Summary for Plant Cell article

Phytochromes are a family of red/far-red light photoreceptors, which positively regulate photomorphogenesis upon red-light perception. Photomorphogenesis is driven by light- induced global transcriptional reprogramming, of which phytochromes are one of the most important regulators. In addition to transcriptional regulation, recent experimental evidence suggest that phytochromes also regulate pre-mRNA splicing of large number of genes, and at least one of the splicing factors, SFPS (Splicing Factor for Phytochrome Signaling), is shown to interact with phytochromes and coordinately modulate pre-mRNA splicing. In this paper, Xin et al. have identified one more splicing factor RRC1 (Reduced Red-light responses in Cry1cry2 background 1) as one of the interacting proteins of SFPS and phyB. Through RNA- seq analysis authors showed that RRC1 regulates the expression and pre-mRNA splicing of large number of genes, both under dark and red- light treatment. Moreover, they also found that RRC1 and SFPS together co-regulate hundreds of splicing events, suggesting they might function, in part, in the same complex and pathway. Finally, they conclude that phytochromes modulate the pre-mRNA splicing by directly interacting with splicing factors and (probably) regulating their activity in response to red-light irradiation. Overall, this paper provides a comprehensive view of yet another novel molecular mechanism of phytochrome- mediated signaling to promote photomorphogenesis in Arabidopsis.

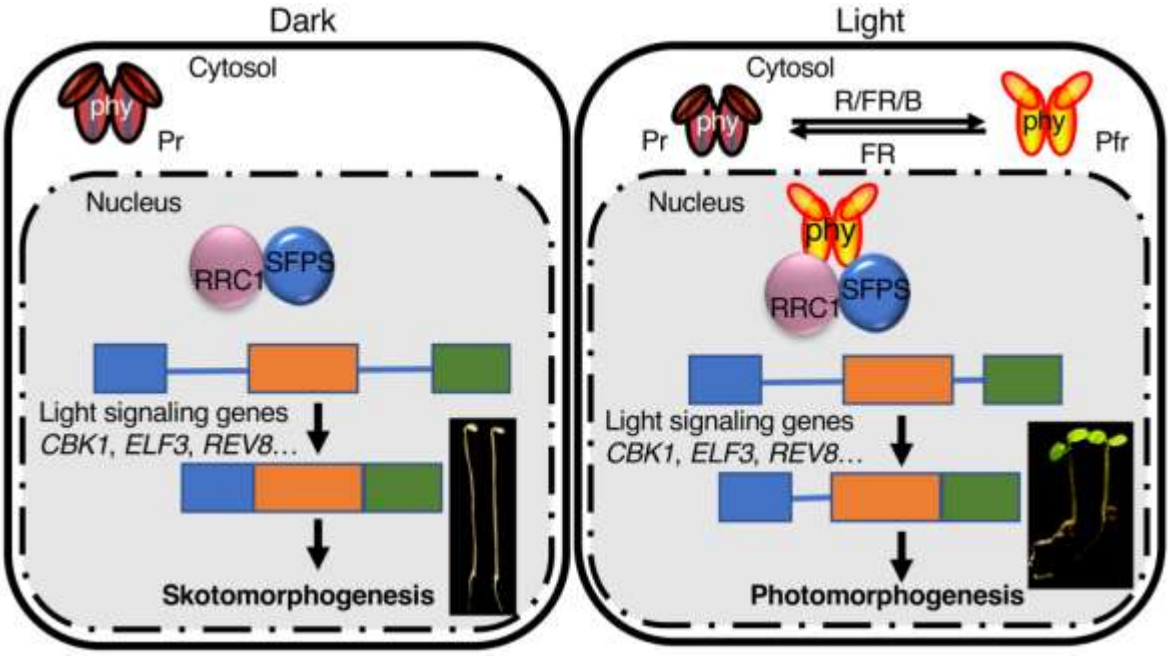

Figure: **Model of RRC1-SFPS function in regulating photomorphogenesis.**
(Left panel) Phytochromes are in the cytosol in darkness. SFPS and RRC1 are localized in the nucleus and regulate pre-mRNA splicing of many genes involved in light signaling and circadian clock pathways. (Right panel) In response to light, phytochromes migrate into the nucleus, directly interact with SFPS and RRC1 and regulate pre-mRNA splicing of light signaling genes (e.g., *CKB1, ELF3, REV8* and others) to fine-tune the light-regulated developmental processes in Arabidopsis.

Summary of Nature Communication article:

Light has a profound impact on plant growth and development not only as a source of energy but also as an informational signal. The phytochrome-family of red/far-red light photoreceptors regulates plant development throughout a plant's life cycle. However, how phytochromes exert such a striking influence on plant development has been the subject of research for decades in many laboratories in the world. In this paper published recently in Nature Communications (https://www.nature.com/articles/s41467-019-12110-y), Huq lab provides a biochemical mechanism for one of the most important questions in the field of phytochrome-mediated light signaling pathways; how do phytochromes trigger rapid phosphorylation and degradation of their target proteins? Light-activated phytochromes induce multiple post-translational modifications onto their signaling partners via direct protein–protein interactions. These modifications, such as phosphorylation, play a critical role in phytochrome signaling cascades. However, little is known about how phytochromes initiate this post-translational modification. They found a long-sought protein kinase complex responsible for the light-induced phosphorylation of PHYTOCHROME INTERACTING FACTOR 1 (PIF1).

CONSTITUTIVE PHOTOMORPHOGENIC 1 (COP1) is one of the best characterized E3 ubiquitin ligases with broad roles as a central repressor of light signaling in plants to cancer biology in mammals. In plants, COP1 interacts with SUPPRESSOR OF PHYA-105 1 family members (SPA1-SPA4) and forms a stable COP1/SPA complex. SPAs contain a Serine/Threonine kinase domain at its N-terminus, a coiled-coil domain in the middle, and four WD-40 repeats at its C-terminal half, which serves as an interaction domain with the substrates. SPA kinases have no known substrate, although the kinase domain of SPA proteins have been shown to be necessary for their biological functions. This story provides evidence that SPA1, a component of the COP1-SPA E3 ubiquitin ligase complex, itself can act as a protein kinase. By performing extensive biochemical, genetic and genomic analyses, they provide strong evidence that SPA1 is a *bona fide* Serine/Threonine kinase that is necessary for the light-induced phosphorylation and degradation of PIF1. They also provide mechanistic details on how the red/far-red light photoreceptor phytochrome B directly interacts with SPA1 kinase in response to red light, and this interaction plays a pivotal role in inducing PIF1 phosphorylation and subsequent degradation to promote seed germination and seedling development. These data highlight the importance of the COP1-associated kinases not only in enhancing the COP1 activity, but also the phosphorylation of their substrates for rapid ubiquitination and subsequent degradation. This is a ground-breaking work discovering a novel kinase that might serve as a model for other kinase-E3 ubiquitin ligase complexes (e.g., mammalian COP1-Trib kinase complex) and help decipher new intriguing roles of COP1 and associated kinases from other systems. This pivotal discovery is expected to redirect the focus of the future research on COP1 and associated kinases in a new direction, and help uncover novel roles of COP1 associated complexes in plants and animal systems.

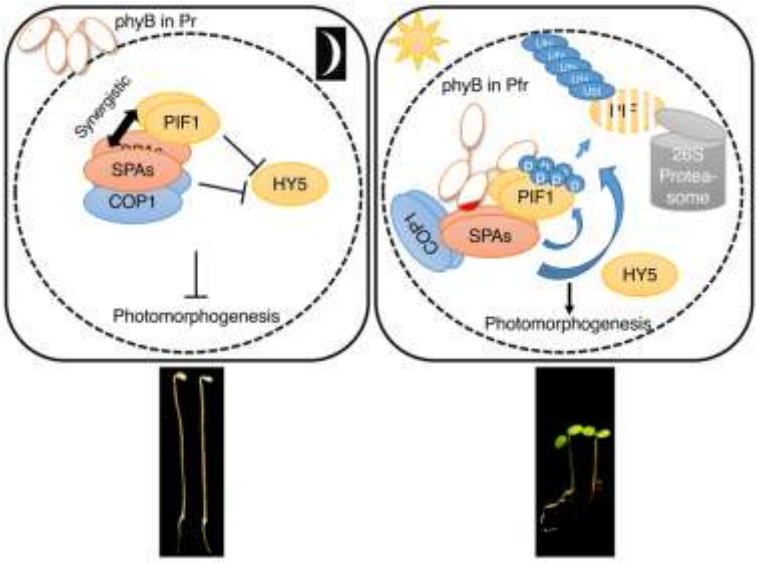

Figure: Model showing how phyB pron phosphorylation and degradation of its intera partner PIF1.

In darkness, inactive Pr form of phyB is present i cytoplasm, while COP1-SPA complex together PIF1 as a co-factor induces degradation of posit acting transcription factors (HY5/HFR1 and pos others). However, upon light exposure, activate form of phyB translocates into the nucleus interacts with PIF1 as well as SPA1 to trigger phosphorylation of PIF1. phyB interacts with mostly through its N-terminal domain, whil extreme C-terminal 80 amino acids (shown as patch) are necessary for SPA1 interaction. stabilizing the phyB-SPA1-PIF1 tripartite com phyB can initiate the light-induced phosphorylati PIF1 by SPA1 kinase. Phosphorylated PIF1 is recognized by the $CUL4^{COP1\text{-}SPA}$ E3 ubiquitin ligas rapid poly-ubiquitylation and subsequent degrad through the 26S proteasome. Degradation of PIF stabilization of HY5 result in promotion