We are IntechOpen, the world’s leading publisher of Open Access books
Built by scientists, for scientists

6,600 Open access books available
177,000 International authors and editors
195M Downloads

154 Countries delivered to
TOP 1% Most cited scientists
12.2% Contributors from top 500 universities

WEB OF SCIENCE™
Selection of our books indexed in the Book Citation Index
in Web of Science™ Core Collection (BKCI)

Interested in publishing with us?
Contact book.department@intechopen.com

Numbers displayed above are based on latest data collected.
For more information visit www.intechopen.com
Chapter 9

Advances in Plant Tolerance to Abiotic Stresses

Geoffrey Onaga and Kerstin Wydra

Additional information is available at the end of the chapter

http://dx.doi.org/10.5772/64350

Abstract

During the last 50 years, it has been shown that abiotic stresses influence plant growth and crop production greatly, and crop yields have evidently stagnated or decreased in economically important crops, where only high inputs assure high yields. The recent manifesting effects of climate change are considered to have aggravated the negative effects of abiotic stresses on plant productivity. On the other hand, the complexity of plant mechanisms controlling important traits and the limited availability of germplasm for tolerance to certain stresses have restricted genetic advances in major crops for increased yields or for improved other traits. However, some level of success has been achieved in understanding crop tolerance to abiotic stresses; for instance, identification of abscisic acid (ABA) receptors (e.g., ABA-responsive element (ABRE) binding protein/ABRE binding factor (AREB/ABF) transcription factors), and other regulons (e.g., WRKYs, MYB/MYC, NACs, HSFs, bZIPs and nuclear factor-Y (NF-Y)), has shown potential promise to improve plant tolerance to abiotic stresses. Apart from these major regulons, studies on the post-transcriptional regulation of stress-responsive genes have provided additional opportunities for addressing the molecular basis of cellular stress responses in plants. This chapter focuses on the progress in the study of plant tolerance to abiotic stresses, and describes the major tolerance pathways and implicated signaling factors that have been identified, so far. To link basic and applied research, genes and proteins that play functional roles in mitigating abiotic stress damage are summarized and discussed.

Keywords: abiotic stress, climate change, crop improvement, transcription, regulatory proteins

1. Introduction

Abiotic stress is defined as the negative impact of non-living factors on living organisms in a specific environment. Abiotic stresses, such as drought, salinity, low or high temperatures and other environmental extremes are the major cause of poor plant growth and reduced crop yields in the world [1]. Drought alone affects 45% of the world’s agricultural land, whereas
19.5% of irrigated agricultural lands are considered saline [2, 3]. Moreover, 16% of the agricultural rice land of the world suffers from flash flooding [4]. A combination of two or more abiotic stresses, e.g., drought and heat stress also occurs in field situations and causes more severe crop yield reductions than a single stress [5]. With increasing challenges posed by climate change, it is predicted that warming, drought, floods and storm events will become even more frequent and severe, and will further reduce crop yields, especially in the tropics and subtropics.

Abiotic stresses commonly induce overproduction of reactive oxygen species (ROS) causing extensive cellular damage and inhibition of physiological processes in plants. Although anti-oxidative mechanisms would be an immediate endogenic choice of the plants to counter ROS production, this mechanism can be impaired by abiotic stresses causing a rise in ROS intracellular concentration and an increase in the damage. To survive under such conditions, plants have evolved intricate mechanisms, allowing optimal responses that enable adaptation or avoidance of the stress. These plant responses are regulated at all levels of organization. At the cellular level, responses include adjustments of the membrane system, modifications of cell wall architecture, changes in cell cycle and cell division, and synthesis of specific endogenous and low-molecular-weight molecules, such as salicylic acid, jasmonic acid, ethylene and abscisic acid [6]. An overview of changes that may occur under different abiotic stress conditions is presented in Figure 1.

At the genomic level, plant responses include the expression of stress-inducible genes involved in direct plant protection against stresses [3, 7, 8]. A broad range of abiotic stress induced genes are divided into two functional categories: and regulatory proteins. The first group consists of genes encoding for membrane proteins, enzymes for osmolyte biosynthesis, detoxification (glutathione S-transferases, superoxide dismutases, dehydrins, dehydroascorbate reductases, quinine reductases and ascorbate peroxidases) and proteins for macromolecular protection (such as LEA protein, anti-freezing proteins, chaperons and mRNA binding protein) [2]. The second group comprises genes encoding for transcription factors (e.g., DREBPs), protein kinases (e.g., SRK2E), receptor protein kinases, ribosomal-protein kinases and signal transduction proteinases (such as phosphoesterases and phospholipase C). Alterations in the phenylpropanoid pathway in which lignin biosynthesis intermediates are produced also occur under abiotic stress conditions. Moreover, increased accumulation of wall-linked phenolic compounds, for instance, in maize root elongation zone and the polyphenol content in cotton have been linked to stress response [9]. The same authors have shown the role of flavonoids, isoflavonoids, terpenoid and nitrogen-containing secondary metabolites such as glucosinolates alkaloids in abiotic stress response.

Thus, abiotic stress tolerance in plants is a complex trait, involving many different metabolic pathways and cellular and molecular components.

In the past 100 years, conventional breeding (Figure 2; based on observed variation and controlled mating) approaches have randomly exploited these plant tolerance mechanisms with limited success. Moreover, in vitro induced variations have also shown little progress in the improvement of plants against abiotic stresses. These conventional breeding approaches are limited by the complexity of stress tolerance traits coupled with less genetic
variation exhibited by most crops due to domestication bottlenecks. The recent reports that the cultivated gene pool of major cereal crops, e.g., rice, maize and wheat, has reduced in genetic variation compared to wild relatives [10–12], raises concern, and could probably undermine the current efforts to identify genetic sources of resistance within the cultivated genepools. It is important, therefore, to consider exploring alternative sources of resistance by incorporating modern techniques into traditional breeding strategies to develop stress-tolerant crops (Figure 2).

Recently, with the support of genomics, targeted genetic studies involving QTL mapping and validation, identification of key regulatory genes, e.g., genes encoding for ABA receptors, developments in transcriptional and post-transcriptional regulation of stress-responsive genes and studies on hormonal interactions during plant response to stress, have provided opportunities for understanding cellular stress responses in plants. Moreover, the emergence of deep sequencing technologies, proteomics, metabolomics and epigenetics, has remarkably provided novel possibilities to understand the biology of plants and consequently to precisely develop stress-tolerant crop varieties. Amongst the techniques that are currently being
exploited to develop stress-tolerant plants, alongside basic molecular biology, there are molecular breeding methods, including development of functional molecular markers to aid in marker-assisted selection, horizontal gene transfer and genome editing tools such as CRISPR/Cas9, to develop genetically modified plants with new or improved characteristics.

In this chapter, we reviewed the plant responses to various abiotic stresses, and focus on genetic and molecular components that function to confer stress tolerance in plants.

2. Advances in plant tolerance to drought

Drought tolerance in plants is the ability to survive and produce stable yields under water scarcity during various stages of crop growth. Principally, drought stress occurs when the soil water potential falls between −0.5 and −1.5 MPa. This affects plants by decreasing the photosynthetic rate through photo-oxidation and enzyme damage, thereby decreasing the amount of assimilates available for export to the sink organs [13]. Besides this, carbohydrate metabolism in plants is severely altered, ultimately affecting both biological and economical yield [14].
Evidence from several studies has shown that plants respond to drought, like many other abiotic stresses, by inducing cellular damage and secondary stresses, such as osmotic and oxidative stresses. These secondary stresses induce initial stress signals (e.g., osmotic and ionic effects and membrane fluidity changes) that are perceived by membrane receptors (sensors). The perceived signals are transmitted downstream to trigger transcription, which is regulated by phytohormones, transcription factor binding proteins (TFBPs), cis-acting elements and miRNAs. Based on the biological functions, the role of these transcriptional regulators and the regulated genes that encode functional proteins or other products to protect plant cells directly from damage is well described [15].

The phytohormone—abscisic acid—acts as a central regulator in the response and adaptation of plants to drought conditions. The various physiological reactions regulated by ABA, including stomatal closure, accumulation of osmoprotectants, changes in gene expression, and other phytohormones have been characterized at the molecular level [16]. The molecular mechanisms of ABA synthesis, transport and signaling in relation to the plant’s response to stress are also reasonably well described [17]. ABA signals are perceived by different cellular receptors. The nucleocytoplasmic receptors PYR/PYL/RCARs (pyrabactin resistance/pyrabactin resistance-like/regulatory component of ABA receptors) have been suggested to be the primary sensors that bind ABA and inhibit type 2C protein phosphatases (PP2Cs) [18]. Inactivation of PP2Cs leads to accumulation of active sucrose non-fermenting-1 (SNF1)-related protein kinases (SnRK2s), which interacts with ABA-responsive TFs, ABA-responsive promoter elements (ABREs) and ABRE-binding protein/ABRE-binding factors (AREB/ABF) to regulate transcription of downstream target genes and related physiological processes [19]. Drought also induces changes in calcium ion levels, which activates calcium-dependent protein kinases (CDPKs) via calmodulin-like domain. The activated CDPKs regulate downstream components of calcium signaling. For instance, OsCPK4 overexpressing rice plants exhibit increased water-holding capacity under drought or salt stress [20]. Genetic manipulation of RLK genes, including OsSIK1 that acts as a positive regulator of drought stress responses, is also well reported [21]. Other secondary signaling molecules, including phosphatases (serine/threonine phosphatases) and phospholipids such as phosphoinositides, nitric oxide, cAMP and sugars, play an important role in signal transduction [22]. Examples of phosphatases include the wheat phosphatase TaPP2Ac-1 that exhibited less wilting under water-deficit conditions than non-transformed controls [23].

Numerous TF families such as myeloblastosis oncogene (MYB), dehydration-responsive element binding proteins (DREB), basic leucine zipper domain (bZIP), WRKYs and the NAC (NAM, ATAF and CUC) are directly or indirectly regulated by endogenous ABA signaling during drought stress [24]. Many MYB genes involved in plant response to drought stress are functionally characterized, including AtMYB15, which was shown to enhance drought tolerance, and sensitivity to ABA [25]. WRKY proteins, including ABA-inducible OsWRKY45, OsWRKY11 and OsWRKY08, are upregulated by drought stress [26]. AP2/ERF family is another large group of plant-specific TFs that have been demonstrated to be effective in enhancing drought tolerance in plants. For instance, overexpression of AP2/ERF genes, e.g., GmERF3 in soybeans, has been reported to enhance tolerance to drought [27]. In addition, DREB2s, e.g.,
ZmDREB2.7, are candidates for drought stress tolerance in maize [28]. The bZIP TFs have also been reported to enhance plant tolerance to stress and hormone signal transduction, e.g., OsbZIP23 in rice [29] and ZmbZIP72 in maize [30]. Within the NAC family, RD26 (responsive to dehydration [26]) was the first NAC gene identified as a regulator in mediating crosstalk between abscisic acid and jasmonic acid (JA) signaling during drought stress responses in Arabidopsis [31]. Overexpression of other NAC genes, including ANAC019, ANAC055 and ANAC072, has been shown to confer drought tolerance in transgenic Arabidopsis [32]. Similarly, overexpression of SNAC1, OsNAC10 and OsNAC5 driven by a root-specific promoter RCc3 confers increased drought resistance under field conditions [33, 34]. The nuclear factor Y (NF-Y) TFs are emerging as important regulators of drought-stress response, particularly with respect to ABA biosynthesis. For instance, ectopic expression of Amaranthus hypochondriacus NF-YC gene (AhNF-YC) in Arabidopsis and overexpression of Bermuda grass Cd1-NF-YC1 in rice has shown that these genes confer drought tolerance [35, 36]. Cd1-NF-YC1 induces expression of both ABA-responsive genes (e.g., OsRAB16A, OsLEA3, OsP5CS1 and OsLIP9) and signaling genes (e.g., OsABI2 and OsNCED3), as well as, ABA independent genes (e.g., OsDREB1A, OsDREB2B and OsDREB1B). In fact there is an increasing evidence that some NAC genes, e.g., SNAC3, contribute to drought resistance and osmotic adjustment independent of ABA [37]. SNAC3 interacts with phosphoglycerate mutase, cytochrome P450 72A1, PP2C, WD domain-containing protein and oxidoreductase to modulate ROS in rice. These findings suggest a complex regulatory mechanisms of drought response and tolerance in plants, involving both ABA and other signaling pathways.

Recent work on inhibitors of phosphoinositide-dependent phospholipases C (PI-PLCs) in Arabidopsis has also provided considerable insight into the drought-stress-related lipid signaling by identifying links of phosphoinositides to the DREB2 pathway [38]. Moreover, overexpression of phosphatidylinositol synthase gene (ZmPIS) in tobacco plants changed membrane lipids’ composition and improved drought stress tolerance [39]. The best characterized lipid derivative, so far, is inositol 1,4,5-trisphosphate (IP₃). IP₃ levels have been shown to increase in response to exogenous ABA in Vicia faba guard cell protoplasts and in Arabidopsis seedlings, for review see [40]. IP₃ acts as a second messenger involved in releasing Ca²⁺ from internal stores such as vacuoles. This pathway has been reported to induce osmotic-stress-responsive genes, as well as ABA stress-responsive genes [40]. Another lipid derivative, phospholipase D (PLD), has been reported by the same authors to be functionally associated with ABA; and the application of phosphatidic acid (PA), a PLD derivative, has been shown to mimic the effect of ABA in inducing stomatal closure [41]. This could probably suggest that lipid signaling is linked to ABA in drought stress response, and it is worthwhile to study how the different lipid derivatives enter in action, either simultaneously or timely synchronized with ABA.

Downstream of the TFs are numerous responsive genes that function either in a constitutive manner (i.e., also expressed under well-watered conditions) or a drought-responsive manner (i.e., expressed only under pronounced water shortage). Amongst them, genes encoding for receptor-like kinases (RLKs) with Ser/Thr kinase domain could play an important role in optimizing plant responses to drought stress [18]. Other genes that have been shown to be up-
or downregulated by drought stress to enable dehydration avoidance or tolerance in various plant species are documented in several studies [18, 42]. Another process, downstream of transcriptional regulatory networks, is the induction of a large range of genes encoding for enzymes involved in osmotic adjustments, osmoprotection, wax biosynthesis and changes in fatty acid composition (Figure 3). Adjustment of osmotic pressure allows the plant to take up more soil water and maintain turgor and cell function for a longer time under drought.

Water-channel proteins, e.g. aquaporins (AQPs), and sugar transporters are believed to facilitate the adjustment of osmotic pressure under stress by transporting water and sugars to the cytosol [42]. More recently, AQPs encoding genes (e.g., MaPIP1;1) were shown to be strongly induced in banana plants exposed to drought [43]. The same authors indicate that overexpression of MaPIP1;1 in Arabidopsis exhibited better growth, reduced water loss and higher survival rates. Li et al. [44] also showed that AQPs were elevated under drought stress in Tibetan Sophora moorcroftiana, which is consistent with the previous reports [45]. However, the same authors indicate conflicting functions of plasma membrane intrinsic proteins (PIPs). For instance, overexpression of GoPIP1, cloned from Galega orientalis, showed increased sensitivity to drought in transgenic Arabidopsis plants. This indicates that AQPs are able to facilitate both tolerance and sensitivity, which warrants further research to delineate AQPs that are potentially helpful in improving drought tolerance in plants.
Studies have also shown that the K⁺ uptake transporter 6 (KUP6) subfamily of transporters act as key factors in osmotic adjustment by balancing potassium homeostasis in cell growth and drought stress responses [46]. KUP6 is apparently under the control of abscisic acid and interacts with ABA-activated SnRK2-type protein kinase, SnRK2E, resulting in phosphorylation of the KUP6 C-terminal domain. This indicates that KUP6 is a downstream target for SnRK2E in the control of water stress responses. However, other interacting proteins, and probably hormones, e.g. auxins, could regulate the activity of KUP6 in the maintenance of water status during drought stress. Indeed, it was reported previously that a variant of KUP6, KUP4/TRH1, facilitates root-specific auxin distribution [47]. This was substantiated by the findings that triple mutants of the KUP genes (i.e., kup2 kup6 kup8 and kup6 kup8 gork) showed enhanced cell expansion and auxin responses in lateral root formation [54]. Moreover, auxin-responsive TFs, LBD18 and LBD29, were highly expressed in the triple mutants in the presence of IAA, indicating that auxin could be modulating K⁺ and proton fluxes during drought stress.

The biosynthesis of osmoprotectants such as amino acid, amines and carbohydrates is another indispensable strategy for plant resistance to drought stress. The most common osmoprotectants are proline (Pro), γ-aminobutyric acid (GABA), glycine betaine (GB), fructans, starch, mono- and disaccharides, trehalose (Tre) and raffinose family oligosaccharides (RFO). The biosynthesis and transport of trehalose and raffinose is particularly relevant in drought stress response. More recently, genes encoding for trehalose and raffinose biosynthesis were significantly upregulated in the roots and leaves of Jatropha curcas under drought [48], suggesting that these compounds may have major impacts on osmotic adjustment and ROS scavenging during drought stress. The same authors indicated that dozens of genes involved in wax biosynthesis, including KCS and WSD, and their regulators (e.g., MYB96, CER) were upregulated more than four-fold in leaves under drought conditions. Overexpression of genes encoding for MYB96, CER KCS and WSD could probably strengthen the hydrophobic barrier that prevents non-stomatal water loss and increase plant tolerance to drought.

Genes encoding for proteins involved in cellular structure stabilization have also been reported to be induced in plant tolerance to drought. For instance, dehydrins (DHNs) function to protect cells from damage caused by drought stress-induced dehydration [49]. Proteins related to lignin biosynthesis, such as caffeoyl-CoA 3-O-methyl-transferases and class III plant peroxidases, were also found to be induced by drought in wild watermelon [50] and in maize roots [51]. In winter triticale, water-deficit-induced leaf rolling was correlated with a higher level of cell wall-bound phenolics in the leaves [52]. These adaptive mechanism could probably limit water loss by restricting the leaf transpiration surface. In addition, carbon/nitrogen-metabolism-related proteins have been reported to be more abundant in roots of soybean [53], wild watermelon [50] and rapeseed [54] after drought treatment, suggesting an increased energy demand as well as enhanced cellular activities in the root tissues during drought stress. The same authors reported a relative increase in the root growth rate and abundance of root-growth-related small G-protein family members such as Ran GTPases, which suggests increased membrane trafficking activity in an effort by the plant roots to absorb water from deep soil layers.
Taken together, the vast amount of data from ‘omic’ tools provide a basis for identification of more functional genes, which could contribute directly to cellular drought stress tolerance. In addition, understanding expression networks of genes encoding for the aforementioned proteins, especially genes involved in cellular structure stabilization, molecular chaperones, enzymes for detoxification of reactive oxygen species, and those for the biosynthesis of sugars, wax and dehydrins, which are important as protectants [55], may allow for the realization of significant genetic gains in breeding for plant tolerance to drought. Further genomic scale investigations will enable understanding of transcriptional regulators behind co-expressed genes and their association with particular genomic regions (QTLs). Although QTL identification for tracing drought tolerance remains a challenge due to the large number of genes influencing drought tolerance traits, continued investigation into the basis of tolerance in crops like *Jatropha curcas* will probably provide a clearer understanding of drought tolerance. Besides this, the mechanism by which drought tolerance associated protein networks effectively protect PSII and granal stability, as well as maintain photosynthetic competence will need further elucidation.

### 3. Advances in plant tolerance to heat stress

Temperatures above the normal optimum cause heat stress (HS) at different levels in all living organisms. Heat stress disturbs cellular homeostasis, and causes denaturation and dysfunction in many proteins, leading to severe retardation in growth, development and even death. In plants, the major sites of heat stress injury are the oxygen-evolving complex (OEC) along with associated biochemical reactions in photosystem II (PSII). Ultimately, efficiency of electron transport is reduced or altered affecting electron flow from OEC towards the acceptor side of PSII. These alterations affect the generation of ATP and the regeneration of Rubisco for carbon fixation [56]. Starch synthesis is also negatively affected by heat stress because of the reduced activity of enzymes such as invertase, sucrose phosphate synthase and ADP glucose pyrophosphorylase. Usually, ROS induction and accumulation in the chloroplasts precedes these changes. Accumulated ROS can severely damage DNA and cause autocatalytic peroxidation of membrane lipids and pigments, altering membrane functions and cell semi-permeability. Physiological changes associated with biochemical damage may include a decrease in chlorophyll a:b ratio, inhibitions of stomatal conductance and net photosynthesis, and low plant water potential. These changes ultimately reduce the partitioning of photosynthates, which morphologically manifest as retarded growth, reduced economic yield and harvest index. Scorching and sunburns of leaves and twigs, branches and stems, leaf senescence and abscission, and fruit discoloration and damage are other morphological damages associated with heat stress [57].

Perception of heat stress by plants usually triggers sensors at the plasma membrane and causes a transient opening of Ca²⁺ channels, possibly via modulation of membrane fluidity (Figure 4) [58]. Upon entry of Ca²⁺, putatively through channels possessing cytosolic C-terminus with a calmodulin-binding domain, multiple kinases are activated.
Figure 4. Hypothetical model for high-temperature signal sensing and induction of molecular pathways leading to plant defence response. Prolonged high-temperature stress causes membrane depolarization leading to Ca$^{2+}$ influx or directly activates apoplastic enzymes including GLPs. Increased levels of cytosolic calcium activate the ROS-producing enzyme, RBOHD, which catalyses ROS production. Effect of temperature on R genes through an unknown pathway is likely to further enhance ROS production. ROS/ Ca$^{2+}$ signaling causes activation of plasma membrane ATPase, which extrudes H$^+$. Alternatively, heat-stress-induced protein damage and protease activity decreases cytosolic pH. Low cytosolic pH and H$_2$O$_2$ accumulation reduces CO$_2$ assimilation, thereby increasing endogenous carbohydrate metabolism. Cytosolic acidification and ATPase activity may also increase accumulation of expansins and methylesterases that eventually affect the cell wall integrity. Activating plasma membrane ATPase is probably reverse phosphorylated by FKBP65 leading to H$^+$ extrusion and K$^+$ intrusion. Apart from its targeted role in the nucleus, FKBP65 could be targeted to the chloroplast through the tat pathway to activate photosystem II 10 kDa poly peptides or for directing chaperone functions. Activated HSPs probably cause chromatin remodelling and histone displacement. In addition to activating PM ion channels, heat-induced changes in membrane fluidity triggers lipid signaling. Plants deploy phospholipids, including phospholipase D (PLD), PIPK (phosphatidylinositol 4,5-bisphosphate kinase), phosphatidic acid (PA), PIP2 (phosphatidylinositol phosphate kinase) and IP3 (D-myo-inositol-1,4,5-trisphosphate) to specific intracellular locations. The accumulation of lipid signaling molecules also triggers Ca$^{2+}$ influx, which initiates downstream signaling, including activation of CDPKs, hormonal changes, transcription factor activation and secondary metabolism. Question marks indicate the unknown players.

The MPK6 activity has been particularly shown to increase under heat stress. MPK6 activates a vacuolar processing enzyme (VPE), which has been suggested to play a role in HS-induced programmed cell death [59]. Transcriptional regulators, such as HSFs, WRKY, Zat and MBF1c, a transcriptional regulator of DREB genes [60], are activated to regulate expression of HSPs and other heat stress response genes.

Heat-induced accumulation of Ca$^{2+}$ in the cytoplasm also activates the ROS-producing enzymes RBOHD and NADPH oxidase, by direct interaction or through activation of calcium-dependent protein kinases (CDPK) that phosphorylate RBOHD [61]. When activated, RBOHD

The MPK6 activity has been particularly shown to increase under heat stress. MPK6 activates a vacuolar processing enzyme (VPE), which has been suggested to play a role in HS-induced programmed cell death [59]. Transcriptional regulators, such as HSFs, WRKY, Zat and MBF1c, a transcriptional regulator of DREB genes [60], are activated to regulate expression of HSPs and other heat stress response genes.

Heat-induced accumulation of Ca$^{2+}$ in the cytoplasm also activates the ROS-producing enzymes RBOHD and NADPH oxidase, by direct interaction or through activation of calcium-dependent protein kinases (CDPK) that phosphorylate RBOHD [61]. When activated, RBOHD
catalyzes the production of ROS, causing membrane depolarization and/or initiation of ROS/redox signaling network, which interacts with the above-mentioned MBF1c, HSFs, MAPKs and SnRKs to trigger downstream signaling networks [61].

Calcium/calmodulin-binding protein kinases (CBK), which also regulate the expression of HSPs, are activated via CaM3. A well-known example is the activation of CBK3, which enhances thermotolerance in A. thaliana seedlings by phosphorylating HsfA1a and a CaM protein phosphatase (PP7) [62]. PP7 interacts with both AtCaM3 and AtHsfA1a. AtCaM3 increases thermotolerance by activating WRKY39 and HSFs, indicating that CBK3 plays a key role in heat stress signaling. The TF Zat is necessary for the activation of WRKYs and ascorbate peroxidase [63]. MBF1c modulates the induction of SA and trehalose, which are regulators of plant stress response [64]. SA has been shown to alleviate heat stress by increasing proline production and restricting the formation of ethylene in heat-stressed plants [65].

Another HS-response-associated signaling pathway was shown in the Hsp90–ROF1 interaction in the cytoplasm and their subsequent translocation to the nucleus. The Hsp90–ROF1 complex localizes in the nucleus only in the presence of HsfA2 [66]. The interaction of these three proteins modulates HSP gene expression under HS. Although, ROF1 has been reported to induce expression of small HSPs, which increases plant survival rate under HS, to date the upstream signal that regulates the subcellular localization of Hsp90–ROF1 remains elusive. Interestingly, just like MBF1c, ROF1 is involved in calcium-dependent phosphorylation of HSFs, which suggests that Ca\(^2+\)-dependent activation of RBOHD or CDPKs could be the upstream signal for ROF1.

Heat stress also triggers lipid signaling. Activation of phospholipase D (PLD) and a phosphatidylinositol 4,5-bisphosphate kinase (PIPK) increases the accumulation of phosphatidic acid (PA), phosphatidylinositol kinase and D-myo-inositol-1,4,5-trisphosphate (IP3); and an active cycling of a G protein appears necessary in this process. The accumulation of lipid signaling molecules could in turn cause the opening of channels and the triggering of Ca\(^2+\) influx [67].

Downstream effects of heat stress signals have been reported to activate a signaling pathway called unfolded protein response (UPR) in the endoplasmic reticulum, which requires specific calcium signals from the plasma membrane [58]. Within the endoplasmic reticulum, the activity of UPRs involves two signaling pathways: one involving proteolytic processing of membrane-associated bZIP TFs and the other involving RNA splicing factor, inositol requiring enzyme-1 (IRE1) and its mRNA target [68]. IRE1 is a dual functional enzyme possessing both serine/threonine protein kinase and endoribonuclease activity. In Arabidopsis, heat signals activate IRE1 to splice bZIP60 mRNA in the cytosol, causing a frameshift, which triggers the synthesis of a tissue factor without a transmembrane domain, but having a nuclear targeting signal [69]. The bZIP60 (bZIP60(s)) spliced forms activate UPR target genes in the nucleus. A cytosolic form of UPR, which is triggered by the presence of unfolded proteins in the cytosol, was also previously reported [70]. Together, these UPRs are associated with the heat shock promoter elements and the involvement of specific HSFs, notably HSFA2, regulated by alternative splicing and non-sense-mediated decay. Under severe HS (42–45°C), a novel post-transcriptional regulatory mechanism governing HSFA2 expression has also been shown to
occur. Moreover, a new splice variant of HSFA2-III is reported to be generated through the use of acryptic 5′ splice site in the intron. HSFA2-III can be translated into the small HSFA2 (S-HsfA2), which binds to the TATA box proximal clusters of HS elements (HSE) in the HSFA2 promoter to activate its own gene expression, thus constituting a positive auto-regulatory loop [71]. Although the TFs interacting with S-HsfA2 are yet to be validated, this finding suggests that severe HS may alter the splicing pattern of Hsf genes, generating isoforms that may auto-activate self-expression and consequently rapidly induce the expression of HSPs required for enhanced response to HS.

Apart from HSFs, overexpression of DPB3-1, which regulates expression of DREB2A and DREB2B, increases thermotolerance [72]. Other studies have also shown the role of bZIP28 [73] and WRKY proteins in plants thermotolerance [74, 75]. Furthermore, the basic helix-loop-helix (bHLH) TF, phytochrome interacting factor 4 (PIF4), was reported to control acclimation to changes in ambient temperature by regulating important hormonal and developmental pathways modulating the acclimation mechanisms [76]. PIF4 alleles control floral timing by modulating FLOWERING LOCUS T (FT). PIF4 also controls early inflorescence internode elongation and high-temperature-induced hypocotyl elongation by modulating levels of free indole-3-acetic acid (IAA) through the triggering of YUC8 (YUCCA8) or TRYPтопHAN AMINOTRANSFERASE OF ARABIDOPSIS (TAA1) gene expression [57, 77]. Thus, PIF4 is a potential regulator of plant responses to high temperature. However, its physical interaction with cryptochrome 1 (CRY1) on nuclear DNA suggests that these two proteins co-regulate temperature responses in plants. Another regulator, E3 ubiquitin ligase CONSTITUTIVE PHOTOMORPHOGENIC1 (COP1), was shown to be essential for plant responses to HS [77]. However, it is not known whether COP1 signaling is independent of PIF4. Orthologs of PIF4 have been identified in several crop species. Thus, if the interaction with other associated proteins is resolved, PIF4 has a potential promise to improve plant tolerance to HS in several crops through genetic engineering.

Other components of heat sensing that could be linked to these signaling pathways include the transcriptional modulator, the nuclear actin-related protein 6 (ARP6), which is part of the Snf-2-related CREB-binding activator protein (SRCAP) encoding a subunit of the SWR1 chromatin remodelling complex is necessary for inserting the alternative histone, H2A.Z, into nucleosomes, while replacing the core histone H2A [78]. Heat stress induces a decrease in H2A.Z occupancy in nucleosomes located at the transcription start site of heat response genes, a process that probably allows nucleosome opening and enhanced transcription of these genes.

Plant adaptation to thermotolerance also involves the activity of superoxide reductase (SOR), S-nitrosoglutathione reductase (GSNOR) and rubisco activase (RCA). The functions of these proteins are reasonably well described in a review by [67]. Other commonly reported antioxidant enzymes produced by plants under HS include superoxide dismutase (SOD), catalase (CAT), guaiacol peroxidase (GPX), ascorbate peroxidase (APX), dehydroascorbate reductase (DHAR), glutathione reductase (GR), glutathione S-transferase (GST) and non-enzymatic antioxidants such as flavanoids, anthocyanin, carotenoids and ascorbic acid (AA) [60]. The accumulation of other osmolytes such as glycine betaine and trehalose is another well-known adaptive mechanism in plants against HS. Generally, most of these compounds are involved
in ROS removal (anti-oxidants), osmotic adjustment, saturation of membrane-associated lipids, protection of photosynthetic reactions, production of polyamines and protein biosynthesis [94], which enable plants to exhibit basal or acquired thermotolerance. Proline and glycine betaine application considerably reduce the $H_2O_2$ production, improve the accumulation of soluble sugars and protect the developing tissues from HS [79]. Tocopherol is another important lipid-soluble redox buffer and an important scavenger of singlet oxygen species and other ROS. Moreover $\alpha$-tocopherol has the highest anti-oxidant activity of all the tocopherol types reported in plants [80].

A number of studies have demonstrated the presence of QTLs associated with most HS-related traits and promise to the use of molecular markers in breeding for heat stress tolerance. More than 50 QTLs have been identified in various crops so far, including maize, wheat, rice, cowpea, lettuce, Medicago truncatula and Brassica napus. Recent studies in transcriptomics [81, 82], proteomics [83, 84], metabolomics [85, 86] and microRNAs [87] have also provided additional information on the mechanisms controlling plant responses to HS. Understanding the relationship between these mechanisms and the genomic regions mapped and delineated as QTLs would validate the genes controlling plant responses to HS, and subsequently improve genetic gains in plant improvement programmes. Besides, the possibility of developing transgenic plants with enhanced tolerance to HS would also gain significance. This approach has already been demonstrated in cotton [88], Arabidopsis [89], tobacco [90] and rice [91], but needs further validation, especially in economically important crops where it has not been applied before. Taken together, heat stress responses discussed here demonstrate that heat stress is a quantitative trait, which requires a combination of several disciplines to improve plant tolerance.

4. Advances in plant tolerance to cold stress

Cold stress occurs at temperatures less than 20ºC and varies with the degree of temperature duration and plant type. Chilling (<20ºC) or freezing (<0ºC) temperatures can trigger the formation of ice in plant tissues, which causes cellular dehydration [92]. Ultimately, cold stress reduces plasma membrane (PM) integrity, causing leakage of intracellular solutes. Cold stress severely affects plant growth and survival, and leads to substantial crop losses in temperate climatic regions and hilly areas of the tropics and subtropics [93]. In rice, for instance, losses due to cold stress can range from 0.5 to 2.5 t/ha and grain yields can drop by up to 26%, especially when low temperatures occur during the reproductive stage [94].

To cope with this adverse condition, plants adapt several strategies such as producing more energy by activation of primary metabolisms, raising the level of anti-oxidants and chaperones, and maintaining osmotic balance by altering cell membrane structure [95]. These mechanisms of plant response to cold stress are closely similar to that of heat stress. However, the difference lies in the fact that membrane rigidification occurs in cold stress as opposed to heat stress. Thus, membrane rigidification is the upstream trigger for the induction cytosolic $Ca^{2+}$ signatures leading to a transient increase in cytosolic $Ca^{2+}$ levels [96]. It is assumed that dimethyl
sulfoxide (DMSO) mediates the perception of membrane rigidification by mechanosensitive Ca\textsuperscript{2+}-channels [97]. Other upstream factors such as changes in the metabolic reactions and metabolite concentrations, protein and nucleic acid conformation could contribute to enhance perception of cold stress. These factors as well, either directly or indirectly, induce an increase in cytosolic Ca\textsuperscript{2+}, which is a well-known upstream second messenger, regulating cold regulated (COR) gene expression.

Cold-stress-induced cytosolic Ca\textsuperscript{2+} signals can be decoded by different pathways. More recently, Ca\textsuperscript{2+} signal was reported to be transduced directly into the nucleus. The concentration of nuclear Ca\textsuperscript{2+} is monitored by a chimera protein formed by the fusion of aequorin to nucleoplasm, which is also transiently increased after cold shock [95]. Aequorin possesses several EF-hand-type binding sites for Ca\textsuperscript{2+} ions. The binding of Ca\textsuperscript{2+} to these sites causes a conformational change in aequorin which enables the monitoring of Ca\textsuperscript{2+} concentration. It has been reported that nuclear Ca\textsuperscript{2+} concentration peaks at about 5–10s later than the cytosolic Ca\textsuperscript{2+} [95]. The same authors have reported that nuclear Ca\textsuperscript{2+} signal may be initiated from the nuclear envelope and is assumed to be propagated by cytosolic Ca\textsuperscript{2+} transients in plants.

In the cytoplasm, a range of Ca\textsuperscript{2+} sensors have been reported, including calmodulin (CaM), CaM-like (CMLs), Ca\textsuperscript{2+}-dependent protein kinases (CDPKs), Ca\textsuperscript{2+}- and Ca\textsuperscript{2+}/CaM-dependent protein kinase (CCaMK), calcineurin B-like proteins (CBLs) and CBL-interacting protein kinases (CIPKs) [98]. Some of the sensors work as negative regulators of cold tolerance in plants, e.g., calmodulin3, a SOS3-like or a CBL calcium-binding protein and a protein phosphatase 2C (AtPP2CA). The positive regulators, e.g., CDPKs and probably some CBLs, relay the Ca\textsuperscript{2+} signal by interacting with and regulating the family of CIPKs. For instance, CBL1 has been shown to regulate cold response by interacting with CIPK7 [99], whereas CAMTA3 has been identified as a positive regulator of CBF2/DREB1C through binding to a regulatory element (CG-1, vCGCGGt) in its promoter [100]. Although CBF2/DREB1C was previously reported to negatively regulate CBF1/DREB1B and CBF3/DREB1A, its expression appears to be necessary for integrating cold-inducible calcium signaling with gene expression, but under transient and tight control to avoid repression of freezing tolerance. Both CBF1/DREB1B and CBF3/DREB1A are required for constitutive expression of cold-inducible genes in Arabidopsis, and play an important role in cold acclimation (see discussion below).

Ca\textsuperscript{2+} influx into the cytoplasm also apparently activates phospholipase C (PLC) and D (PLD), which are precursors for IP\textsubscript{3} and PA, respectively. IP\textsubscript{3} activates IP\textsubscript{3}-gated Ca\textsuperscript{2+} channels that can amplify Ca\textsuperscript{2+} signatures in the cytoplasm, leading to higher induction of COR genes and CBFs, for review see [101].

There are some reports that the chloroplast may also play a role in sensing low temperature [98]. Cold stress is considered to cause excess photosystem II (PSII) excitation pressure, as a result of the imbalance between the capacity for harvesting light energy and the capacity to consume this energy on metabolic activity in the leaves, which probably leads to ROS generation. The damaging effect of ROS on the photosynthetic apparatus presumably leads to photoinhibition, which occurs even under relatively low irradiance [102] and is apparently a mechanism of cold acclimation or freezing tolerance. ROS also acts as the second messenger...
and may reprogramme transcriptome changes through induction of Ca\(^{2+}\) signatures and activation of MAPKs and redox-responsive TFs. The MAPK cascades in *Arabidopsis*, including AtMEKK1/ANP1 (MAPKKK)–AtMKK2 (MAPKK)–AtMPK4/6 (MAPK), positively regulate cold acclimation in plants [103].

The downstream signals that promote the production of COR proteins and cold response to metabolites are reasonably discussed in references [95, 104]. Specific examples include the upregulation of the TFs, CBF/DREB1s (CRT (C-repeat)/DRE binding proteins) [103], which initiate the transcription process. The CBF/DREB1 (mainly CBF3/DREB1A) pathway is controlled by a myelocytomatosis oncogene (MYC)-type TF, inducer of CBF expression1 (ICE1), which binds to the MYC recognition cis-elements (CANNTG) in the promoter of CBF3/DREB1A, and induces the expression of CBF3/DREB1A and its regulons during cold acclimation [103]. The function of ICE1 in cold response is conserved; and overexpression of *Arabidopsis* ICE1 improves chilling tolerance and enhances the accumulation of soluble sugars and proline concentration in cucumber [106]. In rice, OsICE1 and OsICE2 are induced by cold stress and sequentially upregulate OsDREB1B, rice heat shock factor A3 (OsHsfA3) and rice trehalose 6-phosphate phosphatase (OsTTP1). The CBF/DREB1s can bind to CRT/DRE cis-elements, A/GCCGAC, in the promoter of COR genes to regulate expression of COR genes [107]. Moreover, CBF/DREB1 genes are organized in tandem (CBF1/DREB1B-CBF3/DREB1A-CBF2/DREB1C) on *Arabidopsis* chromosome IV and have been reported to be induced at the same time, suggesting that combining these TFs in one genotype could probably improve cold tolerance. However, the inconsistent target specificity amongst the three CBF factors in CBF/DREB1-overexpressing transgenic plants reveals variability in their roles [108]. Indeed, CBF2/DREB1C has been shown to be a negative regulator of both CBF1/DREB1B and CBF3/DREB1A [109], while CBF1/DREB1B and CBF3/DREB1A act as positive regulators of cold acclimation by activating the same subset of CBF/DREB1-target genes [110]. CBF1/DREB1B and CBF3/DREB1A are therefore concertedly required to induce the whole CBF/DREB1-regulon to complete the development of cold acclimation, while the expression of CBF2/DREB1C is tightly controlled to avoid its negative modulation of CBF1/DREB1B and CBF3/DREB1A. The exact mechanism by which this happens is unknown.

Downstream of these TFs are COR genes, which are mainly linked to the onset of tolerance mechanisms and ultimately lead to acclimation. Genes encoding for annexin; hyper-sensitive-induced response (HIR) protein families (e.g., prohibitins and stomatins); dehydrins (e.g., 25 kDa dehydrin-like protein, ERD14, and cold acclimation-specific protein 15 (CAS15)); antioxidants (e.g., superoxide dismutase, catalase and ascorbate peroxidase); HSPs (e.g., HSP70 family being the most abundant); defence-related proteins such as protein disulfide isomerase; disease resistance response proteins, peptidylprolyl isomerase Cyp2 and cysteine proteinase; amino acids, polyamines and polyols; and cellulose synthesis, such as UDP-glucose pyrophosphorylase, are commonly reported in expression studies [111]. Several metabolism-associated proteins, including carbohydrate metabolism enzymes, such as phosphogluconate dehydrogenase, NADP-specific isocitrate dehydrogenase, fructokinase, cytoplasmic malate dehydrogenase, pyruvate orthophosphate dikinase precursors (PPDK), aconitate hydratase, glycine dehydrogenase and enolase, have also been reported to be activated during cold stress.
Thus, several genes and the corresponding proteins are associated with the regulation of the metabolic pathways operating under cold stress. However, identification of functional polymorphism in these genes remains a daunting task. A similar challenge is observed in the QTLs identified, so far, in various crops, including maize, barley, rice, wheat, sorghum and many other economically important crops. Identification of effective cold sensors also remains elusive, as multiple primary sensors are thought to be involved in sensing low temperatures. Thus, a comprehensive understanding of the defence mechanism from sensors, cold signaling, to the defence response will require further research on both upstream and downstream regulations of $ICE1$-$CBF/DREB1$-dependent pathway, as well as proteins that may be functioning independent of this pathway.

5. Advances in plant tolerance to salinity

Salinity is increasingly becoming a major threat to crop production, particularly due to inappropriate irrigation regimes and increasing use of brackish water for irrigation. As much as 6% of the total world land is subjected to salinity [113], and more than 20% of irrigated land is affected by salinity [114]. Moreover, major reductions in cultivated land area, crop productivity and quality that have been reported in the recent past are due to salt-induced stress [115]. Climate-change-associated rise in sea levels and coastal floods are expected to further contribute to this phenomenon in the future.

Salt stress in plants occurs when electrical conductivity of saturated soil paste extract (ECe) reaches 4.0 deci-Siemens per meter (dS/m; approximately 40 mM NaCl). The minimum level may, however, vary from crop to crop. For instance, the salinity threshold for rice is 3.0 dS/m [163]. Beyond this threshold, a yield reduction of 12% per dS/m has been reported to occur. When plants gradually accumulate salts, osmotic stress, nutrient imbalance and oxidative stress occur [116]. These salt effects disrupt intracellular ion homeostasis, membrane function and metabolic activity [117]. As secondary effects, salt-induced osmotic stress decreases root epidermal cell division and elongation rates, reducing primary root growth, eventually resulting in inhibition of growth and reduction of crop yields [118].

Alkalinity stress is a heightened version of salinity stress which has been reported to be much harsher than equimolar salinity, especially at neutral pH [119]. Although it is fairly understood that alkalinity causes osmotic challenge and ionic stress, and precipitates nutrients such as metallic micronutrients and phosphates, and disrupts the integrity of root cellular structure, the molecular signals and adaptive mechanisms are not well understood. Because many saline soils are also alkaline due to the presence of sodium (Na) carbonates, in this section we will exclusively focus on salinity, which is wide spread, and has been extensively researched and discussed in several studies.

To cope with saline soils, plants deploy a range of mechanisms that range from exclusion of $Na^+$ from the cells to tolerance within the cells. When plants are subjected to salinity, a series of responses ranging from genetic molecular expression through biochemical metabolism to physiological processes occur (Figure 5).
Amongst the receptor proteins identified as the first detectors of salt stress are G-protein-coupled receptors, ion channel, receptor-like kinase or histidine kinase. These receptors transduce signals that generate secondary signals such as Ca^{2+}, inositol phosphates, ROS, nitric oxide (NO) and ABA. The signaling pathway associated with increased concentration of cytosolic Ca^{2+} is the most reported.

Cytosolic Ca^{2+} activates calcium-dependent protein kinases (CDPKs), calcineurin B-like proteins (CBLs) and CBL-interacting protein kinases (CIPKs) to transduce signals to downstream protein activity and gene transcription [120]. Transcription factors such as calmodulin-binding transcription activators (CAMTAs), GT element-binding-like proteins (GTLs) and MYBs have been reported to be activated by Ca^{2+}/calmodulin directly [121–123]. Other commonly expressed TFs in response to salt stress include the basic leucine zipper (bZIP), e.g., OsbZIP71 in rice [124], WRKY [125], APETALA2/ETHYLENE RESPONSE FACTOR (AP2/ERF) [126], MYB [127], basic helix–loop–helix [128] and NAC [42] families. These TFs regulate the expression of genes related to water potential decrease, which results from osmotic stress caused by salinity.

Downstream of these TFs, there are several genes associated with salinity tolerance. The most reported are genes encoding for salt exclusion proteins, e.g., SOS1, cation:proton antiporter family1 of Na^{+}/H^{+} anti-porters, salt compartmentalization genes, e.g., vacuolar H^{+} -pyrophosphatase [129], and osmotic adjustment, e.g., pyrroline-5-carboxylate synthetase [130].
The salt overly sensitive (SOS) Ca\(^{2+}\) sensor regulatory mechanism is believed to be conserved in higher plants including monocots and dicots [131]. SOS consists of three functionally interlinked proteins, SOS3/SCaBP8–SOS2–SOS1. SOS3 mainly functions in the roots, while CBL10/SCaBP8, an alternative regulator of SOS2 that has been described as SOS3-like, primarily functions in the shoots. At high Na\(^{+}\) concentrations, increased influx of Ca\(^{2+}\) is perceived by SOS3 that encodes a myristoylated EF hand (a domain of five serially repeated helix–loop–helix calcium-binding motifs). Upon Ca\(^{2+}\) binding, a conformational change occurs and SOS3 activates the downstream serine/threonine protein kinase, SOS2, and recruits it to the plasma membrane. Subsequently, the SOS3–SOS2 complex stimulates the plasma membrane-localized Na\(^{+}/H^{+}\) anti-porter (SOS1), leading to the extrusion of the excess Na\(^{+}\) out of the cells [132]. Different from SOS3, SOS3-like proteins (CBL10/SCaBP8) are phosphorylated by their interacting protein kinases apparently regulating CBL/SCaBP–CIPK/PKS modules [133].

Besides extruding Na\(^{+}\), the adaptive SOS module also links cytosolic Na\(^{+}\) with Ca\(^{2+}\) binding proteins. The PM-localized NHX7/SOS1 and the intracellular localized cation:proton antiporter family1 (CPA1) of Na\(^{+}/H^{+}\) anti-porters (NHX1–NHX4; tonoplast-localized) are a ubiquitous family of transporters that mediate the exchange of K\(^{+}\) or Na\(^{+}\) for H\(^{+}\) while regulating cytoplasmic salt loads [134]. In the cytosol, increased influx of Ca\(^{2+}\) associated with excess Na\(^{+}\) levels is perceived by Ca\(^{2+}\)-binding calmodulins/calmodulin-like proteins, which interact with NHX1 transporters to sequester excess Na\(^{+}\) in the vacuole. In *Arabidopsis*, a calmodulin-like protein, AtCaM15, regulates the tonoplast localized AtNHX1 [135]. The interaction of AtCaM15 with AtNHX1 occurs in the vacuolar lumen and is dependent on Ca\(^{2+}\) and pH. The C-terminus of AtNHX1 has been shown to localize in the cytosol, which might suggest that this strategic placement is targeted for phosphorylation by protein kinases or for sensing changes in cytosolic pH. However, the protein kinase targeting AtNHX1 is unknown, and further studies on the interaction of this transporter with other proteins, especially protein kinases, will be necessary.

Interestingly, at moderate salt levels, the role of these transporters is less clear. Indeed, the *nhx1/nhx2* double mutants are not sensitive to moderate external Na\(^{+}\) concentrations, yet they are sensitive to moderate external K\(^{+}\) concentrations, for review see [134]. Conversely, the trans-Golgi network-localized NHX double knockouts, *nhx5/nhx6*, highly respond to moderate salinity and interfere with vesicle trafficking to the vacuole. This suggests that the endosomal NHXs are more sensitive to Na\(^{+}\) accumulation than vacuolar NHXs. This difference has implications on Na\(^{+}\) tolerance in plants. Recently, another CPA family member, a cation/H\(^{+}\) exchanger (CHX), *GmSALT3*, was shown to improve shoot Na\(^{+}\) exclusion and salt tolerance in soybean [136]. Fluorescent protein fusions suggested that *GmSALT3* and other CHX proteins are localized to the endoplasmic reticulum, further indicating that endosomal NHXs could be more reliable in sensing abnormal Na\(^{+}\) levels in the cell and has a positive implication on salt tolerance in plants.

Other genes encoding for Mannose-1-phosphate guanyl transferase (*OsMPG1*) and the rice homologue of Shaker family K\(^{+}\) channel *KAT1* (*OsKAT1*) have also been reported to confer salinity tolerance [137, 138]. *OsMPG1* is an important enzyme for the biosynthesis of ascorbic acid in plants, whereas *OsKAT1* reduces the cellular Na\(^{+}\) to K\(^{+}\) ratio by increasing the cellular K\(^{+}\) content. Another rice potassium transporter (*OsHAK5*) was shown to accumulate more K\(^{+}\)
and less Na⁺ when constitutively expressed in *Nicotiana tabacum* cv. Bright Yellow 2 under salinity stress [198]. Several other genes were recently identified by Chen et al. [139] while studying the halophyte seashore Paspalum (*Paspalum vaginatum*).

Another process, downstream of transcriptional regulatory networks, involves accumulation of sufficient solutes (e.g., proline and glycine betaine) to balance extra osmotic pressure in the soil solution to maintain turgor [140]. Moreover, plants can also accumulate sufficient Na⁺ and Cl⁻ to balance those in the soil solution, but this is tightly controlled through strict ionic regulation in various cell compartments (‘tissue tolerance’). These tolerance strategies are achieved through a series of ion transporters and their localization in key cell types. Na⁺/H⁺ anti-porter proteins are the key regulators of these tolerance strategies. Examples include TaHKT1;5-D protein, which maintains high cytosolic K⁺/Na⁺ ratios in bread wheat shoots by restricting Na⁺ loads in the root xylem before entering the shoot [141]. Recently, the introgression of the *Triticum monococcum* HKT1;5-A into durum wheat improved shoot Na⁺ exclusion and improved grain yield in the field by 25% [142], indicating the significance and functional stability of these transporters even in interspecific hybrids. Additionally, Eswaran et al. [143] used the yeast Full-length CDNA Over-eXpressor (FOX) gene hunting to identify several salt-responsive genes in *Jatropha curcas*. The late embryogenesis-abundant protein (LEA-5), aquaporins and a cytosolic ascorbate peroxidase-1 (*Apx1*) were amongst the identified genes involved in salinity tolerance. LEA5 are group 5 LEA genes that have been shown to play roles in the combining of concentrated ions and dehydration [143]. This group of LEA proteins have attracted fewer investigations and will require further studies at salt stress conditions. Aquaporin proteins are members of a large multigenic family that regulates a large proportion of water transport across membranes. Aquaporins are rapidly influenced both transcriptionally and post-translationally, and enhance salt stress tolerance in plants. For instance, a plasma membrane intrinsic protein (*GmPIP1;6*, which belongs to a subfamily of aquaporin specifically located in the PM) in soybean increases shoot Na⁺ exclusion and improves the seed yield from a saline field [144]. Orthologous *PIP* proteins are found in *Arabidopsis*, tobacco, barley, rice and wheat. For instance, *GmPIP1;6* is the ortholog of *AtPIP1;2, NIAQPI, HoPIP1;6;1;1* and *TaAQP8*. Overexpression of *NIAQPI* in tobacco increases photosynthetic rate, water use efficiency and yield under salt stress [145]. Overexpression of *TaAQP8, TaNIP* and *TaAQP7* genes in *Arabidopsis* or tobacco also increases salt tolerance of transgenic plants [146–148]. Root stellar cells also confer control over shoot Cl⁻ accumulation [149]. The expression of *GmPIP1;6* in roots was recently shown to be correlated with rapid and longer term changes in root hydraulic conductance (*Lₜ*) in response to shoot treatments and appeared to be more concentrated in stellar tissue [150]. These results indicated that *GmPIP1;6* could be the protein responsible for the control of root water transport, particularly in response to shoot signals. More recently, overexpression of *GmPIP1;6* was shown to significantly increase salt tolerance of soybean by improving root *Lₜ* and Na⁺ exclusion, which provided additional evidence that *GmPIP1;6*’s activity is in the stellar tissue. However, as there is no conclusive interactive or independent role of AQPs in salt tolerance, AQPs could instead be playing an indirect role through their impact on osmotically driven water and solute flow in roots and leaves. Further research will probably provide clear insight as to whether *GmPIP1;6* is responsible for salt regulation in the stellar cells, and whether there are other co-factors involved.
Wheat tonoplast intrinsic protein (TIP2; 2) is also reported to enhance salt tolerance [151]. However, the functional role of this protein is regulated by methylation following salt treatment as is HKT1 in Arabidopsis [152]. This suggests that aquaporin methylation could also play a role in regulating salt tolerance in plants and is worth further exploration.

Accumulation of ROS scavenging enzymes has also been reported to lower cellular damage, maintain photosynthetic energy capture, and improve shoot and root growth under saline conditions. For instance, salt-stress-induced accumulation of SOD has been reported to play a protective role in Canola, S. europaea, S. chilense and K. candel [153–155]. Furthermore, expression levels of anti-oxidant enzymes APX (e.g., Apx1), Trx, Prx, GPX and GST were observed to be enhanced in Tangut nitraria [156] under salinity conditions. Moreover, the same authors have reported that a photosynthetic enzyme, Ferredoxin—NADP (+) reductase (FNR), activity also increased in T. nitraria. Pea plants grown under saline stress also showed an enhancement of both APX activity and S-nitrosylated APX, which suggests that APX plays a significant role in plant tolerance to salt stress. However, apart from ascorbic acid biosynthesis, which has been shown to be modulated by OsMPG1, the molecular regulation of most anti-oxidants in response to salinity remains to be explored.

The recent discovery that salt-tolerant plant growth promoting rhizobacteria (PGPR) populations reduce Na⁺ concentration in the plant shoots [157] provides further insights into plant tolerance to saline conditions. The PGPRs increase the expression of stress-responsive TFs, induce greater proline synthesis, enhance ROS scavenging and improve plant biomass under salinity stress. Therefore, treatment with rhizospheric organisms, and understanding the mechanisms associated with these PGPRs leading to salt tolerance, is an attractive option to improve crop yields under saline conditions.

Fundamental insights into genetic control of salt tolerance mechanisms have also led to identification of more than 100 QTLs in various crops including Arabidopsis, barley, rice and wheat, amongst others. The earlier mentioned salt overly sensitive (SOS) pathway genes and AtCIPK16 are amongst the salt tolerance factors spanning several QTLs identified [158]. CIPK16 is an SNF1-related kinase/CBL-interacting protein kinase, underlying a quantitative trait locus for Na⁺ exclusion in the Arabidopsis Bay-0×Shahadara mapping population. CIPK16 was also recently shown to be expressed in barley and improves Na⁺ exclusion and biomass in a saline field.

Taken together, several genes and proteins have been shown to enhance salt tolerance in plants. However, the limited number of genes with functional polymorphism for salt tolerance makes it difficult to employ marker-assisted breeding for salt tolerance traits. In addition, the complex molecular mechanisms underlying the difference between seedling and reproductive stage salt tolerance in plants, e.g. rice [159], suggest the need for further research. The importance of the apoplastic bypass flow in delivering Na⁺ to the xylem, thus reducing leaf Na⁺ concentration and improving tolerance as suggested by [160], is also worth exploring further. Moreover, more insights into the molecular regulation of salt response will provide avenues for combining tolerance mechanisms to develop varieties that are widely adapted to salt stress.
6. Advances in plant tolerance to submergence/flooding

Over the past 25 years, yield losses caused by flooding have been increasing in various parts of the world, including the United States, China, Europe, Pakistan and Australia [161, 162]. Flooding is expected to increase as a result of erratic weather patterns, including frequent and lengthy storms associated with climate change, and could severely affect food production if mitigation measures are not sought.

Generally, submergence/flooding stress results from reduced oxygen levels in the plant root zone due to the low diffusion rate of oxygen in water. Submergence inhibits electron flows that underpin photosynthesis and aerobic respiration from the air causing energy shortfalls that can prove injurious to the plant [162]. Flooding also leads to accumulation of gases such as ethylene and carbon dioxide by preventing their diffusive escape and oxidative breakdown [163]. A high concentration of ethylene limits root extension, while carbon dioxide can severely damage plant roots. Trapped carbon dioxide may also form bicarbonate ions that can accentuate the effect of high lime content, leading to iron unavailability and chlorosis. The hypoxic environment also leads to restricted production of ATP, forcing cells to rely on glycolysis and fermentation to generate ATP and regenerate NAD+ to cope with the energy crisis [164]. Moreover, survival through prolonged inundation hypoxia involves the use of inorganic pyrophosphate (PPi) as an alternative energy source and induction of enzymes that reduce reactive oxygen species (ROS) or cytoplasmic acidosis, which are equally energy consuming processes. Because translation is a tremendously energy-intensive process, protein synthesis is affected in such oxygen-deprived conditions. Subsequently, essential metabolic processes slow down affecting the overall growth of the plant. In rice, soybean and wheat, various deleterious effects have been observed, such as suppression or reduction of hypocotyl and root elongation, and suppression of lateral root development [162, 164, 165].

Plant tolerance to submergence/flooding is generally a metabolic adaptation in response to anaerobiosis that enables cells to maintain their integrity so that the plant survives hypoxia without major damages. Several defence-related changes occur in submergence tolerant plants, including anatomical (e.g. formation of higher aerenchyma tissue in the nodal region in rice), physiological (more shoot elongation) and biochemical (inhibition of chlorophyll degradation, less utilization of storage carbohydrates and increased activity of anti-oxidative enzymes). At the molecular level, plants need to adapt these several changes in their gene expression profiles as well as cellular protein profiles. We will focus more on molecular adaptation, with a preference for adaptive QTLs, genes and proteins of significance to crop tolerance to flooding.

One of the early responses to submergence involves the differential regulation of a suite of TFs belonging to the ethylene response factor (ERFs) gene family. In rice, a major QTL locus belonging to ERF family, which is responsible for submergence tolerance, was mapped to chromosome 9, designated as Submergence1 (Sub1) [166]. This QTL was reported to account for about 70% of the phenotypic variation under submergence [167]. One of the genes adhered to Sub1 locus is Sub1A, which limits shoot elongation during submergence by repressing gibberellic acid (GA) levels and modulating GA signaling. In the process, the consumption of
energy reserves is reduced, and upon de-submergence, genotypes with \textit{SUB1A} are able to resume development when flood water subsides.

Two \textit{ERF}s, \textit{SNORKEL1 (SK1)} and \textit{SNORKEL2 (SK2)} from Thai deep water accession C9285, also confer submergence adaptation in deep water rice by inducing rapid internode elongation [168]. SKs have also been found in the genomes of accessions of wild \textit{O. rufipogon} from Asia and \textit{O. glumaepatula} from South America but missing in most cultivated rice varieties, which suggests that an ancient genomic region of \textit{Oryza} was lost during the establishment of rice grown in shallow paddies, but was safeguarded in deep water ecosystems. More recently, two QTLs on chromosome 3 and 12, including \textit{O. sativa-GROWTH-REGULATING FACTOR7 (OsGRF7)}, were reported to be involved in GA-dependent stem elongation and meristem maintenance in deep water rice [169]. OsGRF7 on chromosome 12 could probably be a regulator of GA responsiveness for internode elongation, whereas a QTL on chromosome 3 and other QTLs may regulate the \textit{DELLA} function or act downstream of GA signaling. The \textit{DELLA} proteins are the key regulators of GA signaling and suppress plant growth in the absence of GA.

In maize, a major QTL, \textit{Subtol6}, was also recently shown to be associated with submergence tolerance [170]. Based on the expression differences between the parent inbreds, \textit{subtol6} is associated with \textit{HEMOGLOBIN2 (HB2)}, a gene which was previously reported to be associated with plant survival in low oxygen or low ATP conditions [171]. The same authors indicate that haemoglobin proteins in maize repress ROS levels and maintain the energy status of maize cells during hypoxia. Other notable candidate genes, including genes related to \textit{ABA-INSENSITIVE3 (ABI3)/VIVIPAROUS1 (RAV1)}, genes related to accumulation and metabolism of carbohydrates, e.g., alpha subunit of \textit{PYROPHOSPHATE-DEPENDENT FRUCTOSE-6-PHOSPHATE 1-PHOSPHOTRANSFERASE (PFP)} and \textit{ALCOHOL DEHYDROGENASE1 (ADH1)}, have been reported to be highly upregulated in response to submergence [170].

In association with these tolerance genes, a number of other QTLs have also been identified in various crops, including barley, wheat, \textit{Brassica napus}, maize and \textit{Lolium perenne}, amongst others.

In addition to these QTL studies, several proteins have been reported to enhance submergence tolerance in plants. Enzymes involved in primary metabolism are differentially regulated in response to flooding. For instance, UDP-glucose dehydrogenase, UDP-glucose pyrophosphorylase, \(\beta\)-glucosidase G4 and rhamnose synthase, aspartate aminotransferase and lipoygenase have been reported as early flood-responsive proteins in rice and soybeans [164, 172]. The same authors indicate that phenlypropanoid pathway and cell wall synthesis enzymes decrease in abundance during flooding, which could be an energy-conserving adaptive strategy towards enhanced flooding tolerance.

Together these findings suggest that during flooding several processes are inhibited to reduce energy consumption. It is crucial for the plant to preserve some carbohydrates for release of energy to support further growth when the water level recedes. The regulatory genes in this category may also serve some ABA-mediated water stress recovery and inhibition of GA-induced internodal elongation as quiescence strategies adopted by plants [173]. On the other
hand, avoidance mechanisms employed under deep water conditions involve rapid internode elongation. In *R. palustris*, there are populations that show either the quiescence response or the avoidance response to submergence. This divergence shows that quiescence and avoidance are two strategies that can be employed by plants depending on the duration of flooding. Quiescence can be the optimal strategy for short-duration ‘flash’ floods, whereas avoidance via growth could be more reliable in prolonged deep flooding. Notwithstanding the above-mentioned tolerance genes and proteins, a deeper insight into the molecular regulation of quiescence and avoidance, and the associated regulatory networks, is still needed to provide sustainable avenues for improving plants specific to either flooding condition or able to grow in both.

7. Advances in plant tolerance to nutrient imbalances

7.1. Tolerance to nutrient deficiency

A total of 21 mineral nutrients are essential for crop growth and development. Most nutrients in the soil are primarily generated from the weathering of the parent material in the Earth’s crust. Moreover, nutrient levels can vary widely across locations because of initial influence of the composition of the parent material. In most cases, inadequate replenishment from the parent material and from the adsorbed and complexed fractions causes nutrient deficiencies in the soil. In addition, natural factors, including acidity, alkalinity and human activities such as inadequate fertilization also cause nutrient deficiencies. In countries such as India and China, mineral deficiencies have significantly stagnated or limited crop yields. More than 30% of agricultural soils are boron deficient, not only in China and India, but in the whole world. Moreover, zinc deficiency is even more widespread, affecting approximately 50% of the soils. Significant zinc deficiencies occur in sub-Saharan Africa, Turkey, Iran and Pakistan [174].

Several studies have been conducted on understanding plant nutrition; the most noteworthy being the work of the German scientist Justus von Liebig, who stipulated that plant growth is controlled not only by the total resources (nutrients) available, but also by the scarcest resource (the limiting factor). This submission has stimulated a series of studies on nutrient management, including plant breeding for tolerance to nutrient deficiencies. Tolerance to nutrient deficiency is associated with the genotype’s nutrient use efficiency. Genotypic variation in nutrient use efficiency is closely related to root nutrient acquisition capacity and utilization. In this section, we will focus on nitrogen and phosphorus, the two most limiting nutrients that are essential for several biological processes in plants.

7.1.1. Plant tolerance to nitrogen deficiency

Nitrogen is the most limiting nutrient to plant growth in most ecosystems despite its abundance in the atmosphere. This problem occurs because most plants can only take up nitrogen in two solid forms: ammonium ion (NH$_4^+$) and nitrate ion (NO$_3^-$). Ammonium is used less by plants because it is extremely toxic if taken up in large concentrations, so inorganic nitrate is the most usable form obtained by plants from the soil solution. Nitrogen-deficiency effect on
crop yields depends on the growth stage at which it occurs, as well as on its duration and extent [175]. However, reduced radiation interception, low radiation use efficiency, poor dry matter partitioning to reproductive organs, reduced leaf area index and decreased protein content of the plant and seed are the common effects of nitrogen deficiency.

Plants react in many different ways to changes in N provision; and physiological and molecular components governing N uptake, assimilation and remobilization during the plant life cycle have been studied extensively in the past decades, for review see [176, 177]. Three types of responses have been recently unraveled: (i) regulation of root N uptake systems, (ii) plasticity of root system architecture and (iii) fast modulation of shoot growth [178]. The first two responses generally improve efficiency of root N uptake under deficient conditions. The upregulation of specific high-affinity membrane transporters and enhanced foraging by the root system are implicated in these responses. When soil conditions for N uptake are seemingly unfavourable, e.g. limited water availability, plants will quickly slow down the overall N demand, as a nutrient conserving adaptive strategy, to prevent N starvation until conditions for N uptake become favourable.

In various plant species, nitrate transporters play a dominant role in N uptake. In Arabidopsis, three major families of nitrate transporters have been identified: Chlorate resistant 1 (CHL1/NRT1), NRT2 and chloride channel (CLC) [177]. NRT2 belongs to the high-affinity nitrate transporter group while most of the NRT1 family members belong to low-affinity nitrate transporters. The only exception, so far, in the latter group is NRT1.1 that is a dual affinity nitrate transporter. Thus, the high-affinity transporters that have been identified and primarily associated with nitrate uptake from the external environment include NRT1.2, NRT2.1, NRT2.2 and the dual affinity transporter, NRT1.1.

NRT1.1 is functionally regulated by phosphorylation of a threonine residue, Thr101, which facilitates the switching of its activity from a low- to a high-affinity state. AtNRT1.1, which is also induced by auxin and is itself an auxin influx facilitator, is a dimer in the asymmetric unit cell despite being monomeric in solution. At low nitrate levels, AtNRT1.1 is phosphorylated at the dimer interface, dissociates the NRT1.1 dimer, changes into a high-affinity transporter and represses lateral root (LR) development by promoting basipetal auxin transport out of LR primordia (LRP) [179]. At high nitrate levels, NRT1 1 is dephosphorylated, adopts a dimeric structure and adapts a low-affinity transporter configuration. In this state, trafficking of auxin out of the LR is blocked, and auxin accumulates in the LR initials promoting LR development. NRT1.1 is also shown to act upstream of the MADS box ARABIDOPSIS NITRATE REGULATED1 (ANR1) when modulating LR growth [179]. ANR1 mediates localized N response and modulates the proliferation of LRs in N-dense patches. Moreover, NRT1.1 has been shown to regulate genes encoding for other nitrate transporters, including NRT2.1 and NRT3.1 [180]. However, NRT1.1 and NRT2.1 are localized in different cell layers in the roots, and their adaptive/complementary strategy in nitrate uptake is not well elucidated. The NRT1.1-auxin modulation and nitrate signaling has also been a topic of interest and requires elucidation [181].

Amongst the CLC family members, CLCa and CLCb function as proton-nitrate exchanges, and have high selectivity for nitrates over chlorides [182]. Both transporters are known to mediate
nitrate accumulation in the plant vacuoles. Besides the above-mentioned transporters, the acquisition of nitrate is also regulated by slow anion channel (SLAC1) and SLAC1 homologue (SLAH) and nitrate excretion transporter (NAXT-1). Five SLAC genes were previously reported in Arabidopsis. Amongst these genes, SLAC1 and SLAH3 show nitrate transport activity, but their channel activity is co-regulated by kinases (e.g., CPK21) [183]. An efflux component operated by NAXT-1, associated with the nitrate transporter 1/peptide transporter (NRT1/Ptr) family of proteins, mediates nitrate efflux under acid load in the cytosol [184]. Similarly, NRT1.5, which loads nitrates into the xylem for root-to-shoot translocation, also mediates nitrate efflux. However, the proton-coupling mechanism of NAXT1 remains to be elucidated. Two other transporters, NRT1.8 and NRT1.9, have been reported to regulate root-to-shoot nitrate translocation [185, 186]. Both transporters are apparently negative regulators of root-to-shoot nitrate transport. The subsequent nitrate allocation into the vegetative tissues, reproductive tissues and osmotic regulation of guard cells is reasonably described elsewhere [187].

Further studies on signaling, transcriptional and post-translational regulation have revealed evidence that a CBL-interacting protein kinase, CIPK8, regulates the activity of nitrate transporters and the expression of nitrate assimilation genes [188]. Like CIPK8, CIPK23 is also suggested to be activated by a CBL protein, CBL9, but the exact mechanism is elusive. CIPK23 directly interacts with NRT1.1 in the plasma membrane and phosphorylates NRT1.1 at Thr101 to adopt a monomeric structure when the nitrate concentration is low. This process helps plants to adapt to low nitrogen levels.

Several TFs have been implicated in regulating NRT1.1 activity. For instance, the activity of two bZIP TFs in Arabidopsis, ELONGATED HYPOCOTYL5 (HY5) and HY5-HOMOLOG (HYH), was suggested to positively modulate NITRATE REDUCTASE2 (NIA2) and negatively modulate NRT1.1 [189]. The NODULE INCEPTION (NIN)-like TFs have also been shown to play a central role in the regulation of nitrate-inducible genes [190]. Nitrate signaling activates NIN-like transcription factors through their N-terminal regions. The activated factors promote the expression of nitrogen assimilation-related genes and genes encoding regulatory proteins. NLP7 is the most reported in this family of TFs. NLP7 is strongly induced in vascular tissues and root hairs, and is required for the induction of several nitrate uptake and assimilatory genes. Thus, NLP7 is is probably a key regulator of nitrogen utilization mechanisms. More recently, the presence of nitrate in the external solution induced the expression of NRT accessory proteins (NAR), nitrate reductase, nitrite reductase and genes involved in the GS-GOGAT cycle, in Arabidopsis, as well as in maize and other plants [191]. These proteins likely play a role in nitrate sensing.

Strigolactones (SLs), a new class of plant hormones and rhizosphere signaling molecules, also appear to be upregulated in plants under low N conditions [192]; however, the impact of SL levels on root growth is yet to be determined. Changes in root system architecture (RSA) may also be induced depending on the prevailing available organic form of nitrogen, for review see [118]. The most commonly reported organic forms are l-glutamate or carnitine. In Arabidopsis seedlings, l-glutamate inhibits cell division in the root apical meristem (PRM) of the primary root (PR) tip and promotes LR formation and outgrowth. However, several
Arabidopsis auxin-signaling mutants display different levels of sensitivity to l-glutamate, suggesting that l-glutamate is rather a signaling molecule as opposed to a nitrogen source [193]. In addition, the rice glutamate receptor mutants display a host of RSA changes, including short PR and LR, reduced cell division and the cell death of root apical meristem [194], further suggesting that l-glutamate could be a major anchor in the signaling process leading to nitrate uptake and assimilation. This is supported by previous studies that have shown that glutamine synthetase (GS1) from alfalfa causes an increase in photosynthesis and growth under low N fertilization regime [195]. Glutamine synthetase also mediates ammonium assimilation into glutamine. Ammonium form of nitrogen is rapidly assimilated into organic nitrogen forms to avoid tissue toxicity, for review see [196]. Several other reviews have documented the genes and proteins regulating nitrogen use efficiency (NUE) in plants. The reader is referred to excellent reviews by [177, 196]. In addition, more than 50 QTLs for nitrogen use efficiency have been reported in plants, though few of them have been validated. Amongst the identified QTLs are nitrogen deficiency response QTLs in rice, nitrogen supply responses and yield in wheat and nitrogen use efficiency in barley.

Collectively, nitrogen use efficiency in plants is controlled by a complex array of physiological, developmental and environmental interactions that are specific to the genotype of a given species. Notwithstanding the aforementioned N uptake and utilization genes and QTLs, an extensive molecular survey of a wide range of genotypes covering the genetic diversity of a crop could provide further evidence on the genetic control of these trait. This can be achieved using the various available ‘omics’ techniques, combined with agronomic and physiological approaches in order to identify more elements controlling NUE in plants, both universal and specific, for use in crop improvement.

7.1.2. Plant tolerance to phosphorus deficiency

Phosphorus (P) is the second most limiting mineral nutrient in almost all soils, and >30% of the world’s arable land has low P [197]. Phosphorus availability is particularly limiting on highly weathered acid soils of the tropics and subtropics due to its fixation by Al and Fe oxides on the surface of clay minerals. Plants take up phosphorus as phosphate (Pi), either directly by the root system or transferred through the fungal symbiont in arbuscular mycorrhizae host plants. Plants have elaborate sensing and signaling mechanisms in response to Pi deficiency, and both local and systemic signaling in response to Pi deficiency have been reported [197]. Key responses in the plant include changes in the root system architecture (RSA), a reduction in photosynthetic rate; increased activity of high-affinity Pi transporter activities; secretion of APases, ribonucleases and organic acids; membrane phospholipid replacement with glycolipids and sulfolipids; and increased availability of anthocyanin and starch [198]. Putative signaling molecules in response to Pi deficiency include sugars, hormones and microRNAs.

Under limiting Pi conditions, plants can monitor Pi deficiency both locally and systemically, and root foraging strategy to explore top soil layers for Pi is employed. The Pi foraging strategy is accomplished through one of the several different RSA and physiological changes [118]. The local external Pi rather than the systemic Pi status of the whole plant regulates the remodelling of RSA [199]. In maize and some species in the Proteaceae and Casuarinaceae families, the
remodelling of RSA involves production of adventitious roots and cluster roots [200, 201], which increases root surface area for Pi absorption. While a plant Pi receptor is yet to be identified, recent reports have suggested that ethylene biosynthesis and signaling are involved in the Pi-deficiency-triggered remodelling of RSA, for review see [118, 195]. The evidence is supported by previous finding that inhibition of ethylene biosynthesis with 2-aminoethoxyvinyl glycine (AVG) or ethylene perception with Ag⁺ restricted the low Pi-induced meristem exhaustion of the primary root [202]. Correspondingly, application of Ag⁺ was found to reduce the inhibition of primary root growth triggered by Pi deficiency. Moreover, Pi deficiency induced the formation of aerenchyma in adventitious roots, which is similarly induced by ethylene perception.

At the transcriptional level, Lei et al. [203], using an Arabidopsis transgenic line that carries a LUC gene fused to the promoter of the high-affinity Pi transporter, AtPT2, showed that the transcription of AtPT2 is induced by Pi starvation. Using this marker line, the authors identified the Arabidopsis mutant etr1/hps2 (constitutive triple response 1/hyper-sensitive to Pi starvation2), which showed hyper-induction of the AtPT2::LUC gene by Pi deficiency. Interestingly, the expression of AtPT2 was partially blocked in ethylene insensitive 2 (ein2) mutants, but was enhanced in ethylene over producer1 (eto1) mutants. A similar expression pattern was observed for several other Pi starvation-induced (PSI) genes in the hps2 (negative regulator of ethylene response) and ein2 mutants, including high-affinity phosphate transporter, AtPT1 (Pht1;1); a non-coding transcript, At4; an APase, ACP5; a ribonuclease, Rxlink; and miR399d [204]. Enhanced transcription of PSI genes was also observed in the mutant hps3 and hps4, which are ETO1 alleles [205, 206]. ETO1 protein is a member of the broad complex/tramtrack/bric-a-brac (BTB) protein superfamily that participates in substrate recognition during ubiquitin-mediated protein degradation [204, 207]. ETO1 directly binds to the C-terminal of ACS5 and mediates its degradation. When ETO1 is mutated, it causes an overproduction of ethylene in young seedlings [208]. Application of 25 μM ACC to young Arabidopsis seedlings under high Pi conditions barely induces the expression of AtPT2. Under Pi deficiency, however, 0.5 μM ACC dramatically increases AtPT2 expression and induces ectopic root-hair development [203]. Thus, these results provide evidence that ethylene production and signaling is involved in the transcriptional responses of plants to Pi deficiency and primarily integrates with other Pi-deficiency-induced signaling pathways.

The other signaling component involving increased transcription of purple acid phosphatase 10 (AtPAP10) by Pi starvation in the whole seedlings of hps3 and hps4 has been reported [205, 206]. AtPAP10 is a Pi starvation-induced APase (enzymes that scavenge Pi from organophosphate compounds) associated with the root surface. Functional analyses of atpap10 mutants suggest that AtPAP10 is important for plant tolerance to Pi starvation. However, the transcription of AtPAP10 does not significantly increase in ACC-treated seedlings or the constitutive triple response 1 (ctr1) mutant under Pi deficiency, nor does the accumulation of AtPAP10 proteins, which could suggest that ethylene has no effect on AtPAP10 transcription. More recently, Zhang et al. [209] have shown that positive regulation of AtPAP10 depends on sucrose and not ethylene. Moreover, they have also shown that ethylene does not affect AtPAP10 activity without sucrose, but the opposite is true. This suggests that ethylene could be a local
but indirect signal for AtPAP10 activity. However, as discussed before, ethylene could be regulating other components of Pi starvation response at the transcriptional level. Song and Liu [204] have demonstrated that accumulation of anthocyanin is lower in hps2, hps3 and hps4 mutants under low Pi but increases in Pi-starved ein2 mutants. As mentioned before, accumulation of anthocyanin is an indicator of Pi-deficiency response in plants, thus ethylene could be a negative regulator of Pi-deficiency-induced anthocyanin accumulation probably through the regulation of genes involved in anthocyanin synthesis. Thus, ethylene likely participates at both the transcriptional and post-transcriptional levels, and this has implications on Pi starvation response in plants.

The systemic response to P starvation is also carried out through a complex signaling network that involves other plant hormones [210, 211], sugars [212] and nitric oxide [213], collectively resulting in the alteration of carbohydrate distribution between roots and shoots. Amongst the plant hormones, other than ethylene, auxin likely plays a role in response to Pi starvation. However, ethylene likely exerts its influence through regulating auxin activity, as it has been associated with RSA remodelling [198]. Indeed, ethylene has been reported to interact with auxin and sugars, and changes in auxin transport and localization appear to be at least partially responsible for Pi stress-induced LR development [214]. Decreased sensitivity to CK and GA also appears to be at least partially responsible for Pi-stress-induced LR development [215]. Under low Pi, GA has been shown to repress Pi-induced root architecture changes [216]. Moreover, Pi-deficient plants were shown to accumulate DELLA proteins, the negative regulators of GA-induced root growth, which are modulated by auxin.

As discussed before, amongst sugars, sucrose is key to Pi-deficiency response and appears to regulate ethylene activity. Amongst the TFs, phosphate starvation response proteins (e.g., OsPHR1, OsPHR2, PpPHR1, ZmPHR1 and TaPHR1), which bind the promoter sequences of low Pi-induced genes, and their regulator SMALL UBIQUITIN-LIKE MODIFIER1 [AisIZ1; 217], a small ubiquitin-modified E3 ligase, and the downstream PHOSPHATE2 (PHO2), an E2 conjugase, are involved in Pi-deficiency-related transcriptional changes. Other TFs, including the bHLH, PTF1 (e.g., OsPTF1 and ZmPTF1) and MYB2P-1 (e.g., OsMYB2P1), MYB62, WRKY (e.g., WRKY75, WRKY6), bHLH32 and ZAT6 are also involved in the signaling network to regulate plant adaptation to P stress, for review see [218].

Based on genetic analysis, two proteins, the P5 type ATPase encoded by PHOSPHATE DEFICIENCY RESPONSE2 (PDR2), and multicopper oxidase LOW PHOSPHATE ROOT1 (LPR1), were also previously shown to modulate Pi signaling in an endoplasmic-recticum-localized pathway [219]. PDR2 is required for maintaining the levels of the root patterning gene, SCARECROW (SCR), and SHORT-ROOT protein (SHR) trafficking from stele into endodermis. PDR2 was proposed to act upstream of LPR1/LPR2 to adjust meristem activity. A recent study has shown that LPR1 is a ferroxidase [220]. Mutation of LPR1 reduces Fe3+ levels in the meristemic tissues of Pi-deficient plants. In contrast, increased levels of Fe3+ have been reported in pdr2 mutants leading to high production levels of reactive oxygen species (ROS). ROS signaling increases deposition of callose, which has been suggested to impair the trafficking of SHR, thus restricting root tip growth. Thus, PDR2 appears to modulate Pi-deficiency response by limiting Fe3+ accumulation in root tips.
More recently, molecular mechanisms defining the phosphate signaling pathway showed that phosphate uptake 1 (Pup1)-specific protein kinase gene, named phosphorus-starvation tolerance 1 (PSTOL1), was confirmed to be involved in regulating root growth and architecture during early stages of rice growth [221]. Allele-specific markers for this gene have been reported recently [222]. Interestingly, OsPSTOL1 is located within the Kasalath-specific INDEL region and is absent from the rice variety Nipponbare reference genome. Thus, the configuration of the functional mechanism of PSTOL1 is still elusive. We speculate that PSTOL1 could be a local sensor of Pi starvation which transduces signals for sucrose or ethylene biosynthesis or both. The interplay of sucrose accumulation and ethylene biosynthesis is apparently the hallmark of Pi starvation response in plants.

The post-transcriptional regulation as well as long-distance signaling is carried out by microRNAs. As mentioned before, miR399, which is regulated by PHR1, a conserved MYB TF, maintains P homeostasis by regulating P transporter PHO2 [223]. In tomato, overexpression of Arabidopsis miR399 increases both the Pi accumulation and secretion of acid phosphatase and protons in the roots [223]. Thus, miR399 is important for Pi acquisition, and could be acting downstream of sucrose and probably ethylene. Overexpression of miR399 in Arabidopsis also increases P uptake and allocation to the shoot. Moreover, P remobilization from older leaves to young leaves is defective in Arabidopsis miR399 transgenic lines [224]. This suggests that miR399 is important for allocation and remobilization of P. The targets of miR399 include a ubiquitin-conjugating E2 enzyme (UBC24) encoded by PHO2, which is upregulated under P-sufficient conditions and downregulated in P-starved plant roots. Homologues of PHO2/UBC24 have a conserved structure in many species, and their 5’ UTR regions possess multiple miR399-complementary sequences. Thus, the regulatory mechanism of miR399-PHO2 complex is evolutionarily conserved in angiosperms, making it a potential target for improving P nutrition efficiency in plants.

Strigolactones (SL) have also been shown to be induced by low Pi in many species, including tomato, Arabidopsis, pea and rice [225–229]. Strigolactones are terpenoid lactones that function as either endogenous hormones that control plant development or as components of root exudates that promote symbiotic interactions between plants and soil microbes. The production and exudation of SLs may depend on whether the plant is arbuscular mycorrhizal fungi (AMF)-compatible host or an arbuscular mycorrhizal symbiosis (AMS) for Pi and N uptake. A well-known synthetic SL, GR24, apparently increases LR formation under low Pi or decreases LR formation under sufficient Pi. In addition, SL biosynthesis (more axillary growth; max4-1) and signaling (max2-1) mutants have reduced number of root hairs under low Pi condition at the early stages of seedling development. This suggests that SLs mediate plant responses to low Pi; however, the mechanism by which SL exudation affects root growth is not fully understood.

In conclusion, although the molecular components of P stress signaling in plants have been fairly documented, the overall pathway is still less understood and requires further investigation. Nonetheless, the recent developments in whole genome sequencing technologies provide hope for more studies on plants with better P acquisition and utilization. Successes in QTL analysis have also set a stage for subsequent studies. Besides the success story of PSTOL1...
in rice. QTL analysis in common bean has shown the importance of basal roots and adventitious roots for P acquisition [230–232]. Another study by Yan et al. [233] identified a large number of QTLs for Hp exudation, root-hair density and length, associated with P efficiency. Additionally, QTLs for root traits related to P efficiency have also been identified in soybean [234, 235]. Moreover, QTLs controlling P deficiency tolerance were mapped by Zhang et al. [344] using 152 RILs derived from a cross between P-stress-tolerant and P-stress-sensitive parents. Thus, future studies will build on these present discoveries to facilitate genetic improvement for Pi-deficiency tolerance.

7.2. Advances in plant tolerance to nutrient toxicities

Metal toxicity is an important factor limiting the growth of plants in many environments. Some metals, such as copper and zinc, are micronutrients at low concentrations and become toxic at higher levels, whereas others (e.g., aluminium, iron, cadmium, chromium and lead) are well known for their toxicity [236]. These elements can be highly phytotoxic and seriously impair plant root growth. However, some crops are able to tolerate toxic environments, without significant display of toxicity symptoms. Three main strategies are employed by such plants to manage toxic soil compounds: (1) Producing root exudates that bind and neutralize the toxin in the rhizosphere, (2) actively transport the compound into the root, but neutralizing and sequestering it in vacuoles for safe accumulation or eliminating it through exudation and (3) excluding the toxic elements by preventing entry into the plant tissues. For the purpose of this chapter, we will focus on aluminium and iron toxicities as these elements have been frequently reported as major constraints in the production of economically important crops.

7.2.1. Plant tolerance to aluminium toxicity

Aluminium (Al) is a light metal that makes up 7% of the Earth’s crust and is the third most abundant element after oxygen and silicon. Aluminium toxicity is one of the major constraints to crop productivity worldwide, especially in the acid soils of the tropics and subtropics that comprise almost 50% of all non-irrigated arable land in those regions [118, 237]. The soil pH has a crucial role for Al toxicity to occur, by affecting both solubility and the ability of plant roots to absorb Al. Al solubilizes into its toxic form (Al$^{3+}$) when the soil pH drops to 5.5 or less, and is most severe in solutions of low ionic strength and low cation concentrations. Al$^{3+}$ is taken up by plants through diffusion [238], and toxic concentrations of >12 μM are detrimental to root growth. Possible exceptions of Al(OH)$_3$ toxicity at higher pH values have also been reported [239].

The initial effects of Al$^{3+}$ toxicity on the roots include rapid inhibition of cell division and a reduction in root apical cell expansion and elongation. Consequently, plants develop stubby and brittle roots with swollen malformed root tips. Moreover, lateral root initiation and outgrowth are also inhibited. Root-hair malformation is often reported, and nutrient (mainly P, K, Ca and Mg) and water uptake capacity is impaired [238]. Plant responses in the shoots include reduced stomatal opening, chlorosis, foliar necrosis and reduced photosynthetic activity.
Plant tolerance to aluminium toxicity occurs through (1) external avoidance, which involves root secretion of organic acids to chelate $\text{Al}^{3+}$ in the rhizosphere, limiting its diffusion into the roots [240], and (2) true or internal tolerance, which involves regulation of $\text{Al}^{3+}$ uptake, and organic acid chelation and sequestration of aluminium bound substrates [241]. In rice, the latter is the main tolerance mechanism, and is apparently associated with the differential expression and transport properties of membrane transporters, e.g., NRAMP $\text{Al}^{3+}$ transporter 1 (NRAT1) [242]. Most other plant species also vary significantly in these mechanisms; however, there are some tolerance mechanisms that are largely shared. Cereal crops, such as wheat, barley, sorghum (*Sorghum bicolor* L.) and oat were reported to have simple genetic mechanisms of Al tolerance, whereas rice and maize (*Zea mays* L.) have over time developed complicated inheritance controlled by numerous genes/loci involved [118, 243].

Genetic control of organic acid exudation either rests on the Multidrug and Toxin Efflux (MATE) family encoding a citrate transporter or on the membrane localized $\text{Al}^{3+}$-activated malate transporters (ALMT). Several transporters in these families, including *HvAACT1* in barley [244], *TaALMT1* and *TaMATE1* in wheat [245] and *ZmMATE1* and *ZmMATE2* in maize [246] are responsible for organic acid exudation and Al tolerance. Specific markers for *HvAACT1* and the MATE gene, *HvMATE-21*, have been developed and can be used to differentiate tolerant and sensitive barley cultivars. Differences amongst these transporters however exist. For instance, *TaALMT1* encodes a malate transporter on chromosome 4D and is constitutively expressed on root apices, whereas *TaMATE1* reportedly responds to Al stress based on citrate efflux. *ZmMATE1* and *ZmMATE2* co-segregate with two major Al-tolerance QTLs [247]. *ZmMATE1* was shown to be induced by Al and enhances Al tolerance, whereas *ZmMATE2* did not respond to Al [246], suggesting variability in their roles. In sorghum, Al tolerance is controlled by *SbMATE*, encoded by a major Al-tolerant locus *Alts* on chromosome 3 [248]. In *Arabidopsis*, two genes were reportedly responsible for Al tolerance: *AtALMT1* that also encodes a malate transporter responsible for malate efflux on chromosome 1 [249] and *AtMATE* that encodes an Al-activated citrate transporter [389]. These two genes function independently, but both are regulated by the C2H2-type zinc finger transcription factor *STOP1* [250], which is also reportedly induced by with low pH tolerance [366]. In rye, *ScALMT1*, which is mainly expressed in the root apex and upregulated by Al, co-segregates with the *Al4* locus on chromosome 7RS [367]. Another candidate gene *ScAACT1* on chromosome 7RS was mapped to 25 cM from *ScALMT1* [251].

At the transcriptome level, two genes, *SENSITIVE TO ALUMINUM RHIZOTOXICITY1* and 2 (*STAR1* and 2), which encode the nuclear binding domain and the transmembrane domain, respectively, of an ABC transporter, with specificity for uridine diphosphate (UDP) glucose, are upregulated following root exposure to Al$^{3+}$ [252]. Both *STAR* genes were previously reported to be upregulated by the constitutively expressed rice root *ALUMINUM RESISTANT TRANSCRIPTION FACTOR1* (ART1), which also upregulates several other genes implicated in different aluminium tolerance mechanisms [253]. More recently, *ASR5* was reported to act as a key TF that is essential for Al-responsive *STAR1* and other Al response genes [254]. Rice homologues, which encode $\alpha$-expansin (e.g., *EXPA10*), belong to this family of TFs, and have been implicated in the regulation of root elongation and cell wall elasticity. The members of
EXPA10 decrease cell wall extension potential when exposed to Al\textsuperscript{3+} [255] and are downregulated during Al\textsuperscript{3+} stress. The functions of STAR1, STAR2/ALS3 and ALS1 in Al tolerance are fairly conserved and ubiquitous in monocot and dicot species. However, these genes are differentially expressed between species. For instance, the expression and induction levels of these genes in response to Al\textsuperscript{3+} stress are higher in the Al-tolerant species of rice than in the Al-sensitive species of Arabidopsis, suggesting that Al-tolerant species may require increased expression of these conserved Al-tolerance genes to overcome Al\textsuperscript{3+} stress [256]. The same authors show that Tartary buckwheat shows high expression of the Al-tolerance gene homologues under Al\textsuperscript{3+} stress. Al-tolerance in buckwheat is evolutionarily closer to Arabidopsis than rice, suggesting that buckwheat could have rapidly evolved higher expression of Al-tolerance genes to detoxify Al\textsuperscript{3+} than Arabidopsis. In addition, the gene duplication of ART1/STOP1, STAR1 and ALS1 has been suggested to play a significant role in Al tolerance. This is consistent with the previous findings that duplication of key genes responsible for metal translocation and detoxification in Arabidopsis halleri facilitates hyper-accumulation of zinc/cadmium [257]. However, further functional analysis by creating knockdown or knockout mutants will be necessary to provide additional insights into the role of each homologous gene in Al detoxification and accumulation in buckwheat.

An Arabidopsis cell-wall-associated putative endochitinase, CHITINASE A (AtCHIA), likely involved in modulating cell wall extension by regulating chitin levels, has also been suggested to play a role in Al tolerance [258]. Another signal of Al\textsuperscript{3+}-induced cellular response is the induction of 1,3-β-d-glucan synthase, which leads to the accumulation of callose in root apices, especially in endodermal and cortical cell walls [259, 260]. This callose deposition is suggested to be an inhibitory process that may block symplastic and apoplastic flows. Whether callose deposition represents Al\textsuperscript{3+}-induced injury or a defence response to block further Al\textsuperscript{3+} binding and movement remains to be confirmed.

In Arabidopsis, the ethylene receptor gene ETHYLENE RECEPTOR1 (ETR1) and the ethylene signal transducer ETHYLENE INSENSITIVE2 (EIN2) were found to be important for Al\textsuperscript{3+}-induced inhibition of root elongation [261]. These genes apparently regulate Al\textsuperscript{3+}-induced upregulation of the Arabidopsis ethylene synthesis genes 1-AMINOCYCLOPROPANE-1-CARBOXYLIC ACID SYNTHASE2, 6, and 8 and 1-AMINOCYCLOPROPANE-1-CARBOXYLIC ACID OXIDASE1 and 2. Moreover, ET was recently shown to negatively regulate aluminium-induced malate efflux from wheat roots and tobacco cells transformed with TaALMT1 [262], which suggests that ethylene could be a negative regulator of root secretion of organic acids. The upregulation of auxin transporters PIN FORMED2 (PIN2) and auxin influx carrier AUXIN RESISTANT1 (AUX1), which regulate auxin distribution, is associated with the regulation of root elongation in Arabidopsis plants [263]. AUX1 and PIN2 are apparently disrupted by ethylene signal that alters auxin distribution and transport in the roots. He et al. [264] suggests that auxin could be involved in aluminium-induced efflux of malic acid acting on anion channels. Thus, auxin/IAA transport could be a target for Al\textsuperscript{3+} toxicity tolerance if the modulation by ET is attenuated. However, considering several phytohormonal changes that occur during Al stress, molecular mechanisms associated with their interplay will require further
elucidation. Recent evidence that microRNAs are involved in Al stress tolerance [265] also provides new insights into understanding the mechanism of Al\(^{3+}\) tolerance in plants.

Overall, we expect that major advances in understanding physiological and molecular basis for Al tolerance will happen in the near future, considering that the pace at which new genes are being discovered has improved with new sequencing technologies. The future challenge for studying Al tolerance is the identification of new tolerance mechanisms. The discovery of the key molecular regulators, e.g., ASR5, which was recently shown to mediate Al-responsive gene expression to provide Al tolerance in rice, is an indication that several other mechanism of Al tolerance exist in plants. The blocking of Al\(^{3+}\) cell wall binding sites in rice may be one of the major mechanisms of aluminium tolerance that will need further investigation. Studies on barley, wheat and maize have shown variation in gene expression associated with variation in gene sequence, which would require further investigation to understand the regulatory networks affected by this sequence polymorphisms.

7.2.2. Advances in plant tolerance to iron toxicity

The problem of iron toxicity occurs in most wetland rice growing areas of the world, primarily in flooded acidic soils, inland and coastal swamps. Some of the irrigated lands in South and Southeast Asia, Africa and South America are affected [266]. In India alone, about 11.7 million hectares of land are affected by iron toxicity. In Burkina Faso, 300 ha of ferrous iron intoxicated soils were abandoned in the Valley du Kou in 1986, most of which remained uncultivated to date [267]. Iron toxicity is also becoming a major rice yield limiting factor in East Africa, including lowland rice cultivation areas of Uganda [268]. Yield losses in the range of 10% to 100% have been reported [266]. Moreover, toxicity at seedling and early vegetative stages can strongly affect plant growth and hinder development, and can result in complete crop failure.

Three major adaptation mechanisms are generally reported for Fe-toxicity tolerance. The details by which rice plants execute these processes and their molecular components are not yet fully understood, but there are some clues from various studies on rice and other plant species. For instance, plant tolerance by root oxidizing power is mediated by diffusion of molecular oxygen from the shoots to the roots through aerenchyma tissue and its subsequent release in the rhizosphere. Oxidation of Fe\(^{2+}\) in the rhizosphere results in the precipitation of insoluble iron oxides at the root surface, forming iron plaques. These iron plaques not only reduce Fe\(^{2+}\) concentration in the soil solution, but also form a physical barrier against further influx of Fe\(^{2+}\) into the roots.

Plant tolerance by retention of iron in the root or shoot involves compartmentalization. Nicotianamine (NA), Fe-NA complex transporters, VIT proteins, FPN2-like proteins, MIT- and PIC1-like proteins, organic acids, ferritins, Fe-sulphur and other heme proteins that can sequester Fe are all potential candidates for plant tolerance to excess iron through regulated storage and compartmentalization (Figure 6).

In Arabidopsis, apoplastic Fe is mostly found within the stele [269], suggesting that compartmentalization within the stele could restrict excess Fe from reaching the shoot during transport towards the aerial parts. Fe\(^{3+}\) decreases could also occur in association with an
alkalization of apoplastic pH, which reduces Fe$^{2+}$ mobility and chemical stability [269]. Alkalization has been reported to be modulated by ethylene [270], suggesting additional role
of ethylene in regulating Fe\textsuperscript{2+} besides its role in aerenchyma formation. Tissue tolerance of Fe toxicity is mediated by detoxification of free radicals. In rice, expression of several genes involved in oxidative stress control, including peroxidases, glutathione transferase (GST) and cytochromes, was upregulated in roots and shoots in response to excess Fe\textsuperscript{2+} [271]. Similar trends were observed at the protein and enzymatic activity levels of the same genes. Excess iron was reported to induce the activity of superoxide dismutase (SOD) and ascorbate peroxidase (APX) in the leaf sheath and laminae, respectively, in a tolerant variety from *Oryza glaberrima* [272]. The activity of glutathione reductase and peroxidase (POD) was also reported to increase in rice leaf segments exposed to excess iron [273]. Fang et al. [274] also showed that lipid peroxidation resulting from Fe toxicity was inhibited by free radical scavengers such as mannitol and GSH. Moreover, the differential expression of anti-oxidant enzyme activities (SOD, APX, CAT, GR and DHR) was observed between rice varieties contrasting in tolerance of Fe toxicity [275].

Several genetic studies also reflect that iron toxicity tolerance is a complex quantitative trait controlled by a large number of rather small effect quantitative trait loci (QTLs), indicating the involvement of multiple tolerance mechanisms. For instance, Wu et al. [276] identified QTLs for leaf bronzing and shoot dry weight on chromosome 1 and 8, explaining 10–32\% of the phenotypic variation. Interestingly, QTLs associated with enzymatic activity of anti-oxidants in rice leaves were detected in the same region [277]. Similarly, Fukuda et al. [278] detected a region on chromosome 3 responsible for high shoot iron content in a susceptible variety, which co-localize with the QTL previously identified by Shimizu et al. [279] for the same trait. Co-localization of most of these QTLs was captured in an integrative genetic map reflecting mapping studies from different conditions of Fe toxicity [277], which substantiates on recurrent chromosomal regions identified in several QTL studies.

A major limitation of iron toxicity tolerance studies, however, is that most of the QTLs associated with iron toxicity tolerance have not been furthered to cloning of tolerance genes. It is thus critical to devote some effort to fine-map the few, but consistent QTLs mentioned herein in order to increase precision and accelerate candidate gene identification. Subsequently, functional validation of several genes identified in microarray studies will need to be explored. Exploring allelic variation of these genes in contrasting genotypes and evaluating the promising alleles in well designed and efficient phenotyping experiments would provide a basis for their use in marker-assisted breeding (MAB) for Fe-toxicity tolerance.

### 8. Conclusions and perspectives

In this chapter, we have attempted to present the recent advances in crop tolerance to abiotic stresses. Various strategies used by plants to counteract stress, and some success in identifying genomic regions associated with plant tolerance is presented. Interestingly, plants have evolved common regulatory networks in response to abiotic stresses. For instance, drought, salt and cold stress induce calcium influx to activate the downstream second messengers to yield different or similar responses. Calcium influx channels at the membrane (e.g., the recently
reported *hyper-osmolality induced [Ca^{2+}]* increases 1 (OSCA1) from *Arabidopsis thaliana* that is gated by hyper-osmotic stress [28] act in concert with the membrane-located NADPH-oxidase Respiratory burst oxidase Homolog (RboH), generating apoplastic ROS. Intracellular transduction is conveyed by calcium-binding proteins (e.g., CBLs/CIPKs, CDPKs and calci-neurins), a MAP-Kinase cascade and phytohormones (e.g., ABA, ET, JA and SA), which apparently act as integrators of early signals. Depending on the relative temporal patterns of these upstream signals, the activity of TFs and their interacting proteins will decipher specific combinations of genes required to be expressed to boost enzymatic or protein reaction levels necessary to counter the stress perceived. These proteins largely contribute to adaptive response in most plants, e.g., production of compatible osmolytes that helps to reinstall turgidity during drought and synthesis of LEA proteins that prevent protein precipitation. Other examples include chelation/sequestering of ions into cellular compartments in response to toxic elements, induction of anti-oxidative enzymes, induction of molecular chaperones and adaptive regulation of plant hormones. These adaptive strategies and the molecular components involved provide potential molecular genetic targets for enhancing abiotic resistance in crops.

However, many challenges still lie ahead. For example, the regulation of signaling cascades, especially how plants can discriminate the signaling components, and even their specific combinations, to activate specific downstream biological processes for a given stress. A frequent manifestation has been the case of ethylene controversial role in abiotic stress response. Whether the negative regulations associated with ethylene represent a plant strategic mechanism to prime the subsequent useful reaction remains to be confirmed. Also, temporal and specific differences in activation of upstream signaling components will need to be explored to help in identifying molecular components essentially required to counter a given stress. Moreover, the specific downstream components for which much of the studies have been conducted, e.g. transcription factors, transmembrane proteins, transporters, enzymes for osmolyte biosynthesis, hormonal regulators, ROS scavengers and other traits that have been shown to play major roles in plant response to stress, will need classification according to their aptitude and functional significance in response to a given abiotic stress. Morpho-physiological traits associated with stress tolerance would also substantially reinforce the successes in molecular biology if addressed to a greater extent. The use of models for predicting gene effects, particularly when combining multiple traits, will also find greater application in dissecting G × E interactions and will help breeders to improve target varieties. Thus, there is need to integrate molecular tools with precise high-throughput phenotyping and biochemical analysis to confirm the consistency of various molecular findings, and to realize the full benefits of molecular biology in selecting genotypes that are stably tolerant under a given stress, considering the interaction with various environments. Here, we emphasize stresses that have been commonly reported in literature, which would provide a basis for understanding other minor stresses. We also refer to the chapter on biotic stresses and the numerous interactions in signaling pathways and expressions of resistance and tolerance on molecular level towards abiotic and biotic stress in plants. Additional background information can also be found in excellent reviews and references therein.
Acknowledgements

This publication was supported by Erfurt University of Applied Sciences.

Author details

Geoffrey Onaga1 and Kerstin Wydra2*

*Address all correspondence to: kerstin.wydra@fh-erfurt.de

1 National Crops Resources Research Institute, Kampala, Uganda
2 Erfurt University of Applied Sciences, Faculty of Landscape Architecture, Horticulture and Forestry, Erfurt, Germany

References

[1] Acquaah G. Principles of plant genetics and breeding. 2nd ed. Oxford, UK: Wiley; 2012. 20 pp.

[2] Dos Reis SP, Lima AM, de Souza CRB. Recent molecular advances on downstream plant responses to abiotic stress. International Journal of Molecular Sciences. 2012;13:8628–8647. DOI: 10.3390/ijms13078628

[3] Flowers TJ, Yeo AR. Breeding for salinity resistance in crop plants—where next? Australian Journal of Plant Physiology. 1995;22:875–884. DOI: 10.1071/PP9950875

[4] Colmer TD, Pedersen O. Underwater photosynthesis and respiration in leaves of submerged wetland plants: gas films improve CO2 and O2 exchange. New Phytologist. 2008;177:918–926. DOI: 10.1111/j.1469-8137.2007.02318.x

[5] Mittler R. Abiotic stress, the field environment and stress combination. Trends Plant Science. 2006;11:15–19. DOI: 10.1016/j.tplants.2005.11.002

[6] Fujita M, Fujita Y, Noutoshi Y, Takahashi F, Narusaka Y, Yamaguchi-Shinozaki K, Shinozaki K. Crosstalk between abiotic and biotic stress responses: a current view from the points of convergence in the stress signaling networks. Current Opinion in Plant Biology. 2006;9:436–442. DOI: 10.1016/j.pbi.2006.05.014

[7] Chinnusamy V, Schumaker K, Zhu JK. Molecular genetic perspectives on cross-talk and specificity in abiotic stress signaling in plants. Journal of Experimental Botany. 2004;55:225–236. DOI: 10.1093/jxb/erh005
[8] Krasensky J, Jonak C. Drought, salt, and temperature stress-induced metabolic rearrangements and regulatory networks. Journal of Experimental Botany. 2012;63:1593–1608. DOI: 10.1093/jxb/err460

[9] Ramakrishna A, Ravishankar GA. Influence of abiotic stress singles on secondary metabolites in plants. Plant Signaling Behavior. 2011;6:1720–1731. DOI: 10.4161/psb.6.11.17613

[10] Zhu QH, Zheng XM, Luo JC, Gaut BS, Ge S. Multilocus analysis of nucleotide variation of Oryza sativa and its wild relatives: severe bottleneck during domestication of rice. Molecular Biology and Evolution. 2007;24:875–888. DOI: 10.1093/molbev/msn005

[11] Buckler ES, Thornsberry JM, Kresovich S. Molecular diversity, structure and domestication of grasses. Genetical Research. 2001;77:213–218. DOI: 10.1017/S0016672301005158

[12] Haudry A, Cenci A, Ravel C, Bataillon T, Brunel D, Poncet C, Hochu I, Poirier S, Santoni S, Glémard S, David J. Grinding up wheat: a massive loss of nucleotide diversity since domestication. Molecular Biology Evolution. 2007;24:1506–1517. DOI: 10.1093/molbev/msn077

[13] Kim JY, Mahé AB, rangeon J, Prioul JL. A maize vacuolar invertase, IVR2, is induced by water stress. Organ/tissue specificity and diurnal modulation of expression. Plant Physiology. 2000;124:71–84. DOI: 10.1104/pp.124.1.71

[14] Liu F, Jensen CR, Andersen MN. Drought stress effect on carbohydrate concentration in soybean leaves and pods during early reproductive development: its implication in altering pod set. Field Crops Research. 2004;86(1):1–13. DOI: 10.1016/S0378-4290(03)00165-5

[15] Hirayama T, Shinozaki K. Research on plant abiotic stress responses in the post genome era: past, present and future. The Plant Journal. 2010;61(6):1041–1052. DOI: 10.1111/j.1365-313X.2010.04124.x

[16] Umezawa T, Nakashima K, Miyakawa K, Kuromori T, Tanokura M, Shinozaki K, Yamaguchi-Shinozaki K. Molecular basis of the core regulatory network in ABA responses: sensing, signaling and transport. Plant & Cell Physiology. 2010;51:1821–1839. DOI: 10.1093/pcp/pcq156

[17] Boursiac Y, Leran S, Corratge-Faillie C, Gojon A, Krouk G, Lacombe B. ABA transport and transporters. Trends in Plant Science 2013;18:325–333. DOI: http://dx.doi.org/10.1016/j.tplants.2013.01.007

[18] Golldack D, Li C, Mohan H, Probst N. Tolerance to drought and salt stress in plants: unraveling the signaling networks. Frontiers in Plant Science. 2014;5:151. DOI: 10.3389/fpls.2014.00151

[19] Vlad F, Rubio S, Rodrigues A, Sirichandra C, Belin C, Robert N, Leung J, Rodriguez PL, Laurière C, Merlot S. Protein phosphatases 2C regulate the activation of the Snf1-
related kinase OST1 by abscisic acid in *Arabidopsis*. The Plant Cell 2009;21:3170–3184. DOI: 10.1105/tpc.109.069179

[20] Campo S, Baldrich P, Messeguer J, Lalanne E, Coca M, San Segundo B. Overexpression of a calcium-dependent protein kinase confers salt and drought tolerance in rice by preventing membrane lipid peroxidation. Plant Physiology. 2014;165:688–704. DOI: 10.1104/pp.113.230268

[21] Ouyang SQ, Liu YF, Liu P, Lei G, He SJ, Ma B, Zhang WK, Zhang JS, Chen SY. Receptor-like kinase OsSIK1 improves drought and salt stress tolerance in rice (*Oryza sativa*) plants. The Plant Journal. 2010;62:316–329. DOI: 10.1111/j.1365-313X.2010.04146.x

[22] Osakabe Y, Yamaguchi-Shinozaki K, Shinozaki K, Lam-Son Phan T.ABA control of plant macro element membrane transport systems in response to water deficit and high salinity. New Phytologist. 2014;202:35–49. DOI: 10.1111/nph.12613

[23] Xu C, Jing R, Mao X, Jia X, Chang X. A wheat (*Triticum aestivum*) protein phosphatase 2A catalytic subunit gene provides enhanced drought tolerance in tobacco. Annals of Botany. 2007;99:439–450. DOI: 10.1093/aob/mcl285

[24] Banerjee A, Roychoudhury A. WRKY proteins: signaling and regulation of expression during abiotic stress responses. The Scientific World Journal. 2015;2015:807560. DOI: 10.1155/2015/807560

[25] Ding Z, Li S, An X, Liu X, Qin H, Wang D. Transgenic expression of MYB15 confers enhanced sensitivity to abscisic acid and improved drought tolerance in *Arabidopsis thaliana*. Journal of Genetics and Genomics. 2009;36:17–29. DOI: 10.1016/S1673-8527(09)60003-5

[26] Rushton DL, Tripathi P, Rabara RC, Lin J, Ringler P, Boken AK, Langum TJ, Smidt L, Boomsma DD, Emme NJ, Chen X, Finer JJ, Shen QJ, Rushton PJ. WRKY transcription factors: key components in abscisic acid signaling. Plant Biotechnology Journal. 2012;10:2–11. DOI: 10.1111/j.1467-7652.2011

[27] Zhang G, Chen M, Li L, Xu Z, Chen X, Guo J, Ma Y. Overexpression of the soybean GmERF3 gene, an AP2/ERF type transcription factor for increased tolerances to salt, drought, and diseases in transgenic tobacco. Journal of Experimental Botany. 2009;60:3781–3796. DOI: 10.1093/jxb/erp214

[28] Liu S, Wang X, Wang H, Xin H, Yang X, Yan J Li J, Tran LS, Shinozaki K, Yamaguchi-Shinozaki K, Qin F. Genome-wide analysis of ZmDREB genes and their association with natural variation in drought tolerance at seedling stage of *Zea mays* L. PLoS Genetics. 2013;9(9):e1003790. DOI: 10.1371/journal.pgen.1003790

[29] Ji X, Liu G, Liu Y, Zheng L, Nie X, Wang Y. The bZIP protein from Tamarix hispida, ThbZIP1, is ACGT elements binding factor that enhances abiotic stress signaling in transgenic *Arabidopsis*. BMC Plant Biology. 2013;13. DOI: 10.1186/1471-2229-13-151
[30] Ying S, Zhang DF, Fu J, Shi YS, Song YC, Wang TY, and Li Y. Cloning and characterization of a maize bZIP transcription factor, ZmbZIP72, confers drought and salt tolerance in transgenic Arabidopsis. Planta. 2012;235:253–266. DOI: 10.1007/s00425-011-1496-7

[31] Fujita M, Fujita Y, Maruyama K, Seki M, Hiratsu K, Ohme-Takagi M, Tran LS, Yamaguchi-Shinozaki K, Shinozaki K. A dehydration-induced NAC protein, RD26, is involved in a novel ABA-dependent stress-signaling pathway. The Plant Journal. 2004;39:863–876. DOI: 10.1111/j.1365-313X.2004.02171.x

[32] Tran LS, Nakashima K, Sakuma Y, Simpson SD, Fujita Y, Maruyama K, Fujita M, Seki M, Shinozaki K, Yamaguchi-Shinozaki K. Isolation and functional analysis of Arabidopsis stress-inducible NAC transcription factors that bind to a drought responsive cis-element in the early responsive to dehydration stress 1 promoter. The Plant Cell 2004;16:2481–2498. DOI: 10.1105/tpc.104.022699

[33] Jeong JS, Kim YS, Baek KH, Jung H, Ha SH, Do Choi Y, Kim M, Reuzeau C, Kim JK. Root-specific expression of OsNAC10 improves drought tolerance and grain yield in rice under field drought conditions. Plant Physiology. 2010;153:185–197. DOI: 10.1104/pp.110.154773

[34] Jeong JS, Kim YS, Redillas MC, Jang G, Jung H, Bang SW Choi YD, Ha SH, Reuzeau C, Kim JK. OsNAC5 overexpression enlarges root diameter in rice plants leading to enhanced drought tolerance and increased grain yield in the field. Plant Biotechnology Journal. 2013;11:101–114. DOI: 10.1111/pbi.12011

[35] Palmeros-Suárez PA, Massange-Sánchez JA, Martínez-Gallardo NA, Montero-Vargas JM, Gómez-Leyva JF, Délano-Frier JP. The overexpression of an Amaranthus hypochondriacus NF-YC gene modifies growth and confers water deficit stress resistance in Arabidopsis. Plant Science. 2015;240:25–40. DOI: 10.1016/j.plantsci.2015.08.010

[36] Chen M, Zhao Y, Zhuo C, Lu S, Guo Z. Overexpression of a NF-YC transcription factor from Bermuda grass confers tolerance to drought and salinity in transgenic rice. Plant Biotechnology Journal. 2015;13:482–491. DOI: 10.1111/pbi.12270

[37] Fang Y, Liao K, Du H, Xu Y, Song H, Li X, Xiong LA. Stress-responsive NAC transcription factor SNAC3 confers heat and drought tolerance through modulation of reactive oxygen species in rice. Journal of Experimental Botany. 2015;25:3472–3490. DOI: 10.1093/jxb/erv234

[38] Djafi N, Vergnolle C, Cantrel C, Wietrzyński W, Delage E, Cochet F Puyaubert J, Soubigou-Taconnat L, Gey D, Collin S, Balzergue S, Zachowski A, Ruelland E. The Arabidopsis DREB2 genetic pathway is constitutively repressed by basal phosphoinositide-dependent phospholipase C coupled to diacylglycerol kinase. Frontiers in Plant Science. 2013;4:307. DOI: 10.3389/fpls.2013.00307

[39] Zhai SM, Gao Q, Xue HW, Sui ZH, Yue GD, Yang AF, Zhang JR. Overexpression of the phosphatidylinositol synthase gene from Zea mays in tobacco plants alters the
membrane lipids composition and improves drought stress tolerance. Planta. 2012;235:69–84. DOI: 10.1007/s00425-011-1490-0

[40] Zhu J-K. Salt and Drought Stress Signal Transduction in Plants. Annual review of plant biology. 2002;53:247–273. DOI: 10.1146/annurev.arplant.53.091401.143329.

[41] Jacob T, Ritchie S, Assmann SM, Gilroy S. Abscisic acid signal transduction in guard cells is mediated by phospholipase D activity. Proceedings of the National Academy of Sciences of the United States of America. 1999;96:12192–97. DOI: 10.1073/pnas.96.21.12192

[42] Seki M, Narusaka M, Ishida J, Nanjo T, Fujita M Oono Y, Kamiya A, Nakajima M, Enju A, Sakurai T, Satou M, Akiyama K, Taji T, Yamaguchi-Shinozaki K, Carninci P, Kawai J, Hayashizaki Y, Shinozaki K. Monitoring the expression profiles of 7000 Arabidopsis genes under drought, cold and high-salinity stresses using a full-length cDNA microarray. The Plant Journal. 2002;31:279–292. DOI: 10.1046/j.1365-313X.2002.01359.x

[43] Xu Y, Wei H, Juhua L, Jianbin Z, Caihong J, Jianbin Z, Biyu X, Zhiqiang J. A banana aquaporin gene, MaPIP1;1, is involved in tolerance to drought and salt stresses. BMC Plant Biology. 2014;14:59. DOI: 10.1186/s12870-014-0229-y

[44] Li H, Yao W, Fu Y, Li S, Guo Q. De Novo Assembly and discovery of genes that are involved in drought tolerance in Tibetan Sophora moorcroftiana. PLoS ONE 2015;10(1):e011054. DOI: 10.1371/journal.pone.0111054

[45] Li J, Ban L, Wen H, Wang Z, Dzyubenko N, Chapurin V, Gao H, Wang X. An aquaporin protein is associated with drought stress tolerance. Biochemical Biophysical Research Communication. 2015;459:208–213. DOI: 10.1016/j.bbrc.2015.02.052

[46] Osakabe Y, Arinaga N, Umezawa T, Katsura S, Nagamachi K, Tanaka H, Ohiraki H, Yamada K, Seo SU, Abo M Yoshimura E, Shinozaki K, Yamaguchi-Shinozaki K. Osmotic stress responses and plant growth controlled by potassium transporters in Arabidopsis. The Plant Cell 2013;25:609–624. DOI: 10.1105/tpc.112.105700

[47] Vicente-Agullo F, Rigas S, Desbrosses G, Dolan L, Hatzopoulos P, Grabov A. Potassium carrier TRH1 is required for auxin transport in Arabidopsis roots. The Plant Journal. 2004;40:523–535. DOI: 10.1046/j.1365-313X.2004.02230.x

[48] Zhang C, Zhang L, Zhang S, Zhu S, Wu P, Chen Y, Li M, Jiang H, Wu G. Global analysis of gene expression profiles in physic nut (Jatropha curcas L.) seedlings exposed to drought stress. BMC Plant Biology. 2015;15:1. DOI: 10.1371/journal.pone.0139302

[49] Wisniewski ME, Bassett CL, Renault J, Farrell R, Tworkoski T, Artlip TS. Differential regulation of two dehydrin genes from peach (Prunus persica) by photoperiod, low temperature and water deficit. Tree Physiology. 2006;26:575–584. DOI: 10.1093/treephys/26.5.575

[50] Yoshimura K, Masuda A, Kuwano M, Yokota A, Akashi K. Programmed proteome response for drought avoidance/tolerance in the root of a C3 xerophyte (wild water-
melon) under water deficits. Plant Cell Physiology. 2008;49:226–241. DOI: 10.1093/pcp/pcm180

[51] Degenhardt B, Gimmler H. Cell wall adaptations to multiple environmental stresses in maize roots. Journal of Experimental Botany. 2000;51:595–603. DOI: 10.1093/jexbot/51.344.595

[52] Hura T, Hura K, Dziurka K, Ostrowska A, Basczek-Kwinta R, Grzesiak M. An increase in the content of cell wall-bound phenolics correlates with the productivity of triticale under soil drought. Journal of Plant Physiology. 2012;169:1728–1736.61. DOI: 10.1016/j.jplph.2012.07.012

[53] Alam I, Sharmin SA, Kim KH, Yang JK, Choi MS, Lee BH. Proteome analysis of soybean roots subjected to short-term drought stress. Plant and Soil. 2010;333:491–505. DOI: 10.1007/s11104-010-0365-7

[54] Mohammadi PP, Moieni A, Komatsu S. Comparative proteome analysis of drought-sensitive and drought-tolerant rapeseed roots and their hybrid F1 line under drought stress. Amino Acids. 2012;43:2137–2152. DOI: 10.1007/s00726-012-1299-6

[55] Hu H, Xiong L. Genetic engineering and breeding of drought-resistant crops. Annual Reviews of Plant Biology. 2014;65:715–741. DOI: 10.1146/annurev-arplant-050213-040000

[56] Asthir B. Protective mechanisms of heat tolerance in crop plants. Journal of Plant Interactions. 2015;10:202–210. DOI: 10.1080/17429145.2015.1067726

[57] Hasanuzzaman M, Nahar K, Alam MdM, Roychowdhury, Rajib, and Fujita M. Physiological, biochemical, and molecular mechanisms of heat stress tolerance in plants. International Journal of Molecular Science. 2013;14(5):9643–9684. DOI: 10.3390/ijms14059643

[58] Saidi Y, Peter M, Finka A, Cicekli C, Vigh L, Goloubinoff P. Membrane lipid composition affects plant heat sensing and modulates Ca2+-dependent heat shock response. Plant Signaling and Behavior. 2010;5:1530–1533. DOI: 10.4161/psb.5.12.13163

[59] Li Z, Yue HY, Xing D. MAP Kinase 6-mediated activation of vacuolar processing enzyme modulates heat shock-induced programmed cell death in Arabidopsis. New Phytologist. 2012;195:85–96. DOI: 10.3389/fpls.2015.00234

[60] Suzuki N, Sejima H, Tam R, Schlauch K, Mittler R. Identification of the MBF1 heat-response regulon of Arabidopsis thaliana. The Plant Journal. 2011;66:844–851. DOI: 10.1111/j.1365-313X.2011.04550.x

[61] Miller G, Schlauch K, Tam R, Cortes D, Torres MA, Shulaev V, Dangl JL, Mittler R. The plant NADPH oxidase RBOHD mediates rapid systemic signaling in response to diverse stimuli. Science Signaling. 2009;2. DOI: 10.1126/scisignal.2000448
[62] Liu HT, Gao F, Li GL, Han JL, Liu DL, Sun DY, Zhou RG. The calmodulin-binding protein kinase 3 is part of heat-shock signal transduction in *Arabidopsis thaliana*. The Plant Journal. 2008;55:760–773. DOI: 10.1111/j.1365-313X.2008.03544.x

[63] Baniwal SK, Bharti K, Chan KY, Fauth M, Ganguki A, Kotak S, Mishra SK, Nover L, Port M, Scharf KD, Tripp J, Weber C, Zielinski D, von Koskull-Döring P. Heat stress response in plants: a complex game with chaperones and more than twenty heat stress transcription factors. Journal of Biosciences. 2004;29:471–487.

[64] Suzuki N, Bajad S, Shuman J, Shulaev V, Mittler R. The transcriptional co-activator MBF1c is a key regulator of thermotolerance in *Arabidopsis thaliana*. The Journal of Biological Chemistry. 2008, 283:9269–9275. DOI: 10.1074/jbc.M709187200

[65] Khan MIR, Iqbal N, Masood A, Per TS, Khan NA. Salicylic acid alleviates adverse effects of heat stress on photosynthesis through changes in proline production and ethylene formation. Plant Signaling & Behavior. 2013; 8(11):e26374. DOI: 10.4161/psb.26374.

[66] Meiri D, Breiman A. *Arabidopsis* ROF1 (FKBP62) modulates thermotolerance by interacting with HSP90.1 and affecting the accumulation of HsfA2-regulated shHSPs. The Plant Journal 2009;59:387–399. DOI: 10.1111/j.1365-313X.2009.03878.x

[67] Bokszczanin KL, Fragkostefanakis S. Perspectives on deciphering mechanisms underlying plant heat stress response and thermotolerance. Frontiers in Plant Science. 2013;4:315–335. DOI: 10.3389/fpls.2013.00315

[68] Sidrauski C, Walter P. The transmembrane kinase Ire1p is a site-specific endonuclease that initiates mRNA splicing in the unfolded protein response. Cell 1997;90:1031–1039. DOI: 10.1016/S0092-8674(00)80369-4

[69] Deng Y, Humbert S, Liu JX, Srivastava R, Rothstein SJ, Howell SH. Heat induces the splicing by IRE1 of a mRNA encoding a transcription factor involved in the unfolded protein response in *Arabidopsis*. Proceedings of the National Academy of Sciences of the United States of America. 2011;108:7247–7252. DOI: 10.1073/pnas.102117108

[70] Sugio A, Dreos R, Aparicio F, Maule AJ. The cytosolic protein response as a subcomponent of the wider heat shock response in *Arabidopsis*. The Plant Cell 2009;21:642–654. DOI: 10.1105/tpc.108.062596

[71] Liu J, Sun N, Liu M, Liu J, Du B, Wang X, Qi X. An autoregulatory loop controlling *Arabidopsis* HsfA2 expression: role of heat shock-induced alternative splicing. Plant Physiology. 2013;162:512–521. DOI: 10.1104/pp.112.205864

[72] Sato H, Mizoi J, Tanaka H, Maruyama K, Qin F, Osakabe Y, Morimoto K, Ohori T, Kusakabe K, Nagata M, Shinozaki K, Yamaguchi-Shinozakia K. *Arabidopsis* DPB3-1, a DREB2A interactor, specifically enhances heat stress-induced gene expression by forming a heat stress-specific transcriptional complex with NF-Y subunits. The Plant Cell 2014;26:4954–4973. DOI: 10.1105/tpc.114.132928
[73] Gao HB, Brandizzi F, Benning C, Larkin RM. A membrane-tethered transcription factor defines a branch of the heat stress response in *Arabidopsis thaliana*. Proceedings of the National Academy of Sciences of the United States of America. 2008;105:16398–16403. DOI: 10.1073/pnas.0808463105

[74] Li SJ, Fu QT, Huang WD, Yu DQ. Functional analysis of an *Arabidopsis* transcription factor WRKY25 in heat stress. Plant Cell Reports. 2009;28:683–693. DOI: 10.1007/s00299-008-0666-y

[75] Wu XL, Shiroto Y, Kishitani S, Ito Y, Toriyama K. Enhanced heat and drought tolerance in transgenic rice seedlings overexpressing OsWRKY11 under the control of HSP101 promoter. Plant Cell Reports. 2009;28:21–30. DOI: 10.1007/s00299-008-0614-x

[76] Proveniers MCG, van Zanten M. High temperature acclimation through PIF4 signaling. Trends in Plant Science. 2013;18:59–64. DOI: 10.1016/j.tplants.2012.09.002

[77] Ma D, Li X, Guo X, Chu J, Fang S, Yan C, Noel J, Liu H. Cryptochrome 1 interacts with PIF4 to regulate high temperature-mediated hypocotyl elongation in response to blue light. Proceedings of the National Academy of Sciences of the United States of America. 2015;113:224–229. DOI: 10.1073/pnas.1511437113

[78] Erkina TY, Zou Y, Freeing S, Vorobyev VI, Erkine AM. Functional interplay between chromatin remodeling complexes RSC, SWI/SNF and ISWI in regulation of yeast heat shock genes. Nucleic Acids Research. 2010;38:1441–1449. DOI: 10.1093/nar/gkp1130

[79] Rasheed R, Wahid A, Farooq M, Basra SMA. Role of proline and glycinebetaine in improving heat tolerance in sprouting sugarcane (*Saccharum* sp.) buds. Plant Growth Regulation. 2011;65:35–45. DOI: 10.1007/s10725-011-9572-3

[80] Kapoor D, Sharma R, Handa N, Kaur H, Rattan A, Yadav P, Gautam V, Kaur R, Bhardwaj R. Redox homeostasis in plants under abiotic stress. Role of electron carriers, energy metabolism mediators and proteinaceous thiols. Frontiers in Environmental Science. 2015;3:13. DOI: 10.3389/fenvs.2015.00013

[81] Priest HD, Fox SE, Rowley ER, Murray JR, Michael TP, Mockler TC. Analysis of global gene expression in *Brachypodium distachyon* reveals extensive network plasticity in response to abiotic stress. *PLoS* ONE. 2014;9:e87499. DOI: 10.1371/journal.pone.0087499

[82] Zhang Y, Xu L, Zhu X, Gong Y, Xiang F, Sun X, Liu L. Proteomic analysis of heat stress response in leaves of radish (*Raphanus sativus* L.). Plant Molecular Biology Reports. 2013;31:195–203. DOI: 10.1007/s11105-012-0486-7

[83] Chen X, Zhang W, Zhang B, Zhou J, Wang Y, Yang Q, Ke Y, He H. Phosphoproteins regulated by heat stress in rice leaves. Proteome Science. 2011;9:37. DOI: 10.1186/1477-5956-9-37

[84] Li W, Wei Z, Qiao Z, Wu Z, Cheng L, Wang Y. Proteomics analysis of alfalfa response to heat stress. *PLoS* ONE. 2013a;8:e82725. DOI: 10.1371/journal.pone.0082725
[85] Du HM, Wang ZL, Yu WJ, Liu YM, Huang BR. Differential metabolic responses of perennial grass *Cynodon transvaalensis* × *Cynodon dactylon* (C4) and *Poa pratensis* (C3) to heat stress. Physiologia Plantarum. 2011;141:51–264. DOI: 10.1111/j.1399-3054.2010.01432.x

[86] Yu J, Du H, Xu M, Huang B. Metabolic responses to heat stress under elevated atmospheric CO$_2$ concentration in a cool-season grass species. Journal of the American Society for Horticultural Science. 2012b;137:221–228. DOI: 10.1111/j.1399-3054.2010.01432.x

[87] Goswami S, Kumar RR, Rai RD. Heat-responsive microRNAs regulate the transcription factors and heat shock proteins in modulating thermostability of starch biosynthesis enzymes in wheat (*Triticum aestivum* L.) under the heat stress. Australian Journal of Crop Science. 2014;8:697—705.

[88] Hozain M, Abdelmageed H, Lee J, Kang M, Fokar M, Allen RD, Holaday AS. Expression of AtSAP5 in cotton up-regulates putative stress-responsive genes and improves the tolerance to rapidly developing water deficit and moderate heat stress. Journal of Plant Physiology. 2012;169:1261—1270. DOI: 10.1016/j.jplph.2012.04.007

[89] Sasaki K, Saito T, Lämsä M, Oksman-Caldentey KM, Suzuki M, Ohyama K, Muranaka T, Ohara K, Yazaki K. Plants utilize isoprene emission as a thermo tolerance mechanism. Plant Cell Physiology. 2007;48:1254—126. DOI: 10.1093/pcp/pcm104

[90] Macková H, Hronková M, Dobrá J, Turecková V, Novák O, Lubovská Z, Motyka V, Haisel D, Hájek Prášil ITT, Gaudinová A, Storčová H, Ge E, Werner T, Schmülling T, Vanková R. Enhanced drought and heat stress tolerance of tobacco plants with ectopically enhanced cytokinin oxidase/dehydrogenase gene expression. Journal of Experimental Botany. 2013;64:2805—2815. DOI: 10.1093/jxb/ert131

[91] Feng L, Wang KJ, Li Y, Tan Y, Kong J, H L, Y L, Zhu Y. Over expression of SBPase enhances photosynthesis against high temperature stress in transgenic rice plants. Plant Cell Reports. 2007;26:1635—1646. DOI: 10.1007/s00299-006-0299-y

[92] Chinnusamy V, Zhu J, Zhu JK. Cold stress regulation of gene expression in plants. Trends Plant Science. 2007;12:444—451. DOI: 10.1016/j.tplants.2007.07.002

[93] Sanghera GS, Wani SH, Hussain W, Singh NB. Engineering cold stress tolerance in crop plants. Current Genomics. 2011;12:30–43. DOI: 10.2174/138920211794520178

[94] Ye H, Du H, Tang N, Li X, Xiong L. Identification and expression profiling analysis of TIFY family genes involved in stress and phytohormone responses in rice. Plant Molecular Biology. 2009;71:291—305. DOI: 10.1007/s11103-009-9524-8

[95] Miura K, and Furumoto T. Cold signaling and cold response in plants. International Journal of Molecular Science. 2013;14(3):5312-5337. DOI: 10.3390/ijms14035312
[96] Knight MR, Campbell AK, Smith SM, Trewavas AJ. Transgenic plant aequorin reports the effects of touch and cold-shock and elicitors on cytoplasmic calcium. Nature. 1991;352(6335):524–6. DOI: 10.1038/352524a0

[97] Furuya T, Matsuoka D, Nanmori T. Membrane rigidification functions upstream of the MEKK1-MKK2-MPK4 cascade during cold acclimation in Arabidopsis thaliana. FEBS Letters. 2014;588:2025–2030. DOI: 10.1016/j.febslet.2014.04.032

[98] Francia E, Rizza F, Cattivelli L, Stanca AM, Galiba G, Toth B, Hayes PM, Skinner JS, Pecchioni N. Two loci on chromosome 5H determine low-temperature tolerance in a ‘Nure’ (winter) × ‘Tremois’ (spring) barley map. Theoretical Applied Genetics. 2004;108:670–680. DOI: 10.1007/s00122-003-1468-9

[99] Huang C, Ding S, Zhang H, Du H, An L. CIPK7 is involved in cold response by interacting with CBL1 in Arabidopsis thaliana. Plant Science. 2011;181:57–64. DOI: 10.1016/j.plantsci.2011.03.011

[100] Doherty CJ, Buskirk HAV, Myers SJ, Thomashow MF. Roles for Arabidopsis CAMTA transcription factors in cold-regulated gene expression and freezing tolerance. The Plant Cell. 2009;21:972–984. DOI: 10.1105/tpc.108.063958

[101] Chinnusamy V, Zhu J-K, Sunkar R. Gene Regulation during cold stress acclimation in plants. Methods in molecular biology. 2010;639:39–55. DOI: 10.1007/978-1-60761-702-0_3.

[102] Takahashi S, Murata N. How do environmental stresses accelerate photo-inhibition? Trends in Plant Science. 2008;13:178–182. DOI: 10.1016/j.tiplants.2008.01.005

[103] Teige M, Scheikl E, Eulgem T, Doczi R, Ichimura K, Shinozaki K, Dangl JL, Hirt H. The MKK2 pathway mediates cold and salt stress signaling in Arabidopsis. Molecular Cell. 2004;15:141–152. DOI: 10.1016/j.molcel.2004.06.023

[104] Tarkowski ŁP, Van den Ende W. Cold tolerance triggered by soluble sugars: a multifaceted countermeasure. Frontiers in Plant Science. 2015;6. DOI: 10.3389/fpls.2015.00203

[105] Chinnusamy V, Ohta M, Kanrar S, Lee B H, Hong X, Agarwal M, Zhu JK. ICE1. A regulator of cold-induced transcriptome and freezing tolerance in Arabidopsis. Genes and Development. 2003;17:1043–1054. DOI: 10.1101/gad.1077503

[106] Liu L, Duan L, Zhang J, Zhang Z, Mi G, Ren H. Cucumber (Cucumis sativus L.) over-expressing cold-induced transcriptome regulator ICE1 exhibits changed morphological characters and enhances chilling tolerance. Scientia Horticulturae. 2010;124:29–33. DOI: 10.1016/j.scienta.2009.11.018

[107] Medina J, Catala R, Salinas J. The CBFs: three Arabidopsis transcription factors to cold acclimate. Plant Science. 2011;180:3–11. DOI: 10.1016/j.plantsci.2010.06.019

[108] Zeller G, Henz SR, Widmer CK, Sachsenberg T, Ratsch G, Weigel D, Laubinger S. Stress-induced changes in the Arabidopsis thaliana transcriptome analyzed using
whole-genome tiling arrays. The Plant Journal. 2009;58:1068–1082. DOI: 10.1111/j.1365-313X.2009.03835.x

[109] Novillo F, Alonso JM, Ecker JR, Salinas J. CBF2/DREB1C is a negative regulator of CBF1/DREB1B and CBF3/DREB1A expression and plays a central role in stress tolerance in Arabidopsis. Proceedings of the National Academy of Sciences of the United States of America. 2004;101:3985–3990. DOI: 10.1073/pnas.0303029101

[110] Novillo F, Medina J, Salinas J. Arabidopsis CBF1 and CBF3 have a different function than CBF2 in cold acclimation and define different gene classes in the CBF regulon. Proceedings of the National Academy of Sciences of the United States of America. 2007;104:21002–21007. DOI: 10.1073/pnas.0705639105

[111] Krasensky J, Jonak C. Drought, salt, and temperature stress-induced metabolic rearrangements and regulatory networks. Journal of Experimental Botany. 2012;63:1593–1608. DOI: 10.1093/jxb/err460

[112] Lee DG, Ahsan N, Lee SH, Lee JJ, Bahk JD, Kang KY, Lee BH. Chilling stress-induced proteomic changes in rice roots. Journal of Plant Physiology. 2009;166:1–11. DOI: 10.1016/j.jplph.2008.02.001

[113] FAO. Land and Plant Nutrition Management Service [Internet]. 2008. Available from: http://wwwfao.org/ag/agl/agll/spush [Accessed: 2016-05-03]

[114] Munns R, Tester M. Mechanisms of salinity tolerance. Annual Reviews in Plant Biology. 2008;59:651–681. DOI: 10.1146/annurev.arplant.59.032607.092911

[115] Shahbaz M, Ashraf M. Improving salinity tolerance in cereals. Critical Reviews in Plant Science. 2013;32:237–249. DOI: 10.1080/07352689.2013.758544

[116] Volkov V. Salinity tolerance in plants. Quantitative approach to ion transport starting from halophytes and stepping to genetic and protein engineering for manipulating ion fluxes Frontiers in Plant Science. 2015;6:873. DOI: 10.3389/fpls.2015.00873

[117] Hasegawa PM, Bressan RA, Zhu JK and HJ Bohnert. Plant cellular and molecular responses to high salinity. Annual Reviews in Plant Biology. 2000;51:463–499. DOI: 10.1146/annurev.arplant.51.1.463

[118] Jung JKH, McCouch S. Getting to the roots of it: genetic and hormonal control of root architecture. Frontiers in Plant Science. 2013;4:186. DOI: 10.3389/fpls.2013.00186

[119] Wang H, Wu Z, Chen Y, Yang C, Shi D. Effects of salt and alkali stresses on growth and ion balance in rice (Oryza sativa L.). Plant, Soil and Environment. 2011;57:286–294.

[120] Deinlein U, Stephen AB, Horie T, Luo W, Xu G, Schroeder JI. Plant salt tolerance mechanisms. Trends in Plant Science. 2014;19:371–379. DOI: 10.1016/j.tplants.2014.02.001
[121] Pandey N, Ranjan A, Pant P, Tripathi RK, Ateek F, Pandey HP, Patre UV, Sawant SV. CAMTA 1 regulates drought responses in Arabidopsis thaliana. BMC Genomics. 2013;14:216. DOI: 10.1186/1471-2164-14-216

[122] Weng H, Yoo CY, Gosney MJ, Hasegawa PM, Mickelbart MV. Poplar GTL1 is a Ca2+/calmodulin-binding transcription factor that functions in plant water use efficiency and drought tolerance. PLoS ONE. 2012;7:e32925. DOI: 10.1371/journal.pone.0032925

[123] Yoo JH, Park CY, Kim JC, Heo WD, Cheong MS, Park HC, Kim MC, Moon BC, Choi MS, Kang YH, Lee JH, Kim HS, Lee SM, Yoon HW, Lim CO, Yun DJ, Lee SY, Chung WS, Cho MJ. Direct interaction of a divergent CaM isoform and the transcription factor, MYB2, enhances salt tolerance in Arabidopsis. Journal of Biological Chemistry. 2005;280:3697–3706. DOI: 10.1074/jbc.M408237200

[124] Liu C, Mao B, Ou S, Wang W, Liu L, Wu Y, Chu C, Wang X. OsbZIP71, a bZIP transcription factor, confers salinity and drought tolerance in rice. Plant Molecular Biology. 2014;84:19–36. DOI: 10.1007/s11103-013-0115-3

[125] Jiang Y, Deyholos MK. Functional characterization of Arabidopsis NaCl-inducible WRKY25 and WRKY33 transcription factors in abiotic stresses. Plant Molecular Biology. 2009;69:91–105. DOI: 10.1007/s11103-008-9408-3

[126] Yamaguchi-Shinozaki K, Shinozaki K. Improving plant drought, salt, and freezing tolerance by gene transfer of a single stress-inducible transcription factor. In: Novartis Foundation Symposium 236—Rice Biotechnology: Improving Yield, Stress Tolerance and Grain Quality; February 2001; discussion 186-9; 236:176–186; DOI: 10.1002/9780470515778.ch13

[127] Cui MH, Yoo KS, Hyoung S, Nguyen HT, Kim YY, Kim HJ, Ok SH, Yoo SD, Shin JS. An Arabidopsis R2R3-MYB transcription factor, AtMYB20, negatively regulates type 2C serine/threonine protein phosphatases to enhance salt tolerance. FEBS Letters. 2013;587:1773–1778. DOI: 10.1016/j.febslet.2013.04.028

[128] Jiang Y, Yang B, Deyholos MK. Functional characterization of the Arabidopsis bHLH29 transcription factor in abiotic stress. Molecular Genetics and Genomics 2009;282:503–516. DOI: 10.1007/s00438-009-0481-3

[129] Chen Y, Zong J, Tan Z, Li L, Hu B, Chen C, Chen J, Liu J. Systematic mining of salinity-tolerant genes in Halophyte-Zoysia matrella through cDNA expression library screening. Plant Physiology and Biochemistry 2015b;89:44–52. DOI: 10.1016/j.plaphy.2015.02.007

[130] Hur J, Jung KH, Lee CH, An G. Stress-inducible OsP5CS2 gene is essential for salt and cold tolerance in rice. Plant Science 2004;167:417–426. DOI: 10.1016/j.plantsci.2004.04.009
[131] Ismail A, Takeda S, and Nick P. Life and death under salt stress: same players, different timing? Journal of Experimental Botany. 2014;65:2963–2979. DOI: 10.1093/jxb/eru159

[132] Quintero FJ, Martinez-Atienza J, Villalta I, Jiang X, Kim WY, Ali Z, Fujii H, Mendoza I, Yun DJ, Zhu JK, and Pardo JM. Activation of the plasma membrane Na/H antiporter Salt-Overly Sensitive 1 (SOS1) by phosphorylation of an auto-inhibitory C-terminal domain. Proceedings of the National Academy of Sciences of the United States of America. 2011;108:2611–2616. DOI: 10.1073/pnas.1018921108

[133] Du W, Lin H, Chen S, Wu Y, Zhang J, Fuglsang AT, Palmgren MG, Wu W, Guo Y. Phosphorylation of SOS3-like calcium-binding proteins by their interacting SOS2-like protein kinases is a common regulatory mechanism in Arabidopsis. Plant Physiology. 2011;156:2235–2243. DOI: 10.1104/pp.111.173377

[134] Bassil E, Blumwald E. The ins and outs of intracellular ion homeostasis: NHX-type Cation/H+ transporters. Current Opinion in Plant Biology. 2014, 22:1–6. DOI: 10.1016/j.pbi.2014.08.002

[135] Yamaguchi T, Aharon GS, Sottosanto JB, Blumwald E. Vacuolar Na+/H+ antiporter cation selectivity is regulated by calmodulin from within the vacuole in a Ca2+ and pH-dependent manner. Proceedings of the National Academy of Sciences of the United States of America. 2005;102:16107–16112. DOI: 10.1073/pnas.0504437102

[136] Guan R, Qu Y, Guo Y, Yu L, Liu Y, Jiang J, Chen J, Ren Y, Liu G, Tian L, Jin L, Liu Z, Hong H, Chang R, Gilliham M, Qiu L. Salinity tolerance in soybean is modulated by natural variation in GmSALT3. The Plant Journal. 2014;80:937–950. DOI: 10.1111/tpj.12695

[137] Kumar R, Mustafiz A, Sahoo KK, Sharma V, Samanta S, Sopory SK, Pareek A, Singla-Pareek SL. Functional screening of cDNA library from a salt tolerant rice genotype Pokkali identifies mannose-1-phosphate guanyl transferase gene (OsMPG1) as a key member of salinity stress response. Plant Molecular Biology. 2012;79:555–568. DOI: 10.1007/s11103-012-9928-8

[138] Obata T, Kitamoto HK, Nakamura A, Fukuda A, Tanaka Y. Rice shaker potassium channel OsKAT1 confers tolerance to salinity stress on yeast and rice cells. Plant Physiology. 2007;144:1978–1985. DOI: 10.1104/pp.107.101154

[139] Chen Y, Chen C, Tan Z, Liu J, Zhuang L, Yang Z, Huang B. Functional identification and characterization of genes cloned from halophyte Seashore paspalum conferring salinity and cadmium tolerance. Frontiers in Plant Science. 2016;7:102. DOI: 10.3389/fpls.2016.00102

[140] Munns R, Gilliham M. Salinity tolerance of crops – what is the cost? New Phytologist. 2015;208:668–673. DOI: 10.1111/nph.13519

[141] Byrt CS, Xu B, Krishnan M, Lightfoot DJ, Athman A, Jacobs AK, Watson-Haigh NS, Munns R, Tester M, Gilliham M. The Na+ transporter, TaHKT1;5-D, limits shoot Na+
accumulation in bread wheat. The Plant Journal. 2014;80:516–26. DOI: 10.1111/tpj.12651

[142] Munns R, James RA, Xu B, Athman A, Conn SJ, Jordans C, Byrt CS, Hare RA, Tyerman SD, Tester M, Plett D, Gilliham M. Wheat grain yield on saline soils is improved by an ancestral Na⁺ transporter gene. Nature Biotechnology. 2012;30:360–4. 10.1038/nbt.2120

[143] Eswaran N, Parameswaran S, Sathram B, Anantharaman B, Kumar GRK, Tangirala SJ. Yeast functional screen to identify genetic determinants capable of conferring abiotic stress tolerance in *Jatropha curcas*. BMC Biotechnology. 2010;10:23. DOI: 10.3389/fpls.2015.00920

[144] Zhou L, Wang C, Liu R, Han Q, Vandeule RK, Du J, Tyerman S, Shou H. Constitutive overexpression of soybean plasma membrane intrinsic protein GmPIP1;6 confers salt tolerance. BMC Plant Biology. 2014;14:188. DOI: 10.1186/1471-2229-14-181

[145] Sade N, Gebretsadik M, Seligmann R, Schwartz A, Wallach R, Mosheilon M. The role of tobacco Aquaporin I in improving water use efficiency, hydraulic conductivity, and yield production under salt stress. Plant Physiology. 2010;152(1):245–254. DOI: 10.1104/pp.109.145854

[146] Gao Z, He X, Zhao B, Zhou C, Liang Y, Ge R, Shen Y, Huang Z. Overexpressing a putative aquaporin gene from wheat, TaNIP, enhances salt tolerance in transgenic *Arabidopsis*. Plant & Cell Physiology. 2010;51(5):767–775. DOI: 10.1093/pcp/pcq036

[147] Hu W, Yuan Q, Wang Y, Cai R, Deng X, Wang J, Zhou S, Chen M, Chen L, Huang C, Ma Z, Yang G, He G. Overexpression of a wheat aquaporin gene, TaAQP8, enhances salt stress tolerance in transgenic tobacco. Plant & Cell Physiology 2012;53(12):2127–2141. DOI: 10.1093/pcp/pcs154

[148] Zhou QY, Wang L, Cai X, Wang D, Hua XJ, Qu LQ, Lin JX, Chen T. Net sodium fluxes change significantly at anatomically distinct root zones of rice (*Oryza sativa* L.) seedlings. Journal of Plant Physiology. 2011;168(11):1249–1255. DOI: 10.1016/j.jplph.2011.01.017

[149] Henderson SW, Baumann U, Blackmore DH, Walker AR, Walker RR, Gilliham M. Shoot chloride exclusion and salt tolerance in grapevine is associated with differential ion transporter expression in roots. BMC Plant Biology. 2014;14:273. DOI: 10.1186/s12870-014-0273-8

[150] Vandeule RK, Sullivan W, Athman A, Jordans C, Gilliham M, Kaiser BN, Tyerman SD. Rapid shoot-to-root signaling regulates root hydraulic conductance via aquaporins. Plant, Cell and Environment. 2014;37(2):520–538. DOI: 10.1111/pce.12175

[151] Xu C, Wang M, Zhou L, Quan T, Xia G. Heterologous expression of the wheat aquaporin gene TaTIP2.2 compromises the abiotic stress tolerance of *Arabidopsis thaliana*. PLoS ONE. 2013;8:e79618. DOI: 10.1371/journal.pone.0079618
[152] Sani E, Herzyk P, Perrella G, Colot V, Amtmann A. Hyperosmotic priming of Arabidopsis seedlings establishes a long-term somatic memory accompanied by specific changes of the epigenome. Genome Biology. 2013;14:R59. DOI: 10.1186/gb-2013-14-6-r59

[153] Wang X, Fan P, Song H, Chen X, Li X, Li Y. Comparative proteomic analysis of differentially expressed proteins in shoots of Salicornia europaea under different salinity. Journal of Proteome Research. 2009;8:3331–3345. DOI: 10.1021/pr801083a

[154] Zhou S, Sauvé RJ, Liu Z, Reddy S, Hatti S, Hucko SD, Fish T, Thannhauser TW. Identification of salt-induced changes in leaf and root proteomes of the wild tomato, Solanum chilense. Journal of the American Society for Horticultural Science. 2011;136:288–302.

[155] Wang L, Liu X, Liang M, Tan F, Liang W, Chen Y, Lin Y, Huang L, Xing J, Chen W. Proteomic analysis of salt-responsive proteins in the leaves of mangrove Kandelia candel during short-term stress. PLoS ONE. 2014;9:e83141. DOI: 10.1371/journal.pone.0083141

[156] Cheng T, Chen J, Zhang J, Shi S, Zhou Y, Lu L, Wang P, Jiang Z, Yang J, Zhang S, Shi J. Physiological and proteomic analyses of leaves from the halophyte Tangut nitriata reveals diverse response pathways critical for high salinity tolerance. Front in Plant Science. 2015;6:30. DOI: 10.3389/fpls.2015.00030

[157] Nadeem SM, Ahmad M, Zahir ZA, Javaid A, Ashraf M. The role of mycorrhizae and plant growth promoting rhizobacteria (PGPR) in improving crop productivity under stressful environments. Biotechnology Advances. 2014;32:429–448. DOI: 10.1016/j.biotechadv.2013.12.005

[158] Roy SJ, Huang W, Wang XJ, Evrard A, Schmoeckel SM, Zafar ZU, Tester M. A novel protein kinase involved in Na+ exclusion revealed from positional cloning. Plant, Cell and Environment. 2013;36:553–568. DOI: 10.1111/j.1365-3040.2012.02595.x

[159] Platten JD, Egdane JA, Ismail AM. Salinity tolerance, Na+ exclusion and allele mining of HKT1;5 in Oryza sativa and O. glaberrima. Many sources, many genes, one mechanism? BMC Plant Biology. 2013;13:32. DOI: 10.1186/1471-2229-13-32

[160] Zhou S, Zhang Z, Tang Q, Lan H, Li Y, Luo P. Enhanced V-ATPase activity contributes to the improved salt tolerance of transgenic tobacco plants overexpressing vacuolar Na+/H+ antiporter AtNHX1. Biotechnology Letters 2011;33:375–380. DOI: 10.1007/s10529-010-0418-2

[161] Bailey-Serres J, Lee SC, Brinton E. Waterproofing crops: effective flooding survival strategies. Plant Physiology. 2012;160:1698–1709. DOI: 10.1104/pp.112.208173

[162] Jackson MB, Ismail AM. Electrons, water and rice fields: plant response and adaptation to flooding and submergence stress. AoB Plants. 2015;7:plv078. DOI: 10.1093/aobpla/plv078
[163] Arshad M, and Frankenberger WTJ. Production and stability of ethylene in soil. Biology and Fertility of Soils. 1990;10:29-34. DOI: 10.1007/BF00336121

[164] Gibbs J, Greenway H. Mechanisms of anoxia tolerance in plants: growth, survival and anaerobic catabolism. Functional Plant Biology. 2003;30:1-47. DOI: 10.1071/PP98095

[165] Herzog M, Striker GG, Colmer TD, Pedersen O. Mechanisms of waterlogging tolerance in wheat—a review of root and shoot physiology. Plant, Cell and Environment. 2016;39(5):1068–1086. DOI: 10.1111/pce.12676

[166] Jung KH, Seo YS, Walia H, Cao P, Fukao T, Canlas P, Amonpant F, Bailey-Serres J, Ronald PC. The submergence tolerance regulator Sub1A mediates stress-responsive expression of AP2/ERF transcription factors. Plant Physiology. 2010;152:1674–1692. DOI: 10.1104/pp.109.152157

[167] Xu K, Mackill DJ. A major locus for submergence tolerance mapped on rice chromosome 9. Molecular Breeding. 1996;2:219–224. DOI: 10.1007/BF00564199

[168] Hattori Y, Nagai K, Furukawa S, Song XJ, Kawano R, Sakakibara H, Wu J, Matsumoto T, Yoshimura A, Kitano H, Matsuoka M, Mori H, Ashikari M. The ethylene response factors SNORKEL1 and SNORKEL2 allow rice to adapt to deep water. Nature. 2009;460:1026–1030. DOI: 10.1038/nature08258

[169] Nagai K, Kondo Y, Kitaoka T, Noda T, Kuroh T, Angeles-Shim RB, Yasui H, Yoshimura A, Ashikari M. QTL analysis of internode elongation in response to gibberellin in deep water rice. AoB Plants. 2014;6:plu028. DOI: 10.1093/aobpla/plu028

[170] Campbell MT, Proctor CA, Dou Y, Schmitz AJ, Phansak P, Kruger GR, Zhang C, Walia H. Genetic and molecular characterization of submergence response identifies subtol 6 as a major submergence tolerance locus in maize. PLoS ONE. 2015;10(3):e0120385.

[171] Huang S, Hill RD, Wally OSD, Dionisio G, Ayele BT, Jamil SK, Stasolla C. Hemoglobin control of cell survival/death decision regulates in vitro plant embryogenesis. Plant Physiology. 2014;165:810–825. DOI: 10.11.04/pp.114.239335

[172] Jackson MB, Ram PC. Physiological and molecular basis of susceptibility and tolerance of rice plants to complete submergence. Annals of Botany. 2003;91:227–241. DOI: 10.1093/aob/mcf242

[173] Nishiuchi S, Yamauchi T, Takahashi H, Kotula L, Nakazono M. Mechanisms for coping with submergence and waterlogging in rice. Rice. 2012;5:2. DOI: 10.1186/1939-8433-5-2

[174] Voortman RL. Micronutrients in agriculture and the world food system – future scarcity and implications. Centre for World Food Studies (SOW-VU), VU University, Amsterdam 2012;
Advances in Plant Tolerance to Abiotic Stresses

http://dx.doi.org/10.5772/64350

[175] Gui QZ, Bao LM, Chang ZR, Bao CL. Timing and level of nitrogen supply affect nitrogen distribution and recovery in two contrasting oat genotypes. Journal of Plant Nutrition and Soil Science. 2012;175:614–621. DOI: 10.1002/jpln.201100279

[176] Hermans C, Hammond JP, White PJ, Verbruggen N. How do plants respond to nutrient shortage by biomass allocation? Trends in Plant Science. 2006;11:610–617. DOI: 10.1016/j.tplants.2006.10.007

[177] Kant S, Bi YM, Rothstein SJ. Understanding plant response to nitrogen limitation for the improvement of crop nitrogen use efficiency. Journal of Experimental Botany. 2011a;62:1499–1509. DOI: 10.1093/jxb/erq297

[178] Nacry P, Bouguyon E, Gojon A. Nitrogen acquisition by roots: physiological and developmental mechanisms ensuring plant adaptation to a fluctuating resource. Plant and Soil. 2013;370:1–29. DOI: 10.1007/s11104-013-1645-9

[179] Zhang H, Forde BG. An Arabidopsis MADS box gene that controls nutrient-induced changes in root architecture. Science. 1998;279:407–409. DOI: 10.1126/science.279.5354.407

[180] Kotur Z, Mackenzie N, Ramesh S, Tyerman SD, Kaiser BN, Glass AD. Nitrate transport capacity of the Arabidopsis thaliana NRT2 family members and their interactions with AtNAR2.1. New Phytologist. 2012;194:724–731. DOI: 10.1111/j.1469-8137.2012.04094.x

[181] Sun J, Zheng N. Molecular mechanism underlying the plant NRT1.1 dual-affinity nitrate transporter. Frontiers in Physiology. 2015;6:386. DOI: 10.3389/physiol.2015.00386

[182] De Angeli A, Monachello D, Ephritikhine G, Frachisse JM, Thomine S, Gamble F, Barbier-Brygoo H. The nitrate/proton antiporter AtCLCa mediates nitrate accumulation in plant vacuoles. Nature. 2006;442:939–942. DOI: 10.1038/nature05013

[183] Geiger D, Maierhofer T, Al-Rasheid KA, Scherzer S, Mumm P, Liese A, Ache P, Wellmann C, Marten I, Grill E, Romeis T, Hedrich R. Stomatal closure by fast abscisic acid signaling is mediated by the guard cell anion channel SLAHH and the receptor RCAR1. Science Signaling. 2011;4:ra32. DOI: 10.1126/scisignal.2001346

[184] Segonzac C, Boyer JC, Ipotesi E, Szponarski W, Tillard P, Touraine B, Sommerer N, Rossignol M, Girbat R. Nitrate efflux at the root plasma membrane: identification of an Arabidopsis excretion transporter. The Plant Cell. 2007;19:3760–3777. DOI: 10.1105/tpc.106.048173

[185] Li JY, Fu YL, Pike SM, Bao J, Tian W, Zhang Y, Chen CZ, Zhang Y, Li HM, Huang J, Li LG, Schroeder JJ, Gassmann W, Gong JM. The Arabidopsis nitrate transporter NRT1.8 functions in nitrate removal from the xylem sap and mediates cadmium tolerance. The Plant Cell 2010;22:1633–1646. DOI: 10.1105/tpc.110.075242

[186] Wang YY, Tsay YF. Arabidopsis nitrate transporter NRT1.9 is important in phloem nitrate transport. The Plant Cell 2011;23:1945–1957. DOI: 10.1105/tpc.111.083618
[187] Wang YY, Hsu PK, Tsay YF. Uptake, allocation and signaling of nitrate. Trends in Plant Science. 2012;17:458–467. DOI: 10.1016/j.tplants.2012.04.006

[188] Hu H, Wang Y, Tsay Y. AtCIPK8, a CBL-interacting protein kinase, regulates the low-affinity phase of the primary nitrate response. The Plant Journal. 2009;57:264–278. DOI: 10.1111/j.1365-313X.2008.03685.x

[189] Jonassen EM, Sévin DC, Lillo C. The bZIP transcription factors HY5 and HYH are positive regulators of the main nitrate reductase gene in Arabidopsis leaves, NIA2, but negative regulators of the nitrate uptake gene NRT1.1. Journal of Plant Physiology. 2009;166:2071–2076. DOI: 10.1016/j.jplph.2009.05.010

[190] Konishi M, Yanagisawa S. Arabidopsis Nin-like transcription factors play a central role in nitrate signaling. Nature Communications. 2013;4:1503. DOI: 10.1038/ncomms2621

[191] Pinton R, Tomasi N, Zanin L. Molecular and physiological interactions of urea and nitrate uptake in plants. Plant Signaling & Behavior. 2015;11:e1076603. DOI: 10.1080/15592324.2015.1076603

[192] Yoneyama K, Xie X, Kusumoto D. Nitrogen deficiency as well as phosphorus deficiency in Sorghum promotes the production and exudation of 5-deoxystrigol, the host recognition signal for arbuscular. Planta. 2007b;227:125–132. DOI: 10.1007/s00425-007-0600-5

[193] Walch-Liu P, Ivanov II, Filleur S, Gan Y, Remans T, Forde BG. Nitrogen regulation of root branching. Annals of Botany. 2006;97:875–881. DOI: 10.1093/aob/mcj601

[194] Li J, Zhu S, Song X, Chen Y, Chen H, Yu J, Yi K, Liu Y, Karplus VJ, Wu P, Deng XW. A rice glutamate receptor-like gene is critical for the division and survival of individual cells in the root apical meristem. The Plant Cell 2006;18 340–349. DOI: 10.1105/tpc.105.037713

[195] Fuentes SI, Allen DJ, Ortiz-Lopez A, Hernandez G. Over-expression of cytosolic glutamine synthetase increases photosynthesis and growth at low nitrogen concentrations. Journal of Experimental Botany. 2001;52:1071–1081. DOI: 10.1093/jexbot/52.358.1071

[196] Reddy MM, Ulaganathan K. Nitrogen nutrition, its regulation and biotechnological approaches to improve crop productivity. American Journal of Plant Sciences. 2015;6:2745–2798. DOI: 10.4236/ajps.2015.618275

[197] Vance CP, Uhde-Stone C, Allan DL. Phosphorus acquisition and use: critical adaptations by plants for securing a nonrenewable resource. New Phytologist. 2003;157:423–447. DOI:

[198] Song L, Liu D. Ethylene and plant responses to phosphate deficiency. Frontiers in Plant Science. 2015;6:796. DOI: 10.3389/fpls.2015.00796.
[199] Thibaud MC, Arrighi JF, Bayle V, Chiarenza S, Creff A, Bustos R, Paz-Ares J, Poirier Y, Nussaume L. Dissection of local and systemic transcriptional responses to phosphate starvation in Arabidopsis. The Plant Journal. 2010;64:775–789. DOI: 10.1111/j.1365-313X.2010.04375.x

[200] Wang Z, Rahman AB, Wang G, Ludewig U, Shen J, Neumann G. Hormonal interactions during cluster-root development in phosphate-deficient white lupin (Lupinus albus L.). J Plant Physiology. 2015;177:74–82. DOI: 10.1016/j.jplph.2014.10.022

[201] He CJ, Morgan PW, Drew MC. Enhanced sensitivity to ethylene in nitrogen or phosphate-starved roots of Zea mays L. during aerenchyma formation. Plant Physiology. 1992;98:137–142. DOI: 10.1104/pp.98.1.137

[202] Chacón-López A, Ibarra-Laclette E, Sánchez-Calderón L, Gutiérrez-Alanís D, Herrera-Estrella L. Global expression pattern comparison between low phosphorus insensitive 4 and WT Arabidopsis reveals an important role of reactive oxygen species and jasmonic acid in the root tip response to phosphate starvation. Plant Signaling & Behavior. 2011;6:382–392. DOI: 10.4161/psb.6.3.14160

[203] Lei M, Zhu C, Liu Y, Karthikeyan AS, Bressan RA, Raghothama KG, Liu D. Ethylene signaling is involved in regulation of phosphate starvation-induced gene expression and production of acid phosphatases and anthocyanin in Arabidopsis. New Phytologist. 2011b;189:1084–1095. DOI: 10.1111/j.1469-8137.2010.03555.x

[204] Song G, Liu S. Phosphorus speciation and distribution in surface sediments of the Yellow Sea and East China Sea and potential impacts on ecosystem. Acta Oceanologica Sinica. 2015;34:84–91. DOI: 10.1007/s13131-015-0653-4

[205] Yu H, Luo N, Sun L, Liu D. HPS4/SABRE regulates plant responses to phosphate starvation through antagonistic interaction with ethylene signaling. Journal of Experimental Botany 2012;63:4527–4538. DOI: 10.1093/jxb/eru377

[206] Wang L, Dong J, Gao Z, Liu D. The Arabidopsis gene HYPERSENSITIVE TO PHOSPHATE STARVATION3 encodes ETHYLENE OVERPRODUCTION1. Plant Cell Physiology 2012;53:1093–1105. DOI: 10.1093/pcp/pcs072

[207] Christians MJ, Gingerich DJ, Hansen M, Binder BM, Kieber JJ, Vierstra RD. The BTB ubiquitin ligases ETO1. EOL1 and EOL2 act collectively to regulate ethylene biosynthesis in Arabidopsis by controlling type-2 ACC synthase levels. The Plant Journal. 2009;57:332–345. DOI: 10.1111/j.1365-313X.2008.03693.x

[208] Wang KL, Yoshida H, Lurin C, Ecker JR. Regulation of ethylene gas biosynthesis by the Arabidopsis ETO1 protein. Nature. 2004;428:945–950. DOI: 10.1038/nature02516

[209] Zhang Y, Wang X, Lu S, Liu D. A major root-associated acid phosphatase in Arabidopsis, AtPAP10, is regulated by both local and systemic signals under phosphate starvation. Journal of Experimental Botany. 2014;65(22):6577–6588. DOI: 10.1093/jxb/eru377.
miR399d has enhanced phosphorus accumulation through increased acid phosphatase activity. Responses to phosphorus deficiency are modulated by the gibberellin-DELLA signaling pathway.

Verstappen F, Puech-Pages V, Bécard G, Mulder P, Bouwmeester H. Tomato strigolactones are derived from carotenoids and their biosynthesis is promoted by phosphorus deficiency. 

Li YS, Gao Y, Tian QY, Shi FL, Li LH, Zhang WH. Stimulation of root acid phosphatase by phosphorus deficiency is regulated by ethylene in Medicago falcata. Environmental and Experimental Botany. 2011;71:114–120. DOI: 10.1016/j.envexpbot.2010.11.007

Nacry P, Canivenc G, Muller B, Azmi A, Van Onckelen H, Rossignol M. A role for auxin redistribution in the responses of the root system architecture to phosphate starvation in Arabidopsis. Plant Physiology. 2005;138:2061–2074. DOI: 10.1104/pp.105.060061.

Karthikeyan AS, Varadarajan DK, Jain A, Held MA, Carpita NC, Raghothama KG. Phosphate starvation responses are mediated by sugar signaling in Arabidopsis. Planta. 2007;225:907–918. DOI: 10.1007/s00425-006-0408-8

Niu YF, Chai RS, Jin GL, Wang H, Tang CX, Zhang YS. Responses of root architecture development to low phosphorus availability: a review. Annals of Botany. 2013;112(2):391–408. DOI: 10.1093/aob/mcs285.

Karthikeyan AS, Varadarajan DK, Jain A, Held MA, Carpita NC, Raghothama KG. Phosphate starvation responses are mediated by sugar signaling in Arabidopsis. Planta. 2007;225(4):907–918. DOI: 10.1007/s00425-006-0408-8

López-Bucio J, Hernandez-Abreu E, Sanchez-Calderon L, Nieto-Jacobo MF, Simpson J, Herrera-Estrella L. Phosphate availability alters architecture and causes changes in hormone sensitivity in the Arabidopsis root system. Plant Physiology. 2002;129:244–256. DOI: 10.1104/pp.010934

Jiang C, Gao X, Liao L, Harberd NP, Fu X. Phosphate starvation root architecture and anthocyanin accumulation responses are modulated by the gibberellin-DELLA signaling pathway in Arabidopsis. Plant Physiology. 2007;145:1460–1470. DOI: 10.1104/pp.107.103788

iura K, Rus A, Sharkhuu A, Yokoi S, Karthikeyan AS, Raghothama KG, Baek D, Koo YD, Jin JB, Bressan RA, Yun DJ, Hasegawa PM. The Arabidopsis SUMO E3 ligase SIZ1 controls phosphate deficiency responses. Proceedings of the National Academy of Sciences of the United States of America. 2005;102:7760–7765. DOI: 10.1073/pnas.0500778102

Niu YF, Chai RS, Jin GL, Wang H, Tang CX, Zhang YS. Responses of root architecture development to low phosphorus availability: a review. Annals of Botany. 2013;112:391–408. DOI: 10.1093/aob/mcs285

Ticconi CA, Lucero RD, Sakhonwasee S, Adamson AW, Creff A, Nussaume L, Desnos T, Abel S. ER-resident proteins PDR2 and LPR1 mediate the developmental response of root meristems to phosphate availability. Proceedings of the National Academy of Sciences of the United States of America. 2009;106:14174–14179. DOI: 10.1073/pnas.0901778106
[220] Müller J, Toev T, Heisters M, Teller J, Moore KL, Hause G, Dinesh DC, Bürstenbinder K, Abel S. Iron-dependent callose deposition adjusts root meristem maintenance to phosphate availability. Developmental Cell. 2015;33:216–230. DOI: 10.1016/j.devcel.2015.02.007

[221] Gamuyao R, Chin JH, Pariasca-Tanaka J, Pesaresi P, Catausan S, Dalid C, Slamet-Loedin I, Tecson-Mendoza EM, Wissuwa M, Heuer S. The protein kinase Pstol1 from traditional rice confers tolerance of phosphorus deficiency. Nature. 2012;488:535–539. DOI: 10.1038/nature11346

[222] Pariasca-Tanaka J, Chian J, Drame K, Dalid C, Heuer S, Wissuwa m. A novel allele of the P-starvation tolerance gene OsPSTOL1 from African rice (Oryza glaberrima Steud) and its distribution in the genus Oryza. Theoretical Applied Genetics. 2014;127:1387–1398. DOI: 10.1007/s00122-014-2306-y

[223] Gao N, Yanhua S, Min J, Shen W, Shi W. Transgenic tomato overexpressing ath-miR399d has enhanced phosphorus accumulation through increased acid phosphatase and proton secretion as well as phosphate transporter. Plant and Soil. 2010;334:23–136. DOI: 10.1007/s11104-009-0219-3

[224] Chiou T, Aung K, Lin S, Wu C, Chiang S, Su C. Regulation of phosphate homeostasis by microRNA in Arabidopsis. The Plant Cell. 2006;18:412–421. DOI: 10.1105/tpc.105.038943

[225] López-Bucio J, Hernandez-Abreu E, Sanchez-Calderon L, Nieto-Jacobo MF, Simpson J, Herrera-Estrella L. Phosphate availability alters architecture and causes changes in hormone sensitivity in the Arabidopsis root system. Plant Physiology. 2002;129:244–256. DOI: 10.1104/pp.010934

[226] Jiang C, Gao X, Liao L, Harberd NP, Fu X. Phosphate starvation root architecture and anthocyanin accumulation responses are modulated by the gibberellin-DELLA signaling pathway in Arabidopsis. Plant Physiology. 2007;145:1460–1470. DOI: 10.1104/pp.107.103788

[227] Lopez-Raez JA, Charnikhova T, Gomez-Roldan V, Matusova R, Kohlen W, De Vos R, Verstappen F, Puch-Pages V, Bécard G, Mulder P, Bouwmeester H. Tomato strigolactones are derived from carotenoids and their biosynthesis is promoted by phosphate starvation. New Phytologist. 2008;178:863–874. DOI: 10.1111/j.1469-8137.2008.02406.x

[228] Umehara M, Hanada A, Magome H, Takeda-Kamiya N, Yamaguchi S. Contribution of strigolactones to the inhibition of tiller bud outgrowth under phosphate deficiency in rice. Plant and Cell Physiology. 2010;51:1118–1126. DOI: 10.1093/pcp/pcq084

[229] Kohlen W, Charnikhova T, Liu Q. Strigolactones are transported through the xylem and play a key role in shoot architectural response to phosphate deficiency in non-arbuscular mycorrhizal host. Plant Physiology. 2011;155:974–987. DOI: 10.1104/pp.110.164640
[230] Foo E, Yoneyama K, Hugill CJ, Quittenden LJ, Reid JB. Strigolactones and the regulation of pea symbioses in response to nitrate and phosphate deficiency. Molecular Plant. 2012;6:76–87. DOI: 10.1093/mp/sss115

[231] Mayzlish-Gati E, De-Cuyper C, Goormachtig S, Beeckman T, Vuylsteke M, Brewer PB, Beveridge CA, Yermiyahu U, Kaplan Y, Enzer Y, Wininger S, Resnick N, Cohen M, Kapulnik Y, Koltai H. Strigolactones are involved in root response to low phosphate conditions in Arabidopsis. Plant Physiology. 2012;160:1329–1341. DOI: 10.1104/pp.112.202358

[232] Liao H, Yan XL, Rubio G, Beebe SE, Blair MW, Lynch JP. Genetic mapping of basal root gravitropism and phosphorus acquisition efficiency in common bean. Functional Plant Biology. 2004;31:959–970. DOI: 10.1071/FP03255

[233] Beebe SE, Rojas-Pierce M, Yan XL Matthew W. Blair MM, Pedraza F, Muñoz F, Tohme J, Lynch JP. Quantitative trait loci for root architecture traits correlated with phosphorus acquisition in common bean. Crop Science. 2006;46: 413–423. DOI: 10.2135/cropsci2005.0226

[234] Ochoa IE, Blair MW, Lynch JP. QTL analysis of adventitious root formation in common bean under contrasting phosphorus availability. Crop Science 2006, 46:1609–1621. DOI: 10.2135/cropsci2005.12-0446

[235] Yan X, Liao H, Beebe SE, Blair MW, Lynch JP. QTL mapping of root hair and acid exudation traits and their relationship to phosphorus uptake in common bean. Plant and Soil. 2004;265:17–29. DOI: 10.1007/s11104-005-0693-1

[236] Li YD, Wang YJ, Tong YP, Gao JG, Zhang JS, Chen SY. QTL mapping of phosphorus deficiency tolerance in soybean (Glycine max L. Merr.). Euphytica. 2005;142:137–142. DOI: 10.1007/s10681-005-0693-1

[237] Zhang D, Cheng H, Geng L, Kan S, Cui S, Meng Q, Gai J, Yu, D. Detection of quantitative trait loci for phosphorus deficiency tolerance at soybean seedling stage. Euphytica. 2009;167:313–322. DOI: 10.1007/s10681-009-9880-0

[238] Hodson MJ. Metal toxicity and tolerance in plants [Internet]. 2012; Available from: http://www.hodsons.org/MartinHodson/Biochemist12.pdf [Accessed: 2016-05-03]

[239] Uexküll HR, Mutert E. Global extent, development and economic impact of acid soils. Plant and Soil. 1995;171:1–15. DOI: 10.1007/BF00009558

[240] Kochian LV. Cellular mechanisms of aluminum toxicity and resistance in plants. Annual Reviews in Plant Biology. 1995;46:237–260. DOI: 10.1146/annurev.pp.46.060195.001321

[241] Kinraide TB. Assessing the rhizotoxicity of the aluminate ion, Al(OH)₄⁻. Plant Physiology. 1990;93:1620–1625. DOI: 10.1146/annurev.pp.46.060195.001321
Advances in Plant Tolerance to Abiotic Stresses 225
http://dx.doi.org/10.5772/64350

[242] Delhaize E, Ryan PR, Randall PJ. Aluminum tolerance in wheat (Triticum aestivum L.) (II. Aluminum-stimulated excretion of malic acid from root apices). Plant Physiology. 1993;103:695–702. DOI: 10.1104/pp.103.3.695

[243] Klug B, Horst WJ. Spatial characteristics of aluminum uptake and translocation in roots of buckwheat (Fagopyrum esculentum). Physiologia Plantarum. 2010;139:181–191. DOI: 10.1111/j.1399-3054.2010.01355.x

[244] Li JY, Liu J, Dong D, Jia X, McCouch SR, Kochian LV. Natural variation underlies alterations in Nramp aluminum transporter (NRAT1) expression and function that play a key role in rice aluminum tolerance. Proceedings of the National Academy of Sciences of the United States of America. 2014;111:6503–6508. DOI: 10.1073/pnas.1318975111

[245] Bian M, Zhou M, Sun D, Li C. Molecular approaches unravel the mechanism of acid soil tolerance in plants. The Crop Journal. 2013;1:91–104. DOI: 10.1016/j.cj.2013.08.002

[246] Zhou GF, Delhaize E, Zhou MX, Ryan PR. The barley MATE gene, HvAACT1, increases citrate efflux and Al3+ tolerance when expressed in wheat and barley. Annals of Botany. 2013;112:603–612. DOI: 10.1093/aob/mct135

[247] Sasaki T, Yamamoto Y, Ezaki B, Katsuhara M, Ahn SJ, Ryan PR, Delhaize E, Matsutomo H. A wheat gene encoding an aluminum-activated malate transporter. The Plant Journal. 2004;37:645–653. DOI: 10.1111/j.1365-313X.2003.01991.x

[248] Maron LG, Pineros MA, Guimarães CT, Magalhaes JV, Pleiman JK, Mao C, Shaff J, Belicuaus SN, Kochian LV. Two functionally distinct members of the MATE (multidrug and toxic compound extrusion) family of transporters potentially underlie two major aluminum tolerance QTLs in maize. The Plant Journal. 2010;61:728–740. DOI: 10.1111/j.1365-313X.2009.04103.x

[249] Ryan PR, Raman H, Gupta S, Horst WJ, Delhaize E. A second mechanism for aluminum resistance in wheat relies on the constitutive efflux of citrate from roots. Plant Physiology. 2009;149:340–351. DOI: 10.1104/pp.108.129155

[250] Magalhaes JV, Liu J, Guimarães CT, Lana UGP, Alves VM, Wang YH, Schaffert RE, Hoekenga OA, Piñeros MA, Shaff JE, Klein PE, Carneiro NP, Coelho CM, Trick HN, Kochian LV. A gene in the multidrug and toxic compound extrusion (MATE) family confers aluminum tolerance in sorghum. Nature Genetics. 2007;39:1156–1161. DOI: 10.1038/ng2074

[251] Hoekenga OA, Maron LG, Piñeros MA, Cancado GM, Shaff J, Kobayashi Y, Ryan PR, Dong B, Delhaize E, Sasaki T, Matsumoto H, Yamamoto Y, Koyama H, Kochian LV. AtALMT1, which encodes a malate transporter, is identified as one of several genes critical for aluminum tolerance in Arabidopsis. Proceedings of the National Academy of Sciences of the United States of America. 2006;103:9738–9743. DOI: 10.1073/pnas.0602868103
[252] Liu J, Magalhaes JV, Shaff J, Kochian LV. Aluminum-activated citrate and malate transporters from the MATE and ALMT families function independently to confer Arabidopsis aluminum tolerance. The Plant Journal. 2009;57:389–399. DOI: 10.1111/j.1365-313X.2008.03696.x

[253] Silva-Navas J, Benito C, Téllez-Robledo B, El-Moneim A, Gallego FJ. The ScAACT1 gene at the Qalt5 locus as a candidate for increased aluminum tolerance in rye (Secale cereale L.). Molecular Breeding. 2011;30(2):845–856. DOI: 10.1007/s11032-011-9668-5

[254] Huang J, Xia H, Li Z, Xiong Y, Kong G. Soil aluminum uptake and accumulation by Paspalum notatum. Waste Management Research 2009b;27:668–675. DOI:

[255] Fujii M, Yokosho K, Yamaji N, Saisho D, Yamane M, Takahashi H, Sato K, Nakazono M, Ma JF. Acquisition of aluminium tolerance by modification of a single gene in barley. Nature Communications. 2012;3:713. DOI: 10.1038/ncomms1726

[256] Arenhart RA, Lima JC, Pedron M, Carvalho FE, Silveira JA, Rosa SB, Caverzan A, Andrade CM, Schünemann M, Margis R, Margis-Pinheiro M. Involvement of ASR genes in aluminum tolerance mechanisms in rice. Plant Cell & Environment. 2013b; 36:52–67. DOI: 10.1111/j.1365-3040.2012.02553.x

[257] Gao CJ, Xing D, Li LL, Zhang LR. Implication of reactive oxygen species and mitochondrial dysfunction in the early stages of plant programmed cell death induced by ultraviolet-C overexposure. Planta. 2008;227:755–767. DOI: 10.1007/s00425-007-0654-4

[258] Zhu HF, Wang H, Zhu YF, Zou JW, Zhao FJ, Huang CF. Genome-wide transcriptomic and phylogenetic analyses reveal distinct aluminum-tolerance mechanisms in the aluminum-accumulating species buckwheat (Fagopyrum tataricum). BMC Plant Biology. 2015;15:16. DOI: 10.1186/s12870-014-0395-z

[259] Hanikenne M, Talke IN, Haydon MJ, Lanz C, Nolte A, Motte P, Kroymann J, Weigel D, Krämer U. Evolution of metal hyperaccumulation required cis-regulatory changes and triplication of HMA4. Nature. 2008;453(7193):391–395. DOI: 10.1038/nature06877

[260] Kwon H-K, Yokoyama R, Nishitani K. A proteomic approach to apoplastic proteins involved in cell wall regeneration in protoplasts of Arabidopsis suspension-cultured cells. Plant Cell Physiology. 2005;46:843–857. DOI: 10.1093/pcp/pct089

[261] Bhuja P, McLachlan K, Stephens J, Taylor G. Accumulation of 1,3-beta-D-glucans, in response to aluminum and cytosolic calcium in Triticum aestivum. Plant Cell Physiology. 2004;45:543–549. DOI: 10.1093/pcp/pch068

[262] Sivaguru M, Horst WJ, Eticha D, Matsumoto H. Aluminum inhibits apoplastic flow of high-molecular weight solutes in root apices of Zea mays L. Journal of Plant Nutrition and Soil Science. 2006;169:679–690. DOI: 10.1002/jpln.200620603

[263] O’Malley RC, Rodriguez Fl, Esch JJ, Binder BM, O’Donnell P, Klee HJ, Bleecker AB. Ethylene-binding activity, gene expression levels, and receptor system output for
ethylene receptor family members from *Arabidopsis* and tomato. The Plant Journal. 2005;41:651–659. DOI: 10.1111/j.1365-313X.2004.02331.x

[264] Tian Q, Zhan X, Ramesh S, Gillilham M, Tyerman SD, Zhan W-H. Ethylene negatively regulates aluminium-induced malate efflux from wheat roots and tobacco cells transformed with TaALMT1. Journal of Experimental Botany 2014;65:2415–2426. DOI:

[265] Sun P, Tian Q-Y, Chen J, Zhang W-H. Aluminium-induced inhibition of root elongation in *Arabidopsis* is mediated by ethylene and auxin. Journal of Experimental Botany. 2010;61:347–356. DOI: 10.1093/jxb/erp306

[266] He H, He L, Gu M. Interactions between nitric oxide and plant hormones in aluminum tolerance. Plant Signaling and Behavior. 2012;7:469–471. DOI: 10.4161/psb.19312

[267] He HY, He LF, Gu MH. Role of microRNAs in aluminum stress in plants. Plant Cell Reports. 2014;33:831–836. DOI: 10.1007/s00299-014-1565-z

[268] Sahrawat KL. Iron toxicity in wetland rice and the role of other nutrients. Journal of Plant Nutrition. 2005;27:1471–1504. DOI: 10.1081/PLN-200025869

[269] Keita A, Yacouba H, Hayde LG, Schultz B. A single-season irrigated rice soil presents higher iron toxicity risk in tropical savannah valley bottoms. Open Soil Science. 2013;3:314–322. DOI: 10.4236/ojss.2013.37036

[270] Onaga G, Edema R, Asea G. Tolerance of rice germplasm to iron toxicity stress and the relationship between tolerance, Fe^{2+}, P and K content in the leaves and roots. Archives of Agronomy and Soil Science. 2013a;59:213–229. DOI: 10.1080/03650340.2011.622751

[271] Roschttardtz H, Conéjéro G, Divol F, Alcon C, Verdeil JL, Curie C, Mari S. New insights into Fe localization in plant tissues. Frontiers in Plant Science. 2013;4:350. DOI: 10.3389/fpls.2013.00350

[272] Staal M, Cnoddier TD, Simon D, Vandenbussche F, Straeten DVD, Verbelen JP, Elzenaga T, Vissenberg K. Apoplastic alkalization is instrumental for the inhibition of cell elongation in the *Arabidopsis* root by the ethylene precursor 1-aminocyclopropane-1-carboxylic acid. Plant Physiology. 2011;155:2049–2055. DOI: 10.1104/pp.110.168476

[273] Quinet M, Vromman D, Clippe A, Bertin P, Lequeux H, Dufey I, Lutts S, Lefèvre I. Combined transcriptomic and physiological approaches reveal strong differences between short and long term response of rice (*Oryza sativa*) to iron toxicity. Plant Cell & Environment. 2012;35:1837–1859. DOI: 10.1111/j.1365-3040.2012.02521.x

[274] Majerus V, Bertin P, Swenden V, Fortemps A, Lobréaux S, Lutts S. Organ-dependent responses of the African rice to short-term iron toxicity: ferritin regulation and antioxidative responses. Biologia Plantarum 2007;51(2):303–312. DOI: 10.1007/s10535-007-0060-6
Plants being sessile in nature encounter numerous biotic agents, including bacteria, fungi. Significant advances have been made in understanding the plant defence mechanisms expressed against biotic stresses. These mechanisms range from anatomy, physiology, biochemistry to genetics.

However, there are drawbacks and insufficiencies in precisely deciphering and deploying knowledge to develop biotic stress resistant germplasm. In this chapter, we provide a comprehensive overview and discussed plant defence mechanisms involving models and approaches.

References:

[275] Peng XX, Yu XL, Li MQ, Yamauchi M. Induction of peroxidase by Fe 2+ in detached rice leaves. Plant and Soil. 1996;180:159–163. DOI: 10.1007/BF00015422

[276] Fang WC, Wang JW, Lin CC, Kao CH. Iron induction of lipid peroxidation and effects on antioxidative enzyme activities in rice leaves. Plant Growth Regulation. 2001;35(1):75–80. DOI: 10.1023/A:1013879019368

[277] Stein RJ, Lopes SIG, Fett JP. Iron toxicity in field-cultivated rice: contrasting tolerance mechanisms in distinct cultivars. Theoretical and Experimental Plant Physiology. 2014;26:135–146. DOI: 10.1007/s40626-014-0013-3

[278] Wu LB, Shhadi MY, Gregorio G, Matthus E, Becker M, Frei M. Genetic and physiological analysis of tolerance to acute iron toxicity in rice. Rice 2014;7:8. DOI: 10.1186/s12284-014-0008-3

[279] Dufey I, Mathieu AS, Draye X, Lutts S, Bertin P. Construction of an integrated map through comparative studies allows the identification of candidate regions for resistance to ferrous iron toxicity in rice. Euphytica. 2015a;203:59–69. DOI: 10.1007/s10681-014-1255-5

[280] Fukuda A, Shiratsuchi H, Fukushima A, Yamaguchi H, Mochida H, Terao T, Ogiwara H. Detection of chromosomal regions affecting iron concentration in rice shoots subjected to excess ferrous iron using chromosomal segment substitution lines between Japonica and Indica. Plant Production Science. 2012;15:183–191. DOI: 10.1626/pps.15.183

[281] Shimizu A. QTL analysis of genetic tolerance to iron toxicity in rice (Oryza sativa L.) by quantification of bronzing score. Journal of New Seeds. 2009;10(3):171–179. DOI: 10.1080/15228860903064989

[282] Yuan F, Yang H, Xue Y, Kong D, Ye R, Li C, Zhang J, Theprungsirikul L, Shrift T, Krivilsky B, Johnson DM, Swift GB, He Y, Siedow JN, Pei ZM. OSCA1 mediates osmotic-stress evoked Ca2+ increases vital for osmosensing in Arabidopsis. Nature. 2014;514(7522):367-371. DOI: 10.1038/nature13593