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Plant hormone regulation of abiotic stress responses

Rainer Waadt[1,2], **Charles A Seller**[3], **Po-Kai Hsu**[3], **Yohei Takahashi**[3], **Shintaro Munemasa**[4], **Julian I Schroeder**[3,*]

[1]Institute of Technology, University of Tartu, Tartu, Estonia

[2]Institut für Biologie und Biotechnologie der Pflanzen, Westfälische Wilhelms-Universität Münster, Münster, Germany

[3]Division of Biological Sciences, Cell and Developmental Biology Section, University of California San Diego, La Jolla, CA, USA

[4]Graduate School of Environmental and Life Science, Okayama University, Tsushima-Naka, Okayama, Japan

Abstract

Plant hormones are signaling compounds regulating critical aspects of growth, development, and environmental stress responses. Abiotic stresses, such as drought, salinity, heat, cold and flooding have profound effects on plants. Adaptations to such stresses require sophisticated sensing, signaling and response mechanisms for stress tolerance. Here we review recent advances in hormonal control of abiotic stress responses in plants and highlight points of hormonal crosstalk during abiotic stress signaling. Specific topics are addressed, including osmotic stress sensory and signaling mechanisms, hormonal control of gene regulation and plant development during stress, hormone-regulated submergence tolerance and stomatal movements. We further explore how innovative imaging approaches are providing insights into single cell and tissue hormone dynamics. Recent advances open new opportunities for agricultural applications.

Introduction

Plants are a major source for food, fuel and fiber, and important contributors to the ecological diversity and sustainability of our planet. To optimize their growth and productivity under changing environmental conditions, that are intensified due to climate change, plants have developed sophisticated mechanisms to sense and respond to external stresses[1,2]. Among them, abiotic stresses appear in various forms, associated either with weather conditions, i.e. rainfall, temperature and irradiation from the sun, or to the quality of the soil in which plants grow, i.e. the content of water, nutrients and soil contaminants[1]. In particular, changes in water availability and temperature, e.g. heat stress, have been associated with climate change[2].

* jischroeder@ucsd.edu .

Competing interests

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To develop concepts and approaches for protecting plants from the negative effects of abiotic stresses, and to secure the future demands for plant products, we need to understand the mechanisms of plant stress responses at the molecular level. Plant hormones, i.e. abscisic acid (ABA), auxin, brassinosteroid (BR), cytokinin (CK), ethylene (ET), gibberellic acid (GA), jasmonic acid (JA), salicylic acid (SA) and strigolactone (SL) are well known plant growth regulators that mediate adaptations to environmental conditions. For an overview on their functions and respective signaling mechanisms, please refer to a recent review[3] and references therein. The diverse roles of phytohormones in abiotic stress responses are reviewed here, and many of these roles are listed in Table 1.

Here, we review how plant hormones and other signaling compounds mediate plant responses to abiotic stresses, including drought, osmotic stress and flooding. We further summarize the current view on how osmotic stress is sensed by plants, and how this leads to the activation of SnRK2-type protein kinases and interactions with plant hormone signaling modules. Then we elaborate on phytohormone-dependent gene regulatory mechanisms that mediate abiotic stress responses of plants. We also highlight how hormones regulate seed germination, how ABA and auxin coordinate root growth under stress, the effects of stress-dependent hormone responses on flowering time, the ethylene- and gibberellic acid-mediated regulation of plant responses to flooding, and the ABA and abiotic stress sensing mechanisms regulating stomatal aperture. Abiotic stresses further cause bud dormancy, leaf senescence, and organ abscission, which is reviewed elsewhere[4]. Finally, we review how biosensor-based hormone imaging techniques are contributing to the elucidation of hormone dynamics under abiotic stress.

Osmotic stress sensing and signaling

Water uptake from the soil and water movements within plants are driven by water potential gradients. Hypoosmotic stress, such as flooding, leads to cell swelling, whereas hyperosmotic stress, such as drought and salinity, leads to plant wilting. Plants have evolved osmotic stress adaptive mechanisms, including the regulation of cellular osmoticum concentrations, stomatal movements, and plant development through ABA-dependent and ABA-independent pathways[1,5]. Here, we summarize the current understanding of osmotic sensory and signaling mechanisms in plants.

Osmotic- and salt stress sensing.

Plants can sense the alteration of turgor, the mild change of solute concentrations in cells, and the mechanical effects on cellular structures caused by osmotic stress. Calcium signaling is suggested to play a key role in osmosensing because cytosolic-free calcium concentrations ($[Ca^{2+}]_{cyt}$) in plants rapidly and transiently increase within seconds of exposure to osmotic shock[6,7]. The roles of mechanosensitive ion channels in osmotic stress sensing have been investigated (FIG. 1a). MECHANOSENSITIVE CHANNELS OF SMALL CONDUCTANCE-LIKE (MSLs) are non-selective ion channels activated by membrane tension for osmoregulation during hypo-osmolality in organelles, hydration and germination in pollen, touch responses in roots, and cell swelling[8–10]. MID1-COMPLEMENTING ACTIVITY (MCA)-type Ca^{2+}-permeable channels are activated by membrane tension

and are suggested to mediate hypo-osmotic shock and touch sensing in roots[11]. They also function in mediating cold tolerance and cold-induced $[Ca^{2+}]_{cyt}$ increases[12]. Another mechanosensitive ion channel PIEZO1 (PZO1) is required for mechanotransduction at root tips[13]. Interestingly, plant PIEZOs are localized in the vacuolar membrane to regulate $[Ca^{2+}]_{cyt}$ oscillations and tip growth in *Physcomitrium patens* caulonemal cells and mediate vacuole tubulation in the tips of Arabidopsis pollen tubes[14], suggesting a potential function under hypo-osmotic stress.

A potential osmosensor REDUCED HYPEROSMOLALITY-INDUCED $[Ca^{2+}]_{cyt}$ INCREASE 1 (OSCA1) is involved in hyperosmotic stress-induced $[Ca^{2+}]_{cyt}$ increases for osmotic stress tolerance[15] (FIG. 1b). OSCA1 was initially characterized as a hyperosmolality-activated Ca^{2+}-permeable cation channel[15]. Later studies reported that OSCA family proteins function as stretch-activated channels[16,17]. Ca^{2+}-responsive phospholipid-binding BONZAI (BON) proteins were recently reported to mediate hyperosmotic stress tolerance by positively regulating osmotic stress-induced $[Ca^{2+}]_{cyt}$ increases, ABA accumulation, and gene expression[18]. These membrane-associated Ca^{2+}-responsive BON proteins may be involved in osmotic sensing and signaling by regulating the initial $[Ca^{2+}]_{cyt}$ elevation together with plasma membrane Ca^{2+} transporters[19] (FIG. 1b). Defects in plant growth and ABA accumulation under osmotic stress in *bon* mutants can be restored by crossing *bon* mutants with *snc1–11* and *pad4* mutant alleles that are impaired in nucleotide-binding domain and leucine-rich repeat (NLR) immune signaling[18]. Therefore, BONs may confer osmotic stress responses by suppressing NLR immune signaling. Although several osmotic stress-linked mechanisms have been characterized, further research is needed to dissect their differential functions. Notably, the activation of SUCROSE NONFERMENTING 1-RELATED PROTEIN KINASE 2 (SnRK2) kinases in response to hyperosmotic shock is not impaired in *osca* septuple and *bon1 bon2 bon3* triple mutants[18,20] indicating the need to identify additional osmotic stress sensing and signaling mechanisms.

Plants sense salinity and induce rapid $[Ca^{2+}]_{cyt}$ transients[6,21] to trigger salt tolerance responses through the Salt Overly Sensitive (SOS) pathway[1] (FIG. 1c). Under salt stress, the receptor-like kinase FERONIA may sense cell wall defects caused by salinity, and elicits cell-specific $[Ca^{2+}]_{cyt}$ transients for maintaining cell wall integrity[22]. FERONIA potentially also interferes with ABA signaling via interaction with the PROTEIN PHOSPHATASE TYPE 2C (PP2C) ABA INSENSITIVE 2 (ABI2)[23]. In Arabidopsis, the current hypothesis is that plasma membrane glycosyl inositol phosphorylceramide sphingolipids (GIPCs) function in salt sensing[24]. In this model, GIPC lipid formation is catalyzed by the protein MOCA1/IPUT1 (MONOCATION-INDUCED $[Ca^{2+}]_i$ INCREASES 1/Inositol Phosphorylceramide Glucuronosyltransferase 1), and binding of Na^+ ions to GIPC lipids activates Ca^{2+} influx channels[24]. Disruption of the ANNEXIN gene *AtANN4*, a putative Ca^{2+} permeable channel component, impairs salt stress-induced $[Ca^{2+}]_{cyt}$ transients by ~40%, while Ca^{2+}-activated SOS2-SCaBP8/CBL10 complexes negatively regulate AtANN4 by phosphorylation to fine-tune salt tolerance responses[25].

Osmotic stress-induced ABA biosynthesis.

Endogenous ABA concentrations increase approximately 2.5 to 6 hours after exposure to water deficiency[26–29]. Stress-induced *de novo* ABA synthesis depends on the induction of the *NCED3* gene that encodes a 9-cis-epoxycatoteinoid dioxygenase catalyzing the rate-limiting step for ABA biosynthesis[30]. Posttranslational processing of NCED3 in the chloroplast has also been reported to regulate ABA accumulation[31]. In response to water deficiency in roots, a hydraulic signal contributes to a rapid root-to-shoot water deficiency signal to trigger ABA biosynthesis in Arabidopsis leaves and stomatal closure[26]. In addition, a small peptide CLAVATA3/EMBRYO-SURROUNDING REGION-RELATED 25 (CLE25) is induced in the root vasculature during drought stress and moves to aerial tissues to induce *NCED3* gene expression likely through BARELY ANY MERISTEM (BAM1 and BAM3) receptor-like kinases[32] (FIG. 1b). At the transcriptional level, an Arabidopsis NAC transcription factor ATAF1 was suggested to regulate *NCED3* expression to enhance ABA accumulation[33]. Moreover, the NGATHA (NGA) protein family, including four members in Arabidopsis, were identified as transcriptional activators regulating *NCED3* expression through direct binding to a *cis*-acting element (CACTTG) in the 5’ UTR of the *NCED3* promoter[30].

An emerging role of Raf-like MAPKKKs.

An ABA-independent rapid osmotic stress signal transduction pathway and an ABA-dependent pathway converge at the level of SnRK2-type protein kinase activation. The Arabidopsis genome encodes ten *SnRK2* genes. Except for SnRK2.9, the other SnRK2s are activated by osmotic stress, whereas only SnRK2.2, SnRK2.3, and SnRK2.6/OST1 are clearly activated by ABA[34–37]. ABA-dependent SnRK2 activation through the PYRABACTIN RESISTANCE 1 (PYR1)/PYR1-LIKE (PYL)/REGULATORY COMPONENT OF ABA RECEPTOR (RCAR) and PP2C ABA sensing module has been well described[38]. Since SnRK2 activation by osmotic stress is not impaired in ABA-insensitive dominant negative PP2C Arabidopsis mutants[34,39], osmotic stress employs other signaling mechanisms to activate SnRK2 kinases. This ABA-independent pathway is still largely unknown including the identity of the contributing osmotic stress sensors.

SnRK2s have an auto-phosphorylation activity which enhances the kinase activity itself[40]. Previous *in vitro* studies identified a key phosphorylation site at Ser-175 within the activation-loop of the SnRK2.6/OST1 kinase domain[40]. *In vivo* analyses revealed that both ABA and osmotic stress induce phosphorylation at Ser-171 and Ser-175 residues[41].

Recent studies identified Raf-like MAP kinase kinase kinases (Raf-like M3Ks) to be required for phosphorylation-dependent SnRK2 activation via osmotic stress and ABA signaling [20,42–44] (FIG. 1b). SnRK2.6/OST1 was found to be impaired in auto-activation after dephosphorylation by PP2Cs[42]. The re-activation of SnRK2.6/OST1 requires the initial trans-phosphorylation at Ser-171 or Ser-175 by members of the Arabidopsis B2 and B3 subgroup of Raf-like M3Ks[42,44]. Moreover, the *raf-like m3kδ1/δ6/δ7* triple knock-out mutant exhibited not only a reduced SnRK2 kinase activation by ABA but also impairment in SnRK2 activation by osmotic stress[42]. Important functions of B4 subgroup

Raf-like M3Ks in osmotic stress-, but not in ABA-induced rapid SnRK2 activation, were identified[20,43] (FIG. 1b).

Roles for Raf-like M3Ks in ABA responses were initially identified in genetic ABA/stress response screens in Arabidopsis and in moss[45,46]. In the moss *Physcomitrium patens*, the ABA and abiotic stress-responsive Raf-like kinase (*ARK/ANR*), a single ancestral gene similar to the Arabidopsis B3 subgroup, has a role in osmotic stress- and ABA signaling[46–48]. A recent study reported that this ARK/ANR kinase is activated by ABA[47]. The Arabidopsis B3 Raf-like M3K subgroup contains another well-studied gene, *CONSTITUTIVE TRIPLE RESPONSE 1* (*CTR1*), functioning as a negative regulator of ethylene signaling. Ethylene deactivates CTR1 through ethylene receptors, a family of histidine kinases including ETHYLENE RESPONSE 1 (ETR1), which directly binds to CTR1[49]. Interestingly, *Physcomitrium patens* ARK/ANR (also known as *PpCTR1L*) mediates not only ABA signaling in moss, but also ethylene responses[50], which might suggest a role of Raf-like M3Ks as a signaling “hub”. How osmotic stress sensors are linked to Raf-like M3Ks, SnRK2 activation and downstream components remains to be determined.

Gene regulation under abiotic stress

Changes in gene expression mediate many of the effects of phytohormones. Early genomic technologies revealed that abiotic stress-linked ABA increases change the mRNA levels of thousands of genes (e.g. REF.[51]). This, together with the discovery that many classic ABA-insensitive mutations were mapped to transcriptional regulators suggested a prominent role for gene regulation in abiotic stress resistance[52].

ABA-mediated transcriptional regulation and hormone crosstalk.

Early studies discovered a conserved *cis*-acting regulatory element known as the *ABA-RESPONSIVE ELEMENT (ABRE)* in the promoters of drought-induced genes[53]. *ABREs* are recognized by bZIP-type transcription factors (TFs), including a family of four ABRE-binding proteins/ABRE-binding factors (AREBs/ABFs)[54], and the closely related ABA-INSENSITIVE 5 (ABI5)[52,55,56] (FIG. 2). During ABA signaling, ABA-dependent SnRK2-type protein kinases directly phosphorylate and activate AREBs/ABFs and ABI5[57–59]. In Arabidopsis, the four partially redundant AREBs/ABFs are responsible for most of the transcriptional responses to ABA during vegetative growth[60], whereas ABI5 is more important during seed germination[61] (see below). Many of the AREB/ABF targets are other TF genes, implying that a multilevel transcription factor hierarchy controls ABA-dependent transcriptome remodeling[62–65]. A seminal study using ChIP-Seq to profile the genome-wide binding sites of 21 TFs during the ABA response revealed that many binding events are dynamic, and that multiple TFs can target the same gene[64]. Crucially, the ABA-induced binding of some TFs was positively correlated with the presence of adjacent *ABRE* sites, suggesting that some TFs may act cooperatively with AREBs/ABFs. Indeed, several NAC family transcription factors are required for ABA-dependent transcription events including ANAC096, which interacts with ABF2 to activate *RD29A* transcription[62,66,67].

In the absence of abiotic stress, repression of ABA signaling promotes optimal growth. For instance, under non-stress conditions mRNA levels of *ABI5* are low, and *ABI5* transcription

is increased upon exposure to ABA or osmotic stress[55]. This repression of *ABI5* requires the SWI2/SNF2 chromatin remodeling ATPase BRAHMA[68]. In the absence of ABA, BRAHMA inhibits the transcription of *ABI5* by promoting nucleosome occupancy at the transcription start site of the *ABI5* gene. Interestingly, phosphorylation of BRAHMA by SnRK2.2/2.3/2.6 appears to inhibit its action and in turn, this inhibitory phosphate is removed by group A PP2Cs[69] (FIG. 2).

Different hormone pathways interact to control numerous aspects of plant life in the absence of abiotic stress and we direct readers to a review on this topic for a concentrated discussion[70]. Here we focus on how transcriptional regulation enables hormone crosstalk during drought stress responses. For instance, the ABA-induced TF RESPONSE TO DESSICATION 26 (RD26) represses a subset of brassinosteroid-induced genes[62,71]. Furthermore, brassinosteroid-activated TFs repress the expression of *RD26*, suggesting that antagonistic crosstalk between ABA and brassinosteroid signaling contributes to drought stress responses[72] (FIG. 2). Intriguingly, overexpression of the vascular-enriched brassinosteroid receptor BRL3 causes constitutive expression of some drought-induced genes, including *RD26*, and promotes drought resistance[73]. The cytokinin and ABA pathways also converge on the level of transcriptional control. SnRK2-mediated phosphorylation of the type-A ARABIDOPSIS RESPONSE REGULATOR ARR5, a negative regulator of cytokinin signaling, promotes its protein stability thereby downregulating cytokinin responses during drought stress[74]. Oppositely, cytokinin can trigger the degradation of ABI5 to promote seed germination[75] (FIG. 2). Furthermore, dehydration induces the expression of IAA5 and IAA19, two transcriptional repressors of the auxin signaling pathway, indicating that auxin responses are repressed during drought stress[76].

Post-transcriptional abiotic stress responses.

Post-transcriptional processes expand gene regulatory possibilities beyond transcriptional control, and recent research has uncovered the contributions of such mechanisms in shaping ABA responses. The discovery of a mutant in the mRNA cap-binding protein *abh1* exhibiting ABA-hypersensitivity established an early link between mRNA processing and ABA signaling[77]. Alternative mRNA splicing, a process producing multiple distinct mRNA isoforms from a single gene, is modulated by abiotic stress and regulates ABA responses[78–82]. *HAB1* for instance, a group A *PP2C* gene, encodes multiple splice isoforms, of which *HAB1.2* retains an intron leading to a non-functional protein and ABA hypersensitivity[79,80].

Recently mRNA decay has emerged as an additional mechanism contributing to abiotic stress responses. Degradation of mRNA molecules is mediated by mRNA decapping, the removal of the 5’ methyl-guanosine cap[83]. During osmotic stress, ABA-independent subclass I SnRK2-type protein kinases phosphorylate the decapping activator VARICOSE (VCS) leading to the destabilization of some transcripts[84]. The 5’ end of mRNAs can be alternatively modified by the addition of a nicotinamide adenine dinucleotide (NAD^+). In plants, the NAD^+ cap occurs on many transcripts and is thought to down-regulate gene expression by promoting the degradation of marked mRNAs[85,86]. A recent study

demonstrated that the NAD^+-capped transcriptome undergoes extensive changes in response to ABA and that many ABA-induced transcripts lose their NAD^+ caps following ABA treatment possibly promoting their stability[87].

Growth regulation under abiotic stress

Phytohormones regulate many aspects of plant growth and development. Recent discoveries have begun to illuminate how they control different strategies of plant growth and development in response to abiotic stress.

TOR interaction with abiotic stress responses.

The protein kinase TARGET OF RAPAMYCIN (TOR) is a central developmental and metabolic regulator in plants[88]. A reciprocal regulation between ABA and TOR pathways has been proposed to coordinate plant growth and abiotic stress responses[89]. TOR phosphorylates PYL/RCAR ABA receptors to inhibit ABA signaling and promote growth under non-stress conditions, whereas ABA-activated SnRK2 kinases phosphorylate the REGULATORY-ASSOCIATED PROTEIN OF TOR 1B (RAPTOR1B) to inhibit TOR kinase activity and repress growth in response to abiotic stress conditions (FIG. 2).

Gibberellic acid, ABA and the decision to germinate.

The regulation of seed germination promotes seedling survival by coordinating embryo development and emergence with environmental conditions. The balance of two competing hormone signaling pathways, gibberellic acid and ABA, dominates the decision to germinate[52]. During seed maturation a network of transcription factors including the ABA-regulated TFs ABI3, ABI4, and ABI5 induce genes required for seed desiccation and ABA biosynthesis and repress GA biosynthesis genes. Environmental signals, such as cold and light, that trigger seeds to break dormancy do so by flipping the balance towards GA[52]. This antagonistic relationship between ABA and GA arises at multiple points in their respective pathways (FIG. 3a).

DELLA proteins are members of the plant-specific GRAS (GIBBERELLIN-INSENSITIVE, REPRESSOR of *ga1–3*, SCARECROW) family of transcriptional regulators that lack DNA-binding activity and function by interacting with other TFs[90]. DELLAs inhibit GA responses, and GA signaling inactivates DELLAs in part by triggering their proteasomal degradation[90,91]. DELLAs interact with ABI3 and ABI5, and together these protein complexes stimulate the transcription of *SOMNUS*, a key dormancy promoting factor that activates ABA biosynthesis genes and represses GA biosynthesis genes[92]. Interestingly, the action of the DELLA-ABI5 complex is inhibited by the bHLH TF INDUCER OF CBF EXPRESSION1 (ICE1)[93]. Binding of ICE1 blocks the DNA binding activity of ABI5, and this interaction is stimulated by GA treatment, possibly due to the degradation of DELLAs providing a possible mechanism through which prior exposure to cold temperatures may promote germination.

The control of *ABI5* expression appears to be a major regulatory point for multiple environmental signals during germination. The light signaling component ELONGATED HYPCOTYL 5 (HY5) directly activates *ABI5* transcription in response to light[94]. The

DELLA protein RGL2 further promotes ABA signaling by enhancing the transcription of *ABI5*. GA production during germination initiation could then reduce *ABI5* expression through RGL2 degradation[95]. High salinity inhibits seed germination, and two different transcription factors, AGL21 and RSM1, were reported recently to enhance *ABI5* expression during exposure to NaCl[96,97].

Auxin, ABA and root growth under stress.

Root development is shaped by environmental conditions and this topic has been the subject of multiple excellent reviews (e.g. REF.[98]). Here we focus on the mechanisms by which auxin and ABA control the architecture of the root system in response to water and salinity stresses (FIG. 3b).

While high concentrations of exogenous ABA inhibit root growth, lower (nM range) concentrations stimulate primary root growth[99]. Water distribution in soil is uneven, and plants partly address this situation through hydrotropism, the directional growth of roots towards water. Hydrotropism is impaired in ABA deficient mutants and ABA accumulates in root tissues during water stress suggesting an important role for ABA signaling[100]. For hydrotropism the SnRK2.2 kinase is required specifically in cortical cells of the root elongation zone where it promotes the cell elongation necessary for differential growth[101] (FIG. 3b). Low concentrations of ABA (100 nM) stimulate primary root growth by abating PP2C-mediated inhibition of apoplastic H^+ efflux through the AUTOINHIBITED H^+-ATPase 2 (AHA2)[102]. This mechanism provides a contribution to the hydrotropic response[102]. Two recent studies have implicated brassinosteroid signaling in the hydrotropic response as well, although the mechanism is currently unclear[73,103]. By contrast, in high saline environments, lateral roots enter a prolonged growth arrest which requires endodermal ABA signaling[104]. Roots also exhibit preferential growth away from areas of high-salinity – a phenomenon called halotropism[105]. Salt treatment induces internalization of the auxin transporter PIN-FORMED 2 (PIN2) and when roots encounter a longitudinal salinity gradient, auxin accumulates on the side of the root furthest away from the salt source which then leads to root bending[105,106]. Interestingly, hydrotropism does not appear to act through auxin redistribution suggesting that halotropism is a distinct process[107,108].

ABA also regulates root tissue patterning during water stress. Endodermal ABA signaling stimulates xylem differentiation by inducing the expression of the microRNAs miR165/166, two key regulators of vascular development[109,110]. ABA functions within xylem cells as well, where it activates expression of several *VASCULAR-RELATED NAC DOMAIN* (*VND*) TFs which promote xylem differentiation[111].

Research on two related water-dependent root-branching strategies, hydropatterning and xerobranching, has uncovered requirements for auxin and ABA signaling[98]. Lateral roots initiate from pericycle cells within the primary root and their initiation is timed by an auxin-regulated transcriptional network[112,113]. The position of these initiation events was shown to respond to water availability in a process termed hydropatterning[114]. In hydropatterning, differences in water content across the circumference of primary roots lead to preferential lateral root initiation where water is available. Hydropatterning was correlated with auxin biosynthesis and signaling on the side of the root in contact with water[114]. A

recent study demonstrated that hydropatterning requires the auxin response factor ARF7[115] (FIG. 3b). Removal of seedlings from agar plates and their exposure to air triggered the post-translational modification of ARF7 with a SUMO protein and SUMOylated-ARF7 had reduced DNA-binding activity. Roots can encounter large air spaces in soil, and in these regions lateral root formation is repressed. This repression of branching along the entire root circumference has been termed xerobranching. A recent study implicated ABA signaling in the xerobranching response[116]. The roots of barley plants were found to accumulate ABA following a transient water deficit. Short-term ABA treatment of aeroponically grown maize and barley roots led to a zone of lateral root repression showing that ABA can mimic a xerobranching response. Furthermore, ABA treatment disrupted auxin signaling in roots suggesting a possible mechanism for lateral root repression[116] (FIG. 3b).

Gibberellin, ABA and ethylene regulate flowering during abiotic stress.

A core genetic network regulates flowering time in plants, and this network receives inputs from endogenous, environmental, and seasonal cues[117]. Here we explore how hormone signaling intersects with core flowering regulators to mediate the effects of abiotic stress on flowering time.

During periods of prolonged drought many species will accelerate the flowering transition to reproduce before death and this response is known as drought escape[118]. The exact role of ABA during the flowering transition is currently unclear, and puzzlingly *snrk2.2/2.3/2.6* triple mutants are early flowering[119] while ABA-deficient mutants and *areb1/areb2/abf3/abf1* quadruple mutants are late flowering[60,118]. Here we focus on the case of drought-accelerated flowering, where emerging evidence suggests a positive role for ABA signaling. Under long-day conditions flowering time is delayed in ABA biosynthesis mutants and advanced in an ABA hypersensitive *pp2c* triple mutant[118]. Crucially, drought stress magnifies this delay, suggesting that ABA is required to promote drought escape[118]. This positive role of ABA in drought-induced flowering requires the core photoperiod dependent flowering regulator GIGANTEA (GI)[118,120]. Additionally, the drought escape response is abolished in *abf3/abf4* transcription factor double mutants and ABF3/ABF4 can directly induce the transcription of *SUPPRESSOR OF CONSTANS1* (*SOC1*), another key flowering gene[65] (FIG. 3c).

In contrast to drought, salt stress causes an ethylene dependent delay in flowering time in Arabidopsis[121,122]. Salt stress leads to ethylene accumulation by inducing the expression of ethylene biosynthesis genes[121]. Although the underlying mechanism is unclear, ethylene interferes with GA signaling leading to the accumulation of DELLA proteins. DELLAs can then delay flowering by inhibiting the flowering stimulating TF CONSTANS[123]. In addition, salt stress also represses flowering by inducing the degradation of GIGANTEA[124].

Ethylene and gibberellin control flooding responses.

Floods are a major cause for crop loss in agriculture and a clear environmental challenge for some plants in natural ecosystems[125]. Plants possess an array of developmental and physiological strategies to adapt to flooding with different strategies exhibited in different species. Here we discuss advances related to hormone signaling during submergence and

provide focused coverage of recent work on the hormonal control of a flood-escape strategy in rice. We further direct readers to recent reviews on the broader topic of flooding responses[126,127].

The submergence of plant tissues impedes cellular access to O2 and CO_2 which can severely disrupt metabolism. Additionally, restricted gas diffusion underwater leads to an accumulation of ethylene within flooded plant tissues[125]. Prolonged flooding can cause hypoxia which activates a conserved gene expression program that supports plant survival in limiting O2. In Arabidopsis, the transcription of these hypoxia-responsive genes requires five transcription factors known as group VII ETHYLENE RESPONSE FACTORs (ERF-VIIs)[128]. Additionally, *Submergence1*, a major QTL associated with improved flood tolerance in rice, contains a cluster of three related *ERF* genes[129]. Both hypoxia and high concentrations of ethylene enhance the protein stability of ERF-VIIs leading to target gene transcription[130–133] (FIG. 3d). ERF-VII TFs bind to conserved *cis*-regulatory elements and the chromatin accessibility at these regulatory elements increases in response to flooding[128,134]. Interestingly, accessible ERF-VII binding sites were more prevalent in the flood-adapted rice genome than in the dryland-adapted tomato *Solanum pennellii*[134].

Some flood adapted species display an escape strategy where underwater shoots and leaves elongate to emerge into the air[125]. Research on a flooding-tolerant rice variety known as deepwater-rice has begun to reveal how ethylene and gibberellin signaling control this underwater growth response. GA stimulates stem elongation by promoting internode growth, and this relationship has been exploited during plant domestication. In rice, ethylene accumulates in submerged stem and leaf tissues and this elevated ethylene concentration induces the expression of two *ERFs* known as *SNORKEL1* and *SNORKEL2*, two major QTLs associated with deepwater internode elongation[135]. SNORKEL1 and SNORKEL2 may stimulate stem elongation by inducing GA biosynthesis (FIG. 3d). More recently, an additional locus associated with internode elongation was mapped to *SEMIDWARF1* (*SD1*), a key GA biosynthesis gene[136]. In contrast to the more common semidwarf rice variety which carries a null allele of *SD1*, deepwater-rice plants induce *SD1* expression in submerged tissues. The resulting increased GA levels act together with an additional locus called *ACCELERATOR OF INTERNODE ELONGATION1* (*ACE1*) to promote cell division in the intercalary meristem[137].

Interestingly, a recent study reported that compacted soil leads to ethylene accumulation in roots which subsequently inhibits further growth, possibly allowing plants to avoid regions with poor soil aeration[138]. This suggests that elevated ethylene concentration may be a common and early cue for air deficiency stress that plants use to adapt their growth.

Regulation of stomatal movements

Stomatal pores formed by guard cells in the leaf epidermis allow the uptake of CO_2 for photosynthesis in exchange for water. To optimize plant water-use-efficiency, guard cells sense and respond to several abiotic factors, including light, CO_2, and drought. ABA is a central regulator of guard cell physiology (FIG. 4a), and here we discuss the crosstalk with abiotic factors and other hormones.

Stomatal response to drought.

Drought stress has been reported to trigger ABA synthesis in vascular tissues and guard cells[139,140]. ABA signaling in guard cells regulates plasma membrane ion channels to trigger long-term efflux of anions and K^+, resulting in the reduction of guard cell turgor and stomatal closure. Anion release from guard cells and subsequent plasma membrane depolarization is mediated by slow-type (S-type) and rapid-type (R-type) anion channels[141]. A major S-type anion channel in Arabidopsis guard cells is encoded by *SLOW ANION CHANNEL-ASSOCIATED 1* (*SLAC1*)[142,143]. ALUMINUM-ACTIVATED MALATE TRANSPORTER 12/QUICK-ACTIVATING ANION CHANNEL 1 (ALMT12/QUAC1) contributes to 40 percent of R-type anion currents[144]. The anion channel-triggered depolarization in turn induces K^+ efflux through the voltage-dependent outward-rectifying K^+ (K^+_{out}) channel[145] GUARD CELL OUTWARD RECTIFYING K^+ CHANNEL (GORK)[146]. The protein kinase SnRK2.6/OST1 is a major positive regulator of ABA signaling in guard cells[35]. SnRK2.6/OST1 phosphorylates and activates both, SLAC1[147,148] and ALMT12/QUAC1[149]. Group A PP2Cs, as negative ABA signaling regulators, directly dephosphorylate and inactivate not only SnRK2.6/OST1[150,151] but also SnRK2.6/OST1 substrates, such as SLAC1[152]. Ion transport at the vacuolar membrane is also required for ABA-induced stomatal closure and detailed information has been reviewed[153].

Cytosolic Ca^{2+} fine-tunes ABA-mediated stomatal closure by regulating Ca^{2+}-sensor proteins, such as Ca^{2+}-dependent protein kinases (CPKs), that contribute to the activation of SLAC1[152,154]. ABA can induce elevations in $[Ca^{2+}]_{cyt}$ in guard cells, and one of the underlying mechanisms is plasma membrane Ca^{2+} influx through hyperpolarization-activated Ca^{2+}-permeable cation (I_{Ca}) channels[155,156]. ABA-activation of I_{Ca} channels involves several steps. First, SnRK2.6/OST1 triggers extracellular reactive oxygen species (ROS) production which includes SnRK2 regulation of NADPH oxidases[157,158]. ROS mediates I_{Ca} channel activation in the plasma membrane via the hydrogen peroxide (H_2O_2) sensor kinase HYDROGEN-PEROXIDE-INDUCED Ca^{2+} INCREASES 1 (HPCA1)[159] and the receptor-like (pseudo-)kinase GUARD CELL HYDROGEN PEROXIDE-RESISTANT1 (GHR1)[160]. GHR1 also contributes to SLAC1 activation[161]. In addition to Ca^{2+} and ROS, other small molecules such as hydrogen sulfide[162,163] and gamma-aminobutyric acid[164] have been recently shown to modulate guard cell ABA signaling.

Abiotic signal integration in guard cells.

Guard cells can perceive and integrate several environmental stimuli (FIG. 4b). Amongst them, light and CO_2 are major abiotic stimuli that regulate stomatal aperture. Blue and red light induce stomatal opening mechanisms to maximize photosynthesis. Light-induced stomatal opening is mediated by H^+-ATPase activation and subsequent K^+ uptake through voltage-dependent inward-rectifying (K^+_{in}) channels at the guard cell plasma membrane[145,165,166]. ABA suppresses light-induced stomatal opening via inhibition of H^+-ATPases and K^+_{in} channels. Group D PP2Cs (PP2C.Ds) and their negative regulators, the SMALL AUXIN-UP RNAs (SAURs) also contribute to the regulation of H^+-ATPases in Arabidopsis guard cells[167,168]. To which extent auxin is involved in this mechanism remains to be elucidated. Rapid downregulation of K^+_{in} channels is mediated by SnRK2.6/OST1-dependent phosphorylation of the K^+_{in} channel KAT1[169] and also by $[Ca^{2+}]_{cyt}$ elevation[170].

On a slower time-scale, the expression of K^+_{in} channel-encoding genes, including *KAT1*, is inhibited via SnRK2-dependent inactivation of ABA-RESPONSIVE KINASE SUBSTRATE (AKS) transcription factors[171].

High CO_2 induces stomatal closure, whereas low CO_2 induces stomatal opening. In response to ABA, stomata close and do not easily re-open in the short term. In contrast, high CO_2-mediated stomatal closure is rapidly reversible (e.g. REF.[172]). The mechanisms by which high CO_2 mediates stomatal closure and activates SLAC1 differ from those of ABA[172–174]. In contrast to ABA signaling, elevated CO_2 does not rapidly activate SnRK2-type protein kinases[172,175]. Raf-like kinases, such as CONVERGENCE OF BLUE LIGHT AND CO_2 1 (CBC1), CBC2[165] and HIGH LEAF TEMPERATURE 1 (HT1)[176,177], inhibit S-type anion channel activation via an unknown mechanism. CBCs function at a convergence point between blue light and low CO_2-induced stomatal opening signaling pathways[165].

Signaling crosstalk between ABA and other hormones contributes to guard cell abiotic stress responses. The F-box protein MORE AXILLARY GROWTH 2 (MAX2) is a central regulator of both strigolactone and karrikin signaling[178]. MAX2-dependent signaling induces the upregulation of ABA-signaling genes, such as *SnRK2.6/OST1*, thereby enhancing ABA-induced stomatal closure and drought tolerance[179,180]. The type-B ARRs ARR1, ARR10, and ARR12, acting as positive transcriptional regulators of cytokinin signaling, negatively regulate stomatal closure and drought tolerance[181]. ABA and water deficit suppress cytokinin signaling via downregulation of type-B *ARRs*, as a proposed adaptive mechanism to survive drought[181]. It was reported that excess high light stress triggers local and whole-plant systemic stomatal closure, which is likely mediated by the NADPH oxidase RBOHD in coordination with ABA, salicylic acid, and jasmonate signaling[182]. Darkness and high CO_2 however, do not induce stomatal closure in systemic leaves of Arabidopsis[183]. Under heat stress, plants open stomata to cool down the leaves by transpiration. Jasmonate has been suggested to fine-tune stomatal apertures during a combination of heat and other stresses such as high light and wounding[184,185]. Brassinosteroids can positively mediate stomatal opening[186]. In the brassinosteroid-biosynthesis mutant *dwarf5* (*dwf5*), *KAT1* expression is downregulated via an AKS-independent pathway and the light-driven activation of H^+-ATPases remains intact, suggesting that brassinosteroid regulation of stomatal opening is independent of ABA signaling[186].

Monitoring hormone responses in plants

To understand phytohormone signaling processes during abiotic stress, it is important to determine under which stress conditions, and in which cell-types or tissues and time frames phytohormone responses appear. Furthermore, it is relevant to determine at which level (i.e. biosynthesis, transport, perception, transduction, and transcriptional response) abiotic stresses interfere with a certain hormone signaling pathway. Genetically encoded phytohormone indicators (GEPHIs) are biosensors that allow the in vivo monitoring of cellular hormone responses at high spatiotemporal resolution and at various levels. Their functional principles, advantages over other methodologies and their limitations have

been extensively discussed[3,187,188] and are summarized in BOX 1. Here we review their contribution to abiotic stress response analyses in plants.

GEPHIs that enable the direct detection of phytohormone concentration changes have been initially developed for ABA[28,189], followed by reporters for GA, SL and auxin[190–192]. However, only the FRET-based ABA indicators ABACUS1–2μ and ABAleon have thus far been employed in research related to abiotic stress[28,189]. Analyses in Arabidopsis using ABAleon2.1 under non stress conditions revealed the existence of an ABA gradient in roots and comparably higher basal ABA concentrations in guard cells, in the root-hypocotyl junction, and the root-tip[28,193]. Experiments using ABAleon2.1_Tao3s, that were targeted to either side of the ER membrane, indicated that in tobacco protoplasts, ABA levels might be higher in the ER compared to the cytosol[194]. How ABA gradients are maintained, and to what extent ABA biosynthesis and transport pathways contribute to distinct ABA concentration patterns could be further analyzed using ABA biosensors, similar to research previously conducted on gibberellin gradients in Arabidopsis roots[195]. There is increasing evidence that water deficit in Arabidopsis roots first induces the biosynthesis of ABA in leaves, via long-distance signals, before ABA accumulation is detected in roots[26,27,32]. Consistent with these findings, ABA indicator analyses could not detect rapid osmotic- or salt stress-induced ABA elevations in roots under imposed experimental conditions. Instead, ABA elevations were observed only several hours after exposure to stress[28,189]. Further analyses in Arabidopsis also revealed that sulfate and cysteine trigger ABA level increases in guard cells[196], whereas CO_2 elevation did not cause a rapid ABA concentration increase[172,175]. More detailed analyses are required to determine the spatiotemporal parameters of ABA elevation in response to water deficit and the intercellular transport routes of ABA. In the future it will also be interesting to investigate whether recently developed indicators for auxin[192], GA[190] and SL[191] can detect respective hormone dynamics in response to abiotic stress.

Complementary to direct ABA indicators, the FRET-based SNACS reporter monitors downstream SnRK2-type protein kinase activity[175]. In Arabidopsis guard cells, SNACS responded to ABA, but not to elevated CO_2 concentrations, or treatments with methyl jasmonate. These results were consistent with a lack of ABA accumulation under the same experimental conditions, providing evidence for the hypothesis that basal ABA signaling rather than SnRK2 activation contributes to elevated CO_2 and methyl jasmonate responses in guard cells[172,175].

Early on, promoter fragments of marker genes, or synthetic hormone-responsive promoters, were used to drive reporter gene expression as a readout for the detection of phytohormone signaling patterns[3,187,188] (BOX 1). Although several transcriptional reporters were employed for the analyses of abiotic stress responses, most of the research related to abiotic stress focused on ABA. In this context, ABA signaling reporters were employed for the analyses of drought-, osmotic-, salt-, cold and high CO_2 responses[26,172,193,197], contributing to the hypothesis that in response to water shortage, ABA is largely synthesized in shoots rather than in roots of Arabidopsis[26]. Furthermore, the *proRD29A*-based ABA signaling reporter was employed as a readout in genetic screens[197], contributing to the identification of ABA synthesis genes in Arabidopsis[198]. Also synthetic hormone-responsive promoter

(*SP*) reporters were recently utilized for the reconstitution of ABA signaling in yeast[199] and for the analysis of ABA-mediated transcriptional regulation in Arabidopsis[200]. The latter *6xABRE SPs* reported basal ABA-independent activity in the root quiescent center, and ABA-, salt- and osmotic stress-dependent increases in other root tissues[200], albeit with an apparent relatively low dynamic range compared to the *proRAB18:GFP* reporter[193].

Reporters for other phytohormones also contributed to important observations on the roles of auxin, cytokinin and gibberellin in osmotic stress[201], the contribution of cytokinin signaling to the hydrotropic response[202], and the involvement of gibberellin signaling in the salt stress response[121]. The utilization of hormone reporters in species other than Arabidopsis is beginning to emerge (e.g. Kirschner et al.[203]) and will likely aid in determining differences and similarities in hormone signaling between different taxa.

Conclusions

Due to climate change, abiotic stresses, such as drought, salt, heat, and flooding are becoming increasingly challenging for plants[1,2]. Climate change and abiotic stresses can also intensify plant diseases[204]. Such alarming conditions demand innovative approaches. Recent advances in plant biology are providing crucial new insights into how plants sense and respond to abiotic stresses. While translating such findings into field applications remains challenging[205], the advanced understanding of individual hormone-regulated abiotic stress responses, reviewed here, has the potential to provide key insights for developing more resilient crops through both, engineering and mining of traits from more resistant wild crop relatives (e.g. REF.[125]). The elucidation of mechanisms, genes and pathways that control these traits, can provide road maps for applications and translational research into enhancing or protecting yields in response to abiotic stressors. Many of the advances we discuss in this review were made in the model system *Arabidopsis thaliana*. Therefore, research will be needed to determine whether similar or divergent mechanisms are used in crops. Moreover, it has become clear that specific cell types have specific hormone signaling pathways and outputs, and therefore alteration of cell- or tissue-targeted traits will require investigation of hormone signaling mechanisms in those cell types. New tools, including hormone reporters, protein complex identifications, single cell sequencing and other approaches will enable the dissection of abiotic stress-linked cell-type specific and species-specific signal transduction mechanisms. Genetic approaches, including genomics-accelerated breeding and CRISPR-Cas9 gene editing provide new opportunities to accelerate the development of abiotic stress resilient traits. Furthermore, the genomics revolution combined with automated phenotyping are enhancing our ability to understand or predict which of these genes and mechanisms could be primarily used by resilient wild relatives. This can lead to targeted breeding of improved traits into crops. Moreover, enhancing yields of climate change resilient wild varieties through knowledge-guided *de-novo* domestication of crops[206] provides an important new avenue for incorporating beneficial hormone signaling traits. Continued advances at understanding the interplay of plant hormones in diverse responses to abiotic stress will be important for developing abiotic stress resilient crops.

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Glossary

Abiotic stress
Environmental stresses that are associated to the non-living environment such as weather conditions or the quality of the soil in which plants grow.

Phytohormones
Phytohormones are plant-derived compounds that function as plant growth regulators either locally or over long distances and at low (sub μM) concentrations.

Osmotic stress
Osmotic stress derives from differences in the water potential between plant cells and their environment. Hypoosmotic stress leads to cell swelling, whereas hyperosmotic stress leads to plant wilting.

Mechanosensitive ion channels
Ion channels that respond to mechanical forces, e.g. induced by membrane tension.

CLE25
A small peptide that is induced in the root vasculature during drought stress and moves to aerial tissues to induce *NCED3* gene expression.

SnRK2s
A family of protein kinases, including the ABA-activated SnRK2.2/2.3/2.6, that are activated in response to hyperosmotic stress.

B2, B3 and B4 subgroup Raf-like M3Ks
Members of these subgroups of Raf-like M3Ks are involved in the osmotic stress- and ABA-dependent activation of SnRK2-type protein kinases.

Seed dormancy
Inhibition of seed germination during unfavorable environmental conditions requires the downregulation of gibberellic acid signal transduction.

Hydrotropism
The directional growth of roots towards regions of the soil environment with higher water content.

Halotropism
The directional growth of roots away from regions of high salinity.

Xerobranching

A water-responsive root developmental program, where the formation of lateral roots is repressed in regions of the soil environment that lacks water.

Hydropatterning
A water-responsive root developmental program active when water is asymmetrically available around the circumference of the root. Lateral roots preferentially form on the water contacting side.

Drought escape
An adaptive response to prolonged drought stress where plants accelerate the transition to flowering in order to reproduce.

Deepwater rice
Specific varieties of rice (*Oryza sativa)* can survive periods of prolonged flooding by activating a developmental program, that depends on ethylene and gibberellin, to promote stem elongation into the air.

Stomata
Small pores in the leaf epidermis that are formed by guard cells to allow the uptake of CO_2 for photosynthesis in exchange for water loss.

Depolarization and hyperpolarization
Changes in the cell membrane potential, making it more positive or more negative, respectively.

GEPHI
Genetically encoded phytohormone indicators that allow the *in vivo* monitoring of hormone levels and downstream hormone signaling responses.

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Box 1 |

Genetically encoded phytohormone indicators.

Genetically encoded phytohormone indicators (GEPHIs) enable the *in vivo* analysis of hormone concentration changes and subsequent downstream signaling processes at tissue- and cellular resolution. Several GEPHIs have been developed and employed in plants, and more comprehensive information can be found elsewhere[3,187,188].

FRET-based GEPHIs.

To directly monitor hormone concentration changes, Förster Resonance Energy Transfer (FRET)-based indicators have been developed for ABA (ABACUS and ABAleon)[28,189,224], auxin (AuxSen)[192] and gibberellins (GPS1)[190]. These indicators consist of a sensory domain that changes its structure in a hormone-bound configuration, thereby affecting the distance, orientation, and the fluorescence emission ratio of a fluorescent protein (FP) FRET pair upon excitation of the FRET donor FP. ABAleon-type ABA indicators have recently received two updates. Dual-reporting indicators, consisting of ABAleonSD1–3L21 fused via the self-cleaving P2A peptide linker to the red-fluorescing Ca^{2+} indicator R-GECO1 or the pH indicator PA-17, allow the simultaneous monitoring of ABA together with Ca^{2+} or pH[224]. Whereas, ABAleon2.1_Tao3s, that harbor a nanobody recognition domain and a secretion signal, can be recruited by a subcellular targeted nanobody to either side of the endoplasmic reticulum (ER) membrane[194]. For the analysis of signaling processes downstream of ABA perception, the FRET-based SnRK2 Activity Sensor (SNACS) has been developed, providing an approach to investigate the activation of SnRK2-type protein kinases in response to abiotic stresses and SnRK2 interaction with other hormone signaling pathways[175].

Degradation-based hormone reporters.

Several plant hormones induce downstream responses by regulating the ubiquitination and proteasomal degradation of transcriptional repressors[3]. Degradation-based GEPHIs, monitor their protein levels using FP fusions. More sophisticated reporters employ only a hormone-dependent degradation domain (degron-motif) fused to a FP to report an increased hormone concentration and signaling strength via a decrease in FP stability. For achieving a ratiometric readout, reporters for auxin, such as R2D2[225] and qDII[226] co-express a non-degradable FP as a reference.

Synthetic Hormone-activated Cas9-based Repressors (HACRs).

HACRs consist of a deactivated dCas9 fused to a hormone-dependent degradation domain and a fragment of the repressor TOPLESS. The dCas9 component associates with a guide RNA (gRNA) and recruits the HACR to a target promoter. Hormone-dependent degradation of the HACR then leads to a de-repression of the target (reporter) gene[227].

A reporter for ethylene-dependent translational regulation.

The ethylene signaling pathway involves the EIN2 C-terminal domain (CEND) association with 3’ UTRs of EIN3-binding F-box protein (EBF) mRNAs to repress their translation[228,229]. Based on this mechanism, a translational reporter has been

designed consisting of a *FP* coding sequence followed by three tandems of Ethylene Responsive RNA elements containing Poly-Uridylates (*EPUs*). Upon induction of *EIN2*, the translation of this 6x *EPU* reporter is inhibited, and transgenic plants expressing this construct are insensitive towards ethylene[228].

Hormone-activated transcriptional reporters.

Several marker genes for plant hormone signaling have been identified and their promoters have been employed to drive the expression of reporter genes. In addition, the identification of *cis*-elements that are targeted by hormone-specific transcriptional regulators, lead to the development of synthetic promoters (*SPs*). *SPs* contain multiple repeats of *cis*-element-containing promoter fragments upstream of a minimal *35S* promoter to drive reporter gene expression. They have been developed for almost any plant hormone[3,187,188].

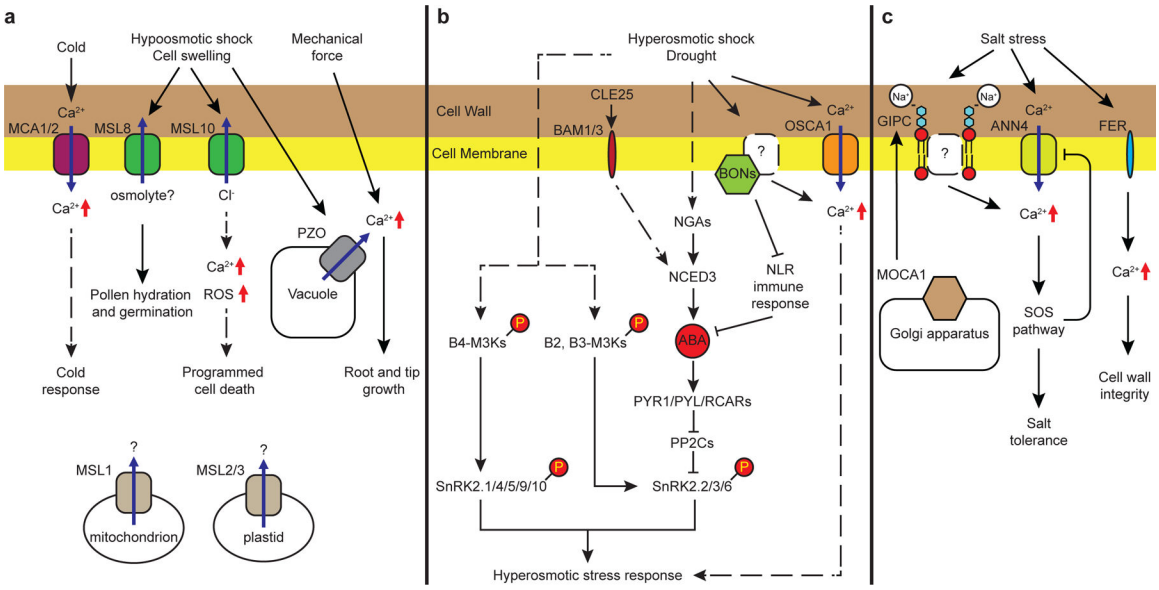

Figure 1 |. Osmotic stress and salinity sensing and signaling in plants.

a | Mechanosensitive channels have been proposed to be involved in sensing the alterations of membrane tension caused by hypoosmotic stress and other abiotic stresses. MSL8 prevents bursting of pollen during hydration and germination[8]. MSL10 potentiates hypoosmotic/cell swelling-induced $[Ca^{2+}]_{cyt}$ transient increases, ROS production, and programmed cell death[10]. MSL1 and MSL2/3 control mitochondrial and plastidial osmotic pressure[223]. MCA1 and MCA2 function in hypoosmotic and cold-induced $[Ca^{2+}]_{cyt}$ transients and regulate cold acclimation responses[12]. Tonoplast-localized PIEZOs (PZO) are required for $[Ca^{2+}]_{cyt}$ oscillations in tip-growing cells[14], and mechanical-induced $[Ca^{2+}]_{cyt}$ increases in the root tip to regulate root penetration into denser barriers[13].

b | Hyperosmotic stress-induced $[Ca^{2+}]_{cyt}$ increases have been reported to function in early hyperosmotic-stress signaling. OSCA1 is an osmotic/mechanical-sensitive channel required for hyperosmotic-induced $[Ca^{2+}]_{cyt}$ increases[15]. Ca^{2+}-responsive phospholipid-binding BONZAI (BON) proteins regulate hyperosmotic-induced $[Ca^{2+}]_{cyt}$ increases and suppress NLR immune signaling to trigger a hyperosmotic stress response[18]. Drought induces ABA biosynthesis *NCED3* gene expression, leading to ABA accumulation. Root-derived CLE25 peptides activate *NCED3* gene expression in the shoot in response to dehydration likely through receptor-like kinases BAM1 and BAM3[32]. NGATHA (NGA) transcription factors are responsible for the drought-induced transcriptional activation of *NCED3*[30]. Hyperosmotic stress activates Raf-like M3Ks via phosphorylation through an unknown osmotic stress sensor-mediated signal transduction mechanism. Members of the B2 and B3 subgroups of Raf-like M3Ks mediate both the rapid osmotic stress-induced and slower, post-ABA synthesis, activation of SnRK2.2/2.3/2.6, whereas the B4 subgroup of Raf-like M3Ks only activate osmotic stress-responsive SnRK2.1/2.4/2.5/2.9/2.10[20,42–44].

c | A Salt-induced $[Ca^{2+}]_{cyt}$ increase triggers tolerance responses through the salt overly sensitive (SOS) pathway. Glycosyl inositol phosphorylceramide sphingolipids (GIPCs) synthesized by Inositol Phosphorylceramide Glucuronosyltransferase (MOCA1/IPUT1) are involved in Na^+ sensing[24]. The Annexin 4 (ANN4)-medicated $[Ca^{2+}]_{cyt}$ increase is feedback

inhibited by the SOS pathway for fine-tuning salt tolerance[25]. FERONIA (FER) is required for maintaining cell wall integrity under salt stress[22].

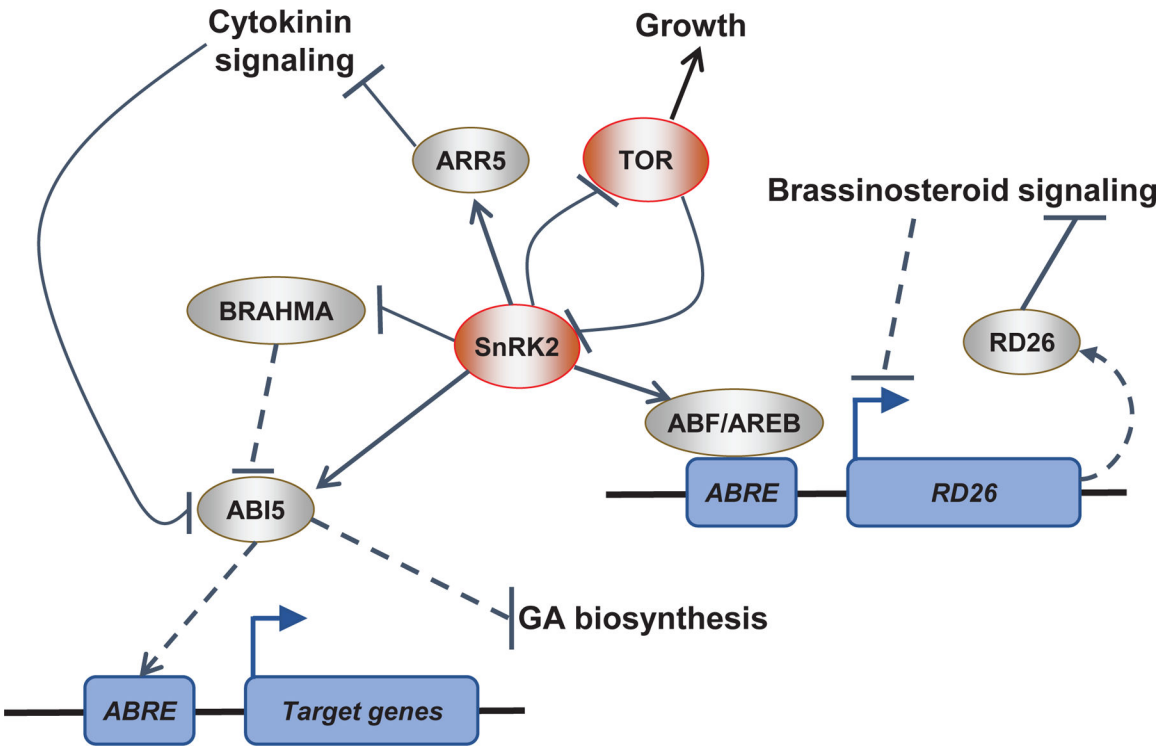

Figure 2 |. Hormonal crosstalk through transcriptional regulation.

Plant hormones control abiotic stress response by altering transcriptional programs. The ABA signaling system intersects with many other hormone pathways during transcription. This figure summarizes the relationships between ABA signaling and key transcriptional regulators during hormonal signaling. During ABA-responses, SUCROSE NONFERMENTING 1-RELATED PROTEIN KINASE 2 (SnRK2) protein kinases phosphorylate *ABA-RESPONSIVE ELEMENT* (*ABRE*)-binding proteins/ABRE-binding factors (AREBs/ABFs) and ABA-INSENSITIVE 5 (ABI5) transcription factors. AREBs/ABFs and ABI5 activate target genes with *ABREs* in their promotors to drive ABA responses. For instance, during drought stress AREBs/ABFs activate transcription of *RESPONSE TO DESSICATION 26* (*RD26*), a transcription factor that can repress brassinosteroid signaling. Additionally, in dormant seeds, ABI5 target genes repress gibberellic acid (GA) biosynthesis and thereby block germination. Cytokinin signaling can repress ABA responses possibly by triggering the degradation of ABI5. ABA-activated SnRK2-type protein kinases promote transcriptional ABA responses by phosphorylating type-A ARABIDOPSIS RESPONSE REGULATOR5 (ARR5), a negative regulator of the cytokinin pathway. Finally, in unstressed conditions the protein kinase TARGET OF RAPAMYCIN (TOR) promotes plant growth by inhibiting SnRK2-type protein kinase-mediated ABA responses through phosphorylation of ABA receptors. Conversely, during stress SnRK2-type protein kinases phosphorylate and inhibit the TOR regulatory protein RAPTOR1B leading to growth repression. Note that not all mechanisms shown here are necessarily present at the same time or in the same cell/tissue.

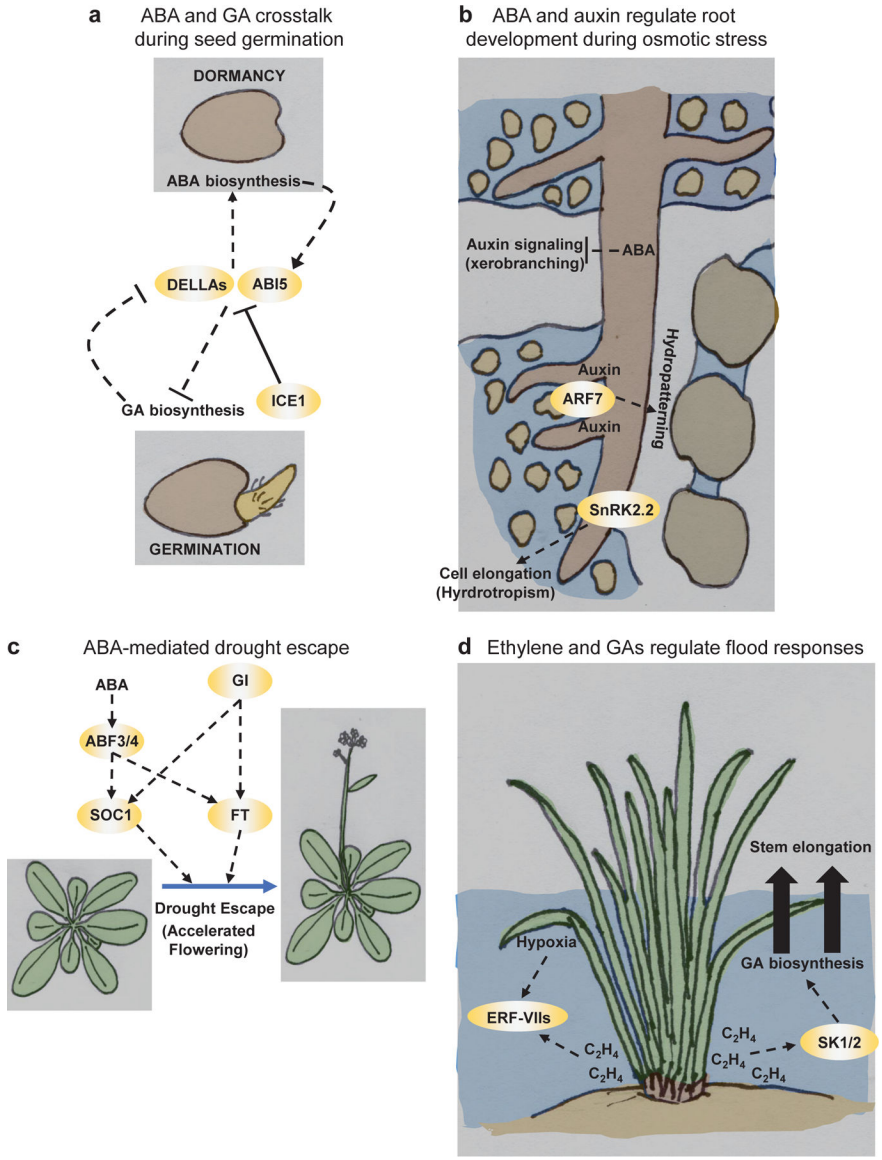

Figure 3 |. Hormonal control of growth and development during abiotic stress.

a | ABA and GA signaling pathways antagonistically control germination. In dormant seeds, DELLA proteins and ABI5 promote ABA signaling by stimulating expression of ABA biosynthesis genes and the *ABI5* gene and inhibit GA responses by repressing GA biosynthesis. INDUCER OF CBF EXPRESSION1 (ICE1) antagonizes DELLA and ABI5 activity to promote germination. During germination, GA levels increase, and GA triggers the destruction of DELLA proteins leading to decreased ABA signaling. **b** | Water is unevenly distributed in soil and large air pockets form between soil particles. Primary roots display hydrotropism or biased growth towards areas of higher water. This process depends on SnRK2.2 protein kinase activity in cortex cells of the elongation zone. When roots enter air spaces lateral root formation is repressed (xerobranching), a process that depends on the ABA inhibition of auxin signaling. Roots growing in areas where water is asymmetrically distributed display a growth program known as hydropatterning,

where lateral roots preferentially form on the water contacting side. In hydropatterning, the auxin response factor ARF7 stimulates preferential lateral root initiation. **c** | During prolonged drought, plants will accelerate flowering to reproduce in a process called drought escape. Under drought stress, the ABA-activated transcription factors ABF3/4 and the floral regulator GIGANTEA (GI) stimulate expression of the flowering inducers *SUPRESSOR OF OVEREXPRESSION OF CO1* (*SOC1*) and *FLOWERING LOCUS T* (*FT*) to advance flowering. **d** | Submerged plant tissues experience hypoxia and elevated ethylene gas (C_2H_4). These cues activate transcription factors known as group VII ETHYLENE RESPONSE FACTORs (ERF-VIIs). ERF-VIIs initiate a conserved hypoxia-induced transcriptional program. In deepwater rice varieties, elevated ethylene activates the ERFs SNORKEL1 and 2 (SK1/2) which induce GA biosynthesis. GA signaling promotes a flood escape strategy where stems elongate to emerge into the air.

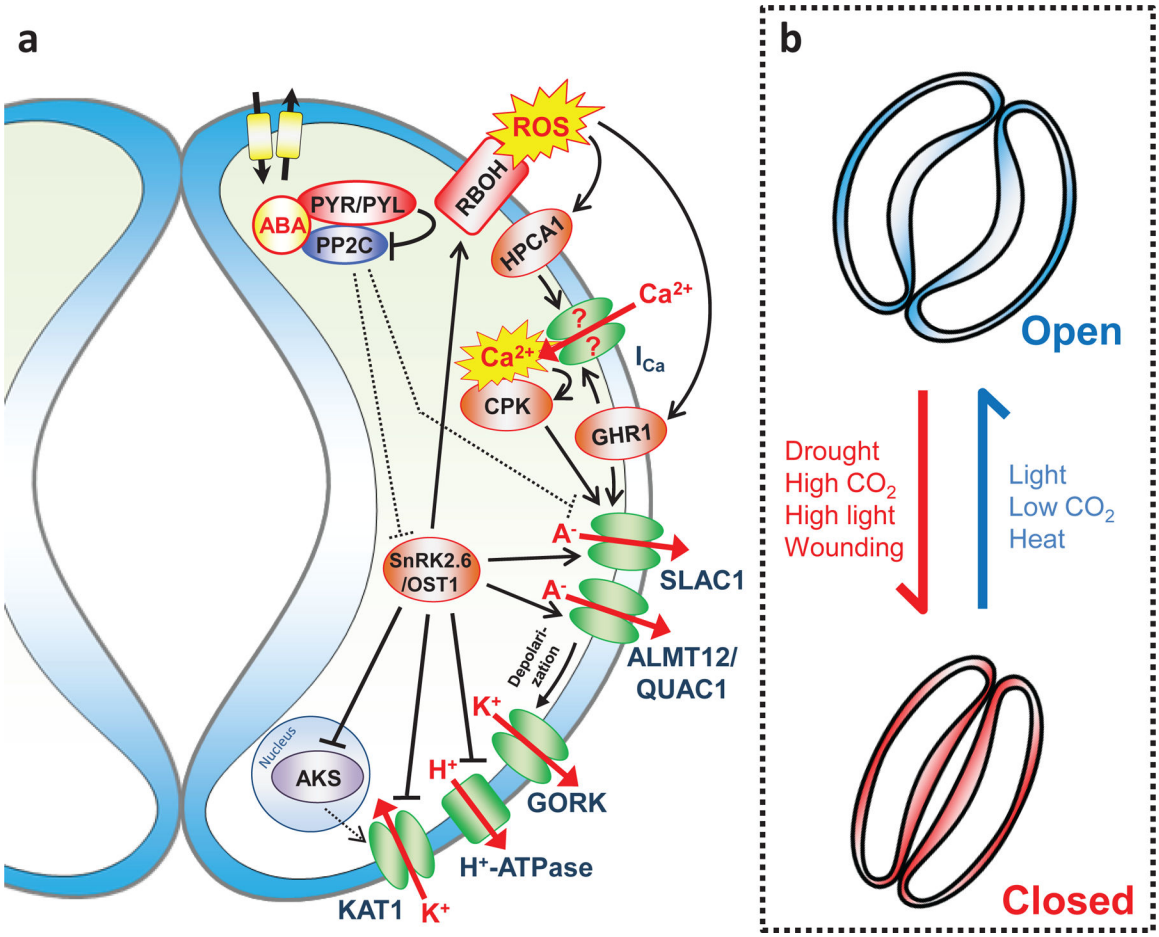

Figure 4 |. Guard cell signal transduction and stomatal responses to environmental stimuli.

a | Schematic model of abscisic acid (ABA) signal transduction in guard cells. ABA transporters mediate ABA import or export from guard cells. In the presence of ABA, the key regulator SNF1-RELATED PROTEIN KINASE 2.6/OPEN STOMATA 1 (SnRK2.6/OST1) phosphorylates and activates SLOW ANION CHANNEL-ASSOCIATED 1 (SLAC1), ALUMINUM-ACTIVATED MALATE TRANSPORTER 12/ QUICK-ACTIVATING ANION CHANNEL 1 (ALMT12/QUAC1), and RESPIRATORY BURST OXIDASE HOMOLOGs (RBOHs). Activation of the S-type anion channel SLAC1 and the R-type anion channel ALMT12/QUAC1 leads to long-term plasma membrane depolarization, which causes K^+ efflux through the voltage-dependent K^+_{out} channel GUARD CELL OUTWARD RECTIFYING K^+ CHANNEL (GORK). Activated RBOH NADPH oxidases produce ROS that mediate HYDROGEN-PEROXIDE-INDUCED Ca^{2+} INCREASES 1 (HPCA1) sensor-dependent activation of Ca^{2+}-permeable I_{Ca} channels, resulting in the elevation of the cytosolic Ca^{2+} concentration ($[Ca^{2+}]_{cyt}$). Elevated $[Ca^{2+}]_{cyt}$ activates Ca^{2+}-sensor proteins including Ca^{2+}-DEPENDENT PROTEIN KINASEs (CPKs) that phosphorylate and activate SLAC1. The (pseudo-)kinase GUARD CELL HYDROGEN PEROXIDE-RESISTANT 1 (GHR1) mediates the activation of I_{Ca} and SLAC1 channels through an unknown mechanism, possibly as scaffolding protein. SnRK2.6/OST1 inhibits the K^+_{in} channel K^+ CHANNEL IN ARABIDOPSIS THALIANA 1 (KAT1), that mediates

K^+ uptake, by direct phosphorylation. In addition, SnRK2.6/OST1 causes a long-term decrease of *KAT1* expression by inhibition of ABA-RESPONSIVE KINASE SUBSTRATE (AKS) transcription factors. ABA also inhibits H^+-ATPase activity through SnRK2.6/OST1, but the detailed mechanism is unknown. Dashed lines indicate steps that are inhibited in the presence of ABA. Note that only guard cell ABA signaling regulating ion transport across the plasma membrane is depicted in this figure. **b** | In addition to drought stress, several other environmental stimuli can be perceived by guard cells and affect stomatal aperture though sophisticated signaling crosstalk and integration.

Table 1 |

Examples of roles of phytohormones in abiotic stress responses.

Phytohormone	Ref.
Abscisic acid (ABA)	
Drought stress in roots induces ABA biosynthesis in shoots via hydraulic signals, and CLE25 peptide-mediated induction of *NCED3* expression.	26,27,32
Osmotic- and salt stress induce the activation of SnRK2-type protein kinases, and SnRK2 activation is mediated by Subgroup B Raf-like kinases.	34,36,39,42,44
Root hydrotropism requires ABA signaling in the cortex of the elongation zone.	101
Salt stress inhibits lateral root formation, which depends on ABA synthesis and endodermal ABA signaling. ABA inhibits lateral root formation through interfering with auxin signaling.	104,116
Salt stress, K^+ and SO_4^{2-} deficiency induce endodermal suberization in an ABA-dependent manner.	207
Cold stress responses are modulated via SnRK2.6/OST1 phosphorylation of the transcription factor ICE1.	208
Heat stress tolerance is reduced in mutants deficient in ABA signaling or biosynthesis.	209
Auxin (IAA)	
Salt stress induces halotropism, the preferential growth away from areas of high salinity, which is mediated by auxin redistribution to induce root bending.	105,106
Hydropatterning, the preferential formation of lateral roots near water, is initiated by auxin signaling, and depends on the auxin response factor ARF7.	114,115
Drought stress induces the expression of *IAA5* and *IAA19*, two transcriptional repressors of auxin responses. Additionally, *iaa* mutants have reduced survival during osmotic stress.	76
Heat stress induces auxin biosynthesis via PIF4, and the stabilization of auxin co-receptors. Auxin signaling via ARFs mediates high temperature-dependent hypocotyl elongation.	210–212
Brassinosteroid (BR)	
Drought stress responses interfere with BR signaling via BR and ABA crosstalk at the level of BES1 and RD26 mediated transcriptional regulation.	71
Cold acclimation and freezing tolerance involve BR signaling through its effect on *COR* and *CBF* gene expression.	213,214
During **Thermomorphogenesis**, PIF4 induces BR biosynthesis, whereas the BR activated transcription factor BZR1 functions in a feedforward loop downstream of auxin and PIF4 to further induce *PIF4* expression.	215,216
Cytokinin (CK)	
Drought- and salt-stress induce the reduction of CK content and signaling, leading to an increased ABA sensitivity, likely via interaction of SnRK2s with type-A and type-B ARRs.	74,181,217
The **osmotic stress-dependent hydrotropic response** depends on the asymmetric distribution of CK signaling in the root tip, which is enhanced at the lower water potential side.	202
Ethylene (ET)	
Salt stress induces the production of ET and ET signaling. ET signaling promotes salt tolerance.	121
Flooding or submergence adaptation depends on ET production and the function of group VII Ethylene Response Factors (ERFs) in Arabidopsis and other related ERF genes in rice (*Sub1A* and *SNORKEL*), likely inducing GA biosynthesis.	128,129,135
Metal deficiency reduces endodermal suberization in an ET-dependent manner.	207
Gibberellic acid (GA)	
Under **drought stress** conditions GA signaling interferes with ABA signaling via DELLA protein interactions with the ABA-regulated TF ABF2.	218
Salt stress reduces the levels of bioactive GAs, likely via ABA signaling. *della*-quadruple mutants are hypersensitive to salt stress.	121
Cold stress responses are mediated via DELLA accumulation and interactions with GRF-type TFs.	219

Phytohormone	**Ref.**
Heat stress induces GA biosynthesis and the degradation of DELLAs in a COP1-dependent manner to regulate hypocotyl elongation.	220
Water submergence triggers GA production to induce internode elongation in rice.	135–137
Jasmonic acid (JA)	
Cold stress induces the production of JA. JAZ degradation in response to cold releases ICE1 and ICE2 from JAZ-mediated repression.	221
Heat stress promotes the accumulation of the JA receptor COI1 to enhance downstream JA responses.	185
Strigolactone (SL)	
Drought- and salt stress responses are positively modulated by SL via ABA-dependent and ABA-independent pathways.	179,222

Abbreviations:
CLE25, CLAVATA3/ENHANCER OF SHOOT REGENERATION-RELATED 25
NCED3, NINE-CIS-EPOXYCAROTENOID DIOXIGENASE 3
SnRK2, SUCROSE NONFERMENTING 1-RELATED PROTEIN KINASE
ARF, AUXIN RESPONSE FACTOR
ICE1, INDUCER OF CBF EXPRESSION 1
IAA5 and IAA9, INDOLE-3-ACETIC ACID INDUCIBLE 5 and 9
PIF4, PHYTOCHROME-INTERACTING FACTOR4
BRL3, BRI1-LIKE 3
BES1, BRI1-EMS-SUPPRESSOR 1
RD26, RESPONSIVE TO DESSICATION 26
COR, COLD REGULATED
CBF, C-REPEAT BINDING FACTOR
ARR, ARABIDOPSIS RESPONSE REGULATOR
ERF, ETHYLENE RESPONSE FACTOR
DELLA, plant-specific GRAS family proteins functioning as repressors of the GA signaling pathway
ABF, *ABRE*-BINDING FACTOR
GRF, GROWTH REGULATORY FACTOR
COP1, CONSTITUTIVE PHOTOMORPHOGENETIC 1
JAZ, JASMONATE-ZIM-DOMAIN PROTEIN
COI1, CORONATINE-INSENSITIVE 1