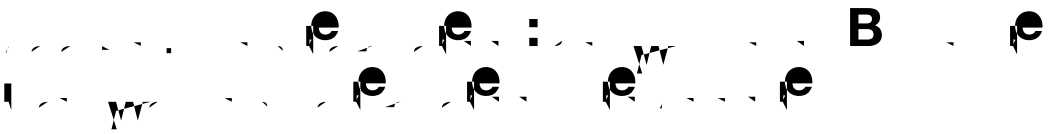
Developmental Cell

Review



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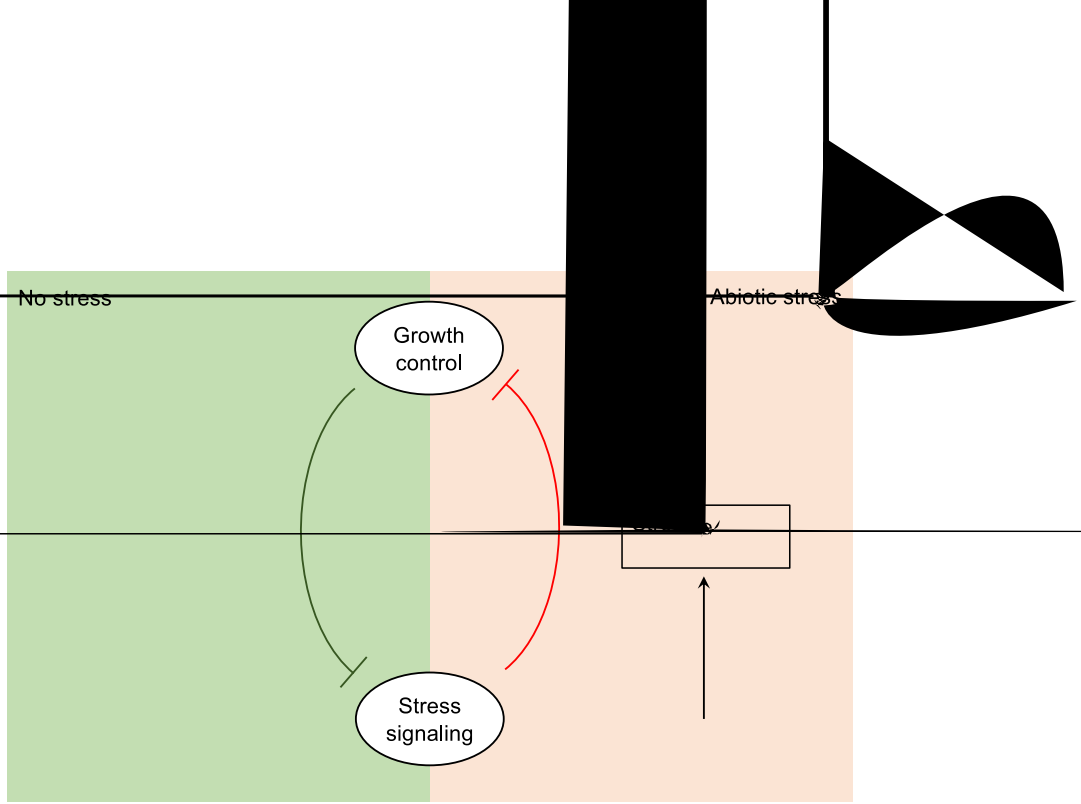
SUMMARY

Defense against stress and active suppression of growth are two complementary strategies by which plants respond to adverse environments. Although beneficial for plant survival, active growth inhibition is often undesirable for crop productivity. Compared with the knowledge on how plants defend against stress-caused cellular impairment, much less is known about how stress signaling regulates plant growth and vice versa. Here, we review recent progress in this area and discuss recent studies suggesting that reciprocal regulation between stress-response and growth-control pathways occurs at multiple levels. Understanding this regulatory network will be critical for resetting the balance between stress resistance and growth in order to engineer stress-resistant and high-yielding crops.

INTRODUCTION

Plants are considered to be under stress when environmental conditions are not ideal for growth. How adverse environments



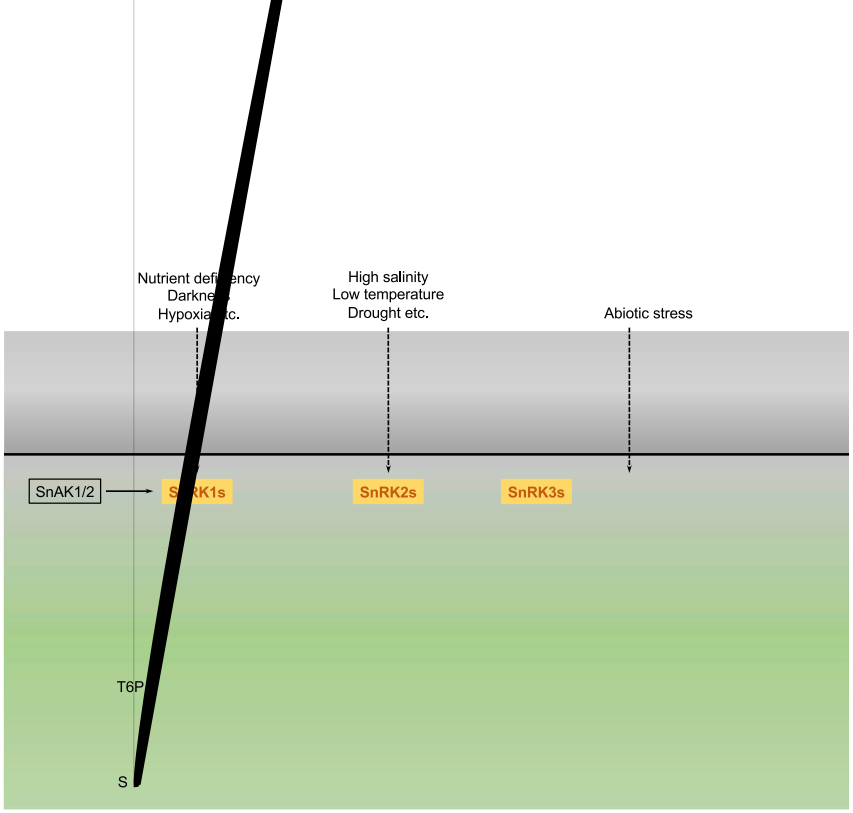
even when they are transferred to unstressed environments where resources are not limiting (Chapin, 1991), suggesting that an intrinsic process shaped by evolution controls the growth rate. In most plants, the stress-response program is sensitive to mild stresses, which prepares the plants for the possibility of more severe stress in the future. The stress signaling network actively represses cellular anabolic activities and plant growth early in the stress response even when the cellular energy status is not affected. This type of regulation becomes evident in transgenic plants overexpressing master regulators of abiotic stress responses, where the plant stress resistance in terms of survival is increased but growth is severely compromised (Kasuga et al., 1999). On the other hand, growth-promoting pathways have been found to actively repress the stress program (Wang et al., 2018a). Understanding the reciprocal regulation between the genetic programs for stress response and growth may be the key to breaking or resetting the stress-growth trade-off and thereby for engineering hardier but high-yielding crops.

Abiotic stresses cause various physiological and molecular changes in plants. While the specific sensing mechanisms depend on the type of stresses, the resulting signal transduction processes rely on similar signaling modules. We review here major components of abiotic stress signaling and how they are connected to growth regulation pathways (Figure 2). We focus on key protein kinases such as SnRKs (SNF1/AMPK-related protein kinases) and TOR (target of rapamycin) that are instrumental in regulating cellular energy homeostasis, stress responses, and growth. The signaling of stress-related phytohormones such as abscisic acid (ABA) also involves the SnRKs and extensive crosstalk with the growth program. At the end of our review, we will discuss possible strategies that can be used to reset the balance between growth and stress resistance in order to increase crop productivity under stress conditions.

STRESS DEFENSE SIGNALING PATHWAYS

Stress Sensing and Early Signaling

Because they are sessile, plants are extremely sensitive to stressful environments. Nevertheless, we know little about the



Testerink and Munnik, 2011; Waszczak et al., 2018). Stress-induced increases in cytosolic calcium concentration (denoted $[Ca^{2+}]_i$) vary in intensity, frequency, and subcellular location. Calcium transients can be detected in *Arabidopsis* guard cells within 15 s after osmotic stress treatment (Yuan et al., 2014). Calcium signals can then be detected by calcium-binding proteins, which usually feed the signal to an interacting protein kinase or to a kinase directly fused to them, such as the calcium-dependent protein kinases (CDPKs or CPKs). ROS in plants can be produced from multiple organelles including chloroplasts, mitochondria, and peroxisomes, or by the plasma membrane-localized Rboh NADPH oxidases. In particular, apoplastic ROS produced by RbohD and RbohF (respiratory burst oxidase homologs D and F) may stimulate specific calcium and electrical signals and mediate rapid systemic signaling in response to stress (Choi et al., 2016). This type of signal was found to propagate within *Arabidopsis* at ~8.4 cm per minute (Miller et al., 2009). Various abiotic stresses also facilitate the production of phosphatidic acid (PA), which is catalyzed by phospholipase Ds (PLDs) and which plays positive or negative roles under different stress conditions (Hong et al., 2016; Testerink and Munnik, 2011). In the guard cell, drought-induced PA production is required for RbohD/F-mediated ROS accumulation and stomatal closure (Zhang et al., 2009b). Stress signaling in plants also involves different families of kinases including those in the MAPK (mitogen-activated protein kinase) module (de Zelicourt et al., 2016), the SNF1-related protein kinases (SnRKs), CDPKs, and RLKs. For instance, MPK3, MPK4, and MPK6 can be activated within 2 min of exposure to drought, salt, or low temperature stresses (Droillard et al., 2002; Ichimura et al., 2000; Zhao et al., 2017).

overexpression of *KIN10*, in contrast, delays developmental transitions and improves survival under nutrient-shortage conditions (Baena-González et al., 2007). In the presence of exogenous sugars, however, SnRK1 knockdown promotes growth while SnRK1 overexpression reduces growth of *Arabidopsis* seedlings (Baena-González et al., 2007).

The composition of the SnRK1 protein complex and the upstream signals of SnRK1s differ in important ways from their counterparts in yeast or animals. In mammals, AMPKs function as heterotrimeric complexes that are composed of the catalytic α subunit and the non-catalytic β & γ subunits. In contrast, functional plant SnRK1 complexes contain one α subunit, one β subunit, and a plant-specific βγ subunit (Emanuelle et al., 2015). The chimeric βγ subunit is characterized by an N-terminal CBM (carbohydrate-binding module) domain, which typically occurs on AMPK β subunits, and four CBS (cystathionine b synthase) motifs in the C-terminal region (Emanuelle et al., 2015). Although AMP, and less effectively ADP, activates AMPK via multiple independent mechanisms (Gowans et al., 2013), the kinase activity of SnRK1 complexes is insensitive to AMP/ADP inhibition, and the CBM domains from the β or βγ subunit cannot actually bind carbohydrates (Emanuelle et al., 2015). A recent study indicated that the catalytic subunit of KIN10 or KIN11 is functional without protein complex formation (Ramon et al., 2019). Under normal growth conditions, the majority of the α subunit is excluded from the nucleus by association with the myristoylated β subunits but can translocate into the nucleus under stress treatments to mediate downstream transcriptional changes, while the β subunits remain in the cytosol or plasma membrane (Ramon et al., 2019).

The direct energy signals regulating SnRK1 activities remain to be elucidated. In *Arabidopsis*, two homologous kinases, SnAK1 (SnRK1 activating kinase 1)/GRIK2 (geminivirus rep interacting kinase 2) and SnAK2/GRIK1, activate the SnRK1s by phosphorylating the activation loop (Crozet et al., 2010; Shen et al., 2009). Additional evidence indicates that sugar phosphates are negative regulators of SnRK1 kinase activity (Nunes et al., 2013b; Toroser et al., 2000; Zhang et al., 2009a). T6P (trehalose 6-phosphate), G6P (glucose 6-phosphate), and G1P (glucose 1-phosphate) achieve 50% inhibition (IC50) of SnRK1 *in vitro* at 5.4 μM, 0.48, and ~10 mM, respectively (Nunes et al., 2013b; Toroser et al., 2000). Among them, T6P is of particular interest because its IC50 of SnRK1 inhibition is in the range of its physiological concentration. Moreover, T6P concentration is correlated with sucrose concentration *in vivo* (known as the Suc-T6P nexus model) (Figueroa and Lunn, 2016). T6P partly inhibits SnRK1 activity by disrupting the interaction between SnAKs and SnRK1 (Figueroa and Lunn, 2016; Zhai et al., 2018; Zhang et al., 2009a). In addition, two plant-specific KID (protein kinase A-interacting domain)-containing proteins, SKIN1 (SnRK1A-interacting negative regulator 1) and SKIN2, directly interact with and repress the function of SnRK1A in rice seedlings (Lin et al., 2014).

SnRK1s promote the cellular low energy response partly through the C/S_1 bZIP (group-C and S_1 bZIP) transcription factors (Dröge-Laser and Weiste, 2018). *Arabidopsis* contains five group-S_1 bZIPs and four group-C bZIPs. The bZIPs from different groups form heterodimers to activate the expression of enzymes involved in alternative metabolism under stress, such as the proline dehydrogenase (ProDH), which degrades proline to provide energy (Llorca et al., 2015; Weltmeier et al., 2006). SnRK1 directly phosphorylates bZIP63, a group-C bZIP, to promote its dimerization with itself or other bZIPs, and to activate downstream genes (Mair et al., 2015). On the other hand, bZIP11 (group S_1) downregulates T6P levels by enhancing the expression of T6P phosphatase genes (Ma et al., 2011). Transcriptome analyses in plants in which the expression of the five *S_1 bZIP* genes was knocked down indicated that over half of the differentially regulated genes are also targets of SnRK1s (Pedrotti et al., 2018). Interestingly, all S_1 bZIPs contain a conserved upstream open reading frame (uORF) that stalls translation when the sucrose level is high (Wiese et al., 2004), indicating that S_1 bZIPs are repressed at the translational level in the absence of stress.

SnRK2s are monomeric kinases that regulate plant responses to multiple abiotic stresses such as drought, salinity, cold, and heat. Experiments with *Arabidopsis* and rice showed that essentially all SnRK2s, except for AtSnRK2.9, can be activated by hyperosmotic stress (Boudsocq et al., 2004; Kobayashi et al., 2004). A subgroup of Raf-like protein kinases (RAFs) are very quickly activated by osmotic stress and then phosphorylate and activate SnRK2s (Lin et al., 2020; Soma et al., 2020; Takahashi et al., 2020). Based on the phylogenetic relationship deduced from protein sequences and functional analyses, SnRK2s can be further divided into 3 groups (Belin et al., 2006; Kulik et al., 2011; Yoshida et al., 2006). Group 3 SnRK2s (SnRK2.2/3/6 in *Arabidopsis*) are strongly activated by the plant hormone ABA (Boudsocq et al., 2004; Fujii and Zhu, 2009; Kobayashi et al., 2004). In response to osmotic stress, the ABA-unresponsive group 1 SnRK2s (SnRK2.1/4/5/9/10 in *Arabidopsis*) translocate to processing bodies, where they phosphorylate VARICOSE (VCS), a main component of the mRNA-decapping complex, and regulate mRNA decay and root architecture under salt stress (Kawa et al., 2020; McLoughlin et al., 2012; Soma et al., 2017). The SnRK2 proteins show high redundancy, and stress-hypersensitive phenotypes are only observed in high-order *snrk2* mutants (Fujii et al., 2011; Fujii and Zhu, 2009). Overexpression of either *SnRK2.6* or *SnRK2.8* not only confers hypersensitivity to ABA in terms of the inhibition of seed germination and of hypocotyl and root elongation but also promotes growth and biomass accumulation of Arabidopsis (Shin et al., 2007; Zheng et al., 2010), suggesting that SnRK2s play a role in promoting growth when the environment is favorable.

SnRK3s are involved in the regulation of plant responses to diverse abiotic stresses and particularly to ionic stresses (such as high Na^+, low K^+, and low nitrate) and high pH. SnRK3s are also called CIPKs (CBL-interacting protein kinases)/PKS (protein kinases related to SOS2) because SnRK3s function together with CBLs (calcineurin B-like)/SCaBPs (SOS3-like calcium-binding proteins), a family of EF-hand calcium-binding proteins. The signature FISL/NAF motif of SnRK3s functions as an autoinhibitory domain as well as the binding site for CBLs/SCaBPs (Albrecht et al., 2001; Guo et al., 2001). Stress-triggered Ca^{2+} binds to the CBLs/SCaBPs and induces conformational changes in the SCaBP-SnRK3 protein complex, causing a release of its autoinhibition. The SOS (SALT OVERLY SENSITIVE) signaling pathway mediating salt stress responses is a well-characterized SCaBP-SnRK3 module. When plants are under high salinity, Ca^{2+}-bound

SOS3 and SCaBP8 interact with and activate SOS2/SnRK3.11, which then phosphorylates and activates SOS1, a Na^+/H^+ antiporter located on the plasma membrane (Zhu, 2002). Similar modules also mediate low potassium, high magnesium, and high pH signaling (Kudla et al., 2018; Zhu, 2016). *Arabidopsis* contains 10 CBLs/SCaBPs and 25 SnRK3s. The large number of potential combinations of SCaBPs and SnRK3s and the observation of calcium signals under various stress conditions suggest that the SCaBP-SnRK3 module is widely used for plant signaling under other conditions. In rice, OsCIPK15 integrates hypoxia and sugar-starvation signals and activates SnRK1A (Lee et al., 2009). In *Arabidopsis*, CIPK14 also regulates the glucose response and physically interacts with KIN10/SnRK1.1 and KIN11/SnRK1.2 (Yan et al., 2014). On the other hand, SnAK1/GRIK1 and SnAK2/GRIK2 can phosphorylate and activate SOS2 under salt stress (Barajas-Lopez et al., 2018). These observations indicate crosstalk among SnRK subfamilies.

The ABA Signaling Pathway

ABA plays a major role in various abiotic stress responses and is generally regarded as a stress hormone. ABA is an isoprenoid hormone synthesized from carotenoids (Nambara and Marion-Poll, 2005). ABA mediates developmental processes such as seed maturation and dormancy as well as stress responses including stomatal closure, leaf senescence, and growth inhibition. Abiotic stresses such as drought and high salinity induce the biosynthesis of ABA, which then mediates stress response through phosphorylation-dependent signaling cascades.

The RCAR (regulatory component of ABA receptor)/PYR1 (pyrabactin resistance 1)/PYL (PYR1-like) proteins, hereafter referred to as PYLs, and type 2C protein phosphatases (PP2Cs) function as receptors and coreceptors, respectively, for ABA (Ma et al., 2009; Park et al., 2009). When *de novo* synthesis of ABA is induced by stress signals, ABA enters the hydrophobic binding pocket of the START domain of PYLs, which triggers a conformational change that closes the pocket and creates a binding surface for PP2Cs (Melcher et al., 2009). Binding of the PP2C to the ABA-PYL complex increases the affinity between ABA and PYLs by ~100-fold (Ma et al., 2009). Among the 76 PP2Cs in *Arabidopsis*, only the nine members from clade A (12 clades in total) have major roles in ABA signaling. Under normal growth conditions, clade A PP2Cs associate with and repress the activity of group 1 SnRK2s (Fujii and Zhu, 2009; Melcher et al., 2009). When ABA binds to PYLs and PP2Cs, SnRK2s are released from PP2C inhibition and can then phosphorylate downstream substrates, such as ABF/AREB (ABA-responsive-elements-binding factor/ABA-responsive-elements-binding protein) transcription factors for stress-responsive gene regulation, Rbohs for ROS production, and ion channels for stomatal closure (Sato et al., 2009; Sirichandra et al., 2009; Umezawa et al., 2013; Wang et al., 2013).

SnRK1s are also negatively regulated by PP2Cs. *KIN10/SnRK1.1* overexpression plants are ABA hypersensitive (Jossier et al., 2009). SnRK1 promotes seed maturation possibly through ABI3 (Radchuk et al., 2010, 2006). SnRK1 phosphorylates and stabilizes FUS3 to promote seed maturation under heat stress (Chan et al., 2017; Tsai and Gazzarrini, 2012). SnRK1 also phosphorylates ABI5 and AREBP *in vitro* (Bitrián et al., 2011; Zhang et al., 2008). Two clade A PP2Cs, ABI1 (ABA insensitive 1) and PP2CA (At3g11410), directly interact with the KA1 (kinase associated 1) domain of KIN10 and inhibit KIN10 activity by dephosphorylation (Rodrigues et al., 2013). A mutant deficient in four *PP2C* genes (*abi1*, *pp2ca*, *hai1*, and *hab1*) has defects in post-stress inactivation of SnRK1 activity, and exogenous application of ABA enhances SnRK1 activity (Rodrigues et al., 2013). Between 20%–30% of KIN10/SnRK1.1-regulated genes overlap with ABA-regulated genes (Rodrigues et al., 2013).

PP2Cs also have negative roles in SnRK3 regulation of transporters such as SOS1 and AKT1 (*Arabidopsis* K^+ transporter 1) (Lan et al., 2011; Ohta et al., 2003). The phosphatase ABI2 (ABA insensitive 2) binds to the PPI (protein phosphatase interaction) domain of SOS2 and inhibits SOS1 activation by SOS2 (Ohta et al., 2003). Subsequent research demonstrated that multiple SnRK3s can interact with specific members of clade A PP2Cs, indicating that this is a common mechanism for regulation of SnRK3-mediated signaling (Lan et al., 2011).

ABA has long been known for its role in inhibiting plant growth, although the mechanism is not well studied. Overexpression of any of the ABA-dependent transcription factors ABF2/AREB1/bZIP36, ABF3/bZIP37, or ABF4/AREB2/bZIP38 enhances plant survival under severe drought but reduces plant growth under normal conditions (Fujita et al., 2005; Furihata et al., 2006; Kang et al., 2002; Kim et al., 2004). Interestingly, loss-of-function mutations in a subgroup of rice ABA receptors, *OsPYL1*, *OsPYL4*, and *OsPYL6*, improve plant growth and grain yield in paddy fields. The *ospyl1/4/6* mutants show only some mild defects in stomatal closure and seed dormancy (Miao et al., 2018). These results suggest that some ABA receptor genes have evolved specialized functions in growth regulation because *PYL1*, *4*, and *6* in rice seem to mainly function in growth inhibition rather than drought resistance.

The Effects of Abiotic Stress on Energy Supply

Prolonged abiotic stress usually reduces the plant energy supply by inhibiting photosystem II activity (Gururani et al., 2015). For example, plants respond to water deficit by reducing stomatal opening in order to reduce water use. This leads to a series of negative effects on the photosystem. First, stomatal closure (aka. reduced stomatal conductance) and reduced mesophyll conductance of CO_2 (the diffusion rate of CO_2 through mesophyll cells) limit the internal CO_2 concentration and thereby contribute to a decreased photosynthesis rate (Flexas et al., 2006). Drought-induced synthesis of ABA is necessary for stomatal closure and decreased mesophyll conductance (Alexandersson et al., 2010; Bauer et al., 2013; Mizokami et al., 2015). Second, decreased CO_2 availability leads to a decline in the energy consumption of the Calvin-Benson cycle and to the over-reduction of the photosynthetic electron transport chain by excess light energy (Lawlor and Tezara, 2009). This leads to the accumulation of ROS, including singlet oxygen ($^1O_2$) and hydrogen peroxide (H_2O_2) produced by the chloroplast. The oxidative stress caused by ROS mainly affects chloroplast protein synthesis and photosystem II repair (Tikkanen et al., 2014). Consistent with the postulation that excess light stress is a main consequence of drought, transcriptome analyses showed that the expression of 70% of the high-light (excess-light)-induced genes is also increased under drought treatment (Estavillo et al., 2011).

In response to abiotic stress, plants divert substantial resources to prevent or repair damage caused by stress to maintain cellular homeostasis, as reflected by dramatic changes in the transcriptomic, proteomic, and metabolomic profiles of stressed plants within minutes or hours (Chinnusamy et al., 2004; Cramer et al., 2011; Kilian et al., 2007; Shulaev et al., 2008). Contrary to the presumption that stressed plants are under carbon and energy shortage due to the processes discussed above, physiological studies have revealed that the concentrations of soluble carbohydrates often increase in plants under water deficit (Hummel et al., 2010; Martínez-Noël and Tognetti, 2018; Muller et al., 2011). A common phenomenon observed in many tested plant species under stress is that the growth rate drops faster, albeit to a different extent, than the photosynthetic rate (Muller et al., 2011), indicating that stressed plants actively suppress growth (carbon consumption) to ensure adequate energy supply.

RECIPROCAL REGULATION BETWEEN STRESS AND GROWTH PROGRAMS

Repression of the stress response is necessary to ensure proper growth when there is no stress. ABA and osmotic stress responses in plants were recently found to be inhibited by the growth-promoting TOR kinase under favorable conditions (Wang et al., 2018a). Many components of the ABA signaling pathway, from ABA receptors to ABA-activated transcription factors (TFs), are also regulated by the ubiquitin proteasome system (UPS), which helps repress ABA signaling under normal conditions and which accelerates the stress response by maintaining a high turnover rate of proteins (Stone, 2019).

Under adverse environments, stress defense is activated, and growth is inhibited as part of the stress response. How growth is regulated under stress has been less studied than other stress responses. Plant growth relies on cell proliferation and cell expansion, and abiotic stress generally impedes plant growth by repressing both cell division and cell expansion. The decreases in cell division and cell expansion make different contributions to growth arrest in different natural accessions of *Arabidopsis* under drought stress, indicating that the mechanism of growth regulation under stress can be flexible (Aguirrezabal et al., 2006).

In addition, there is complex crosstalk between plant hormones. ABA generally functions antagonistically with the "growth-related" hormones like gibberellic acid (GA), brassinosteroids, cytokinin, and auxin. However, the relationship between ABA and other hormones may depend on the type of tissues and organs, the duration and intensity of the stress, and the developmental stage of the plant. We do not further consider this crosstalk among hormones in this review because the topic has been recently reviewed (Jameson and Song, 2016; Li et al., 2016; Müller and Munné-Bosch, 2015; Pinheiro and Chaves, 2011; Wang et al., 2020; Yu et al., 2015).

TOR Complex as a Central Regulator of Plant Growth

The evolutionarily conserved TOR complex functions as a master regulator of nutrient sensing and plant growth. Yeast and animals contain two TOR complexes, TORC1 and TORC2 (TOR complex 1 and 2), that differ in the composition of their subunits, while plants have only one complex, which is equivalent to TORC1 (van Dam et al., 2011). The TOR complex in *Arabidopsis* is composed of three subunits: TOR, RAPTOR, and LST8. The kinase subunit TOR is encoded by a single gene, and the latter two subunits are each encoded by two genes (*RAPTOR1*/*RAPTOR1B* and *RAPTOR2*/*RAPTOR1A*; *LST8-1* and *LST8-2*), among which *RAPTOR1*/*RAPTOR1B* and *LST8-1* are predominant. Null mutations of *TOR* cause embryo arrest at the globular stage (Menand et al., 2002; Ren et al., 2011), while a reduction in *TOR* expression levels or activity or a mutation in *RAPTOR1* and *LST8-1* results in growth defects, including impaired meristem-driven growth, reduced apical dominance, deformed floral organs, and delayed senescence (Anderson et al., 2005; Deprost et al., 2005; Moreau et al., 2012; Ren et al., 2012; Salem et al., 2018; Xiong et al., 2013). Transcriptome analyses of the effect of glucose on 3-day-old *Arabidopsis* seedlings revealed that glucose-triggered transcriptional changes depend on TOR activity (Xiong et al., 2013). TOR is required for the upregulation of genes involved in glycolysis, translation, and other anabolic processes, and downregulation of genes involved in autophagy, stress responses, and degradation of biomolecules in response to glucose treatment (Xiong et al., 2013). Direct substrates of TOR include S6K (ribosomal protein S6 kinase) and E2F TFs, which are master regulators of translation and the cell cycle, respectively (Figure 2) (Mahfouz et al., 2006; Schepetilnikov et al., 2013; Xiong et al., 2013). As is true for the SnRK1 complex, the direct signals that activate TOR remain to be discovered because many of the known regulators of TOR in yeast and animal systems are not present in plants (Roustan et al., 2016). Exogenous or photosynthesis-derived glucose activates TOR, and this effect can be blocked by inhibitors of glycolysis or respiration (Xiong et al., 2013). TOR activity is downregulated by sulfur deficiency; this regulation likely operates through the glucose pathway (Dong et al., 2017). In the shoot meristem, TOR is required to integrate the sugar and light signals in order to activate meristem activity (Li et al., 2017; Pfeiffer et al., 2016); light activates TOR via auxin and the small GTPase ROP2 (Rho-related protein 2) (Li et al., 2017).

Reciprocal Regulation between SnRKs and TOR

The activity of TOR is inhibited by various abiotic stresses. The kinase activities of TOR and its substrate protein S6K1 are downregulated in response to cold and osmotic stress (Dong et al., 2019; Mahfouz et al., 2006; Wang et al., 2017). TOR activity is downregulated within the first 2 h after cold treatment but subsequently recovers (Wang et al., 2017), possibly reflecting the acclimation process during which growth is slowly recovered. A reduction in TOR or S6K1 activity results in hypersensitivity to cold stress (Deprost et al., 2007; Dong et al., 2019; Mahfouz et al., 2006). Overexpression of TOR in *Arabidopsis* enhances growth under control conditions but not under cold stress conditions (Dong et al., 2019). However, overexpression of TOR in rice enhances biomass accumulation, yield, and water-use efficiency when water availability is limited (Bakshi et al., 2017).

SnRK1 and TOR have largely overlapping and opposite effects in regulating cellular activities, and their antagonistic roles have been recently reviewed (Baena-González and Hanson, 2017; Margalha et al., 2019; Rodriguez et al., 2019). In animals, the Raptor subunit of TOR is a known substrate of AMPK (Gwinn

et al., 2008). A proteomics-based study in *Arabidopsis* also recently identified TOR subunits as KIN10-interacting proteins and determined that KIN10 phosphorylates RAPTOR1 *in vitro* (Nukarinen et al., 2016). This suggests that SnRK1 phosphorylates and inactivates TOR in plants (Figure 2), an inference that is supported by genetic analyses of the role of SnRK1 and TOR in the regulation of autophagy. Autophagy is a regulated self-degradation and resource-recycling process in response to nutrient shortage and abiotic stresses. SnRK1 and TOR are positive and negative regulators, respectively, of autophagy. Repression of TOR or overexpression of SnRK1 increases autophagy under normal growth conditions, but overexpression of TOR or repression of SnRK1 decreases autophagy induced by nutrient deficiency and various abiotic stresses (Chen et al., 2017; Pu et al., 2017). However, inhibition or activation of both SnRK1 and TOR results in phenotypes similar to those resulting from the manipulation of TOR activity alone, indicating that SnRK1 activates autophagy by inactivating TOR (Soto-Burgos and Bassham, 2017).

A recent study revealed the mechanism by which SnRK2s and TOR reciprocally regulate each other (Figure 2) (Wang et al., 2018a). Osmotic stress or ABA inhibits TOR kinase activity, as measured by T449 phosphorylation on the TOR substrate S6K1 (Wang et al., 2018a). ABA promotes SnRK2-catalyzed phosphorylation of RAPTOR1 on Ser897 and reduces TOR kinase activity by disrupting the association of RAPTOR1 and TOR (Wang et al., 2018a). On the other hand, TOR phosphorylates the PYL ABA receptors at a conserved serine residue (Ser119 in PYL1). This phosphorylation is sufficient to disrupt the binding of ABA to PYLs, thus, preventing the activation of SnRK2s (Wang et al., 2018a). TOR signaling is therefore involved in repressing ABA and stress responses in unstressed plants. Consistent with a negative role of TOR in ABA signaling, inhibition of TOR kinase activity enhances the response to ABA. The *raptor1* mutants are more sensitive to ABA than the wild type in terms of decreases in seed germination rate, chlorophyll content, and ABA-induced gene expression levels (Wang et al., 2018a).

No direct link between SnRK3/CIPK proteins and TOR has been reported. However, considering the important role of SnRK3s/CIPKs in mediating responses to nutrient stresses such as nitrogen and phosphate starvation and other ionic stresses, it would not be surprising to find that SnRK3 signaling also crosstalks with TOR signaling.

Regulation of Cell Division

As in other eukaryotes, cyclins and cyclin-dependent kinases (CDKs) in plants function as the main molecular drivers of the cell cycle. *Arabidopsis* contains at least 50 cyclins and 12 CDKs, which are classified into 10 and 6 groups, respectively, and which are usually named by a combination of letters and numbers (CYCD3;1, for example, is the name for cyclin 3;1 in group D) (Gutierrez, 2009; Wang et al., 2004). CDKs are usually expressed at a steady level, but their activity requires interaction with specific cyclins, whose levels vary during the cell cycle. The CDKA/CDCD complexes function through RBR (retinoblastoma related) to regulate the activity of the E2F/DP family of TFs, which mediate the transition from G1 to S phase; CDKA drives the G2/M transition when it is complexed with B-, D-, or A-type cyclins (Gutierrez, 2009). Both RBR and E2Fs are also directly regulated by the TOR kinase (Van Leene et al., 2019; Xiong et al., 2013). The activity of cyclin-CDK complexes are negatively regulated by CDK inhibitors (CKIs), which in plants include the ICK/KRP family (inhibitor of cyclin-dependent kinase/Kip-related protein, seven members) and the SMR family (SIAMESE-related, 17 members).

Drought and salt stresses inhibit cell division by downregulating the expression of cyclin and CDK genes and by upregulating the expression of CKI genes. Transcriptome analyses indicated that multiple *CYC* and *CDK* transcripts are downregulated within 24 h after osmotic stress (Skirycz et al., 2011a). Salt treatment of *Arabidopsis* strongly reduces CDK kinase activity and *CYCB1;2* promoter activity within 2 h (West et al., 2004). The expression of some *ICK/KRP* and *SMR* genes are induced by abiotic stress or ABA (Peres et al., 2007; Wang et al., 1998; Yi et al., 2014). Expression of *ICK1*, the first identified *CKI* gene from the ICK/KRP family, is induced by ABA (Wang et al., 1998). *SMR5* and *SMR7* are upregulated by H_2O_2 and high light, and this regulation is required for repair of ROS-induced DNA damages (Yi et al., 2014). Multiple *SMR* genes are induced by mild drought stress in developing *Arabidopsis* leaves, and *SMR1* is required for drought-induced growth repression (Dubois et al., 2018).

Cell Expansion and Cell Wall Signaling

Plant cell expansion requires coordination between cell wall loosening and biosynthesis, i.e., these processes must be balanced so that cell wall integrity is not impaired. The cell wall can be divided into the primary and secondary cell wall. The latter is lignified and in general cannot be extended. Primary (growing) cell walls are mainly composed of polysaccharides, including cellulose, hemicellulose, and pectin, and 1%–5% structural proteins. Cell wall loosening requires the action of expansins, xyloglucan hydrolases, and pectin methyltransferases (Cosgrove, 2018). Because the activity of expansins is promoted by low pH and because plasma-membrane-localized proton ATPases (H^+-ATPases) are required for apoplastic acidification, H^+-ATPases are also important regulators of cell enlargement.

The plant cell wall is the organelle directly exposed to the environment. Cell wall integrity can be compromised by biotic or abiotic factors. The cell wall is thus involved in sensing and transducing environmental signals and coordinating plant growth and the stress response. Plants contain hundreds of RLKs, which in principle can sense changes in cell wall integrity and regulate cell growth. A class of malectin-like receptor kinases, named CrRLK1Ls (*Catharanthus roseus* receptor-like kinase1-likes), are important for monitoring cell wall integrity and for mediating cell wall extension during growth, during PAMP-triggered immunity responses, and during responses to various abiotic stresses (Franck et al., 2018). The CrRLK1L family in *Arabidopsis* contains at least 17 members, including several characterized members like FERONIA (FER), THESEUS1 (THE1), and ERULUS (ERU) (Franck et al., 2018).

FER is the best characterized member of the CrRLK1L family. The loss-of-function *fer* mutants are semi-dwarf and exhibit developmental defects in trichome and root hair formation, pollen tube reception, and pavement cell morphogenesis, indicating deficiencies in polar growth (Duan et al., 2010; Escobar-Restrepo et al., 2007; Guo et al., 2009). FER is also required for mechanical signaling. Compared with wild-type seedlings, *fer*

mutants display abolished or reduced $[Ca^{2+}]_i$ signals in response to cell wall stretching and decreased induction of touch-responsive genes, unstable root cell expansion profiles, and various growth phenotypes indicative of impaired mechanical development (Shih et al., 2014). FER interacts with cell-wall-localized proteins, LRX3/4/5 (LEUCINE-RICH REPEAT/EXTENSIN 3/4/5) to regulate vacuole expansion during cellular elongation (Dünser et al., 2019). FER functions as a receptor for RALF1 (rapid alkalinization factor 1), a secreted peptide that inhibits cell elongation (Haruta et al., 2014). The RALF1-FER interaction activates FER, which phosphorylates AHA2 (plasma membrane H^+-ATPase 2) at Ser899, causing inhibition of proton transport and cessation of cell wall extension (Haruta et al., 2014). FER also contributes to plant defense by positively regulating PAMP-triggered immunity. The *fer* mutants are more susceptible to the bacterial pathogens *Pseudomonas syringae* pv. *tomato* DC3000 (Stegmann et al., 2017). FER promotes the ligand-induced formation of immune complexes FLS2 (flagellin-sensing 2)-BAK1 (BRI1-associated kinase 1) and EFR (elongation factor TU receptor)-BAK1, whereas the RALF1 (rapid alkalization factor 1) peptide inhibits plant immunity by binding to FER (Stegmann et al., 2017). These results indicate that FER and possibly other CrRLK1Ls act as signaling hubs that integrate intrinsic and external signals to regulate plant growth and stress responses.

CrRLK1Ls as Signaling Nodes in Plant Growth and Stress Response

CrRLK1Ls are implicated in stress response because the expression of most *CrRLK1Ls* is decreased by abiotic stresses (Lindner et al., 2012), although only the function of FER has been characterized under abiotic stress. Loss-of-function *fer* mutants are hypersensitive to salt, cold, and heat stress and to ABA, indicating that FER helps maintain plant growth under abiotic stress conditions (Chen et al., 2016; Yu et al., 2012; Zhao et al., 2018). Under salt stress, Na^+ displaces Ca^{2+} in the cell wall, causing disruption of pectin and other changes in the cell wall (Byrt et al., 2018). During the acclimation to salt stress, cells in the elongation zone of *fer* mutant roots initiate the cell elongation process but eventually rupture due to the failure to reinforce salt-damaged cell walls, suggesting that FER is required for this reinforcement (Feng et al., 2018). The functioning of FER in salt tolerance requires a group of cell wall-localized LRX proteins, i.e., LRX3, LRX4, and LRX5 (Zhao et al., 2018). The *lrx3/4/5* triple mutant plants have a salt hypersensitive phenotype similar to that of the *fer-4* mutant (Zhao et al., 2018). Both LRX3/4/5 and FER proteins interact with a group of phylogenetically related RALF peptides (RALF22/23) (Zhao et al., 2018). Salt stress induces RALF22/23 maturation, and RALF overexpression phenocopies the *fer* and *lrx3/4/5* mutants, possibly by promoting the cytosolic internalization of FER (Zhao et al., 2018). These data suggest that the LRX3/4/5-RALF22/23-FER module coordinates plant growth and stress responses (Figure 2). Salt-induced cell wall damage may be sensed by the LRX proteins, which release the RALF peptides to promote FER internalization.

ABA signaling also participates in the regulation of FER. The PP2C phosphatase ABI2 directly interacts with the intracellular kinase domain of FER and inhibits its phosphorylation (Chen et al., 2016). ABA enhances FER phosphorylation in a PYL-dependent manner, while attenuating ABA signaling partially rescues the ABA- and stress-hypersensitive phenotype of *fer* (Chen et al., 2016). ABA and FER signaling likely converge on the H^+-ATPase AHA2 because ABA was also reported to promote AHA2 phosphorylation and inhibit its activity (Planes et al., 2015). The extensive crosstalk between stress/ABA signaling and FER signaling supports the notion that FER is a hub for plant growth regulation under stress.

FER was also found to negatively regulate ABA signaling (Figure 2). FER directly interacts with GEF1/4/10 (guanine exchange factor 1/4/10) to activate the GTPase ROP11/ARAC10 (Rho of plants 11), which in turn activates the phosphatase activity of ABI2 (Yu et al., 2012). Loss-of-function mutations in *FER*, *GEF1/4/10*, or *ROP11/ARAC* result in hypersensitivity to ABA (Yu et al., 2012). FER-activated Rho GTPases were also reported to promote ROS production during root hair development (Duan et al., 2010). Considering the important functions of ROS in abiotic stress signaling, it is possible that the stress and FER pathways also crosstalk through ROS. Among all CrRLK1Ls, only *FER* and *THE1* are expressed in most tissues (Lindner et al., 2012). The other CrRLK1Ls may also be involved in cell wall integrity sensing and in crosstalk with stress signaling in specific cells and tissues.

Reciprocal Regulation at the Transcript Level

Extensive crosstalk between the stress signaling and growth regulation pathways also occurs at the level of transcriptional and post-transcriptional regulation. Reciprocal repression between growth-related TFs and stress-related TFs is evident in transcriptome and ChIP-seq (chromatin immunoprecipitation sequencing) analyses (Liu et al., 2018; Song et al., 2016; Xie et al., 2019). The miR396-growth-regulating factors (GRF)/GIF regulatory module is a good example (Figure 2). GRFs belong to a small family (typically 8–20 in land plants, 9 in *Arabidopsis*) of plant-specific TFs that generally promote plant growth and development at virtually every stage of the plant life cycle (Omidbakhshfard et al., 2015). GRFs extensively crosstalk with the signaling pathways of growth-promoting hormones including GA, brassinosteroids, and auxin to regulate plant growth and crop yield (Che et al., 2015; Gao et al., 2015; Lee et al., 2018; Li et al., 2018; Tang et al., 2018; Zhang et al., 2018a). In angiosperms, the majority of *GRFs* are post-transcriptionally repressed by a highly conserved microRNA family, miR396 (Beltramino et al., 2018). In addition, GRF activity is stimulated by GIFs (GRF-interacting factors). The expression of miR396 is upregulated by low temperature, high salinity, drought, and UV stress (Beltramino et al., 2018; Casadevall et al., 2013; Chen et al., 2015; Li et al., 2019; Yang and Yu, 2010; Yuan et al., 2019), which results in decreased *GRF* transcripts. GRF7 directly binds to and represses the *DREB2A* gene, a master transcription factor that regulates the osmotic stress response (Kim et al., 2012). The *grf7* mutants display similar phenotypes as *DREB2A* overexpressing plants, in which growth is decreased under favorable conditions and survival is increased under abiotic stress (Kim et al., 2012; Liu et al., 1998). Importantly, transcriptome analyses of the *grf7* mutant identified more than 200 genes that are derepressed under normal conditions and that are involved in osmotic stress response and ABA biosynthesis and signaling, including *NCED3*, *ABI1*, and *ABF4/AREB2* (Kim

et al., 2012). These data indicate a pivotal role of GRF7 in repressing the stress response. On the other hand, the *miR396* expression level is significantly decreased in *GRF1* and *GRF3* overexpression plants, indicating reciprocal regulation between miR396 and GRFs (Hewezi and Baum, 2012).

STRATEGIES TO IMPROVE PLANT GROWTH UNDER STRESS

Several observations suggest that the stress-growth trade-off can be manipulated. First, among the plant species that are adapted to harsh environments, some can achieve the highest biomass under favorable growth conditions, aka the highest yield potential (Henry, 2010; Zhang et al., 2018b). Second, natural variations in stress resistance (as indicated by relative growth under stress conditions) exist among different accessions of the same species (Bechtold et al., 2018; Clauw et al., 2016). Third, intraspecific hybrids usually show a concomitant enhancement in both growth and stress tolerance (Miller et al., 2015).

Many examples in the literature also support the optimistic view that stress resistance can be increased without a significant yield penalty, i.e., it may be possible to break the growth-stress trade-off and reset the balance. Positive regulators of stress response have been traditionally overexpressed to increase plant survival under abiotic stress. However, many of these manipulations also resulted in retarded plant growth. These observations are consistent with the previously discussed reciprocal inhibition between stress and growth pathways. A prediction from the reciprocal inhibition model is that desensitizing the stress-response pathway to sacrifice stress resistance will help improve yield. This can be achieved by increasing the expression or activity of growth regulators or by decreasing the sensitivity of stress pathways. Overexpression of *GA5*, a GA biosynthesis gene, together with *DREB1A/CBF3* in *Arabidopsis* increased both biomass accumulation and survival under drought (Kudo et al., 2019). Similarly, increasing the level of cytokinin during the maturation period significantly improved growth and survival rate under drought (Rivero et al., 2007). On the other hand, mutating the stress resistance-promoting factors *PYL1/4/6* enhances the growth and grain yield of rice (Miao et al., 2018). There are more cases in which improved growth has been achieved under stress conditions, but without much understanding of the underlying mechanism. Overexpression of the transcription factor AtNF-YB1, for example, increases the growth, yield, and survival of *Arabidopsis* and maize under drought (Nelson et al., 2007). Transcriptome analyses indicated that NF-YB1 regulates different sets of genes than the CBF or ABF TFs (Nelson et al., 2007). Ectopic production of melatonin in plants enhances abiotic stress tolerance (Wang et al., 2018b). Transcriptome analyses indicated that, in addition to the induction of some stress-related genes, melatonin upregulates genes involved in photosynthesis and nitrogen assimilation (Shi et al., 2015). A common theme in these examples is that certain aspects of the growth program is strengthened in these plants.

To design crops that can better maintain growth under stress, we need an improved understanding of the critical components of the stress and growth pathways and of their crosstalk in a tissue- and temporal-specific manner. For example, the repression of growth by SnRK1 is relieved in the hypocotyl of plants under high temperature; the T6P-SnAK1/GRIK1-KIN10 module regulates the phosphorylation status of the PIF4 transcription factor, a master regulator of thermomorphogenesis (Hwang et al., 2019). Recent advances in the application of T6P in agriculture also indicate that SnRK1 has tissue-specific functions. Both increases or decreases in the level of T6P, which inhibits SnRK1 activity and is part of the Suc-T6P nexus, have been found to increase yield under drought conditions (Griffiths et al., 2016; Nuccio et al., 2015). The decrease of T6P levels by the overexpression of a trehalose phosphate phosphatase in maize phloem companion cells increases SnRK1 activity, the expression of *SWEET* sugar transporter genes, and sucrose flux into the grain (Oszvald et al., 2018). On the other hand, an increase in T6P levels in wheat kernels helps inhibit SnRK1 activity and promotes starch biosynthesis and seed growth (Griffiths et al., 2016; Paul et al., 2018).

CONCLUSIONS AND PERSPECTIVES

Plants in the wild constantly face stresses, and ideal growth conditions for any plant may only be achieved in a controlled environment. Thus, being under stress is the "normal" state, and plant growth in the wild is usually inhibited. Contrary to the presumption that the growth-stress trade-off is due to limits in energy/carbon supply, increasing evidence indicates that the trade-off mainly results from the active suppression of growth by stress signaling pathways. The regulatory networks for stress response and growth regulation crosstalk at multiple levels. Further studies will elucidate critical links and will suggest strategies for increasing stress resistance with little or no yield penalty. Many traits such as stress-induced flowering (Jung and Müller, 2009) and senescence (Mittler and Blumwald, 2010) increase survival but reduce crop productivity. Selection against some of these traits (e.g., selection for delayed senescence) has been effective in breeding programs (Richards et al., 2010; Rivero et al., 2007). It follows that, rather than increasing crop productivity by making plants hypersensitive to stress, we should increase crop productivity by desensitizing the stress response. One caveat is that such crops may fail under very severe stresses. Regardless, our ability to achieve both stress resistance and high productivity is likely to increase with a better understanding of the relationships between growth and stress-response pathways.

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