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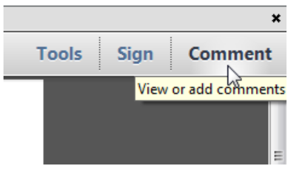

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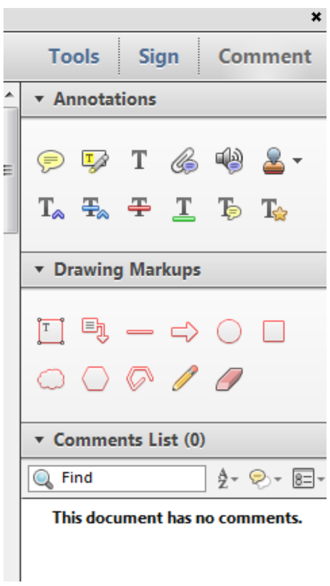

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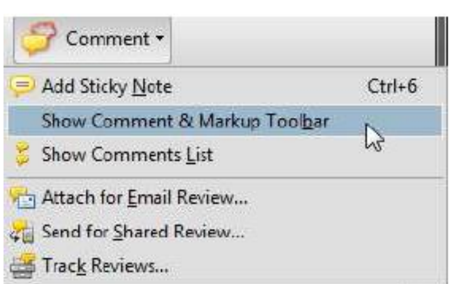

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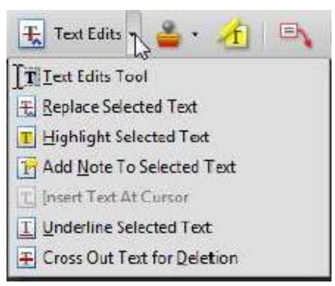

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REVIEW PAPER

The light and dark sides of nitric oxide: multifaceted roles of nitric oxide in plant responses to light

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Abstract

Light drives photosynthesis and informs plants about their surroundings. Regarded as a multifunctional signaling molecule in plants, nitric oxide (NO) has been repeatedly demonstrated to interact with light signaling cascades to control plant growth, development and metabolism. During early plant development, light-triggered NO accumulation counteracts negative regulators of photomorphogenesis and modulates the abundance of, and sensitivity to, plant hormones to promote seed germination and de-etiolation. In photosynthetically active tissues, NO is generated at distinct rates under light or dark conditions and acts at multiple target sites within chloroplasts to regulate photosynthetic reactions. Moreover, changes in NO concentrations in response to light stress promote plant defenses against oxidative stress under high light or ultraviolet-B radiation. Here we review the literature on the interaction of NO with the complicated light and hormonal signaling cascades controlling plant photomorphogenesis and light stress responses, focusing on the recently identified molecular partners and action mechanisms of NO in these events. We also discuss the versatile role of NO in regulating both photosynthesis and light-dependent stomatal movements, two key determinants of plant carbon gain. The regulation of nitrate reductase (NR) by light is highlighted as vital to adjust NO production in plants living under natural light conditions.

Keywords: De-etiolation, germination, light stress, nitric oxide, photomorphogenesis, photoreceptor, phytochrome, reactive oxygen species, stomata, UV-B.

Introduction

Light not only drives photosynthesis to produce sugars but is also one of the most reliable abiotic cues that informs plants about their surrounding environment. Plants are exposed to an ever-changing light environment, influenced by factors as diverse as shading from clouds and overlapping leaves, to gradual variations in the number of consecutive hours of light (i.e. photoperiod) throughout the year. Due to their extraordinary ability to continually monitor light quality, intensity, duration and direction, plants can coordinate flexible short- and long-term responses that facilitate growth and survival. Light-regulated development responses, also regarded as plant photomorphogenesis, include seed germination, photoperiodic flowering, shade avoidance and phototropism (Chen *et al.*, 2004; Franklin and Quail, 2010). Light perception is also vital to adjust the circadian clock, allowing the synchronization of plant growth and metabolism with the daily light/dark cycle (Sanchez *et al.*, 2020).

The information provided by the light environment is perceived by multiple plant photoreceptors: UV-B RESISTANCE LOCUS8 (UVR8) detects ultraviolet-B radiation (UV-B, 280–315 nm), phototropins (PHOTs) and cryptochromes (CRYs) both sense UV-A (315–400 nm) and blue light (BL, 320–500 nm), and phytochromes (PHYs) are sensitive to red (RL, max=660 nm) and far-red (FRL, max=730 nm) light. These photoreceptors convert light signals into physiological responses by initiating intricate downstream signal transduction cascades. As natural light is composed of different wavelengths, plants living under natural light conditions are regularly exposed to a range of wavelengths at the same time, which causes the simultaneous activation of multiple photoreceptors of the same or distinct families. The integration of stimuli from different regions of the light spectrum relies on multiple shared hubs in the signal transduction pathways triggered by each photoreceptor. Examples of these hub signaling proteins include ubiquitin ligases, notably CONSTITUTIVE PHOTOMORPHOGENESIS1 (COP1), and transcription factors (TFs) such as ELONGATED HYPOCOTYL5 (HY5) and PHYTOCHROME INTERACTING FACTORs (PIFs; Xu *et al.*, 2015; Jing and Lin, 2020). HY5 and its homolog HYH stimulate photomorphogenic development by binding directly to promoters of a large number of photomorphogenesis-related genes (Osterlund *et al.*, 2000; Lee *et al.*, 2007), whereas PIFs and PIF-like (PILs) proteins are major repressors of photomorphogenic responses (Leivar and Quail, 2011; Jing and Lin, 2020).

Plant hormones and other small signaling molecules are also responsible for shaping plant growth and development in response to the light environment (Seo *et al.*, 2009; Vanhaelewyn *et al.*, 2016). The small molecule nitric oxide (NO) has emerged as part of the signaling cascades controlling light-dependent plant responses such as seed germination, stomatal movements, light stress responses, and photosynthesis, amongst others (Beligni and Lamattina, 2000; Lozano-Juste and León, 2011; Melo *et al.*, 2016; Li *et al.*, 2018). The first report on the influence of NO on plant photomorphogenesis dates back to the year 2000, when Lamattina's group revealed that NO donors could replace, to different degrees, the light requirements for repressing hypocotyl and internode elongation, and promoting seed germination and seedling greening (Beligni and Lamattina, 2000). Since then, light was shown to regulate NO metabolism at several steps of the plant life cycle, and some new mechanisms behind the crosstalk between NO and photoreceptor-mediated signaling cascades have been characterized (Lozano-Juste and León, 2011; Melo *et al.*, 2016; Li *et al.*, 2018). In this review, we cover recent breakthroughs on NO signaling action in plant photomorphogenesis, light-dependent stomatal movement, photosynthetic reactions, and light stress responses, and highlight how NO metabolism is affected by distinct light conditions. As different light-controlled processes can affect the ability of plants to germinate, acclimate, survive and reproduce in natural and agricultural ecosystems, we also discuss the practical implications and biotechnological relevance of further understanding NO and light signaling interaction as a means to enhance productivity and stress resistance of crop plants.

Shedding light on nitric oxide metabolism

Nitric oxide metabolism in plants: a brief overview

The capacity of leaves to emit NO into the atmosphere has been reported well before the recognition of this gaseous free radical as a critical signaling molecule in plant development and stress responses (Klepper, 1979). Despite this, the mechanisms by which plant cells control NO homeostasis are still under intense debate (Astier *et al.*, 2018; Kolbert *et al.*, 2019a; León and Costa-Broseta, 2020).

Various reductive and oxidative routes for NO production in plants have been proposed, but the *in vivo* relevance and molecular mechanisms of NO biosynthesis have not been clarified so far (Fig. 1). In his pioneering study, Klepper (1979) demonstrated that treatment with photosynthesis-inhibiting herbicides induced NO emission from soybean leaves under dark conditions, in a process that was dependent on nitrite (NO_2^-) accumulation. The relationship between nitrogen metabolism and NO synthesis was further established by Dean and Harper (1986), who suggested the involvement of nitrate reductase (NR) in NO synthesis. NR catalyzes the reduction of nitrate (NO_3^-) to NO_2^-, which is further reduced to ammonium by nitrite reductase, before being converted into amino acids (Yoneyama and Suzuki, 2019). However, NO_2^- is now widely considered an important substrate for NO synthesis in plants, as it can also be reduced to NO (Astier *et al.*, 2018; Kolbert *et al.*, 2019a).

In vitro and *in vivo* assays have indicated that NR is indeed able to reduce NO_2^- to NO, which may account for 1% of its overall activity (Yamasaki *et al.*, 1999; Rockel *et al.*, 2002; Planchet *et al.*, 2005). In addition to directly generate NO, NR plays a pivotal role of providing NO_2^- to be reduced to NO by other pathways (Salgado *et al.*, 2013). Non-enzymatic reduction of NO_2^- to NO occurs at low pH and in the presence of reductants (as phenolic acids), conditions that are found in the apoplast (Bethke *et al.*, 2004). NO_2^- can also be reduced to NO by the electron transport chains of plant mitochondria and chloroplasts (Gupta *et al.*, 2005; Jasid *et al.*, 2006; Alber *et al.*, 2017), and by plasma membrane-bound nitrite: NO reductase activity in roots (Stöhr *et al.*, 2001). More recently, the molybdoenzyme amidoxime-reducing component of the alga *Chlamydomonas reinhardtii* was demonstrated to have a NO-forming nitrite reductase activity (Chamizo-Ampudia *et al.*, 2016; León and Costa-Broseta, 2020). This enzyme interacts with NR, providing electrons and NO_2^- for NO synthesis. Despite some genomic evidence, such a mechanism has not yet been functionally confirmed in higher plants (León and Costa-Broseta, 2020).

NO synthesis has also been proposed to occur through oxidative pathways using *L*-arginine (*L*-Arg) or related molecules as substrates. *L*-Arg-dependent NO production has been reported in different compartments of plant cells, indicating the existence of a nitric oxide synthase (NOS) activity similar to that found in mammals (Corpas and Barroso, 2017; Santolini *et al.*, 2017). Despite the detection of this NOS-like activity, a gene with homology to mammalian and algal *NOS* has not been identified in land plants, suggesting

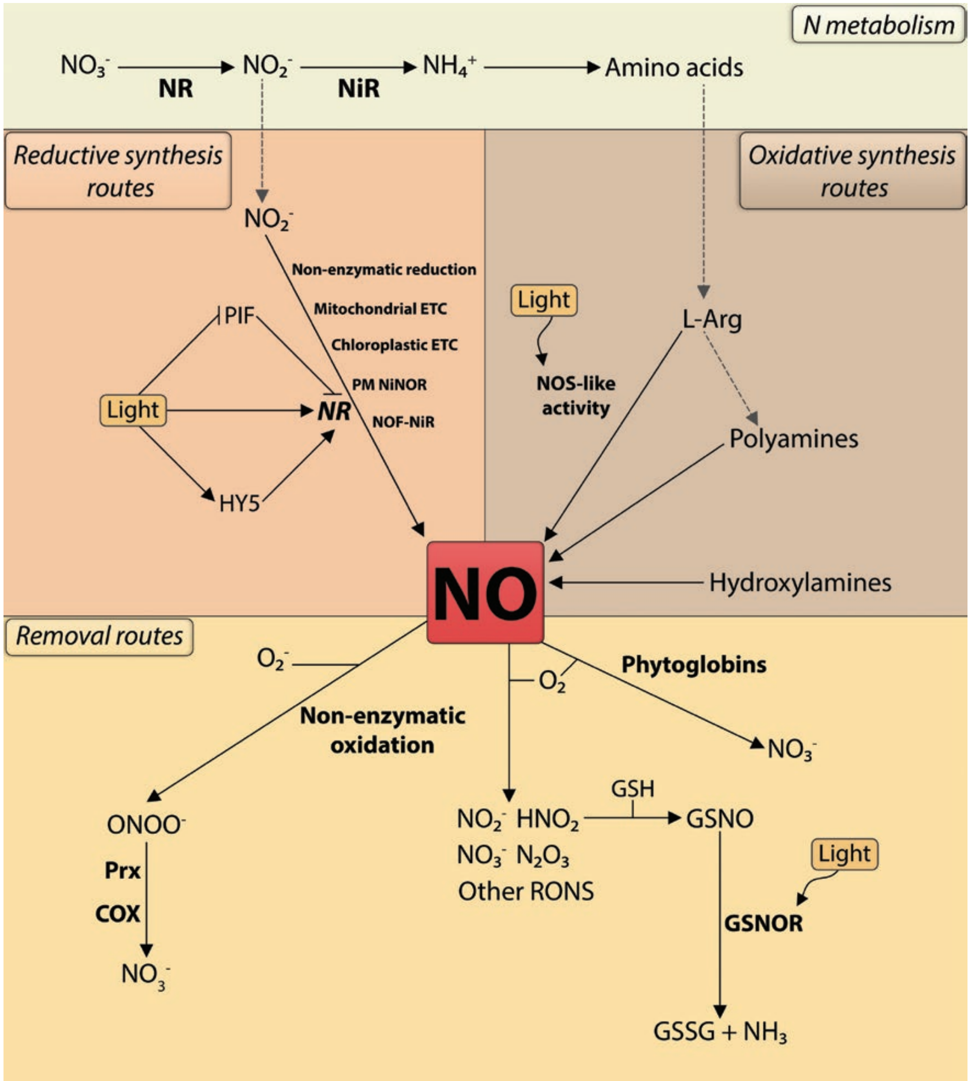

Fig. 1. Mechanisms of nitric oxide (NO) synthesis and removal in plants and their regulation by light stimuli. Nitrate reductase (NR) catalyzes the reduction of nitrate (NO_3^-) to nitrite (NO_2^-), which is further reduced to ammonium (NH_4^+) by nitrite reductase (NiR). NO_2^- can also be reduced to NO either non-enzymatically at low pH or enzymatically via NR, plasma membrane-bound nitrite: NO reductase (PM NiNOR), NO-forming nitrite reductase (NOF-NiR) or the electron transport chains (ETC) of plant mitochondria and chloroplasts. NO may also be generated by the oxidation polyamines and hydroxylamines or from L-arginine (L-Arg) via nitric oxide synthase (NOS)-like activity. NO removal involves the action of phytoglobins as well as the non-enzymatic oxidation of NO to NO_3^-. NO can react with the thiol group of reduced glutathione (GSH) generating S-nitrosoglutathione (GSNO). GSNO can be converted to oxidized glutathione (GSSG) and ammonia (NH_3) via GSNO reductase (GSNOR) activity. Light promotes both NR- and NOS-like- synthesis of NO. *NR* gene transcription is promoted and repressed by the positive and negative regulators of photomorphogenesis, ELONGATED HYPOCOTYL5 (HY5) and PHYTOCHROME INTERACTING FACTOR (PIF), respectively. Light is also involved in the post-translational activation of NR enzyme. Degradation of GSNO via GSNOR is also promoted by light. COX, cytochrome *c* oxidase; HNO_2, nitrous acid; N, nitrogen; N_2O_3, dinitrogen trioxide; O_2, molecular oxygen; O_2^-, superoxide anion; $ONOO^-$, peroxynitrite; Prx, peroxiredoxin; RONS, reactive oxygen and nitrogen species.

the absence of a canonical NOS in these organisms (Jeandroz *et al.*, 2016). Similarly, NO production from polyamines and hydroxylamines have been reported, but the involved mechanisms remain completely unknown (Tun *et al.*, 2006; Rümer *et al.*, 2009).

In addition to biosynthesis, mechanisms of NO degradation are pivotal for controlling the homeostasis of this signaling molecule in plant cells (Fig. 1). Non-enzymatic pathways for NO removal in aqueous aerobic solutions include the oxidation of NO or its derivatives to form NO_2^- or NO_3^- (Wendehenne *et al.*, 2001), and the reaction of NO with superoxide anion to form peroxynitrite ($ONOO^-$; de Oliveira *et al.*, 2008). $ONOO^-$ is an oxidant related to tyrosine (Tyr) nitration, but can be converted to NO_2^- by cytochrome *c* oxidase (Pearce *et al.*, 2002) or peroxiredoxins (Romero-Puertas *et al.*, 2007).

Some products of NO oxidation can react with thiol groups, yielding *S*-nitrosothiols (RSNO). *S*-nitrosoglutathione (GSNO) is the most abundant low-molecular-weight RSNO in cells, acting as an intracellular reservoir of NO, besides having signaling functions *per se* (Broniowska *et al.*, 2013). Although GSNO degradation may occur non-enzymatically, the enzyme GSNO reductase (GSNOR) plays a vital role in converting GSNO to oxidized glutathione and ammonia, thus regulating intracellular NO concentrations and protein *S*-nitrosation (Fig. 1; Leterrier *et al.*, 2011; Lindermayr, 2018; Jahnová *et al.*, 2019). Phytoglobins also control NO concentrations in plants

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by catalyzing the oxidation of NO to NO_3^- (Stasolla *et al.*, 2019).

Over the years, Arabidopsis mutants defective in specific NO production and degradation pathways, particularly NR (single *nia1*, *nia2* and double *nia1nia2* mutants) and GSNOR (*gsnor*), have played a significant role in clarifying NO metabolism under different contexts (Desikan *et al.*, 2002; Lozano-Juste & León, 2010, 2011; Kwon *et al.*, 2012). In addition, two mutants with alterations in plastid biogenesis, the *nitric oxide associated 1* (*noa1*) and *NO overproducer1* (*nox1*), have also been widely used in NO research because of their reduced and increased NO accumulation, respectively (He *et al.*, 2004; Flores-Pérez *et al.*, 2008; Fu *et al.*, 2016 ; Li *et al.*, 2018).

Nitric oxide metabolism at the beginning of plant photomorphogenic development

Plant photomorphogenesis initiates with seed germination and the subsequent establishment of emergent seedlings as competent autotrophic organisms (Seo *et al.*, 2009). During this challenging step of the plant life cycle, NO production appears to be up-regulated (Lozano-Juste and León, 2011; Melo *et al.*, 2016; Li *et al.*, 2018).

Although *L*-Arg-dependent NO biosynthesis has been reported to occur under some circumstances, such as in dark-grown barley and wheat seedlings transferred to light (Zhang *et al.*, 2006; Li *et al.*, 2013), NR activity has been predominantly reported as the primary source of NO during early plant photomorphogenesis (Lozano-Juste and León, 2011; Melo *et al.*, 2016; Li *et al.*, 2018). For instance, light-triggered increments in NO production in germinating seeds and de-etiolating seedlings were accompanied by concomitant elevations in NR gene expression and enzymatic activity, which depended on PHY activation (Melo *et al.*, 2016; Li *et al.*, 2018). Moreover, high concentrations of gibberellins (GAs) repressed NO production in darkness (Lozano-Juste and Léon, 2011), and GAs negatively regulated NR activity in Arabidopsis seedlings (Zhang *et al.*, 2011b). Accordingly, PHY-mediated light perception has long been shown to promote the transcription of genes involved in nitrogen assimilation, including *NR* (Lillo and Appenroth, 2001). It is known that *NITRATE REDUCTASE 2* (*NIA2*), which is the NR-encoding gene predominantly expressed in Arabidopsis green tissues, is stimulated and repressed by positive and negative regulators of photomorphogenesis, HY5/HYH and PIF4, respectively (Jonassen *et al.*, 2009 a, b; Fig. 1).

Light not only influences NO generation, but also NO degradation (Fig. 1). In dark-grown tomato seedlings, either RL- or BL-triggered NO generation was followed by an increase of the NO scavenging capacity by cotyledons, which correlated with increased RSNO content and GSNOR activity (Zuccarelli *et al.*, 2017). Furthermore, hypocotyl GSNOR activity was higher in pea seedlings under a 12 h photoperiod than under continuous darkness, reinforcing the role of this enzyme in the regulation of NO homeostasis in the light (Kubienová *et al.*, 2014).

Diel fluctuations in nitric oxide production in green tissues: a central role for nitrate reductase?

Although seed plants often initiate their life in the subterranean environment, a permanent transition to repetitive day/night cycles takes place as soon as seedlings emerge from the soil. Therefore, for most of their life cycle, plants continuously monitor the diel cycle by combining inputs from the photoreceptor-mediated detection of light stimuli and the rhythmic nature of light-dependent photosynthetic reactions (Sanchez *et al.*, 2020).

NO production has been shown to vary significantly over the 24 h light-dark cycle (Rockel *et al.*, 2002). Interestingly, the influence of light on NO homeostasis in green, mature leaves has been outlined in the very first report describing NO production by plants. Klepper (1979) observed that short-term NO emission by soybean leaves upon 2,4-D treatment was much higher in darkness than in the presence of light. The herbicide was shown to promote the accumulation of NO_2^-, a substrate for NO synthesis. The inhibitory effect of light on NO evolution was related to the activation of nitrite reductase, which decreased NO_2^- concentrations in the cells.

In contrast to Klepper's results, which were obtained in a particular experimental condition (i.e. herbicide treatment), subsequent studies showed a different scenario, in which light exposure promoted NO production in green plant tissues (Wildt *et al.*, 1997; Rockel *et al.*, 2002; Planchet *et al.*, 2005), which seems to be linked to the influence of this environmental cue on transcriptional and post-translational regulation of NR. NR gene expression and enzyme activity fluctuate within the 24 h cycle, in part due to the robust control by the circadian clock (Jones *et al.*, 1998; Lillo *et al.*, 2001; Freschi *et al.*, 2009). At the post-translational level, light regulates the phosphorylation state of NR. In the dark, NR is phosphorylated at a conserved serine residue, which allows the binding of 14-3-3 proteins and divalent cations, leading to NR inactivation (Lillo *et al.*, 2004). In the presence of light, NR is dephosphorylated by a photosynthesis-dependent process, resulting in its activation (Lillo and Appenroth, 2001; Lillo *et al.*, 2004).

In agreement, spinach leaves maintained under dark conditions emitted less NO than in the light, which was consistent with lower NR activity and NO_2^- concentrations, whereas the illumination of dark-grown sunflower plants led to a rapid increase of NO flux (Rockel *et al.*, 2002). In contrast, when illuminated leaves were transferred to darkness, a transient increase in NO production was observed, which correlated with transient NO_2^- accumulation. As NO_2^- concentrations decreased, the NO flux decayed to values below than those of light-exposed leaves (Rockel *et al.*, 2002). This "light-off peak" of NO emission in light-dark transition, as well as the strong induction of NR-dependent NO evolution by light, were also reported in a study with tobacco leaves (Planchet *et al.*, 2005). It is noteworthy that in the pioneering work of Klepper (1979), a decay of NO emission by soybean plants was observed after 2 h of darkness; a response presumably related to NR inactivation via dark-induced protein phosphorylation.

Nitric oxide action in plant photomorphogenesis

Interaction of nitric oxide and light signaling in germinating seeds

Studies in Arabidopsis have started to elucidate the NO-PHY interplay during seed germination (Batak *et al.*, 2002; Li *et al.*, 2018). Amongst the five Arabidopsis PHY proteins (PHYA-E), PHYA and PHYB are most relevant for seed germination in response to FRL and RL, respectively (Seo *et al.*, 2009), with PHYB being particularly important during early events of seed germination. In the presence of RL, PHYB moves from the cytosol to the nucleus, where it promotes the degradation of PIFs and promotes the transcription of *HY5* (Shen *et al.*, 2005). As part of a fail-safe mechanism, LONG HYPOCOTYL IN FAR-RED (HFR1), which is known to sequester PIF1 and restrain *PIF1* transcriptional activity, requires light to accumulate in plant cells (Shi *et al.*, 2013). In this signaling context, NR-derived NO production was demonstrated to promote PHYB-mediated seed germination by both down-regulating *PIF1* transcription, and stabilizing HFR1 protein (Li *et al.*, 2018). Therefore, NO fine-tunes light-regulated seed germination by intensifying the HFR1-PIF1 regulatory module, which in turn alleviates PIF1-mediated repression of genes associated with the hormonal and metabolic rewiring required for germination. NO has also been reported to participate in PHYA-mediated germination (Batak *et al.*, 2002); however, the mechanism behind PHYA-NO interaction in imbibed seeds remains elusive.

A central aspect in light-regulated germination is the influence of the photosensory systems on the relative abundance of, and sensitivity to, plant hormones such as abscisic acid (ABA) and GAs (Seo *et al.*, 2009; Barrero *et al.*, 2014). As a dormancy-relieving molecule and promoter of seed germination, NO closely interacts with both these hormonal classes to fine-tune the germination process, according to the environmental conditions (Bethke *et al.*, 2007; Liu *et al.*, 2009; Sanz *et al.*, 2015; Fig. 2A). Analysis of Arabidopsis mutants with altered NO amounts, as well as treatment with NO donors, revealed that NO alleviates seed dormancy by reducing ABA sensitivity in imbibed seeds (Bethke *et al.*, 2006; Lozano-Juste and León, 2010). The regulation of the abundance of ABA INSENSITIVE5 (ABI5), a TF responsible for ABA-mediated post-germinative seedling arrest (Lopez-Molina *et al.*, 2001), represents a central hub of NO action during seed germination and initial seedling growth (Gibbs *et al.*, 2014; Albertos *et al.*, 2015). NO was demonstrated to control *ABI5* transcription via regulation of the stability of group VII ethylene response factors (ERFs), with NO-mediated degradation of ERFVIIs proposed as the basis of NO sensing during germination and other plant responses (Gibbs *et al.*, 2014). Moreover, *S*-nitrosation stimulates the degradation of ABI5 and promotes seed germination and seedling growth, whereas ABI5 protein accumulation perturbs the inhibition of seed germination by reducing endogenous NO concentrations (Albertos *et al.*, 2015). NO also alleviates the inhibitory effect of ABA on seed germination by *S*-nitrosation and inactivation of SNF1-RELATED PROTEIN KINASE 2.2 (SnRK2.2), and presumably SnRK2.3 (Wang *et al.*, 2015a), which are protein kinases involved in ABI5 phosphorylation and activation (Nakashima *et al.*, 2009). Considering that ABI5 is also a convergence point of light and ABA signaling during seed germination, with HY5 acting as a direct activator of *ABI5* expression (Chen *et al.*, 2008), it seems plausible to anticipate some role for ABI5 in NO-light crosstalk in germinating seeds. Another relevant mechanism controlling the sensitivity of plant tissues to ABA relies on the Tyr nitration-mediated inactivation of PYR/PYL/RCAR (PYRABACTIN RESISTANCE 1/PYR1-LIKE/REGULATORY COMPONENTS OF ABA RECEPTORS) family of ABA receptors, which is described as a rapid NO-mediated mechanism to locally restrict hormone action (Castillo *et al.*, 2015). As seed imbibition promotes both NO and hydrogen peroxide (H_2O_2) increase

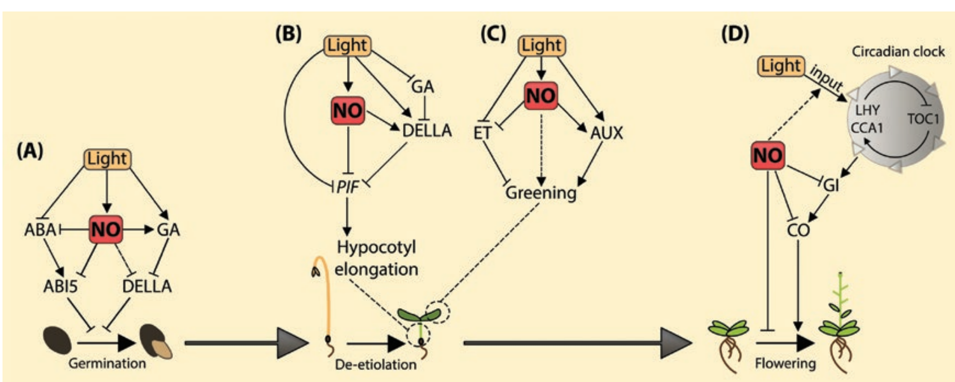

Fig. 2. NO, light and hormone interaction in plant photomorphogenesis. (A) In light-dependent seed germination, NO promotes abscisic acid (ABA) degradation, represses the accumulation of ABA INSENSITIVE5 (ABI5), up-regulates gibberellin (GA) biosynthesis, and possibly facilitates DELLA degradation. (B) During seedling de-etiolation, NO inhibits hypocotyl elongation through the repression of GA accumulation, reduction in PHYTOCHROME INTERACTING FACTOR (*PIF*) expression and promotion of DELLA accumulation. (C) NO also mediates light-triggered cotyledon greening by repressing ethylene (ET) synthesis and promoting auxin (AUX) accumulation and signaling. (D) In photoperiodic floral transition, NO affects the light-dependent inputs to, and output components from, the circadian clock, causing delayed flowering. Output components of the circadian clock, such as CO (CONSTANS) and GI (GIGANTEA), are major regulators of flowering time. Dashed lines indicate potential pathways. CCA1, CIRCADIAN CLOCK ASSOCIATED 1; LHY, LATE ELONGATED HYPOCOTYL; TOC1, TIMING OF CAB EXPRESSION1.

(Liu *et al.*, 2010), monitoring the impacts of *in vivo* Tyr nitration of ABA receptors at early stages of seed germination remains an interesting topic for future investigation.

Besides controlling ABA sensitivity, NO influences ABA and GA abundance in imbibed seeds. NO is released at the endosperm layer within hours after seed imbibition and accelerates ABA degradation by promoting the transcription of the ABA 8′-hydroxylase gene *CYP707A2* (Liu *et al.*, 2009). Aleurone cells also respond to NO by up-regulating key genes encoding the GA biosynthetic enzyme GA_3 oxidase (*GA3ox1* and *GA3ox2*), which in turn leads to structural changes in the protein storage vacuoles of these cells (Bethke *et al.*, 2007). NO further promotes the hydrolysation of storage starch (Zhang *et al.*, 2005; Wu *et al.*, 2013) and the expression of cell wall loosening-related genes (Li *et al.*, 2018) in germinating seeds, two key processes also regulated by the ABA/GA balance. Therefore, NO may represent a key piece of the puzzle interconnecting PHY-PIF signaling cascade, ABA catabolism and GA biosynthesis during light-dependent seed germination (Seo *et al.*, 2009; Barrero *et al.*, 2014; Fig. 2A). In agreement, the overproduction of NO conferred by the *nox1* mutation was shown to intensify the promotive effect of HFR1 on the expression of *CYP707A2*, *GA3ox1* and *GA3ox2*, as well as cell wall loosening-related genes, in imbibed seeds of Arabidopsis (Li *et al.*, 2018). Since NO interferes with DELLA accumulation during hypocotyl elongation (Lozano-Juste and León, 2011), determining whether DELLA stability is also influenced by NO during seed germination remains an interesting topic for future research.

An incomplete picture of NO interaction with other signaling molecules and photoreceptors during light-regulated seed germination is also emerging. This includes the action of phospholipase D (PLD)-mediated phosphatidic acid (PA) production as a downstream signal of NO in light-induced lettuce seed germination (An and Zhou, 2017), and NO, ABA and BL interaction during tomato seed germination under osmotic stress (Piterková *et al.*, 2012). In addition, salt-induced accumulation of ETHYLENE INSENSITIVE 3 (EIN3), a critical ethylene-related TF, requires NO production under light in imbibed Arabidopsis seedlings (Li *et al.*, 2016). Since EIN3 protein stability during early plant development is regulated by light in a PHYB-dependent manner (Shi *et al.*, 2016), and ethylene is known to affect seed germination in several species (Arc *et al.*, 2013), a crosstalk between NO, ethylene and PHY signaling cascades may be relevant during seed germination, particularly under unfavorable conditions.

Nitric oxide and light signaling interplay during seedling de-etiolation

Seedlings growing through the soil must adjust their growth to absent or limited light supply via etiolated growth (i.e. skotomorphogenesis). After emerging from the soil, seedlings may encounter adequate light conditions and initiate de-etiolated, autotrophic growth, which involves deceleration of hypocotyl elongation, unfolding of cotyledons, and opening of the apical hook, amongst other processes (Seluzicki *et al.*, 2017).

Decades of research in plant photobiology have progressively dissected the molecular mechanisms repressing and promoting seedling photomorphogenesis under dark and light conditions, respectively (Seluzicki *et al.*, 2017). DELLA proteins physically interact with PIF1, PIF3, and PIF4 to impede these TFs from binding to their targets, which culminates in the inhibition of hypocotyl elongation (Feng *et al.*, 2008). Evidence points out that NO is also part of the light-GA signaling crosstalk controlling Arabidopsis hypocotyl growth (Lozano-Juste and León, 2011; Fig. 2B). Light and GAs antagonistically regulate hypocotyl elongation by promoting the accumulation and degradation of DELLA proteins, respectively (Feng *et al.*, 2008). NO-deficient mutants display more elongated hypocotyls than the wild type exclusively under RL, and this phenotypic difference is linked to higher transcript abundance of *PIF1*, *PIF3*, and *PIF4*, reduced DELLA accumulation, and altered GA sensitivity (Lozano-Juste and León, 2011). In contrast, treatment with increasing concentrations of a NO donor resulted in progressively shorter hypocotyls under RL, a response directly correlated with DELLA accumulation (Lozano-Juste and León, 2011). PIF3 was also identified as the TF most highly associated with NO sensitivity in etiolated seedlings (Castillo *et al.*, 2018). As in seed germination, the NO-mediated regulation of the turnover of ERFVIIs is proposed to regulate NO sensing in etiolated hypocotyls (Gibbs *et al.*, 2014).

The switch from heterotrophic to autotrophic growth in de-etiolating seedlings requires the conversion of etioplasts into green, photosynthetically active chloroplasts. Exogenous NO has been recurrently shown to induce or intensify chlorophyll accumulation and chloroplast maturation during early plant development, as reported in dark-grown wheat seedlings (Beligni and Lamattina, 2000; Liu *et al.*, 2013), apple embryos under photoperiodic conditions (Krasuska *et al.*, 2015), *PHY*-deficient tomato seedlings under RL conditions (Melo *et al.*, 2016), and barley seedlings transferred from dark to white light conditions (Zhang *et al.*, 2006). Furthermore, the progressive light-mediated chlorophyll accumulation in etiolated tissues is reported to be accompanied by a gradual increase in NO production (Zhang *et al.*, 2006; Melo *et al.*, 2016), with the intensity of chloroplast maturation correlated with the NO production rates across photomorphogenic mutants (Melo *et al.*, 2016). NO-mediated repression of ethylene synthesis and promotion of auxin accumulation and signaling were characterized as essential to allow the transcription of plastid division and differentiation genes in tomato seedlings (Melo *et al.*, 2016; Fig. 2C). As these two hormonal classes are highly regulated by light at both metabolic and signaling levels (Halliday *et al.*, 2009; Rodrigues *et al.*, 2014), and are implicated in many other aspects of seedling de-etiolation (Zhong *et al.*, 2014; de Wit *et al.*, 2016), the interplay between auxin, ethylene and NO in early events of plant photomorphogenesis remains a promising target for future research.

The interplay between PHY, PIF3 and NO also seems to coordinate root growth in light, as NO-mediated root growth in light-exposed Arabidopsis seedlings was directly linked to changes in PHYB and PIF3 protein accumulation (Bai *et al.*, 2014). Furthermore, NO was reported to mediate light-triggered morphological changes in rice seminal roots, acting upstream of auxin and ethylene (Chen *et al.*, 2015).

The role of nitric oxide in other plant photomorphogenic responses: what are we missing?

Compared with early events in plant photomorphogenesis, much less is known about the involvement of NO in light-regulated developmental processes that take place later in the plant life cycle. Floral transition, for instance, can be regulated by seasonal changes in day length (i.e. photoperiodic flowering; Song *et al.*, 2013), and is repressed by NO (He *et al.*, 2004; Kwon *et al.*, 2012; Zhang *et al.*, 2017). In Arabidopsis, PHY- and CRY-dependent inputs to the circadian clock affect the expression of key components of the central oscillators, such as CCA1 (CIRCADIAN CLOCK ASSOCIATED 1), LHY (LATE ELONGATED HYPOCOTYL), and TOC1 (TIMING OF CAB EXPRESSION 1), whereas CO (CONSTANS) and GI (GIGANTEA) act as output components of the circadian clock to regulate flowering time (Song *et al.*, 2013; Sanchez *et al.*, 2020). Reports indicate that NO down-regulates *CO* and *GI* expression (He *et al.*, 2004; Zhang *et al.*, 2019; Fig. 2D), and both these output components of the circadian clock can be *S*-nitrosated (Zhang *et al.*, 2019). NO-mediated changes in transcript abundance of the input gene *CRY1* and the central oscillator genes *LHY*, *CCA1* and *TOC1* were also reported (Zhang *et al.*, 2019), which can further explain the repressive role of NO on light/circadian regulation of floral transition in Arabidopsis. In animal systems, NO is necessary for circadian photic entrainment, and the daily NOS-dependent NO production is responsible for generating phase shifts of circadian rhythms (Golombek and Rosenstein, 2010; Vinod and Jagota, 2016). Whether daily changes in NO production are also linked to circadian rhythms in plants remains to be investigated. Fruit growth and ripening are also critically influenced by both NO (Corpas *et al.*, 2018; Palma *et al.*, 2019) and light signaling (Bianchetti *et al.*, 2018; Cruz *et al.*, 2018; Alves *et al.*, 2020), but the interaction between these two pathways remains to be investigated in this context. Moreover, given the multiple links between NO and auxins (Freschi, 2013), and the critical role of auxins in photomorphogenic responses, including phototropism and shade-avoidance responses (de Wit *et al.*, 2016), further investigation about NO–auxin crosstalk in plant photomorphogenesis is needed.

Light as an energy source: nitric oxide action in carbon assimilation

Role of nitric oxide in mediating light-dependent stomatal movements

Light intensity and quality are major determinants of photosynthetic rate and sugar synthesis in plants. As gateways linking the intercellular gas spaces to the external environment, stomatal movements balance atmospheric CO_2 uptake by leaves, which is vital for photosynthesis, along with water loss to the atmosphere. To carry out this critical role, guard cells integrate a multitude of external and endogenous stimuli to modulate stomatal aperture (Matthews *et al.*, 2020). Amongst them, light promotes stomatal opening in C_3 and C_4 species via two pathways: (i) the guard cell-specific response to BL, which saturates at low fluence rates (~10 μmol m^{-2} s^{-1}; Shimazaki *et al.*, 2007), triggers photosynthesis-independent stomatal opening at early morning; whereas (ii) the RL-triggered stomatal opening requires high fluence rates and is believed to coordinate stomatal behavior and photosynthesis (Matthews *et al.*, 2020).

Under BL, phototropins are activated via autophosphorylation and initiate a signaling cascade within the guard cells, involving the protein kinase BLUE LIGHT SIGNALLING 1 (BLUS1), and type 1 protein phosphatase (PP1), among other components (Takemiya *et al.*, 2006; Matthews *et al.*, 2020). This BL-triggered signaling cascade promotes H^+ pumping by activating H^+-ATPase in the plasma membrane of the guard cells, causing membrane hyperpolarization and driving the uptake of K^+ into guard cells through inward-rectifying K^+ channels (Takemiya *et al.*, 2006; Shimazaki *et al.*, 2007; Hayashi *et al.*, 2011; Fig. 3). The uptake of K^+, combined with the accumulation of the counter-ions malate (produced via starch degradation) and Cl^- in the vacuole, drives water movement into guard cells leading to swelling and stomatal pore opening (Matthews *et al.*, 2020). BL-triggered stomatal opening can be reversed by ABA to minimize water loss during day time (Goh *et al.*, 1996), with ABA inhibiting plasma membrane H^+-ATPase, and promoting membrane depolarization and K^+ efflux from the guard cells (Schroeder and Hagiwara, 1990; MacRobbie, 1992; Thiel *et al.*, 1992; Goh *et al.*, 1996; Zhang *et al.*, 2004).

Over the last two decades, NO has been repeatedly implicated as a downstream signal in ABA-induced stomatal closure (Desikan *et al.*, 2002; Neill *et al.*, 2002; Garcia-Mata *et al.*, 2003; Bright *et al.*, 2006; Murata *et al.*, 2015), as the NO concentrations in guard cells usually increase following ABA treatment, whereas the application of NO scavengers prevents ABA-induced stomatal closure (García-Mata and Lamattina, 2001; Neill *et al.*, 2002; Zhang *et al.*, 2004). ABA-induced NO production was also shown to cause *S*-nitrosation of SnRK2.6 (also known as OPEN STOMATA 1-OST1), inactivating this central component of ABA signaling in guard cells (Wang *et al.*, 2015b). However, other lines of evidence suggest that, rather than acting as an intermediate of ABA, NO would be limited to fine-tune stomatal apertures through alternative pathways (van Meeteren *et al.*, 2020).

Although NO action in stomatal closure under rapid dehydration is currently under debate (van Meeteren *et al.*, 2020), the role of NO in coordinating stomatal aperture in response to light/dark cycles in well-hydrated plants remains unquestioned (Ribeiro *et al.*, 2009; Wilson *et al.*, 2009). In turgid epidermal strips, NO acts downstream to H_2O_2 in signaling during stomatal closure, as supported by multiple lines of evidence. Stomatal closure in response to NO and H_2O_2 is more efficiently induced in light than in the dark, and higher concentrations of both these molecules in guard cells were observed following the light to dark transition (She *et al.*, 2004; He *et al.*, 2005). Also, NO- and H_2O_2-scavengers prevent both light- and dark-induced stomatal opening and closure, respectively (She *et al.*, 2004; Garcia-Mata and Lamattina, 2007; Ribeiro *et al.*, 2009), with exogenous H_2O_2 inducing rapid NO synthesis in

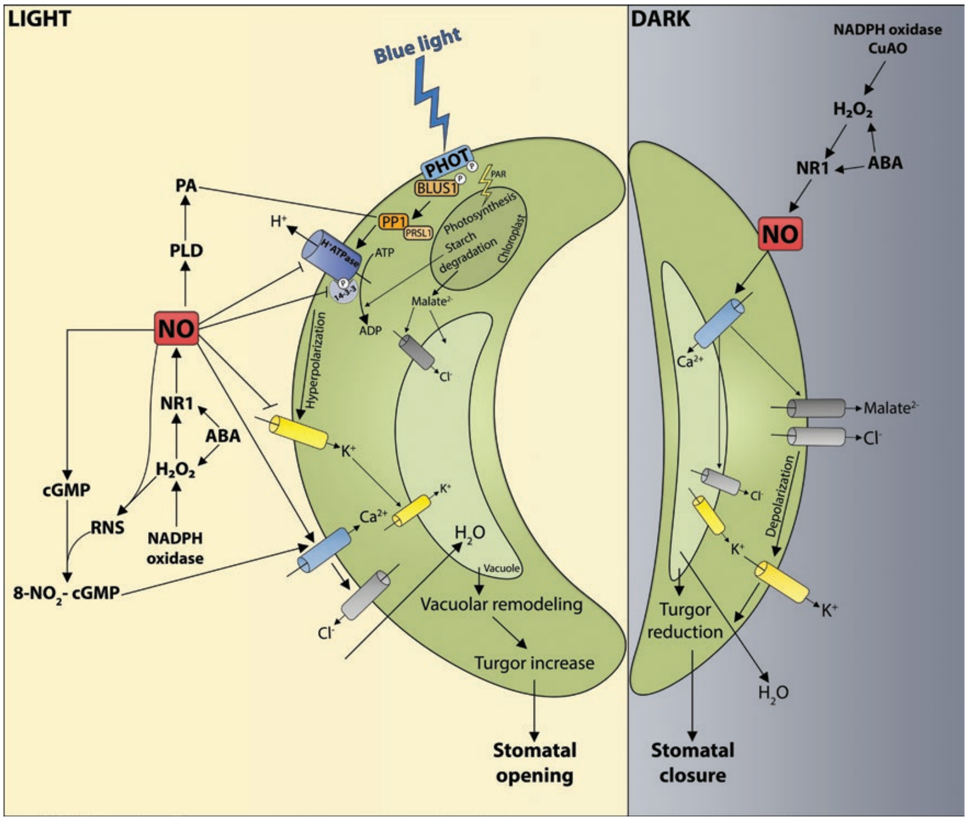

Fig. 3. NO action in light-regulated stomatal movement. In the presence of blue light, phototropins (PHOT) initiate a signaling cascade involving the protein kinase BLUE LIGHT SIGNALLING 1 (BLUS1), type 1 protein phosphatase (PP1) and its regulatory subunit (PRLS1). Guard cell photosynthesis provides ATP for H^+-ATPase, while the signal from BLUS1 activates plasma membrane H^+-ATPase by the phosphorylation and subsequent binding of a 14-3-3 protein, promoting H^+ pumping, which hyperpolarizes the plasma membrane and drives K^+ into guard cells. The accumulation of K^+ and counter-ions (Cl^- and malate^{2-}) drives water movement into the guard cells, increasing cell turgor and opening the stomatal pore. Preceded by hydrogen peroxide (H_2O_2) generation, nitrate reductase1 (NR1)-mediated nitric oxide (NO) synthesis promotes phospholipase D (PLD)-dependent phosphatidic acid (PA) production, which inhibits PP1 and represses H^+-ATPase. Abscisic acid (ABA) is known to promote both H_2O_2 and NR1-mediated NO generation in guard cells. NO can react with reactive oxygen species, such as H_2O_2, generating nitrogen reactive species (RNS), leading to the accumulation of 8-nitro-cGMP in guard cells, which in turn triggers stomatal closure in the light by favoring Ca^{2+} influx. In the dark, NAD(P)H oxidase- and copper amine oxidase (CuAO)-mediated H_2O_2 production triggers NR1 activation and NO production, leading to Ca^{2+} signaling-dependent events that culminate in stomatal closure.

guard cells (Lum *et al.*, 2002; Lü *et al.*, 2005; Bright *et al.*, 2006; Yan *et al.*, 2007; Wang *et al.*, 2010). Pharmacological and genetic data suggest NR, particularly NR1/NIA1, as the primary biosynthetic source of NO in guard cells during ABA-induced stomatal closure (Bright *et al.*, 2006), with H_2O_2 synthesis by NADPH oxidase isoforms AtrbohD/F preceding NO synthesis by NR1/NIA1 (Bright *et al.*, 2006; Fig. 3). In addition to AtrbohD/F, copper amine oxidase (CuAO) is also reported as the H_2O_2 source that precedes NO accumulation and cytosolic alkalinization during dark-induced stomatal closure (Huang *et al.*, 2015).

NO was also shown to inhibit BL-specific, but not RL-induced, stomatal opening via the repression of multiple BL-regulated processes, such as H^+-ATPase activity (Zhang *et al.*, 2007), PA production via PLD (Distéfano *et al.*, 2008; Takemiya and Shimazaki, 2010), and K^+ influx across the guard cell plasma membrane (Zhao *et al.*, 2012, 2013). During ABA inhibition of light-induced stomatal opening, there is cross-talk between NO and Ca^{2+} (García-Mata and Lamattina, 2007; Ribeiro *et al.*, 2009), possibly by the *S*-nitrosation of Ca^{2+}-dependent ion channels (Sokolovski and Blatt, 2004). NO also acts upstream to cyclic GMP (cGMP) in guard cells (Neill *et al.*, 2002), and reactive oxygen species (ROS) can react with NO to form reactive nitrogen species (RNS), which in turn lead to the formation of the nitrated cGMP derivative 8-nitro-cGMP. While cGMP induces stomatal opening in the dark, 8-nitro-cGMP triggers stomatal closure in the light by repressing Ca^{2+} channels (Joudoi *et al.*, 2013; Fig. 3).

A role for NO in UV-B-mediated stomatal closure is also proposed. UV-B induces NO production in the cytosol and chloroplasts of guard cells (He *et al.*, 2005), and both UV-B-triggered NO generation and stomatal closure are repressed by NR inhibitors and a NO scavenger (He *et al.*, 2011). GPA1, the Gα-subunit of heterotrimeric G proteins, is also reported to activate H_2O_2 production by AtrbohD/F followed by NR1/NIA1-dependent NO production during UV-B-mediated stomatal closure (He *et al.*, 2013). Moreover, ethylene production was shown to precede NO accumulation during UV-B-triggered stomatal closure (He *et al.*, 2011), whereas treatment with ethylene reduced NO amounts in guard cells and promoted stomatal opening under dark conditions (Song *et al.*, 2011).

Light and NO also interact to regulate stomatal development and patterning. Supporting this claim, Fu *et al.* (2016) revealed that NO treatment, as well as *nox1* and *noa1* mutations, affect stomatal development by affecting the expression of genes encoding SPEECHLESS (SPCH), MUTE and FAMA, which are TFs responsible for initiating stomatal development that also are responsive to the PHY-CRY-COP1 signaling system (Casson *et al.*, 2009; Kang *et al.*, 2009).

Chloroplasts and photosynthesis: multiple target sites of nitric oxide action in the solar powerhouse of green plants

Mature chloroplasts are the solar powerhouses of green plants, and also a focal point of ROS and NO production in illuminated plants. The effects of NO on the plant photosynthetic system have been extensively examined, leading to the identification of a large number of target sites of NO action in chloroplasts (reviewed by Misra *et al.*, 2014).

In photosystem II (PSII), NO can reversibly bind to the non-heme iron localized between Q_A and Q_B ($Q_AFe^{2+}Q_B$) and cause a ten-fold decrease in electron transfer between Q_A and Q_B (Diner and Petrouleas, 1990; Petrouleas and Diner, 1990; Fig. 4). *In vivo* confirmation that Q_A- Q_B electron transfer rate is reduced by NO donors was obtained, being linked to inhibited charge recombination reactions of Q_A^- with the S_2 state of the oxygen-evolving complex (OEC) and decreased maximum quantum efficiency of PSII (Wodala *et al.*, 2010).

A second target site of NO action in PSII is the catalytic manganese cluster of the OEC (Schansker *et al.*, 2002; Fig. 4). In the presence of NO, the oxygen oscillation patterns of PSII-enriched membranes changed due to the NO-related reduction of the Mn cluster to the S_2 state (Schansker *et al.*, 2002; Sarrou *et al.*, 2003). As a consequence, NO inhibits primary oxygen-evolving reactions, as demonstrated *in vitro* in isolated thylakoids (Vladkova *et al.*, 2011) and intact chloroplasts (Jasid *et al.*, 2006). NO may also affect the donor side of PSII due to the interaction of NO with the second redox active tyrosine residue (Y_D) of D2 protein. The rapidly formed Y_D–NO complex has lower redox potential than the parent Tyr and can act as an electron donor in PSII instead of Tyr Y_Z and the water-splitting Mn complex (Sanakis *et al.*, 1997).

As for photosystem I (PSI), P700 chlorophyll fluorescence measurements in intact pea leaves revealed that GSNO promoted PSI quantum efficiency and modestly increased the pool size of electrons in the intersystem chain, indicating that NO may influence PSI photochemistry *in vivo* (Wodala and Horváth, 2008). Furthermore, Twigg *et al.* (2009) demonstrated that NO binds to reduced heme c_n in the cytochrome b_6f complex (Fig. 4), though the consequential effect of this NO binding has not been revealed so far. It is known, however, that NO_2^--dependent NO production is implicated in cytochrome b_6f degradation in nitrogen- or sulfur-starved *C. reinhardtii* (Wei *et al.*, 2014; de Mia *et al.*, 2019).

Treatment of isolated thylakoid membranes with NO donors revealed that NO strongly inhibits photosynthetic ATP synthesis, and that the inhibition can be reversed by the addition of bicarbonate (Takahashi and Yamasaki, 2002; Fig. 4). Electron transport rate, light-triggered ΔpH formation, and ATP hydrolysis were also diminished by NO. In guard cell protoplasts, exogenous NO reversibly inhibited the linear electron transport chain, reducing the amount of ATP and NADPH available for osmoregulation (Ördög *et al.*, 2013). Additionally, the catalytic component (CF1) of ATP synthase was found to be *S*-nitrosated after treatment

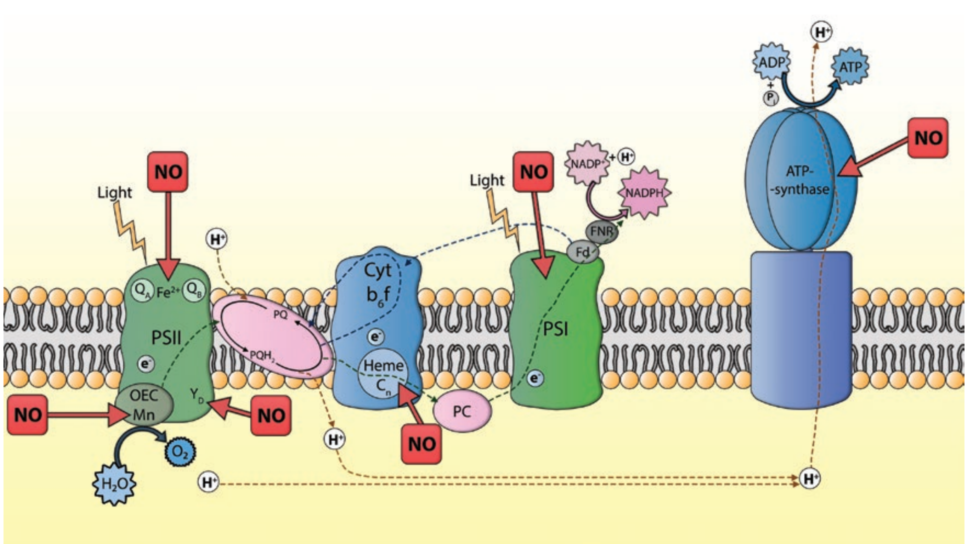

Fig. 4. Target sites of NO in the photosynthetic electron transport chain. NO inhibits the oxygen-evolving complex (OEC) by reducing Mn clusters, while NO affects the activity of photosystem II (PSII) through direct binding to non-heme iron (Fe^{2+}) between plastoquinones QA and QB. NO also binds to both the second redox active tyrosine residue (Y_D) of D2 protein and the reduced heme c_n in the cytochrome b_6f complex (cytb_6f). Moreover, NO influences photosystem I (PSI) photochemistry and strongly inhibits photosynthetic ATP synthesis, possibly due to *S*-nitrosation of the catalytic component (CF1) of ATP synthase. PQH_2, reduced, mobile plastoquinone pool; PC, plastocyanin; Fd, ferredoxin; FNR, ferredoxin-NADP$^+$ oxidoreductase.

with NO gas or GSNO (Lindermayr *et al.*, 2005); however, the consequent alteration in ATP synthase activity has not been revealed.

NO also affects numerous enzymes involved in CO_2 assimilation, including the most abundant key enzyme in the Calvin cycle, ribulose-1,5-bisphosphate carboxylase/oxygenase (RuBisCO). Lindermayr *et al.* (2005) first analysed *S*-nitrosation in a photosynthetically active tissue and identified several chloroplast proteins as targets for *S*-nitrosation, including RuBisCO and RuBisCO activase. Subsequently, *S*-nitrosation-triggered inhibition of RuBisCO was demonstrated both *in vivo* and *in vitro* (Abat *et al.*, 2008), with both subunits of the enzyme undergoing *S*-nitrosation in response to low temperature (Abat and Deswal, 2009), and six Cys-SNO sites recently identified (Qiu *et al.*, 2019). Other photosynthesis-related proteins identified as targets for *S*-nitrosation are involved in light-dependent reactions (e.g. PsbP1 or ATPA), in all three phases of the Calvin cycle (e.g. phosphoglycerate kinase), components of carbon concentration mechanisms (e.g. phosphoenolpyruvate carboxylase, carbonic anhydrase) and glycolytic enzymes (e.g. aldolase, triosephosphate), amongst others (Lindermayr *et al.*, 2005; Abat *et al.*, 2008; Abat and Deswal, 2009; Fares *et al.*, 2011; Tanou *et al.*, 2012; Kato *et al.*, 2013; Vanzo *et al.*, 2014; Hu *et al.*, 2015; Kolbert *et al.*, 2019b). RuBisCO activase and both subunits of RuBisCO enzyme are also subjected to *in vivo* nitration at specific Tyr residues, as well as several other chloroplast-localized proteins, including the PSII protein D1 (Galetskiy *et al.*, 2011; Lozano-Juste *et al.*, 2011; Ramos-Artuso *et al.*, 2019). Therefore, based on the proteomic data available so far, it appears that the activity of numerous photosynthetic proteins (e.g. RuBisCO activase, RuBisCO) is under dual regulation by *S*-nitrosation and Tyr nitration, implicating that NO tightly controls photosynthetic activity at the post-translational level.

Multiple high-throughput analysis revealed that NO also modulates photosynthesis at the transcriptional level, as revealed by the significant proportion of photosynthesis- and chloroplast-related functional categories within the NO-responsive genes (Polverari *et al.*, 2003; Parani *et al.*, 2004; Begara-Morales *et al.*, 2014; Hussain *et al.*, 2016; León *et al.*, 2016). Furthermore, NO treatment influences the abundance of intermediates of photorespiration (glycerate) and Calvin cycles (sedoheptulose-7-phosphate and ribose-5-phosphate), as well as downstream products of photosynthesis (León *et al.*, 2016).

As chloroplasts are hotspots of NO production and action, and this molecule regulates multiple aspects of the photosynthetic machinery, intensive research has been devoted to evaluating the practical implications of adjusting NO concentrations as a strategy to ameliorate the photosynthetic performance of plants under stress conditions (reviewed by Misra *et al.*, 2014).

Multifunctional role of nitric oxide in plant light stress responses

Nitric oxide as a protective molecule against light stress-induced disturbances in redox homeostasis

Throughout their life cycle, plants can face both seasonal and sporadic deviations from optimal light conditions, including excessive or insufficient light intensity. Either irradiances below the light-compensation point or far above the light saturation point of photosynthesis, collectively known as light stress, can lead to oxidative stress, photoinhibition, and limited plant growth and development (Krause *et al.*, 2012; Zhang *et al.*, 2018). Enrichment in UV radiation, particularly UV-B, can also be a source of light stress for plants (Mackerness, 2000). Whereas low-fluence UV-B contributes to plant photomorphogenesis (Wu *et al.*, 2016), high levels of this radiation can cause DNA damage, photooxidation of pigments, inhibition of photosynthetic activity, and reduction of biomass accumulation (Greenberg *et al.*, 1997; An *et al.*, 2005).

Chloroplasts and the photosynthetic apparatus are particularly sensitive to excess visible light and UV-B radiation (Powles, 1984; Aro *et al.*, 1993). The oxygen produced by PSII during photosynthesis can potentially increase ROS generation, especially under excessive light (Aro *et al.*, 1993; Mackerness *et al.*, 2001). Therefore, disturbances in redox homeostasis are arguably one of the most frequent metabolic consequences of light stress (Fig. 5). Light stress-induced production of ROS (e.g. singlet oxygen, superoxide anion, H_2O_2 and hydroxyl radicals) may lead to lipid peroxidation and damage to the cell membranes, consequently inhibiting photosynthesis, respiration and plant growth (Asada, 2006; Xu *et al.*, 2013). As one of the first lines of plant defense against oxidative stress, non-enzymatic antioxidants (e.g. ascorbate and glutathione) and antioxidant enzymes (e.g. catalase, ascorbate peroxidase and superoxide dismutase) are frequently up-regulated by plant cells to avoid or minimize light stress-induced cellular damage (Jansen *et al.*, 1998; Kim *et al.*, 2010).

High amounts of visible light or UV-B modulate NO production in plant cells (Wang *et al.*, 2006; Corpas *et al.*, 2008; Choudhury *et al.*, 2018), which in turn activates plant antioxidant defenses under these circumstances (Xu *et al.*, 2013; Simontacchi *et al.*, 2015). For example, the transfer of Arabidopsis plants from low light conditions (50 μmol m^{-2} s^{-1}) to excessive light (1000 μmol m^{-2} s^{-1}) increased endogenous NO concentration within minutes; a response also coupled with the accumulation of glutathione (Choudhury *et al.*, 2018). Short-term high light stress (above 1000 μmol m^{-2} s^{-1} for 4 h) stimulated NOS-like activity and RSNO production in pea plants, whereas GSNOR activity remained unaltered (Corpas *et al.*, 2008). When two varieties of tall fescue grass (*Festuca arundinacea*) with contrasting tolerance to light stress were treated with ABA followed by high light exposure, a significant increase in NO release and NOS-like activity, linked to the activation of antioxidant defenses, was observed in the high light-tolerant variety (Xu *et al.*, 2013). Similarly, UV-B stress was demonstrated to promote NO and ROS accumulation in maize seedlings, with pharmacological treatments indicating that both ROS and NO mediate UV-B-induced ethylene biosynthesis (Wang *et al.*, 2006). Data from the literature support either NR (Wang *et al.*, 2006; Zhang *et al.*, 2011a) or NOS-like activity (Xu *et al.*, 2013) as the source of NO production during light stress responses, depending on the species. In the green algae *C. reinhardtii*, very high light intensity (3000 μmol m^{-2} s^{-1}) triggered non-enzymatic NO production, which in turn repressed carotenoid synthesis, consequently leading to

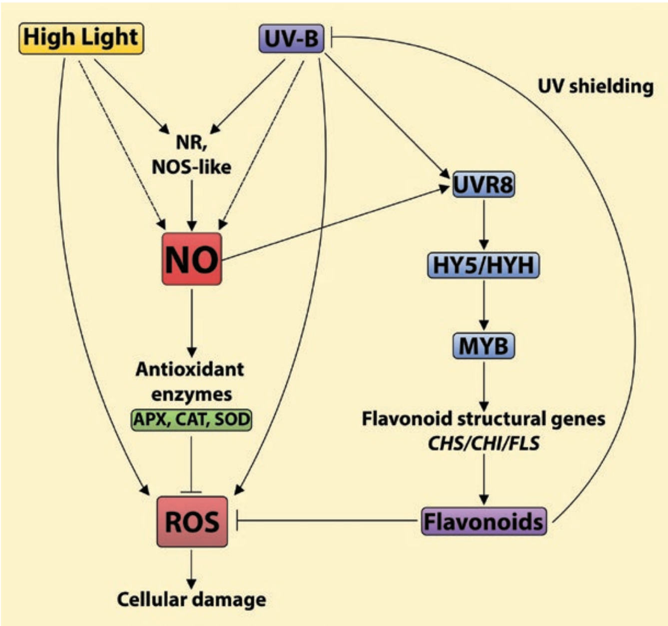

Fig. 5. Protective roles of nitric oxide in light stress responses. High light and UV-B promote NO generation via both nitrate reductase (NR) and NO synthase-like (NOS-like) activity and also trigger the accumulation of reactive oxygen species (ROS). NO promotes the expression and activity of antioxidant enzymes such as ascorbate peroxidase (APX), catalase (CAT) and superoxide dismutase (SOD). UV-B-triggered activation of the photoreceptor UVR8 up-regulates genes encoding transcription factors, such as ELONGATED HYPOCOTYL5 (*HY5*), HY5-homolog (*HYH)* and *MYB*, which in turn promote the transcription of flavonoid structural genes. NO-mediated accumulation of *UVR8* transcripts intensifies the synthesis of flavonoids, which in turn alleviates oxidative stress and minimizes UV absorption by the plant tissues. Dashed lines indicate potential pathways. CHI, chalcone isomerase; CHS, chalcone synthase; FLS, flavanol synthase.

singlet oxygen ($^1O_2$) over accumulation, lipid peroxidation, enhanced expression of oxidative stress-related genes and irreversible PSII inactivation (Chang *et al.*, 2013). On the other hand, less extreme high-light conditions (1600 µmol m^{-2} s^{-1}) induced a burst in both NR and NOS-dependent NO generation in *C. reinhardtii*, which was associated with autophagy activation, probably via an interplay with H_2O_2 (Kuo *et al.*, 2020). In Arabidopsis, both NO and H_2O_2 interact during the induction of cell death (Murgia *et al.*, 2004), and $^1O_2$ overproduction is associated with high light-induced cell death (Shumbe *et al.*, 2016), suggesting a connection between ROS and NO in light stress-triggered cell death, although this has yet to be demonstrated.

Exogenous NO, applied as sodium nitroprusside (SNP), also promotes antioxidant defenses and ameliorates oxidative stress caused by excessive light (Xu *et al.*, 2010) or UV-B exposure (Santa-Cruz *et al.*, 2014; Hu *et al.*, 2016). The ameliorative action of NO on chloroplast function under UV-B stress was confirmed by SNP-induced reduction of thylakoid membrane protein oxidation, prevention of chlorophyll loss and limited accumulation of H_2O_2 , as well as the restorative effect on PSII activity in UV-B treated common bean leaves (Shi *et al.*, 2005). Moreover, UV-B-triggered increase in the activity of antioxidant enzymes was further intensified upon SNP treatment (Shi *et al.*, 2005). In soybean, NO production also mediates UV-B-triggered induction of heme oxygenase, an enzyme associated with antioxidant defenses (Santa-Cruz *et al.*, 2010). As in land plants, NO treatment induces antioxidant defenses and alleviates UV-B-induced chlorophyll degradation and damage to the photosynthetic apparatus in green algae (Chen *et al.*, 2010) and cyanobacteria (Xue *et al.*, 2007). NO also promotes enzymatic antioxidant defenses under low light conditions (Fu *et al.*, 2014; Zhang *et al.*, 2018; Hu *et al.*, 2019). For example, NO production was suggested as being necessary to promote the ascorbate-glutathione (AsA-GSH) cycle in *Brassica pekinensis* seedlings exposed to moderately low light stress (100 µmol m^{-2} s^{-1}) in the presence of a nitrate-containing hydroponic solution (Hu *et al.*, 2019). Although catalase, superoxide dismutase and other central players in the AsA-GSH cycle are regulated by *S*-nitrosation and/or Tyr nitration (Begara-Morales *et al.*, 2016), the relevance of these NO-dependent post-translational modifications for the induction of antioxidant responses under light stress remains to be investigated.

In a contrasting situation, ROS can promote NO accumulation during light stress (Lin *et al.*, 2012). Working with the catalase-deficient rice mutant *nitric oxide excess1* (*noe1*), Lin *et al.* (2012) demonstrated that the distinctive over accumulation of H_2O_2 in leaves of this genotype was responsible for promoting NR-dependent NO production upon high light treatment. In this same study, *GSNOR* overexpression in *noe1* plants failed to reduce leaf H_2O_2 concentrations, suggesting that NO acts downstream of H_2O_2 during light stress-induced programmed cell death in rice leaves (Lin *et al.*, 2012). In agreement, H_2O_2 was also characterized as an upstream signal in UV-B-induced NO production in hypocotyls of radish sprouts (Wu *et al.*, 2016). Under some circumstances, however, no correlation between antioxidant metabolism and NO protective action against excessive light has been observed, as seen in neotropical tree seedlings treated with NO-releasing chitosan nanoparticles under full sun (Lopes-Oliveira *et al.*, 2019).

Screening out UV radiation: nitric oxide and flavonoid biosynthesis

As an additional line of defense against UV radiation damage, plants have evolved mechanisms for screening out UV radiation through the accumulation of UV-absorbing phenolic compounds, particularly flavonoids such as flavonols, anthocyanins and chalcones (Fig. 5). UV-B, perceived by UVR8, is known to control multiple TFs (e.g. HY5, HYH, MYB) responsible for regulating the transcription of key components of the phenylpropanoid biosynthetic pathway in plant cells (Kliebenstein *et al.*, 2002; Heijde *et al.*, 2013; Huang *et al.*, 2014; Wu *et al.*, 2016). In agreement, constitutively active UVR8 variants and *UVR8*-deficient mutants are characterized by increased and reduced anthocyanins levels, respectively (Kliebenstein *et al.*, 2002; Heijde *et al.*, 2013; Huang *et al.*, 2014; Wu *et al.*, 2016).

Both H_2O_2 and NO interplay with the UVR8 signaling pathway to regulate flavonoid accumulation (Fig. 5). Early evidence in Arabidopsis, based on enzyme inhibitors and free radical scavengers, indicated that UV-B-triggered up-regulation of *CHALCONE SYNTHASE* (*CHS*), which encodes a key enzyme in the phenylpropanoid pathway, was not affected by ROS scavengers, but was reduced by NOS inhibitors or NO scavengers (Mackerness *et al.*, 2001). In addition, UV-B was shown to promote H_2O_2 and anthocyanin accumulation, whereas treatment with SNP, H_2O_2, and their combination promoted the transcript abundance of both *UVR8* and structural genes responsible for anthocyanin biosynthesis (Wu *et al.*, 2016; Fig. 5). More recently, studies performed in the anthocyanin-over accumulating *Anthocyanin fruit* (*Aft*) tomato accession revealed that both NR transcript and activity are promoted by co-irradiation with blue light and UV-B, and pharmacological evidence supported a role for NR-mediated NO generation in the control of anthocyanin biosynthesis in tomato fruit skin (Kim *et al.*, 2020). As flavonoids have both the capacity to shield the tissue by UV absorption and also scavenge excessive ROS production (Harborne and Williams, 2000), their accumulation in the cells offers a dual benefit to plants facing excessive white light or UV irradiance. Moreover, in line with the well-described role of flavonols as inhibitors of auxin transport and root development (Silva-Navas *et al.*, 2016), the over accumulation of flavonoids in NO-deficient Arabidopsis mutants has been linked to the reduced root growth phenotype found in light-grown seedlings of these genotypes (Sanz *et al.*, 2014). In addition, UV-B radiation has been reported to cause dose-dependent inhibition of root growth in soybean seedlings by modulating the production of NO, ROS and multiple plant hormones (Zhang *et al.*, 2019).

The extended landscape of nitric oxide and light interaction in plant stress responses

Light and NO can also co-regulate plant responses to other abiotic stresses (Lee *et al.*, 2008; Liu and Guo, 2013; Kumari *et al.*, 2019). In sunflower seedling cotyledons, the biosynthesis of the osmolyte glycine betaine (GB) was differentially modulated by NO under light and dark conditions, with light restricting its NO-induced accumulation (Kumari *et al.*, 2019). In *gsnor1* missense and null Arabidopsis mutants, unusual thermotolerance has been observed depending on the light conditions (Lee *et al.*, 2008). Whereas *gsnor1* null mutants were not able to heat-acclimate, *gsnor1* missense mutants exhibited typical heat-acclimation responses when grown under light but not in the dark (Lee *et al.*, 2008).

NO is also known to closely interact with ethylene to regulate flooding-induced plant responses, including aerenchyma formation (Wany *et al.*, 2017) and acclimation to hypoxia (Hartman *et al.*, 2019). Since light conditions vary greatly depending on floodwater depth and clarity, light availability may also play a relevant role in controlling NO biosynthesis and removal during natural flooding conditions (Sasidharan *et al.*, 2018). In addition, NO is also known to inhibit chlorophyll catabolism and promote the stability of photosynthetic complexes in thylakoid membranes during dark-induced senescence in Arabidopsis (Liu and Guo, 2013).

Conclusions and future perspectives

Accumulating evidence indicates that light stimuli exert a positive influence on NO production, very frequently via increments in NR transcription and enzyme activity. Moreover, NO has been shown to interact with central components of signaling cascades initiated by photoreceptors, including signaling hub proteins (e.g. PIFs, HFR1, HY5), as well as plant hormones (e.g. ABA, GA, ethylene, auxins), during light-dependent plant responses.

Some cutting-edge insights into NO–light signaling crosstalk have recently been achieved during seed germination and de-etiolation, including the identification of a NO sensing mechanism (NO-mediated ERFVIIs degradation; Gibbs *et al.*, 2014), NO-interacting partners (e.g. PIFs, HFR1, DELLA; Lozano-Juste and León, 2011) and downstream responses to NO action (e.g. regulation of starch metabolism, cell wall loosening). However, a multitude of other signaling steps leading to light-induced seed germination and de-etiolation remains to be investigated as possible targets of NO action. Additional research efforts are also required to identify photoreceptors and light signaling proteins susceptible to NO-mediated PTMs under physiologically relevant conditions.

Although the initiation of seed germination in response to adequate environmental conditions and the acquisition of photoautotrophic capacity in emerging seedlings are life-or-death issues for plants, they usually occupy a brief moment in the plant photomorphogenic life. Therefore, a comprehensive picture of NO action in plant photomorphogenesis requires an intensification of research efforts in other light-modulated developmental responses. Photoperiod flowering, shade-avoidance responses, fruit development and leaf senescence are some examples of light-modulated developmental responses that are gaining momentum in photo-biotechnology endeavors to promote crop productivity (Ganesan *et al.*, 2016). However, very limited, or non-existent, information is available about the involvement of NO in these processes. Also, as many plant photomorphogenic responses are regulated by the inter- and intra-class interaction of photoreceptors, research on the interplay between NO and light signaling should consider this additional level of complexity.

NO production and signaling are also of great biotechnological relevance in the context of carbon gain, not only due to the central role of NO in the complex signal transduction pathways responsible for light-dependent stomatal movements, but also because the target sites of NO action in chloroplasts are multiple and diversified. However, a closer look at the available literature reveals that very little is known about the *in vivo* regulatory role of NO on chloroplast function, despite this organelle is a hotspot of NO production. Moreover, determining whether NO represents a unifying signal to control stomatal movements in response to light, drought, and other environmental factors is another critical question open for future investigation. Additional research is also needed to dissect how photoreceptors and light signaling proteins are linked to NO production and removal systems in guard cells.

As in other abiotic stresses, NO promotes plant antioxidant defenses under unfavorable light conditions. Under intense

UV-B radiation, NO promotes the synthesis of UV-absorbing phenolic compounds, which fulfill the dual role of screening out UV radiation and acting as non-enzymatic antioxidants. Despite the importance of light stress for both crop and non-crop plants, the mechanisms behind NO interplay with other signaling elements controlling the induction of enzymes involved in antioxidant defenses and phenolic compound synthesis remain poorly characterized.

The emergence of more precise and robust gene modification tools applicable to both model and crop species, combined with the wealth of information derived from several decades of investigation in plant photobiology, suggest a bright future for research on the interaction between NO and light signaling from both scientific/academic and agronomical/economic points of view.

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