., 1985). Recirculated K^+ can be an important source of K^+ in roots, particularly with NO_3^--grown plants, and phloem-delivered K^+ from shoots may be a signal that modulates K^+ influx into the root (Peuke and Jeschke, 1993; White, 1997). The relatively high permeability of plant cells to K^+ confers on the ion the ability to impose short- and long-term modifications upon the electrical potential difference across the plasma membrane ($\Delta\Psi_{PM}$) that is primarily established and maintained by the H^+-ATPase. This can be readily seen when changes in the $[K^+]_{ext}$ result in permanent hyperpolarization (upon reduction of K^+) or depolarization (upon increase in K^+) of $\Delta\Psi_{PM}$ (Pitman et al., 1970; Cheeseman and Hanson, 1979; Kochian et al., 1989; Maathuis and Sanders, 1996a; Rodríguez-Navarro, 2000). Notably, in some species such as rice (*Oryza sativa*), or the halophyte *Triglochin maritima*, ammonium (NH_4^+), and sodium (respectively) can also adjust $\Delta\Psi_{PM}$ (Jefferies, 1973; Wang et al., 1994). Nevertheless, in most plants $\Delta\Psi_{PM}$ is only transiently modified by either ion (Higinbotham et al., 1964; L'Roy and Hendrix, 1980; Cheeseman, 1982; Cheeseman et al., 1985).

K^+ accumulates to considerable concentrations in cytosolic and vacuolar compartments. The cytosolic K^+ pool appears to be relatively stable, although estimates of cytosolic K^+ concentration ($[K^+]_{cyt}$) can range widely, between 30 and 320 mM, tending to a set point of around 100 mM (Walker et al., 1996). This range, and the exact stringency of $[K^+]_{cyt}$ homeostasis, reflects, in part, some disagreement arising from the use of different methods (see Britto and Kronzucker, 2008). By contrast with the cytosolic pool, the concentration of the vacuolar K^+ pool has been found to vary greatly, between 10 and 500 mM, depending on the plant examined and the K^+ growth condition (Leigh and Wyn Jones, 1984; Marschner, 1995). A stable $[K^+]_{cyt}$ is considered necessary for optimal enzyme activity, and its disruption may underlie ion toxicities such as brought about by high sodium or ammonium provision (Mills et al., 1985; Hajibagheri et al., 1987, 1988; Speer and Kaiser, 1991; Walker et al., 1996; Flowers and Hajibagheri, 2001; Carden et al., 2003; Halperin and Lynch, 2003; Kronzucker et al., 2003, 2006; Szczerba et al., 2006, 2008a).

High-affinity K^+ transport

As with most mineral nutrients, the primary acquisition of K^+ from the external environment follows a biphasic pattern, described as the sum of two uptake mechanisms at the plasma membrane, and distinguishable in terms of saturability, flux capacity, differential sensitivity by physicochemical treatments, and mechanism (Epstein and Bloom, 2005; Gierth and Mäser, 2007; Lebaudy et al., 2007; Britto and Kronzucker, 2008). The high-affinity transport system (HATS) is a saturable system catalyzing the thermodynamically active uptake of K^+ at low concentrations (<1 mM). The active influx of K^+ is thought to be coupled to the passive influx of H^+ down its electrochemical gradient, which is maintained by proton-pumping ATPase complexes in the plasma membrane (Cheeseman et al., 1980; Rodríguez-Navarro et al., 1986; Kochian et al., 1989; Maathuis and Sanders, 1994; Briskin and Gawienowski, 1996). Despite some disagreement concerning the precise stoichiometry of H^+/K^+ symport, it is generally accepted to be 1:1 (Maathuis and Sanders, 1994; Maathuis et al. 1997), as has also been demonstrated in bacterial (Bakker and Harold, 1980) and fungal systems (Rodríguez-Navarro et al., 1986). In plants, the K_M value for HATS ranges from 13 to 130 μM, with a V_{max} of between 1.8 and nearly 150 $\mu mol\,g^{-1}\,h^{-1}$, depending on the experimental system investigated (Epstein et al., 1963; Kochian and Lucas, 1982, 1983; Wrona and Epstein, 1985; Maathuis and Sanders, 1996a, b).

The magnitude of HATS-mediated K^+ influx has been shown to be inversely correlated with tissue K^+ content (Glass, 1976; Kochian and Lucas, 1982; Siddiqi and Glass, 1986), although it is unclear how plants interpret K^+ status and adjust transport rates appropriately. HATS-mediated K^+ influx is also severely reduced by NH_4^+ provision (Scherer et al., 1984; Vale et al., 1987, 1988a, b; Wang et al., 1996; Spalding et al., 1999; Santa-María et al., 2000; Bañuelos et al., 2002; Kronzucker et al., 2003; Martínez-Cordero et al., 2004; Szczerba et al., 2006, 2008b; Nieves-Cordones et al., 2007), such that reduced K^+ uptake and accumulation in the presence of NH_4^+ is a characteristic symptom of NH_4^+ toxicity (Kirkby, 1968; Van Beusichem et al., 1988; Peuke and Jeschke, 1993; Gerendás et al., 1997; Hirsch et al., 1998; Britto and Kronzucker, 2002; Martínez-Cordero et al., 2005). The mechanism by which NH_4^+ inhibits K^+ influx in the HATS range, while not firmly established, may result from direct competition between NH_4^+ and K^+ for entry into the cell (Vale et al., 1987; Wang et al., 1996; White, 1996). Similarly, Na^+ has been shown to suppress HATS-mediated K^+ influx, particularly at millimolar $[Na^+]_{ext}$ (Cheeseman, 1982; Jeschke, 1982; Schachtman and Schroeder, 1994; Rubio et al., 1995; Gassmann et al., 1996; Maathuis et al., 1996; Santa-María et al., 1997; Martínez-Cordero et al., 2005; Kronzucker et al., 2006, 2008; Nieves-Cordones et al., 2007), although a few studies suggest that Na^+ has only a weak effect (Epstein, 1961; Epstein et al., 1963), or indeed may stimulate HATS-mediated K^+ influx (Rubio et al., 1995; Spalding et al., 1999). Conflicting information on the effects of Na^+ may arise from differences in experimental systems and approaches (e.g., between heterologous expression systems, excised roots and intact plants, or between measurements of unidirectional and net fluxes).

Several genes have been identified that appear to encode HATS transporters. They are grouped into four major families: HAK/KUP/KT (K^+/H^+ symporters), HKT/TRK (K^+/H^+ or K^+/Na^+ symporters), CPA (cation/H^+ antiporters), and Shaker channels (to be discussed below), with additional candidates found in other (mostly channel-type) transport families. With the exception of ion channels, these transporters mediate active K^+ symport with H^+ (Maathuis and Sanders, 1999; Rodríguez-Navarro, 2000; Mäser et al., 2001; Gierth and Mäser, 2007; Grabov, 2007), with the majority of HATS-mediated influx catalyzed by members of the HAK/KUP/KT family (Figure 1), particularly under conditions of K^+ starvation (Gierth and Mäser, 2007). Initially identified in *Escherichia coli*, this type of transporter was found to be significantly different from previously identified bacterial TRK K^+ transporters (Schleyer and Bakker, 1993). Homologous amino acid sequences were subsequently identified in the yeast *Schwanniomyces occidentalis* (Bañuelos et al., 1995) and in barley (*Hordeum vulgare*, Santa-María et al., 1997). Supporting the hypothesis that HAK/KUP/KT functions in the acquisition of K^+ at low $[K^+]_{ext}$, K^+ starvation has been found to promote *HAK* transcript abundance in a wide

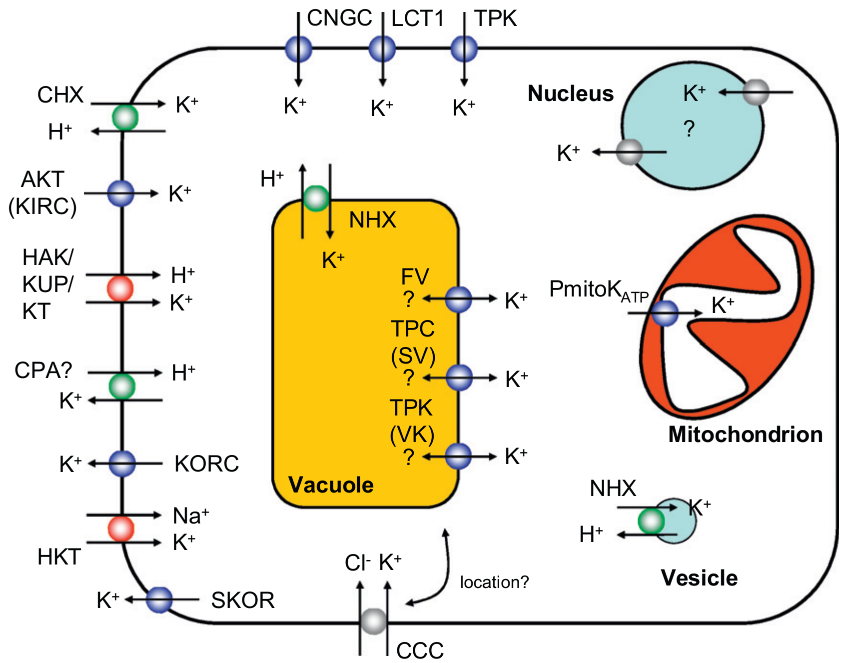

Figure 1. A summary of known and putative potassium transporters in the plant root cell. *Abbreviations*: CNGC = cyclic nucleotide-gated channels; LCT1 = low-affinity cation transporter; TPK = tandem-pore K^+ channel; CCC = cation/chloride cotransporter; SKOR = stelar K^+ outward rectifier; HKT = high-affinity K^+ transporter; KORC = K^+ outward-rectifying channel; CPA = cation/H^+ antiporter; HAK/KUP/KT = high-affinity K^+ symporter family; KIRC = K^+ inward-rectifying channel; CHX = cation/H^+ exchanger; PmitoK$_{ATP}$ = ATP-sensitive plant mitochondrial K^+ channel; NHX = Na^+/H^+ exchanger; FV = fast-activating vacuolar channel; TPC = two-pore channel; SV = slow-activating vacuolar channel; VK = vacuolar K^+ channel.

variety of plant systems, including barley, rice, *Arabidopsis thaliana*, *Capsicum annum*, *Mesembryanthemum crystallinum*, *Solanum lycopersicum*, and *Phragmites australis* (Santa-María et al., 1997; Bañuelos et al., 2002; Su et al., 2002; Ahn et al., 2004; Armengaud et al., 2004; Martínez-Cordero et al., 2004; Shin and Schachtman, 2004; Gierth et al., 2005; Nieves-Cordones et al., 2007; Takahashi et al., 2007). Conversely, *HAK* transcription decreases or is eliminated under K^+-replete conditions. These findings help explain tracer studies showing that HATS-mediated K^+ influx is reduced with high K^+ provision, and increased with K^+ starvation (Glass, 1976; Kochian and Lucas, 1982; Siddiqi and Glass, 1986). Further corroboration indicating that HAK/KUP/KT transporters mediate HATS fluxes is found in studies showing that transporter abundance and/or transport activity are/is inhibited by Na^+ (Santa-María et al., 1997; Quintero and Blatt, 1997; Fu and Luan, 1998; Su et al., 2002; Martínez-Cordero et al., 2005; Nieves-Cordones et al., 2007) and NH_4^+ (Santa-María et al., 2000; Bañuelos et al., 2002; Martínez-Cordero et al., 2004, 2005; Vallejo et al., 2005; Nieves-Cordones et al., 2007). Some evidence also suggests that influx of Na^+ occurs via high-affinity K^+ transporters (Santa-María et al., 1997; Takahashi et al., 2007), in support of earlier physiological studies (see above). However, it should be noted that AtKUP transporters have not yet been localized to the plasma membrane. A closely related transporter, OsHAK10, has been localized to the tonoplast (Bañuelos et al., 2002), supporting the idea that HAK/KUP/KT transporters mobilize K^+ from the vacuole under K^+ deficiency (Rodríguez-Navarro and Rubio, 2006), a role also suggested by a recent proteomics study that found five members of the KUP family in tonoplast-enriched *Arabidopsis* membrane fractions (Whiteman et al., 2008).

Localization studies have shown that HAK/KUP/KT transporters are expressed throughout the plant, including in floral, foliar, and stem tissue (Kim et al., 1998; Rubio et al., 2000; Bañuelos et al., 2002; Su et al., 2002). This indicates that this family does not simply mediate primary K^+ uptake from soil. For instance, a mutation in the *AtKT2/AtKUP2* gene was shown to alter turgor-driven cell expansion in the shoot (Elumalai et al., 2002).

An interesting outcome of the molecular analyses of transport proteins is that the distinction between HATS and LATS is not as rigid as previously thought. For example, AtKUP1, from *A. thaliana*, appears to mediate K^+ uptake at both low and high $[K^+]_{ext}$ in yeast and *Arabidopsis*-suspension cells (Fu and Luan, 1998; Kim et al., 1998), although some of the evidence is problematic (very low fluxes in wild-type and transformant, lack of conformity to kinetic models, and the background presence of endogenous transporters; Rodríguez-Navarro, 2000). Nevertheless, AtKUP1 displays properties of both HAK/KUP/KT family members, and plant K^+ Shaker channels (described below), including the presence of 12 transmembrane-spanning domains, characteristic of HAK/KUP/KT transporters, and an amino acid sequence of IYGD (isoleucine-tyrosine-glycine-aspartate), similar to the GYGD/E (glycine-tyrosine-glycine-aspartate/glutamate) motif found in the pore domain of K^+ channels (Chérel, 2004). In addition, AtKUP1 shows sensitivity both to Na^+ and to the channel inhibitors tetraethylammonium (TEA^+), cesium (Cs^+), and barium (Ba^{2+}, Fu and Luan, 1998). Another *A. thaliana* transporter, AtKT2/AtKUP2, rescued yeast mutants defective in K^+ uptake when supplied with $\geqslant$2.5 mM K^+, while yeast growth was substantially reduced when $[K^+]_{ext}$ was reduced to 1 mM (Quintero and Blatt, 1997), suggesting that not all members of the HAK/KUP/KT family operate in the high-affinity range.

Unlike HAK/KUP/KT, the role of the HKT/TRK family (Figure 1) in mediating high-affinity K^+ transport in plants has been questioned since its initial characterization by Schachtman and Schroeder (1994). Hailed as the first identification of a gene encoding high-affinity K^+ transport, *HKT1* was isolated from a cDNA library derived from K^+-deprived wheat (*Triticum aestivum*). *HKT1* showed sequence similarity with other TRK-type K^+ transporters (i.e., from yeast), and functionally complemented yeast deficient in K^+ uptake (Schachtman and Schroeder, 1994). However, K^+ transport via HKT varies with the expression system used to test its function, is strongly influenced by the presence of Na^+, and, most importantly, depends on the member of the *HKT* gene family under investigation. Studies using *Xenopus* oocytes and yeast have indicated that one role for HKT family members may be that of a K^+/Na^+ symporter at low $[Na^+]_{ext}$, and as a Na^+-specific transporter at higher $[Na^+]_{ext}$ (Rubio et al., 1995; Gassmann et al., 1996; Golldack et al., 2002; Garciadeblás et al., 2003; Haro et al., 2005). However, tests for coupled K^+/Na^+ symport in intact plants have shown that micromolar $[Na^+]_{ext}$ stimulates neither K^+ uptake nor plant growth (Maathuis et al., 1996; Box and Schachtman, 2000). Other evidence supports a limited role for the HKT family in K^+ uptake, at least under K^+-starved conditions (Uozumi et al., 2000; Horie et al., 2001; Garciadeblás et al., 2003; Haro et al., 2005), but these transporters may be much more important in Na^+ uptake by plants (Uozumi et al., 2000; Horie et al., 2001; Garciadeblás et al., 2003; Kader et al., 2006; Horie et al., 2007) and for its internal allocation, particularly in its removal from the xylem, and circulation through the phloem (Fairbairn et al., 2000; Berthomieu et al., 2003; Garciadeblás et al., 2003; Su et al., 2003; Rus et al., 2004, 2006; Sunarpi et al., 2005; Kader et al., 2006; Davenport et al., 2007). The HKT family has served as an important demonstration of the diversity and complexity of ion transport physiology, and sounds a note of caution in the interpretation of results from heterologous expression systems and their applicability *in planta*.

The plant cation, proton antiporter (CPA) superfamily has also been implicated in the mediation of K^+ uptake, despite functional analyses describing cation antiporters more as regulators of cellular ion homeostasis by expulsion of stress-inducing ions such as Na^+ (Pardo et al., 2006; Apse and Blumwald, 2007; Figure 1). Indeed, the most well-characterized CPA transporters are members of the CPA1 family, which predominantly mediate Na^+/H^+ exchange, either intracellularly or across the plasma membrane (Brett et al., 2005). However, one such member, NHX1, has been shown to also mediate K^+ transport in leaf tonoplast vesicles from tomato plants (*S. lycopersicum*, Zhang and Blumwald, 2001), while Venema et al. (2003) characterized a novel NHX gene from tomato plants (*LeNHX2*), closely related to *A. thaliana* NHX5 (Yokoi et al., 2002), that encodes an intracellular K^+/H^+ exchanger. LeNHX2 has been shown to affect plant growth, salt tolerance, and K^+ compartmentation, and appears to be localized to small intracellular vesicles (Rodríguez-Rosales et al., 2008).

More speculatively, some members of the CPA2 family may encode K^+/H^+ exchangers. KHA1 from *Saccharomyces cerevisiae* belongs to this family and appears to mediate an intracellular K^+ flux (Maresova and Sychrova, 2005, 2006), while a number of closely related transporters have been identified in plants by structural homology (Sze et al., 2004). Cellier et al. (2004) demonstrated increased transcript abundance of a gene (*AtCHX17*) encoding a putative K^+/H^+ *anti*porter in response to K^+ starvation and Na^+ stress. While the group hypothesized that the antiporter may function in K^+ acquisition, it is difficult to envisage how it would function in energetic terms, since both K^+ uptake and H^+ extrusion would likely be against the respective electrochemical gradients for each ion. Shin and Schachtman (2004) also observed transient transcriptional up-regulation by K^+ deprivation of the *KEA5* gene, which putatively encodes another K^+ antiporter in the CPA2 family. Like KHA1, other members of this family may operate intracellularly, including AtCHX23 and AtCHX20, which have been located in the chloroplast envelope (Song et al., 2004) and endosomal membranes (Padmanaban et al., 2007), respectively. While results suggest that CHX and KEA transporters participate in cellular K^+ homeostasis, determination of their precise roles needs further attention.

Low-affinity K^+ transport

The low-affinity transport system (LATS) for K^+ predominantly functions at high external concentrations (generally above 1 mM), and is generally considered to be channel-mediated (Epstein et al., 1963; Kochian and Lucas, 1982; Kochian et al., 1985; Gassmann and Schroeder, 1994; Maathuis and Sanders, 1995; White and Lemtiri-Chlieh, 1995; White, 1996; Hirsch et al., 1998), largely because of its high flux capacity and sensitivity to channel inhibitors. Pharmacological agents that have been extensively tested on channel-mediated transport systems in animals (Hille, 1992), including TEA^+, Cs^+, Ba^{2+}, calcium (Ca^{2+}), lanthanum (La^{3+}), and quinidine, have powerful effects on plant systems, demonstrating strong similarities between the two kingdoms (Leonard et al., 1975; Ketchum and Poole, 1990; Blatt, 1992; Wegner et al., 1994; Roberts and Tester, 1995; White and Lemtiri-Chlieh, 1995; Nocito et al., 2002; also see below).

Unlike HATS, uptake in the LATS range is thermodynamically passive (Maathuis and Sanders, 1996a). However, a consequence of both the passive uptake of K^+ and its active uptake via H^+/K^+ symport is an electrogenic entry of net positive charge, which requires the active removal of protons to maintain electrical neutrality (see Gerendás and Schurr, 1999; Rodríguez-Navarro, 2000). Were neutralization not to occur, K^+ influx (e.g., with channel-mediated rates between 1×10^6 and 1×10^8 ions s^{-1} protein^{-1}; Maathuis et al., 1997) could cause a precipitous depolarization of the plasma membrane and the loss of its normal electrical properties (Britto and Kronzucker, 2006). Therefore, a distinction between K^+ HATS and LATS, based upon energy requirement, must include the more subtle distinction between the coupling of K^+ and H^+ influx, which drives K^+ entry against an electrochemical potential gradient in the case of HATS, and the expulsion of H^+ following active or passive K^+ entry for charge balancing, in the case of both HATS and LATS.

LATS-mediated K^+ influx can be further distinguished from HATS by its lack of down-regulation at high external $[K^+]$, despite both increased tissue K^+ levels (Szczerba et al., 2006), and a progressively depolarized plasma membrane (Pitman et al., 1970; Cheeseman and Hanson, 1979; Kochian et al., 1989; Maathuis and Sanders, 1996a). In addition, the linear increase of the flux often observed in response to K^+ supply, under steady-state (Szczerba et al., 2006) and non-steady-state conditions (Kochian and Lucas, 1982), sharply contrasts with the characteristically saturable response in the HATS range. However, it should be noted that LATS has also been described by Michaelis–Menten kinetics, depending on the experimental approach used, with "K_M" and "V_{max}" values being consistently high when saturation is observed (Epstein et al., 1963; Kochian and Lucas, 1982, 1983; Kochian et al., 1985; Wrona and Epstein, 1985; Fu and Luan, 1998). The identification of ion channels as likely mediators of LATS transport has removed much of the disagreement concerning the uniqueness of the LATS mechanism, despite recent discoveries of ion transporters with dual-affinity characteristics (Hirsch et al., 1998; Fu and Luan, 1998; Liu et al., 1999; see above).

LATS influx is also NH_4^+-insensitive, in contrast to HATS (Spalding et al., 1999; Santa-María et al., 2000; Kronzucker et al., 2003; Szczerba et al., 2006), to the extent that increasing $[K^+]_{ext}$ into the LATS-dominated range can alleviate the symptoms of NH_4^+ toxicity that appear at lower $[K^+]_{ext}$ (Mengel et al., 1976; Cao et al., 1993; Gerendás et al., 1995; Santa-María et al., 2000; Kronzucker et al., 2003; Szczerba et al., 2006, 2008a). Because K^+ and NH_4^+ are univalent cations with similar hydrated atomic radii, it has been suggested that they share a common transporter, and that K^+ may alleviate NH_4^+ toxicity by competing with NH_4^+ at the transport level (Kielland, 1937; Wang et al., 1996; White, 1996; Nielsen and Schjoerring, 1998; Hess et al., 2006). Recent $^{13}NH_4^+$ work in barley has confirmed the K^+-dependent reduction of toxic NH_4^+ fluxes (Szczerba et al., 2008a).

In contrast to NH_4^+, Na^+ suppresses K^+ influx in both LATS and HATS ranges (Rains and Epstein, 1967; Benlloch et al., 1994; Flowers and Hajibagheri, 2001; Fuchs et al., 2005; Kronzucker et al., 2006, 2008; Wang et al., 2007). The reasons for this are unclear, but Na^+ may directly inhibit K^+ uptake, possibly because Na^+ itself utilizes K^+ LATS transporters (Wang et al., 2007), or because Na^+ stress brings about decreased expression of K^+-specific LATS transporters (Golldack et al., 2003).

An impressive array of ion channels has been characterized in plant systems by use of multiple experimental approaches. Electrophysiological analyses of guard cells, xylem parenchyma cells, and root protoplasts have revealed the presence of K^+-specific channels that are inwardly rectifying and activated by membrane hyperpolarization (Lebaudy et al., 2007). Expression studies complementing yeast mutants deficient in K^+ uptake yielded the genetic sequence of the first two inwardly rectifying K^+ channels discovered in plants, KAT1 (expressed in guard cells) and AKT1 (expressed predominantly in roots, with other AKT isoforms found throughout the plant; Anderson et al., 1992; Sentenac et al., 1992; Lebaudy et al., 2007; Figure 1). Both KAT1 and AKT1, as well as many of their homologs, share numerous genetic and physiological features with animal Shaker-type K^+ transporters, including six transmembrane domains; a voltage sensor domain located at the fourth transmembrane domain and rich in basic amino acids; a pore region located between the fifth and sixth transmembrane domains, containing the highly conserved GYGD amino acid sequence; and a putative cyclic-nucleotide-binding domain located near the C-terminus (Maathuis et al., 1997; Czempinski et al., 1999; Zimmermann and Sentenac, 1999; Chérel, 2004; Gambale and Uozumi, 2006; Gierth and Mäser, 2007; Lebaudy et al., 2007). They are also inhibited by K^+-channel-specific inhibitors such as TEA^+, Ba^{2+}, and La^{3+} (Wegner et al., 1994; Bertl et al., 1995; Müller-Röber et al., 1995; Véry et al., 1995; Lewis and Spalding, 1998; Nielsen and Schjoerring, 1998). In addition, Shaker K^+ channels in both animal and plant systems have been shown to assemble in the plasma membrane as tetramers (MacKinnon, 1991; Daram et al., 1997). Unlike high-affinity K^+ transporters, *AKT1* transcript levels do not respond to K^+ starvation in most systems, consistent with its mediation of K^+ uptake at high external $[K^+]$ (Lagarde et al., 1996; Su et al., 2001; Pilot et al., 2003). One notable exception was found by Buschmann et al. (2000), who showed an increase in *AKT1* transcript abundance and K^+ currents in K^+-starved wheat, suggesting that K^+ channels in wheat may play a greater role in K^+ scavenging than in other species.

Electrophysiological analyses showing the NH_4^+-insensitivity of specific Shaker-type K^+ channels in plants confirm previous physiological studies (Bertl et al., 1995; Müller-Röber et al., 1995; White, 1996; Hirsch et al., 1998; Moroni et al., 1998; Spalding et al., 1999; Su et al., 2005). In one compelling study, differential sensitivity to NH_4^+ in HATS and LATS was exploited to demonstrate the ability of AKT1 to mediate K^+ transport in the high-affinity range: after inhibition of HATS with NH_4^+ in *A. thaliana*, *akt1* mutants grew very poorly at low $[K^+]_{ext}$, while wild-type seedlings were much less affected, indicating that AKT1 could scavenge K^+ at concentrations as low as 10 μM (Hirsch et al., 1998; Spalding et al., 1999).

Less well understood is the role of K^+ channels in mediating Na^+ fluxes, and the effect of Na^+ stress upon K^+ channel activity. It has been demonstrated that increasing extracellular Na^+ can reduce K^+ channel transcript abundance in *A. thaliana*, *M. crystallinum*, and *O. sativa* (Su et al., 2001; Golldack et al., 2003; Pilot et al., 2003), and it has been suggested that AKT1 mediates Na^+ fluxes (Golldack et al., 2003; Obata et al., 2007; Wang et al., 2007). Interestingly, Qi and Spalding (2004) found that a cytosolic $[Na^+]$ of only 10 mM completely inhibited AKT1-mediated inward currents in *Arabidopsis* protoplasts examined using whole-cell patch-clamping. Essah et al. (2003), however, found no difference in Na^+ accumulation in *A. thaliana akt1* mutants as compared with wild-type seedlings, and, similarly, Obata et al. (2007) found either the same, or lower, Na^+ content in yeast and rice cells expressing *OsAKT1* (overexpressing, in the case of rice), relative to untransformed cells. Buschmann et al. (2000), in a patch-clamp study with AKT1 from wheat (TaAKT1), concluded that K^+ and Na^+ currents are not mediated by the same transporter. Kronzucker et al. (2006, 2008) found that an approximately 400-fold range in $[K^+]_{ext}$ had little effect on Na^+ influx in barley seedlings grown with 100 mM $[Na^+]_{ext}$, while, by contrast, Na^+ stress profoundly inhibited K^+ uptake. These results suggest that under certain circumstances Na^+ may utilize K^+ channels, but this should not be taken as a general rule.

Other important K^+ channels within plants

In addition to mediating primary K^+ uptake, channels play an important role in long-distance K^+ fluxes via the vasculature. Early work on channels in root xylem parenchyma cells showed TEA^+ and La^{3+} inhibition (Wegner et al., 1994), and subsequent investigations attributed a component of xylem K^+ loading to the activity of SKOR, a Shaker-type efflux channel found in stelar parenchyma cells (Gaymard et al., 1998; Figure 1). SKOR-deficient *A. thaliana* mutants showed a 50% reduction in shoot K^+ content, while root content was unaffected (Gaymard et al., 1998). High NH_4^+ reduces K^+ flux to the shoot, and shoot K^+ content, by as much as 90% (Kronzucker et al., 2003), suggesting that xylem loading, possibly mediated by SKOR and other transporters, is sensitive to NH_4^+

(Santa-María et al., 2000; Kronzucker et al., 2003; Szczerba et al., 2006, 2008b). Similarly, phloem K^+ loading and unloading may be mediated by another Shaker-type channel, AKT2, which was identified in phloem cells using β-glucuronidase (GUS) reporting and *in situ* hybridization (Marten et al., 1999; Lacombe et al., 2000; Deeken et al., 2000). K^+ starvation increases transcript abundance of *SKOR* and *AKT2*, while abscisic acid (ABA) shows opposing effects on the two genes, reducing *SKOR* mRNA abundance, while increasing that of *AKT2* (Marten et al., 1999; Lacombe et al., 2000, Deeken et al., 2000, 2002; Pilot et al., 2001, 2003). This dual effect is consistent with the role of ABA during water stress: reduced K^+ transport to the shoots, and increased delivery of K^+ to the roots via the phloem, may be critical in increasing the osmotic strength of roots deprived of water.

In contrast to the inward flux of K^+ through KAT1 in guard cells, the rapid removal of K^+ during stomatal closure has been attributed in large part to the GORK Shaker channel (Ache et al., 2000). Indeed, *gork* gene mutations or disruptions in the protein-mediated regulation of the GORK channel have been shown to disrupt water relations in plants (Hosy et al., 2003; Becker et al., 2003).

Several other channel types have been shown to transport K^+ in plants, including the tandem-pore K^+ (TPK) channels (Czempinski et al., 1999; Zimmermann and Sentenac, 1999; Mäser et al., 2001, 2002; Ashley et al., 2006; Lebaudy et al., 2007). TPK transporters, found in plant, animal, and fungal systems, have between two and eight transmembrane domains, with either an individual pore or, more frequently, two pores, separated by two transmembrane domains, each containing a GYGD sequence, similar to Shaker channels (Zimmermann and Sentenac, 1999; Mäser et al., 2001, 2002; Czempinski et al., 1999; Ashley et al., 2006; Lebaudy et al., 2007). Unlike with Shaker-type channels, however, TPK subunits do not appear to form heteromeric proteins (Voelker et al., 2006). TPK channels have been identified in roots, leaves, and flowers, localizing to the tonoplast or plasma membrane (Figure 1), with regulatory sites for Ca^{2+} binding and phosphorylation (Czempinski et al., 1997, 2002; Moshelion et al., 2002; Latz et al., 2007). Although a number of putative plant TPK channels have been identified, *in planta* function has only been determined for two members: AtTPK4, located at the plasma membrane, which participates in pollen and pollen-tube K^+ transport (Becker et al., 2004); and TPK1, a tonoplast-localized channel that is Ca^{2+}-activated, pH-sensitive, and voltage-insensitive (Gobert et al., 2007). Based on these characteristics, TPK1 has been suggested to be the VK (vacuolar K^+) channel, previously identified by electrophysiological means (Ward and Schroeder, 1994; Allen and Sanders, 1996; Bihler et al., 2005).

Although not a TPK channel, another two-pore channel (TPC1) having Shaker family-type structure with 12, rather than six, membrane-spanning domains, and showing Ca^{2+} and K^+ transport capabilities, has been identified (Furuichi et al., 2001; Peiter et al., 2005). Electrophysiological analysis of this tonoplast-localized channel (Figure 1) in protoplasts showed ion conductances identical to those previously attributed to slow vacuolar (SV) channels (Hedrich and Neher, 1987; Ward and Schroeder, 1994; Allen and Sanders, 1995; Peiter et al., 2005). Moreover, *A. thaliana* mutants either overexpressing *TPC1*, or having a *TPC1* knockout, exhibited SV-type channel conductances that were, respectively, either enhanced or silenced (Peiter et al., 2005). Despite a possible molecular identity for SV channels, high abundance in the tonoplast, and considerable interest in its role, no *in planta* function has yet been assigned to it. However, it has been suggested to mediate K^+ fluxes into and out of the vacuole (Allen and Sanders, 1996; Ivashikina and Hedrich, 2005), as well as Ca^{2+} fluxes from the vacuole to the cytosol (Pottosin and Schönknecht, 2007).

Cyclic-nucleotide gated channels (CNGCs, Mäser et al., 2001, 2002; Trewavas et al., 2002; Ashley et al., 2006), like TPK channels, comprise an important emerging class of K^+ transporter (Figure 1). CNGCs share structural homology with Shaker channels, having six transmembrane domains, with a pore domain located between the fifth and sixth transmembrane units (Talke et al., 2003). CNGCs and some Shaker channels also share the characteristic of activation by cyclic nucleotides (Véry and Sentenac, 2003). The cyclic-nucleotide-binding domain for CNGCs is located in the carboxyl terminus of the protein, along with a calmodulin-binding domain (Talke et al., 2003). However, unlike Shaker channels, CNGCs do not have a consistent pore sequence comparable to GYGD (Talke et al., 2003). Of the identified CNGCs, two have been shown to have equal K^+ and Na^+ conductance (AtCNGC1 and AtCNGC4; Balague et al., 2003; Hua et al., 2003), and a third has been implicated in K^+ uptake (AtCNGC10, Borsics et al., 2007). However, it has been suggested that CNGCs mainly function in mediating Na^+, Ca^{2+}, or nonselective cation transport in plants, a role that may also describe TPC1 (Maathuis and Sanders, 2001; Demidchik et al., 2002; Demidchik and Maathuis, 2007).

Regulatory mechanisms

A number of regulatory mechanisms have been identified for K^+ transporters, particularly those of the Shaker family. In patch-clamp studies, Schroeder and Fang (1991) and Su et al. (2005) observed that decreased K^+ supply reduced current conductance and activation of guard cell K^+ channels, and concluded that these channels were inactivated at micromolar [K^+], in contrast to evidence that some Shaker channels continue to mediate K^+ currents at similarly low $[K^+]_{ext}$ (Hirsch et al., 1998; Brüggemann et al., 1999). In this case, low $[K^+]_{ext}$ was suggested to trigger a conformational change in the channel's pore region, essentially reducing its diameter and conductivity (Zhou et al., 2001; Hertel et al., 2005; Su et al., 2005). However, pore size alone does not determine channel activity, only the likelihood of permeability for an ion; other channel properties, such as ion-binding affinity and activation-sensor modulation, also play key roles (Zhou and MacKinnon, 2004; Lockless et al., 2007).

Ca^{2+} signaling and protein phosphorylation may also be central to ion sensing in plants. As mentioned previously, H^+-ATPase activity establishes electrical and pH gradients across the plasma membrane, which coexist with ion gradients, notably that of K^+. As changes occur in $[K^+]_{ext}$, the K^+ and H^+ gradients will adjust appropriately, unless the shift in $[K^+]_{ext}$ is severe, when another mechanism, possibly involving a Ca^{2+} signal cascade, may be elicited. This secondary reaction may recruit other molecules, such as calmodulin, to activate or deactivate a transporter, or initiate a signaling cascade that will ultimately modify gene transcription. The plasma membrane will now have a new complement of transporters, establishing a new steady-state in response to a changed external K^+ environment. Recently, a sophisticated Ca^{2+} signal transduction pathway, corresponding to the above hypothesis, and describing a specific regulatory mechanism for AKT1, was elucidated (Figure 2): the ankyrin domain of AKT1 interacts with a protein kinase (CIPK23) that activates AKT1 by phosphorylation, and is targeted by calcineurin B-like calcium sensors (CBL1 and CBL9), which are in turn activated by Ca^{2+} (Li et al., 2006; Xu et al., 2006; Lee et al., 2007). The Ca^{2+} signal is initiated by an unknown low K^+ sensor. AKT1 channel-inactivation can be achieved by dephosphorylation, via a 2C-type protein phosphatase (Lee et al., 2007). *cipk23* mutants of *A. thaliana* show impaired growth under low K^+ conditions (Cheong et al., 2007), further suggesting that K^+ channels may have an important role in K^+ scavenging (Hirsch et al., 1998; Buschmann et al., 2000). Previously, the function of the ankyrin domain of AKT1 was unknown but postulated to interact with the cytoskeleton, as described for animal systems (Davies et al., 1991; Bennett, 1992; Mills and Mandel, 1994).

Ca^{2+} may also regulate plant K^+ channels by interacting with guanine nucleotide-binding proteins ("G proteins"; Kelly et al., 1995; Wegner and

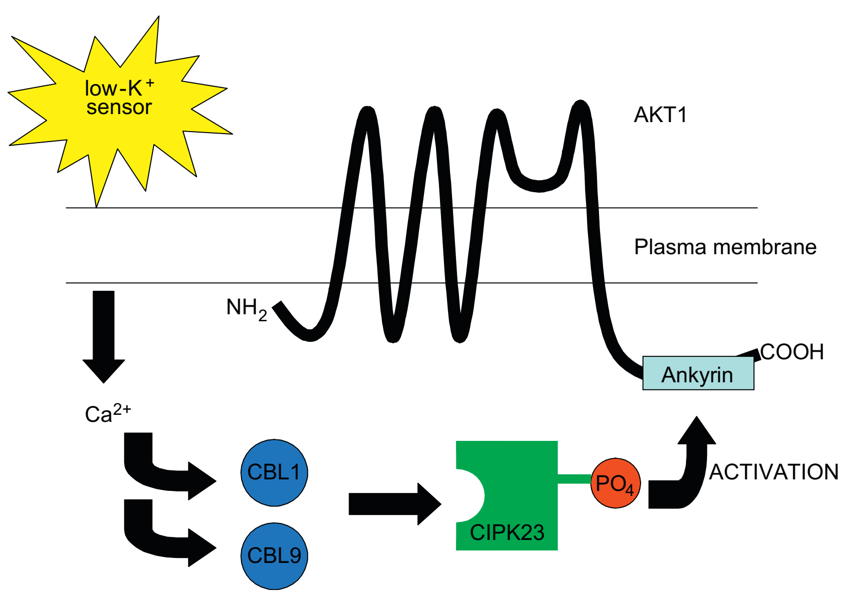

Figure 2. Activation mechanism for AKT1 under conditions of low $[K^+]_{ext}$. This diagram illustrates how recent work has delineated fine details of K^+ flux regulation. For details, see text. Based upon Lee et al., 2007. CBL = calcineurin B-like calcium sensor; CIPK = CBL-interacting protein kinase; Ankyrin = conserved region on C-terminus of Shaker family channels.

De Boer, 1997; Wang et al., 2001). While G proteins are known to regulate animal K^+ channels, there is scant information about their role in plant K^+ transport (Assmann, 2002). However, a role for G proteins in the control of stomatal aperture via modulation of K^+ channel currents has been suggested (Fan et al 2008).

Another class of proteins known to interact with K^+ channels are the 14-3-3 proteins, which have a wide range of functions in both plants and animals (Mackintosh, 2004), including regulation of high-affinity transporters such as the H^+-ATPase (De Boer, 2002). 14-3-3 proteins regulate K^+ channels intracellularly (van den Wijngaard et al., 2001; Latz et al., 2007), and at the plasma membrane (Saalbach et al., 1997; van den Wijngaard et al., 2005), and recent evidence demonstrates their role in modifying the recruitment of K^+ channels to the plasma membrane (Sottocornola et al., 2008).

Recently, a novel protein, OsARP, was identified in rice, and found to regulate tonoplast transport, stimulating Na^+ accumulation when overexpressed in tobacco (Uddin et al., 2008). Similar sequences can be found in a number of plants including *A. thaliana*, *Beta procumbens*, *Picea sitchensis*, *Populus trichocarpa*, and *Vitis vinifera*. While it is unclear how this protein works, and whether it plays a role in K^+ compartmentation, this discovery indicates that further investigation into protein–protein interactions of plant transporters will yield interesting and important results.

Characteristics of Shaker channels may also be modified via the variable composition of heteromeric complexes, as suggested by the indiscriminate, *in vivo* assembly of functional aggregates of heterogeneous channel subunits derived from different plant organs (e.g., roots and shoots), or even different plant species (e.g., *A. thaliana* and *S. tuberosum*; Dreyer et al., 1997; Baizabal-Aguirre et al., 1999; Pilot et al., 2001, 2003; Reintanz et al., 2002; Xicluna et al., 2007; Bregante et al., 2008). Distinct heteromeric channels vary in current conductances and sensitivities to H^+, Cs^+, and Ca^{2+}, reflecting unique subunit combinations (Dreyer et al., 1997; Baizabal-Aguirre et al., 1999; Reintanz et al., 2002; Xicluna et al., 2007). While the only *in planta* example of this type of regulation has been observed in protoplasts with heteromers composed of AtKC1 and AKT1 subunits (Dreyer et al., 1997), such a feature of Shaker channels may provide a mechanism for acclimation to abiotic stress or rapidly changing environmental conditions, via assembly of novel transporter complexes.

A relatively new research area focuses on the role of reactive oxygen species (ROS) in signal mediation. Shin and Schachtman (2004) found that K^+ deficiency leads to H_2O_2 release, which induces the expression of genes, such as *AtHAK5*, encoding K^+ transporters. H_2O_2 pretreatment of seeds has also been shown to increase Na^+ tolerance in wheat; under Na^+ stress, H_2O_2-treated plants had greater K^+ content than controls (Wahid et al., 2007). Interestingly, runaway ROS production may be curtailed via the ROS-dependent activation of the ATP-sensitive plant mitochondrial K^+ channel ($PmitoK_{ATP}$), located in the inner mitochondrial membrane (Pastore et al., 2007). Activation of this channel catalyzes K^+ transport into the matrix, and may reduce cellular redox stress by dissipating the membrane potential and discharging reducing equivalents.

K^+ efflux and K^+-use efficiency

Improvement of plant nutrient use efficiency, including that of K^+, is an agronomically important research area (Lea and Azevedo, 2007; Gerendás et al., 2008; Jia et al., 2008). Of particular interest is a plant's ability to maximize K^+ uptake, by increasing influx, decreasing efflux, or both. K^+ influx and efflux can both increase substantially with K^+ provision (Le Bot et al., 1998; Szczerba et al., 2006), resulting in a condition of futile cycling that may have toxic consequences (Britto and Kronzucker, 2006). One such consequence is the substantial energy required for the active removal of K^+ under high $[K^+]_{ext}$, a "leak-pump" condition similar to what is observed for Na^+ under NaCl stress (Szczerba et al., 2006, 2008a). Thus, the investigation of K^+ efflux is an essential area of practical importance.

Despite functional characterization of root K^+ efflux, including the electrophysiological identification of outward-rectifying K^+ currents from root hairs and from cortical and xylem parenchyma cells (Ketchum et al., 1989; Schachtman et al., 1991; Wegner and De Boer, 1997), little is known about the molecular identity of these transporters. Two members of the Shaker family have been identified as participating in outward-rectifying currents: GORK, in root hairs, and SKOR, in the stele (Gaymard et al., 1998; Ivashikina et al., 2001; Becker et al., 2003), while the identity of cortical K^+ efflux channels is an open question.

Also as yet unaddressed at the molecular level is the identity of the transporter(s) mediating K^+ efflux against its electrochemical potential gradient, at millimolar $[K^+]_{ext}$ (Szczerba et al., 2006). Candidates for this role are likely to come from the

CPA superfamily, such as a CHX transporter, and/or K^+ efflux may be mediated by the Na^+ pump, SOS1. Although it has been claimed that SOS1 discriminates strongly against K^+ in favor of Na^+ (Pardo et al., 2006), unlike the closely related tonoplast Na^+/H^+ exchanger NHX1, this point has not been satisfactorily demonstrated. Studies by Quintero et al. (2002) and Shi et al. (2002) showed that *sos* mutants had altered K^+ content or uptake, but did not demonstrate a direct role for SOS1 in K^+ transport. By contrast, both Zhang and Blumwald (2001) and Venema et al. (2002) showed that the NHX1 protein can mediate Na^+ or K^+ transport, with similar kinetics. Moreover, Gaxiola et al. (1999) showed that K^+ can stimulate NHX expression, and Venema et al. (2003) demonstrated that LeNHX2 from tomato discriminates in favor of K^+ over Na^+.

While much more work is necessary to determine the molecular identities of outwardly directed K^+ transporters in the plasma membrane, this work may prove beneficial in reducing fertilizer usage for economic and environmental reasons. K^+-use efficiency is a key agronomic measurement, and it may be critical to maximize cellular K^+ use efficiency before other gains can be made at the whole-plant level.

K^+ transport and root zonation

An interesting aspect of K^+ transport is the relative contribution, and localization, of HATS and LATS across the variety of soil conditions encountered by plant roots. Kochian and Lucas (1983) identified the root periphery as HATS-enriched, while the cortex became more important under conditions of greater K^+ provision. This view has gained some molecular support, in that expression of the high-affinity *HAK* transporter was found to be greater in the root epidermis than in the cortex (Su et al., 2002; Gierth et al., 2005), although this is a species-specific pattern (Fulgenzi et al., 2008). AKT1, by contrast, is expressed throughout the root, in both cortical and epidermal layers (Su et al., 2001; Golldack et al., 2003). Another member of the Shaker family, AtKC1, which forms functional channels only in association with AKT1, is restricted to the root epidermis (Ivashikina et al., 2001; Pilot et al., 2003). Thus, the expression of AKT1 represents a pattern that supports both high and low-affinity transport, consistent with its ability to transport K^+ under both HATS and LATS conditions (Hirsch et al., 1998; Buschmann et al., 2000).

Longitudinally, a different localization pattern can be seen. AKT1 has been found in root apical cells, and in the remainder of the root (Hirsch et al., 1998; Vallejo et al., 2005), while, by contrast, HAK transcripts in barley were found to be present in high abundance only above the first 10 mm from the root tip (the area already occupied by AKT1, Vallejo et al., 2005). Unfortunately, information describing longitudinal expression patterns of other K^+ transporters is lacking. While broad tissue localization to "root" or "shoot" can be found, transport candidates such as LCT1 or CNGCs must also be mapped along the root axis to more fully assess their functional roles.

The contribution of AKT1 to K^+ transport under various K^+ conditions remains a topic of interest. It has been claimed that AKT1 may account for 55–63% of K^+ uptake under conditions of low $[K^+]_{ext}$ (Spalding et al., 1999), even though only a small fraction of cells may participate in this. This is because a very hyperpolarized membrane is necessary to ensure that the electrochemical gradient is adequate for passive K^+ uptake (Hirsch et al., 1998). It remains to be demonstrated how much of the root is involved in K^+ uptake, particularly under these conditions of low K^+. One suggestion is that AKT1 is most important under conditions of NH_4^+ supply (Rodríguez-Navarro and Rubio, 2006), implicating a greater role of the root tip under such conditions. Ultimately, it may be found that the root is divided into functional segments specializing in different mechanisms of K^+ transport, with each segment's importance depending on external $[K^+]$ and on the presence of potentially toxic ions.

Concluding remarks

Understanding the diversity of K^+ transporters in plants can be a daunting task, particularly as new evidence increases the variety of known K^+ uptake and efflux mechanisms. From the initial description of K^+ uptake as the two systems, HATS and LATS, to the diversity of K^+ transporters that have now been identified at the molecular level, our understanding of K^+ transport has grown tremendously. However, several key questions remain unanswered. It is clear that there is redundancy in the K^+ transport machinery of plants, but an integrated picture of how these transporters cooperate is still incomplete. It also remains unclear how energy is conserved to mediate K^+ transport, although recent investigations have shown that, in addition to H^+- or Na^+-coupled K^+ transport, plants possess cation-chloride cotransporters (CCCs), such as found in animals (Colmenero-Flores et al., 2007).

It may also emerge that the regulation of K^+ transport in plants is closely associated with water transport, a finding recently discovered in virus–host interactions (Gazzarrini et al., 2006). Such investigations may lead to new insights concerning the interactions between K^+ and NH_4^+ or Na^+, ions that have been shown to inhibit K^+ uptake or bring about K^+ loss (Rubio et al., 1995; Shabala and Cuin, 2008; Szczerba et al., 2008b). However, at a more basic level, it still is not understood how K^+ sensing occurs in plants, nor what may be the preliminary signals initiating the downstream cascades that activate K^+ transport. However, a recent study by Nieves-Cordones et al. (2008) found that changes in $\Delta\Psi_{PM}$ could affect the gene expression of a K^+ transporter, supporting the hypothesis described above.

It is evident that further studies, at both molecular and whole-plant levels, are needed to help unravel the matrix of K^+ transporters, and the signals and regulators that affect their activities. If goals of higher potassium-use efficiency are to be realized, a variety of approaches will be necessary to more adequately comprehend the complexity of K^+ transport. It is clear that investigations focusing on K^+ transport are as critical today as they were nearly 50 years ago, when the dual-pattern of K^+ uptake was initially characterized by Epstein et al. (1963).

Acknowledgements

We thank Drs. E Blumwald, E Katz, and V Martínez for useful discussion of this manuscript. The work was supported by grants from the Natural Sciences and Engineering Research Council of Canada (NSERC) and the Canada Research Chair (CRC) program.

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