Role of PINOID-mediated COP1 phosphorylation in Arabidopsis photomorphogenesis is overemphasized

Huanhuan Jin and Ziqiang Zhu

Etiolated Arabidopsis seedlings have long hypocotyls and closed yellow cotyledons (skotomorphogenesis); however, light-grown plants exhibit short hypocotyls and opened green cotyledons (photomorphogenesis) (1). It has been revealed that E3 ubiquitin ligase CONSTITUTIVELY PHOTOMORPHOGENIC 1 (COP1) is a central negative regulator for plant photomorphogenesis. COP1 directly targets a plethora of transcription factors for degradation in darkness, while light suppresses COP1 activity to stabilize these transcription factors (2). Many of these transcription factors are able to associate with light-responsive promoters to trigger a proportion of transcriptomic alternations and then establish photomorphogenesis (3).

Lin et al. (4) argue that they find Ser/Thr kinase PINOID (PID) directly phosphorylates COP1 and inhibits COP1 activity during photomorphogenesis. Although we appreciate this interesting finding, we have several concerns on the interpretation of their genetic data and particularly whether the PID-mediated COP1 phosphorylation matters in photomorphogenesis.

The authors identified one mutation (pid-15) that successfully reverses the short hypocotyl of cop1-6 (cop1 weak allele) back to normal (4). They further demonstrated that PID directly phosphorylates COP1 at Ser20.

It was explained that in cop1-6, PID phosphorylates the residue COP1 protein and inhibits its activity, while in cop1-6 pid-15 double mutant, loss of PID dephosphorylates COP1 and reactivates the residue COP1 activity, which completely recovers the normal etiolation growth (4). If it stands, we will assume that the PID-mediated COP1 phosphorylation is critical for COP1 activity. However, every YFP tag-fused phosphorylation dead mimic (YFP-COP1 S20A) or phosphorylation active mimic (YFP-COP1 S20D) completely rescues cop1-6 mutants. Although the authors argue that YFP-COP1 S20D has weaker activity than YFP-COP1 S20A, the hypocotyl lengths of YFP-COP1 S20D/cop1-6 are already equal to the wild-type plants. This result indicates that the PID-mediated COP1 phosphorylation at Ser20 is dispensable for COP1 activity. In addition, the abundance of COP1 target protein HY5 was not changed in PID overexpression lines, suggesting that enhancing PID levels did not significantly reduce COP1 E3 ligase activity.

Although we question the biological consequence of PID-mediated COP1 phosphorylation in photomorphogenesis, we do not doubt that pid suppresses cop1 phenotype and PID–COP1 physical interactions. In contrast, to put COP1 downstream of PID, we propose that PID might act downstream of COP1 based on the authors’ observations (4). The pid-15 mutants have a normal etiolation growth phenotype, while pid-15 cop1-6 resembles pid-17. PID is not likely to be a degradation target for COP1, because PID protein levels are the same in Col-0 and cop1-6 plants. However, the authors do not examine PID kinase activity in cop1 mutants and even do not mention too much on the biological function of PID. PID phosphorylates auxin efflux carriers and modulates auxin homeostasis (5–7). Since auxin is crucial for hypocotyl elongation, we suggest that it is required to reexamine the impact of COP1–PID interaction and check whether COP1 affects PID kinase activity and auxin flow.

In summary, we do not believe that pid-15 suppression of cop1-6 is caused by PID-mediated COP1 phosphorylation. Instead, it is urged to pursue other possibilities, like whether COP1 affects PID kinase activity on auxin efflux proteins.


*College of Life Sciences, Nanjing Normal University, Nanjing 210023, China

Author contributions: H.J. performed research; Z.Z. designed research; and Z.Z. wrote the paper.

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*To whom correspondence should be addressed. Email: zqzhu@njnu.edu.cn.