Salt Tolerance Mechanisms of Plants

Annual Review of Plant Biology

Zelm, Eva; Zhang, Yanxia; Testerink, Christa

https://doi.org/10.1146/annurev-arplant-050718-100005

Annual Review of Plant Biology

Salt Tolerance Mechanisms of Plants

Eva van Zelm,* Yanxia Zhang,* and Christa Testerink

Laboratory of Plant Physiology, Wageningen University, 6700 AA Wageningen, The Netherlands; email: christa.testerink@wur.nl

Annu. Rev. Plant Biol. 2020. 71:403–33

First published as a Review in Advance on March 13, 2020

The *Annual Review of Plant Biology* is online at plant.annualreviews.org

https://doi.org/10.1146/annurev-arplant-050718-100005

*These authors contributed equally to this article

Keywords

ABA, auxin, osmotic stress, ionic stress, salinity, developmental plasticity

Abstract

Crop loss due to soil salinization is an increasing threat to agriculture worldwide. This review provides an overview of cellular and physiological mechanisms in plant responses to salt. We place cellular responses in a time- and tissue-dependent context in order to link them to observed phases in growth rate that occur in response to stress. Recent advances in phenotyping can now functionally or genetically link cellular signaling responses, ion transport, water management, and gene expression to growth, development, and survival. Halophytes, which are naturally salt-tolerant plants, are highlighted as success stories to learn from. We emphasize that (*a*) filling the major knowledge gaps in salt-induced signaling pathways, (*b*) increasing the spatial and temporal resolution of our knowledge of salt stress responses, (*c*) discovering and considering crop-specific responses, and (*d*) including halophytes in our comparative studies are all essential in order to take our approaches to increasing crop yields in saline soils to the next level.

Contents

INTRODUCTION

Soil salinity is an increasingly severe global problem, as salt hampers plant growth and development and reduces crop yield. In addition to naturally occurring soil salinity, the amount of salinization increases as a result of irrigation practices and climate change; the latter acts through either a rise in sea levels or increased evaporation in periods of drought (reviewed in 144). Crop loss due to increased soil salinity is now affecting migration patterns of farmers in coastal agricultural regions such as Bangladesh (22), stressing the impact and urgency of the problem. The detrimental effect of NaCl on plants is caused by both the reduction of water availability as sodium accumulates in the soil (124) and the toxic effect of sodium and chlorine ions on plants. Halophytes are plants that are adapted to salinized environments. They employ specialized strategies to deal with salinity (47). However, most crop species are salt sensitive—glycophytes—and optimizing them to produce more salt-tolerant plants is a strategy to increase crop yield on salinized agricultural land.

Halophyte: a plant that is able to thrive under high salt concentrations (>200 mM NaCl)

Glycophyte: a plant that can only grow and reproduce under relatively low concentrations of salt

Soil salinity induces both osmotic and toxicity stress in plants, resulting in growth inhibition, developmental changes, metabolic adaptations, and ion sequestration or exclusion (124). Osmotic and ion toxicity effects were thought to be temporally and spatially separated, with sodium ions in the soil rapidly reducing water availability and a slow accumulation of sodium in the shoot

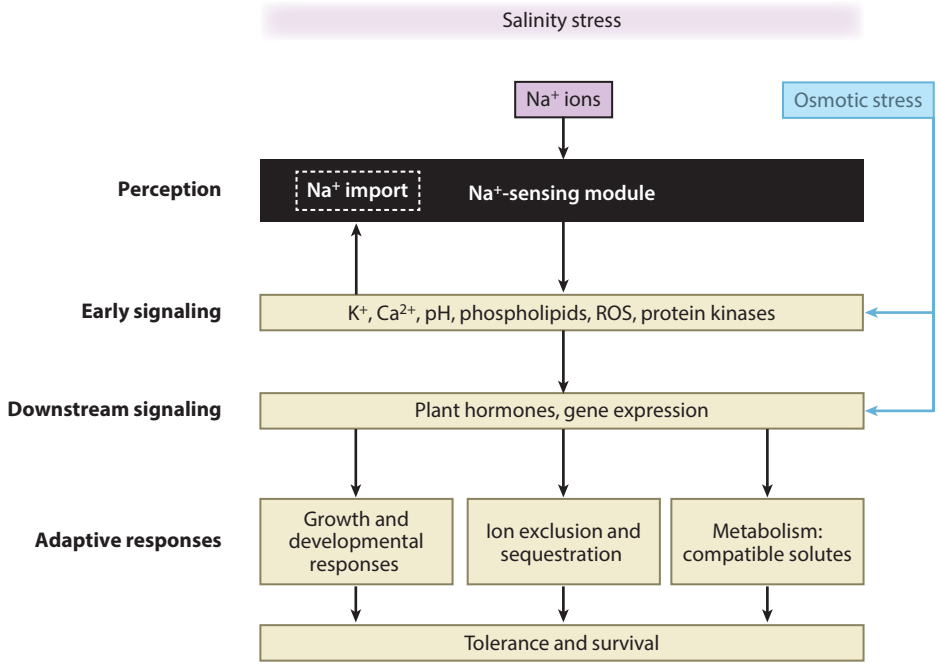


Figure 1

Sodium sensing and import are the black box of salt stress responses. Na^+ induces specific downstream responses, but the sodium-sensing mechanism of plants remains to be identified. Sensing could occur either inter- or extracellularly, before or after Na^+ import. After the initial perception, early signaling responses are induced, including K^+ transport, Ca^{2+} signaling, H^+ transport, phospholipid modifications, reactive oxygen species (ROS) induction, and protein kinase activity. In turn, these early Na^+-induced signals reduce sodium import. Downstream of the early signaling phase, phytohormone levels change through both biosynthesis and transport, and gene expression levels are altered in a manner that is both dependent on and independent from phytohormones. Finally, the salt-induced signaling cascade results in adaptive responses, such as modulation of growth and development, ion transport, and production of compatible solutes to compensate for the osmotic pressure of Na^+. Every step in the chain of signaling plays a role in mounting an adequate response to salinity and, finally, survival on saline soils. Part of the early signaling and downstream signaling response overlaps with osmotic stress–induced pathways.

inhibiting photosynthesis. This spatial and temporal separation suggested that early salt stress responses are due to general osmotic or drought stress and that sodium-specific responses are induced later. While there is indeed a significant overlap between osmotic and salt stresses in early and downstream signaling (**Figure 1**), this theory could be challenged by recent findings of both rapid salt-specific signal transduction and fast sodium-induced growth responses in roots (23, 51).

In this review, we link new findings in cellular responses to salt-induced changes in growth rate. At the cellular level, advances in the past decade have focused on salt-induced early signaling, demonstrating a major role for both calcium waves and reactive oxygen species (ROS) and their downstream targets, while the cell wall is often implicated as a modulator of cell expansion during salt stress. Furthermore, gene expression, mRNA stability, and translational regulation are altered to change protein abundance during salt stress. We discuss how different ion channels and transporters affect sodium/potassium homeostasis at the cellular level, with consequences for their in planta function. The plant hormones auxin and abscisic acid (ABA) have a major role in growth and

Reactive oxygen species (ROS): oxygen-containing reactive chemical species playing crucial roles in cellular signaling

Auxin: a phytohormone that regulates different aspects of plant growth and development throughout a plant's life span

Abscisic acid (ABA): a stress-induced phytohormone

architectural adaptations. We place local hormone signaling, gene expression, and ion transport in a tissue context to discuss their effect on organ-specific growth and whole plant survival.

The knowledge on signaling pathways and growth adaptations mostly comes from work on the model species *Arabidopsis thaliana* and has not yet been transferred to crops. To be able to increase salt tolerance in crop species we need to understand which growth adaptations to salt are beneficial for specific species. Plant salt tolerance is a complex trait that can be scored in different ways, including by ion accumulation, tissue-specific growth rates, biomass production, survival, and seed production. Depending on the crop, it will be desirable to optimize different salt responses in order to increase yield.

FROM SALT PERCEPTION TO EARLY SIGNALING AND ION TRANSPORT

First Steps: Sodium Import and Perception

The earliest cellular responses to salt, sodium import and sodium sensing, are arguably the least understood, and they remain a black box in salt-induced signaling pathways (**Figure 1**). Salt can enter the root through nonselective cation channels (NSCCs), which transport sodium across the plasma membrane (27, 28) (**Figure 2*a***). NSCCs are regulated by different salt-induced signals, such as calcium, 3′,5′-cyclic guanosine monophosphate (cGMP), and ROS. Other channels and transporters may also contribute, but their actual role in sodium import in planta is debated. The hypothesized action and regulation of sodium import have recently been critically assessed (94).

Despite recent advances, the mechanisms by which plants perceive salt is another open question (**Figure 1**). It has been proposed that plants sense osmotic changes rather than sodium ions, while sodium-specific responses occur much later through the toxic effects of sodium (or chloride) on the leaves (124). However, rapid salt-specific responses, such as salt-specific calcium waves, were recently identified in roots (23). Furthermore, the rapid and sodium-specific effect of salt on root growth direction (halotropism) predicts the presence of a root-based sodium sensor (51). Sodium may be sensed intercellularly, extracellularly, or by ion transporters at the plasma membrane (97a). Recently, significant progress has been made with the identification of MONOCATION-INDUCED $[Ca^{2+}]_i$ INCREASES 1 (MOCA1) likely functioning in extracellular salt sensing, including but not restricted to Na^+ ions (71). The *moca1* mutant lacks the early response calcium waves that occur in response to Na^+, K^+, or Li^+ ions. Functioning as a glucuronosyltransferase, MOCA1 produces glycosyl inositol phosphorylceramide (GIPC) sphingolipids at the plasma membrane. These GIPCs can bind monovalent cations and, upon binding, are hypothesized to bind and open a Ca^{2+} channel to induce downstream responses to salinity (**Figure 2*a***). In addition, salt-induced changes in the cell wall are perceived via FERONIA (FER), a receptor-like kinase (RLK) (**Figure 2*e***) (45). However, downstream signaling of this receptor happens several hours after salt application, and not during early salt-induced signaling responses (**Figure 2**). It is likely that no single sodium sensor exists, but rather that different aspects of salt stress are sensed and integrated through different signaling routes.

Receptor-like kinases (RLKs): transmembrane proteins with both extracellular domains and intracellular kinase domains to interact with various protein partners and downstream targets

Producing Ca^{2+} and ROS: The Early Sodium Signaling Wave

Increases in cytosolic Ca^{2+} concentration, ROS production, and cGMP are some of the earliest-described salt stress responses (32, 85, 117) (**Figure 2*b***). Three different types of calcium fluxes in response to sodium have been described: cellular calcium spikes and fast- and late-response calcium waves (23, 45, 85). The use of fluorescent and luminescent calcium biosensors has made it possible to track calcium fluxes throughout tissues. Although various stresses showed a cellular

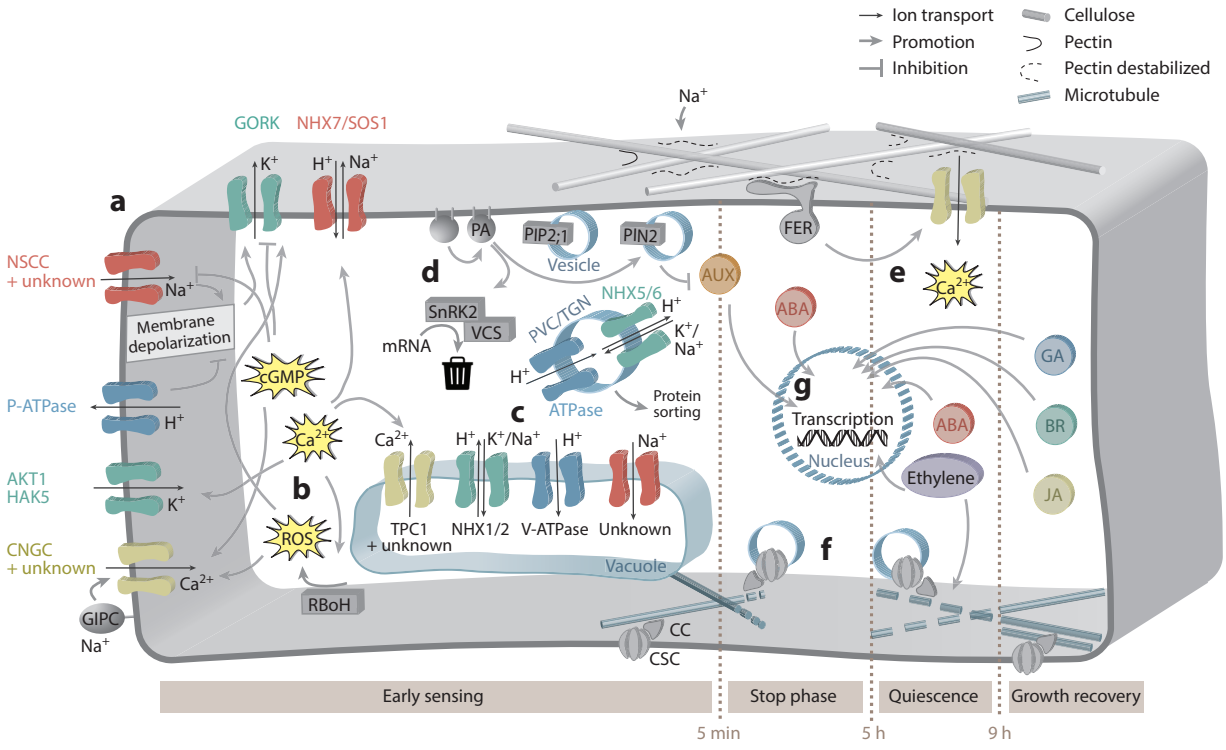


Figure 2

Cellular salt stress signaling over time. Cellular responses can be placed in different phases after salt application. Early signaling represents changes observed within 5 min of salt stress application. Growth rate decreases during the stop phase (within 5 min to 5 h of salt application in the main root), stays low during the quiescent phase (within 5–9 h of salt application in the main root), and partly recovers during the growth recovery phase (starting at 9 h after salt application in the main root). (*a*) When sodium enters the cell [through nonselective cation channels (NSCCs) and other, unidentified channels], the membrane depolarizes. This change in membrane voltage influences ion transport, because many K^+ and Ca^{2+} channels are regulated by voltage. The activity of P-ATPases can repolarize the membrane. Glycosyl inositol phosphorylceramide (GIPCs) stimulate Ca^{2+} import through an unknown Ca^{2+} channel, when bound by sodium. (*b*) Besides the Ca^{2+} influx, Na^+ import induces a rise of 3′,5′-cyclic guanosine monophosphate (cGMP) levels, and production of reactive oxygen species (ROS), together forming the three major early signaling compounds. cGMP induces Ca^{2+} import, reduces import of Na^{2+}, and decreases K^+ efflux. There is a positive feedback loop between ROS and Ca^{2+}, where Ca^{2+} induces ROS production by RBoHD, RBoHF, and RBoHJ and ROS induces Ca^{2+} import and promotes K^+ efflux in stomata. Ca^{2+}, in turn, induces Na^+ efflux through several CBL-CIPK pathways. (*c*) Ca^{2+} also induces Ca^{2+} release from the vacuole, thus reinforcing the Ca^{2+} signal. Na^+ is sequestered in the vacuole through transporters that have yet to be identified. The antiporters NHX1 and NHX2 influence the Na^+/K^+ ratio, as they exchange either Na^+ or K^+ for H^+. Similarly, NHX5 and NHX6 maintain the pH and Na^+/K^+ balance of the prevacuolar compartments (PVCs) and *trans*-Golgi network (TGN), which affect protein sorting. NHXs are fueled by ATPases that transport H^+ in the opposite direction. (*d*) Salt induces enzymes that convert phospholipids, including the production of phosphatidic acid (PA). PA causes the internalization of auxin transporter PIN2. The SnRK2 subclass I (SnRK2) protein kinases interact with PA and migrate to processing bodies, where they interact with VCS to regulate mRNA degradation. (*e*) In the cell wall, Na^+ is hypothesized to decrease pectin crosslinking, which might be sensed by the receptor-like kinase FERONIA (FER), causing a late-response Ca^{2+} wave at the end of the quiescent phase, just before the start of growth recovery. Inside the cell, cortical microtubules depolymerize starting 30 min after salt application. The microtubule network is rebuilt during the quiescent phase, and eventually microtubule bundling is increased by salt. (*f*) Cellulose synthesis complexes (CSCs), responsible for cellulose synthesis, are bound to microtubules by companion of cellulose synthesis (CC) proteins, and are internalized during microtubule depolymerization. (*g*) Transcriptional changes occur after the early signaling phase. Auxin distribution rapidly changes, first through altered transport and later by local biosynthesis. Downstream ABA-dependent transcriptional networks are upregulated during the stop and quiescent phases. Ethylene plays a role both in repolymerizing microtubules and in transcriptional changes. Gibberellic acid (GA), Brassinosteroids (BR), and jasmonic acid (JA) play an important role during the growth recovery phase.

increase in calcium, tissue-specific subtle differences were found in the amplitude and oscillation of calcium peaks induced by osmotic and salt stress (85). Subsequently, local salt application showed that the propagation of long-range calcium waves was induced by high salt, but not by osmotic stress treatment. These waves are almost instant, initiate 10 s after salt application, and can propagate throughout the root and even reach the leaves within 30 s (23). As described earlier, GIPC produced by MOCA1 can be bound by monovalent cations and initiate a Ca^{2+} influx; however, the involved Ca^{2+} channel remains to be identified (71) (**Figure 2*a***). The propagation of Ca^{2+} waves is facilitated by TWO-PORE CHANNEL1 (TPC1), which mediates Ca^{2+} release from the vacuole; consequently, the speed of the long-range calcium signals in *tpc1* mutants was drastically reduced (23) (**Figure 2*c***). In general, calcium waves and spikes are not a sodium-specific phenomenon, as various other stimuli such as touch, cold, and osmotic stress are also known to induce changes in cellular calcium concentrations; however, the peak amplitude, peak oscillation pattern, and wave propagation do show salt specificity.

Salt overly sensitive (SOS) pathway: a salt stress–induced cellular signaling pathway that comprises SOS1, SOS2, and SOS3 to regulate sodium concentration in the cytosol

14–3–3 proteins: a family of conserved regulatory molecules with the ability to bind to various signaling proteins, such as protein kinases, phosphatases, and receptors

To mediate the output of calcium fluxes, calcineurin B–like proteins (CBLs) bind calcium and cause protein phosphorylation through their interaction with CBL-interacting protein kinases (CIPKs). Over the years, different CBL-CIPKs have been found to coordinate a set of cellular responses to sodium by decoding the Ca^{2+} signals produced (reviewed in 111). The salt overly sensitive (SOS) pathway is the best-characterized CBL-CIPK pathway (**Figure 2*b***). Calcium is sensed by SOS3/CBL4 (104), which binds to SOS2/CIPK24 (57, 103). The SOS2-SOS3 complex phosphorylates the H^+/cation antiporter SOS1/NHX7 (57), which can transport sodium out of the cell. Mutation of any of the components of the SOS pathway results in severely reduced salt tolerance in *Arabidopsis*, crop species, and halophytic species (reviewed in 68, 111). This pathway was extended with CBL10, which is more abundant in shoots (86, 140). CBL10 can also form a complex with SOS2, and this complex plays a role in sodium sequestration in the vacuole. However, it remains unknown which transporter is activated by the CBL-CIPK complex in this case. An additional pathway activating SOS2 is mediated by SOS2-LIKE PROTEIN KINASE5 (PKS5) and 14–3–3 proteins (178). PKS5 can phosphorylate SOS2 in control conditions, repressing SOS2 by inducing binding with 14–3–3 proteins. During salt stress, calcium binds the 14–3–3 proteins and PKS5 activity is repressed, showing an alternative pathway through which calcium can modulate SOS2 activity. The annexin AtANN4 mediates salt stress–induced Ca^{2+} increase, while its subsequent phosphorylation by SOS2 attenuates the Ca^{2+} wave to generate a salt-specific calcium signal (106). Other CBL-CIPKs are involved in regulating energy status and proton and potassium transport (reviewed in 111). One example is CPK3, which phosphorylates the vacuolar TWO-PORE K+ CHANNEL 1 (TPK1), and *cpk3* and *tpk1* mutants both show increased salt sensitivity (98) (**Table 1**). Together, these recent advances in understanding CBL-CIPK pathways have begun to reveal how calcium fluxes induce different cellular salt stress responses.

Salt stress rapidly generates elevated levels of apoplastic ROS molecules (**Figure 2*b***), such as hydrogen peroxide, singlet oxygen, superoxide, and hydroxyl radicals, which disrupt redox homeostasis and cause oxidative damage to plant cells (117). The RESPIRATORY BURST OXIDASE HOMOLOGs (RBOHs) are plant ROS-producing NADPH oxidases. In *Arabidopsis*, the expression patterns of 10 RBOH genes (*RBOHA–RBOHJ*) are changing dynamically, and they generate ROS waves upon salt stress over a 24-h period (176), indicating that the complex ROS production network is constantly active and plays a central role during early salt response.

ROS initially interact with polyamines to increase cytosolic free Ca^{2+}, which by binding N-terminal EF-hand motifs of Rbohs causes the activation and phosphorylation of Rboh proteins (130, 182). Mathematical modeling showed that under salt stress, crosstalk between ROS and Ca^{2+} signaling is necessary to spread the Ca^{2+} signal between cells (43). Accordingly, the *atrbohd atrbohf* double mutant, which is hypersensitive to salt stress, exhibited a reduction in cytosolic free

Table 1 Selection of *Arabidopsis* K^+ channels and transporters with a function in Na^+ transport or K^+ homeostasis during salt stress

Family	Abbreviation	Total	Member	Selectivity	Cellular localization	Function during salt stress	Reference(s)
Shaker-type potassium channels	Kv-like	9	AKT1	K^+ influx	Plasma membrane	During salt stress, uptake of potassium in the root is decreased because of membrane depolarization	151
			KAT1	K^+ influx	Plasma membrane	Guard cell K uptake, SnRK2.6 regulated	149
			GORK	K^+ efflux	Plasma membrane	K^+ efflux caused by membrane depolarization	151
Two-pore K^+ channel	TPK	6	TPK1	K^+	Tonoplast	Ca^{2+}-dependent phosphorylation and activation during salt stress	98, 109
High-affinity K^+ transporter	HKT	1	HKT1;1	Na^+	Plasma membrane	Na^+ loading in the shoot phloem and Na^+ unloading in the root xylem; in adult plants, HKT1 overexpression increases salt tolerance	15, 118, 160
K^+ uptake permease/high-affinity K^+ transporter/K^+ transporter	KUP/HAK/KT	13	HAK5	K^+/Na^+	Plasma membrane	K^+ uptake under low K^+ saline conditions; single mutation can change selectivity from K^+ to Na^+	2, 128
			KUP6	K^+ efflux	Plasma membrane	Phosphorylated by SnRK2.6 and involved in stomatal closure as response to ABA	135
K^+ efflux antiporters	KEA	6	KEA1/KEA2	$K^+ \leftrightarrow H^+$	Chloroplast	Influences chloroplast function and osmoregulation in control conditions; phenotype can be rescued by salt application	96
Na^+/H^+ exchangers	NHX	8	NHX1/NHX2	$K^+/Na^+ \leftrightarrow H^+$	Tonoplast	Vacuolar K^+ homeostasis during salt stress	9
			NHX5/NHX6	$K^+/Na^+ \leftrightarrow H^+$	TGN/PVC	K^+ homeostasis and pH regulation of endomembrane compartments, affecting protein sorting during salt stress	12, 142
			NHX7/SOS1	$Na^+ \leftrightarrow H^+$	Plasma membrane	Exclusion of sodium from the root; *sos1* mutants are more salt sensitive	141, 153, 154

(*Continued*)

Table 1 (*Continued*)

Family	Abbreviation	Total	Member	Selectivity	Cellular localization	Function during salt stress	Reference(s)
Cation/H^+ exchangers	CHX	28	CHX13	$K^+/Na^+ \leftrightarrow H^+$	Plasma membrane	Required for root halotropic response in low K^+ conditions	29, 186
			CHX17	$K^+/Na^+ \leftrightarrow H^+$	Plasma membrane/PVC	K^+ accumulation in the root during salt stress	19
			CHX21	$K^+/Na^+ \leftrightarrow H^+$	Plasma membrane	Affects leaf Na^+ concentration during salt stress	58

This table shows the channel/transporter families of *Arabidopsis* (as described in 113), abbreviation of family name, total number of members in the family (as described in 113), family member, ion selectivity [antiports (↔), might transport both ions (/)], cellular localization [*trans*-Golgi network (TGN), prevacuolar compartment (PVC)] and function during salt stress for channels and transporters mentioned in this review.

Ca^{2+} and plasma membrane Ca^{2+} influx (107). Moreover, the calcium signaling complex CBL1/9-CIPK26 interacts with and phosphorylates AtRbohF (35). Thus, in the early stages of salt stress, ROS and Ca^{2+} signals work together to affect cellular pH and ion homeostasis (**Figure 2**).

Phospholipid and Protein Kinase Signaling

Modifications in membrane phospholipids act as signaling components during salt stress. Within 5 min of exposure to both salt and osmotic stress, several phospholipid signals are produced, including polyphosphoinositides and phosphatidic acid (PA). The latter is produced by phospholipase C or phospholipase D (PLD) activity in *Chlamydomonas* and several plant species, including *Arabidopsis* and rice (164, 165). Several *pld* mutants are more salt sensitive (8). As some C2 domain–containing PLDs use calcium as a cofactor (139), PA formation is yet another putative downstream response of calcium signaling during salt stress. Furthermore, Ca^{2+}-independent ζ -type PLDs influence the direction of root growth during salt stress (51, 93). PA itself can bind and influence various downstream proteins in ABA signaling and auxin transport (116), two hormone signaling pathways with a major role in salt stress signaling. PA also influences sodium transport through MITOGEN-ACTIVATED PROTEIN KINASE6, which has SOS1/NHX7 as a downstream target (180). In rice, MITOGEN-ACTIVATED PROTEIN KINASE6 is a downstream effector of the lectin RLK SALT INTOLERANCE I, which is dephosphorylated by Protein Phosphatase 2A under salt and mediates plant salt sensitivity by regulating ethylene homeostasis (101, 187). In summary, PA influences protein localization and activation during salt and osmotic stress, thereby influencing hormone signaling and sodium transport (**Figure 2*d***).

Phospholipids: a class of lipid molecules that are the main component of cellular membranes

Phosphatidic acid (PA): a type of phospholipid serving as a rapid and transient signaling molecule upon stress responses

Other PA-binding proteins are the protein kinase family SUCROSE NONFERMENTING1-RELATED PROTEIN KINASE2 (SnRK2) subclass 1 proteins (76, 163) (**Figure 2*d***). These protein kinases are activated by osmotic and salt stress in an ABA-independent manner, in contrast to subclasses 2 and 3, which are required for ABA responses (95). During osmotic stress, ABA-independent SnRKs localize to intercellular foci within 5 min of salt application (115, 157). Recently, these foci were found to be mRNA-processing bodies, in which SnRK2s bind to and phosphorylate VARICOSE (VCS), an mRNA-decapping protein involved in RNA stability (157). VCS and other members of the decapping protein complex, including DECAPPING and SM-like proteins, as well as 5′-to-3′ exoribonucleases, are required for 5′-to-3′ mRNA degradation. The ABA-independent SnRKs influence mRNA levels of aquaporins and the enzyme CYP97B2, involved in auxin biosynthesis (80) (see the section titled Auxin Transport and Local Auxin Response). Finally, the isoforms SnRK2.4 and 2.10 influence root growth and branching during salt

stress (80, 115). In general, the stability of coding mRNAs was previously shown to be affected by osmotic stress (127), and coordinated degradation of mRNA through regulation of several 5′-to-3′ degradation components is an emerging response to salt stress (reviewed in 81).

Maintaining Sodium/Potassium Homeostasis Through Cellular Ion Transport

Proton pumps play an essential role in cellular ion transport and sodium sequestration. P-type ATPases (P-ATPases) localize at the plasma membrane and maintain a negative membrane potential (**Figure 2*a***). During salt stress, the positive charge of sodium ions depolarizes the membrane potential. Consequently, the P-ATPases become more active to counteract this depolarization, decreasing sodium import and potassium export in poplar (159). Moreover, P-ATPases fuel the exclusion of sodium from the root, a process mediated by the sodium/proton antiporter SOS1/NHX7 (52). Similarly, the tonoplast-localized H^+-ATPases build up the proton-motive force necessary for sodium sequestration in the vacuole (11) (**Figure 2*c***). These functions of H^+-ATPases show how H^+-ATPases influence the transport of other ions through their effect on membrane polarity and pH of intercellular compartments.

The cellular balance between sodium and potassium is important for plant survival in saline soils. The molecular similarity between the two causes potassium replacement by sodium, although it cannot take over the function of potassium in cellular processes (14). A multitude of channels, transporters, and antiporters play a role in maintaining sodium/potassium homeostasis during salt stress (4), one of which is the family of NA^+/H^+ EXCHANGERS (NHXs) (**Table 1**). The plasma membrane–localized NHX7/SOS1 predominantly transports sodium and is important for the exclusion of sodium from the root (**Figure 2*b***) (68). Apart from the plasma membrane–localized NHX7 and NHX8, the other members of the NHX family localize to intercellular compartments and can transport both potassium and sodium, which has demonstrated relevance for salt tolerance of crop plants (4). In *Arabidopsis*, overexpression of *NHX1* increases salt tolerance and sodium accumulation in the shoot during salt stress (6). Because of this accumulation and the tonoplast localization of NHX1 and NHX2, these NHXs are hypothesized to influence sodium sequestration in the vacuole. However, in planta they are likely involved mainly in vacuolar potassium homeostasis (**Figure 2*c***) because of their equal affinity for potassium (70, 110). This hypothesis was confirmed by a decrease in vacuolar potassium content in *nhx1 nhx2* double mutants accompanied by an increase in sodium content in *nhx1 nhx2* leaf tissue during salt stress (9). The impaired potassium homeostasis resulted in decreased growth and delayed stomatal closure. In addition, *nhx5 nhx6* mutants exhibit increased salt sensitivity. Endosome-localized NHX5 and NHX6 increase the pH of endomembrane compartments and thereby influence the sorting of transmembrane proteins (**Figure 2*c***) (12, 142). Taken together, these data show that NHX7/SOS1 has an important role in sodium export, while the endomembrane- and tonoplast-localized NHXs seem to be important for protein sorting, intercellular potassium transport, and endomembrane pH maintenance during salt stress.

Compared with NHXs, considerably less is known about the role of other potassium transporters and antiporters. However, the role of HIGH-AFFINITY POTASSIUM TRANSPORTER 5 (HAK5) has been shown to facilitate potassium transport under conditions of low extracellular potassium or high salt (128) (**Figure 2*a***; **Table 1**). A single amino acid mutation increases HAK5's potassium affinity and decreases its affinity for sodium in a heterologous system (2). HAK5 expression is upregulated in the loss-of-function mutant of the ethylene production regulator *ETO1*, which was associated with higher K^+ levels in *eto1* mutants during salt stress (69). Furthermore, different shaker-type potassium channels are important for maintaining sodium/potassium homeostasis (reviewed in 151) (**Figure 2*a***). GATED OUTWARDLY-RECTIFYING K^+ CHANNEL (GORK) and ARABIDOPSIS K^+ TRANSPORTER 1 (AKT1) are responsible

for potassium efflux during salt stress and influx during osmotic stress, respectively. Furthermore, K^+ CHANNEL IN ARABIDOPSIS THALIANA 1 (KAT1) and GORK have a role in stomatal closure in response to ABA (**Table 1**). Finally, CATION/H^+ EXCHANGER 13 (CHX13) has a role in root growth direction on a salt gradient, while CHX17 influences K^+ accumulation in the root and CHX21 influences Na^+ accumulation in the shoot (**Table 1**).

Quiescent phase: a plant root growth stage of extremely slow growth

Although the expression of many other cation transporters changes during salt stress (108), the biological role of these transporters has not been determined in many cases. Moreover, most of our knowledge of transporters that have a role in sodium/potassium homeostasis is at either the cellular or whole-organ level, but rarely integrated between these levels. Recent advances in our understanding of calcium transport are partly due to the generation of high-resolution reporters. Similar reporters for sodium and potassium could yield insights into the functions of transporters and the relevance of tissue-specific sodium/potassium balance, as well as the transporters' subcellular and tissue-specific function.

CELLULAR COMPONENTS FACILITATING A MULTIPHASE GROWTH RESPONSE

Salt-Induced Growth Rate Phases

After early signaling, the growth rate changes, going through four different growth phases. In the main root, salt rapidly decreases the rate of growth and induces a temporary pause in growth referred to as the quiescent phase (53); thereafter, the growth rate partially recovers, approximately 9 to 13 h after salt application (**Figure 2**). A similar growth curve has been described in lateral roots; however, here the quiescent phase could last more than 2 days (36). In lateral roots of Col-0 seedlings, the growth rate was reduced by 90%, whereas the primary root growth rate was reduced by only 50%. Less is known about salt-induced changes in the shoot growth rate. Leaf elongation rates in barley show a similar pattern after salt application to that of roots (48). Here the quiescent phase is shorter; growth recovery starts between 30 and 50 min after stress application, and the growth rate eventually is reduced by 60%.

The severity and length of salt-induced quiescence are regulated by hormone levels (53). The ABA levels and ABA signaling transcripts were found to correlate with the quiescent phase. ABA concentrations were reduced during the recovery phase, whereas jasmonic acid (JA), brassinosteroids (BR), and gibberellic acid (GA) levels rise, and downstream transcriptional programs are induced (**Figure 2*g***). GA induction was observed from 5 to 8 h after salt treatment, when it could function in growth recovery, followed by a reduction at 24 h (53), consistent with earlier reports of reduction of bioactive GAs after several days of salt treatment (1). JA biosynthesis and signaling are activated by salt stress and consequently inhibit plant cell elongation and primary root growth (169). Accordingly, a JA insensitive mutant *jai3-1* recovers sooner after salt treatment and grows faster at the homeostasis stage compared with wild type, indicating that JA inhibits root growth during and just after the growth recovery phase (53). Modulation of growth rate coincides with changes in cellular water and solute management, photosynthesis, cell elongation, and cell division.

The Cell Wall as a Modulator of Cell Expansion

Cell expansion and division are both crucial for maintaining growth. Salt application reduces cell division (174) and induces cell swelling close to the root tip (45). Cell swelling is influenced by the structure of the cell wall, and the production of the cell wall's load-bearing component, cellulose, and cellulose crosslinking by pectins are modified upon exogenous salt application (reviewed in 46). Calcium ions, normally required for pectin crosslinks, can be replaced by sodium ions, likely

compromising pectin structure (120). Within 1.5 h of salt application, the microtubule network starts to be broken down and repolymerized, a process that completes after 4–8 h (40). Because the tips of microtubules are bound by cellulose synthesis complexes (CSCs), CSCs are internalized and delivered back to the plasma membrane during the microtubule repolymerization phase (40). Companion of cellulose synthesis (CC) proteins are a part of the CSCs and facilitate both the bundling of microtubules and the association of CSCs to microtubules (83). In *cc1* mutants, CSCs dissociate from the microtubules, resulting in decreased growth and cellulose synthesis during salt stress. Interestingly, the timing of these cell wall modifications precede the above-described changes in growth rate; CSC internalization and microtubule depolymerization coincide with the quiescent phase and microtubule bundling preceding the recovery phase (**Figure 2*f***).

Genome-wide association study (GWAS): a genetic approach to associate natural genetic variation with particular traits

Recently, new regulators of salt-induced microtubule dynamics have been identified. Microtubule depolymerization requires changes in the activity of the kinase PROPYZAMIDE HYPERSENSITIVE1 (PHS1) and SPIRAL1 (SPR1). PHS can phosphorylate and inhibit polymerization of α-tubulin. Normally, this function is inhibited through the phosphatase function of PHS1 itself; however, osmotic stress blocks this phosphatase activity, causing phosphorylation and depolymerization of α-tubulin (49). SPR1 binds to the plus ends of microtubules, where it inhibits the depolymerization of microtubules. Salt induces 26S proteasome–mediated degradation of SPR1 and thereby causes depolymerization of microtubules (173). In contrast, microtubule repolymerization and bundling require ethylene signaling (34) and PA recruitment of microtubule-associated protein 65 (183). Taken together, both microtubule depolymerization and repolymerization as well as increased bundling are necessary to maintain long-term growth rates and tolerance.

A class of receptors, the RLKs, can perceive salt-induced changes in cell wall properties. One RLK with a clear role in salt stress signaling is FERONIA (FER) (45) (**Figure 2*e***). As a response to high salt stress, FER mediates late-response calcium waves, which are necessary to prevent cell bursting. Consequently, *fer* mutants show increased salt-induced cell swelling and eventually bursting close to the root tip. FER is thought to directly perceive decreased crosslinking of pectin, because it binds pectin in vitro and addition of exogenous calcium or borate can complement the *fer* phenotype, likely by promoting pectin crosslinking. Alternatively, FER might indirectly perceive changes in the cell wall structure through LEUCINE-RICH REPEAT EXTENSINS and the peptides RAPID ALKALINIZATION FACTOR (RALF) 22/23 (185). Another RLK with a possible role in salt stress, MIK2/LRR-KISS, was identified in a genome-wide association study (GWAS) for natural variation in biomass during salt stress (74). MIK2/LRR-KISS expression levels can be linked to salt tolerance (42, 74), and the receptor might perceive changes in cell wall composition, since an *mik2/lrr-kiss* mutant shows a decreased response to cellulose synthesis inhibition (171). Beside the peptides RALF22/23, salt induces the expression of the plant elicitor peptide 3 (PEP3), which can be perceived by PEP receptor 1 (PEPR1) and eventually increase salt tolerance (125). This could be another perception mechanism of cell wall modifications, because PEP3 expression is induced by cell wall damage (42). In summary, components of the cell wall are affected by salt stress and can modulate cell expansion. FER, MIK2/LRR-KISS and PEPR1 are three examples of receptors that could perceive these changes in the cell wall either directly or through the exclusion of small signaling peptides such as RALFs or PEPs. This perception is in turn required to induce important responses to salt stress, likely leading to salt adaptation and tolerance.

Osmotic Adjustment

Loss of water, due to decreased osmotic pressure, is one of the problems plants face when growing in salinized soils. To maintain cell volume and turgor in this situation, plants can compensate

for changes in osmotic pressure with organic solutes. Accumulation of osmolytes in the cytoplasm for osmotic adjustment is one of the major salt tolerance mechanisms of halophytes (47). These osmolytes are low-molecular-weight compounds including proline, sugar alcohols, sorbitol, quaternary ammonium compounds, and α-amino nitrogen. Metabolomic profiling analysis of *Arabidopsis*, rice, and lotus reveals that the balance between amino acids and organic acid is a conserved metabolomic response upon salt treatment (148). The osmolyte profiles may vary from species to species upon salt stress. For instance, some halophytic species accumulate sucrose as compatible solute, while others synthesize proline (reviewed in 156).

Suberin: a complex insoluble plant cell wall polymer made from long-chain fatty acids and glycerol to prevent mineral ions and water from passing through the root endodermis

Osmotic adjustment in response to salt stress decreases the growth rate by redirecting energy sources to the production of organic solutes (121). This major allocation of resources is estimated to be impossible without uptake of NaCl, because it would take away too many of the resources produced by photosynthesis, reducing those available to maintain growth. This calculation showed that plants need to take up sodium to maintain turgor; however, in this case they need to be able to deal with the toxic effects of sodium (122), described as tissue tolerance.

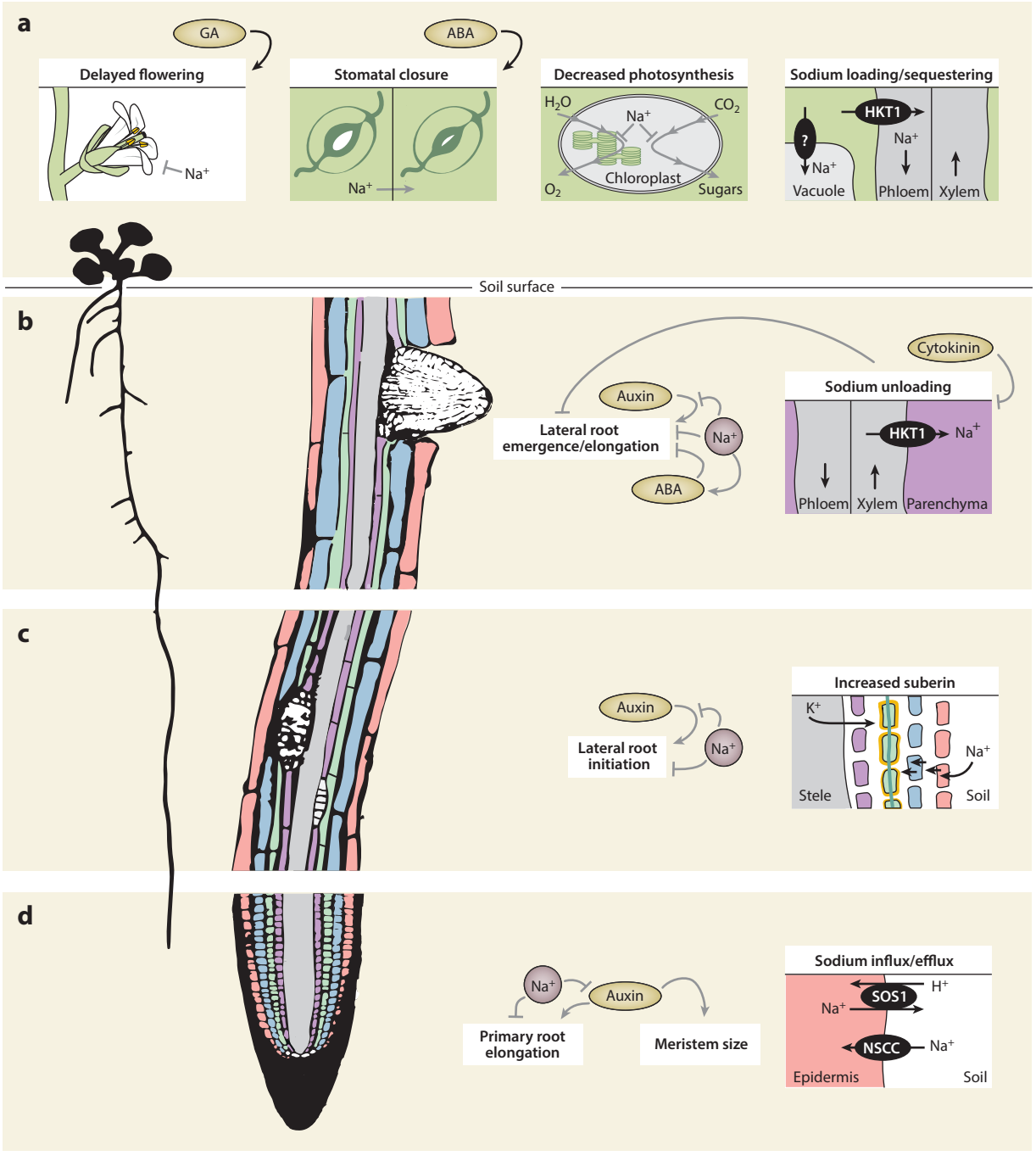
Salt Affects Photosynthesis

In addition to reallocating resources for osmotic adjustment, a decrease in photosynthesis reduces available resources and therefore growth in response to salt. The closure of stomata reduces the amount of CO_2 available for fixation, although increasing the CO^2 concentration can only partially recover the photosynthetic rate (21). This finding suggests that there is also an ionic effect, or at least a stomatal closure–independent effect, of sodium on photosynthesis. The activity of CO_2-fixing enzymes decreases during salt stress, and interestingly the tolerance of these enzymes for Na^+ in vitro differs among species (17), although the relevance of this observation for in planta photosynthetic capacity has not been addressed. The proton-motive force necessary for energy production in chloroplasts depends on close coordination between pH and electro potential changes over thylakoid membranes (17). Sodium ions can disturb this balance because of their positive charge and effect on pH. Ion channels and transporters with a potential role in maintaining chloroplast function during salt stress have recently been reviewed (17). For example, a mutant that lacks chloroplast-localized K^+ Efflux Antiporter 1 (KEA1) and KEA2 shows decreased photosynthetic capacity, and applying sodium can rescue this phenotype (**Table 1**). In addition, chloroplasts produce retrograde signals to communicate chloroplast status, affecting signaling pathways relevant to salt stress responses (25). Overall, Na^+ influences photosynthesis by disrupting the proton-motive force and chloroplast function and by interfering with CO_2-fixing enzymes (**Figure 3*a***).

SPATIAL ASPECTS OF SALT STRESS RESPONSES

The Endodermis as a Barrier

The above-described cellular processes take place within the context of a tissue. Thus, tissue-specific responses and transport mechanisms eventually determine organ growth and development during salt stress, starting with ion and water transport throughout the plant. There are three transport routes of water and solutes between the soil and the root vasculature: apoplastic transport through the extracellular space, symplastic transport through neighboring cells connected by plasmodesmata, and transcellular transport through cells via channels and transporters. All three transport routes can contribute to the import of salt into the root. The endodermal tissue surrounding the stele is important in regulating apoplastic and transcellular transport due to the deposition of lignin and suberin (reviewed in 30). Lignin is deposited in bands around endodermal



(Caption appears on following page)

Figure 3 (*Figure appears on preceding page*)

Relevance of tissue-specific ion transport, hormone signaling, and tissue growth for salt-induced phenotypic changes. (*a*) In the *Arabidopsis* shoot, Na^+ delays flowering, and induces abscisic acid (ABA)-mediated stomatal closure to reduce water loss. Na^+ decreases the photosynthetic capacity by interfering with the proton-motive force and reducing the effectivity of CO^2-fixing enzymes. To decrease the shoot Na^+ levels, Na^+ can be both sequestered in the vacuole and loaded into the phloem by HKT1 for transport back to the root. [Flower image adapted from Figshare (F. Bouché, 2018_Flower_*Arabidopsis*, **https://figshare.com/articles/Flower_Arabidopsis_2018/7159928**); CC BY 4.0.] (*b*) In the root, HKT1 unloads Na^+ and transports it back to parenchymal cells, a process that is inhibited by cytokinin. The HKT1-induced accumulation of sodium in the root can reduce lateral root growth in young seedlings. In this part of the root Na^+ reduces lateral root growth by producing ABA and reducing auxin. (*c*) The initiation of lateral roots is possibly decreased through posttranscriptional changes in AUXIN RESPONSE FACTORS (ARFs). The endodermis forms an important barrier for Na^+ influx to the stele; increased suberization in lower parts of the root blocks the transcellular transport of Na^+ to the stele, while the Casparian strip blocks the efflux of K^+ from the stele. (*d*) Sodium that enters the root through nonselective cation channels (NSCCs) can be returned to the soil by SOS1/NHX7. At the root tip, salt decreases meristem size and primary root elongation, partly through a reduction in local auxin levels. (*a–d*) Overall, the degree of primary and lateral root growth inhibition can vary between genotypes, resulting in different root architectural responses to salt stress. Auxin produced in the shoot is transported down the root stele and moves back up through the epidermis. Salt-induced changes in global auxin transport decrease auxin-mediated growth and development, while local auxin biosynthesis can contribute to lateral root growth and initiation. ABA is synthesized both in leaves and around the root stele; in the root, it inhibits lateral root elongation, while it promotes stomatal closure in the leaves. In the end, tissue-specific responses work together to generate a complex phenotypic response to salt, eventually defining salt tolerance. Seedling and root drawings are based on pictures by Iko Koevoets.

cells; together these bands form the Casparian strip and regulate the apoplastic transport of solutes to the stele. Suberin is a waxy substance that is deposited between the cell wall and the plasma membrane and thereby regulates influx and efflux of both water and solutes during transcellular transport through the endodermis.

The integrity of the Casparian strip affects the deposition of suberin, making it difficult to determine the independent effects of these two barriers in ion transport (30). The recent identification of the RLK SHENGEN3 (SGN3) has resolved this problem, because SGN3 is required not only for Casparian strip integrity but also for increased suberin deposition upon sensing Casparian strip defects (31). *sgn3* mutants demonstrate the function of the Casparian strip without increased suberin deposition. Interestingly, the Casparian strip alone does not influence the accumulation of most ions, including sodium, in control conditions, while potassium content is decreased (136). This finding suggests that the Casparian strip plays a role in blocking potassium leakage from the stele. In contrast, suberin deposition mutants show a higher permeability to sodium in particular (reviewed in 30). Thus, the endodermis could play a dual role in maintaining a low sodium/potassium ratio by both minimizing potassium leakage from the stele and limiting sodium import into the xylem (**Figure 3*c***). Suberization deposition dynamically changes during salt stress, resulting in continuous suberization alongside major parts of the root (7). This increase in suberin deposition is dependent on endodermal ABA signaling, wherein ethylene signaling plays an antagonistic role by decreasing suberization in metal-deficient conditions. Suberin-deficient genotypes show higher sodium accumulation after salt treatment, and both root growth and seed production are reduced, suggesting that salt stress–induced suberization could contribute to salt tolerance (7).

Casparian strip: a strip formed of bands of lignin around endodermal cells that influences the transport of ions and nutrients

At a concentration of 100 mM NaCl outside the root, the hydraulic conductivity (i.e., water uptake capacity) of the root decreases by 70% (18). Interestingly, in *Arabidopsis*, there is natural variation in this hydraulic conductivity (161), suggesting that plants employ different water management strategies during salt stress. Suberin decreases water flow (158); therefore, increased suberization may reduce hydraulic conductivity. Hydraulic conductivity is facilitated to a large extent by aquaporins, which are pores that facilitate water transport over membranes (67, 137). Both their localization and their activity are altered by salt stress and downstream sodium signals (reviewed in 102) (**Figure 2*d***). Finally, the closure of stomata largely influences transpiration

and therefore water transport throughout the plant. ABA is a major regulator of stomata closure, mediated by the above-described signaling components such as calcium, ROS, and PA (89) (**Figure 3*a***). In summary, stomatal closure decreases transpiration while, in parallel, aquaporin regulation and suberin deposition reduce hydraulic conductivity of the root. While the relevance of reduced transpiration to minimization of water loss is obvious, the relevance of reduced root hydraulic conductivity in response to salt is still debated. Water import might facilitate the passive import of sodium ions, or reduced hydraulic conductivity could prevent backflow of water into the saline soil.

HKT1 Function in Ion Transport

Sodium concentration in shoots is affected by the regulation of sodium transport from root to shoot by the family of HIGH-AFFINITY POTASSIUM TRANSPORTERS (HKTs) (**Figure 3*a*,*b***). The members of this family can be divided into two classes according to their affinity for either sodium or potassium, caused by a single amino acid substitution in the pore region of the transporter (112). The HKT1 subclass transports sodium, while the HKT2 subclass transports potassium. In *Arabidopsis*, AtHKT1;1 is the only member of the HKT family. AtHKT1;1 both inhibits root-to-shoot transport and facilitates shoot-to-root transport of sodium by loading sodium into the phloem stream (15) and unloading sodium from the xylem flow into xylem parenchymal cells, respectively (**Figure 3*a*,*b***; **Table 1**) (160). In other species, the role of HKTs in sodium transport is more complex because of the differing localization and affinity among members of the HKT1 families, as we discuss in the section titled Halophytes and the section titled Transgenic Approaches in Model Plants and Crops.

Overexpression of AtHKT1;1 in the stele increases salt tolerance in adult plants grown in hydroponics (118); however, high expression of AtHKT1;1 can also have a detrimental effect, possibly due to its role in root development, as shown by investigations of natural variation in AtHKT1;1 expression (13, 75). On average, accessions from coastal regions were shown to contain a higher sodium leaf content, accompanied by enrichment of a weak allele of AtHKT1;1 (13). Subsequently, AtHKT1;1 was identified in a GWAS for root system architecture changes in response to salt, demonstrating that the higher expression of AtHKT1;1 resulted in fewer and shorter lateral roots, a phenotype that could be partially rescued by addition of potassium to the medium (75). Furthermore, the beneficial effect of AtHKT1;1 overexpression was shown to be dependent on the plant developmental stage during salt application. Even earlier in development, salt stress inhibits seed germination, which coincides with upregulation of AtHKT1;1. The *calmodulin-binding transcription activator6* (*camta6*) mutant is salt tolerant during germination and shows no upregulation of AtHKT1;1 (155). However, an *athkt1;1 camta6* double mutant regains salt sensitivity during germination. Thus, depending on the expression levels, AtHKT1;1 can be either beneficial or harmful in seed germination under saline conditions. Lastly, AtHKT1;1 is known to be repressed by the hormone cytokinin (CK), causing an increase in leaf sodium content (114). Salt stress reduces CK levels, and the accumulation of CK negatively regulates plant salt tolerance. In summary, when the root system is already established, overexpression of AtHKT1;1 can be beneficial by reducing shoot sodium concentration. However, in earlier developmental stages the high sodium/potassium ratio in the root inhibits lateral root formation, and upregulation of AtHKT1;1 is associated with salt sensitivity during germination.

Production and Transport of the Master Regulator ABA

Phytohormones are crucial endogenous chemical signals coordinating plant growth and development both under optimal conditions and during environmental challenges. ABA has key roles

in stomatal closure and regulation of root growth under abiotic stress, and guard cells and various root tissues are the hot spots for their functions (**Figure 3**) (53, 89). Abiotic stresses, including salinity and water deficit, induce the expression of ABA biosynthetic genes (e.g., *NINE-CIS-EPOXYCAROTENOID DIOXYGENASEs* (*NCEDs*), *ABA DEFICIENTs* (*ABAs*), and *ALDEHYDE OXIDASE 3*) in specific vascular tissues (10, 41, 146). In the root, expression of *ABA1–ABA3* and *NCED3* is triggered in epidermis, cortex, endodermis, and root stele layers within 3 h of salt treatment (53). ABA has been proposed to be transported from root to guard cells to promote stomatal closure; however, grafting experiments in tomato show that stomatal closure does not rely on ABA production in the root (64).The expression pattern of the rate-limiting ABA biosynthesis gene *NCED3* suggests that ABA can be synthesized in leaf vascular parenchymal cells and transported to guard cells (41, 77). The ATP-binding cassette (ABC) B-type transporters ABCG25 and ABCG40 transport ABA from leaf vascular cells to guard cells to mediate stomatal closure during drought stress (77, 97). The combination of tissue-specific ABA production and high-resolution ABA reporters could elucidate how ABA moves throughout the plant and may offer opportunities to determine ABA origin and spatial regulation in response to salt. Förster resonance energy transfer sensors have been developed for ABA, although they are not yet being used widely due to their interference with ABA signaling (72, 172). A newly developed ABA-responsive element (ABRE)-based promoter reporter could be an alternative tool to resolve spatial ABA signaling (175).

The perception of ABA by PYRABACTIN RESISTANCE/PYRABACTIN RESISTANCE-LIKE (PYR/PYL) leads to inactivation of PP2C/ABI1, which induces the phosphorylation activity of subclass III SnRKs (SnRK2.2, SnRK2.3 and SnRK2.6). Various downstream phosphorylation targets of SnRK2 kinases mediate processes including ion transport, ROS production, gene transcription, and closing of stomata. OST1/SnRK2.6 interacts with and phosphorylates specific ion channels [KAT1, K^+ UPTAKE PERMEASE 6 (KUP6), and SLOW ANION CHANNEL-ASSOCIATED 1 (SLAC1)] to mediate K^+ efflux and anion currents in guard cells, promoting stomatal closure during salt and osmotic stress (135, 149, 168). In addition, ABA-mediated gene expression is dependent on transcription factors acting downstream as substrate of SnRK2s, as well as target genes that contain *ABRE*s in their promoters (50). Additional transcription factor families containing other stress-responsive *cis*-regulatory elements in their promoters may mediate ABA-responsive gene expression; these include NAC-, MYB-, HD-Zip-, AP2/ERF-, and WRKY-type transcription factors, which are induced in various plant species in response to salt (reviewed in 63).

Aside from ABA-dependent transcriptional regulation, the dehydration-responsive element (DRE) is the major *cis*-regulatory element regulating gene expression response to salt stress (126). *Rd29A* and the AP2/ERF family transcription factor *DEHYDRATION-RESPONSIVE ELEMENT BINDING PROTEIN2A* (*DREB2A*), both of which contain DRE in their promoters, are rapidly upregulated by salt treatment (179). GROWTH-REGULATING FACTOR7 (GRF7) binds to the *DREB2A* promoter and interacts with the DREB2A protein. Knockout mutants of *grf7* exhibited a greater survival rate under high salt, and a transcriptomic analysis revealed significant increases in osmotic stress– and ABA-responsive gene expression in this mutant, including expression of *DREB2A* (87). Interestingly, the same study also supports crosstalk between ABA-dependent and -independent pathways in regulation of gene expression under salt stress, since *GRF7* also suppresses ABA-responsive gene expression. An ABRE element is consistently present in the promoter of *DREB2A*, and expression of *DREB2A* is regulated by the ABA signaling cascade, implying crosslinking between ABA-dependent and -independent gene regulation pathways (88). Taken together, major advances have been made in deciphering the ABA signaling cascade and downstream responses, while the relative contribution of tissue-specific ABA production and transport remains a debated topic.

Auxin Transport and Local Auxin Response

A hallmark of a plant's response to abiotic stress is root developmental plasticity (91). Long-distance shoot–root downward auxin transport and local cellular movements to maintain auxin maxima are indispensable for plant root growth and adaptation to environmental cues. Both global auxin transport and intracellular auxin movements contribute to maintenance of local auxin maxima (reviewed in 92). Salt and osmotic treatments induce PIN-FORMED2 (PIN2) efflux facilitator internalization (189). When exposed to a salt gradient, plant roots exhibit a salt avoidance (halotropism) response due to rapid (within 1 h) asymmetric auxin distribution mediated by PIN2 internalization (51). The response is enhanced by elevation of the auxin influx carrier AUX1 at the non-salt-exposed side of the roots, as well as a transient accumulation of PIN1 (51, 170). This internalization of PIN2, but not AUX1, is facilitated by the PA-producing phospholipases PLDζ 1 and PLDζ 2 (51, 93) (**Figure 2*d***). Apart from PIN and AUX auxin polar transporters, ABCB transporters exhibit spatiotemporal expression patterns in response to salinity (20, 181), which might also contribute to the dynamic auxin distribution to mediate plant growth response.

Auxin biosynthesis pathways are temporally and spatially regulated in response to a range of salt stresses in multiple plant species (38, 61, 92, 138, 177). Short-term (3-h) high salinity stress (≥100 mM NaCl) of cucumber seedlings induces expression of YUCCA genes, which are part of the major Trp-dependent auxin biosynthesis pathway, leading to increased IAA levels (177). In addition, the *Brassica*-specific indole-3-acetaldoxime (IAOx) auxin biosynthesis pathway genes *CYP79B2* and *CYP79B3* show induction in both roots and shoots of *Arabidopsis* after 24 h of mild salt stress, while the double mutant shows reduced root branching on salt, suggesting a role for the IAOx pathway in regulating salt stress responses (75). Auxin is perceived through binding to the F-box protein complex TRANSPORT INHIBITOR RESPONSE1/AUXIN SIGNALING F-BOX (TIR1/AFB); auxin binding promotes interaction with the AUXIN/INDOLE-3-ACETIC ACID (AUX/IAA) family of transcription repressors, leading to the ubiquitination and degradation of AUX/IAAs and expression of AUXIN RESPONSE FACTOR (ARF) genes (100). Salinity suppresses auxin response by downregulating expression of the auxin receptor complex TIR1/AFB2 (65). In *Arabidopsis*, there are 29 *AUX/IAA* genes with functional specialization and differentiation in various aspects of plant development. Among them, AXR3/IAA17 is stabilized by the accumulation of nitric oxide under salt stress (105). Although the role of most AUX/IAA genes in salt stress response is largely unknown, an in vitro yeast one-hybrid screening assay (152) showed that stress-responsive DREB/CBF-type transcription factors bind to the promoters of a number of *Arabidopsis* AUX/IAA genes (IAA5, IAA6, and IAA19). Together, changes in auxin transport along with local tuning of auxin synthesis and responses coordinate roots' tissue-specific growth responses to salt.

Salt Inhibits Root Growth and Reshapes Root System Architecture

As mentioned above, plants have evolved strategies to coordinate hormone signals to dynamically modulate their spatial growth and reshape their architecture in response to environmental changes. Roots are the frontline organs in contact with salt in the soil, and they need to adapt in order to maintain growth and uptake of nutrients and water. Salt stress in general reduces root mass and modifies the distribution of different root system architecture components, differentially affecting the growth rate of the main root and lateral roots and inhibiting lateral root formation. Automated phenotyping platforms have been developed for two- and three-dimensional imaging systems, allowing the capture of root dynamic growth in response to environmental factors (73, 119, 143).

SUMOylation: a posttranslational protein modification process that involves addition of small ubiquitin-like modifiers

Lateral root initiation: prior to the lateral root primordia formation, xylem pole lateral root founder cells undergo nuclear migration and the first asymmetric cell division, a process regulated by auxin, auxin response factors, and their downstream targets

Salt inhibition of both main and lateral roots was observed when measured after 4–8 days (36, 73). Salt reduces lateral root elongation in *Arabidopsis* Col-0, but seems not to affect lateral root density when observed macroscopically, by counting emerged lateral roots (73). Yet, lateral root development is partially blocked by salt stress in developmental stages V–VI just prior to emergence (37, 115). Lateral root growth is modulated primarily via auxin (99) (**Figure 3*b***). ARFs are crucial mediators for lateral root development in response to abiotic stress factors including salt. As an example, ARF4 posttranscriptionally targeted by microRNA390 is involved in lateral root development under salt in poplar, while ARF7 is posttranslationally SUMOylated on the dry side of *Arabidopsis* roots (60, 133). SUMOylation negatively affects the DNA-binding activity of ARF7 to the promoter of *LBD16*, affecting lateral root initiation during hydropatterning (133). While SUMOylation has been reported to affect salt responses as well (24), its role in salt-affected root architecture is unknown (**Figure 3*c***). Other than auxin, endodermal ABA signaling is required for salt-induced lateral root growth quiescence (37). One study showed that the ABA-SnRK2.2/SnRK2.3/SnRK2.6 core signaling pathway promotes lateral root growth quiescence induced by ABA treatment (184). Interestingly, the same study showed that *PYL8* promotes lateral root growth recovery by interacting with MYB77 to enhance auxin signaling and that auxin is able to restore the lateral root phenotype of *pyl8* knockout mutants. On the other hand, the ABA-independent SnRK2.10 affects lateral root emergence under salt, likely via the PA signaling pathway (115). Although the influence of salt on different developmental stages of lateral root primordia is still largely unknown, these data suggest interplay between ABA and auxin in mediating lateral root growth modulation by salt (**Figure 3*b***).

Root system architecture phenotyping of 31 different *Arabidopsis* accessions under salt revealed natural variation in balancing growth between primary and lateral roots. For example, some accessions exhibit greater reduction in primary root elongation than in lateral root elongation as well as changes in lateral root numbers, while others respond similarly in terms of reduction of primary root length, lateral root length, and alteration of lateral root numbers. These different root system architecture strategies in response to salt are associated in part with differences in ABA sensitivity and shoot Na^+/K^+ ratio (73). Furthermore, GWAS have exploited the natural variation in both root architecture remodeling under salt and halotropic responses to salinity to uncover the genetic controls underlying root responses (29, 75, 79). To date, few studies have focused on crops, and much still can be learned from the interspecific variation among architectural responses to salt and other abiotic stresses (91).

Aboveground Development, Growth, and Reproduction in Salt

Salt stress limits the growth and development of aboveground tissues, although the molecular mechanisms are not as intensively studied as those for roots. So far, it is not clear how the shoot apical meristem and shoot architecture are regulated by salt. A recent study showed that low soil NaCl levels reduced far red (shade)-induced hypocotyl elongation through BR and ABA signaling pathways (59). Inhibition of the shade avoidance response by salt involves repression of PHYTOCHROME INTERACTING FACTOR 4 and 5, which are important transcription factors that link shoot responses to light via auxin signaling. In addition, salt delayed flowering time in *Arabidopsis* wild-type plants, while it had the opposite effect on quadruple DELLA mutants (1), implying that GA signaling modulates salt-affected flowering time via DELLA proteins (**Figure 3*a***). Moreover, BROTHER OF FT AND TFL1, which belongs to the FT/TERMINAL FLOWER 1 gene family, is induced by salt and delays flowering time under salt, likely via the ABA pathway (147). A recent study showed that the flowering time regulator GIGANTEA (GI) interacts with and inhibits SOS2-mediated SOS1 phosphorylation in the absence of salt, whereas salt induces degradation of GI, thereby releasing SOS2 to activate the SOS pathway, involved in regulating

ion homeostasis (90). In summary, salt likely affects flowering time through multiple signaling pathways, the molecular mechanisms of which remain elusive.

Carbon Partitioning and Translocation

To sustain local tissue growth and cope with salt stress, plants are responsive in the distribution of their photosynthesis-fixed carbon, which is the major source of starch and soluble sugars. An emerging role of starch metabolism in determining plant fitness under abiotic stresses has been reviewed elsewhere (167). Under optimal conditions, starch accumulates during the day and breaks down in the dark for sucrose production and energy (56). Under osmotic stress or soil water deficit, carbon partitioning to starch is decreased, leading to osmolyte accumulation and carbon translocation to the *Arabidopsis* roots (39, 166). Similarly, salt stress induces carbon partitioning to sugars, but not to starch, in both source (leaf) and sink (root) tissues during the day in *Arabidopsis* (33). In addition, salt treatment increases the level of root sugars, including fructose and glucose, in the root of tissue-specific *Arabidopsis* AtHKT1;1 overexpressors, whereas these sugars were decreased by salt in the shoot of an *htk1;1* mutant, where an increase in tricarboxylic acid cycle intermediates was observed (62). In contrast, carbon translocation towards the roots of tomato is limited by salt stress (162). These findings imply that tissue-specific sodium content is likely playing a role in regulating spatial carbon influx and therefore limiting tissue growth.

SUCCESS STORIES: PLANTS SHOWING BEST PRACTICES FOR DEALING WITH SALINITY

Halophytes

Halophytes are plants that are able to grow and reproduce under high salt concentrations (>200 mM NaCl). In nature, most halophytic species occur in saltwater areas (marine areas or swamplands) or in dry desert regions. Halophytes share salt tolerance mechanisms with glycophytes, and they have evolved specific additional features to adapt to high-salinity conditions. For instance, osmoprotection is a general response to salt that is achieved by osmotic adjustment or ion membrane transport so as to maintain cellular osmotic and turgor pressure. Under salt stress, primary and secondary metabolites, including proline and sugar alcohols, function as osmolytes in both halophytes and glycophytes. (47). Prestress metabolite levels in the halophyte *Eutrema salsugineum* (previously known as *Thellungiella halophila*) are significantly higher than in its glycophytic relative *Arabidopsis* (54). This may explain its greater capacity for osmotic adjustment. In line with this, salt stress stimulates the accumulation of proteins involved in starch and sucrose metabolism in the leaf of *E. salsugineum* (55). Similar to glycophytes, halophytes also use osmoprotective and ion-detoxification strategies consisting of Na^+ removal from cytosol, Na^+ transport from root cells to xylem, and ion compartmentation in the vacuoles, involving SOS1, HKT1, and NHXs ion transporters, respectively. Under salt stress, most halophytes accumulate more Na^+ in their shoots than in their roots while retaining higher levels of K^+ than do glycophytes and, thus, a more optimal K^+/Na^+ ratio (47, 134). These physiological observations suggest that halophytes may employ different mechanisms in ion transportation and homeostasis in salt. Indeed, recent molecular studies showed that expression of *SOS1* and *HKT1* in the halophytes *E. salsugineum* (*EsSOS1*) and *Schrenkiella parvula* (previously *Eutrema parvula*) (*EpHKT1;2*) confers stronger salt tolerance than does that of their *Arabidopsis* homologues (*AtSOS1* and *AtHKT1;1*) (3, 66). The halophyte *Salicornia* (**Figure 4*a***) has a constitutively high level of *SOS1* expression but undetectable *HKT1* (78). In addition, next-generation sequencing has recently broadened opportunities to investigate the genomic basis of ion transportation in halophytes. For example, the *S. parvula* genome has three tandem duplicates of *SpNHX8* and two copies of *HKT1*, which are associated with higher

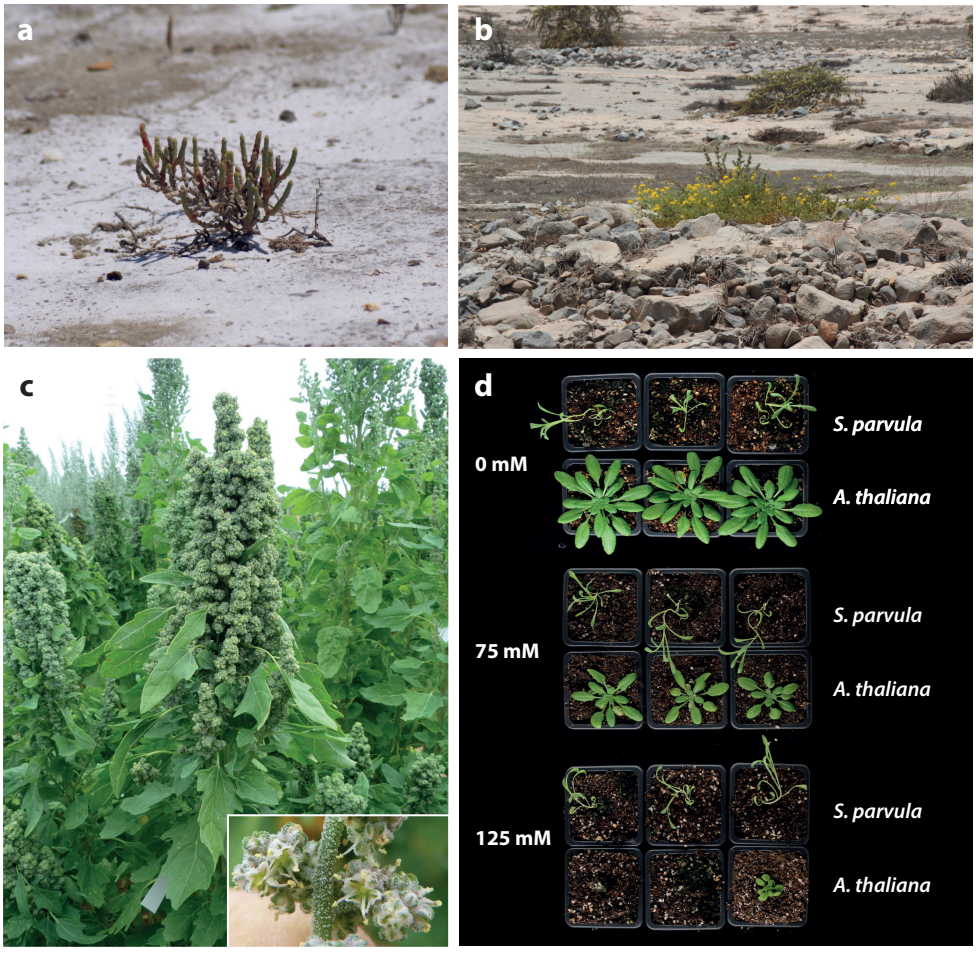


Figure 4

Success stories of salt-tolerant plants: a variety of plant species with relatively high salt tolerance. (*a*) The halophyte *Salicornia* in southern France. (*b*) *Solanum pennellii*, a wild relative of tomato that can cope with high soil salinization, in Peru. (*c*) The salt-tolerant crop quinoa on experimental fields at Wageningen University. (*d*) Growth of the model species *Arabidopsis thaliana* and its halophyte relative *Schrenkiella parvula* at 0, 75, and 150 mM NaCl. Panel *a* picture by Ronald Pierik and Christa Testerink. Panel *b* courtesy of Remco Stam. Panel *c* courtesy of Robert van Loo. Panel *d* courtesy of Hongfei Li.

transcript levels of these two genes when compared with *AtNHX8* and *AtHKT1;1* (132). This finding suggests that genomic variation may contribute to the salt tolerance of these species. In addition, *S. parvula* and *E. salsugineum*, but not *Arabidopsis*, have higher *SOS1* expression and similar genomic structures in the promoter and upstream regions of *SOS1* (131).

Secretion of salt via epidermal bladder cells (EBCs) is another salt tolerance mechanism developed by some halophytic species. Quinoa (*Chenopodium quinoa*) is a halophytic crop (**Figure 4**) containing EBCs, removal of which results in increased salt sensitivity (84). The developmental origin of EBCs is largely unclear, although they are considered to be similar to *Arabidopsis* trichome cells. A transcriptomic analysis of quinoa EBCs revealed high expression of abiotic stress–responsive genes, ion transporters, and sugar transporters, while photosynthesis-related

genes were downregulated, a finding consistent with differences between mesophyll and EBC chloroplasts (16, 188). In addition, with regard to ion transporters, EBCs show higher expression of *HKT1*, *NHX1*, *HAK5*, and the anion exchanger CLC-c but lower expression of *SOS1* when compared with leaf and root tissue (16). Further study of the development and functional mechanisms of EBCs will be crucial for understanding the salt tolerance mechanism of quinoa. Now that its genome has been sequenced (188), quinoa is an ideal halophytic food crop species to study in an effort to develop other, more tolerant crops.

Transgenic Approaches in Model Plants and Crops

Few success stories about improving crops' salt tolerance have been reported (26, 121). In several species, including tomato, overexpression of *NHX1* led to a positive effect on salt tolerance (11). In cereals, expression of *Arabidopsis CIPK16* and *AVP1* in barley improved salt tolerance in greenhouse and field conditions, respectively (145, 150). Introgression of an *HKT1* allele from an ancestral wheat relative into durum wheat improved Na^+ exclusion from the shoot and salt tolerance in the field, with increased grain yield under these conditions (123). For other species, modulation of HKT1 expression could be a promising strategy, as natural variation occurs in many different species. More subtle regulation of HKT1 may be required, however, as the beneficial effect of HKT1 expression depends on the genetic background and age of the plants (75, 118). For example, the higher salt tolerance of the wild tomato species *Solanum pennellii* (**Figure 4*b***) versus *Solanum lycopersicum* may be due in part to an HKT1 gene with lower Na^+ affinity (5). Another promising research direction toward improvement of salt tolerance is modulation of the rhizosphere. A positive effect of plant growth–promoting rhizobacteria and mycorrhiza has been reported in several species, but the mechanisms need to be determined before they can be used as a reliable way to improve salt tolerance (44, 129).

With the notable exception of quinoa, most crops are glycophytes like the model species *Arabidopsis* (**Figure 4*c***). As such, knowledge of mechanisms of salt responses and tolerance of *Arabidopsis* is expected to contribute to the development of more salt stress–resilient crops. However, knowledge gaps remain, limiting this strategy. For example, we lack an understanding of how salt is perceived by the plant and of which signaling pathways affect the different responses that together determine the complex trait of salt tolerance (**Figure 1**). Better knowledge of time and spatial resolution is needed to steer a plant's response to salt in a favorable direction. This direction may differ among individual crops, depending on whether the harvestable parts are root, shoots, or fruits, as well as on other environmental constraints.

Research on halophytic species has shown that ion sequestration, synthesis and transport of compatible solutes, and more optimal developmental plasticity play a crucial role in tolerance. While it is not clear whether halophyte tolerance mechanisms could be transferred to crops without a yield penalty, this is an interesting direction to explore further. In this case, the knowledge gap lies in our understanding of the physiology of halophytes (82). To this end, a focus on a selected number of genetically tractable model species would likely help us exploit the natural salt tolerance of halophytes as a means of improving our crops.

SUMMARY POINTS

1. The development of high-resolution calcium biosensors and the identification of downstream CBL-CIPK pathways have identified Ca^{2+} waves as an early signal in response to salt and resulted in the identification of a new cation-sensing mechanism.

2. By targeting and phosphorylating downstream components, both ABA-independent SUCROSE NONFERMENTING1-RELATED PROTEIN KINASE2 (SnRK2s) and ABA-dependent SnRK2s play pivotal roles in transcriptional regulation and posttranscriptional regulation during salt stress response.
3. Components of the cell wall modulate growth rate changes in response to sodium stress and have a function in signaling responses through receptor-like kinases (RLKs).
4. Salt induces multiphase changes in growth rate, as well as changes in root system architecture and a salt avoidance response of the main root. These responses are mediated by several hormones, including auxin and ABA.
5. Na^+ exclusion from the root, modulation of root–shoot transport, and cellular compartmentalization of Na^+, as well as maintenance of cytoplasmic osmotic balance, are crucial aspects of salt tolerance.
6. ABA is the major phytohormone responsible for salt and osmotic stress signaling in guard cells and root tissues, regulating growth, development, and metabolism.
7. HKT1 plays an important role in root stellar cells, modulating sodium transport to the shoot. Tissue-specific expression of HKT1 at specific developmental stages can contribute to plant salt tolerance.

FUTURE ISSUES

1. Sodium-specific early signaling and growth responses have been identified. Identification of upstream pathways could help in deciphering the sodium-sensing mechanism in plants.
2. For various Na^+ and K^+ transporters and channels the cellular function is known. High-resolution Na^+ and K^+ biosensors could demonstrate relevance for cellular and tissue-specific sodium/potassium homeostasis as well as the importance of local homeostasis for salt tolerance.
3. The sodium specificity of many salt-induced growth, molecular, and cellular responses remains debated. Disentangling the osmotic and sodium stress responses could help improve salt stress tolerance.
4. New phenotyping approaches offer the possibility to monitor how plants balance salt tolerance as well as growth and development, guiding effective strategies for improving crop yield.
5. As phenotypes differ between mild and severe salt stress, the underlying cellular and molecular mechanisms that modify salt responses and tolerance could be profoundly different as well.
6. Molecular and physiological comparisons between halophytes and related glycophytes could reveal new strategies to effectively cope with salt stress.
7. ABA plays an indispensable role in salt and osmotic response. However, other phytohormone signaling pathways and their interactions with ABA signaling deserve to be further explored.

8. Higher cellular and tissue resolution of the action of hormones and ion transporters should help reveal how salt-induced changes affect plant growth and development in saline conditions.

DISCLOSURE STATEMENT

The writing of this review was funded by an ERC (European Research Council) Consolidator grant (Sense2SurviveSalt) to C.T. The authors are not aware of any affiliations, memberships, funding, or financial holdings that might be perceived as affecting the objectivity of this review.

ACKNOWLEDGMENTS

We thank Charlotte Gommers, Nora Gigli Bisceglia, and Ayodeji Deolu-Ajayi for critical reading. We thank Hongfei Li for providing pictures of *S. parvula* in comparison with *Arabidopsis*, Robert van Loo for the quinoa photographs, Remco Stam for the *S. pennellii* picture, and Iko Koevoets for providing root microscopy and seedling pictures for **Figure 3**.

LITERATURE CITED

1. Achard P, Cheng H, De Grauwe L, Decat J, Schoutteten H, et al. 2006. Integration of plant responses to environmentally activated phytohormonal signals. *Science* 311:91–94
2. Alemán F, Caballero F, Ródenas R, Rivero RM, Martínez V, Rubio F. 2014. The F130S point mutation in the *Arabidopsis* high-affinity K^+ transporter AtHAK5 increases K^+ over Na^+ and Cs^+ selectivity and confers Na^+ and Cs^+ tolerance to yeast under heterologous expression. *Front. Plant Sci.* 5:430
3. Ali A, Khan IU, Jan M, Khan HA, Hussain S, et al. 2018. The high-affinity potassium transporter EpHKT1;2 from the extremophile *Eutrema parvula* mediates salt tolerance. *Front. Plant Sci.* 9:1108
4. Almeida DM, Margarida Oliveira M, Saibo NJM. 2017. Regulation of Na^+ and K^+ homeostasis in plants: towards improved salt stress tolerance in crop plants. *Genet. Mol. Biol.* 40:326–45
5. Almeida P, de Boer GJ, de Boer AH. 2014. Differences in shoot Na^+ accumulation between two tomato species are due to differences in ion affinity of HKT1;2. *J. Plant Physiol.* 171:438–47
6. Apse MP, Aharon GS, Snedden WA, Blumwald E. 1999. Salt tolerance conferred by overexpression of a vacuolar Na^+/H^+ antiport in *Arabidopsis*. *Science* 285:1256–58
7. **Barberon M, Vermeer JE, De Bellis D, Wang P, Naseer S, et al. 2016. Adaptation of root function by nutrient-induced plasticity of endodermal differentiation. *Cell* 164:447–59**
8. Bargmann BO, Laxalt AM, ter Riet B, van Schooten B, Merquiol E, et al. 2009. Multiple PLDs required for high salinity and water deficit tolerance in plants. *Plant Cell Physiol.* 50:78–89
9. Barragan V, Leidi EO, Andres Z, Rubio L, De Luca A, et al. 2012. Ion exchangers NHX1 and NHX2 mediate active potassium uptake into vacuoles to regulate cell turgor and stomatal function in *Arabidopsis*. *Plant Cell* 24:1127–42
10. Barrero JM, Rodríguez PL, Quesada V, Piqueras P, Ponce MR, Micol JL. 2006. Both abscisic acid (ABA)-dependent and ABA-independent pathways govern the induction of *NCED3*, *AAO3* and *ABA1* in response to salt stress. *Plant Cell Environ.* 29:2000–8
11. Bassil E, Blumwald E. 2014. The ins and outs of intracellular ion homeostasis: NHX-type cation/H^+ transporters. *Curr. Opin. Plant Biol.* 22:1–6
12. **Bassil E, Ohto MA, Esumi T, Tajima H, Zhu Z, et al. 2011. The *Arabidopsis* intracellular Na^+/H^+ antiporters NHX5 and NHX6 are endosome associated and necessary for plant growth and development. *Plant Cell* 23:224–39**
13. Baxter I, Brazelton JN, Yu D, Huang YS, Lahner B, et al. 2010. A coastal cline in sodium accumulation in *Arabidopsis thaliana* is driven by natural variation of the sodium transporter AtHKT1;1. *PLOS Genet.* 6:e1001193

7. Shows how salt induces suberization in lower parts of the root in an ABA-dependent manner.

12. Elucidates the function of the endomembrane localized NHX5/6 and its effect on protein sorting during salt stress.

14. Benito B, Haro R, Amtmann A, Cuin TA, Dreyer I. 2014. The twins K^+ and Na^+ in plants. *J. Plant Physiol.* 171:723–31
15. Berthomieu P, Conejero G, Nublat A, Brackenbury WJ, Lambert C, et al. 2003. Functional analysis of AtHKT1 in *Arabidopsis* shows that Na^+ recirculation by the phloem is crucial for salt tolerance. *EMBO J.* 22:2004–14

16. Describes salt secretion and gene expression changes in channels and transporters in epidermal bladder cells of the halophyte crop quinoa.

16. Böhm J, Messerer M, Müller HM, Scholz-Starke J, Gradogna A, et al. 2018. Understanding the molecular basis of salt sequestration in epidermal bladder cells of *Chenopodium quinoa*. *Curr. Biol.* 28:3075–85

17. Bose J, Munns R, Shabala S, Gilliham M, Pogson B, Tyerman SD. 2017. Chloroplast function and ion regulation in plants growing on saline soils: lessons from halophytes. *J. Exp. Bot.* 68:3129–43
18. Boursiac Y, Chen S, Luu DT, Sorieul M, van den Dries N, Maurel C. 2005. Early effects of salinity on water transport in Arabidopsis roots. Molecular and cellular features of aquaporin expression. *Plant Physiol.* 139:790–805
19. Cellier F, Conéjéro G, Ricaud L, Luu DT, Lepetit M, et al. 2004. Characterization of *AtCHX17*, a member of the cation/H^+ exchangers, CHX family, from *Arabidopsis thaliana* suggests a role in K^+ homeostasis. *Plant J.* 39:834–46
20. Chai C, Subudhi PK. 2016. Comprehensive analysis and expression profiling of the *OsLAX* and *OsABCB* auxin transporter gene families in rice (*Oryza sativa*) under phytohormone stimuli and abiotic stresses. *Front Plant Sci.* 7:593
21. Cheeseman JM. 2013. The integration of activity in saline environments: problems and perspectives. *Funct. Plant Biol.* 40759–74
22. Chen J, Mueller V. 2018. Coastal climate change, soil salinity and human migration in Bangladesh. *Nat. Climate Change* 8:981–85

23. Shows how salt induces immediate long-range Ca^{2+} signals that travel through the root.

23. Choi WG, Toyota M, Kim SH, Hilleary R, Gilroy S. 2014. Salt stress–induced Ca^{2+} waves are associated with rapid, long-distance root-to-shoot signaling in plants. *PNAS* 111:6497–502

24. Conti L, Price G, O'Donnell E, Schwessinger B, Dominy P, Sadanandom A. 2008. Small ubiquitin-like modifier proteases OVERLY TOLERANT TO SALT1 and -2 regulate salt stress responses in Arabidopsis. *Plant Cell* 20:2894–908
25. Crawford T, Lehotai N, Strand A. 2018. The role of retrograde signals during plant stress responses. *J. Exp. Bot.* 69:2783–95
26. Deinlein U, Stephan AB, Horie T, Luo W, Xu G, Schroeder JI. 2014. Plant salt-tolerance mechanisms. *Trends Plant Sci.* 19:371–79
27. Demidchik V, Maathuis FJM. 2007. Physiological roles of nonselective cation channels in plants: from salt stress to signalling and development. *New Phytol.* 175:387–404
28. Demidchik V, Tester M. 2002. Sodium fluxes through nonselective cation channels in the plasma membrane of protoplasts from Arabidopsis roots. *Plant Physiol.* 128:379–87
29. Deolu-Ajayi AO, Meyer AJ, Haring MA, Julkowska MM, Testerink C. 2019. Genetic loci associated with early salt stress responses of roots. *iScience* 21:458–73
30. Doblas VG, Geldner N, Barberon M. 2017. The endodermis, a tightly controlled barrier for nutrients. *Curr. Opin. Plant Biol.* 39:136–43
31. Doblas VG, Smakowska-Luzan E, Fujita S, Alassimone J, Barberon M, et al. 2017. Root diffusion barrier control by a vasculature-derived peptide binding to the SGN3 receptor. *Science* 355:280–84
32. Donaldson L, Ludidi N, Knight MR, Gehring C, Denby K. 2004. Salt and osmotic stress cause rapid increases in *Arabidopsis thaliana* cGMP levels. *FEBS Lett.* 569:317–20
33. Dong SY, Zhang J, Beckles DM. 2018. A pivotal role for starch in the reconfiguration of ^{14}C-partitioning and allocation in *Arabidopsis thaliana* under short-term abiotic stress. *Sci. Rep.* 8:9314
34. Dou L, He K, Higaki T, Wang X, Mao T. 2018. Ethylene signaling modulates cortical microtubule reassembly in response to salt stress. *Plant Physiol.* 176:2071–81
35. Drerup MM, Schlucking K, Hashimoto K, Manishankar P, Steinhorst L, et al. 2013. The Calcineurin B-like calcium sensors CBL1 and CBL9 together with their interacting protein kinase CIPK26 regulate the *Arabidopsis* NADPH oxidase RBOHF. *Mol. Plant* 6:559–69

36. Duan L, Dietrich D, Ng CH, Chan PM, Bhalerao R, et al. 2013. Endodermal ABA signaling promotes lateral root quiescence during salt stress in *Arabidopsis* seedlings. *Plant Cell* 25:324–41
37. Duan L, Sebastian J, Dinneny JR. 2015. Salt-stress regulation of root system growth and architecture in *Arabidopsis* seedlings. *Methods Mol. Biol.* 1242:105–22
38. Dunlap JR, Binzel ML. 1996. NaCI reduces indole-3-acetic acid levels in the roots of tomato plants independent of stress-induced abscisic acid. *Plant Physiol*. 112:379–84
39. Durand M, Porcheron B, Hennion N, Maurousset L, Lemoine R, Pourtau N. 2016. Water deficit enhances C export to the roots in *Arabidopsis thaliana* plants with contribution of sucrose transporters in both shoot and roots. *Plant Physiol*. 170:1460–79
40. Endler A, Kesten C, Froehlich A, Zhang Y, Ivakov A, et al. 2015. A mechanism for sustained cellulose synthesis during salt stress. *Cell* 162:1353–64
41. Endo A, Sawada Y, Takahashi H, Okamoto M, Ikegami K, et al. 2008. Drought induction of Arabidopsis 9-*cis*-epoxycarotenoid dioxygenase occurs in vascular parenchyma cells. *Plant Physiol*. 147:1984–93
42. Engelsdorf T, Gigli-Bisceglia N, Veerabagu M, McKenna JF, Vaahtera L, et al. 2018. The plant cell wall integrity maintenance and immune signaling systems cooperate to control stress responses in *Arabidopsis thaliana*. *Sci. Signal*. 11:eaao3070
43. Evans MJ, Choi WG, Gilroy S, Morris RJ. 2016. A ROS-assisted calcium wave dependent on the AtRBOHD NADPH oxidase and TPC1 cation channel propagates the systemic response to salt stress. *Plant Physiol*. 171:1771–84
44. Evelin H, Devi TS, Gupta S, Kapoor R. 2019. Mitigation of salinity stress in plants by arbuscular mycorrhizal symbiosis: current understanding and new challenges. *Front. Plant Sci.* 10:470
45. Feng W, Kita D, Peaucelle A, Cartwright HN, Doan V, et al. 2018. The FERONIA receptor kinase maintains cell-wall integrity during salt stress through Ca^{2+} signaling. *Curr. Biol.* 28:666–75
46. Feng W, Lindner H, Robbins NE, Dinneny JR 2nd. 2016. Growing out of stress: the role of cell- and organ-scale growth control in plant water-stress responses. *Plant Cell* 28:1769–82
47. Flowers TJ, Munns R, Colmer TD. 2015. Sodium chloride toxicity and the cellular basis of salt tolerance in halophytes. *Ann. Bot.* 115:419–31
48. Fricke W, Akhiyarova G, Wei W, Alexandersson E, Miller A, et al. 2006. The short-term growth response to salt of the developing barley leaf. *J. Exp. Bot.* 57:1079–95
49. Fujita S, Pytela J, Hotta T, Kato T, Hamada T, et al. 2013. An atypical tubulin kinase mediates stress-induced microtubule depolymerization in *Arabidopsis*. *Curr. Biol*. 23:1969–78
50. Fujita Y, Fujita M, Shinozaki K, Yamaguchi-Shinozaki K. 2011. ABA-mediated transcriptional regulation in response to osmotic stress in plants. *J. Plant Res.* 124:509–25
51. Galvan-Ampudia CS, Julkowska MM, Darwish E, Gandullo J, Korver RA, et al. 2013. Halotropism is a response of plant roots to avoid a saline environment. *Curr. Biol.* 23:2044–50
52. Gaxiola RA, Palmgren MG, Schumacher K. 2007. Plant proton pumps. *FEBS Lett*. 581:2204–14
53. Geng Y, Wu R, Wee CW, Xie F, Wei X, et al. 2013. A spatio-temporal understanding of growth regulation during the salt stress response in *Arabidopsis*. *Plant Cell* 25:2132–54
54. Gong Q, Li P, Ma S, Indu Rupassara S, Bohnert HJ. 2005. Salinity stress adaptation competence in the extremophile *Thellungiella halophila* in comparison with its relative *Arabidopsis thaliana*. *Plant J*. 44:826–39
55. Wang X, Chang L, Wang B, Wang D, Li P, et al. 2013. Comparative proteomics of *Thellungiella halophila* leaves from plants subjected to salinity reveals the importance of chloroplastic starch and soluble sugars in halophyte salt tolerance. *Mol. Cell. Proteom.* 12:2174–95
56. Graf A, Smith AM. 2011. Starch and the clock: the dark side of plant productivity. *Trends Plant Sci*. 16:169–75
57. Halfter U, Ishitani M, Zhu JK. 2000. The *Arabidopsis* SOS2 protein kinase physically interacts with and is activated by the calcium-binding protein SOS3. *PNAS* 97:3735–40
58. Hall D, Evans AR, Newbury HJ, Pritchard J. 2006. Functional analysis of CHX21: a putative sodium transporter in *Arabidopsis*. *J. Exp. Bot*. 57:1201–10

45. Elucidates the role of FERONIA in late-response calcium waves and cell swelling during salt stress, possibly by monitoring the salt-induced changes in pectins.

51. Describes how the main root avoids high salt concentrations: a response called halotropism, which is facilitated by PIN2 internalization and asymmetric auxin distribution.

53. Shows how root growth responses to salt consist of different phases that are mediated by hormone-induced transcriptional changes.

59. Hayes S, Pantazopoulou CK, van Gelderen K, Reinen E, Tween AL, et al. 2019. Soil salinity limits plant shade avoidance. *Curr. Biol.* 29:1669–76
60. He F, Xu C, Fu X, Shen Y, Guo L, et al. 2018. The microRNA390/TRANS-ACTING SHORT INTERFERING RNA3 module mediates lateral root growth under salt stress via the auxin pathway. *Plant Physiol.* 177:775–91
61. Heydarian Z, Yu M, Gruber M, Coutu C, Robinson SJ, Hegedus DD. 2018. Changes in gene expression in *Camelina sativa* roots and vegetative tissues in response to salinity stress. *Sci. Rep.* 8:9804
62. Hill CB, Jha D, Bacic A, Tester M, Roessner U. 2013. Characterization of ion contents and metabolic responses to salt stress of different *Arabidopsis AtHKT1;1* genotypes and their parental strains. *Mol. Plant* 6:350–68
63. Hoang XLT, Nhi DNH, Thu NBA, Thao NP, Tran LSP. 2017. Transcription factors and their roles in signal transduction in plants under abiotic stresses. *Curr. Genom.* 18:483–97
64. Holbrook NM, Shashidhar VR, James RA, Munns R. 2002. Stomatal control in tomato with ABA-deficient roots: response of grafted plants to soil drying. *J. Exp. Bot.* 53:1503–14
65. Iglesias MJ, Terrile MC, Windels D, Lombardo MC, Bartoli CG, et al. 2014. MiR393 regulation of auxin signaling and redox-related components during acclimation to salinity in *Arabidopsis*. *PLOS ONE* 9:e107678
66. Jarvis DE, Ryu CH, Beilstein MA, Schumaker KS. 2014. Distinct roles for SOS1 in the convergent evolution of salt tolerance in *Eutrema salsugineum* and *Schrenkiella parvula*. *Mol. Biol. Evol.* 31:2094–107
67. Javot H, Lauvergeat V, Santoni V, Martin-Laurent F, Guclu J, et al. 2003. Role of a single aquaporin isoform in root water uptake. *Plant Cell* 15:509–22
68. Ji H, Pardo JM, Batelli G, Van Oosten MJ, Bressan RA, Li X. 2013. The Salt Overly Sensitive (SOS) pathway: established and emerging roles. *Mol. Plant* 6:275–86
69. Jiang C, Belfield EJ, Cao Y, Smith JA, Harberd NP. 2013. An *Arabidopsis* soil-salinity-tolerance mutation confers ethylene-mediated enhancement of sodium/potassium homeostasis. *Plant Cell* 25:3535–52
70. Jiang X, Leidi EO, Pardo JM. 2014. How do vacuolar NHX exchangers function in plant salt tolerance? *Plant Signal. Behav.* 5:792–95

71. Identification of GIPCs produced through MOCA1 in sensing of monovalent cations, including Na^+.

71. Jiang Z, Zhou X, Tao M, Yuan F, Liu L, et al. 2019. Plant cell-surface GIPC sphingolipids sense salt to trigger Ca^{2+} influx. *Nature* 572:341–46

72. Jones AM, Danielson JA, Manojkumar SN, Lanquar V, Grossmann G, Frommer WB. 2014. Abscisic acid dynamics in roots detected with genetically encoded FRET sensors. *eLife* 3:e01741
73. Julkowska MM, Hoefsloot HC, Mol S, Feron R, de Boer GJ, et al. 2014. Capturing Arabidopsis root architecture dynamics with ROOT-FIT reveals diversity in responses to salinity. *Plant Physiol.* 166:1387–402
74. Julkowska MM, Klei K, Fokkens L, Haring MA, Schranz ME, Testerink C. 2016. Natural variation in rosette size under salt stress conditions corresponds to developmental differences between *Arabidopsis* accessions and allelic variation in the LRR-KISS gene. *J. Exp. Bot.* 67:2127–38

75. Shows the natural variation in root system architecture remodeling in response to salt stress in *Arabidopsis* and characterization of the underlying genetic loci.

75. Julkowska MM, Koevoets IT, Mol S, Hoefsloot H, Feron R, et al. 2017. Genetic components of root architecture remodeling in response to salt stress. *Plant Cell* 29:3198–213

76. Julkowska MM, McLoughlin F, Galvan-Ampudia CS, Rankenberg JM, Kawa D, et al. 2015. Identification and functional characterization of the *Arabidopsis* Snf1-related protein kinase SnRK2.4 phosphatidic acid–binding domain. *Plant Cell Environ.* 38:614–24
77. Kang J, Hwang JU, Lee M, Kim YY, Assmann SM, et al. 2010. PDR-type ABC transporter mediates cellular uptake of the phytohormone abscisic acid. *PNAS* 107:2355–60
78. Katschnig D, Bliek T, Rozema J, Schat H. 2015. Constitutive high-level SOS1 expression and absence of HKT1;1 expression in the salt-accumulating halophyte *Salicornia dolichostachya*. *Plant Sci.* 234:144–54
79. Kawa D, Julkowska MM, Sommerfeld HM, ter Horst A, Haring MA, Testerink C. 2016. Phosphate-dependent root system architecture responses to salt stress. *Plant Physiol.* 172:690–706
80. Kawa D, Meyer AJ, Dekker HL, Abd-El-Haliem AM, Gevaert K, et al. 2020. SnRK2 protein kinases and mRNA decapping machinery control root development and response to salt. *Plant Physiol.* 182:361–77
81. Kawa D, Testerink C. 2017. Regulation of mRNA decay in plant responses to salt and osmotic stress. *Cell Mol. Life Sci.* 74:1165–76

82. Kazachkova Y, Eshel G, Pantha P, Cheeseman JM, Dassanayake M, Barak S. 2018. Halophytism: What have we learnt from *Arabidopsis thaliana* relative model systems? *Plant Physiol.* 178:972–88
83. Kesten C, Wallmann A, Schneider R, McFarlane HE, Diehl A, et al. 2019. The companion of cellulose synthase 1 confers salt tolerance through a tau-like mechanism in plants. *Nat. Commun.* 10:857
84. Kiani-Pouya A, Roessner U, Jayasinghe NS, Lutz A, Rupasinghe T, et al. 2017. Epidermal bladder cells confer salinity stress tolerance in the halophyte quinoa and Atriplex species. *Plant Cell Environ.* 40:1900–15
85. Kiegle E, Moore CA, Haseloff J, Tester M, Knight MR. 2000. Cell-type-specific calcium responses to drought, salt and cold in the *Arabidopsis* root. *Plant J.* 23:267–78
86. Kim B-G, Waadt R, Cheong YH, Pandey GK, Dominguez-Solis JR, et al. 2007. The calcium sensor CBL10 mediates salt tolerance by regulating ion homeostasis in Arabidopsis. *Plant J.* 52:473–84
87. Kim JS, Mizoi J, Kidokoro S, Maruyama K, Nakajima J, et al. 2012. *Arabidopsis* GROWTH-REGULATING FACTOR7 functions as a transcriptional repressor of abscisic acid– and osmotic stress–responsive genes, including *DREB2A. Plant Cell* 24:3393–405
88. Kim JS, Mizoi J, Yoshida T, Fujita Y, Nakajima J, et al. 2011. An ABRE promoter sequence is involved in osmotic stress-responsive expression of the *DREB2A* gene, which encodes a transcription factor regulating drought-inducible genes in *Arabidopsis. Plant Cell Physiol.* 52:2136–46
89. Kim TH, Bohmer M, Hu HH, Nishimura N, Schroeder JI. 2010. Guard cell signal transduction network: advances in understanding abscisic acid, CO_2, and Ca^{2+} signaling. *Annu. Rev. Plant Biol.* 61:561–91
90. Kim WY, Ali Z, Park HJ, Park SJ, Cha JY, et al. 2013. Release of SOS2 kinase from sequestration with GIGANTEA determines salt tolerance in *Arabidopsis. Nat. Commun.* 4:1352
91. Koevoets IT, Venema JH, Elzenga JTM, Testerink C. 2016. Roots withstanding their environment: exploiting root system architecture responses to abiotic stress to improve crop tolerance. *Front. Plant Sci.* 7:1335
92. Korver RA, Koevoets IT, Testerink C. 2018. Out of shape during stress: a key role for auxin. *Trends Plant Sci.* 23:783–93
93. Korver RA, van den Berg T, Meyer AJ, Galvan-Ampudia CS, ten Tusscher KHWJ, Testerink C. 2020. Halotropism requires phospholipase Dζ 1-mediated modulation of cellular polarity of auxin transport carriers. *Plant Cell Environ.* 43:143–58
94. Kronzucker HJ, Britto DT. 2011. Sodium transport in plants: a critical review. *New Phytol.* 189:54–81
95. Kulik A, Wawer I, Krzywinska E, Bucholc M, Dobrowolska G. 2011. SnRK2 protein kinases—key regulators of plant response to abiotic stresses. *OMICS* 15:859–72
96. Kunz H-H, Gierth M, Herdean A, Satoh-Cruz M, Kramer DM, et al. 2014. Plastidial transporters KEA1, -2, and -3 are essential for chloroplast osmoregulation, integrity, and pH regulation in *Arabidopsis. PNAS* 111:7480–85
97. Kuromori T, Miyaji T, Yabuuchi H, Shimizu H, Sugimoto E, et al. 2010. ABC transporter AtABCG25 is involved in abscisic acid transport and responses. *PNAS* 107:2361–66
97a. Lamers J, van der Meer T, Testerink C. 2020. How plants sense and respond to stressful environments. *Plant Physiol.* 182:In press. **https://doi.org/10.1104/pp.19.01464**
98. Latz A, Mehlmer N, Zapf S, Mueller TD, Wurzinger B, et al. 2013. Salt stress triggers phosphorylation of the *Arabidopsis* vacuolar K^+ channel TPK1 by calcium-dependent protein kinases (CDPKs). *Mol. Plant* 6:1274–89
99. Lavenus J, Goh T, Roberts I, Guyomarc'h S, Lucas M, et al. 2013. Lateral root development in *Arabidopsis*: fifty shades of auxin. *Trends Plant Sci.* 18:455–63
100. Leyser O. 2018. Auxin signaling. *Plant Physiol.* 176:465–79
101. Li C-H, Wang G, Zhao J-L, Zhang L-Q, Ai L-F, et al. 2014. The receptor-like kinase SIT1 mediates salt sensitivity by activating MAPK3/6 and regulating ethylene homeostasis in Rice. *Plant Cell* 26:2538–53
102. Li G, Santoni V, Maurel C. 2014. Plant aquaporins: roles in plant physiology. *Biochim. Biophys. Acta Gen. Subj.* 1840:1574–82
103. Liu J, Ishitani M, Halfter U, Kim CS, Zhu JK. 2000. The *Arabidopsis thaliana SOS2* gene encodes a protein kinase that is required for salt tolerance. *PNAS* 97:3730–34
104. Liu J, Zhu JK. 1998. A calcium sensor homolog required for plant salt tolerance. *Science* 280:1943–45

105. Liu W, Li RJ, Han TT, Cai W, Fu ZW, Lu YT. 2015. Salt stress reduces root meristem size by nitric oxide-mediated modulation of auxin accumulation and signaling in Arabidopsis. *Plant Physiol.* 168:343–56
106. Ma L, Ye J, Yang Y, Lin H, Yue L, et al. 2019. The SOS2-SCaBP8 complex generates and fine-tunes an AtANN4-dependent calcium signature under salt stress. *Dev. Cell* 48:697–709
107. Ma L, Zhang H, Sun L, Jiao Y, Zhang G, et al. 2012. NADPH oxidase AtrbohD and AtrbohF function in ROS-dependent regulation of Na^+/K^+ homeostasis in *Arabidopsis* under salt stress. *J. Exp. Bot.* 63:305–17
108. Maathuis FJM. 2006. The role of monovalent cation transporters in plant responses to salinity. *J. Exp. Bot.* 57:1137–47
109. Maathuis FJM. 2011. Vacuolar two-pore K^+ channels act as vacuolar osmosensors. *New Phytol.* 191:84–91
110. Maathuis FJM, Ahmad I, Patishtan J. 2014. Regulation of Na^+ fluxes in plants. *Front. Plant Sci.* 5:467
111. Manishankar P, Wang N, Köster P, Alatar AA, Kudla J. 2018. Calcium signaling during salt stress and in the regulation of ion homeostasis. *J. Exp. Bot.* 69:4215–26
112. Maser P, Hosoo Y, Goshima S, Horie T, Eckelman B, et al. 2002. Glycine residues in potassium channel-like selectivity filters determine potassium selectivity in four-loop-per-subunit HKT transporters from plants. *PNAS* 99:6428–33
113. Maser P, Thomine S, Schroeder JI, Ward JM, Sze H, et al. 2001. Phylogenetic relationships within cation transporter families of Arabidopsis. *Plant Physiol.* 126:1646–67
114. Mason MG, Jha D, Salt DE, Tester M, Hill K, et al. 2010. Type-B response regulators ARR1 and ARR12 regulate expression of AtHKT1;1 and accumulation of sodium in *Arabidopsis* shoots. *Plant J.* 64:753–63
115. McLoughlin F, Galvan-Ampudia CS, Julkowska MM, Caarls L, van der Does D, et al. 2012. The Snf1-related protein kinases SnRK2.4 and SnRK2.10 are involved in maintenance of root system architecture during salt stress. *Plant J.* 72:436–49
116. McLoughlin F, Testerink C. 2013. Phosphatidic acid, a versatile water-stress signal in roots. *Front. Plant Sci.* 4:525
117. Miller G, Suzuki N, Ciftci-Yilmaz S, Mittler R. 2010. Reactive oxygen species homeostasis and signalling during drought and salinity stresses. *Plant Cell Environ.* 33:453–67
118. Moller IS, Gilliham M, Jha D, Mayo GM, Roy SJ, et al. 2009. Shoot Na^+ exclusion and increased salinity tolerance engineered by cell type–specific alteration of Na^+ transport in *Arabidopsis*. *Plant Cell* 21:2163–78
119. Morris EC, Griffiths M, Golebiowska A, Mairhofer S, Burr-Hersey J, et al. 2017. Shaping 3D root system architecture. *Curr. Biol.* 27:R919–30
120. Morris ER, Powell DA, Gidley MJ, Rees A, Rees D. 1982. Conformations. I. Polymorphism and interactions of pectins between gel and solid states of calcium polygalacturonate. *J. Mol. Biol.* 155:507–16
121. Munns R, Gilliham M. 2015. Salinity tolerance of crops—what is the cost? *New Phytol.* 208:668–73
122. Munns R, James RA, Gilliham M, Flowers TJ, Colmer TD. 2016. Tissue tolerance: an essential but elusive trait for salt-tolerant crops. *Funct. Plant Biol.* 43:1103–13
123. Munns R, James RA, Xu B, Athman A, Conn SJ, et al. 2012. Wheat grain yield on saline soils is improved by an ancestral Na^+ transporter gene. *Nat. Biotechnol.* 30:360–64
124. Munns R, Tester M. 2008. Mechanisms of salinity tolerance. *Annu. Rev. Plant Biol.* 59:651–81
125. Nakaminami K, Okamoto M, Higuchi-Takeuchi M, Yoshizumi T, Yamaguchi Y, et al. 2018. AtPep3 is a hormone-like peptide that plays a role in the salinity stress tolerance of plants. *PNAS* 115:5810–15
126. Nakashima K, Shinwari ZK, Sakuma Y, Seki M, Miura S, et al. 2000. Organization and expression of two *Arabidopsis DREB2* genes encoding DRE-binding proteins involved in dehydration- and high-salinity-responsive gene expression. *Plant Mol. Biol.* 42:657–65
127. Narsai R, Howell KA, Millar AH, O'Toole N, Small I, Whelan J. 2007. Genome-wide analysis of mRNA decay rates and their determinants in *Arabidopsis thaliana*. *Plant Cell* 19:3418–36
128. Nieves-Cordones M, Alemán F, Martínez V, Rubio F. 2010. The *Arabidopsis thaliana* HAK5 K^+ transporter is required for plant growth and K^+ acquisition from low K^+ solutions under saline conditions. *Mol. Plant* 3:326–33

123. Shows that in saline soils the presence of the TmHKT1;5 locus in wheat cultivars significantly reduces leaf sodium content and increases grain yield.

129. Numan M, Bashir S, Khan Y, Mumtaz R, Shinwari ZK, et al. 2018. Plant growth promoting bacteria as an alternative strategy for salt tolerance in plants: a review. *Microbiol. Res.* 209:21–32
130. Ogasawara Y, Kaya H, Hiraoka G, Yumoto F, Kimura S, et al. 2008. Synergistic activation of the *Arabidopsis* NADPH oxidase AtrbohD by Ca^{2+} and phosphorylation. *J. Biol. Chem.* 283:8885–92
131. Oh DH, Dassanayake M, Haas JS, Kropornika A, Wright C, et al. 2010. Genome structures and halophyte-specific gene expression of the extremophile *Thellungiella parvula* in comparison with *Thellungiella salsuginea* (*Thellungiella halophila*) and *Arabidopsis*. *Plant Physiol*. 154:1040–52
132. Oh DH, Hong H, Lee SY, Yun DJ, Bohnert HJ, Dassanayake M. 2014. Genome structures and transcriptomes signify niche adaptation for the multiple-ion-tolerant extremophyte *Schrenkiella parvula*. *Plant Physiol*. 164:2123–38
133. Orosa-Puente B, Leftley N, von Wangenheim D, Banda J, Srivastava AK, et al. 2018. Root branching toward water involves posttranslational modification of transcription factor ARF7. *Science* 362:1407–10
134. Orsini F, D'Urzo MP, Inan G, Serra S, Oh DH, et al. 2010. A comparative study of salt tolerance parameters in 11 wild relatives of *Arabidopsis thaliana*. *J. Exp. Bot*. 61:3787–98
135. Osakabe Y, Arinaga N, Umezawa T, Katsura S, Nagamachi K, et al. 2013. Osmotic stress responses and plant growth controlled by potassium transporters in *Arabidopsis*. *Plant Cell* 25:609–24
136. Pfister A, Barberon M, Alassimone J, Kalmbach L, Lee Y, et al. 2014. A receptor-like kinase mutant with absent endodermal diffusion barrier displays selective nutrient homeostasis defects. *eLife* 3:e03115
137. Postaire O, Tournaire-Roux C, Grondin A, Boursiac Y, Morillon R, et al. 2010. A PIP1 aquaporin contributes to hydrostatic pressure–induced water transport in both the root and rosette of Arabidopsis. *Plant Physiol*. 152:1418–30
138. Prerostova S, Dobrev PI, Gaudinova A, Hosek P, Soudek P, et al. 2017. Hormonal dynamics during salt stress responses of salt-sensitive *Arabidopsis thaliana* and salt-tolerant *Thellungiella salsuginea*. *Plant Sci*. 264:188–98
139. Qin W, Pappan K, Wang X. 1997. Molecular heterogeneity of phospholipase D (PLD). *J. Biol. Chem.* 272:28267–73
140. Quan R, Lin H, Mendoza I, Zhang Y, Cao W, et al. 2007. SCABP8/CBL10, a putative calcium sensor, interacts with the protein kinase SOS2 to protect *Arabidopsis* shoots from salt stress. *Plant Cell* 19:1415–31
141. Quintero FJ, Ohta M, Shi H, Zhu J-K, Pardo JM. 2002. Reconstitution in yeast of the *Arabidopsis* SOS signaling pathway for Na^+ homeostasis. *PNAS* 99:9061–66
142. Reguera M, Bassil E, Tajima H, Wimmer M, Chanoca A, et al. 2015. pH regulation by NHX-type antiporters is required for receptor-mediated protein trafficking to the vacuole in Arabidopsis. *Plant Cell* 27:1200–17
143. Rellán-Alvárez R, Lobet G, Lindner H, Pradier PL, Sebastian J, et al. 2015. GLO-Roots: an imaging platform enabling multidimensional characterization of soil-grown root systems. *eLife* 4:e07597
144. Rengasamy P. 2006. World salinization with emphasis on Australia. *J. Exp. Bot.* 57:1017–23
145. Roy A, Sahoo D, Tripathy BC. 2013. Involvement of phytochrome A in suppression of photomorphogenesis in rice seedling grown in red light. *Plant Cell Environ*. 36:2120–34
146. Ruiz-Sola MA, Arbona V, Gómez-Cadenas A, Rodríguez-Concepción M, Rodríguez-Villalón A. 2014. A root specific induction of carotenoid biosynthesis contributes to ABA production upon salt stress in *Arabidopsis*. *PLOS ONE* 9:e90765
147. Ryu JY, Lee HJ, Seo PJ, Jung JH, Ahn JH, Park CM. 2014. The *Arabidopsis* floral repressor BFT delays flowering by competing with FT for FD binding under high salinity. *Mol. Plant* 7:377–87
148. Sanchez DH, Siahpoosh MR, Roessner U, Udvardi M, Kopka J. 2008. Plant metabolomics reveals conserved and divergent metabolic responses to salinity. *Physiol. Plant.* 132:209–19
149. Sato A, Sato Y, Fukao Y, Fujiwara M, Umezawa T, et al. 2009. Threonine at position 306 of the KAT1 potassium channel is essential for channel activity and is a target site for ABA-activated SnRK2/OST1/SnRK2.6 protein kinase. *Biochem. J.* 424:439–48
150. Schilling RK, Marschner P, Shavrukov Y, Berger B, Tester M, et al. 2014. Expression of the *Arabidopsis* vacuolar H^+-pyrophosphatase gene (*AVP1*) improves the shoot biomass of transgenic barley and increases grain yield in a saline field. *Plant Biotechnol. J.* 12:378–86

151. Shabala S, Cuin TA. 2008. Potassium transport and plant salt tolerance. *Physiol. Plant.* 133:651–69
152. Shani E, Salehin M, Zhang Y, Sanchez SE, Doherty C, et al. 2017. Plant stress tolerance requires auxin-sensitive Aux/IAA transcriptional repressors. *Curr. Biol.* 27:437–44
153. Shi H, Quintero FJ, Pardo JM, Zhu JK. 2002. The putative plasma membrane Na^+/H^+ antiporter SOS1 controls long-distance Na^+ transport in plants. *Plant Cell* 14:465–77
154. Shi H, Ishitani M, Kim C, Zhu JK. 2000. The *Arabidopsis thaliana* salt tolerance gene *SOS1* encodes a putative Na^+/H^+ antiporter. *PNAS* 97:6896–901
155. Shkolnik D, Finkler A, Pasmanik-Chor M, Fromm H. 2019. CALMODULIN-BINDING TRANSCRIPTION ACTIVATOR 6: a key regulator of Na^+ homeostasis during germination. *Plant Physiol.* 180:1101–18
156. Slama I, Abdelly C, Bouchereau A, Flowers T, Savouré A. 2015. Diversity, distribution and roles of osmoprotective compounds accumulated in halophytes under abiotic stress. *Ann. Bot.* 115:433–47
157. Soma F, Mogami J, Yoshida T, Abekura M, Takahashi F, et al. 2017. ABA-unresponsive SnRK2 protein kinases regulate mRNA decay under osmotic stress in plants. *Nat. Plants* 3:16204
158. Steudle E, Peterson CA. 1998. How does water get through roots? *J. Exp. Bot.* 49:775–88
159. Sun J, Dai S, Wang R, Chen S, Li N, et al. 2009. Calcium mediates root K^+/Na^+ homeostasis in poplar species differing in salt tolerance. *Tree Physiol.* 29:1175–86
160. Sunarpi Horie T, Motoda J, Kubo M, Yang H, et al. 2005. Enhanced salt tolerance mediated by AtHKT1 transporter–induced Na unloading from xylem vessels to xylem parenchyma cells. *Plant J.* 44:928–38
161. Sutka M, Li G, Boudet J, Boursiac Y, Doumas P, Maurel C. 2011. Natural variation of root hydraulics in Arabidopsis grown in normal and salt-stressed conditions. *Plant Physiol.* 155:1264–76
162. Suwa R, Fujimaki S, Suzui N, Kawachi N, Ishii S, et al. 2008. Use of positron-emitting tracer imaging system for measuring the effect of salinity on temporal and spatial distribution of ^{11}C tracer and coupling between source and sink organs. *Plant Sci.* 175:210–16
163. Testerink C, Dekker HL, Lim Z-Y, Johns MK, Holmes AB, et al. 2004. Isolation and identification of phosphatidic acid targets from plants. *Plant J.* 39:527–36
164. Testerink C, Munnik T. 2005. Phosphatidic acid: a multifunctional stress signaling lipid in plants. *Trends Plant Sci.* 10:368–75
165. Testerink C, Munnik T. 2011. Molecular, cellular, and physiological responses to phosphatidic acid formation in plants. *J. Exp. Bot.* 62:2349–61
166. Thalmann M, Pazmino D, Seung D, Horrer D, Nigro A, et al. 2016. Regulation of leaf starch degradation by abscisic acid is important for osmotic stress tolerance in plants. *Plant Cell* 28:1860–78
167. Thalmann M, Santelia D. 2017. Starch as a determinant of plant fitness under abiotic stress. *New Phytol.* 214:943–51
168. Vahisalu T, Kollist H, Wang YF, Nishimura N, Chan WY, et al. 2008. SLAC1 is required for plant guard cell S-type anion channel function in stomatal signalling. *Nature* 452:487–91
169. Valenzuela CE, Acevedo-Acevedo O, Miranda GS, Vergara-Barros P, Holuigue L, et al. 2016. Salt stress response triggers activation of the jasmonate signaling pathway leading to inhibition of cell elongation in *Arabidopsis* primary root. *J. Exp. Bot.* 67:4209–20
170. van den Berg T, Korver Testerink RA Tusscher KHWJ C. 2016. Modeling halotropism: a key role for root tip architecture and reflux loop remodeling in redistributing auxin. *Development* 143:3350–62
171. Van der Does D, Boutrot F, Engelsdorf T, Rhodes J, McKenna JF, et al. 2017. The *Arabidopsis* leucine-rich repeat receptor kinase MIK2/LRR-KISS connects cell wall integrity sensing, root growth and response to abiotic and biotic stresses. *PLOS Genet.* 13:e1006832
172. Waadt R, Hitomi K, Nishimura N, Hitomi C, Adams SR, et al. 2014. FRET-based reporters for the direct visualization of abscisic acid concentration changes and distribution in *Arabidopsis*. *eLife* 3:e01739
173. Wang S, Kurepa J, Hashimoto T, Smalle JA. 2011. Salt stress–induced disassembly of *Arabidopsis* cortical microtubule arrays involves 26S proteasome–dependent degradation of SPIRAL1. *Plant Cell* 23:3412–27
174. West G, Inze D, Beemster GT. 2004. Cell cycle modulation in the response of the primary root of Arabidopsis to salt stress. *Plant Physiol.* 135:1050–58

175. Wu R, Duan L, Pruneda-Paz JL, Oh DH, Pound M, et al. 2018. The *6xABRE* synthetic promoter enables the spatiotemporal analysis of ABA-mediated transcriptional regulation. *Plant Physiol*. 177:1650–65
176. Xie YJ, Xu S, Han B, Wu MZ, Yuan XX, et al. 2011. Evidence of Arabidopsis salt acclimation induced by up-regulation of HY1 and the regulatory role of RbohD-derived reactive oxygen species synthesis. *Plant J*. 66:280–92
177. Yan S, Che G, Ding L, Chen Z, Liu X, et al. 2016. Different cucumber *CsYUC* genes regulate response to abiotic stresses and flower development. *Sci. Rep.* 6:20760
178. Yang Z, Wang C, Xue Y, Liu X, Chen S, et al. 2019. Calcium-activated 14–3–3 proteins as a molecular switch in salt stress tolerance. *Nat. Commun.* 10:1199
179. Yoshida T, Mogami J, Yamaguchi-Shinozaki K. 2014. ABA-dependent and ABA-independent signaling in response to osmotic stress in plants. *Curr. Opin. Plant Biol.* 21:133–39
180. Yu L, Nie J, Cao C, Jin Y, Yan M, et al. 2010. Phosphatidic acid mediates salt stress response by regulation of MPK6 in *Arabidopsis thaliana*. *New Phytol*. 188:762–73
181. Yue RQ, Tie SG, Sun T, Zhang L, Yang YJ, et al. 2015. Genome-wide identification and expression profiling analysis of *ZmPIN*, *ZmPILS*, *ZmLAX* and *ZmABCB* auxin transporter gene families in maize (*Zea mays* L.) under various abiotic stresses. *PLOS ONE* 10:e0118751
182. Zepeda-Jazo I, Velarde-Buendia AM, Enríquez-Figueroa R, Bose J, Shabala S, et al. 2011. Polyamines interact with hydroxyl radicals in activating Ca^{2+} and K^+ transport across the root epidermal plasma membranes. *Plant Physiol*. 157:2167–80
183. Zhang Q, Lin F, Mao T, Nie J, Yan M, et al. 2012. Phosphatidic acid regulates microtubule organization by interacting with MAP65-1 in response to salt stress in *Arabidopsis*. *Plant Cell* 24:4555–76
184. Zhao Y, Xing L, Wang X, Hou YJ, Gao J, et al. 2014. The ABA receptor PYL8 promotes lateral root growth by enhancing MYB77-dependent transcription of auxin-responsive genes. *Sci. Signal.* 7:ra53
185. Zhao C, Zayed O, Yu Z, Jiang W, Zhu P, et al. 2018. Leucine-rich repeat extensin proteins regulate plant salt tolerance in *Arabidopsis*. *PNAS* 115:1312328
186. Zhao J, Cheng N-H, Motes CM, Blancaflor EB, Moore M, et al. 2008. AtCHX13 is a plasma membrane K^+ transporter. *Plant Physiol*. 148:796–807
187. Zhao J-L, Zhang L-Q, Liu N, Xu S-L, Yue Z-L et al. 2019. Mutual regulation of receptor-like kinase SIT1 and B'κ-PP2A shapes the early response of rice to salt stress. *Plant Cell* 31:2131–51
188. Zou C, Chen A, Xiao L, Muller HM, Ache P, et al. 2017. A high-quality genome assembly of quinoa provides insights into the molecular basis of salt bladder–based salinity tolerance and the exceptional nutritional value. *Cell Res*. 27:1327–40
189. Zwiewka M, Nodzynski T, Robert S, Vanneste S, Friml J. 2015. Osmotic stress modulates the balance between exocytosis and clathrin-mediated endocytosis in *Arabidopsis thaliana*. *Mol. Plant* 8:1175–87