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PIN-mediated polar auxin transport regulations in plant tropic responses

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Summary

Tropisms, growth responses to environmental stimuli such as light or gravity, are spectacular examples of adaptive plant development. The plant hormone auxin serves as a major coordinative signal. The PIN auxin exporters, through their dynamic polar subcellular localizations, redirect auxin fluxes in response to environmental stimuli and the resulting auxin gradients across organs underlie differential cell elongation and bending. In this review, we discuss recent advances concerning regulations of PIN polarity during tropisms, focusing on PIN phosphorylation and trafficking. We also cover how environmental cues regulate PIN actions during tropisms, as well as the crucial role of auxin feedback on PIN polarity during bending termination. Finally, the interactions between different tropisms are reviewed to understand plant adaptive growth in the natural environment.

Introduction

Unlike in animals, plant locomotion in response to environmental stimuli is limited. Instead, plants have evolved a robust system of tropic responses to quickly adapt their growth in the face of changing local environment. Tropisms are directional growth adaptations to light (phototropism), gravity (gravitropism), obstacles (obstacle avoidance), salinity (halotropism) and water (hydrotropism). Phototropism enables plants to react to changes in the direction of light by bending shoots towards the light source, or roots away from it (reviewed in de Wit *et al.*, 2016; Harmer & Brooks, 2018). Gravitropism orients plant growth direction along the gravity vector, so that roots grow downwards, and shoots grow upwards, to position the plant body correctly and to obtain resources for development (reviewed in Rakusová *et al.*, 2015). Obstacle avoidance helps plant roots to realign their growth when encountering obstacles in soil (Lee *et al.*, 2019). Halotropism and hydrotropism enable plants to sense salinity and water potential in the local environment and direct root growth accordingly (reviewed in Dietrich, 2018; Harmer & Brooks, 2018).

In most tropic responses, redirection of the cell-to-cell directional auxin movement in response to external cues is the main mechanism for generating asymmetric auxin distribution and differential cell elongation. It has been well established that the directional auxin flow in plant tissues is achieved via polarly localized PIN auxin exporters (Petrášek *et al.*, 2006; Glanc *et al.*, 2018). Influx carriers, AUXIN-RESISTANT1/LIKE AUX1 (AUX1/LAX) (Band *et al.*, 2014), and some members of the B subclass of ATP-BINDING CASSETTE (ABCB) efflux carriers also contribute to polar auxin flow (reviewed in Geisler & Murphy, 2006). A number of studies support the notion that PIN-mediated directional auxin flow is crucial for plant phototropism (Friml *et al.*, 2002; Ding *et al.*, 2011), root and shoot gravitropism (Friml *et al.*, 2002; Kleine-Vehn *et al.*, 2010; Rakusová *et al.*, 2011; Baster *et al.*, 2013), obstacle avoidance (Lee *et al.*, 2019), halotropism (Galvan-Ampudia *et al.*, 2013), and possibly also for hydrotropism (Nakajima *et al.*, 2017).

Here we give an overview of PIN-mediated polar auxin transport in phototropism, gravitropism, obstacle avoidance, halotropism and hydrotropism (Fig. 1). We also discuss the underlying molecular mechanisms of PIN polarity switches in these tropic responses. Furthermore, we summarize how plants transmit environmental signals into PIN polarity switches in tropisms, as well as the interactions between distinct tropic responses under natural conditions.

PIN-mediated directional auxin fluxes during tropic responses

Phototropism

Light is an essential environmental resource for plant development. Plants can detect light quality, intensity and direction, which results in cellular responses to optimize growth and survival (reviewed in Chen *et al.*, 2004). Light-oriented shoot bending was first observed in dark-grown seedlings of oat (*Avena sativa*) and canary grass (*Phalaris canariensis*), and this phenomenon was termed phototropism (Darwin, 1880). Early experiments indicated that, during phototropism, an unknown signal moves from the irradiated side of the seedling to the shaded side, triggering differential growth and thus bending (Darwin, 1880; Went, 1928). Later, the plant hormone auxin was identified as the mobile signal responsible for phototropism (Kogl & Haagen-Smits, 1931). Together with this early work, the Cholodny–Went hypothesis, a hallmark of which is asymmetric auxin distribution, was formulated to explain phototropic responses in plants (reviewed in Christie & Murphy, 2013). Several studies have confirmed that lateral auxin transport occurs during phototropism; however, the underlying mechanism remained unknown for a long time (Briggs *et al.*, 1957; Briggs, 1963; Pickard & Thimann, 1963).

It was later suggested that auxin exporters of the PIN family mediate polar auxin transport during phototropism (Fig. 1a; Friml *et al.*, 2002; Ding *et al.*, 2011). Among PINs, PIN3 is the major mediator of lateral auxin flow during phototropism (Ding *et al.*, 2011). By default, PIN3 displays apolar localization in endodermal cells of etiolated hypocotyls. However, following unilateral blue light stimulation, PIN3 gradually polarizes to and is stabilized at the endodermal cell sides away from the light source (Ding *et al.*, 2011). This polarized PIN3 distribution would (re)direct auxin flow to the shaded side of the hypocotyl, where auxin accumulation can be visualized by auxin response reporter (Ding *et al.*, 2011). This provides a possible mechanism for translating external light direction into internal auxin flow to generate auxin asymmetry driving bending towards light. Nonetheless, whether there is a causal link between PIN3 polarization and bending remains unclear, mainly because pronounced PIN3 polarization is observed with too much of a delay compared with the phototropic growth response.

The blue light receptor phototropins (PHOT1 and PHOT2) play a key role in phototropism. PHOT1 senses both low- and high-intensity blue light, whereas PHOT2 is more specific towards high light intensities (Sakai *et al.*, 2001; Inada *et al.*, 2004). Following blue light perception, the signal transducer NON-PHOTOTROPIC HYPOCOTYL (NPH3) is dephosphorylated in a PHOT1-dependent manner, resulting in release of NPH3 from the plasma membrane to the cytosol (Haga *et al.*, 2015). When light intensity increases, PHOT2 mediates the relocation or stabilization of NPH3 at plasma membrane (Zhao *et al.*, 2018). In addition, the transcription of *ROOT PHOTOTROPISM 2* (*RPT2*) is increased at high blue light intensities. RPT2 binds to the oxygen or voltage sensing 1 (LOV1) domain of PHOT1 to suppress PHOT1 autophosphorylation activity, resulting in altered NPH3 phosphorylation and phototropic response (Haga *et al.*, 2015; Kimura *et al.*, 2020). PHOT1 and PHOT2 therefore mediate plant phototropism in response to different blue light intensities via regulation of NPH3 dynamics. However, how light intensity influences PIN polarization remains unknown.

Whereas shoots grow towards blue light, roots exhibit a negative phototropic response (Fig. 1a; reviewed in Kutschera & Briggs, 2012). Blue light-mediated reorientation of root growth requires a local response in the transition zone of the root meristem and utilizes auxin exporters in the root tip to establish asymmetric auxin accumulation (Wan *et al.*, 2012; Zhang *et al.*, 2013). At the illuminated side of the root, light inhibits PIN2 vacuolar degradation, and hence PIN2 accumulates at the plasma membrane; however, at the shaded side of the root, PIN2 disappears from the plasma membrane through vacuole-targeted degradation (Kleine-Vehn *et al.*, 2008a). PIN2 is therefore distributed differentially at the plasma membranes on the irradiated and shaded sides of the root, which leads to asymmetric auxin distribution across the root (Wan *et al.*, 2012). Furthermore, PIN3 is, by default, apolar in the root columella cells at the root tip; however, following blue light illumination, it polarizes to the lateral membranes towards the light source (Zhang *et al.*, 2013). As a consequence, auxin accumulates at the illuminated root side to promote growth, and roots grow away from the light (Fig. 1a). These observations from the root are unexpected given that PIN3 polarizes there towards the light source (not observed in shoots) and auxin accumulation promotes growth during phototropism (whereas growth is inhibited during root gravitropism). Thus, it is possible that light impacts on PIN polarization or PIN abundance at the plasma membrane to control auxin movement during negative root phototropism but the mechanism requires more clarification.

In addition to the key role of blue light in phototropism, red light and UV-B illumination also trigger phototropic responses in shoots (Goyal *et al.*, 2016; Vanhaelewyn *et al.*, 2019). In de-etiolated *Arabidopsis* seedlings, the red/far red (R/FR) light receptor, phytochrome B (phyB), regulates phototropism by modulating auxin biosynthesis and transport (Goyal *et al.*, 2016). UV-B was also observed to induce a phototropic response in *Arabidopsis* stems via the UVR8 receptor, also involving auxin transport and response (Vanhaelewyn *et al.*, 2019). Red light receptors, phytochromes, and PHYTOCHROME KINASE SUBSTRATE (PKS1) have also been linked to a positive root phototropic response (Ruppel *et al.*, 2001; Kiss *et al.*, 2003; Molas & Kiss, 2008; Vandenbrink *et al.*, 2016). However, the link between red light and PIN polarization in root remains an open question.

Gravitropism

Similar to light, gravity modulates plant growth, causing roots and shoots to grow downwards and upwards, respectively. The asymmetric auxin distribution between the opposite sides of a gravistimulated shoot or root is again achieved via differential subcellular PIN distribution, resulting in organ bending (Fig. 1b). The strong agravitropic phenotype of *pin2* roots indicates that PIN2 is the main player mediating auxin transport in root gravitropism, transporting auxin from the root tip where gravity is

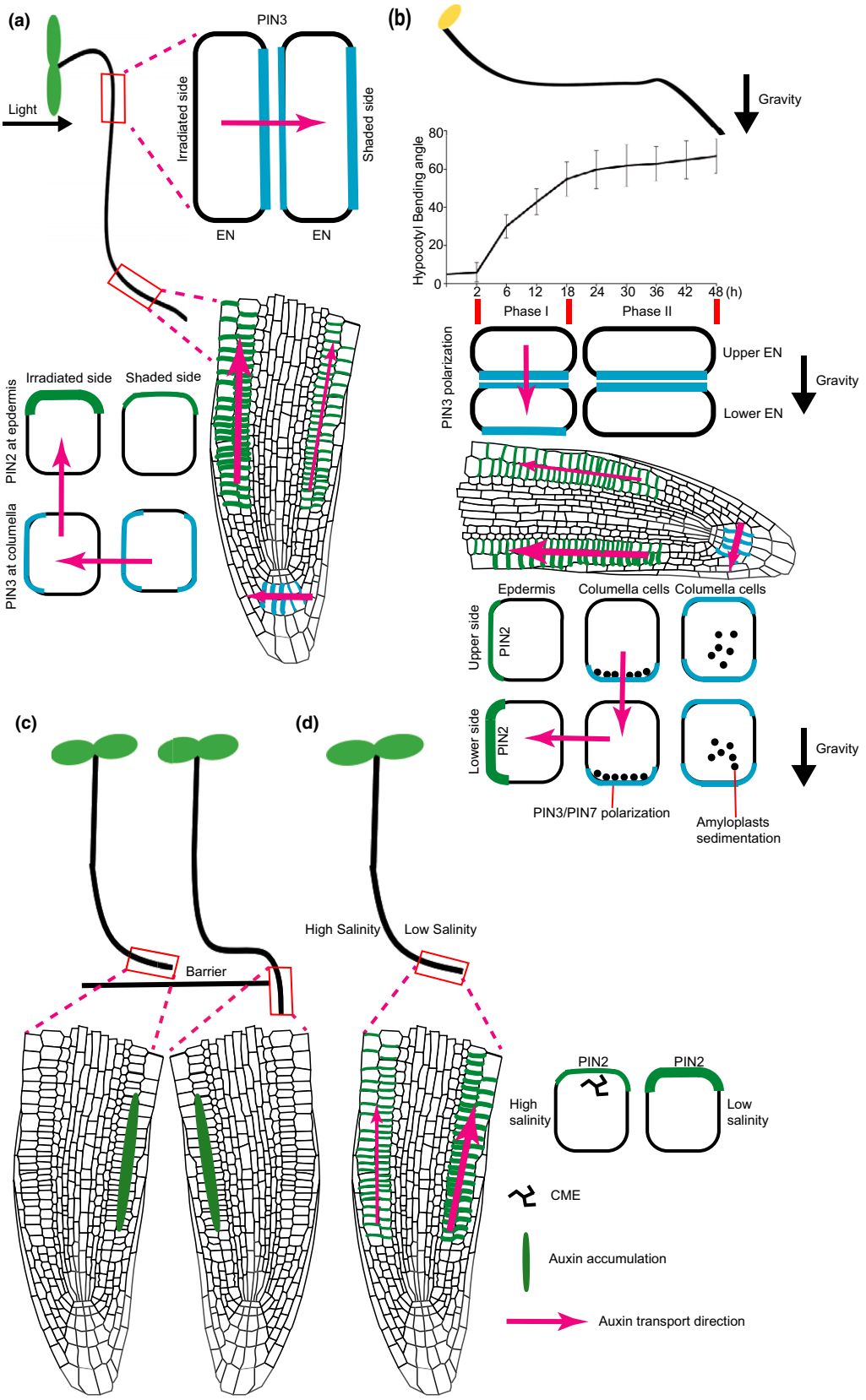

Fig. 1 PIN polarization-dependent polar auxin fluxes during tropisms. (a) Phototropism in shoots and roots. In shoots, light triggers PIN3 (in blue) polarization to the shaded side of the hypocotyl, thus driving auxin movements to promote hypocotyl growth at the shaded side; shoots then bend towards the light source. In primary roots, light inhibits vacuole-targeted PIN2 degradation at the irradiated side of the root to generate differential PIN2 abundance at the plasma membrane and thus auxin transport capacity between irradiated and shaded sides of the root. On the other hand, PIN3 polarizes to the irradiated side of primary roots. Consequently, auxin accumulates at the irradiated side of the roots to promote growth and roots bend away from the light (Wan *et al*., 2012; Zhang *et al*., 2013). Note that in this model, contrary to all other cases of tropism, auxin accumulation promotes growth in negative tropic responses of the root. EN, endodermal cells. (b) Gravitropism in shoots and primary roots. In shoots, hypocotyl bending kinetics highly correlate with two distinct PIN polarization events (bending data adapted from Rakusová *et al*., 2019; error bars are SE). In the early phase (I), gravity induces PIN3 polarization to the lower side of the endodermal cells to mediate auxin transport to the lower side of the hypocotyls where auxin accumulation promotes growth and the shoot bends upward. In the late phase (II), the higher auxin concentrations at the lower hypocotyl side, by a feedback regulation, promote PIN3 polarization to the inner side of endodermal cells, thus re-establishing symmetry of PIN3 localization and auxin distribution to terminate bending. In gravistimulated primary roots, amyloplasts sediment to the bottom side of columella cells, and then PIN3 and PIN7 polarize to the bottom side of the columella cells, redirecting auxin movement downwards. In parallel, PIN2 at the lower root side changes its abundance in epidermis, thus reinforcing the initial auxin asymmetry with more auxin accumulating at the lower root side where it inhibits growth, leading to downward bending. (c) Asymmetric auxin distribution during obstacle avoidance. When roots contact with the barrier (first bending occurs), roots bend away from the barrier, and keep growing in parallel to the end of the barrier. When roots reach the end of barrier (second bending occurs), they grow downwards. In these two bending events, auxin is asymmetrically distributed between the convex and concave sides of the root to guide growth and avoid the obstacle. (d) Halotropism in primary roots. When roots face a high-salinity area, high salinity initiates clathrin-mediated PIN2 internalization, resulting in a reduction of PIN2 abundance at the plasma membrane. Through this mechanism, auxin accumulates at the side that faces low-salinity conditions and the roots bend away from the high-salinity area. CME, clathrin-mediated endocytosis. The thicker pink arrow means more auxin is transported to this side.

perceived to the elongation zone, where growth is regulated (Luschnig *et al*., 1998; Baster *et al*., 2013; Zhang *et al*., 2019; Tan *et al*., 2020a). In addition to PIN2, the AUX1 auxin importer also mediates auxin transport to the elongation zone (Swarup *et al*., 2001). Following gravistimulation, the abundance of PIN2 in the epidermis of the upper root side decreases, while on the lower root side it increases. This asymmetric abundance of PIN2 then potentiates polar auxin transport to the root elongation zone, and roots grow downwards as a result (Baster *et al*., 2013). In addition, increased auxin concentrations at the lower root side promote expression of the small secretory peptide *GOLVEN* (*GLV*). Differential expression of *GLV* genes in epidermal and cortical cells exacerbates differential PIN2 distribution through regulation of its trafficking (Whitford *et al*., 2012). Similarly, asymmetric distribution of the phytohormone gibberellin during root gravitropism (Löfke *et al*., 2013) regulates PIN2 trafficking (Salanenka *et al*., 2018), thus aiding the auxin transport asymmetry between upper and lower root sides.

Root gravitropism also requires PIN3 and PIN7 activities (Fig. 1b). By default, both of these transporters are localized at the plasma membrane in columella cells (the site of gravity perception) in an apolar manner. Following gravistimulation, PIN3 and PIN7 polarize to the new lower sides of columella cells, thus driving auxin flow towards the lower side of the root tip (Friml *et al*., 2002; Kleine-Vehn *et al*., 2010). Gravity-induced PIN3 and PIN7 polarization in columella cells and asymmetric PIN2 degradation in epidermal cells would both therefore independently contribute to asymmetric auxin distribution with auxin accumulation at the lower root side where it inhibits growth (reviewed in Gallei *et al*., 2020), causing downward bending.

In shoots, PIN3 redirects auxin flow for shoot gravitropism (Fig. 1b; Rakusová *et al*., 2011). PIN3 exhibits an apolar distribution in hypocotyl endodermal cells. Following gravistimulation, PIN3 relocates to the lower side of the endodermal cells, presumably redirecting auxin flow to the lower hypocotyl side where auxin accumulates, promotes growth and thereby initiates hypocotyl bending. Interestingly, PIN4 and PIN7 contribute to polar auxin transport in the absence of PIN3 (Rakusová *et al*., 2011).

Lateral organs typically emerge from primary organs with a defined gravitropic setpoint angle (GSA), which is also controlled by auxin (Rosquete *et al*., 2013; Roychoudhry *et al*., 2013). In the early stages of lateral root emergence, PIN3 is expressed and symmetrically distributed at the plasma membrane of lateral root columella cells. Then, PIN3 quickly repolarizes to the bottom side to establish asymmetric auxin distribution and to enforce GSA. When lateral roots reach their GSA, the overall PIN expression level is decreased, and PIN proteins are symmetrically localized at the plasma membrane to maintain nondifferential growth along the GSA. Hence, PIN polarity and expression are temporally controlled to limit auxin fluxes in lateral root GSA. In shoots, a similar PIN polarization-dependent auxin flow is also suggested to control lateral shoot GSA, and the $SCF^{TIR1/AFB}$ auxin signaling is involved in this process (Roychoudhry *et al*., 2013). In addition, TILLER ANGLE CONTROL1 (TAC1) and LAZY1-LIKE (LZY) proteins contribute to the control of lateral shoot GSA, possibly also via regulation of PIN polarization and auxin transport (reviewed in Roychoudhry & Kepinski, 2015; Nakamura *et al*., 2019).

Thus, in gravitropism of both roots and shoots, PIN repolarization-dependent redirection of auxin fluxes appears to be the major mechanism of generating asymmetric auxin distribution to initiate bending.

Obstacle avoidance

Roots grow downwards to acquire water with nutrients and to anchor the plant in the soil. Upon encountering an obstacle, however, roots must reorient their growth (Fig. 1c; Massa & Gilroy, 2003; Lee *et al*., 2019; Zhang & Friml, 2020). A recent study showed that PIN-mediated polar auxin transport facilitates root bending away from obstacles (Lee *et al*., 2019). Asymmetric auxin distribution is detected between the concave and convex root sides which arise when a root is deformed by an obstacle. However, it is difficult to determine which PIN family member specifically

affects auxin flow during obstacle avoidance, even though the *pin2* mutant shows a clear bending defect as well as a defective auxin distribution (Lee *et al.*, 2019).

Halotropism

Apart from normal development, auxin is also involved in stress responses (reviewed in Blakeslee *et al.*, 2019). Salinity, a devastating abiotic stress, affects root growth (Liu *et al.*, 2015), gravitropism (Sun *et al.*, 2008) and auxin distribution (Fu *et al.*, 2019; Wang *et al.*, 2019). The phenomenon in which a root grows away from an area of high salt concentration is termed halotropism (Fig. 1d; Galvan-Ampudia *et al.*, 2013). Salinity induces internalization of PIN2 from the plasma membrane at the side of the root proximal to the area of high salt concentration, and therefore differential PIN2 distribution redirects auxin flow to the root side without salt. The mechanism behind differential PIN2 endocytic trafficking may be related to the osmotic regulation of exo- and endocytic trafficking (Zwiewka *et al.*, 2015). These events result in the root growing away from the high salt concentration area (Galvan-Ampudia *et al.*, 2013). Nevertheless, the roles in halotropism of other PINs, such as PIN1 and PIN3, as well as the role of the influx transporter AUX1, are somewhat elusive (Galvan-Ampudia *et al.*, 2013).

Hydrotropism

In addition to salinity of the local environment, water potential, in general, also influences root growth. Roots mediate water uptake from the soil and have developed a robust adaptive response called hydrotropism to sense different water potentials in the local environment and to direct their growth accordingly (reviewed in Dietrich, 2018; Harmer & Brooks, 2018). Pharmacological studies indicate that auxin transport is required for hydrotropism in some species, but not in *Arabidopsis thaliana* (Shkolnik *et al.*, 2016; Morohashi *et al.*, 2017; Nakajima *et al.*, 2017). In *Arabidopsis*, auxin transport inhibitors 2,3,5-triiodobenzoic acid (TIBA) and naphthylphthalamic acid (NPA) (Abas *et al.*, 2021) do not interfere with hydrotropism, and the auxin response reporters DII-Venus and DR5 remain unaffected during the root hydrotropic response (Shkolnik *et al.*, 2016). However, the putative cucumber (*Cucumis sativus*) PIN2 ortholog, *CsPIN5*, may direct auxin flow in hydrotropism (Morohashi *et al.*, 2017). While polar auxin transport is not necessary for root hydrotropism in *Arabidopsis*, the auxin response components are required (reviewed in Dietrich, 2018). Nevertheless, abscisic acid (ABA) has been shown to play an essential role in hydrotropism, which interestingly appears linked to the modulation of PIN2 expression and auxin transport (Xu *et al.*, 2013).

In conclusion, PINs switch their subcellular polarity or endocytic trafficking and degradation during various tropisms to achieve asymmetric auxin distribution across organs (Fig. 1). The auxin gradient ultimately leads to differential cell elongation in roots or shoots, resulting in their bending. However, in some cases, the involvement of polar auxin transport is inconclusive and requires further investigation.

Subcellular PIN trafficking and kinase-mediated PIN phosphorylation for PIN polarity switches in tropisms

PIN-mediated auxin transport is vital for tropic responses in plants, but how is the subcellular localization of PIN proteins controlled to establish auxin fluxes? Accumulating evidence supports subcellular PIN trafficking (reviewed in Adamowski & Friml, 2015; Narasimhan *et al.*, 2021) and kinase-mediated PIN phosphorylation (reviewed in Barbosa *et al.*, 2018; Tan *et al.*, 2021) as conserved mechanisms that enable rapid changes in PIN polarities and redirections of polar auxin movement during tropic responses.

PIN proteins undergo dynamic cycling between the plasma membrane and the endosomal compartments in a process termed endocytic recycling. The exocytotic step of endocytic recycling is inhibited by the fungal toxin brefeldin A (BFA) (Geldner *et al.*, 2001, 2003; reviewed in Friml, 2010). Brefeldin A inhibits some ADP-RIBOSYLATION FACTOR GUANINE-NUCLEOTIDE EXCHANGE FACTORS (ARF-GEFs), activators of small ARF GTPases that are essential for vesicle formation and other aspects of the endomembrane system function (reviewed in Singh & Jürgens, 2017). The ARF-GEF GNOM was identified as the target of the BFA-mediated inhibition of PIN trafficking (Geldner *et al.*, 2003). It has been observed that BFA treatments interfere with gravity- or light-mediated PIN polarization, resulting in defective bending. However, the BFA-resistant *GNOM*^{M696L} mutant shows a normal PIN polarization and bending response to gravity or light (Kleine-Vehn *et al.*, 2010; Ding *et al.*, 2011; Rakusová *et al.*, 2011). These observations indicate that GNOM-mediated PIN trafficking is vital to PIN polarity switches in tropisms. Interestingly, MIZ2/GNOM-mediated trafficking has also been reported to play an important role in root hydrotropism, albeit via a mechanism distinct from its role in polar auxin transport (Miyazawa *et al.*, 2009).

As part of endocytic recycling, PIN proteins are continuously internalized from the plasma membrane via clathrin-mediated endocytosis (Narasimhan *et al.*, 2020). The vesicular clathrin coat is composed of CLATHRIN HEAVY CHAIN (CHC) and CLATHRIN LIGHT CHAIN (CLC) proteins. The *chc* or *clc* mutants display strong defects in PIN trafficking, PIN polar localization, auxin distribution and tropic responses (Kitakura *et al.*, 2011; Wang *et al.*, 2013; Zhang *et al.*, 2017). The dominant-negative *chc1* mutant exhibits an agravitropic root and hypocotyl with altered PIN localization and auxin distribution (Kitakura *et al.*, 2011). The loss-of-function *clc2 clc3* double mutant exhibits a defective light-mediated PIN3 repolarization, as well as reduced phototropic bending (Zhang *et al.*, 2017). In addition, the *clc2 clc3* double mutant also shows a reduced root gravitropic bending and defective PIN2 trafficking (Wang *et al.*, 2013). Furthermore, clathrin-mediated PIN2 endocytosis is crucial for the asymmetric auxin distribution associated with halotropism (Galvan-Ampudia *et al.*, 2013).

With these observations in mind, it must be noted that clathrin coats are involved not only in endocytosis but also in the formation of vesicles that participate in other trafficking events, specifically in the vacuolar pathway and, potentially, in some forms of exocytosis (reviewed in Robinson & Pimpl, 2014). Furthermore, the ARF-

GEF GNOM is also found at the plasma membrane besides acting at the Golgi apparatus (Naramoto *et al.*, 2010, 2014). Thus, it is difficult to conclude precisely by which means these factors contribute to PIN polarity control. Nonetheless, PIN trafficking is clearly required for PIN relocation and the subsequent formation of auxin gradients during tropic responses.

Additional observations in support of the relocation of PIN proteins by means of endocytic recycling during tropisms come from studies focused on a process called transcytosis, which is defined as the movement of a cargo from one polar domain to another (Kleine-Vehn *et al.*, 2008b). It has been observed that, in gravistimulated roots or hypocotyls, inhibition of protein synthesis does not affect gravity-induced PIN3 polarization to the bottom cell side, suggesting that PIN3 is translocated from the pre-existing pools (Kleine-Vehn *et al.*, 2010; Rakusová *et al.*, 2011). The transcytosis of PINs is therefore suggested as an essential mechanism for the rapid modulation of polar auxin transport in tropisms.

Another important regulatory mechanism for PIN trafficking and polarity in general, and during tropisms, is kinase-mediated PIN phosphorylation (reviewed in Barbosa *et al.*, 2018; Tan *et al.*, 2021). Phosphorylation of PIN proteins at the central hydrophilic loop is sufficient to modulate PIN polarity (Michniewicz *et al.*, 2007; Huang *et al.*, 2010; Zhang *et al.*, 2011; reviewed in Ganguly *et al.*, 2012). Several protein kinase families, including the plant-specific AGCVIII family (named after cyclic adenosine monophosphate (cAMP)-dependent and cyclic guanosine monophosphate (cGMP)-dependent protein kinases, as well as protein kinase C), the MITOGEN-ACTIVATED PROTEIN KINASE (MAPK) family, and the CDPK-RELATED KINASE5 (CRK5), phosphorylate PIN proteins and control PIN polarity and transport activity in many developmental processes including tropism (reviewed in Barbosa *et al.*, 2018; Tan *et al.*, 2021).

Within the AGCVIII kinase family, PINOID (PID) and its paralogs WAVY ROOT GROWTH (WAGs) have been well studied in PIN polarity regulation. PINOID and WAGs phosphorylate PINs at three conserved serine sites (S1–S3), and this phosphorylation triggers changes in PIN polarity (Friml *et al.*, 2004; Dhonukshe *et al.*, 2010; Huang *et al.*, 2010). PINOID and WAGs act together and form a positive feedback loop with their interactors, the MAB4/MEL scaffold proteins, to limit lateral diffusion of PINs in their polar domains (Glanc *et al.*, 2021). PINOID loss-of-function and gain-of-function mutants exhibit defective PIN3 polarization as well as bending defects in response to light and gravity (Ding *et al.*, 2011; Rakusová *et al.*, 2011). Hence, PID-mediated PIN3 phosphorylation is essential for establishing the auxin gradient during phototropic and gravitropic responses (Ding *et al.*, 2011; Rakusová *et al.*, 2011; Grones *et al.*, 2018). ROOTS CURL IN NPA 1 (RCN1) encodes a regulatory subunit of PROTEIN PHOSPHATASE 2A (PP2A), and the *rcn1* mutant displays a reduced phosphatase activity and reduced root gravitropic bending curvature, suggesting that PP2A-mediated PIN dephosphorylation is also vital to PIN polarity regulation (Michniewicz *et al.*, 2007) and gravitropism (Sukumar *et al.*, 2009).

Another group of AGCVIII kinases, D6 PROTEIN KINASE (D6PK) and D6 PROTEIN KINASE-LIKEs (D6PKLs), have been implicated in auxin transport regulation. Loss of D6PK and D6PKLs activity led to typical auxin-related phenotypes that correlate with altered polar auxin transport (reviewed in Barbosa *et al.*, 2018; Tan *et al.*, 2021). D6PK phosphorylates PINs at serines S1–S3 and two additional serine sites S4 and S5, and this phosphorylation is independent of the PID kinase. In contrast to PID, D6PKs phosphorylate PINs but do not affect PIN protein polarity. Instead, D6PKs regulate the PIN transport activity. Reduced PIN transport capacity in *d6pk* and *d6pkl* mutants is correlated with defective shoot gravitropic and phototropic responses (reviewed in Barbosa *et al.*, 2018; Tan *et al.*, 2021), whereas D6PK does not play a major role in root gravitropism (Grones *et al.*, 2018).

The *Arabidopsis* 3-PHOSPHOINOSITIDE-DEPENDENT PROTEIN KINASE 1 (PDK1) and PDK2, which also belong to the AGCVIII kinase family, have been shown to modulate PID and D6PK kinase activity, and the *pdk1 pdk2* mutant exhibits decreased gravitropic and phototropic bending (reviewed in Armengot *et al.*, 2016; Tan *et al.*, 2020b; Xiao & Offringa, 2020). This indicates that there might be another layer in PIN polarity and activity regulation during tropisms. In summary, AGCVIII family kinases probably have distinct but partially overlapping functions in regulating PIN polarity and auxin transport during tropisms.

MITOGEN-ACTIVATED PROTEIN KINASE cascades play essential roles in plant growth and development (reviewed in Xu & Zhang, 2015). Auxin activates unknown MAPKs in the root (Mockaitis & Howell, 2000), and MAPKs repress auxin signaling (Kovtun *et al.*, 1998), indicating a crosstalk between auxin and MAPKs. In the *bushy and dwarf1* (*bud1*) gain-of-function mutant of MAP KINASE KINASE7 (MKK7), the hypocotyl is hyperbending after gravity stimulation (Dai *et al.*, 2006). MITOGEN-ACTIVATED PROTEIN KINASE6 (MPK6) has been shown to be the substrate of MKK7 (Jia *et al.*, 2016). MKK7-MPK6 cascade directly phosphorylates Ser 337 of PIN1, a conserved phosphorylation site present in PIN3 at Ser 317 (Jia *et al.*, 2016). However, there is little evidence linking PIN3 polarity regulation to the MKK7-MPK6 cascade during shoot gravitropic response. It is also known that T227, T248 and T286 in the PIN hydrophilic loop can be phosphorylated by MPK6 (Dory *et al.*, 2018). The three threonines are part of the TPRXS (N/S) motifs at the S1–S3 phosphorylation sites, suggesting a possible link between AGCVIII and MAPK kinases in the regulation of PIN polarity via phosphorylation.

A rapid transient increase of Ca^{2+} concentration in the root is observed following auxin treatment (Monshausen *et al.*, 2011) and after gravity stimulation (reviewed in Tatsumi *et al.*, 2014). Increase in Ca^{2+} concentrations suppresses *PIN1* gain-of-function phenotypes and leads to defects in basal PIN1 localization (Zhang *et al.*, 2011). This indicates a possible involvement of the Ca^{2+} signaling pathway in PIN polarity regulation. CRK5 is a candidate, which might convert elevated Ca^{2+} concentrations into altered PIN polarity in root gravitropism (Rigó *et al.*, 2013). CRK5 phosphorylates the PIN2 hydrophilic loop *in vitro*, and defective phosphorylation of PIN2 in the *crk5* mutant leads to a delayed root gravitropic response (Rigó *et al.*, 2013). CRK5 is able to phosphorylate the hydrophilic loop of PIN3 *in vitro* as well (Baba

et al., 2019). However, the phosphorylation site(s) of CRK5 on PINs, which are essential for tropism, are unknown.

Environmental cue-induced PIN polarity switches during tropisms

Plants employ different means to regulate and maintain PIN polarities and auxin gradients in different contexts. A key question regarding the role of PINs in tropisms is how plants translate environmental signals into PIN polarity switches.

Light

During shoot phototropism, the blue light receptor phototropins sense blue light at the upper part of the shoot and initiate shoot phototropic bending (Preuten *et al.*, 2013; reviewed in Liscum *et al.*, 2014). It has been suggested that the signal transducer NPH3 integrates the light signal and auxin gradient establishment during shoot phototropic response (Haga *et al.*, 2005, 2015; reviewed in Christie *et al.*, 2018). This is also supported by a failure to establish auxin gradient in the rice *nph3* mutant following blue light illumination (Haga *et al.*, 2005). NPH3 localizes to the plasma membrane and interacts with the blue light receptor PHOT1, and this interaction is transiently disrupted by light, which also induces NPH3 internalization from the plasma membrane to the cytosolic microdomain aggregates (Haga *et al.*, 2015). This PHOT1-driven NPH3 subcellular localization switch correlates with the phosphorylation status of NPH3. In the dark, NPH3 is phosphorylated, whereas following light illumination, it is rapidly dephosphorylated (Pedmale & Liscum, 2007). The reversible light-induced PHOT1–NPH3 interaction, the change of NPH3 subcellular localization and phosphorylation status is proposed to be part of a signaling mechanism that determines PIN-mediated lateral auxin distribution in phototropism, even though there is no direct evidence for PIN polarization defects in the *nph3* mutant (Haga *et al.*, 2005, 2015; Pedmale & Liscum, 2007; reviewed in Christie *et al.*, 2018). Moreover, etiolated seedlings lacking the PHYTOCHROME INTERACTING BASIC HELIX–LOOP–HELIX FACTORS (PIFs) display reduced NPH3 dephosphorylation and altered auxin distribution (Sun *et al.*, 2013; Sullivan *et al.*, 2019). PHOT1 also interacts with and phosphorylates the ATP-BINDING CASSETTE B19 (ABCB19) auxin transporter (Christie *et al.*, 2011). The phosphorylated ABCB19 protein exhibits reduced auxin transport activity. The elevated auxin pool may be subsequently channeled by PIN3 to the shaded side of the elongation zone; the hypocotyl then bends towards the light source (Christie *et al.*, 2011). Light also represses the transcription of PID, contributing to PIN3 polarization during phototropism (Ding *et al.*, 2011). Loss of D6PK activity does not affect light-induced dephosphorylation of NPH3; however, PIN3 phosphorylation-based polar auxin transport is reduced, ultimately leading to a phototropic defect (Willige *et al.*, 2013). Therefore, it has been suggested that the light-induced phosphorylation gradient via NPH3, PID and D6PKs, which is crucial for PIN polarization and activity, is part of the mechanism that drives lateral auxin distribution during phototropism (reviewed in de Wit *et al.*, 2016). Nonetheless, this model requires experimental confirmation because the timing of visible PIN3 relocation seems to be too slow to account for the initial mechanism of auxin gradient formation.

Gravity

Plants sense gravity via statoliths, starch-filled organelles present in statocytes (gravity-sensing cells), in the root columella and shoot endodermis (reviewed in Vandenbrink & Kiss, 2019). Following gravity stimulation, statoliths reorientate, thus providing information on the direction of the gravity vector. This triggers a biochemical cascade to initiate PIN polarization and resulting asymmetric auxin distribution for directional root or shoot growth. Some evidence suggests that the plant cell cytoskeleton, lipids and the TRANSLOCON OF THE OUTER ENVELOPE OF CHLOROPLASTS (TOC) complex play essential roles in gravity signal transduction (reviewed in Vandenbrink & Kiss, 2019). The J-domain protein, ALTERED RESPONSE TO GRAVITY 1 (ARG1), and its paralog ARG1-LIKE 2 (ARL2) are expressed in the root, and the *arg1* and *arl2* mutations exhibit defective gravity sensing and PIN polarization (Harrison & Masson, 2008). Root and shoot gravitropism depend on NEGATIVE GRAVITROPIC RESPONSE OF ROOT (NGR) proteins, also referred to as LAZY1-LIKE (LZY) proteins (Ge & Chen, 2016; Taniguchi *et al.*, 2017). The *ngr1,2,3/lzy2,3,4* triple mutant shows normal starch sedimentation, but PIN3 polarizes to the upper cell sides after gravity stimulation; thus, the root grows upwards (Ge & Chen, 2019). The LAZY protein is localized at the plasma membrane of columella cells. After gravity stimulation, it polarizes to the bottom side of the columella cell and recruits the RCC1-LIKE DOMAIN (RLD) protein to form a complex. This LAZY–RLD complex then guides PIN polarization and auxin flow, thus bridging the gap between gravity-induced amyloplast sedimentation and polar auxin movement (Furutani *et al.*, 2020). In addition, the isolation of a series of *shoot gravitropism* (*sgr*) mutants provides vital insights into the mechanism of gravity sensing and PIN polarity regulation (Fukaki *et al.*, 1996; Nakamura *et al.*, 2011). Besides, a secondary gravity-sensing site may also exist in the root distal elongation zone (DEZ) (Wolverton *et al.*, 2002; Mancuso *et al.*, 2006). One possible mechanism would be that DEZ cells detect locally exerted force by the protoplast on the cell wall to sense gravity, as suggested in Chara and rice root tip (Staves *et al.*, 1992, 1997). Plant cells perceive gravitational pressure presumably via activation of mechanosensitive ion channels to reorganize the cytoskeleton network (reviewed in Soga, 2013). We anticipate that our understanding of how gravity sensing influences PIN-mediated auxin transport and therewith mediates tropic bending will benefit from novel technologies such as the vertical microscope (von Wangenheim *et al.*, 2017) and from detailed genetic and integrative studies.

Salinity

Significant efforts have been made to understand the mechanism of salt perception and signal transduction. Phosphatidic acid (PA), a minor membrane phospholipid, is essential for plant growth and

development in response to salinity. Phosphatidic acid binds to PID and enhances PID-dependent PIN phosphorylation under salt treatment (Wang *et al.*, 2019). Inhibition of PA synthesis also alters PIN localization and the halotropic response (Korver *et al.*, 2019). Furthermore, PA has been shown to modulate clathrin-mediated endocytosis (Antonescu *et al.*, 2010). It is therefore likely that plants sense the changes in PA steady-state concentrations, and then recruit PID or employ endocytosis to modulate PIN-mediated auxin distribution during the halotropic response.

Water potential

Where and how plants sense differentials in water potential in the root has been a subject of major research efforts. Identification of key genes involved in hydrotropism provided important insights (Eapen *et al.*, 2003; Kobayashi *et al.*, 2007; Miyazawa *et al.*, 2009; Saucedo *et al.*, 2012; reviewed in Dietrich, 2018). It has been proposed that the root elongation zone captures the water gradient, leading to a differential growth of cortex cells (Dietrich *et al.*, 2017). The *mizu-kussei 1* (*miz1*) mutant shows a reduced hydrotropic response, and MIZ1 plays a role in sensing water potential in the early phase of hydrotropism (Kobayashi *et al.*, 2007; Dietrich *et al.*, 2017). The role of water uptake and transport in root hydrotropism has also been investigated. It has been shown that PLASMA MEMBRANE INTRINSIC PROTEINS (PIPs), a subfamily of plasma membrane-localized aquaporin channels, contribute to water transport, root hydraulic conductivity and hydrotropism (Sutka *et al.*, 2011; reviewed in Li *et al.*, 2014; Dietrich, 2018). Thus, a hypothetical model for water potential perception has been proposed (Dietrich *et al.*, 2017, 2018). In this model, MIZ1 senses water potential (Kobayashi *et al.*, 2007; Dietrich *et al.*, 2017), and low water potential influences the presence or aquaporin activity of PIPs at the membrane. This modulation of PIPs results in altered hydraulic conductivity or ABA concentration, which ultimately leads to differential cell elongation and root bending. Meanwhile, MIZ2/GNOM is required for rapid cycling of PIPs and the interaction between PIPs and RECEPTOR-LIKE KINASES (RLKs) serves to maintain aquaporin activity during hydrotropism (reviewed in Dietrich, 2018).

Auxin feedback on PIN polarity to terminate bending

While a lot of effort focused on the mechanism of bending initiation, the termination of tropic responses received comparatively less attention. Hypocotyl bending response is terminated by a mechanism that involves a feedback control on PIN polarity (Fig. 1b; Rakusová *et al.*, 2016). When auxin gradually accumulates on the lower hypocotyl side, this triggers PIN3 repolarization to the inner side of endodermal cells which (re)establishes a symmetric PIN localization and restoration of the symmetric auxin distribution across the organ, resulting in bending termination. This auxin-triggered feedback on PIN repolarization requires the $SCF^{TIR1/AFB}$ auxin signaling (Han *et al.*, 2020), clathrin-mediated endocytosis, GNOM-mediated trafficking, PID-mediated phosphorylation (Rakusová *et al.*, 2016), actin cytoskeleton (Rakusová *et al.*, 2019) and Myosin XI activity (Han *et al.*, 2021). Such an auxin feedback mechanism ensures the fine-tuning of auxin fluxes for termination of asymmetric growth during shoot gravitropic and phototropic responses.

Interactions among different tropisms

Plants are exposed to a complex environment and must balance their cellular actions in response to a combination of environmental cues for optimal adaption. Several studies have been carried out to dissect the interactions among different tropisms in an effort to understand adaptive growth under natural conditions.

Light-grown plants must integrate light and gravity signals, with light playing a dominant role in hypocotyl tropic growth. Several studies have demonstrated that light inhibits the shoot gravitropic response to promote phototropism through different light receptors (Lariguet & Fankhauser, 2004; Ohgishi *et al.*, 2004; Kim *et al.*, 2011). Higher-order blue light receptor mutants display random gravitropism, indicating that light is important for maintaining a proper gravitropic response (Lariguet & Fankhauser, 2004; Ohgishi *et al.*, 2004). Red light illumination inhibits shoot gravitropism by triggering a conversion of gravity-sensing endodermal amyloplasts into chloroplasts, dependent on phytochromes and PIFs (Kim *et al.*, 2011). These plastids with diminished gravity-sensing function lead to a reduced gravitropic response (Kim *et al.*, 2011). In addition, PIFs directly bind LAZY4, an essential regulator of PIN polarization during gravitropism (Ge & Chen, 2019; Furutani *et al.*, 2020), and activate *LAZY4* transcription to inhibit shoot gravitropism (Yang *et al.*, 2020). Light also modulates actin cytoskeleton organization via RICE MORPHOLOGY DETERMINANT (RMD) to control statolith mobility and auxin distribution, thus negatively regulating gravitropism (Song *et al.*, 2019). In summary, based on the available data, light promotes phototropism through inhibition of gravitropism by modulating gravity sensing, actin organization and auxin transport.

An interaction between halotropism and gravitropism has been described where salt treatment leads to reduced root gravitropic bending (Sun *et al.*, 2008). This salt-induced reduction of the root gravitropic response correlates with a rapid degradation of amyloplasts and with the suppression of PIN2 endocytic recycling and degradation (Sun *et al.*, *l.*, *l.*, 2008). In turn, it has been suggested that light attenuates root halotropism by preventing the perception of a salinity gradient (Yokawa *et al.*, 2014). However, the mechanistic basis of this observation remains unknown.

To grow hydrotropically, roots must overcome gravitropism. It has been shown that a moisture gradient or water stress induces a decrease in starch content and the rapid degradation of amyloplasts in root columella cells (Takahashi *et al.*, 2003). This mechanism helps the root, at least partially, to overcome gravitropism to promote hydrotropism. On the other hand, PHOSPHOLIPASE Dζ2 (PLDζ2) participates in root hydrotropism through PIN2-mediated suppression of root gravitropism (Taniguchi *et al.*, 2010). Light also affects hydrotropic growth. Seedlings grown in the light or dark behave differently during hydrotropic responses. Dark-grown seedlings show a reduced hydrotropic curvature compared with light-grown seedlings, and it has been suggested that this is a result of the light-dependent regulation of expression and

subcellular localization of the MIZ1 regulator in the root cap (Moriwaki *et al.*, 2012).

These rather scattered observations provide initial and valuable insights into the molecular and cellular mechanisms of crosstalk between various tropic responses in the face of a complex environment. However, the underlying mechanisms, including a role of PIN-mediated polar auxin transport in these interactions, require further investigation.

Concluding remarks and future perspectives

Tropisms, reorientations of plant growth in reaction to environmental stimuli, are robust adaptive responses that coordinate plant growth in a changing environment. In many cases, PIN-mediated asymmetric auxin distribution plays an indispensable role in generating and/or maintaining the auxin gradient in the stimulated organs. Rearrangement of polar auxin fluxes determined by PIN polarity switches and the regulation of PIN abundance are common themes in most tropic responses. The application of new tools and approaches expands our understanding of the molecular mechanisms of PIN polarizations in response to various environmental stimuli.

PIN-mediated cell-to-cell polar auxin movement is a central mechanism for differential cell growth and organ bending in most tropic responses. More importantly, PIN polarity rearrangements in response to environmental stimuli, such as light or gravity, redirect auxin fluxes, thus providing a common mechanism to translate external signal into auxin-induced growth response.

The signaling pathways and mechanisms that plants employ to trigger PIN polarity switches or to modulate PIN abundance by altering PIN trafficking pathways still remain incompletely understood. Multidisciplinary approaches utilizing genetics, biochemistry, advanced imaging tools and other cutting-edge techniques will be helpful in future studies to better understand PIN polarity regulation in tropisms.

In a natural environment, plants are subject to multiple and sometimes contradictory environmental cues to control their growth. To achieve optimal fitness under complex conditions, plants need to integrate various environmental cues to coordinate cellular responses and growth. The question of how plants coordinate the response to these often conflicting stimuli is a particularly interesting and intricate one. Therefore, it is a popular subject to mathematical modeling, which greatly aids our understanding of the mechanisms by which environmental stimuli lead to changes in cellular activities, tissue growth and ultimately to tropic responses at a whole-plant scale (Moulton *et al.*, 2020; Levernier *et al.*, 2021). These broader concepts, along with the elucidation of molecular mechanisms by which PIN polarity regulations resulting from distinct tropic stimuli are integrated, will greatly help us to understand how plants adapt to changing environments.

A comprehensive understanding of PIN-mediated directional auxin transport in plant tropic responses will require the unraveling not only of the complex signaling pathways described earlier but also of how these pathways interact with each other. The basic knowledge gained from *Arabidopsis* will provide essential insights into the engineering of crops for better growth and yield under adverse environmental conditions.

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HH and JF conceived the idea; HH designed the figure; HH, MA, LQ, SSA and JF wrote the manuscript.

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