Review

What molecular mechanism is adapted by plants during salt stress tolerance?

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Salt stress harmfully shocks agricultural yield throughout the world affecting production whether it is for subsistence or economic outcomes. The plant response to salinity consists of numerous processes that must function in coordination to alleviate both cellular hyper-osmolarity and ion disequilibrium. Salt tolerance and yield stability are complex genetic traits that are difficult to establish in crops since salt stress may occur as a catastrophic episode, be imposed continuously or intermittently and become gradually more severe at any stage during development. Molecular biology research has provided new insight into the plant response to salinity and identified genetic determinants that effect salt tolerance. Recent confirmation that many salt tolerance determinants are ubiquitous in plants has led to the use of genetic models, like *Arabidopsis thaliana*, to further dissect the plant salt stress response. Since many of the most fundamental salt tolerance determinants are those that mediate cellular ion homeostasis, this review will focus primarily on the functional essentiality of ion homeostasis mechanisms in plant salt tolerance. The transport systems that facilitate cellular capacity to utilize Na^+ for osmotic adjustment and growth and the role of the Salt-Overly-Sensitive (SOS) signal transduction pathway in the regulation of ion homeostasis and salt tolerance will be particularly emphasized. The objective of the review is to know "What molecular mechanism is adopted by plants during salt stress tolerance?" A conclusion will be presented that integrates cellular based stress signaling and ion homeostasis mechanisms into a functional paradigm for whole plants and defines biotechnology strategies for enhancing salt tolerance of crops.

Key words: Hyper-osmolarity, ion disequilibrium, *Arabidopsis thaliana,* homeostasis, catastrophic episode.

INTRODUCTION

Many species of higher plants, including most crops, are subjected to growth inhibition under high-NaCl conditions. The salt-induced inhibition of plant growth, so-called salt stress, is caused not only by osmotic effects on water uptake but also by variable effects on plant cell metabolism under salt stress. While the first component can bring about water deficit, the excess of a specific ion can cause toxicity and or induce nutritional disorders (Greenway and Munns, 1980). Natural boundaries imposed by soil salinity also limit the caloric and the nutritional potential of agricultural production. These constraints are most acute in areas of the world where food distribution is problematic because of insufficient infra-

structure or political instability (Yokoi et al., 2002). Water and soil management practices have facilitated agricultural production on soils marginalized by salinity but additional gain by these approaches seems problematic. On the horizon are crop improvement strategies that are based on the use of molecular marker techniques and biotechnology and can be used in conjunction with traditional breeding efforts (Ribaut and Hoisington, 1998).

DNA markers should enhance the recovery rate of the isogenic recurrent genome after hybridization and facilitate the introgression of quantitative trait loci necessary to increase stress tolerance. Molecular marker techniques were used successfully to transfer alleles of interest from wild relatives into commercial cultivars (Tanksley and McCouch, 1997). The basic resources for biotechnology are genetic determinants of salt tolerance and yield stability. Implementation of biotechnology

strategies to achieve this goal requires that substantial research effort be focused to on identify salt tolerance effectors and the regulatory components that control these during the stress episode (Hasegawa et al., 2000b).

Further knowledge obtained about these stress tolerance determinants will be additional resource information for the dissection of the plant response to salinity, which will reveal how plants sense salt stress, transduce signals to mediate a defensive response and define the signal pathway outputs or effectors that accomplish the processes required for stress survival and alleviation and steady-state growth in the saline environment. Molecular genetic and plant transformation advances have made it feasible to assess biotechnological strategies based on activated signal cascades, engineered biosynthetic pathways, targeted gene or protein expression or alteration of the natural stress responsiveness of genes for development of salt tolerant crops (Hasegawa et al., 2000b; Zhu, 2007). The molecular identities of key ion transport systems that are fundamental to plant salt tolerance are now known (Hasegawa et al., 2000b). More recently, the SOS salt stress signaling pathway was determined to have a pivotal regulatory function in salt tolerance, fundamental of which is the control of ion homeostasis (Hasegawa et al., 2000b; Sanders, 2000; Zhu, 2000). This review will summarize research on plant ion homeostasis in saline environments and present a model that integrates current understanding of salt stress sensing, which leads to the activation of the SOS pathway and the regulation of ion transport systems that facilitate ion homeostasis.

GENETIC DIVERSITY IN PLANTS FOR SALT TOLERANCE

The extensive genetic diversity for salt tolerance that exists in plant taxa is distributed over numerous genera (Flowers et al., 2007; Greenway and Munns, 1980). Most crops are salt sensitive or hypersensitive plants (glycophytes) in contrast to halophytes, which are native flora of saline environments. Some halophytes have the capacity to accommodate extreme salinity because of very special anatomical and morphological adaptations or avoidance mechanisms (Flowers et al., 2007). However, these are rather unique characteristics for which the genes are not likely to be introgressed easily into crop plants. Research of recent decades has established that most halophytes and glycophytes tolerate salinity by rather similar strategies often using analogous tactical processes (Hasegawa et al., 2000b). The cytotoxic ions in saline environments, typically Na^+ and Cl^-, are compartmentalized into the vacuole and used as osmotic solutes (Blumwald et al., 2000; Niu et al., 2005). It follows then that many of the molecular entities that mediate ion homeostasis and salt stress signaling are similar in all plants (Hasegawa et al., 2000b). In the fact, the paradigm for ion homeostasis that

facilitates plant salt tolerance resembles that described for yeast (Bressan et al., 1998; Serrano et al., 1999). The fact that cellular ion homeostasis is controlled and effected by common molecular entities made it feasible to use model genetic organismal systems for the dissection of the plant salt stress response (Bressan et al., 1998; Serrano et al., 1999; Hasegawa et al., 2000a; Sanders, 2000; Zhu, 2000, 2001). Research on the plant genetic model Arabidopsis has increased greatly our understanding of how cellular salt tolerance mechanisms are integrated and coordinated in an organismal context, and are linked to essential phenological adaptations. Since Arabidopsis is a glycophyte, a salt tolerant genetic model will be required to delineate if salt tolerance is affected most by form or function of genes or more by differences in the expression of common genes due either to transcriptional or post-transcriptional control (Zhu, 2007).

MOLECULAR CELLULAR MECHANISMS FOR SALT TOLERANCE

High salinity causes hyperosmotic stress and ion disequilibrium that produce secondary effects or pathologies (Hasegawa et al., 2000b; Zhu, 2001). Fundamentally, plants cope by either avoiding or tolerating salt stress. That is, plants are either dormant during the salt episode or there must be cellular adjustment to tolerate the saline environment. Tolerance mechanisms can be categorized as those that function to minimize osmotic stress or ion disequilibrium or alleviate the consequent secondary effects caused by these stresses. The chemical potential of the saline solution initially establishes a water potential imbalance between the apoplast and symplast that leads to turgor decrease, which if severe enough can cause growth reduction (Bohnert et al., 2005). Growth cessation occurs when turgor is reduced below the yield threshold of the cell wall. Cellular dehydration begins when the water potential difference is greater than can be compensated for by turgor loss (Taiz and Zeiger, 1998). The cellular response to turgor reduction is osmotic adjustment. The cytosolic and organellar machinery of glycophytes and halophytes is equivalently Na^+ and Cl- sensitive; so osmotic adjustment is achieved in these compartments by accumulation of compatible osmolytes and osmoprotectants (Bohnert et al., 2005; Bohnert and Jensen, 1996). However, Na^+ and Cl^- are energetically efficient osmolytes for osmotic adjustment and are compartmentalized into the vacuole to minimize cytotoxicity (Blumwald et al., 2000; Niu et al., 2005). Since plant cell growth occurs primarily because of directional expansion mediated by an increase in vacuolar volume, compartmentalization of Na^+ and Cl^- facilitates osmotic adjustment that is essential for cellular development. Movement of ions into the vacuole might occur directly from the apoplast into the vacuole through membrane vesiculation or a cytological process that juxtaposes the plasma mem-

brane to the tonoplast (Hasegawa et al., 2000b). Then compartmentalization could be achieved with minimal or no exposure of the cytosol to toxic ions. However, it is not clear presently the extent to which processes like these contribute to vacuolar ion compartmentalization. The bulk of Na^+ and Cl^- movement from the apoplast to the vacuole likely is mediated through ion transport systems located in the plasma membrane and tonoplast. Presumably, tight coordinate regulation of these ion transport systems is required in order to control net influx across the plasma membrane and vacuolar compartmentalization. The SOS signal pathway is a pivotal regulator of, at least some, key transport systems required for ion homeostasis (Hasegawa et al., 2000a; Sanders, 2000; Zhu, 2000).

OSMOLYTES AND OSMOPROTECTANTS

As indicated previously, salt tolerance requires that compatible solutes accumulate in the cytosol and organelles where these function in osmotic adjustment and osmoprotection (Rhodes and Hanson, 1993). Some compatible osmolytes are essential elemental ions, such as K^+, but the majority are organic solutes. Compatible solute accumulation as a response to osmotic stress is an ubiquitous process in organisms as diverse as bacteria to plants and animals. However, the solutes that accumulate vary with the organism and even between plant species. A major category of organic osmotic solutes consists of simple sugars (mainly fructose and glucose), sugar alcohols (glycerol and methylated inositols) and complex sugars (trehalose, raffinose and fructans) (Bohnert and Jensen, 1996). Others include quaternary amino acid derivatives (proline, glycine betaine, ß-alanine betaine, proline betaine), tertiary amines (1,4,5,6-tetrahydro-2-mehyl-4-carboxyl pyrimidine), and sulfonium compounds (choline osulfate, dimethyl sulfonium propironate) (Nuccio et al., 1999). Many organic osmolytes are presumed to be osmoprotectants, as their levels of accumulation are insufficient to facilitate osmotic adjustment. Glycine betaine preserves thylakoid and plasma membrane integrity after exposure to saline solutions or to freezing or high temperatures (Rhodes and Hanson, 1993).

Furthermore, many of the osmoprotectants enhance stress tolerance of plants when expressed as transgene products (Bohnert and Jensen, 1996; Zhu, 2001). An adaptive biochemical function of osmoprotectants is the scavenging of reactive oxygen species that are byproducts of hyperosmotic and ionic stresses and cause membrane dysfunction and cell death (Bohnert and Jensen, 1996). A common feature of compatible solutes is that these compounds can accumulate to high levels without disturbing intracellular biochemistry (Bohnert and Jensen, 1996). Compatible solutes have the capacity to persevere the activity of enzymes that are in saline solutions. These compounds have minimal affect on pH or charge balance of the cytosol or luminal compart-

ments of organelles. The synthesis of compatible osmolytes is often achieved by diversion of basic intermediary metabolites into unique biochemical reactions. Often, stress triggers this metabolic diversion. For example, higher plants synthesize glycine betaine from choline by two reactions that are catalyzed in sequence by choline mono-oxygenase (CMO) and betaine aldehyde dehydrogenase (BADH) (Rhodes and Hanson, 1993). Pinitol is synthesized from myo-inositol by the sequential catalysis of inositol-O-methyltransferase and ononitol epimerase (Bohnert and Jensen, 1996).

ION HOMEOSTASIS

Since NaCl is the principal soil salinity stress, a research focus has been the transport systems that are involved in utilization of Na^+ as an osmotic solute (Blumwald et al., 2000; Hasegawa et al., 2000b; Niu et al., 2005). Research of more than 30 years previously, established that intracellular Na^+ homeostasis and salt tolerance are modulated by Ca^{2+} and high $[Na^+]ext$ negatively affects K^+ acquisition (Rains and Epstein, 1967). Na^+ competes with K^+ for uptake through common transport systems and does this effectively since the $[Na^+]ext$ in saline environments is usually considerably greater than $[K^+]ext$. Ca^{2+} enhances K^+/Na^+ selective intracellular accumulation (Maathuis et al., 1996; Rains and Epstein, 1967). Research of the last decade has defined many of the molecular entities that mediate Na^+ and K^+ homeostasis and given insight into the function of Ca^{2+} in the regulation of these transport systems. Recently, the SOS stress-signaling pathway was identified to be a pivotal regulator of plant ion homeostasis and salt tolerance (Hasegawa et al., 2000b; Sanders, 2000). This signaling pathway functionally resembles the yeast calcineurin cascade that controls Na^+ influx and efflux across the plasma membrane (Bressan et al., 1998). Expression of an activated form of calcineurin in yeast or plants enhances salt tolerance further implicating the functional similarity between the calcineurin and the SOS pathways (Mendoza et al., 1996; Pardo et al., 1998). Little is known about the mechanistic entities that are responsible for Cl-transport or the regulation of Cl- homeostasis (Hedrich, 1994).

ION TRANSPORT SYSTEMS

H^+ pumps in the plasma membrane and tonoplast energize solute transport necessary to compartmentalize cytotoxic ions away from the cytoplasm and to facilitate the function of ions as signal determinants (Maeshima, 2000; Maeshima, 2001; Morsomme and Boutry, 2000; Ratajczak, 2000). The plasma membrane localized H^+ pump is a P-type ATPase and is primarily responsible for the high pH and membrane potential gradient across this

membrane (Morsomme and Boutry, 2000). A vacuolar type H^+ - ATPase and a vacuolar pyrophosphatase generate the pH and membrane potential across the tonoplast (Drozdowicz and Rea, 2001; Maeshima, 2001). The activity of these H^+ pumps is increased by salt treatment and induced gene expression may account for some of the up regulation (Hasegawa et al., 2000b; Maeshima, 2001). Recently, the plasma membrane H^+ ATPase was confirmed as a salt tolerance determinant based on analyses of phenotypes caused by the semi-dominant aha4-1 mutation (Vitart et al., 2001). The mutation to AHA4, which is expressed predominantly in the roots, causes a reduction in root and shoot growth (relative to wild type) of plants that are grown on medium supplemented with 75 mM NaCl. Decreased root length of salt treated aha4-1 plants is due to reduce cell length. In NaCl supplemented medium, leaves of aha4-1 plants accumulate substantially more Na^+ and less K^+ than those of wild type. It is postulated that AHA4 functions in the control of Na^+ flux across the endodermis (Vitart et al., 2001).

TRANSMEMBRANE SODIUM MOVEMENTS

Blumwald et al. (1987), for example, have claimed the existence of Na^+/H^+ antiport in tonoplast vesicles of sugar beet based upon the response to Na^+ of pH-dependent acridine orange fluorescence quenching. The Na^+ effect is increased by Na^+ pretreatment, sensitive to amiloride (an inhibitor of an analogous transporter in various animal systems) and to a number of promising amiloride analogs (Blumwald et al. 1987). Unfortunately, there have not so far been reports which have included direct measurement of Na^+ fluxes or confirmation of these results with different probes. Nevertheless, the results to date show promise for the difficult task of isolating and identifying a membrane ion transporter. At the plasmalemma, if Na^+ entry is not always down a substantial electrochemical gradient, rather little addition of Na^+ to the external. At the cellular level, the steady state must be maintained either by the very effective exclusion of Na^+ initially or by the extrusion or turnover of internal pools. No plant is a perfect excluder; even the most easily killed species have significant Na^+ levels in their roots. Rapid turnover rates, on the other hand, are probably common in both mesophytes and halophytes (Cheeseman, 1988; Lazof and Cheeseman, 2007). Influx appears to be passive down an electrochemical gradient and independent of either H^+ or K^+ movements. Both influx and efflux of Na^+ to roots are unresponsive to modifiers of plasmalemma H^+ pumping, energization level, or transport activity such as fusicoccin, N, N-dicyclohexylcarbodiimide (DCCD) and p-fluoromethoxycarbonyl cyanide phenylhydrazone (FCCP). Though the evidence is far from complete, it should also not be discounted that Na^+ movement involves mechanisms other than those mediated by transmembrane transporters; for example, it has recently been resuggested,

based on very high estimates of unidirectional Na^+ movements, that the fluxes may involve vesiculation and turnover of a sub-cytoplasmic compartment (Lazof and Cheesman, 2007). Finally, there are numerous complexities in the study of Na^+ uptake and organismal response, which cloud the interpretation of even apparently straightforward studies. These include the interactions of Na^+, Ca^{2+}, K^+, membrane surface properties, root cell development, and growth (Cramer et al., 2007). Beyond this, the nature of the transport systems involved in the distribution and compartmentation of Na^+ at the organismal level is largely unknown. Though the potential, integrated system complexity is great, including, at least, sequestration within specific cells and tissues of the root, stem base, and leaves and retransport from shoots or sequestered pools to the roots for excretion, it is possible to model (schematically) the acquisition and allocation of Na without additional basic cellular-level transporting systems.

Ca^{2+} SIGNALING AND SALT OVERLY SENSITIVE (SOS) SIGNAL TRANSDUCTION PATHWAY

Jian-Kang Zhu and co-workers identified three genetically linked Arabidopsis loci (SOS_1, SOS_2 and SOS_3), which are components of a stress-signaling -pathway that controls ion homeostasis and salt tolerance (Hasegawa et al.2000a; Sanders, 2000; Zhu, 2000, 2001). Genetic analysis of Na^+/Li^+ sensitivity established that sos1 is epistatic to sos2 and sos3 (Zhu, 2000). These sos mutants also exhibit a K^+ deficient phenotype in medium supplemented with μM $[K^+]$ ext and $[Ca^{2+}]$ ext. Na^+ and K^+ deficiency of sos2 and sos3 is suppressed with mM $[Ca^{2+}]$ ext (Zhu et al., 1998). SOS_1 exhibits hyperosmotic sensitivity unlike sos3 and sos2. Together, these results indicate that the SOS signaling pathway regulates Na^+ and K^+ homeostasis and is Ca^{2+} activated. SOS_3 encodes a Ca^{2+} binding protein with sequence similarity to the regulatory B subunit of calcineurin (protein phosphatase 2B) and neuronal Ca^{2+} sensors (Ishitani et al., 2000; Liu and Zhu, 1998). Interaction of SOS_3 with the SOS_2 kinase (Liu et al., 2000) and SOS_2 activation is Ca^{2+} dependent (Halfter et al., 2000). The in plant function of SOS_3 as a salt tolerance determinant is dependent on Ca^{2+} binding and Nmyristoylation (Ishitani et al., 2000).

The SOS_2 serine/threonine kinase (446 amino acids) has a 267 amino acid N-terminal catalytic domain that is similar in sequence to yeast SNF1 (sucrose nonfermenting) kinase and the mammalian AMPK (AMP-activated protein kinase) (Liu et al., 2000; Zhu, 2000). The kinase activity of SOS_2 is essential for its salt tolerance determinant function (Zhu, 2000). The SOS_2 C-terminal regulatory domain interacts with the kinase domain to cause auto inhibition. A 21 amino acid motif in the regulatory domain of SOS_2 is the site where SOS_3 interacts with the kinase and is the auto inhibitory domain of the kinase

(Guo et al., 2001). Binding of SOS_3 to this motif blocks auto inhibition of SOS_2 kinase activity. Deletion of the auto inhibitory domain results in constitutive SOS_2 activation, independent of SOS_3. Also, a Thr168 to Asp mutation in the activation loop of the kinase domain constitutively activates SOS_2 (Hasegawa et al., 2000b; Sanders, 2000; Zhu, 2001). Ca^{2+} binds to SOS_3, which leads to interaction with SOS_2 and activation of the kinase. Among the SOS signal pathway outputs are transport systems that facilitate ion homeostasis. The plasma membrane sited Na^+/H^+ antiporter SOS_1 is controlled by the SOS pathway at the transcriptional and post-transcriptional level (Guo et al., 2001; Zhu, 2001). Recently, functional disruption of AtHKT1 was shown to suppress the salt sensitive phenotype of SOS_{3-1}, indicating that the SOS pathway negatively controls this Na^+ influx system (Rus et al., 2001). Also, the SOS pathway negatively controls expression of AtNHX family members that are implicated as determinants in the salt stress response (Yokoi et al., 2001).

$[Ca^{2+}]$ ext enhances salt tolerance and salinity stress elicits a transient $[Ca^{2+}]$ cytoplasmic increase, from either an internal or external source, that has been implicated in adaptation (Knight et al., 1997, Läuchli, 1990). Data from recent experiments with yeast has provided insight into Ca^{2+} activation of salt stress signaling that controls ion homeostasis and tolerance (Matsumoto et al., 2001). The hyperosmotic component of high salinity induces a short duration (1 min) rise in cytoplasmic $[Ca^{2+}]$ that is due substantially to influx across the plasma membrane through the Cch1p and Mid1p Ca^{2+} transport system. The transient increase in cytoplasmic $[Ca^{2+}]$ activates the PP2B phosphatase calcineurin (a key intermediate in salt stress signaling controlling ion homeostasis) leading to the transcription of ENA1, which encodes the P-type ATPase that is primarily responsible for Na^+ efflux across the plasma membrane (Nakamura et al., 1993; Mendoza et al., 1994; Matsumoto et al., 2001). The model proposes that the hyperosmotically-induced localized cytoplasmic $[Ca^{2+}]$ transient activates calmodulin that is tethered to Cch1p-Midp (Elhers and Augustine, 1999; Sanders et al., 1999; Matsumoto et al., 2001). Calmodulin in turn activates signaling through the calcineurin pathway, which mediates ion homeostasis and salt tolerance (Matsumoto et al., 2001). Components of the SOS pathway, either SOS3 or upstream elements, might be associated with an osmotically responsive channel through which Ca^{2+} influx could initiate signaling through the pathway. It is notable that a new elevated cytoplasmic $[Ca^{2+}]$ steady state is established in yeast cells, that are maintained in medium supplemented with NaCl, after the hyperosmotic induction of the short duration $[Ca^{2+}]$cyt transient (Matsumoto et al., 2001). It is likely that the newly established cytoplasmic $[Ca^{2+}]$ contributes to cellular capacity for growth in salinity. The vacuolar membrane H^+/Ca^{2+} antiporter Vcx1p and endomembrane localized Ca^{2+} ATPases are pivotal effectors that regulate

the amplitude and duration of the $[Ca^{2+}]$ cyt transient (Miseta et al., 1999). The cytoplasmic $[Ca^{2+}]$ steady state established in salt containing medium presumably also involves coordination of channel activation that facilitates influx from external and internal sources and energy dependent transport systems that compartmentalize the divalent cation. It is reasonable to assume that the salt induced cytoplasmic $[Ca^{2+}]$ transient detected in plant cells (Knight et al., 1996) and, perhaps, a new cytoplasmic $[Ca^{2+}]$ steady-state are controlled by the ECA and ACA Ca^{2+}-ATPases and CAX1 and 2 transporters which are orthologs of Vcx1p (Sze et al., 2000). Nevertheless, Ca^{2+} has at least two roles in salt tolerance, a pivotal signaling function in the salt stress response leading to adaptation and a direct inhibitory effect on a Na^+ entry system.

CARBON ACHIEVEMENT AND DISTRIBUTION

Seemann and Critchley (1985) considered the effects of salt stress on the gas exchange characteristics of bean (Plaseoliis vta/lgunis). Under conditions of severely reduced growth and leaf accumulation of Cl in particular. stomatal limitations were manifested by a decrease in intercellular CO and 6' 3C. Nonstomatal reductions reflected effects on both the photochemical processes (a decrease in quantum efficiency for CO, uptake) and ion apparent, *in vivo* (but not *in vitro*) ribulose bisphosphate carboxylic activity. Both the total leaf N and carboxylase concentrations were little changed.

Robinson et al. (1983) showed a similar result in their study of spinach leaves and chloroplasts. Comparing plants grown with 0 and 200 mM NaCl. Overall growth was reduced approximately 65%, as was stomatal conductance, which in this case became the more significant limiting factor in total carbon fixation. Leaf photosynthetic capacity, that is, the maximal, unlimited rate per unit area or unit Chl was altered only about 1(0)'. However variable fluorescence was unchanged. In isolated intact chloroplasts, CO, dependent O. evolution was reduced only 20 to 50% and electron transport was unchanged by salinity. It is now frequently noted, as it was in these studies, that the reduction of growth is greater than the decrease in realized or potential photosynthesis and the reduction of shoot growth is much greater than the reduction of root growth. Such single point comparisons should be interpreted with caution, however. The experimental convenience may have little to do with the biological information content because, first, the care based upon plants in that highly abnormal state. 'Control,' in which there are essentially no limitations placed on growth by resource availability and second, the adjustment of growth or metabolism in a variable environment with limited resources is the quintessence of organismal function in plants. In defining the more general systems for control and integration, therefore, more attention must be directed

toward the dynamics of adjustment as environmentally imposed limitations change.

Seemann and Critchley (1985) commented in closing that the overall reduction in long-term growth probably reflected the reduction in carbon allocation to new leaves and long-term potential photosynthesis. In source-sink studies, it has frequently been shown that an increase in carbon usage may increase the rate of fixation. The importance of photosynthetic responses in the organismal context should, therefore, also consider the fate of the fixed carbon. The range of responses was nicely demonstrated in a comparative study of salinity responses in three San Francisco Bay area halophytes by Pearcy and Ustin (1984). They demonstrated that at moderate salinity (for these plants) salinization was accompanied by an increase in photosynthetic capacity (per unit leaf area) with little change or a slight increase in net fixation under growth lighting and CO, conditions. Overall growth responses were considerably different, however, ranging from stimulation to severe reduction, reflecting the degree to which carbon was reallocated from shoot to root growth. In Salicornia, the small growth response was associated with a 33%c decrease in root-shoot ratio. In Scirplus, a drastic reduction in total growth (80%) was associated with a 90Cc increase in root-shoot ratio. It is broadly acceptable that total carbon usage can be partitioned to growth (production of cell walls and integral cellular machinery), maintenance (turnover and repair), transport (generally not separable from maintenance in practice) and storage.

CONCLUSIONS

Recent progress in the elucidation of salt stress signaling and effector output determinants that mediate ion homeostasis has uncovered some potential biotechnology tactics that may be used to obtain salt tolerant crop plants. The recent demonstration that a constitutively activated SOS_2 kinase can be achieved by deletion of the auto inhibitory domain or by site-specific modifications to the catalytic domain of the protein kinase offers an approach to regulate stress signaling that controls ion homeostasis. Furthermore, over expression of AtNHX1 enhances plant salt tolerance, presumably by increasing vacuolar Na^+ compartmentalization that minimizes the toxic accumulation of the ion in the cytosol and facilitates growth in the saline environment. Thus regulation of the numerous salt tolerance determinants can be coordinated for an effective plant response but many of the costs associated with salt tolerance in nature might be minimized because some essential evolutionary necessities can be compensated for by agricultural practices.

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