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REVIEW

The regulation and plasticity of root hair patterning and morphogenesis

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ABSTRACT

Root hairs are highly specialized cells found in the epidermis of plant roots that play a key role in providing the plant with water and mineral nutrients. Root hairs have been used as a model system for understanding both cell fate determination and the morphogenetic plasticity of cell differentiation. Indeed, many studies have shown that the fate of root epidermal cells, which differentiate into either root hair or non-hair cells, is determined by a complex interplay of intrinsic and extrinsic cues that results in a predictable but highly plastic pattern of epidermal cells that can vary in shape, size and function. Here, we review these studies and discuss recent evidence suggesting that environmental information can be integrated at multiple points in the root hair morphogenetic pathway and affects multifaceted processes at the chromatin, transcriptional and post-transcriptional levels.

KEY WORDS: Cell fate, Root hairs, Phosphate deficiency, Phenotypic plasticity, Auxin, Strigolactones, Chromatin remodeling

Introduction

The development of multicellular organisms requires the specification of cells into various types that differ in shape, size, composition and function. Owing to their reiterative and potentially indeterminate mode of growth, plants remain highly responsive to environmental cues post-embryonically. In addition, and to compensate for their lack of motility, plants exhibit a high degree of plasticity in cell size, patterning and differentiation that allows them to adapt their development to an ever-changing environment. Thus, a wide range of information is integrated into the decisions that adjust plant growth and maximize fitness. These responses are systemic: information communicated from above-ground parts orchestrates the physiological responses of roots and tunes developmental programs, and, conversely, roots are exposed to a plethora of signals that alter gene expression and trigger adaptive responses. Understanding the mechanisms that drive this plastic development of plants is of utmost importance against the background of a changing climate and the negative environmental aspects of intensive agriculture. This knowledge also forms the basis for the generation of crops with more resilient performance under unfavorable environmental conditions, which in turn sets the stage for more sustainable, low-input agricultural ecosystems.

One model system that has been used extensively to study how plant cells integrate external and internal signals to calibrate morphogenetic programs is the root hair. Root hairs are tubular outgrowths that play a key role in providing the plant with water and mineral nutrients (Fig. 1A). Owing to their extreme polar growth, root hairs extend the reach of roots radially, thereby minimizing the generation of depletion zones and increasing nutrient absorption by interception. Root hairs are also involved in beneficial endosymbiotic interactions with a variety of microorganisms such as nitrogen-fixing bacterial symbionts (Oldroyd and Dixon, 2014), and are hence often used as a model for understanding such interactions (see Box 1).

In recent years, studies have shown that the pathway of root hair morphogenesis, which is determined by genetically fixed, intrinsic developmental programs, and the environmental pathways that allow plants to adapt to prevailing conditions are intertwined in a sophisticated way. Knowledge of how these edaphic, genetic and systemic signals are integrated to orchestrate the physiological responses and developmental programs of roots is essential to gain a comprehensive picture of the mechanisms underlying phenotypic plasticity. In this Review, we summarize progress in understanding how the complex interplay of intrinsic programs with external cues shapes the form and function of root hair cells. Although *Arabidopsis* has been widely used as a tool to elucidate the molecular pathways of such integrated pathways, obvious differences between species show that different solutions have evolved, and other model species have thus emerged for evaluating processes that cannot be studied in *Arabidopsis*. We also, therefore, integrate knowledge derived from other root hair models in comparison with and in addition to what has been learned from *Arabidopsis*.

An overview of root hair development and patterning

Root hairs develop from a group of specialized epidermal cells referred to as trichoblasts (Leavitt, 1904) and are arranged in the epidermis in a species-dependent pattern. Owing to their shorter cycle time, trichoblasts are generally shorter than non-hair-forming atrichoblasts, less vacuolated and have denser cytoplasm (Grierson et al., 2014). These differences between the two cell types are evident prior to root hair initiation. In principle, three different types of root hair patterns (Fig. 1B) can be distinguished (Leavitt, 1904; Dolan and Costa, 2001; Marzec et al., 2014). In species exhibiting a type I pattern (e.g. rice), root hairs develop in a random pattern; the variable proportion of cells that form root hairs allows for plastic responses to the environment (Cormack, 1947). In species with a type II pattern (e.g. grasses like *Brachypodium*), root hairs derive from asymmetric cell divisions after which the larger cell enters the non-hair cell fate whereas the smaller cell develops into a root hair. Finally, in the case of the type III pattern, which is seen in Brassicaceae like *Arabidopsis* and in some of their sister families such as Capparaceae, Tovariceae and Resedaceae, root hairs are organized in cell files that are interspersed with non-hair cell files (Bünning, 1951; Pemberton et al., 2001).

The initial development of root hairs involves position-dependent cell fate specification pathways within the epidermis. Manipulation

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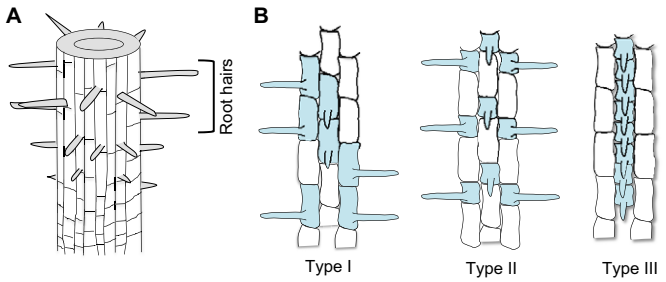

Fig. 1. Root hair formation and root epidermal patterning. (A) The development of root hairs in the root epidermis. (B) Types of root epidermal patterning. In type I, any root epidermal cell can produce a root hair. In type II, root hairs derive from asymmetric cell divisions after which the larger cell enters the non-hair cell fate whereas the smaller cell develops into a root hair. Type III is characterized by alternating files of root hair and non-root hair cells.

of the position of the cell, for example by laser ablation after which the position of the cells is altered, revealed that the fate of epidermal cells in *Arabidopsis* is defined by positional information and not by cell lineage (Berger et al., 1998). More specifically, epidermal cells that are in contact with two cells in the underlying cortical layer (i.e. those in a 'H position'; Fig. 2A) develop into a root hair cell, whereas cells that are in contact with only one cortical cell (i.e. those in an 'N position') enter the non-hair cell developmental program.

The process of root hair formation can be separated into two discrete phases: initiation and tip growth (Fig. 2B; Dolan et al., 1994; Grierson et al., 2014). After cell specification, whereby a patterned assignment of cell fates leads to morphologically distinguishable trichoblasts and atrichoblasts, the establishment of planar polarity allows selection of the root hair initiation site in a complex, auxin-mediated process referred to as planar polarity determination. During this event, anisotropically expanding epidermal cells switch to polar growth (Nakamura et al., 2012; Balcerowicz et al., 2015). Root hair initiation commences with positioning of a polar initiation site at the basal end of the trichoblast, where local cell wall loosening leads to the formation of a disc-shaped area shortly before the cell ceases to elongate longitudinally. Subsequently, bulge formation occurs, which involves the accumulation of ROP protein, endoplasmic reticulum and filamentous actin as well as local acidification of the cell wall. After bulge formation, tip growth begins with the formation of a rapidly growing tubular structure, sustained by rapid exocytosis of cell wall and membrane materials in the root hair apex. Tip growth commences after trichoblasts cease to elongate and is governed by the establishment of a calcium gradient and highly polarized and dynamic actin cytoskeleton. The velocity and duration of tip growth defines the final size of the root hair; in *Arabidopsis*, root hair length can reach 1 mm or more (Grierson et al., 2014).

Cell fate decisions during root hair development

In *Arabidopsis*, the fate of cells in the epidermis is regulated by various signaling and transcription factors (Fig. 3). Root hair cell fate is negatively regulated by an activator complex that comprises the R2R3-type MYB transcription factor WEREWOLF (WER), the basic helix-loop-helix (bHLH) protein GLABRA 3 (GL3) or its paralog ENHANCER OF GLABRA 3 (EGL3), and the WD repeat protein TRANSPARENT TESTA GLABRA 1 (TTG1) (Grebe, 2012; Schiefelbein et al., 2014; Balcerowicz et al., 2015). This complex promotes the expression of the homeodomain protein GLABRA 2 (GL2), which ultimately blocks the hair pathway. In trichoblasts, the expression of *WER* is reduced by a positional signal induced by JACKDAW (JKD) in the cortex (Hassan et al., 2010) that is thought to be stronger in the H position and to be perceived by the leucine-rich repeat receptor-like kinase SCRAMBLED (SCM) (Kwak et al., 2005). *WER* repression in H positions requires TORNADO 1, a plant-specific protein with a leucine-rich repeat ribonuclease inhibitor-like domain (Kwak et al., 2015). The reduced abundance of WER allows the formation of a complex in which WER is substituted by the R3 MYB protein CAPRICE (CPC) or its functional paralogs ENHANCER OF TRY AND CPC 1 (ETC1), ETC3 or TRYPTICHON (TRY) (Schiefelbein et al., 2014). This complex does not support expression of *GL2* and the cell therefore enters the root hair cell fate program. Cell fate assignment is reinforced by the migration of CPC from the N to the H position and the movement of GL3/EGL3 in the opposite direction, which has

Box 1. The root hair 'infectome'

Root hairs from soybean and *Medicago truncatula* represent the major models for investigating legume-rhizobium interactions (Wan et al., 2005; Libault et al., 2010; Nguyen et al., 2012; Breakspear et al., 2014). In root hairs of soybean, ~2000 genes are differentially expressed during infection with *Bradyrhizobium japonicum* (Libault et al., 2010). A time-course analysis revealed that, in an early phase, transcriptional regulators are activated followed by their putative targets, which are involved in gibberellin biosynthesis and in the modification of cell walls, i.e. root hair deformation-related genes (Libault et al., 2010). Phosphoproteomic analysis of soybean root hairs after *B. japonicum* inoculation indicated that proteins associated with signal transduction and hormone signaling were post-translationally modified (Nguyen et al., 2012). Analyses of *M. truncatula* root hairs in response to infection with *Sinorhizobium meliloti* and bacterial Nod factor application showed that rhizobial infection is associated with re-activation of the cell cycle, DNA metabolism and intensive hormonal regulation by strigolactones, gibberellic acid and brassinosteroids. In particular, auxin signaling at infection sites was found to be required for cell wall extension. Notably, several genes that are regulated by rhizobium infection are conserved between soybean and *Medicago*, suggesting that the core genes that are required for this symbiotic interaction evolved prior to lineage diversion (Breakspear et al., 2014).

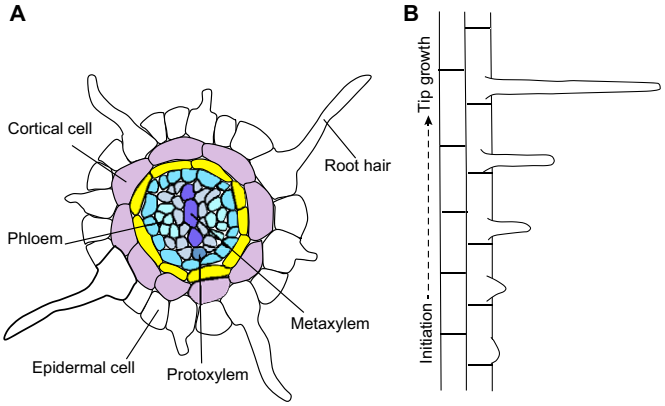

Fig. 2. Root hair development. (A) Cross-section of an *Arabidopsis* root in the differentiation zone, highlighting the various cell types present within the root. Note that epidermal cells that are in contact with only one cell in the underlying cortical layer enter the non-hair cell developmental program, whereas those in contact with two cortical cells develop into a root hair. (B) The key stages of root hair formation: initiation and tip growth. Root hair initiation commences with the positioning of a polar initiation site at the basal end of the trichoblast. Subsequently, a bulge forms and tip growth begins, leading to the formation of a rapidly growing tubular structure.

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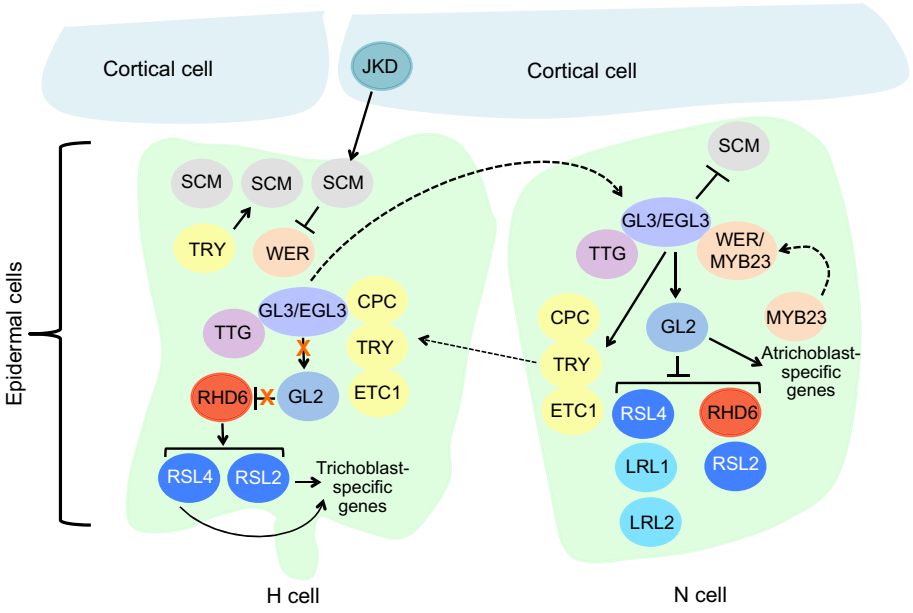

Fig. 3. Position-dependent cell fate specification in the *Arabidopsis* root epidermis. Transcriptional activation is indicated by arrows; blunted lines represent transcriptional repression. Dashed lines indicate intra/intercellular protein movement.

been referred to as a mutual support system (Grierson et al., 2014; Savage et al., 2008; Benítez and Alvarez-Buylla, 2010).

Several feedback mechanisms also support the different cell fate assignments of neighboring cells. For example, the activator complex promotes the expression of a functional paralog of WER, MYB23, establishing a positive-feedback loop in N cells (Kang et al., 2009). A negative-feedback loop between the activator complex and *SCM* also exists, leading to reduced *SCM* expression in N cells (Kwak and Schiefelbein, 2008). Moreover, in *try* but not in *cpc* mutants a reduction of SCM protein is observed, implying positive feedback via TRY on the preferential accumulation of SCM in trichoblasts (Kwak and Schiefelbein, 2014). Interestingly, the activator complex promotes the expression of both positive regulators (GL2) and inhibitors (CPC, ETC1 and TRY) of non-hair cell fate (Schiefelbein et al., 2014).

Root hair formation is modulated by environmental signals

Both the length and the abundance of root hairs are responsive to environmental signals; this ensures the optimal acquisition of soil resources. In particular, mineral nutrients with limited mobility in most soil systems, such as inorganic phosphate (Pi), Mn, Fe and Zn, can affect root hair morphogenesis (Ma et al., 2001; Müller and Schmidt, 2004; Yang et al., 2008).

The best-explored environmentally induced changes in root hair morphogenesis are the responses to Pi deficiency. Pi is an essential plant nutrient and its deficiency leads to deleterious effects on plant development and growth. Pi signaling and homeostasis in plants is reviewed elsewhere (Chiou and Lin, 2011; Lin et al., 2014; Briat et al., 2015) and here we focus on the response to Pi deficiency. The Pi starvation response (PSR) is a multifaceted adaptation that aims to improve Pi acquisition and the internal recycling of Pi. It is composed of metabolic, physiological and morphological components. The major modules of the PSR control the reprogramming of lipid and carbohydrate metabolism to improve the internal use of Pi, control Pi influx via dynamic regulation of the activity of high-affinity Pi transporters, and increase the rhizospheric Pi pool by excretion of Pi-releasing enzymes such as RNases and purple acid phosphatases. In addition, root architectural changes, comprising restricted elongation of primary roots, an increase in the density and length of lateral roots, and the formation of denser and longer root hairs, lead collectively to an increase in the absorptive area, particularly in the Pi-rich topsoil layer. The coordination of shoot and root responses involves trafficking of systemic signals, such as sugar, microRNAs and hormones, through the vasculature to tune the responses in roots (Lin et al., 2014). The morphological responses of roots are thought to be chiefly controlled by the local concentration of Pi (Ticconi et al., 2004; Thibaud et al., 2010), but it may be assumed that local and systemic signals are integrated and that long distance signals might also influence some or all root morphological responses.

In Pi-deficient plants, root hair length is increased by about twofold and hairs are more abundant than in plants grown with sufficient amounts of Pi (Ma et al., 2001). In addition, ectopic root hairs form in N positions, albeit at a relatively low frequency (Savage et al., 2013). A comparison of the root hair response to Pi starvation in different *Arabidopsis* accessions revealed that not all of the lines under investigation show an increase in root hair density and/or length, possibly indicating that the various accessions use different strategies to cope with low Pi (Stetter et al., 2015). Notably, polyploidy has a positive effect on root hair length and density under Pi-replete conditions, but does not contribute to the root hair phenotype of Pi-deficient plants. Furthermore, changes in root hair density and length are not always coupled, suggesting that at least partly separate regulatory pathways control the two traits.

The Pi deficiency-induced increase in root hair density is mainly caused by attenuated longitudinal cell elongation. Strikingly, the longitudinal length of root epidermal cells is dictated by the positional signal that determines cell fate. Mutants that are defective in the expression of *SCM*, which is required for perception of the signal (Kwak et al., 2005), or *WER*, the gateway for positional information into the patterning cascade (Song et al., 2011), form shorter cells than do wild-type plants (Savage et al., 2013). Mathematical modeling indicates that the cortical bias determines the probability of N-positioned cells; a weaker signal delays atrichoblast cell fate resolution and reduces the time for the cell to elongate, resulting in uniquely short, trichoblast-like cells (Savage et al., 2013). In other words, reduced signal strength (as in the case of Pi deficiency) delays the cell fate decision, giving cells in the N position less time to exit the default (root hair) pathway and to acquire an atrichoblast-like length.

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Although restricted elongation of root cells is the dominant mechanism of increasing root hair frequency, genotypes that cannot perceive the signal form more ectopic root hairs in N positions when subjected to Pi deficiency (Savage et al., 2013). Thus, positional bias largely represses the formation of ectopic root hairs, and an additional mechanism that drives the formation of root hairs in N positions can be assumed. In addition, the results are consistent with a Pi deficiency-induced decrease in cortical bias caused by reduced signal strength, or compromised detection or transduction of the signal, which restricts elongation of the cells and allows additional hair assignment in N positions (Savage et al., 2013).

In line with the above findings, it was shown that contact of the root tip with Pi-depleted media was sufficient to restrict longitudinal cell elongation (Svistoonoff et al., 2007). However, this response was not observed when plants were grown on Fe-depleted media (Ward et al., 2008), which led to the suggestion that reduced primary root growth is caused mainly by Fe toxicity. A more recent study showed that although Fe is required for primary root growth inhibition upon Pi deficiency, restricted cell elongation is highly adaptive and is an integral part of the Pi starvation response (Müller et al., 2015). The deposition of Fe and callose in root meristems induced by Pi starvation might compromise symplastic communication in the root stem cell niche and restrict cell elongation. Both LOW PHOSPHATE ROOT 1 (LPR1), a cell wall-localized ferroxidase expressed specifically in the root meristem and in the elongation zone, and the P5-type ATPase PHOSPHATE DEFICIENCY RESPONSE 2 (PDR2) are involved in this process (Ticconi et al., 2009; Müller et al., 2015). Previous work has shown that *pdr2* mutants have a more sensitive response to the activity of both Pi and Fe (Ticconi et al., 2009). Together, these results support a scenario in which the oxidation of ferrous Fe and subsequent apoplastic deposition of ferric oxides and callose in the interior cell layers of Pi-deprived root tips reduces cell-to-cell signaling between cortical and epidermal cells, leading to reduced positional signaling, shorter epidermal cells and, subsequently, a higher frequency of root hairs.

Mechanisms underlying the plasticity of hair cell formation

The Fe, Mn or Pi starvation-induced formation of root hairs in positions normally occupied by non-hair cells (Perry et al., 2007) is indicative of a mechanism that induces the formation of ectopic hairs in response to restricted phyto-availability of immobile mineral nutrients. A number of studies have thus aimed to determine how such external cues affect the WER-based signaling pathway: because cell fate is decided by competition between the WER and CPC complexes, any changes that perturb this competition would alter the cell fate decisions at an early time point in the development of epidermal cells. Together, these studies have identified a number of signals and cues, some of which are integrated into the WER patterning cascade, to modulate hair cell formation in response to environmental signals.

Brassinosteroids act upstream of WER to alter cell fate

Brassinosteroids (BRs) are plant steroid hormones that have recently entered the stage as determinants of epidermal cell fate. BRs are crucial for regulation of *WER* expression and patterning, and thus act upstream of the transcription factor cascade that determines the fate of epidermal cells (Kuppusamy et al., 2009). Low BR signaling reduces CPC levels as a consequence of decreased *WER* expression and reduces the number of hair cells in H positions (Kuppusamy et al., 2009). A genetic analysis revealed that the strength of BR signaling is inversely correlated with the expression of the WER target GL2, reducing its transcription in non-hair cells when BR signaling is compromised (Cheng et al., 2014). BRs also induce the phosphorylation of EGL3 and TTG1 via the GSK3-like kinase BIN2, thereby promoting the movement of EGL3 to N cells and negatively regulating the activity of the WER-GL3–EGL3-TTG1 complex (Cheng et al., 2014).

Lipid metabolism can influence the WER cascade

During root hair patterning, the paralogous R3 MYB proteins CPC, ETC1, ETC3 and TRY appear to play similar but not completely redundant roles in root hair patterning (Schiefelbein et al., 2009; Tominaga-Wada and Wada, 2014). However, it was shown that CPC, ETC1 and TRY acquire additional functions when plants are subjected to Pi deficiency, including but not limited to roles in the transport of Fe and Zn, lipid metabolism, and the acquisition, uptake and storage of Pi (Chen and Schmidt, 2015). Their roles in the regulation of lipid metabolism are of particular interest, given that links between lipid signaling and hair cell differentiation have previously been reported. In response to Pi starvation, plants employ a metabolic pathway in which phospholipids (PLs) are hydrolyzed and replaced by glycolipids to liberate Pi, a process that has been designated as ‘membrane lipid remodeling’ (Nakamura et al., 2009, 2014). Mutants defective in the expression of genes involved in this pathway exhibit a Pi deficiency-specific root hair phenotype (Chandrika et al., 2013), indicative of intricate interplay between root hair differentiation and lipid metabolism under conditions of Pi deficiency. The pronounced changes in lipid metabolism might affect the biological activity of the main players in the battle for cell fate (Fig. 4). For example, it is known that binding to phosphatidic acid (PA) is crucial for the nuclear localization of WER; this PA-WER interaction modulates root hair formation and elongation by a feedback mechanism that supports WER function in non-hair cells and PA abundance in hair cells (Yao et al., 2013). In this scenario, the movement of WER into the nucleus promotes the production of GL2, which negatively

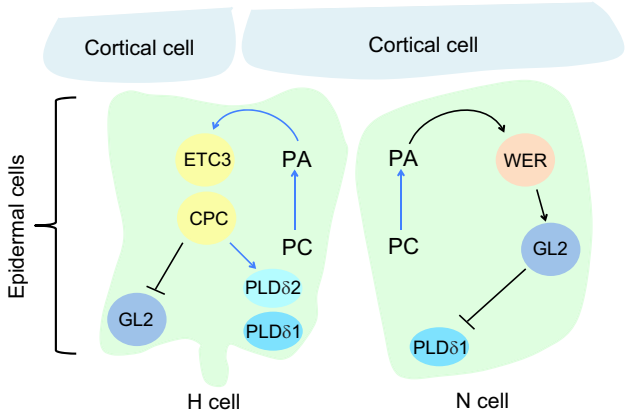

Fig. 4. A Pi deficiency-induced feedback loop leads to the assignment of additional hair cell fates. In non-hair cells, the binding of WER to PA promotes WER translocation to the nucleus where it promotes GL2 expression. Under control conditions, PA is mainly produced by PLDδ1. In root hair cells, CPC competes with WER for binding to PA (and/or other lipids) and positively regulates the expression of PLDδ2, which supports root hair formation. Under Pi-deficient conditions, the expression of *PLDδ2* and of the CPC paralog *ETC3* is increased, leading to the assignment of additional hair fate. Under Pi-deficient conditions, CPC also supports expression of *PLDδ2*, increasing PA production and reinforcing the feedback loop. Pi deficiency-induced processes are denoted in blue. Arrows indicate positive regulation; blunted lines denote repression.

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regulates the expression of phospholipase Dζ1 (PLDζ1) and attenuates the generation of PA, leading to the formation of non-hair cells (Fig. 4) (Ohashi et al., 2003). In hair cells, the relatively higher levels of CPC (compared with those in non-hair cells) counteract WER action and relieve the repression of PLDζ1 by GL2. Likewise, in non-hair cells of Pi-deficient plants, increased levels of CPC (or CPC paralogs) partly compromise the attenuation of PLDζ1 and PA to support root hair formation. In addition, under Pi-deficient conditions, the generation of PA is supported by PLDζ2, which is robustly induced by Pi starvation (Li et al., 2006; Chen and Schmidt, 2015). Mutants defective for both *PLDζ1* and *PLDζ2* thus form fewer root hairs when grown on Pi-depleted medium (Li et al., 2006). Moreover, full induction of PLDζ2 upon Pi starvation is dependent on functional CPC (Chen and Schmidt, 2015), which establishes a feed-forward mechanism that promotes the hair cell fate by continuous PA production (Fig. 4).

Regulation of cell fate at the chromatin level

Tuning the accessibility of DNA to the transcriptional machinery, for example by altering histone composition, DNA methylation and chromatin structure, can define gene expression in cells. Not surprisingly, therefore, chromatin structure appears to be of crucial importance for adaptive strategies in plants, although this remains largely understudied. The first evidence for chromatin level-mediated regulation of environmentally responsive genes originated from a mutant defective in ACTIN-RELATED PROTEIN 6 (ARP6), a component of the SWR1 chromatin remodeling complex (Deal et al., 2005). SWR1 is involved in depositing histone H2A.Z/H2B dimers (in place of canonical H2A/H2B pairs), which alters chromatin structure by promoting intramolecular folding (Fan et al., 2002). In *Arabidopsis*, defective *ARP6* expression results in incorrect H2A.Z deposition and in constitutive activation of a suite of Pi starvation-inducible (PSI) genes. Accordingly, *arp6* mutants form long and dense root hairs under Pi-replete conditions, resembling Pi-deficient plants (Smith et al., 2010). A possible scenario, which explains the role of H2A.Z in this response, involves the binding of *trans*-regulatory elements to the promoters of a subset of PSI genes. Whether root hair genes are regulated directly by H2A.Z, or whether the root hair phenotype is a result of altered Pi deficiency, remains to be elucidated.

A forward genetic screen for mutants with Pi deficiency-specific inhibition of root hair growth was used to identify the plant homeodomain (PHD) protein ALFIN-LIKE 6 (AL6) as being essential for root hair elongation under conditions of Pi deficiency (Chandrika et al., 2013). AL6 can bind to trimethylated lysine 4 of histone H3 (H3K4me3), thereby providing a binding platform for *trans*-acting regulators (Sims and Reinberg, 2006; Lee et al., 2009). In homozygous *al6* mutants, a suite of Pi-responsive genes show decreased expression, including *ETC1* and genes involved in lipid membrane remodeling. It is thus tempting to speculate that AL6 affects early processes in Pi deficiency-induced root hair morphogenesis, probably involving post-transcriptional control of proteins that dictate cell fate and/or the post-translational regulation of such proteins via protein-lipid interactions.

Histone deacetylases have also been implicated in hair cell formation. Both defects in and overexpression of the histone deacetylase gene *HDA18* weakens the pattern of hair and non-hair cells in *Arabidopsis* leading to the formation of root hairs in ectopic positions (Xu et al., 2005; Liu et al., 2013). In addition, altered expression of *HDA6* affects the expression of *ETC1* and *GL2* by binding to the promoters of these genes and causes subsequent changes to the acetylation status of the *ETC1* and *GL2* promoters (Li et al., 2015). Mutations in *HDA6* lead to the formation of ectopic root hairs, indicating that chromatin-level regulation affects early stages of root hair morphogenesis (Li et al., 2015). Similarly, variations in the expression of *HDA19* affect the longitudinal elongation of root cells and, consequently, root hair density, an effect that is more pronounced under Pi-deficient conditions (Chen et al., 2015). When grown on Pi-depleted media, *HDA19*-overexpressing lines show dramatically increased formation of ectopic root hairs. These results have been interpreted as an effect of histone acetylation on the strength of positional bias, a scenario in which increased HDA19 activity results in a weaker signal, resembling the situation observed in Pi-deficient plants.

Reactive oxygen species-dependent changes in root hair morphogenesis

Reactive forms of molecular oxygen (reactive oxygen species, ROS) are omnipresent by-products of aerobic metabolism that are highly reactive towards cellular proteins, DNA and lipids, but they also play important roles in signaling and development (Schippers et al., 2012). Signal transduction via ROS probably derived from the necessity to monitor and combat potentially harmful ROS species, which requires a sophisticated detection system. Environmental conditions can cause subtle changes in ROS homeostasis, opening a route through which nutritional cues can be sensed and translated into adaptive changes in gene expression. Indeed, the spatially distinct regulation of ROS production and distribution is crucial for root hair development, and has been shown to affect Ca^{2+} fluxes and cell wall properties during polarized expansion of the cell (Takeda et al., 2008; Monshausen et al., 2007).

The bHLH transcription factor UPBEAT1 (UPB1), which controls the generation of and the balance between the two main ROS species H_2O_2 and O_2^- by regulating the expression of H_2O_2-generating class III peroxidases and NADPH oxidases, has been shown to control the transition from proliferation to differentiation during hair cell formation (Tsukagoshi et al., 2010). PFT1, a subunit of the Mediator complex, also appears to be involved in controlling ROS balance, acting to regulate the expression of class III peroxidases. In *pft1* mutants, compromised ROS homeostasis results in a marked reduction in root hair density and length, indicating that ROS distribution is crucial for both initiation and elongation of root hairs (Sundaravelpandian et al., 2013). Furthermore, in mutants defective for the Rho GTPase GDP dissociation inhibitor SUPERCENTIPEDE1 (SCN1), ROS production is not focused to a single point, and trichoblasts produce multiple hairs from one initiation site (Carol et al., 2005). This indicates that not only the amount but also the distribution of ROS is crucial for proper root hair morphogenesis.

ROS homeostasis is also linked to nutritional signals. For example, ROS production is an early response to K^+ deprivation (Shin and Schachtman, 2004), and the generation of ROS is associated with differential expression of peroxidases and genes involved in K^+ uptake. In the NADPH oxidase mutant *root hair defective 2* (*rhd2*), which produces hairs that burst during the transition to tip growth, the upregulation of K^+-responsive genes is compromised (Foreman et al., 2003; Shin and Schachtman, 2004). High Mg^{2+} concentrations also compromise tip-focused ROS accumulation and root hair elongation, indicating that the expression of root-hair genes is affected by the Mg^{2+} regime via ROS signaling. Consistent with this, no dependence of root hair elongation on Mg^{2+} supply was observed in *rhd2* plants (Niu et al., 2014). Both the concentration and distribution of ROS are decreased

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in response to Pi deficiency and these changes have been associated with Pi deficiency-induced meristem exhaustion (Chacón-López et al., 2011). In support of this assumption, the *low phosphorus insensitive 4* (*lpi4*) mutant shows a less pronounced decrease in hydrogen peroxide maximum in response to Pi starvation than do wild-type plants and fails to display the attenuated primary root growth that is typical of Pi-deficient plants (Chacón-López et al., 2011). Moreover, a mutant that is hypersensitive to Pi starvation (*hypersensitive to Pi starvation 7*, *hps7*) accumulates high amounts of hydrogen peroxide (Kang et al., 2014). Notably, changes in ROS distribution upon nutrient starvation appear to be nutrient specific (Shin et al., 2005). Based on these results, it is reasonable to assume that ROS homeostasis plays a key role in mediating responses to the nutrient regime affecting the initiation, elongation and shape of root hairs.

The control of root hair elongation and length

The length of the root hair is determined by the duration of tip growth (Knox et al., 2003). Pi deficiency-induced prolonged growth of root hairs is directly correlated with the intensity of a translational pulse of the bHLH transcription factor ROOT HAIR DEFECTIVE 6-LIKE 4 (RSL4) (Yi et al., 2010; Datta et al., 2015). *RSL4* is a direct target of ROOT HAIR DEFECTIVE 6 (RHD6), a VIIIc subfamily bHLH transcription factor that promotes the early stages of root hair development, and is suggested to represent the conversion point of internal and environmental cues (Masucci and Schiefelbein, 1996). RSL4 is expressed in *Arabidopsis* trichoblasts in the elongation zone immediately prior to initiation of root-hair outgrowth, and it controls a suite of genes encoding proteins that are required for root hair morphogenesis (Yi et al., 2010; Datta et al., 2015). Of note, RSL4 is rapidly degraded after root hair elongation is initiated; Pi deficiency significantly increases the synthesis and half-life of RSL4 (Datta et al., 2015). The function of RSL4 appears to be conserved; natural and synthetic allopolyploid wheat form longer root hairs than their diploid progenitors, a trait that was positively correlated with the expression level of *TaRSL4* (Han et al., 2016). This result is in line with the findings of Stetter et al. (2015), who observed longer and denser root hairs in polyploid *Arabidopsis* accessions. Consistent with a crucial function of root hairs particularly in nutrient-poor environments, overexpression of *TaRSL4* led to increased shoot fresh biomass under such conditions (Han et al., 2016).

Interestingly, RSL proteins are not only positive regulators of root hair formation but are also crucial for the development of rhizoids and caulonemal cells in the moss *Physcomitrella patens* (Menand et al., 2007a,b). Group XI bHLH transcription factors encoded by *LOTUS JAPONICUS ROOTHAIRLESS1-LIKE* (*LRL*) genes also play an important role in root hair morphogenesis in both *Physcomitrella* and in angiosperms. Notably, LRL genes are involved in Pi deficiency responses in rice (Yi et al., 2005). Two *LRL* genes in *Physcomitrella*, *PpLRL1* and *PpLRL2*, are involved in the Pi deficiency-induced transition from chloronema to caulonema (Tam et al., 2015). It thus appears that RSL and LRL genes are part of a conserved 'rooting response' evolved in the Silurian period that is recruited under conditions of Pi starvation to improve Pi acquisition (Menand et al., 2007a,b; Tam et al., 2015). Recently, *RSL1*, *RSL2*, *LRL1*, *LRL2* and *RHD6* were shown to be direct targets of GL2 (Lin et al., 2015). Of these genes, *RSL2* is responsive to Pi deficiency (Lan et al., 2012a), linking the activity of GL2 with the perception of environmental information. Interestingly, two functional paralogs of GL2 targets, *RSL4* and *LRL3*, are also responsive to external signals, indicating that some of the apparent genetic redundancy could be explained by the recruitment of these genes for specific functions in response to the prevailing environmental conditions.

Root hair elongation, and hence length, is also dependent on phosphoinositides, a family of minor membrane phospholipids that contain a phosphorylated inositol head group and play crucial roles in signaling events (Xue et al., 2009). Phosphatidylinositol 3-phosphate [PtdIns(3)*P*], for example, is essential for root hair elongation (Lee et al., 2008). *ROOT HAIR DEFECTIVE 4* encodes a phosphatidylinositol-4-phosphate phosphatase required for the organization of polarized secretion during root hair development with selectivity for PtdIns(4)*P*; *rhd4-1* mutants form short hairs with randomly arranged bulges (Thole and Nielsen, 2008). Similar to RSL4, B-type phosphatidylinositol phosphate 5-kinase (PIP5K) enzymes act as quantitative factors for root hair elongation in response to Pi deficiency in *Arabidopsis* (Kusano et al., 2008). These kinases produce phosphatidylinositol 4,5-bisphosphate [PtdIns(4,5)P_2], which is required for reorganization of the actin cytoskeleton (Boss and Im, 2012). Mutants harboring defects in *PIP5K3* and *PIP5K4* produce short root hairs, and double mutants lose responsiveness to Pi deficiency (Wada et al., 2015). Both genes are upregulated by Pi deficiency, and based on these findings it was suggested that PIP5K genes play a key role in transducing the Pi deficiency signal into root hair elongation upon Pi starvation (Wada et al., 2015).

Regulatory intervention by auxin

The plant hormone auxin is also a key regulator of root hair formation (Grebe et al., 2002; Masucci and Schiefelbein, 1996; Balcerowicz et al., 2015). Proper auxin distribution is required for correct cell fate assignment, the determination of planar polarity, and initiation site selection during root hair development. In addition, auxin is an important modulator of root hair shape and size, allowing for the integration of developmental and environmental information. Contrary to what might be expected, preferential expression of the auxin influx carrier AUX1 was detected in non-hair cells, whereas no or weak AUX1 expression was detectable in hair cells (Jones et al., 2009; Ikeda et al., 2009). Thus, it appears that non-hair cells sustain the auxin supply for hair cells. In *wer* roots, in which all epidermal cells develop into root hairs, AUX1 is absent in the epidermis and hairs are shorter than those of the wild type. Root hairs of *wer* mutants can be restored to wild-type length by auxin application, supporting the assumption that auxin supply from non-hair cells is required for proper root hair elongation (Jones et al., 2009). In the rice *osaux1* mutant, too, root hairs are considerably shorter than those of the wild type (Yu et al., 2015). In contrast to its *Arabidopsis* ortholog, *OsAUX1* is expressed in root hairs but not in non-hair cells, probably reflecting the different patterning of root hairs of the two species (Yu et al., 2015). As we discuss below, a number of studies have investigated how auxin might influence the various stages of root hair development.

The intersection between auxin and genes in the hair cell formation pathway

Auxin acts downstream of RHD6 to positively regulate root hair formation (Masucci and Schiefelbein, 1996). Both the expression of the RHD6 target *RSL4* and root hair length are increased by auxin in wild-type plants but not in *rsl2 rsl4* double mutants (Yi et al., 2010). It thus appears that auxin acts primarily via RSL4. However, only 34 of the 90 genes that were found to be auxin responsive were affected in *rsl4* mutants (Bruex et al., 2012; Yi et al., 2010), indicating that auxin can act via different routes on root hair morphogenesis

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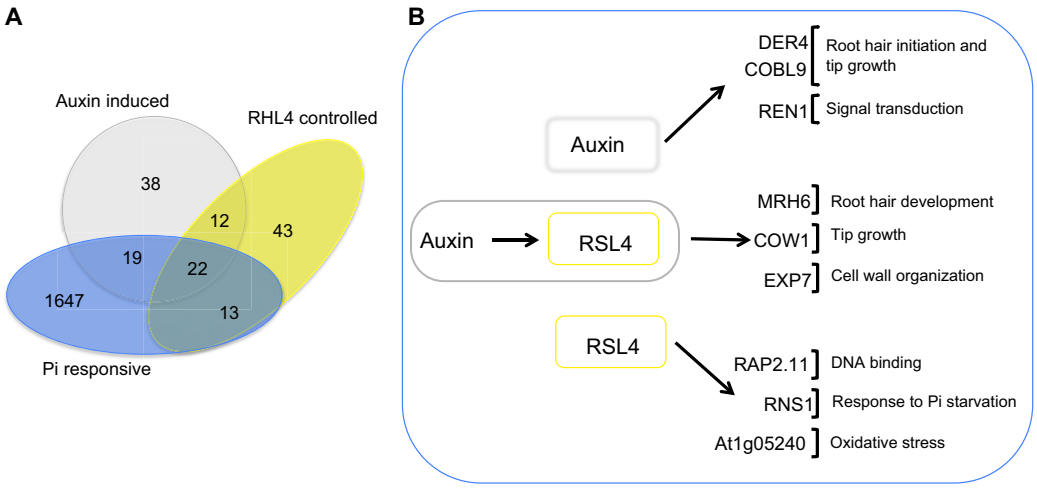

Fig. 5. Genes regulated by auxin, RHL4 and Pi deficiency. (A) Venn diagram showing the relationship among auxin-inducible, RHL4-controlled and Pi-responsive genes. (B) Putative action of Pi-responsive genes in root hair morphogenesis. The partly separate regulation of auxin- and RSL-regulated genes implies both joined and independent pathways. Only a small number of target genes are shown. Data are from Lan et al. (2012a), Yi et al. (2010) and Bruex et al. (2012).

(Fig. 5). Similarly, it appears that RSL4 controls both auxin-responsive and non-responsive genes, possibly inducing parallel but separate routes to promote root hair elongation.

The *tiny root hair 1*(*trh1*) mutant is also defective in the transition to tip growth and develops short root hairs (Rigas et al., 2001). Mutations in *TRH1*, which encodes a member of the KT/KUP/HAK potassium transporter family, cause distorted auxin distribution in the root apex due to de-regulated expression of the auxin transporter *PIN1* (Rigas et al., 2013). TRH1 thus appears to be a crucial node that translates environmental information into auxin signals. Daras et al. (2015) showed that TRH1 acts independently of hair cell specification genes and RHD6, and it was also shown that the expression of *TRH1* is not dependent on RSL4 (Yi et al., 2010), suggesting that TRH1 probably acts upstream of RSL4 in the environmental pathway (Fig. 6). Notably, *trh1* plants are rescued by Pi (Müller and Schmidt, 2004), indicating that the increase in auxin responsiveness by Pi starvation overcomes the misdistribution of auxin in this mutant. Similarly, when grown on Pi-depleted media, the auxin-insensitive mutants *axr1* and *axr2*, which form almost no root hairs under control conditions, are indistinguishable from wild type (Schmidt and Schikora, 2001), suggesting that either an increase in auxin responsiveness compensates for the genetic defects or that the Pi-deficient phenotype is not dependent on auxin signaling. Alternatively, Pi deficiency could feed in downstream of the auxin signaling pathway.

Iron deficiency and auxin: a special case

Whereas Pi starvation augments auxin responsiveness and signaling in *Arabidopsis* root cells (Lan et al., 2012b; López-Bucio et al., 2002; Pérez-Torres et al., 2008), Fe-deficient plants show decreased expression of the synthetic auxin-responsive promoter reporter DR5 compared with control plants (Lan et al., 2012b). Fe deficiency induces the formation of branched root hairs, a response that has been interpreted as an alternative to longer root hairs to increase the surface of the roots (Müller and Schmidt, 2004). Branched root hairs in *Arabidopsis* are typical of Fe deficiencies, whereas other species, such as sunflower, cucumber and tomato, produce normal but more dense root hairs in response to Fe deficiency (Landsberg, 1996; Li and Schmidt, 2010; Schikora and Schmidt, 2002). The formation of branched root hairs was also observed in auxin-related *Arabidopsis* mutants, such as *axr1*, *aux1* and *axr2*, and in mutants defective in actin filament organization or vesicle transport (e.g. *tip1*) (Guimil and Dunand, 2007). In contrast to Pi starvation, growing plants on Fe-free media did not rescue the *axr1* and *axr2* phenotype, indicating that auxin is required for inducing the phenotype typical of Fe-deficient plants (Schmidt and Schikora, 2001).

Interestingly, plants that harbor a mutated form of UBIQUITIN-CONJUGATING ENZYME 13A (UBC13A) do not form branched root hairs in response to Fe deficiency (Li and Schmidt, 2010). UBC13 is the only enzyme that catalyzes ubiquitination at lysine 63 (K63), which, in contrast to K48-linked chains, does not target proteins for proteosomal degradation (Hofmann and Pickart, 1999). The *Arabidopsis* genome contains two highly similar UBC13 proteins, UBC13A and UBC13B (Wen et al., 2006). It was shown that *ubc13a ubc13b* double mutants form fewer and shorter root hairs compared with wild-type plants, bearing a phenotype resembling that of auxin-deficient mutants such as *trh1* (Li and Schmidt, 2010; Wen et al., 2014; Vicente-Agullo et al., 2004; Pitts et al., 1998). In *ubc13a ubc13b* plants, the abundance of Aux/IAA (indole-3-acetic acid) proteins is increased, compromising the

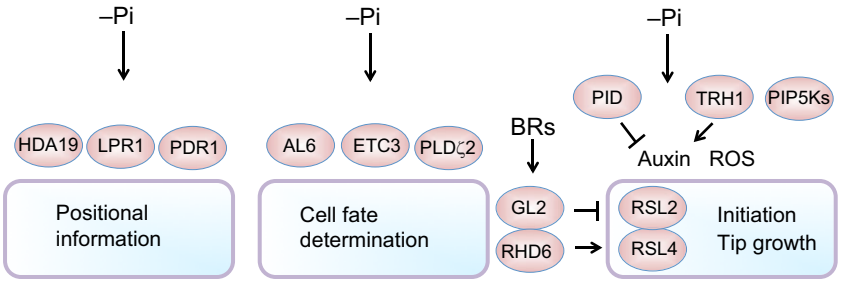

Fig. 6. The integration of environmental cues into the root epidermal cell morphogenetic pathway. External signals, such as Pi deficiency (–Pi), can be translated into signals that affect early stages of differentiation via modulation of the positional signal and the activity of transcription factors that positively affect root hair cell fate. In addition, external cues can act on root hair initiation and the elongation of epidermal cells. Signals are likely to be transduced via various plant hormones, such as strigolactones, brassinosteroids (BRs), ethylene and auxin.

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activation of auxin-responsive genes via auxin response factors (Wen et al., 2014; Guilfoyle et al., 1998). How UBC13 affects Aux/IAAs remains enigmatic. RGLG is the only reported E3 ligase that interacts with UBC13 (Yin et al., 2007). In contrast to expectations, *rglg1 rglg2* double mutants show constitutive root hair branching independent of Fe supply, and they are also partly defective in the response to Fe deficiency (Li and Schmidt, 2010; Pan and Schmidt, 2014; Pan et al., 2015). Similar to *trh1* mutants, the phenotype of *rglg1 rglg2* mutants is rescued by Pi starvation (Li and Schmidt, 2010). Recently, it was shown that the proteolytic turnover of PIN2 is mediated by RGLG via K63-linked ubiquitylation, altering auxin availability in *Arabidopsis* roots (Leitner et al., 2012). Furthermore, *rglg1 rglg2* mutants show reduced auxin levels and responsiveness (Yin et al., 2007). A plausible scenario for Fe-deficient plants involves recruitment of RGLG proteins into the nucleus by UBC13 where they are required for processes associated with DNA repair or genome stability. The finding that both UBC13 and RGLG are involved in DNA repair supports this proposed mechanism (Pan and Schmidt, 2014).

Ethylene promotes root hair morphogenesis via the auxin route

The plant hormone ethylene plays a role in numerous stress responses including Pi deficiency (Kazan, 2015; Nagarajan et al., 2011) and is involved in a large set of developmental responses. Auxin/ethylene interactions have been thoroughly described (Vanstraelen and Benková, 2012; Muday et al., 2012; Robles et al., 2013; Lee and Cho, 2013) and will not be discussed here further. Ethylene promotes root hair morphogenesis, acting on the morphogenetic pathway at the same point as auxin, i.e. downstream of RHD6 (Masucci and Schiefelbein, 1996). The large overlap of auxin- and ethylene-induced genes (Bruex et al., 2012) suggests that the pathways for root hair formation are largely congruent.

Emerging interactions between strigolactones and auxin during root hair morphogenesis

Recently, another group of plant hormones, the carotenoid-derived strigolactones (SLs), have been associated with various aspects of plant development, including root hair formation. SLs are produced by a wide range of embryophytes, including mosses (Delaux et al., 2012). It has been suggested that the primary role of SLs is to modulate development in order to ensure competitive strength for limited soil resources (Brewer et al., 2013). SLs increase the length and density of root hairs, and under conditions of Pi starvation promote the formation of lateral roots (Kapulnik et al., 2011). Relatively little information is available regarding the precise mode of action of SLs, but it appears that modulation of auxin transport is the dominating mechanism (Koltai et al., 2010; Ruyter-Spira et al., 2011). In roots, specifically, the expression, polar localization and endocytosis of the auxin efflux carrier PIN2 are affected by SLs (Pandya-Kumar et al., 2014).

Conclusions

The results outlined here highlight that external stimuli can modulate various stages of root hair patterning and morphogenesis, leading to a multifaceted, highly plastic response (Fig. 6). External cues can be perceived at or upstream of the transcription factor cascade that determines cell fate. Alternatively, they can alter the strength of positional cues and/or change the abundance of positive or negative *trans*-acting factors either transcriptionally or by subcellular or intercellular localization of these factors. Such environmental signals, acting through plant hormones and/or transcription factors, such as RSL4, affect the number, position and length of root hairs through induction of a subset of genes that are crucially involved in cell wall remodeling. Although most of the information summarized here is derived from studies on Pi-deficient plants, it seems plausible that multiple entry sites exist for a large suite of external signals. In addition, largely unexplored regulatory layers, such as epigenetic changes that affect the transcriptional competency of key genes, DNA-encoded information that affects translational fitness of mRNAs and translation efficiency via translation rate, or the accuracy of protein folding, might fine-tune cell size and patterning. This complexity of post-embryonic plasticity mirrors the sophisticated mechanism that determines cell fate in the root epidermis. The seemingly redundant possibilities for external signals to enter the developmental pathway may provide the basis for an efficient integration of various inputs, such as hormones, *cis*-acting factors, chromatin level regulation and post-translational modifications.

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Competing interests

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