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Review

Thriving under Stress: How Plants Balance Growth and the Stress Response

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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 affect plant growth is not only a fundamental scientific question but is also of vital importance for agriculture and food security. Adverse environments were estimated to cause an overall yield loss of ~70% in key agricultural crops, i.e., the average yield was only ~30% of the genetic yield potential (Boyer, 1982; Shinozaki et al., 2015; Vij and Tyagi, 2007; Zurbriggen et al., 2010). Abiotic stress caused by deficiencies or excesses in environmental factors including water, salt, light, temperature, and nutrients can substantially reduce plant growth and productivity and even survival (Figure 1). Abiotic stress reduces plant growth because, by definition, when plants are under stress, the environmental conditions are not optimal for growth processes including cell division and expansion. Drought stress, for example, inhibits plant growth because water is needed for cell turgor (the pressure on the cell wall exerted by enclosed liquid) that drives cell expansion; cold stress reduces plant growth because the activities of enzymes and other proteins are lower under cold temperatures. Slower plant growth under stress, however, is not only a passive consequence of the adverse environment. Plants under stress also actively slow their growth in order to adapt to the stress conditions (Figure 1). This "active" growth inhibition is achieved through stress-triggered cell signaling.

Researchers often measure stress resistance as the rate of plant growth or survival under stress relative to control conditions. The two measures of stress resistance (relative growth versus survival) may be inconsistent (Skirycz et al., 2011b) because survivability can be achieved by sacrificing growth and vice versa. In this review, we refer to "stress resistance" as the relative ability to grow, unless specified otherwise. The effect of stress on plant growth can be measured as a decrease in plant growth rate or as a decrease in biomass accumulation. For example, the leaf elongation rate of cereals responds to hyperosmotic stress within seconds and is one of the most sensitive plant responses to stress (Hsiao et al., 1976). Some plants can increase the growth of certain plant parts as a response to specific stresses; they can, for example, increase root growth in response to mild drought or increase stem growth in response to low light or flooding conditions (Xu et al., 2006; Zhao et al., 2014). This type of stress-induced growth of a specific organ is usually achieved by sacrificing the growth of other parts of the plant. In these cases, whole plant biomass accumulation better reflects the overall effect of the stress.

Scientists have been trying to mitigate the negative effects of stress on crop productivity through genetic engineering but with limited success. Although many genes involved in plant abiotic stress signaling and response have been identified, translating the knowledge into crops with enhanced stress resistance remains challenging (Tardieu, 2012; Zhu, 2016). Among the main crops, only one transgenic maize cultivar with a stress-resistance trait (drought resistance) has been commercialized so far (Castiglioni et al., 2008). The seemingly unavoidable trade-off between growth and stress resistance is usually explained by energy and resource limitations: plants under stress must divert energy and resources away from growth and toward a stress response. Increasing evidence indicates, however, that plants actively repress growth under stress conditions as an adaptive strategy to maximize survival. Plant species that are adapted to desert, tundra, or other harsh environments grow slowly

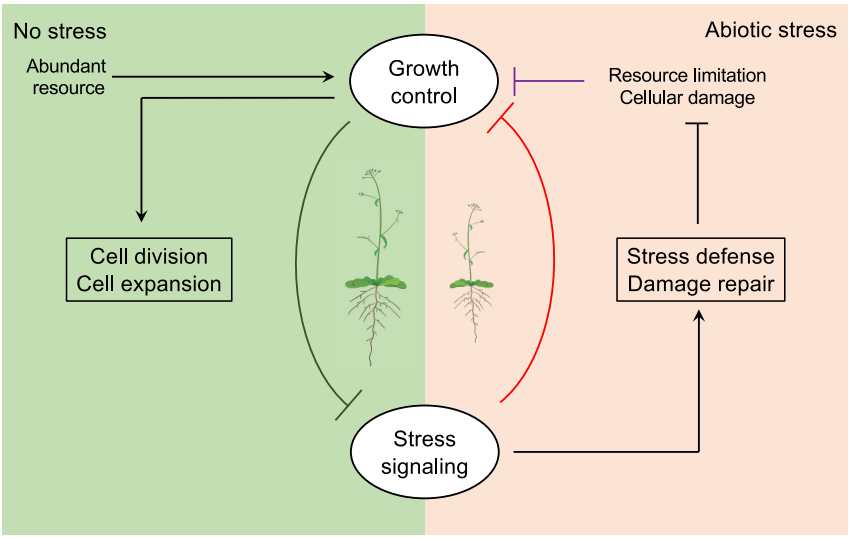

Figure 1. The Relationship between Stress Signaling and Plant Growth
Abiotic stress passively inhibits plant growth (purple path) by causing cellular damage and/or limiting resources (e.g., carbon dioxide, nutrients, and energy). Through stress sensing and signaling, abiotic stress actively inhibits growth (red path) and also activates stress responses to prevent and repair cellular damages caused by the stress. When resources are abundant and stress is absent, growth signaling is activated, repressing stress signaling (green path) in addition to promoting growth. Arrows indicate positive regulation, and bars indicate negative regulation.

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 molecular mechanisms underlying stress sensing despite its importance. In principle, any early perturbations in a cellular component in response to the stress can serve as a stress-sensing mechanism as long as the perturbation is recognized and amplified by molecular machineries in the cell (Zhu, 2016). For example, drought-induced hyperosmotic stress causes changes in turgor pressure, plasma membrane curvature, and cellular osmotic potential. Thus, plasma membrane-localized mechanosensors or cytoplasm-localized sensors have been proposed to sense osmotic stress in plants (Christmann et al., 2013; Zhu, 2016). Similarly, stress sensing through perception of stress-triggered cell wall changes may be linked to growth regulation because cell expansion-driven growth involves constant remodeling of the cell wall. In this context, receptor-like kinases (RLKs), which are greatly amplified in most plant genomes, may function to sense various stress-induced changes in the plant cell wall. The stress sensing and signaling likely occur in multiple waves, which may explain the observed multiple waves of transcriptome changes (Kollist et al., 2019). Inter-organ signals may also play a role. As noted earlier, an osmotic stress-caused decrease in the leaf elongation rate can be detected within seconds (Chazen and Neumann, 1994). The initial effects of hyperosmotic stress may be transmitted as hydraulic signals at the speed of sound from roots to shoots (Malone, 1993; Wildon et al., 1992). A hyperosmolality-gated calcium channel called OSCA1 (reduced hyperosmolality-induced $[Ca^{2+}]_i$ increase 1) was identified through a genetic screen (Yuan et al., 2014). Loss-of-function mutations in *OSCA1* lead to reduced cytosolic calcium transients and impaired stomatal closure in response to hyperosmotic treatment (Yuan et al., 2014), although the mutant plants exhibit no evident growth phenotypes under stress. Using a similar screening system, Jiang et al. identified glycosyl inositol phosphorylceramide (GIPC) sphingolipids in the plasma membrane as the sensor of sodium ion because loss-of-function mutations in the glucuronosyltransferase MOCA1 (monocation-induced $[Ca^{2+}]_i$ increase 1) results in loss of calcium spikes in response to ionic stress and reduced growth under salt stress (Jiang et al., 2019). The downstream effectors of OSCA1 and MOCA1 remain to be determined.

Once a stress is sensed by the plant cell, the signal is relayed and amplified by second messengers such as calcium, reactive oxygen species (ROS), phospholipids, and nitric oxide (NO), along with different types of protein kinases (Kudla et al., 2018;

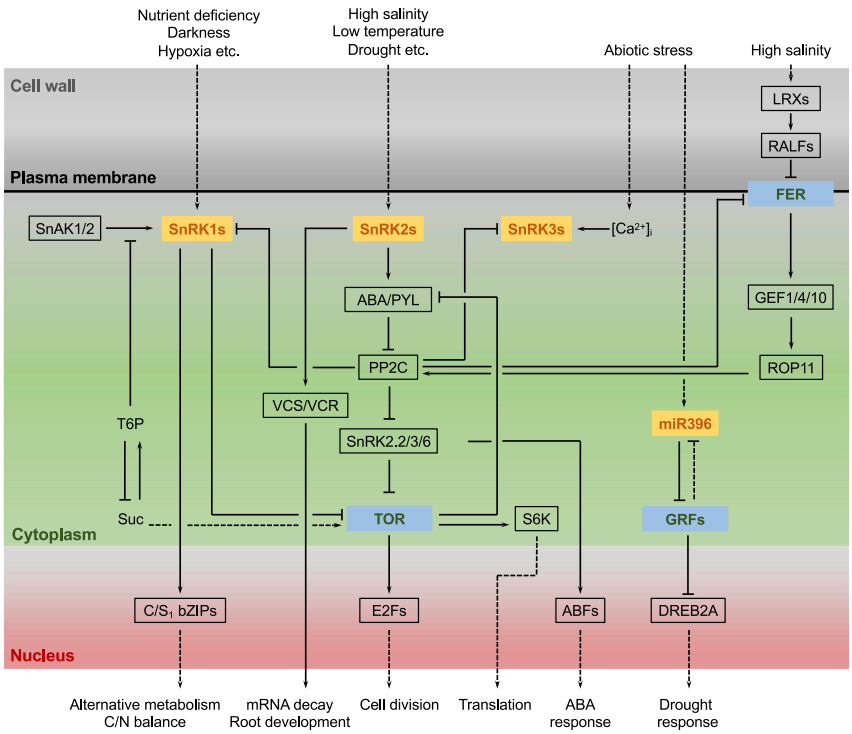

Figure 2. Critical Molecular Components in Stress and Growth Signaling Pathways and Their Roles in the Reciprocal Regulation of the Stress Response and Growth

Red letters in golden rectangles and green letters in cyan rectangles indicate critical nodes for stress and growth signaling pathways, respectively. Solid and dashed lines indicate direct and indirect regulation, respectively. Arrows indicate positive regulation, and bars indicate negative regulation.

The Central Role of SnRKs in Mediating Stress Responses

Higher plants extensively use the SNF1-related protein kinases (SnRKs) to mediate stress signaling under various types of stress (Figure 2). Based on phylogenetic analyses of the kinase domain, SnRKs can be divided into three subfamilies: SnRK1, SnRK2, and SnRK3. The SnRK2 and SnRK3 subfamilies are greatly expanded in embryophytes (Jamsheer et al., 2019). *Arabidopsis thaliana*, for example, contains three SnRK1s, ten SnRK2s, and 25 SnRK3s (Hrabak et al., 2003). Among them, SnRK1s are most similar to the classical SNF1 (sucrose non-fermentable 1) protein from yeast and to the AMPKs (AMP-dependent protein kinases) from animals, both of which are activated by reduced cellular energy states. SnRK2s are characterized by an acidic patch in the C-terminal domain, while SnRK3s contain a signature FISL/NAF motif (named after the conserved amino acid residues within the motif) near the C terminus of the kinase domain (Hrabak et al., 2003). SnRK2s are activated by the phytohormone ABA and abiotic stresses such as drought and salinity, whereas SnRK3s act as part of calcium signaling to mediate plant development as well as responses to ionic and nutrient stresses such as high sodium, potassium deficiency, and nitrogen nutrient starvation.

Like their orthologs in fungi and animals, SnRK1s in plants function as the principle regulators of cellular energy homeostasis. SnRK1 activity can be activated by low nutrient stress or energy starvation as a result of prolonged darkness, hypoxia, or chemical inhibition of photosynthesis and can be inhibited by exogenous sucrose or glucose (Ananieva et al., 2008; Baena-González et al., 2007; Bledsoe et al., 2017; Coello and Martínez-Barajas, 2014; Nunes et al., 2013a). Once activated, SnRK1 kinases globally upregulate catabolic activities and repress anabolic processes (Baena-González et al., 2007). Among the three *SnRK1* genes in *Arabidopsis*, *KIN10* (SNF1 kinase homolog 10)/*SnRK1.1* and *KIN11* (SNF1 kinase homolog 11)/*SnRK1.2* are believed to be the major players given that *KIN12*/*SnRK1.3* expression cannot be detected in most tissues. The *kin10 kin11* double mutant is embryonic lethal because SnRK1s are required for metabolic regulation during normal growth and development (Baena-González et al., 2007). When grown in soil, *kin10 kin11* knockdown lines have small stature, impaired starch mobilization at night, strong accumulation of anthocyanin, and early senescence before flowering;

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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REFERENCES

Aguirrezabal, L., Bouchier-Combaud, S., Radziejwoski, A., Dauzat, M., Cookson, S.J., and Granier, C. (2006). Plasticity to soil water deficit in Arabidopsis thaliana: dissection of leaf development into underlying growth dynamic and cellular variables reveals invisible phenotypes. Plant Cell Environ. *29*, 2216–2227.

Albrecht, V., Ritz, O., Linder, S., Harter, K., and Kudla, J. (2001). The NAF domain defines a novel protein-protein interaction module conserved in Ca2+-regulated kinases. EMBO J. *20*, 1051–1063.

Alexandersson, E., Danielson, J.A., Råde, J., Moparthi, V.K., Fontes, M., Kjellbom, P., and Johanson, U. (2010). Transcriptional regulation of aquaporins in accessions of Arabidopsis in response to drought stress. Plant J. *61*, 650–660.

Ananieva, E.A., Gillaspy, G.E., Ely, A., Burnette, R.N., and Erickson, F.L. (2008). Interaction of the WD40 domain of a myoinositol polyphosphate 5-phosphatase with SnRK1 links inositol, sugar, and stress signaling. Plant Physiol. *148*, 1868–1882.

Anderson, G.H., Veit, B., and Hanson, M.R. (2005). The Arabidopsis AtRaptor genes are essential for post-embryonic plant growth. BMC Biol. *3*, 12.

Baena-González, E., and Hanson, J. (2017). Shaping plant development through the SnRK1-TOR metabolic regulators. Curr. Opin. Plant Biol. *35*, 152–157.

Baena-González, E., Rolland, F., Thevelein, J.M., and Sheen, J. (2007). A central integrator of transcription networks in plant stress and energy signalling. Nature *448*, 938–942.

Bakshi, A., Moin, M., Kumar, M.U., Reddy, A.B., Ren, M., Datla, R., Siddiq, E.A., and Kirti, P.B. (2017). Ectopic expression of Arabidopsis target of rapamycin (AtTOR) improves water-use efficiency and yield potential in rice. Sci. Rep. *7*, 42835.

Barajas-Lopez, J.D., Moreno, J.R., Gamez-Arjona, F.M., Pardo, J.M., Punkkinen, M., Zhu, J.K., Quintero, F.J., and Fujii, H. (2018). Upstream kinases of plant SnRKs are involved in salt stress tolerance. Plant J. *93*, 107–118.

Bauer, H., Ache, P., Lautner, S., Fromm, J., Hartung, W., Al-Rasheid, K.A., Sonnewald, S., Sonnewald, U., Kneitz, S., Lachmann, N., et al. (2013). The stomatal response to reduced relative humidity requires guard cell-autonomous ABA synthesis. Curr. Biol. *23*, 53–57.

Bechtold, U., Ferguson, J.N., and Mullineaux, P.M. (2018). To defend or to grow: lessons from Arabidopsis C24. J. Exp. Bot. *69*, 2809–2821.

Belin, C., de Franco, P.O., Bourbousse, C., Chaignepain, S., Schmitter, J.M., Vavasseur, A., Giraudat, J., Barbier-Brygoo, H., and Thomine, S. (2006). Identification of features regulating OST1 kinase activity and OST1 function in guard cells. Plant Physiol. *141*, 1316–1327.

Beltramino, M., Ercoli, M.F., Debernardi, J.M., Goldy, C., Rojas, A.M.L., Nota, F., Alvarez, M.E., Vercruyssen, L., Inzé, D., Palatnik, J.F., and Rodriguez, R.E. (2018). Robust increase of leaf size by Arabidopsis thaliana GRF3-like transcription factors under different growth conditions. Sci. Rep. *8*, 13447.

Bitrián, M., Roodbarkelari, F., Horváth, M., and Koncz, C. (2011). BAC-recombineering for studying plant gene regulation: developmental control and cellular localization of SnRK1 kinase subunits. Plant J. *65*, 829–842.

Bledsoe, S.W., Henry, C., Griffiths, C.A., Paul, M.J., Feil, R., Lunn, J.E., Stitt, M., and Lagrimini, L.M. (2017). The role of Tre6P and SnRK1 in maize early kernel development and events leading to stress-induced kernel abortion. BMC Plant Biol. *17*, 74.

Boudsocq, M., Barbier-Brygoo, H., and Laurière, C. (2004). Identification of nine sucrose nonfermenting 1-related protein kinases 2 activated by hyperosmotic and saline stresses in Arabidopsis thaliana. J. Biol. Chem. *279*, 41758–41766.

Boyer, J.S. (1982). Plant productivity and environment. Science *218*, 443–448.

Byrt, C.S., Munns, R., Burton, R.A., Gilliham, M., and Wege, S. (2018). Root cell wall solutions for crop plants in saline soils. Plant Sci. *269*, 47–55.

Casadevall, R., Rodriguez, R.E., Debernardi, J.M., Palatnik, J.F., and Casati, P. (2013). Repression of growth regulating factors by the microRNA396 inhibits cell proliferation by UV-B radiation in Arabidopsis leaves. Plant Cell *25*, 3570–3583.

Castiglioni, P., Warner, D., Bensen, R.J., Anstrom, D.C., Harrison, J., Stoecker, M., Abad, M., Kumar, G., Salvador, S., D'Ordine, R., et al. (2008). Bacterial RNA chaperones confer abiotic stress tolerance in plants and improved grain yield in maize under water-limited conditions. Plant Physiol. *147*, 446–455.

Chan, A., Carianopol, C., Tsai, A.Y., Varatharajah, K., Chiu, R.S., and Gazzarrini, S. (2017). SnRK1 phosphorylation of FUSCA3 positively regulates embryogenesis, seed yield, and plant growth at high temperature in Arabidopsis. J. Exp. Bot. *68*, 4219–4231.

Chapin, F.S. (1991). Integrated responses of plants to stress. BioScience *41*, 29–36.

Chazen, O., and Neumann, P.M. (1994). Hydraulic signals from the roots and rapid cell-wall hardening in growing maize (Zea mays L.) leaves are primary responses to polyethylene glycol-induced water deficits. Plant Physiol. *104*, 1385–1392.

Che, R., Tong, H., Shi, B., Liu, Y., Fang, S., Liu, D., Xiao, Y., Hu, B., Liu, L., Wang, H., et al. (2015). Control of grain size and rice yield by GL2-mediated brassinosteroid responses. Nat. Plants *2*, 15195.

Chen, J., Yu, F., Liu, Y., Du, C., Li, X., Zhu, S., Wang, X., Lan, W., Rodriguez, P.L., Liu, X., et al. (2016). FERONIA interacts with ABI2-type phosphatases to facilitate signaling cross-talk between abscisic acid and RALF peptide in Arabidopsis. Proc. Natl. Acad. Sci. USA *113*, E5519–E5527.

Chen, L., Luan, Y.S., and Zhai, J.M. (2015). Sp-miR396a-5p acts as a stress-responsive genes regulator by conferring tolerance to abiotic stresses and susceptibility to Phytophthora nicotianae infection in transgenic tobacco. Plant Cell Rep. *34*, 2013–2025.

Chen, L., Su, Z.Z., Huang, L., Xia, F.N., Qi, H., Xie, L.J., Xiao, S., and Chen, Q.F. (2017). The AMP-activated protein kinase KIN10 is involved in the regulation of autophagy in Arabidopsis. Front. Plant Sci. *8*, 1201.

Chinnusamy, V., Schumaker, K., and Zhu, J.K. (2004). Molecular genetic perspectives on cross-talk and specificity in abiotic stress signalling in plants. J. Exp. Bot. *55*, 225–236.

Choi, W.G., Hilleary, R., Swanson, S.J., Kim, S.H., and Gilroy, S. (2016). Rapid, long-distance electrical and calcium signaling in plants. Annu. Rev. Plant Biol. *67*, 287–307.

Christmann, A., Grill, E., and Huang, J. (2013). Hydraulic signals in long-distance signaling. Curr. Opin. Plant Biol. *16*, 293–300.

Clauw, P., Coppens, F., Korte, A., Herman, D., Slabbinck, B., Dhondt, S., Van Daele, T., De Milde, L., Vermeersch, M., Maleux, K., et al. (2016). Leaf growth response to mild drought: natural variation in Arabidopsis sheds light on trait architecture. Plant Cell *28*, 2417–2434.

Coello, P., and Martínez-Barajas, E. (2014). The activity of SnRK1 is increased in Phaseolus vulgaris seeds in response to a reduced nutrient supply. Front. Plant Sci. *5*, 196.

Cosgrove, D.J. (2018). Diffuse growth of plant cell walls. Plant Physiol. *176*, 16–27.

Cramer, G.R., Urano, K., Delrot, S., Pezzotti, M., and Shinozaki, K. (2011). Effects of abiotic stress on plants: a systems biology perspective. BMC Plant Biol. *11*, 163.

Crozet, P., Jammes, F., Valot, B., Ambard-Bretteville, F., Nessler, S., Hodges, M., Vidal, J., and Thomas, M. (2010). Cross-phosphorylation between Arabidopsis thaliana sucrose nonfermenting 1-related protein kinase 1 (AtSnRK1) and its activating kinase (AtSnAK) determines their catalytic activities. J. Biol. Chem. *285*, 12071–12077.

de Zelicourt, A., Colcombet, J., and Hirt, H. (2016). The role of MAPK modules and ABA during abiotic stress signaling. Trends Plant Sci. *21*, 677–685.

Deprost, D., Truong, H.N., Robaglia, C., and Meyer, C. (2005). An Arabidopsis homolog of RAPTOR/KOG1 is essential for early embryo development. Biochem. Biophys. Res. Commun. *326*, 844–850.

Deprost, D., Yao, L., Sormani, R., Moreau, M., Leterreux, G., Nicolaï, M., Bedu, M., Robaglia, C., and Meyer, C. (2007). The Arabidopsis TOR kinase links plant growth, yield, stress resistance and mRNA translation. EMBO Rep. *8*, 864–870.

Dong, Y., Silbermann, M., Speiser, A., Forieri, I., Linster, E., Poschet, G., Allboje Samami, A., Wanatabe, M., Sticht, C., Teleman, A.A., et al. (2017). Sulfur availability regulates plant growth via glucose-TOR signaling. Nat. Commun. *8*, 1174.

Dong, Y., Teleman, A.A., Jedmowski, C., Wirtz, M., and Hell, R. (2019). The Arabidopsis THADA homologue modulates TOR activity and cold acclimation. Plant Biol. (Stuttg.) *21*, 77–83.

Dröge-Laser, W., and Weiste, C. (2018). The C/S1 bZIP network: a regulatory hub orchestrating plant energy homeostasis. Trends Plant Sci. *23*, 422–433.

Droillard, M., Boudsocq, M., Barbier-Brygoo, H., and Laurière, C. (2002). Different protein kinase families are activated by osmotic stresses in

Arabidopsis thaliana cell suspensions. Involvement of the MAP kinases AtMPK3 and AtMPK6. FEBS Lett. *527*, 43–50.

Duan, Q., Kita, D., Li, C., Cheung, A.Y., and Wu, H.M. (2010). FERONIA receptor-like kinase regulates Rho GTPase signaling of root hair development. Proc. Natl. Acad. Sci. USA *107*, 17821–17826.

Dubois, M., Selden, K., Bediée, A., Rolland, G., Baumberger, N., Noir, S., Bach, L., Lamy, G., Granier, C., and Genschik, P. (2018). SIAMESE-RELATED1 is regulated posttranslationally and participates in repression of leaf growth under moderate drought. Plant Physiol. *176*, 2834–2850.

Dünser, K., Gupta, S., Herger, A., Feraru, M.I., Ringli, C., and Kleine-Vehn, J. (2019). Extracellular matrix sensing by FERONIA and leucine- rich repeat extensins controls vacuolar expansion during cellular elongation in Arabidopsis thaliana. EMBO J. *38*.

Emanuelle, S., Hossain, M.I., Moller, I.E., Pedersen, H.L., van de Meene, A.M., Doblin, M.S., Koay, A., Oakhill, J.S., Scott, J.W., Willats, W.G., et al. (2015). SnRK1 from Arabidopsis thaliana is an atypical AMPK. Plant J. *82*, 183–192.

Escobar-Restrepo, J.M., Huck, N., Kessler, S., Gagliardini, V., Gheyselinck, J., Yang, W.C., and Grossniklaus, U. (2007). The FERONIA receptor-like kinase mediates male-female interactions during pollen tube reception. Science *317*, 656–660.

Estavillo, G.M., Crisp, P.A., Pornsiriwong, W., Wirtz, M., Collinge, D., Carrie, C., Giraud, E., Whelan, J., David, P., Javot, H., et al. (2011). Evidence for a SAL1-PAP chloroplast retrograde pathway that functions in drought and high light signaling in Arabidopsis. Plant Cell *23*, 3992–4012.

Feng, W., Kita, D., Peaucelle, A., Cartwright, H.N., Doan, V., Duan, Q., Liu, M.C., Maman, J., Steinhorst, L., Schmitz-Thom, I., et al. (2018). The FERONIA Receptor Kinase Maintains Cell-Wall Integrity during Salt Stress through Ca2+ Signaling. Curr. Biol. *28*, 666–675.e5.

Figueroa, C.M., and Lunn, J.E. (2016). A tale of two sugars: trehalose 6-phosphate and sucrose. Plant Physiol. *172*, 7–27.

Flexas, J., Bota, J., Galmés, J., Medrano, H., and Ribas-Carbó, M. (2006). Keeping a positive carbon balance under adverse conditions: responses of photosynthesis and respiration to water stress. Physiol. Plant. *127*, 343–352.

Franck, C.M., Westermann, J., and Boisson-Dernier, A. (2018). Plant Malectin-like receptor kinases: from cell wall integrity to immunity and beyond. Annu. Rev. Plant Biol. *69*, 301–328.

Fujii, H., Verslues, P.E., and Zhu, J.K. (2011). Arabidopsis decuple mutant reveals the importance of SnRK2 kinases in osmotic stress responses in vivo. Proc. Natl. Acad. Sci. USA *108*, 1717–1722.

Fujii, H., and Zhu, J.K. (2009). Arabidopsis mutant deficient in 3 abscisic acid-activated protein kinases reveals critical roles in growth, reproduction, and stress. Proc. Natl. Acad. Sci. USA *106*, 8380–8385.

Fujita, Y., Fujita, M., Satoh, R., Maruyama, K., Parvez, M.M., Seki, M., Hiratsu, K., Ohme-Takagi, M., Shinozaki, K., and Yamaguchi-Shinozaki, K. (2005). AREB1 is a transcription activator of novel ABRE-dependent ABA signaling that enhances drought stress tolerance in Arabidopsis. Plant Cell *17*, 3470–3488.

Furihata, T., Maruyama, K., Fujita, Y., Umezawa, T., Yoshida, R., Shinozaki, K., and Yamaguchi-Shinozaki, K. (2006). Abscisic acid-dependent multisite phosphorylation regulates the activity of a transcription activator AREB1. Proc. Natl. Acad. Sci. USA *103*, 1988–1993.

Gao, F., Wang, K., Liu, Y., Chen, Y., Chen, P., Shi, Z., Luo, J., Jiang, D., Fan, F., Zhu, Y., and Li, S. (2015). Blocking miR396 increases rice yield by shaping inflorescence architecture. Nat. Plants *2*, 15196.

Gowans, G.J., Hawley, S.A., Ross, F.A., and Hardie, D.G. (2013). AMP is a true physiological regulator of AMP-activated protein kinase by both allosteric activation and enhancing net phosphorylation. Cell Metab. *18*, 556–566.

Griffiths, C.A., Sagar, R., Geng, Y., Primavesi, L.F., Patel, M.K., Passarelli, M.K., Gilmore, I.S., Steven, R.T., Bunch, J., Paul, M.J., and Davis, B.G. (2016). Chemical intervention in plant sugar signalling increases yield and resilience. Nature *540*, 574–578.

Guo, H., Li, L., Ye, H., Yu, X., Algreen, A., and Yin, Y. (2009). Three related receptor-like kinases are required for optimal cell elongation in Arabidopsis thaliana. Proc. Natl. Acad. Sci. USA *106*, 7648–7653.

Guo, Y., Halfter, U., Ishitani, M., and Zhu, J.K. (2001). Molecular characterization of functional domains in the protein kinase SOS2 that is required for plant salt tolerance. Plant Cell *13*, 1383–1400.

Gururani, M.A., Venkatesh, J., and Tran, L.S. (2015). Regulation of photosynthesis during abiotic stress-induced photoinhibition. Mol. Plant *8*, 1304–1320.

Gutierrez, C. (2009). The Arabidopsis cell division cycle. Arabidopsis Book *7*, e0120.

Gwinn, D.M., Shackelford, D.B., Egan, D.F., Mihaylova, M.M., Mery, A., Vasquez, D.S., Turk, B.E., and Shaw, R.J. (2008). AMPK phosphorylation of raptor mediates a metabolic checkpoint. Mol. Cell *30*, 214–226.

Haruta, M., Sabat, G., Stecker, K., Minkoff, B.B., and Sussman, M.R. (2014). A peptide hormone and its receptor protein kinase regulate plant cell expansion. Science *343*, 408–411.

Henry, R.J. (2010). Evaluation of plant biomass resources available for replacement of fossil oil. Plant Biotechnol. J. *8*, 288–293.

Hewezi, T., and Baum, T.J. (2012). Complex feedback regulations govern the expression of miRNA396 and its GRF target genes. Plant Signal. Behav. *7*, 749–751.

Hong, Y., Zhao, J., Guo, L., Kim, S.C., Deng, X., Wang, G., Zhang, G., Li, M., and Wang, X. (2016). Plant phospholipases D and C and their diverse functions in stress responses. Prog. Lipid Res. *62*, 55–74.

Hrabak, E.M., Chan, C.W., Gribskov, M., Harper, J.F., Choi, J.H., Halford, N., Kudla, J., Luan, S., Nimmo, H.G., Sussman, M.R., et al. (2003). The Arabidopsis CDPK-SnRK superfamily of protein kinases. Plant Physiol. *132*, 666–680.

Hsiao, T.C., Acevedo, E., Fereres, E., and Henderson, D.W. (1976). Stress metabolism - water stress, growth, and osmotic adjustment. Philos. Trans. R. Soc. Lond. B *273*, 479–500.

Hummel, I., Pantin, F., Sulpice, R., Piques, M., Rolland, G., Dauzat, M., Christophe, A., Pervent, M., Bouteillé, M., Stitt, M., et al. (2010). Arabidopsis plants acclimate to water deficit at low cost through changes of carbon usage: an integrated perspective using growth, metabolite, enzyme, and gene expression analysis. Plant Physiol. *154*, 357–372.

Hwang, G., Kim, S., Cho, J.Y., Paik, I., Kim, J.I., and Oh, E. (2019). Trehalose-6-phosphate signaling regulates thermoresponsive hypocotyl growth in Arabidopsis thaliana. EMBO Rep. *20*, e47828.

Ichimura, K., Mizoguchi, T., Yoshida, R., Yuasa, T., and Shinozaki, K. (2000). Various abiotic stresses rapidly activate Arabidopsis MAP kinases ATMPK4 and ATMPK6. Plant J. *24*, 655–665.

Jameson, P.E., and Song, J. (2016). Cytokinin: a key driver of seed yield. J. Exp. Bot. *67*, 593–606.

Jamsheer, K.M., Jindal, S., and Laxmi, A. (2019). Evolution of TOR-SnRK dynamics in green plants and its integration with phytohormone signaling networks. J. Exp. Bot. *70*, 2239–2259.

Jiang, Z., Zhou, X., Tao, M., Yuan, F., Liu, L., Wu, F., Wu, X., Xiang, Y., Niu, Y., Liu, F., et al. (2019). Plant cell-surface GIPC sphingolipids sense salt to trigger Ca2+ influx. Nature *572*, 341–346.

Jossier, M., Bouly, J.P., Meimoun, P., Arjmand, A., Lessard, P., Hawley, S., Grahame Hardie, D., and Thomas, M. (2009). SnRK1 (SNF1-related kinase 1) has a central role in sugar and ABA signalling in Arabidopsis thaliana. Plant J. *59*, 316–328.

Jung, C., and Müller, A.E. (2009). Flowering time control and applications in plant breeding. Trends Plant Sci. *14*, 563–573.

Kang, J.Y., Choi, H.I., Im, M.Y., and Kim, S.Y. (2002). Arabidopsis basic leucine zipper proteins that mediate stress-responsive abscisic acid signaling. Plant Cell *14*, 343–357.

Kasuga, M., Liu, Q., Miura, S., Yamaguchi-Shinozaki, K., and Shinozaki, K. (1999). Improving plant drought, salt, and freezing tolerance by gene transfer of a single stress-inducible transcription factor. Nat. Biotechnol. *17*, 287–291.

Kawa, D., Meyer, A.J., Dekker, H.L., Abd-El-Haliem, A.M., Gevaert, K., Van De Slijke, E., Maszkowska, J., Bucholc, M., Dobrowolska, G., De Jaeger, G., et al. (2020). SnRK2 protein kinases and mRNA decapping machinery control root development and response to salt. Plant Physiol. *182*, 361–377.

Kilian, J., Whitehead, D., Horak, J., Wanke, D., Weinl, S., Batistic, O., D'Angelo, C., Bornberg-Bauer, E., Kudla, J., and Harter, K. (2007). The AtGenExpress global stress expression data set: protocols, evaluation and model data analysis of UV-B light, drought and cold stress responses. Plant J. *50*, 347–363.

Kim, J.-S., Mizoi, J., Kidokoro, S., Maruyama, K., Nakajima, J., Nakashima, K., Mitsuda, N., Takiguchi, Y., Ohme-Takagi, M., Kondou, Y., et al. (2012). Arabidopsis growth-regulating factor7 functions as a transcriptional repressor of abscisic acid- and osmotic stress-responsive genes, including DREB2A. Plant Cell *24*, 3393–3405.

Kim, S., Kang, J.Y., Cho, D.I., Park, J.H., and Kim, S.Y. (2004). ABF2, an ABRE-binding bZIP factor, is an essential component of glucose signaling and its overexpression affects multiple stress tolerance. Plant J. *40*, 75–87.

Kobayashi, Y., Yamamoto, S., Minami, H., Kagaya, Y., and Hattori, T. (2004). Differential activation of the rice sucrose nonfermenting1-related protein kinase2 family by hyperosmotic stress and abscisic acid. Plant Cell *16*, 1163–1177.

Kollist, H., Zandalinas, S.I., Sengupta, S., Nuhkat, M., Kangasjärvi, J., and Mittler, R. (2019). Rapid responses to abiotic stress: priming the landscape for the signal transduction network. Trends Plant Sci. *24*, 25–37.

Kudla, J., Becker, D., Grill, E., Hedrich, R., Hippler, M., Kummer, U., Parniske, M., Romeis, T., and Schumacher, K. (2018). Advances and current challenges in calcium signaling. New Phytol. *218*, 414–431.

Kudo, M., Kidokoro, S., Yoshida, T., Mizoi, J., Kojima, M., Takebayashi, Y., Sakakibara, H., Fernie, A.R., Shinozaki, K., and Yamaguchi-Shinozaki, K. (2019). A gene-stacking approach to overcome the trade-off between drought stress tolerance and growth in Arabidopsis. Plant J. *97*, 240–256.

Kulik, A., Wawer, I., Krzywińska, E., Bucholc, M., and Dobrowolska, G. (2011). SnRK2 protein kinases–key regulators of plant response to abiotic stresses. Omics *15*, 859–872.

Lan, W.Z., Lee, S.C., Che, Y.F., Jiang, Y.Q., and Luan, S. (2011). Mechanistic analysis of AKT1 regulation by the CBL-CIPK-PP2CA interactions. Mol. Plant *4*, 527–536.

Lawlor, D.W., and Tezara, W. (2009). Causes of decreased photosynthetic rate and metabolic capacity in water-deficient leaf cells: a critical evaluation of mechanisms and integration of processes. Ann. Bot. *103*, 561–579.

Lee, K.W., Chen, P.W., Lu, C.A., Chen, S., Ho, T.H., and Yu, S.M. (2009). Coordinated responses to oxygen and sugar deficiency allow rice seedlings to tolerate flooding. Sci. Signal. *2*, ra61.

Lee, S.-J., Lee, B.H., Jung, J.-H., Park, S.K., Song, J.T., and Kim, J.H. (2018). GROWTH-REGULATING FACTOR and GRF-INTERACTING FACTOR specify meristematic cells of gynoecia and anthers. Plant Physiol. *176*, 717–729.

Li, A.L., Wen, Z., Yang, K., and Wen, X.P. (2019). Conserved miR396b-GRF regulation is involved in abiotic stress responses in Pitaya (Hylocereus polyrhizus). Int. J. Mol. Sci. *20*.

Li, S., Tian, Y., Wu, K., Ye, Y., Yu, J., Zhang, J., Liu, Q., Hu, M., Li, H., Tong, Y., et al. (2018). Modulating plant growth-metabolism coordination for sustainable agriculture. Nature *560*, 595–600.

Li, W., Herrera-Estrella, L., and Tran, L.P. (2016). The yin-yang of cytokinin homeostasis and drought acclimation/adaptation. Trends Plant Sci. *21*, 548–550.

Li, X., Cai, W., Liu, Y., Li, H., Fu, L., Liu, Z., Xu, L., Liu, H., Xu, T., and Xiong, Y. (2017). Differential TOR activation and cell proliferation in Arabidopsis root and shoot apexes. Proc. Natl. Acad. Sci. USA *114*, 2765–2770.

Lin, C.R., Lee, K.W., Chen, C.Y., Hong, Y.F., Chen, J.L., Lu, C.A., Chen, K.T., Ho, T.H., and Yu, S.M. (2014). SnRK1A-interacting negative regulators modulate the nutrient starvation signaling sensor SnRK1 in source-sink communication in cereal seedlings under abiotic stress. Plant Cell *26*, 808–827.

Lin, Z., Li, Y., Zhang, Z., Liu, X., Hsu, C.-C., Du, Y., Sang, T., Zhu, C., Wang, Y., Satheesh, V., et al. (2020). A RAF-SnRK2 kinase cascade mediates early osmotic stress signaling in higher plants. Nat. Commun. *11*, 613.

Lindner, H., Müller, L.M., Boisson-Dernier, A., and Grossniklaus, U. (2012). CrRLK1L receptor-like kinases: not just another brick in the wall. Curr. Opin. Plant Biol. *15*, 659–669.

Liu, C., Wang, B., Li, Z., Peng, Z., and Zhang, J. (2018). TsNAC1 is a key transcription factor in abiotic stress resistance and growth. Plant Physiol. *176*, 742–756.

Liu, Q., Kasuga, M., Sakuma, Y., Abe, H., Miura, S., Yamaguchi-Shinozaki, K., and Shinozaki, K. (1998). Two transcription factors, DREB1 and DREB2, with an EREBP/AP2 DNA binding domain separate two cellular signal transduction pathways in drought- and low-temperature-responsive gene expression, respectively, in Arabidopsis. Plant Cell *10*, 1391–1406.

Llorca, C.M., Berendzen, K.W., Malik, W.A., Mahn, S., Piepho, H.P., and Zentgraf, U. (2015). The elucidation of the interactome of 16 Arabidopsis bZIP factors reveals three independent functional networks. PLoS One *10*, e0139884.

Ma, J., Hanssen, M., Lundgren, K., Hernández, L., Delatte, T., Ehlert, A., Liu, C.-M., Schluepmann, H., Dröge-Laser, W., Moritz, T., et al. (2011). The sucrose-regulated Arabidopsis transcription factor bZIP11 reprograms metabolism and regulates trehalose metabolism. New Phytol. *191*, 733–745.

Ma, Y., Szostkiewicz, I., Korte, A., Moes, D., Yang, Y., Christmann, A., and Grill, E. (2009). Regulators of PP2C phosphatase activity function as abscisic acid sensors. Science *324*, 1064–1068.

Mahfouz, M.M., Kim, S., Delauney, A.J., and Verma, D.P. (2006). Arabidopsis TARGET OF rapamycin interacts with RAPTOR, which regulates the activity of S6 kinase in response to osmotic stress signals. Plant Cell *18*, 477–490.

Mair, A., Pedrotti, L., Wurzinger, B., Anrather, D., Simeunovic, A., Weiste, C., Valerio, C., Dietrich, K., Kirchler, T., Nägele, T., et al. (2015). SnRK1-triggered switch of bZIP63 dimerization mediates the low-energy response in plants. eLife *4*, e05828.

Malone, M. (1993). Hydraulic signals. Phil. Trans. R. Soc. Lond. B *341*, 33–39.

Margalha, L., Confraria, A., and Baena-González, E. (2019). SnRK1 and TOR: modulating growth-defense trade-offs in plant stress responses. J. Exp. Bot. *70*, 2261–2274.

Martínez-Noël, G.M.A., and Tognetti, J.A. (2018). Sugar signaling under abiotic stress in plants. Plant Metabolites and Regulation under Environmental Stress (Academic Press), pp. 397–406.

McLoughlin, F., Galvan-Ampudia, C.S., Julkowska, M.M., Caarls, L., van der Does, D., Laurière, C., Munnik, T., Haring, M.A., and Testerink, C. (2012). The Snf1-related protein kinases SnRK2.4 and SnRK2.10 are involved in maintenance of root system architecture during salt stress. Plant J. *72*, 436–449.

Melcher, K., Ng, L.-M., Zhou, X.E., Soon, F.F., Xu, Y., Suino-Powell, K.M., Park, S.Y., Weiner, J.J., Fujii, H., Chinnusamy, V., et al. (2009). A gate-latch-lock mechanism for hormone signalling by abscisic acid receptors. Nature *462*, 602–608.

Menand, B., Desnos, T., Nussaume, L., Berger, F., Bouchez, D., Meyer, C., and Robaglia, C. (2002). Expression and disruption of the Arabidopsis TOR (target of rapamycin) gene. Proc. Natl. Acad. Sci. USA *99*, 6422–6427.

Miao, C., Xiao, L., Hua, K., Zou, C., Zhao, Y., Bressan, R.A., and Zhu, J.K. (2018). Mutations in a subfamily of abscisic acid receptor genes promote rice growth and productivity. Proc. Natl. Acad. Sci. USA *115*, 6058–6063.

Miller, G., Schlauch, K., Tam, R., Cortes, D., Torres, M.A., Shulaev, V., Dangl, J.L., and Mittler, R. (2009). The plant NADPH oxidase RBOHD mediates rapid systemic signaling in response to diverse stimuli. Sci. Signal. *2*, ra45.

Miller, M., Song, Q., Shi, X., Juenger, T.E., and Chen, Z.J. (2015). Natural variation in timing of stress-responsive gene expression predicts heterosis in intraspecific hybrids of Arabidopsis. Nat. Commun. *6*, 7453.

Mittler, R., and Blumwald, E. (2010). Genetic engineering for modern agriculture: challenges and perspectives. Annu. Rev. Plant Biol. *61*, 443–462.

Mizokami, Y., Noguchi, K., Kojima, M., Sakakibara, H., and Terashima, I. (2015). Mesophyll conductance decreases in the wild type but not in an ABA-deficient mutant (aba1) of Nicotiana plumbaginifolia under drought conditions. Plant Cell Environ. *38*, 388–398.

Moreau, M., Azzopardi, M., Clément, G., Dobrenel, T., Marchive, C., Renne, C., Martin-Magniette, M.L., Taconnat, L., Renou, J.P., Robaglia, C., and Meyer, C. (2012). Mutations in the Arabidopsis homolog of LST8/GβL, a partner of the target of rapamycin kinase, impair plant growth, flowering, and metabolic adaptation to long days. Plant Cell *24*, 463–481.

Muller, B., Pantin, F., Génard, M., Turc, O., Freixes, S., Piques, M., and Gibon, Y. (2011). Water deficits uncouple growth from photosynthesis, increase C content, and modify the relationships between C and growth in sink organs. J. Exp. Bot. *62*, 1715–1729.

Müller, M., and Munné-Bosch, S. (2015). Ethylene response factors: a key regulatory hub in hormone and stress signaling. Plant Physiol. *169*, 32–41.

Nambara, E., and Marion-Poll, A. (2005). Abscisic acid biosynthesis and catabolism. Annu. Rev. Plant Biol. *56*, 165–185.

Nelson, D.E., Repetti, P.P., Adams, T.R., Creelman, R.A., Wu, J., Warner, D.C., Anstrom, D.C., Bensen, R.J., Castiglioni, P.P., Donnarummo, M.G., et al. (2007). Plant nuclear factor Y (NF-Y) B subunits confer drought tolerance and lead to improved corn yields on water-limited acres. Proc. Natl. Acad. Sci. USA *104*, 16450–16455.

Nuccio, M.L., Wu, J., Mowers, R., Zhou, H.-P., Meghji, M., Primavesi, L.F., Paul, M.J., Chen, X., Gao, Y., Haque, E., et al. (2015). Expression of trehalose-6-phosphate phosphatase in maize ears improves yield in well-watered and drought conditions. Nat. Biotechnol. *33*, 862–869.

Nukarinen, E., Nägele, T., Pedrotti, L., Wurzinger, B., Mair, A., Landgraf, R., Börnke, F., Hanson, J., Teige, M., Baena-Gonzalez, E., et al. (2016). Quantitative phosphoproteomics reveals the role of the AMPK plant ortholog SnRK1 as a metabolic master regulator under energy deprivation. Sci. Rep. *6*, 31697.

Nunes, C., O'Hara, L.E., Primavesi, L.F., Delatte, T.L., Schluepmann, H., Somsen, G.W., Silva, A.B., Fevereiro, P.S., Wingler, A., and Paul, M.J. (2013a). The trehalose 6-phosphate/SnRK1 signaling pathway primes growth recovery following relief of sink limitation. Plant Physiol. *162*, 1720–1732.

Nunes, C., Primavesi, L.F., Patel, M.K., Martinez-Barajas, E., Powers, S.J., Sagar, R., Fevereiro, P.S., Davis, B.G., and Paul, M.J. (2013b). Inhibition of SnRK1 by metabolites: tissue-dependent effects and cooperative inhibition by glucose 1-phosphate in combination with trehalose 6-phosphate. Plant Physiol. Biochem. *63*, 89–98.

Ohta, M., Guo, Y., Halfter, U., and Zhu, J.K. (2003). A novel domain in the protein kinase SOS2 mediates interaction with the protein phosphatase 2C ABI2. Proc. Natl. Acad. Sci. USA *100*, 11771–11776.

Omidbakhshfard, M.A., Proost, S., Fujikura, U., and Mueller-Roeber, B. (2015). Growth-regulating factors (GRFs): a small transcription factor family with important functions in plant biology. Mol. Plant *8*, 998–1010.

Oszvald, M., Primavesi, L.F., Griffiths, C.A., Cohn, J., Basu, S.S., Nuccio, M.L., and Paul, M.J. (2018). Trehalose 6-phosphate regulates photosynthesis and assimilate partitioning in reproductive tissue. Plant Physiol. *176*, 2623–2638.

Park, S.-Y., Fung, P., Nishimura, N., Jensen, D.R., Fujii, H., Zhao, Y., Lumba, S., Santiago, J., Rodrigues, A., Chow, T.-F., et al. (2009). Abscisic acid inhibits type 2C protein phosphatases via the PYR/PYL family of START proteins. Science *324*, 1068–1071.

Paul, M.J., Gonzalez-Uriarte, A., Griffiths, C.A., and Hassani-Pak, K. (2018). The role of trehalose 6-phosphate in crop yield and resilience. Plant Physiol. *177*, 12–23.

Pedrotti, L., Weiste, C., Nägele, T., Wolf, E., Lorenzin, F., Dietrich, K., Mair, A., Weckwerth, W., Teige, M., Baena-González, E., and Dröge-Laser, W. (2018). Snf1-RELATED KINASE1-controlled C/S1-bZIP signaling activates alternative mitochondrial metabolic pathways to ensure plant survival in extended darkness. Plant Cell *30*, 495–509.

Peres, A., Churchman, M.L., Hariharan, S., Himanen, K., Verkest, A., Vandepoele, K., Magyar, Z., Hatzfeld, Y., Van Der Schueren, E., Beemster, G.T., et al. (2007). Novel plant-specific cyclin-dependent kinase inhibitors induced by biotic and abiotic stresses. J. Biol. Chem. *282*, 25588–25596.

Pfeiffer, A., Janocha, D., Dong, Y., Medzihradszky, A., Schöne, S., Daum, G., Suzaki, T., Forner, J., Langenecker, T., Rempel, E., et al. (2016). Integration of light and metabolic signals for stem cell activation at the shoot apical meristem. eLife *5*, e17023.

Pinheiro, C., and Chaves, M.M. (2011). Photosynthesis and drought: can we make metabolic connections from available data? J. Exp. Bot. *62*, 869–882.

Planes, M.D., Niñoles, R., Rubio, L., Bissoli, G., Bueso, E., García-Sánchez, M.J., Alejandro, S., Gonzalez-Guzmán, M., Hedrich, R., Rodriguez, P.L., et al. (2015). A mechanism of growth inhibition by abscisic acid in germinating seeds of Arabidopsis thaliana based on inhibition of plasma membrane H+-ATPase and decreased cytosolic pH, K+, and anions. J. Exp. Bot. *66*, 813–825.

Pu, Y., Luo, X., and Bassham, D.C. (2017). TOR-dependent and -independent pathways regulate autophagy in Arabidopsis thaliana. Front. Plant Sci. *8*, 1204.

Radchuk, R., Emery, R.J., Weier, D., Vigeolas, H., Geigenberger, P., Lunn, J.E., Feil, R., Weschke, W., and Weber, H. (2010). Sucrose non-fermenting kinase 1 (SnRK1) coordinates metabolic and hormonal signals during pea cotyledon growth and differentiation. Plant J. *61*, 324–338.

Radchuk, R., Radchuk, V., Weschke, W., Borisjuk, L., and Weber, H. (2006). Repressing the expression of the sucrose NONFERMENTING-1-RELATED PROTEIN kinase gene in pea embryo causes pleiotropic defects of maturation similar to an abscisic acid-insensitive phenotype. Plant Physiol. *140*, 263–278.

Ramon, M., Dang, T.V.T., Broeckx, T., Hulsmans, S., Crepin, N., Sheen, J., and Rolland, F. (2019). Default activation and nuclear translocation of the plant cellular energy sensor SnRK1 regulate metabolic stress responses and development. Plant Cell *31*, 1614–1632.

Ren, M., Qiu, S., Venglat, P., Xiang, D., Feng, L., Selvaraj, G., and Datla, R. (2011). Target of rapamycin regulates development and ribosomal RNA expression through kinase domain in Arabidopsis. Plant Physiol. *155*, 1367–1382.

Ren, M., Venglat, P., Qiu, S., Feng, L., Cao, Y., Wang, E., Xiang, D., Wang, J., Alexander, D., Chalivendra, S., et al. (2012). Target of rapamycin signaling regulates metabolism, growth, and life span in Arabidopsis. Plant Cell *24*, 4850–4874.

Richards, R.A., Rebetzke, G.J., Watt, M., Condon, A.G., Spielmeyer, W., and Dolferus, R. (2010). Breeding for improved water productivity in temperate cereals: phenotyping, Quantitative trait loci, markers and the selection environment. Functional Plant Biol. *37*, 85–97.

Rivero, R.M., Kojima, M., Gepstein, A., Sakakibara, H., Mittler, R., Gepstein, S., and Blumwald, E. (2007). Delayed leaf senescence induces extreme drought tolerance in a flowering plant. Proc. Natl. Acad. Sci. USA *104*, 19631–19636.

Rodrigues, A., Adamo, M., Crozet, P., Margalha, L., Confraria, A., Martinho, C., Elias, A., Rabissi, A., Lumbreras, V., González-Guzmán, M., et al. (2013). ABI1 and PP2CA phosphatases are negative regulators of Snf1-related protein kinase1 signaling in Arabidopsis. Plant Cell *25*, 3871–3884.

Rodriguez, M., Parola, R., Andreola, S., Pereyra, C., and Martínez-Noël, G. (2019). TOR and SnRK1 signaling pathways in plant response to abiotic stresses: do they always act according to the "yin-yang" model? Plant Sci. *288*, 110220.

Roustan, V., Jain, A., Teige, M., Ebersberger, I., and Weckwerth, W. (2016). An evolutionary perspective of AMPK-TOR signaling in the three domains of life. J. Exp. Bot. *67*, 3897–3907.

Salem, M.A., Li, Y., Bajdzienko, K., Fisahn, J., Watanabe, M., Hoefgen, R., Schöttler, M.A., and Giavalisco, P. (2018). RAPTOR controls developmental growth transitions by altering the hormonal and metabolic balance. Plant Physiol. *177*, 565–593.

Sato, A., Sato, Y., Fukao, Y., Fujiwara, M., Umezawa, T., Shinozaki, K., Hibi, T., Taniguchi, M., Miyake, H., Goto, D.B., and Uozumi, N. (2009). Threonine at position 306 of the KAT1 potassium channel is essential for channel activity and is a target site for ABA-activated SnRK2/OST1/SnRK2.6 protein kinase. Biochem. J. *424*, 439–448.

Schepetilnikov, M., Dimitrova, M., Mancera-Martínez, E., Geldreich, A., Keller, M., and Ryabova, L.A. (2013). TOR and S6K1 promote translation reinitiation of uORF-containing mRNAs via phosphorylation of eIF3h. EMBO J. *32*, 1087–1102.

Shen, W., Reyes, M.I., and Hanley-Bowdoin, L. (2009). Arabidopsis protein kinases GRIK1 and GRIK2 specifically activate SnRK1 by phosphorylating its activation loop. Plant Physiol. *150*, 996–1005.

Shi, H., Jiang, C., Ye, T., Tan, D.X., Reiter, R.J., Zhang, H., Liu, R., and Chan, Z. (2015). Comparative physiological, metabolomic, and transcriptomic analyses reveal mechanisms of improved abiotic stress resistance in Bermudagrass by exogenous melatonin. J. Exp. Bot. *66*, 681–694.

Shih, H.W., Miller, N.D., Dai, C., Spalding, E.P., and Monshausen, G.B. (2014). The receptor-like kinase FERONIA is required for mechanical signal transduction in Arabidopsis seedlings. Curr. Biol. *24*, 1887–1892.

Shin, R., Alvarez, S., Burch, A.Y., Jez, J.M., and Schachtman, D.P. (2007). Phosphoproteomic identification of targets of the Arabidopsis sucrose

nonfermenting-like kinase SnRK2.8 reveals a connection to metabolic processes. Proc. Natl. Acad. Sci. USA *104*, 6460–6465.

Shinozaki, K., Uemura, M., Bailey-Serres, J., Bray, E.A., and Weretilnyk, E. (2015). Responses to abiotic stress. In Biochemistry and Molecular Biology of Plants, B.B. Buchanan, W. Gruissem, and R.L. Jones, eds. (Willey), pp. 1051–1100.

Shulaev, V., Cortes, D., Miller, G., and Mittler, R. (2008). Metabolomics for plant stress response. Physiol. Plant. *132*, 199–208.

Sirichandra, C., Gu, D., Hu, H.C., Davanture, M., Lee, S., Djaoui, M., Valot, B., Zivy, M., Leung, J., Merlot, S., and Kwak, J.M. (2009). Phosphorylation of the Arabidopsis AtrbohF NADPH oxidase by OST1 protein kinase. FEBS Lett. *583*, 2982–2986.

Skirycz, A., Claeys, H., De Bodt, S., Oikawa, A., Shinoda, S., Andriankaja, M., Maleux, K., Eloy, N.B., Coppens, F., Yoo, S.-D., et al. (2011a). Pause-and-stop: the effects of osmotic stress on cell proliferation during early leaf development in Arabidopsis and a role for ethylene signaling in cell cycle arrest. Plant Cell *23*, 1876–1888.

Skirycz, A., Vandenbroucke, K., Clauw, P., Maleux, K., De Meyer, B., Dhondt, S., Pucci, A., Gonzalez, N., Hoeberichts, F., Tognetti, V.B., et al. (2011b). Survival and growth of Arabidopsis plants given limited water are not equal. Nat. Biotechnol. *29*, 212–214.

Soma, F., Mogami, J., Yoshida, T., Abekura, M., Takahashi, F., Kidokoro, S., Mizoi, J., Shinozaki, K., and Yamaguchi-Shinozaki, K. (2017). ABA-unresponsive SnRK2 protein kinases regulate mRNA decay under osmotic stress in plants. Nat. Plants *3*, 16204.

Soma, F., Takahashi, F., Suzuki, T., Shinozaki, K., and Yamaguchi-Shinozaki, K. (2020). Plant Raf-like kinases regulate the mRNA population upstream of ABA-unresponsive SnRK2 kinases under drought stress. Nat. Commun. *11*, 1373.

Song, L., Huang, S.C., Wise, A., Castanon, R., Nery, J.R., Chen, H., Watanabe, M., Thomas, J., Bar-Joseph, Z., and Ecker, J.R. (2016). A transcription factor hierarchy defines an environmental stress response network. Science *354*, aag1550.

Soto-Burgos, J., and Bassham, D.C. (2017). SnRK1 activates autophagy via the TOR signaling pathway in Arabidopsis thaliana. PLoS One *12*, e0182591.

Stegmann, M., Monaghan, J., Smakowska-Luzan, E., Rovenich, H., Lehner, A., Holton, N., Belkhadir, Y., and Zipfel, C. (2017). The receptor kinase FER is a RALF-regulated scaffold controlling plant immune signaling. Science *355*, 287–289.

Stone, S.L. (2019). Role of the ubiquitin proteasome system in plant response to abiotic stress. Int. Rev. Cell Mol. Biol. *343*, 65–110.

Takahashi, Y., Zhang, J., Hsu, P.K., Ceciliato, P.H.O., Zhang, L., Dubeaux, G., Munemasa, S., Ge, C., Zhao, Y., Hauser, F., and Schroeder, J.I. (2020). MAP3-kinase-dependent SnRK2-kinase activation is required for abscisic acid signal transduction and rapid osmotic stress response. Nat. Commun. *11*, 12.

Tang, Y., Liu, H., Guo, S., Wang, B., Li, Z., Chong, K., and Xu, Y. (2018). OsmiR396d affects gibberellin and brassinosteroid signaling to regulate plant architecture in rice. Plant Physiol. *176*, 946–959.

Tardieu, F. (2012). Any trait or trait-related allele can confer drought tolerance: just design the right drought scenario. J. Exp. Bot. *63*, 25–31.

Testerink, C., and Munnik, T. (2011). Molecular, cellular, and physiological responses to phosphatidic acid formation in plants. J. Exp. Bot. *62*, 2349–2361.

Tikkanen, M., Mekala, N.R., and Aro, E.M. (2014). Photosystem II photoinhibition-repair cycle protects photosystem I from irreversible damage. Biochim. Biophys. Acta *1837*, 210–215.

Toroser, D., Plaut, Z., and Huber, S.C. (2000). Regulation of a plant SNF1-related protein kinase by glucose-6-phosphate. Plant Physiol. *123*, 403–412.

Tsai, A.Y., and Gazzarrini, S. (2012). AKIN10 and FUSCA3 interact to control lateral organ development and phase transitions in Arabidopsis. Plant J. *69*, 809–821.

Umezawa, T., Sugiyama, N., Takahashi, F., Anderson, J.C., Ishihama, Y., Peck, S.C., and Shinozaki, K. (2013). Genetics and phosphoproteomics reveal a protein phosphorylation network in the abscisic acid signaling pathway in Arabidopsis thaliana. Sci. Signal. *6*, rs8.

van Dam, T.J., Zwartkruis, F.J., Bos, J.L., and Snel, B. (2011). Evolution of the TOR pathway. J. Mol. Evol. *73*, 209–220.

Van Leene, J., Han, C., Gadeyne, A., Eeckhout, D., Matthijs, C., Cannoot, B., De Winne, N., Persiau, G., Van De Slijke, E., Van de Cotte, B., et al. (2019). Capturing the phosphorylation and protein interaction landscape of the plant TOR kinase. Nat. Plants *5*, 316–327.

Vij, S., and Tyagi, A.K. (2007). Emerging trends in the functional genomics of the abiotic stress response in crop plants. Plant Biotechnol. J. *5*, 361–380.

Wang, G., Kong, H., Sun, Y., Zhang, X., Zhang, W., Altman, N., DePamphilis, C.W., and Ma, H. (2004). Genome-wide analysis of the cyclin family in Arabidopsis and comparative phylogenetic analysis of plant cyclin-like proteins. Plant Physiol. *135*, 1084–1099.

Wang, H., Qi, Q., Schorr, P., Cutler, A.J., Crosby, W.L., and Fowke, L.C. (1998). ICK1, a cyclin-dependent protein kinase inhibitor from Arabidopsis thaliana interacts with both Cdc2a and CycD3, and its expression is induced by abscisic acid. Plant J. *15*, 501–510.

Wang, L., Li, H., Zhao, C., Li, S., Kong, L., Wu, W., Kong, W., Liu, Y., Wei, Y., Zhu, J.-K., and Zhang, H. (2017). The inhibition of protein translation mediated by AtGCN1 is essential for cold tolerance in Arabidopsis thaliana. Plant Cell Environ. *40*, 56–68.

Wang, P., Xue, L., Batelli, G., Lee, S., Hou, Y.J., Van Oosten, M.J., Zhang, H., Tao, W.A., and Zhu, J.K. (2013). Quantitative phosphoproteomics identifies SnRK2 protein kinase substrates and reveals the effectors of abscisic acid action. Proc. Natl. Acad. Sci. USA *110*, 11205–11210.

Wang, P., Zhao, Y., Li, Z., Hsu, C.-C., Liu, X., Fu, L., Hou, Y.J., Du, Y., Xie, S., Zhang, C., et al. (2018a). Reciprocal regulation of the TOR kinase and ABA receptor balances plant growth and stress response. Mol. Cell *69*, 100–112.e6.

Wang, Q., Yu, F., and Xie, Q. (2020). Balancing growth and adaptation to stress: crosstalk between brassinosteroid and abscisic acid signaling. Plant Cell Environ. *43*, 2325–2335.

Wang, Y., Reiter, R.J., and Chan, Z. (2018b). Phytomelatonin: a universal abiotic stress regulator. J. Exp. Bot. *69*, 963–974.

Waszczak, C., Carmody, M., and Kangasjärvi, J. (2018). Reactive oxygen species in plant signaling. Annu. Rev. Plant Biol. *69*, 209–236.

Weltmeier, F., Ehlert, A., Mayer, C.S., Dietrich, K., Wang, X., Schütze, K., Alonso, R., Harter, K., Vicente-Carbajosa, J., and Dröge-Laser, W. (2006). Combinatorial control of Arabidopsis proline dehydrogenase transcription by specific heterodimerisation of bZIP transcription factors. EMBO J. *25*, 3133–3143.

West, G., Inzé, D., and Beemster, G.T. (2004). Cell cycle modulation in the response of the primary root of Arabidopsis to salt stress. Plant Physiol. *135*, 1050–1058.

Wiese, A., Elzinga, N., Wobbes, B., and Smeekens, S. (2004). A conserved upstream open reading frame mediates sucrose-induced repression of translation. Plant Cell *16*, 1717–1729.

Wildon, D.C., Thain, J.F., Minchin, P.E.H., Gubb, I.R., Reilly, A.J., Skipper, Y.D., Doherty, H.M., O'Donnell, P.J., and Bowles, D.J. (1992). Electrical signalling and systemic proteinase inhibitor induction in the wounded plant. Nature *360*, 62–65.

Xie, Z., Nolan, T., Jiang, H., Tang, B., Zhang, M., Li, Z., and Yin, Y. (2019). The AP2/ERF transcription factor TINY modulates brassinosteroid-regulated plant growth and drought responses in Arabidopsis. Plant Cell *31*, 1788–1806.

Xiong, Y., McCormack, M., Li, L., Hall, Q., Xiang, C., and Sheen, J. (2013). Glucose-TOR signalling reprograms the transcriptome and activates meristems. Nature *496*, 181–186.

Xu, K., Xu, X., Fukao, T., Canlas, P., Maghirang-Rodriguez, R., Heuer, S., Ismail, A.M., Bailey-Serres, J., Ronald, P.C., and Mackill, D.J. (2006). Sub1A is an ethylene-response-factor-like gene that confers submergence tolerance to rice. Nature *442*, 705–708.

Yan, J., Niu, F., Liu, W.Z., Zhang, H., Wang, B., Lan, W., Che, Y., Yang, B., Luan, S., and Jiang, Y.Q. (2014). Arabidopsis CIPK14 positively regulates glucose response. Biochem. Biophys. Res. Commun. *450*, 1679–1683.

Yang, F.X., and Yu, D. (2010). Overexpression of Arabidopsis MiR396 enhances drought tolerance in transgenic tobacco plants. Acta Bot. Yunnanica *31*, 421–426.

Yi, D., Alvim Kamei, C.L., Cools, T., Vanderauwera, S., Takahashi, N., Okushima, Y., Eekhout, T., Yoshiyama, K.O., Larkin, J., Van den Daele, H., et al. (2014). The Arabidopsis SIAMESE-RELATED cyclin-dependent kinase inhibitors SMR5 and SMR7 regulate the DNA damage checkpoint in response to reactive oxygen species. Plant Cell *26*, 296–309.

Yoshida, R., Umezawa, T., Mizoguchi, T., Takahashi, S., Takahashi, F., and Shinozaki, K. (2006). The regulatory domain of SRK2E/OST1/SnRK2.6 interacts with ABI1 and integrates abscisic acid (ABA) and osmotic stress signals controlling stomatal closure in Arabidopsis. J. Biol. Chem. *281*, 5310–5318.

Yu, F., Qian, L., Nibau, C., Duan, Q., Kita, D., Levasseur, K., Li, X., Lu, C., Li, H., Hou, C., et al. (2012). FERONIA receptor kinase pathway suppresses abscisic acid signaling in Arabidopsis by activating ABI2 phosphatase. Proc. Natl. Acad. Sci. USA *109*, 14693–14698.

Yu, S.M., Lo, S.F., and Ho, T.D. (2015). Source-sink communication: regulated by hormone, nutrient, and stress cross-signaling. Trends Plant Sci. *20*, 844–857.

Yuan, F., Yang, H., Xue, Y., Kong, D., Ye, R., Li, C., Zhang, J., Theprungsirikul, L., Shrift, T., Krichilsky, B., et al. (2014). OSCA1 mediates osmotic-stress-evoked Ca2+ increases vital for osmosensing in Arabidopsis. Nature *514*, 367–371.

Yuan, S., Zhao, J., Li, Z., Hu, Q., Yuan, N., Zhou, M., Xia, X., Noorai, R., Saski, C., Li, S., and Luo, H. (2019). MicroRNA396-mediated alteration in plant development and salinity stress response in creeping bentgrass. Hortic. Res. *6*, 48.

Zhai, Z., Keereetaweep, J., Liu, H., Feil, R., Lunn, J.E., and Shanklin, J. (2018). Trehalose 6-phosphate positively regulates fatty acid synthesis by stabilizing WRINKLED1. Plant Cell *30*, 2616–2627.

Zhang, D., Sun, W., Singh, R., Zheng, Y., Cao, Z., Li, M., Lunde, C., Hake, S., and Zhang, Z. (2018a). GRF-interacting factor1 regulates shoot architecture and meristem determinacy in maize. Plant Cell *30*, 360–374.

Zhang, H., Li, Y., and Zhu, J.K. (2018b). Developing naturally stress-resistant crops for a sustainable agriculture. Nat. Plants *4*, 989–996.

Zhang, Y., Andralojc, P.J., Hey, S.J., Primavesi, L.F., Specht, M., Koehler, J., Parry, M.A.J., and Halford, N.G. (2008). Arabidopsis sucrose non-fermenting-1-related protein kinase-1 and calcium-dependent protein kinase phosphorylate conserved target sites in ABA response element binding proteins. Ann. Appl. Biol. *153*, 401–409.

Zhang, Y., Primavesi, L.F., Jhurreea, D., Andralojc, P.J., Mitchell, R.A., Powers, S.J., Schluepmann, H., Delatte, T., Wingler, A., and Paul, M.J. (2009a). Inhibition of SNF1-related protein kinase1 activity and regulation of metabolic pathways by trehalose-6-phosphate. Plant Physiol. *149*, 1860–1871.

Zhang, Y., Zhu, H., Zhang, Q., Li, M., Yan, M., Wang, R., Wang, L., Welti, R., Zhang, W., and Wang, X. (2009b). Phospholipase dalpha1 and phosphatidic acid regulate NADPH oxidase activity and production of reactive oxygen species in ABA-mediated stomatal closure in Arabidopsis. Plant Cell *21*, 2357–2377.

Zhao, C., Wang, P., Si, T., Hsu, C.C., Wang, L., Zayed, O., Yu, Z., Zhu, Y., Dong, J., Tao, W.A., and Zhu, J.-K. (2017). MAP kinase cascades regulate the cold response by modulating ICE1 protein stability. Dev. Cell *43*, 618–629.e5.

Zhao, C., Zayed, O., Yu, Z., Jiang, W., Zhu, P., Hsu, C.C., Zhang, L., Tao, W.A., Lozano-Durán, R., and Zhu, J.K. (2018). Leucine-rich repeat extensin proteins regulate plant salt tolerance in Arabidopsis. Proc. Natl. Acad. Sci. USA *115*, 13123–13128.

Zhao, Y., Xing, L., Wang, X., Hou, Y.J., Gao, J., Wang, P., Duan, C.G., Zhu, X., and Zhu, J.K. (2014). The ABA receptor PYL8 promotes lateral root growth by enhancing MYB77-dependent transcription of auxin-responsive genes. Sci. Signal. *7*, ra53.

Zheng, Z., Xu, X., Crosley, R.A., Greenwalt, S.A., Sun, Y., Blakeslee, B., Wang, L., Ni, W., Sopko, M.S., Yao, C., et al. (2010). The protein kinase SnRK2.6 mediates the regulation of sucrose metabolism and plant growth in Arabidopsis. Plant Physiol. *153*, 99–113.

Zhu, J.K. (2002). Salt and drought stress signal transduction in plants. Annu. Rev. Plant Biol. *53*, 247–273.

Zhu, J.K. (2016). Abiotic stress signaling and responses in plants. Cell *167*, 313–324.

Zurbriggen, M.D., Hajirezaei, M.R., and Carrillo, N. (2010). Engineering the future. Development of transgenic plants with enhanced tolerance to adverse environments. Biotechnol. Genet. Eng. Rev. *27*, 33–56.