REVIEW

Nitric oxide as a key component in hormone-regulated processes

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Abstract Nitric oxide (NO) is a small gaseous molecule, with a free radical nature that allows it to participate in a wide spectrum of biologically important reactions. NO is an endogenous product in plants, where different biosynthetic pathways have been proposed. First known in animals as a signaling molecule in cardiovascular and nervous systems, it has turned up to be an essential component for a wide variety of hormone-regulated processes in plants. Adaptation of plants to a changing environment involves a panoply of processes, which include the control of CO_2 fixation and water loss through stomatal closure, rearrangements of root architecture as well as growth restriction. The regulation of these processes requires the concerted action of several phytohormones, as well as the participation of the ubiquitous molecule NO. This review analyzes the role of NO in relation to the signaling pathways involved in stomatal movement, plant growth and senescence, in the frame of its interaction with abscisic acid, auxins, gibberellins, and ethylene.

Keywords Abscisic acid · Auxins · Ethylene · Gibberellins · Nitric oxide

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Introduction

For many years, nitric oxide (NO) was identified as a component of polluted air and blamed for exerting toxic effects over photosynthesis and plant growth (Clyde Hill and Bennett 1970). Plants can itself produce NO, in addition to respond to that offered by the environment, as it was first observed in soybean (Klepper 1979).

Two centuries after its discovery in 1772, NO burst onto the scene associated with important functions in vasculature and nervous system in animals (Culotta and Koshland 1992). The identification of biological targets and functions of NO in mammals led to investigate whether or not NO was implicated in physiological processes that take place in plants. It was soon described that NO participates in maturation and senescence, in addition to being an essential component in plant responses against biotic stress (Delledonne et al. 1998; Durner et al. 1998; Leshem et al. 1998). Since then, it has become evident that reactive intermediates and free radicals, formerly considered toxic compounds, constitute in fact important endogenous signals in cellular metabolism (Delledonne et al. 1998; Durner et al. 1998; Crawford 2006; Foyer and Noctor 2009), and that they participate in the modulation of hormonal responses (Lamattina et al. 2003; Wendehenne et al. 2004; Bartoli et al. 2012) regulating plant growth and developmental processes (Correa-Aragunde et al. 2007).

NO in a biological environment: target molecules and effects

NO is a small, uncharged, scarcely polar molecule, thus freely diffuses across membranes from one compartment to the other. From a chemical point of view it is a free radical, with an unpaired electron which makes it paramagnetic and allows its detection by employing electron paramagnetic resonance (Puntarulo et al. 2010).

In biological systems, NO has a low steady state concentration determined by the rates of synthesis and decay reactions. In hydrophobic environments, such as biomembranes, NO reacts with radical species, acting as antioxidant and breaking lipid peroxidation processes (Rubbo et al. 2000; O'Donnell and Freeman 2001).

Biologically important reactions of NO involve those with, (a) transition metal ions, including those found in metalloproteins, (b) free radicals, such as superoxide anion (O_2^-) and intermediates in lipid peroxidation, and (c) thiols groups present in proteins and peptides (Fig. 1).

Regarding the reactions between NO and transition metals, they constitute an important mechanism to modulate the activity of proteins, being the activation of soluble guanylate cyclase (sGC) the most studied example (Ignarro et al. 1984). Research performed in plants showed that NO induces a transient increase in endogenous cGMP concentrations (Durner et al. 1998; Neill et al. 2003). In addition, inhibitors of sGC block the NO-induced activation of both the expression of phenylalanine ammonia-lyase (PAL) genes and enzyme activity (Durner et al. 1998). Auxin responses during the adventitious root formation include NO/cGMP signaling as it is discussed later.

Other regulatory properties of NO which involve posttranslational modification of proteins are exerted through *S*-nitrosylation (also named as *S*-nitrosation) of thiol groups and nitration of tyrosine residues (Table 1). As a whole, NO-mediated protein modifications constitute the basis for NO signaling.

The reaction between NO and O_2^-, is among the fastest biological reactions ($k = 4$–16×10^9 M^{-1} s^{-1}, Huie and Padmaja 1993), and leads to the formation of a short-lived strong oxidant, peroxynitrite (Ischiropoulos et al. 1992). This reaction establishes a link between reactive oxygen and nitrogen species metabolism (Wink and Mitchell 1998). In plants, the simultaneous generation of O_2^- and NO has a synergistically function in defense responses (Asai et al. 2008), as well as in plants exposed to abiotic stress (Tanou et al. 2009).

Nitration, consisting in the addition of a nitro group (–NO_2) into a protein tyrosine residue, can alter protein function and is considered a molecular footprint of peroxynitrite presence in cells (Radi 2004). Detection of nitrated proteins has been reported in several species under
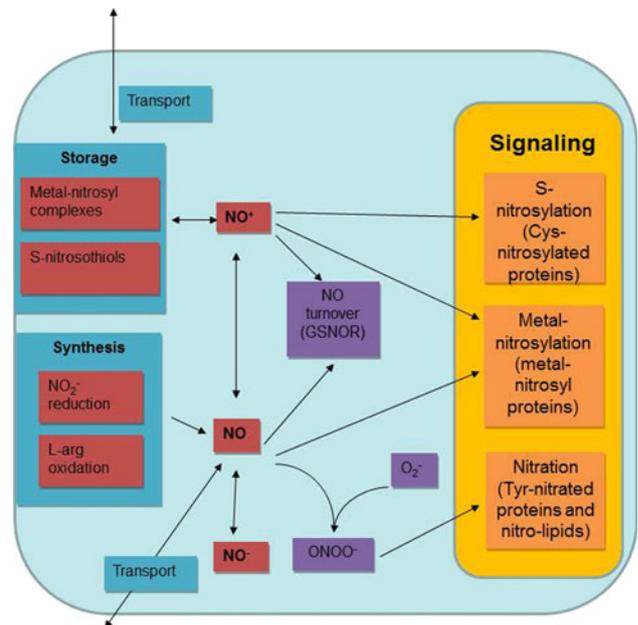

Fig. 1 NO metabolism and targets. NO steady state concentration in cells is determined by its production (diffusion from external sources, synthesis and release from storage compounds) and consumption (chemical reactions, GSNOR activity, diffusion). The broader chemistry of NO involves two interrelated redox forms, apart from NO radical: nitrosonium cation (NO$^+$) and nitroxyl anion (NO$^-$), which can be found in living systems (Hughes 1999). Low molecular iron-nitrosyl complexes and S-nitrosothiols are proposed as NO$^+$ carriers and donors in biological systems (Ramirez et al. 2011) buffering the concentration of free NO (Stamler et al. 1992). The enzyme GSNOR metabolizes GSNO to glutathione sulphinamide as main product (Jensen et al. 1998). Regulatory effects of NO are mediated by reversible Cys-nitrosylation of proteins (e.g., auxin receptor TIR1, and ion channel proteins), metal nitrosylation of enzymes (e.g., activation of sGC), and Tyr-nitration of proteins (e.g., cytochrome *c*, and Mn-SOD). A signaling role for nitroalkenes and nitroalkene thiol adducts on transcription factors has been proposed in animals (Rubbo and Radi 2008)

physiological or stress conditions (Morot-Gaudry-Talarmain et al. 2002; Saito et al. 2006; Romero-Puertas et al. 2007; Bechtold et al. 2009; Chaki et al. 2009; Jasid et al. 2009). Among the proteins prone to be modified by nitration Rubisco activase, ATP synthase subunit α, and glutamine synthetase 2 in pathogen-challenged Arabidopsis have been described (Cecconi et al. 2009; Lozano-Juste et al. 2011). In chloroplasts, tyrosine nitration sites have been also identified, mainly in PSI, PSII, cytochrome b6/f and ATP synthase complex (Galetskiy et al. 2011).

As mentioned above, other important mechanism through which NO exerts its effects is by means of S-nitrosylation of sulfhydryl centers (Lamotte et al. 2006). The NO carrier S-nitrosoglutathione (GSNO) is an excellent nitrosating agent, which can transfer the NO$^+$ group to a variety of nucleophiles (Hughes 1999; Stamler et al. 2001). The level of GSNO in vivo depends on both NO synthesis

Table 1 Examples of NO-related post-translational modification in plant proteins

Protein	Description	References
Glyceraldehyde-3-phosphate dehydrogenase	*S*-nitrosylation *A. thaliana* Reversible inhibition	Lyndermayr et al. (2005)
Methionine adenosyltransferase (MAT)	S-nitrosylation *A. thaliana* Inhibition of activity	Lyndermayr et al. (2006)
Ribulose 1,5 bisphophate carboxylase/oxygenase RUBISCO	S-nitrosylation *B. juncea* and *K. pinnata* Inhibition of activity	Abat and Deswal (2009); Abat et al. (2008)
Auxin receptor TIR1/AFB	S-nitrosylation *A. thaliana* Enhances TIR1-Aux/IAA interaction	Terrile et al. (2012)
Salicylic acid-binding protein AtSABP3	S-nitrosylation *A. thaliana* Inhibition of activity and SA binding	Wang et al. (2009)
Glycolate oxidase	S-nitrosylation *P. sativum* Peroxisomes exposed to abiotic stress	Ortega-Galisteo et al. (2012)
Dehydroascorbate reductase	S-nitrosylation *Solanum tuberosum* Inhibition of activity	Kato et al. (2012)
Ferredoxin–NADP reductase	Tyr-nitration *Helianthus annuus* Inhibition of activity	Chaki et al. (2011)
Methionine synthase	Tyr-nitration Arabidopsis Inhibition of activity	Lozano-Juste et al. (2011)
Glyceraldehyde-3-phosphate dehydrogenase	Tyr-nitration *A. thaliana* Inhibition of activity	Lozano-Juste et al. (2011)
NADP-isocitrate dehydrogenase	Tyr-nitration *P. sativum* Inhibition of activity	Begara-Morales et al. (2013)
PSI and PSII proteins	Tyr-nitration *A. thaliana* Regulate the stability and turnover	Galetski et al. (2011)

and GSNO turnover, which is performed by the activity of the enzyme nitrosoglutathione reductase (GSNOR) (Barroso et al. 2006). Thus, GSNOR activity regulates the global level of S-nitrosylation in plants (Feechan et al. 2005; Malik et al. 2011).

Studies on S-nitrosylated proteins in *Arabidopsis thaliana* leaves and tobacco cells suggested that S-nitrosylation is a specific and regulated event (Lyndermayr et al. 2005; Lyndermayr and Durner 2009; Astier et al. 2011; 2012). S-nitrosylation of the auxin receptor TIR1 imposed by NO constitute a relevant example of post-translational

modification with high impact in biological functions (Terrile et al. 2012).

Some effects of NO in plant cells are exerted through the second messenger Ca^{2+} (Gould et al. 2003). Changes in free cytosolic Ca^{2+}, and its release from specific intracellular stores, induced by NO have been described in different plant species exposed to elicitors or treated with exogenous NO (Lecourieux et al. 2002). In tobacco cells, treatment with cryptogein or the NO donor diethylamine NONOate promoted an increase in cytosolic free Ca^{2+} concentration, involving phosphorylation events (Lamotte

et al. 2004). In this line of evidence, it was also observed in *N. plumbaginifolia* that NO induces an increase of Ca^{2+} through the activation of protein kinases (Lamotte et al. 2006). Interestingly, Calcium and calcium-dependent protein kinases (CDPKs) are involved in the auxin-NO crosstalk regulation of adventitious root formation (Lanteri et al. 2006). In addition, ABA-mediated stomatal closure includes NO-dependent Ca^{2+} mobilization as it is discussed later.

Finally, the pattern of gene expression can be affected by NO level in plants (Grün et al. 2006). In *A. thaliana,* changes in the expression profiles were analyzed following infiltration with the NO donor sodium nitroprusside (SNP). More than 100 genes showed altered expression patterns with SNP treatment, which belong to the functional categories of signal transduction, defense or cell death, reactive oxygen species (ROS) generation and removal, photosynthetic processes, cellular trafficking, and basic metabolism (Polverari et al. 2003). Other studies disclosed that NO-regulated genes in plants include enzymes involved in jasmonic acid (JA) and ethylene synthesis, enzymes of phenylpropanoid synthesis, auxin-responsive proteins as well as enzymes involved in glutathione synthesis (Wendehenne et al. 2004; Innocenti et al. 2007). Interestingly, a microarray analysis performed for Arabidopsis plants exposed to the NO donor SNP indicated that 10 % of genes that respond to NO treatment are transcription factors (Parani et al. 2004). As mentioned above, NO likely exerts its effects on gene expression imposing post-traslational modifications of transcription factors, regulatory proteins or nuclear proteins through S-nitrosylation of cysteine residues, tyrosine nitration, and metal nitrosylation (Lyndermayr et al. 2005; Grün et al. 2006; Serpa et al. 2007; Corpas et al. 2010).

A new biochemical pathway for NO catabolism was described based on the reaction with the plant hormone cytokinin. In addition, this finding represents a novel mechanism for NO-hormone interaction (Liu et al. 2013).

In the following sections, we describe some examples highlighting the role of NO as a mediator in hormone-regulated processes in plants (Fig. 2). Likely they represent only a small portion of the processes that take place in higher plants, particularly when they are exposed to environmental challenges, involving a cross-talk between NO and hormones. The selected examples describe events with great biological importance.

ABA says NO to stomatal opening and plant water loss

Stomata are pores formed by two differentiated cells named guard cells located in the epidermis of terrestrial plants. The opening of the stomatal pore is finely regulated
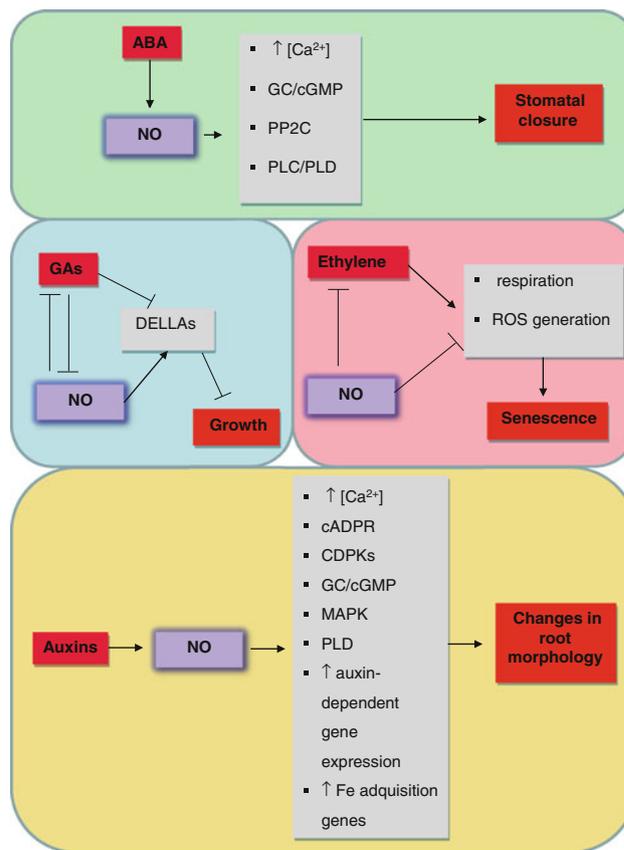

Fig. 2 NO and hormone-regulated processes. Schematic representation of NO acting as a regulatory molecule in ABA, GAs, ethylene and auxin-mediated processes. ABA increases NO level in guard cells and several targets or effectors downstream NO were identified in ABA-mediated stomatal closure. An interplay between NO and GAs through DELLAs pathway is currently suggested. Ethylene and NO exert opposite effects over senescence, as well as postharvest conservation of plant products. In roots auxins presence led to an increase in NO levels, and both NR and NOS-like activities were described as NO sources (Abu-Abied et al. 2012; Jin et al. 2011). Several effectors acting downstream NO were identified mediating changes in root architecture. *ABA* abscisic acid, *cADRP* cyclic ADP-ribose, *CDPKs* calcium-dependent protein kinases, *cGMP* cyclic guanosine monophosphate, *GAs*, gibberellins, *MAPK* mitogen-activated protein kinase, *NO* nitric oxide, *NOS* nitric oxide synthase, *NR* nitrate reductase, *PLC* phospholipase C, *PLD* phospholipase D, *PP2C* protein phosphatase 2C, *ROS* reactive oxygen species, *sGC* soluble guanylate cyclase

to facilitate CO_2 uptake, for CO_2 fixation during photosynthesis and to avoid transpirational water loss.

Therefore, optimal photosynthesis and water management by plants rely on an exquisite integration of several inputs linking signaling molecules and second messengers coming from different, circumstantial, and dynamical plant requirements.

Environmental parameters such as light, CO_2 levels, humidity, soil water status and biotic stresses regulate stomatal aperture (Assmann 1993; Hetherington and

Woodward 2003; Schroeder et al. 2001). In addition, endogenous stimuli under the control of plant hormones influence the stomatal aperture. The size of the stomatal pore depends on variations of the turgor that results in volume changes of the guard cells (Blatt 2000). Guard cells lack plasmodesmata; therefore, transport across plasma membrane and the modulation of ion channel activity play key roles determining the entrance and sorting of osmotically active solutes (Blatt 2000; Schroeder et al. 2001).

Stomatal guard cells are acknowledged as a model system for the study of signal transduction in plants. The best-characterized response is that generated by the phytohormone ABA. When ABA molecules reach guard cell, the surface is internalized by the ABC transporter At ABCG40 (Kang et al. 2010). Once sensed by the PYR/PYL/RCAR receptor, ABA triggers a complex signaling network that results in a reduction of the guard cell turgor pressure and the closure of the stomatal pore (Blatt 2000; Schroeder et al. 2001). Some of the key steps in this network are: (a) the inactivation of plasma membrante H^+-ATPase, (b) cytoplasmatic Ca^{2+} increases, and (c) plasma membrane anion channel activation (both rapid and slow anion channels) (Kim et al. 2010). Anion efflux generates and maintains a depolarization of the guard cell plasma membrane which in turn promotes K^+ efflux through the activation of the outward rectifying K^+ channels and reduces K^+ influx through the deactivation of inward-rectifying K^+ channels, resulting in a net loss of solutes and the concomitant reduction of turgor pressure and stomatal closure (Kim et al. 2010; Dreyer and Uozumi 2011).

The first report on NO regulation of stomatal movement showed that the NO donor SNP-induced stomatal closure in three different plant species *Vicia faba*, *Salpichroa organifolia*, and *Tradescantia* spp. (García-Mata and Lamattina 2001). Soon after, NO was linked to guard cells ABA-dependent signaling network. Two independent works used the NO-detection fluorescent probe DAF-2 DA to show that ABA induces endogenous NO production in *Pisum sativum* and *Vicia faba* (García-Mata and Lamattina 2002; Neill et al. 2002). It was also demonstrated that the NO scavenger cPTIO blocks ABA-dependent stomatal closure, supporting that NO is required for the ABA-regulated signaling pathway leading to stomatal closure (García-Mata and Lamattina 2002; Neill et al. 2002). Further studies showed that AtrboHD and AtrboHF-mediated ROS production, recognized as an early event in ABA-signaling networks, are required for endogenous NO production (Bright et al. 2006) indicating that ROS are upstream of NO in ABA-dependent stomatal closure.

Several targets of NO have been identified within ABA-regulated guard cell signaling. It has been reported that NO deactivates inward-rectifying K^+ channels and activates anion channels, contributing to the loss of the turgor

pressure that precedes stomatal closure (García-Mata et al. 2003). Both of these ion channels are Ca^{2+} dependent. In this context, it has been also demonstrated that NO increases guard cell cytoplasmic Ca^{2+} concentration, in a GC/cGMP-dependent manner via Ca^{2+} release from intracellular Ca^{2+} stores (García-Mata et al. 2003). Later it was shown that the Ca^{2+} insensitive outward rectifying Ca^{2+} channel GORK was also regulated by NO, possible via the nitrosylation of the channel protein (Sokolovski and Blatt 2004).

Another effector pointed out to interact with NO is the protein phosphatase 2C (PPC2), which binds and stabilizes the ABA-PYL/PYR/RCAR complex (Fujii et al. 2009; Ma et al. 2009; Park et al. 2009; Santiago et al. 2009). It was shown that, even though PP2C mutants *abi1-1* and *abi2-1* produced NO in response to ABA, they do not close the stomata in response to NO, indicating that PP2C might be downstream of NO (Desikan et al. 2002). It was recently reported that NO interaction with ABI1 was also mediated by the GC/cGMP pathway (Dubovskaya et al. 2011).

Another component reported to bind to and regulate ABI1 is the phospholipid signal phosphatidic acid (PA) (Jacob et al. 1999). PA is rapidly formed in response to drought stress and it can be synthesized through two independent ways: (a) from the hydrolysis of phosphatidylinositol 4,5-biphosphate (PIP2) in a reaction catalyzed by phospholipase C (PLC), or (b) by phospholipase D (PLD) which hydrolyzes structural phospholipids, such as phosphatidilcholyne to PA and cholyne (Distéfano et al. 2008). ABA activates both PLC and PLD (Jacob et al. 1999; Staxen et al. 1999). In Arabidopsis guard cells, NO induces the activity of both PLC and PLD, and the inhibition of both enzymes resulted in a reduction of ABA-dependent stomatal closure (Distéfano et al. 2008; 2012).

As was reported, ABA induces stomatal closure and inhibits light-induced stomatal opening via two separate signaling processes (Schroeder et al. 2001). Interestingly, it was demonstrated that NO is required in ABA signaling both for stomatal closure induction and for inhibition of light-induced stomatal opening (García-Mata and Lamattina 2007; Zhang et al. 2007).

Despite the responses generated by ABA are the most studied processes in guard cells, other hormones have been reported to regulate stomatal movement under different physiological conditions. Such is the case of ethylene (Liu et al. 2010), methyl jasmonate (MeJA) (Saito et al. 2009), cytokinins (Xiao-Ping and Xi-Gui 2006), auxins (Xiao-Ping and Xi-Gui 2006), and salicylic acid (Hao et al. 2010). Interestingly, NO seems to be a common second messenger participating in all hormone-regulated signaling pathways controlling stomatal aperture processes and plant gas exchange with the environment.

Interactions between NO and gibberellins: the DELLAs pathway

A main role of plant hormones is to contribute to the integration of the multiple exogenous and endogenous signals perceived by plants. Integration of those signals into a common pathway allows a coordinated growth and developmental response, which is particularly relevant when plants are exposed to environmental stresses and it was undoubtedly a key element for the evolutionary success of vascular plants in early land colonization. Research performed over the last 15 years disclosed a pivotal role of DELLA proteins, a subfamily of likely nuclear transcriptional regulators, in the integration of hormonal responses (Harberd et al. 2009; Sun 2011). Some mutant versions of these proteins display an altered function that confer dwarf or semidwarf phenotypes, being the later critical to increase grain production during the green revolution (Hedden 2003). In turn, plants carrying disruptions in DELLA coding genes display a slender phenotype. Detailed studies performed with Arabidopsis and crop plants indicate that these proteins have the capacity to repress plant growth. The growth restriction imposed by them is commonly relieved by the action of gibberellins (GAs) which, by interacting with GID1 receptors, bind DELLAs and allow the recruitment of the GA-GID1-DELLA module to the E3 ubiquitin ligase SLY1, thus favoring DELLAs degradation at the proteasome and subsequently promoting growth (Peng et al. 1999; Fu et al. 2002; Ueguchi-Tanaka et al. 2005).

Several lines of evidence suggest a possible interplay between GAs and NO. Both regulators are known to influence to a large list of common processes, among them: hypocotyl elongation and plant responses to light (Peng et al. 1999; Beligni and Lamattina 2000; Fu et al. 2004), primary root growth (Correa-Aragunde et al. 2004; Fu and Harberd 2003; Negi et al. 2010; Fernández-Marcos et al. 2011) as well as responses to several stresses, particularly salinity (Achard et al. 2006; Bai et al. 2011). Some of these responses also involve the interaction with other hormones, which is clearly shown with auxins in the control of primary root growth (Fu and Harberd, 2003). Both DELLAs and NO modulate the antioxidant response of plants (Achard et al. 2008; Bai et al. 2011; Moriconi et al. 2012). Interestingly, NO and GAs exert opposite effects in several, but not all, of the physiological processes in which they participate, suggesting that there is some degree of antagonism between them. Indirect evidence suggesting that such antagonism could involve a differential effect of NO and GAs on the DELLAs pathway was obtained early for *Hordeum vulgare* aleurone layers. In this tissue, GAs-induced programmed cell death (PCD) was delayed by the addition of the NO donors SNP and *S*-nitroso-*N*-acetyl-DL-

penicillamine (SNAP) (Beligni et al. 2002). It was also showed that in this plant species the expression of GAM-YB, a GA-regulated transcriptional activator of α-amylase expression was induced by GAs through the release of the repression imposed by SLN1, the barley DELLAs homologous protein (Gubler et al. 2002). Since alternative explanations could be advanced to explain those observations, it remains to be determined at what level NO interferes with GA-induced signaling pathways including PCD activation. A direct dissection of the interaction between NO and the GA-GID1-DELLA module for a specific physiological process has been only recently offered. As a result of studies performed with *nia1,2noa1-2* Arabidopsis plants, which are deficient in NO accumulation, an "slender-like" phenotype was observed when plants were grown in white or red light but not in darkness, blue or far red light (Lozano-Juste and León 2011). In turn, exogenously applied NO led to an increase in the accumulation of DELLAs proteins and shortened the hypocotyls as observed in altered function phenotypes. These data are consistent with the idea that endogenous NO levels regulate DELLAs accumulation and contribute to determine hypocotyl length. The interaction between NO and DELLAs in this particular phenomenon could be explained because of an effect of NO on the steps that lead to DELLAs turnover and activity. In this regard, it was observed that NO promoted DELLAs accumulation through negative modulation of SLY1, but also that NO could eventually reduce the synthesis of GAs in a specific pool by down regulating the *GA20ox3* gene (Lozano-Juste and León 2011). In addition, those authors observed that NO generation was down regulated by GAs (Lozano-Juste and León 2011), indicating the existence of a reciprocal interaction between both regulators. It seems now evident that NO and GAs could probably interact in other processes where the GA-GID1-DELLA module is involved. In this regard, recent studies indicated that the inhibitory effect of SNP on Arabidopsis root elongation is partially reverted by the addition of GAs being moderately diminished in plants carrying a disruption in four of the five *DELLA* genes (Fernández-Marcos et al. 2012). Interestingly, evidence for a potentially opposite regulation has been reported in wheat plants exposed to high aluminum concentrations. In this case, the addition of SNP stimulates GAs accumulation and subsequently apical root growth (He et al. 2012). These apparently conflicting reports could be explained by differences between Arabidopsis and wheat in the regulation of root growth or by an interaction between NO and GAs superimposed to the GA-GID1-DELLA module.

The usual pathway of DELLAs degradation involves the contribution of GAs. However, evidence of GAs-independent DELLAs degradation has been obtained. From the three Arabidopsis GID1 proteins, it is thought that

AtGID1B has the capacity to bind DELLAs in a GA-independent mode (Sun 2011). Interestingly it has been reported that transcript levels of *AtGID1B* are diminished in the *nia1,2noa1-2* mutant (Lozano-Juste and León 2011), suggesting the possibility that NO could eventually modulate DELLAs stability through both GAs-dependent and GAs-independent pathways.

These recent findings open an exciting perspective, while posing important questions. One of them is to what extent the antagonism between GAs and NO observed in other processes also involves the GA-GID1-DELLA module. Besides, it should be noted that while there is now some knowledge about the way by which NO exerts its effects on GAs signaling during the transition from darkness to white light, the precise mode by which GAs modulate NO generation deserves to be further examined. In this context, an additional question refers to the timing of the evolutionary coalescence of NO signaling and the DELLA-GID system. The last one probably became functional during the early steps of vascular plant evolution (Sun 2011; Depuydt and Hardtke 2011). As mentioned above, there is some controversy on the identity and relative contribution of the pathways that participate in NO synthesis in plants, being their evolutionary trend essentially unknown. Further research on this subject could help to understand how those two regulatory pathways became under reciprocal control giving additional complexity and flexibility to plant responses to multiple environmental stimuli.

Role of NO in auxin-mediated processes

Among the broad spectrum of auxin-mediated effects in plants, this section is focused on the role of auxins in root morphology (Muday and Haworth 1994; Klerk et al. 1999) and in plant responses to iron deficiency (Li and Li 2004).

The principal auxin receptors have been identified as F-box protein components of an SCF ubiquitin E3 ligase complex, belonging to the TIR1/AFB family (Dharmasiri et al. 2005; Kepinski and Leyser 2005). Auxins stabilize the formation of a heterodimer between TIR1/AFB and the Aux/IAA repressor proteins, stimulating the degradation of the transcriptional repressors Aux/IAA proteins through the covalent addition of ubiquitin molecules (Tan et al. 2007).

Auxin-regulated processes mediated by NO have been described in roots from different species and include root tip elongation, adventitious root formation, primary root growth inhibition, gravitropic response, cell cycle activation and root hair development (reviewed in Correa-Aragunde et al. 2007; Fernandez-Marcos et al. 2011). The first report for NO participation in an auxin-dependent process was performed in maize root segments exposed to NO donors (Gouvêa et al. 1997), where it was found that NO and auxins elicited the same plant response, in this case the root tip elongation. The application of NO donors mimics the effect of auxins (Chen et al. 2012), even in promoting rooting of juvenile and mature cuttings of woody plants (Abu-Abied et al. 2012). Moreover, the presence of NO was described as a necessary requirement for crown roots primordia initiation in rice seedlings (Xiong et al. 2009).

Transient increase in NO levels in tissues were observed during adventitious root development in cucumber explants and in lateral root formation in tomato, in both cases induced by auxins (Pagnussat et al. 2002; Correa-Aragunde et al. 2004). Accordingly, application of exogenous indole-3-butyric-acid (IBA) induces both lateral root initiation and NO raises in primordia of *A. thaliana* roots (Kolbert et al. 2008). Auxin-induced adventitious roots, lateral roots, and radical hair formation as well as root gravitropic response were prevented by the application of the specific NO scavenger 2-(4-carboxyphenyl)-4,4,5,5-tetramethylimidazoline-1-oxyl-3-oxide potassium salt (cPTIO) pointing out a key role for endogenous NO in mediating these processes (Pagnussat et al. 2002; Correa-Aragunde et al. 2004; Hu et al. 2005; Lombardo et al. 2006). Moreover, when polar auxin transport is blocked employing 1-napthylphlamic acid, the accumulation of NO in the interfascicular cells is completely avoided (Yadav et al. 2011).

From these reports it is clear that NO is an important molecule operating downstream of auxin through signaling pathways during root growth and development.

One possible interaction between NO levels and auxins has been proposed in cadmium-stressed plants where application of NO reduced auxin degradation through the inhibition of IAA oxidase activity, thus improving cadmium tolerance (Xu et al. 2010).

Evidence for a NO/cGMP signaling has been described in auxin-dependent adventitious root formation in explants of mung bean (Bai et al. 2012), supporting previous findings demonstrating that NO and cGMP are involved in the auxin response during the adventitious rooting process in cucumber (Pagnussat et al. 2002; Pagnussat et al. 2003). The involvement of a MAPK signal cascade in adventitious root formation induced by auxins and NO was also reported (Pagnussat et al. 2004).

These results led to proposing a model for auxin and NO signaling consisted of auxin-induced NO accumulation in tissues, which triggers a cascade involving cGMP, cADPR, increases in cytosolic $[Ca^{2+}]$, and CDPKs or an alternative cGMP-independent pathway involving MAPKs cascade (Pagnussat et al. 2002; 2003; 2004; Xiong et al. 2009; Bai et al. 2012). In addition, phosphatidic acid accumulation was pointed out as an early signaling component during the adventitious root formation induced by auxins and NO in cucumber (Lanteri et al. 2008).

A mechanism explaining NO participation in auxin-mediated processes has been recently developed for Arabidopsis roots based on NO molecular targets (Terrile et al. 2012). As it was stated, adequate auxins concentration leads to a transient increase in NO levels, which in turn produces the S-nitrosylation of the auxin receptor TIR1. This post-translational modification stimulates TIR1/AFB-Aux/IAA interaction, which eventually leads to a modulation of auxin-dependent gene expression (Terrile et al. 2012).

There are different aspects of iron metabolism that can be affected by NO (Wink and Mitchell 1998). In plants, NO reverts the symptoms of iron deficiency probably by increasing the availability of internal iron or facilitating the delivery of iron through the formation of mono- and dinitrosyl iron complexes (Graziano et al. 2002; Graziano and Lamattina 2007b; Ramirez et al. 2011). In addition, NO acts as a signaling molecule mediating iron deficiency responses through the upregulation of the expression of iron uptake-related genes and the nuclear encoded ferritin genes (Murgia et al. 2002).

Iron deficiency leads to an increase in NO level in root epidermis, which is necessary for the expression of genes associated with iron uptake in tomato roots, the basic helix-loop-helix transcription factor *LeFER*, the ferric-chelate reductase *LeFRO1*, and the Fe(II) transporter *LeIRT1* (Graziano and Lamattina 2007a).

Strategy I plants (nongraminaceous monocots and dicots) develop a range of responses against Fe-deficiency stress, among them the activation of a plasmalemma ferric-chelate reductase (Robinson et al. 1999) is a key component, since it allows the enzymatic reduction of Fe(III) to Fe(II), required for iron uptake. It has been described that auxins are able to stimulate root ferric reductase activity in Fe-sufficient cucumber, bean, and *Plantago lanceolata* plants (Schmidt and Bartels 1996; Li and Li 2004). The use of different auxin and NO-related Arabidopsis mutants (*aux1-7*, *axr1-3*, *noa1*, and *nia1 nia2*) led to the conclusion that NO acts as a signal downstream of auxins leading to ferric-chelate reductase induction under Fe scarcity (Chen et al. 2010). The authors developed a model in which Fe deficiency leads to an increase in auxin levels, with the subsequent enhance of NO concentration. NO would in turn act as a signal activating ferric-chelate reductase activity through a FIT-mediated transcriptional regulation of *FRO2* (Chen et al. 2010). The basic helix-loop-helix transcription factor FIT (FER-LIKE FE DEFICIENCY-INDUCED TRANSCRIPTION FACTOR) is a key transcription factor in the Fe deficiency response required for high-level expression of *FRO2* and *IRT* in Arabidopsis (Bauer et al. 2007). Moreover, NO and ethylene increase the accumulation of FIT counteracting its proteasomal degradation (Meiser et al. 2011; Lingam et al. 2011).

Another morphological response in plants exposed to Fe scarcity is root branching, where both auxins and NO have demonstrated to be involved (Benková and Bielach 2010). Root branching was inhibited in Fe-deficient plants treated with the NO scavenger cPTIO or the auxin transport inhibitor NPA (Jin et al. 2011). It was suggested that NO should act as a downstream signal of IAA in mediating Fe-deficiency-induced root branching (Jin et al. 2011).

Probably, NO and auxins are implicated in a general response against nutrient deficiency in plants, as it was described for the development of lateral roots and cluster roots in phosphorus-deficient plants (Nacry et al. 2005; Wang et al. 2010; Meng et al. 2012).

Interplay between NO and ethylene in plant senescence

The senescence of plant organs is the last stage in their development consisting in the degradation and remobilization of molecules to other growing tissues (Nooden et al. 1997). Degradation of chlorophyll, proteins, antioxidants and water imbalance are processes involved in the senescence of plant organs highly regulated by hormones. Ethylene is largely known as a key hormone that accelerates leaf, flower and fruit senescence. In contrast, NO has been observed participating in active growth and delaying the development of the senescence syndrome in plants (Lamattina et al. 2003; Guo and Crawford 2005). Young plant organs present high NO rate emission, but it decreases during maturation showing ethylene formation an opposite trend (Leshem et al. 1998). This implies that NO and ethylene productions are inversely affected during plant development.

A specific association between these two signaling molecules has been established in some studies. NO is able to decrease ethylene emission through the downregulation of its synthesis (Zhu and Zhou 2007; Manjunatha et al. 2012). All the elements of the ethylene synthesis pathway affected are not clearly established. However, it is consistently observed in several reports that the final step in ethylene synthesis catalyzed by 1-aminocyclopropane carboxylic acid oxidase is down regulated by NO (Zaharah and Singh 2011; Zhu et al. 2006). Furthermore, the S-nitrosylation by NO inhibits the activity of enzymes involved in ethylene synthesis (Kaur and Deswal 2010). This work shows that ethylene synthesis is highly controlled by NO.

Besides, NO may affect the response of different plant organs to ethylene. It is well known that the sensitivity of the tissues to a hormone depends on the active participation of specific receptors and downstream components of hormone signaling. NO might have an influence on ethylene signaling pathway, but this interrelationship has not been deeply studied yet. Information about the cross-talk

between these two physiological players is scarce and remains to be further studied in detail.

The antagonistic effect of NO on ethylene-associated senescence disorders has been used for the manipulation of the development of undesirable biochemical modifications of edible plant organs. Exogenous NO treatment was successfully used for the extension of postharvest life of many fruits (Cheng et al. 2009; Wills et al. 2000; Zhu et al. 2006). NO treatment delays the climacteric peak of ethylene production and the progress of fruit ripening of several species. Associated with ethylene, there is a raise in the respiratory activity and in ROS generation that are involved in the acceleration of deteriorative processes during ripening. Respiration activity is proportional to the product deterioration, since many organic compounds are metabolized (Kader 2002). It was also observed that NO decreases respiration through the inhibition of cytochrome c oxidase (Millar and Day 1996).

Another beneficial aspect observed during postharvest is the improvement of fruit quality. Ascorbic acid or vitamin C is an important nutritional metabolite that declines during ripening (Kader 2002). While ethylene decreases ascorbic acid concentrations (Gergoff-Grozeff et al. 2013) NO stimulates its accumulation (Jin et al. 2009). Mitochondrial activity, which is affected by both NO and ethylene, regulates ascorbate synthesis (Millar and Day 1996; Bartoli et al. 2006). The precise mechanism linking NO, ethylene, ascorbic acid, and respiration during plant edible organ storage remains to be studied. It is worth noting that other nutritional attributes were also improved by NO treatment on several fruits (Manjunatha et al. 2012 and references therein).

Similarly, flower vase conservation of a climacteric species is improved by NO (Leshem et al. 1998; Bowyer et al. 2003). NO extended the postharvest life of carnation flowers, increasing the flower fresh weight and the antioxidant activity and reducing the oxidative damage (Zeng et al. 2011). The mechanism of action of NO on ethylene metabolism and sensitivity in cut flowers is not known.

All this evidence shows that NO might affect different metabolic traits leading to the improvement of extension and quality of plant products for human consumption.

Concluding remarks

Undoubtedly, NO has a key role in signal transduction network in plants influencing the action of hormones. In addition to the mentioned interactions with ABA, auxins, ethylene, and gibberellins, other research suggests that NO is involved in jasmonic acid (Huang et al. 2004), salicylic acid (Zottini et al. 2007), and brassinosteroids (Hayat et al. 2010) actions. Temporally and spatially controlled changes

in NO steady state concentration have an impact on second messengers, enzymatic activities or activation of transcription factors. Even though important molecular targets of NO have begun to be discovered, and many others are likely in the way to be identified in the near future, one important question is how plants regulate cellular NO steady state concentrations. Different pathways contribute to the consumption, among them the enzyme nitrosoglutathione reductase (GSNOR) (Lee et al. 2008) and the molecular targets mentioned along the text. However, the sources of NO, which probably rely on local substrates availability such as L-arg, O_2, or nitrite concentrations, as well as changes in pH, remain unclear (Fröhlich and Durner 2011).

One of the main drawbacks in research involving NO is the difficulty inherent in its detection. Detection is not a simple issue due to both the low concentrations and the relative high chemical reactivity of NO. The detection method employing the commonly used diaminofluorescein (DAF) derivatives is indirect, and actually relies in the reaction of a product of NO oxidation with the fluorophore, thus it could be affected by the presence of oxidants (Jourd'heuil 2002). Detection of NO in a complex biological system by this method should be confirmed, so far as possible, by the use of alternative methodologies (Besson-Bard et al. 2008).

An additional difficulty issue is related to the way in which NO levels are manipulated in experimental conditions. Research on NO participation in biological processes is mainly based on the use of NO scavengers, NO donors, and inhibitors of endogenous NO synthesis. Reinforcing the idea of using different methodological approaches, it was observed under certain conditions, that the presence of the NO scavenger cPTIO could result in an unexpectedly increase in the observed DAF fluorescence (Arita et al. 2006), which could be likely attributed to the indirect mode of this detection as it was pointed out. Regarding the NO donors, it is important to bear in mind that each compound may have a different kinetic of NO release, and can differ from other in the chemical form of NO generated (NO· or NO^+) (Floryszak-Wieczorek et al. 2006). This could help to explain contradictory observations depending on the NO donor employed (Murgia et al. 2004). Compounds may also vary in their capacity for acting as nitrosylating agents, and sometimes there are other active compounds that are being released together with NO (Bethke et al. 2006). Finally, the use of inhibitors of endogenous NO synthesis needs a note of caution. Despite the fact that at least seven different pathways have been proposed to produce NO in plants (Gupta et al. 2011), there is a lack of knowledge regarding the relevance of the different sources under physiological conditions. Nitrate reductase (NR) is the unique enzymatic activity identified in plants with the ability to reduce nitrite

to NO. This enzymatic activity is frequently inhibited in vivo employing tungstate, which is an inhibitor of molybdate-containing enzymes. Thus, tungstate has shown to exert other effects unrelated with the inhibition of NR activity (Xiong et al. 2012). Other important pathway for NO production is related with the oxidation of the amino-acid L-arg. Nitric oxide synthases (NOS) are present in almost all known organisms except in higher plants, where no NOS genes or enzymes have been identified yet. Just in 2010 was described the first cannonical NOS gene in a photosynthetic organism, the microalgae *Ostreococcus tauri* (Foresi et al. 2010). The use of L-arg analogs that are inhibitors of classical mammalian NOS activities have shown to be active in plant research, often in high concentrations (Gupta et al. 2011). In this regard, genetic approaches involving the use of mutant plants with altered endogenous NO levels constitute a valuable tool for research supporting pharmacological studies.

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