Review

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Plant salt-tolerance mechanisms

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Crop performance is severely affected by high salt concentrations in soils. To engineer more salt-tolerant plants it is crucial to unravel the key components of the plant salt-tolerance network. Here we review our understanding of the core salt-tolerance mechanisms in plants. Recent studies have shown that stress sensing and signaling components can play important roles in regulating the plant salinity stress response. We also review key Na^+ transport and detoxification pathways and the impact of epigenetic chromatin modifications on salinity tolerance. In addition, we discuss the progress that has been made towards engineering salt tolerance in crops, including marker-assisted selection and gene stacking techniques. We also identify key open questions that remain to be addressed in the future.

Soil salinization and its impact on plants

Soil salinization is a growing problem for agriculture worldwide. Salt accumulation in arable soils is mainly derived from irrigation water that contains trace amounts of sodium chloride (NaCl) and from seawater [1,2]. Increased soil salt concentrations decrease the ability of a plant to take up water and, once Na^+ and Cl^- are taken up in large amounts by roots, both Na^+ and Cl^- negatively affect growth by impairing metabolic processes and decreasing photosynthetic efficiency [1,3]. Thus, plant salt stress can be subdivided into early-occurring osmotic stress and accumulating ionic Na^+ stress [4,5] with additional Cl^- stress (reviewed in [6]). Plants enact mechanisms to mitigate osmotic stress by reducing water loss while maximizing water uptake. Furthermore, plants minimize the harmful effects of ionic Na^+ stress by exclusion of Na^+ from leaf tissues and by compartmentalization of Na^+, mainly into vacuoles [5,7]. Despite these tolerance mechanisms, salt stress decreases crop yields and is leading to continuing loss of arable land. Such losses are compounded by the additional challenge that agriculture needs to provide enough nutrition for a world population that is rapidly expanding (estimated to reach 9.6 billion by the year 2050) and which has a steadily increasing quality of life [8,9]. In this context, engineering crops to enhance salt-tolerance mechanisms is a promising approach to address these challenges. In this review, we examine the key mechanisms that mediate plant salt tolerance and give an overview of recent literature on salinity stress sensing and signaling as well as regulation of gene expression as part of the salt stress response in plants. Furthermore, the understanding of the plant Na^+ transport network is updated and an evaluation of methods that can help with the engineering of salt-tolerant crops is made.

Sensory mechanisms of salt stress

To mount an effective response to cope with salt stress, plants have developed the ability to sense both the hyperosmotic component and the ionic Na^+ component of the stress. These two sensory modalities are evident in that some responses to NaCl remain distinct from responses to purely osmotic stress. A high salt concentration in the soil solution produces hyperosmotic stress on roots. To date, the molecular identities of plant hyperosmotic sensors and Na^+ sensors have remained elusive. The *Arabidopsis* (*Arabidopsis thaliana*) histidine kinase receptor protein HK1 has been shown to complement the loss of the yeast osmosensor Sln1 [10] and overexpression/loss-of-function lines exhibit drought and osmotic stress-associated phenotypes [11,12]. Plants exhibit many physiological responses to osmotic stress. However, recent research has shown that some of these responses are altered in *hk1* mutants, but others remain unaffected. Therefore, other proteins must still be perceiving the osmotic stress in the *hk1* mutant [13]. Plant hyperosmotic sensors are likely to be closely coupled with Ca^{2+} channels given that plants exhibit a rapid rise in cytosolic Ca^{2+} levels within seconds of exposure to NaCl or mannitol [14]. This Ca^{2+} response originates within the roots [15] and occurs in several cell types [16,17]. This observation has led to speculation that hyperosmotic stress may be sensed by a mechanically gated Ca^{2+} channel [18]. In support of a mechano-osmotic sensory modality, mutations affecting cuticle development interfere with many osmotic-induced responses, including downstream abscisic acid (ABA) production [19]. The cuticle provides structural support to the plasma membrane/cell wall and could alter the water diffusibility into the cell.

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Thus, altering cuticle properties may affect the mechanical properties of water stress on the cell. Other second messengers are also induced by salt or hyperosmotic stress and are linked to Ca^{2+} signaling, for example, reactive oxygen species (ROS) [20] (Figure 1), and *Arabidopsis* annexins have been reported to mediate both NaCl and ROS-induced Ca^{2+} responses [21,22]. Downstream of Ca^{2+}, kinases may become activated, including calcium-dependent protein kinases (CDPKs) [23,24] and calcineurin B-like proteins (CBLs) with CBL-interacting protein kinases (CIPKs) [25], which may transduce the hyperosmotic signal to downstream protein activity and gene transcription. Furthermore, transcription factors may be activated by Ca^{2+}/calmodulin directly, including calmodulin-binding transcription activators (CAMTAs) [26], GT element-binding-like proteins (GTLs) [27], and MYBs [28]. Although the rapid Ca^{2+} increase is a hallmark response to osmotic stress, there may also exist Ca^{2+}-independent osmotic sensory mechanisms. Genetic identification of osmotic and Na^+ sensors is likely to be instrumental in resolving these early sensory mechanisms.

Gene regulation in roots in response to salt stress

Transcription factors are integral in linking salt sensory pathways to many tolerance responses. Core sets of transcription factor family genes are differentially expressed in response to elevated external salinity [29], including basic leucine zipper (bZIP) [30], WRKY [31], APETALA2/ETHYLENE RESPONSE FACTOR (AP2/ERF) [32], MYB [33], basic helix–loop–helix (bHLH) [34], and NAC [35] families. These transcription factors, in turn, regulate the expression levels of various genes that may ultimately influence the level of salt tolerance of plants (Figure 1). To counteract the water potential decrease resulting from the osmotic component of enhanced salinity, genes relevant for inorganic ion uptake and osmolyte synthesis are upregulated [36]. To some extent, transcriptional regulation of these stress response genes in plants is mediated

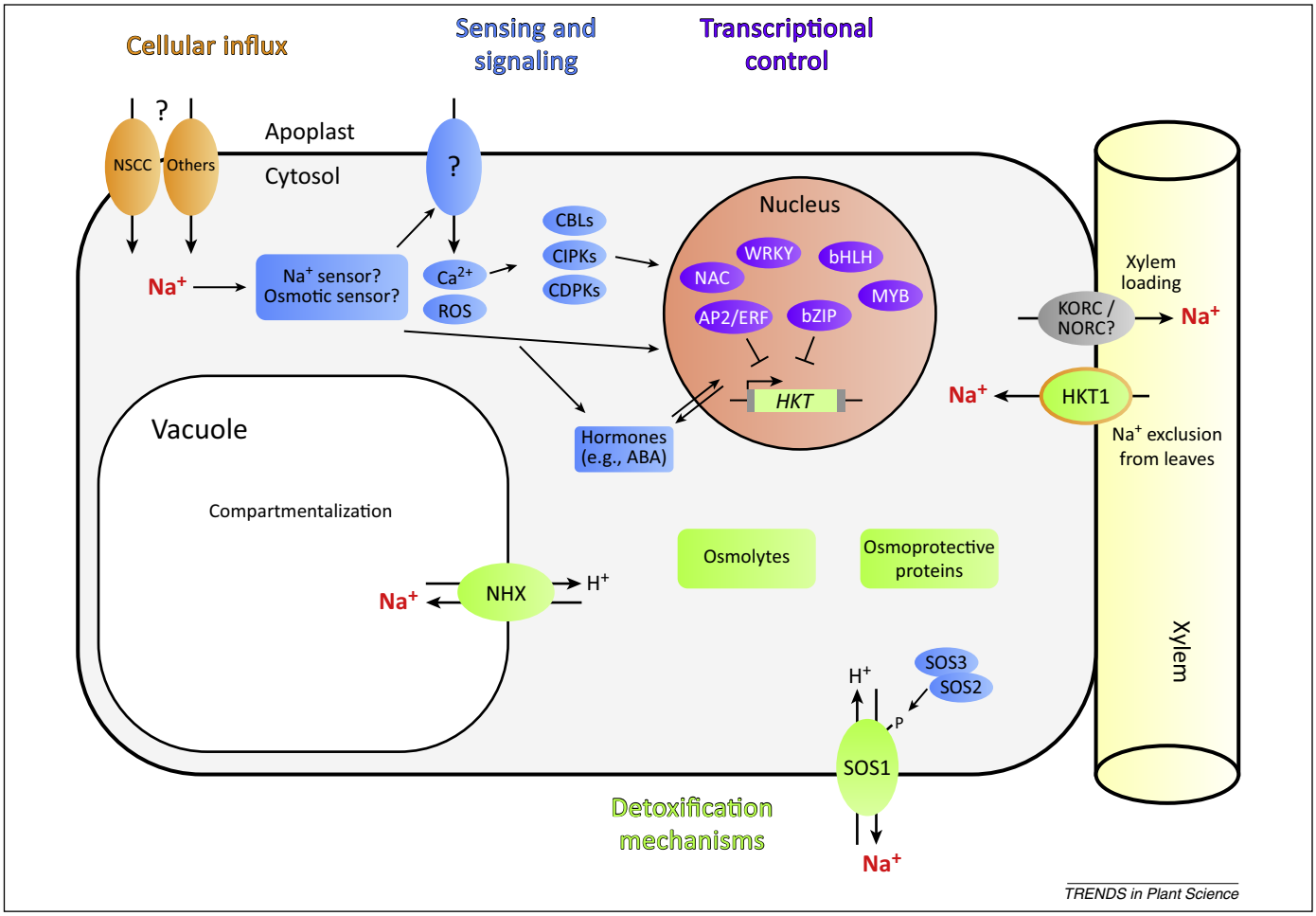

Figure 1. Overview of cellular Na^+ transport mechanisms and important components of the salt stress response network in plant root cells. Na^+ (depicted in red) enters the cell via NSCCs and other, as yet largely unknown membrane transporters (cellular Na^+-influx mechanisms highlighted in orange). Inside the cell, Na^+ is sensed by an as yet unidentified sensory mechanism. At the next step, Ca^{2+}, ROS, and hormone signaling cascades are activated. CBLs, CIPKs, and CDPKs are part of the Ca^{2+}-signaling pathway (sensing and signaling components highlighted in blue), which can alter the global transcriptional profile of the plant (transcription factor families in the nucleus depicted in purple; an AP2/ERF and a bZIP transcription factor that negatively regulate *HKT* gene expression are shown as an example). Ultimately, these early signaling pathways result in expression and activation of cellular detoxification mechanisms, including HKT, NHX, and the SOS Na^+ transport mechanisms as well as osmotic protection strategies (cellular detoxification mechanisms highlighted in light green). Furthermore, the Na^+ distribution in the plant is regulated in a tissue-specific manner by unloading of Na^+ from the xylem. Abbreviations: NSCCs, nonselective cation channels; ROS, reactive oxygen species; CDPKs, calcium-dependent protein kinases; CBLs, calcineurin B-like proteins; CIPKs, CBL-interacting protein kinases; AP2/ERF, APETALA2/ETHYLENE RESPONSE FACTOR; bZIP, basic leucine zipper; NHX, Na^+/H^+ exchanger; SOS, SALT OVERLY SENSITIVE.