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# Molecular Links Between Light and Auxin Signaling Pathways

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## Abstract

Light influences plant development and may act in part by modifying responses to the hormone auxin. Physiological studies have suggested correlations between light conditions and regulation of auxin transport. Genetic and biochemical studies are beginning to indicate specific proteins that may link light perception with auxin responses. Mutations in several genes encoding transcription factors affect both light and auxin responses, as do mutations that affect protein turnover. Therefore, light and auxin may control turnover of transcription factors. Biochemical and gene expression studies are needed to reveal more precisely how auxin and light signals interact.

**Key words:** Light; Auxin; Phytochrome; Aux/IAA proteins

## Introduction

Plants cannot move and must accommodate to the spot where they germinate. Therefore, they have evolved mechanisms to sense their local environment and adjust their physiology and development so as to optimize growth and reproduction. Light is an especially important environmental variable, and plants use light both as an energy source for photosynthesis and as a signal to activate and modify endogenous developmental programs. Light regulates numerous developmental events including seed germination, stem elongation, apical hook opening, leaf expansion, phototropism, chloroplast development, and flower initiation. The phytohormone auxin can regulate many of the same cellular and developmental processes as light. Auxin promotes cell enlargement required for stem elongation, leaf expansion, and tropic growth; it inhibits lateral shoot outgrowth; and it promotes lateral root formation. The overlapping functions of light and auxin imply that there might be a functional connection between them, and indeed considerable evidence is accumulating that light and auxin signaling pathways are intertwined. In this review, we focus on several recent results that suggest molecular links between light perception and auxin response pathways.

## Light Modulates Auxin Physiology

Various studies have shown correlations between light responses and changes in auxin levels or auxin transport. For example, red light inhibits stem elongation whereas auxin promotes it, suggesting the simple model that light might decrease auxin levels or inhibit auxin action in elongating stems. Red light decreased auxin levels in epidermal cells of both elongating maize mesocotyl and pea epicotyls (Behringer and Davies 1992; Jones and others 1991). Conversely, a partially light-insensitive phyto-

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chrome-deficient mutant of *Nicotiana plumbaginifolia* had increased auxin levels in leaves of young plants (Kraepiel and others 1995). Also consistent with a role of auxin in light-regulated hypocotyl elongation, *Arabidopsis axr1-12* mutant seedlings, which have impaired auxin responses, elongated significantly less than wild-type seedlings when grown in far-red rich light (Steindler and others 1999).

Several results suggest that light regulation of auxin levels in elongating stems might be due to changes in auxin transport into or out of the expanding cells. Auxin is produced in young leaves and transported basipetally down stems by a polar auxin transport system (see review by Muday in this issue). Light decreased the amount of auxin transported through etiolated maize shoots, and this decrease in transport correlated with decreased growth (Jones and others 1991). In contrast, dim red light increased auxin transport in cucumber hypocotyls relative to dark-grown hypocotyls (Shinkle and others 1998). In this case, the increase of auxin transport correlated with decreased apical and increased basal hypocotyl growth, suggesting that transport-mediated changes in auxin distribution control local growth rates (Shinkle and others 1998).

One mechanism by which light may affect auxin transport is by inducing production of flavonoids. Flavonoids can inhibit auxin transport (Jacobs and Rubery 1988), and *Arabidopsis tt4* flavonoid biosynthetic mutants have higher auxin transport rates than wild-type plants (Brown and others 2001). These mutants also have morphological phenotypes that might be caused by increased auxin transport, including decreased shoot apical dominance and increased numbers of lateral roots (Brown and others 2001). As light can induce flavonoid production, these results suggest that light might inhibit auxin transport by inducing flavonoid synthesis. This model would not explain cases in which light increases auxin transport, so light probably also regulates transport by other mechanisms.

A second possible connection between light-induced flavonoid synthesis and auxin transport has been revealed by analyses of *Arabidopsis big/doc1/tir3* mutants. These mutants express light-inducible genes in the dark and also have decreased auxin transport rates (Gil and others 2001; Li and others 1994; Ruegger and others 1997). BIG is a protein of over 500 kD of unknown biochemical function, and has a homolog in animals (Gil and others 2001). As the *big* mutants overexpress flavonoid biosynthetic genes, increased flavonoid production could possibly explain the decreased auxin transport. On the other hand, the increased expression of light-inducible genes in the dark in the mutant was suppressed by a second mutation that causes auxin overproduction, suggesting that altered auxin distribution might cause the gene expression changes (Gil and others 2001). Further work will be required to understand which of these models may be correct.

Other experiments support the notion that auxin transport is more important under some light conditions than others. For example, the auxin efflux carrier NPA inhibited hypocotyl growth in light-grown *Arabidopsis* seedlings, and phyA, phyB, and cry1 photoreceptors were involved in this NPA effect (Jensen and others 1998). However, NPA had little effect on hypocotyl elongation of *Arabidopsis* seedlings in the dark, although dark-grown seedling hypocotyls have been used for classical studies of auxin-induced elongation (Gray and others 1998; Jensen and others 1998). Thus it seems that auxin may control growth distinctly depending on both the light level and the developmental stage of the plant. *Arabidopsis procuste* mutants have a dark-specific defect in hypocotyl elongation, also suggesting that mechanisms of growth control are different under different light conditions (Desnos and others 1996).

Finally, tissue-specific factors probably also influence interactions between light and auxin responses. In leaves, auxin and light each promote leaf cell expansion (Cosgrove 1994; Jones and others 1998). In this case, light might trigger a self-reinforcing loop whereby expanding leaves produce auxin that in turn induces the leaves to continue to grow. Auxin synthesized in growing apices might then promote lateral root formation by increasing basipetal auxin flow from the shoot, explaining why light can also promote root formation (Jensen and others 1998; Reed and others 1998b). In both shoots and roots, light can also induce phototropic growth and enhance gravitropic growth, and these effects also involve asymmetric auxin transport that leads to differential elongation on opposite sides of an organ. These issues are discussed in more detail in the article by Muday in this issue.

Taken together, these results suggest that light can modulate the amount of auxin production or auxin transport, and that this leads to quantitative changes in local auxin concentrations that correlate with local tissue growth rates. In some cases these correlations may be sufficient to explain how light may cause quantitative morphological changes. However, molecular studies of light and auxin signal transduction have produced several results that complicate these simple notions, and suggest that light may also modulate auxin signaling pathways.

## Light-Regulated Transcription Factors Affect Auxin Responses

Plants perceive light using several families of photoreceptors: the red/far red-sensing phytochromes, the blue/UV-A-sensing cryptochromes, phototropins responsible for phototropism, and unidentified UV-B photoreceptors. *Arabidopsis* has five genes encoding phytochromes (*PHYA-PHYE*). phyA primarily mediates far-red light responses, whereas phyB and other phytochromes primarily mediate red light responses. Upon light activation, phyA and phyB move from the cytoplasm to the nucleus where they can interact with downstream signaling components including transcription factors. Downstream signaling components may be either specific to a certain phytochrome or shared by more than one phytochrome. PIF3 is a basic helix-loop-helix protein and binds to G-box elements in promoters of light-responsive genes (Martínez-García and others 2000; Ni and others 1998). PIF3 interacts directly with phyA or phyB, and preferentially interacts with the active Pfr form of each (Martínez-García and others 2000; Ni and others 1999; Zhu and others 2000). Although PIF3 is not known to regulate auxin responses, and therefore provides no obvious molecular link between auxin and light, these results have shown that phytochromes can interact directly with nuclear transcription factors to regulate gene expression responses.

Other transcription factors may provide a more direct connection between light and auxin signaling. For example, HY5 is a bZIP protein (Oyama and others 1997), and loss-of-function *hy5* mutants have increased hypocotyl elongation only when grown in the light (Koornneef and others 1980). They also have defects in processes known to be regulated by auxin, such as altered gravitropic and touching responses in roots, enhanced initiation and elongation of lateral roots, and reduced secondary thickening of the root and hypocotyl (Oyama and others 1997). Although HY5 protein has not been shown to interact directly with a photoreceptor, the stability of HY5 protein is regulated by light. Thus, in dark-grown plants, HY5 protein is turned over rapidly, whereas in the light it is stabilized (Osterlund and others 2000). Destabilization of nuclear HY5 in darkness is thought to be mediated by the COP1 protein, which is present in the nucleus only in the dark, and can interact with HY5 (Hardtke and others 2000). COP1 has a RING-finger motif, which is found in many ubiquitin-protein ligases, and COP1 might act as a ubiquitin ligase to target HY5 for degradation. Action of any of several photoreceptors can regulate both COP1 nuclear abundance and HY5 stability (Osterlund and Deng 1998; Osterlund and others 2000), suggesting that light may regulate HY5 through its action on COP1 nuclear abundance. As discussed below, protein turnover plays a key role in both light and auxin response pathways. Components of the COP9 signalosome discussed below are also required for HY5 degradation.

A third *Arabidopsis* transcription factor, ATHB-2 (also called HAT4), is a homeodomain-leucine zipper (HD-zip) protein. ATHB-2 is required for the shade avoidance response to far-red light, most likely acting as a negative regulator of gene expression (Steindler and others 1999). Overexpressed *ATHB-2* caused decreased cotyledon expansion and increased hypocotyl elongation in light (Schena and others 1993; Steindler and others 1999). Elevated *ATHB-2* level also inhibited lateral root formation, and this latter phenotype could be rescued by exogenous auxin (Steindler and others 1999). Conversely, antisense *ATHB2* plants had short hypocotyls and enlarged cotyledons (Schena and others 1993; Steindler and others 1999). Consistent with the model that ATHB2 inhibits light response, expression of *ATHB2* is down-regulated by phytochrome activation (Carabelli and others 1996).

## Mutations in Aux/IAA Genes Affect Light Signaling

HY5 and ATHB2 levels are each regulated by light, and mutation of each of them also causes auxin-related phenotypes. Conversely, a fourth class of transcriptional regulator, the Aux/IAA proteins, are known to be regulated by auxin, and also affect light responses. There are 29 *Aux/IAA* genes in *Arabidopsis,* and gain-of-function mutations in nine of them cause defects in auxin responses (Reed 2001). These mutations also affect auxin-regulated gene expression (Abel and others 1995; Leyser and others 1996; Rogg and others 2001; Q. Tian and J.W. Reed, unpublished results). Aux/IAA proteins probably regulate transcription by modifying activity of ARF (auxin response factor) proteins, with which they can dimerize. ARF proteins bind to auxin response elements in promoters of auxin-regulated genes (Hagen and Guilfoyle 2001). Auxin may therefore regulate ARF activity, possibly through effects on interacting Aux/IAA proteins. Most *Aux/IAA* genes are themselves induced by auxin (Abel and others 1995), indicating that they are both regulators and targets of auxin transcriptional regulation.

Gain-of-function mutations in several *Aux/IAA* genes stabilize the corresponding proteins (Colon-

Carmona and others 2000; Ouellet and others 2001; Worley and others 2000), raising the possibility that auxin regulates Aux/IAA protein turnover in addition to transcription of their genes. Mutations in *TIR1* and *ASK1* genes encoding components of the ubiquitin ligase $SCF^{TIR1}$ cause auxin resistance (Gray and others 1999; Ruegger and others 1998), and Aux/IAA proteins may be targets for $SCF^{TIR1}$ targeted ubiquitination and protein turnover. Mutations in *AXR1*eliminate modification of $SCF^{TIR1}$ by the ubiquitin-related protein Rub (del Pozo and Estelle 1999). This modification is required for $SCF^{TIR1}$ activity, and *axr1* mutations also cause auxin resistance (Estelle and Somerville 1987). Auxin might regulate ubiquitin ligase activity, or it might cause modification of the Aux/IAA proteins themselves.

Several of the gain-of-function Aux/IAA mutants have light-related phenotypes. Mutants in *SHY2/IAA3, AXR2/IAA7,* and *AXR3/IAA17* each have short hypocotyls both in the light and in the dark, and they can make leaves in the dark (Kim and others 1998; Kim and others 1996; Leyser and others 1996; Nagpal and others 2000; Reed and others 1998a; Tian and Reed 1999; Timpte and others 1994). *shy2* mutant plants also express several light-regulated genes in darkness (Kim and others 1998). These results raise the possibility that light may normally activate these genes or proteins to induce morphological responses such as leaf development. Consistent with this possibility, oat phyA can interact with some Aux/IAA proteins *in vitro* and in the yeast two-hybrid system (Colon-Carmona and others 2000; Soh and others 1999). In addition, phyA can phosphorylate any of several Aux/IAA proteins (IAA1, IAA3, IAA4, IAA17, and Ps-IAA4/5) (Colon-Carmona and others 2000). These *in vitro* interactions were not light-dependent. However, phytochromes move to the nucleus in response to light (Reed 1999), and nuclear localization could trigger interactions between phytochromes and Aux/IAA proteins. If phyA or other phytochromes indeed interact with Aux/IAA proteins *in vivo,* they might regulate the stability or activity of Aux/IAA proteins by phosphorylating them, which could in turn modulate the gene regulatory activity of ARFs. Assays of the effects of light on Aux/IAA protein abundance and localization will provide key tests of these ideas.

A further potential link between Aux/IAA proteins and light responses has recently emerged from characterizations of *Arabidopsis* plants deficient in the COP9 signalosome. Mutations in any of several genes encoding components of this large protein complex cause expression of numerous light-inducible genes in dark-grown seedlings (Deng and Quail 1999; Neff and others 2000). Numerous other genes are also inappropriately expressed (Mayer and others 1996), but these mutations are lethal, precluding extensive analyses of the mutant phenotypes. However, an antisense line for *CSN5,* encoding one component of the COP9 signalosome, has a weaker phenotype (Schwechheimer and others 2001). This line had several auxin-related phenotypes, including increased apical dominance, reduced hypocotyl growth, auxin-resistant root growth, fewer lateral roots, reduced root hair elongation, and reduced gravitropic response in roots. It also degraded a PsIAA6::luciferase fusion protein more slowly than wild-type plants, suggesting that increased Aux/IAA protein activity may explain the auxin-resistant phenotypes in the antisense line. By analogy with the de-etiolated phenotypes of gain-of-function *Aux/IAA* mutants, increased Aux/IAA activity could also account for the leaf development and short hypocotyl phenotypes of *cop9* and other COP9 complex component mutants. As mentioned above, the COP9 complex is also required for HY5 protein turnover, and HY5 is stabilized by light. It will be interesting to determine whether light also regulates Aux/IAA protein turnover, and whether other mutations that cause similar phenotypes such as *det1* and *cop1* also affect Aux/IAA protein stability.

Another twist to this story is that the COP9 signalosome acts as a Rub-deconjugating enzyme, and can remove Rub from cullin (Lyapina and others 2001; Schwechheimer and others 2001). As mentioned above, AXR1 is part of a Rub-conjugating enzyme. Thus, deficiency of either Rub-conjugation or Rub-deconjugation activity may stabilize Aux/IAA proteins and cause auxin resistance. This seeming paradox may be explained if cycles of Rub conjugation and deconjugation are needed for $SCF^{TIR1}$ activity. Another possibility is that Rub modification of cullin changes the specificity of SCF complexes for different Aux/IAA protein substrates, rather than simply turning SCF ubiquitin ligase activity on or off. Finally, it is possible that other activities of the COP9 signalosome are more relevant. For example, the human COP9 signalosome can phosphorylate the p53 tumor suppressor protein to target it for degradation (Bech-Otschirand others 2001). In any event, it appears that protein turnover regulates both light and auxin responses. A key question is whether light and auxin regulate ubiquitination of common substrates.

### *GH3* Genes May Also Mediate Light and Auxin Responses

Two members of another auxin-regulated gene class have also been implicated in light responses. The

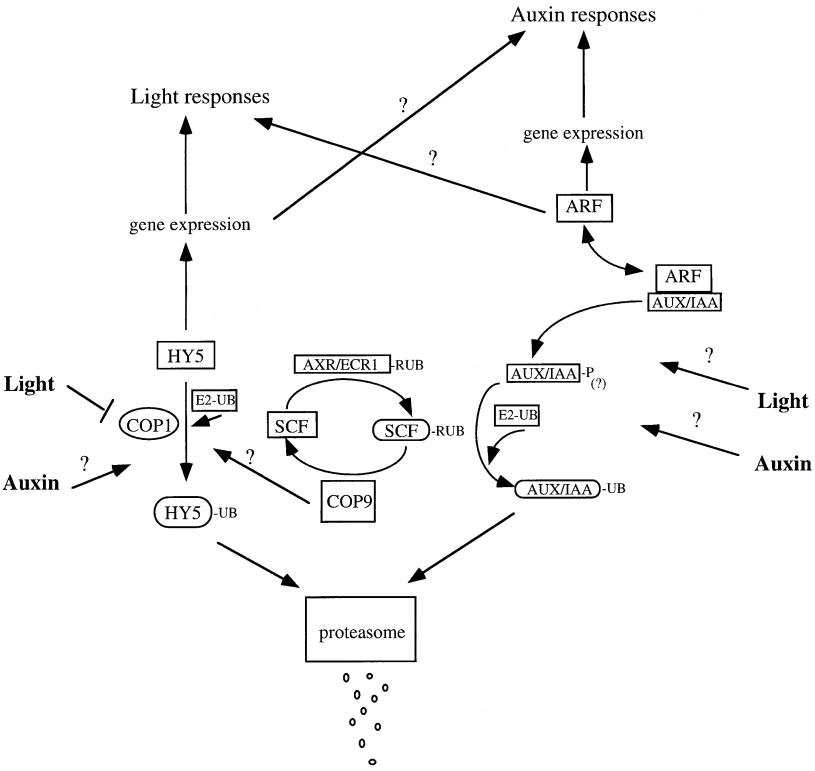


**Figure 1.** A model to explain how auxin and light may regulate common developmental processes. Auxin and light may interact at multiple levels, including regulating stability or activity of common proteins, and regulating common target genes. Not shown in this model is potential regulation of auxin synthesis or transport by light.

*fin219* mutant has a far-red light-specific long hypocotyl phenotype, suggesting that it has a defect in phyA responses (Hsieh and others 2000). The mutation mapped to the *GH3-11* gene, a member of the *GH3* class of auxin-inducible genes, suggesting that these genes are also important for light responses. Interestingly, although a *GH3-11* transgene could rescue the mutant phenotype, no sequence change could be found in this gene in the mutant, leading to the conclusion that the phenotype was caused by an epigenetic change that decreased expression (Hsieh and others 2000). Recently, *jar1* mutations, which cause resistance to jasmonic acid as well as pathogen response phenotypes, were also found to affect the *GH3-11* gene (P. Staswick, personal communication). The *jar1* mutations include putative nulls, and have normal light responses. These results raise the possibilities that the *FIN219* gene was misidentified, that some peculiarity of the epigenetic expression pattern caused the light response phenotypes, or that the decreased light response of *fin219* arose from simultaneous effects on multiple genes.

A second gene of this family, *GH3–6/DFL*, also affects light responses. Dominant *dfl1-D* mutants have reduced hypocotyl growth under blue, red, and far-red light, but normal hypocotyl growth in the dark (Nakazawa and others 2001). They also have altered auxin responses, such as reduced number of lateral roots and auxin-resistant root growth. These phenotypes are caused by overexpression of *GH3-6*. In wild-type plants, expression of *GH3–6/DFL1* is regulated by auxin, but not by light. Overexpression of *GH3–11/FIN219/JAR1* also caused hypersensitivity to light (Hsieh and others 2000). The light specificity of the mutant phenotypes suggests either that *GH3–6/DFL* and *GH3–11/FIN219/JAR1* are light signaling components or that they affect processes, such as auxin transport-dependent hypocotyl elongation, that can be seen in the light but not in the dark.

## A Model For Further Studies

We have incorporated many of these molecular results in a model whereby light and auxin each regulate activity of transcription factors, and these in turn control expression of genes that effect growth responses (Figure 1). In many cases, turnover of the transcription factors may be regulated. Except for light regulation of HY5 turnover, we do not know whether auxin or light regulates these components directly, or whether the identified transcriptional regulators are simply required for activity of more direct targets of signal transduction. Further biochemical analyses should clarify this issue, and genetic and gene expression studies should reveal whether the light-auxin link is central to light re-

sponses, or just one of several parallel pathways induced by light.

A further challenge will be to integrate these results into the context of development of a plant over time. Cells in different organs or tissues have distinct regulatory makeups; moreover they are exposed to varying amounts of light, auxin, and other signals at different times. Models of biochemical pathways will therefore have to account for these variations in both signal inputs and response characteristics. At that point we may be able to explain the physiology of plant growth more precisely.

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