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PLANTS SENSORY-RESPONSE MECHANISMS FOR SALINITY AND HEAT STRESS

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ABSTRACT

Plants, being sessile in nature, have evolved efficient sensory mechanisms to cope with the dynamic environment. Their survival depends on a network of robust and densely interconnected systems for rapid signal perception, transduction, reaction and adaptation to biotic and abiotic stimuli. Among the abiotic stresses, heat and salt stress are the major threats to modern agriculture especially in the scenario of climate change. At cellular level, signal perception and response is critical for the establishment of stress tolerance. This review gives an insight of sensory and response mechanisms induced by salt and heat stress. Saline conditions activate membrane bound sensors including ion transporters followed by activation of second messengers and signaling cascades (like TFs, MAPKs, SnRK2s and SOS pathways) for the activation of response mechanisms. Heat stress affects membrane properties and initiate sensory events followed by activation of response mechanisms (like TFs, HSFs, chaperones and osmo-protectants). Such observations emphasize the importance of signal messengers as well as transcription factors for cellular performance of plants under confrontational circumstances. Better understanding of these stress-sensory mechanisms can facilitate the breeding programs for the development of climate-resilient plants which may perform better in a challenging environment and can contribute to the global food security.

Keywords: Salinity, Heat, sensory mechanisms, stress response, climate change.

INTRODUCTION

Plant survival depends on a network of robust and densely interconnected systems for rapid signal perception, reaction and adaptation to suboptimal environmental conditions. Under stress conditions, plants are unable to show complete genetic potential, which negatively affects plant growth, development and agronomic yield. But to feed the fast growing population, there is a need for almost 70% sustainable increase in major food crop production (FAOSTAT, 2009). This increase could be achieved by overcoming the yield losses due to environmental stresses without increasing input demand. Abiotic stresses are responsible for more than 50% of the primary crop yield loss (Albacete *et al.*, 2014) and deterioration of agricultural lands. Among major abiotic stresses, salinity and heat stress play a predominant role in limiting crop yield.

Soil salinity is a serious problem for plants. It affects plants by disturbing the water extraction capacity of roots and continuous uptake of salts that becomes toxic for plant. Primarily, the salt stress affects seed germination, plant growth, photosynthesis, water relation, nutrient balance, plant yield and may lead to even death of the plant (Munns and Tester, 2008; Yadav *et al.*, 2011; Arefian *et al.*, 2014; Parihar *et al.*, 2015). Plants have developed various physiological, biochemical and molecular mechanisms to survive on saline soils (Gupta and Huang, 2014).

Heat stress is another major abiotic constraint for crop productivity. According to predictions, at the end of 21st century temperature of the growing season in the tropics and subtropics will surpass the highest seasonal temperature observed during 1900-2006 (Battisti and Naylor, 2009). Global warming has profound and diverse effects on plants. During 1980–2008 high temperature has decreased world maize and wheat production by 3.8 and 5.5%, respectively (Lobell and Field, 2007; Lobell *et al.*, 2011). Worldwide yields of the six most important crops (wheat, rice, maize, soybeans, barley, and sorghum) have declined by 0.6~8.9% for every 1°C rise in temperature (Lobell and Field, 2007). Climate change models have forecasted that there will be more frequent and intense heat waves in the future, causing a gradual increase in ambient temperature and it will be a major threat for agricultural production (Suzuki *et al.*, 2014). High temperature interrupts plant growth, development and physiological processes, thus contributing significantly to yield reduction. Major consequences of heat stress include production of reactive oxygen species (ROS) and disturbance in the photosynthetic activities of plants (Davies *et al.*, 2006; Hasanuzzaman *et al.*, 2013)

Therefore, it is critical to understand how plants feel the heat and adapt protective measures for survival.

In the open environment, plants are simultaneously exposed to multiple stresses. Generally, plants have to respond to combinations of two or more stresses. Recent findings suggest that plants exhibit unique responses under such conditions (Zhang *et al.*, 2015; Sham *et al.*, 2015; Saucedo-GarcÃ- a*et al.*, 2015; Nigam *et al.*, 2015; Krasensky and Jonak, 2012). Sometimes, plant responses to a combination of stresses become even more complex. It is because, the responses to the collective stresses are mostly controlled by diverse, and occasionally contrasting, signaling pathways that may have negative effects on each other. Therefore, it is really important to decipher the mechanisms that execute the integration of stress perception, signal transduction and plant responses.

Current review is focused on mechanisms of plants for sensing and response to two major environmental constraints: salt and heat stress.

Sensing the stress stimulus: In general, signal transduction pathway includes signal perception, activation of second messengers, modulation of intracellular Ca^{+2} levels and triggering a protein phosphorylation cascade. Depending upon the nature of stimulus, either one or both classes of following proteins are activated: proteins directly involved in cellular protection or transcription factors that modulate stress-regulated genes.

In the stress signaling cascade certain second messengers also act as sensor for the detection of stress signal. Among different classes of Ca^{2+} sensors calmodulin (CaM), calcineurin B-like proteins (CBLs) and CBL-interacting protein kinases (CIPKs), are the best characterized (Reddy *et al.*, 2011; Yu *et al.*, 2014). These transducers of Ca^{2+} signaling are also involved in stress signal sensing. This is because the sensing and the signaling events of calcium and other ions like Na^{+}, are very closely interconnected and are very difficult to be dissociated as the sensing event immediately transmits signal (DeFalco *et al.*, 2010; Conde *et al.*, 2011).

Other second messengers include cyclic nucleotides cAMP/cGMP (Walker and Berkowitz, 2013), Polyphosphoinositides (Munnik and Vermeer, 2010), nitric oxide (NO) (Agurla *et al.*, 2014), sugars (Rolland *et al.*, 2002), abscisic acid (ABA) (Raghavendra *et al.*, 2010) and jasmonates (JA) (Wasternack and Hause, 2013).

Sensing salt stress stimuli: Plants can sense and exhibit an effective response against hyperosmotic and ionic components of salt stress (Munns and Tester, 2008). The Arabidopsis Histidine Kinase receptor protein (HK1) is an important candidate for osmo-sensors because it can balance the loss of the yeast osmo-sensor Sln1 and overexpression/loss-of function plants exhibit drought and osmotic stress-associated phenotypes (Urao *et al.*, 1999; Tran *et al.*, 2007; Wohlbach *et al.*, 2008; Kumar *et al.*, 2013; Kushwaha *et al.*, 2013). Some of the physiological activities induced by osmotic stress were altered in hk1 mutants, but others remained unaffected. Therefore, other proteins must still perceive the osmotic stress in the hk1 mutant (Kumar *et al.*, 2013). It is assumed that hyperosmolality and other environmental stimuli increase cytosolic free calcium concentration ($[Ca^{2+}]$) and osmosensing Ca^{2+} channels in animals and bacteria function as osmosensors (Kung, 2005; Arnadóttir and Chalfie, 2010). In *Arabidopsis thaliana*, hyperosmolality gated calcium-permeable channel (OSCA1) is important for osmosensing (Yuan *et al.*, 2014). Similarly, a plasma membrane localized receptor-like protein (NtC7) functions in osmotic adjustment and its transcripts are upregulated rapidly after salt, osmotic and wounding stress (Tamura *et al.*, 2003).

Membrane embedded Mechanosensitive channels (MS) are important for plant osmosensing machinery (Peyronnet *et al.*, 2014). These channels serve as sensors and effectors of physical responses. The most extensively characterized MS channels belong to the MscL (MS channel Large conductance) or MscS (Small conductance) families (Hamill and Martinac, 2001; Martinac *et al.*, 2008). Upon membrane stretching in response to environmental stimuli, MS channels are immediately activated along with cellular ion fluxes and ROS variations (Monshausen and Gilroy, 2009; Coutand, 2010). Therefore, it is speculated that MS channels, are involved either directly or indirectly in modulating ion fluxes, especially Ca^{2+} (Peyronnet *et al.*, 2014). As the cytosolic Ca^{2+} increase is a hallmark of osmotic stress, MS channels are thought to play role in osmosensing and subsequent responses (Figure 1).

Salinity and hyperosmotic exposure trigger several early signaling events including ROS formation (Miller *et al.*, 2010; Jiang *et al.*, 2013), the induction of PLD-mediated phosphatidic acid production (Bargmann *et al.*, 2009; Darwish *et al.*, 2009; Yu *et al.*, 2010), accumulation of nitric oxide (Khan *et al.*, 2012), formation of cAMP/cGMP (Donaldson *et al.*, 2004), synthesis of abscisic acid (ABA) (Dinneny, 2014) and transient increases in cytosolic Ca^{2+} concentrations (Laohavisit *et al.*, 2012; Laohavisit *et al.*, 2013). Arabidopsis annexins mediate both NaCl and ROS-induced Ca^{+2} responses in roots (Laohavisit *et al.*, 2012; Laohavisit *et al.*, 2013; Richards *et al.*, 2014). Although the rapid Ca^{2+} increase is an important response to osmotic stress but Ca^{2+}-independent osmotic sensory mechanisms may also be present. Genetic identification of osmotic and Na^{+} sensors will be very helpful to know about these early sensory mechanisms.

Na^{+} imparts the ionic component of salt stress. Extracellular Na^{+} is thought to be detected by a membrane receptor while intracellular Na^{+} is expected to be detected either by membrane proteins or by one of the Na^+-sensitive enzymes in the cytoplasm (Zhu, 2003; Maathuis *et al.*, 2014; Maathuis, 2014). The molecular nature of Na^+ sensors is still unclear, the plasma membrane Na^+/H^+ antiporter SOS1 (Salt Overly Sensitive1) is a possible candidate because its cytoplasmic end is assumed to be involved in Na^+ sensing (Zhu, 2003).

Under normal conditions, plant cells sustain a high level of K^+/Na^+ in the cytosol and such control is mediated by a tight regulation of ion flux. It involves primary active transport and secondary transport mediated by H^+-ATPases and co-transporters/channels, respectively (Hauser and Horie, 2010). A rise of Na^+ in root zone promotes the entry of Na^+ ions inside the plant body either through plasma-membrane non-selective cation channels (NSCCs), HKT transporters, or via anatomical ‘leaks’ in the root endodermis (Demidchik and Tester, 2002; Munns and Tester, 2008; Hamamoto *et al.*, 2015).

Sensing high temperature stress stimulus: Plants can detect temperature changes as small as 1°C (Penfield, 2008) and activate response mechanisms (Saidi *et al.*, 2011; Conde *et al.*, 2011; Grover *et al.*, 2013; Cerný *et al.*, 2014; Gao *et al.*, 2014; Liu *et al.*, 2015). Despite a number of dedicated efforts, still it is difficult to nominate the primary heat sensor(s). Plasma membrane Cyclic Nucleotide Gated Calcium Channels (CNGCs) (Younousse Saidi *et al.*, 2009; Finka *et al.*, 2012), two unfolded protein sensors, ER-UPR and Cyt-UPR (Sugio *et al.*, 2009; Deng *et al.*, 2011) and a histone variant (H2A.Z) containing nucleosomes (Kumar and Wigge, 2010) are considered putative heat sensors. Heat stress activates CNGCs, ER-UPR, and Cyt-UPR, and triggers signaling through multiple kinases as well as transcriptional regulators of the HSR, such as HSFs, MBF1c, and Rboh. Protein translation capacity of chloroplasts generates the retrograde signals to activate HsfA2-dependent heat-responsive genes in the nucleus. Thus, the chloroplasts are proposed as heat sensors as well (Liu *et al.*, 2015).

Plasma membrane is also considered as an important sensor for heat stress (Figure 2). It perceives temperature changes and transduces them into the nucleus where the transcriptome is rehabilitated (Saidi *et al.*, 2009; Conde *et al.*, 2011). Membrane lipid composition plays a key role in regulating calcium dependent heat-signaling pathway (Saidi *et al.*, 2010). Elevation in external temperature brings about the activation of Ca^{2+} channels in plasma membrane causing the Ca^{2+} influx inside the plant cells. Moreover, heat stress leads to the alteration in properties of plasma membrane (Hofmann, 2009), which signals the activation of heat responsive (HR) regulatory networks. Other metabolites such as nitric oxide (NO) and hydrogen peroxide (H_2O_2) are also produced in response to heat stress and probably activate similar molecular mechanisms for improving the heat stress tolerance of plant (Hua, 2009; Saidi *et al.*, 2011).

Response to stress

Salt stress response: When $[Na^+]_{cyt}$ surpasses a threshold, response mechanisms are initiated (Figure 1), which involve vacuolar sequestration and/or extrusion of Na^+ to the apoplast and a reduction in Na^+ conductance of membranes (Maathuis, 2014). Once an osmotic or ionic stress component is sensed by plants, a signaling cascade involving different second messengers is initiated followed by activation of downstream kinases, calcium-dependent protein kinases (CDPKs) and calcineurin B-like proteins (CBLs) with CBL-interacting protein kinases (CIPKs) (Weinl and Kudla, 2009; Boudsocq and Sheen, 2013). It may transduce the signal to downstream transcription factors including Calmodulin Binding Transcription Activators (CAMTAs) (Pandey *et al.*, 2013), GT-element-binding-like proteins (GTLs) (Weng *et al.*, 2012) and MYBs (Yoo *et al.*, 2005; Xie *et al.*, 2014). Although a prompt variation of $Ca2^+$ is a hallmark response to osmotic stress, there may also exist Ca^{2+}- independent osmotic sensory mechanisms. Genetic identification of osmotic and Na^+ sensors is likely to be instrumental in resolving these early sensory mechanisms (Figure 1).

During stress conditions, a family of sensor transmitters, Calcineurin B-like proteins (CBL) and their respective kinase effectors, CBL-interacting protein kinases (CIPK), together make a primary element in signaling cascade responsible for Ca^{2+} signaling and is named as CIPK/CBL pathway (Yu *et al.*, 2014). The CIPK-CBL complex regulates downstream targets like ion channels and transporters (Manik *et al.*, 2015) and play significant role against various abiotic stresses (Luan *et al.*, 2009). Opening of Ca^{2+} channels due to abiotic stress signals (Figure 3) causes a rapid influx of Ca^{2+} ions, that starts a cascade of stress signaling (White, 2000; Sanders *et al.*, 2002). The Ca^{2+} signal stimulates the binding of calcineurin B-like protein, CBL, with Ca^{2+} leading to the formation of CBL-CIPK-type kinases complex which is responsible for decoding the Ca^{2+} signals (Zhu *et al.*, 2013). The interaction occurs when a conserved serine residue within the C terminus of CBLs gets phosphorylated by the interacting CIPKs which interact with CBL via CIPK-NAF domain (Hashimoto *et al.*, 2012). This interaction then leads to the phosphorylation of downstream target proteins which leads to the development of stress tolerance.

An important adoption of plants for salinity tolerance is the extrusion of Na^+ ions from plant body, which is brought about by SOS signaling pathway (Zhu *et al.*, 1998; Ji *et al.*, 2013). Salt stress induces the increase in cytosolic Ca^{2+}. A calcium sensor SOS3 binds with Ca^{2+} and activates a serine/threonine protein kinase SOS2 of the SnRK3 family of protein kinases (Zhu *et al.*, 1998; Halfter *et al.*, 2000; Liu *et al.*, 2000; Hrabak *et al.*, 2003; Zhu, 2003). The SOS3 & SOS2 complex activates plasma membrane-localized Na^+/H^+ antiporter SOS1. This complex is responsible for a significant extrusion of Na^+ ions from the cytosol (Shi *et al.*, 2000; Qiu *et al.*, 2002; Quintero *et al.*, 2002; Zhu, 2003; Quan *et al.*, 2007; Quintero *et al.*, 2011). This signaling cascade is not as simple as it was thought to be. The activation of SOS1 does not completely depend on SOS3–SOS2 complex (Shabala *et al.*, 2005). Thus, the regulation of salt stress and extrusion of Na^+ ions by SOS pathway is far more complex process and is not the only pathway responsible for maintaining ion-homeostasis and extruding excess Na^+ ions from plant body.

Plants also compartmentalize Na^+ ions to prevent their excess build-up in the cell. During salt stress, compartmentalization of Na^+ ions is crucial for plant growth. Na^+ ions enter the cytoplasm and are then transported to the vacuole via Na^+/H^+ antiporter (Gupta and Huang, 2014). Accumulation of Na^+ ions in vacuoles lead to increased osmotic adjustment and increased sequestration of Na^+ ions in root and leaf vacuoles (Munns and Tester, 2008).

Reduced rate of photosynthesis resulting from salinity induced oxidative stress, leads to the increased formation of reactive oxygen species (ROS), which are rapidly detoxified by various cellular enzymes (Apel and Hirt, 2004; Foyer and Noctor, 2005; Bose *et al.*, 2014). RSA1, a nuclear-localized calcium-binding protein, its interacting partner, RITF1, and a transcription factor bHLH are involved in the regulation of salt stress by hunting reactive oxygen species (ROS) and regulating *SOS1* gene (Guan *et al.*, 2013).

One of the major and most important responses of plants to environmental stresses is the biosynthesis of ABA which activates a number of genes involved in respective stress response (Dong *et al.*, 2015) and on the other hand, such responses could follow ABA-dependent or ABA-independent pathways (Agarwal and Jha, 2010). Under stress conditions, ABA responsive genes get activated. The activation of these genes, results from the binding of ABA induced TFs to their respective binding sites/cis-regulatory elements in the promoter region of these genes. The ABA induced TFs include AREB, DREB2A, DREB 2B, MYB/MYC, bZIP & RD22BP1 (Zhu, 2002; Yoshida *et al.*, 2014; E *et al.*, 2014). In response to salt stress, the basic leucine zipper transcription factor called AREB binds to ABRE elements leading to the activation of RD29B stress responsive gene, which is involved in providing tolerance against salinity. Two of these ABRE motifs have been reported in Arabidopsis, which are involved in the regulation of LEA-like (late embryogenesis abundant) protein encoding RD29B gene (Narusaka *et al.*, 2003). The TFs, DREB2A and DREB2B, are drought inducible and their expression is regulated by ABA dependent pathways. These genes are responsible for the activation of RD29A stress responsive gene, leading to the maintenance of osmotic equilibrium during drought stress. The MYB/MYC TFs get induced at the late stages of stress response. RD2BP1 and MYB/MYC TFs cause the activation of RD22 gene in response to stress (Tuteja and Sopory, 2008).

Heat stress response: Heat stress differently affects the stability of several membranes, proteins, cytoskeleton and RNA structures. Cytoskeleton de-polymerization, protein conformation, metabolic reactions and membrane fluidity are regarded as the temperature sensitive processes (Ruelland and Zachowski, 2010). Moreover, high temperature negatively affects the efficiency of enzymatic reactions, which disturbs the major physiological and metabolic processes (Hasanuzzaman *et al.*, 2013).

Plants have developed several physiological and molecular pathways to cope with heat stress (Figure 2). Plant response to heat stress is regulated both by genetic and epigenetic mechanisms (Qu *et al.*, 2013; Sanchez and Paszkowski, 2014; Liu *et al.*, 2015). Heat stress-responsive genes and proteins in plants include protein kinases, transcription factors, heat shock proteins (HSPs) and catalases (Qu *et al.*, 2013). Moreover, plants might regulate the heat stress response at the post-transcriptional level by inducing a global mRNA decay process (Merret *et al.*, 2013).

Various secondary messengers; nitric oxide (NO), hydrogen peroxide (H_2O_2), Ca^{2+} ions and plasma membrane are basic elements of heat stress signaling pathways (Bolwell, 1999; Durner and Klessig, 1999; Smékalová *et al.*, 2014; Pandey *et al.*, 2015). The downstream components; mitogen-activated protein kinases, Hsp90 and calmodulins are involved in the activation of Heat-shock transcription factors (Hsf). Heat signal transduction induced through different pathways causes activation of Hsfs, the expression of Hsps and the onset of thermos-tolerance (Saidi *et al.*, 2011).

The expression of Hsfs depends on the intensity of stress. Upon very high temperature overexpression of Hsf causes the activation of all the heat stress responsive genes. There are multiple copies of Hsfs in plants like 17 in tomato, 19 in rice (Baniwal *et al.*, 2004) and 21 in Arabidopsis (Nover *et al.*, 2001). These genes are grouped into three classes A, B and C, depending on the differences in the protein structural features like flexible linkers and oligomerization domains (Nover *et al.*, 2001; Baniwal *et al.*, 2004). Each Hsp subfamily has a different mechanism of action, some of them cause the degradation of misfolded proteins, some prevent the aggregation of Hsps by binding to different intermediates of folding (Hsp70 and Hsp60) and Hsp100 re-activates the already aggregated proteins. HsfA8 functions as redox sensing transcription factor within the stress-responsive transcriptional network (Giesguth *et al.*, 2015). These proteins are specific in expression and are expressed at certain developmental stages of plant like seed germination, embryogenesis, microsporogenesis and fruit maturation (Prasinos *et al.*, 2005).

Previous studies on Arabidopsis concluded that Arabidopsis mitogen-activated protein kinase MPK6 become activated under heat stress (Sinha *et al.*, 2011). MPK6 specifically activates HsfA2 (Evrard *et al.*, 2013) followed by activation of MAPKs. MAPK-dependent responses to heat stress in alfalfa and tomato plants have previously been described (Link *et al.*, 2002; Sangwan *et al.*, 2002). ZmMAPK1 overexpressing *A. thaliana* plants respond better to drought and heat stress (Wu *et al.*, 2015).

Heat stress response mechanisms have also shown correlation with epigenetic gene silencing in Arabidopsis. This has been confirmed by inducing mutations in gene silencing pathways. The mutated plants showed hypersensitivity to heat stress. The response to temperature stress at transcription level co-ordinates with RNA-dependent DNA methylation pathway for the induction of resistance (Popova *et al.*, 2013).

A significant fraction of the HSPs seems to be co-regulated by cytokinin. It is argued that temperature and cytokinin signaling pathways are interconnected through Ca^{2+} ions fluxes. Furthermore, the response of proteome to cytokinin treatment at low and standard temperatures partially minimizes the effects of heat shocks on it. Therefore, cytokinin may directly contribute in heat signaling pathway. Finally, most of the heat shock and cytokinin responsive proteins are present together in the chloroplast. (Cerný *et al.*, 2014)

Besides Hsps, there are certain other genes as well, found to be up regulated by heat stress. Arabidopsis cytosolic ascorbate peroxidase gene (APX1) is up regulated by heat stress. A functional heat shock element (HSE) is located in its 5'-promoter region (Storozhenko *et al.*, 1998). Under stressful conditions plants accumulate H_2O_2 in their cytosol, which may lead to cell death. The accumulation of toxic H_2O_2 is prevented by the expression of APX1 which is a major hydrogen peroxide-scavenging enzyme in the cytosol (Mittler *et al.*, 2004). In response to a combined heat and drought stress treatment, APX1-deficient mutant (apx1), accumulated more hydrogen peroxide in the cytosol and was considerably more sensitive to the stress combination than wild type. This shows that APX1 plays a key role in providing thermo-tolerance to plants (Koussevitzky *et al.*, 2008).

In *Arabidopis thaliana* MBF1c (multi-protein binding factor 1c) plays a key role in the regulation of thermo-tolerance. MFB1c interacts with heat inducible TPS5 (trehalose phosphate synthase 5) to function during heat stress and the mutants deficient for TPS5 are sensitive to high temperature (Suzuki *et al.*, 2008). Along with MFB1c, AtSAP5, a nuclear localized protein, affects cellular responses to abiotic stresses i.e. water deficiency, salinity, osmotic imbalance and heat stress (Kang *et al.*, 2011).

Li *et al*. (2011) categorized the function of WRKY33, WRKY26, and WRKY25 as effective controllers of response against heat stress. This categorization was based on the analysis of both their loss-of-function and over-expression phenotypes, and potential upstream and downstream targets of these genes. It is suggested that these three regulators exhibit fractional redundancy in thermo-tolerance and help in coordination between Hsps-related signaling and MBF1c (ethylene)-activated pathways that facilitate responses to heat stress. Similarly, members of DREB family are also important for plant response to heat stress (Qu *et al.*, 2013). There are also other genes involved in Hsp independent pathways. AtPARK13 is a heat stress-induced protease which is localized in mitochondria. It confers thermo-tolerance in Arabidopsis and degrades miss-folded proteins (Basak *et al.*, 2014).

Cross-talk of salt and heat stress response in plants: Heat and salinity stress combination may affect agronomic traits of plants, either positively or negatively (Suzuki *et al.*, 2014) and direct rearrangements in metabolic and regulatory networks (Krasensky and Jonak, 2012). Therefore, an understanding of such mechanisms could be fruitful in developing multiple stress tolerant plants. To fight with these types of abiotic stresses, plants modulate their physiological and biochemical pathways. Like, salt stress causes the activation of pathways which are aimed at detoxifying the ROS and maintaining the ion-homeostasis. In the same way heat stress induces the metabolic pathways which employ heat shock proteins for the development of thermo-tolerance.

Correlation among different regulatory networks is referred to as cross talk. Studies have revealed the existence of cross-talk among the heat and non-heat stress regulatory pathways, where Hsfs act as intersecting points in response to various types of stresses including thermal and non-thermal stresses like drought, salt, cold and heat stresses. Genome-wide transcriptional profiling led to the finding that unlike other stresses, osmotic, cold, and salt treatments induced a strong Hsps expression .The degree of crosstalk varies depending on the intensity of stress (Swindell *et al.*, 2007). Among all the four abiotic stresses stated before, the highest induction of Hsp100 is caused by both heat and salt stresses. Hsf and Hsps gene families have shown to be widely induced by heat, drought and salt treatments, whereas, Hsp100, Hsp90 and Hsp70 families are more specific in response to stresses (Hu *et al.*, 2009). Moreover, plant response to a combination of heat and drought stress could be different from individual drought or heat stress. This is because of the two correlated multi-gene defense pathways as well as specific expression of 454 transcripts during combination of heat and drought stress (Rizhsky *et al.*, 2004). AtHsfA6a works as a transcriptional activator of stress-responsive genes following the ABA-dependent signaling system. Moreover, plants over-expressing AtHsfA6a were highly sensitive to ABA and showed increased resistance against drought and salt stresses (Hwang *et al.*, 2014). DREB2A is involved in the upregulation of heat and salinity stress responsive genes (Sakuma *et al.*, 2006). In response to multiple stresses some proteins work together and activate genes coding for regulators as miRNAs and TFs (Nigam *et al.*, 2015). These regulatory genes are responsible for downstream target genes regulation for providing stress resistance. It is very important to understand the relationship between regulators and target genes *in vivo.*

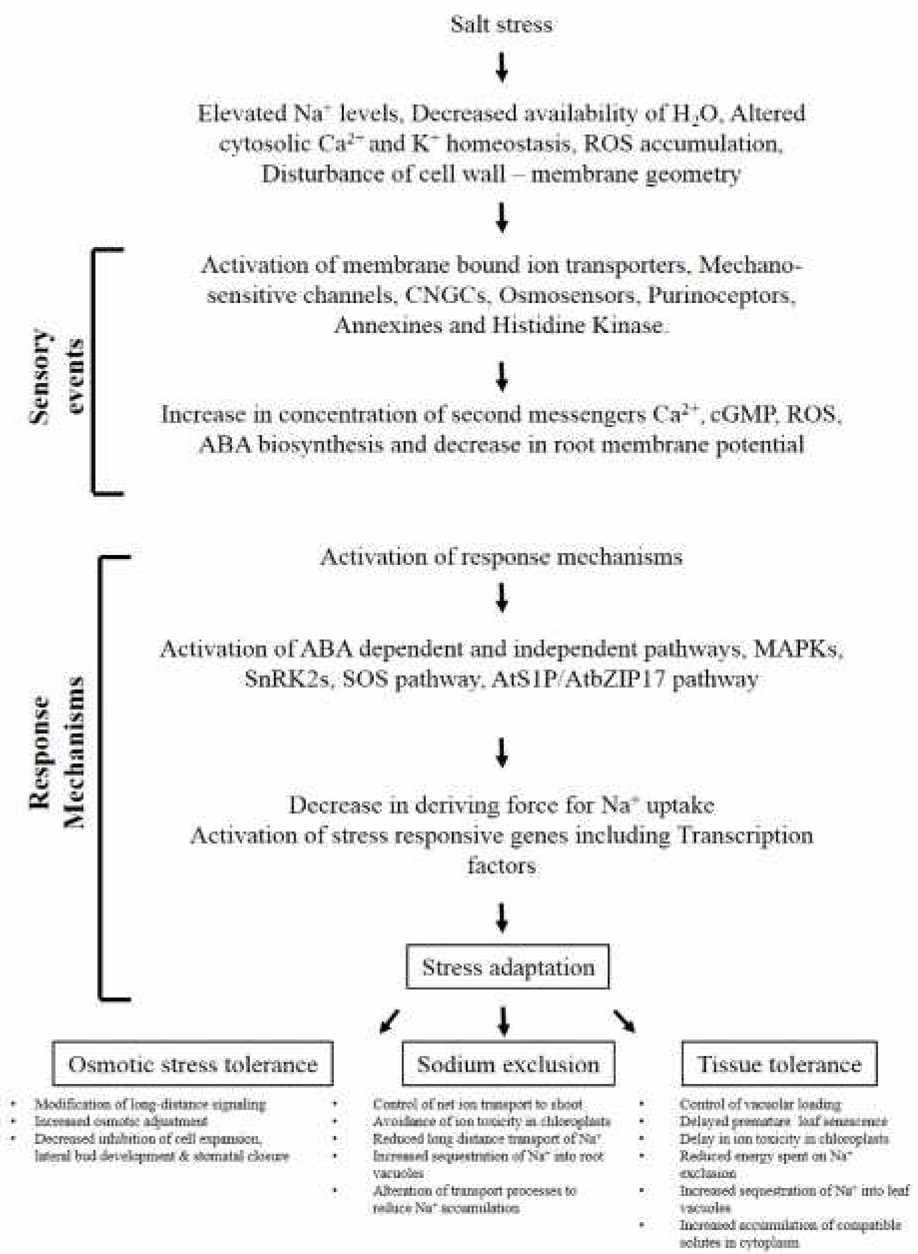

Figure 1. A general overview of different sensory-response events involved in plant response to salinity stress. Partly adopted from Munns and Tester (2008) and Conde *et al*. (2011).

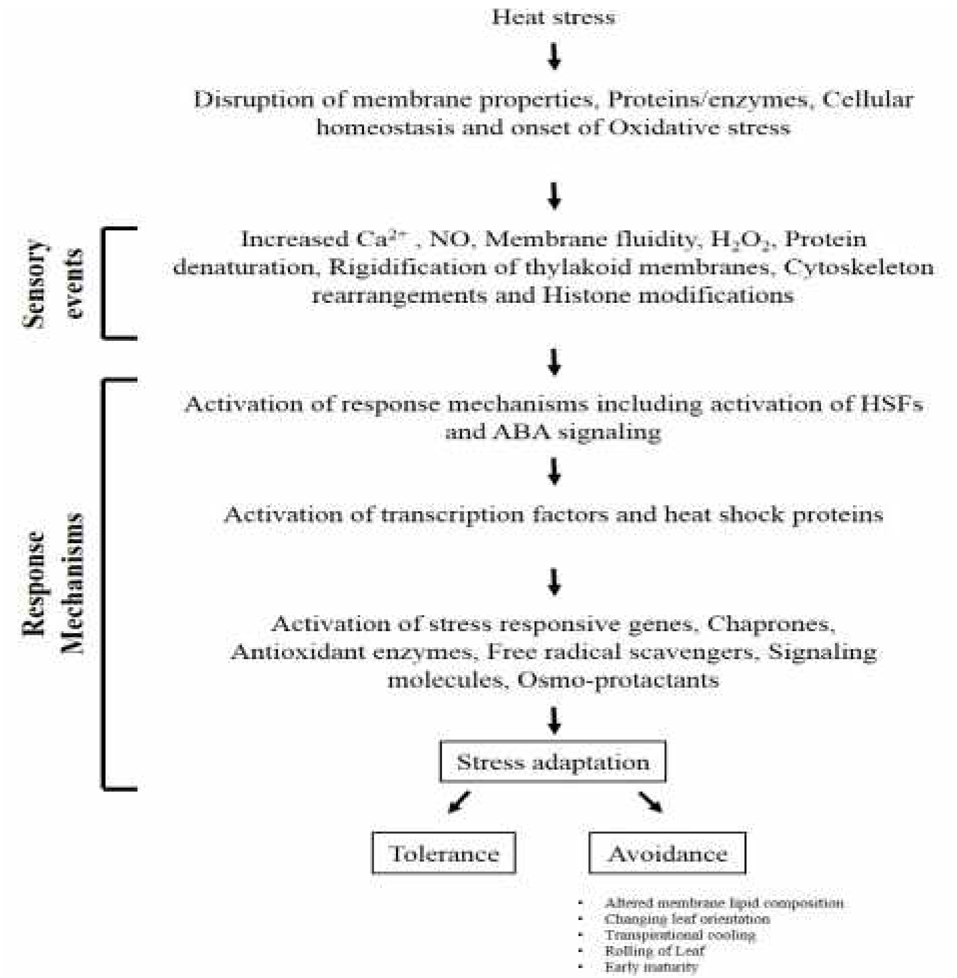

Figure 2. A general overview of different sensory-response events involved in plant response to heat stress. Partly adopted from Wahid *et al*. (2007), Saidi *et al*. (2011), Hasanuzzaman *et al*. (2013)

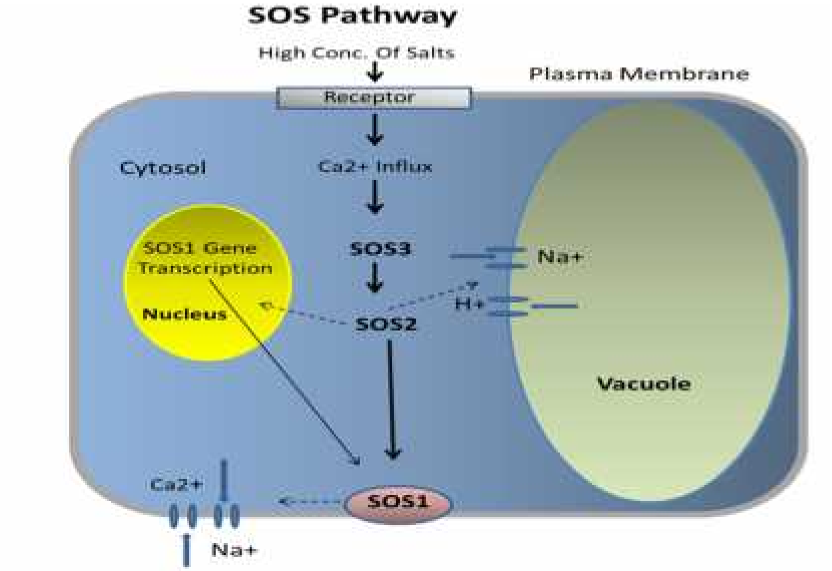

Figure 3:High concentration of salts in the external environment causes Ca2$^+$ influx which leads to the activation of SOS3 and subsequently SOS2 & SOS1 genes. These genes cause the extrusion of Na$^+$ ions from the plant body, preventing the excess build-up of Na$^+$

Conclusions and perspectives: It has clearly been established that plants deploy special sensory-response mechanisms to cope with challenging environment. Starting from the onset of stress, plants respond through a chain of inter-connected and inter-dependent events. Over the years, a significant amount of information has been gathered for plant stress response mechanisms. Current understanding of the epigenetic regulatory mechanisms (like chromatin regulation mediated by histone modification and DNA methylation, and RNA-mediated regulation) involved in plant abiotic stress responses has gradually been improved in recent years (Kim *et al*., 2015; Mittal *et al*., 2016). Still there are many unanswered questions. The putative nature of stress sensors (Shabala *et al.*, 2015a,b) adds complexity to our understandings. An intense investigation is suggested to explore novel genetic resources (including wild parents and extremophiles) for the identification of stress sensors. As plasma membrane is the only barrier between cellular contents and the environment, membrane bound transport proteins might be given more attention.

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Authors contributions: IR, HN, MHS and FA planned, designed and drafted the manuscript. MHS, AU and FA generated the figure. RMA, MAA, AU, FR, MAf, MAb, and FA read and approved the final manuscript.

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