Physiologia Plantarum
An International Journal for Plant Biology

MINIREVIEW

The physiology of channel-mediated K^+ acquisition in roots of higher plants

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Received 1 October 2013;
revised 17 January 2014

doi:10.1111/ppl.12174

K^+ channels are among the best-characterized classes of membrane protein in plants. Nevertheless, in-planta demonstrations of traits emerging from molecular characterizations have often been insufficient or lacking altogether. Such linkages are, however, critical to our basic understanding of plant nutrition and to addressing 'real-world' issues that are faced in environmental and agricultural settings. Here, we cover some of the recent advances in K^+ acquisition with particular focus on voltage-gated K^+ channel functioning and regulation in roots, and highlight where linkages to in-planta behavior have been successfully made and, conversely, where such linkages are yet to be made.

Introduction

Potassium (K^+) is an essential macronutrient for plant growth and development (Marschner and Marschner 2011). Thus, understanding the nature of plant K^+ acquisition is of truly fundamental importance, accentuated by the common occurrence of soil K^+ deficiencies (Römheld and Kirkby 2010, White 2013). Contemporary research on K^+ uptake is based on a strong foundation laid out in early physiological studies that kinetically mapped the primary acquisition of K^+ from external solutions with radiotracers (Epstein et al. 1963, Glass 1976, Kochian and Lucas 1988). With the advent of molecular technologies, these earlier physiological studies have been augmented by in-depth mechanistic examinations, particularly with respect to the function of K^+ channels, which mediate thermodynamically passive fluxes of K^+ across biological membranes (i.e. down its electrochemical gradient). The first molecular studies of plant K^+ channels were reported in 1992, based on work using the model species Arabidopsis (*Arabidopsis thaliana*; Anderson et al. 1992, Sentenac et al. 1992). We now know that some 15 genes code for K^+ channels in Arabidopsis; six encode non-voltage-gated channels and nine voltage-gated (Shaker-like) ones; the latter comprise the best-characterized transporter class in plants (for review, see Dreyer and Uozumi 2011). The class of voltage-gated K^+ channels can be further divided into four subcategories, based on their function: (1) inward-rectifying (K_{in}) channels, (2) silent (K_{silent}) channels, (3) weakly-rectifying (K_{weak}) channels and (4) outward-rectifying (K_{out}) channels. K_{in} and K_{out} channels mediate the inward and outward transport of K^+ across cell membranes, respectively. K_{silent} channels modify functional properties of some K_{in} channels. Lastly, K_{weak} channels have been shown to play important roles in K^+ translocation.

For the purpose of this minireview, we shall focus on the role of voltage-gated K^+ channels in primary K^+ acquisition in roots of higher plants. We summarize several recent investigations related to channel function and

Abbreviations – AIP, AKT1-interacting PP2C; AKT, Arabidopsis K^+ transporter; BUS, backup system; CBL, calcineurin B-like protein; CIPK, CBL-interacting protein kinase; CNGC, cyclic-nucleotide-gated channel; HAK, high-affinity K^+ transporter; K_{in}, inward-rectifying K^+ channel; K_{out}, outward-rectifying K^+ channel; K_{silent}, silent K^+ channel; K_{weak}, weakly rectifying K^+ channels; NSCC, non-selective cation channel; PP2C, protein phosphatase 2C; ROS, reactive oxygen species; SNARE, soluble *N*-ethylmaleimide-sensitive factor protein attachment protein receptor; TEA^+, tetraethylammonium; WT, wild type;

regulation, including (1) their involvement in primary K^+ uptake, (2) the engagement of signaling cascades and post-translational modifications in K^+-channel gating, and (3) the intersection between vesicular trafficking, K^+-channel function, and cell expansion. Many of these studies have employed detailed molecular-genetic and electrophysiological approaches to address questions of gene expression, channel localization, post-translational modifications, and protein–protein interactions, amongst others, typically in heterologous expression systems, such as *Xenopus* oocytes, yeast or insect cells, or in isolated protoplasts. However, the linking back of such studies to the level of the intact plant has often been limited to recording phenotypic differences (growth responses), external K^+ depletion and/or tissue K^+ content, and wild type (WT) controls to knock-out or over-expressor mutant lines for a gene of interest. However, it is critical that in-planta demonstrations of molecular discoveries be forthcoming, if any extrapolations to plant performance under field conditions are to be made. We here discuss important caveats related to generalizing findings from single-cell (heterologous/protoplast) systems to the level of the intact plant, and from those that have remained limited to the Arabidopsis model to other plants, such as cereals.

Primary K^+ acquisition: can K^+ channels do it all? Where do we stand now?

The involvement of specific channels in primary K^+ uptake

Two decades ago, (Kochian and Lucas 1993) speculated on the involvement of K^+ channels in mediating primary K^+ acquisition at very low (micromolar) external K^+ concentrations ($[K^+]_{ext}$), and this question continues to be relevant today (e.g. see Coskun et al. 2013, Coskun and Kronzucker 2013). The argument of Kochian and Lucas' was made in light of K^+-channel studies in heterologous expression systems that demonstrated channel-mediated K^+ uptake at very low $[K^+]_{ext}$ (e.g. 20 μ*M*; see Schachtman et al. 1992), which led to the speculation that channels may mediate nutritionally relevant, high-affinity K^+ acquisition in plant roots, in addition to their recognized function at high substrate concentrations (Szczerba et al. 2006). However, thermodynamic considerations presented by Kochian and Lucas questioned this conclusion: in maize, they showed that high-affinity (e.g. at $[K^+]_{ext}$ <25 μ*M*) K^+ uptake in roots was a thermodynamically active process and, thus, could not be mediated by channels, which conduct strictly passive fluxes. (Maathuis and Sanders 1993) came to a similar conclusion in the Arabidopsis case (see also Maathuis and Sanders 1994). This conclusion appears to have been superseded, however, by the demonstration by (Hirsch et al. 1998) that AtAKT1 (the primary K_{in} channel in Arabidopsis roots) can mediate K^+ uptake at $[K^+]_{ext}$ as low as 10 μ*M*, given that the H^+-ATPase can generate a sufficiently polarized membrane to provide an inwardly directed electrochemical gradient for passive K^+ influx (Dreyer and Blatt 2009). Importantly, this study was conducted in roots exposed to high (millimolar) concentrations of ammonium (NH_4^+), applied to inhibit the high-affinity (non-AtAKT1) K^+-acquisition system (Cao et al. 1993; see also Spalding et al. 1999, Qi et al. 2008). By contrast, Kochian and Lucas (1993) and Maathuis and Sanders (1993) used either a simplified growth medium (0.2 m*M* $CaSO_4 \pm K^+$) or a modified Murashige and Skoog (MS) medium, with NO_3^- as the sole N source. Thus, it may well be the case that channels only operate in-planta under low-K^+ conditions in Arabidopsis when NH_4^+ is present. We return to this important caveat in the sections below.

An important feature of K^+ channels, which appears to be conserved across kingdoms, is the ability of the selectivity filter in the channel pore to undergo conformational change and 'collapse' when $[K^+]_{ext}$ is lowered, inactivating the channel (Zhou et al. 2001, Bernèche and Roux 2005). The threshold value of $[K^+]_{ext}$, below which inactivation occurs, varies considerably among plant Shaker-like channels. For example, while AtKAT1 from guard cells can operate at micromolar concentrations (Hertel et al. 2005), AtAKT1 is mostly inactive under such conditions (Geiger et al. 2009). The physiological role of such channel inactivation might be to restrict K^+ leakage via these channels when the electrochemical gradient favors K^+ efflux (Dreyer and Uozumi 2011). The higher inactivation threshold in AtAKT1 compared with AtKAT1 may reflect the importance of minimizing K^+ leakage in roots, given that soil $[K^+]$ can vary considerably and at times be precariously low (Dreyer and Uozumi 2011; also see below regarding K^+ leakage in AtAKT1-AtKC1 channel complexes). It is important to note, however, that such functional characterizations stem from patch-clamp analyses in isolated protoplasts or heterologous systems, where ionic conditions are significantly different from those in whole-plant studies. This makes it difficult to compare and reconcile patch-clamp studies to whole-plant studies, such as in the case of AtAKT1 where in-planta analysis has demonstrated channel function at $[K^+]_{ext}$ as low as 10 μ*M* (Hirsch et al. 1998), which appears at odds with patch-clamp studies (see above). Despite these difficulties, such comparative studies are not impossible (see e.g. Tyerman and Skerrett 1999), and in fact are critical if we are to synthesize findings at different levels of

biological organization, and ultimately apply them to practical ends such as agriculture.

The applicability of the Arabidopsis model of K^+ acquisition (Hirsch et al. 1998, Aleman et al. 2011) to important other plant species was recently questioned by Coskun et al. (2013), who found that, unlike in Arabidopsis, roots of intact barley (*Hordeum vulgare*) seedlings could not conduct channel-mediated K^+ uptake at $[K^+]_{ext}$ <100 µ*M*, in the presence of high NH_4^+. However, it was shown that when NH_4^+ was withdrawn in the short term (5 min), K^+ acquisition in intact barley roots from $[K^+]_{ext}$ as low as 22.5 µ*M* was greatly stimulated (over 2.5-fold), and this stimulation could be attributed to channel activity. Interestingly, similar stimulations have also been observed in Arabidopsis single- and double-knock-out mutants for AtAKT1 and AtHAK5 (the primary high-affinity transporter in roots; Gierth et al. 2005), revealing the concerted and complex nature of K^+-uptake mechanisms. Moreover, it was demonstrated under high (5 m*M*) $[K^+]_{ext}$, that K^+ uptake (in the presence of high NH_4^+) could be stimulated twofold if nitrate (NO_3^-) or chloride (Cl^-) anions were introduced for a brief period (5 min), and >3.5-fold if NH_4^+ was simultaneously withdrawn (see also Coskun and Kronzucker 2013). Similar 'anion effects', under conditions of thermodynamically passive K^+ uptake (i.e. at high $[K^+]_{ext}$), were examined in detail by (Kochian et al. 1985), but have remained relatively unexplored since then. The mechanism underlying these effects remains particularly elusive, considering that molecular characterizations of K_{in} channels currently leave no room for anion-induced modifications (e.g. see Doyle et al. 1998). Given the significant increases in K^+ acquisition observed, a focus on the mechanistic underpinnings of this trait is clearly needed (see above; Fig. 1).

Distinct from the role of Shaker-like K^+ channels, that of non-selective cation channels (NSCCs) in mediating K^+ uptake in-planta is important to consider. NSCCs are an extensively characterized class of ion channels (for review, see Demidchik et al. 2002, Kronzucker and Britto 2011), and although usually discussed in the context of sodium (Na^+) into plant root cells under salinity (Munns and Tester 2008; see also Kronzucker and Britto 2011), ion-selectivity series have shown NSCCs to mediate fluxes of other cations, such as K^+, NH_4^+, Rb^+, Cs^+, Li^+ and tetraethylammonium (TEA^+), at rates broadly similar to those of Na^+ (Kronzucker and Britto 2011). In some cases, K^+:Na^+ selectivity ratios for NSCCs have been shown to reach 3:1 (Demidchik et al. 2002, Demidchik and Maathuis 2007), and given their ostensible capacity for high rates of transmembrane transport (albeit at very high Na^+ concentrations; e.g. Essah et al. 2003), it would follow that in-planta

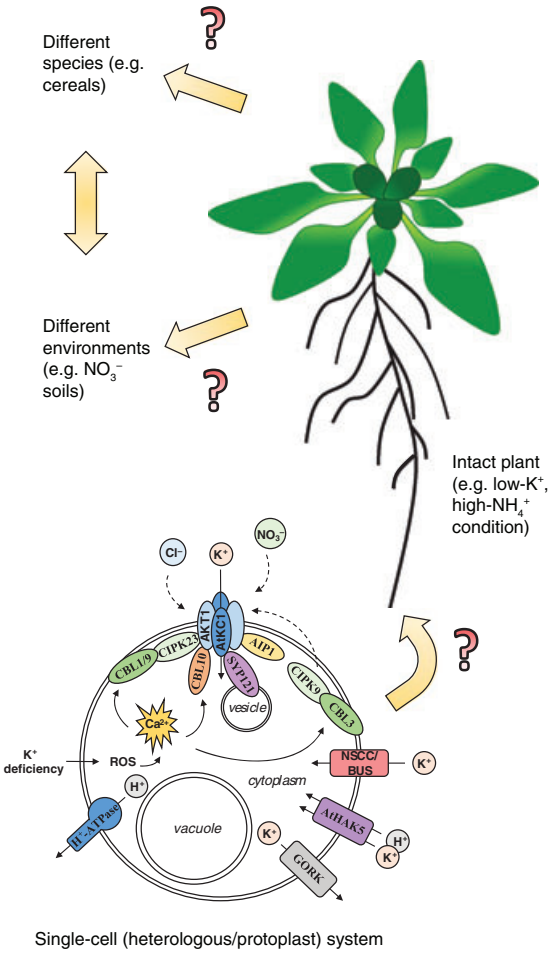

Fig. 1. Summary of cellular mechanisms related to K^+-channel function and regulation in roots of Arabidopsis and its relation to higher levels of organization. In single-cell (heterologous/protoplast) systems, the K^+-channel heterotetrameric complex AtAKT1-AtKC1 can mediate K^+ acquisition from low (micromolar) external concentrations ($[K^+]_{ext}$) and in the presence of NH_4^+. External K^+ deficiency is sensed by the cell and results in ROS production which leads to cellular Ca^{2+} signaling cascades. Ca^{2+} binds to CBL proteins to either directly modulate AtAKT1 (see CBL10) or activate protein kinases (CIPK23) that phosphorylate AtAKT1 and activate the channel complex. By contrast, the protein phosphatase AIP1 can dephosphorylate AtAKT1 and thereby inactivate the complex. CBL3-CIPK9 also regulates K^+ acquisition, although via indirect mechanisms. The SNARE protein SYP121 interacts with AtKC1 to activate the channel complex and also facilitates membrane trafficking and cell expansion via vesicular fusion. Anions (Cl^- and NO_3^-) may also modulate channel activity, albeit only at high (millimolar) $[K^+]_{ext}$. Other potential means of K^+ acquisition involve the non-selective cation channels (NSCCs), which may or may not represent the K^+ 'backup system' (BUS). Also, the high-affinity transporter AtHAK5 mediates H^+-coupled K^+ symport under conditions of thermodynamically active K^+ uptake (powered by the H^+-ATPase). Lastly, the K^+ channel GORK mediates cellular K^+ efflux under passive-release conditions. Importantly, in-planta links to many cellular-mechanistic characterizations require further exploration. Once such links are made, broader applications can be developed, such as with species (e.g. cereals) and to conditions relevant to agriculture.

NSCC-mediated K^+ transport should be measureable. However, our extensive recent pharmacological profiling of K^+ uptake in intact barley roots revealed no effect of well-established NSCC inhibitors, suggesting a lack of NSCC involvement (Coskun et al. 2013). Nevertheless, the role of NSCCs in K^+ acquisition remains an important avenue of research, particularly as it may pertain to the so-called 'backup system' [or systems ('BUS')] for K^+ transport, as recently described in studies using *athak5 atakt1* double-knock-out mutants in Arabidopsis (Pyo et al. 2010, Caballero et al. 2012, Coskun et al. 2013). This system was shown to rescue *athak5 atakt1* mutants at high $[K^+]_{ext}$ (>1 m*M*), although K^+ uptake at $[K^+]_{ext}$ as low as 22.5 μ*M* may also occur via this system (Coskun et al. 2013). It could furthermore be important in mediating K^+ uptake under conditions where both AtHAK5 and AtAKT1 are inhibited, such as under salinity and/or NH_4^+ stress (Britto and Kronzucker 2002, Kronzucker et al. 2013). Although no 'BUS' genes have yet been identified, cyclic-nucleotide-gated channels (CNGCs) have been implicated as potential candidates (Caballero et al. 2012; see also Kaplan et al. 2007), and this deserves increased attention.

Low-K^+ sensing and channel phosphorylation/dephosphorylation networks

Soil-K^+ depletion is an important environmental issue that may be linked to K^+ channels (Ashley et al. 2006), and an increasing number of molecular studies are providing mechanistic detail of K^+-channel functioning and regulation in Arabidopsis (for review, see Luan 2009, Wang and Wu 2013). This includes the identification of signaling cascades associated with low-K^+ sensing of the external environment and the subsequent phosphorylation/dephosphorylation networks associated with K_{in} channels (Fig. 1). These mechanistic characterizations have provided interesting insight into K^+-transport function and regulation at the molecular-cellular level, although the scope of many of these studies (e.g. Arabidopsis seedlings grown under low-K^+, high-NH_4^+ conditions) currently limits the general applicability of these findings, and the ecological and agronomic significance of the findings to low-K^+ soils await further testing at the whole-plant level.

The majority of contemporary studies investigating molecular aspects of K^+ transport rely, understandably, on the Arabidopsis model, by expressing Arabidopsis genes in heterologous systems or by analyzing Arabidopsis mutant lines. However, as mentioned above, channels do not appear to operate in-planta in the example of barley under steady-state conditions of low K^+ and high NH_4^+. Similarly, in tomato (*Solanum lycopersicum*), an NH_4^+-sensitive system, probably mediated by LeHAK5, dominates high-affinity K^+ uptake in the presence of NH_4^+, indicating that in this species, the contribution of the LKT1 channel (an AKT1 homolog) to K^+ acquisition under K^+-starved conditions may be low (Nieves-Cordones et al. 2007). Thus, the Arabidopsis model may not be as broadly applicable to other plant species as is often stated, or assumed. Also, low-K^+, high-NH_4^+ growth conditions are typical of many studies in this domain, as a means of isolating channel activity (Hirsch et al. 1998, Spalding et al. 1999); however, such conditions cannot speak to the involvement of channels under other growth conditions, such as low or moderate NH_4^+, or under NO_3^- supply, an often dominant N source in the environment, especially under agricultural conditions (Britto and Kronzucker 2013). For example, unpublished results from our lab suggest that channels also do not operate at $[K^+]_{ext}$ <100 μ*M* in NO_3^--grown barley. Moreover, as mentioned above, earlier work in NO_3^--grown Arabidopsis unequivocally demonstrated that K^+ uptake under low-K^+ conditions was a thermodynamically active process, and thus channels could not operate (Maathuis and Sanders 1993). Therefore, generally, caution is in order when extrapolating findings from studies that utilize Arabidopsis under narrow growth conditions to other conditions and other species, such as those relevant to agriculture (e.g. cereals; Fig. 1). Indeed, channels may not participate in K^+ acquisition in many settings outside the laboratory. Nevertheless, the mechanistic questions regarding channel involvement under low-K^+ conditions remain interesting and are worth exploring.

Although direct evidence of specific K^+ sensors located either in the plasma membrane or cytoplasm in roots of higher plants is currently lacking, a large body of work has begun to characterize the signaling cascades associated with low-$[K^+]_{ext}$ sensing and channel function in roots (for review, see Amtmann et al. 2006, Schachtman and Shin 2007, Wang and Wu 2013). One of the earliest signaling events under low-K^+ stress involves the production of reactive oxygen species (ROS) (Shin and Schachtman 2004, Shin et al. 2005). ROS production has been suggested to be an upstream regulator of Ca^{2+} signaling (Li et al. 2006, Lebaudy et al. 2007, Laohavisit et al. 2012), itself linked to Ca^{2+} binding to calcineurin-B-like protein1 (CBL1) and CBL9. These proteins, in turn, directly target CBL-Interacting protein kinase23 (CIPK23), forming a protein complex responsible for phosphorylating and activating AtAKT1-channel complexes (Li et al. 2006, Xu et al. 2006, Lee et al. 2007; see also Grefen and Blatt 2012), resulting in channel uptake from low $[K^+]_{ext}$ in roots of Arabidopsis. Moreover, a specific 2C-type

protein phosphatase (PP2C), AIP1 (for AKT1-interacting PP2C 1), has been identified and shown to physically interact with and inactivate AtAKT1 (Lee et al. 2007, Lan et al. 2011). Recently, new CBL/CIPK proteins identified to participate in K^+ acquisition under low-K^+ conditions have been discovered. For example, CBL10 has been shown to directly modulate AtAKT1 activity (Ren et al. 2013), while the CBL3–CIPK9 complex has been shown to affect K^+ transport under low-K^+ stress, although it does not appear to specifically interact with AtAKT1 or other key K^+ transporters in roots (Liu et al. 2013).

The intersection between the SYP121-KC1-AKT1 tripartite and cellular expansion

It has been shown that two K^+-channel subunits, AtKC1 (K_{silent}) and AtAKT1 (K_{in}), form a functional heterotetrameric K^+-channel complex in the plasma membrane of WT Arabidopsis root hairs (Reintanz et al. 2002). Without AtKC1 expression (i.e. in *atkc1* knock-out genotypes), altered gating sensitivity was observed in AtAKT1 homomers, resulting in an activation threshold shifted positive by 50–70 mV and in K^+ 'leakage' via the channels at membrane voltages (V_m) positive of the equilibrium potential (E_{K^+}) (Reintanz et al. 2002, Duby et al. 2008). These effects were linked to the impaired plant growth of *atkc1* mutants under low-K^+, high-NH_4^+ conditions, relative to WT (Geiger et al. 2009). Interestingly, in another study on AtKC1, AtKC1 knock-out genotypes showed enhanced tolerance to low-K^+, high-NH_4^+ conditions, in contradiction to the Geiger et al. study (Wang et al. 2010). This phenotype was attributed to the lack of AtKC1's restrictive regulation of AtAKT1, thus higher K^+ uptake was observed, resulting in higher tissue K^+ content and plant biomass. To our knowledge, this inconsistency has not been directly addressed; however, it may have to do with differences in knock-out constructs being used in each study. Nevertheless, this highlights an important caveat with respect to gene-disrupting technology and the significance of potential pleiotropic effects. It also points out the importance of thorough physiological examination. Because measurement of K^+ depletion and tissue K^+ content were only conducted by Wang et al., direct phenotypic comparisons between the studies could not be made. Moreover, as mentioned, such measurements are only indirect assessments of channel function (or lack thereof) and direct measures of influx in-planta should be conducted (see above). In another interesting case, AtKC1 knock-out lines displayed major growth (biomass) reductions under 'standard' (i.e. K^+-replete, NO_3^--supply) growth conditions after 8 weeks (Jeanguenin et al. 2011). It was concluded that loss of AtKC1 expression was more severe (with respect to biomass decline) than any individual knock-out of a single K_{in} or K_{weak} subunit (i.e. AKT1, AKT2, KAT1 or KAT2) under these conditions. This was explained in part by AtKC1's ability to indiscriminately bind to the aforementioned K^+-channel subunits. Thus, it is clear that AtKC1 is vital for proper K^+-channel function. What is not as clear is the ecophysiological relevance of these studies. Characterizing a mutant under narrow growth conditions can be rather limited in utility. For example, the AtAKT1-homomer-mediated K^+ 'leakage' was only observed in heterologous systems (Duby et al. 2008, Geiger et al. 2009). In fact, Arabidopsis *atakt1* knock-out lines display similar outwardly-directed K^+ currents in both protoplast (Hirsch et al. 1998) and in-planta (Shabala et al. 2006) systems, revealing the lack of AtAKT1's involvement in cellular K^+ efflux. Moreover, K^+ efflux in Arabidopsis root hairs has been clearly ascribed to the function of the K_{out} channel GORK (Ivashikina et al. 2001, Reintanz et al. 2002). This raises questions of the relevance of the 'leaky' AtAKT1 homomer outside of laboratory conditions.

Adding further complexity to AtAKT1's characterization is the realization that the soluble *N*-ethylmaleimide-sensitive factor protein attachment protein receptor (SNARE), SYP121, binds to AtKC1, forming a tripartite complex with AtAKT1, essential for proper K^+-channel functioning (Honsbein et al. 2009). SNAREs are a ubiquitous superfamily of proteins responsible for vesicle targeting and fusion (i.e. membrane trafficking; Grefen and Blatt 2008). Thus, it has been hypothesized that SYP121 is an important link between ion transport and membrane/cell expansion (Grefen and Blatt 2008). In support of this idea, it has been demonstrated that SYP121 is most highly expressed in cells that undergo significant expansions and growth, such as root hairs and root epidermal cells in the elongation zone (Grefen and Blatt 2008). Importantly, these are also regions of high K^+ uptake linked to channel activity (Lagarde et al. 1996, Hirsch et al. 1998, Reintanz et al. 2002). Moreover, as the predominant osmoticum in plant cells, K^+ contributes to turgor and cell expansion (Dolan and Davies 2004). Thus, understanding the mechanism behind membrane trafficking and K^+-channel function and its control will be critical to understanding cellular growth, proliferation, and stress response in the context of K^+ transport (Grefen et al. 2011). As with *atkc1* and *atakt1* (see above), *syp121* knock-out lines display suppressed K^+ currents in Arabidopsis root epidermal protoplasts and also suppress K^+ uptake and growth in seedlings under low-K^+, high-NH_4^+ conditions (Honsbein et al. 2009). Of course, this raises the question of SYP121's role under K^+-replete conditions, or with low/moderate-NH_4^+, or NO_3^- backgrounds, which

will require further exploration (see above), as will questions pertaining to the relevance of this mechanism under conditions of active K^+ uptake, where channels do not participate. As mentioned, channel-mediated K^+ uptake may only occur under very specific growth scenarios. What is the link between membrane trafficking and AtHAK5-mediated K^+ acquisition, for example? Qi et al. (2008) have provided evidence suggesting that K^+ deprivation affects the trafficking of AtHAK5 from the endoplasmic reticulum to the cell membrane. Whether a link to cell expansion exists here is yet to be determined, as are the molecular determinants of this regulation. Our understanding of the fascinating role of SNARE proteins at the intersection of ion transport and cellular expansion is in its infancy. Clearly, much remains to be discovered in this critical area.

Conclusion

In many ways, we come to the same conclusion arrived at two decades ago by Kochian and Lucas (1993): that K^+ channels cannot 'do it all' in terms of primary K^+ acquisition in roots of higher plants. In light of mounting molecular characterizations of K^+-channel function and regulation, particularly under conditions of 'low-K^+ stress', this assessment remains particularly relevant. Even so, we maintain that, in general, studies in this area would benefit from more robust in-planta demonstrations. It appears that channel-mediated K^+ acquisition under low (micromolar) K^+ supply is limited to conditions of high (millimolar) NH_4^+, and has only been demonstrated convincingly in Arabidopsis. Other species, such as barley, do not appear to engage channel activity under comparable conditions (Coskun et al. 2013). Only under recovery from NH_4^+ toxicity (i.e. NH_4^+ withdrawal) do channels become relevant to K^+ acquisition from a low-K^+ environment in the cereal. Moreover, under NO_3^- provision, K^+ channels do not appear to participate in such acquisition, even in Arabidopsis (Maathuis and Sanders 1993). Thus, we must be cautious of the generic utility of the Arabidopsis model, especially if the goal is to understand K^+ acquisition in roots of plants growing in K^+-deficient soils (Fig. 1).

Acknowledgements – Funding for this minireview was generously provided by the Natural Sciences and Engineering Research Council of Canada (NSERC) and the Canada Research Chair (CRC) program.

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Edited by J. K. Schjτrring