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Dawning of a new era: photomorphogenesis as an integrated molecular network

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Plant growth is shaped by the dynamic integration of environmental, developmental, and metabolic cues. Information from many of these input pathways feeds into the highly connected network of small molecule phytohormones. Signal transduction components for most plant hormones are known and mapping of hormone interactions within the network is well underway. Recent investigations of seedling photomorphogenesis, using well-established physiological and genetic tools in combination with sophisticated application of newer genomic technologies, provide a systems-level view of early seedling development. Factors, such as light, the circadian clock, and organ-specific developmental programs, profoundly influence the hormone network. The integrative approaches described here clarify the mechanisms of signal integration while revealing the flexibility of such relationships.

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Introduction

In plants, physiology and development are inextricably linked: morphogenesis reflects external environmental conditions as much as endogenous cues. Developmental, metabolic, and environmental information is encoded, transported, and ultimately translated into phenotype through a complex network of hormones. Alterations in this hormone network lead to profound changes in plant architecture, a fact highlighted by molecular analysis of agricultural stocks. Strains which can produce high yields in the crowded, low-light growing conditions found in farm plots frequently harbor mutations in hormone biosynthesis or response genes [1,2].

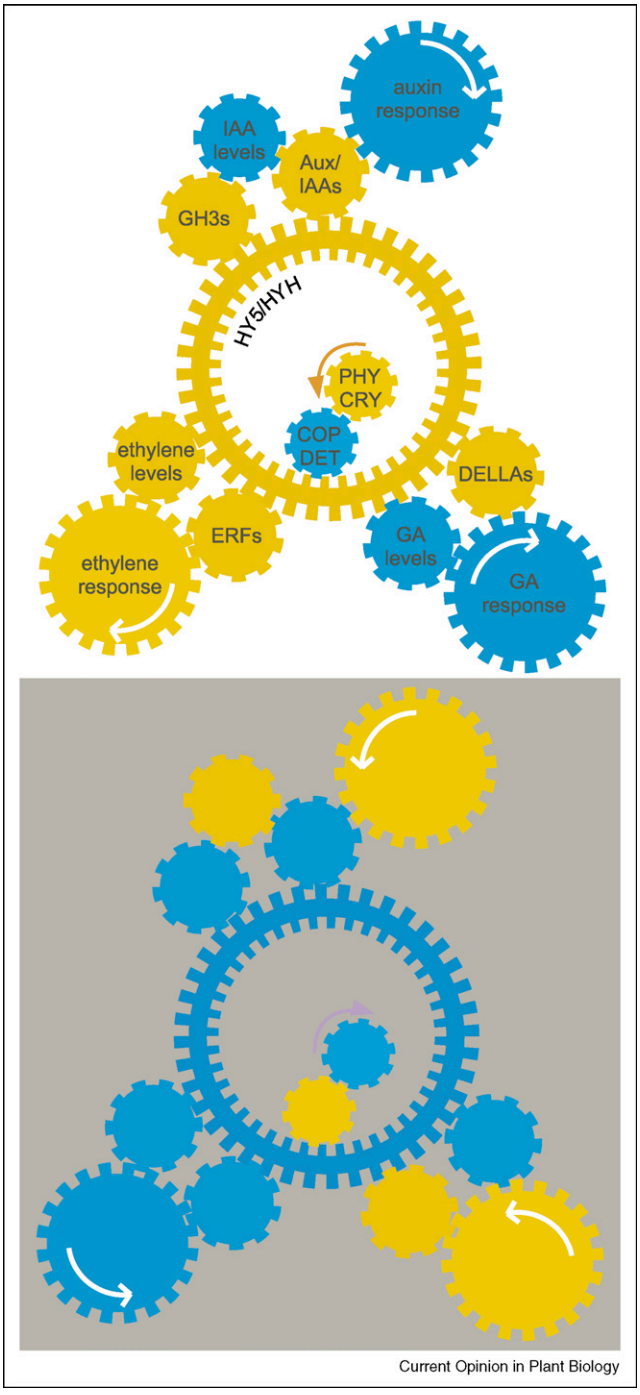
The highly plastic *Arabidopsis* seedling has been an excellent system for dissecting the interaction between internal and external cues during light-directed development (photomorphogenesis) [3]. Light is among the most significant early environmental influences on seedling morphology. Low light environments promote stem elongation while inhibiting cotyledon expansion, whereas higher light levels promote cotyledon growth at the expense of hypocotyl elongation. A number of phytohormones are known to influence the photomorphogenetic program, including gibberellins, auxins, brassinosteroids, cytokinin, and ethylene [3]. The individual pathways controlling levels and response to each hormone are largely known, and, in many cases, the interactions between pathways are beginning to be understood at a molecular level. This review will focus on recent studies investigating how the hormone network interfaces with other factors — such as light, the circadian clock, and organ-specific developmental programs — during photomorphogenesis.

Light and the hormone network

The antagonistic relationship between light response and growth-promoting hormones — such as auxin, gibberellins, and brassinosteroids — has been uncovered through physiological and genetic studies, but the molecular map connecting these pathways has been elusive [3]. In *Arabidopsis* seedlings, light triggers a massive wave of transcriptional re-programming, largely under the control of two sets of photoreceptors: the red/far-red-sensing phytochromes (PHYA-E) and the UV-A/blue-light-sensing cryptochromes (CRY1 and CRY2) [4]. Light stimulation triggers accumulation of the bZIP transcription factor ELONGATED HYPOCOTYL 5 (HY5), which is required for hypocotyl inhibition in all light conditions. CONSTITUTIVE PHOTOMORPHOGENIC 1 (COP1), a ubiquitin ligase, targets HY5 for degradation in the dark. The COP1–HY5 interaction is disrupted in the light, allowing rapid accumulation of HY5 and reduced hypocotyl elongation. Recent studies described below have uncovered new direct links between photoreceptors, HY5 and hormone response.

Many lines of evidence connect the phytohormone auxin to the light response in seedlings. For example, many mutants with reduced auxin response are deetiolated in the dark [5] and both light and auxin responses require a common set of proteins involved in ubiquitin-mediated protein degradation [6]. The *hy5* mutant was one of the first mutants identified with reduced light response [7]. Careful analysis of *hy5* plants revealed that certain aspects of their phenotype, particularly in root architecture, can

Figure 1



A highly simplified representation of molecular connections underlying photomorphogenesis. In this drawing, each gear represents a small molecule, protein or protein family. Gears are identical between panels. Direct linking of gears reflects evidence for direct interaction between the factors. Light perception by phytochrome (PHY) and cryptochrome (CRY) photoreceptors is indicated in the center of each panel. Light stimulation of the photoreceptors during the day is designated with an orange arrow (top panel), while the purple arrow specifies reduced light levels at night (bottom panel). Rotation of gears in a given direction leads to increased levels or activity for some factors (shown in yellow) and decreased levels or activities of other factors (shown in blue).

be attributed to increased auxin response [8]. Recent work on double mutants of *HY5* and *HY5-HOMOLOG* (*HYH*), a *HY5* paralog, reveal a state of constitutive auxin-induced gene expression in the mutants, suggesting that one mechanism for HY5 action is through repression of auxin-induced genes [9•]. Global transcriptome analysis of double mutants exposed to just six hours of white light show overexpression of genes encoding members of the three classic early response gene classes — Aux/IAA transcriptional co-repressors, GH3 auxin conjugating enzymes, and SAURs of unknown function. Interestingly, several genes involved in GA and ethylene metabolism were also mis-regulated in the double mutant. A genome-wide analysis of HY5-binding sites found evidence for direct binding of HY5 to the promoters of multiple genes required for auxin signaling, including Aux/IAAs and members of the Auxin Response Factor family of transcription factors [10•]. In addition, HY5 was found bound to the promoters of some ethylene-responsive factors (ERFs) and members of the DELLA gene family, transcriptional regulators from the ethylene and GA pathways, respectively.

Defects in gibberellin (GA) biosynthesis or response result in light-grown phenotypes in dark-grown seedlings [11]. Elegant work by Achard and colleagues extends this observation to the molecular level by showing phytochrome-mediated light perception regulates levels of repressors of the GA response [12••]. GA is perceived by the GA INSENSITIVE DWARF1 (GID1) receptors which promote degradation of nuclear-localized growth repressors called DELLAs via interaction with the F-box protein SLEEPY1 [13]. In this new work, a DELLA-fusion protein is barely detectable in dark-grown, rapidly elongating hypocotyl cells, while it rapidly accumulates in the same cells following exposure to light [12••]. Consistent with this observation, light inhibition of hypocotyl growth is reduced in quadruple DELLA mutants. The authors also show that changes in DELLA levels are coincident with local transcriptional effects on genes involved in GA metabolism, suggesting that the effects on DELLA proteins are a downstream effect of phytochrome-mediated changes in bioactive GA levels. PHYTOCHROME INTERACTING FACTOR 5 (PIF5), a light-labile phytochrome-interacting transcription factor, inhibits seed germination by regulating the transcription

For example, light activation of the photoreceptors leads to inhibition of the COP/DET/FUS repressors of photomorphogenesis, which in turn, leads to increased levels of HY5 and HYH transcription factors. HY5 and HYH turn on expression of genes encoding GH3 and Aux/IAA proteins which reduce auxin (IAA) levels and signaling, respectively, inhibiting auxin response. Increased HY5 levels also lead to decreased response to gibberellins (GA) and increased ethylene response. For details, please refer to the text. The circadian clock and organ-specific factors push gears into and out of contact with other gears, creating outputs fine-tuned for specific conditions. This is analogous to the way a shift stick alters the arrangement of engaged gears in a transmission as the driver changes from one gear to another.

of genes involved in GA metabolism and signal transduction [14,15•]. PIF5 directly binds to the promoters of *GA INSENSITIVE* (*GAI*) and *REPRESSOR OF ga1* (*RGA1*), two genes encoding DELLA repressors, but its effects on genes involved in GA metabolism appear to be indirect [15•]. PIF5, or other related transcription factors, may play a similar role in regulation of GA response during seedling photomorphogenesis.

A simplified model for these interactions between light and hormones is shown in Figure 1. Additional detailed temporal analysis of early timepoints following light exposure should elucidate the chronology of light signaling and hormone responses. The effectiveness of such an approach has already been demonstrated for dissection of timing and contribution of various photoreceptors [16]. It would be interesting to examine the timing of the disappearance of DELLAs in the double mutant to test for interactions between the auxin and GA pathways at these early timepoints. Moreover, pinpointing where in the plant body the increased auxin response is detected immediately following light exposure would be quite interesting to compare to the regional changes in DELLA localization.

It is also worth noting that light effects on hormone response is not a one-way street — changes in hormone biosynthesis or response may influence plants' response to light. For instance, plants with increased sensitivity to brassinosteroids show reduced light response [17]. A molecular model for hormone modification of light sensitivity is emerging from studies of the cytokinin pathway. Cytokinin is perceived by a family of sensor histadine kinases, initiating a phosphorelay which ends with a family of *Arabidopsis* response regulators (ARRs). Phosphorylation on Type-B ARRs relieves autorepression, allowing them to induce expression of a number of early response genes, including Type-A ARRs which act as negative regulators of the pathway [18,19]. Phytochrome B (PHYB) has been shown to directly interact with a Type-A response regulator called ARR4 [18,20]. Recent work shows that cytokinin-induced phosphorylation of ARR4 modulates seedling sensitivity to red light through its direct interaction with PHYB [19,21]. Cytokinin also influences a subset of cryptochrome-mediated responses, including accumulation of anthocyanin genes but not hypocotyl inhibition [21]. Accumulation of HY5 is at least one point of convergence between the two pathways. An interesting next step would be incorporating all three pathways — phytochrome, cryptochrome, and cytokinin — into a model for white light conditions.

Diurnal and circadian effects on the hormone network

The circadian clock enhances plant fitness by facilitating optimal correspondence between internal and external environments [22]. Sensitivity to many signals is modulated by the clock. The time of day when the signal is perceived determines the magnitude of response, a phenomenon called gating [23]. Many areas of growth in young seedlings are under clock control, including hypocotyl elongation [24]. While most work on clock regulation of seedling growth has focused on 'free-running' conditions where plants are held in constant light or constant darkness, a recent analysis focused on plants grown in day/night cycles [25]. Under these more natural conditions, the coincidence of specific light and time-of-day cues together drive rhythmic growth. Two transcription factors, PIF4 and PIF5, were identified as crucial integrators of light and clock signals.

The hormone network is also under clock control. Expression of *CONSTITUTIVE PHOTOMORPHOGENIC DWARF* (*CPD*), a gene encoding a rate-limiting enzyme in brassinosteroid biosynthesis, was found to be under dual control of phytochrome-mediated light response and circadian regulation [26]. Quantitative analysis of endogenous brassinosteroids demonstrated striking diurnal fluctuations in levels of the most active brassinosteroid, brassinolide. Rhythmic synthesis of ethylene in seedlings has also been shown to be clock-regulated, though rhythmic growth is maintained even in mutants with altered ethylene biosynthesis or perception. This suggests that circadian growth patterns are largely ethylene-independent [27]. Past studies have shown that gibberellin [28] and auxin [29] levels are also clock-regulated in some tissues.

In addition to effects on biosynthesis, the clock can also alter hormone responsiveness. Early studies demonstrated that sensitivity to auxin treatment varied over the course of the day [30]. Recent work has provided a molecular framework to understand this phenomenon [31••]. Covington and Harmer used global transcriptome analysis to identify genes whose fluctuating expression levels in constant light conditions suggested circadian regulation. Within this list, genes associated with auxin — including genes involved in auxin biosynthesis, efflux, conjugation, perception, and gene regulation — were significantly overrepresented. The authors were then able to show that auxin effects on both gene expression and hypocotyl elongation were under clock control. *De novo* auxin biosynthesis does not appear to drive circadian effects, as exogenous application of high levels of auxin did not significantly alter rhythmic patterns of growth, expression of a reporter of auxin signaling or expression patterns of clock-associated genes. This is consistent with previous experiments showing that decapitated floral stems exposed to a constant supply of auxin maintain rhythmic growth patterns [29]. The exact point(s) of interaction between the clock and hormone pathways are still unknown, but will probably emerge soon with the wealth of tools now available to address such questions. These studies, as well as one suggesting that many

hormones influence some aspect of clock function [32], contribute significantly to understanding seedling growth while also providing a cautionary note about appropriate controls for all studies of hormone effects.

Regional specificity in hormone interactions

Differential responsiveness, or competence, of subpopulations of cells to global signaling molecules is essential for normal development. Many recent studies have elaborated the diverse roles of hormone networks in the regulation of stem cells and in the production of lateral organs [33]. In seedling photomorphogenesis, growth, and perhaps hormone response, is differentially partitioned between embryonic stems and leaves depending on the light environment: light inhibits cell expansion in the hypocotyl but promotes it in the cotyledons. Several recent studies have detailed the tremendous impact of developmental context on interactions within the hormone network.

Studies of natural variation in root architecture uncovered a root-specific feedback loop between brassinosteroid biosynthesis and auxin response [34]. A putative transcription factor called BREVIS RADIX (BRX) is highly induced by auxin and is required for brassinosteroid biosynthesis in the seedling root. The Umkirch-1 accession carries a loss-of-function *brx* allele resulting in strongly reduced root meristem size and overall root growth. These severe defects, as well as the strongly altered transcriptome profile of such roots, can be largely rescued by application of exogenous brassinosteroids or constitutive expression of *CPD*, the likely target of BRX regulation in the brassinosteroid biosynthetic pathway. In the absence of threshold levels of brassinosteroids, auxin response is severely impaired, as has been seen in other tissues [35]. Auxin has a crucial role in establishing and maintaining the root apical meristem [36], and the reduced auxin response caused by low brassinosteroid levels is probably the proximate cause of the *brx* phenotype. Together, this work provides a mechanistic explanation for the observed organ-specificity of the brassinosteroid–auxin interaction. Whether a similar mechanism for integration of auxin response and brassinosteroid biosynthesis exists in aerial tissues, using a different or redundant transcription factor, remains an open question.

A study dissecting the relationship between auxin and ethylene brings into focus both the organ-specific nature of hormone interactions, as well as the complex organization of such relationships. Stepanova *et al.* found that ethylene-mediated inhibition of seedling root elongation depends on an intact auxin pathway, whereas ethylene effects on hypocotyl elongation are auxin-independent [37••]. Specifically, auxin activity is needed in root transition zones to allow full activation of the ethylene-regulated ETHYLENE-INSENSITIVE 3 (EIN3) transcription factor in the zone of elongation. This conclusion was further bolstered by a second study using quantitative analysis of hormone levels, targeted gene expression, and kinematic growth analysis to show that auxin transported from the root apex to the elongation zone is required for ethylene-mediated inhibition of root growth [38••]. Together, these stories provide the first detailed map of both temporal and spatial constraints on hormone interactions at the molecular level. Interestingly, while global transcriptome analysis supported the largely linear ethylene dependency on auxin for root growth effects, it also revealed a diverse set of other relationships, including ethylene-dependent auxin effects and effects solely under the control of one hormone [37••]. The genes showing these alternative profiles may promote other aspects of auxin and ethylene responses.

Conclusions

It is clear that simple models cannot explain the seedling hormone network [39]. The findings described here reveal the contours of a highly reticulate, multilevel network directing photomorphogenesis. The challenge ahead is synthesizing the remaining disconnected pieces into a cohesive map of dynamic, organ-specific responses under various environmental conditions. These new studies prove that attaining such a systems-level view is not only feasible but essential for truly understanding the mechanisms of plant growth and development.

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