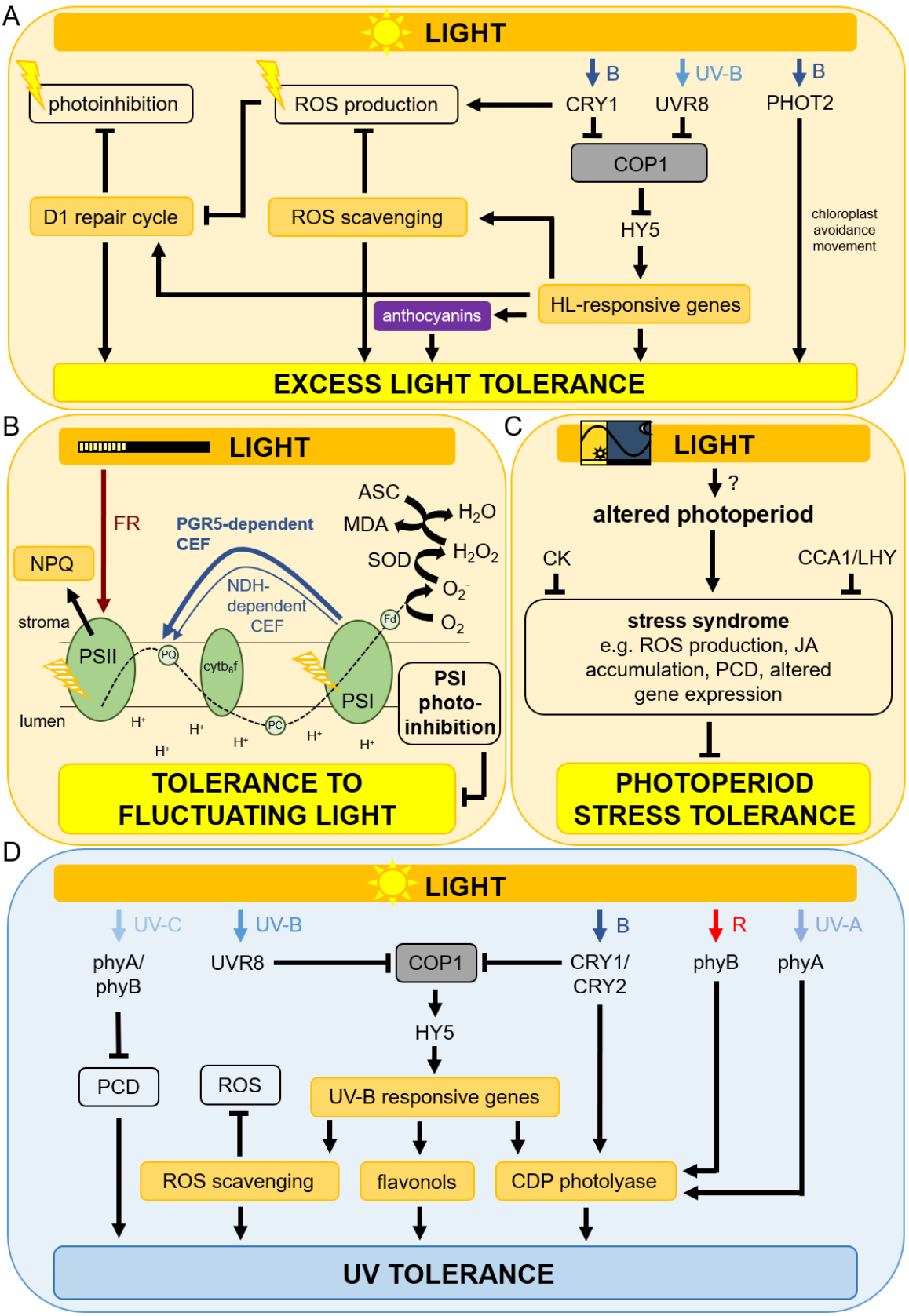

Figure 1. Signaling pathways involved in the response to light stress.

Figure 1. Signaling pathways involved in the response to light stress.
Light stress in plants can be caused by excess light, prolongation of the photoperiod, fluctuating light or UV light. (A) Excess light stress results in photoinhibition and ROS production which are counteracted by the D1 repair cycle and ROS scavenging enzymes. Upon perception of excess light, high light (HL)-responsive genes are induced resulting in the accumulation of anthocyanins but also resulting in an upregulation of genes involved in D1 repair and ROS scavenging. A specific role for blue light and UV-B, through CRY1 and UVR8 via COP1/HY5 has been shown in the regulation of HL-responsive genes. Moreover, CRY1 itself, upon light perception, produces ROS by the interconversion of the flavin redox states caused by photoexcitation. Chloroplast avoidance movement mediated through PHOT2 also contributes to enhanced high light tolerance. (B) Fluctuating light results in PSI photoinhibition. To deal with fluctuating light, plants developed alternative cyclic electron transport forces (CEF), of which the PGR5-dependent CEF has a bigger role as the NDH-dependent CEF. Together with the Mehler and water-water cycle, these CEFs act as alternative electron sinks. All these mechanisms avoid PSI photoinhibition causing tolerance to fluctuating light. Only FR light is known to ameliorate the energy dissipation via NPQ in PSII. (C) Photoperiod stress is caused by a prolongation of the light period resulting in a stress syndrome characterized by ROS production, jasmonic acid (JA) accumulation and eventually programmed cell death (PCD). Both cytokinin (CK) and CCA1/LHY are negative regulators of photoperiod stress. Recurrent photoperiod stress events reduce the stress response to subsequent stresses. (D) To overcome UV stress, plants induce UV-B-responsive genes, including genes encoding ROS scavenging enzymes, flavonols and CDP photolyases. The UV-B-responsive genes can be regulated via UVR8 and CRY1 in a COP1/HY5 dependent manner. The CPD photolyases can also be induced through phyB and phyA. Also, UV-C irradiance, perceived by phyA or phyB, results in an inhibition of PCD. For more detailed information about the different pathways, please refer to section 2. Abbreviations: B, blue light; R, red light; FR, far-red light; UV, ultraviolet light; ROS, reactive oxygen species; CPD, cyclobutene pyrimidine dimers; PS, photosystem; ASC, ascorbate; MDA, monodehydroascorbate; SOD, superoxide dismutase; PQ, plastoquinone; PC, plastocyanin; Fd, ferredoxin; cytb_6f, cytochrome b_6f; NPQ; non-photochemical quenching.

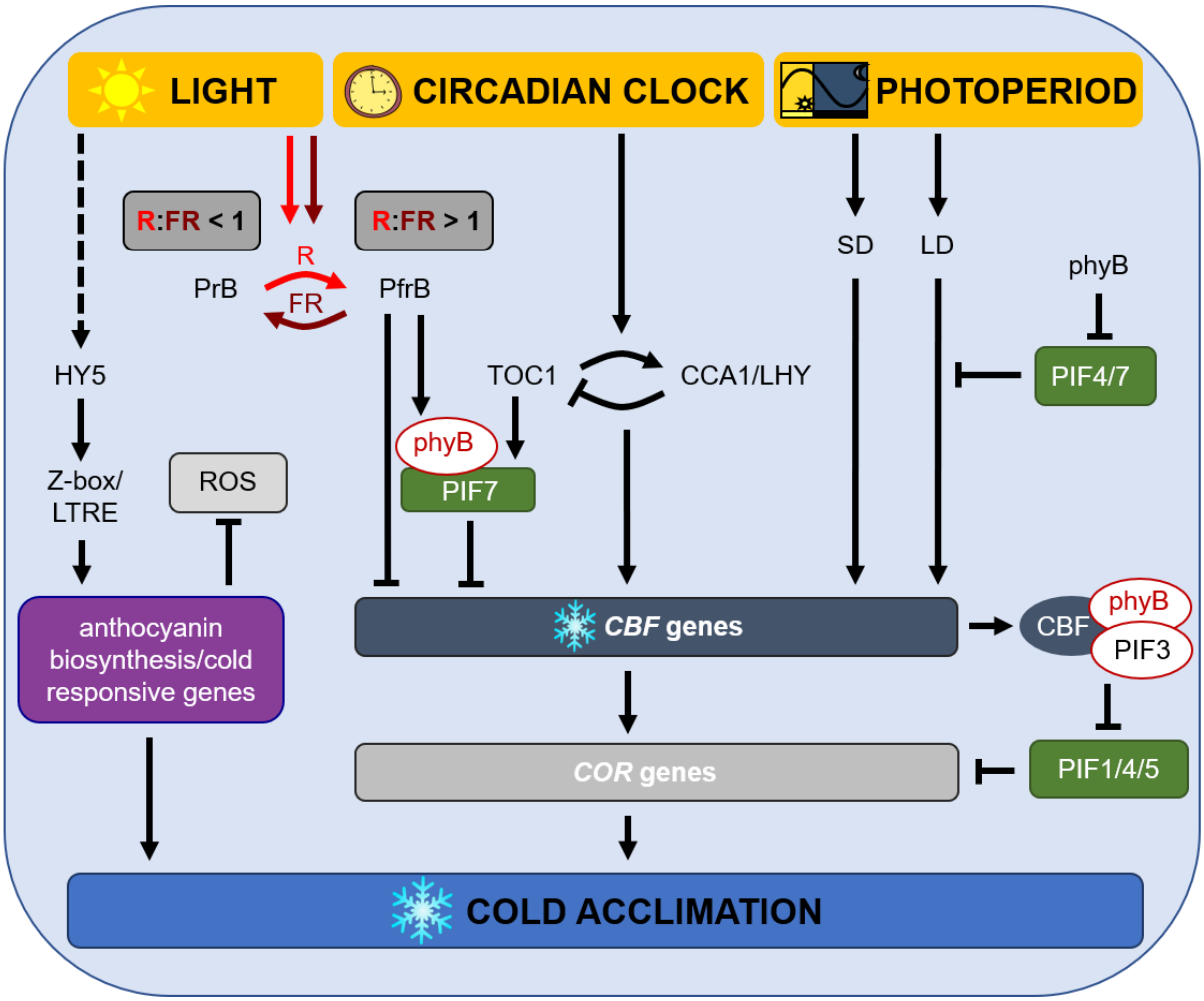

Figure 2. Signaling pathways involved in the crosstalk between light and cold acclimation.
Upon cold temperatures, the circadian clock-regulated *CBF* genes are induced resulting in cold acclimation. Upon light perception, HY5 is activated which induces the expression of anthocyanin biosynthesis and cold-responsive genes through the Z-box/LTRE thereby reducing reactive oxygen species (ROS) resulting in cold acclimation. Changes in light quality (R:FR) are sensed by phyB which exists in an active PfrB and an inactive PrB form. Under high R:FR ratios (> 1), PfrB represses *CBF* gene expression, while low R:FR ratios (< 1) caused by e.g. increased twilight during autumn causes cold acclimation by decreasing the amount of active PfrB. PIF7 which represses *CBF* gene expression is under control of TOC1, a central component of the circadian clock, and under the control of phyB. Under SD photoperiod, *CBF* genes are strongly induced causing cold acclimation. Under warmer LD, *PIF4* and *PIF7* which are under the control of phyB are higher expressed resulting in an inhibition of *CBF* gene expression. As days shorten, e.g. during autumn, this repression falls away resulting in cold acclimation. CBF proteins interact with phyB and PIF3 causing degradation of PIF1, 4 and 5 which releases *COR* genes from PIF repression. For more information concerning the different pathways, please refer to section 3.1. Abbreviations: R, red light; FR, far-red light, SD, short day; LD, long day; ROS, reactive oxygen species; LTRE, low temperature responsive element.

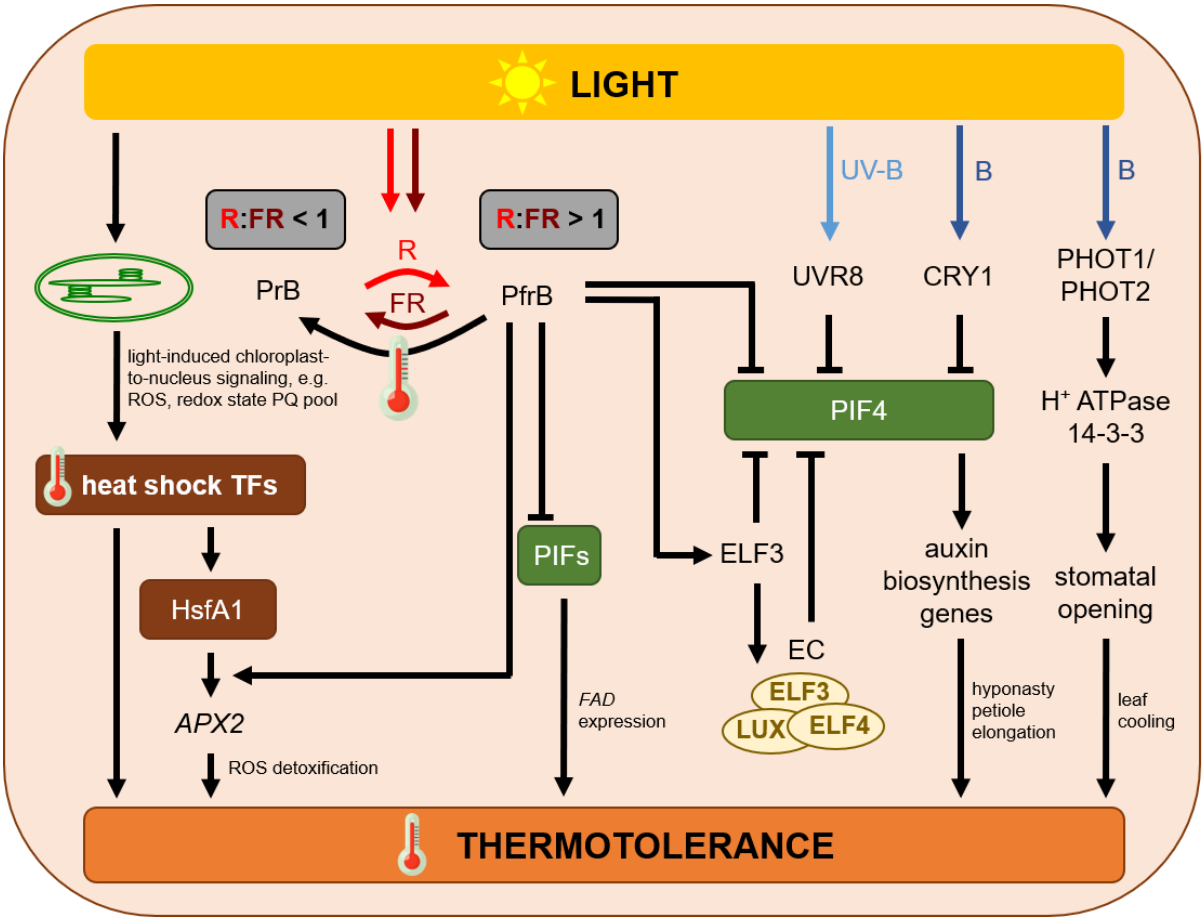

Figure 3. Light signaling pathways contributing to thermortolerance.
High temperature causes the induction of heat shock transcription factors (TFs) which results in thermotolerance. Under influence of light, a chloroplast-to-nucleus signal contributes to the induction of heat shock TFs. Independent of this chloroplast signal, the increase in HsfA1 upon heat stress causes a phyB-dependent increase in *APX2* expression resulting in ROS detoxification. The different photoreceptors are also involved in acquiring thermotolerance. Especially phyB, which is a thermosensor, and PIF4 play a central role. Thermal reversion and low R:FR ratios result in an inactivation of phyB, thereby resolving its inhibitory effect on PIF4. In turn, PIF4 stimulates the expression of auxin biosynthesis genes to regulate morphological adaptations like hyponasty or petiole elongation contributing to thermotolerance. PIF4 is inhibited by UVR8 and CRY1 as well. Also, other PIFs are regulated by phyB affecting *FAD* expression and fatty acid desaturation. phyB also influences ELF3 abundance which blocks PIF4 activity in an evening clock-independent and -dependent pathway involving also LUX and ELF4, other components of the EC. Blue light perceived by phototropins results in stomatal opening and increased leaf cooling. For more information concerning the different pathways, please refer to section 3.2. Abbreviations: R, red light; FR, far-red light; B, blue light; UV, ultraviolet light; ROS, reactive oxygen species; TFs, transcription factors; HsfA1, heat shock factor protein A1, PIFs, phytochrome interacting factors; EC, evening complex, *FAD, fatty acid desaturase*.

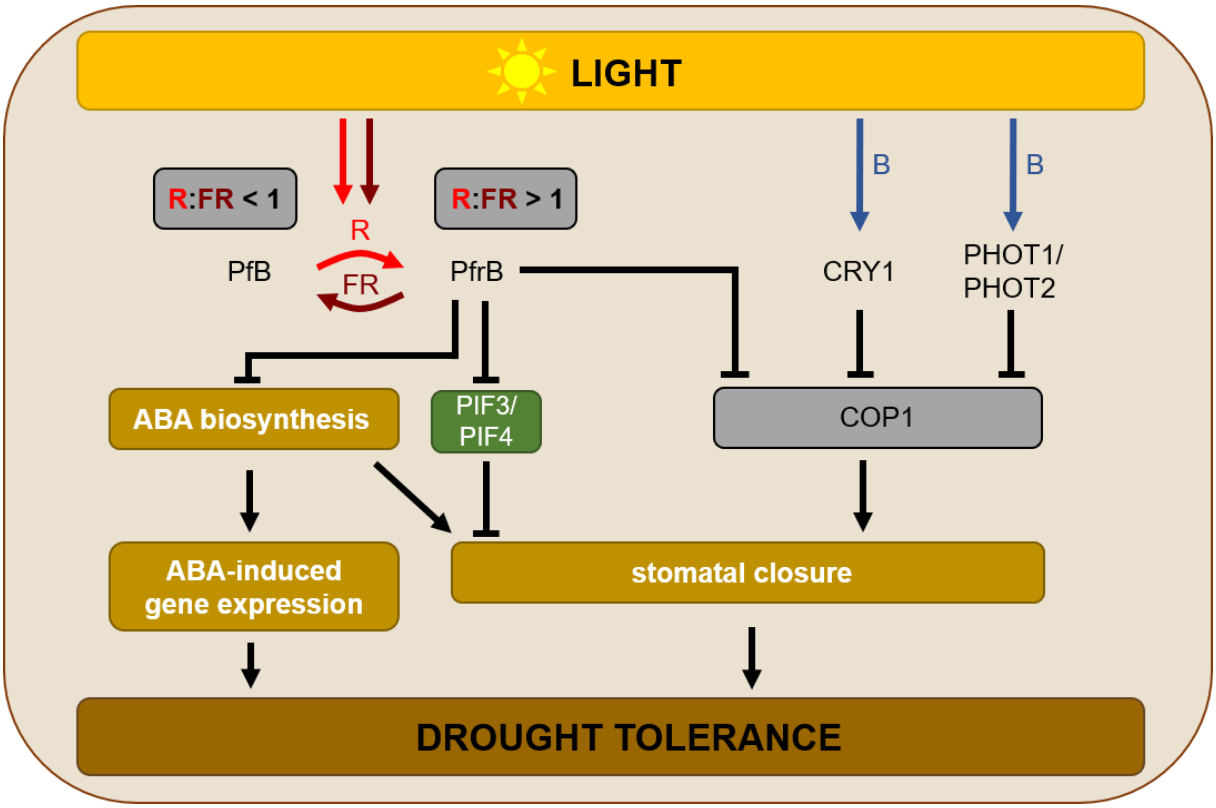

Figure 4. Light signaling pathways contributing to drought tolerance.
Drought stress results in biosynthesis of ABA leading to ABA-dependent gene regulation causing drought tolerance. In addition, stomata close preventing water loss via transpiration. Under shade conditions (low R:FR ratio), ABA biosynthesis is stimulated which results in the induction of ABA-induced gene expression causing drought tolerance. Low R:FR ratios also increase the sensitivity to ABA causing increased drought tolerance. Also, phyB acts on stomatal opening in a COP1- or PIF3/PIF4-dependent way to regulate stomatal closure. Blue light perceived by CRY1 and PHOT1/PHOT2 represses the inhibitory function of COP1 on stomatal opening resulting in closed stomata thereby increasing drought tolerance. For more information concerning the different pathways, please refer to section 4. Abbreviations: ABA, abscisic acid; R, red light; FR, far-red light; B, blue light.

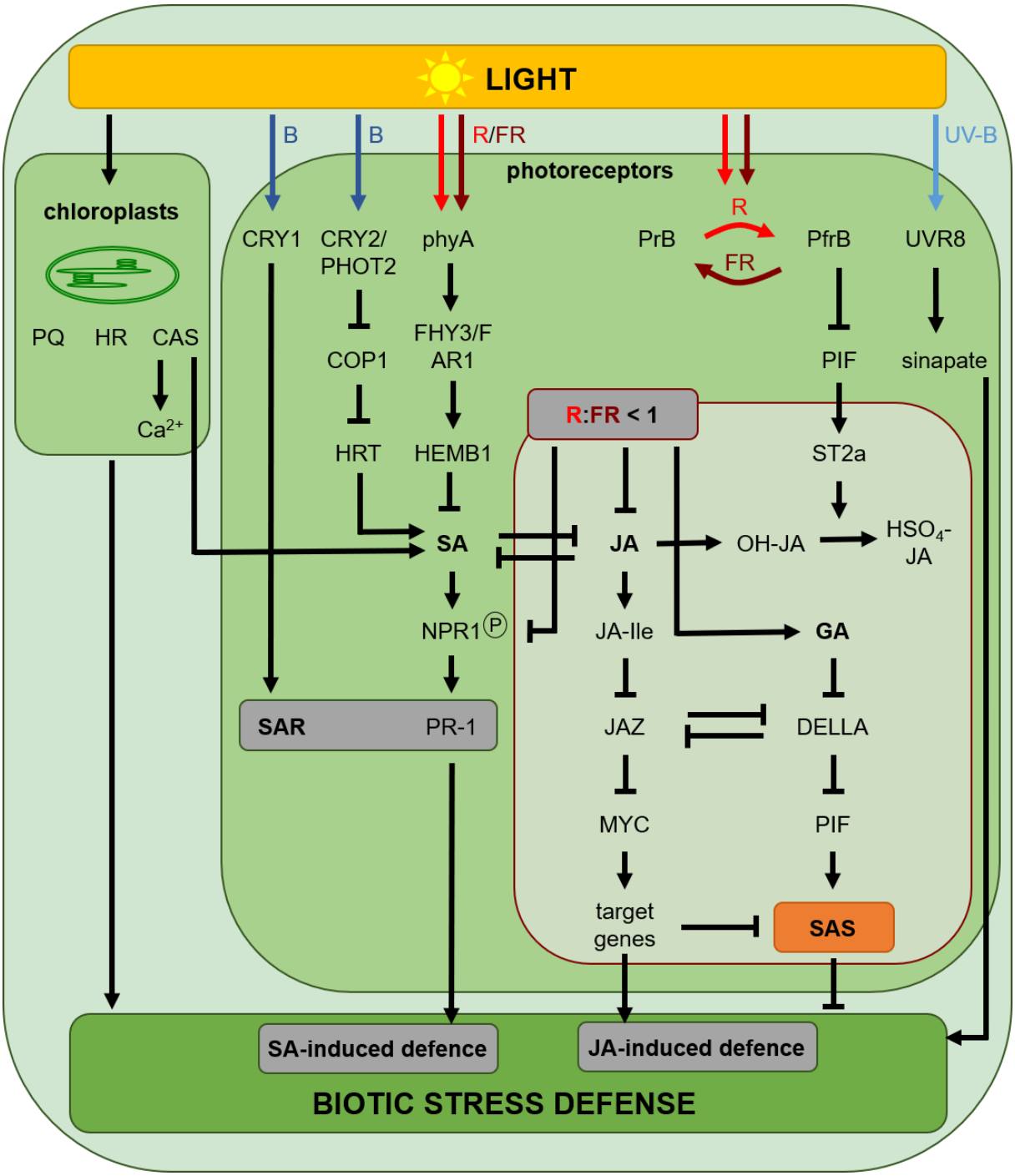

Figure 5: Signaling pathways involved in the crosstalk between light and biotic stress.

Figure 5. Signaling pathways involved in the crosstalk between light and biotic stress defense.

Plant responses to pathogen or herbivore attack are affected by light. During biotic stress defense, plants perceive and mediate light signals via chloroplasts or photoreceptors. The chloroplast redox status, especially the redox status of the plastoquinone (PQ) pool, influences the plant defense to pathogens. The functionality of chloroplasts plays an important role in mediating the plant hypersensitive response (HR). The chloroplast-located calcium-sensing receptor CAS mediates calcium signals affecting biotic stress defense. In addition, CAS controls the accumulation of salicylic acid (SA). In *Arabidopsis* plants, SA levels are regulated by blue light via a CRY2/PHOT2-mediated and by red light via phytochrome-regulated pathways. CRY2 and PHOT2 negatively regulate COP1 which in turn regulates the stability of the R protein HRT (Hypersensitive Response to TCV), thereby influencing pathogen resistance. The homologous, phytochrome-regulated TFs FHY3 and FAR1 influence SA-induced defense by controlling *HEMB1* expression. The plant SA content influences the protein kinase NPR1 which regulates the transcription of SA-induced defense genes, such as *PR-1*, representing a marker for systemic acquired resistance (SAR). CRY1 promotes *PR-1* gene expression. During shade (low R:FR ratios), phosphorylation of NPR1 is inhibited which affects SA-induced defense. Shade environments attenuate jasmonic acid (JA)-induced defense. The *Arabidopsis* sulfotransferase ST2a which is regulated by PIFs and responsible for the formation of HSO_4-JA thereby decreasing levels of active JA is upregulated under FR light conditions. The stability of JAZ proteins is enhanced during low R:FR ratios in a phyB-dependent manner leading to attenuated defense responses. In addition, gibberellin (GA) activity is enhanced during shade resulting in decreased DELLA protein functionality. DELLAs represent negative regulators of the shade avoidance syndrome (SAS) and are involved in preventing interaction of JAZ proteins with its targets. UV-B light perceived by the UVR8 receptor enhances sinapate production involved in biotic stress defense. For more information concerning the different pathways, we refer to section 5. Abbreviations: R, red light; FR, far-red light; B, blue light; UV, ultraviolet light; TFs, transcription factors.