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Plant salt tolerance and Na^+ sensing and transport

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Salinity is a global challenge to agricultural production. Understanding Na^+ sensing and transport in plants under salt stress will be of benefit for breeding robustly salt-tolerant crop species. In this review, first, possible salt stress sensor candidates and the root meristem zone as a tissue harboring salt stress-sensing components are proposed. Then, the importance of Na^+ exclusion and vacuolar Na^+ sequestration in plant overall salt tolerance is highlighted. Other Na^+ regulation processes, including xylem Na^+ loading and unloading, phloem Na^+ recirculation, and Na^+ secretion, are discussed and summarized. Along with a summary of Na^+ transporters and channels, the molecular regulation of Na^+ transporters and channels in response to salt stress is discussed. Finally, some largely neglected issues in plant salt stress tolerance, including Na^+ concentration in cytosol and the role of Na^+ as a nutrient, are reviewed and discussed.

Contents

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1. Introduction

Sodium constitutes the sixth most abundant element on earth [1], and sodium salts dominate in many saline soils of the world [2]. The current progressive increase in soil salinization may result in a ~30% loss of the arable land within the next 25 years [3]. To meet the projected food demand of 9.3 billion people by 2050, global agricultural production must be increased by 60% from its 2005–2007 levels [4]. This urgent need requires a large effort to improve agricultural production. One feasible way to cope with this challenge is to breed robustly salt-tolerant crops. Understanding the mechanisms underlying plant salt tolerance would be of benefit for breeding such crops and mitigating future food shortages.

Na^+ is generally not essential for plants. The similarity of the hydrated ionic radii of Na^+ and K^+ leads to Na^+ toxicity in plants under salt stress [5]. Accumulation of high Na^+ in the cytosol can not only cause K^+ deficiency and thus disrupt various enzymatic processes, but also impose an energetic burden on the cell owing to the requirement of organic solute synthesis to compensate for the export of Na^+ for osmotic adjustment [6]. More than 50 enzymes are activated by K^+, which cannot be substituted with Na^+ [7]. Also, oxidative stress is always accompanied by salt stress in plants [8]. Thus, understanding how Na^+ is sensed and transported in plants under saline conditions could help researchers or breeders breed crops with robust salt tolerance.

The present review is focused mainly on how plants sense Na^+ and control its transport under salt stress. The molecular identity of transporters and channels involved in Na^+ transport and its molecular regulation in response to salt stress are discussed.

2. Na^+ as an issue

Our earth is a salty planet [9,10]. Saline soils cover 3.1% (397 million ha) of the total land area of the world [11]. High concentrations of salts in soils account for large decreases in the yields of a wide variety of crops worldwide and result in annual losses in the billions of dollars [7,12]. For example, soil salinity developed through shallow water-tables costs the farming economy in Australia about $300 million per year [13].

The onset of salinity stress on plants can be divided into two phases: osmotic phase (rapid response to osmotic pressure) and then ionic phase (ionic toxicity from accumulated Na^+) [6,14]. In terms of salinity stress tolerance, plants can be divided into halophytes and glycophytes. Most crop species are glycophytes. Many glycophytes are particularly intolerant to salt, being inhibited by NaCl concentrations around 25–50 mmol L^{-1} [15]. Soil solutions with ECe (Electrical Conductivity of a saturated soil Extract) higher than 4 dS m^{-1} (corresponding to roughly 40 mmol L^{-1} NaCl) are regarded as saline [6]. Salinity stress can reduce germination rate [14,16], survival rate [16,17], growth [17], biomass/yield [14,16,18], leaf area [19], and leaf chlorophyll content [20,21]. It can impact photosynthesis-associated traits and reduce stomatal conductance and net CO_2 assimilation rate [20]. It can not only cause Na^+ accumulation in plants but also induce root [22,23] and mesophyll K^+ loss [24–26].

3. Na^+ as a nutrient

Given that Na^+ is one of the most soluble minerals and is easily accessible to plants to increase osmotic potential, absorb water, and sustain turgor, uptake of Na^+ is desirable, although excess Na^+ may become toxic to plants [27]. Na^+ influx into cells against an electrochemical gradient is mediated mainly by non-selective cation channels and the sodium transporter HKT1 [28]. Low to moderate Na^+ concentrations are commonly found to be benign and even beneficial, and can even stimulate growth of many plant species when they are K^+-deprived [29]. This effect may be due to the replacement by Na^+ of K^+ in the vacuole, making more potassium available to the cytosol [30–32]. For example, halophyte *Salicornia europaea* showed stimulated growth under 200 mmol L^{-1} NaCl compared to the non-saline condition [33]. Thus, to better improve salt tolerance in glycophytes, researchers have looked to the mechanisms used by halophytes to use Na^+ as a nutrient or prevent Na^+ accumulation in the cell cytosol [32,34–36]. For example, Shabala et al. [36] suggested that it will be possible in the near future to transform the trichomes of crop species to epidermal bladder cells, which are used in halophytes for storage of excess Na^+. The various strategies used by halophytes and glycophytes in response to salt stress are compared in Fig. 1.

4. Na^+ concentration in plant cell cytosol: some inconsistent results

To date, the role of the concentration of Na^+ in the cell cytosol in plant salt tolerance is debated. Some researchers claim ~30 mmol L^{-1} as a threshold of cytosolic Na^+ concentration [6,7], while others suggest a range between 50 and 200 mmol L^{-1} [37]. Carden et al. [38] found that Na^+ concentration in cytosol ranged between ~10 and 30 mmol L^{-1} in salt-stressed barley root cortical cells. Anil et al. [39] found

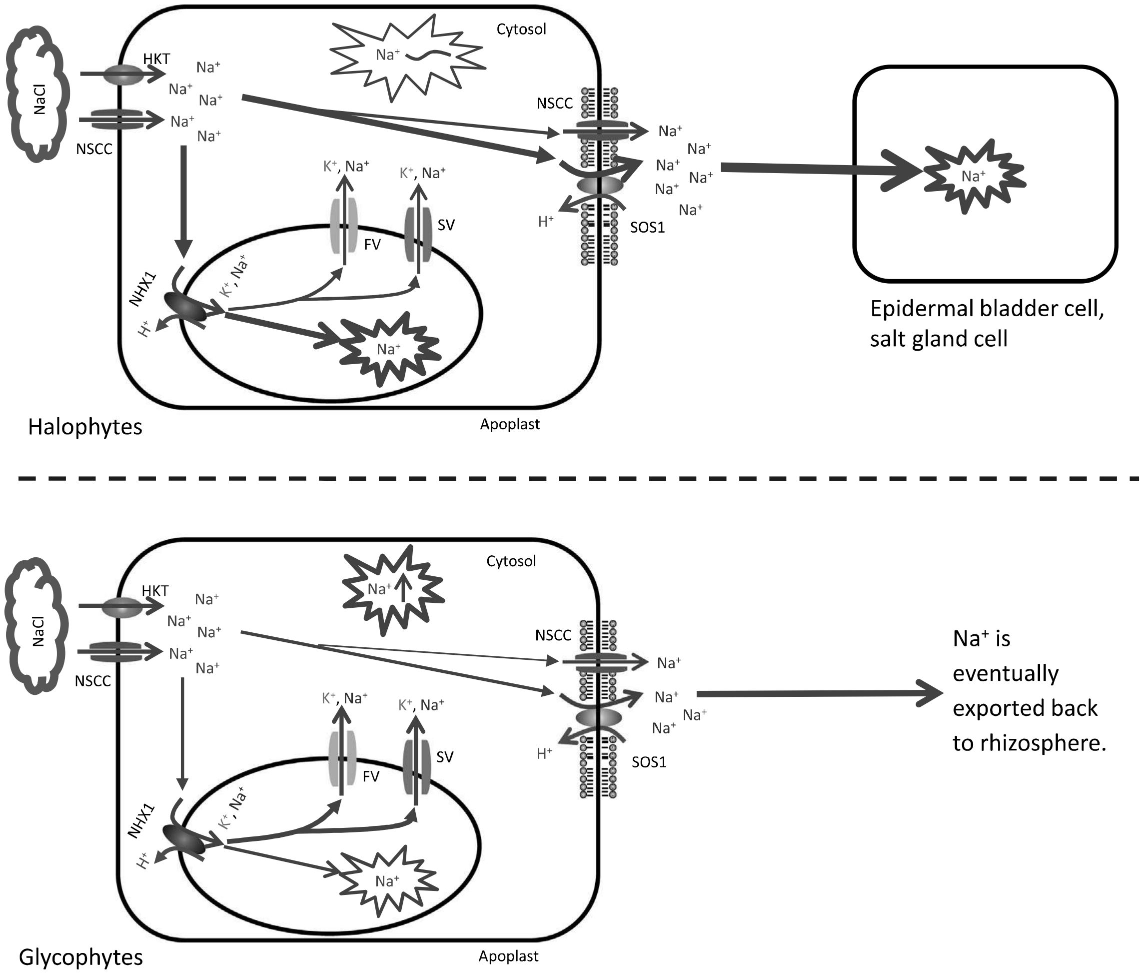

Fig. 1 – Different strategies of halophytes and glycophytes in response to salt stress. The thicknesses of lines represent the proposed contributions.

the cytosolic Na^+ concentration to range from ~5 to ~12 mmol L^{-1} in rice suspension cells under control conditions, whether they were derived from salt-tolerant or -sensitive varieties. Halperin and Lynch [40] measured the Na^+ concentration in *Arabidopsis* root hairs at lower than 65 mmol L^{-1} under salt stress. Na^+ concentration in the cell cytosol in halophytes under saline conditions is proposed to range between 60 and 200 mmol L^{-1} [41]. Differences in plant species' ability to tolerate Na^+ in the cytosol and sensitivities of measurement methods may partially account for these disparate findings.

5. Na^+ sensing in plants

5.1. *Possible salt sensors for perception of Na^+*

Unlike in animal cells, no specific salt sensors have been identified in plant cells to date. Thus, our knowledge of how plant perceive salt stress and thus encode the corresponding signals remains limited. Cramer et al. [42] found that Ca^{2+} can mitigate the loss of membrane integrity and minimize cytosolic K^+ leakage and proposed that displacement of Ca^{2+} by Na^+ from the root cell plasmalemma is a primary response to salt stress. However, Kinraide [43] claimed that the Ca^{2+}-displacement hypothesis is often of minor importance to salt stress response. SOS1 (salt overly sensitive 1) Na^+/H^+ antiporters [44], histidine kinases [45], and AHK1/ATHK1 [46] have also been suggested to be potential salt sensors or osmosensors, though clear evidence is still lacking. Shabala et al. [47] suggested some putative salt stress sensors/proteins involved in early signaling events, including exchangers and transporters have Na^+-binding proteins similar to mammalian cell Na^+ sensors, SOS1 Na^+/H^+ antiporters, NCX Na^+/Ca^{2+} exchangers, NSCC/NADPH oxidase tandem, mechanosensory channels and transporters, cyclic nucleotide receptors, purino-receptors, annexins, and H^+-ATPase/GORK tandem. Na^+-activated K^+ channels in animal tissues are able to translate changes in Na^+ levels into K^+ fluxes [48], triggering signaling cascades. The binding of salt stress-induced increases of cyclic nucleotides to their receptors, e.g. CNGCs, can activate this CNGC Ca^{2+}-permeable channels, and thus the increase of cyclic nucleotides could be translated into a massive cytosolic Ca^{2+} uptake, which can affect Ca^{2+} signaling

[47]. Similarly, sensing of salt-induced eATP (extracellular ATP) by plasma membrane purinoceptors can be translated into other signaling events, such as ROS (reactive oxygen species) and cytosolic Ca^{2+} signature [49]. Annexin is involved in ROS-induced cytosolic Ca^{2+} elevation under salt stress [50] and is suggested to be a key component in root cell adaption to salt stress [51]. K^+ acts as an intrinsic uncoupler of plasma membrane H^+-ATPase, and its binding to the cytoplasmic phosphorylation domain site involving Asp^{617} amino acid residue induces dephosphorylation of plasma membrane H^+-ATPase [52]. Thus, if a GORK channel is located near the H^+-ATPase (H^+-ATPase/GORK tandem), the depolarization-activated GORK channel-mediated K^+ efflux (early salt-stress signaling event [53,54]) can result in reduced cytosolic K^+ and thus may prompt activation of H^+-ATPase to restore plasma membrane potential, allowing modifying/affecting salt-induced K^+ efflux signaling events.

5.2. *Root meristem zone: a tissue harboring salt sensors?*

The root meristem is located at the very tip of the root. Root morphological change is always observed in root nutrient foraging [55]. This observation suggests that roots can perceive changes in environmental factors, such as nutrient distribution and salinity level. In most cases, the root is the first plant organ that encounters salinity. Thus, Na^+ enters first into roots and is then transported to shoots. Wu et al. [56] found that salt-tolerant bread wheat varieties had significantly higher cytosolic Na^+ in the root meristem zone than salt-sensitive varieties, although no difference in vacuolar Na^+ fluorescence intensity was found in the root meristem zone. This finding suggests that salt-tolerant wheats could have more ability to buffer or tolerate increased Na^+ in the cell cytosol in root meristem zone than salt-sensitive wheats. Further, by removal of the root meristem zone from salt-tolerant wheat varieties, Na^+ distribution in mesophyll cells was altered and a salt-sensitive phenotype resulted [57]. Taken together, these findings suggest that the root meristem zone can act as a salt stress sensor, or at least a tissue that harbors salt stress-sensor components.

6. Regulation of Na^+ transport in plants under salt stress

6.1. *The importance of Na^+ exclusion in plant salt tolerance*

The importance of Na^+ exclusion in protecting plants against salinity stress is widely accepted. Under salt stress, net Na^+ accumulation in plant cells is determined by the ion-exchange activity of Na^+ influx and efflux. Na^+ influx occurs mainly through ion channels such as the high-affinity K^+ transporter HKT and non-selective cation channels (NSCC), and Na^+ efflux is known to be mediated by SOS1, a Na^+/H^+ antiporter. In the presence of elevated levels of external Na^+, under saline conditions, Na^+ efflux from plant cells is an active process [58]. To date, SOS1, expressed mainly in the root apex in *Arabidopsis* [59], is the only transporter that has been characterized in Na^+ export from the cytosol to the apoplast. Cuin et al. [60] showed that among eight tested varieties, the most salt-tolerant wheat variety Kharchia 65 had the strongest root Na^+ exclusion ability. Overexpression of *SOS1* can also enhance salt tolerance in transgenic plants [61,62]. Loss of SOS1 function resulted in a hyper-salt-sensitive phenotype in the halophytic *Arabidopsis* relative *Thellungiella salsuginea* [63]. This finding further confirmed the important role of the SOS1 Na^+/H^+ antiporter in Na^+ exclusion and overall plant salt tolerance. Moreover, to date, studies showing the important role of Na^+ exclusion in overall salt tolerance have been based mostly on shoot/leaf or even whole-plant Na^+ content [64–69]. Whether this restricted Na^+ accumulation in shoot/leaves is achieved mainly by root Na^+ export or shoot Na^+ exclusion, or by both of these processes with tight regulation/coordination at different growth stages and time scales, however, has remained unclarified.

6.2. *The importance of vacuolar Na^+ sequestration in plant salt tolerance*

SOS1-mediated Na^+ export from cytosol to apoplast (against an Na^+ gradient) is an energy-consuming process. Given that most of the cell volume is occupied by vacuole and most metabolism occurs in the cytoplasm, one way for plants to alleviate Na^+ toxicity in the cytosol is to store Na^+ in the vacuole. Vacuolar Na^+ sequestration is a common and important mechanism in plant salt tolerance, and is mediated by Na^+/H^+ antiporters [70–72]. Prevention of cytoplasmic Na^+ elevation, maintenance of the cytosolic K^+/Na^+ ratio, and control of vacuolar osmotic potential in plants under salt stress can be achieved by, or is associated with, vacuolar Na^+ sequestration [73]. To date, the best-known transporter for vacuolar Na^+ sequestration is the NHX1 Na^+, K^+/H^+ exchanger. Overexpression of NHX1, a Na^+, K^+/H^+ exchanger, improves salt tolerance in many species including *Arabidopsis* [70], tomato [74], rice [75], and tobacco [76], showing the importance of vacuolar Na^+ sequestration in plant overall salt tolerance. Significantly more Na^+ in excised leaves accumulated in tolerant than in sensitive barley genotypes, suggesting the important role of vacuolar Na^+ sequestration in overall salt tolerance [77]. Also, salt-tolerant wheat varieties showed significantly higher vacuolar Na^+ fluorescence intensity in mature root cells than did sensitive varieties [56,60]. Under overexpression of *OsNHX1*, transgenic rice cells survived better under saline condition and showed significantly higher growth rate and total Na^+ content than the wild type (WT) [78]. Taken together, these findings show clearly that vacuolar Na^+ sequestration is an important trait contributing to plant overall salt tolerance.

After sequestration of Na^+ in vacuoles, another important concern is to prevent Na^+ leakage from vacuole to cytosol. Loss of control of this step could result in futile Na^+ cycling between vacuole and cytosol, imposing a high energy burden on the plant. FV (fast-activating) and SV (slow-activating) channels are tonoplast Na^+ and K^+-permeable channels that control Na^+ leakage from vacuole to cytosol. Negative control of FV and SV channel activity has been shown in the salt-stressed halophyte quinoa to reduce such leakage [79], suggesting that efficient control of Na^+ leakage from vacuole to cytosol could be an important mechanism in plant overall salt stress tolerance.

6.3. Control of xylem Na^+ loading and unloading

Roots absorb ions and then transfer them to shoots via xylem loading, so that control of xylem Na^+ loading is important in plant overall salt tolerance. To date, SOS1 Na^+/H^+ antiporter [58,80,81], CCC co-transporter [82], and SKOR channel (if xylem loading of Na^+ is passive) [83] have been shown to be involved in xylem Na^+ loading. Shi et al. [59] suggested that SOS1 plays a role in xylem Na^+ loading in *Arabidopsis* under mild salt stress. Yadav et al. [84] showed that enhanced xylem Na^+ loading and higher overall salt tolerance was achieved in tobacco by overexpression of *SbSOS1*. Recently, a reduction in overall net xylem Na^+ loading and accumulation in the shoot and thus improved salt tolerance were observed in wheat *Nax* (locus for Na^+ exclusion) lines following downregulation of SOS1-like Na^+/H^+ antiporter [85]. Besides SOS1, a CCC co-transporter that is preferentially expressed at the xylem/symplast boundary has also been suggested to play a role in xylem Na^+ loading [82]. With respect to Na^+ transport in xylem, besides Na^+ loading into xylem, Na^+ unloading from xylem is another important mechanism. HKT transporters play a main role in this process. Sunarpi et al. [86] showed that the AtHKT1 transporter located on the plasma membrane in xylem parenchyma cells in leaves played a role in Na^+ unloading from xylem vessels to parenchyma cells. Huang et al. [87] suggested that TmHKT7-A2, which is associated with *Nax1* locus, could control xylem Na^+ unloading in roots and sheaths. Also, Byrt et al. [65] showed that HKT1;5 (HKT8) is strongly associated with *Nax2* locus in durum wheat and its orthologous locus *Kna1* in bread wheat removes Na^+ from xylem in roots and leads to a high K^+/Na^+ ratio in leaves. Jaime-Perez et al. [88] showed that the SlHKT1;2 Na^+-selective transporter plays an important role in Na^+ unloading from xylem in tomato shoots and thus modulates its Na^+ homeostasis under salinity. Fig. 2 presents a schematic diagram of the control of xylem Na^+ loading and unloading.

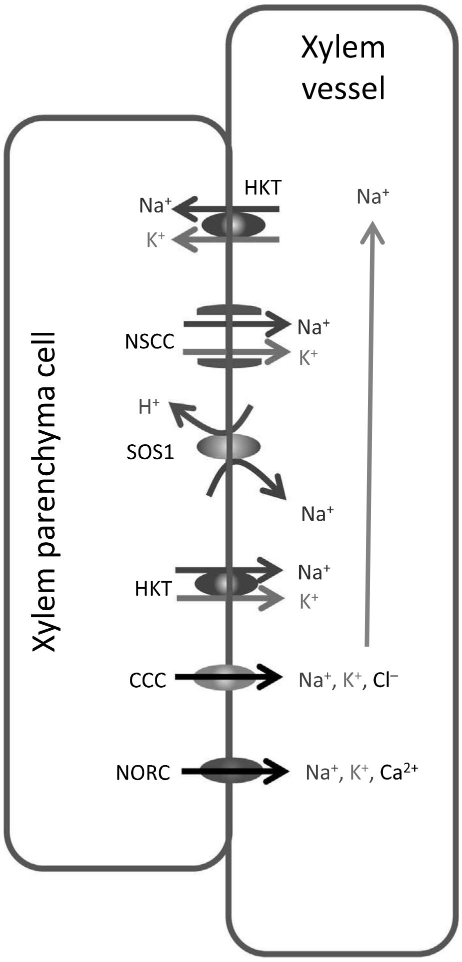

Fig. 2 – Control of xylem Na^+ loading and unloading.

6.4. Na^+ recirculation from shoot to root via phloem

Na^+ recirculation from shoots to roots has been suggested as an efficient way to protect leaf cells from Na^+ toxicity [89]. Because leaf vacuolar Na^+ sequestration ability is poor, Na^+ recirculation from shoots to roots via phloem sap is probably the main mechanism involved in prevention of Na^+ delivery to leaf cells in most salt-sensitive plants [90]. Apart from shoot growth rate, the rate of recirculation of Na^+ to the roots via phloem has been suggested as an important factor affecting Na^+ concentrations in shoots [91]. In several species, such as lupine, clover, sweet pepper, and maize, recirculation of Na^+ to roots via phloem played a role in overall salt tolerance [7]. Berthomieu et al. [90] showed that expression of the *AtHKT1* gene was restricted to phloem tissues in all organs in *Arabidopsis*, and that the *AtHKT1* gene was involved in Na^+ recirculation from shoots to roots probably by mediating Na^+ loading into phloem sap in the shoots and unloading it in roots. However, in *Arabidopsis*, a role of *AtHKT1* in control of both Na^+ accumulation in roots and retrieval of Na^+ from xylem, without involvement in root influx or recirculation in the phloem, was suggested by Davenport et al. [91]. Ren et al. [92] showed that HKT-type transporter encoded by *SKC1* (*shoot K^+ concentration 1*) gene might be involved in the recirculation of Na^+ by unloading it from the xylem in rice. Kobayashi et al. [93] found that an OsHKT1;5 Na^+ selective transporter associated with the *SKC1* locus is localized in cells adjacent to the xylem in roots, and is involved in mediating Na^+ exclusion in phloem to protect young leaf blades of rice under salt stress.

6.5. Na^+ secretion

In halophytic plants, ion secretion by specialized salt glands is a well-known mechanism for regulating mineral content. Under high-salinity conditions, these specialized cells can serve as a peripheral Na^+ storage organ, mitigate the elevation of cytosolic Na^+, and thus improve survival [94]. Salt glands secrete both Na^+ and K^+ in Rhodes grass, but the ability to secrete Na^+ is greater than K^+ [95]. Special salt glands in *Aeluropus littoralis* excreted salts consisting mostly of sodium chloride [96]. Chen et al. [97] found that when *Avicennia marina* plants were transferred to increasingly strong saline solutions, increased numbers of salt glands in leaves were found and rates of salt secretion greatly increased. Agarie et al. [94] showed that epidermal bladder cells in the common ice plant

(*Mesembryanthemum crystallinum*) contribute to salt tolerance by maintaining ion sequestration and homeostasis within photosynthetically active tissues. Bonales-Alatorre et al. [79] found that old leaves in quinoa had significantly higher Na^+ concentration in leaf sap than young leaves under salt stress, whether or not the leaves were brushed to remove bladder cells.

7. Transporters and channels involved in Na^+ transport in plants under salt stress

7.1. Na^+ transporters

In contrast to HKT transporters subfamily 2 members, which show superior K^+ conductance, all members of HKT transporter subfamily 1 have a serine at the first pore loop (for the motif S-G-G-G) and show preferential Na^+ conductance. For example, *Nax1* and *Nax2* QTL (quantitative trait locus) in durum wheat are respectively linked to HKT1;4 and HKT1;5 transporters [66], and *Kna1* in bread wheat is linked to an HKT1;5 transporter [65]. The role of HKT1:X transporters in Na^+ unloading and recirculation in salt stressed plants was mentioned in Section 6.3 and 6.4. For example, Kobayashi et al. [93] found that the OsHKT1;5 Na^+ selective transporter, which is associated with the *SKC1* locus, is localized in cells adjacent to the xylem in roots, and is involved in mediating Na^+ exclusion in phloem to protect young leaf blades of rice under salt stress.

As mentioned earlier, vacuolar Na^+ sequestration is an important mechanism in plant salt tolerance. In 1999, Apse et al. [70] showed that transgenic plants overexpressing *AtNHX1* had markedly increased salt stress tolerance and biomass. NHX1, a Na^+, K^+/H^+ exchanger, plays a crucial role not only in Na^+ accumulation in vacuoles but in pH regulation and K^+ homeostasis, regulating processes from vesicle trafficking and cell expansion to plant development [1,98,99]. Most of the NHX family members (AtNHX1, AtNHX2, AtNHX3, AtNHX4, ItNHX1, ItNHX2, and OsNHX1) are located on the tonoplast; AtNHX7/SOS1 and AtNHX8, and other NHXs (AtNHX5, AtNHX6, and LeNHX2) are located on the plasma membrane and the endomembrane system, respectively [100]. The intracellular NHX transporters compose subclass 1 of the cation/proton antiporter (CPA) family. To date, most members of the CPA family have been identified as Na^+/H^+ antiporters, but a few are K^+/H^+ antiporters, including CHX13, CHX17, CHX20, and CHX23 in the CPA2 family [101].

Besides vacuolar Na^+ sequestration, another important pathway for controlling Na^+ distribution in plant cells is Na^+ exclusion/export. To date, SOS1 Na^+/H^+ antiporter is the only reported antiporter responsible for Na^+ export from plant cells [102,103]. SOS1 activity is regulated by SOS2, a serine/threonine protein kinase (CIPK24) and SOS3, a myristoylated calcium-binding protein (CBL4) [104–106]. SOS3 recruits SOS2 to the plasma membrane, and then this CBL-CIPK complex activates SOS1 by phosphorylation, dramatically increasing Na^+/H^+ exchange activity [58]. Moreover, the existence of an ATP-driven Na^+ transport mediated by a Na^+-ATPase at the plasma membrane has been shown in lower plants, such as the marine alga *Heterosigma akashiwo* [107] and the moss *Physcomitrella patens* [108].

7.2. Na^+ channels

NSCCs are a large family of channels that lack selectivity for cations. They are typically permeable to a wide range of monovalent cations [109] and are located on both the plasma membrane and the tonoplast. They can be divided into voltage-dependent NSCCs (depolarization-activated, hyperpolarization-activated), voltage-independent NSCCs, ROS-activated NSCCs, amino acid-activated NSCCs, cyclic nucleotide-gated NSCCs, etc. Electrophysiological studies suggest that Na^+ influx across the plasma membrane occurs via NSCC/VIC in root cortical cells [10,58,110]. Maathuis and Sanders [111] found that cyclic nucleotide-regulated VIC (voltage-independent cation channels) channels showed no selectivity among monovalent cations in *Arabidopsis* root cells. Channels and transporters involved in Na^+ transport in plants under salt stress are summarized in Fig. 3.

7.3. Molecular regulation of Na^+ transporters/channels in response to salt stress

To date, SOS1 is the only known anti-transporter responsible for Na^+ export from cytosol to apoplast. Usually, expression of the *SOS1* gene is upregulated in salt stressed plants [63,102,112]. The functional activity of SOS1 mediated Na^+ export could be influenced by SOS2 [104], SOS3 [106], the assembly of SOS2-SOS3 complex [113], and H^+-ATPase, which can increase H^+ efflux to energize Na^+ efflux through SOS1 antiporters [114]. SOS1 activity could also be influenced by ROS or ROS signaling-associated components. *SOS1* mRNA stability is increased in *Arabidopsis* under H_2O_2 treatment, and NADPH oxidase is also involved in the upregulation of SOS1 mRNA stability [115]. Also, SOS1 interacts with RCD1 (radical-induced cell death), a regulator of oxidative stress responses, and functions in oxidative stress tolerance in *Arabidopsis* [116]. Reduced ROS production and increased SOS1 expression was found in *pao1pao5* (polyamine oxidase, PAO) *Arabidopsis* mutants than in the WT under salt stress [117].

As with *SOS1*, overexpression of *NHX1* to increase plant salt tolerance has been shown in many plant species [61,62,70]. Although the role of AtNHX1 in K^+ accumulation in the vacuole was discovered in recent years [98,99,118], this finding cannot completely rule out the involvement of NHX1 in vacuolar Na^+ sequestration, especially under high salinity [81,119]. Usually, the *NHX1* gene is upregulated in salt-stressed plants, including *Arabidopsis* [120], barley [121], and alfalfa [122]. However, a clear decrease in the transcript level of *NHX1* in wheat roots was observed under salt stress, while almost no change in the *NHX1* transcript level was found in leaves [123]. Also, in contrast to the successfully improved salt stress tolerance in *Arabidopsis* [70], tomato [74], rice [75], and tobacco [76], overall salt tolerance was not enhanced in *Arabidopsis* [61] and barley [124] by expression of the *NHX1* Na^+/H^+ exchanger gene. These conflicting results raise the questions of the importance of tissue specificity in plant salt-stress tolerance.

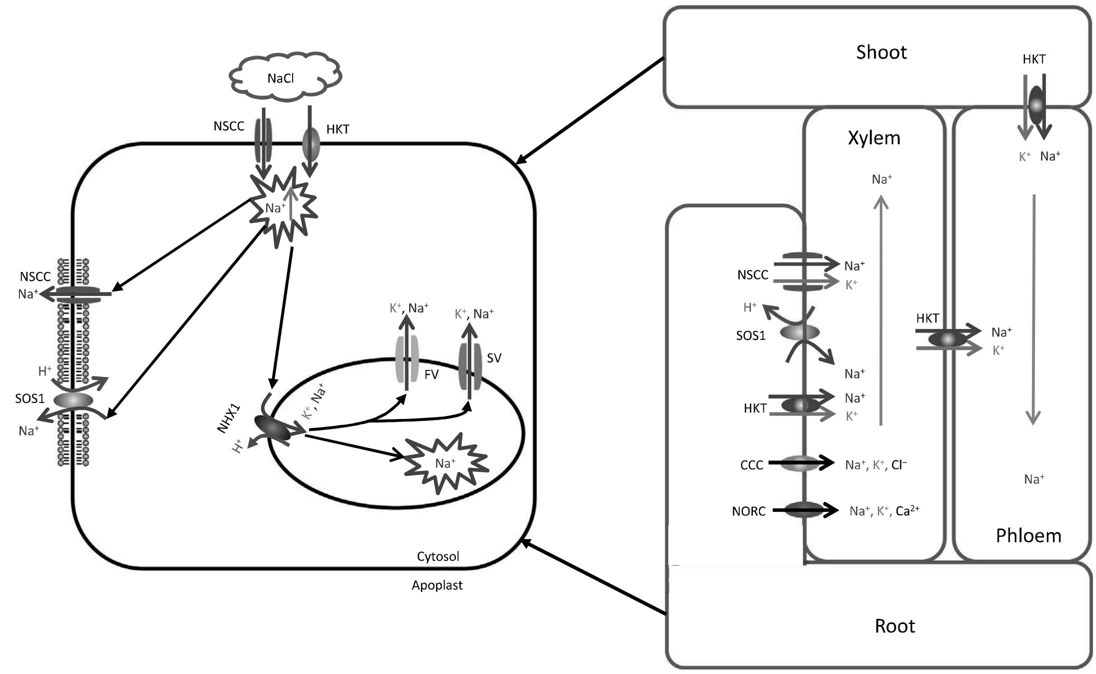

Fig. 3 – Na^+ transport in plants under salinity stress.

NHX1 is known to be fuelled by an H^+ gradient across the tonoplast that is maintained by vacuolar H^+-ATPase and vacuolar PPase [125]. Expressing a halophyte vacuolar H^+-ATPase subunit c1 (*SaVHAc1*) in rice plants resulted in higher chlorophyll content and yield than in its WT [126]. Overexpression of vacuolar PPase AVP1 improved salt tolerance in transgenic *Arabidopsis* relative to the WT, showing a healthy growth of transgenic *Arabidopsis* in the presence of 250 mmol L^{-1} NaCl compared with the WT, which died after 10 days [127]. These results suggest that manipulating vacuolar H^+-ATPase and PPase could allow regulating NHX1 activity and eventually plant overall salt tolerance. Other known factors in the regulation of NHX1 activity are SOS2 [128] and CaM15 [129]. Also, CBL10 can interact with SOS2 to protect *Arabidopsis* shoots from salt stress [130]. Tang et al. [131] showed that PtCBL10A and PtCBL10B interact with PtSOS2 in the vacuolar membrane to regulate shoot salt tolerance in poplar. Thus, CBL10 is also proposed to regulate NHX1 activity [132]. Two recent reviews [81,133] have also focused on molecular regulation of Na^+ transporters/channels in response to salt stress.

8. Conclusion

Although plant salt tolerance at the level of Na^+ transport is well characterized, the initial plant perception of salt stress and its transduction to subsequent signaling cascades is still obscure. In this review, some suggested putative salt stress sensors have been described. The root meristem zone as a tissue harboring salt stress-sensing components has been proposed. The importance of Na^+ exclusion and vacuolar Na^+ sequestration in plant salt tolerance has been highlighted. The molecular regulation of Na^+ transporters/channels in response to salt stress has been discussed. Although over-accumulation of Na^+ is toxic to plants, low levels of Na^+ can stimulate plant growth especially under K^+ deprivation. Inconsistent cytosolic Na^+ concentrations reported in the literature may be attributed to the diversity of plant species or the sensitivities of measurement methods.

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