COP1 is a negative regulator of seed germination in Strigolactone signaling.

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Strigolactones (SLs) are host factors that stimulate seed germination of parasitic plant species *Striga*. To understand the roles of SLs in seed germination, it is necessary to develop a tractable experimental system using model plants such as Arabidopsis. We reported that thermoinhibition, which involves exposing seeds to high temperatures, uncovers a clear role for SLs in promoting Arabidopsis seed germination. Both SL biosynthetic and signaling mutants showed increased sensitivity to seed thermoinhibition. The synthetic strigolactone GR24 does not rescued max2-1 seed germination.

Hormone analysis revealed that SLs alleviate thermoinhibition by modulating levels of the two plant hormones, GA and ABA. Recently, we also reported that GR24 directly bind the HTL α/β hydrolase in Arabidopsis in vitro. Strigolactones promoted an interaction between HTL and the F-box protein MAX2 in yeast. We also found *htl* mutant in our GR24 insensitive screening using thermoinhibiton. These results suggest that HTL is involved in SL signaling during seed germination in Arabidopsis. Molecular analysis using a hypocotyl elongation assay showed SLs regulate the nuclear localization of the COP1 ubiquitin ligase, which in part determines the levels of light regulators such as HY5. Genetic analysis revealed that *cop1* single mutant, *cop1 max2* double mutant and *cop1 htl-3* double mutant showed thermo-tolerant seed germination phenotype. These results indicated that COP1 is a negative regulator of seed germination and is genetically at or downstream of MAX2 and HTL in SL signaling with respect to seed germination.