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Review

Chemical signaling under abiotic stress environment in plants

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Many chemicals are critical for plant growth and development and play an important role in integrating various stress signals and controlling downstream stress responses by modulating gene expression machinery and regulating various transporters/pumps and biochemical reactions. These chemicals include calcium (Ca^{2+}), cyclic nucleotides, polyphosphoinositides, nitric oxide (NO), sugars, abscisic acid (ABA), jasmonates (JA), salicylic acid (SA) and polyamines. Ca^{2+} is one of the very important ubiquitous second messengers in signal transduction pathways and usually its concentration increases in response to the stimuli including stress signals. Many Ca^{2+} sensors detect the Ca^{2+} signals and direct them to downstream signaling pathways by binding and activating diverse targets. cAMP or cGMP protects the cell with ion toxicity. Phosphoinositides are known to be involved both in transmission of signal across the plasma membrane and in intracellular signaling. NO activates various defense genes and acts as a developmental regulator in plants. Sugars affect the expression of many genes involved in photosynthesis, glycolysis, nitrogen metabolism, sucrose and starch metabolism, defense mechanisms and cell cycle regulation. ABA, JA, SA and polyamines are also involved in many stress responses. Cross-talk between these chemical signaling pathways is very common in plant responses to abiotic and bitotic factors. In this article we have described the role of these chemicals in initiating signaling under stress conditions mainly the abiotic stress.

Introduction

Under unfavourable environmental condtions plant growth and development is severely affected which impede the productivity and thus prevent them from reaching their full genetic potential. Indeed, abiotic stresses are the primary cause leading to worldwide crop loss and reducing the average yield for most crops by more than 50%.[1] In many plants in response to these stresses various genes gets upregulated resulting in altered metabolic functions, which can mitigate the effect of stress and lead to plant adaptation. Plant responds to stresses as individual cells and synergistically as a whole organism. Stress signal is first perceived by the receptors present on the membrane of the plant cells. Following this the signal information is transduced downstream resulting in the activation of various stress responsive genes (Fig. 1). The products of these stress genes ultimately lead to stress tolerance response or plant adaptation and help the plant to survive and surpass the unfavorable conditions.[2-6] The response could also lead to growth inhibition or cell death, which will depend upon how many and which kinds of genes are up or down regulated in response to the stress (Fig. 1). Overall, the stress response is a coordinated action of many genes encoding signaling proteins/factors, including protein modifiers (methylation, ubiquitination, glycosylation, etc.,), adaptors and scaffolds.[7]

Various chemicals are known to be involved in signal transduction pathways. During the last couple of decades a number of second messengers and hormones that are altered in response to stresses have been identified. Among these the role of calcium and ABA have been well studied and have been reviewed earlier.[8,9] In addition to these, in recent years, the role of cyclic nucleotides, polyphosphoinositides, NO, sugars, JA and SA and other hormones have been elucidated. In this review we have briefly covered the recent work on the role of all these chemicals and their downstream signaling components especially under abiotic stress conditions.

Calcium Signaling

Calcium (Ca^{2+}) is an essential plant macro-nutrient element that is taken up by roots and delivered to the shoot via the xylem where it regulate many physiological processes.[7,8,10-13] The plant cell contains a number of vesicular compartments, which stores Ca^{2+} that can be released into the cytoplasm when required. Specific channels/pumps regulate the movement of Ca^{2+} in and out of cells and organelles.[7,13] Using various techniques it has been shown that Ca^{2+} concentrations in apoplasts ranges between 0.1 to 1.0 mM in vacuoles and ~1 mM in endoplasmic reticulum lumen and these levels can go upto 50 mM. Plastids and mitochondria contain millimolar levels of Ca^{2+} but much less than endoplasmic reticulum. The cytosolic free Ca^{2+} concentration is maintained typically at 200 nM.[14] However, the Ca^{2+} content of the cytosol is far higher than this because of the high affinity for Ca^{2+} to a range of Ca^{2+} binding proteins.

The changes in the cytoplasmic calcium concentrations following stress perception have been reported. It was in 1996 that Knight et

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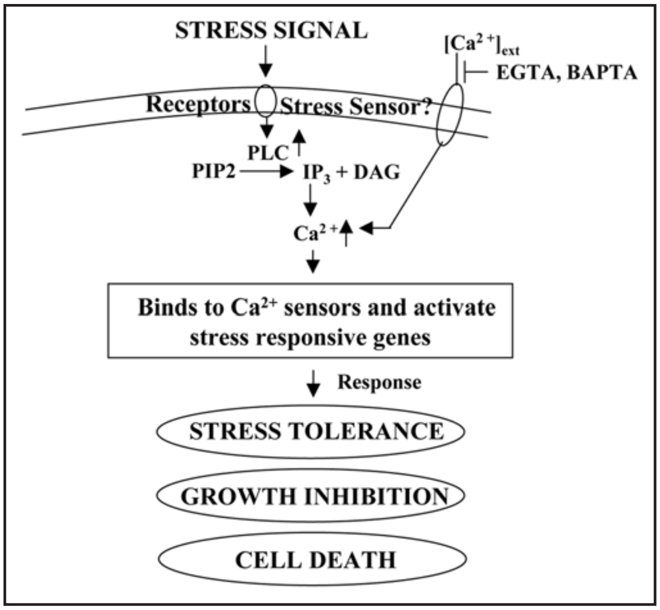

Figure 1. Generic pathway for plant response to stress. The extracellular stress signal is first perceived by the membrane receptors and then activate large and complex signaling cascade intracellularly including the generation of secondary signal molecules. The stress signal can first activates phospholipase C (PLC), which hydrolyses PIP_2 to generate IP_3 and DAG resulting in an increase in the level of Ca^{2+} ions in the cytosol, which is sensed by calcium sensor. The signal cascade results into the expression of multiple stress responsive genes, the products of which can provide the stress tolerance directly or indirectly. Overall, the stress response could be coordination action of many genes, which may cross-talk with each others.

al., showed biphasic changes in calcium levels in response to cold stress. Similarly water stress and heat induced changes in calcium levels were also reported.[15-16] The relationship of these changes with gene expression was also indicated in earlier studies.[17] Infact it was found that the changes in calcium were cell type specific.[18] Some of this and other work on the role of calcium in stress signaling have been reviewed.[19,20] Recently, using various activators and inhibitors, it was found that calcium may be involved even in long-term processing if signals in plants in response to abiotic stresses.[21] It thus became evident that calcium is one of the principal candidates for functioning as central node in overall 'signaling web' and plays important role in providing stress tolerance. Recently its role in salinity tolerance has been reviewed.[8,13] High salinity leads to increase cytosolic Ca^{2+}, which initiates the stress signal transduction pathways for stress tolerance.[6,7]

While the source of calcium resulting in its increase in the cytoplasm after stress has not be very well studied. Ca^{2+} release can be primarily from extracellular source (apoplastic space) as addition of EGTA or BAPTA was shown in many cases to block calcium effects (Fig. 1). Ca^{2+} release may also result from activation of PLC (phospholipase C), leading to hydrolysis of PIP_2 to IP_3 and subsequent release of Ca^{2+} from intracellular Ca^{2+} stores. Furthermore, calcium-binding proteins (calcium sensors) can provide an additional level of regulation in the calcium signaling. These sensor proteins recognize and decode the information provided in the calcium signatures,[8] relay the information downstream to initiate a phosphorylation cascade leading to regulation of gene expression (Fig. 1). On the functional basis, CaBP can be classified as trigger proteins and buffer proteins. Trigger proteins get active only after binding to Ca^{2+} and then interact with other proteins in signal transduction pathway and alter their activity. Buffer proteins bind to the elevated level to sequestre Ca^{2+}. Only few CaBP of trigger type, which are involved in stress mediated Ca^{2+} signaling, have been identified in plants such as calmodulin (CaM), CaM-binding proteins, Ca^{2+} dependent protein kinases and phosphatases.[12]

Calmodulin is one of the important CaBP and contains four calcium binding sites (Fig. 2A). Upon receiving stimuli, there is an increase in the intracellular Ca^{2+}. The increase in calcium is sensed by various proteins like calmodulin or directly by calcium dependent protein kinases (CDPKs). The CDPKs have a catalytic domain and calcium binding domain like calmodulin (Fig. 2B). There are other calcium binding proteins also which can sense calcium concentration. All these proteins can trigger downstream processes, which can elicit specific response (Fig. 2C). The response is terminated by OFF mechanisms that restore Ca^{++} to the resting level. Various physical and chemical signals have been shown to induce mRNA corresponding to CaM and CaM-related genes and in some cases Ca^{2+} signals, for example generated via wind and cold stimuli, operate through CaM gene expression (Fig. 2C). From various studies it is clear that CaM is a very important protein in Ca^{2+} signaling and is involved in various process in plants including biotic and abiotic stresses.[8,12,22] CaM can also regulate gene expression by binding to specific transcription factors.[8,23]

Plants possess many unique, putative Ca^{2+}-sensors, including a large family (50 in Arabidopsis) of calmodulin-like proteins termed CMLs. These CaM like proteins differ from the CaM in containing more than 148 amino acid residues and have between one to six EF hand motifs. They possess limited homology to CaM (75% identity with canonical CaM isoforms.[12,24] Some of these CMLs have been implicated in Ca^{2+}-based stress response but most remain unstudied. Recently, Venderbeld and Snedden (2007)[25] suggested that these CMLs likely play important roles as sensors in Ca^{2+}-mediated developmental and stress response pathways. In Ca^{2+}-mediated signaling many proteins (CaBP) are bound and regulated by CaM and known as CaMBP. In plants some of the examples of CaMBP, which are stimulated by CaM are small nuclear NTPases, glutamate decarboxylase, NAD-kinases, Ca^{2+}-ATPases (ER and tonoplast located), kinesin-like CaMBP, heat-shock proteins and few transcription factors of basic helix-loop-helix family. In Arabidopsis AtCAM3 promoter binds to the leucine zipper family of transcription factor TGA3, while CaM itself acts as an enhancer of TGA 3 binding with its Cis-elements. All these suggests that Ca^{2+}-mediated signaling coupled to gene expression might go via CaM and CaM binding transcription factors and could lead to specificity of the response. It was shown that a Calcium/CaM dependent kinase that was stress regulated phosphorylated a protein that showed binding to stress regulated promoter.[20]

There are several proteins that bind Ca^{2+} but do not contain EF-hand motifs. These include, the phospholipase D (PLD), annexins, calreticulin, calnexin and Pistil-expressed Ca^{2+}-binding protein (PCP). The activity of PLD, which cleaves membrane phospholipids into a soluble head group and phosphatidic acid, is regulated by $[Ca^{2+}]_{cyt}$ through a Ca^{2+}/phospholipids binding-site termed as the 'C2 domain'.[26] PLD activity is implicated in cellular

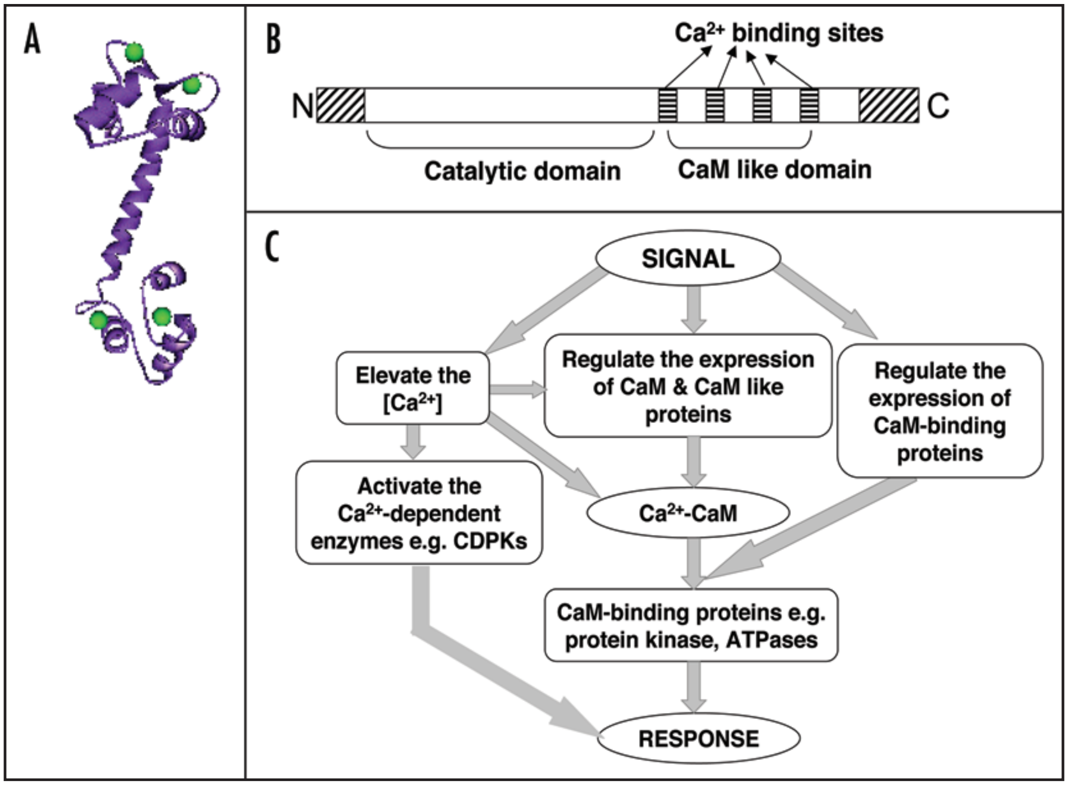

Figure 2. The units of the calcium signaling network. A major protein that regulates a number of calcium mediated signals is calmodulin, which has four calcium binding sites (A). On receiving stimulii, calcium mobilizing signals act on various ON mechanisms that lead to an increase in the intracellular Ca^{++}. The increase in calcium is sensed by various proteins like calmodulin or directly by calcium dependent protein kinases (CDPKs). The CDPKs have a catalytic domain and calcium binding domain like calmodulin (B). There are other calcium binding proteins also which can sense calcium concentration. All these proteins can trigger downstream processes which can elicit specific response (C). The response is terminated by OFF mechanisms that restore Ca^{++} to the resting level.

responses to ethylene and ABA, α amylase synthesis in aleurone cells, stomatal closure, pathogen responses, leaf senescence and drought tolerance.[27] Plants posses several PLD isoforms that differ in their affinity for Ca^{2+} and their modulation by phosphoinositides, free fatty acids and lysolipids.[26] These biochemical modulators of PLD activity are the substrates or products of phospholipase C, which generates IP_3, diacylglycerol (DAG), phospholipase A_2 and diacylglycerol kinase, both of which are regulated by CaM. It is suggested that $[Ca^{2+}]_{cyt}$ signaling cascades might coordinate the activities of these diverse enzymes to effect specific responses to the environmental stimuli.[26]

Annexins are a family of proteins in plants and animals that bind phospholipids in a Ca^{2+} dependent manner and contain four to eight repeats of about 70 amino acids.[8] Annexins are encoded by twelve genes in verterbrates and by eight in higher plants. These proteins are involved in organization and function of biological membranes.[8] Although exact function of plant annexin is not known, but they are implicated in secretory processes and some have ATPase, peroxidase activities.[8] On the basis of existing literature and experimental evidence, Górecka et al., (2007a)[28] have proposed that plant annexins may have a role in stress response. Infact, Górecka et al., (2007b)[29] have shown that Annexin At1 of *Arabidopsis thaliana* (AnnAt1), which is one of eight proteins of this family in *A. thaliana*, play important role in pH-mediated cellular response to environmental stimuli. The ssame group also suggested that the pH-sensitive ion channel activity of AnnAt1 might play a role in intracellular ion homeostasis.

Calnexin (CNX) is a 90 kDa integral memberane protein of the endoplasmic reticulum (ER). It acts as a molecular chaperone that share several functions, including Ca^{2+} binding, lectin-like activity and recognition of misfolded proteins.[30,31] CNX also contributes to the quality control of non-glycosylated polytopic membrane proteins by binding to misfolded or unassembled transmembrane domains. It also plays a role in the degradation of misfolded proteins. Its role in stress has not yet been studied, however it may regulates the endoplasmic reticulum stress response in a manner unique to plants.

Several reports indicate that Ca^{2+} regulates the transcription of target genes by affecting changes in the phosphorylation status of specific transcription factors. ABA-induced expression of two genes in aurea mutants of tomato, *rd-29A*, a dessication-induced gene and *kin2*, a cold-responsive gene are also reported to be Ca^{2+}-regulated. Analysis of transcriptome changes in response to increase in cytosolic calcium revealed that ABRE-related sequences also act as calcium responsive cis-elements.[32] It seems therefore that Ca^{2+}, can control many cellular processes in plant cell by interacting with various proteins and kinases, which result in the regulation of expression of various genes. Recently a novel family of transcription factors that bind calmodulin, named CAMTA (calmodulin-binding transcription activators) have been identified and indicated to be involved in stress induced calcium mediated gene expression in Arabidopsis.[33,34] The presence of CAMTA has also been shown in rice.[35] All CAMTAs have a novel type of sequence specific DNA binding domain and also have IQ motif known to be associated with binding to CaM.

CAMTA can also interact with other proteins to and act also as co-activator of transcription.[36]

In addition to regulating gene expression, another pathway called SOS (salt overly sensitive) pathway, has been implicated in calcium-mediated pathway for salinity stress tolerance.[37] As salt stress imposes a major environmental threat to agriculture, understanding the basic physiology and genetics of cell under salt stress is crucial for developing any transgenic strategy. The *SOS* genes (*SOS1–SOS3*) were isolated through positional cloning. Since *sos* mutants are hypersensitive to salt, their characterization resulted in the discovery of a novel pathway, which has helped in our understanding the mechanism of salt stress tolerance in plants. The *sos* mutants i.e., *sos1*, *sos2* and *sos3*, could accumulate more proline under salt stress and this accumulation seems to correlate with the extent of damage caused by salt stress.[38] All these 3 mutants i.e., *sos1, sos2* and *sos3* showed a similar spectrum of phenotype indicating that these three *SOS* genes function in the same or related pathway. The fact that double mutants of these sos genes did not have any additive effect on the salt sensitive phenotype indicated that these genes work in a same pathway leading to salt tolerance.[39] However, among the three SOS loci, SOS1 plays the greatest role in imparting salt stress tolerance. *Sos1* mutant plants are more sensitive to Na^+ and Li^+ stress in comparison with *sos2* and *sos3* mutants.[40] Genetic analysis confirmed that SOS1-SOS3 function in a common pathway of salt tolerance. This pathway also emphasizes the significance of Ca^{2+} signal in reinstating cellular ion homeostasis. SOS3, a Ca^{2+} sensor, transduces the signal downstream after activating and interacting with SOS2 protein kinase. This SOS3-SOS2 complex activates the Na^+/H^+ antiporter activity of SOS1 thereby reestablish cellular ion homeostasis. Recently, SOS4 and SOS5 have also been characterized.[37] *SOS4* encodes a pyridoxal (PL) kinase that is involved in the biosynthesis of pyridoxal-5-phosphate (PLP), an active form of vitamin B6. SOS5 has been shown to be a putative cell surface adhesion protein that is required for normal cell expansion. Under salt stress, the normal growth and expansion of a plant cell becomes even more important and SOS5 helps in the maintenance of cell wall integrity and architecture.

In addition to the nature of calcium signal, discussed earlier, the specificity of Ca^{2+} signals will also depend on the concentration and activity of these proteins. The specificity of the response will also depend upon the source of Ca^{2+}. For example in response to elicitor, GA and water stress, the source of Ca^{2+} is from extracellular or apoplastic while in response to wind and touch the source is from intracellular stores. However, for cold shock induced responses the source of Ca^{2+} could be both extracellular stores and intracellular which is IP_3-mediated.

The multifunctional role of Ca^{2+} suggests that it participates in many signaling pathways in the plant cell. In fact an indirect increase in $[Ca^{2+}]_{cyt}$ in plant cell is mediated by the increase in intracellular concentrations of other second messengers such as cGMP, cAMP, IP_3 etc., in response to stimuli. It is clear now that the elevation of Ca^{2+} concentration in cytoplasm is a key event in the plant cell for transduction of various signals to a biological effect. This suggests that Ca^{2+} is a communication point in the cytoplasm for the cross-talk between different signal transduction pathways and finally helps interchanging the information.

Signaling via Cyclic Nucleotides

cAMP and cGMP are ubiquitous second messengers and play very important role in cell signaling. In fact cAMP was the first intracellular messenger to be discovered. The levels of cAMP and cGMP in the cell are determined by the activities of cyclases (which catalyze their synthesis) and phosphodieserase (PDE; which hydrolzye them), as shown below:

$$\text{ATP} \xrightarrow{\text{adenylate cyclase}} \text{3', 5'-cAMP} + \text{PPi}$$

$$\text{GTP} \xrightarrow{\text{guanylate cyclase}} \text{3', 5'-cGMP} + \text{PPi}$$

$$\text{3', 5'-cAMP} + H_2O \xrightarrow{\text{cAMP PDE}} \text{5'-AMP}$$

$$\text{3', 5'-cGMP} + H_2O \xrightarrow{\text{cGMP PDE}} \text{5'-GMP}$$

There have been a number of reports showing the presence of cyclic AMP in plants. A cDNA encoding adenylate cyclase was also reported from ax1-141 tobacco line, which also suggested the existence of cAMP signaling in plant. Its role has been suggested in flowering, in auxin-triggered cell division in higher plants, in the cell cycle control of tobacco cell culture, and in the activation of ion channels that were permeable for Ca^{2+} and were negatively regulated by CaM.

Kohler et al., (1999)[41] have reported a new gene family in Arabidopsis, which share features with cyclic-nucleotide-gated channels from animals and inward-rectifying K^+ channels from plants. This gene family comprised of six members of cyclic nucleotide-gated channels, with significant protein sequence homology among them. An extracellular nucleotide PDE from tomato cultured cells has been purified. The purified enzyme has a molecular mass of 70 kDa, a pH optimum of 6.2 and an isoelectric point of 8.1. The enzyme was shown to induce with RNase LE and acid phosphatase upon phosphate starvation of suspension cultured tomato cells. These authors also provided evidence that tomato cyclic nucleotide PDE was an accessory ribonucleolytic activity required for complete degradation of extracellular RNA.

In Arabidopsis, cGMP was shown to modulate the activity of inward rectifying K^+ channels KAT1 and AKT1. High salinity stress is a major cause of sodium toxicity in plants. Still not much is known about the mechanisms that underlie Na^+ influx. Maathuis and Sanders (2001)[42] reported that sodium uptake in Arabidopsis roots is regulated by cyclic nucleotides. The presence of micromolar concentrations of cAMP or cGMP at the cytoplasmic side of the plasma membrane evoked a rapid decrease in channel open probability. The short-term unidirectional Na^+ ions influx was also reduced in the presence of cyclic nucleotides. In summary, the data of Maathuis and Sanders (2001)[42] suggested that Arabidopsis plants might contain a cyclic nucleotide-based signaling pathway that directly affects Na^+ ions transport via voltage independent channels. In a later study, Maathuis (2006)[43] studied transcriptome following treatment with membrane permeable cGMP and found changes in many transcripts including those involved in defence and cGMP also modulated Na^+ and K^+ fluxes.

Phosphoinositide Signaling

Phosphoinositides are key components of the inositol 1,4,5-trisphosphate/diacylglycerol-based signaling pathway in animal cells. In plant cells different phosphoinositide isomers have also been identified.[44] Phosphoinositides are the inositol-containing phospholipids of plasma membrane of plant cells and are known to be involved both in transmission of signal across the plasma membrane and in intracellular signaling. The inositol head group can be phosphorylated at several different positions, giving rise to a number of multiple phosphorylated species like phosphatidylinositol (PI), PI-4-monophosphate (PI(4)P or PIP) and PI 4, 5-biphosphate (PI(4,5) P_2 or PIP_2).[45] These polyphosphoinositides are continuously being formed in the inner leaflet of the plasma membrane and are acted upon by set of specific kinases and phosphatases and thereby are kept in a state of constant turnover. One such kinase of significance is PI3 kinase (PI3Kase). Phospholipid metabolism is involved in hyperosmotic-stress responses in plants.[46]

In plants phospholipases are required for signal transduction in many events like during seed germination, in auxin-stimulated cell elongation, oil biosynthesis during embryo maturation, for membrane reorganization/lipid recycling, and in light mediated processes.[47,48] The action of phospholipases can generate three important signal molecules IP3, DAG and PA (Fig. 1). PLD catalyzes the hydrolysis of the phosphodiester bond between the phosphate and the polar group (inositol, serine, glycerol, choline, ethanolamine) and can produce phosphatidic acid. PI specific PLC uses PIP_2 as a substrate to release DAG and inositol triphosphate (IP_3). Overall, phospholipases they play an important role in plant growth and development including their role in stress tolerance.[47,48] Their catalytic properties are often controlled by phosphorylation, interaction with other proteins (e.g., G proteins), as well as interactions with other lipids.[48] The signaling through PI3K, phosphotidic acid, IP3 and DAG are described below.

Phosphatidylinositol 3-kinases (PI3Ks). The PI3K family of enzymes phosphorylates D-3 position of inositol ring (i.e., 3' hydroxyl group) of PI, PIP and PIP_2 resulting in the formation of PI-3-phospate (PI-3P), PI-3,4-biphosphate (PI-3,4-P_2) and PI-3,4,5-triphosphate (PI-3,4,5-P_3) respectively.[49] Both PI(3,4) P_2 and PI(3,4,5)P_3 are thought to be second messenger molecules that interact with target proteins to change their activity within the cell. In animal systems P13K has been shown to be a component of a many tyrosine kinase complexes that transduce growth factor signal(s) for cell division. The PI3-Ks are now known to be involved in a plethora of cellular processes, ranging from mitogenesis, membrane trafficking and ruffling, to glucose uptake, oxidative burst responses, chemotaxis and apoptosis. PI3K is composed of a catalytic (110 kDa) and regulatory (85 kDa) subunit.[49]

In plant, this is known to be present in Arabidopsis, soybean and carrot.[50-52] The Arabidopsis PI3K (AtVPS34) was shown to be involved in plant growth and development.[50] In soybean, PI3K was reported to be induced during nodule organogenesis and was shown to be associated with membrane proliferation.[51] In carrot, PI3K activity was associated with the cytoskeleton.[52] In carrot suspension cells, PI3K has been shown to be localized on the transcription sites present in the nucleus and nucleolus.[52] Stomatal guard cells have been reported to contain PI3P and PI3K activity. Guard cells generate reactive oxygen species (ROS) in response to abscisic acid (ABA), which leads to stomatal closing. The upstream steps of the ABA-induced ROS generation pathway are not well studied. Park et al., (2003)[53] reported that PI3K inhibitors wortmannin or LY294002 inhibited ABA-induced ROS generation and stomatal closing, suggested the role of this in ABA-induced ROS generation in guard cells. It has been also reported that activation of PI3K and its product PI3P are required for auxin-induced production of ROS and root gravitropism.[54] Recently, Leshem et al., (2007)[55] have shown that salt stress responses, such as increased plasma membrane endocytosis and the intracellular production of ROS, were coordinated by phospholipid-regulated signaling pathways. These authors observed that induction of PI3K-mediated endocytosis by salt stress leads to intracellular production of ROS and salt tolerance, which suggested that ROS act in the signal transduction of the salt tolerance response.[55]

Phosphatidic acid (PA). PA and its phosphorylated derivative diacyl-glycerol pyrophosphate (DAG-PP) are lipid molecules that have been implicated in plant stress signaling.[56] It is one of the second messenger generated by activation of two different signaling pathways, those of PLC and PLD.[6,48] PLC hydrolyses PIP_2 into IP_3 and DAG. IP_3 diffuses into the cytosol, where it releases Ca^{2+} from intracellular stores, whereas DAG remains in the membrane, where it is phosphorylated to PA by DAG kinase.[48] However, PLD produces PA directly by hydrolysing structural phospholipids such as phosphatidylcholine.[57] DAG kinase was reported to be the dominant producer of PA that was subsequently metabolized to DAG-PP.[48] Since PA formation was reported to correlated with the high turnover of polyphosphoinositides, it was suggested that elicitor treatment activates PLC to produce DAG, which in turn acts as substrate for DAG kinase.[48]

In plants, PA level increases within minutes of wide variety of stress treatments including ethylene, wounding, osmotic stress, oxidative stress, drought, pathogen elicitors and abscisic acid.[53] The enhanced PA signaling in plants is attenuated by phosphorylating it to DAG-PP via a PA kinase. Whether the formation of DAG-PP is just a mechanism for attenuating PA levels or whether DAG-PP also functions as a signaling molecule remains to be seen. Several downstream targets of PA have been identified, including protein kinases and ion channels.[58] In general PA does not act by itself but it cross talks with other pathways, such as those controlled by Ca^{2+}, G-proteins, protein kinases, MAP kinases and reactive oxygen species. Recently, it was found that PA binds to CTR1, which is a negative regulator of ethylene signaling pathway.[59] In addition to PA, a PA metabolite, DAG pyrophosphate (DGPP), has been found in plants and this species is absent in higher animals. Its levels are very low under normal condtions but increase in response to stress. It is broken down by DGPP phosphatase.[60]

IP_3 and DAG. As mentioned above, in response to a stimulas the PLC gets activated and hydrolyzes PIP_2 to release two important second messengers viz IP_3 (inositol 1,4,5, trisphosphate) and DAG.[8,48] IP_3 is known in animal systems to release calcium from endoplasmic reticulum while in plants it has been shown that vacuole is the major store house for calcium.[8,48] It has been shown, using TLC and gas chromatography, and comparing constitutive and stress inducible poyphospohinositides, that there was loss in unsaturated phosphatidylinositol which are also channeled towards IP3 production.[61] IP3 has also been shown to play important role in abiotic stress signaling. Pical et al., (1999)[44] have shown that phosphatidylinositol 4,5-bisphosphate, diacylglycerol pyrophosphate and phosphatidylcholine in *Arabidopsis thaliana* cells increases rapidly in response to salinity and hyperosmotic stress. Drobak and Watkins (2000)[46] found that salts and osmotic agents increased the levels of IP3 by 15-fold as one of the initial response. Takahashi et al., (2001)[62] also reported that hyperosmotic stress induced a rapid and transient increase in inositol IP3 independent of abscisic acid in Arabidopsis cell culture.

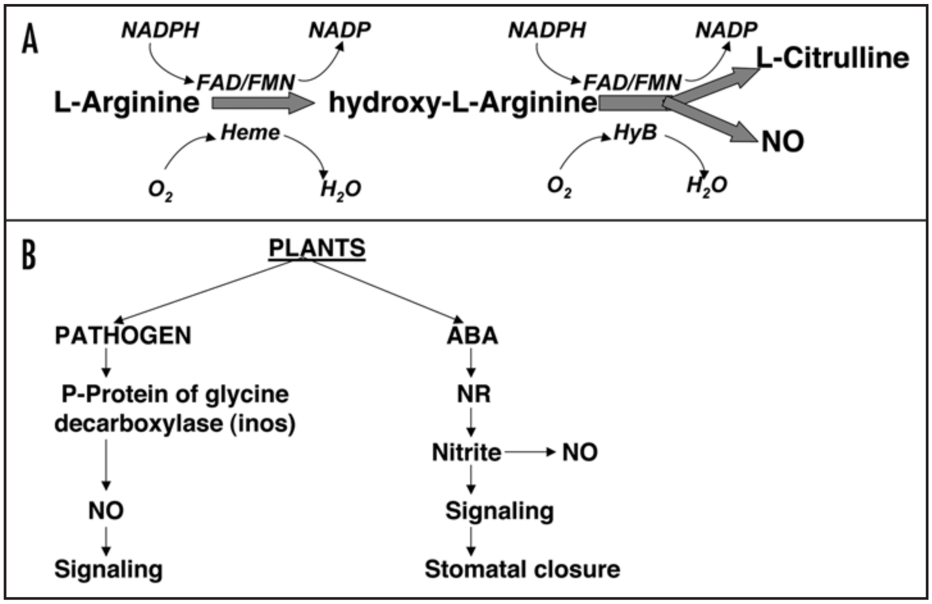

Figure 3. NO as a signaling molecule. The NO is synthesized from arginine (A). In plants the NO is synthesized by an enzyme that has homology with the p-protein of alanine decarboxylase in response to pathogen. It can also be synthesized by the activity of nitrate reductase as was shown in ABA mediated stomata closure response (B).

Though DAG has been shown to increase in response to exogenous stimuli, the downstream signal transduction pathways have not been well characterized in plants. In animal systems it activates protein kinases of C type. In plants there are only a few reports on the existence of PKC and its role has been shown in light and in stress responses.[63] As mentioned above DAG could also get converted to PA, which can then act as a signal molecule.

Proline accumulation is reported to occur in various plant organisms in response to environmental stresses. Recently role of PLC in proline accumulation was shown by the use of U73122, which inhibits PLC activity and it also inhibited the enzyme P5CS that is involved in proline accumuation.[64] These studies have indicated IP3 mediated calcium release may be important for stress mediated proline accumulation. Moreover, the utilization of diacylglycerol kinase and InsP(3)-gated calcium release receptor inhibitors suggested that InsP(3) or its derivatives are essential for proline accumulation upon salt stress, involving calcium as a second messenger in ionic stress signaling.[64,65] These results also demonstrated the specific involvement of lipid signaling pathway to discriminate between ionic and nonionic stresses.[64] Recently, Tang et al., (2007)[65] reported that in *Arabidopsis thaliana*, cytosolic Ca^{2+} oscillations are synchronized to extracellular Ca^{2+} concentration oscillations largely through the Ca^{2+}-sensing receptor CAS. CAS regulates concentrations of IP3, which in turn directs release of Ca^{2+} from internal stores. The oscillating amplitudes of cytosolic Ca^{2+} and extracellular Ca^{2+} are controlled by soil Ca^{2+} concentrations and transpiration rates.

Nitric Oxide Signaling

In 1867, amyl nitrate was used clinically by Brunton for the treatment of angina pectoris. Today we know that amyl nitrate is a compound that liberates nitric oxide (NO).[66] NO is an intra- and extracellular messenger that mediates diverse signaling pathways in target cells and is known to play important role in many physiological processes. An extensive work in the animal systems has led to the acceptance of NO as a component of second messenger cascades of hormone and physiological signals.[66] NO is synthesized as one of the product of a reaction in which L-arginine is metabolized to L-citrulline by nitric oxide synthase as shown in Figure 3A. In this reaction NADPH is used as an electron donor and O_2 as co-substrate. The cofactors FMN, FADH and tetrahydopterin are also required. There are at least three isoforms of nitric oxide synthase (NOS). Two of these are constitutive and one is inducible (iNOS or NOSII). Of the two constitutive forms is classified as neuronal (n NOS or NOSI) and the other as endothelial (eNOS or NOSIII). The constitutive NOS isoforms require calcium and calmodulin for their activity. The NOSI is a 160 kDa, NOSII a 135 kDa and NOSIII a 135–140 kDa protein.[66] From the N-terminal end lie the domains for binding to arginine, H4B, heme and calmodulin and from the C-terminal end binding sites for NADPH, FMN and FAD are present. As active NOS is a homodimer. Nitric oxide has been shown to modulate its action via stimulating proteins like ADP ribosyl transferase, cycloxygenases and more importantly via soluble guanylate cyclases. It can also inhibit the activity of lipoxygenase, H+ ATPase, ribonuceotide reductase, aconitase etc. In plants the presence and the activity of NOS has been reported in many systems by western blot analysis using antibodies against animal NOS and also by checking the conversion of labeled arginine to citrulline.[66]

NO is soluble in both aqueous and lipid media, but is relatively reactive and easily oxidized to other nitrogen oxides. It reacts with superoxide to form peroxynitrite, with other cellular components such as transition metals and haem-containing proteins and with thiol groups to form S-nitrosothiols. Thus, diffusion of NO within the plant may be relatively restricted. It is possible that NO is transported as chemical precursors such as nitrite or as nitrosothiols that might function as NO reservoirs. Studies have also revealed a role for NO as signaling molecule in plant, as it activates various defense genes and acts as developmental regulator.[66] It was however been shown that in plants nitrate reductase may be involved in generating NO by catalyzing NAD(P)H dependent reduction of nitrate to NO.[66] Infact this pathway of NO generation was found to be involved in ABA-induced stomatal closure (Fig. 3B). In detailed studies on pathogen signaling, it was found that after pathogen attack the machinery for NO synthesis, NOS or other NO-generating enzymes may be activated thus leading to an increase in NO which inturn could activate guanylate cyclase leading to an increase in cGMP. Details of downstream signaling targets, however, are not very well worked out.

As a developmental regulator, NO promotes germination, leaf extension and root growth, and delays leaf senescence and fruit maturation. Moreover, NO acts as a key signal in plant resistance to incompatible pathogens by triggering resistance-associated hypersensitive cell death. In addition, NO activates the expression of several defense genes (e.g., pathogenesis-related genes, phenylalanine ammonialyase, chalcone synthase) and could play a role in pathways leading to systemic acquired resistance.[67] A novel role for NO in the regulation of lateral root development in tomato has been reported which probably operates in the auxin signaling transduction pathway.[68] NO has been associated with plant defense responses during microbial attack, and with induction and/or regulation of programmed cell death.[69] The expression of genes in response to NO has also been shown to mediate via SA and JA signaling pathway.[70]

Recently, Ali et al., (2007)[71] showed that *Arabidopsis thaliana* cyclic nucleotide gated channel-2 (CNGC2/DND1) conducts Ca^{2+} into cells and provide a model linking this Ca^{2+} current to downstream NO production. Plants without functional CNGC2 lack this cell membrane Ca^{2+} current and do not display hypersensitive response (HR); providing the mutant with NO complements this phenotype. The bacterial pathogen-associated molecular pattern elicitor lipopolysaccharide activates a CNGC Ca^{2+} current, which may be linked to NO generation due to buildup of cytosolic Ca^{2+}/calmodulin.[67] Recently, it was found that NO is required for the production of lipid second messenger, PA, via activation of PLC and DAG in tomato cell cultures treated with xylanase, a fungal elicitor. PA was required for xylanase induced ROS production.[72] These results indicated that PLC/DAG kinase-derived PA represents a novel downstream component of NO signaling cascade during plant defense. This also shows cross talk between two different signaling pathways during plant defense. Graziano and Lamattina, (2007)[73] recently reported that NO accumulation is required for molecular and physiological responses to iron deficiency in tomato roots. NO action in plants also seems to be closely linked to SA and hence it could mediate many processes via SA signaling pathway. Infact biochemical, molecular and genetic studies reveal an interaction of NO, ROS, Ca^{2+}, SA, JA and ethylene in many biotic stress responses and also in developing cross tolerance in plants.[69-72]

NO is also known to play a role in regulation of stomatal closure. Little is known about the signalling events occurring downstream of NO. Previous studies have shown that NO modulates cytosolic calcium concentration and the activation of plasma membrane ion channels.[69-73] There is some evidence to show that calcium and MAPK can mediate NO signaling.[74] Zhang et al., (2007)[75] have found that NO and H_2O_2 regulate K/Na ratio in callus of Populus eupharatica under salt stress. Recently, Distefano et al., (2008)[76] have provided a evidence that supported the involvement of the lipid second messenger PA in NO signalling during stomatal closure.

Sugar Signaling

The production, utilization, mobilization and allocation of photosynthate in various tissues at different stages of development are highly regulated as these processes are required to meet the carbon and energy demands of the system. Sugars are essential to plant growth and metabolism, both as energy source and as structural components. Sugars such as sucrose serve dual functions as transported carbohydrates in vascular plants and as signal molecules that regulate gene expression and plant development.[77] Sugars affect the expression of many genes involved in photosynthesis, glycolysis, nitrogen metabolism, sucrose and starch metabolism, defence mechanisms and cell cycle regulation and therefore studies have been undertaken to find out sugar sensing and signal transduction pathways.[78] In many systems hexokinase 1 (HXK1) has been implicated to be an evolutionarily conserved glucose sensor that integrates nutrient and hormone signals to govern gene expression and plant growth in response to environmental cues.[79] However, the molecular mechanism through which HXK1 controls the expression of genes encoding proteins involved in photosynthesis is not well understood. Arabidopsis plants shows that there may be broadly two glucose signal transduction pathways: hexokinase dependent, and hexokinase independent pathway.[78] Recent research demonstrates that a previously unknown HXK1 nuclear complex controls the expression of specific photosynthetic genes, a process that is independent of glucose metabolism but requires two unexpected partners, VHA-B1 and RPT5B,[80] which suggested that metabolic enzymes can play unique roles in signal transduction by directly controlling gene expression in the nucleus. In plants the sugar signaling may also cross talk to other signaling pathways.

To understand the molecular mechanisms of sugar responses, Li et al., (2007)[81] have reported an *Arabidopsis thaliana* mutant, high sugar response8 (*hsr8*), which showed enhanced sugar-responsive growth and gene expression. These authors observed that light-grown *hsr8* plants exhibited increased starch and anthocyanin and reduced chlorophyll content in response to glucose treatment, while dark-grown *hsr8* seedlings showed glucose-hypersensitive hypocotyl elongation and development. Their further findings revealed a pathway that signals changes in the cell wall through PRL1 (pleiotropic regulatory locus1) to altered gene expression and sugar-responsive metabolic, growth and developmental changes.[81] The level and activity of sucrose synthase (SUS) enzyme was found to be high in sink organs but low in source organs. Recently, Qiu et al., (2007)[82] reported that light and metabolic signals regulating SUS protein degradation in maize (*Zea mays*) leaves during deetiolation. These findings suggested that SUS degradation is important to supply residues for the synthesis of other proteins required for autotrophic metabolism.

Starch is known to be the main carbohydrate store in plants. Regulation of starch metabolism, in particular in response to environmental cues, is of primary importance for carbon and energy flow in plants. Recently, Kempa et al., (2007)[83] provided an evidence that MsK4, a novel *Medicago sativa* Glycogen synthase kinase 3-like kinase, connects stress signalling with carbon metabolism. MsK4 was found to be a plastid-localized protein kinase that is associated with starch granules. High-salt stress rapidly induced the in vivo kinase activity of MsK4. Plants overexpressing *MsK4* showed improved tolerance to salt stress, which also accumulated significantly more starch and showed modified carbohydrate content compared with wild-type plants. These important finding suggested that MsK4 is an important regulator that adjusts carbohydrate metabolism to environmental stress.[83] Baena-Gonzalez et al., (2007)[84] have shown that protein kinases KIN10 and KIN11 are the central points in linking stress, sugar and developmental signals to regulate metabolism, energy balance, growth and survival under stress.

Signaling via Phytohormones and Growth Regulators

Phytohormones are critical for plant growth and development and play an important role in integrating various stress signals and controlling downstream stress responses. It is now well established that the stress-induced gene products are also involved in the generation of regulatory molecules like ABA. However, in recent years regulation and involvement of other hormones like cytokinins,[85] auxins,[86,87] ethylene[88] and brassinosteroids[89,90] have been indicated. There are indications that volatile organic compounds, which are involved in signaling in plant-insect interactions[91,92] may also be induced by exposure of plants to high light and high temperature conditions.[93] Thus in addition those regulators which were thought to be involved in only biotic signaling are now being shown to be involved in abiotic signaling too suggesting cross talk between the abiotic and biotic pathways. In this context here we described the role of ABA, SA, JA and polyamines, whose production under stress can initiate the second round of signaling.

Abscisic acid (ABA). It is known to be generated as a endogenous messenger during a plant's life cycle to control various physiological processes such as seed dormancy and delays its germination, development of seeds, promotion of stomatal closure, embryo morphogenesis, synthesis of storage proteins and lipids, organ senescence and also defense against pathogens.[9,94] Since various stresses induce ABA synthesis, therefore it is now considered as a plant stress hormone.[6-9,95] Plants have to adjust ABA levels constantly in responce to changing physiological and environmental conditions. The application of ABA to plant mimics the effect of a stress condition.[9] As many abiotic stresses ultimately results in desiccation of the cell and osmotic imbalance, there is an overlap in the expression pattern of stress genes after cold, drought, high salt or ABA application.[9] This suggests that various stress signals and ABA share common elements in the signaling pathway and these common elements cross-talk with each other, to maintain cellular homeostasis.[9] Various transcription factors are known to regulate the ABA-responsive gene expression.[6,9]

Main function of ABA seems to be the regulation of plant water balance and osmotic stress tolerance. Several ABA deficient mutants namely *aba1*, *aba2* and *aba3* have been reported for Arabidopsis.[96] ABA deficient mutants for tobacco, tomato and maize have also been reported.[95] Without any stress treatment the growth of these mutants is comparable to wild type plants. Under drought stress, ABA deficient mutants readily wilt and die if the stress persists. Under salt stress also ABA deficient mutants show poor growth.[97] In addition, ABA is required for freezing tolerance, which also involves the induction of dehydration tolerance genes.[97] Studies suggest that osmotic stress imposed by high salt or drought is transmitted through at least two pathways; one is ABA-dependent and the other ABA-independent. Cold exerts its effects on gene expression largely through an ABA-independent pathway.[9] The transcript accumulation of *RD29A* gene and the Proline accumulation in plants are reported to be regulated in both ABA-dependent and ABA-independent manner.[98] Auxin homeostasis is also known to be directly linked to growth regulation with stress through interactions with SA and ABA signals.[99] Several studies have demonstrated that ABA, drought, cold and high salt result in rapid increase in calcium levels in plant cells.[6-9,95] The salinity stress-induced upregulation of transcript of pea DNA helicase 45 (*PDH45*) followed ABA-dependent pathway,[100] while calcineurin B-like protein (*CBL*) and CBL-interacting protein kinase (*CIPK*) from pea followed the ABA-independent pathway.[101] Overall, the ABA-dependent pathways are involved essentially in osmotic stress gene expression. The role of ABA in drought tolerance in plant has been repored by Xiong et al., (2006).[102] Recently, Cao et al., (2007)[103] have reported that ABA and stress signals induce *Viviparous1* (*Vp1*) expression in seed and vegetative tissues of maize. The *Vp1* encodes a B3 domain-containing transcription factor that is a key regulator of seed maturation in maize.

It is now clear that to survive environmental stresses plants must respond to ABA. Though lot of work on the involvement and relation ship of this hormone to abiotic stress response, especially drought stress, has been done, and ABA-dependent *Cis* elements (ABRE) have been reported in many stress responsive genes, the receptor of this hormone was not found till recently. Razem et al., (2006)[104] have shown that an RNA binding protein, FCA, which also has role in flowering, is ABA receptor. This work should now reveal how under stress the flowering behaviour is altered in different plants. To obtain more information about ABA signaling network, Zhang et al., (2008)[105] carried out a genetic screen about ABA supersensitive mutant (*absg1* and *absg2*) on the basis of an estradiol-inducible activation-tagged Arabidopsis mutants and reported that the disturbance of small RNA pathways enhanced ABA response and multiple stress responses in Arabidopsis. This report provided more physiological evidences that linked small RNA pathway and ABA signaling.

Jasmonates (JA). Jasmonates are lipid-derived signaling molecules involved in induced systemic resistance, wounding and stress responses as well as in plant growth and development. Many of them alter gene expression positively or negatively in a regulatory network with synergistic and antagonistic effects in relation to other plant hormones such as salicylate, auxin, ethylene and abscisic acid.[106] It is reported in tobacco that, wounding causes rapid activation of two mitogen-activated protein kinases (MAPKs), wound-induced protein kinase (WIPK) and salicylic acid (SA)-induced protein kinase (SIPK), and the subsequent accumulation of jasmonic acid. It is found that activation of WIPK is required for the production of wound-induced JA. Recently, Seo et al., (2007)[107] suggested that WIPK and SIPK play an important role in JA production in response to wounding, and that they function cooperatively to control SA biosynthesis. In Arabidopsis the systemic immunity uses conserved defense signaling pathways, which is found to mediate by jasmonates.[108] These authors suggested that jasmonate signaling mediate long-distance information transmission. Moreover, the systemic transcriptional response shares extraordinary overlap with local herbivory and wounding responses, indicating that jasmonates may be pivotal to an evolutionarily conserved signaling network that decodes multiple abiotic and biotic stress signals.

Recentlt, Walia et al., (2007)[109] reported the jasmonic acid-mediated adaptation of barley to salinity stress. The JA-pre-treated salt-stressed plants accumulated low levels of Na^+ in the shoot tissue compared with untreated salt-stressed barley plants after several days of exposure to stress. Their study suggested that three JA-regulated genes, arginine decarboxylase, ribulose 1.5-bisphosphate carboxylase/oxygenase (Rubisco) activase and apoplastic invertase are possibly involved in salinity tolerance mediated by JA.

Salicylic acid (SA). It is also a plant signaling molecule and known to play a critical role in defense responses after pathogen attack. It antagonizes gene induction by the stress signaling molecule JA. Dat et al., (1998)[110] reported that SA can induced thermotolerance in mustard seedlings. Borsani et al., (2001)[111] showed an evidence for a role of SA in the oxidative damage generated by NaCl and osmotic stress in Arabidopsis seedlings. We have earlier reported that the CBL and CIPK genes from pea were induced in response to SA and wounding.[101] Several SA-responsive genes are known to regulate by basic/leucine zipper-type transcription factors of the TGA family. TGA factors interact with NPR1, a central regulator of many SA-induced defense responses including SA/JA antagonism. An *ATPase C* gene from Pennisetum was found to be upregulated by salicylic acid and its promoter was found to bind nuclear factors to TGA cis elements.[112] Recently, Ndamukong et al., (2007)[113] reported that glutaredoxin acts as regulatory protein of SA-dependent signaling pathways. Koo et al., (2007)[114] reported that overexpression of SA carboxyl methyltransferase reduces SA-mediated pathogen resistance in *Arabidopsis thaliana*. Their results indicated that in the absence of SA, methyl salicylate alone cannot induce a defense response, yet it serves as an airborne signal for plant-to-plant communication. Recently, Wang et al., (2007)[115] reported that SA inhibits pathogen growth in plants through repression of the auxin signaling pathway.

Polyamines. Polyamines, a group of small aliphatic amines, are reported to play role in plant development. These are also known to accumulate under salt stress conditions in different plant systems, resulting in presumed protective effects, acting as free radical scavengers, stabilizing cellular membranes and maintaining cellular ionic balance under these conditions. Jiménez-Bremont et al., (2007)[116] reported that in response to long-term salt stress the levels spermidine and spermine were modulated suggesting their role in salt stress. These authors also suggested that there is a connection between polyamine metabolism, abiotic stress and abscisic acid. Polyamines role in other stress responses has also been shown.[117,118] It was found by Yamaguchi et al., (2006)[119] that an arabidopsis double knock out plant that cannot produce spermine showed higher sensitivity to high salt. This mutant was also later found to be sensitive to drought and the mutant phenotype could be cured by the addition of spermine but not by spermidine.[120] The genes involved in spermine and spermidine synthesis have also been found to be regulated by salinity and ABA.[116] By using patch clamp techniques to protoplasts, it was found that polyamines affect K^+/Na^+ homeostasis.[121] This could be one of the mechanism by which polyamines could help in salinity tolerance in plants. Regulation of polyamines biosynthesis has also been reported in plants under drought stress. In cacao, expression pattern of five genes encoding enzymes involved in polyamines biosynthesis were studied in response to drought and correlated with the levels of putresicine, spermine and spermidine.[122] In many studies overexpression of these genes has also been shown to confer stress tolerance in plants.[118] There is evidence of cross-talk of polyamine with NO. Exogenous addition of polyamine has been shown to induce the production of NO although the mechanism for this is still not understood and needs more experiments.[123]

Conclusion and Perspectives

The essential feature of plant survival to changing environmental conditions is their ability to continuously monitor fluctuations in light conditions, temperature variations, water and nutrient availability, CO_2 levels etc. The sensitivity of the sensors to evaluate the intensity and limits of variation lead to the induction of physiological defense mechanisms. In this review we have dealt with the nature of chemicals which are produced following perception of stress environment and carry the message in the cell to respond either directly of indirectly by regulating the expression of genes leading to altered functions or enhance the capacity of the already existing defense metabolites. These chemicals remain in a state of flux; their levels are increased or decreased according to the demand put on plants by the intensity and the nature of the abiotic stress. Thus abiotic stress signaling is an important area with respect to increase in crops yield under sub-optimal conditions.

During the last few years the information on the nature of chemicals and the signaling pathways that they are involved has been generated. Earlier, it was thought that calcium, phosphoinositides and ABA were the key molecules that participated in abitoic stress signaling. Recently, calcium signaling is also reported to involved in the regulation of plant cell cycle progression in response to abiotic stress.[124] However, as it has been described in this review chemicals like SA, JA, NO, which were thought to respond to biotic stresses, have now been implicated in abiotic stresses also. The role of other molecules like simple sugars and polyamines have also been elucidated which has been covered in this review. It seems that there may be many others, which may also be playing the role as signal molecules. Lately, chemicals like glutathione and trehalose-6-phosphate, which are involved as antioxidant and osmolyte, have also been shown to act as signaling molecules and induce the expression of genes.[125,126] Genetic analyses have revealed extensive interactions between sugar and plant hormone signaling. Important and complex roles for Snf1-related kinases (SnRKs), extracellular sugar sensors and trehalose metabolism in plant sugar signaling are now also emerging.[78]

In future more work should be done to find out their exact role. An effort should also be taken up to find out the function of brassinosteroids in more details. Cellular perception of NO may occur through its reaction with biologically active molecules that could function as 'NO-sensors'. But exactly how NO evolution relates to its bioactivity in planta remains to be established.[127] We envisage that more molecules will be discovered that play an important role in signal transduction under abiotic stresses. The overall progress of research on chemical regulated stress responsive genes and their products reflect their central role in plant growth and development under stress conditions. The mechanisms, by which the plants maintain their physiology and development under abiotic stress using different chemical signals at different times and in response to different stress conditions, and cross talk between each of them, still remained to be fully understood. Overall, it is becoming clear that chemicals action enforces a sophisticated regulation at all levels.

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