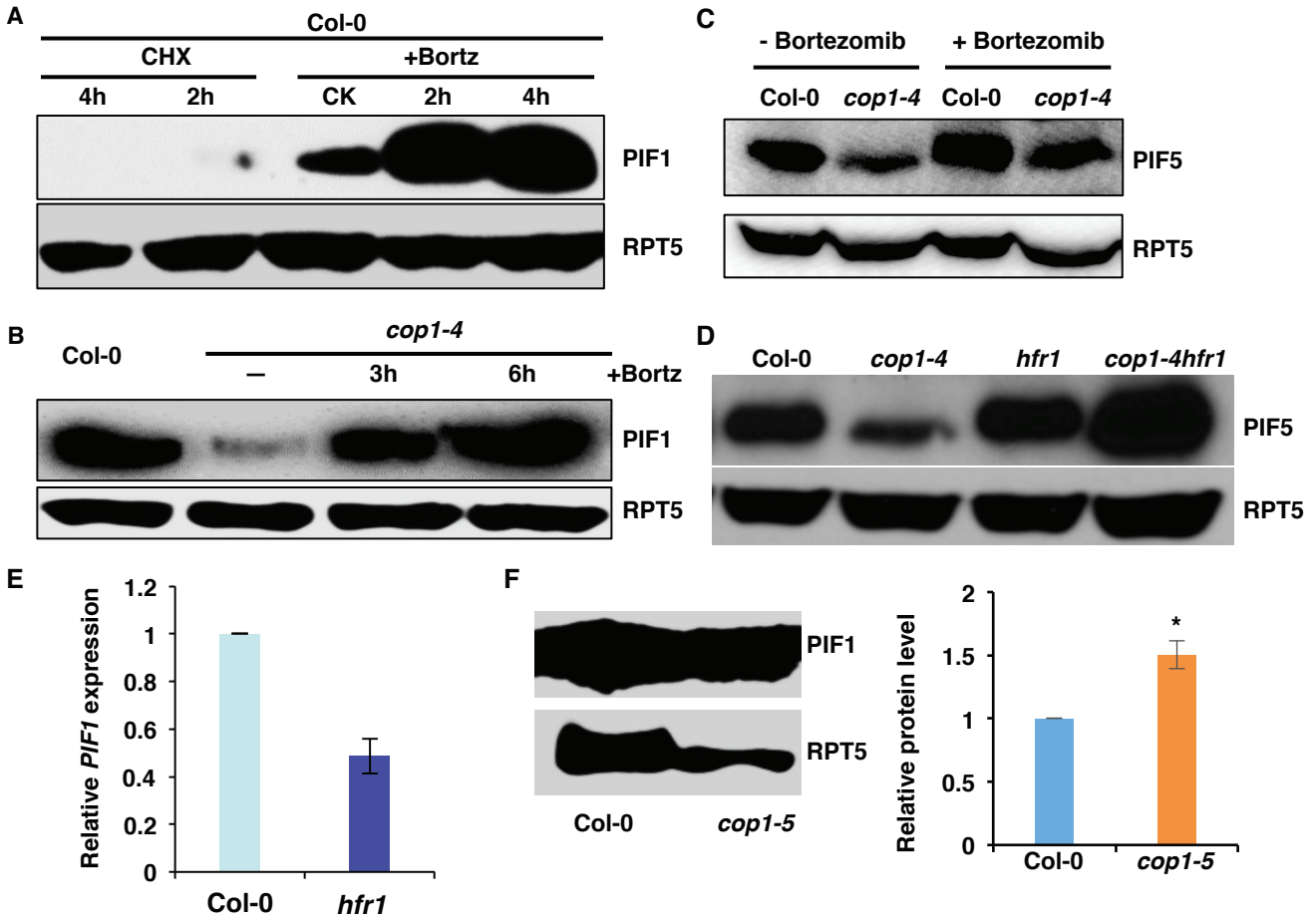
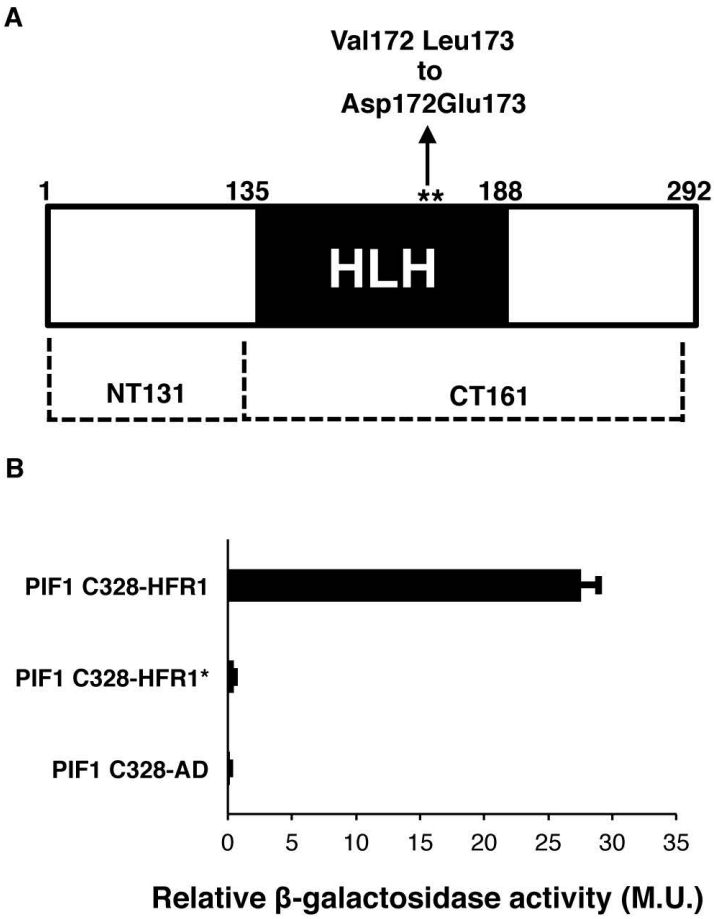
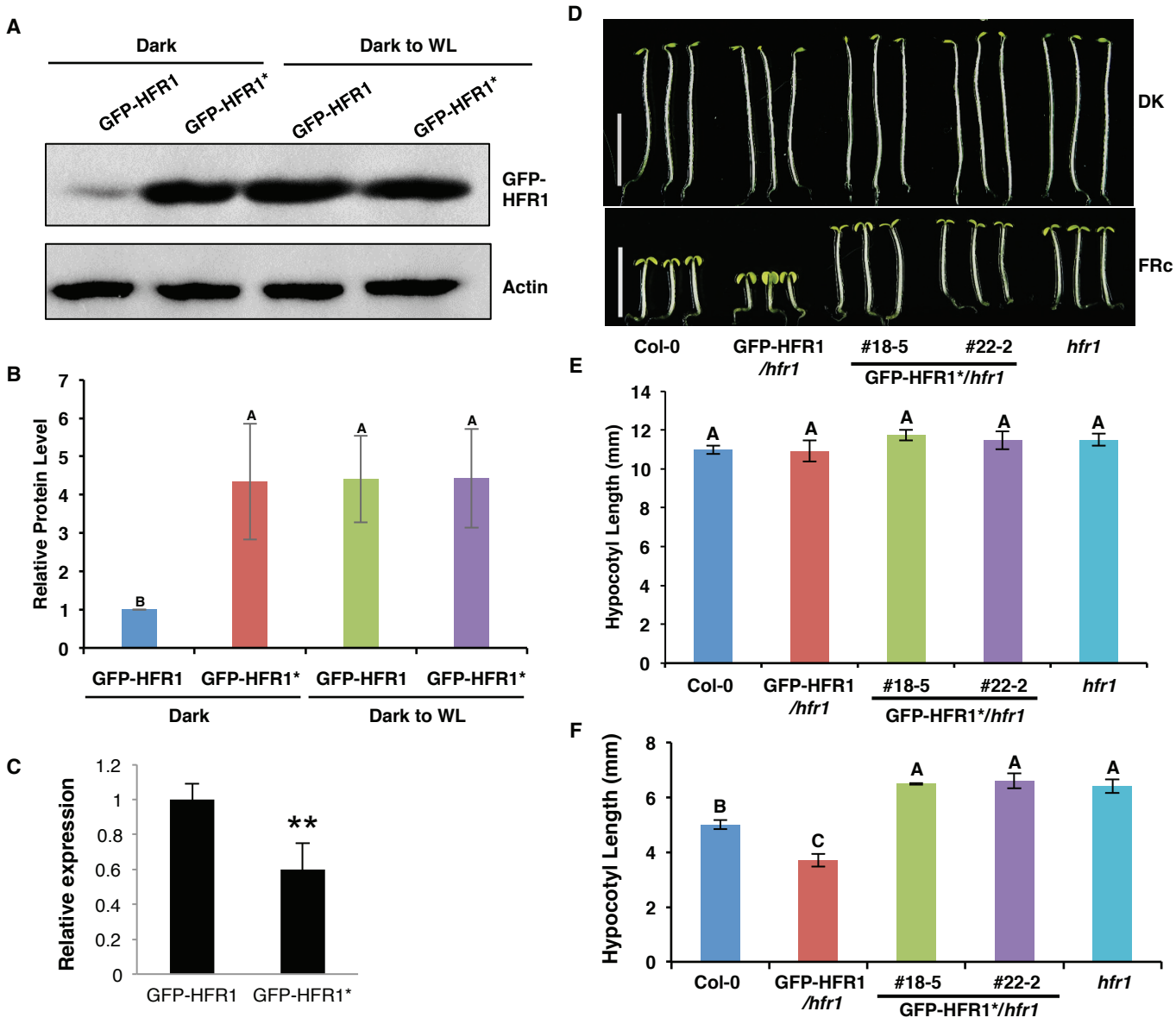


Supplemental Figure 1: COP1 and HFR1 are involved in the 26S proteasome mediated degradation PIF1 and PIF5 in the dark.

(A) Immunoblot shows PIF1 level in 5-day-old wild type Col-0 dark-grown seedlings treated with 20 mM cycloheximide (CHX) or proteasome inhibitor (40 μM Bortezomib) for the indicated hours before protein extraction in the dark. CK is a control without any treatment in the dark. Total protein was separated on an 8% SDS-PAGE gel, blotted onto PVDF membrane and probed with anti-PIF1 or anti-RPT5 antibodies. (B) Immunoblot shows the PIF1 level in 4-day-old wild type Col-0 or *cop1-4* dark-grown seedlings with and without proteasome inhibitor (40 μM Bortezomib) pretreatment for the indicated time before protein extraction in the dark. Total protein was separated on an 8% SDS-PAGE gel, blotted onto PVDF membrane and probed with anti-PIF1 or anti-RPT5 antibodies. (C) Immunoblot shows the PIF5 level in 4-day-old wild type Col-0 or *cop1-4* dark-grown seedlings with and without proteasome inhibitor (40 μM Bortezomib) pretreatment for the 3 hours before protein extraction in the dark. Total protein was separated on an 8% SDS-PAGE gel, blotted onto PVDF membrane and probed with anti-PIF5 or anti-RPT5 antibodies. (D) Immunoblot shows the PIF5 level in 4-day-old wild type Col-0, *cop1-4*, *hfr1*, and *cop1-4hfr1* dark-grown seedlings. Immunoblot was performed as described (C). (E) RT-qPCR data showing the relative expression of *PIF1* in wild-type and *hfr1-201* mutant. RNA was extracted from 4-day-old dark grown wild-type Col-0 and *hfr1-201* seedlings and reverse transcribed into cDNA. (F) PIF1 is more abundant in *cop1-5* compared to wild type. (Left) Immunoblot blot shows the PIF1 level in wild type Col-0 and *cop1-5*. Total protein was extracted from 4-day-old seedlings grown on the MS media in darkness. (Right) Quantification of PIF1 protein level using RPT5 as a control. * indicates statistically significant differences between means of protein levels ($p<0.05$). The error bars indicate standard deviation (n=3).

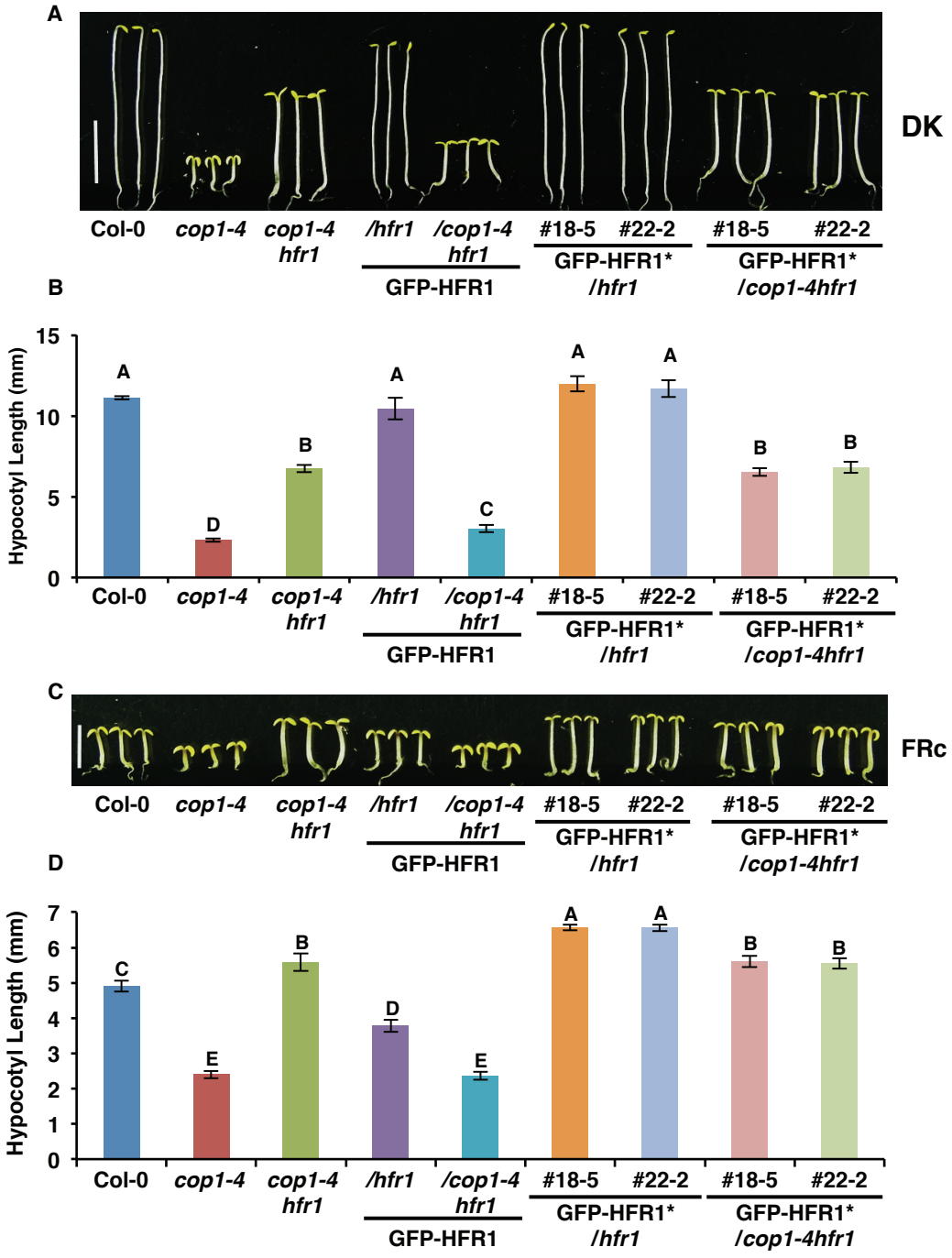


Supplemental Figure 2: HFR1* does not interact with PIF1 in yeast 2-hyrbid assays. A) The domain structure of HFR1. The N-terminal 131 domain of HFR1 is responsible for interaction with COP1 and triggered the 26 proteasome mediated degradation, the C-terminal 161 domain (CT161) is involved in forming heterodimer with PIF1/3/4/5 to block PIF's transcriptional activity for binding to DNA. The ** indicate mutated version of the HFR1 protein (HFR1*) that substitutes two conserved residues Val172 Leu173 to Asp172Glu173 in the HLH domain, which can interfere with the dimerization. B) Quantitative yeast-two hybrid assay showing HFR1 directly interacts with the C-terminal bHLH domain of PIF1 (C328). PIF1 C328 was fused with GAL4 DNA binding domain (pGBT9). Full-length HFR1 and mutant HFR1* deficient in interaction with PIF1 were fused with GAL4 activation domain (pGAD424). Mutant HFR1* and empty vector (pGAD424) was used as negative control. AD: empty vector pGAD424. β-galactosidase units are Miller units. LacZ assays were performed in triplicate and error bars indicate standard deviation.



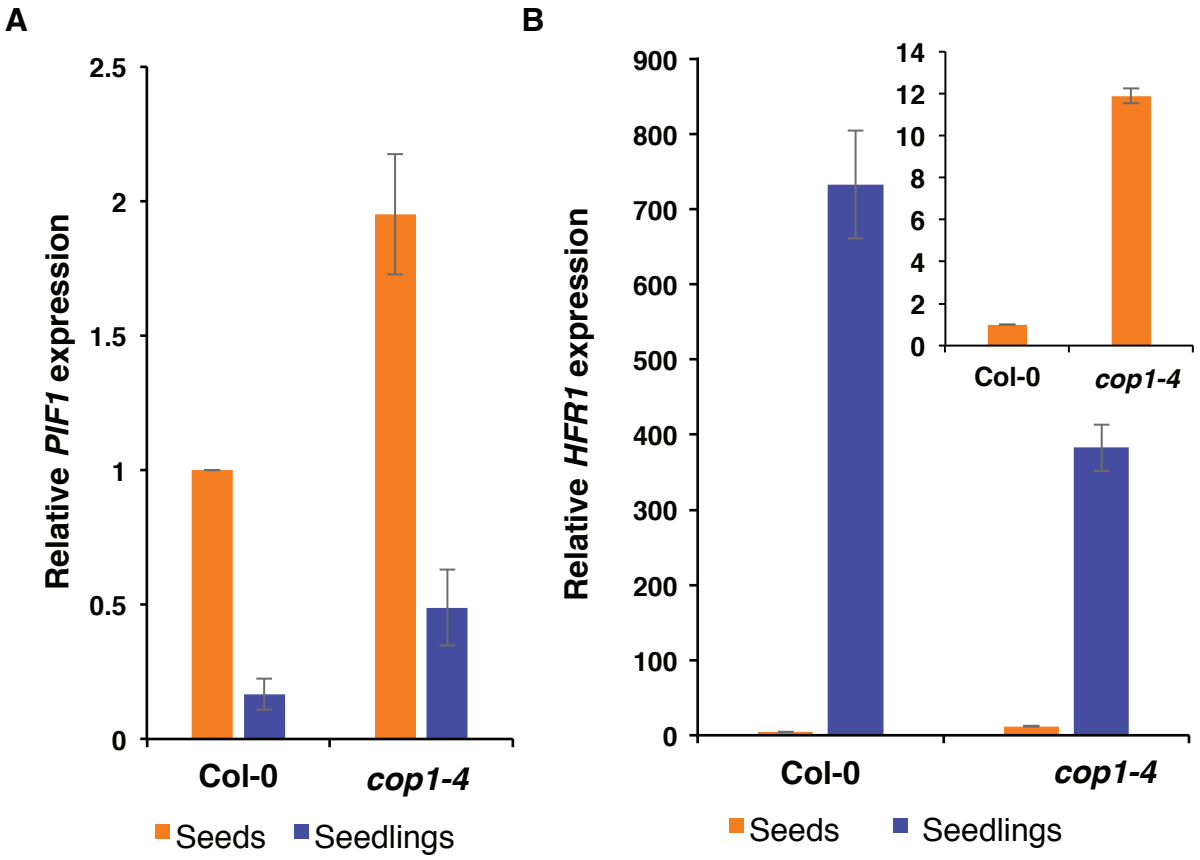
Supplemental Figure 3: HFR1* is stable in the dark and is non-functional *in vivo*.

(A) Immunoblot shows the GFP-HFR1 and GFP-HFR1* protein levels. Two batches of *Arabidopsis* seedlings expressing GFP-HFR1 or GFP-HFR1* were grown in the dark for 4 days and then one batch of seedlings was transferred to white light (WL) condition for 6 hours before total protein was extracted. Total protein was separated on 8% SDS-PAGE gel, blotted onto PVDF membrane and probed with anti-GFP or anti-Actin antibodies. (B) Quantification of GFP-HFR1 and GFP-HFR1* protein levels using Actin as a control. The letters "A" to "B" indicate statistically significant differences between means of relative protein levels of the indicated genotypes, ($p<0.05$). The error bars indicate standard deviation (n=3). C) Quantification of *GFP-HFR1* and *GFP-HFR1** mRNA levels using *PP2A* as a control in lines used in (A). Four-day-old dark-grown seedlings were used for RNA isolation. Error bars show standard deviation. ** $p<0.01$ (Student two-tailed *t*-test). D) Photographs of seedlings of various genotypes as indicated grown in the dark for 5 days or grown in the dark for 21 hours and then transferred to continuous FR light (0.45 µmol/m^2/s) for 4 days. White bar=5mm. (E and F) Bar graphs showing the hypocotyl lengths for the seedlings grown in the dark (E) or far-red light (F). Error bars indicate standard deviation. The letters "A" to "E" indicate statistically significant differences between means for hypocotyl lengths ($p<0.05$), (n>30, three biological replicates).



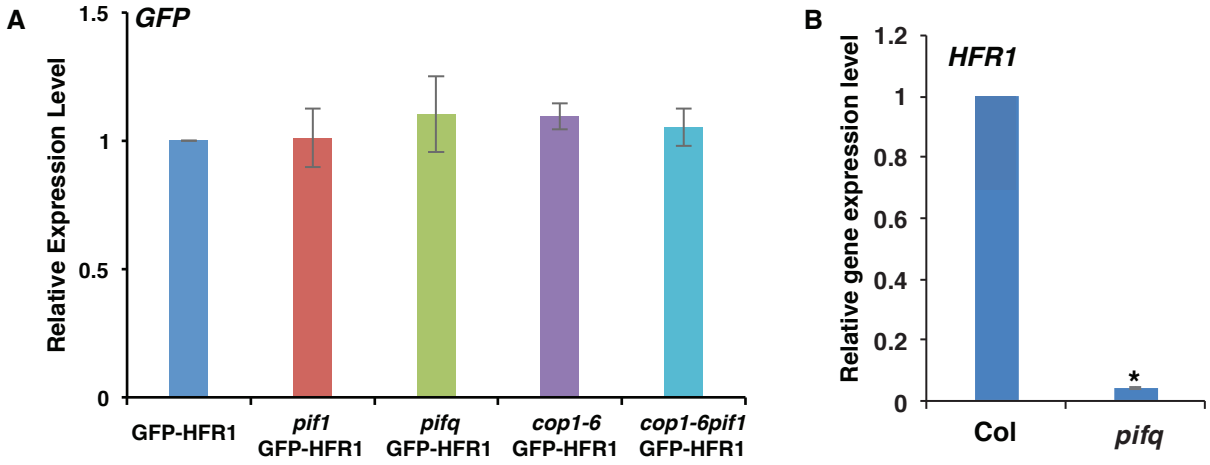
Supplemental Figure 4: GFP-HFR1* does not rescue phenotype in the *cop1-4* background.

(A and C) Photographs of seedlings of various genotypes as indicated grown in the dark for 5 days (A) or grown in the dark for 21 hours and then transferred to continuous FR light (0.45 μmol/m^2/s) for 4 days (C). White bar=5mm. (B and D) Bar graphs showing the hypocotyl lengths for the seedlings shown in A and C. Error bars indicate standard deviation. The letters "A" to "E" indicate statistically significant differences between means for hypocotyl lengths ($p<0.05$), ($n>30$, three biological replicates).



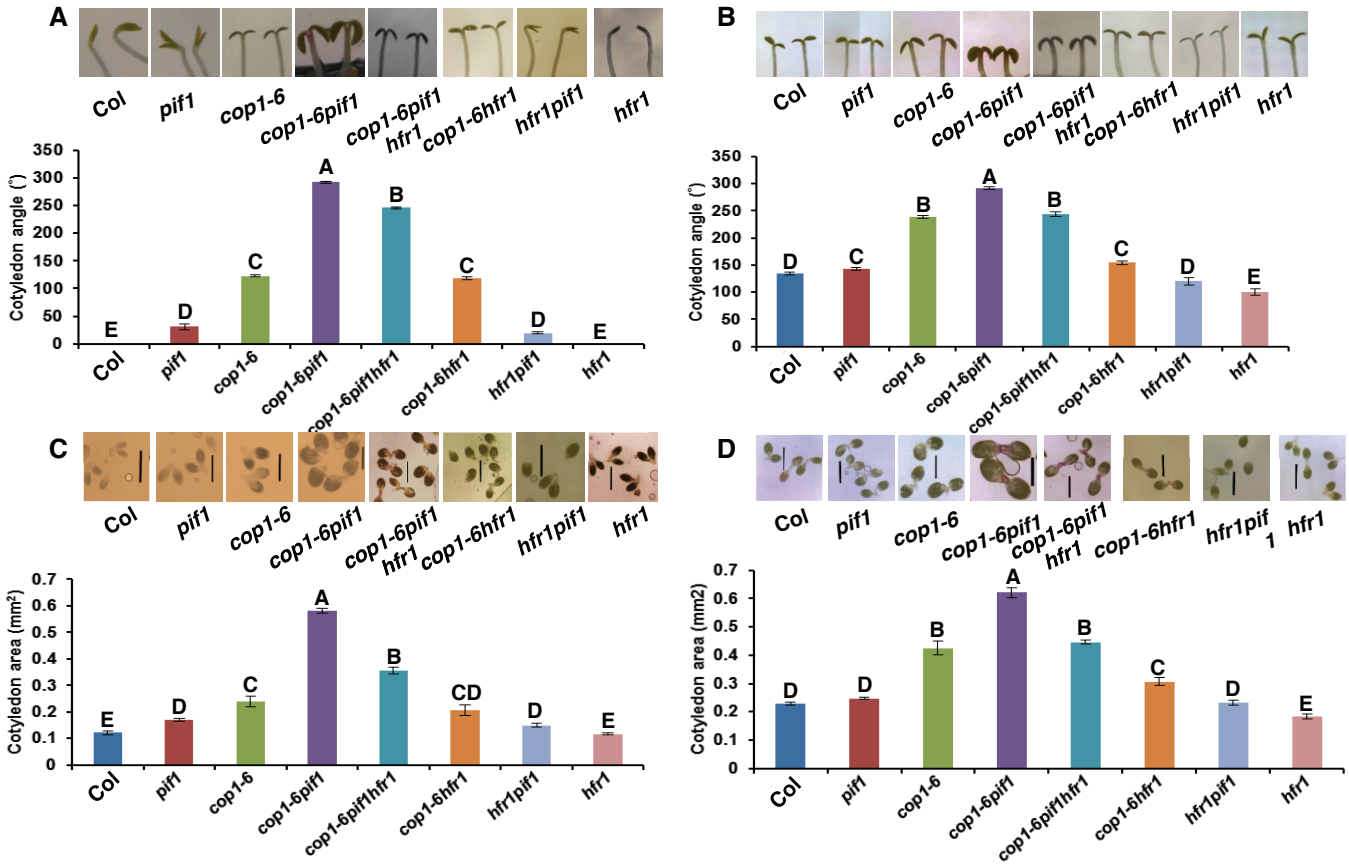
Supplemental Figure 5: Expression of *PIF1* and *HFR1* in seeds and seedlings.

PIF1 is expressed more in the seeds compared to seedlings (A), while *HFR1* is highly expressed at the seedling stage compared to seed stage (B). RT-qPCR data showing the relative expression of *PIF1* and *HFR1* in wild-type (Col-0) and *cop1-4* seedlings compared to seeds. RNA was extracted from 4-day-old dark grown wild-type Col-0, *cop1-4* seedlings and imbibed seeds. *PP2A* (At1g13320) was used as a control for normalization of the expression data. Inset in (B) shows *HFR1* expression in Col-0 and *cop1-4* seeds.



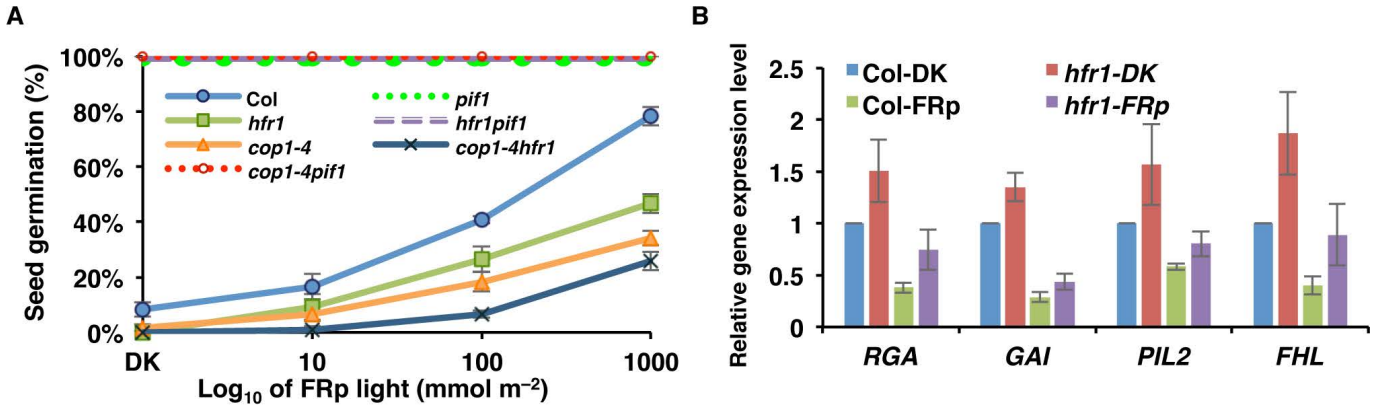
Supplemental Figure 6: *GFP* and native *HFR1* mRNA levels in various backgrounds.

(A) Bar graph showing the *GFP* mRNA levels in the different genotypes as indicated. *GFP* mRNA level was determined using RT-qPCR assays using primers designed from the *GFP* region. Total RNA was isolated from 4-day-old dark-grown seedlings for RT-qPCR assays (n= 3 independent biological repeats). *PP2A* was used as an internal control. GFP-HFR1 was set as 1 and the relative gene expression levels were calculated. Error bars indicate standard deviation. (B) Bar graph shows the native *HFR1* mRNA level in the wild type (Col-0) and *pifq* based on RNA-seq data as described {Zhang, 2013 #419}. Error bars indicate standard deviation. *, indicates significant difference ($p<0.05$).



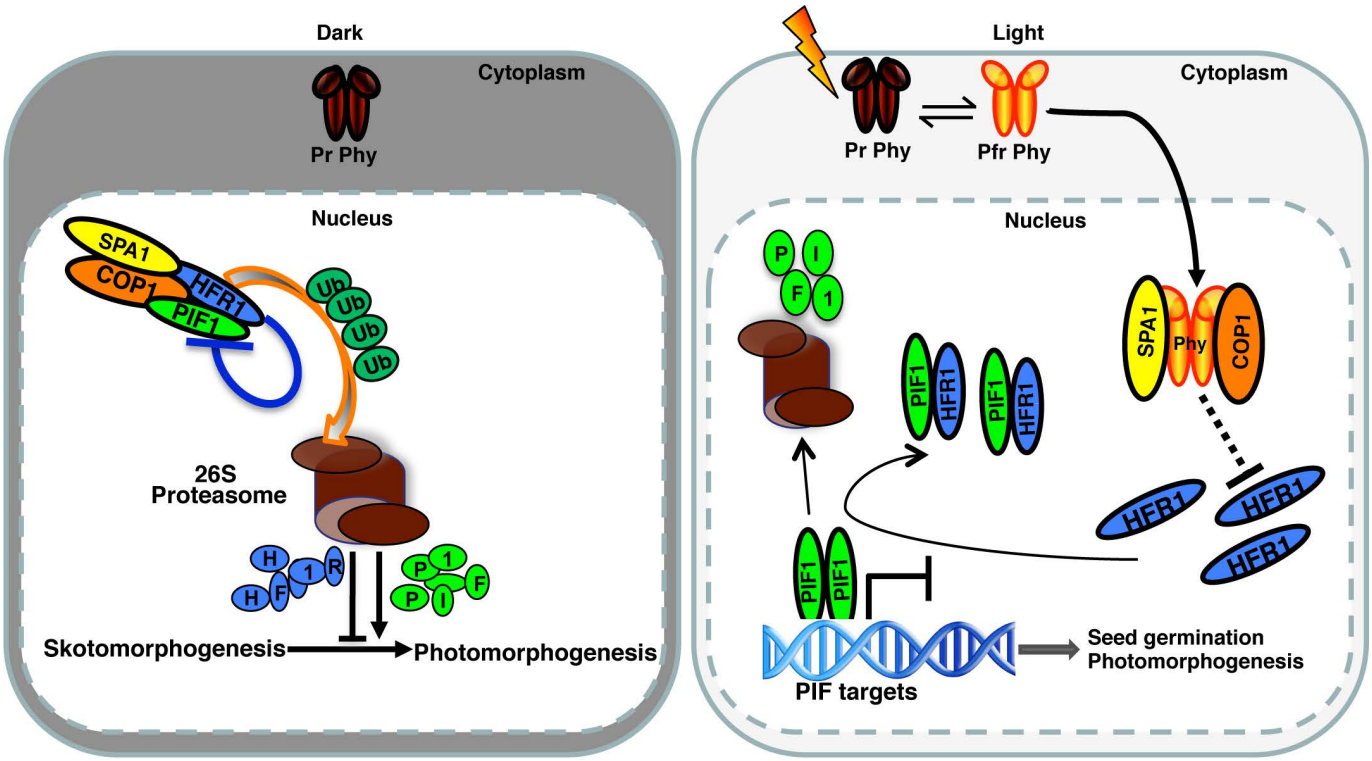
Supplemental Figure 7: *hfr1* partially suppresses the synergistic promotion of photomorphogenesis in the *cop1-6pif1* background in the dark and far-red light.

(A-B) (Top) Photographs of cotyledon angles of dark and FRc light grown seedlings, including wild type, *pif1, cop1-6, cop1-6pif1, cop1-6pif1hfr1, cop1-6hfr1, hfr1pif1* and *hfr1*. Seedlings were grown either in the dark for 5 days (A) or grown in the dark for 21 hours then transferred to continuous FRc (0.06 µmol/m^2/s) for 4 days (B). (Bottom) Bar graph showing cotyledon angles of various genotypes as indicated. (C-D) (Top) Photographs of cotyledon areas of dark and FRc light grown seedlings. (Bottom) Bar graph showing cotyledon areas of various genotypes as indicated above. Error bars indicate standard deviation. The letters "A" to "E" indicate statistically significant differences between means for hypocotyl lengths, cotyledon angle and cotyledon area of the indicated genotypes, ($p<0.05$), ($n>30$, three biological replicates).



Supplemental Figure 8: HFR1 promotes seed germination under far-red light

(A) Line graph shows the percent of seeds germinated for various genotypes as indicated in the dark and an increasing amount of far-red light intensities. Same stage seeds of Col-0, *pif1*, *hfr1*, *hfr1pif1*, *cop1-4*, *cop1-4hfr1* and *cop1-4pif1* were surface sterilized within 1 hour of imbibition and plated on the MS plates. They were exposed to far-red light (34 μmol/m^2/s) for 5 mins before being kept in the dark for 48 hours. The seeds were then either kept in the dark continuously or treated with increasing amount of far-red light as indicated and then wrapped again to keep in the dark for 6 additional days before being quantified. The error bars indicate standard deviation (n=40, three biological repeats). (B) The bar graph shows the increased expression of PIF1 direct target genes in the *hfr1* mutant seeds compared with wild type Col-0 seeds both under dark and far-red light conditions. Seeds of Col-0 and *hfr1* mutant were plated on MS plates supplemented with 100 μM paclobutrazol within 1 hour. Then they were exposed to far-red light (34 $\mu molm^{-2}\ s^{-1}$) for 5 min and kept in the dark for 48 hours. Total RNA was isolated from either 48 hours old dark-grown seeds or 48 hours old dark-grown seeds exposed to far-red light (100 $\mu molm^{-2}$) for 1 hour. Error bars indicate standard deviation (n= 3 independent biological repeats).



Supplemental Figure 9: Model showing the reciprocal degradation of PIF1 and HFR1 by COP1 during the transition from skotomorphogenesis to photomorphogenesis.

(Left) PIF1, COP1, SPA1 and HFR1 directly interact with each other to form a complex. PIF1 promotes the COP1-mediated ubiquitination and subsequent degradation of HFR1 through the 26S proteasome-mediated pathway. HFR1, in one hand, suppresses the transcriptional activity by blocking the DNA binding ability of PIF1; on the other hand, also promotes the PIF1 ubiquitination and degradation by the 26S proteasome pathway. (Right) Under light, the active Pfr form of phytochrome migrates into the nucleus and inhibits the COP1/SPA complex. This results in increased abundance of HFR1, which inhibits PIF1 function to promote seed germination and seedling de-etiolation. PIF1 is also degraded under light resulting in inhibition of PIF1 activated gene expression.

Table S1

Supplemental Table 1: Primer sequences used in experiments described in the text.

Gene	Forward	Reverse
For qRT-PCR		
GFP	AAGCTGACCCTGAAGTTCATCTGC	CTTGTAGTTGCCGTCGTCCTTGAA
PP2A	TATCGGATGACGATTCTTCGTGCAG	GCTTGGTCGACTATCGGAATGAGAG
RGA	CATTCCCGGAAACGCGATTTATCAG	TCACCGTCGTTCCTATGACTCCAC
GAI	AGCGTCATGAAACGTTGAGTCAGTG	TGCCAACCCAACATGAGACAGC
PIL2	CACCACCATGGATGATACTCTTC	TTCTTGCAAAGGGCCAAAGATCC
FHL	TCTGAGCATCAAGCCTCTCTTG	TCATCGCTGGTTTTTGTGTTCT
HFR1	ATTGGCCATTACCACCGTTTAC	TGAGGAGAAGAAGCTGGTGATG
PIF1	TGAATCCCGTAGCGAGGAAACAA	TTCCACATCCCATTGACATCATCTG
For HFR1*GFP site directed mutagenesis		
HFR1-pENTRY Cloning	CACCATGTCGAATAATCAAGCTTTCATGG	TAGTCTTCTCATCGCATGGGAAGAAAAATCC
HFR1- Mutagenesis	CAAGACGGACAAGGTTTCGGATGAGGACAAGACCATAGAG	CTCTATGGTCTTGTCCTCATCCGAAACCTTGTCCGTCTTG
For Yeast two hybrid assay		
HFR1	CGAGAATTCATGTCGAATAATCAAGCTTTC	CCTGTCGACTCATAGTCTTCTCATCGCATG
*HFR1**	CTGGAATTCATGTCGAATAATCAAGCTTTC	CTGGTCGACTCATAGTCTTCTCATCGCATG
PIF1-C328	CTGGAATTCAGAGGGGATTTTAATAACGG	CTGGTCGACTTAACCTGTTGTGTGGTTTCC