Title: Environmental control of hypocotyl elongation

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Abstract

The hypocotyl is the embryonic stem connecting the primary root to the cotyledons. Hypocotyl length varies tremendously depending on the conditions. This developmental plasticity and the simplicity of the organ explains its success as a model for growth regulation. Light and temperature are prominent growth-controlling cues, using shared signaling elements. Mechanisms controlling hypocotyl elongation in etiolated seedlings reaching the light differ from those in photoautotrophic seedlings. However, many common growth regulators intervene in both situations. Multiple photoreceptors including phytochromes, which also respond to temperature, control the activity of several transcription factors thereby eliciting rapid transcriptional reprogramming. Hypocotyl growth often depends on sensing in green tissues and interorgan communication comprising auxin. In the hypocotyl auxin in conjunction with other hormones determine epidermal cell elongation. Plants facing cues with opposite effects on growth control hypocotyl elongation through intricate mechanisms. We discuss the status of the field and finish by highlighting open questions.

List of key terms

De-etiolation: the processes occurring when a dark-grown seedling reaches the light, which comprises rapid inhibition of hypocotyl elongation.

High red to far-red ratio (high R/FR): Sunlight has a R/FR slightly above 1, which is considered as high R/FR.

Low red to far-red ratio (low R/FR): a low R/FR occurs under a plant canopy or in the proximity of plants.

Canopy shade: a light condition combining low R/FR, reduced PAR, low red and low blue.

Shade-avoidance response: a suite of physiological and morphological adaptations triggered by canopy shade.

Neighbor proximity response: a suite of physiological and morphological adaptations triggered by low R/FR alone.

Thermomorphogenesis: a suite of physiological and morphological adaptations triggered by elevated ambient temperature.

CUL4$^{COP1/SPA}$: a cullin4 comprising multimeric ubiquitin E3 ligase controlling the degradation of numerous transcriptional regulators.

Phytochromes (phy): red and far-red sensing photoreceptors controlling growth and developmental transitions in plants. phyA and phyB play distinct roles.

Cryptochromes: blue sensing photoreceptors controlling growth and developmental transitions in plants.

UVR8: UV-B sensing photoreceptor controlling growth and developmental transitions in plants.

Phytochrome Interacting Factors (PIFs): bHLH class transcription factors interacting with and regulated by light activated phytochromes (in particular phyB).

Auxin: a plant hormone regulating many facets of plant development and growth and a key factor controlling hypocotyl elongation.

Acid growth theory: a hypothesis proposing that auxin application to the hypocotyl leads to apoplast acidification and elongation.

Anisotropic growth: a condition when growth rates are not equal in all directions, which is what occurs in epidermal hypocotyl cells.

Cell wall loosening: relaxation of stress following the rearrangement of cell wall components (e.g., changes in polymer interactions).

Resource allocation: hypocotyls are sink organs requiring resources such as sucrose to be transported from green tissues to fuel growth.

Circadian clock: an internally driven rhythm of approximatively 24 hours that regulates plant physiology and growth.

A Summary Points list highlighting the central points of the article

1. Hypocotyl elongation in light and dark-grown seedlings is controlled by cellular expansion, particularly of epidermal cells.

2. Environmental regulation of auxin biosynthesis and signaling often underlies the control of hypocotyl elongation.

3. Brassinosteroids and gibberellic acid are also important growth promoting hormones but there is scarce evidence for environmental regulation of their levels.

4. Environmental control of hypocotyl elongation largely depends on rapid and organ specific changes in gene expression.

5. Phytochrome Interacting Factor (PIFs) are bHLH transcription factors playing a central role in hypocotyl growth control.

6. Multiple photoreceptors and environmental cues control PIF abundance and activity

7. Ubiquitin E3 ligases, most prominently CUL4$^{COP1/SPA}$ are regulated by light and control the abundance of transcription factors.

8. Thermomorphogenesis and shade avoidance rely on highly similar signal transduction mechanisms.

Future Issues

1. How allocation of resources from roots and green tissues is controlled to cover the demands of hypocotyl elongation is poorly understood.

2. Hypocotyl growth is controlled distally and locally, our understanding of local growth control remains scarce.

3. Due to limited spatial and temporal resolution we have a poor understanding of the role of translation, epigenetics and post-transcriptional mechanisms in hypocotyl growth control.

4. The mechanisms underlying environmental control of cell wall extensibility remain poorly understood.

5. Environmental change triggers rapid responses (e.g. burst in auxin production), which are well understood but how this leads to prolonged growth promotion is less clear.

6. Cell biology of inner tissues (endodermis, vasculature) during hypocotyl growth control remains under-explored.

Brief annotations to some references

Du, M. et al. (2022) ‘Biphasic control of cell expansion by auxin coordinates etiolated seedling development’, Science Advances, 8(2), pp. 1–13. Available at: https://doi.org/10.1126/sciadv.abj1570.

Elegant paper showing how changes in auxin concentration can explain the growth dynamics of etiolated hypocotyls.

Costigliolo Rojas, C. et al. (2022) ‘Organ-specific COP1 control of BES1 stability adjusts plant growth patterns under shade or warmth’, Developmental Cell, p. S1534580722004920. Available at: https://doi.org/10.1016/j.devcel.2022.07.003.

This paper provides strong evidence for BES1 and COP1 controlling the differential growth response occurring in hypocotyls versus cotyledons.

Fendrych, M., Leung, J. and Friml, J. (2016) ‘Tir1/AFB-Aux/IAA auxin perception mediates rapid cell wall acidification and growth of Arabidopsis hypocotyls’, eLife, 5(September2016), pp. 1–19. Available at: https://doi.org/10.7554/eLife.19048.

Compelling evidence supporting the role of the canonical auxin signaling pathway during hypocotyl growth promotion.

Hayes, S. et al. (2019) ‘Soil Salinity Limits Plant Shade Avoidance’, Current Biology, 29(10), pp. 1669-1676.e4. Available at: https://doi.org/10.1016/j.cub.2019.03.042.

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Ince, Y.Ç. et al. (2022) ‘A combination of plasma membrane sterol biosynthesis and autophagy is required for shade-induced hypocotyl elongation’, Nature Communications, 13(1), p. 5659. Available at: https://doi.org/10.1038/s41467-022-33384-9.

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Paper providing strong evidence for phytochromes acting as a thermosensors controlling hypocotyl growth depending on temperature and light.

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Paper providing strong evidence for phytochromes acting as a thermosensors controlling hypocotyl growth depending on temperature and light

Lau, K. et al. (2019) ‘ Plant photoreceptors and their signaling components compete for COP 1 binding via VP peptide motifs ’, The EMBO Journal, 38(18), pp. 1–18. Available at: https://doi.org/10.15252/embj.2019102140.

Insightful mechanistic study identifying a mechanism by which photoreceptors control the activity of COP1.

Procko, C. et al. (2016) ‘The epidermis coordinates auxin-induced stem growth in response to shade’, Genes and Development, 30(13), pp. 1529–1541. Available at: https://doi.org/10.1101/gad.283234.116.

Elegant paper showing the key role of auxin in the epidermis to promote hypocotyl elongation in the shade.

Shi, H. et al. (2016) ‘Seedlings Transduce the Depth and Mechanical Pressure of Covering Soil Using COP1 and Ethylene to Regulate EBF1/EBF2 for Soil Emergence’, Current Biology, 26(2), pp. 139–149. Available at: https://doi.org/10.1016/j.cub.2015.11.053.

This neat paper identifies mechanism explains how mechanical stress elicited by soil regulates etiolated hypocotyl elongation and thickening.

Figure legends

Figure 1: Hypocotyl elongation behaviors at different developmental stages

(A) Hypocotyl growth during different stages of seedling development, discussed in sections 1 to 4. (1) Etiolated seedlings are elongating quickly while growing in the dark. Growth is slowed down and hypocotyls thicken when soil is more compact. (2) Once seedlings reach the soil surface they undergo de-etiolation which includes inhibition of hypocotyl elongation and can be mediated by several photoreceptors and light qualities. (3,4) Fully de-etiolated photo-autotrophic seedlings barely elongate in an open habitat at lower temperatures. However, vegetation proximity or cover and warm temperatures induce hypocotyl elongation in shade-avoiding species. (B) Overview of the major environmental signals and responses of etiolated seedlings and main hormone signaling pathways regulating these. Hypocotyl elongation growth is achieved by anisotropic cell expansion, which is characterized by the processes summarized on the box on the right.

Figure 2: Overview of important signaling mechanisms underlying environmental regulation of hypocotyl elongation.

A: Core signaling network that controls hypocotyl elongation in response to environmental cues. Regulated proteins in response to light and temperature changes are highlighted as indicated in the legend. B: PIFs as critical transcription factors for the regulation of hypocotyl elongation, either as master regulators of other transcriptional regulators, or as work horses for the expression of diverse growth promoting genes. C: Simplified signaling pathways of the main hormones discussed in this review.

Introduction

The hypocotyl of a dicot plant is the embryonic stem that connects the primary root with the shoot. Along the stem axis, it is delimited by the primary root and the cotyledonary node. It persists throughout the lifetime of the plant and its tissues and cell types are already well defined in the mature embryo. The Arabidopsis hypocotyl comprises an epidermis, which is the main tissue restricting or allowing growth, two cell layers of cortex, endodermis and vasculature [1,2]. The basic cellular organization does not change during germination until radial growth begins at late seedling stages, but some tissues differentiate during or after germination. The vasculature is largely undifferentiated in the mature embryo but the pericycle and protophloem sieve tube precursor cells can be identified. During germination the vasculature differentiates quickly and has a bilateral symmetry of two protophloem poles and two protoxylem poles embedded in parenchyma and surrounded by the 1-cell layer pericycle [1]. At later Arabidopsis seedling stages, the hypocotyl undergoes radial secondary growth which was recently reviewed [3] and is not covered here. Likewise, formation of adventitious roots from the hypocotyl has been reviewed by others [4]. Gravity and directional light cues regulate asymmetric hypocotyl growth responses known as gravitropism and phototropism, respectively, for which we refer the reader to other recent reviews [5,6].

Hypocotyl development and elongation depend dramatically on environmental conditions, which is the focus of this review. Here, we review hypocotyl elongation and its underlying signaling mechanisms along a developmental gradient including etiolated hypocotyls, growth inhibition during de-etiolation and how environmental cues regulate elongation in de-etiolated, photoautotrophic seedlings (Figure 1A). As the hypocotyl has been a popular model system, the large body of available literature forced us to focus on the major regulatory pathways and relatively recent publications. Therefore, we have chosen to summarize such mechanisms with an emphasis on their physiological relevance without covering complex regulatory mechanisms in depth. Most of the described evidence is from experiments with the popular model organism *Arabidopsis thaliana*, however we included some evidence from other species where relevant.

1. Hypocotyl growth of etiolated seedlings

1.1 The start of hypocotyl growth during germination

In nature seeds typically start to germinate when they become moist and temperature and oxygen availability are favorable [7]. Seeds of many plant species also need light to germinate. In the lab, germination of *Arabidopsis thaliana* seeds is therefore induced and synchronized by exposing moist

and cold-treated (stratified) seeds to light. Germination is usually considered as completed with emergence of the radicle [7], however it has been argued that some elongation growth in the root – hypocotyl transition zone and lower hypocotyl should be regarded as part of the germination process [8]. Regardless of the definition of the end of germination, slight hypocotyl elongation precedes radicle emergence [8] but will increase rapidly after germination.

1.2 Fast hypocotyl elongation in darkness

Seeds germinating at the soil surface are exposed to light immediately and will undergo a developmental program referred to as photomorphogenesis (section 2). However, seeds can germinate underground and develop in the absence of light undergoing a developmental program called skotomorphogenesis, resulting in so-called etiolated seedlings. This section focuses on the signaling underlying skotomorphogenesis.

Etiolated seedlings are characterized by fast hypocotyl elongation, small yellow cotyledons and a closed apical hook that protects the shoot apical meristem from damage while being pushed upwards through the soil. Fast elongation of etiolated hypocotyls depends on hormone signaling and protein stability regulators which converge on the regulation of a large network of transcription factors.

The most relevant protein stability regulators are members of the COP/DET/FUS group which are required for maintaining skotomorphogenesis. They include components of E3-ligase complexes or regulators of E3 ligase activity: the COP9 signalosome which is essential for the activity of Cullin-RING-E3 ligases (CRLs), the CULLIN4 - CONSTITUTIVELY PHOTOMORPHOGENIC1 - SUPPRESSOR OF PHYA-105 ($CUL4^{COP1/SPA}$) and the CUL4 – COP10-DET1-DDB1 ($CUL4^{CDD}$) E3 ligase complexes [9–11].

COP9 signalosome mutants have pleiotropic defects given that they alter the activity of numerous CRLs [10]. $CUL4^{CDD}$ has a role in transcription factor regulation (see below and section 4). -The mechanisms of $CUL4^{COP1/SPA}$are particularly well understood. Its substrate binding moiety is composed of two COP1 and two SPA proteins (SPA1 to SPA4 in Arabidopsis) [11]. Other CRLs regulating hypocotyl elongation in other conditions will be mentioned later.

Two distinct mechanisms of action of the $CUL4^{COP1/SPA}$ complex have been described which both contribute to the maintenance of skotomorphogenesis: Firstly, the ubiquitylation and degradation of transcription factors that promote the opposing developmental program, photomorphogenesis, and secondly the indirect stabilization of transcription factors which promote skotomorphogenesis [11].

Among the many photomorphogenesis promoting transcription factors targeted by $CUL4^{COP1-SPA}$ are ELONGATED HYPOCOTYL 5 (HY5), HY5 HOMOLOG (HYH), LONG HYPOCOTYL IN FAR RED (HFR) 1, and several B-BOX DOMAIN (BBX) proteins (for a complete list of COP1 targets see [11]). Since HY5 is a very strong inducer of photomorphogenesis, its inhibition by COP1 is a central mechanism to maintain skotomorphogenesis. Its homolog HYH has similar functions. HY5 lacks a trans-activation domain but cooperates with three B-box proteins, BBX20, 21 and 22 to regulate expression of its targets. BBX20, 21 and 22 are also degraded via COP1 and simultaneous stabilization of HY5 and BBX21 leads to de-etiolation in darkness [12].

Members of the PHYTOCHROME INTERACTING FACTOR (PIF) family of bHLH transcription factors are required for skotomorphogenesis and strong hypocotyl growth in darkness as they induce expression of growth-promoting genes [13,14]. Their abundance and activity is tightly regulated both in etiolated seedlings and in response to light thereby modulating hypocotyl elongation in a variety of growth conditions (see also sections 2-5). There are eight PIFs (Figure 2B) in Arabidopsis with various

functions in plant development, and primarily PIF 1,3,4 and 5 promote skotomorphogenesis [13]. Control of PIF abundance via degradation is a hallmark of de-etiolation, while in darkness they are turned over to a smaller extent. Several processes maintain PIF stability and activity in darkness. CUL4^{CDD} enhances PIF activity by destabilizing negative PIF regulators, the DELLA proteins (see below) [15] and maintains PIF stability by an unknown mechanism [16]. CUL4$^{COP1\text{-}SPA}$ binds to PIF3 which protects it from phosphorylation by BRASSINOSTEROID INSENSITIVE 2 (BIN2) and subsequent ubiquitylation [17,18].

The most prominent hormones required for hypocotyl elongation in darkness are auxin, gibberellic acid (GA) and brassinosteroids (BR) [19–21] (Figures 1B, 2C). Auxin has a biphasic, dose dependent role in etiolated hypocotyl elongation: Initially, within roughly the first half day after radicle emergence, auxin concentrations are high allowing only very slow growth. Over time, auxin concentrations decrease, possibly due to dilution in enlarging cells, and eventually reach levels that are in the concentration range where growth is promoted [22]. From this point, auxin is instrumental in enhancing hypocotyl elongation through activation of plasma membrane localized proton pumps and induction of genes that are relevant for elongation growth by controlling AUXIN RESPONSE FACTORS (ARFs) transcription factors (Figure 2B,C, section 6) [20,23].

GA levels are higher in the dark than during the first hours of light exposure [24] and seedlings deficient in GA have short hypocotyls in darkness [21]. GA acts primarily by facilitating interaction of its receptor, GA INSENSITIVE DWARF 1 (GID1), with DELLA proteins which promotes their degradation. DELLA proteins bind to PIFs and prevent them from binding to their targets but also induce their degradation (Figure 2C). Therefore, in darkness, GA suppression of DELLAs contributes to PIFs activity [21,25].

Like auxin, BR is required for etiolated hypocotyl elongation at low concentrations but is inhibitory at higher levels [19,26,27]. Transcriptional responses to BR are mediated by transcription factors BZR1 and BES1 which are de-repressed in response to BR due to inactivation of their negative regulator BIN2 (Figure 2C) [19]. Some of the BZR1 and BES1 target genes directly play a role in elongation and include regulators of the cytoskeleton and cell wall [28,29]. BES1/BZR1, PIFs and ARFs are highly interdependent for target gene regulation and are regulated similarly by DELLAs [30]. How the genes regulated by these hormone signaling pathways carry out elongation growth is further discussed in section 6.

1.3 Mechanical obstacles in the soil and soil compaction affect hypocotyl growth

While an etiolated seedling is growing upwards through the soil, it is likely to experience compressed soil or other mechanical obstacles (Figure 1A,B). This leads to an ethylene response resulting in thicker and shorter hypocotyls, which is important for seedling emergence through the soil and survival [31]. The underlying mechanism downstream of ethylene production and sensing involves induction of the transcriptional regulator ETHYLENE RESPONSE FACTOR 1 (ERF1) by the ethylene responsive transcription factor ETHYLENE INSENSITIVE 3 (EIN3) [32]. In addition, COP1 is also involved in regulating this response: In the dark, COP1 indirectly stabilizes EIN3 by targeting EIN3-BINDING F BOX PROTEIN 1 (EBF1) and EBF2 for degradation, thereby preventing them from targeting EIN3 for degradation [31].

2. De-etiolation

2.1 Role of photoreceptor signaling during photomorphogenesis

Once a seedling has reached the soil surface light exposure triggers de-etiolation: the apical hook opens, cotyledons expand and become green, and hypocotyl elongation ceases (Figure 1A). This developmental switch can be initiated by different light colors, which activate specific photoreceptors. This applies to red, far red and blue light. Green light is an exception as seedlings de-etiolating in green light grow longer hypocotyls than in darkness. This effect is independent of the known photoreceptors but depends on BR signaling [33]. UV-B light also suppresses elongation but the experimental framework is typically a comparison of white light with and without UV-B supplementation at varying developmental stages, therefore we will focus on all UV-B mediated processes separately (section 3.3).

Here we describe the mechanisms of photomorphogenesis which are relevant for suppressing hypocotyl elongation, which have a large overlap with those that control hook opening and greening but are not always identical.

De-etiolation mechanisms are dominated by the action of transcription factors and result in largely overlapping changes in gene expression irrespective of the light color (except green) [34]. Changes in gene expression are accompanied by complex epigenetic modifications [35] that are not exhaustively described here. Additional mechanisms have been described, including rapid increase in translation activity by light [36], light dependent promoter usage [37], light dependent alternative splicing [38] and mRNA modification [39]. However, how these mechanisms specifically tune hypocotyl growth remains poorly understood. This is in large part due to the lack of spatial resolution of many of these genome-wide studies. Few studies have specifically analyzed the hypocotyl transcriptome, where growth is inhibited by light, separately from cotyledons, where light promotes growth. Analyzing cotyledon and hypocotyl transcriptomes in response to white light separately, showed a strongly organ-dependent light response [40]. De-etiolation processes are also tissue-specific, such as xylem differentiation. For the transition to photoautotrophic growth, establishment of functional xylem tissue is critical to establish water supply to the sites of photosynthesis. This differentiation is mainly triggered by blue light. In darkness, PIFs maintain high expression of peptide ligand CLE44, which contributes to activation of its receptor, PXY/TDR, an inhibitor of differentiation. Suppression of PIFs by light leads to a decrease of CLE44 levels, PXY/TDR activation and subsequently xylem differentiation [41].

A recent study using a single cell RNA sequencing approach in de-etiolating seedlings shows that light responses differ according to cell types and that shoot tissues are much more responsive than roots tips [42]. More studies including spatial and temporal elements are required to understand hypocotyl growth regulation.

2.1.1 Suppression of hypocotyl elongation by blue light

Three classes of photoreceptors regulate hypocotyl growth during de-etiolation in blue light: phototropin, phytochromes and cryptochrome.

Phototropins are UV-A and blue light receptors, with two members in Arabidopsis, phot1 and phot2. While the main function of phototropins is perception of light direction they also have a fast and transient role in de-etiolation [6]. Phot1 is the photoreceptor responsible for this very early response which is detectable within 30 seconds of light treatment and lasts for about 30 minutes [43]. Phytochromes also contribute to hypocotyl growth suppression in blue light with *phyA* mutant behaving similarly to *cry1* in the early stages of growth inhibition [44].

Cryptochromes (cry1 and cry2 in Arabidopsis) are the primary photoreceptors triggering blue-light-induced de-etiolation [45] and which are responsible for more than half of the gene expression differences between dark and blue light [45]. Cry1 dominates hypocotyl growth suppression at medium

or high light intensities, while cry2 primarily affects the process in response to low blue light intensities [46]. Cry1 and cry2 differ in their protein stability: cry1 is more stable in blue light, while cry2 is rapidly degraded even at low intensities. Blue light dependent degradation of both cryptochromes is mediated by $CUL4^{COP1\text{-}SPA}$ and $CUL3^{LRBs}$ [47,48]. Stabilization of COP1 by the deubiquitinating enzymes UBP12 and UBP13 contributes to fine-tuning hypocotyl elongation at low blue light intensities [49].

Blue light activated cryptochromes inhibit the action of the $CUL4^{COP1\text{-}SPA}$ complex by several mechanisms. Among them cry1 and cry2 inhibit $CUL4^{COP1\text{-}SPA}$ following light-regulated interaction with SPA1 [11]. Moreover, activated cry2 competes with COP1 for binding to conserved valine-proline (VP) motifs of target proteins, making them less accessible for degradation [50,51]. In addition, blue light enhances the degradation of SPA1 and SPA2 and leads to nuclear depletion of COP1 separating it from its target transcription factors. Altogether these mechanisms decrease degradation of photomorphogenesis promoting transcription factors [11].

Another mechanism of cryptochrome action relies on light-regulated binding and activation of CRYPTOCHROME-INTERACTING BASIC-HELIX-LOOP-HELIX (CIB) transcription factors. However this mediates cry2 regulation of flowering time rather than the regulation of hypocotyl elongation [45]. In contrast, regulation of PIF transcription factors, which are related to CIBs, is important for de-etiolation in blue light [52]. The mechanism is expected to involve light-regulated interaction of cry1 with PIF4 and PIF5 leading to their inhibition as shown for cry1-mediated responses to elevated temperature and shade (sections 3,4) [53,54].

During de-etiolation in blue light, changes in hormone levels are not always established while it is known that light regulates hormone signaling pathways. Blue light antagonizes the auxin pathway, resulting in down-regulation of ARF target genes [55]. To our knowledge, auxin abundance during de-etiolation in blue light has not been measured, but given the general similarities with mechanisms in red light induced de-etiolation (section 2.1.2) and the ability of cry1 to suppress expression of auxin biosynthetic genes at warm temperatures [53], it is likely that auxin levels decrease in blue light. There is however evidence for inhibition of auxin signaling by cry1 through direct interaction of photoactivated cry1 with ARFs to prevent their DNA binding [55]. Activated cry1 also interacts with several AUX/IAAs, which prevents them from binding to the TIR1 auxin receptor. This protects IAAs from degradation thereby allowing them to inhibit ARFs [56].

GA levels only transiently decrease in response to blue light [57]. Therefore, regulation of GA levels alone cannot explain its strong effect on hypocotyl growth. Again light also acts on GA signaling with photoactivated cry1 binding to DELLAs and GID1, which prevents DELLA degradation [58–60]. This is expected to decrease PIF stability or activity and hence limit hypocotyl elongation [21,25,61].

To our knowledge, changes in BR in response to blue light have not been reported. However, cry1 interacts with BZR1 and BES1 to prevent their target gene expression [62,63]. In addition, activated cry1 promotes nuclear exclusion of BZR1 via increasing the interaction of BIN2 with BZR1 [63]. Collectively this results in reduced BR-mediated hypocotyl elongation.

2.1.2 Red and far red light

Red and far red light also suppress hypocotyl elongation as part of the de-etiolation process [64], mediated by phytochromes. Arabidopsis possesses five phytochromes of which phyA and phyB are the most relevant during de-etiolation. Due to differences in their abundance, stability and signaling cascades, phyB is the major photoreceptor mediating elongation suppression in red light and phyA in far red light. The ability of plants to de-etiolate in far red light by phyA signaling is thought to allow

plants to develop in shaded environments where phyB is largely inactivated and blue light intensity is low (see section 3)[64].

Phytochrome and cryptochromes mediate de-etiolation through analogous processes. Light-regulated transcription is key but other less well understood processes can contribute. A first step for phytochrome-regulated gene expression is nuclear import of the light activated photoreceptors. In the nucleus phyB localization into so-called photobodies depends on the precise light environment, a process that is believed to underlie phytochrome activity [64,65].

The known mechanisms by which phyA and phyB inhibit $CUL4^{COP1\text{-}SPA}$ are similar to the cryptochrome mediated mechanisms [11]: photoactivated phyA and phyB de-stabilize the interaction of COP1 with SPA proteins [66,67]; both phyA and phyB lead to rapid nuclear exclusion of COP1 [11] as well as degradation of SPA1 and SPA2 [68].

PIFs are suppressed by phytochrome activation by sequestration as well as degradation [69]. Suppression of PIF target gene expression due to sequestration by activated phyB has been shown for PIF1, 3 and – less exhaustively – PIF4 [64,69,70], while PIF1, 3, 4 and 5 are degraded in response to red and / or far red light [18,69]. Several E3 ligases responsible for ubiquitylation of PIFs prior to degradation by the 26S proteasome have been identified [71]. Another degradation mechanism involves HEMERA (HMR), which is stabilized by photoactivated phyB and plays a role in PIF1 and PIF3 degradation in photobodies [72,73].

Hormone pathways are regulated by de-etiolation in red light at multiple points. Auxin abundance drops quickly after red light exposure [74]. In addition, ARF6, ARF8 and AUX/IAA are directly regulated by binding of activated phyB [55,56]. Gene expression data suggests that phyB mediated light signal transduction down-regulates GA biosynthetic genes which is predicted to result in lower GA concentrations and higher DELLA protein accumulation [75]. phyB suppresses BR signaling, but changes in BR abundance have to our knowledge not been reported. Like cry1, phyB interacts with BZR1 and BES1 to suppress their transcriptional activities [76,77].

Besides these hormone and transcription factor-based mechanisms, photoreceptors also control gene expression by epigenetic changes during de-etiolation. The histone H2A can be exchanged for the variant H2A.Z, which typically results in more compact and therefore less transcribed chromatin, with the help of a protein complex that includes SERRATED LEAVES AND EARLY FLOWERING (SEF / SWC6) and ACTIN-RELATED PROTEIN 6 (ARP6). Photoactivated cry1 and phyB associate with this complex to promote enrichment of H2A.Z on promoters of growth enhancing genes [78,79]. What remains to be clarified is whether these epigenetic changes follow transcriptional reprogramming elicited by PIFs or other transcription factors as observed during shade avoidance and thermomorphogenesis [80,81].

2.2 Timing of hypocotyl elongation during de-etiolation

Daily growth rhythms have been observed in many plant species and in different organs, including the hypocotyl [82]. In experiments where seedlings are germinated in light-dark cycles and transferred to constant conditions, growth is fastest around subjective dusk and slowest around subjective dawn [83]. However, in short day light-dark cycles (16h light, 8h darkness), elongation rhythms differ as growth is fastest around dawn [84]. This growth rhythm depends on circadian expression of *PIFs* as well as light- and clock-regulated PIF activity and stability [85]. The underlying mechanisms are complex, were recently reviewed and define a window of PIF activity around dawn [85].

This co-incidence of high PIF4 and PIF5 expression with low inhibition by the clock or light only exists in short days. In long days (16h light, 8h darkness) by contrast, suppression of PIFs remains strong until the end of the night and the growth peak around dawn is not observed. Over time, this leads to less overall elongation in long day and explains why hypocotyls are longer in short than in long days [85].

3. Light cues regulating hypocotyl growth in photo-autotrophic seedlings

After de-etiolation, seedlings which are growing in light conditions indicative of a favorable light environment largely cease to elongate their hypocotyl. At this stage, seedlings have used up seed reserves and obtain resources for growth and maintenance from photosynthesis.

However, less favorable habitats can induce hypocotyl elongation of photoautotrophic seedlings, especially vegetation proximity (section 3.1), canopy cover (section 3.2), or warm temperatures (section 4). In addition, these elongation responses are themselves modified by other environmental conditions including biotic and abiotic stresses such as herbivory or salinity (section 5).

The same photoreceptors described above control hypocotyl growth of photoautotrophic seedlings when vegetation proximity or shade alter the light spectrum and hence photoreceptor signaling. Moreover, in addition to being a photoreceptor phyB also contributes to temperature sensing (section 4).

3.1 Neighbor detection

Green plant tissue effectively absorbs red, blue and UV light, but most of the far-red is transmitted or reflected. Plants sense the proximity to potential competitors as the reflected light is enriched in far red light resulting in a low ratio of red to far red light (R/FR) (Figure 1A) [86]. The primary photoreceptor sensing neighbor proximity is phyB, which is activated by red and inactivated by far red light, making it sensitive to R/FR. While during de-etiolation the strong activity of phyA leads to suppression of hypocotyl elongation by FR light, in photoautotrophic seedlings phyA levels are much reduced and hypocotyl elongation is primarily controlled by phyB. Therefore, in contrast to the de-etiolation scenario, additional FR light enhances hypocotyl elongation rather than suppressing it. Importantly, the overall photosynthetically active radiation (PAR) is not affected by this neighbor detection scenario [86].

Reduced phyB activity in low R/FR releases suppression of PIFs. In photoautotrophic seedlings, the dominating PIFs controlling low R/FR-induced hypocotyl elongation are PIF7 with contributions of PIF4 and PIF5, while PIF1 and PIF3 play a minor role [87,88]. Low R/FR inhibits active phyB dependent phosphorylation and degradation of PIF4 and 5 [64]. phyB does not appear to strongly regulate PIF7 protein abundance. Rather, it controls the PIF7 phosphorylation state which affects PIF7 subnuclear localization and propensity to form nuclear speckles [80,88]. The key role of PIFs in cotyledons is to induce expression of *YUCCA* genes *YUC* 2, 5, 8 and 9, which encode flavin mono-oxygenases that catalyze the rate-limiting step in auxin biosynthesis [88–92]. Auxin is subsequently transported from the main site of synthesis – the cotyledons – to the hypocotyl where it induces elongation. The PIN auxin transporters PIN3, 4 and 7 are required for auxin transport and their expression is also induced in low R/FR [90,91,93]. Within 1 hour auxin reaches the hypocotyl – especially the epidermis –where it triggers elongation growth [93,94]. While low R/FR sensing and auxin production primarily occur in cotyledons and young leaves, auxin homeostasis also occurs in the hypocotyl [95].

Other hormone pathways, especially GA and BR, also control low R/FR induced hypocotyl elongation. Unlike auxin, changes in their abundance are not always clearly established. The abundance of GA

increases slightly in very low R/FR [96] but GA is required for elongation in low R/FR [97]. Signaling downstream of GA is controlled by R/FR by down-regulating DELLA protein abundance leading to higher PIF activity [97,98]. BR levels do not increase in response to low R/FR [96]. Rather, BR signaling is regulated, and this is downstream of auxin since auxin leads to up-regulation of BZR1 [94].

As in etiolated seedlings, PIFs, BZR1/BES1 and ARFs likely work in concert as promoters of differentially regulated genes are bound by PIF4, PIF5, ARF6 and BZR1 [91]. Moreover, an additional transcription factor, CYCLING DOF FACTOR 2 (CDF2) functions in concert with PIFs to contribute to hypocotyl growth regulation [99].

Low R/FR leads to highly distinct expression reprogramming in hypocotyls versus cotyledons [91,92]. Auxin emerged as a key player in these organ specific responses: Expression of auxin responsive genes is similar in both organs at first but then becomes more sustained in hypocotyls [91]. In cotyledons, PIFs induce auxin synthesis, while in the hypocotyl PIF-mediated gene expression may require concerted action with ARFs [92]. Moreover, many genes associated with cell growth are selectively up-regulated in hypocotyls in a PIF-dependent manner [91,92].

PIF target genes induced in low R/FR include inhibitors of the growth response such as HFR1, PHYTOCHROME INTERACTING FACTOR 3 -LIKE 1 (PIL1) and PHY RAPIDLY REGULATED 1 and 2 proteins [64]. HFR1 directly inhibits PIFs by forming non-DNA binding heterodimers thereby constituting a negative feedback loop, which are typical in growth regulatory mechanisms [100,101]. Interestingly, in the shade tolerant plant *Cardamine hirsuta*, phyA and HFR1 activity are stronger than in Arabidopsis explaining at least in part why in this species low R/FR does not elicit strong hypocotyl elongation [102,103].

COP1 and SPA proteins are required for elongation in low R/FR. They are particularly important for the induction of auxin biosynthetic and other growth-related genes while dispensable for the induction of *PIL1* and *HFR1* expression [104]. Moreover, it is likely that $CUL4^{COP1/SPA}$ in low R/FR, as in canopy shade, promotes the degradation PIF inhibitors (e.g. HFR1, PIL1) allowing increased elongation (see section 3.2).

3.2 canopy shade

In contrast to neighbor detection, canopy cover reduces not only the R/FR ratio but also overall PAR, including intensity of red, blue and UV light.

Since responses to the R/FR ratio and LB are mediated by different photoreceptors, it can be useful to study blue depletion alone, even though blue depletion (‘LB’ for low blue) does not have a known equivalent in nature. LB, like low R/FR, promotes hypocotyl elongation by cell elongation [105]. We will first cover mechanisms underlying the LB response before discussing what happens in more realistic canopy shade conditions.

The relevant photoreceptors for regulating hypocotyl elongation in response to LB are cry1 and cry2, which are less activated in this condition [54,106]. Signaling components regulated by cry1 overlap but are not identical to those operating in low R/FR. PIF4 and PIF5 are required for elongation in LB while with PIF7 plays a minor role [54,107]. Light activated cryptochromes were proposed to directly interact with and inhibit PIF4 and PIF5, an inhibition that is released in LB allowing these PIFs to promote hypocotyl elongation [53,54]. *PIF4* and *PIF5* transcript expression increases in LB leading to enhanced protein accumulation [54,108]. PIFs are responsible for substantial transcriptional changes in LB, including targets involved in elongation growth and cell wall modification [54,92]. However, compared to the low R/FR response, their contribution to transcriptional reprogramming is less pervasive [92].

Auxin synthesis and transport are necessary for hypocotyl elongation in LB [54,105,106]. However, in contrast to low R/FR, auxin levels do not increase, known auxin regulated genes are less affected by LB than low R/FR, and transcriptome-wide changes are less impaired in an auxin biosynthetic mutant [54,92]. A possible explanation for these seemingly contradictory results is that LB leads to a modest, temporally, or spatially restricted increase in auxin production that was not detected.

The role of GA for elongation in LB appears reminiscent of its role in low R/FR: blocking GA synthesis reduces hypocotyl elongation, and DELLAs inhibit part of the elongation response [97]. BR response genes are up-regulated in LB [92]. Auxin and BR are both required for full hypocotyl elongation in LB as inhibition of either BR or auxin signaling leads to partial suppression of elongation in LB while both hormones contribute additively to hypocotyl elongation [105,109]. One explanation for this additive response is that auxin and BR signaling up-regulate mostly different genes of the *XTH* (endotransglucosylase/hydrolase) family, which mediate cell elongation [105].

Canopy shade is simulated in the lab either by combining low R/FR with LB or with green filters and white light containing FR. Both conditions provide good mimics of real shade [109,110]. As expected from specific light treatments (LB or LRFR) in canopy shade PIF4, PIF5 and PIF7 all contribute strongly to the response [109,111]. COP1 accumulates in the nucleus in response to canopy shade [112]. It is likely that the combined effects of regulated PIF and $CUL4^{COP1/SPA}$ activity triggers transcriptional reprogramming required for hypocotyl elongation [111].

Two differences in PIF regulation can explain the enhanced elongation observed in canopy shade compared to low R/FR. Firstly, PIF stability is higher in canopy shade [109], secondly, HFR1 levels are lower due to degradation by COP1 [111]. PIF abundance however depends on the duration of shade exposure. In persistent canopy shade, lasting over 1 day, PIF4 protein abundance declines in cotyledon mesophyll cells, but remains high in the hypocotyl vasculature, where it probably regulates downstream auxin signaling components allowing responses to persistent shade in the absence of high auxin levels (see below) [113].

As in low R/FR and low blue, auxin plays an important role in hypocotyl elongation in canopy shade [113]. Initially, canopy shade causes a burst in auxin production, but auxin concentrations eventually drop to the pre-shaded levels, even though rapid hypocotyl elongation continues. This apparent paradox can be explained by an increase in auxin sensitivity over time [113,114]. Further links between auxin and shade signaling have recently been reviewed in depth [115]. GA signaling is enhanced by canopy shade due to degradation of DELLAs by COP1 [110] and DELLA sequestration by BBX24 [116]. Significant changes in BR levels in response to canopy shade have not been observed in either cotyledons or hypocotyls [117]. BR signaling however is regulated in canopy shade in an organ-specific manner: BZR1 and BES1 accumulate in nuclei in the hypocotyl, while their nuclear abundance decreases in cotyledons, hence their nuclear abundance correlates with growth regulation since cotyledons expansion is reduced in shade. This opposite behavior in the two organs depends on both COP1 and PIF4 and likely involves degradation of BZR1/BES1 by COP1 in the cotyledons but not the hypocotyl [117].

3.3 Role of UV light

Direct sunlight includes UV-B light (280-315 nm) which suppresses hypocotyl elongation [118]. UV-B can cause hypocotyl growth inhibition due to DNA damage [118,119], therefore the experimental settings to study its photomorphogenic effects involve very low irradiances of UV-B to avoid the confounding effects of UV-B stress [119]. In these conditions, UV-B regulated hypocotyl elongation depends on the UV-B receptor UVR8 [120].

UVR8 exists as a dimer when inactive and upon UVB light perception by specific tryptophane residues it monomerizes and localizes to the nucleus, where it exerts its action [118,119]. Therefore, the dynamics of monomerization and dimerization is important for its activity and inactivation of the photoreceptor through re-dimerization also regulates UV-B signaling [119].

Like the cryptochromes and phytochromes, activated UVR8 reprograms gene expression by inhibiting $CUL4^{COP1/SPA}$ and through less clearly established mechanisms by directly interacting with transcription factors [119,121]. $CUL4^{COP1/SPA}$ inhibition does not involve a light-regulated interaction with SPA proteins but rather, UVR8-COP1 interaction: The C-terminus of UVR8 binds strongly to the substrate binding site of COP1 thereby preventing degradation of photomorphogenesis promoting factors, including HY5, its co-activators BBX21 and BBX22 and HFR1 [50,119,121–123]. Stabilization of HY5 contributes to higher HY5 gene expression as this transcription factor promotes its own expression. HY5-dependent expression reprogramming makes a large contribution to UV-B-induced transcriptional changes [50,119,121]

Additional mechanisms contribute to the UV-B transcriptional response. UVR8 promotes PIF4 and PIF5 degradation, reducing their occupancy on target gene promoters, including *YUC8*, *IAA19* and *IAA29* [124,125]. The details underlying this process are is still unknown, but based on what happens in de-etiolated seedlings it could involve inhibition of COP1-mediated protection of PIFs [124]. Stabilization of HFR1 in UV-B further contributes to reduced PIF activity. UVR8 also interacts directly with transcription factors. One example is WRK36 which inhibits HY5 transcription. Interaction of UVR8 with WRK36 releases this inhibition [126]. In addition, nuclear UVR8 directly interacts with BES1 and its interactor BIM1, inhibiting both [127]. However, nuclear-localized photoactivated UVR8 is not sufficient to control gene expression in a *cop1* mutant background, suggesting that COP1 is also required for these UVB-triggered responses [119].

4. The effect of temperature and flooding on hypocotyl elongation

In de-etiolated Arabidopsis seedlings, increasing the temperature up to 30°C promotes hypocotyl growth, while higher temperatures cause stress and inhibit elongation. Here we will focus on the effects on growth caused by mildly elevated temperatures, or thermomorphogenesis.

The physiological relevance of hypocotyl growth promotion by warm temperature is not as intuitive as during shade avoidance. Two hypotheses which are not mutually exclusive were proposed. First, elongating the hypocotyl may, as shown for petioles, promote cooling capacity [128]. Second, at warmer temperature respiration increases and photosynthetic capacity decreases meaning that in crowded environments shading is even more problematic [129]. This second hypothesis provides a possible explanation for the highly similar pathways eliciting hypocotyl growth by warmth and shade [129]. Moreover, it explains the hypocotyl growth response in various combinations of temperature and light conditions (section 5.1).

Defining temperature sensors is challenging given that essentially all biochemical reactions are temperature regulated and the properties of biological material such as membranes change with temperature [130,131]. Three sensors controlling thermomorphogenesis have been described: phyB and ELF3 whose activities decrease in warm temperatures and PIF7 which is more abundant in warm temperatures [130,131]. These three temperature-regulated processes all contribute to enhanced PIF activity during thermomorphogenesis.

In the absence of light, phyB thermodynamically reverts to the inactive conformation in a process called dark or thermal reversion. Increasing rates of thermal reversion at higher temperature reduce the pool of active phyB, thus promoting hypocotyl elongation [132,133]. Moreover, warm temperatures promote disassembly of nuclear photobodies, inhibiting phyB activity [134]. This may in part be due to

the activity of PCH1, which stabilizes active phyB in nuclear bodies. Indeed, at warm temperatures PCH1 expression and stability are reduced thereby further decreasing the stability of active phyB [135].

ELF3, a component of the transcriptional inhibitory Evening Complex (EC), is inactivated in warm temperature by forming aggregates by liquid-liquid phase separation through a prion-like domain present in its structure [136]. In addition, two E3 ligases, namely XBAT31 and XBAT35 degrade ELF3 in response to warm temperatures [137,138]. This results in decreased EC activity and hence increased expression of PIF4, a central regulator of thermomorphogenesis [130,139]. In addition, HEAT SHOCK PROTEIN 90 (HSP90) has been implicated in promoting degradation of ELF3 at elevated temperature [140].

PIF7 is essential for temperature induced hypocotyl elongation and PIF7 protein levels increase rapidly in warm temperatures due to enhanced translation, a process regulated by an RNA hairpin in its 5' UTR [141,142]. PIF4 and PIF7 share many growth promoting target genes and loss-of-function mutants have similar phenotypes, leading to the proposal that they may act as heterodimers to promote hypocotyl elongation in warm conditions. However, in some situations (combining warmth and low R/FR), PIF7 can function in the absence of PIF4 indicating that the PIF4-PIF7 heterodimer hypothesis may only explain growth regulation in some conditions [143].

In addition to the temperature-regulated mechanisms controlling PIF4 and PIF7 accumulation described above, several regulators of PIF4 abundance have thermomorphogenesis defects that can be attributed to altered PIF4 levels, namely BLADE-ON-PETIOLE (BOP), HMR and REGULATOR OF CHLOROPLAST BIOGENESIS (RCB) [144–146]. While these studies emphasize the importance of PIF4, it is unclear whether these mechanisms specifically contribute to temperature-regulated hypocotyl elongation.

Warm temperature also increases COP1 nuclear levels, possibly due to inactivation of phyB as in the case of shade avoidance [112,147]. This promotes COP1-mediated degradation of HY5 and DELLAs before a detectable change in GA levels [147]. This reduces the inhibitory activity of DELLAs on PIFs [110]. Moreover, HY5 and PIF4 bind to shared target genes but regulate them in opposite ways. By reducing HY5 levels, PIF4 can more effectively enhance expression of growth promoting genes [110,131,148,149]. $CUL4^{CDD}$ is also required for warmth-induced hypocotyl elongation through a process involving the regulation of PIF4 protein abundance [110,131,148,149].

Auxin, BR and GA are required for promoting hypocotyl elongation during thermomorphogenesis. IAA and GA levels increase [53,110,150] which can be explained by the transcriptional responses discussed above (e.g. PIF-mediated *YUC* expression) [130,150,151]. As in shade, increased auxin levels in hypocotyls depend on transport from the cotyledons [152]. While BR levels do not change significantly in Arabidopsis in response to warmth [117], the transcriptional response and measurements in *Brassica rapa* suggest that there may be small changes [117]. BR signaling is enhanced by promoting nuclear accumulation of BZR1 [150,153]. Temperature regulation of BES1 abundance depends on COP1 and is opposite in hypocotyls and cotyledons, reflecting the opposite growth responses of these organs at elevated temperature and in shade [117]. In addition to these transcriptional responses enhancing the BR pathway in elongating hypocotyls, temperature-regulated phosphorylation of a MAP4K also contributes to a stronger BR response in Arabidopsis and wheat [154]. How this protein kinase connects to the BR pathway remains to be uncovered but importantly this pathway regulates temperature-controlled elongation in dicots and monocots [154].

Long-term transcriptional reprogramming in response to warm temperatures involves epigenetic changes. The abundance of the repressive histone variant H2A.Z in the promoter of the temperature-responsive genes decreases at warmer temperatures. This is at least partially regulated by interaction

between PIF4 and the chromatin remodeling complex INO80-EIN6 ENHANCER [81]. Moreover, INO80-C is required for the deposition of H3K4me3 marks, associated with active transcription, in PIF4-target gene promoters. Similar events take place in response to PIF7 regulated gene expression in low R/FR [80,155]. Other histone deacetylases, demethylases and chromatin remodeling factors are also involved in thermomorphogenesis [156].

Another condition which causes hypocotyl elongation is submergence, and this is mediated by ethylene. Notably, ethylene has opposing roles in darkness and in the light as it inhibits elongation in etiolated seedlings but promotes it in light-grown seedlings. PIF3 is the most relevant PIF for elongation in this condition and its abundance increases in response to ethylene or submergence [157]. The elicited transcriptional changes resembles those in the response to shade [158]. Hypocotyl elongation in shade however does not require ethylene sensing, in contrast to petiole elongation [106]. It remains to be clarified how the ethylene signaling network connects to shade regulated mechanisms in different organs.

5. Integration of hypocotyl elongation signals with biotic and abiotic stresses

The mechanisms described so far demonstrate that the same signaling components are regulated by multiple different environmental cues which can affect elongation in either the same or opposing directions. Here we describe how such signals are integrated.

5.1 Light cues and temperature

Combination of light and temperature cues regulate hypocotyl elongation in an integrated fashion [129]. Growth-promoting effects of temperature are reduced by higher light irradiances [133]. Conversely, shade-induced elongation is enhanced at warm temperatures [129]. This strong additivity requires PIF7, auxin and some unidentified factors [143].

5.2 Effect of UV light on shade and temperature induced elongation

UV-B counteracts hypocotyl elongation elicited by other environmental cues. In canopy shade, UV-B is filtered out by vegetation [86]. Nevertheless, plants experience sun flecks due to perturbations in the canopy and changes in solar elevation. UVR8 perceives these sun flecks and inhibits hypocotyl elongation [159]. Different mechanisms are relevant depending on the duration of UV-B. PIF5 is rapidly degraded in the presence of UV-B as UVR8 lifts stabilization of PIF5 by COP1 [124,159]. In addition, UVR8 suppresses PIF activity by promoting HFR1 accumulation [123]. In the longer term, UV-B enhances HY5 and HYH expression, resulting in the suppression of growth promoting genes and GA signaling [160]. Thermomorphogenesis is also inhibited by UV-B. Photoactivated UVR8 inhibits accumulation of PIF4, thereby reducing the expression of growth-promoting genes and hypocotyl elongation [161].

5.3 Interaction of water availability with light and temperature cues

Plant growth critically depends on water availability and signaling networks regulate elongation before water shortage puts cell integrity at risk [162,163]. This conservative growth strategy is presumably favorable for long-term survival.

Addition of PEG to the growth media (to decrease the osmotic potential) is frequently used to simulate drought in the lab. In etiolated seedlings, hypocotyl elongation decreases by half on PEG when the osmotic potential is reduced from -0,1MPa to -0,5MPa [164]. Similar and more negative water potentials also suppress elongation in light grown-photoautotrophic seedlings without affecting cotyledon expansion, and this effect is particularly strong in canopy shade [163].

Drought increases the concentration of the hormone abscisic acid (ABA) which suppresses hypocotyl elongation [165] by interacting with the classical hypocotyl growth promoting pathways. ABA treatment of etiolated seedlings inhibits the H+-ATPase by limiting the phosphorylation of the penultimate Thr [165] (see section 6). In canopy shade, PEG addition reduces PIF3, 4 and 5 expression as well as PIF4 protein stability, causing reduced expression of typical growth promoting PIF target genes [163]. Conversely, shade reduces ABA related gene expression in hypocotyls, indicating a reciprocal negative relationship between shade and drought signaling [91]. Thermomorphogenesis is also suppressed by ABA application, which is an interesting combination as high temperatures and drought often occur simultaneously [166].

Interestingly, the antagonism of shade or temperature signaling and ABA has been exploited in the search for possibilities of overcoming growth - drought resistance trade-offs. Simultaneous over-expression of the drought regulator DEHYDRATION RESPONSE ELEMENT B1A (DREB1A) and PIF4 resulted in plants which are more drought resistant than the WT without the growth deficit of sole DREB1A over-expressors [167].

High salt concentrations reduce water availability and have a toxic effect on plant tissues [168]. It is therefore a similar but not identical abiotic stress as drought. Salinity inhibits hypocotyl elongation triggered in neighbor shade in Arabidopsis and other species [162] via an increase in ABA and ABA signaling [168] and suppression of PIF activity [162]. The mechanism is based on regulation of BR signaling by ABA. When confronted with shade and salinity, the interaction of ABA signaling with PIF dependent BR signaling suppresses BES1 and the concerted activation of targets with PIF4 and PIF5 [162].

Additional mechanisms for the interaction of salinity and shade have been reported. The circadian clock component ELF3 may regulate the ABA – shade signaling interaction, but mechanisms are unknown [162]. Moreover, two kinases, FERONIA and SALT OVERLY SENSITIVE 2 regulate light responses by phosphorylation of phyB and PIFs, respectively, though the relevance for hypocotyl elongation has not been studied in detail [169–171].

5.4 interaction of defense signals and elongation signals

Like drought and salinity, pathogen attack frequently reduces plant growth [172]. The mechanism underlying growth inhibition and its interaction with growth promoting cues is best understood in the context of herbivore attack, which we cover here. Jasmonic acid (JA) is the primary hormone induced by herbivory, which triggers defense reactions as well as growth inhibition. JA treatment is frequently used to activate the signaling and transcriptional response elicited by herbivore attack [172]. A trade-off between growth and defense is therefore inherent in JA signaling. The ecological significance of this trade-off has been the focus of many debates [172]. Balancing usage of resources may be one reason, but balancing advantages and disadvantages of tall growth habits could be more important: While being tall helps for access to sunlight in a competition scenario, it probably also makes plants more prone to herbivore attack [172]. In white light, hypocotyl length suppression by JA treatment is well documented [98,173,174], while in response to neighbor proximity, either the shade signal [98] or the defense signal can dominate [175], likely depending on intensities or time of light, shade and JA signal.

In the absence of JA, defense responses are inhibited by Jasmonate ZIM-domain (JAZ) proteins as they suppress several MYC transcription factors which induce transcriptional defense responses. Upon JA application, JAZs are degraded, and MYCs are de-repressed. This impacts hypocotyl growth control and its balance with defense mechanisms in several ways. Binding of JAZ to DELLAs weakens

the interaction of DELLAs with PIFs. Therefore, in the absence of JA, JAZ decreases DELLA-mediated PIF suppression while in response to JA, degradation of JAZ makes DELLAs more available to sequester PIFs [173]. This mechanism may also explain why JA does not suppress hypocotyl elongation in some shade conditions: degradation of DELLAs in the shade releases not only PIFs to promote growth but also JAZ proteins to suppress defense [98]. Moreover, low R/FR promotes PIF-mediated expression of a JA catabolizing enzyme thereby weakening defense responses and mitigating the effects of JA on neighbor-induced growth. Whether this mechanism contributes to hypocotyl growth control remains to be established [176]. Another proposed mechanism involves FAR-RED ELONGATED HYPOCOTYLS 3 (FHY3) and FAR-RED IMPAIRED RESPONSE 1 (FAR1) which regulate growth and defense by modulating both PIF and MYC-regulated transcription [175]. A third mechanism is based on HY5. In the presence of JA, MYCs promote HY5 expression by directly binding to its promoter, which leads to positive regulation of photomorphogenesis and shorter hypocotyls [174].

Additional mechanisms underlying JA-mediated growth and defense and its interaction with shade pathways have recently been summarized [177]. Taken together, the defense and elongation growth signaling pathways are highly connected to balance growth and defense reactions [177,178].

6. Growth processes

The signaling pathways described so far converge on the regulation of elongation suppressing transcriptional regulators (e.g. HY5, HYH, BBX20-21) as well as a set of interdependent transcription factors that promote elongation, namely PIFs, BZR1, BES1 ARF6 and ARF8. The genes controlled by these transcription factors either carry out growth-related functions quite directly or through downstream transcription factors (Figure 2B) [89]. The regulated growth mechanisms follow similar principles, even though global transcriptomics changes differ to some extent depending on the growth-modulating stimulus [92]. We first describe general concepts of hypocotyl cell elongation growth, how they connect to signaling networks described above, and finally comment on the resources that provide material for elongation.

6.1 Cellular processes underlying hypocotyl elongation

Hypocotyl elongation does not require new cell division of epidermal and cortex cells but relies on elongation of the cells present since the end of embryogenesis [90,179]. The spatial dynamics of elongation in etiolated seedlings has been described as an acropetal wave: elongation growth speeds up first at the base of the hypocotyl and then moves towards the cotyledons [22,179]. In light grown seedlings hypocotyl growth promotion occurs through preferential elongation of the cells in the middle of the hypocotyl [90,105]. Outwards facing epidermal cell walls are thicker than those of inner tissues both in etiolated and light-grown seedlings. In etiolated seedlings, this cell wall is very thick at the time of emergence from the seed and becomes thinner during fast growth while cell wall material deposition occurs later. In light-grown seedlings cell wall thickness rather increases with time but the outer epidermal wall also becomes thinner during fast growth [180]. This shows that although deposition of new cell wall material is ultimately required to allow growth, deposition of such material can be uncoupled from fast hypocotyl elongation [181,182]. Periods of fast growth depend on prior deposition of cell wall material [183,184], while during the actual fast elongation phase, re-organization of existing cell wall material (e.g. cellulose and pectin) contributes more to cell elongation than new synthesis [183,184].

Cell elongation requires high turgor pressure, but current evidence supports the idea that growth is regulated at the level of cell wall loosening. The primary cell wall of hypocotyl cell expands in the

diffuse mode (as opposed to tip growth observed e.g. in pollen tubes). The cell wall is a complex material and its structure as well as the key elements regulating its irreversible expansion are still debated [185–187]. Nevertheless, there is substantial evidence for a critical role of the "Acid Growth Theory" in connecting auxin-mediated growth promotion with loosening of the primary cell wall and hence hypocotyl elongation [185,188,189]. Importantly, the links between apoplasm acidification and cellular growth are not identical in shoots and roots, and we specifically discuss what occurs in hypocotyls [185,189]. Acidification is caused by activation of plasma membrane H+ ATPases through phosphorylation at their penultimate Thr residues [20,190]. AHA phosphorylation is observed within 10 seconds of auxin treatment [191]. The plasma membrane localized TRANSMEMBRANE-KINASE (TMK)1 and TMK4 are required for this fast AHA phosphorylation [191]. Maintenance of AHA phosphorylation state then requires inhibition of the type 2C protein phosphatase PP2C-D, which de-phosphorylates H+ATPases, [192]. This inhibition is carried out by SMALL AUXIN UP-RNA (SAUR) proteins (e.g. SAUR19). Expression of SAURs is induced by auxin via the classical nuclear localized auxin receptors TRANSPORT INHIBITOR RESPONSE 1 (TIR1) and AUXIN SIGNALING F BOX PROTEINs (AFBs). SAUR19 binds to and inactivates PP2C-D, preventing de-phosphorylation of H+ ATPases [192,193]. A substantial body of evidence supports the notion that this regulatory mechanism primarily operates in the epidermis which limits the hypocotyl growth potential [94,194].

Cell wall acidification leads to cell wall loosening by different mechanisms. Members of a group of non-enzymatic cell wall proteins, expansins, are induced (gene expression as well as activity) in response to auxin and the concomitant pH drop. They likely act by weakening non-covalent links between cell wall polymers, making the cell wall more flexible [186,187]. Several groups of cell wall proteins can increase cell wall loosening by enzymatic action, acting on different cell wall polymers. Xyloglucan endotransglycolases/hydrolases (XTHs) cut xyloglucan polymers, though their effect on measurable cell wall extensibility may be indirect [186]. Pectin modifying enzymes can also affect cell wall flexibility and elongation, such as demethylesterification which enhances elongation capacity [183,186,195,196], and higher activity of pectin degrading the enzymes POLYGALACTURONASE INVOLVED IN EXPANSION (PGX) family can increase cell expansion by shortening pectin polymers [197]. Glycoproteins in the cell wall are also important for regulating cell wall extensibility, such as arabinogalactan proteins (AGPs) and extensins [196].

Anisotropic growth is coordinated by the cytoskeleton, namely microtubules, and is not directly auxin regulated [198]. In quickly elongating hypocotyl cells, an increase in transverse cortical microtubules is observed, which directs cellulose deposition [199], allowing elongation in the longitudinal direction, perpendicular to the direction of microtubule and cellulose fiber orientation [186]. Therefore, re-organization of the microtubule cytoskeleton is necessary for elongation and proteins involved in microtubule stability regulation also affect hypocotyl elongation [29,200–202]. The importance of microtubule orientation during rapid hypocotyl elongation was established genetically [203]. In addition to the role of cellulose microfibrils in orienting hypocotyl epidermal cell growth, pectins have also been implicated in the regulation of this anisotropic growth process [204].

6.2 Regulation of cellular growth by signaling pathways

While auxin synthesis is regulated by light and temperature cues and is the hallmark hormone to cause cell wall acidification, many other signaling component impinge on this pathway. SAUR gene expression can be directly up-regulated by binding of PIFs, BZR1 and ARFs to their promoters [30,40] and they are therefore not only up-regulated by auxin signaling but also by BR and GA [190,205]. JA on the other hand suppresses *SAUR* expression, by suppressing the action of PIFs [173]. Growth repression by ABA involves de-phosphorylation of H+-ATPases and suppression of *SAUR* transcription by two drought induced transcription factors, Arabidopsis zinc-finger protein 1 (AZF1) and AZF2 which bind directly to promoters of *SAUR* genes [165,206].

Cell wall loosening proteins are also directly transcriptionally regulated. Expansins are upregulated in many growth-promoting conditions [54,207], some are directly bound by PIFs at their promoters [89] and for EXPANSIN 8 (EXP8) there is evidence for direct binding by PIF4, BZR1 and ARF6 [30]. Expression of many *XTH* genes is correlated with hypocotyl elongation in response to shade in a PIF dependent manner [54,89,91,105,112]. The promoters of several genes which encode pectin modifying enzymes are directly bound by PIF5 [89]. Pectin modification is also involved in cell wall stiffening and hypocotyl thickening in response to ethylene, where hypocotyl expansion is reduced [208]. A role for AGPs as likely PIF targets has been proposed during low R/FR-induced hypocotyl elongation, although available results suggest a complex role of this large gene family [91,196]. Further investigations are required to understand the distinct contribution of different AGPs. Finally, several extensins are up-regulated in response to LB in a PIF4/5 dependent manner [54]. Identifying how each of these groups of proteins regulates different aspects of cell wall extensibility remains an important challenge to better understand environmentally-controlled hypocotyl elongation.

Two microtubule associated proteins of the WAVE-DAMPENED2-LIKE (WDL) protein family negatively affect hypocotyl elongation by stabilizing cortical microtubules and preventing their transverse orientation. WDL3 is degraded by $CUL4^{COP1\text{-}SPA}$ in darkness, allowing more elongation than in the light where it is more abundant [202]. WDL5 is up-regulated by ethylene in de-etiolated seedlings to shorten hypocotyls [201]. Moreover, accumulation of the microtubule associated protein, SPIRAL1 (SPR1) is regulated by PIF4 during thermomorphogenesis. SPR1 promotes transverse orientation of microtubules and is required for proper hypocotyl elongation during thermomorphogenesis [209]. While the role of microtubules in anisotropic hypocotyl cell growth is clear we lack an integrated view of the multiple actors regulating this process.

Cellulose synthases are regulated by the light environment transcriptionally and post-translationally. In response to low R/FR for instance, ‘cellulose biosynthetic process’ is an up-regulated GO term in hypocotyls [92]. In addition, several photoreceptor mutants have altered cellulose content and phyB clearly plays a role in regulating the speed of the cellulose synthase complex [181]. One mechanism of direct CESA regulation has been proposed: BIN2 can phosphorylated and inactivate CELLULOSE SYNTHASE A1 (CESA1). Since BR suppresses BIN2, maintenance of active CESA1 contributes to hypocotyl elongation promotion by BR [210]. Carbon status and cellular metabolism (e,g. starch) can have an impact on CESA phosphorylation as well as localization, therefore it is likely that cellulose synthase activity is controlled by metabolic cues and light signaling [181].

6.3 Resource allocation for hypocotyl elongation

Etiolated seedlings rely on their seed reserves, in particular triacylglycerides (TAGs) which are stored in cotyledons and endosperm to fuel seedling establishment and hypocotyl elongation [211]. Accordingly, mutants impaired in ß-oxidation, the glyoxylate cycle or gluconeogenesis from oxaloacetate have shorter hypocotyls in darkness but this can be rescued by sucrose addition to the growth media [212]. GA is required for TAG mobilization to fuel hypocotyl elongation [211]. In addition to TAGs, storage protein can serve as carbon source for etiolated hypocotyl elongation via an alternative gluconeogenesis pathway [212].

De-etiolating seedlings have not yet fully established their photosynthetic capacity and therefore also rely on seed reserves for their growth and metabolism. During de-etiolation in red or far red light, phytochromes promote reserve mobilization from oil bodies in cotyledons, mainly to establish photosynthetic capacity as hypocotyl elongation is simultaneously slowing down [213].

As hypocotyls are sink organs, resources required to fuel growth in photo-autotrophic seedlings are imported from cotyledons. Regulation of this process was studied during low R/FR-induced hypocotyl

growth promotion. In this situation increased hypocotyl elongation requires sucrose transport mediated by sucrose efflux transporters SWEET11 and SWEET12, and the sucrose – proton symporter SUC2 [214]. Sucrose synthesis may also be regulated in particular the steps directing carbon towards sucrose rather than starch given that mutants unable to accumulate starch have constitutively long hypocotyls [214]. Enhanced carbon allocation to the hypocotyl contributes to cell wall, protein and lipid biogenesis [214]. This includes hypocotyl-specific, PIF-mediated control of plasma-membrane sterol biosynthesis, which was proposed to contribute to additional membrane material required for cellular growth [92].

Low R/FR-induced hypocotyl elongation requires transport of sucrose and auxin from the cotyledons to the hypocotyl. There is a complex yet poorly understood interdependency between these two growth enhancing substances. For example, higher levels of endogenous sucrose leads to enhanced hypocotyl elongation and this response requires PIF7 which is known to regulate auxin biosynthesis [214]. The role of carbon metabolic signals for elongation growth is frequently studied by the addition of sucrose to the growth media. This increases hypocotyl length of de-etiolating seedlings, mainly by lengthening the time window of elongation [215]. Providing additional resources for growth could be one reason, however, PIFs are required for this growth response arguing for a signaling role, where sucrose induces auxin synthesis via PIFs, and therefore elongation [215,216]. Two prominent carbon status signaling players are involved in the effect of sucrose on hypocotyl elongation: trehalose-6-phosphate (T6P) which promotes and SnRK1 which suppresses elongation on sucrose [207]. This sugar signaling mechanism has been implicated in the regulation of PIF4 during temperature-induced hypocotyl elongation [217]. Altogether, even though it is clear that changes in carbon metabolism are needed to provide the building blocks for hypocotyl elongation, its dual role as energy / material source and signaling component is not entirely understood and neither are the mechanisms connecting the effects of sucrose and auxin.

In LB and canopy shade, hypocotyls elongate even though light and therefore assimilated carbon decreases. It is likely that resources are mobilized less from newly assimilated carbon than from pre-existing material as autophagic processes and autophagy markers are up-regulated specifically in response to LB [92]. A functional autophagy pathway is required for elongation in LB more than in low R/FR, while new lipid synthesis is less critical for elongation in LB, than in low R/FR. Therefore, anabolic processes appear to play a larger role to fuel elongation in low R/FR, while catabolic metabolism is more important in LB [92]. Importantly, in canopy shade-simulating conditions a combination of anabolic and catabolic processes is required to promote hypocotyl elongation [92].

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A

BL BL RL FR BL
PHOT1 CRY1 phyB phyA
BL RL FR UV-B
phyB—FR
no nearby plants
neighbour detection low R/FR
canopy shade
1. Etiolated (with / without compaction)
2. De-etiolating seedling
3. Elongation responses of photoautotrophic seedlings to different types of shade
4. Elongation at warm temperature

B

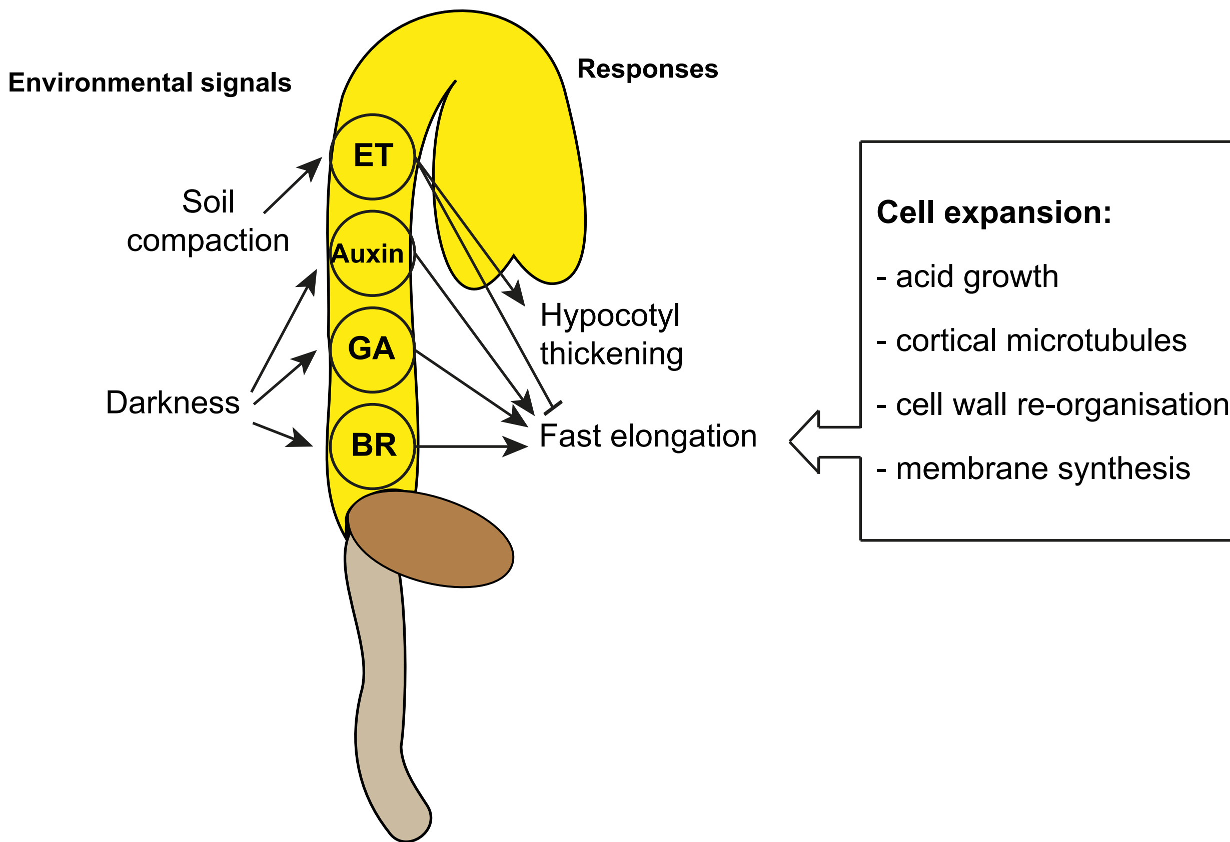
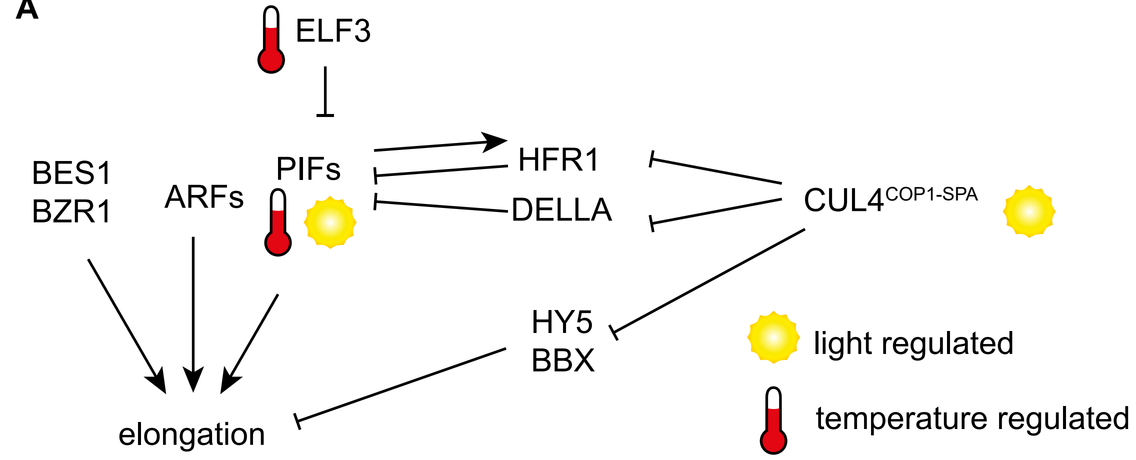
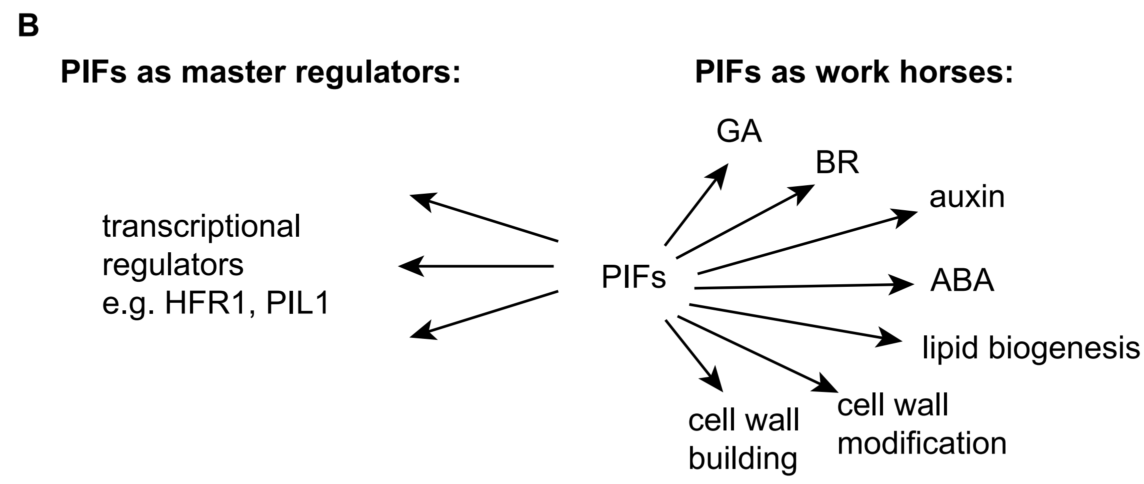


Figure 1: Hypocotyl elongation behaviors at different developmental stages
(A) Hypocotyl growth during different stages of seedling development, discussed in sections 1 to 4. (1) Etiolated seedlings are elongating quickly while growing in the dark. Growth is slowed down and hypocotyls thicken when soil is more compact. (2) Once seedlings reach the soil surface they undergo de-etiolation which includes inhibition of hypocotyl elongation and can be mediated by several photoreceptors and light qualities. (3,4) Fully de-etiolated photo-autotrophic seedlings barely elongate in an open habitat at lower temperatures. However, vegetation proximity or cover and warm temperatures induce hypocotyl elongation in shade-avoiding species. (B) Overview of the major environmental signals and responses of etiolated seedlings and main hormone signaling pathways regulating these. Hypocotyl elongation growth is achieved by anisotropic cell expansion, which is characterized by the processes summarized on the box on the right.





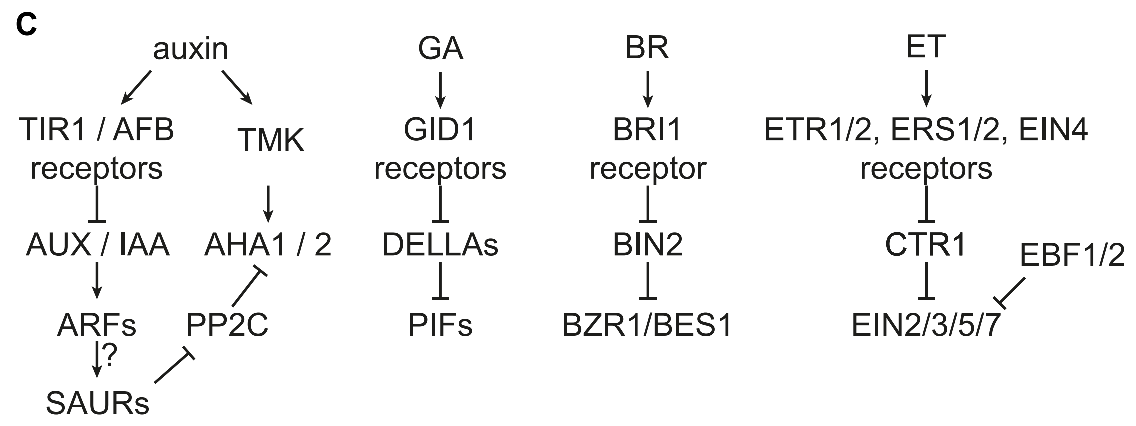


Figure 2: Overview of important signaling mechanisms underlying enviro-mental regulation of hypocotyl elongation.
A: Core signaling network that controls hypocotyl elongation in response to environ-mental cues. Regulated proteins in response to light and temperature changes are highlighted as indicated in the legend. B: PIFs as critical transcription factors for the regulation of hypocotyl elongation, either as master regulators of other transcrip-tional regulators, or as work horses for the expression of diverse growth promoting genes. C: Simplified signaling pathways of the main hormones discussed in this review.