Potassium Transport and Signaling in Higher Plants

Yi Wang and Wei-Hua Wu

State Key Laboratory of Plant Physiology and Biochemistry, National Center of Plant Gene Research (Beijing), College of Biological Sciences, China Agricultural University, Beijing 100193, China; email: wuwh@public3.bta.net.cn

Annu. Rev. Plant Biol. 2013. 64:451–76

First published online as a Review in Advance on January 16, 2013

The *Annual Review of Plant Biology* is online at plant.annualreviews.org

This article's doi:
10.1146/annurev-arplant-050312-120153

Keywords

K^+ deficiency, K^+ channel, K^+ transporter, calcium, CIPK, ROS

Abstract

As one of the most important mineral nutrient elements, potassium (K^+) participates in many plant physiological processes and determines the yield and quality of crop production. In this review, we summarize K^+ signaling processes and K^+ transport regulation in higher plants, especially in plant responses to K^+-deficiency stress. Plants perceive external K^+ fluctuations and generate the initial K^+ signal in root cells. This signal is transduced into the cytoplasm and encoded as Ca^{2+} and reactive oxygen species signaling. K^+-deficiency-induced signals are subsequently decoded by cytoplasmic sensors, which regulate the downstream transcriptional and posttranslational responses. Eventually, plants produce a series of adaptive events in both physiological and morphological alterations that help them survive K^+ deficiency.

Contents

INTRODUCTION

Potassium (K^+) is the most important and abundant cation in living plant cells and plays crucial roles in many fundamental processes, including enzyme activation, membrane transport, anion neutralization, and osmoregulation (27). As one of the principal nutrient elements, it significantly affects multiple plant physiological processes, including photosynthesis and transport/translocation of assimilation products, and consequently affects overall plant growth and development. Owing to the limited availability of K^+ in soils, most plants living in the natural environment often suffer from K^+-deficiency stress. To adapt to K^+-deficient environments, plants have evolved a complex signaling and physiological regulatory network that helps them survive under K^+-deficiency stress (13, 62, 129, 146, 155). Molecular genetic studies of the model plant *Arabidopsis* and the availability of *Arabidopsis* genome sequences and knockout mutants have begun to shed light on the complex network underlying plant responses to K^+-deficiency stress.

K+ TRANSPORT COMPONENTS IN HIGHER PLANTS

As one of the major nutrient ions, K^+ is absorbed and accumulated in plant cells, and it constitutes 2–10% of plant dry weight (81). The cytoplasmic K^+ concentration ($[K^+]_{cyt}$) in plant cells is relatively stable at approximately 100 mM, which is the optimal K^+ concentration for cytoplasmic enzyme activities (81, 152). The K^+ accumulated in vacuoles is the largest K^+

pool in plant cells; the vacuolar K^+ concentration is variable (between 10 mM and 200 mM) depending on the K^+ supply and tissue type (81, 152). Compared with the high K^+ concentration in plant cells, the typical concentration at the surfaces of roots in the soil is extremely low, varying from 0.1 to 1 mM (92, 133). Plant root cells therefore absorb K^+ from the soil against the K^+ concentration gradient, a process conducted by plant K^+ transport components such as K^+ transporters and channels. K^+ translocation from roots to shoots or from source to sink inside plants is also mediated by K^+ transporters and channels.

Classification of K^+ Transport Components

Early investigations indicated that K^+ uptake in higher plants shows typical dual-affinity (high and low) mechanisms, which perform at different external K^+ concentrations ($[K^+]_{ext}$) and alternate depending on the external K^+ supply (39). The high-affinity K^+ uptake mechanism is mediated primarily by K^+ transporters at low $[K^+]_{ext}$ (below 0.2 mM) (39, 94); the low-affinity K^+ uptake mechanism is mediated primarily by K^+ channels at relatively high $[K^+]_{ext}$ (above 0.3 mM) (39, 133). It seems that plant cells can perceive the $[K^+]_{ext}$ and employ the appropriate K^+ uptake components for a given concentration.

In many higher-plant species, a large number of genes encoding K^+ transporters and channels have been cloned and identified (97, 149, 157). These transporters and channels differ in their energetic coupling, affinity, and selectivity for K^+ and constitute the complicated K^+ transport system in plant cells.

The genes from three families—*Shaker*, *TPK* (*tandem-pore* K^+), and *Kir* (K^+ *inward rectifier*)*–like*—encode K^+ channel proteins in higher plants (**Table 1**) (45, 79, 149). The plant Shaker K^+ channels have a protein structure similar to that of animal Shaker K^+ channels initially cloned from *Drosophila*. Each Shaker K^+ channel subunit harbors six transmembrane segments and one pore loop, and four subunits form a whole K^+ channel complex (**Table 1**). Plant Shaker channels exhibit remarkable K^+ selectivity and voltage sensitivity. They are divided into three categories based on their voltage dependence: Inward-rectifying channels are activated by hyperpolarization potential and mainly mediate K^+ uptake, outward-rectifying channels are activated by depolarization and function in K^+ efflux, and weakly rectifying channels could be activated by hyperpolarization and may mediate both K^+ uptake and K^+ release depending on the membrane potentials.

Plant TPK and Kir-like channels were previously classified as KCO channels but are now considered two distinct families on the basis of their different channel structures. TPK channels possess four transmembrane segments and two pore-loop domains in tandem (**Table 1**), similar to their animal counterpart KCNK channels. Kir-like channels are related to animal Kir channels and possess two transmembrane segments and one pore-loop domain. Unlike Shaker channels, there seems to be no voltage sensor for TPK and Kir-like channels, implying their weak sensitivity to membrane potentials. But some TPK and Kir-like channels possess EF hand domains (putative Ca^{2+}-binding sites) located in the cytosolic C-terminal region, suggesting their sensitivity to Ca^{2+} (79).

The plant K^+ transporters derive mainly from several gene families, including *KUP/HAK/KT*, *HKT*, *NHX*, and *CHX* (50). The transporters encoded by the *KUP/HAK/KT* family are homologs of the bacterial K^+ transporter KUP and fungal K^+ transporter HAK1. Some of the KUP/HAK/KT transporters have been verified as performing K^+ transport activities in plant cells (**Table 1**) (38, 41, 51, 70, 123). The analogs of HKT transporters in fungi (Trk transporters) and bacteria (KtrB transporters) function as K^+ symporters coupling with H^+ or Na^+. However, studies in plants have indicated that most plant HKT transporters function as Na^+ transporters (50), and only a few are Na^+/K^+ symporters (46, 124). Most

Table 1 K^+ channels and transporters in *Arabidopsis*

Family/name	Protein structure	Organ(s)/tissue(s)[a]	Functions	Reference(s)
Shaker				
AKT1	6 TMSs, 1 P-loop, CNBD, Anky, KHA	Root (root hairs, epidermis, cortex), leaf (primordia, mesophyll, hydathodes, guard cells)	Inward-rectifying K^+ channel, K^+ uptake into root cells	51, 60, 76, 80, 84, 117, 134, 141, 167
AtKC1		Root (root hairs, epidermis, cortex), leaf (epidermis, hydathodes, trichome)	Silent K^+ channel, assembly with Shaker inward K^+ channels and regulation of K^+ uptake into cells	35, 36, 48, 67, 122, 154
SPIK		Pollen (pollen, pollen tubes)	Inward-rectifying K^+ channel, K^+ uptake into pollen tubes, pollen tube development regulation	102
AKT2		Root (phloem), stem, leaf (phloem, mesophyll, epidermis, guard cells), flower (sepal)	Weakly rectifying K^+ channel, K^+ circulation in phloem	26, 44, 75, 95, 99
KAT1		Leaf (guard cells)	Inward-rectifying K^+ channel, K^+ uptake into guard cells, stomatal regulation	8, 74, 128, 140, 142, 144
KAT2		Leaf (guard cells, phloem)	Inward-rectifying K^+ channel, K^+ uptake into guard cells, stomatal regulation	115, 144
SKOR		Root (stellar tissues), pollen	Outward-rectifying K^+ channel, K^+ release into xylem, K^+ translocation from roots to shoots	47
GORK		Root (root hairs, epidermis), leaf (guard cells)	Outward-rectifying K^+ channel, K^+ release from guard cells, stomatal regulation	1, 66
TPK				
TPK1	4 TMSs, 2 P-loops, EF hand, 14-3-3 binding	Root (root tip, elongation zone), leaf (mesophyll, guard cells), flower (sepals, anthers, pollen)	Vacuolar K^+ channel, K^+ release from vacuole, stomatal closure, intracellular K^+ homeostasis	28, 52, 78, 151
TPK4		Pollen (pollen, pollen tubes)	PM K^+ channel, control of pollen PM voltage	18, 151
KUP/HAK/KT				
KUP1	10–14 TMSs	Stem, leaf, flower	Dual-affinity K^+ transporter, K^+ uptake into cells	41, 70
KUP2		Root (root tip), stem (hypocotyls, inflorescence stem), leaf (cotyledons, young leaves)	Low-affinity K^+ transporter, K^+-dependent cell expansion	38, 41
KUP4		Root, stem, leaf, flower	High-affinity K^+ transporter, K^+ translocation and root hair elongation	41, 123, 150
HAK5		Root (epidermis, stele)	High-affinity K^+ transporter, K^+ uptake into root cells	51, 117, 118

(*Continued*)

Table 1 (*Continued*)

Family/name	Protein structure	Organ(s)/tissue(s)[a]	Functions	Reference(s)
NHX				
NHX1/NHX2	10–12 TMSs	Stem (cortical tissue), leaf (vascular tissue, guard cells), flower (petals, stamens, anthers), silique (developing embryo)	Vacuolar $Na^+(K^+)/H^+$ antiporter, vacuolar pH and K^+ homeostasis, turgor regulation, plant growth, flower development, stomatal function	10, 15, 16, 147
CHX				
CHX13	10–12 TMSs	Root, flower, pollen	High-affinity K^+ transporter, K^+ uptake into root cells	170
CHX17		Root (epidermis, cortical cells)	$Na^+(K^+)/H^+$ antiporter, K^+ uptake into root cells and K^+ homeostasis	24
CHX20		Leaf (guard cells)	$Na^+(K^+)/H^+$ antiporter, K^+ homeostasis and pH regulation, guard cell osmoregulation	107
CHX21		Pollen (pollen, pollen tubes)	$Na^+(K^+)/H^+$ antiporter, pollen tube targeting to ovules	88
CHX23		Root, stem, leaf (cotyledons), flower (sepals), pollen	$Na^+(K^+)/H^+$ antiporter, pH homeostasis and chloroplast development, pollen tube targeting to ovules	88, 139

Information adapted from References 45, 50, 53, 79, and 149. Abbreviations: 14-3-3 binding, 14-3-3 protein binding domain; Anky, ankyrin domain; CNBD, putative cyclic nucleotide-binding domain; EF hand, EF hand domain; KHA, domain rich in hydrophobic and acidic residues; P-loop, pore-loop domain; PM, plasma membrane; TMS, transmembrane segment domain.

[a]The expression profiles of the K^+ channels and transporters in different organs, tissues, or cell types.

members of the NHX and CHX families have been identified as Na^+/H^+ antiporters, and a few are K^+/H^+ antiporters (**Table 1**) (15, 16, 24, 88, 107, 139, 170).

Physiological Functions of K^+ Transporters and Channels

In *Arabidopsis*, Shaker K^+ channel AKT1 and KUP/HAK/KT transporter HAK5 are expressed primarily in the roots and function in K^+ uptake from the external environment (51, 60, 76). These two transport proteins mediate almost all K^+ absorption in *Arabidopsis* roots (51, 117, 141). For K^+ translocation between different organs or tissues inside a plant, K^+ secretion from root cortex cells into the xylem is mediated by the outward-rectifying channels, such as SKOR (47, 159); K^+ loading and unloading in phloem tissues are facilitated mainly by the weakly rectifying K^+ channels, such as AKT2 (44, 75, 95).

Stomatal guard cells and pollen cells are regarded as two special types of cells that possess unique K^+ transport components (63, 98, 110, 138, 157). Both inward and outward K^+ channels were initially identified in stomatal guard cells, mediating K^+ uptake and release, respectively (65, 131, 132). Further functional characterization showed that the inward K^+ channels KAT1 and KAT2 control the K^+ influx across the plasma membrane (PM) during stomatal opening (8, 74, 115, 144), and the outward K^+ channel GORK conducts the K^+ efflux during stomatal closure (1, 66). In pollen and pollen tube cells, the pollen-specific Shaker channel

SPIK mediates K^+ influx across the PM, which regulates pollen tube growth and development (102). Another TPK channel, TPK4, is also pollen specific and controls the membrane potential across the pollen PM during pollen tube growth (18).

Inside plant cells, the K^+ ions could be distributed and recycled between the cytoplasm and vacuole (K^+ pool). The voltage-independent K^+-selective channels were initially identified at the tonoplast of stomatal guard cells, and they are crucial for vacuolar K^+ release during stomatal closure (158). Further functional characterization showed that the tonoplast-located TPK1 functions in K^+ transport across the vacuolar membrane and plays a role in intracellular K^+ homeostasis, which is important for several physiological activities, such as stomatal movement and seed germination (28, 52). Other TPK channels (TPK2, TPK3, and TPK5) are also targeted to the tonoplast (151), but their physiological functions remain unknown.

K^+ transporters also participate in intracellular K^+ compartmentation in plant cells. The two major tonoplast-located NHX members, NHX1 and NHX2, are highly expressed in leaves (10, 15, 16). These two Na^+,K^+/H^+ antiporters mediate K^+ uptake into the vacuole, which in *Arabidopsis* is essential for K^+ pool creation, flower development, and stomatal movement (15, 16, 147).

K^+-DEFICIENCY-SENSING MECHANISMS

Compared with the high $[K^+]_{cyt}$ (~100 mM), the typical $[K^+]_{ext}$ in soil is extremely low (0.1–1 mM) (92, 133). Although plants cannot move to avoid this environmental stress, they have evolved K^+-deficiency-sensing and subsequent adaptive mechanisms that help them survive under K^+-deficiency stress. Plant roots are the main organs that directly contact the external medium or soils, and therefore the K^+-deficiency sensing is initiated mainly in root cells, especially in the epidermis and root hairs. The K^+-deficiency signal is first perceived at the PM of root epidermal cells and then transduced into the cytoplasm. After perceiving the signal, plant cells undergo a series of biochemical and physiological reactions that include both short- and long-term responses (129). The short-term responses occur within a few hours and involve several signal components, including the membrane potential, reactive oxygen species (ROS), and phytohormones (93, 137). During the early sensing period, the $[K^+]_{cyt}$ may be not affected even though the $[K^+]_{ext}$ is low (129). As the K^+-deficiency stress continues over days or weeks, the $[K^+]_{cyt}$ is significantly reduced and the long-term responses are then initiated (129), which results in metabolic (7, 12) and morphological (62) changes.

Hyperpolarization of the Membrane Potential

The membrane potential across the PM in living plant cells is the main driving force that energizes the K^+ absorption from soils against the K^+ concentration gradient. The membrane potential is dependent on $[K^+]_{ext}$ (93) and is established and maintained primarily by H^+-ATPase activity (108). The root cell membrane potential is highly specific to $[K^+]_{ext}$ and displays a linear relationship with $\log[K^+]_{ext}$ (93). The membrane potential is hyperpolarized by low $[K^+]_{ext}$ (93, 105, 130) and depolarized by high $[K^+]_{ext}$ (141), and the change in membrane potential may act as one of the sensing signals in plant responses to K^+ deficiency. The earliest detected event in plant responses to K^+ deficiency is the hyperpolarization of the cell membrane potential, which appears within a few minutes of a decrease in $[K^+]_{ext}$ (**Figure 1**) (93, 105, 130).

Membrane hyperpolarization caused by a decrease in $[K^+]_{ext}$ would obviously affect the activities of voltage-gated K^+ channels. Because they are activated by hyperpolarization, the Shaker AKT1-like channels are considered the main channel components that mediate K^+ influx into root cells in many plant species; these channels include AKT1 in *Arabidopsis* (60, 76), OsAKT1 in rice (42), ZMK1 in maize (113),

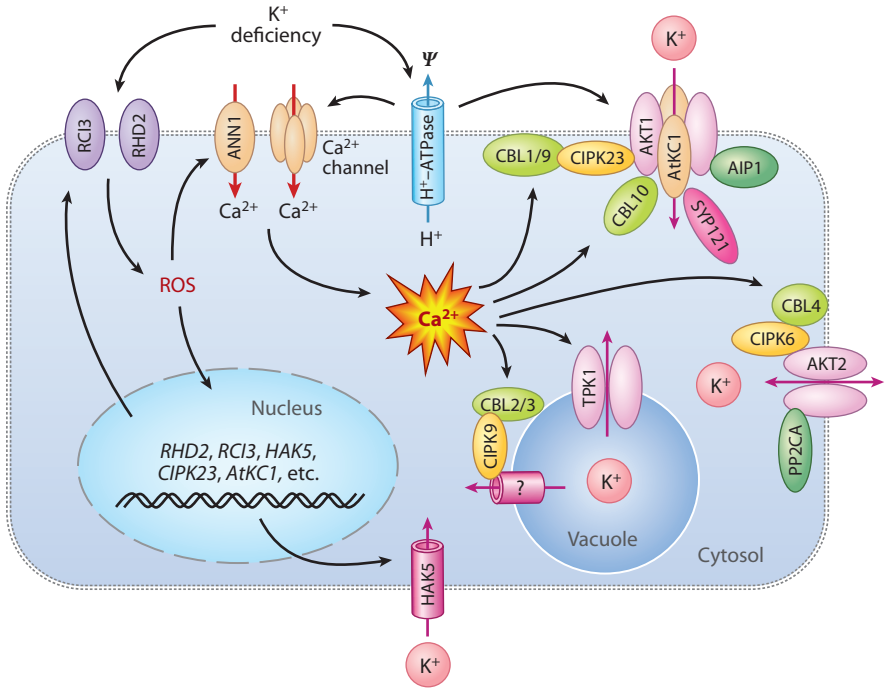

Figure 1

K^+ transport and signaling pathways in *Arabidopsis* responses to K^+ deficiency. The perception of K^+-deficiency stress leads to enhanced H^+-ATPase (most likely AHA2) activity, which then results in plasma membrane (PM) hyperpolarization (93, 105) and extracellular acidification (19). The hyperpolarized membrane potential (ψ) activates the inward K^+ channel AKT1 and the Ca^{2+}-permeable channels. Meanwhile, the extracellular acidification energizes the K^+ uptake mediated by a K^+ transporter such as HAK5. K^+ deficiency also induces ROS accumulation in root cells via RHD2 (137) and RCI3 (71), which further regulates the transcription of K^+-deficiency-responsive genes such as *HAK5* (71, 72). The elevated ROS also activates the PM-located Ca^{2+}-permeable ANN1 channels that mediate Ca^{2+} flux into the cytoplasm (77). The activation of Ca^{2+}-permeable channels by membrane hyperpolarization and ROS results in the elevation of cytoplasmic Ca^{2+}, and this Ca^{2+} signal can be perceived and transduced downstream by specific Ca^{2+} sensors such as CBL proteins. At the PM, CBL1 (and/or CBL9) recruits the cytoplasm-located kinase CIPK23 to the PM, where CIPK23 activates AKT1-mediated K^+ uptake via phosphorylation (84, 167). AKT1 channel activity can also be regulated by CBL10 (122a), AIP1 (a protein phosphatase) (80), AtKC1 (a K^+ channel subunit) (36, 48, 154), and SYP121 (a SNARE protein) (64). The CBL4-CIPK6 complex enhances AKT2-mediated K^+ flux by facilitating the translocation of the AKT2 channel from the endoplasmic reticulum to the PM in a kinase-interaction-dependent but phosphorylation-independent way (58). At the tonoplast, CBL3 (or CBL2) interacts with CIPK9 and activates an unknown tonoplast-located K^+ channel or transporter, facilitating K^+ release from the vacuole into the cytoplasm (87, 109). In addition, the tonoplast channel TPK1 mediates the K^+ efflux from the vacuole into the cytoplasm.

TaAKT1 in wheat (22), LKT1 in tomato (56), and SKT1 in potato (172). These channels contain voltage sensors and can perceive changes in membrane potential, and are activated under hyperpolarization conditions. The direction of K^+ flux across these channels depends on the K^+ equilibrium potential (E_K) and actual membrane potential (E_m). When E_m is more negative than E_K, K^+ influx across these channels occurs; otherwise, the cytoplasmic K^+ effluxes out of the cells (48, 57). To ensure net K^+ absorption, root cells must establish a more negative E_m to

provide enough driving force for K^+ influx and avoid K^+ efflux under K^+-deficient conditions.

Extracellular Acidification

Many KUP/HAK/KT transporters from different plant species have been reported as high-affinity K^+ transporters involved in K^+ uptake under K^+-deficient conditions (**Table 1**). For example, HAK5 contributes approximately half the K^+ absorption in *Arabidopsis* roots under K^+-deficient conditions (51, 117, 141). The members of this family had been proposed to function as K^+/H^+ symporters (53), although this needs to be further confirmed by biophysical analyses. Thus, the establishment of the transmembrane proton gradient is essential for KUP/HAK/KT transporter activity. The H^+-ATPases are the main contributors that generate not only membrane potential but also the transmembrane proton gradient (108). K^+ deficiency could enhance the activity of H^+-ATPase, most likely AHA2 (33, 55), leading to H^+ extrusion and extracellular acidification (**Figure 1**) (19). The K^+-deficiency-induced PM hyperpolarization activates K^+ channels, which may help plants to absorb as much K^+ as they do under K^+-deficient conditions. The K^+-deficiency-caused extracellular acidification may simultaneously energize the transporter-mediated K^+ uptake, and the external acidic conditions could enhance the K^+ uptake activities of AKT1-like channels (42, 56, 113). The H^+-ATPases are clearly important components in the initial sensing of plant responses to K^+ deficiency.

Plasma Membrane–Located K^+ Sensors

Because plant roots are the sites of initial K^+-deficiency sensing, it is reasonable to expect their cells to contain sensors for K^+-deficiency signals. These K^+ sensors could plausibly be located at the PMs of epidermal cells or root hairs and have the capacity to sense changes in external K^+ concentration. Unfortunately, no K^+ sensor has yet been identified at the molecular level.

The identification of a nitrate (NO_3^-) sensor in *Arabidopsis*, however, may provide some clues for K^+ sensor identification. The NO_3^- transporter CHL1 shows a dual-affinity binding property to NO_3^- ions and functions as an NO_3^- sensor in *Arabidopsis* roots (61). The previous data had indicated that the inward K^+ channels may function as the K^+ sensor when the $[K^+]_{ext}$ is reduced (130). Therefore, the K^+ transporter AKT1 could plausibly act as a K^+ sensor in *Arabidopsis* roots. First, AKT1 is the major inward K^+ channel expressed primarily in *Arabidopsis* roots and located at the PM of epidermal cells (76, 134). Second, AKT1-mediated K^+ uptake in *Arabidopsis* roots shows a typical dual-affinity character. AKT1 functions not only at the low-affinity range but also at the high-affinity range, even when the $[K^+]_{ext}$ is decreased to 10 μM (60, 141). AKT1 can perceive $[K^+]_{ext}$ fluctuations and shift its uptake kinetic phases in a $[K^+]_{ext}$-dependent manner. This switch between two uptake phases may also depend on the AKT1 phosphorylation status, which is controlled by the protein kinase CIPK23 (84, 167). Similarly, the NO_3^- sensing also relies on the phosphorylation status of the CHL1 protein (61).

Other evidence might also support the hypothesis that the AKT1 inward K^+ channel acts as a K^+ sensor. The initial event of plant responses to K^+ deficiency is membrane potential hyperpolarization in epidermal cells, as discussed above (60, 141), but this hyperpolarization completely disappears in *akt1* mutant plants (60, 141). Furthermore, investigations using *Xenopus* oocytes have shown that the membrane potentials shift significantly in the negative direction (hyperpolarization) in the oocytes expressing AKT1 channels when the $[K^+]_{ext}$ decreases to micromolar concentrations (154). These results suggest that AKT1 may sense the changes in $[K^+]_{ext}$ and subsequently affect the membrane potential controlled by PM-located H^+-ATPases, most likely AHA2 (33, 55). It seems that a communication mechanism may exist between AKT1 and

H^+-ATPases. Recent reports have shown that K^+ ions could bind to the C terminus of the H^+-ATPases and act as an intrinsic negative regulator of H^+-ATPases by uncoupling ATP hydrolysis activity and proton pumping activity (21). One may further hypothesize that K^+ deficiency impairs AKT1-mediated K^+ uptake and causes the subsequent decrease in $[K^+]_{cyt}$, and that the decrease in $[K^+]_{cyt}$ near the PM may enhance H^+-ATPase activation. As a result, the membrane is hyperpolarized and the K^+ channel or transporter-mediated K^+ uptake is enhanced.

Cytoplasmic K^+ Sensors

At the early stage of plant responses to K^+ deficiency, plant cells may mobilize K^+ ions from the vacuole (K^+ pool) into the cytoplasm to compensate for the decreased K^+ uptake from the external medium (152, 164). The $[K^+]_{cyt}$ is therefore not significantly reduced in the short term. In the longer term, the $[K^+]_{cyt}$ gradually decreases, ultimately affecting physiological processes in plant cells. The activities of many enzymes in plant cells require K^+ ions as a cofactor (165). Thus, these K^+-activated enzymes may act as K^+ sensors in the cytoplasm. Among these enzymes, pyruvate kinase is considered the most likely candidate (5, 12). This kinase is highly sensitive to cytoplasmic K^+ (121), and its activity can be directly inhibited by low $[K^+]_{cyt}$ in root cells after plants are subjected to long-term K^+-deficiency stress (12). This idea is supported by the findings that the cytoplasmic pyruvate content in root cells is significantly reduced under K^+-deficient conditions and is restored after K^+ resupply (7, 12). The decrease in pyruvate content leads to glycolysis inhibition and subsequently affects many metabolic pathways (such as the TCA cycle and GS/GOGAT/GDH cycle) (12) by which the K^+-deficiency signals are transduced to downstream metabolic processes.

Ca^{2+} AND ROS SIGNALING

Cytoplasmic Ca^{2+} is the most important second messenger in living plant cells in the responses to various biotic and abiotic stresses (125, 161). $[Ca^{2+}]_{cyt}$ elevation is considered the universal response of plants under different stresses. In addition, a particular and unique Ca^{2+} signal called the Ca^{2+} signature—which refers not only to a different $[Ca^{2+}]_{cyt}$ but also to a distinct spatial and temporal pattern—may be elicited under a particular stress (59, 125, 161). ROS, as another important signal component, is also involved in plant stress responses (9, 101) and plays a key role in generating the cytoplasmic Ca^{2+} signature (31, 101). These diverse Ca^{2+} signatures elicited by different stresses have been proposed to be subsequently decoded by various cytoplasmic Ca^{2+} sensors, which trigger the downstream responses such as protein modification and transcriptional regulation (20, 163).

Ca^{2+} Influx Under K^+-Deficient Conditions

Ca^{2+} sensors are involved in K^+ uptake in *Arabidopsis* roots, especially under K^+-deficient conditions (84, 167), which suggests that plant cells may require a specific Ca^{2+} signal for K^+-deficiency stress. The elevation of $[Ca^{2+}]_{cyt}$ induced by low $[K^+]_{ext}$ has been reported in guard cells (4), and recent experiments have shown that K^+ deficiency in the external medium could also induce $[Ca^{2+}]_{cyt}$ elevation in *Arabidopsis* root cells (**Figure 1**) (L.K. Shen, Y. Wang & W.H. Wu, unpublished data), although this K^+-deficiency Ca^{2+} signal needs to be further characterized.

Ca^{2+} signals can be generated by Ca^{2+} flux across the PM and/or intracellular membranes mediated by Ca^{2+}-permeable channels (59, 161). Most of the Ca^{2+}-permeable channels in plant cells are considered nonselective cation channels (59). In addition to TPC1 (a two-pore channel), the Ca^{2+}-permeable channels in *Arabidopsis* may be encoded by 40 candidate genes derived from two gene families: *CNGC* (*cyclic nucleotide gated channel*) and *GLR* (*glutamate receptor channel*). Some of these channels have been functionally characterized as Ca^{2+}-permeable channels (40, 83, 100, 112).

However, the mechanisms by which these channels function to generate specific Ca^{2+} signals remain unclear.

Many nonselective cation channels are activated under a hyperpolarized membrane potential and are therefore called hyperpolarization-activated Ca^{2+} channels (31, 59). Some of these channels have been identified in plant root epidermal cells as well as at root hair tips (30, 148). They may be activated by the hyperpolarized membrane potential induced by K^+-deficiency stress. The Ca^{2+} influx mediated by these channels may subsequently generate the specific Ca^{2+} signal for K^+-deficiency stress (**Figure 1**). The increased cytoplasmic Ca^{2+} could further activate the endomembrane nonselective cation channels and lead to Ca^{2+} release from the intracellular Ca^{2+} pool. For example, AtTPC1, as a vacuole-located SV (slow vacuolar)–type channel, could also mediate Ca^{2+} release from the vacuole into the cytoplasm (112). Functional characterization of these Ca^{2+}-permeable channels to describe their roles in K^+-deficiency Ca^{2+} signaling will clearly be an important area in the study of plant responses to K^+ deficiency.

ROS Signaling

ROS, as an important signal component, plays crucial roles in plant stress response, development, pathogen reaction, and many other physiological processes (9, 101). Nutrient deprivation—including deficiencies in K^+, nitrogen, phosphorus, and sulfur—could also induce ROS signals in plant root cells (129, 136, 137). The K^+-deficiency-induced ROS signal, which in *Arabidopsis* occurs in the discrete region of the root just behind the elongation zone, can be detected 6 h after K^+ deficiency begins and increases within 30 h (**Figure 1**) (136, 137). It has been reported that ROS generation is mediated by various oxidases as well as peroxidases. A recent study showed that RCI3, a peroxidase from the type III peroxidase family, is involved in ROS generation in *Arabidopsis* root responses to K^+ deficiency (**Figure 1**) (71). The RCI3-mediated ROS production is also essential for the induction of *HAK5* gene expression under K^+-deficient conditions (71).

ROS is also important for Ca^{2+} signal generation in plant cells because it activates Ca^{2+}-permeable channels (31). In plant root cells, PM-located NADPH oxidase produces extracellular ROS, and these additional ROS molecules directly activate PM-located Ca^{2+}-permeable channels (ROS-activated channels) that mediate Ca^{2+} flux into the cytoplasm (**Figure 1**) (32, 111). One of the ROS-activated channels was recently identified at the molecular level in *Arabidopsis* root cells (77). The membrane protein annexin (ANN1) could form a Ca^{2+}-permeable channel at the PMs of root hairs and epidermal cells (**Figure 1**). The loss-of-function mutant for *ANN1*, *Atann1*, lacks the hydroxyl radical–activated Ca^{2+}-permeable conductance, and so $[Ca^{2+}]_{cyt}$ elevation is impaired (77).

Ca^{2+} Sensors

It is believed that Ca^{2+} signals should be perceived by Ca^{2+} sensors, decoded, and further transduced. Obviously, there must be different Ca^{2+} sensors in the cytoplasm of plant cells so that the different Ca^{2+} signals can be recognized. In higher plants, the proposed Ca^{2+} sensors are derived mainly from four gene families: *CaM*, *CML* (*CaM-like protein*) (171), *CBL* (*calcineurin B–like protein*) (73), and *CDPK* (*Ca^{2+}-dependent protein kinase*) (54), which collectively comprise approximately 100 genes in the *Arabidopsis* genome (20). The plethora of Ca^{2+} sensors fulfills the need for specific sensing of diverse Ca^{2+} signals. Many of these Ca^{2+}-binding proteins (Ca^{2+} sensors) participate in plant responses to environmental stresses such as high salt, drought, cold, and heat (20). Some members of the CBL protein family have been reported to function in plant responses to K^+ deficiency (84, 167).

CBL proteins are similar to both regulatory calcineurin B subunits and neuronal Ca^{2+} sensors in animals (90). They have been identified only in higher plants, suggesting their specific roles in plant signaling processes

(73). Ten CBL isoforms have been identified in *Arabidopsis*, and they display high amino acid sequence identity, ranging from 20% to 90% (73), which may result in functional redundancy among close members. Each CBL protein contains four EF hand domains that endow CBLs with Ca^{2+}-binding capabilities (104). The first identified CBL proteins involved in K^+-deficiency response were CBL1 and CBL9, which participate in the positive regulation of AKT1-mediated K^+ uptake in *Arabidopsis* roots under K^+-deficient conditions (**Figure 1**) (84, 167). Recent data have shown that CBL10 could also regulate AKT1 channel activity but displays a negative effect on AKT1-mediated K^+ uptake (**Figure 1**) (122a). CBL4 is involved in the regulation of AKT2 traffic from the endoplasmic reticulum to the PM (**Figure 1**) (58). Moreover, CBL2 and CBL3 may function in the regulation of K^+ transport between the vacuole and cytoplasm (**Figure 1**) (87). Although these Ca^{2+} sensors have been shown to play a role in the regulation of K^+ nutrition, the mechanisms for how they respond to specific Ca^{2+} signals remain unclear.

POSTTRANSLATIONAL REGULATION OF K^+ CHANNELS

After perceiving K^+-deficiency signals, plants initiate a series of signaling events to modulate biochemical and physiological processes. The accumulated experimental evidence of the past decade has revealed that K^+ channels are modulated at the posttranslational level under K^+-deficiency stress. Modulation of K^+ channel proteins may result in enhancement of K^+ uptake from the environment or mobilization of K^+ ions from the K^+ pool (vacuoles), which may help plants adapt to K^+-deficiency stress.

CBL-CIPK Complexes

The CBL proteins are considered major Ca^{2+} sensors involved in the perception and transduction of K^+-deficiency-induced Ca^{2+} signals in plant cells (84, 167). Many studies have demonstrated that they interact exclusively with a group of serine/threonine kinases called CIPK (CBL-interacting protein kinase) proteins, which are similar to SNF (sucrose nonfermenting) protein kinases in yeast and animals (3, 135, 160). The interaction between CBLs and CIPKs requires the presence of Ca^{2+} ions (135). By forming CBL-CIPK complexes, the Ca^{2+} signals are transduced to downstream target proteins by CIPK-facilitated phosphorylation. In *Arabidopsis*, 10 CBLs and 26 CIPKs constitute a diverse and complicated interacting network for the plant Ca^{2+} signaling system (3, 89, 160). Owing to the specificity and overlap of CBL-CIPK interactions, CBLs and CIPKs display broad functional redundancy (3). Several CBL-CIPK signaling pathways function in plant responses to various abiotic stresses, such as excess salt, high or low pH, and nutrient deprivation (43, 61, 69, 84, 119, 120, 167).

Xu et al. (167) showed that the CBL-CIPK complex participates in the regulation of plant K^+ uptake under K^+-deficiency stress. CBL1 and/or CBL9 interact with CIPK23 and recruit it to the root cell PM, where it phosphorylates the K^+ channel AKT1 (**Figure 1**) (84, 167). *CIPK23* transcription can be induced by K^+-deficiency stress (**Figure 1**) (167), suggesting that CIPK23 activity and subsequent AKT1 phosphorylation could be enhanced. Eventually, K^+ uptake mediated by AKT1 channels is enhanced under K^+-deficient conditions (167). Another CBL protein, CBL10, was recently identified as a negative regulator of the AKT1 channel (**Figure 1**) (122a). CBL10 interacts directly with the AKT1 channel and inhibits AKT1-mediated K^+ flux into the cytoplasm. This finding suggests a novel regulatory mechanism of CBL proteins—that they may directly regulate target proteins in a CIPK-independent manner. The CBL4-CIPK6 complex was also found to be involved in the regulation of another Shaker K^+ channel, AKT2, although the phosphorylation of AKT2 by CIPK6 was not observed (58). The CBL4-CIPK6 complex enhances AKT2-mediated K^+ currents by facilitating the translocation of AKT2 channels from the endoplasmic reticulum to the PM in a kinase-interaction-dependent but

phosphorylation-independent way (**Figure 1**) (58).

Recent experimental results have shown that another CBL-CIPK complex, CBL3-CIPK9, functions in the tonoplast and may regulate K^+ transport between the cytoplasm and the vacuole (**Figure 1**) (87). CIPK9 is the CIPK family member closest to CIPK23, and *CIPK9* loss of function results in a phenotype tolerant of K^+-deficient conditions (87); the overexpressing line of *CIPK9*, in contrast, is sensitive to K^+-deficiency stress. These results suggest that CBL3-CIPK9 may modulate a tonoplast-located K^+ channel or transporter and act as a negative regulator to control K^+ transport from the vacuole to the cytoplasm, especially under K^+-deficient conditions (87, 109).

Most of these CBL-CIPK pathways regulate the activities of ion channels or transporters at the PM or tonoplast. Localization analysis has indicated that most of the CBL proteins target to the PM (CBL1/4/5/9/10) or tonoplast (CBL2/3/6), with the exceptions of CBL7 and CBL8 (17). The CIPK proteins, in contrast, do not show specific localization, and most of them exhibit cytoplasmic and nucleoplasmic localization (17). CBLs seem to play a crucial role in determining the specific subcellular localization of CBL-CIPK complexes. By interacting with CBLs, CIPK proteins may be recruited to different membranes. It is plausible that more ion channels or transporters are being regulated by CBL-CIPK complexes and that CBL-CIPK-mediated signaling mechanisms play a crucial role in ion homeostasis in plant cells.

Phosphorylation and Dephosphorylation of K^+ Channels

More and more evidence supports the idea that the activities of plant ion channels and transporters are significantly regulated by phosphorylation (49, 61, 84, 120, 167) and dephosphorylation (26, 80). In *Arabidopsis*, the AKT1 channel is phosphorylated by the serine/threonine kinase CIPK23, which enhances the K^+ uptake activity of AKT1 under K^+-deficient conditions (84, 167). The phosphorylated AKT1 could be dephosphorylated by the 2C-type protein phosphatase (PP2C) AIP1 (AKT1-interacting PP2C 1), resulting in the inhibition of AKT1 activity (**Figure 1**) (80). It has been suggested that the kinase CIPK23 and phosphatase AIP1 both interact with AKT1, forming a molecular complex to regulate AKT1 activity by switching its phosphorylation status (80).

Another Shaker K^+ channel, AKT2, is also regulated by phosphorylation and dephosphorylation (26). The phosphorylation status of AKT2 is crucial for its channel activity and physiological function in plant cells (44, 99). The modification of AKT2 by phosphorylation significantly regulates AKT2 function in K^+ circulation in the phloem, which serves as a "K^+ battery" and assists the H^+-ATPase in energizing transmembrane transport (44). Unfortunately, the protein kinase(s) that may phosphorylate AKT2 channels have not been identified at the molecular level. However, AtPP2CA does interact with AKT2 and inhibit AKT2-mediated K^+ currents by direct dephosphorylation of AKT2 (**Figure 1**) (26). Phosphorylation and dephosphorylation of K^+ channel proteins are clearly important mechanisms for the regulation of K^+ uptake and translocation in plant cells, and may also benefit plant adaptation to K^+-deficiency stress.

Other Regulatory Proteins of K^+ Channels

Studies in animals have shown that different K^+ channel subunits from the same subfamily can assemble and form heterotetrameric channels that exhibit new channel properties, apparently different from those of homotetrameric channels (106). The plant Shaker K^+ channels show high structural similarity with animal K^+ channels, indicating that a similar heterotetramerization might also exist in plant cells. Structural analysis shows that in plant cells the C-terminal regions of plant Shaker K^+ channels seem to assist channels to form functional heterotetramers (29, 37). Early studies indicated that plant Shaker α-subunits

from different tissues, species, and subfamilies could assemble indiscriminately and form heterotetrameric K^+ channels with diverse characteristics (35, 166). The best-investigated heterotetrameric channel in plant cells is the AKT1-AtKC1 complex in *Arabidopsis*. AtKC1 itself could not form a functional homotetrameric K^+ channel (122), but it acts as a general regulatory α-subunit that negatively modulates many inward Shaker K^+ channels, including AKT1, AKT2, KAT1, and KAT2 (36, 48, 67, 154). AtKC1 interacts with AKT1 to form an AKT1-AtKC1 heterotetrameric K^+ channel, and it inhibits AKT1-mediated K^+ currents by negatively shifting the voltage dependence of the AKT1 channel (**Figure 1**) (36, 48, 154). This negative regulation may limit AKT1-mediated K^+ leakage in *Arabidopsis* roots under K^+-deficient conditions (48, 154).

Because many plant K^+ channels harbor regulatory domains in the cytoplasm, they could be modulated by various cytoplasmic regulatory components, such as trafficking proteins (64, 142), 14-3-3 proteins (78, 140), and K^+ channel β-subunits (169). SYP121 is an identified SNARE protein that mediates the traffic and positional anchoring of K^+ channels to the PM (142). It preferentially interacts with the AtKC1 channel rather than AKT1 and promotes the gating of the AKT1 channel, suggesting that it plays a role in regulating K^+ uptake in *Arabidopsis* roots (**Figure 1**) (64). The 14-3-3 proteins are also involved in regulating K^+ channel activities in plant cells. Sottocornola et al. (140) revealed that the maize 14-3-3 protein GF14-6 can directly interact with the *Arabidopsis* KAT1 channel and that it increases KAT1-mediated K^+ currents in vitro by shifting the channel activation voltage to a more positive direction. A similar activation by 14-3-3 proteins was also observed in the tonoplast. In *Arabidopsis*, the 14-3-3 protein GRF6 interacts with TPK1, an important K^+ channel, in the tonoplast and strongly enhances TPK1 activities (**Figure 1**) (78).

In addition, plant Shaker K^+ channels possess three main regulatory domains—CNBD (putative cyclic nucleotide-binding domain), Anky (ankyrin domain), and KHA (domain rich in hydrophobic and acidic residues)—that function in cyclic nucleotide binding, subunit tetramerization, and interaction with regulatory proteins (45, 149). Investigations of the AKT1 channel have indicated that AKT1 could be modulated by several regulatory proteins (36, 48, 122a, 154, 167). Thus, the identification and characterization of more plant K^+ channel regulatory proteins would increase our understanding of regulatory mechanisms of plant responses to K^+-deficiency stress.

TRANSCRIPTIONAL REGULATION IN PLANT RESPONSES TO K^+ DEFICIENCY

Transcriptional regulation has been widely investigated in plant responses to environmental stimuli. A number of marker genes for responses to specific stresses have been characterized. Transcriptomic analyses of plant responses to nutrient deficiencies have been performed using microarray technology, which provides the genome-wide gene expression profiles for the study of transcriptional regulation in plant responses to nutrient deficiencies. Previous transcriptomic data revealed that many genes are transcriptionally regulated by phosphorus and nitrogen deprivation (153, 162). Under K^+-deficient conditions, some K^+-related genes—including those encoding K^+ transporters, regulatory factors, and signaling components—are also regulated at the transcriptional level (11, 51, 91).

K^+ Transporter Genes

Investigations of plant responses to nutrient deficiencies (e.g., deficiencies in K^+, nitrogen, phosphorus, or sulfur) have indicated that the transcription of many ion transporter genes is significantly induced when the substrate concentration in the growing medium is significantly decreased, and such a transcriptional induction may eventually enhance nutrient absorption from the external environment (6). Transcriptional analysis has revealed that many

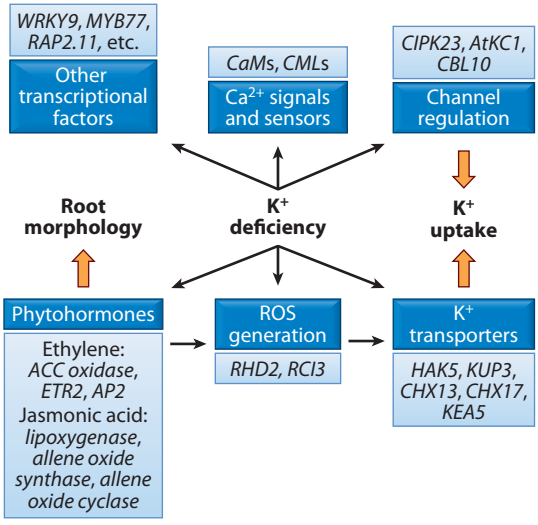

Figure 2

Transcriptional regulation of K^+-deficiency-responsive genes in *Arabidopsis*. K^+ deficiency induces transcriptional changes in many functional genes related to signaling components, including phytohormones (11, 137), ROS generation (71, 137), K^+ transporters (2, 24, 51, 70, 137, 170), Ca^{2+} signals and sensors (11, 91), and other transcriptional factors (72, 137). Some ethylene-related genes are induced by K^+ deficiency, which leads to ethylene accumulation (137). Ethylene, as an important signal molecule in plant responses to K^+ deficiency, stimulates ROS generation and root morphological changes (68). The K^+-deficiency-induced genes *RHD2* and *RCI3* encode enzymes for ROS generation (71, 137), which involves the transcriptional regulation of the K^+ transporter gene *HAK5* (71, 72). Some regulatory factors of K^+ channels (such as *CIPK23*) are also transcriptionally regulated by K^+ deficiency (25, 122a, 137, 167).

K^+ transporter genes, rather than K^+ channel genes, respond to K^+-deficient conditions. It has been hypothesized that the K^+ transporters are regulated primarily at the transcriptional level, whereas K^+ channels are regulated primarily at the posttranslational level.

In *Arabidopsis*, the transcription of some K^+ transporter genes—including *AtHAK5*, *AtKEA5*, *AtKUP3*, *AtCHX13*, and *AtCHX17*—is induced by K^+ deficiency (**Figure 2**) (2, 24, 50, 70, 137, 170). Among these genes, *AtHAK5* shows remarkable transcriptional induction by K^+ deficiency, and is also regarded as the marker gene in *Arabidopsis* response to K^+ deficiency (51, 137). The increase of *AtHAK5* transcription under K^+-deficient conditions is also dependent on the ROS signal generation and transcription factor RAP2.11 (**Figure 2**) (68, 71, 72, 137). Notably, the K^+ transporters from the *HAK* gene family seem to be the major components whose transcription is induced under K^+-deficient conditions. The *AtHAK5* homologs from other plant species, such as *LeHAK5* (156), *HvHAK1* (127), *CaHAK1* (96), and *OsHAK1* (14), all respond to K^+ deficiency. Although few K^+ channel genes are transcriptionally regulated by K^+ deficiency, a Shaker K^+ channel from wheat *TaAKT1* is induced by K^+ deficiency (22).

K^+ Channel Regulator Genes

Unlike *AtHAK5*, *AKT1* transcription is not induced by K^+ deficiency (114). However, *CIPK23*, which encodes the positive regulator of the AKT1 channel (as described above), showed significant transcriptional change under K^+-deficiency stress (**Figure 2**) (25, 167). In addition, *CBL10*, which encodes a negative regulator of the AKT1 channel, showed transcriptional inhibition under K^+-deficiency stress (**Figure 2**) (122a). Interestingly, another gene that encodes an AKT1 negative regulator, *AtKC1*, is induced by K^+ deficiency at the transcriptional level (**Figure 2**) (137). A K^+-deficiency-induced increase of *AtKC1* transcription may subsequently increase AtKC1 activity, which negatively shifts the voltage dependence of AKT1 channels and reduces K^+ leakage through AKT1 under K^+-deficient conditions (48, 154). These results suggest that the transcriptional regulation may be a more important mechanism for AKT1 regulator genes than *AKT1* itself.

Other K^+-Deficiency-Responsive Genes

ROS, Ca^{2+}, ethylene, and jasmonic acid have been considered the major signals in plant responses to K^+-deficiency stress (11, 129, 137). Transcriptomic analysis has revealed that a series of genes related to ROS metabolism, Ca^{2+} signal perception, and ethylene and jasmonic acid synthesis are significantly

induced by K^+ deficiency. In *Arabidopsis*, the NADPH oxidase gene *RHD2* and the peroxidase gene *RCI3*, which are involved in ROS generation, are significantly upregulated following K^+ deprivation (**Figure 2**) (71, 137). Furthermore, the induction of these two genes is required for the transcriptional regulation of a series of ROS-dependent genes in plant responses to K^+ deficiency (71, 137). However, in *rbd2* mutant plants that lack ROS production, many K^+-deficiency-induced genes, such as *WRKY9* and *AtHAK5*, are no longer upregulated following K^+-deficiency stress (137). In addition, two ethylene-synthesis-related genes—those encoding the AP2 transcription factor and ACC oxidase—are also induced under K^+-deficient conditions in wild-type plants but not in *rbd2* mutant plants (**Figure 2**) (137). These results suggest that both ROS and ethylene are involved in plant responses to K^+-deficiency stress and that ethylene may be the upstream signal of ROS generation (**Figure 2**) (68).

The genes related to jasmonic acid biosynthesis—such as those encoding the jasmonic biosynthetic enzymes lipoxygenase, allene oxide synthase, and allene oxide cyclase—are also significantly induced by K^+ deficiency but quickly reduced after K^+ resupply, suggesting jasmonic acid's prominent role in plant signaling in response to K^+ deficiency (**Figure 2**) (11). Plant Ca^{2+} sensors, including many *CaM* and *CML* genes, are regarded as important K^+-responsive genes, and their transcription is also induced under K^+-deficient conditions (**Figure 2**) (11, 91). These transcriptionally regulated genes may participate in perceiving and transducing K^+-deficiency signals and play a role in plant adaptation to K^+-deficiency stress (11, 137).

ROOT GROWTH UNDER K^+-DEFICIENT CONDITIONS

K^+ is considered the main osmotic cation in plant root cells, and it generates turgor pressure for cell expansion in the elongation zone of plant roots (34). K^+ deficiency significantly affects root growth and root architecture, including inhibiting primary root growth and stimulating root hair elongation (68, 146). More and more data have revealed that the root morphology under K^+-deficient conditions is significantly dependent on external ammonium (NH_4^+) concentration and regulated by phytohormones such as ethylene and auxin.

Root Growth and NH_4^+

NH_4^+ can be utilized as an important nitrogen source for higher plants. However, high NH_4^+ concentrations in the environment may generate toxicity and dramatically repress plant root growth (23). This repression is pronounced when the available K^+ in the external medium is reduced but reverses when the K^+ is resupplied (**Figure 3**) (23, 167). It is likely that the NH_4^+ inhibition of root growth is due to competition and interference with K^+ uptake in plant root cells. NH_4^+ and K^+ ions have similar properties in terms of hydrated ion diameter, charge value, and hydration energy, which causes the absorption interference of these two ions (143, 145).

In higher plants, the NH_4^+ uptake is mediated mainly by NH_4^+ transporters (**Figure 3**) (168). However, some K^+ transporters and channels—such as AKT1, AtHAK5, and HvHKT2.1—are also permeable to NH_4^+ (145). As the external NH_4^+ concentration increases, the K^+ uptake is competitively inhibited by NH_4^+ absorption via these K^+ transporters and channels (145). It should be noted that AKT1-like K^+ channels are insensitive to high external NH_4^+ concentrations, whereas the K^+ transporters, such as AtHAK5, HvHAK1, and CaHAK1, are considered NH_4^+-sensitive components whose K^+ transport activities are inhibited by external NH_4^+ (**Figure 3**) (96, 126, 141). Furthermore, the transcriptional induction of K^+ transporter genes by K^+-deficiency stress is also impaired in the presence of NH_4^+ (96, 118). It seems that a high NH_4^+

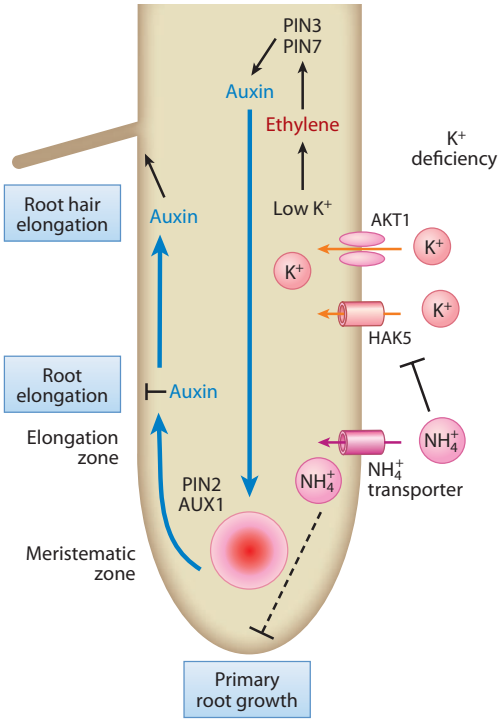

Figure 3

Root growth regulation by NH_4^+ and phytohormones in *Arabidopsis* responses to K^+ deficiency. Under K^+-deficient conditions, external NH_4^+ competitively inhibits the K^+ uptake mediated by the NH_4^+-sensitive K^+ transporter HAK5, but does not inhibit the NH_4^+-insensitive inward K^+ channel AKT1 (96, 126, 141, 145). Meanwhile, the NH_4^+ is absorbed directly into plant cells by NH_4^+ transporters and inhibits the primary root growth (23, 146, 168). Ethylene is induced by K^+-deficiency stress (137) and enhances auxin transport toward roots by increasing *PIN3* and *PIN7* transcription in the central cylinder, resulting in auxin accumulation in the meristematic zone (103). At the root tip, ethylene enhances auxin transport from the root apex to the elongation zone by increasing *PIN2* and *AUX1* transcription (103). Eventually, the accumulated auxin in the elongation zone reduces the primary root elongation but stimulates the root hair initiation and elongation.

concentration could inhibit both K^+ transporter activity and transcription under K^+-deficient conditions.

Root Growth and Phytohormones

The phytohormones have been proposed to participate in plant root morphology under K^+-deficient conditions. It is interesting that the addition of ethylene or auxin to the external medium leads to the inhibition of plant primary root growth and root hair elongation and that such a phenotype change is very similar to the phenotype change observed in K^+-deficient plants (68, 103). This phenomenon indicates that ethylene and auxin may participate in the root morphology regulation under K^+-deficient conditions. Ethylene is the initial responsive phytohormone to K^+ deficiency, and its content is significantly increased only 6 h after K^+ deficiency begins (**Figure 3**) (137). That several genes related to ethylene generation are significantly induced by K^+ deficiency is consistent with this finding (137). Moreover, the phenotype changes in primary root growth and root hair elongation are also observed in ethylene-treated plants and in constitutive ethylene response mutants (68, 116), and the inhibition of primary root growth by K^+ deficiency is relieved in *etr1* and *ein2* mutant plants (68). These results suggest that the root morphology under K^+-deficient conditions requires the ethylene signaling pathway and depends on ETR1 and EIN2 (68).

The inhibition of primary root growth by K^+ deficiency can be reversed in auxin-resistant mutants, indicating that auxin is another important phytohormone involved in the regulation of root architecture under K^+-deficient conditions (23). The detection of *DR5*:*GUS* expression in roots shows that auxin accumulates in central cylinder cells in the distal elongation zone following K^+ deprivation (150). This auxin redistribution under K^+-deficient conditions is likely caused by the alteration of auxin transport, which requires TRH1, a putative K^+ transporter belonging to the KUP/HAK/KT family (150). Recent studies have revealed that ethylene and auxin may function together, forming auxin-ethylene crosstalk, and synergistically or antagonistically control plant root growth and development (103). Ethylene could enhance auxin synthesis and transport in plant roots so that the accumulated auxin in the elongation zone reduces primary root growth but stimulates root hair elongation (**Figure 3**) (103). One may further hypothesize that ethylene acts as an upstream component of the auxin signaling pathway in plant responses to K^+ deficiency. The ethylene signal initially induced

by K^+ deficiency regulates auxin synthesis and distribution.

CROSSTALK BETWEEN K^+ AND OTHER MINERAL NUTRIENTS

Studies on signaling in plant responses to nutrient deficiencies have shown that different nutrient deficiencies may induce the same signaling component (such as ROS) at the early stage of nutrient deficiency (129, 136). ROS signal elevation is always observed in plant roots when plants are subjected to different nutrient deficiencies (e.g., deficiencies in K^+, nitrogen, phosphorus, or sulfur) (129, 136). In addition, many nutrition-related genes are always induced by deprivation of different nutrients (136). These results indicate that crosstalk may exist among different signaling pathways for plant responses to different nutrient deficiencies.

Recent investigations have indicated that K^+ nutrient signaling has a close relationship with nitrogen nutrition at both the transcriptional and posttranslational levels (**Figure 4**) (146). In *Arabidopsis*, K^+ deficiency not only induces the expression of K^+ transporter genes but also alters the transcription of NO_3^- transporter genes. Several NO_3^- transporter genes, including *NRT1.5*, *NRT2.1*, *NRT2.3*, and *NRT2.6*, are downregulated in plant roots under K^+-deficient conditions, but normal transcription quickly resumes after K^+ resupply (**Figure 4**) (11, 85). NRT2.1 is a high-affinity transporter involved in NO_3^- uptake in roots (86), and NRT1.5 is responsible for NO_3^- loading from pericycle cells into the xylem (85). Interestingly, K^+ translocation from roots to shoots is reduced in *nrt1.5* mutants (85). These findings suggest that NO_3^- uptake and transport may somehow relate to K^+ supply levels and that K^+ transport in plants is also dependent on NO_3^- transport (85).

Studies on the posttranslational regulation of K^+ channels and NO_3^- transporters have indicated that they might share the same (or a similar) regulatory mechanism. The protein kinase CIPK23, a positive regulator of the K^+ channel AKT1, modulates the activity of the NO_3^- transporter CHL1 (**Figure 4**) (61, 167). The K^+ transport activity of the AKT1 channel is activated by CIPK23 via phosphorylation, so that the AKT1-mediated K^+ uptake is enhanced under K^+-deficient conditions (167). Similarly, after phosphorylation by CIPK23, CHL1 shifts to the high-affinity mode and mediates NO_3^- uptake under low-NO_3^- conditions (61). CIPK23 may act as an important node of the plant nutrient regulatory network and simultaneously control the uptake of both K^+ and NO_3^- (61).

The importance of coregulating K^+ and nitrogen nutrition is clearly to maintain the balance of K^+ and NO_3^- uptake, translocation, and metabolic processes in plants. K^+ deficiency inhibits the activities of pyruvate kinase and NO_3^- reductase, consequently decreasing

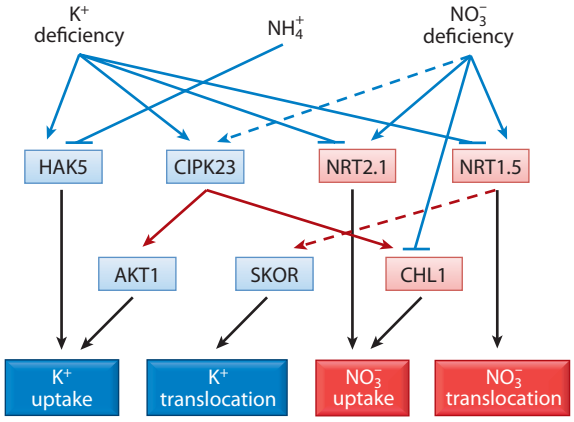

Figure 4

Schematic model of the crosstalk between K^+ and nitrogen signaling in *Arabidopsis* responses to K^+ and nitrogen deficiencies. The K^+ transporter HAK5 and K^+ channel AKT1 play a major role in K^+ uptake in *Arabidopsis* roots (51, 60, 76, 117), and the NO_3^- transporters CHL1 and NRT2.1 function in NO_3^- uptake (61, 86). The K^+ channel SKOR and NO_3^- transporter NRT1.5 regulate the root-to-shoot translocation of K^+ and NO_3^-, respectively (47, 85). K^+ deficiency enhances *HAK5* and *CIPK23* transcription (25, 50, 137, 167) but reduces *NRT2.1* and *NRT1.5* transcription (11, 85). NH_4^+ inhibits *HAK5* transcription under K^+-deficient conditions (96, 118). Nitrogen deficiency induces *NRT2.1* and *NRT1.5* transcription but downregulates *CHL1* expression (82, 85). The increased CIPK23 regulates activities of both AKT1 and CHL1 by phosphorylation, which coordinately modulates K^+ uptake and NO_3^- uptake (61, 167). NRT1.5-mediated NO_3^- translocation also affects the root-to-shoot K^+ translocation that may be conducted by SKOR channels (85).

the carbon flux into the TCA cycle and reducing amino acids in plant roots, respectively (12). However, the GS/GOGAT/GDH cycle activity in roots is enhanced after K^+ deprivation, which maintains carbon flux into amino acids in the face of reduced glycolysis (12). This coregulation of K^+ and nitrogen nutrition may also function in the balance of carbon and nitrogen metabolism and modulate the carbon/nitrogen ratio in amino acids under K^+-deficient conditions (12).

CONCLUSIONS AND FUTURE CHALLENGES

This review summarizes the most recent findings in plant signaling and transport regulation in response to K^+ deficiency. Because of space limitations, we have focused on the components involved in the regulation of K^+ uptake in roots as well as K^+ translocation from root to shoot. Other research fields closely related to K^+ signaling, such as the regulation of stomatal movement (110, 138, 157) and pollen tube growth (63, 88, 98), have also made great progress and are facing similar challenges.

The extensive genomic, molecular genetic, and physiological studies of the past two decades have shed light on the signaling mechanisms and transport regulation of plant responses to K^+ deficiency. Many K^+ transporters and channels in higher plants—especially *Arabidopsis*—have been cloned, and some have been functionally characterized. These K^+ transporters and channels are regarded not only as the major components of K^+ uptake and translocation but also as potential K^+ sensors for plant responses to K^+ deficiency. However, the functions and regulatory mechanisms of many K^+ transporters and channels remain unknown. Thus, future investigations should give attention to further functional characterization of these channels and transporters and to the regulatory mechanisms of these components. Analyzing the coordination among various channels and transporters is also an important task, one that would benefit our understanding of the complex signaling network for plant responses to K^+ deficiency.

Comprehensive study of the regulatory mechanisms of K^+ transporters and channels is another important issue. Activities of K^+ channels and transporters can be modulated at transcriptional and/or posttranslational stages following the perception of K^+-deficiency signals. Previous studies have shown that many signaling components, such as Ca^{2+}, ROS, and phytohormones, are involved in plant signaling responses to K^+ deficiency. Thus, how to integrate these different signaling pathways into a comprehensive network needs extensive further investigation.

K^+, as an essential mineral nutrient for plant growth and development, is a key factor in crop yield and quality. Studying the regulatory mechanisms of plant responses to K^+ deficiency may therefore lead to improvements in crop K^+ utilization efficiency (KUE). However, most of the work in this field has used *Arabidopsis*; thus, an important future issue is how to transfer this knowledge of *Arabidopsis* to crop species. Faced with the challenges of the worldwide food supply, this work is expected to generate genetically modified crops with improved KUE, although there are many questions to be answered before moving to practice in farmland.

SUMMARY POINTS

1. Plant K^+ transporters and channels are encoded by several gene families and differ in their energetic coupling, affinity, and selectivity for K^+ ions.
2. The Shaker K^+ channel AKT1 and KUP/HAK/KT transporter HAK5 are expressed primarily in *Arabidopsis* roots and mediate most of the plant K^+ absorption from the external environment.

3. Transcriptional and posttranslational regulation of K^+ transporters and channels are major molecular mechanisms for K^+ transport and signaling in plant cells.
4. Ca^{2+}, ROS, and phytohormone signals are involved in plant signaling in response to K^+-deficiency stress. ROS regulates Ca^{2+} signal generation, and ethylene may regulate ROS generation.
5. The CBL-CIPK pathways play crucial roles in the perception of K^+-deficiency-induced Ca^{2+} signals and the regulation of K^+ channels or transporters at the posttranslational level.
6. Ethylene may act as an important signal molecule in plant responses to K^+ deficiency and may be involved in ROS generation and root morphological changes under K^+-deficient conditions.
7. Changes in root morphology under K^+-deficiency stress are dependent on external NH_4^+ and regulated by ethylene and auxin.
8. K^+ nutrient signaling shows crosstalk and coregulation with nitrogen signaling in plant cells at both transcriptional and posttranslational levels.

DISCLOSURE STATEMENT

The authors are not aware of any affiliations, memberships, funding, or financial holdings that might be perceived as affecting the objectivity of this review.

ACKNOWLEDGMENTS

Work in W.H.W.'s laboratory is supported primarily by the National Natural Science Foundation of China (#30830013, #31121002, and #30900269), the China Key Basic Research Program (#2011CB100300), and the Program of Introducing Talents of Discipline to Universities (111 Program #B06003). We apologize to all colleagues whose work could not be cited owing to space limitations.

LITERATURE CITED

1. Ache P, Becker D, Ivashikina N, Dietrich P, Roelfsema RG, et al. 2000. GORK, a delayed outward rectifier expressed in guard cells of *Arabidopsis thaliana*, is a K^+-selective, K^+-sensing ion channel. *FEBS Lett.* 486:93–98
2. Ahn SJ, Shin R, Schachtman DP. 2004. Expression of *KT/KUP* genes in Arabidopsis and the role of root hairs in K^+ uptake. *Plant Physiol.* 134:1135–45
3. Albrecht V, Ritz O, Linder S, Harter K, Kudla J. 2001. The NAF domain defines a novel protein-protein interaction module conserved in Ca^{2+}-regulated kinases. *EMBO J.* 20:1051–63
4. Allen GJ, Chu SP, Harrington CL, Schumacher K, Hoffmann T, et al. 2001. A defined range of guard cell calcium oscillation parameters encodes stomatal movements. *Nature* 411:1053–57
5. Amtmann A, Armengaud P. 2009. Effects of N, P, K and S on metabolism: new knowledge gained from multi-level analysis. *Curr. Opin. Plant Biol.* 12:275–83
6. Amtmann A, Blatt MR. 2009. Regulation of macronutrient transport. *New Phytol.* 181:35–52
7. Amtmann A, Troufflard S, Armengaud P. 2008. The effect of potassium nutrition on pest and disease resistance in plants. *Physiol. Plant.* 133:682–91
8. Anderson JA, Huprikar SS, Kochian LV, Lucas WJ, Gaber RF. 1992. Functional expression of a probable *Arabidopsis thaliana* potassium channel in *Saccharomyces cerevisiae*. *Proc. Natl. Acad. Sci. USA* 89:3736–40

9. Apel K, Hirt H. 2004. Reactive oxygen species: metabolism, oxidative stress, and signal transduction. *Annu. Rev. Plant Biol.* 55:373–99
10. Apse MP, Sottosanto JB, Blumwald E. 2003. Vacuolar cation/H^+ exchange, ion homeostasis, and leaf development are altered in a T-DNA insertional mutant of *AtNHX1*, the *Arabidopsis* vacuolar Na^+/H^+ antiporter. *Plant J.* 36:229–39
11. Armengaud P, Breitling R, Amtmann A. 2004. The potassium-dependent transcriptome of *Arabidopsis* reveals a prominent role of jasmonic acid in nutrient signaling. *Plant Physiol.* 136:2556–76
12. Armengaud P, Sulpice R, Miller AJ, Stitt M, Amtmann A, et al. 2009. Multilevel analysis of primary metabolism provides new insights into the role of potassium nutrition for glycolysis and nitrogen assimilation in Arabidopsis roots. *Plant Physiol.* 150:772–85
13. Ashley MK, Grant M, Grabov A. 2006. Plant responses to potassium deficiencies: a role for potassium transport proteins. *J. Exp. Bot.* 57:425–36
14. Bañuelos MA, Garciadeblas B, Cubero B, Rodríguez-Navarro A. 2002. Inventory and functional characterization of the HAK potassium transporters of rice. *Plant Physiol.* 130:784–95
15. Barragan V, Leidi EO, Andres Z, Rubio L, De Luca A, et al. 2012. Ion exchangers NHX1 and NHX2 mediate active potassium uptake into vacuoles to regulate cell turgor and stomatal function in *Arabidopsis*. *Plant Cell* 24:1127–42
16. Bassil E, Tajima H, Liang YC, Ohto MA, Ushijima K, et al. 2011. The *Arabidopsis* Na^+/H^+ antiporters NHX1 and NHX2 control vacuolar pH and K^+ homeostasis to regulate growth, flower development, and reproduction. *Plant Cell* 23:3482–97
17. Batistič O, Waadt R, Steinhorst L, Held K, Kudla J. 2010. CBL-mediated targeting of CIPKs facilitates the decoding of calcium signals emanating from distinct cellular stores. *Plant J.* 61:211–22
18. Becker D, Geiger D, Dunkel M, Roller A, Bertl A, et al. 2004. AtTPK4, an *Arabidopsis* tandem-pore K^+ channel, poised to control the pollen membrane voltage in a pH- and Ca^{2+}-dependent manner. *Proc. Natl. Acad. Sci. USA* 101:15621–26
19. Behl R, Raschke K. 1987. Close coupling between extrusion of H^+ and uptake of K^+ by barley roots. *Planta* 172:531–38
20. Bouché N, Yellin A, Snedden WA, Fromm H. 2005. Plant-specific calmodulin-binding proteins. *Annu. Rev. Plant Biol.* 56:435–66
21. Buch-Pedersen MJ, Rudashevskaya EL, Berner TS, Venema K, Palmgren MG. 2006. Potassium as an intrinsic uncoupler of the plasma membrane H^+-ATPase. *J. Biol. Chem.* 281:38285–92
22. Buschmann PH, Vaidyanathan R, Gassmann W, Schroeder JI. 2000. Enhancement of Na^+ uptake currents, time-dependent inward-rectifying K^+ channel currents, and K^+ channel transcripts by K^+ starvation in wheat root cells. *Plant Physiol.* 122:1387–97
23. Cao Y, Glass AD, Crawford NM. 1993. Ammonium inhibition of *Arabidopsis* root growth can be reversed by potassium and by auxin resistance mutations *aux1*, *axr1*, and *axr2*. *Plant Physiol.* 102:983–89
24. Cellier F, Conejero G, Ricaud L, Luu DT, Lepetit M, et al. 2004. Characterization of *AtCHX17*, a member of the cation/H^+ exchangers, CHX family, from *Arabidopsis thaliana* suggests a role in K^+ homeostasis. *Plant J.* 39:834–46
25. Cheong YH, Pandey GK, Grant JJ, Batistič O, Li L, et al. 2007. Two calcineurin B-like calcium sensors, interacting with protein kinase CIPK23, regulate leaf transpiration and root potassium uptake in *Arabidopsis*. *Plant J.* 52:223–39
26. Chérel I, Michard E, Platet N, Mouline K, Alcon C, et al. 2002. Physical and functional interaction of the *Arabidopsis* K^+ channel AKT2 and phosphatase AtPP2CA. *Plant Cell* 14:1133–46
27. Clarkson DT, Hanson JB. 1980. The mineral nutrition of higher plants. *Annu. Rev. Plant Physiol.* 31:239–98
28. Czempinski K, Frachisse JM, Maurel C, Barbier-Brygoo H, Mueller-Roeber B. 2002. Vacuolar membrane localization of the *Arabidopsis* "two-pore" K^+ channel KCO1. *Plant J.* 29:809–20
29. Daram P, Urbach S, Gaymard F, Sentenac H, Cherel I. 1997. Tetramerization of the AKT1 plant potassium channel involves its C-terminal cytoplasmic domain. *EMBO J.* 16:3455–63
30. Demidchik V, Davenport RJ, Tester M. 2002. Nonselective cation channels in plants. *Annu. Rev. Plant Biol.* 53:67–107

31. Demidchik V, Maathuis FJ. 2007. Physiological roles of nonselective cation channels in plants: from salt stress to signalling and development. *New Phytol.* 175:387–404
32. Demidchik V, Shabala SN, Davies JM. 2007. Spatial variation in H_2O_2 response of *Arabidopsis thaliana* root epidermal Ca^{2+} flux and plasma membrane Ca^{2+} channels. *Plant J.* 49:377–86
33. DeWitt ND, Hong B, Sussman MR, Harper JF. 1996. Targeting of two Arabidopsis H^+-ATPase isoforms to the plasma membrane. *Plant Physiol.* 112:833–44
34. Dolan L, Davies J. 2004. Cell expansion in roots. *Curr. Opin. Plant Biol.* 7:33–39
35. Dreyer I, Antunes S, Hoshi T, Müller-Röber B, Palme K, et al. 1997. Plant K^+ channel α-subunits assemble indiscriminately. *Biophys. J.* 72:2143–50
36. Duby G, Hosy E, Fizames C, Alcon C, Costa A, et al. 2008. AtKC1, a conditionally targeted Shaker-type subunit, regulates the activity of plant K^+ channels. *Plant J.* 53: 115–23
37. Ehrhardt T, Zimmermann S, Müller-Röber B. 1997. Association of plant K^+in channels is mediated by conserved C-termini and does not affect subunit assembly. *FEBS Lett.* 409:166–70
38. Elumalai RP, Nagpal P, Reed JW. 2002. A mutation in the Arabidopsis *KT2/KUP2* potassium transporter gene affects shoot cell expansion. *Plant Cell* 14:119–31
39. Epstein E, Rains D, Elzam O. 1963. Resolution of dual mechanisms of potassium absorption by barley roots. *Proc. Natl. Acad. Sci. USA* 49:684–92
40. Frietsch S, Wang YF, Sladek C, Poulsen LR, Romanowsky SM, et al. 2007. A cyclic nucleotide-gated channel is essential for polarized tip growth of pollen. *Proc. Natl. Acad. Sci. USA* 104:14531–36
41. Fu HH, Luan S. 1998. AtKUP1: a dual-affinity K^+ transporter from Arabidopsis. *Plant Cell* 10:63–73
42. Fuchs I, Stolzle S, Ivashikina N, Hedrich R. 2005. Rice K^+ uptake channel OsAKT1 is sensitive to salt stress. *Planta* 221:212–21
43. Fuglsang AT, Guo Y, Cuin TA, Qiu Q, Song C, et al. 2007. *Arabidopsis* protein kinase PKS5 inhibits the plasma membrane H^+-ATPase by preventing interaction with 14-3-3 protein. *Plant Cell* 19:1617–34
44. Gajdanowicz P, Michard E, Sandmann M, Rocha M, Correa LGG, et al. 2011. Potassium (K^+) gradients serve as a mobile energy source in plant vascular tissues. *Proc. Natl. Acad. Sci. USA* 108:864–69
45. Gambale F, Uozumi N. 2006. Properties of Shaker-type potassium channels in higher plants. *J. Membr. Biol.* 210:1–19
46. Gassmann W, Rubio F, Schroeder JI. 1996. Alkali cation selectivity of the wheat root high-affinity potassium transporter HKT1. *Plant J.* 10:869–82
47. Gaymard F, Pilot G, Lacombe B, Bouchez D, Bruneau D, et al. 1998. Identification and disruption of a plant Shaker-like outward channel involved in K^+ release into the xylem sap. *Cell* 94:647–55
48. Geiger D, Becker D, Vosloh D, Gambale F, Palme K, et al. 2009. Heteromeric AtKC1 • AKT1 channels in *Arabidopsis* roots facilitate growth under K^+-limiting conditions. *J. Biol. Chem.* 284:21288–95
49. Geiger D, Scherzer S, Mumm P, Marten I, Ache P, et al. 2010. Guard cell anion channel SLAC1 is regulated by CDPK protein kinases with distinct Ca^{2+} affinities. *Proc. Natl. Acad. Sci. USA* 107:8023–28
50. Gierth M, Mäser P. 2007. Potassium transporters in plants—involvement in K^+ acquisition, redistribution and homeostasis. *FEBS Lett.* 581:2348–56
51. Gierth M, Mäser P, Schroeder JI. 2005. The potassium transporter *AtHAK5* functions in K^+ deprivation-induced high-affinity K^+ uptake and *AKT1* K^+ channel contribution to K^+ uptake kinetics in Arabidopsis roots. *Plant Physiol.* 137:1105–14
52. Gobert A, Isayenkov S, Voelker C, Czempinski K, Maathuis FJM. 2007. The two-pore channel *TPK1* gene encodes the vacuolar K^+ conductance and plays a role in K^+ homeostasis. *Proc. Natl. Acad. Sci. USA* 104:10726–31
53. Grabov A. 2007. Plant KT/KUP/HAK potassium transporters: single family—multiple functions. *Ann. Bot.* 99:1035–41
54. Harmon AC, Gribskov M, Harper JF. 2000. CDPKs—a kinase for every Ca^{2+} signal? *Trends Plant Sci.* 5:154–59
55. Harper JF, Surowy TK, Sussman MR. 1989. Molecular cloning and sequence of cDNA encoding the plasma membrane proton pump (H^+-ATPase) of *Arabidopsis thaliana*. *Proc. Natl. Acad. Sci. USA* 86:1234–38

56. Hartje S, Zimmermann S, Klonus D, Mueller-Roeber B. 2000. Functional characterisation of LKT1, a K^+ uptake channel from tomato root hairs, and comparison with the closely related potato inwardly rectifying K^+ channel SKT1 after expression in *Xenopus* oocytes. *Planta* 210:723–31
57. Hedrich R, Kudla J. 2006. Calcium signaling networks channel plant K^+ uptake. *Cell* 125:1221–23
58. Held K, Pascaud F, Eckert C, Gajdanowicz P, Hashimoto K, et al. 2011. Calcium-dependent modulation and plasma membrane targeting of the AKT2 potassium channel by the CBL4/CIPK6 calcium sensor/protein kinase complex. *Cell Res.* 21:1116–30
59. Hetherington AM, Brownlee C. 2004. The generation of Ca^{2+} signals in plants. *Annu. Rev. Plant Biol.* 55:401–27
60. Hirsch RE, Lewis BD, Spalding EP, Sussman MR. 1998. A role for the AKT1 potassium channel in plant nutrition. *Science* 280:918–21
61. Ho CH, Lin SH, Hu HC, Tsay YF. 2009. CHL1 functions as a nitrate sensor in plants. *Cell* 138:1184–94
62. Hodge A. 2004. The plastic plant: root responses to heterogeneous supplies of nutrients. *New Phytol.* 162:9–24
63. Holdaway-Clarke TL, Hepler PK. 2003. Control of pollen tube growth: role of ion gradients and fluxes. *New Phytol.* 159:539–63
64. Honsbein A, Sokolovski S, Grefen C, Campanoni P, Pratelli R, et al. 2009. A tripartite SNARE-K^+ channel complex mediates in channel-dependent K^+ nutrition in *Arabidopsis*. *Plant Cell* 21:2859–77
65. Hosoi S, Iino M, Shimazaki K. 1988. Outward-rectifying K^+ channels in stomatal guard cell protoplasts. *Plant Cell Physiol.* 29:907–11
66. Hosy E, Vavasseur A, Mouline K, Dreyer I, Gaymard F, et al. 2003. The *Arabidopsis* outward K^+ channel *GORK* is involved in regulation of stomatal movements and plant transpiration. *Proc. Natl. Acad. Sci. USA* 100:5549–54
67. Jeanguenin L, Alcon C, Duby G, Boeglin M, Cherel I, et al. 2011. AtKC1 is a general modulator of *Arabidopsis* inward Shaker channel activity. *Plant J.* 67:570–82
68. Jung JY, Shin R, Schachtman DP. 2009. Ethylene mediates response and tolerance to potassium deprivation in *Arabidopsis*. *Plant Cell* 21:607–21
69. Kim BG, Waadt R, Cheong YH, Pandey GK, Dominguez-Solis JR, et al. 2007. The calcium sensor CBL10 mediates salt tolerance by regulating ion homeostasis in Arabidopsis. *Plant J.* 52:473–84
70. Kim EJ, Kwak JM, Uozumi N, Schroeder JI. 1998. *AtKUP1*: an Arabidopsis gene encoding high-affinity potassium transport activity. *Plant Cell* 10:51–62
71. Kim MJ, Ciani S, Schachtman DP. 2010. A peroxidase contributes to ROS production during *Arabidopsis* root response to potassium deficiency. *Mol. Plant* 3:420–27
72. Kim MJ, Ruzicka D, Shin R, Schachtman DP. 2012. The *Arabidopsis* AP2/ERF transcription factor RAP2.11 modulates plant response to low-potassium conditions. *Mol. Plant* 5:1042–57
73. Kudla J, Xu Q, Harter K, Gruissem W, Luan S. 1999. Genes for calcineurin B-like proteins in *Arabidopsis* are differentially regulated by stress signals. *Proc. Natl. Acad. Sci. USA* 96:4718–23
74. Kwak JM, Murata Y, Baizabal-Aguirre VM, Merrill J, Wang M, et al. 2001. Dominant negative guard cell K^+ channel mutants reduce inward-rectifying K^+ currents and light-induced stomatal opening in Arabidopsis. *Plant Physiol.* 127:473–85
75. Lacombe B, Pilot G, Michard E, Gaymard F, Sentenac H, et al. 2000. A Shaker-like K^+ channel with weak rectification is expressed in both source and sink phloem tissues of Arabidopsis. *Plant Cell* 12:837–51
76. Lagarde D, Basset M, Lepetit M, Conejero G, Gaymard F, et al. 1996. Tissue-specific expression of *Arabidopsis AKT1* gene is consistent with a role in K^+ nutrition. *Plant J.* 9:195–203
77. Laohavisit A, Shang Z, Rubio L, Cuin TA, Véry AA, et al. 2012. *Arabidopsis* Annexin1 mediates the radical-activated plasma membrane Ca^{2+}- and K^+-permeable conductance in root cells. *Plant Cell* 24:1522–33
78. Latz A, Becker D, Hekman M, Muller T, Beyhl D, et al. 2007. TPK1, a Ca^{2+}-regulated Arabidopsis vacuole two-pore K^+ channel is activated by 14-3-3 proteins. *Plant J.* 52:449–59
79. Lebaudy A, Véry AA, Sentenac H. 2007. K^+ channel activity in plants: genes, regulations and functions. *FEBS Lett.* 581:2357–66

80. Lee SC, Lan WZ, Kim BG, Li L, Cheong YH, et al. 2007. A protein phosphorylation/dephosphorylation network regulates a plant potassium channel. *Proc. Natl. Acad. Sci. USA* 104:15959–64
81. Leigh RA, Wyn Jones RG. 1984. A hypothesis relating critical potassium concentrations for growth to the distribution and functions of this ion in the plant cell. *New Phytol.* 97:1–13
82. Lejay L, Tillard P, Lepetit M, Olive FD, Filleur S, Daniel-Vedele F, Gojon A. 1999. Molecular and functional regulation of two NO_3^- uptake systems by N- and C-status of *Arabidopsis* plants. *Plant J.* 18:509–19
83. Leng Q, Mercier RW, Yao W, Berkowitz GA. 1999. Cloning and first functional characterization of a plant cyclic nucleotide-gated cation channel. *Plant Physiol.* 121:753–61
84. Li L, Kim BG, Cheong YH, Pandey GK, Luan S. 2006. A Ca^{2+} signaling pathway regulates a K^+ channel for low-K response in *Arabidopsis*. *Proc. Natl. Acad. Sci. USA* 103:12625–30
85. Lin SH, Kuo HF, Canivenc G, Lin CS, Lepetit M, et al. 2008. Mutation of the *Arabidopsis NRT1.5* nitrate transporter causes defective root-to-shoot nitrate transport. *Plant Cell* 20:2514–28
86. Little DY, Rao H, Oliva S, Daniel-Vedele F, Krapp A, et al. 2005. The putative high-affinity nitrate transporter NRT2.1 represses lateral root initiation in response to nutritional cues. *Proc. Natl. Acad. Sci. USA* 102:13693–98
87. Liu LL, Ren HM, Chen LQ, Wang Y, Wu WH. 2013. A protein kinase CIPK9 interacts with calcium sensor CBL3 and regulates K^+ homeostasis under low-K^+ stress in *Arabidopsis*. *Plant Physiol.* 161:266–77
88. Lu YX, Chanroj S, Zulkifli L, Johnson MA, Uozumi N, Cheung A, Sze H. 2011. Pollen tubes lacking a pair of K^+ transporters fail to target ovules in *Arabidopsis*. *Plant Cell* 23:81–93
89. Luan S. 2008. The CBL-CIPK network in plant calcium signaling. *Trends Plant Sci.* 14:37–42
90. Luan S, Kudla J, Rodriguez-Concepcion M, Yalovsky S, Gruissem W. 2002. Calmodulins and calcineurin B–like proteins: calcium sensors for specific signal response coupling in plants. *Plant Cell* 14(Suppl.):S389–400
91. Ma TL, Wu WH, Wang Y. 2012. Transcriptome analysis of rice root responses to K^+ deficiency. *BMC Plant Biol.* 12:161
92. Maathuis FJM. 2009. Physiological functions of mineral macronutrients. *Curr. Opin. Plant Biol.* 12:250–58
93. Maathuis FJM, Sanders D. 1993. Energization of potassium uptake in *Arabidopsis thaliana*. *Planta* 191:302–7
94. Maathuis FJM, Sanders D. 1994. Mechanism of high-affinity potassium uptake in roots of *Arabidopsis thaliana*. *Proc. Natl. Acad. Sci. USA* 91:9272–76
95. Marten I, Hoth S, Deeken R, Ache P, Ketchum KA, et al. 1999. AKT3, a phloem-localized K^+ channel, is blocked by protons. *Proc. Natl. Acad. Sci. USA* 96:7581–86
96. Martinez-Cordero MA, Martinez V, Rubio F. 2005. High-affinity K^+ uptake in pepper plants. *J. Exp. Bot.* 56:1553–62
97. Mäser P, Thomine S, Schroeder JI, Ward JM, Hirschi K, et al. 2001. Phylogenetic relationships within cation transporter families of Arabidopsis. *Plant Physiol.* 126:1646–67
98. Michard E, Alves F, Feijó J. 2009. The role of ion fluxes in polarized cell growth and morphogenesis: the pollen tube as an experimental paradigm. *Int. J. Dev. Biol.* 53:1609–22
99. Michard E, Dreyer I, Lacombe B, Sentenac H, Thibaud JB. 2005. Inward rectification of the AKT2 channel abolished by voltage-dependent phosphorylation. *Plant J.* 44:783–97
100. Michard E, Lima PT, Borges F, Silva AC, Portes MT, et al. 2011. Glutamate receptor-like genes form Ca^{2+} channels in pollen tubes and are regulated by pistil D-serine. *Science* 332:434–37
101. Mori IC, Schroeder JI. 2004. Reactive oxygen species activation of plant Ca^{2+} channels. A signaling mechanism in polar growth, hormone transduction, stress signaling, and hypothetically mechanotransduction. *Plant Physiol.* 135:702–8
102. Mouline K, Véry AA, Gaymard F, Boucherez J, Pilot G, et al. 2002. Pollen tube development and competitive ability are impaired by disruption of a Shaker K^+ channel in *Arabidopsis*. *Genes Dev.* 16:339–50
103. Muday GK, Rahman A, Binder BM. 2012. Auxin and ethylene: collaborators or competitors? *Trends Plant Sci.* 17:181–95

104. Nagae M, Nozawa A, Koizumi N, Sano H, Hashimoto H, et al. 2003. The crystal structure of the novel calcium-binding protein AtCBL2 from *Arabidopsis thaliana*. *J. Biol. Chem.* 278:42240–46
105. Nieves-Cordones M, Miller AJ, Aleman F, Martinez V, Rubio F. 2008. A putative role for the plasma membrane potential in the control of the expression of the gene encoding the tomato high-affinity potassium transporter HAK5. *Plant Mol. Biol.* 68:521–32
106. Ottschytsch N, Raes A, Van Hoorick D, Snyders DJ. 2002. Obligatory heterotetramerization of three previously uncharacterized Kv channel α-subunits identified in the human genome. *Proc. Natl. Acad. Sci. USA* 99:7986–91
107. Padmanaban S, Chnroj S, Kwak JM, Li X, Ward J, et al. 2007. Participation of endomembrane cation/H^+ exchanger AtCHX20 in osmoregulation of guard cells. *Plant Physiol.* 44:82–93
108. Palmgren MG. 2001. Plant plasma membrane H^+-ATPases: powerhouses for nutrient uptake. *Annu. Rev. Plant Physiol. Plant Mol. Biol.* 52:817–45
109. Pandey GK, Cheong YH, Kim BG, Grant JJ, Li L, et al. 2007. CIPK9: a calcium sensor-interacting protein kinase required for low-potassium tolerance in *Arabidopsis*. *Cell Res.* 17:411–21
110. Pandey S, Zhang W, Assmann SM. 2007. Roles of ion channels and transporters in guard cell signal transduction. *FEBS Lett.* 581:2325–36
111. Pei ZM, Murata Y, Benning G, Thomine S, Klusener B, et al. 2000. Calcium channels activated by hydrogen peroxide mediate abscisic acid signalling in guard cells. *Nature* 406:731–34
112. Peiter E, Maathuis FJM, Mills LN, Knight H, Pelloux J, et al. 2005. The vacuolar Ca^{2+}-activated channel TPC1 regulates germination and stomatal movement. *Nature* 434:404–8
113. Philippar K, Fuchs I, Luthen H, Hoth S, Bauer CS, et al. 1999. Auxin-induced K^+ channel expression represents an essential step in coleoptile growth and gravitropism. *Proc. Natl. Acad. Sci. USA* 96:12186–91
114. Pilot G, Gaymard F, Mouline K, Chérel I, Sentenac H. 2003. Regulated expression of *Arabidopsis* Shaker K^+ channel genes involved in K^+ uptake and distribution in the plant. *Plant Mol. Biol.* 51:773–87
115. Pilot G, Lacombe B, Gaymard F, Cherel I, Boucherez J, et al. 2001. Guard cell inward K^+ channel activity in *Arabidopsis* involves expression of the twin channel subunits KAT1 and KAT2. *J. Biol. Chem.* 276:3215–21
116. Pitts RJ, Cernac A, Estelle M. 1998. Auxin and ethylene promote root hair elongation in *Arabidopsis*. *Plant J.* 16:553–60
117. Pyo YJ, Gierth M, Schroeder JI, Cho MH. 2010. High-affinity K^+ transport in *Arabidopsis*: AtHAK5 and AKT1 are vital for seedling establishment and postgermination growth under low-potassium conditions. *Plant Physiol.* 153:863–75
118. Qi Z, Hampton CR, Shin R, Barkla BJ, White PJ, et al. 2008. The high affinity K^+ transporter AtHAK5 plays a physiological role *in planta* at very low K^+ concentrations and provides a caesium uptake pathway in *Arabidopsis*. *J. Exp. Bot.* 59:595–607
119. Quan R, Lin H, Mendoza I, Zhang Y, Cao W, et al. 2007. SCABP8/CBL10, a putative calcium sensor, interacts with the protein kinase SOS2 to protect *Arabidopsis* shoots from salt stress. *Plant Cell* 19:1415–31
120. Quintero FJ, Ohta M, Shi H, Zhu JK, Pardo JM. 2002. Reconstitution in yeast of the *Arabidopsis* SOS signaling pathway for Na^+ homeostasis. *Proc. Natl. Acad. Sci. USA* 99:9061–66
121. Ramírez-Silva L, Ferreira ST, Nowak T, Tuena de Gómez-Puyou M, Gómez-Puyou A. 2001. Dimethylsulfoxide promotes K^+-independent activity of pyruvate kinase and the acquisition of the active catalytic conformation. *Eur. J. Biochem.* 268:3267–74
122. Reintanz B, Szyroki A, Ivashikina N, Ache P, Godde M, et al. 2002. AtKC1, a silent *Arabidopsis* potassium channel α-subunit modulates root hair K^+ influx. *Proc. Natl. Acad. Sci. USA* 99:4079–84
122a. Ren XL, Qi GN, Feng HQ, Zhao S, Zhao SS, et al. 2013. Calcineurin B-like protein CBL10 directly interacts with AKT1 and modulates K^+ homeostasis in *Arabidopsis*. *Plant J.* In press. doi: 10.1111/tpj.12123
123. Rigas S, Desbrosses G, Haralampidis K, Vicente-Agullo F, Feldmann KA, et al. 2001. *TRH1* encodes a potassium transporter required for tip growth in Arabidopsis root hairs. *Plant Cell* 13:139–51
124. Rubio F, Gassmann W, Schroeder JI. 1995. Sodium-driven potassium uptake by the plant potassium transporter HKT1 and mutations conferring salt tolerance. *Science* 270:1660–63

125. Rudd JJ, Franklin-Tong VE. 2001. Unravelling response-specificity in Ca^{2+} signaling pathways in plant cells. *New Phytol.* 151:7–33
126. Santa-María GE, Danna CH, Czibener C. 2000. High-affinity potassium transport in barley roots. Ammonium-sensitive and -insensitive pathways. *Plant Physiol.* 123:297–306
127. Santa-María GE, Rubio F, Dubcovsky J, Rodríguez-Navarro A. 1997. The *HAK1* gene of barley is a member of a large gene family and encodes a high-affinity potassium transporter. *Plant Cell* 9:2281–89
128. Schachtman DP, Schroeder JI, Lucas WJ, Anderson JA, Gaber RF. 1992. Expression of an inward-rectifying potassium channel by the *Arabidopsis KAT1* cDNA. *Science* 258:1654–58
129. Schachtman DP, Shin R. 2007. Nutrient sensing and signaling: NPKS. *Annu. Rev. Plant Biol.* 58:47–69
130. Schroeder JI, Fang HH. 1991. Inward-rectifying K^+ channels in guard cells provides a mechanism for low-affinity K^+ uptake. *Proc. Natl. Acad. Sci. USA* 88:11583–87
131. Schroeder JI, Hedrich R, Fernandez JM. 1984. Potassium-selective single channels in guard cell protoplasts of *Vicia faba*. *Nature* 312:361–62
132. Schroeder JI, Raschke K, Neher E. 1987. Voltage dependence of K^+ channels in guard-cell protoplasts. *Proc. Natl. Acad. Sci. USA* 84:4108–12
133. Schroeder JI, Ward JM, Gassmann W. 1994. Perspectives on the physiology and structure of inward-rectifying K^+ channels in higher plants: biophysical implications for K^+ uptake. *Annu. Rev. Biophys. Biomol. Struct.* 23:441–71
134. Sentenac H, Bonneaud N, Minet M, Lacroute F, Salmon JM, et al. 1992. Cloning and expression in yeast of a plant potassium ion transport system. *Science* 256:663–65
135. Shi J, Kim KN, Ritz O, Albrecht V, Gupta R, et al. 1999. Novel protein kinases associated with calcineurin B–like calcium sensors in Arabidopsis. *Plant Cell* 11:2393–405
136. Shin R, Berg RH, Schachtman DP. 2005. Reactive oxygen species and root hairs in *Arabidopsis* root response to nitrogen, phosphorus and potassium deficiency. *Plant Cell Physiol.* 46:1350–57
137. Shin R, Schachtman DP. 2004. Hydrogen peroxide mediates plant root cell response to nutrient deprivation. *Proc. Natl. Acad. Sci. USA* 101:8827–32
138. Sirichandra C, Wasilewska A, Vlad F, Valon C, Leung J. 2009. The guard cell as a single-cell model towards understanding drought tolerance and abscisic acid action. *J. Exp. Bot.* 60:1439–63
139. Song CP, Guo Y, Qiu Q, Lambert G, Galbraith DW, et al. 2004. A probable $Na^+(K^+)/H^+$ exchanger on the chloroplast envelope functions in pH homeostasis and chloroplast development in *Arabidopsis thaliana*. *Proc. Natl. Acad. Sci. USA* 101:10211–16
140. Sottocornola B, Visconti S, Orsi S, Gazzarrini S, Giacometti S, et al. 2006. The potassium channel KAT1 is activated by plant and animal 14-3-3 proteins. *J. Biol. Chem.* 281:35735–41
141. Spalding EP, Hirsch RE, Lewis DR, Qi Z, Sussman MR, et al. 1999. Potassium uptake supporting plant growth in the absence of AKT1 channel activity. *J. Gen. Physiol.* 113:909–18
142. Sutter JU, Campanoni P, Tyrrell M, Blatt MR. 2006. Selective mobility and sensitivity to SNAREs is exhibited by the *Arabidopsis* KAT1 K^+ channel at the plasma membrane. *Plant Cell* 18:935–54
143. Szczerba MW, Britto DT, Ali SA, Balkos KD, Kronzucker HJ. 2008. NH_4^+-stimulated and -inhibited components of K^+ transport in rice (*Oryza sativa* L.). *J. Exp. Bot.* 59:3415–23
144. Szyroki A, Ivashikina N, Dietrich P, Roelfsema MRG, Ache P, et al. 2001. KAT1 is not essential for stomatal opening. *Proc. Natl. Acad. Sci. USA* 98:2917–21
145. ten Hoopen F, Cuin TA, Pedas P, Hegelund JN, Shabala S, et al. 2010. Competition between uptake of ammonium and potassium in barley and *Arabidopsis* roots: molecular mechanisms and physiological consequences. *J. Exp. Bot.* 61:2303–15
146. Tsay YF, Ho CH, Chen HY, Lin SH. 2011. Integration of nitrogen and potassium signaling. *Annu. Rev. Plant Biol.* 62:207–26
147. Venema K, Quintero FJ, Pardo JM, Donaire JP. 2002. The *Arabidopsis* Na^+/H^+ exchanger AtNHX1 catalyzes low affinity Na^+ and K^+ transport in reconstituted liposomes. *J. Biol. Chem.* 277:2413–18
148. Véry AA, Davies JM. 2000. Hyperpolarization-activated calcium channels at the tip of *Arabidopsis* root hairs. *Proc. Natl. Acad. Sci. USA* 97:9801–6
149. Véry AA, Sentenac H. 2003. Molecular mechanisms and regulation of K^+ transport in higher plants. *Annu. Rev. Plant Biol.* 54:575–603

150. Vicente-Agullo F, Rigas S, Desbrosses G, Dolan L, Hatzopoulos P, et al. 2004. Potassium carrier TRH1 is required for auxin transport in *Arabidopsis* roots. *Plant J.* 40:523–35
151. Voelker C, Schmidt D, Mueller-Roeber B, Czempinski K. 2006. Members of the Arabidopsis AtTPK/KCO family form homomeric vacuolar channels *in planta*. *Plant J.* 48:296–306
152. Walker DJ, Leigh RA, Miller AJ. 1996. Potassium homeostasis in vacuolate plant cells. *Proc. Natl. Acad. Sci. USA* 93:10510–14
153. Wang R, Guegler K, Labrie ST, Crawford NM. 2000. Genomic analysis of a nutrient response in Arabidopsis reveals diverse expression patterns and novel metabolic and potential regulatory genes induced by nitrate. *Plant Cell* 12:1491–509
154. Wang Y, He L, Li HD, Xu J, Wu WH. 2010. Potassium channel α-subunit AtKC1 negatively regulates AKT1-mediated K^+ uptake in *Arabidopsis* roots under low-K^+ stress. *Cell Res.* 20:826–37
155. Wang Y, Wu WH. 2010. Plant sensing and signaling in response to K^+-deficiency. *Mol. Plant* 3:280–87
156. Wang YH, Garvin DF, Kochian LV. 2002. Rapid induction of regulatory and transporter genes in response to phosphorus, potassium and iron deficiencies in tomato roots. Evidence for cross talk and root/rhizosphere-mediated signals. *Plant Physiol.* 130:1361–70
157. Ward JM, Mäser P, Schroeder JI. 2009. Plant ion channels: gene families, physiology, and functional genomics analyses. *Annu. Rev. Physiol.* 71:59–82
158. Ward JM, Schroeder JI. 1994. Calcium-activated K^+ channels and calcium-induced calcium release by slow vacuolar ion channels in guard cell vacuoles implicated in the control of stomatal closure. *Plant Cell* 6:669–83
159. Wegner LH, Raschke K. 1994. Ion channels in the xylem parenchyma of barley roots. *Plant Physiol.* 105:799–813
160. Weinl S, Kudla J. 2009. The CBL-CIPK Ca^{2+}-decoding signaling network: function and perspectives. *New Phytol.* 184:517–28
161. White PJ, Broadley MR. 2003. Calcium in plants. *Ann. Bot.* 92:487–511
162. Wu P, Ma LG, Hou XL, Wang MY, Wu YR, et al. 2003. Phosphate starvation triggers distinct alterations of genome expression in Arabidopsis roots and leaves. *Plant Physiol.* 132:1260–71
163. Wu WH, Wang Y, Chul LS, Lan WZ, Luan S. 2010. Regulation of ion channels by the calcium signaling network in plant cells. In *Ion Channels and Plant Stress Responses*, Signal. Commun. Plants, ed. V Demidchik, F Maathuis, pp. 111–35. Berlin: Springer-Verlag
164. Wyn Jones RJ. 1999. Cytoplasmic potassium homeostasis: review of the evidence and its implication. In *Frontiers in Potassium Nutrition: New Perspectives on the Effects of Potassium on Physiology of Plants*, CSSA Spec. Publ., ed. DM Oosterhuis, GA Berkowitz, pp. 13–22. Atlanta: Potash Phosphate Inst.
165. Wyn Jones RJ, Pollard A. 1983. Proteins, enzymes and inorganic ions. In *Encyclopedia of Plant Physiology*, ed. A Lauchli, A Pirson, pp. 528–62. Berlin: Springer-Verlag
166. Xicluna J, Lacombe B, Dreyer I, Alcon C, Jeanguenin L, et al. 2007. Increased functional diversity of plant K^+ channels by preferential heteromerization of the Shaker-like subunits AKT2 and KAT2. *J. Biol. Chem.* 282:486–94
167. Xu J, Li HD, Chen LQ, Wang Y, Liu LL, et al. 2006. A protein kinase, interacting with two calcineurin B-like proteins, regulates K^+ transporter AKT1 in *Arabidopsis*. *Cell* 125:1347–60
168. Yuan L, Loque D, Kojima S, Rauch S, Ishiyama K, et al. 2007. The organization of high-affinity ammonium uptake in *Arabidopsis* roots depends on the spatial arrangement and biochemical properties of AMT1-type transporters. *Plant Cell* 19:2636–52
169. Zhang X, Ma J, Berkowitz GA. 1999. Evaluation of functional interaction between K^+ channel α- and β-subunits and putative inactivation gating by co-expression in *Xenopus laevis* oocytes. *Plant Physiol.* 121:995–1002
170. Zhao J, Cheng NH, Motes CM, Blancaflor EB, Moore M, et al. 2008. AtCHX13 is a plasma membrane K^+ transporter. *Plant Physiol.* 148:796–807
171. Zielinski RE. 1998. Calmodulin and calmodulin binding proteins in plants. *Annu. Rev. Plant Physiol. Plant Mol. Biol.* 49:697–725
172. Zimmermann S, Talke I, Ehrhardt T, Nast G, Müller-Röber B. 1998. Characterization of SKT1, an inwardly rectifying potassium channel from potato, by heterologous expression in insect cells. *Plant Physiol.* 116:879–90

Annual Review of Plant Biology
Volume 64, 2013

Contents

Indexes

Errata

An online log of corrections to *Annual Review of Plant Biology* articles may be found at http://www.annualreviews.org/errata/arplant