Signal transduction pathways under abiotic stresses in plants

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All abiotic stresses reduce plant growth and yield. The products of stress-inducible genes which could be directly protecting against these stresses include the enzymes responsible for the synthesis of various osmoprotectants like late embryogenesis abundant (LEA) proteins, antifreeze proteins, chaperones and detoxification enzymes. Another group of gene products involved in gene expression and signal transduction pathways includes transcription factors, protein kinases and enzymes involved in phosphoinositide metabolism. In the present article, the signal transduction pathways involving mitogen activated protein kinases (MAPK) under osmotic/oxidative stress have been discussed. Studies indicate that in addition to connections between MAPK modules used by osmotic stress and ROS signalling, there is also specificity in the individual pathways. The role of external stimuli and of IP3 as a second messenger in releasing Ca^{+2} from cellular stores has been reported. Interaction between phosphoinositide-specific phospholipase C activity and IP3 level has been indicated. Abiotic stresses and ABA biosynthesis suggested connection between cold, drought, salinity and ABA signal transduction pathways. Calcium-dependent signalling that leads to LEA type genes and salt overly sensitive signalling that result in ion homeostasis has been discussed. Crosstalk among various transduction pathways under abiotic stresses in regulation of metabolism has been reviewed.

PLANTS are subjected to various abiotic stresses because of unavoidable environmental conditions which adversely affect their growth and development. Drought, or more generally inadequate availability of water, and salt stress due to soil or quality of irrigated water, are the main abiotic stresses to which crops are exposed in India. Depending upon the extent of stress, the plants try to adapt to the changing environmental conditions. For example, under osmotic and ionic stresses, the plants must get adequate amount of water for their growth and development of reproductive structures. Therefore, under these conditions, the adaptive mechanisms should be directed to this objective. The closure of stomata limits water loss and the integrity of the photosynthetic and carbon fixation apparatus is maintained by the initiation of a series of physiological processes[1,2]. Although most of the biochemical factors necessary for stress tolerance acquisition are present in all species, the difference is how fast and how persistent this machinery is activated, and how the stress is perceived and how the signals are further transduced into a series of responses[3,4].

In addition to external abiotic signals, a variety of internal signals such as hormones and solutes modify plant cell growth and development. A cascade of complex events involving several interacting components required for initial recognition of signal and subsequent transduction of these signals to the physiological response is triggered. The cascade of events is called signal transduction, which normally acts through second messengers that can trigger the molecular events leading to physiological response, often by modification of gene expression[5].

Studies on molecular responses of plants to various types of stresses indicate that different types of stresses provide the cells with different information. The multiplicity of the information generated by different types of stresses makes the response of plants complex and hence also the stress signalling pathways[6–8]. Many genes that are induced or upregulated by osmotic stresses have been identified[9–11]. Gene expression profiling using cDNA microarrays or gene chips has come up as an important tool in identifying many more genes that are regulated by drought or salt stress[12–17].

Various signal pathways can operate independent of each other or they may positively or negatively modulate other pathways. Different signalling pathways may also share components and second messengers to achieve their objectives. This interdependence of various pathways on each other is called crosstalk among themselves. As a result, many signals could interact in a cooperative fashion with each other[18]. Plant cells are connected with neighbouring cells by means of plasmadesmata which could allow signalling molecules to pass directly from cell to cell[19].

Products of stress-inducible genes

The products of stress-inducible genes are classified into two groups[17]. (i) Those which directly protect against stresses, and these are the proteins that function by protecting cells from dehydration. They include the enzymes responsible for the synthesis of various osmoprotectants like late embryogenesis abundant (LEA) proteins, antifreeze proteins, chaperones and detoxification enzymes. (ii) The second group of gene products includes transcription factors, protein kinases

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and enzymes involved in phosphoinositide metabolism. This group of gene products regulates gene expression and signal transduction pathways. Stress-inducible genes have been used to improve the stress tolerance of plants by gene transfer[20–22]. The signal transduction pathways in plants under environmental stresses have been divided into three major types[23]: (i) osmotic/oxidative stress signalling that makes use of mitogen activated protein kinase (MAPK) modules; (ii) Ca^{+2}-dependent signalling that leads to activation of LEA-type genes such as dehydration responsive elements (DRE)/cold responsive sensitive transcription factors (CRT) class of genes, and (iii) Ca^{+2}-dependent salt overly sensitive (SOS) signalling that results in ion homeostasis.

The application of genetic analysis in plant stress tolerance is limited because of the scarcity of the availability of suitable phenotypes for mutant screening. Seed germination had been used as a marker to screen salt-tolerant mutants in *Arabidopsis*[24,25]. However, salt tolerance of the resistant mutants thus isolated was not extended to vegetative stages, implying that the mechanism of salt tolerance during germination is different from that of vegetative salt tolerance. However, in certain cases, treatments showing better seedling growth under abiotic stresses could sustain the effect till seed maturity, thus influencing yield in a positive manner[26–28]. After a large-scale screening for salt tolerant mutants, it was found that salt resistance at germination was not observed at seedling growth and these mutants were not defective in the expression of stress-induced genes examined[29]. These data suggested that the isolation of mutations in salt stress signal transduction pathways by salt tolerance germination was difficult. Two mutants for salt tolerance during growth at seedling stage of *Arabidopsis* were isolated. In one pst 1 mutant, it was shown that salt tolerance was due to enhanced scavenging of reactive oxygen species (ROS)[30]. The reduced growth of plants under abiotic stresses is also not a reliable criterion for screening a mutagenized population, as many other factors also affect plant growth.

No mutations in abiotic stress receptors, phosphoinositol module, calcium-dependent protein kinases (CDPKs) and MAPK modules in relation to abiotic stress signalling have been reported in plants. The transcriptional factors identified from mutations in the ABA signal transduction pathway seemed to be mainly functioning in seed development[30]. Therefore, because of the scarcity of abiotic stress-specific phenotypes for conventional genetic screening and lack of mutants with respect to signalling pathways, molecular genetic analysis using stress-responsive promotor-driven receptors was suggested as an alternative approach to genetically dissect different types of abiotic stress signalling pathways[31].

Osmotic/oxidative stress signalling by MAPK modules

On exposure to water deficit or salinity stresses, plants lower the osmotic potential of the cell cytosol and accumulate compatible osmolytes[32–36]. In glycophytes, the capacity for sodium compartmentalization and osmolyte biosynthesis is limited; however, an increased production of compatible osmolytes such as proline, glycine, betaine and polyols can reduce stress damage to plant cells. This is an adaptive strategy and transgenic plants with increased osmolyte production or decreased degradation showed improved salt and drought tolerance[37,38]. Salt tolerance was also increased in transgenic plants engineered to produce new osmolytes absent in the parental lines or in plants that overexpressed the genes whose products limit the production of these osmolytes[39,40]. The introduction of fructan synthesizing genes in tobacco plant enhanced the resistance to drought stress[41]. These osmolytes may protect proteins from misfolding and alleviate the toxic effects of ROS[40,42,43]. Upregulation of protein biosynthetic genes such as pyrroline-5-carboxylate synthase (P5CS) has been reported under salt or drought stresses[44,45]. Increased biosynthesis of proline in plants may involve regulation by feedback inhibition. Tobacco plants expressing a mutated P5CS protein that disabled negative regulation, accumulated more proline under both stress and unstressed conditions and showed increased salt tolerance[43].

The ionic and osmotic stresses create secondary stresses like oxidative stress due to generation of ROS, e.g. hydrogen peroxide, hydroxyl radicals and superoxide anions in plants[30,43]. The major function of compatible solutes is to alleviate oxidative stress damage. The transgenic plants overexpressing ROS scavenging enzymes such as superoxide dismutase, catalase and glutathione S-transferase (GST) showed increased tolerance to salt stress[39]. A genetic screen for salt-tolerant growth in *Arabidopsis* resulted in the isolation of several salt tolerant pst mutants. The enhanced salt tolerance in pst 1 mutants was associated with increased ROS detoxification as a result of activation of superoxide dismutase and ascorbate peroxidase[30]. A connection between osmotic stresses and oxidative stresses has been indicated in *Arabidopsis*, where it has been shown that several MAPK components were activated or their gene expression induced by salt and other stresses[31,46].

The stress-responsive genes can be classified into two classes, i.e. early and delayed response genes[47]. The former are induced quickly and transiently, while the latter are activated more slowly and their expression is sustained. The early response genes encode transcription factors that activate downstream delayed response genes[48], as shown in Figure 1. The signalling pathways for abiotic stresses have been reported to converge at multiple steps[30,49]. This was based on a comprehensive mutational analysis in which *Arabidopsis* single-gene mutation was found to affect responses to all or a combination of these signals[50].

The MAP kinase pathways are intracellular signal modules that mediate signal transduction from the cell surface to the nucleus. These kinases seem to be widely used as osmolarity signalling modules. The completion of *Arabidopsis thaliana* (*AT*) genome sequence has helped in the identi-

fication and isolation of gene families encoding MAPKs and their immediate upstream regulators, MAPK kinase (MAPKK) and MAPKK kinase (MAPKKK) on the basis of sequence conservation. The environmental signals are first perceived by specific receptors that upon activation initiate a cascade to transmit the signal intracellularly and in many cases activate nuclear transcription factors to induce the expression of specific sets of genes.

MAPKs are signalling modules that phosphorylate specific serine/threonine residues on the target protein substrate and regulate a variety of cellular activities[48]. The MAPK phosphorylation system serves as a link between upstream receptors and downstream targets, thereby regulating many important cellular functions. MAPKs are activated in response to drought and other environmental stresses[51]. MAPK genes encode polypeptides whose sequence and function are highly conserved among eukaryotes[48]. The MAPK cascade consists of three functionally interlinked protein kinases: MAPKKK, MAPKK, and MAPK[51]. In this phosphorylation module, a MAPKKK is phosphorylated directly downstream of the stimulus. The activated MAPKKK then phosphorylates and activates a particular MAPKK, which in turn phosphorylates and activates a MAPK. Activated MAPK is imported into the nucleus, where it phosphorylates and activates specific downstream signalling components, such as transcription factors to induce cellular responses[52] (Figure 2).

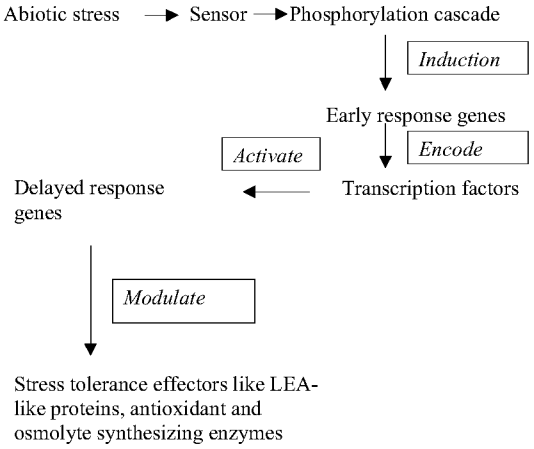

Figure 1. A simplified presentation of the effect of abiotic stresses at molecular level.

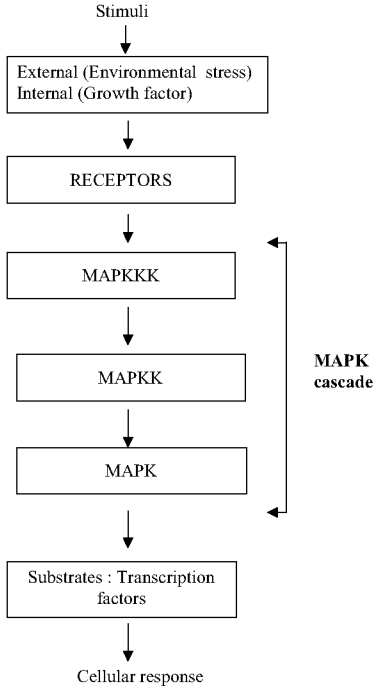

Figure 2. Model of MAPK cascade depicting how MAPK phosphorylation system serves as a link between upstream receptors and downstream signalling components such as transcription factors to induce cellular response.

Nine MAPK genes have been identified from rice. Each MAPK encodes a distinct protein kinase that plays a role in mediating drought tolerance. Using an *in vitro* system, OsMSRMK2 transcripts (mRNA) have been shown to accumulate at significant levels in 15 min after initiation of drought stress[53].

The two-component sensor receptor regulatory system involving histidine kinase for perception of various environmental stresses, has been found to exist in plants. When its extracellular sensor domain perceives a signal, the cytoplasmic histidine residue is autophosphorylated and the phosphoryl moiety is then passed to an aspartate receiver, in a response regulator which may constitute part of the sensor protein or a separate protein. The two-component system may couple with a downstream MAPK or directly phosphorylate specific targets to initiate cellular responses. There is accumulating evidence indicating that plants rapidly activate MAPK when exposed to multiple abiotic stress stimuli[54,55]. The best characterized two-component histidine kinase is the yeast osmosensor SLNI. Together with XPDI–SSK1 response regulator, this two-component signal unit regulates the high osmolarity glycerol (HOG) MAPK cascade, resulting in the production of glycerol to survive osmotic stress. Other MAPK modules (MAPKKK–MAPKK–MAPK) that are involved in osmotic stress signalling have been identified in alfalfa as SIMKK–SIMK[55] and in tobacco as Nt MEKZ–SIPK/WIPK[56,57]. Salt stress can activate different MAPKs at different times after the onset of stress[58]. The diverse and multiple stress responses of MAPKs suggest that there is a fundamental difference in functional specificity of MAPKs with respect to drought/salt response. Understanding of the MAPK cascade can provide insight to understanding and solving the problem of drought/salt stress in agricultural crops.

The role of MAPK modules in plant tolerance to abiotic stress was further illustrated by a study, where a tobacco MAPKKK ANP orthologue, NPK1 was expressed in an active form in *Arabidopsis*. NPK1 mediates H_2O_2-regulated gene expression in plants[59]. In addition to the existence of connections among MAPK modules used by osmotic stress

and ROS signalling, there is also specificity in the individual pathways[60].

Secondary signal molecules in signal transduction pathways

Abiotic stresses result in transient increases in cytosolic Ca^{+2} either through influx from the apoplastic space or release from the internal stores[8,61]. The primary increase in cytosolic calcium leads to output 1 and generates a secondary signal (hormone and second messenger) which initiates another cascade of signalling events and stimulates a second round of transient calcium increases, resulting in output 3. Secondary signalling molecules such as ROS can also regulate signal transduction without calcium (output 2; Figure 3). Internal Ca^{+2} release is regulated by ligand-sensitive Ca^{+2} channels. Inositol polyphosphates, cyclic ADP ribose, nicotinic acid adenine dinucleotide phosphate could act as second messengers. These molecules have been found to be able to induce Ca^{+2} release in plant cells[62]. One of these second messengers has been reported to be inositol triphosphate (IP_3). During stress, phosphoinositide-specific phospholipase C (PI-PLC) hydrolyses phosphatidyl-inositol 4,5 bisphosphate (PIP_2) upon activation. During stress, changes in phospholipid composition have been detected in plants[63]. PIP_2 itself is a signal molecule[64]. During stress, the major role of phospholipids may be to serve as precursors for the generation of second messengers. During osmotic stress, plant cells may increase the production of PIP_2 by upregulating the expression of *PI5K*, a gene that encodes a phosphatidyl inositol 4-phosphate 5-kinase functioning in the production[65] of PIP_2 (Figure 4). PIP_2 levels were found to be increased in ATH cells cultured under osmotic stress[66,67]. Drought and salt stress also upregulated the mRNA levels for certain PI-PLC isoforms[68,69].

In plants, the role of exogenous IP_3 in releasing Ca^{+2} from cellular stores has been reported[61,62,67]. Inhibition of PI-PLC activity eliminated IP_3 increase[67,70] and inhibited the osmotic stress induction of the stress-responsive genes *RD29A* and *COR47*. The stress hormone ABA also elicited the increase in $1P_3$ levels in *Vicia faba* guard cell protoplasts[71] and *Arabidopsis* seedlings[45,72].

Since IP_3 has a critical role in signalling, its level must be tightly regulated. Though information on turnover of IP_3 in plants is limited, it has been observed that modifying inositol-5-phosphatase dosage could regulate stimulus-induced endogeneous IP_3 levels and thus affecting stress and ABA signal transduction[72,73].

Phospholipase D (PLD) can also be involved in transduction of stress signals. PLD hydrolyses phospholipids to generate phosphatidic acid (PA), another second messenger in animal cells that can activate PI-PLC and protein (Figure 5) kinase C[74]. PA may also serve as a second messenger in plants[75]. Drought and hyperosmolarity activated PLD and increased PA level in plants[76–78]. PLD appeared to be activated by osmotic stress through a G protein[76] independent of ABA. Drought stress-induced PLD activities were found to be higher in drought-sensitive than in drought-tolerant cultivars of cowpea[79], suggesting that a high PLD activity may jeopardize membrane integrity as PA is a non-bilayer lipid favouring hexagonal phase formation and may destabilize membranes at high concentrations[75]. So excess PLD may have negative impact on plant stress tolerance. *Arabidopsis* deficient in PLD_α was found to be more tolerant to freezing stress[23].

Role of ABA in signalling

Abiotic stress causes an increase in ABA biosynthesis, which is then rapidly metabolized following the removal of stress[80–83]. Many stress-responsive genes are upregulated by ABA[9,10,84]. ABA is a regulatory molecule involved in drought stress tolerance. The main function of ABA is to regulate osmotic stress tolerance via cellular dehydration tolerance genes and to regulate plant water balance

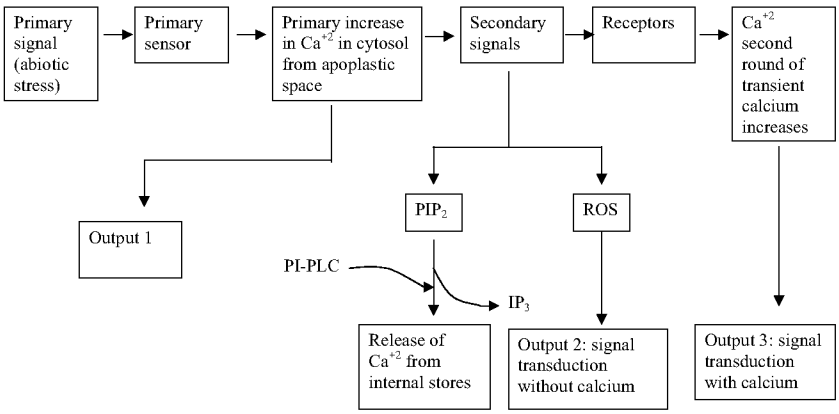

Figure 3. Role of secondary signal molecules in signal transduction pathways as affected by abiotic stresses[7,8,61,62].

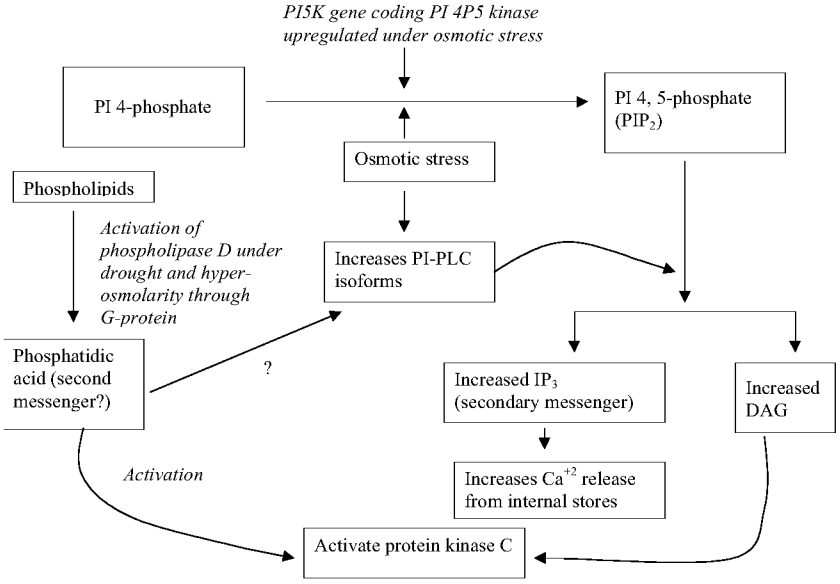

Figure 4. Role of PI 4P5 kinase in signal transduction[62,66–69,75]. PI-4-phosphate, Phosphatidyl inositol 4-phosphate; PI 4,5 phosphate, Phosphatidyl inositol 4,5 phosphate; PIP_2. Phosphatidyl inositol bisphosphate; PI-PLC, Phosphatidyl inositol phospholipase C.

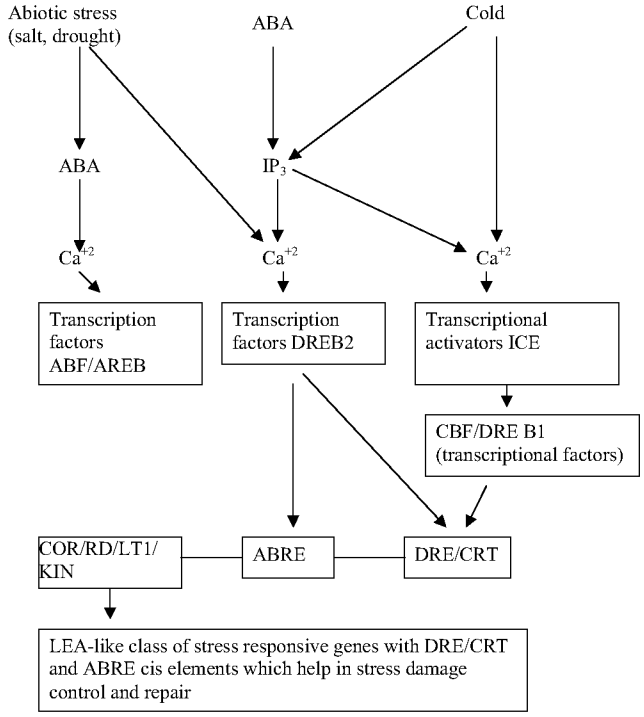

Figure 5. Pathways for activation of LEA-like class of stress-responsive genes[21–23,45,50]. COR, Cold-regulated; KIN, Cold-induced; LTI, Low temperature-induced; RD, Responsive to dehydration; ABF, ABA responsive element binding factor; CBF, Cold responsive element binding factor; ICE, Inducer of CBF expression; DRE, Dehydration responsive element; ABRE, Abscisic acid responsive element.

through guard cells. ABA is also induced by salt and to a lesser extent by cold stress. ABA-inducible genes have the ABA-responsive element (ABRE) (C/T)ACGTGGC in their promoters. Basic leucine zipper factors (bZIP) function in signal transduction by binding to the ABRE element in stress-inducible genes. Many bZIP factors have now been identified, including AREB binding protein. They could activate the dehydration-responsive *RD29B* gene[85,86]. Recent work has focused on determining the roles of bZIP factors *in vivo*[87,88]. Two well-studied genes for dehydration response are *RD22* and *RD29B*. The *RD22* gene is induced by ABA under drought and salt stress, and requires protein synthesis for mRNA expression. The *RD29* response involves two components, *RD29A* and *RD29B*, with temporally distinct expression. The *RD29A* gene is expressed within 20 min of stress induction, whereas rd29B peaks 3 h later[89].

Various studies conducted using transgenic *Arabidopsis* that expressed the firefly luciferase reporter gene (*LUC*) under control of the *RD29A* promotor which contained both ABRE and DRE/CRT suggested extensive connections between cold, drought, salinity and ABA signal transduction pathways[50]. It has been suggested that stress signalling pathways for the expression of LEA-like genes are not completely independent of ABA. During genetic screening, a mutant with reduced expression of *RD29A–LUC* under osmotic stress was isolated[50]. Two of the loci defined in these mutants, i.e. LOS 5 and LOS 6 were characterized and the genes isolated. In LOS 5, the expression of many stress-responsive genes like *RD29A*, *COR15*, *COR47*, *RD22* and *P5CS* were severely reduced during salt stress[45]. LOS 5 plants were defective in drought-induced ABA biosynthesis. When exogenous ABA was applied, salt induction of *RD29A–LUC* was restored to the wild type level, indicating the role of ABA in osmotic stress

regulation of gene expression[45]. In LOS 6 mutants, osmotic stress induction of *RD29A*, *COR15A*, *KIN1*, *COR47*, *RD19* and *ADH* was lower than that in wild type plants[23]. LOS 6 plants were also defective in drought-induced ABA biosynthesis[23].

Characterization of the LOS 5 and LOS 6 mutants revealed a critical role of ABA in mediating osmotic stress regulation of gene expression. Since ABA deficiency did not appear to significantly affect the expression of *DREB2A* (which codes for a drought-stress specific transcription factor), it was thought that ABA signalling may be required for regulating the activity of *DREB2A* or its associated factors in the activation of the DRE class of genes[45] (Figure 5). This level of interaction between ABA signalling and osmotic stress signalling may indicate a synergistic interaction between them in activating the expression of stress-responsive genes[45].

Calcium dependent signal transduction pathways

The increase in calcium by secondary signal molecules is perceived by various calcium-binding proteins. In abiotic stress signalling, CDPKs and the SOS3 family of Ca^{+2} sensors are involved in coupling of this calcium signal to specific protein phosphorylation cascades. CDPKs are serine/threonine protein kinases with a C-terminal cadmodulin-like domain with up to 4 EF hand motifs that can directly bind Ca^{+2}. Some CDPKs have N-terminal myristoylation motif, suggesting their potential association with membranes. CDPKs are activated by abiotic stresses[90]. In rice plants, a membrane associated CDPK was activated by cold treatment[91]. Overexpression of *OSCDP7* resulted in increased cold and osmotic stress tolerance in rice. A potential role for CDPK in directly shuttling information to the nucleus to activate gene expression has been suggested[92]. The role of CDPK has been discussed in the activation of transcription factors which induce gene expression of LEA-like proteins. Another group of calcium sensors in plants is the SOS3 family of calcium-binding proteins. Their role has been implicated in ion homeostasis.

Ca^{+2}-dependent signalling that leads to activation of LEA-type genes

The most common and widely reported genes that are stress regulated are the LEA or LEA-like genes. These are highly expressed in seeds during desiccation stage and in vegetative tissues in response to water deficit[93,94]. Overexpression of individual LEA genes has been reported to confer stress tolerance in transgenic rice[95]. One group of genes in *Arabidopsis*, e.g. RD (responsive to dehydration) is strongly induced by salt and drought stresses. The products of these genes have resemblance to LEA proteins and hence are called LEA-like proteins. These genes are not expressed under normal conditions. The enhanced expression of transcription factors that regulate the expression of these genes increased the tolerance of transgenic plants to drought and salt stress. It showed the protective effect of these proteins, which could be due to the prevention of denaturation of key proteins[96] by acting as chaperones[97].

Ca^{+2}-dependent SOS signalling that regulates homeostasis

Restoring ion homeostasis in plants disturbed by salt stress represents a crucial response. Plant responses in countering ionic stress caused by high salinity include restricting salt intake, increased extrusion, compartmentalization and controlled long-distance transport to aerial parts. Additionally, to avoid cellular damage and nutrient deficiency, plant cells need to maintain adequate K^+ nutrition and a favourable K^+/Na^+ ratio in the cytosol[98,99].

Calcium has been observed to have a protective effect under sodium stress both in solution culture and in soils that had increased calcium supply. This effect could be due to increased availability of cytosolic Ca^{+2}. When the *Arabidopsis ACA4* gene that codes for vacuolar Ca^{+2}-ATPase was expressed in yeast, it increased the salt tolerance of the yeast cells[100]. An early detectable response to sodium stress is the rise in cytosolic free calcium concentration[8]. Sodium stress is sensed by an unknown receptor and calcium signal serves as a second messenger. In *Arabidopsis*, genetic studies suggested that the sensor protein for this salt-induced calcium signature is the Ca^{+2}-binding protein SOS3. A loss of function mutation in this protein renders the plant hypersensitive to salt stress. Sodium extrusion[101,102] is achieved by plasma-membrane localized Na^+/H^+ antiporter SOS1. Mutations in SOS1 rendered the mutant plants sensitive[103] to Na. Plasma membrane vesicles from *Arabidopsis* plants have a Na^+/H^+ antiporter activity, which was enhanced by pretreatment with salt stress[104].

Another mechanism to reduce accumulation of cytosolic Na^+ is achieved by the action of Na^+/H^+ antiporters on the tonoplast. The proton gradient that drives the antiporter is generated by tonoplast H^+-ATPases and pyrophosphatases (Figure 6). Transgenic *Arabidopsis* plants overexpressing the vacuolar H^+-pyrophosphatase, AVP1 showed increased tolerance to salt stress[105]. Similarly, overexpression of one of the vacuolar Na^+/H^+ antiporters, At NHX1, increased the salt tolerance of the *Arabidopsis* plants[106].

Whatever is the mechanism of response of plants to abiotic stresses, a transient increase in cytosolic Ca^{+2} must be coupled with downstream signalling events to mediate stress adaptation. In *Arabidopsis* salt stress signalling, the Ca^{+2} signal is perceived by the calcineurin-β-like Ca^{+2} sensor[96] SOS3. However, unlike the calcineurin-β in yeast that acts through activation of a protein phosphatase, SOS3 interacts with and activates[107] protein kinase SOS2. Thus SOS3 resembles an adapter or scaffold protein that mediates the interaction

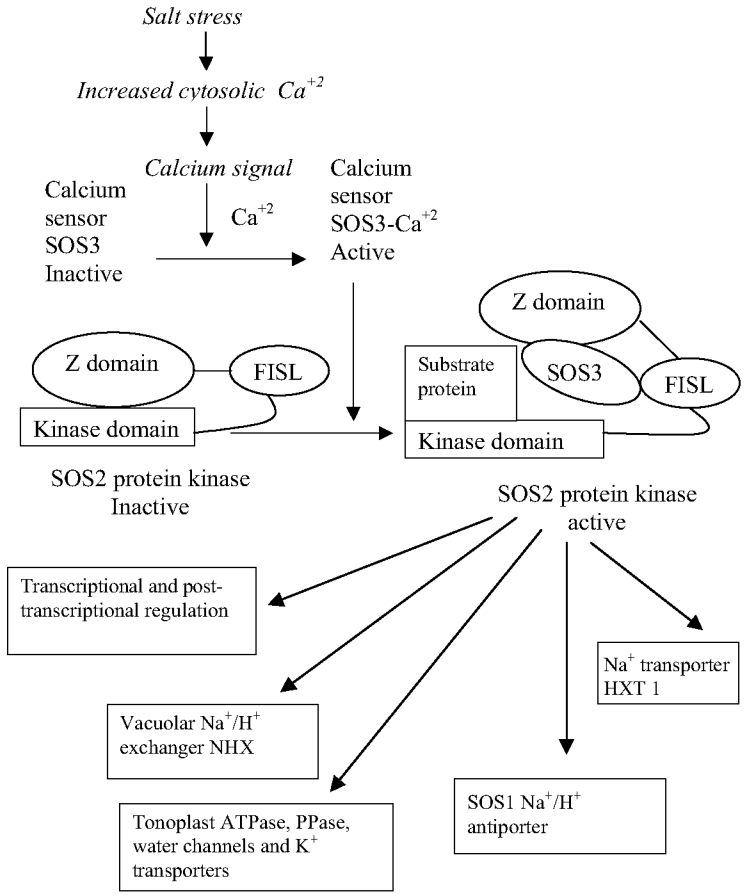

Figure 6. Pathways showing activation of SOS2 protein kinase by calcium sensor, SOS3 and regulation of ion homeostasis[101–109].

of SOS2 with other proteins such as ion transporters. This property of SOS3 was suggested due to the requirement of its myristolylation for full action in salt tolerance[108]. The interaction of SOS3 and SOS2 was studied by analysing the functional domains of both proteins. The regulatory and catalytic domains of SOS2 interact with each other, resulting in auto inhibition of kinase[109] (Figure 6). A 21 amino acid motif (FISL motif, refers to the conserved amino acid residue) in the regulatory domain mediates the interaction of SOS2 with SOS3. This interaction appears to result in auto inhibition and provides the substrate access to the SOS2 kinase domain[109]. Possibly, through this phosphorylation, the activated SOS2 could modulate the activity of ion transporters such as SOS_1 and HKT_1.

Conclusions

The multiple stress responses on various kinds of genes and their transcribed products involved in a variety of cellular functions are important in understanding and solving the problems of drought/salt stress tolerance. Different signal transduction pathways act independently and also have a significant crosstalk among themselves. It makes their understanding under abiotic stimuli complex. Multiple genes which are affected under abiotic stresses indicate that there could not be a single marker for stress tolerance. Biochemists and molecular biologists should look forward for defined set of markers to predict tolerance towards a particular type of stress with a definite degree of assurance.

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