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RESEARCH HIGHLIGHT

The role of calcium sensor-interacting protein kinases in plant adaptation to potassium-deficiency: new answers to old questions

Anna Amtmann[1], Patrick Armengaud[1]

[1]*Plant Sciences Group, Institute of Biomedical and Life Sciences, University of Glasgow, Glasgow G12 8QQ, UK. a.amtmann@bio.gla.ac.uk*

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Potassium (K^+) is an essential macronutrient for all living organisms and large amounts are required for plant growth and development. In many regions of Asia K^+-fertilization has been neglected and soils have become K^+-depleted. K^+-deficiency in the field diminishes not only crop production but also leads to environmental problems due to inefficient usage and leaching of nitrate. Consequences of K^+-deficiency on crop production range from decreased biomass, nutritional quality and taste of the crops to inferior harvest and storage properties, as well as increased susceptibility to disease. Effects of K^+-deficiency on plant physiology include decreased photosynthetic rate, impaired tissue allocation of sugars and amino acids, decreased protein synthesis, and lack of control over turgor and gas exchange [1]. K^+-uptake and its re-distribution within the plant is facilitated by a plethora of membrane transport proteins displaying an astonishing diversity with respect to their affinity and selectivity for K^+, mode and direction of transport, tissue specific expression, membrane localization and regulation [2]. Microarray experiments have shown that – in contrast to transporters of other macronutrients – genes encoding K^+-transporters display surprisingly little responsiveness to the external nutrient supply [3]. This observation probably reflects that because of its vital role in maintenance of cell turgor and membrane potential K^+-transport has to respond very quickly to changes in the environment. Hence, post-translational control mechanisms are required.

Two recent studies have provided exciting new information on this issue. Wu and colleagues [4] and Luan and colleagues [5] identified a calcineurin B-like protein (CBL)-interacting protein kinase CIPK23 and two upstream elements, CBL1 and CBL9, as regulators of AKT1. AKT1 is a Shaker-type voltage-gated ion channel that mediates the uptake of K^+ at hyperpolarized membrane voltages [2]. The importance of AKT1 for K^+-uptake from the root environment had previously been proven in *Arabidopsis* akt1 knock-out mutants, which show impaired growth in low external K^+-concentrations, when high-affinity K^+-transporters are inhibited by ammonium [6]. The CIPK/CBL regulatory system links K^+-uptake to cytoplasmic Ca^{2+}, the most important secondary messenger in plants, and is thus reminiscent of the SOS signalling pathway, which controls cellular Na^+-homeostasis [2].

The paper by Pandey *et al*. in a recent issue of Cell Research [7] identifies another member of the CIPK family, CIPK9, as playing an important role in plant adaptation to K^+-deficiency. The authors report that two independent *Arabidopsis* T-DNA insertion knock-out lines for CIPK9 show impaired growth under conditions of low K^+-supply. The response is specific for K^+ as the phenotype is caused by depletion of the growth medium for K^+ but not for other ions. However, in contrast to the phenotype caused by knock-out of CIPK23, root and shoot total tissue K^+-contents were unchanged in cipk9 mutants compared to wildtype.

The study raises the question which processes other than K^+ acquisition are important for plant growth in K^+-deficient conditions. One possibility is that CIPK9, as CIPK23, interacts with a K^+-channel, but that unlike AKT1 this channel does not reside in the root plasma membrane. Experiments with K^+-selective microelectrodes have shown that under varying extracellular K^+-concentrations cytoplasmic K^+-concentrations in root cells are maintained at a constant level at the cost of vacuolar K^+ [8]. Thus, the

vacuolar K^+-pool is used as a flexible store for cellular K^+-homeostasis. Several K^+-permeable channels in the tonoplast could facilitate K^+ release from the vacuole under K^+-deficient conditions [2] but the question how these channels 'sense' the external K^+-concentrations has long puzzled researchers in the field. The possibility that CIPK9 directly regulates a vacuolar K^+-channel thereby linking channel gating to external K^+ via a cytoplasmic Ca^{2+} signal is therefore intriguing. K^+-homeostasis operates not only at the cellular level but also at the tissue level. This is apparent in the fact that K^+-deficiency symptoms appear first in older leaves. Effective re-location of K^+ from older into younger leaves requires regulation of plasma membrane and tonoplast K^+-transporters in a number of different cell types, and CIPK9 could be an essential component of this regulatory network.

Another possibility is that CIPK9 regulation targets aspects of plant adaptation to low K^+ that are not linked to K^+-transport. Although cellular and tissue K^+-homeostasis can protect metabolically active cells from serious K^+-deficiency for a limited period of time, it is clear that a plant that experiences long-term K^+-deficiency will have to re-prioritise its growth, development and metabolism to achieve maximal seed production with limited resources. Research in our lab has identified jasmonic acid (JA) as a potential central integrator of the adaptation process [9]. Microarray analysis showed that a large percentage of the K^+-responsive transcriptome is related to JA, and a rise of JA during K^+-deficiency, as well as the specificity of this response for K^+-deficiency, have since been confirmed [A Amtmann, P Armengaud, unpublished data]. JA is well known to play a role in growth inhibition, senescence and stomatal closure; processes that are crucial for plant adaptation to K^+-deficiency. Our microarray study also identified CIPK9 as being transcriptionally regulated by K^+, and subsequent profiling of the K^+-responsive transcriptome in JA-signalling mutants showed that CIPK9 regulation is independent of JA-signalling. In the light of these findings it is exciting that Pandey *et al*. [7] report enhanced expression of CIPK9 after wounding, another well-known stimulus for JA biosynthesis. CIPK9 could therefore be an essential upstream component of JA-mediated adaptive responses to K^+-deficiency.

A number of experiments are now required to further characterize the physiological role of CIPK9. Yeast two-hybrid assays should be carried out to identify both upstream (e.g. CBLs) and downstream (e.g. K^+-transporters) interactors of CIPK9. To test the possibility that CIPK9 is involved in more general aspects of plant adaptation to low K^+ cipk9 mutants should be subjected to microarray analysis and the transcriptional profile compared with available data from wildtype plants. To position CIPK9 within the K^+-signalling network, dependence of its transcriptional K^+-responsiveness to a putative ROS-upstream signal [10],

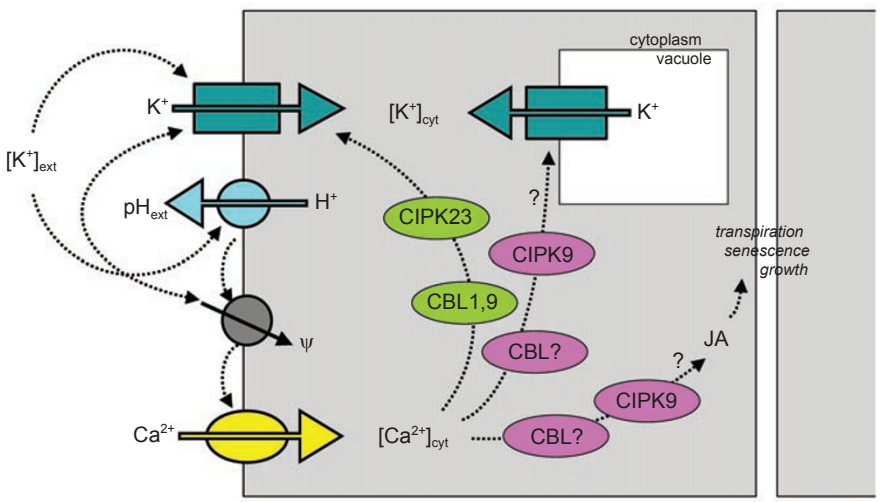

Figure 1 Putative functions of CBL/CIPK pathways in K^+-signalling. Through its effect on plasma membrane K^+- and H^+-conductance a decrease in external K^+ leads to membrane hyperpolarisation and subsequent activation of voltage-dependent Ca^{2+}-channels. Calcineurin B-like sensor proteins (CBLs) detect the rise in cytoplasmic Ca^{2+} and activate CBL-interacting protein kinases (CIPKs). Possible targets of CIPK regulation are plasma membrane K^+-channels facilitating K^+-uptake from the external medium, tonoplast K^+-channels mediating K^+-release from the vacuole, and upstream elements of hormonal pathways integrating a range of physiological adaptations.

and its requirement for a JA-downstream signal should be evaluated.

The recent discovery of the CIPK/CBL regulatory system has made a major contribution to our knowledge of how plants perceive external K^+ (Figure 1), a question that has occupied researchers for some 50 years. Future studies should aim to explore the function of this system in a whole-plant context, thus enhancing systemic understanding of a phenomenon that is not only of great scientific interest but also of central importance for sustainable agriculture worldwide.

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