PLANT SIGNALING & BEHAVIOR
2019, VOL. 14, NO. 11, e1665455 (15 pages)
https://doi.org/10.1080/15592324.2019.1665455

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REVIEW

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Calcium signaling and salt tolerance are diversely entwined in plants

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ABSTRACT

In plants dehydration imposed by salinity can invoke physical changes at the interface of the plasma membrane and cell wall. Changes in hydrostatic pressure activate ion channels and cause depolarization of the plasma membrane due to disturbance in ion transport. During the initial phases of salinity stress, the relatively high osmotic potential of the rhizosphere enforces the plant to use a diverse spectrum of strategies to optimize water and nutrient uptake. Signals of salt stress are recognized by specific root receptors that activate an osmosensing network. Plant response to hyperosmotic tension is closely linked to the calcium (Ca^{2+}) channels and interacting proteins such as calmodulin. A rapid rise in cytosolic Ca^{2+} levels occurs within seconds of exposure to salt stress. Plants employ multiple sensors and signaling components to sense and respond to salinity stress, of which most are closely related to Ca^{2+} sensing and signaling. Several tolerance strategies such as osmoprotectant accumulation, antioxidant boosting, polyaminses and nitric oxide (NO) machineries are also coordinated by Ca^{2+} signaling. Substantial research has been done to discover the salt stress pathway and tolerance mechanism in plants, resulting in new insights into the perception of salt stress and the downstream signaling that happens in response. Nevertheless, the role of multifunctional components such as Ca^{2+} has not been sufficiently addressed in the context of salt stress. In this review, we elaborate that the salt tolerance signaling pathway converges with Ca^{2+} signaling in diverse pathways. We summarize knowledge related to different dimensions of salt stress signaling pathways in the cell by emphasizing the administrative role of Ca^{2+} signaling on salt perception, signaling, gene expression, ion homeostasis and adaptive responses.

ARTICLE HISTORY
Received 24 July 2019
Revised 26 August 2019
Accepted 28 August 2019

KEYWORDS
Calcium; salinity; secondary messengers; osmoprotection; signaling pathway

Introduction

Soluble salts are abundantly available in the soil as sources of essential nutrients for plant growth and development. Nevertheless, when the amount of salts in the soil exceeds a particular threshold, plant health and productivity are impaired. Over-salinized soil is a global concern that threatens approximately 20% of irrigated land with significant crop reduction.[1,2] Salt stress frequently inhibits growth and influences key developmental stages, triggering premature senescence and death during prolonged exposure.[3,4] Salinization has an osmotic effect on plant cells, leading to their shrinkage by dehydration. This effect is transient, with cells regaining their original volume within few hours of their initial exposure to chronic salt concentrations. Despite this recovery, cell division and mostly cell expansion are negatively impacted, resulting in reduced leaf and root growth. Various plant developmental stages comprising germination, vegetative and reproductive growth are also affected by salt stress. Plants dynamically respond to salt exposure, which in extreme conditions their response is accompanied with programmed cell death.[5] Osmotic stress caused by a high salt environment constrains water uptake from rhizosphere, resulting in nutrient deficiency and dehydration damage to the plant. Plant responses to these constrains range from regulating external ion uptake to dynamic ion hemostasis pattern. For instance, phosphorus (P) uptake is reduced under saline conditions because Ca^{2+} inhibits P ions uptake by the root system, while in plant cells an increase in sodium (Na^+) influx is accompanied with a decrease in potassium (K^+) uptake and transport.[6–8] As K^+ is a pivotal component for modulating ion hemostasis and osmotic pressure, plants should define an adaptable strategy to regulate cation transporters in the plasma membrane (PM) to fulfill hampered ion hemostasis during saline stress event.

Plants utilize multiple strategies to alleviate salt stress damage. Na^+ influx is powered by voltage-insensitive monovalent-cation channels (VIC).[9] These channels are regulated using bivalent cations such as Ca^{2+} to maintain ion hemostasis. Over the course of evolution, plants have developed the capacity to detoxify excessive Na^+ levels that accompany high salt concentrations. Recent studies have identified two classes of Na^+/H^+ antiporters responsible for Na^+ detoxification:

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Na^+/H^+ exchanger (NHX1) and salt overly sensitive (SOS1), respectively localized to the vacuole and PM.[10,11] Alternatively, to maintain an optimal osmotic pressure and alleviate the salt stress damage, Na^+ accumulation in the cell is mediated by compartmentalization in the vacuole.[12] This regulatory mechanism is triggered when the cell senses salt stress. Although the molecular mechanism of hyperosmotic perception in plants is still unknown, in yeast these signals are perceived in the PM and communicated downstream by means of the mitogen-activated proteins kinase (MAPK) pathway.[4] Chloride (Cl^-) ions are also toxic when they accumulate in the cytoplasm. Plants that are capable of Cl^- compartmentalization in both shoot and root systems are potentially tolerant to NaCl stress. Alternatively, regulation of Cl^- transport has also been suggested for induction of salt tolerance.[13] In plant cells, ion concentration is directly regulated by cation-chloride co-transporters (CCCs).[14–17] CCCs function has been described in mammals in which K^+ and/or Na^+ with the anion Cl^- transport in a 1:1 ratio and promote electroneutral transport.[18]

Under salinity stress, abscisic acid (ABA) biosynthesis is up-regulated in guard cells, resulting in stomatal closure. This reduces photosynthetic capacity and triggers photoinhibition and oxidative stress. The biosynthesis and signaling activity of other phytohormones such as ethylene are also influenced by salinity stress. Induction of ethylene synthesis under a variety of stresses including salinity stress is well known. Biosynthesis of ethylene or its direct precursor, ACC (1-aminocyclopropane-1-carboxylate) is induced to a remarkable degree by salt stress.[19] Therefore, ethylene accumulates in plants encumbered with salt shock. Transgenic plants, with ethylene overexpression display enhanced stress tolerance. Nevertheless, inconsistent findings also suggest an inhibitive effect of ethylene in the induction of salt tolerance. These contradictions lead us to speculate a dynamic mode of action for ethylene based on its concentration. In fact, various levels of ethylene may play distinct roles in regulation of salt stress responses. However, the mechanism/s controlling ethylene levels under salt stress condition could be a major point of interest. Earlier reports showed that under saline stress conditions, Ca^{2+}-dependent protein kinases (CDPKs) phosphorylate the ACC synthase (ACS) protein stabilizing its conformation, leading to an increased ethylene content.[20] Ca^{2+} levels act as a secondary messenger, becoming elevated early on when plants encounter biotic and abiotic stresses.[21,22] Points of convergence in the ethylene and Ca^{2+} signaling pathways have been well characterized.[23–26] Ethylene compromises the crosstalk between CDPK and MAPK. In general, the perception of salinity stress at the PM triggers a plethora of responses ranging from Ca^{2+} oscillation, gene expression, changes in phytohormone homeostasis and alteration in osmotic pressure. Furthermore, activated signaling machinery will ultimately lead to the saline stress responses. However, among all, Ca^{2+} oscillation plays multifaceted roles to rule out detrimental effects of saline shock by building up multiple cross nods through generation of dynamic amplitudes.[27] This makes it capable of adjusting a range of variable downstream signaling cascades and influences the plant compatibility.

In this review, recent findings about Ca^{2+} signaling-related salt stress responses are discussed. In light of these findings, Ca^{2+} involvement in salt stress responses, with emphasis on salt perception, signaling events and Ca^{2+}-mediated metabolic blueprints, will be discussed. Taken as a whole, these results underscore the fact that Ca^{2+} signaling and salt stress signaling and responses are inseparable.

Mechanism of salt stress perception

In plants, strong growth inhibition in different organs has been reported as a result of exposure to salt stress.[28] Na^+ influx into the cell is mediated via the Na^+ transporter HKT.[29] However, due to the high hydrated ionic radii similarity between Na^+ and K^+, PM ion transporters are generally not capable of discriminating their influx. The result is Na^+ toxicity during exposure to high salt concentrations. This unidirectional influx disturbs various enzymatic processes and due to lack of need for high level of Na^+, it becomes parallel with osmotic pressure impairment. Growth retardation can be due to changes in cell wall structure and disturbed cell division and expansion.[30] Hampered growth, particularly in the leaves, leads to a sharp decline in plant biomass since reduced leaf area is accompanied by lower photosynthetic efficiency. It has been shown that salt-tolerant maize plants exhibit higher cell extensibility under salt stress.[31] By contrast, hypersensitive Arabidopsis mutants are defective in cell-wall organization.[32] Although the mechanism underlying cell wall expansion under osmotic shock is not completely known, cell-wall integrity during salt stress has been associated with the FERONIA (FER) receptor kinase activity. FER, a PM localized receptor kinase, plays a critical role in maintaining cell wall structure in plants exposed to saline shock; this role is mainly related to the Ca^{2+} signaling cascade. Upon exposure to salt stress, cell wall structure is damaged by Na^+; this damage is perceived by FER which in turn activates a Ca^{2+} channel via unknown pathway, resulting in a transient Ca^{2+} signaling in the root cells (Figure 1).[28,33] Na^+ is sensed by salt sensitive receptors comprising SOS1, Na^+/H^+ antiporters, histidine kinases, AHK1/ATHK1[34–36] and nonselective cation channels (NSCCs).[29,37] Elevation of Na^+ concentration in the surrounding environment leads to an influx of Na^+ into the cells through NSCCs. The two main categories of NSCCs in *A. thaliana* are cyclic nucleotide gated channels (CNGCs) and glutamate-activated channels (GLRs). CNGCs contribute to the regulation of ion homeostasis, which positively correlates with salt tolerance in Arabidopsis. CNGCs have also been proposed to be involved in Ca^{2+} influx and signaling.[38,39] CNGS interacts with calmodulin (CaM), a Ca^{2+}-modulated protein, in IQ-motif and regulates downstream signaling (Figure 1).[40] Notably polymorphisms in IQ-peptide has been reported which suggests that there are diverse modes of action for CaM signaling. This explains how Ca^{2+}-CaM binding to the CNGS induces flexible machinery to fine tune induced Ca^{2+} signals.

In animal cells, Na^+/Ca^{2+} exchangers (NCXs) play important roles in Ca^{2+} homeostasis. Bioinformatic approaches in Arabidopsis have revealed a *AtNCL* gene encoding a PM-

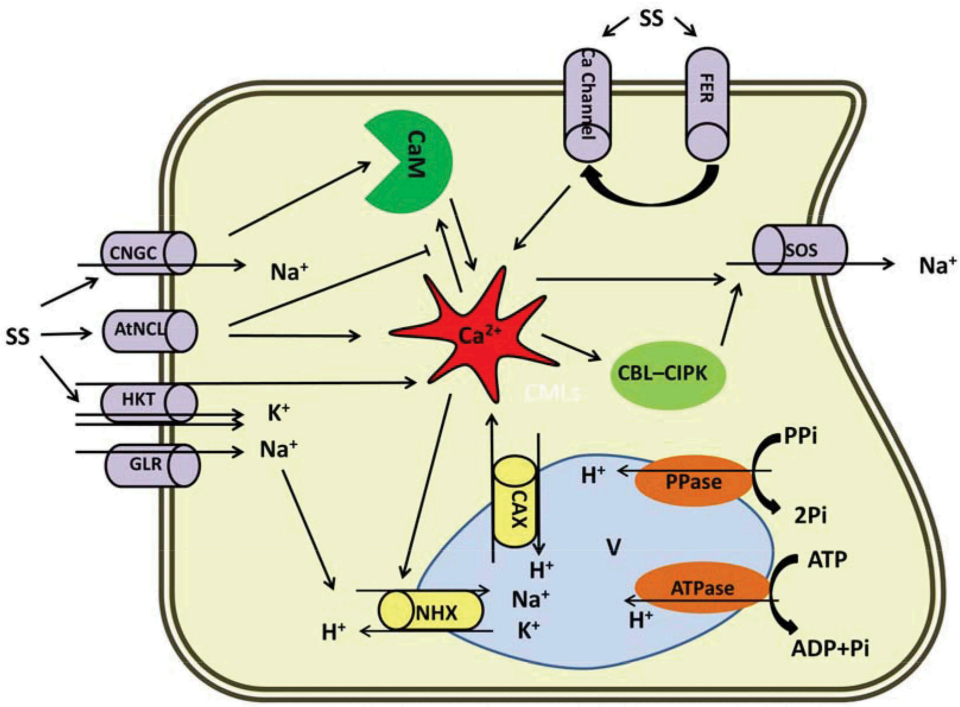

Figure 1. Diagrammatic representation of the Ca^{2+} role in salt stress responses in plants. Prominent role of Ca^{2+} under salt has been proposed in this scheme. Na^+ influx to the cytosol is mediated by CNGC, HKT, GLR and AtNCl. Na^+ efflux is derived by SOS. NHX and CAX are involved in Na^+/H^+ and Ca^{2+}/H exchanges through vacuolar membrane, respectively. Ca^{2+} in the center implicates the Na^+ current either by direct interaction with ion channels or indirectly through Ca^{2+} related modules (CBL-CIPK and CaM). FER activates Ca^{2+} channels via unknown pathway and bears a transient Ca^{2+} signaling. AtNCL: Na^+/Ca^{2+} exchanger-like protein, CAX: Vacuolar H^+/Ca^{2+} antiporter, CNGS: Cyclic nucleotide gated channels, FER: FERONIA, GLR: Glutamate-activated channels, HKT: Na^+ transporter, NHX: Vacuolar located Na^+/H^+ antiporters, SOS: Salt overly sensitive, SS: Salt stress.

localized protein that has the capacity to bind Ca^{2+} and possesses NCX-like activity.[41] It has been known that *AtNCL* is highly expressed under salt stress leading to Ca^{2+} ions being extruded (Figure 1). Consistently, plants defective in AtNCL functions exhibit elevated cytosolic Ca^{2+} levels and root surface Ca^{2+} fluxes.[42,43] This suggests that AtNCL terminates Ca^{2+} signaling under salt stress, although because of availability of various transporters in response to Ca^{2+} signaling, a dynamic interaction between transporters across the PM is highly likely.

The potential functions of the SOS signaling pathway that are involved in the Na^+ exclusion and ion homeostasis have been well described under salt stress.[44,45] Previous studies reported that elevated Ca^{2+} levels can protect plants from Na^+ toxicity. When Na^+ is sensed by the root, the SOS signal transduction cascade is activated in the cell through Ca^{2+} oscillation and inhibits excessive ion accumulation generated by salt stress. Elevated concentrations of the Ca^{2+} generated by continuous Na^+ influx into the cell activates the SOS proteins (a Ca^{2+} binding protein), in return SOS antiporters (such as SOS1) contribute to the Na^+ exclusion and cellular ion (Na^+/K^+) homeostasis.[46] This retro-controlling mechanism mediated by Ca^{2+} signaling balances the Na^+ concentration in root cells under saline conditions. Salt stress signaling is regulated by ethylene biosynthesis and signaling in *sos2* and *sos3* mutants; changes in the expression of ethylene responsive genes (ERF) has been observed due to exposure to salt stress.[47] Recent studies provide evidence that activation of SOS2 gene is responsive to phosphorylation and activation of ethylene insensitive 3 (EIN3), which leads to ethylene and salt-inducible ERF1 (ESE1) expression. This implies that the function of the SOS gene is closely linked to ethylene signaling during plant response to abiotic stresses.

Earlier reports pointed out that Ca^{2+} and Na^+ can improve plant growth, photosynthesis, water and ion transport and these roles are mainly related to the direct interaction of Na^+–Ca^{2+} at the surface of the PM, which is powered by Ca^{2+} signaling.[48] Lynch and coworkers for the first time reported the involvement of Ca^{2+} in salt stress responses by discovering a rapid rise in intracellular Ca^{2+} in root protoplasts of maize (*Zea mays* L.) within seconds of being exposed to saline shock.[49] Furthermore, induction of Ca^{2+} signals in response to osmotic and salt stresses are demonstrated by aequorin-based luminescence imaging of Ca^{2+} reporter protein.[50] Ca^{2+} signaling is now regarded as a secondary messenger in stress signaling pathways, such as salt stress.[51–54] It is noteworthy that, elevation in cytosolic Ca^{2+} in response to osmotic stress appeared to be a conserved signaling event in Plantae kingdom, which is manifested by the similar function across the higher plants to the basal-branching moss taxon *Physcomitrella patens*.[55,56] The Ca^{2+} signaling pathway initiates with elevation in cytosolic Ca^{2+} content and is regulated by Ca^{2+} transporters such as cyclic CNGCs, doubled-pore Ca^{2+} channels (TPCs), Ca^{2+}-ATPases and a vacuolar H^+/Ca^{2+} antiporter (CAXs) (Figure 1).[57] Moreover, it was postulated that an initial Ca^{2+}-dependent signaling network involves in the salt stress responses comprising Ca^{2+} transport and downstream targets, such as CaM, CMLs (CaM-like

protein), CDPKs, CBLs (calcineurin B-like protein) and CIPKs (CBL-interacting protein kinase).[52,53]

Both osmotic and ionic stresses have been implicated in triggering similar Ca^{2+} oscillations in Arabidopsis, suggesting a unique Ca^{2+} readout under both stress events.[58,59] The amplitude and duration of the Ca^{2+} transient are determined by the activity of vacuolar membrane H^+/Ca^{2+} antiporter (Vcx1p) and endomembrane localized Ca^{2+}-ATPases.[60,61] Ca^{2+} transients are also involved in activation of responsible channels for ion influx and energy dependent transport systems that contribute to the divalent cation compartmentalization. It has been reported that the salt stress-induced elevation in cytosolic Ca^{2+} and the new cytosolic Ca^{2+} status is regulated by CAX1, ECA (ATP-driven Ca^{2+} pump) and ACA Ca^{2+}-ATPases (auto-inhibited Ca^{2+}-ATPases).[62] In general, two lines of evidence suggest that Ca^{2+} triggers salt stress responses: first, by direct inhibition of Na^+ influx and second, as a downstream signaling component in response to salt stress.[63]

Ca^{+2} signaling and regulation of ion channels under salt stress conditions

NaCl is an immense challenge for salt-imposed plants; as the process of accumulation, exclusion, translocation and organelle sequestration of Na^+ and Cl^- are the major cause of salt-induced damages in plants.[64,65] Na^+ is the most abundant toxic ion in saline soils to enter the plant cells through Na^+-permeable transporters, by way of an unknown molecular mechanism. It was assumed that Na^+ can enter to the cell through nonselective cation transporters, such as K^+ transporters. When plant roots encounter with a high dosage of Na^+, three outcomes explain the exorbitant Na^+ influx to the epidermal cells; (i) accumulation in the vacuoles, (ii) redirection to the soil or apoplastic area, and (iii) shift into the adjacent cells via plasmodesmata.[66] In these ways, numerous transporters, ions, Ca^{2+} sensors, and their downstream interacting counterparts function in association to regulate the efflux of excess Na^+ ions and to gain the ion hemostasis.[67,68] Hyperosmolarity-gated Ca^{2+} permeable channel (OSCA1), a putative candidate for perception of salt stress,[69] Na^+/H^+ antiporters (NHX), V-type H^+-ATPase and H^+-PPase are responsible for compartmentalization of Na^+ into the vacuoles (Figure 1). These processes require the activation of a complex signaling network, which is initiated by Ca^{2+} signals in response to high levels of Na^+[70,71]

Investigating NaCl-induced Ca^{2+} signaling in plants using ratiometric Ca^{2+} sensor, YC3.6, revealed that the localized application of NaCl results in a drastic and transient elevation in local Ca^{2+} and triggers a systemic feedback in distinct sites.[72] However, it is not clarified whether physical (osmotic), chemical (ionic) or both features of Na^+ trigger Ca^{2+} signaling responses.[56] Under salt stress, cytosolic Ca^{2+} transiently activates CaM protein, which switches on the signaling networks through the calcineurin pathway so that salt-induced damage associated with high Na^+ levels is mitigated.[73] One of these networks consists of a Ca^{2+} sensor protein-kinase network like CBL-CIPK signaling complex that modulates protein transporters and serve the K^+ and Na^+ influx and transport to regulate ion hemostasis under salinity (Figure 1).[74]

An Na^+ transporter was discovered to function in wheat as a 'high-affinity K^+-transporter' and referred to as HKT according to its similarity to the high-affinity K^+ transport in bacteria and fungi.[75] In the absence of K^+, this membrane protein is capable of Na^+ transport with higher capacity (26 fold);[76] hence HKT was further known as Na^+–K^+ symporter.[77] Notably, it has been identified that a rice HKT protein encodes by OsHKT2;4 also functions as a Ca^{2+}-permeable NSCC.[75] It has also been reported that Na^+ transport via NSCCs is sensitive to Ca^{2+}, which may explain the inhibitory effect of Ca^{2+} on Na^+ influx into the root epidermal cells. However, it remained controversial that whether the Ca^{2+} function is associated with the intracellular regulatory protein or may be regulated independently.

Different Na^+ transport modes have also been suggested in plants by investigation of *sos3-1 hkt1* double mutants in Arabidopsis. It was found that increased extracellular Ca^{2+} impedes the *hkt1* knockout mutant to suppress Na^+ sensitivity of *sos3*-1, leading researchers to postulate about the inhibitory effect of Ca^{2+} on Na^+ influx in plants.[63] Likewise, analysis of plant cell patches revealed the existence of both Ca^{2+} in/sensitive Na^+ transport responses.[63,78] According to the electrophysiological and in plants mutants dissection studies, two Na^+ influx classifications have been defined in plants; one is directly inhibited by Ca^{2+}, and the other is HKT.[63] Given that Ca^{2+} functions as second messenger, the most probable scenario could be that the HKT links Ca^{2+} to the downstream signal transduction events.[79] Consistently, a recent report has revealed that MtCML40 in *MediCago truncatula* triggers a salt stress response by targeting MtHKT-dependent Na^+ accumulation.[80] As Ca^{2+} has potentially displayed an array of physiological roles in both signaling and nutrient uptake, HKT could be considered as a Ca^{2+} mediating candidate in modulation of plant growth and development under salt stress.[75]

Na^+/H^+ (NHX) transporter has been suggested as a salt tolerance responsive transporter, since its overexpression is associated with increased tolerance to salt stress and conversely, plants with truncated NHX exhibit decreased tolerance.[81–83] Moreover, NHX5/NH6X double mutants are hypersensitive to salt stress.[84] Another NHX member localized to the PM, namely SOS1, has been suggested to contribute to Na^+ loading into xylem vessels, though SOS1-related genes that are expressed in epidermal cells are rather low. These all can imply that NHX resembles to contribute in systemic retrieval and distribution of Na^+ in plants.[66]

Salt susceptible mutants have shown K^+ deficiency, which is consistent with the challenge of K^+ and Na^+ influx through similar transporter.[85–87] Notably, independent influx of Ca^{2+} maintain its regulatory effect on K^+/Na^+ levels irrespective of K^+/Na^+ ratio. However, Ca^{2+} mainly contributes to K^+ uptake, which results in a fine tuning of ion transport particularly when excessive levels of Na^+ are present.[88] Ca^{2+} may function in ion transport by interacting with NSCCs. Alternatively, as a second messenger, Ca^{2+} signaling triggers CBL–CIPK network with further effect on the SOS signal transduction pathway.[63,66,85] In fact, under salinity, a causal link occurs between Na^+ level regulated by NHX and SOS function

modulated by CBL–CIPK signaling network, which is built up by Ca^{2+} signaling pathway.

To date several mutants of SOS genes have been empirically identified by a positional cloning approach.[89] The SOS pathway consists of three major components including SOS3, a Ca^{2+} sensor (a myristoylated Ca^{2+}-binding protein), SOS2 a serine/threonine protein kinase and SOS1 that functions as PM-Na^+/H^+ antiporter.[89–91] According to previous studies, involvement of Ca^{2+} in salt stress signaling facilitates ion hemostasis.[54,92,93] Salt perception by PM sensors induces the cytoplasmic Ca^{2+} oscillation. Furthermore, Ca^{2+} read out, sensed by SOS3 (also known as CBL4), is decoded by Ca^{2+}-sensing proteins such as CBLs and their cooperative partners, CIPKs (Figure 2) [94] and ultimately brings SOS3 and SOS2 interaction. The myristoylation motif of SOS3 results in SOS3/SOS2 protein kinase complex, which further phosphorylates and activates SOS1. SOS1 facilitates excess of Na^+ ions efflux and thus, contributes to Na^+ ion homeostasis and long distance Na^+ transport (Figure 2).[95–97] SOS2 is also found to function in ion homeostasis by interacting with vacuolar Na^+/H^+ antiporter (NHX) and regulating Na^+/H^+ ratio, which results in sequestration of excess Na^+ ions into the vacuole (Figure 2).[98] Moreover, SOS2 is shown to regulate SOS1 and CAX1 independently of SOS3 resulting from external stimuli[99] and contributes to Ca^{2+} homeostasis, demonstrating a metabolic link between Na^+ and Ca^{2+} homeostasis in plants (Figure 2).[100–103] This leads us to speculate that the salt stress responses, at least in part, are controlled by Ca^{2+} signal transduction pathway.

As a second messenger, Ca^{2+} binds to the protein sensors and transmits stress cues to the downstream signaling routs. Several CBLs and their target kinases have been described as components of salt stress responses. Na^+ efflux from the cytoplasm is mediated by an array of proteins comprising SOS3, its interacting kinase SOS2 and SOS1.[101] The fact that *sos1* mutant is hypersensitive to salt and contains more Na^+ in comparison to the wild type, supports the postulation that the SOS1 protein may cause Na^+ efflux from the cytoplasm. Mutations of SOS2 and SOS3 also produced a salt-hypersensitive phenotype; however, similar levels of Na^+ in these mutants with wild type suggest distinct regulatory mechanisms for SOS2 and SOS3 compared to SOS1.[104] CBL1 and its interacting kinase CIPK1, along with CBL9 with its kinase counterpart, CIPK3, were also found to contribute in salt stress tolerance signaling.[105–107] CBL/CIPK signaling modules are involved in salt tolerance through modulation of PM processes. However, their metabolic mode of action in plants that are exposed to salt stress is not fully clarified.

For the first time, Kim and colleague (2007) reported lower Na^+ content in a hyper salt-sensitive *cbl10* mutant, which suggests a novel role for CBL10 in the regulation of a pathway for salt tolerance (Figure 2).[104] In fact, plants with the capacity to mitigate excessive Na^+ levels, either by limiting influx or by enhancing efflux, exhibit higher tolerance to salt stress. An alternative hypothesis posits that lower Na^+ content in the cell is a function salt sequestration into the vacuoles. In this way defect/s in vacuolar Na^+ sequestration inhibit Na^+ transfer from cytoplasm to the vacuole which would be accompanied by salt susceptibility. This can be a plausible explanation for higher sensitivity of *cb110* mutant plants when compare to the wild type, though they are more sensitive to salt shock.[104] Consistently with this hypothesis, plants overexpressing AtNHX1 possess over-accumulation of Na^+ while maintaining capability for salt tolerance.[108]

CBL10 interacts with the SOS2/SOS1 complex[104,109,110] and localizes it to the PM where it mediates SOS1 induction and promotes Na^+ efflux from aerial parts of the plants.[109] The contribution of CBL10-SOS2 to salt stress responses by storing Na^+ in the vacuole has also been documented; nevertheless, its

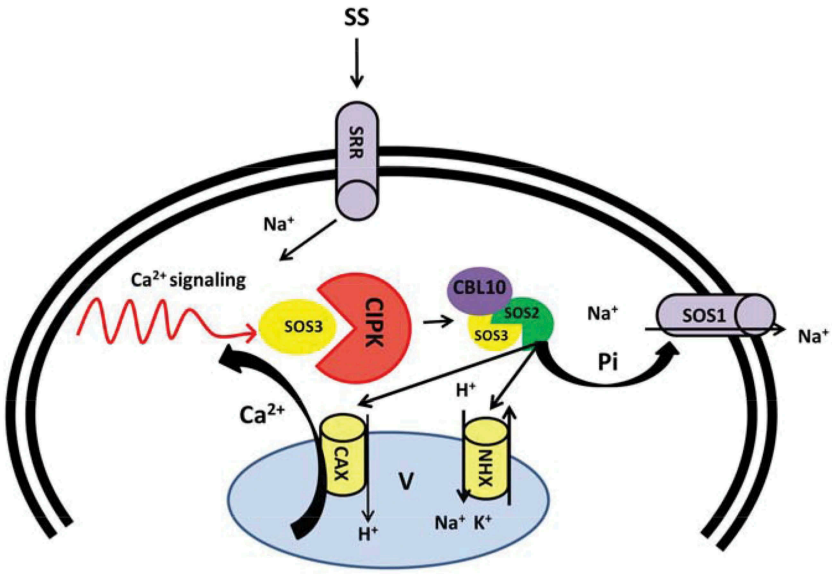

Figure 2. Schema model of SOS pathway regulated by Ca^{2+} under saline shock. Ca^{2+} signaling triggered by salt stress activate SOS3 causes interaction with SOS2. SOS3/SOS2 complex activates SOS1 by direct phosphorylation driven by SOS2 and result in Na^+ efflux. Alternatively, SOS2 regulates vacuolar channels (CAX and NHX) and balance Na^+ vacuolar sequestration. CAX: Vacuolar H^+/Ca^{2+} antiporter, CBL: Calcineurin B-like protein, CIPK: CBL-interacting protein kinase, NHX: Vacuolar located Na^+/H^+ antiporters, SOS: Salt overly sensitive, SS: Salt stress. SRR: Sal responsive receptors.

vacuolar or endosomal target proteins have not been identified thus far.[104] Alternatively, CBL10 triggers a stress response by interacting with AKT1 to hinder its accumulation at the PM and function as a negative regulator of K^+ uptake and thus regulates the Na^+ hemostasis.[111] These finding reveal the related function of Ca^{2+} signaling/modules in salt tolerance mechanism in plants. According to the direct or indirect interactions of Ca^{2+} with salt responses queues, a generic role for Ca^{2+} signaling in the context of salt stress is conclusive.

Convergence of Ca^{2+} and salt stress signaling pathways

In response to salt stress, an array of genes including *CAMTA* (CaM binding transcription activator) and *MYB* have been shown to be up-regulated by Ca^{2+}/CaM in *Aegilops tauschii*, implying the putative role of the Ca^{2+} signaling pathway in salt stress responses in plants.[93,112,113] Transcriptomic analysis of the wild-type and *myb59* mutant of *A. thaliana* using microarrays has revealed 45 differentially regulated transcripts with 33 and 12 genes up- and down-regulated, respectively. Twenty-five percent of up-regulated transcripts possess the expression of Ca^{2+}-binding proteins and the rest are functionally involved in Ca^{2+} homeostasis, transport and signal transduction pathways.

Involvement of these genes in Ca^{2+} signaling has been confirmed according to the DAVID enrichment analysis.[114] Comparison between wild-type, *myb59* and overexpressed (OE) lines demonstrated that the genes involved in Ca^{2+} signaling (*CML24, CML35, CML47, KIC* and *PILS4*) have been constitutively expressed. Ca^{2+} signaling role in regulation of stomatal movement has been reportedly suggested.[115–117] Likewise, Ca^{2+} has null effect on stomata of *myb59* while in OE and wild type resulted in stomatal movement. This suggests that under salt stress, involvement of MYB59 in stomatal functioning through Ca^{2+} signaling is highly likely, as expressed MYB59 by salt stress can suppress CAX1 and trigger stomatal closure.[54]

It has been well documented that the expression of a *Medicago truncatula CML, MtCML40*, is up-regulated under salt stress. Surprisingly, overexpression of *MtCML40* negatively affect the expression of *MtHKT1;1* and *MtHKT1;2*, two genes responsible for encoding proteins with function in mitigation of exceed Na^+ from shoots. This can indirectly connect MtCML40 regulatory role to the Ca^{2+} signaling through a MtHKT-dependent Na^+ accumulation process when plants suffer from saline shock.

Gene duplication is defined as one of the major adaptation mechanisms developed by plants to cope with unexpected environmental stimuli. Gene duplication effect on plant salt tolerance has been investigated on the duplicated *CBL10* genes of *Eutrema salsugineum* as a salt-tolerant plant. Results have indicated that, down-regulation of duplicated *CBL10* genes (*EsCBL10a* and *EsCBL10b*) reduced the plant growth in response to salinity, while higher reduction was observed when expression level reduced in each gene individually, implying a distinct function for both genes in response to salt stress. Based on a cross-species complementation assay, *EsCBL10b* is a potential activator of SOS pathway while *EsCBL10a* exhibits a different function than *EsCBL10b*. Notably, N-terminus modification in homologues of EsCBL10s results in different functions of each protein. Regardless of different functions of *EsCBL10a* and *EsCBL10b*, their duplication increases Ca^{2+}-mediated signaling capacity in *Eutrema salsugineum*.[118] Moreover, it is believed that elevated number of active Ca^{2+} sensors causes salt tolerance and adaption. *Eutrema penlandii* comprises three Ca^{2+} sensors, EsCBL10a, EsCBL10b and EsSOS3, with both individual and combined functions. It has been revealed that co-expression of *EsCBL10a* and *EsSOS3* in *Atsos3* increases root growth, suggesting an additive effect of these genes on salt tolerance and thus root growth.[118]

It has been shown that salt tolerance is associated with higher K^+/Na^+ ratio[119] and enhanced Na^+ efflux.[120] Transcriptome analysis in wheat genotypes revealed that NHX1 and the SOS pathway-related gene expression levels in salt-tolerant genotypes were higher in comparison with their expression in the other genotypes.[121] Moreover, similar research in *Aegilops tauschii* during long-term exposure to saline stress demonstrated that genes encoding transporters and channels including *SOS1, NHXs, HAKs*, with contribution to K^+ hemostasis have been up-regulated.[93] In addition, it was shown that *ENA1*, which encodes the *P*-type ATPase required for Na^+ efflux is expressed by elevation in cytosolic Ca^{2+} concentration.[63,122]

Transcriptomic analysis of *Camellia sinensis* revealed that expression levels of Ca^{2+}*-ATPases* and *CAX* genes were upregulated in response to salt stress, this suggest the existence of overlapping expression patterns with Ca^{2+} signaling and salt stress responses.[53] Moreover, three *MAPK* kinase and five *MAPK* kinase genes were also shown to be induced under salinity.[53] As *MAPK* signaling pathway is mediated by Ca^{2+}-dependent regulation pathway this suggests the involvement of Ca^{2+} in salt stress responses.[123]

WRKY gene expression under salt stress is also regulated by CaM-mediated Ca^{2+} signaling pathway.[53,124–126] In this regard, it was reported that in *Camellia sinensis* multiple *WRKY* genes, including *WRKY7, WRKY33, WRKY18* and *WRKY40*, were up-regulated under salinity.[53] Among 74 *WRKYs* identified in Arabidopsis, expression of 18 genes drive salt stress responses in the root, whereas overexpression of *AtWRKY25* and *AtWRKY33* increased salt tolerance in the shoot.[127,128]

Studying the gene expression pattern in soybean under saline–alkaline and drought stress conditions revealed that genes associated with Ca^{2+} signaling are up-regulated in all stress treatments suggesting that Ca^{2+} signal transduction is casually linked to the external stimuli responses.[129]

In summary, physiological and metabolic analyses under salt stress condition demonstrated the involvement of several genes and transcription factors in salt stress responses that are linked to Ca^{2+}-dependent signaling pathways. Future research on the role of Ca^{2+} signaling as stress signaling molecules on

differential expression of genes can open a new avenue to reveal the controlling mechanism of salt stress in plants.

Polyamine/Ca^{2+}-dependent salt stress responses

Polyamines (PAs), mainly known as putrescine (Put), spermidine (Spd) and spermine (Spm), are well recognized for their profound effects on plant growth, development and adaptation against various environmental cues.[130] It has been well-established that PAs play indispensable roles in multiple cellular processes, such as cell division, differentiation, transcriptional regulation, translation, membrane and cell wall stabilization and programmed cell death.[131–133] Transcriptome analysis of PAs in plants subjected to a wide array of environmental stressors has shown that modulation of PA levels is associated with tolerance and stress amelioration and is often accompanied by changes in expression level of PA biosynthesis-related genes.[131,134] Studies concerning the regulation of PA concentrations in plants revealed that several genes involved in PA biosynthetic pathways are unregulated under salt stress.[134,135] Activation and stimulation of arginine decarboxylase (ADC), which is involved in PA biosynthesis, in response to salinity has been regarded as a key regulator of adaptive responses in plants.[136] An investigation on expression levels of PAs biosynthetic genes in 18 varieties of *Oryza sativa* demonstrated that ADC gene is induced under salt stress.[137]

Physiological studies have provided a wealth of information on the role of PAs degradation pathway driven by PAs oxidase enzyme in response to stress stimuli.[138] Recent studies have indicated that the PAs metabolic pathway is in intricate crosstalk with other signaling molecules such as ABA, H_2O_2 and γ-aminobutyric acid (GABA).[139,140] In this way, it has been shown that GABA enhances salt tolerance in some plant species such as lettuce.[141] PA metabolism also stimulates NO production that may link PAs-mediated stress responses to the other stress mediators such as Ca^{2+} ions and protein kinases.

Under salinity conditions, PAs as positively charged molecules could substantially interact with negatively charged proteins including ion channels and affect their conductivity.[142,143] Garufi et al. (2007) suggested that PAs may regulate the activity of numerous ion channels indirectly by affecting PM potential by activation of H^+-ATPase through enhancement of interactions with 14-3-3 proteins, a family of highly conserved regulatory molecules (Figure 3).[144] PAs directly inhibit both types (fast and slow acting) of NSCCs in vacuolar membrane with a high affinity.[145,146] Consistently, it has been reported that PAs induce stomatal closure by regulating KAT-1 like voltage-dependent K^+ channel. PAs also reduce Na^+ influx through the NSCCs transporters, thus diminishing the Na^+-induced membrane depolarization and inhibiting K^+ losses.[147,148] In pea leaves, the NSCC inhibition by external PAs, Put or Spm, incorporate to decreases in the salt-induced membrane depolarization and K^+ efflux.[146] Inhibitory effects of PAs in NSCC-mediated Na^+ flow have been reported in leaf[146] and root[142] tissues. In this regard, PAs indirectly can be linked to the Ca^{2+} signaling as discussed transporters are modulated by Ca^{2+} or related modules.

The H_2O_2 production in PAs-depleted cells, affect the PM ion transport in a ROS-dependent manner and activate Ca^{2+} signaling. Salt stress-induced decrease in the activity of the PM and vacuolar H^+ pumps can be substantially retained by exogenous application of PAs (Figure 3).[149] Spm-deficient

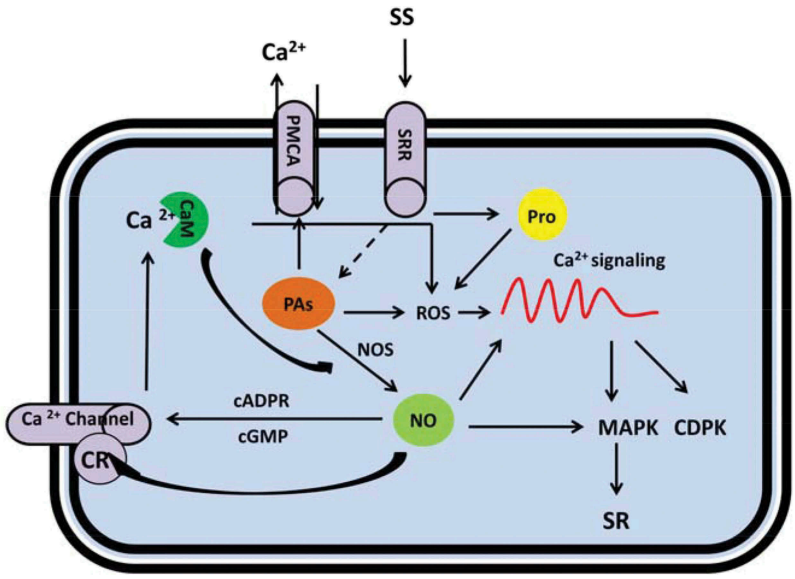

Figure 3. Metabolic scheme for regulation of salt stress responses by polyamines (PAs), nitric oxide (NO) and proline mediated by cytosolic Ca^{2+} signaling. Under salt stress, elevated PAs content affect Ca^{2+} level in the cell by either direct activation of PMCA and contributes to Ca^{2+} excursion (this will result in a steady state level of Ca^{2+} for normal metabolism in the cell); or indirectly by stimulation of NO and ROS production that results in downstream Ca^{2+} signaling. Similarly by salt shock, enhanced proline content influences the Ca^{2+} read out by ROS engagement; while NO is multiply associated with Ca^{2+} signaling comprising: direct manipulation of the Ca^{2+} signaling, effect on Ca^{2+} channels and regulation of MAPKs. CR: Cysteine residue, cGMP: Cyclic GMP, cADPR: Cyclic ADP ribose (cADPR), CaM: Calmodulin, CDPK: Ca^{2+} dependent protein kinases, MAPK: mitogen-activated proteins kinase, NO: Nitric oxide, PAs: Polyamines, PMCA: plasma membrane-Ca^{2+}-ATPase, Pro: Proline, ROS: Reactive oxygen species, SR: Salt responses, SRR: Salt responsive receptors.

mutant plants have constitutively active CAX channels implying negative role of PAs on Ca^{2+} current in vacuolar channels.[150]

Ca^{2+} influx across the PM plays a central role in ROS signaling. Free oxygen radicals like H_2O_2 and $^{\bullet}OH$ can activate hyperpolarization of Ca^{2+}-permeable channels. $^{\bullet}OH$ is also able to activate both outward K^+ current and voltage-dependent conductance (ROSIC). Unexpectedly, PAs potentiates $^{\bullet}OH$-induced K^+ efflux, as well as ROSIC in isolated protoplasts. Thus, it is conceivable that adsorption of PAs may sensitize ROSIC for activation by $^{\bullet}OH$. ROS and PAs suppress the activity of some constitutively expressed K^+ channels. In prolonged stress condition elevated cytosolic Ca^{2+} level may cause detrimental effect on the normal cell metabolism. Therefore, active Ca^{2+} efflux systems play a prominent role in maintaining basal Ca^{2+} content in the cell.[150–152] PAs activate Ca^{2+} efflux systems, such as PM and endomembrane Ca^{2+}-ATPase (PMCA) pumps and exchangers, thus balancing Ca^{2+} cytosolic levels (Figure 3).

These data are well in line with the idea that the PAs metabolic or catabolic pathways are joined to Ca^{2+} signaling with potential to provide salt stress responses in plants, although precise regulation mechanism remains elusive.

NO/Ca^{2+} regulated responses to salinity

NO has emerged as a potent signaling molecule in plant cells with various protective functions against abiotic stresses.[153] This membrane-permeable free radical has also been associated with a wide range of physiological processes, such as root hair growth,[154] germination[155] stomatal movements,[156] flowering,[157] root nodulation[158] and pollen tube growth.[159] Dual effects of NO have been well defined: high cellular concentrations triggers cellular damage due to the generation of the nitro-oxidative pool while at low content it contributes to different redox-regulated gene expression and establishment of plant stress tolerance.[160] An increasing number of studies report the modulation of NO metabolism during different abiotic stress conditions, such as high light intensity,[161] low[162] and high temperature,[163] mechanical wounding[164] and salinity.[165,166] Arginine-dependent NO production by NO synthase (NOS) enzyme is the main pathway for NO synthesis in animals.[167] A protein displaying NOS activity namely AtNOS1 has also been identified in *A. thaliana* (Figure 3).[168] Considering the fact that NO is a molecule related to PAs by a common precursor of l–arginine, PAs like Spm and Spd have also been suggested to trigger NO production in plant (Figure 3).

Direct and indirect interactions with various signal transduction components, such as protein kinases, secondary messengers (e.g. Ca^{2+}, cGMPs, phosphatidic acid and ROS), and phytohormones have also been proposed for NO.[169–171] The function of NO in the signaling network might be mediated by mobilization of intracellular Ca^{2+} or interaction with Ca^{2+} channels,[172] NO signaling has been shown to induce Ca^{2+} signaling in plants (Figure 3).[173] This implies a feedback loop underlying Ca^{2+} and NO signaling (at least in part) are entwined. During salt stress, NO-induced elevated cytosolic Ca^{2+} levels modulate the activity of protein kinases, such as CDPKs and MAPKs.[174,175] The primary targets of NO in plant cells might include MAPK. MAPKs can be activated in response to extracellular signals such as osmotic stress that cause the activation of signal transduction pathways resulting in altered gene expression (Figure 3).[176] NO has been associated with ion transport and regulation of ion channels in mammalian tissues while in plant direct evidence of regulation of ion channel by NO comes mainly from electrophysiological studies in guard cells.[156,177] ABA-induced stomatal closure via cytosolic Ca^{2+} has been implied to be associated with NO boost in the guard cells.[115,178]

Ca^{2+} channels are affected by NO through direct involvement with S-nitrosylation (binding of NO to the cysteine residue) or indirectly by effect on cyclic GMP (cGMP) and cyclic ADP ribose (cADPR) functionality to release Ca^{2+} from intracellular stores (Figure 3). Lamotte et al. (2006) emphasized the role of NO in activation of both PM and intracellular Ca^{2+}-permeable channels via signaling cascades. In this way, PM depolarization, cADPR and protein kinases activity by NO was accompanied with Ca^{2+} reporter apoaequorin activation in *Nicotiana plumbaginifolia* subjected to hyperosmotic stress.[179] Elevated levels of cytosolic Ca^{2+} elicits downstream physiological responses to a given signal and enhance/maintain NO generation. NO biosynthesis driven by NOS is regulated by plant-NOS activity on Ca^{2+} and CaM signaling basis (Figure 3).[180,181]

NO is known to be involved in plants tolerance to salt stress by improving antioxidative defense system, osmolyte accumulation and ionic homeostasis.[182] The antioxidant role of NO during establishment of salinity tolerance is mainly due to the maintenance of cellular redox homeostasis and boosting of antioxidant metabolites, e.g. ascorbate, reduced glutathione, and osmolytes, e.g. proline and soluble sugar.[183] Moreover, NO possesses antioxidant properties and might function as a signal to enhance the activity of H^+-ATPase in PM and proton-pomp in tonoplast.[184–186]

An positive effect of exogenous NO on oxidative metabolism has been reported in NaCl-treated chickpea plants.[187] Hasanuzzaman et al. (2011) demonstrated that ROS detoxification in *Triticum aestivum* under saline condition can be modulated by exogenous application of NO.[188] Under salt stress NO maintain a high level of K^+ ion, which is accompanied with low Na^+ content in the cytosol by mediation of PM channels, such as H^+-ATPase.[189] Consistently, investigations conducted on salt-treated callus cell cultures showed that NO regulates PM-H^+-ATPase activity, thus elevating the K^+/Na^+ ratio, leading to salt acclimation.[190,191]

These findings forged ahead our current understanding on the retro-controlling mechanism in response to salt stress powered by a system with three components involving salt perception and Ca^{2+}/NO signaling. However, the metabolic route/s involved in this complex chain is still baffling and further studies are required to unravel the precise mechanism/s underlying salt-Ca^{2+}-NO orchestration.

Osmoprotectant responses to salt and Ca^{2+} signaling

Various physiological and biochemical adaptive strategies have been developed in plants to minimize the detrimental

effects of stress events, such as salinity. For instance, plants synthesize and accumulate compatible soluble substrates or osmoprotectants that are implicated to grow and develop under such conditions.[192] Osmoprotectants are highly soluble organic compounds with low molecular weight and naturally safe at high concentrations.[193] As evident from a series of *in vivo* and *in vitro* investigations on physiology, biochemistry, genetics and molecular biology of plants, osmoprotectants potentially provide adaptive steady status under various unfavorable conditions, such as drought,[194] salinity,[195] temperature[196] and heavy metal stresses.[197] Compatible solutes are integrated in adjustment of the plants turgor pressure against salinity stress. Reduction of the cytoplasm osmotic potential driven by compatible solutes permits the maintenance of the cell water content and retention of turgor pressure under stress conditions.[198] In fact, osmotic adjustment as a fast acting adaptive response allows the plant to regulate ion homeostasis and maintain their turgor pressure and cell volumes under salinity shock[199]

The major osmolytes that are primarily synthesized and accumulated in the cytoplasm in response to abiotic stress can be characterized in three classes comprising (i) ammonium compounds such as PAs, glycinebetaine, b-alanine betaine, dimethyl-sulfonio propionate and choline-sulfate, (ii) sugars and sugar alcohols namely fructan, mannitol, D-ononitol and sorbitol and (iii) amino acids so-called proline and glycine betaine.[200] Among them, proline, glycine betaine and mannitol are the most prominent osmoprotectants. Proline, an α-amino acid derivative compound, is the main and widely distributed compatible solutes with high capacity in maintaining osmotic balance in the cell under saline condition.[201] Proline accumulates in response to saline stress and contributes to the alleviation of cytoplasmic acidosis and maintaining of the NADP/NADPH balance to conduct the natural metabolism.[202] Additionally, the strong conformational rigidity of proline structure in retaining proteins stability and membrane structure when is paralleled with its integration to ROS scavenging makes it a vital key candidate for growth and survival under osmotic stress. Szabados and Savoure (2010) demonstrated that proline functions as a molecular chaperone capable of maintaining protein integrity with potential to enhance the activity of various enzymes attributed to the ability of this compound to form hydro-phil/phobic features dealing with various proteins.[201] Investigations on the accumulation of proline in seven rice (*Oryza sativa* L.) cultivars showed that increased proline concentration progressively correlates with increased salt stress tolerance.[203] Although, several lines of evidence indicate that under various stress circumstances, elevated levels of proline assume a protective role, excessive levels of proline are toxic due to its effect on increasing ROS production (Figure 3). ROS production is mediated by Ca^{2+}/CaM signaling. Ca^{2+}/CaM deals with the ROS in two divergent mechanisms. On one hand a feedback loop activates NAD(H) kinase and provides NADP(H) as primary substrate for ROS; on the other hand it induces the antioxidant enzymes activity and mitigates ROS accumulation.[204] Ca^{2+} has also been proposed to stimulate antioxidant enzyme activity and maintain cellular redox potential in rice seedling under saline stress condition.[205] This cross link between proline, Ca^{2+} and salt stress indicates that collapsed ion hemostasis derived by salt stress is partially ruled by Ca^{2+} signaling. However, knowledge about Ca^{2+} interrelation with diverse osmolytes is scant and requires further research to formulate the specific metabolic pathway.

Conclusion

To date, prevalent research has been conducted on salinity stress in plants and as a result diverse metabolic pathways have been elucidated, leading to the identification of important mechanisms for induction of salt tolerance in plants. Meanwhile, parallel investigations into Ca^{2+} signaling have revealed diverse roles for Ca^{2+} in the context of salt stress as well. Despite of these findings, the multifaceted role of Ca^{2+} in plant stress responses has been underestimated. In this review, the authors attempted to demonstrate the comprehensive effects of Ca^{2+}, in terms of both quantity and signaling, in various aspects of plant responses to salt stress. Accordingly, the effect of Ca^{2+} in salinity begins from the initial stage of salinity perception, activating a signal transduction pathway to force Na^+. Ca^{2+} dynamically affects diverse Na^+ receptors and modulates salt-derived downstream signaling pathways. Consequently, Ca^{2+} can be considered as one of the most versatile and important molecules in the response to salinity. Due to the prominent role of Ca^{2+} in salinity stress, better understanding of Ca^{2+} signaling pathway can pave the way for release of salt-tolerant crops. This addresses the Ca^{2+} signaling as an imperative field for future research.

Acknowledgments

Authors want to sincerely thank Massimo Bosacchi from KWS Gateway Research Center for critical revising of the manuscript.

Funding

Financial supports from Iran National Science Foundation (INSF) Grant number 97007583 to Dr. Maryam Seifikalhor and Grant number 96006991 to Dr. Sasan Aliniaeifard are gratefully acknowledged.

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