

Hydrogen Peroxide and Nitric Oxide Metabolism in Chloroplasts



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Abstract Chloroplasts are ubiquitous organelles with a notable oxidative metabolism that are involved in free energy transduction through photophosphorylation. In addition, thioredoxin-mediated redox regulation of Calvin cycle enzymes has been shown to determine the efficiency of carbon assimilation. These provide significant evidence that chloroplasts are key cellular sites of reactive oxygen species (ROS) production, including molecules like superoxide ($O_2^{\bullet-}$) and H_2O_2 . In addition to ROS, there are compelling indications that nitric oxide (NO) can be generated in chloroplasts by both reductive and oxidative pathways. NO is involved in many physiological and biochemical processes in plants, including photosynthesis. However, many unanswered questions remain concerning how, when, and where NO is generated (enzymatically or nonenzymatically) in higher plants. ROS and reactive

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nitrogen species (RNS) play a key role in signaling, but they can also be deleterious via oxidation of cell components when overproduced, as a consequence of redox imbalance and/or adverse conditions. In chloroplasts, besides the redox state and regulation of thiol groups of cysteinyl residues in proteins, identification of other types of posttranslational protein modifications (PTMs) such as Tyr nitration, *S*-nitrosylation, and glutathionylation has been reported. Therefore, this chapter is focused on photosynthetic light/dark reactions, antioxidant defense and thiol-based redox regulation, ROS production, NO synthesis, and its implication together with that of ROS, in PTM-mediated regulation, with the aim of drawing a picture of the importance of all of them in the regulation of the chloroplast function.

Keywords Plant stress · Chloroplast · Hydrogen peroxide · Nitric oxide · Posttranslational modification · Reactive nitrogen species · Reactive oxygen species

1 Introduction

In plant cells, generation of ROS (reactive oxygen species) and RNS (reactive nitrogen species) is characteristic of a number of metabolic reactions that take place in different cell compartments. The best known ROS—superoxide anion ($O_2^{\bullet -}$), hydrogen peroxide (H_2O_2), single oxygen (1O_2), and hydroxyl radical ($^{\bullet}OH$)—are all strong oxidizing compounds and therefore potentially harmful for cell integrity by causing loss of redox homeostasis. Among them, H_2O_2 is thermostable and can be produced in all compartments by the dismutation of $O_2^{\bullet -}$, with the electron transport in mitochondria, but mainly that in chloroplast during photosynthesis and in peroxisomes during photorespiration, being the most important ROS sources (Foyer and Shigeoka 2011). Besides their toxicity, ROS are also considered to be powerful signaling molecules that regulate plant growth and development as well as biotic and abiotic stress responses (Mittler et al. 2004; Baxter et al. 2014; del Río 2015).

H_2O_2 in chloroplasts, even at low levels, is a potent inhibitor of photosynthesis due to its capacity to oxidize the thiol-modulated enzymes of the Calvin cycle. Therefore, chloroplasts are equipped with a robust ROS detoxification system including nonenzymatic and enzymatic components, able to maintain a strict control of ROS content produced during the normal metabolism, and increased under unfavorable stress conditions. The ascorbate-glutathione (ASC-GSH) cycle and the water-water cycle are in charge of metabolizing H_2O_2 and to dissipate excess of excitation energy in chloroplasts (Foyer and Shigeoka 2011), whereas superoxide dismutases (SODs) metabolize $O_2^{\bullet -}$. Other enzymes involved in H_2O_2 and hydroperoxides detoxification are glutathione peroxidase (GPX) and peroxiredoxin (Prx), which function together with sulfiredoxin (Srx) and thioredoxin (Trx) in chloroplasts ROS detoxification and redox homeostasis (Bernier-Villamor et al. 2004; Iglesias-Baena et al. 2010, 2011; Mock and Dietz 2016).

Thioredoxins (Trxs) are an important class of regulatory small proteins presenting two cysteines in their active site that modulate the activity of target thiol proteins by

reducing specific disulfide bounds (Montrichard et al. 2009). In chloroplasts, in the photosynthetic electron transport (PET), reducing equivalents are transferred to specific proteins containing redox-active cysteine residues mediated by the thioredoxin system (Yoshida et al. 2015). This Trx system, composed of ferredoxin (Fd)-Trx reductase (FTR) and Trx, ensures light-responsive control of multiple key functions (Crawford et al. 1989; Barajas-López et al. 2012) as well as the thiol-based H_2O_2 reduction through the activity of Prxs. Thus, these proteins participate in the cell signaling network, where redox signals, comprising ROS and redox balance sensors, are perceived and transmitted (Sevilla et al. 2015a; Calderón et al. 2018).

Nitric oxide (NO) is a gaseous small reactive molecule which exists in three different forms, i.e., NO^+ (nitrosonium cation), NO^- (nitroxyl anion), and $\cdot NO$ (nitric oxide radical). As a free radical, NO and its derived molecules are designated as reactive nitrogen species (RNS) including peroxyntirite ($ONOO^-$), *S*-nitrosothiols (SNOs), and *S*-nitrosoglutathione (GSNO) (Yu et al. 2014). There is evidence of the NO production in plants due to a variety of sources, including an L-arginine-dependent nitric oxide synthase (NOS)-like enzymes (Corpas et al. 2017), nitrite and nitrate reductase (NR), or nonenzymatic sources, occurring in different cell compartments (see below). Additionally, an excess of NO can act synergistically with ROS and result in nitro-oxidative stress eliciting undesirable damaging effects in plant cells. In chloroplasts, photosynthesis is affected not only by ROS but also by reaction with NO. Thus under conditions of high NO content in chloroplast, the generation of RNS may provoke an impairment of the photosynthetic machinery (Jasid et al. 2006). On the other hand, as an endogenous metabolite, NO has various signaling functions. NO and RNS can interact at different levels, as part of a complex network of signaling processes as well as a response mechanism against environmental stress conditions (Turkan 2017; Umbreen et al. 2018). Interaction of RNS with ROS occurs during plant growth and development in processes such as seed germination and flowering (Zafra et al. 2016) and in defense (Asai et al. 2008; Romero-Puertas et al. 2008; Lin et al. 2012) and abiotic stress responses (Wang et al. 2006; del Río 2015). Prxs are important proteins at the crossroads of the ROS and NO signaling pathways, because they can reduce the H_2O_2 , peroxyntirite, and organic peroxides generated in the cell, through their catalytic cysteine residues by using the thiol-containing Trx system (Mock and Dietz 2016). Moreover, transduction and specificity of ROS and RNS bioactivity under physiological processes as well as under stress conditions involve posttranslational modification of proteins, such as sulfenylation, *S*-glutathionylation, nitration, and *S*-nitrosylation (Mittler et al. 2004; Corpas et al. 2009; Lozano-Juste et al. 2011; Ortega-Galisteo et al. 2012; Lázaro et al. 2013; Camejo et al. 2015).

In this chapter, we will focus on the role of antioxidant and redox-modulated enzymes in chloroplasts and the effects played by ROS, mainly H_2O_2 and NO on the photosynthesis functionality through specific targets. The sources of NO in plants cells together with some examples of posttranslational modification driven by ROS and RNS in chloroplasts proteins are also presented.

2 ROS Metabolism

Oxygen (O_2) represents approximately 21% of the volume of the composition of the terrestrial atmosphere. The reduction of the oxygen to water provides the energy that allows the impressive complexity of the superior organisms, but when its reduction is not complete, the generated species are called ROS.

2.1 ROS Generation in Plants

These reactive species are generated as products of the aerobic metabolism in the different cellular compartments as a by-product of a wide variety of processes, among them those which imply transport of electrons such as respiration in mitochondria and photosynthesis in chloroplasts (Asada 2006; Sevilla et al. 2015b) or during photorespiration and oxidation of fatty acids in peroxisomes and glyoxysomes (del Río and López-Huertas 2016). Also certain enzymatic activities generate ROS, such as NADPH-oxidoreductases and peroxidases of the plasmatic membrane and cell wall, respectively (Mittler et al. 2004) (Table 1).

A common feature of ROS is the generation of oxidative damage to proteins inactivating enzymes by oxidizing thiol groups or producing redox changes in the metals of the active centers. They also cause breaks and mutations in the DNA oxidizing the aromatic compounds of this molecule, as well as altering the functionality of the lipids in the membranes forming hydroperoxides.

Light energy distribution in the photosynthetic process regulates ROS production in leaf tissues (Tikkanen et al. 2014). In chloroplasts, ROS are generated within the electron transport chains (ETC) of PSII and PSI, and this production is increased as consequence of stressful conditions when CO_2 is limited and ATP synthesis is impaired (Nishiyama and Murata 2014; Noctor et al. 2014). The oxygen-evolving complex (OEC) in PSII liberates O_2 from H_2O , and electrons are then transferred to PSI via ETC from plastocyanin (PC) to ferredoxin (Fd) (Tikkanen and Aro 2014). FNR can then reduce $NADP^+$ to NADPH from the reduced Fd (Fig. 1) (Rochaix

Table 1 ROS generation in different plant cell compartments

Localization	Process	ROS
Chloroplasts	Photosynthesis PSI, PSII	$O_2^{\bullet-}$
	Excited chlorophyll	1O_2
Mitochondria	Respiration CI and CIII	$O_2^{\bullet-}$
Peroxisomes	Xantine oxidase	$O_2^{\bullet-}$
	Glycolate oxidase	H_2O_2
	Fatty acids β -oxidation	H_2O_2
Plasma membrane	NADPH oxidase	$O_2^{\bullet-}$
Cell wall	Peroxidases, Mn^{2+} and NADH	$H_2O_2/O_2^{\bullet-}$
Apoplast	Oxalate and amine oxidase	H_2O_2

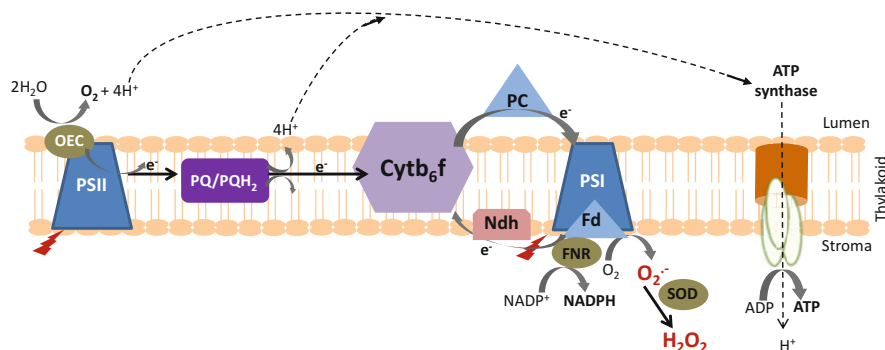


Fig. 1 Generation of ROS in the photosynthetic electron transport chain. Linear electron flow (continuous arrows) is catalyzed by PSII from water oxidation through PQ pool to Cytb₆f and plastocyanin to PSI and ferredoxin. Ultimately, FNR reduces NADP⁺ to NADPH. Electrons can be derived to O₂ to generate O₂^{•-} which dismutates to H₂O₂ by SOD. Cyclic electron flow occurs through PSI and Cytb₆f using Fd, NADPH, and Ndh complex or directly from Fd to Cytc in the Cytb₆ complex. Cytb₆f cytochrome b₆ complex, Fd ferredoxin, FNR Fd reductase, Ndh NADH dehydrogenase, OEC oxygen-evolving complex, PSI photosystem I, PSII photosystem II, PC plastocyanin, PQ plastoquinone, SOD superoxide dismutase (modified from Rochaix 2013)

2013). The cyclic electron flow (CEF) occurs through PSI and Cytb₆f mainly in the stroma by two pathways, one involving Fd, NADPH, and the NADH dehydrogenase (Ndh) complex and the other involving Fd and Cytc in the Cytb₆f complex. Electron transport coupled to proton translocation generates a pH gradient that drives ATP synthase to produce ATP (Rochaix 2014). These reactions produce ROS, which may cause oxidative damage and disruption of OEC, mainly under continuous exposure to high light (HL), so provoking photoinhibition (Murata et al. 2007; Gururani et al. 2015). Additionally, the pseudo-CEF comprises the so-called Mehler reaction, which is very important in the dark-light induction phase or during CO₂-limited photosynthesis (Asada 2006; Michelet and Krieger-Liszky 2012). This reaction consists of the direct reduction of dioxygen at the acceptor side of PSI with consequent production of O₂^{•-}. This ROS can be generated under certain stress conditions, such as high light intensity and a low concentration of CO₂, diminishing NADP availability in the chloroplast and thus increasing the electronic flow to O₂^{•-}. In turn, the O₂^{•-} can protonate to HO₂[•] in the internal surface of thylakoid membrane (lumen) or dismutate to H₂O₂ spontaneously or by the Fe and Cu,Zn superoxide dismutase enzymes (SODs), in the external surface of the membrane of the thylakoid or in the stroma (Gómez et al. 2004; Asada 2006; Pospíšil 2012) (Fig. 2). Also, chloroplastic NADPH oxidase-like activity has been shown to mediate H₂O₂ generation in *Brassica napus* (Tewari et al. 2012). This H₂O₂ is mainly detoxified by the activity of ascorbate peroxidase (APX) present in thylakoids and stroma (Gómez et al. 2004) in collaboration with the other components of the so-called ascorbate-glutathione (ASC-GSH) cycle, first described in chloroplasts, and/or by the activity of the different peroxiredoxins (Prxs) (Dietz 2016) in accordance with thioredoxins

(Trxs) and sulfiredoxins (Srxs). All these systems collaborate in the so-called water-water cycle, which, in fact, supposes the reduction of $O_2^{\cdot-}$ into H_2O . It has two quite important main functions under redox-stress conditions: photoprotection and ATP/NADPH ratio balancing (Asada 2006; Eberhard et al. 2008; Foyer and Shigeoka 2011). Another interesting photo-protective strategy involves a class of flavodiiron proteins (FLVs), located in the proximity of PSII and/or PSI. These proteins present several domains, including a flavodoxin-like and an NAD(P)H-flavin reductase-like domain. They are present in *Chlamydomonas*, bryophyte, and gymnosperms although they have been reported in Archaea and anaerobic bacteria and are able to reduce O_2 to water using NADPH as electron donor. They are believed to protect PSI from light stress, under changing light conditions (Gerotto et al. 2016), while another form of the protein FLV2/FLV4 seems to be active in photoprotection of PSII (Allahverdiyeva et al. 2015).

ROS can also be generated in the Fe-S centers in which the Fe^{2+} is involved in Fenton's reaction, where H_2O_2 produces $\cdot OH$, one of the most powerful oxidants (Halliwell and Gutteridge 2015). Another reactive species generated in these organelles is 1O_2 as product of the activity lipoxygenase (Triantaphylides and Havaux 2009), and it is also linked to the excitation of chlorophylls associated with the

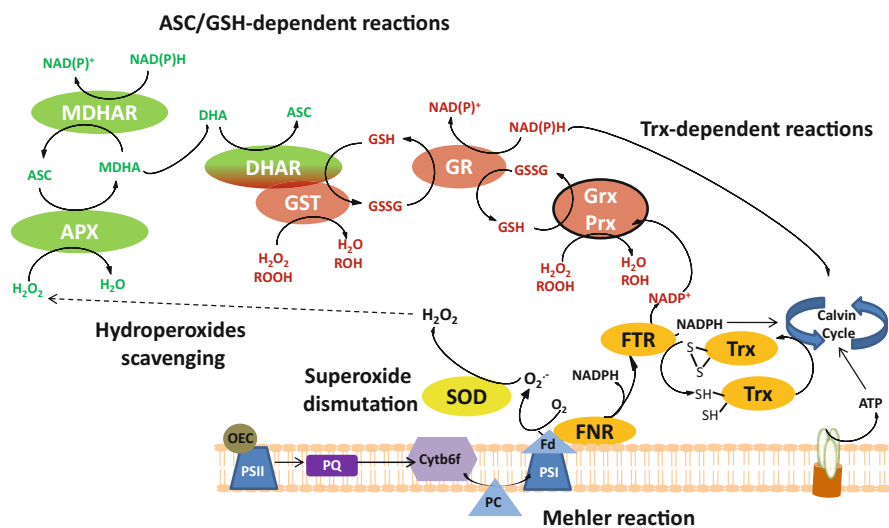


Fig. 2 The water-water cycle and the antioxidant system involved in ROS scavenging in chloroplasts. Pseudo-cyclic electron flow occurs in the PSI (Mehler reaction) to generate $O_2^{\cdot-}$ which dismutates to H_2O_2 . ASC-GSH cycle in coordination with the Trx/Prx/Grx system is in charge of the control of hydroperoxide ($ROOH$, H_2O_2) levels. *APX* ascorbate peroxidase, *Cytb6f* cytochrome b6f complex, *DHAR* dehydroascorbate reductase, *Fd* ferredoxin, *FNR* Fd reductase, *GST* glutathione S-transferase, *GR* glutathione reductase, *GRX* glutaredoxin, *MDHAR* monodehydroascorbate reductase; PSI, photosystem I, *PSII* photosystem II, *Prx* peroxiredoxin, *Trx* thioredoxin, *TR* thioredoxin reductase, *PC* plastocyanin, *SOD* superoxide dismutase (modified from Foyer and Noctor 2011)

electron transport. Specifically it is produced in the PSII and its light-harvesting antennae (Shapiguzov et al. 2012) and is able to promote cell death and to regulate gene expression (Telfer 2014; Laloi and Havaux 2015). Carotenoids are the main $^1\text{O}_2$ scavengers in the chloroplast, and its oxidized form has been shown to induce gene expression of singlet oxygen signaling pathway but with scarce effect on H_2O_2 -responsive gene expression (Ramel et al. 2012).

Several alternative electron pathways have been proposed to increase ATP in the chloroplast, and among them, cyclic electron flow around photosystem I (CEF) is the most important. In this cycle, electrons flow from PSI to PQ, producing ATP without generating of NADPH. This cycle has been reported to be activated by H_2O_2 produced by the imbalance of chloroplast redox state in vivo (Strand et al. 2015), probably through the regulation of proteins involved in the antimycin A-insensitive pathway of CEF or by inactivation of Calvin cycle enzymes.

2.2 ROS Scavenging

2.2.1 ASC-GSH Cycle and SOD

Related to the scavenging and redox control of H_2O_2 , as mentioned above, the ASC-GSH cycle has been described in chloroplast and later on in other cellular compartments as mitochondria, peroxisomes, apoplast, and cytosol (Jiménez et al. 1997; del Río and López-Huertas 2016), involving the antioxidants ascorbate and glutathione and the enzymes ascorbate peroxidase (APX), monodehydroascorbate reductase (MDHAR), dehydroascorbate reductase (DHAR), and glutathione reductase (GR). In this cycle, the H_2O_2 is eliminated by the APX using ASC as electron donor (Fig. 2). The MDHA formed, and from this, the DHA resulting spontaneous dismutation is used for recycling the ASC by the action of NADH-dependent MDHAR and by DHAR, which uses reduced GSH as electron donors, respectively. DHA can also suffer an irreversible hydrolysis, so DHAR may have a significant role in the maintenance of reduced ASC pool at cellular level (Gallie 2013). The role of this cycle in chloroplast has been reported under many different stress situations (Noctor and Foyer 1998; Gómez et al. 1999; Sharma and Dubey 2005; Lázaro et al. 2013; Bartoli et al. 2017; Ortiz-Espín et al. 2017a, b). As an example of the beneficial role of one of the components APX, overexpression of this enzyme in chloroplast, cytosol, and peroxisome was reported to enhance tolerance to salinity, cold, and heat stresses, diminishing the oxidative damage induced (Badawi et al. 2004; Lu et al. 2007; Wei-Feng et al. 2008). On the other hand, mutants lacking stromal and thylakoidal APX are more susceptible to photooxidative damage induced by high light (Kangasjärvi et al. 2008), and increase in ASC and GSH is a response to high irradiation (Bartoli et al. 2009). An antagonist effect of H_2O_2 and $^1\text{O}_2$ has been reported in chloroplast when overexpression of tAPX decreased H_2O_2 levels but increased $^1\text{O}_2$ -mediated response in an excess mutant *Arabidopsis flu* (*Arabidopsis* fluorescent, Laloi et al. 2007). An increase in chloroplast H_2O_2 was

reported in pea plants under salinity together with augmented Fe-SOD, Cu, Zn-SOD II and APX, DHAR, and GR activities (Gómez et al. 1999). These last two enzymes seem to be responsible for the regeneration of the oxidized antioxidants ascorbate and glutathione in this condition and, all together, collaborate to cope with the stress situation. Later on, an increase in H₂O₂ by a decrease in tAPX parallel to increased thylakoid- and stroma-located Fe-SOD and Cu,Zn-SOD activities was shown in these pea plants under salinity (Gómez et al. 2004), with a proposed role for these isoenzymes in the signaling events leading to plant acclimation. All these examples demonstrate the fine tuning of the different organelles through the regulation of the SOD isoenzymes and ASC-GSH components in the protection and acclimation to changing environment.

Alternatively, the regeneration of ASC may also take place due to other redox compounds such as glutaredoxin (Grx) and Trx system (Meyer et al. 2012; Potters et al. 2002), as described below. Oxidized glutathione (GSSG) can also diminish due to the NADPH-dependent thioredoxin reductase (NTR) (Barranco-Medina et al. 2007; Martí et al. 2009; Cejudo et al. 2014). In consequence, the cellular redox status will depend partly on the thiol and disulfide groups of the antioxidants, among others, the antioxidant GSH, and the proteins Trx, Prx, Srx, and Grx. On the other hand, it is considered that the redox state in the different cellular compartments depends to a great extent on the relative rate between reduced and oxidized groups of GSH and Trx (Go and Jones 2010; Foyer and Noctor 2013; Yoshida et al. 2018).

The accepted role of ROS/RNS as part of signaling events is changing the traditional concept of antioxidant systems in charge of their control, to that of “ROS processes systems,” as recently suggested by Foyer (2018). In fact, cellular components that interact with ROS are able to transmit oxidative signals from the different cell compartments, including chloroplasts, as described for phytohormones involved in plant growth and stress response (Bartoli et al. 2013; Xia et al. 2015; Foyer et al. 2017).

2.2.2 Thioredoxins

Thioredoxins (Trxs) are small oxidoreductase proteins of around 12–14 kDa involved in the reduction of the disulfide bonds of other proteins through a dithiol-disulfide exchange, in this way modulating the stability and function of their target proteins (Sevilla et al. 2015a, b). These enzymes present an active preserved site formed by a few amino acids and two cysteine residues (-WC(G/P)PC-). The maintenance in the cell of the reduced state of Trx depends on an external electron donor in every cellular compartment. In this way, mitochondrial, cytoplasmic, and nuclear Trxs are reduced by NADPH-dependent TR (NTR), which uses NADPH as a donor of electrons via FAD, whereas the chloroplastic Trxs use the electrons proceeding from the ET via ferredoxin (Fd) to be reduced by Fd/TR (FTR) (Balsera et al. 2014; Cejudo et al. 2014). A different NADPH NTR (NTRC) has been described in chloroplasts and nonphotosynthetic plastids presenting two sites, one Trx and another NTR, in the same polypeptide chain (Serrato et al. 2004;

Kirchsteiger et al. 2012), allowing to function also as Trx on the target chloroplastic 2-Cys Prx (Spinola et al. 2008; Pérez-Ruiz et al. 2009). This is an alternative system to the ferredoxin-dependent one, using NADPH produced by ET in the photosynthetic process during the day or in the first reactions of the oxidative pentose phosphate pathway during the night (Pérez-Ruiz et al. 2006; Alkhalfioui et al. 2007).

Location of Trxs is mainly cytoplasmic and chloroplastic, although there are members of the family in mitochondria, nucleus, apoplast, and endoplasmic reticulum (Gelhaye et al. 2004; Martí et al. 2009; Pulido et al. 2009; Meng et al. 2010; Calderón et al. 2017a). Among the chloroplastic Trxs, several types are described in *Arabidopsis*: Trxs *f* and *m* with important functions in the regulation of photosynthesis, specifically in the Calvin cycle (Jacquot et al. 1978; Wolosiuk et al. 1979), and also in the light-driven synthesis of ATP, catalyzed by ATP synthase. Recently, it has been described that this Trx-dependent redox modulation of ATP synthase may be involved in regulating the dark stability of the photosynthetic apparatus, most likely by controlling thylakoid membrane transport of proteins and ions (Kohzuma et al. 2017).

Other chloroplasts Trxs include Trx *x* involved in the oxidative stress by activation of 2-Cys Prxs (Mestres-Ortega and Meyer 1999; Pulido et al. 2010), Trx *y* with the capacity to reduce different plastidial Prxs (Collin et al. 2004; Lemaire et al. 2003), and Trx *z* (Arsova et al. 2010), presenting an important function in the development of the chloroplast by controlling the transcription of specific genes (Rivas et al. 2004; Pfalz et al. 2006). Trx *z* is required for the PEP polymerase and the PEP-dependent transcription of plastid genes like *psbA* (D1 protein of PSII) (Dietz and Pfannschmidt 2011). Interestingly, this Trx *z* has a unique function, due to its property of being reduced by other Trxs (Chibani et al. 2011). A role for Trx *x*, Trx *y* together with the atypical CDSP32 has also been described in the response to oxidative stress in chloroplasts (Broin 2002; Collin et al. 2003, 2004). CDSP32 protein has an important role during drought (Broin 2002), and the APR (adenosine 5'-phosphosulfate reductase) proteins, similar to PDI, participate in the sulfur metabolism through the reduction of sulfate to sulfite, among other functions (Martin et al. 2005). As an interesting example, another chloroplast stroma-localized atypical Trx from *Arabidopsis*, designated as Trx-like2 (TrxL2), has been characterized as presenting redox-active properties with an unusually lower negative redox potential (Yoshida et al. 2018). This Trx interacts with a range of chloroplast redox-regulated proteins with the special characteristic that it is able to efficiently oxidize, but not reduce, its target proteins Rubisco activase (RCA), fructose-1,6-bisphosphatase (FBPase), and sedoheptulose-1,7-bisphosphatase (SBPase) efficiently. In fact, Trx *f* could do it as well but much less efficiently. TrxL2 is then a redox-mediator protein specialized in oxidizing redox-regulated proteins and is strongly dependent on 2-Cys Prx and H₂O₂. Interestingly, it seems that TrxL2/2CP allows chloroplasts to turn off the photosynthetic metabolism at night as an acquired mechanism of land plants.

Taking into account the identification of target proteins and the phenotype of transformed plants, we can assume that the main function of Trxs is their action as redox proteins and antioxidants. A multifunction has been described for Trxs in several processes including plant development via regulation of the embryo

formation, control of intercellular communication, reserve mobilization, chloroplast structure and biogenesis, carbon and phosphate metabolism, plant cell death, photorespiration, plant respiration, ATP generation, stress response, and redox regulation of cellular homeostasis in the different compartments including chloroplasts (Reichheld et al. 2007; Martí et al. 2009, 2011; Arsova et al. 2010; Lázaro et al. 2013; Calderón et al. 2017a, 2018; Ying et al. 2017). The participation of Trx in the control of the redox state of Cys residues in the cellular proteome is a key function because it allows the modulation of the protein function in various cellular cycles such as the Calvin cycle, cellular cycle, or tricarboxylic acid cycle (Daloso et al. 2015; Calderón et al. 2017a). As an antioxidant it is able to transfer electrons in a cascade of oxido-reduction to eliminate ROS. An example in chloroplast is the activation by reduction of PrxIIE, an enzyme able to eliminate H_2O_2 , among other hydroperoxides (Gama et al. 2008).

One interesting concept is the regulation by reductants of the ROS production and antioxidant regeneration, described by Foyer and Noctor (2016). In chloroplast, a “push-pull model” was presented by these authors for the dependence of ROS generation on reductants in light, when Fd-NTR/Trx pools become more reduced. In this situation, an increase in ROS is favored by an excess of reductants, which has been described as “reductive stress.”

2.2.3 Peroxiredoxins and Sulfiredoxins

Peroxiredoxins (Prxs) are a family of peroxidases dependent on thiol groups that catalyze the scavenging of peroxides (ROOH) from H_2O_2 , alkyl hydroperoxides, and peroxynitrite to water and the corresponding alcohol (ROH) (Bryk et al. 2000; Rhee et al. 2005; Pedrajas and Bárcena 2018). In plants, the Prxs are classified into four subclasses depending on the number and position of the conserved Cys residues: 1-Cys-Prxs, 2-Cys Prxs, Prx associated to bacterioferritin (Prx Q), and Prx type II (associated to the YLR109 protein). In this last group, there are several members in *Arabidopsis*, poplar, pea, and rice with cytoplasmic, PrxIIC; plastidial, PrxIIE; and mitochondrial location, PrxIIF (Horling et al. 2002; Dietz et al. 2006; Barranco-Medina et al. 2007; Martí et al. 2009). It has been demonstrated that Prxs participate in several processes as 1-Cys-Prx does in seed germination (Haslekås et al. 2003) and in photooxidative stress in chloroplasts (Prx Q) (Rey et al. 2005).

During the catalytic cycle, Prxs are oxidized by H_2O_2 to sulfenic form (Cys-SOH) in the peroxidatic cysteine. The disulfure form and/or sulfenic form is then specifically reduced by cellular thiol groups as Trx or Grx (Barranco-Medina et al. 2007, 2009; Meyer et al. 2012; Couturier et al. 2013) (Fig. 3, lower part). Moreover, the sulfenic form can be over-oxidized by H_2O_2 to the more stable sulfinic form (Cys-SO₂H) with concomitant inactivation of the peroxidase activity, allowing a transient H_2O_2 accumulation which could act as signaling event in response to the oxidative situation (D’Autreaux and Toledano 2007). The action mechanism of Prx is not limited only to its peroxidase function but also to the chaperone modulation

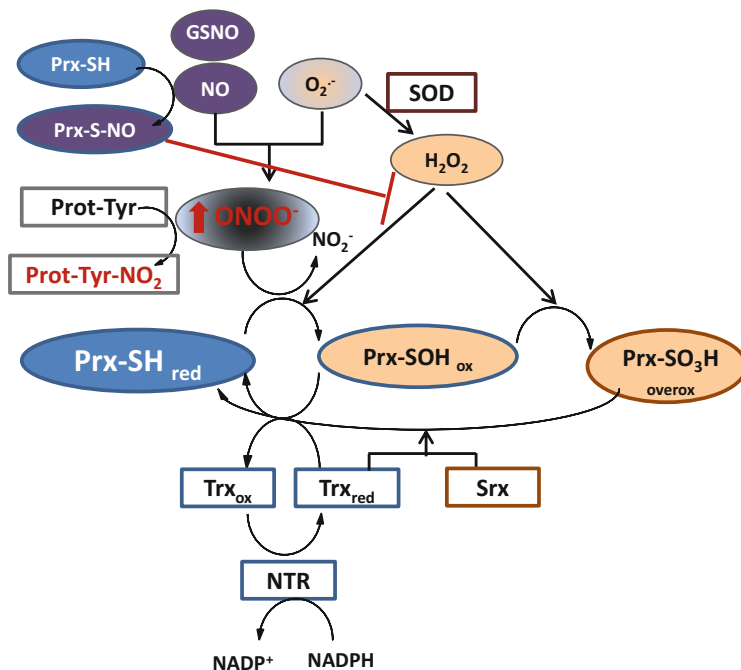


Fig. 3 Proposed mechanism of action of reactive oxygen and nitrogen species on PrxIIIE. Dismutation of $O_2^{\bullet -}$ by superoxide dismutase (SOD) generates H_2O_2 which oxidizes and inactivates the peroxidase PrxIIIE, which is regenerated from its oxidized (ox) forms by the thioredoxin (Trx)/thioredoxin reductase (NTR)/sulfiredoxin (Srx) system at expense of NADPH. NO/ $O_2^{\bullet -}$ reaction leads to the formation of ONOO⁻ which could be metabolized by reduced (red) PrxIIIE. The S-nitrosylation of PrxIIIE by NO or nitrosoglutathione (GSNO) inhibits its peroxynitrite reductase activity (in red) provoking an increase in Tyr nitration of target proteins (modified from Romero-Puertas et al. 2007; Iglesias-Baena et al. 2010)

protecting cellular structures from over-oxidation (Finkemeier et al. 2005; Barranco-Medina et al. 2008, 2009). On the other hand, changes in oligomerization and redox states lead to postulate that Prxs could translate and inform about cellular H_2O_2 . In this sense, these proteins could act as very sensitive H_2O_2 sensors linking oxidative metabolism to a variety of processes dependent on redox state in the different compartments.

The over-oxidized form of Prx can be reverted by sulfiredoxin (Srx) in an ATP-dependent manner in the 2-Cys Prx (Iglesias-Baena et al. 2010). This mechanism operates for plant Srx, which presents a double localization: mitochondrial and chloroplastic (Iglesias-Baena et al. 2010, 2011). However, the hyper-oxidation to a sulfonic form is considered to be an irreversible mechanism. The function of the Srx relates to the maintenance of the redox balance. As an example, it has been shown that under H_2O_2 treatment *AtSrx* gene expression increased and that KO *AtSrx* mutants present higher oxidative stress than wild-type plants. Also, paraquat

treatment provokes an increase in ROS levels in chloroplast of KO mutants, whereas this hypersensitivity is reversed on having restored AtSrx's expression by genetic transformation. This information indicates that the expression and AtSrx's function in chloroplasts are essential for a suitable response of the plant to the conditions of stress (Liu et al. 2006). This role in the balance redox, accompanied by its specificity for Prx and the need for a reductant, such as Trx, to carry out Srx activity, as well as the union of Trx-Prx, has led to the description of the existence in the cell of a system of redox regulation formed by Trx, Prx, and Srx—the so-called Trx/Prx/Srx system. These proteins participate in the signaling network to perceive and transmit mainly ROS and redox sensors (Sevilla et al. 2015a, b).

3 NO Metabolism

3.1 NO Synthesis in Plants

Biosynthesis of NO in plants is still a matter of debate, and several sources have been proposed as possible pathways for NO generation (Gupta et al. 2011; Mur et al. 2013), which depend upon either reductive or oxidative routes taking place in different cell compartments, including cytosol, mitochondria, chloroplasts and plastids, peroxisomes, and apoplast, where RNS and ROS accumulate during stress (Jasid et al. 2006; Corpas et al. 2009; Igamberdiev et al. 2014; del Río 2015; Astier et al. 2018). In mammals, NO is synthesized via an oxidative mechanism by NO synthase (NOS: EC 1.14.13.39), which consists of three well-characterized isoforms, endothelial NOS (eNOS), neuronal NOS (nNOS), and inducible NOS (iNOS) (Alderton et al. 2001), and which was originally isolated from macrophages. NOS proteins use L-arginine as substrate, generating a hydroxyl-arginine intermediate, to produce citrulline and NO. The reaction is NADPH dependent, utilizes O₂ as a co-substrate, and needs the presence of tetrahydrobiopterin (BH₄). Recently, a related protein has been discovered in *Ostreococcus tauri*, a single-cell green algae that shares a common ancestor with higher plants, bacteria, and human. The NOS sequence possesses 45% similarity to human NOS and exhibited NOS activity in vitro, showing similar properties to animal NOS proteins (Foresi et al. 2010). Genes encoding a structurally related NOS enzyme have not been identified in higher plants, even though over 1000 transcriptome sequences of land plants have been analyzed for the presence of canonical NOS sequences (Jeandroz et al. 2016). These results indicate that the NOS activity reported in plants comes from a different type of enzyme (Gas et al. 2009). Taken together, the emerging evidence supports that NO generation could occur from multiple reductive and oxidative sources in land plants and also in different cellular localization.

3.2 Sources of NO in Plants

Reductive routes are dependent upon nitrite as the primary substrate and include reduction via nitrate reductase (NR EC 1.7.1.1), a plasma membrane-bound nitrite-NO reductase (NiNOR) and mitochondrial nitrite reduction. Nitrate reductase (NR) is a key enzyme for NO production in plants and facilitates NO homeostasis (Chamizo-Ampudia et al. 2011). NR in addition to the reduction of nitrate (NO_3^-) to nitrite (NO_2^-) can also catalyze the reduction of nitrite to NO. However, the efficiency of this reaction is low, and it requires small oxygen tensions and light and high nitrite concentrations (Rockel et al. 2002; Igamberdiev et al. 2014). NO production via NR was demonstrated either in vivo as in vitro (Rockel et al. 2002; Seligman et al. 2008). It has been shown that under in vitro conditions, commercial purified maize NR generated NO under ambient air conditions (Yamasaki et al. 1999; Ischiropoulos 2003). In *Arabidopsis*, NR enzyme is encoded through two homologous genes (*Nia1* and *Nia2*) (Chaki et al. 2015). Double *nia1nia2* mutants were used to demonstrate the involvement of NR in some physiological processes. Thus, NR was shown to be involved in the NO generation during the control of stomatal closure induced by ABA and other physiological processes as flower development (Seligman et al. 2008) and plant immunity (Thalineau et al. 2016).

The presence of a PM-bound NiNOR enzyme has been suggested in tobacco roots plasma membrane (Stöhr et al. 2001). The nitrite as substrate for NiNOR is probably provided by plasma membrane-bound NR in a coupled reaction. Unfortunately, the identity of NiNOR remains to be determined. Moreover the nonenzymatic reduction of nitrite leading to the formation of NO and nitrate is known to be favored at acidic pH values, such as those in apoplasts and plastids (Cooney et al. 1994; Bethke et al. 2004).

Mitochondria also support the reduction of nitrite to NO (Tischner et al. 2004; Modolo et al. 2005; Igamberdiev et al. 2014) in the mitochondrial inner membrane, probably via cytochrome c oxidase and/or reductase (Planchet and Kaiser 2006). This process contributes to ATP production under hypoxic conditions (Stoimenova et al. 2007). Pharmacological evidence also suggests that complex III and AOX are both involved in nitrite to NO reduction, although a clear mechanism is established only for cytochrome oxidase under hypoxia (Gupta and Igamberdiev 2016).

In peroxisomes, as other source, NO can also be generated by reversible regulation of *S*-nitrosoglutathione (GSNO) by the action of GSNO reductase (GSNOR) (Corpas et al. 2013a).

Regarding the oxidative NO sources, evidence for the production of NO through an *L*-Arg-dependent NOS-like activity and via polyamine-/hydroxylamine-mediated synthesis has been described (Delledonne et al. 2001; Corpas et al. 2006, 2017; Durner et al. 1998). In this regard, Guo and Crawford (2005) showed that the *Arabidopsis* protein named NO synthase 1 (AtNOS1) is targeted to the mitochondria. This protein was further characterized as a functional small GTPase and therefore renamed AtNOA1 (nitric oxide associated 1) (Moreau et al. 2008) which might be implied in mitochondrial biogenesis. To date, in plant mitochondria, in

contrast with mammalian tissue, the production of NO by a NOS-like enzyme remains elusive (Astier et al. 2018).

The localization of a NOS-like activity has been also reported in the subcellular compartments of peroxisomes and chloroplasts, both organelles taking part in NO production in environmental conditions (Jasid et al. 2006; Corpas and Barroso 2014).

On the other hand, increases in the concentrations of the polyamines spermine and spermidine induce NO release, but the actual reaction mechanism has not yet been determined. Recently it has been reported that in *Arabidopsis thaliana*, copper-containing amino oxidase 1 (CuAO1) mediates the generation of NO in response to both polyamine and ABA (Tun et al. 2006).

3.3 NO Generation in Chloroplasts

Early evidence obtained by fluorescence microscopy (Foissner et al. 2000; Gould et al. 2003) and immunogold electron microscopy (Barroso et al. 1999) suggested that chloroplasts participate in NO synthesis in plants. Moreover, it was reported that chloroplasts were the first organelle where NO increased after elicitation of rape (*B. napus*) cells with cryptogein (Foissner et al. 2000). Further evidence obtained in soybean showed that at least two pathways for NO production are operative in chloroplasts: one of them was dependent on L-Arg-dependent NOS-like enzyme activity and the other on nitrite, as suggested by in vitro exposure assays (Jasid et al. 2006). In this regard, based on loss of function of *Atnoa1* mutants, it has been also sustained that chloroplasts are involved in NO generation (Gas et al. 2009), and it is known that AtNOA1, as a cyclic GTPase, is involved in protein translation in the chloroplast (Gas et al. 2009; Chen et al. 2010). The decrease in accumulation of NO in *Atnoa1* mutants has been indirectly linked to the inability of that mutant to fix carbon, resulting in a decrease in fumarate stores, which is inherently leading to a decrease in L-arginine accumulation and chloroplast dysfunction (Van Ree et al. 2011). Phenotype of maize NOA1 knockdown is lethal. Recently, the study of generation of NO in *B. napus* without any external supply of arginine or NO donors suggests the involvement of a NOS-like protein, but not nitrate reductase, in NO generation in the leaf chloroplasts and protoplasts (Tewari et al. 2013). Additionally, the reduction of nitrite to NO by ascorbic acid under microlocalized pH conditions has also been proposed in chloroplasts (Beligni et al. 2002). The light-mediated reduction of NO₂ by carotenoids is also a source of NO generation (Cooney et al. 1994).

3.4 NO Targets in Chloroplasts

There are interesting reviews on the effects of NO in chloroplasts mainly in the photosynthetic process (Misra et al. 2014; Simontacchi et al. 2015; Turkan 2018). In this chapter, we present some examples using mutant plants with modified endogenous NO content as well as results obtained after exogenous treatments with NO donors. Early studies reported the reduction in CO₂ assimilation by NO (Hill and Bennett 1970). Furthermore, both photosynthesis and photorespiration have been found to be affected by NO in different plants at concentrations below those required to result in visible injuries (Yamasaki 2000; Takahashi and Yamasaki 2002). Elsewhere, it has been reported that NO is involved in light-mediated greening of barley seedlings (Zhang et al. 2006) and it stimulates chlorophyll biosynthesis and chloroplast differentiation by increasing iron availability (Graziano et al. 2002; Kumar et al. 2010).

The analysis of *NOA1*-suppressed mutants which presented low contents of NO has revealed a lower PSII efficiency in comparison with WT plants, suggesting a decrease in the energy transfer efficiency (Liu et al. 2016). NO is able to bind reversibly to several sites in PSII and inhibit electron transport, so decreasing photosynthetic activity in isolated thylakoids of spinach (Sanakis et al. 1997). In this regard, by applying exogenous NO or different NO donors on leaves and/or isolated chloroplasts, several electron transport chain components of PSII have been identified as target sites of NO, including the non-heme iron between Q_A and Q_B quinone binding sites, which results in an important decrease of the electron transport rate between both Q_A and Q_B (Diner and Petrouleas 1990). Also, NO binds at the catalytic Mn cluster of the oxygen-evolving complex (OEC) (Schansker et al. 2002) and interacts with the tyrosine residue (Y_D) of the D2 protein (Sanakis et al. 1997). The action of NO as a reductant in destabilizing the excited states of the Mn cluster complex is well demonstrated, as is the decrease of photosynthetic oxygen evolution in a NO-dependent manner (Schansker et al. 2002). Regarding the Yp-NO couple, NO binding seems to result in a decreased redox potential that becomes a more efficient electron donor in isolated thylakoids than the immediate redox-active tyrosine residue Yz in D1 protein (Vlaskova et al. 2011). As another example, the interaction of NO with cytochrome b₆f complex—the electron transport mediator between PSII and PSI complex—has also been reported. The possible involvement of this NO cytb₆f interaction in signaling pathways, by modulation of cyclic electron flow around PSI, and so regulating redox potential in chloroplasts, has been suggested (Twigg et al. 2009). Interestingly, it has been observed that mutants that exhibit constitutively high rates of cyclic electron flow (CEF) also present increased production of H₂O₂. Further evidence appeared that H₂O₂ resulting from imbalances on plant chloroplasts redox state acts as signaling agent to activate CEF in vivo (Strand et al. 2015).

On the other hand, most of the studies employing exogenous application of NO and/or application of NO chemical donors find that NO effects in photosynthesis are dependent on the nature and concentration of the donor used and also on

environmental conditions. Contradictory results may be obtained if they are analyzed under nonstress or under stressful conditions (Misra et al. 2014). As an example, leaves treated with sodium nitroprusside (SNP) and GSNO showed decreased maximum quantum efficiency (Fv/Fm). However, SNP-treated leaves under different abiotic stresses (including drought, salinity, heat, UVB, metal toxicity) had higher Fv/Fm when compared with the nonstressed SNP-treated leaves, pointing to the stimulating effect of the NO donor treatment on PSII photochemistry, in contrast to that described under nonstress conditions. This positive effect of SNP on PSII has been correlated with an enhancement in the proportion of the open PSII reaction centers (Gupta et al. 2012; Manjunatha et al. 2012). Other protective effects have also been reported by NO under stressful conditions. As an example, and related to that commented on above, NO has been shown to prevent chlorophyll loss. This was shown in sunflower plants subjected to Cd-induced chlorophyll decay (Laspina et al. 2005), where the results were similar to those reported in mesophyll cells from Fe-deficient maize plants, in which SNP prevented leaf chlorosis by significantly increasing chlorophyll content and chloroplasts development (Graziano et al. 2002). A protective effect of NO was also reported in relation to carbonyl and lipid peroxidation contents in chloroplasts (Jasid et al. 2006; Galatro et al. 2013).

Additionally, the effects of NO on metabolism related to photosynthesis have been reported to depend on its concentrations. As an example, Abat et al. (2008) described that NO slows photosynthesis through inhibiting Rubisco by *S*-nitrosylation in a dose-dependent manner (see below). In this context, an enhanced carbonic anhydrase activity was induced by micromolar levels of SNP treatment in tomato, which indirectly resulted in a constant supply of CO₂ to ribulose-1,5-bisphosphate carboxylase/oxygenase (Rubisco). Opposite effects were observed at high NO concentrations, which decreased carbonic anhydrase as well as Rubisco activities (Ferreira et al. 2008; Hayat et al. 2011). This may also be related to the fact that elevated NO content induces the closure of stomata (García-Mata and Lamattina 2001).

4 ROS/RNS and Stress

It has been well established that ROS/RNS generation is a key feature of stress. Thus, the control of ROS/RNS levels by modulation of their production and scavenging can change the role of these species as toxic molecules to signaling ones, so enabling a good response and adaptation to the changing stressful conditions. Interestingly, chloroplasts as described above, together with other cell compartments, are ROS/NO producers and use oxygen/nitrogen derived undesired metabolites to send signals to other compartments (retrograde signaling), so provoking metabolic responses to avoid severe damages (Locato et al. 2018). In this way, redox and antioxidant systems are coordinated to counteract the oxidative stress situation imposed depending on the stress type, duration, and sensibility. As an example, it has been shown that H₂O₂ increase after downregulation of sAPX and

tAPX in defective *Arabidopsis* mutants provokes a negative regulation of a cold stress response but a positive regulation under biotic stress. In these situations, a different gene expression was observed, pointing to the specificity of the response (Maruta et al. 2012).

Another interesting concept related to retrograde regulation is the fact that not only ROS generated in the chloroplasts are involved as intermediates in signaling between this organelle and the nucleus, as reported during acclimation of photosynthesis (Galvez-Valdivieso and Mullineaux 2010), but that photosynthetic functions are also regulated by cues perceived in the apoplast (Shapiguzov et al. 2012), pointing to a larger network collaborating for the stress adaptation. Other plastidial components like glutathione and the Trx system are not only involved in dynamic regulation of photosynthesis but also seem to be able to communicate this redox state in the organelle to the cytoplasm (Bashandy et al. 2010; Noctor et al. 2012).

Several chloroplastic redox hubs, including the plastoquinone and the glutathione pools and the thioredoxin system, provide not only dynamic local regulation of photosynthesis but may also communicate the chloroplast redox status to the cytosol (Marty et al. 2009; Bashandy et al. 2010; Foyer and Noctor 2011; Noctor et al. 2012; Rochaix 2012). For example, the redox state of plastoquinone, a component of the photosynthetic electron transfer chain, is monitored through the thylakoid-associated protein kinase State Transition 7 (STN7). STN7-dependent phosphorylation of chloroplast proteins leads, on the one hand, to optimization of photosynthesis in response to changing light conditions (via the reversible reallocation of light-harvesting antennae called state transitions) and, on the other, to a retrograde signal (Bonardi et al. 2005; Rochaix 2012). Another redox sensor in chloroplast is SAL1 phosphatase, which is inhibited by oxidation (provoking the dimerization of the protein) and glutathionylation. SAL1 dephosphorylates PAP (phosphonucleotide 30-phosphoadenosine 50-phosphate) into AMP (adenosine monophosphate) (Chan et al. 2016). PAP has been shown to accumulate under drought and high light stress, and the translocation to the cytosol provokes RNA cleavage and transcription termination (Chan et al. 2016). In this way, SAL1 is considered as a sensor of changing redox status in the organelle, and ROS-induced accumulation of this and other metabolites may serve as retrograde signals to provoke an adequate stress response.

Another emerging concept is that all types of oxidative modifications may be involved in signaling (Foyer et al. 2017) and ROS can act as death or life signals. In fact, PTMs induced by ROS/RNS are being considered as part of the redox signaling network inducing (or not) the repair, regulation or even desirable cell death, as part of the acclimation or fight against external invasion (biotic stress) or changing abiotic stressful environments.

5 ROS-/RNS-Mediated Protein Modifications

The specificity of redox signaling is achieved by reactive oxygen and nitrogen species generated in the vicinity of certain susceptible proteins in the different cell compartments in which an appropriate environment may support redox-based post-translational modifications (PTMs) (Umbreen et al. 2018). Interestingly, the reversibility of certain modifications is also part of the specificity as well as of the interactions among the different ROS/RNS players and the proteins involved in this reversibility.

5.1 Sulfenylation

ROS have a strong impact in the chemistry of thiol groups in proteins. Cysteine sulfenylation ($-SOH$) is a posttranslational modification related to the ROS-mediated oxidative product of a thiol ($-SH$) (Akter et al. 2017). An efficient photosynthetic carbon metabolism is necessary, and plants have evolved to adjust their physiology toward light fluctuations. One important mechanism is the reversible activation-inactivation during light/dark cycles due to reduction-oxidation switches of Cys residues, termed as redox regulation (Yoshida et al. 2018).

A lot of work is going on to discover the sensor proteins of ROS as a means of transduction of stimuli able to provoke a response to cope with the stress situations. Indeed, plant protein sulfenylation (sulfenome) under oxidative stress is considered a validated method in which reactive cysteine thiols are identified. Several chloroplastic proteins have thus been identified such as some specific Calvin cycle enzymes (Michelet et al. 2013), ribulose biphosphate carboxylase, ATP synthase, NADP-dependent malate dehydrogenase, adenosine kinase 1, and glutamine synthetase 2 (Akter et al. 2017). Another experimental approach is the identification of chloroplastic thioredoxin redox-regulated protein targets. In this sense, a lot of research has been carried out with many different proteins identified that cover the main processes in the organelle, including gene expression, photosynthesis, Calvin cycle, biogenesis of plastids, translation, biosynthetic and antioxidant metabolisms, and stress response (review by Montrichard et al. 2009; Nikkanen et al. 2017). Usually oxidation of the proteins provokes inactivation of the enzymatic activity pointing thioredoxins as key components for protein functionality in both normal metabolism and stress responses. As an example, oxidation of Rubisco induces conformational changes and the PSII efficiency decreases, correlated with the sulfenylation state of the enzyme (Akter et al. 2017). Other redox-regulated proteins in the thylakoid lumen are immunophilin, FKBP13, polyphenol oxidase, and violaxanthin de-epoxidase, which are oxidatively activated by the oxygen release at the PSII site (Buchanan and Luan 2005). Also oxidation of peroxiredoxins provokes oligomerization, leading to a loss in the peroxidase function but gaining a role as chaperone (Barranco-Medina et al. 2009), although Cerveau et al. (2016) suggested

that chaperone function of chloroplastic 2-Cys Prx may not be essential in plants, due to the absence of abundant high-molecular-weight complexes under water deficit or photooxidative conditions. All these are examples that point to ROS-induced posttranslational modification sulfenylation as a key event in the regulation of the structure and function of essential processes in the chloroplasts with a high impact in the metabolism of living cells.

5.2 *S-Nitrosylation and Tyr Nitration*

NO mediates several posttranslational modifications (PTMs) in plants, such as protein tyrosine nitration and *S*-nitrosylation, which are processes that have a high impact in the activity of several enzymes collaborating with other defense mechanisms in the adaptation or tolerance to stress situations (Romero-Puertas et al. 2013; Chaki et al. 2011; Camejo et al. 2013, 2015). In the protein nitration, a nitro group ($-\text{NO}_2$) from nitric oxide is added to Tyr, Trp, Cys, or Met residues of a target protein (Corpas et al. 2009). *S*-nitrosylation, in turn, is the result of the covalent binding of an NO group to a Cys residue (Seth and Stamler 2011). Since both PTMs are involved in signaling, they are presented in more detail below.

Peroxynitrite results from the reaction between NO and $\text{O}_2^{\bullet-}$ and together with GSNO, acts as a natural reservoir and contributes to the control of NO in plant cells. ONOO⁻ presents a very strong nitrating capacity to mediate Tyr nitration. Various studies on the nitro-Tyr proteome have revealed 127 proteins, with several targets located in different organelles including chloroplasts and mitochondria in *Arabidopsis* (Galetskiy et al. 2011a,b; Lozano-Juste et al. 2011; Tanou et al. 2012). In chloroplasts, nitrated enzymes are related to several processes, such as Calvin cycle, photosynthesis, ROS metabolism, and protein synthesis (Table 2). This PTM can irreversibly modify protein conformation, also affecting the catalytic activity and susceptibility to proteolysis (Corpas et al. 2009). As examples of its effect, Tyr nitration inhibits the activity of FNR and chloroplast SOD 3 (Chaki et al. 2011; Holzmeister et al. 2014). Other inactivated enzymes are G3PDH, carbonic anhydrase (Lozano-Juste et al. 2011; Chaki et al. 2013), or the destabilization of the electron transport from the PSI to Fd after Tyr nitration of the D1 protein (Galetskiy et al. 2011a), pointing to nitration as being related to photodamage and disassembly of complexes leading to protein degradation (Galetskiy et al. 2011a). NO treatment of thylakoid membranes isolated from pea leaves provoked the inhibition of the electron transport by binding of NO to several PSII sites as described above (Wodala et al. 2008), and Tyr nitration of D2 protein induced a decrease in the redox potential enough to become a more efficient electron donor than D1 protein (Vladkova et al. 2011). The presence of nitrotyrosine has been considered as a marker for strong nitrosative stress, due to the increase in ONOO⁻ generation as a consequence of environmental and/or biotic stress conditions, although physiological protein nitration also occurs. Moreover, Tyr nitration is quite specific because only certain Tyr residues in target proteins are preferentially nitrated (Corpas et al. 2013b). As an

Table 2 Some Tyr-nitrated proteins in chloroplasts of photosynthetic organisms. For abbreviated protein name see text

Protein	Reference	Organism and treatment
<i>Calvin cycle</i>		
Rubisco	Cecconi et al. (2009)	Hypersensitive response <i>Arabidopsis</i>
	Tanou et al. (2012)	Citrus leaf
Rubisco	Lozano-Juste et al. (2011)	<i>Arabidopsis</i>
Rubisco activase	Cecconi et al. (2009)	Hypersensitive response <i>Arabidopsis</i> Sunflower hypocotyls
	Chaki et al. (2009) Tanou et al. (2012)	Citrus leaf
Glyceraldehyde 3-P dehydrogenase	Lozano-Juste et al. (2011)	<i>Arabidopsis</i>
	Tanou et al. (2012)	Citrus leaf
Phosphoglycerate kinase	Lozano-Juste et al. (2011)	<i>Arabidopsis</i>
	Tanou et al. (2012)	Citrus leaf and root
Phosphoribulokinase	Lozano-Juste et al. (2011)	<i>Arabidopsis</i>
	Tanou et al. (2012)	Citrus leaf
Sedoheptulose-1,7-bisphosphatase	Tanou et al. (2012)	Citrus leaf
Carbonic anhydrase	Chaki et al. (2009, 2013)	Sunflower hypocotyls
	Lozano-Juste et al. (2011)	<i>Arabidopsis</i>
Fructose-1,6-bisphosphate aldolase	Tanou et al. (2012)	Citrus leaf
<i>Photosynthetic proteins</i>		
Lhcb1, Lhcb2, Lhcb4, Lhcb5, Lhcb6 (PSII)	Galetskiy et al. (2011b)	<i>Arabidopsis</i> light stress
Lhcb6 (PSII)	Galetskiy et al. (2011b)	<i>Arabidopsis</i> light stress
PsbA protein of D1 subunit (PSII)	Galetskiy et al. (2011b)	<i>Arabidopsis</i> light stress
PsbD protein of D2 subunit (PSII)	Galetskiy et al. (2011b)	<i>Arabidopsis</i> light stress
PsbC (CP43)(PSII)	Galetskiy et al. (2011b)	<i>Arabidopsis</i> light stress
	Tanou et al. (2012)	Citrus leaf
HCF136 factor (PSII)	Lozano-Juste et al. (2011)	<i>Arabidopsis</i>
PbsP: Oxygen-evolving enhancer protein 2-1 (PSII)	Lozano-Juste et al. (2011)	<i>Arabidopsis</i>
Cyt b ₆ f	Galetskiy et al. (2011a, b)	<i>Arabidopsis</i> light stress

(continued)

Table 2 (continued)

Protein	Reference	Organism and treatment
ATP synthase gamma chain 1,	Lozano-Juste et al. (2011)	<i>Arabidopsis</i>
Lhca3 (PSI)	Galetskiy et al. (2011b)	<i>Arabidopsis</i> light stress
Lhca4 (PSI)	Galetskiy et al. (2011b)	<i>Arabidopsis</i> light stress
PsaF (PSI)	Galetskiy et al. (2011b)	<i>Arabidopsis</i> light stress
P700 apoprotein A (PSI)	Tanou et al. (2012)	Citrus leaf
Ferredoxin-NADP reductase	Chaki et al. (2011)	Heat stress sunflower hypocotyl
Ferredoxin-nitrite reductase	Chaki et al. (2011)	Heat stress sunflower hypocotyl
<i>Others</i>		
Elongation factor Tu	Cecconi et al. (2009)	Hypersensitive response <i>Arabidopsis</i>
Glutamine synthetase	Lozano-Juste et al. (2011)	<i>Arabidopsis</i>
	Chaki et al. (2009)	Sunflower hypocotyls
	Cecconi et al. (2009)	Hypersensitive response <i>Arabidopsis</i>
	Tanou et al. (2012)	Citrus leaf
Ketol-acid reductoisomerase	Lozano-Juste et al. (2011)	<i>Arabidopsis</i>
Phosphoglucomutase	Lozano-Juste et al. (2011)	<i>Arabidopsis</i>
Aspartate aminotransferase	Lozano-Juste et al. (2011)	<i>Arabidopsis</i>
	Tanou et al. (2012)	Citrus root
Malate dehydrogenase	Lozano-Juste et al. (2011)	<i>Arabidopsis</i>
Signal recognition particle 54 kDa protein,	Lozano-Juste et al. (2011)	<i>Arabidopsis</i>
3-Oxoacyl-[acyl-carrier-protein] synthase I	Lozano-Juste et al. (2011)	<i>Arabidopsis</i>
Thiazole biosynthetic enzyme	Lozano-Juste et al. (2011)	<i>Arabidopsis</i>
3-Oxoacyl-[acyl-carrier-protein] reductase	Lozano-Juste et al. (2011)	<i>Arabidopsis</i>
Magnesium-chelatase subunit chlD	Tanou et al. (2012)	Citrus leaf
Diaminopimelate decarboxylase 1	Tanou et al. (2012)	Citrus leaf
Antigen peptide transporter-like 1	Tanou et al. (2012)	Citrus leaf
Glutamate synthase	Tanou et al. (2012)	Citrus leaf
Pyruvate orthophosphate dikinase		
Sulfite reductase	Tanou et al. (2012)	Citrus leaf

(continued)

Table 2 (continued)

Protein	Reference	Organism and treatment
NAD-dependent epimerase	Tanou et al. (2012)	Citrus leaf
Lipoxygenase	Tanou et al. (2012)	Citrus root
Serine-type peptidase	Tanou et al. (2012)	Citrus root
Acylaminoacyl-peptidase1	Tanou et al. (2012)	Citrus root
Phosphoserine aminotransferase	Tanou et al. (2012)	Citrus root
Hsp70	Tanou et al. (2012)	Citrus root
6-phosphogluconolactonase	Tanou et al. (2012)	Citrus root
Glyoxalase	Tanou et al. (2012)	Citrus root
Carboxymethylenebutenolidase	Tanou et al. (2012)	Citrus root
Carbonic anhydrase	Chaki et al. (2011)	Heat stress sunflower hypocotyl

example, in control growth conditions, *Arabidopsis* PSII-LHCII complexes present low levels of phosphorylation and high nitration, while under high light conditions, the opposite occurred (Galetskiy et al. 2011b).

S-nitrosylation is mediated by NO and GSNO, with the latter also provoking S-glutathionylation (Camejo et al. 2015; Calderón et al. 2017b). S-nitrosylation is reversible, and thus it appears to be suitable for a signaling function. It has been reported that it affects protein activities, structure, localization, and interaction between proteins in animal and plant systems (Hess et al. 2005; Camejo et al. 2015; Zaffagnini et al. 2016). Several targets of S-nitrosylation have been identified in different cell compartments including chloroplasts (some are listed in Table 3). Several targets are involved in photosynthesis (in both, light-harvesting and carbon fixation), redox and antioxidant homeostasis, and amino acid biosynthesis, among others. Although this PTM may increase enzyme activity (Astier et al. 2012), the chloroplastic targets are mainly inhibited by S-nitrosylation (Lindermayr et al. 2005).

Related to GSNO metabolism, it is interesting to point out that GSNOR is the only enzyme capable of metabolizing GSNO, in a NADH-dependent manner, transforming GSNO into GSSG and ammonia. Regulation of GSNO cellular homeostasis allows GSNO to control the level of protein nitrosothiols. In fact, KO GSNO *Arabidopsis* plants presented higher levels of S-nitrosylated proteins than control wild-type plants (Feechan et al. 2005; Chaki et al. 2011), while the overexpression provoked decreased levels (Lin et al. 2012). This change in the nitrosylated pattern may affect the functioning of photosynthesis among other processes. Moreover, it has been proposed that a high cross talk exists among the different PTMs and that the influence of S-nitrosylation may be comparable with that of ubiquitination and phosphorylation (Zaffagnini et al. 2016).

Table 3 Some S-nitrosylated proteins in chloroplasts of photosynthetic organisms. For abbreviated protein name see text

Protein	Reference	Organism and treatment
<i>Calvin cycle</i>		
Rubisco	Abat and Deswal (2009) Abat et al. (2008)	GSNO treatment Low temperature NO treatment poplar
	Vanzo et al. (2016)	<i>Brassica juncea</i> , <i>Kalanchoe pinnata</i>
Rubisco	Romero-Puertas et al. (2008)	Hypersensitive response <i>Arabidopsis</i> and <i>Pseudomonas</i>
Rubisco	Lindermayr et al. (2005)	<i>Arabidopsis</i> , GSNO and NO treatment
Rubisco	Kato et al. (2013)	Potato GSNO treatment
Rubisco	Tanou et al. (2012)	Citrus leaf
Rubisco	Camejo et al. (2013)	Salinity, pea leaf
Rubisco activase	Vanzo et al. (2014, 2016)	NO treatment poplar
	Tanou et al. 2012	Citrus leaf
Ribose 5-P isomerase	Vanzo et al. (2014, 2016)	NO treatment poplar
Phosphoribulokinase	Vanzo et al. (2014, 2016)	NO treatment poplar
	Romero-Puertas et al. (2008)	Hypersensitive response
	Kato et al. (2013)	<i>Arabidopsis</i> and <i>Pseudomonas</i>
	Tanou et al. (2012)	Potato GSNO treatment Citrus leaf
Sedoheptulose-1,7-bisphosphatase	Vanzo et al. (2014, 2016)	NO treatment poplar
	Tanou et al. (2012)	Citrus leaf
Triose phosphate isomerase	Vanzo et al. (2014, 2016)	NO treatment poplar
	Lindermayr et al. (2005)	Salinity <i>Arabidopsis</i>
	Tanou et al. (2012)	<i>Arabidopsis</i> , GSNO and NO treatment Citrus root
Glyceraldehyde 3-P dehydrogenase	Vanzo et al. (2014, 2016)	NO treatment poplar
	Romero-Puertas et al. (2008)	Hypersensitive response
	Lindermayr et al. (2005)	<i>Arabidopsis</i> and <i>Pseudomonas</i> <i>Arabidopsis</i> , GSNO and NO treatment
	Tanou et al. (2012)	Citrus leaf
Phosphoglycerate kinase	Vanzo et al. (2014, 2016)	NO treatment poplar
	Lindermayr et al. (2005)	<i>Arabidopsis</i> , GSNO and NO treatment
	Tanou et al. (2012)	Citrus leaf

(continued)

Table 3 (continued)

Protein	Reference	Organism and treatment
Fru-1,6-bisphosphate aldolase	Lindermayr et al. (2005)	<i>Arabidopsis</i> , GSNO and NO treatment
	Tanou et al. (2012)	Citrus leaf
Fructose-1,6-bisphosphatase	Serrato et al. (2018)	In vitro GSNO and SNAP treatment, in vivo GSNO
Ribulose-5-phosphate-3-epimerase	Tanou et al. (2012)	Citrus leaf
<i>Photosynthetic proteins</i>		
PsbQ protein (PSII)	Romero-Puertas et al. (2008)	Hypersensitive response <i>Arabidopsis</i> and <i>Pseudomonas</i>
	Tanou et al. (2012)	Citrus leaf
PsbO protein (PSII)	Lindermayr et al. (2005)	<i>Arabidopsis</i> , GSNO and NO treatment
	Tanou et al. (2012)	Citrus leaf
PsbC protein (PSII)	Tanou et al. (2012)	Citrus leaf
PsbS protein (PSII)	Tanou et al. (2012)	Citrus leaf
PsbP protein (PSII)	Tanou et al. (2012)	Citrus leaf
PsbE(PSII)	Tanou et al. (2012)	Citrus leaf
P680 (PSII)	Lindermayr et al. (2005)	<i>Arabidopsis</i> , GSNO and NO treatment
D2 protein (PSII)	Tanou et al. (2012)	Citrus leaf
Lhcb protein (PSII)	Tanou et al. (2012)	Citrus leaf
Cyt b ₆ f	Lindermayr et al. (2005)	<i>Arabidopsis</i> , GSNO and NO treatment
	Tanou et al. (2012)	Citrus leaf
Plastocyanin	Kato et al. (2013)	Potato GSNO treatment
P700 apoprotein A (PSI)	Tanou et al. (2012)	Citrus leaf
PsaC (PSI)	Tanou et al. (2012)	Citrus leaf
PsbA (PSI)	Tanou et al. (2012)	Citrus leaf
ATP synthase F1 (subunits α and β)	Tanou et al. (2012)	Citrus leaf
Antioxidants		
Prx IIE	Romero-Puertas et al. (2008); Lindermayr et al. (2005)	Hypersensitive response <i>Arabidopsis</i> and <i>Pseudomonas</i>
Peroxisredoxin	Tanou et al. (2012)	Citrus root
Fe-SOD	Holzmeister et al. (2014)	In vitro <i>Arabidopsis</i> peroxytrite treatment
	Tanou et al. (2012)	Citrus leaf
Dehydroascorbate reductase	Lindermayr et al. (2005)	<i>Arabidopsis</i> , GSNO and NO treatment
Monodehydroascorbate reductase	Tanou et al. (2012)	Citrus root

(continued)

Table 3 (continued)

Protein	Reference	Organism and treatment
<i>Others</i>		
Putative translation elongation factor EF-Tu precursor, chloroplast	Romero-Puertas et al. (2008)	Hypersensitive response <i>Arabidopsis</i> and <i>Pseudomonas</i>
Aldolase	Kato et al. (2013)	Potato GSNO treatment
Cysteine synthase 1	Kato et al. (2013)	Potato GSNO treatment
Transketolase	Tanou et al. (2012)	Citrus leaf
HSP 70	Tanou et al. (2012)	Citrus leaf
4-Hydroxy-3-methylbut-2-en-1-yl diphosphate synthase	Tanou et al. (2012)	Citrus leaf
Chaperonin 60 (subunits α and β)	Tanou et al. (2012)	Citrus leaf
Glutamine synthase	Tanou et al. (2012)	Citrus leaf
NAD dependent epimerase	Tanou et al. (2012)	Citrus root
Carbonic anhydrase	Tanou et al. (2012)	Citrus root
Major latex protein	Tanou et al. (2012)	Citrus root

6 ROS/RNS Cross Talk

NO and H₂O₂ have been reported to act synergically in a dose-dependent manner during plant pathogen interaction (Delledonne et al. 2001; Zaninotto et al. 2006). Moreover, it has been reported that ROS activate NO biosynthesis and vice versa and that these reactive species collaborate to induce PCD (de Pinto et al. 2012). In fact, an increase in *S*-nitrosylating agents like GSNO and NO has been shown during cell death in tobacco TBY-2 cells after oxidative H₂O₂ treatments (de Pinto et al. 2013, Ortiz-Espín et al. 2015). In this sense, as referred above, protein PTMs are emerging as a key component of the signaling cross talk in cellular metabolism, and some examples are described below to illustrate this.

The *S*-nitrosylation of several phosphatases and kinases provokes changes in phosphorylation status (Hess and Stamler 2012), and the oxidative stress also influences protein succinylation and acetylation (Zhou et al. 2017) although the functional significance of these PTMs remains unclear. Another example is the regulation of the H₂O₂ scavenger 2-Cys Prx by NTRC/Trx system in chloroplasts (Kirchsteiger et al. 2012). Also PrxIIE has been described as able to detoxify ONOO⁻, and in this way it can interfere with Tyr kinase signaling (Romero-Puertas et al. 2007), while *S*-nitrosylation inhibited the peroxidase function of this PrxIIE, interfering in ROS detoxification and signaling (Fig. 3, upper part). In fact chloroplast PrxIIE has been reported among the proteins specifically *S*-nitrosylated during the plant immune response (Lindermayr et al. 2005; Romero-Puertas et al. 2007) and in induced leaf death by H₂O₂ treatment in wild rice and *noe1* rice mutant (Lin et al. 2012). Elsewhere, Tyr nitration has also been reported to prevent Tyr phosphorylation, affecting this important regulatory mechanism (Galetskiy et al. 2011b). Thus,

PTMs mediated by NO are crucial components in ROS/RNS cross talk that regulate the transduction of these species during stress response. In animal systems, TR/Trxs are involved in protein *S*-denitrosylation, e.g., caspase-3 (Sengupta and Holmgren 2013), where Trx is found as an *S*-nitrosylated intermediate. However, in plants, little is known about this capacity. In fact, denitrosylation/*trans*-nitrosylation is an additional function for Trx that is important in regulating apoptotic processes in animal systems (Sun et al. 2013). *S*-nitrosylation of chloroplastic Trxs in plants has scarcely been reported: Trxm5 was found to be modified in a *nitric oxide excess1* (*noe*) mutants and not in WT plants and also in *Brassica juncea* seedlings under cold stress (Lindermayr et al. 2005; Sehrawat and Deswal 2014). However cytosolic Trx and GSNO have been reported as being involved in NPR1 (non-expressor of pathogenesis-related gene 1) translocation to the nucleus in *Arabidopsis* (Tada et al. 2008; Lindermayr et al. 2010), although the mechanism is not completely elucidated. Also the involvement of cytosolic Trxh5 as a denitrosylase of different proteins including TGA1 (GACG motif binding factor 1, a basic leucine zipper (bZIP) protein) has been described by Kneeshaw et al. (2014) in the immune response. All these examples may represent a link between redox changes and gene regulation provoked by pathogens (Maldonado-Alconada et al. 2011).

Cross talk between oxidative and nitrosative stress can also be shown through the carbonylation and *S*-nitrosylation of several proteins including Prx under salinity (Tanou et al. 2009) in which *S*-nitrosylation can prevent the irreversible protein oxidation. Also mitochondrial Prx IIF was found as *S*-nitrosylated in a time-dependent manner, probably associated with increased NO contents in the organelle under salt stress (Camejo et al. 2013). Later on, we described the functional switch of the *S*-nitrosylated protein from peroxidase to *trans*-nitrosylase (Camejo et al. 2015), as a new function of Prx involved in the cross talk of the ROS/RNS in plants (Calderón et al. 2017b).

7 Future Perspectives

In chloroplasts, the signaling function of ROS is related to the thiol redox regulatory network in which redox proteins peroxiredoxin and thioredoxin act as redox sensors and transmitters. It is now accepted that NO also has a key role to play in signaling in plant cells. However, there is a substantial lack of information on the intricacies of ROS and RNS signaling, which hinders determining its regulatory role in physiological processes and during the abiotic stress response. In this sense, the advances in cellular imaging techniques and real-time detection tools to measure localized ROS and NO production would substantially enhance our understanding of ROS/RNS signaling in chloroplasts. The sources of NO in plants have been difficult to determine, and there is an important debate about how to exactly identify NO produced in plant cells. Thus, at present, nitric oxide research basically centers on the characterization of a NOS enzyme and on deciphering the importance of each of the enzymes and sources involved in the physiological production of NO. These challenges are important and complex because some previous evidence has indicated that the importance of each of

these sources to the physiological production of NO will, likely, depend on the species, the cells/tissues, the environment under which the plants are grown, and of course, the signaling pathways active under those specific conditions.

The recently developed experimental tools have enabled the identification of a range of PTMs in chloroplast proteins. Our knowledge about protein redox regulation on photosynthetic reactions and antioxidant defense has considerably increased in the last decade, and there has been important progress in the signaling role of the redox state of thiols in Trxs and Prxs through PTMs, although the regulation of most of the metabolic pathways in the chloroplast is poorly understood. Because a specific amino acid residue can be targeted by different PTM types (e.g., Cys sulfenylation or *S*-glutathionylation, *S*-nitrosylation/denitrosylation), many challenges remain regarding thiol specificity. Moreover, ROS/NO interaction can result in Tyr nitration. These PTMs may have either antagonistic or cooperative effects; thus, understanding when and how they are coordinated to allow specific proteins to respond and their repercussion in the regulation of various metabolic pathways in chloroplasts during normal physiological processes, as well as under stressful environmental conditions, is a challenging task.

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