Review

CellPress

Tuning plant signaling and growth to survive salt

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Salinity is one of the major abiotic factors threatening food security worldwide. Recently, our understanding of early processes underlying salinity tolerance has expanded. In this review, early signaling events, such as phospholipid signaling, calcium ion (Ca^{2+}) responses, and reactive oxygen species (ROS) production, together with salt stress-induced abscisic acid (ABA) accumulation, are brought into the context of long-term salt stress-specific responses and alteration of plant growth. Salt-induced quiescent and recovery growth phases rely on modification of cell cycle activity, cell expansion, and cell wall extensibility. The period of initial growth arrest varies among different organs, leading to altered plant morphology. Studying stress-induced changes in growth dynamics can be used for screening to discover novel genes contributing to salt stress tolerance in model species and crops.

How flexibility enables increased abiotic stress tolerance

Plants are flexible organisms, with the potential to develop a plethora of morphological patterns depending on the growth conditions to which they are exposed. This morphological flexibility has enabled plants to colonize almost every corner of the globe and to survive in the harshest conditions. Today, the demand for food is on the rise, while arable lands are becoming scarce [1]. To meet future demands, an increase in plant yield in environmentally challenging conditions is essential.

High soil salinity is one of the major abiotic factors limiting crop yield. Halophytes have evolved functional and structural adaptations, such as excretion of salt through secretory glands, ensuring their survival in saline environments [2]. Most crop plants are glycophytes, which are not able to withstand high levels of salt stress and do not have morphological structures allowing salt excretion. By contrast, glycophytes exhibit natural variation in their salinity tolerance between as well as within plant species. This natural variation can be used for the identification of genetic components underlying salinity tolerance in glycophytes [3,4]. Populations comprising natural accessions of model and crop species, such as Arabidopsis (*Arabidopsis thaliana*), rice (*Oryza sativa*), and maize (*Zea mays*), are now broadly used for genome-wide association studies (GWAS). Intraspecific variation can also be a valuable source of allelic variation in previously characterized molecular players, leading to new insights into the salinity tolerance mechanisms [5]. To produce new crop varieties, with reliable and high yield in saline environments, the physiology and allelic variations underlying local adaptations to salinity stress need to be understood.

Salt stress limits plant growth by increasing the osmotic potential of the soil and, thus, decreasing water uptake by the roots. In the long term, accumulation of sodium ions (Na^+) in the shoot compromises plant growth by reducing photosynthesis rate if Na^+ are not compartmentalized at the cellular or intercellular level. Growth of individual plant organs is reduced by salinity stress with varying magnitude, leading to altered general plant morphology, such as a change in the root:shoot ratio [3]. These salt-induced changes in plant morphology are likely to affect the performance of a plant under saline conditions. Recent discoveries in the field of salt stress physiology provide increased understanding of plant acclimation responses resulting in altered plant development and ultimately increased salinity stress tolerance. In this review, we summarize the signaling pathways crucial for early salt stress signaling and the acclimation responses leading to altered plant morphology, which significantly affects plant performance under salt stress conditions.

First aid: early signaling responses to salt stress

The first reactions of a plant to salinity occur within seconds to hours upon exposure to salt stress. An overview of early signaling responses is presented in Figure 1. Sodium ions enter the root epidermal and cortical cells through nonselective cation channels (NSCCs) [6]. The influx of Na^+ induces depolarization of the plasma membrane, activating potassium (K^+) outward-rectifying channels (KOR) and reducing net passive potassium uptake through inward-rectifying K^+ channels [7].

Salt stress additionally causes an increase in osmotic soil potential, resulting in water loss and reduction in turgor pressure. One of the mechanisms potentially involved in sensing changes in turgor pressure involves activation of mechanosensitive receptor kinase cyclase, resulting in rapid (<5 s) accumulation of cGMP, which inhibits sodium influx by deactivation of NSCC channels [8]. Accumulation of cGMP is also thought to underlie

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Keywords: salt stress; developmental plasticity; cellular signaling; Root System Architecture; osmotic stress; potassium starvation; root development; natural variation.

1360-1385/

 http://dx.doi.org/10.1016/j.tplants.2015.06.008

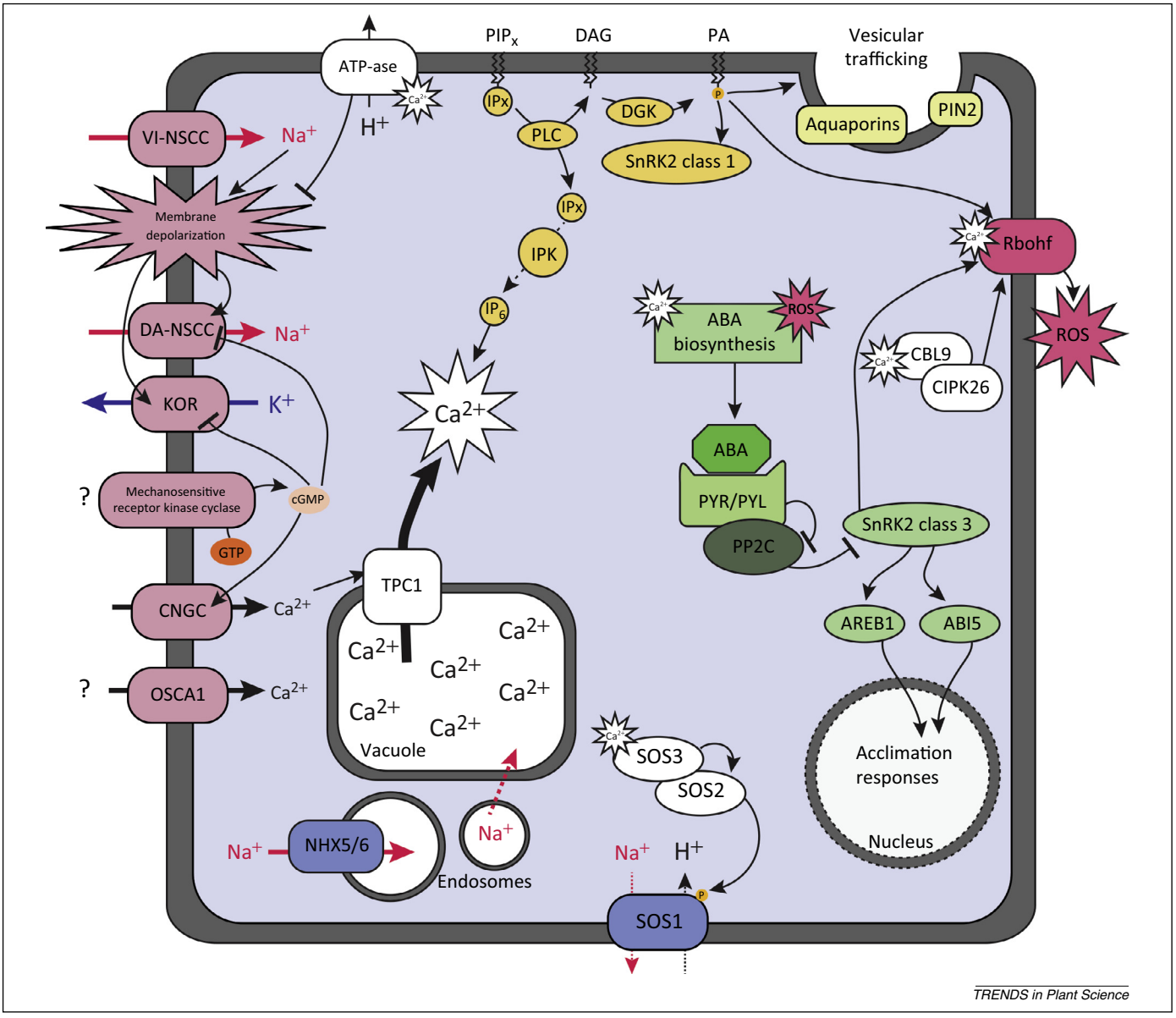

Figure 1. Early signaling in response to salinity stress. Upon salt stress exposure, sodium ions (Na^+) enter the cell through voltage independent nonselective cation channels VI-(NSCCs), inducing membrane depolarization [6], depolarization activated NSCC (DA-NSCC) and potassium ion (K^+) outward rectifier channels (KOR) [7]. Additionally, changes in turgor pressure might be sensed through mechanosensitive receptor kinase cyclases, inducing production of cGMP or reduced hyperosmolality-induced calcium ion ([Ca^{2+}]) increase 1 (OSCA1), a plasma membrane-localized Ca^{2+} channel [10]. cGMP inhibits KOR and induces opening of cyclic nucleotide gated channels (CNGC), responsible for apoplastic Ca^{2+} influx [9]. Increased cytosolic Ca^{2+} release is accomplished by activation of slow-activating vacuolar/two pore channel 1 (SV/TPC1) [12]. High cytosolic Ca^{2+} activates among others calcium-binding calmodulin (CBL9). This interacts with calcium-induced protein kinase (CIPK26), which targets respiratory burst oxidase homolog F (RbohF), inducing production of reactive oxygen species (ROS) [86]. Salt stress-induced phospholipase C (PLC) contributes to production of phosphatidic acid (PA) via phospholipase D (PLD)/diacylglycerol kinase (DGK) pathways, as well as an increase in inositol phosphate 6 (IP_6) through phosphorylation of inositol phosphates (IP) by IP kinase [29]. The increase in PA affects vesicular trafficking of PIN-FORMED 2 (PIN2) [76] and possibly aquaporins, abscisic acid (ABA)-independent sucrose non-fermenting like kinase 2.6 (SnRK 2.6) class 1 proteins, and the activity of RbohF [31]. An increase in cytosolic Ca^{2+} and ROS induces ABA accumulation [21], which is sensed by pyrabactin resistance (PYR)/PYR1-like (PYL) receptors, releasing ABA-dependent protein phosphatase 2C (PP2C) from ABA-dependent SnRK2 class 3, which in turn activates the transcription factors ABA responsive element-binding protein (AREB1) and ABA-insensitive 5 (ABI5) and increases ROS through activation of RbohF [25]. Reduction of cytosolic sodium is guided through activation of the salt overly sensitive (sos) signaling pathway [34], compartmentalization of sodium in endosomes by Na^+/H^+ exchanger 5/6 (NHX5/6) [37], and possibly subsequent fusion with the vacuole. Positive and negative regulatory actions are indicated by arrows and lines with bars, respectively.

initial Ca^{2+} signal mobilization through activation of cyclic nucleotide gated channels (CNGC) [9] allowing influx of apoplastic Ca^{2+} into the cytosol. Another osmo-sensing mechanism involves reduced hyperosmolality-induced [Ca^{2+}] increase 1 (OSCA1), a plasma membrane-localized protein that is responsible for increased Ca^{2+} influx in response to osmotic stress [10]. However, both proposed osmo-sensing mechanisms are yet to be validated for their role in salt stress-induced osmotic signaling and induction of Ca^{2+} influx.

The initial increase in cytosolic Ca^{2+} occurs within seconds after application of salt stress and is further facilitated through activation of slow-activating vacuolar/two pore channel 1 (SV/TPC1), resulting in release of vacuolar Ca^{2+} [11]. In response to local salt treatment, mobilization of vacuolar Ca^{2+} through TPC1 results in