

Molecular genetic analysis of signal transduction controlling photomorphogenic development in *Arabidopsis thaliana*

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Photomorphogenesis is an important developmental phenomenon of higher plants. However, it is not fully understood how light stimuli perceived by multiple photoreceptors are transduced and integrated to affect developmental program.

Light affects plant growth and development throughout the life cycle. However, light signals do not function autonomously but should be integrated with endogenous developmental factors such as the plant hormone auxin to specify correct developmental decisions. We previously reported that the *Arabidopsis shy2-1D* mutation alters various light responses, including highly photomorphogenic development in darkness. Here we show that the mutation also alters various auxin responses, including constitutive formation of lateral roots and reduced sensitivity to auxin in inhibition of hypocotyl and root growth. The mutation is a gain of function mutation occurred in the *IAA3* gene, one of the *Aux/IAA* family genes encoding putative transcription factors of auxin-responsive genes. These results suggest that *IAA3/SHY2* may play important roles in both light- and auxin-mediated development. *IAA3/SHY2* expression was induced in the cotyledon but was reduced in the hypocotyl by light, providing a possible explanation on how light exerts the opposing effect on the growth of these organs. Considering that *Aux/IAA* proteins and auxin response transcription factors interact with one another, we propose that *IAA3/SHY2* may integrate light signals into auxin-mediated developmental responses.

We are also trying to understand the light signaling pathway specific to phytochrome A (PhyA) that mediates most, if not all, various plant responses to far-red (FR) light. Here, we report a novel genetic mutation that impairs a variety of responses in the PhyA-signaling pathway of *Arabidopsis thaliana*. The mutation was isolated by screening seedlings that show reduced sensitivity to continuous far-red (FRc) light irradiation, but not to continuous red (Rc) light irradiation. The mutation named *fin2-1* is not allelic to a *PHYA* mutation. Furthermore, immunoblot analysis indicated that the amount of the phytochrome A apoprotein in the *fin2-1* mutant was comparable to that in wild type. Seedling of the *fin2-1* mutant showed defects in hypocotyl growth inhibition and apical hook and cotyledon opening in FRc light but not in Rc light. The results showed that the mutation occurred in a downstream signaling component potentially specific to PhyA. Other PhyA-mediated