Chapter 1
Reactive Nitrogen Species and Nitric Oxide

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Abstract Free radical nitric oxide (NO) is a biological messenger with diverse functions in plant physiology, including in stress physiology. Together with NO, related molecules called reactive nitrogen species (RNS), e.g. peroxynitrite or *S*-nitrosothiols, are associated with plant metabolism under both physiological and stress conditions. These molecules are able to react with wide spectrum of biomolecules, and they may act as a transporters and reservoirs for NO in a broad range of plant cell signalling affairs. It is possible that some of these reactions, nitration and *S*-nitrosylation, have the same importance as phosphorylation. In this chapter, the current recognition of both the properties, chemical reactions and physiological roles of NO and reactive nitrogen species in plants is reviewed.

Keywords Nitration · Nitrosothiols · Peroxynitrite · *S*-nitrosylation

1.1 Introduction

Originally, nitric oxide (NO) was recognized as a component of environmentally polluting NO_x complex (NO_2 and NO) from combustion processes such as fossil fuel burning or automobile combustion engines. Surprisingly, in 1998, the Nobel Assembly awarded the Nobel Prize in Medicine and Physiology to Robert Furchgott, Louis Ignarro and Ferid Murad for their discoveries concerning NO as a signalling molecule in the cardiovascular system. Since then, NO has become one of the most vigorously researched molecules of biological chemistry. Now, it is well known that this bioactive molecule is involved in many animal physiological processes, such as vasorelaxation, platelet inhibition, neurotransmission,

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cytotoxicity, smooth muscle contraction and relaxation, egg fertilization, immunoregulation and apoptosis (Schmidt and Walter 1994; Stamler 1994; Jeffrey and Snyder 1995; Lloyd-Jones and Bloch 1996; Wink and Mitchell 1998; Gonzales-Zulueta et al. 2000; Hess et al. 2005; Siddiqui et al. 2011).

With the finding of these roles of NO in animal cells, various studies have reported its presence in the plant kingdom and its diverse function in plant cells. As the list of physiological functions of NO has been growing, it has become evident that our knowledge of the pathways and molecular mechanisms responsible for NO effects is poorly understood (Astier et al. 2012). In addition, the term reactive nitrogen species (RNS) was introduced to designate other NO-related molecules, such as *S*-nitrosothiols, *S*-nitrosoglutathione, peroxynitrite, among others, which have relevant roles in multiple physiological processes in animal and plant cells (Halliwell and Gutteridge 2007). With the aim of promoting interest of plant biologists in NO, the following chapter will endeavour to summarize current recognition of both properties and physiological roles of NO and RNS in plants.

1.2 Nitric Oxide

1.2.1 Properties of Nitric Oxide

NO or nitrogen monoxide (systematic name) is a gaseous free radical, existing—due to its ability to adopt an energetically more favourable electron structure either by gaining or by losing an electron—in three redox-related species: nitric oxide radical ($NO^{\cdot}$), nitrosonium cation (NO^{+}) and nitroxyl anion (NO^{-}) (Stamler et al. 1992; Wojtaszek 2000). It is one of the smallest diatomic molecules which contains an unpaired electron in its π_2 orbital but remains uncharged. It is soluble in water (0.047 cm^3/cm^3 H_2O at 20 °C, 1 atm), with increasing solubility in the presence of ferrous salts (Anderson and Mansfield 1979; Neill et al. 2003). NO may not only easily migrate in the hydrophilic regions of the cell, such as the cytoplasm, but also due to its lipophilic character also freely diffuse through the lipid phase of membranes without the aid of specific membrane transporters (Arasimowicz and Floryszak-Wieczorek 2007) at a rate of 50 μm per second (Corpas et al. 2010).

Being the reactive free radical, it has a relatively short half-life which is estimated to be <6 s (Bethke et al. 2004). However, its half-life is dependent on many factors. For example, at low concentrations (<1 μmol l^{-1}), NO can have half-life of minutes to hours and could thus diffuse over several cell layers or over longer distances in intercellular spaces. At higher concentrations, NO has shorter half-life, in order of seconds (Henry et al. 1997). In addition, NO half-life depends on the local concentrations of its targets, i.e. oxygen, hydrogen peroxide, proteins,

haemoproteins, bound iron and copper, cysteine, and ascorbic acid (Stöhr and Ullrich 2002).

Due to its instable nature, NO has a very rich chemistry (Mengel et al. 2013). It reacts with molecules, which are likely to be produced temporally and spatially alongside NO (Neill et al. 2003): for example, very well documented is a simultaneous generation of NO and superoxide radical (O_2^{-}) in plant peroxisomes, mitochondria and chloroplasts (Corpas et al. 2001; del Río et al. 2006; Jasid et al. 2006; Blokhina and Fagerstedt 2010).

1.2.2 Various Roles of NO in Plant Physiology

NO first came to prominence within the context of plant defence regulation during plant–pathogen interactions (Delledonne et al. 1998; Durner et al. 1998). Now, it is well known that NO is involved in the stimulation of seed (Beligni and Lamattina 2000) and pollen (Šírová et al. 2011) germination, modulation of plant growth and development (Durner and Klessig 1999), regulation of cell elongation during primary root growth (Fernández-Marcos et al. 2012), plant maturation and senescence (Leshem et al. 1998; Guo and Crawford 2005; Wilhelmová et al. 2006), floral regulation (He et al. 2004), mediation of stomatal movement (García-Mata and Lamattina 2001; Neill et al. 2002; Guo et al. 2003; Desikan et al. 2004; Bright et al. 2006), gravitropism (Hu et al. 2005), mitochondria functionality (Zottini et al. 2002), photosynthesis regulation (Takahashi and Yamasaki 2002) or involvement of light-mediated greening (Zhang et al. 2006a). In addition, NO is involved in responses to various stresses, such as drought, salt, and heat stresses, risk element stress, disease resistance and apoptosis (Durner and Klessig 1999; García-Mata and Lamattina 2002; Zhao et al. 2004, 2007; Zhang et al. 2006b; Procházková et al. 2012). In addition, NO plays an important role in symbiotic organisms, particularly between legumes and *Sinorhizobium* (Baudouin et al. 2006). NO also acts as a regulator of gene expression at the transcriptional-level regulation of disease resistance processes (Polverari et al. 2003) and the expression of stress-related transcription factors and signalling-related kinases (Parani et al. 2004), and by the interaction with other signalling molecules such as salicylic acid and jasmonic acid (Grün et al. 2006; Lozano-Juste et al. 2011).

Recently, the differential intracellular role of NO has been described as well. For example, NO has recently been shown to modulate mitochondrial alternative oxidase activity to influence the generation of reactive oxygen species (ROS), net NO production and shift primary metabolism towards amino acid biosynthesis via inhibition of aconitase (Cvetkovska and Vanlerberghe 2012; Gupta et al. 2012). In peroxisomes, NO nitrosylates proteins such as catalase, glyoxylate oxidase and malate dehydrogenase are involved in photorespiration, β-oxidation and the detoxification of ROS (Ortega-Galisteo et al. 2012).

1.3 Peroxynitrite

1.3.1 Properties of Peroxynitrite

Reaction between NO and O_2^- results in spontaneous formation of peroxynitrite ($OONO^-$) by a diffusion-limited reaction (Huie and Padmaja 1993), as shown in Eq. 1.1.

$$ON^{\cdot} + O_2^{\cdot -} \rightarrow ONOO^- \tag{1.1}$$

The rate constant of the reaction has been determined by several methods to be within the range of 4–16 $\times$ 10^9 M^{-1} s^{-1} (Arasimowicz-Jelonek and Floryszak-Wieczorek 2011). In essence, NO and superoxide form $OONO^-$ every time they collide. No enzyme is required to produce $OONO^-$ because no enzyme can possibly catalyse any reaction as fast. NO is the only known biological molecule that reacts faster with superoxide and is produced in high enough concentrations to outcompete endogenous levels of superoxide dismutase. Consequently, the kinetics and thermodynamics of the reaction of superoxide with NO make the formation of $OONO^-$ inevitable in vivo (Pacher et al. 2007). In addition, $OONO^-$ can be also produced by an enzyme nitrate reductase in the presence of oxygen and NAD(P)H (Bethke et al. 2004). In plants, low levels of $OONO^-$ are likely to be formed continuously in photosynthesizing chloroplasts, whereas higher levels are likely to be synthesized in response to stress, which induces the production of both NO and ROS (Vandelle and Delledonne 2011).

Direct scavenging of O_2^- towards $OONO^-$ may suggest the antioxidant role of NO in relation to this ROS, since in this situation, NO may disrupt the chain of reactions leading to more toxic ROS. On the other hand, $OONO^-$, as will be mentioned later, may cause serious damage to cell structures.

$OONO^-$ is a relatively short-lived reactive nitrogen species at the physiological pH range and temperature, which may readily migrate through biological membranes and interact with target molecules also in surrounding cells within the radius of one or two cells (~5–20 μm) (Szabó et al. 2007). This molecule is surprisingly stable in solution, considering its strong oxidizing potential, and that it is 36 kcal/mol higher in energy than its isomer nitrate. The unusual stability of $ONOO^-$ results in part because it folds into a stable *cis*-conformation where the negative charge is localized over the entire molecule (Tsai et al. 1994). The molecules are further stabilized by forming strong hydrogen bonds with two or three waters (Tsai et al. 1995).

The first papers suggesting that $ONOO^-$ could be a biological oxidant were shown on animal cells in 1990 (Beckman 1990; Beckman et al. 1990). In plants, at first, the generation of $ONOO^-$ was demonstrated during plant response to biotic stress. Saito et al. (2006) observed intracellular time-dependent $ONOO^-$ production in tobacco BY-2 cells treated with INF1 elicitin secreted by *Phytophthora infestans*. Gaupels et al. (2011) found a significant accumulation of $ONOO^-$ at 3–4 h in *Arabidopsis* challenged with an avirulent *Pseudomonas syringae* pv.

tomato, which significantly increased at 7–8 h after pathogen treatment. Corpas et al. (2009b) described the generation of $ONOO^-$ also during abiotic stress: they found $ONOO^-$ production in *Arabidopsis* roots exposed to salinity stress.

At physiological pH, $ONOO^-$ equilibrates rapidly with peroxynitrous acid (HOONO) that rapidly decomposes to the highly reactive hydroxyl radical ($HO^{\cdot}$) as shown below in Eq. 1.2.

$$OONO^- + H^+ \leftrightarrow HOONO \rightarrow HO^{\cdot} + NO_2^{\cdot} \quad (1.2)$$

It has been reported that this reaction is a far more effective in producing hydroxyl radical than the Fenton reaction or the iron-catalysed Haber–Weiss reaction (Beckman et al. 1990). In biological systems, the reaction may be relevant mainly in hydrophobic phases to initiate lipid peroxidation and nitration processes (Radi et al. 1991; Szabó et al. 2007).

$ONOO^-$ interacts with proteins, lipids and DNA via direct one- or two-electron oxidation reactions or indirectly through the formation of highly reactive radicals. Surprisingly, in plants, $ONOO^-$ does not appear to be as toxic as in animal tissues (Delledonne et al. 2001). Why $ONOO^-$ is not very toxic to plant cells is still unclear. One hypothesis could be the existence of specific detoxifying mechanisms absent in animals. Among them, flavonoids that are known to display a strong antioxidant capacity attracted an attention. However, *transparent testa* mutants impaired in flavonoid biosynthesis are not susceptible to $ONOO^-$ treatment (Vandelle and Delledonne 2011).

$ONOO^-$ reacts with target molecules through two possible pathways. First, peroxynitrite anion or peroxynitrous acid can react directly with a certain target molecule in an overall second-order process (e.g. thiol oxidation). Second, peroxynitrous acid can first homolyse to form nitrogen dioxide and hydroxyl radicals, which in turn react with the target molecule. The latter processes are first order in peroxynitrite but zero order in target, because the formation of the radicals is rate-limiting. To this last type of reaction belong tyrosine nitration and lipid peroxidation (Alvarez and Radi 2003).

The main product from peroxynitrite decay in the absence of targets is nitrate (Anbar and Taube 1954), while secondary reactions of the radicals can also lead to nitrite and dioxygen, particularly at alkaline pH (Pfeiffer et al. 1997; Coddington et al. 1999; Alvarez and Radi 2003).

1.3.2 Reactions of $ONOO^-$ with Proteins

The reaction of $ONOO^-$ with proteins occurs through three possible pathways:

1. $ONOO^-$ reactions with prosthetic group, particularly transition metal centres:

   The reactions of $ONOO^-$ with transition metal centres, particularly those containing haeme and non-haeme iron, copper and manganese ions, are some of the fastest known for $ONOO^-$ (Alvarez and Radi 2003).

2. In the same way as with other Lewis acids (LA), such as the proton or carbon dioxide, the reaction proceeds to form a Lewis adduct which in turn homolyses to yield NO_2 and the corresponding oxyradical ($^{\cdot}O–LA^{-}$) (Radi et al. 2000), as shown in Eq. 1.3.

$$\begin{aligned} ONOO^{-} + LA \rightarrow ONOO - LA^{-} \rightarrow {}^{\cdot}NO_2 + {}^{\cdot}O - LA^{-} \\ \rightarrow {}^{\cdot}NO_2 + O = {}^{\cdot}LA^{-} \end{aligned} \tag{1.3}$$

3. Peroxynitrite can also oxidize reduced metal centres by two electrons yielding the oxyradical or oxo-compound accompanied by the formation of nitrite instead of nitrogen dioxide. This is particularly relevant in the case of reduced cytochrome c oxidase. In the case of the one electron oxidation of cytochrome c, which has all six coordination positions occupied, peroxynitrite reacted with the reduced but not the oxidized form, oxidizing the Fe^{2+}–Fe^{3+} possibly through an outer sphere electron transfer process (Thomson et al. 1995).

Jasid et al. (2006) showed that $ONOO^{-}$ oxidizes chloroplastic proteins and diminishes both the oxygen evolution and the fluorescence yield of photosystem (PS) II in a dose-dependent manner.

1.3.3 Reactions of $ONOO^{-}$ with Amino Acids

(a) Cysteine, methionine, tryptophan and histidine oxidation

Peroxynitrite may alter protein structure and function by reacting with various amino acids in the peptide chain. The predominant and the fastest (10^{3}–10^{7} M^{-1} s^{-1}) reaction is with cysteine (Alvarez and Radi 2003). It has been described in animal cells that $ONOO^{-}$ directly oxidizes methionine, forming methionine sulphoxide, and to a lesser extent ethylene and dimethyldisulphide (Szabó 2003). $ONOO^{-}$ can also oxidize tryptophan yielding *N*-formylkynurenine, oxindole, hydropyrroloindole and nitrotryptophan (Alvarez and Radi 2003). The physiological role of this modification, if any, is unclear (Vandelle and Delledonne 2011). The proteomic analysis of inflamed neurons has shown that several nitrotryptophan-containing proteins contain functional tryptophan residues that interact with other molecules. These proteins appear to be involved in energy metabolism, protein synthesis and stress responses, and it has been suggested that tryptophan nitration may modulate specific interactions between these proteins and their targets (Ikeda et al. 2007; Vandelle and Delledonne 2011). In plants, Galetskiy et al. identified 138 tyrosine and tryptophan nitration sites, mainly in PSI, PSII, cytochrome b6/f and ATP-synthase complex indicating that protein nitration belongs to one of the prominent posttranslational protein modifications in photosynthetic apparatus (Galetskiy et al. 2011a).

In animal cells, $ONOO^{-}$ modifies histidine through a radical mechanism, forming a histidinyl radical, a mechanism involved in the inactivation of Cu, Zn-SOD

by $ONOO^-$ (Alvarez et al. 2004). In plant cells, Gonzalez-Perez et al. (2008) revealed that the specific inhibition site of $ONOO^-$ in PSII is in the plastoquinone Q_AFe^{2+} niche of the PSII acceptor side. $ONOO^-$ interacts with the non-haeme Fe^{2+}; however, the type of the redox reaction between peroxynitrite and the non-haeme Fe^{2+} is not known. They suggested that the products of the one- or two-electron oxidation are able to oxidize or to nitrate the Fe^{2+}-coordinated histidine residues or to induce Fe^{2+} release and as a consequence to destroy the magnetic coupling between Q_A and the non-haeme Fe^{2+} (Gonzalez-Perez et al. 2008).

(b) Tyrosine nitration

Protein tyrosine nitration is a covalent protein modification resulting from the addition of a nitro ($-NO_2$) group adjacent to the hydroxyl group on the aromatic ring of tyrosine residues (Gow et al. 2004). A stable product 3-nitrotyrosine is formed by the addition of −NO2 to the ortho position of tyrosine (Dixit et al. 2009). This biochemical event induces change of the tyrosine molecule into a negatively charged hydrophilic nitrotyrosine moiety and causes a marked shift of the local pKa of the hydroxyl group from 10.07 in tyrosine to 7.50 in nitrotyrosine impinging on the protein function (Turko and Murad 2002). Tyrosine nitration is considered to be a selective process, and proteins have usually approximately 3–4 mol% of tyrosine, but only one or two of these tyrosines may become preferentially nitrated, this depending on several factors, such as protein structure, nitration mechanism and environment, where the protein is located (Bartesaghi et al. 2007; Corpas et al. 2009a). Bayden et al. (2011) suggested that despite the moderately hydrophilic nature of tyrosine, its relatively high degree of surface exposure (only 15 % of tyrosine residues are buried) and the fact that most proteins contain tyrosine (natural abundance 3.2 %), only a limited number of proteins are nitration targets and this does not depend on their abundance.

Tyrosine nitration has been shown to be capable of changing the function of a protein in several ways: (1) gain of function as well as no effect on function; and (2) inhibition of function, which is much more common result of protein tyrosine nitration (Radi 2004; Corpas et al. 2009a). For example, Alvarez et al. (2011) reported the inhibition of *Arabidopsis* O-acetylserine(thiol)lyase A1 by tyrosine nitration. It has been also demonstrated that nitration of tyrosine residue may either prevent further phosphorylation or stimulate phosphorylation (Shi et al. 2007; Rayala et al. 2007). In animal cells, nitrotyrosine has been used as a biomarker of nitrosative stress. In plants, nitrotyrosine is often used as a marker of nitrosative stress during abiotic stress in the same way as lipid peroxidation or protein carbonylation, and like this, it was used, e.g., for salinity stress (Valderrama et al. 2007) and during sunflower–mildew interaction (Chaki et al. 2009). Also under high light, which is a major stress factor often leading to over-excitation of the photosynthetic apparatus and production of ROS, tyrosine nitration was demonstrated (Galetskiy et al. 2011a).

However, evidence accumulates that this modification also has a signalling function in plant cells. For example, Cecconi et al. (2009) reported that tyrosine nitration, as a key process of redox signalling, could be involved in Rubisco

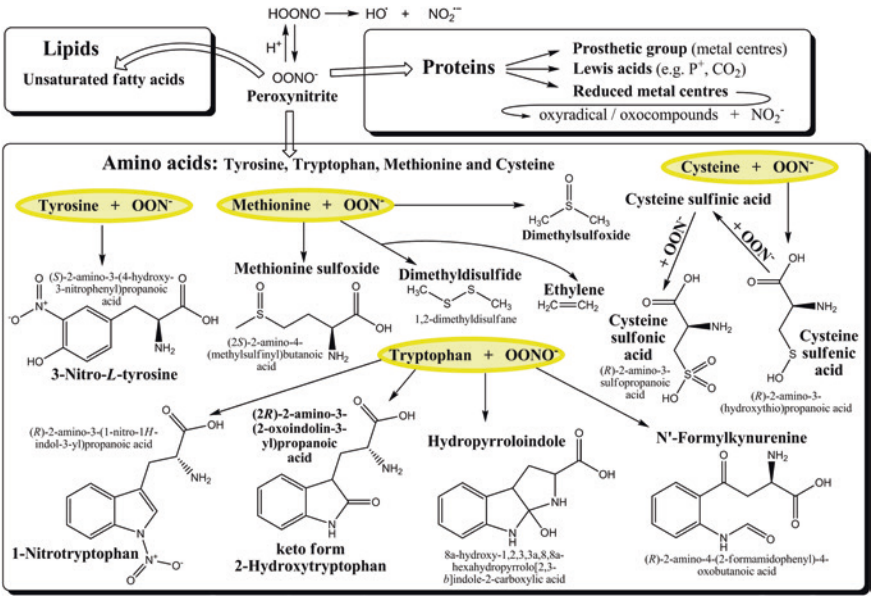

Fig. 1.1 Summary of some reactions of peroxynitrite

large subunit and Rubisco activase modulation during hypersensitive response. Similarly, Gaupels et al. (2011) proposed that $ONOO^-$ transduces the NO signal by modifying protein function via tyrosine nitration during the hypersensitive defence response. In addition, the conformation change in the stromal loop between membrane α-helices IV and V due to nitration might provide a signal for the degradation of D1 protein (Galetskiy et al. 2011b). The reactions of peroxynitrite with amino acids are described in Fig. 1.1.

1.3.4 Reactions of $ONOO^-$ with Lipids

A major aspect of peroxynitrite-dependent cytotoxicity relies on its ability to trigger lipid peroxidation in membranes (Radi et al. 1991). Exposure of low-density lipoprotein to $ONOO^-$ results in the oxidation of unsaturated fatty acids. The mechanism of initiation is unclear but may involve either abstraction of a bis-allylic hydrogen by the 'the hydroxyl radical-like' activity $ONOO^-$ or induced homolysis of $ONOO^-$ by the unsaturated fatty acid (Hogg et al. 1992).

$ONOO^-$ can also rapidly oxidize tocopherols: α-tocopherol is oxidized by two electrons to α-tocopheryl quinone, a form that is not easily repaired by cellular reductants (Hogg et al. 1993). Desel et al. (2007) found that *Brassica napus*, *Nicotiana tabacum* and *Arabidopsis thaliana*, with high levels of endogenous γ-tocopherol, produced 5-nitro-γ-tocopherol during seed germination. It has

been speculated that nitration of γ-tocopherol in plant tissues may be an important mechanism for in vivo modulation of NOx levels (Arasimowicz-Jelonek and Floryszak-Wieczorek 2011). However, research in nitrolipids is in an early stage of investigation, and there is virtually no information available regarding these two nitration processes in plant systems, this being a new area of reactive nitrogen species metabolism that needs to be explored (Corpas et al. 2009a).

1.3.5 Reactions of $ONOO^-$ with DNA

$ONOO^-$ can also react with DNA (Radi 2004). DNA can be damaged by $ONOO^-$ by both introducing oxidative modifications in nucleobases and in sugar–phosphate backbone (Pacher et al. 2007). $ONOO^-$ is able to attack the sugar–phosphate backbone by abstracting a hydrogen atom from the deoxyribose moiety, resulting in the opening of the sugar ring and the generation of DNA strand breaks (Burney et al. 1999; Niles et al. 2006; Pacher et al. 2007). Rubio et al. (1996) have also reported that treatment of deoxynucleosides with $ONOO^-$ leads to the formation of 2-thiobarbituric acid-reactive substances.

Among the nucleobases, guanine is the most reactive with $ONOO^-$ because of its low reduction potential (Yu et al. 2005). The major product of guanine oxidation is 8-oxoguanine, which further reacts with peroxynitrite, yielding cyanuric acid and oxazolone (Niles et al. 2006). Following the formation of abasic sites that can be cleaved by endonucleases in vivo, it gives DNA single-strand breaks (Burney et al. 1999). Unfortunately, similarly as in the case of nitrolipids, research in 8-nitroguanine is in an early stage of investigation in plant systems (Corpas et al. 2009a).

However, increasing studies conducted mainly under in vitro conditions have reported that $ONOO^-$ cannot only be considered as a cytotoxic agent (Altug et al. 1999) but might also act as a potent modulator of the redox regulation in various cell signal transduction pathways (Liaudet et al. 2009; Arasimowicz-Jelonek and Floryszak-Wieczorek 2011).

1.4 Nitrosothiols

The formation of nitrosothiols is still debated. The direct reaction of thiol groups with NO is too slow to occur in vivo; instead, it is assumed that N_2O_3 is the main nitrosylating species in aerobic conditions although the formation of dinitrogen trioxideis controversially discussed (Folkes and Wardman 2004; Ridnour et al. 2004). Other reactive nitrogen species described to mediate *S*-nitrosothiol formation are nitrosonium and nitroxyl ions (Ridnour et al. 2004). Nitroso groups can also be transferred between thiols in a process termed as transnitrosylation. Transnitrosylation occurs between proteins and between proteins and low molecular weight nitrosothiols (e.g.

S-nitrosylated glutathione GSNO) in animals; however, in plants, evidence for this mechanism is lacking (Hogg 2002; Nakamura and Lipton 2013).

S-nitrosothiols are generally more stable in solution compared to NO; therefore, they can participate in the transport, storage and delivery of NO and consequently contribute to posttranslational modifications involved in cell signalling and in stress processes (Foster et al. 2003; Benhar et al. 2006; Leterrier et al. 2011).

One of the families of the most abundant low molecular mass *S*-nitrosothiols is GSNO. GSNO results from the reaction between NO and reduced glutathione (GSH) in a process called *S*-nitrosylation or by a process of transnitrosation from other *S*-nitrosothiols with GSH. *S*-nitrosylation appears to take place through either the formation of N_2O_3 or the addition of NO to a glutathionyl radical formed during this reaction (Broniowska et al. 2013). *S*-nitrosylation of proteins is rapidly reversible, making it an attractive candidate for involvement in signal transduction (Grennan 2007). Increasing evidence suggests that *S*-nitrosylation plays a regulatory role in plant physiology. For example, it can play a negative regulatory role in ethylene biosynthesis via *S*-nitrosylation of S-adenosylmethionine, which is a precursor for ethylene biosynthesis (Lindermayr et al. 2006). In *Arabidopsis* leaves exposed to NO gas, 52 *S*-nitrosylated proteins, including stress and redox-related, metabolic, signalling and cytoskeletal proteins, were identified (Lindermayr et al. 2005). In addition, it was reported that the activity of peroxiredoxin IIE, which detoxifies peroxynitrite and hydrogen peroxide, was inhibited by *S*-nitrosylation (Romero-Puertas et al. 2007). Similarly, isoforms of glyceraldehyde dehydrogenase were inhibited after treatment with GSNO in *Arabidopsis* (Holtgrefe et al. 2008). In the same way, the Rubisco activity was inhibited in *Kalanchoe pinnata* (Abat et al. 2008).

GSNO may function both as an intracellular NO reservoir and as a transporter for NO throughout the cell (Singh et al. 1996) and therefore can affect the process of transnitrosation equilibrium between GSNO and *S*-nitrosylated proteins. In this sense, it has been proposed a mechanism of GSNO formation mediated by cytochrome *c* (Basu et al. 2010; Leterrier et al. 2011).

GSNO seems to be an important molecule during plant responses to various abiotic and biotic stresses. For example, under treatment of heavy metals, GSNO content decreased in pea and *Arabidopsis* (Barroso et al. 2006; Leterrier et al. 2012). On the other hand, its content increased under high temperature in sunflower (Chaki et al. 2011a) and after mechanical wounding in sunflower and *Arabidopsis* (Chaki et al. 2011b; Espunya et al. 2012).

The key enzyme regulating GSNO pools is nitrosoglutathione reductase (GSNOR). GSNOR reduces GSNO to ultimately produce glutathione disulphide and ammonia (Mur et al. 2012). Because of the ubiquitous nature of GSNOR, it has been suggested that this enzyme serves more to protect against nitrosative stress than as cell signalling factor (Lindermayr and Durner 2009).

The family of low molecular mass *S*-nitrosothiols includes other molecules such as *S*-nitrosocysteine and *S*-nitrosocysteinylglycine.

Most proteins possess cysteine residues, but the affinity of this amino acid residue to NO can be very different (Stamler et al. 1997). The vicinity of acid

and base catalysts may help in *S*-nitrosylation formation, although this does not account for all the *S*-nitrosylation sites already founded. Inspection of known *S*-nitrosylated proteins revealed that the presence of a hydrophobic environment, which enables the formation of *S*-nitrosylating species via the reaction between oxygen and NO, also promotes *S*-nitrosylation (Stamler et al. 2001).

Linking nitrosothiol on cysteine residues mediates NO signalling functions of a broad spectrum of mammalian proteins, including caspases, the main effectors of apoptosis. Plant metacaspases can be kept inactive through *S*-nitrosylation of a critical cysteine residue but are insensitive to *S*-nitrosylation when matured (Belenghi et al. 2007). This discrepancy could be related to differences in NO householding between plants and mammals. Whereas mammals control internal NO levels very strictly by fine regulation of activities of the various NO synthase isoforms, plants must deal with atmospheric NO as well as with internal leakage of NO that accumulates under physiological growth conditions due to its production from nitrite (Belenghi et al. 2007).

In addition, there is another group of SNOs called high molecular mass SNOs which are produced by NO binding to sulfhydryl (–SH) groups present in specific cysteine residues of proteins (Corpas et al. 2013).

1.5 Conclusion

Research in the field of NO in plant systems is a challenge; however, it is evident that these researches lag behind the research in the animal system. For example, there is a considerable lack of knowledge regarding, e.g., transnitrosylation, signalling functions of various RNS and explanation of a lesser $ONOO^-$ toxicity comparing to animal cells. Hence, additional research is necessary to explain all these doubts.

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