Diverse roles of jasmonates and ethylene in abiotic stress tolerance

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Jasmonates (JAs) and ethylene (ET), often acting cooperatively, play essential roles in regulating plant defense against pests and pathogens. Recent research reviewed here has revealed mechanistic new insights into the mode of action of these hormones in plant abiotic stress tolerance. During cold stress, JAs and ET differentially regulate the C-repeat binding factor (CBF) pathway. Major JA and ET signaling hubs such as JAZ proteins, CTR1, MYC2, components of the mediator complex, EIN2, EIN3, and several members of the AP2/ERF transcription factor gene family all have complex regulatory roles during abiotic stress adaptation. Better understanding the roles of these phytohormones in plant abiotic stress tolerance will contribute to the development of crop plants tolerant to a wide range of stressful environments.

Facing the reality: abiotic stress tolerance in a changing world

Abiotic stress factors such as salinity, heat, cold, drought, and flooding cause widespread crop losses throughout the world. Preventing such losses is particularly important in the face of a rapidly expanding world population that exerts an immense pressure on human populations to produce more food and feed. Although incremental yield increases on existing agricultural land through intensification are possible, especially in developing countries, it is clear that the rapidly-increasing demand for food and feed production will eventually require a significant expansion of the land under cultivation. However, any dramatic increase in global arable land through extensification must come either at the expense of the precious forests of the world or by expanding crop production into marginal areas [1,2] where stress factors can be more prevalent. The timing and the relative strength of abiotic stresses are expected to be less predictable and more extreme, respectively, in the future [3]. Moreover, the proportion of agricultural land affected from multiple stresses (e.g., heat and drought) is expected to rise significantly under climate change [4]. As argued in a recent editorial in the journal *Science*, an urgent and coordinated action will be necessary to better manage available natural resources such as water to mitigate the negative effects of climate change on agriculture [5]. Therefore, enhancing intrinsic resilience of plants to a wide range of stress factors is of ever-increasing importance to secure our future food supply. Luckily, plants display an amazing diversity and are able to adapt to a wide range of environments. Clearly, if intrinsic factors underpinning plant adaptation to their environment are better understood, then this knowledge can be harnessed to improve tolerance to abiotic stresses [6].

Plant hormones and stress tolerance

Plant hormones play important roles in regulating responses to a wide variety of internal and external stimuli. Traditionally, salicylic acid (SA), JAs, and ET are associated with plant defense, whereas gibberellins (GAs), auxins (IAAs), brassinosteroids (BRs), and cytokinins are associated with plant development. Abscisic acid (ABA) is the principal hormone that regulates plant responses to abiotic stresses. However, it is becoming increasingly evident that all plant hormones can have direct and/or indirect effects on multiple plant functions. For instance, SA, JA, and ET are also involved in plant development and abiotic stress tolerance, whereas IAAs and GAs play roles in biotic and abiotic stress tolerance [7–9]. This review will briefly discuss recent studies that have revealed mechanistic insights into the roles of JAs and ET, which often collaborate during defense against necrotrophic pathogens [10], in abiotic stress tolerance. The role of SA in abiotic stress tolerance has recently been reviewed [11] and will not be covered here. Throughout the review, the term 'tolerance' is used to refer to the relative ability of a plant genotype (e.g., a mutant) to withstand a stress factor better than wild type.

Cold and freezing stress

Low temperature and freezing stress adversely affect plant growth and development in many parts of the world. Plants grown in temperate regions have the ability to develop tolerance to low temperatures after a brief exposure to cold, and this process is known as 'cold acclimation'. In recent years, significant progress has been made towards better understanding the molecular basis of cold and freezing tolerance in plants [12]. Briefly, INDUCER OF CBF EXPRESSION 1 (ICE1) and ICE1-like basic

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helix-loop-helix (bHLH) transcription factors (TFs) c(e.g., ICE2) positively regulate cold tolerance by activating *C-REPEAT BINDING FACTOR*s (*CBF*s), which encode AP2/ERF family TFs. CBFs, by binding to the C-repeat (CRT)/dehydration-responsive element, activate *COLD REGULATED* (*COR*) genes with roles in stress protection [12].

Roles of JAs in cold and freezing tolerance

Recent studies have revealed unexpected roles for JAs as positive regulators of cold and freezing tolerance (Figures 1 and 2). Firstly, exposure to cold rapidly elevates endogenous JA levels by inducing JA biosynthesis genes such as *LOX1*, *AOS1*, *AOC1*, and *JAR1* in *Arabidopsis* (*Arabidopsis thaliana*) [13], and *OsAOS*, *OsOPR1*, *OsAOC*, and *OsLOX2* in rice (*Oryza sativa*) [14]. Furthermore, exogenous JA treatment enhances freezing tolerance in *Arabidopsis*. Finally, *Arabidopsis* mutants (i.e., *lox2*, *aos*, *jar1*, and *coi1*) deficient in JA biosynthesis or signaling display increased sensitivity to freezing stress relative to wild type plants [13].

Recently, a role for *Arabidopsis* JAZ repressors (Box 1) as regulators of cold stress tolerance has also been shown [13]. Under normal growth conditions, JAZ1 and JAZ4, physically interact and suppress the transcriptional activities of ICE1 and ICE2 (Box 1). This prevents non-specific activation of cold stress responses. Under cold stress, increased JA levels trigger COI1-mediated degradation of JAZs, and this releases ICEs from repression. ICE1 and ICE2 then activate *CBF*s by binding to the DRE/CRT box promoter sequence element found in the ICE-regulon promoters [13] (Figure 2).

bHLH TFs acting downstream from JAZs are also involved in the regulation of cold responses. In banana (*Musa accuminata*), MaMYC2a and MaMYC2b, homologs of the

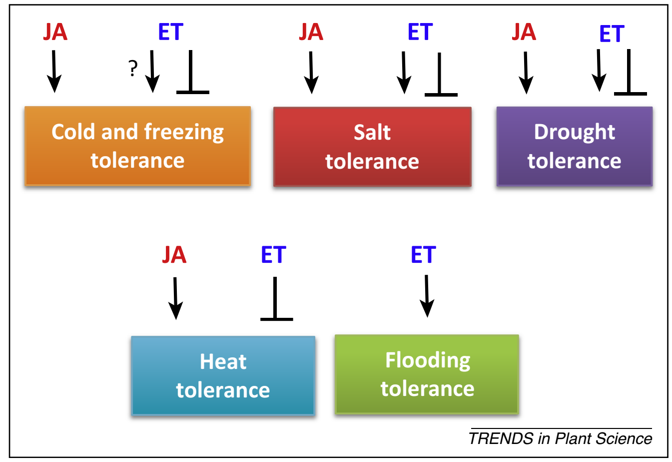

Figure 1. Comparable and contrasting roles of JAs and ET in the regulation of plant responses to major abiotic stress factors. JAs and ET and their signaling pathways can differentially regulate stress tolerance in a species-specific manner. Positive and negative regulatory actions are indicated by arrows and lines with bars, respectively, whereas '?' indicates regulatory events that require further investigation. See text for additional details. Abbreviations: ET, ethylene; JA, jasmonate.

Arabidopsis MYC2 TF (Box 1), physically interact with MaICE1, the banana homolog of the *Arabidopsis* ICE1 protein. A similar interaction occurs between MYC2 and ICE1 in *Arabidopsis* [15,16], potentially providing another point of interaction at the level of master regulators of these two pathways.

The *Arabidopsis* SENSITIVE TO FREEZING 6 (SFR6) protein regulates both the cold and the JA pathway [17,18]. Before its identification as the MEDIATOR16 (MED16) subunit of the plant mediator complex and a regulator of JA-responsive defense gene expression [19,20], SFR6 was known to act downstream from CBFs during the regulation of cold stress [17,18,21]. More recent evidence

Box 1. Plant hormone signaling pathways

JA biosynthesis and signaling

JA biosynthesis and signaling have been extensively reviewed [113–117]. Briefly, as shown in Figure 2 in main text, JAs are synthesized by sequential action of several plastid, peroxisome, and cytoplasmic enzymes. The bioactive jasmonyl-isoleucine (Ja-Ile) is sensed by CORONATINE INSENSITIVE1–JASMONATE ZIM DOMAIN (COI1–JAZ) receptor complexes, and this leads to the degradation of JAZ proteins acting as repressors of transcription factors such as the master regulator MYC2 [115]. This enables MYC2 and related proteins to regulate JA responses by binding to the G-box promoter element found in the promoter of JA-responsive genes. More recently, other bHLH proteins that act as repressors rather than activators of JA responses have also been identified [118–120]. Presumably, the coordinated action of activators and repressors delivers a controlled output that prevents collateral effects. PFT1/MED25, encoding a subunit of the plant mediator complex, also plays an important role in the regulation of JA responses by interacting with several JA-responsive TFs [23,24].

ET biosynthesis and signaling

The gaseous hormone ET is an essential regulator of plant stress responses and development [121]. In *Arabidopsis*, ET is perceived by a family of five receptors, namely ET RESPONSE1 (ETR1), ETR2, ETHYLENE RESPONSE SENSOR1 (ERS1), ERS2 and ETHYLENE INSENSITIVE4 (EIN4). In the absence of ET, receptors negatively regulate the signaling pathway by activating CONSTITUTIVE TRIPLE RESPONSE1 (CTR1) and F-Box proteins EIN3-BINDING F BOX PROTEIN 1 (EBF1) and EBF2 involved in the proteasome-mediated degradation of key signaling components ETHYLENE INSENSITIVE2 (EIN2), EIN3 and EIN3-LIKE1 (EIL1). EIN2 is a membrane protein, whereas EIN3 and EIL1 are TFs that regulate other TFs belonging to WRKY, AP2/ERF, and NAC TF gene families. ET binding to receptors initiates signaling events that prevents the proteasome-mediated degradation of EIN2, EIN3, and EIL1 by the action of the Raf-like MAPKKK (MITOGEN ACTIVATED PROTEIN KINASE KINASE KINASE). The *Arabidopsis* ETHYLENE OVERPRODUCER1 (ETO1) gene encoding a putative E3 ubiquitin ligase and its paralogous ETO1-LIKE 1 (EOL1) and EOL2 modulate ET biosynthesis [122] by interacting with the ET biosynthetic enzyme ACS5 and inhibiting its activity by proteasome-dependent degradation [123]. As a result, the *eto1* mutant shows increased ET accumulation and has been conveniently used to test the effect of ET on various plant processes including stress tolerance (text for details).

ABA biosynthesis and signaling

ABA is considered to be the primary plant hormone regulating abiotic stress responses. Plant responses to abiotic stress are also regulated by ABA-independent pathways with extensive crosstalk taking place between ABA-dependent and ABA-independent pathways. Plant genes responding to ABA contain the ABA RESPONSE ELEMENT (ABRE) in their promoters. ABRE BINDING FACTORS (AREB/ABF) are bZIP TFs that bind to ABREs and regulate osmotic stress tolerance in an ABA-dependent manner. Abiotic stress tolerance can also be regulated in an ABA-independent manner by the AP2/ERF TFs DEHYDRATION RESPONSIVE ELEMENT BINDING PROTEINs (DREB1) and DREB2. DREB1/CBF and DREB2 regulate cold and heat and osmotic stress responses, respectively, by binding to the DRE/CRT sequence element found in stress-responsive gene promoters [124].