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Review

NO signaling in plant immunity: A tale of messengers

Pauline Trapet [a,e], Anna Kulik [b,e], Olivier Lamotte [c,e], Sylvain Jeandroz [d,e], Stéphane Bourque [a,e], Valérie Nicolas-Francès [a,e], Claire Rosnoblet [a,e], Angélique Besson-Bard [a,e], David Wendehenne [a,e,*]

[a] Université de Bourgogne, UMR 1347 Agroécologie, BP 86510, F-21000 Dijon, France
[b] INRA, UMR 1347 Agroécologie, BP 86510, F-21000 Dijon, France
[c] CNRS, UMR 1347 Agroécologie, BP 86510, F-21000 Dijon, France
[d] AgroSup Dijon, UMR 1347 Agroécologie, BP 86510, F-21000 Dijon, France
[e] ERL CNRS 6300, BP 86510, 21000 Dijon, France

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ABSTRACT

Nitric oxide (NO) is a free radical gas involved in a myriad of plant physiological processes including immune responses. How NO mediates its biological effects in plant facing microbial pathogen attack is an unresolved question. Insights into the molecular mechanisms by which it propagates signals reveal the contribution of this simple gas in complex signaling pathways shared with reactive oxygen species (ROS) and the second messenger Ca^{2+}. Understanding of the subtle cross-talks operating between these signals was greatly improved by the recent identification and the functional analysis of proteins regulated through S-nitrosylation, a major NO-dependent post-translational protein modification. Overall, these findings suggest that NO is probably an important component of the mechanism coordinating and regulating Ca^{2+} and ROS signaling in plant immunity.

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Contents

Introduction

Nitric oxide (NO) is a free radical gas produced by most, if not all organisms. The last fifteen years brought several landmarks to the field of NO functions in plant physiology. NO is now recognized to act as a ubiquitous cell signaling molecule involved in various processes such as root growth, iron uptake and sequestration, flowering, pollen tube growth, stomatal closure or hormonal signaling (Besson-Bard et al., 2008b; Simontacchi et al., 2013). It has also been linked to the plant adaptive responses to pathogenic and symbiotic micro-organisms (Frederickson Matika and Loake, 2013; Puppo et al., 2013) as well to abiotic stresses (Corpas et al., 2011). Undoubtedly, research devoted to its role in plant immunity

* Corresponding author at: Université de Bourgogne, UMR 1347 Agroécologie, ERL CNRS 6300, 17 rue Sully, BP 86510, F-21000 Dijon, France. Tel.: +33 380693722; fax: +33 380693753.
E-mail address: wendehen@dijon.inra.fr (D. Wendehenne).

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has received particular attention since the publication of pioneer works underlying the ability of plants to use NO as a signal to defend themselves from invaders (Delledonne et al., 1998; Durner et al., 1998). It is now well documented that NO produced in plant cells challenged by pathogens, MAMPs (Microbe-Associated Molecular Pattern) and DAMPs (Damage-Associated Molecular Pattern) is integrated in signaling cascades leading to the expression of defense-related genes, the production of secondary metabolites and, ultimately, to HR (Hypersensitive Response) (Bellin et al., 2013; Gaupels et al., 2011a,b; Leitner et al., 2009). Genetic, biochemical and pharmacological studies point to nitrate reductase (NR) and a still unidentified enzyme related to mammalian nitric oxide synthase as the main enzymatic sources for NO in plant immune responses. However, how NO is indeed generated remains largely unclear and controversial (Moreau et al., 2010).

As a ubiquitous messenger, a main and still ongoing question is how the information encrypted in the pathogen-induced increases in NO concentration helps to define the outcome of the response. Emerging data highlight that NO is a component of complex but poorly understood networks of signaling compounds including reactive oxygen species (ROS), hormones and the second messengers Ca^{2+}, cyclic GMP, cyclic AMP and cyclic ADP-ribose (Courtois et al., 2008; Gaupels et al., 2011a,b; Jeandroz et al., 2013; Ma et al., 2012). Several studies also support a role for lipids and protein kinases in regulating and/or mediating NO-induced responses (Courtois et al., 2008; Mandal et al., 2012). Why does it have to be so complicated? The response probably relies on NO chemistry. Due to its redox nature, NO is capable of wide range of physiologically relevant chemical reactions. Indeed, as a free radical possessing an unpaired electron, NO reacts with species containing unpaired electrons such as superoxide ($O_2^{\cdot-}$) and with transition metals, notably iron (Stamler et al., 1992). Furthermore, through derivatives such as the nitrosonium ion (NO^+) and higher oxides of nitrogen (notably NO_2, N_2O_3) NO reacts with thiolate or, in the case of peroxynitrite ($ONOO^-$), with tyrosine (Ferrer-Sueta and Radi, 2009; Martínez-Ruiz et al., 2013). Basically, this means that contrary to conventional signaling molecules, NO is unlikely to interact with a unique defined receptor but rather with numerous target proteins.

A central physiological NO-based protein modification appears to be effected by S-nitrosylation. This process designs the reversible incorporation of an NO moiety by covalent bounding to a reactive Cys residue (Stamler et al., 2001). Nowadays, the literature review identified more than 100 proteins putatively S-nitrosylated in plants (see for instance Lindermayr et al., 2005; Tanou et al., 2009). These proteins are involved in all key cellular functions. However, few of them were identified in biological conditions in which the influence of endogenously produced NO was investigated. Furthermore, a role for S-nitrosylated proteins in plant immunity has been ascribed for only a handful of candidates. This includes *Arabidopsis thaliana* NADPH oxidase AtRBOHD (Respiratory Burst Oxidase Homologue D) (Yun et al., 2011), Peroxiredoxin II E (PrXII; Romero-Puertas et al., 2007), NPR1 (Nonexpressor of Pathogenesis-Related gene 1; Tada et al., 2008), the transcription factor TGA1 (TGACG sequence-specific binding protein 1) (Lindermayr et al., 2010) and SABP3 (Salicylic Acid-Binding Protein 3) (Wang et al., 2009a,b). S-nitrosylation influences the activity and function of these proteins in different ways: by impacting the binding of cofactors as reported for AtRBOHD and SABP3, by interfering with active-site Cys residues as shown for PrXII, by promoting the formation of disulfide bonds and, consequently, the switches between the monomeric/oligomeric states in the specific case of NPR1 and, conversely, by protecting Cys residues from formation of disulfide bonds as suspected for TGA1. It might also facilitate protein translocation between subcellular compartments as well as protein interactions as shown for NPR1 and TGA1. Several recent reviews have precisely addressed the incidence of NO on the structure, activities and functions of these proteins (see for instance Astier et al., 2012; Frederickson Matika and Loake, 2013; Kovacs and Lindermayr, 2013) but, with the exception of AtRBOHD and PrxII, these aspects will not be covered here. Beside S-nitrosylation, tyrosine nitration and metal S-nitrosylation represent other NO-dependent post-translational modifications of proteins that might regulate cellular functions (Besson-Bard et al., 2008b; Jacques et al., 2013; Vandelle and Delledonne, 2011). Involvement and incidences of these processes in plant defenses have garnered less attention and, so far, are poorly understood.

As stated above, the picture that has formed indicates that the propagation and regulation of NO signaling entails cross-talk with other cell signals and, probably, with pathways involving NO-independent post-translational modifications such as phosphorylation. The purpose of this review is to summarize studies highlighting the occurrence of cross-talks operating between NO and Ca^{2+}-or ROS-based signaling in plant immunity. The potential generality and importance of S-nitrosylation in these cross-talks are also discussed.

Cross-talk between NO and Ca^{2+} in immune signaling

NO, a component of Ca^{2+}-dependent signaling cascades

Increasing evidences document the occurrence of a cross-talk between NO and Ca^{2+} signaling pathways in cells challenged by pathogen attack, MAMPs or DAMPs. First, MAMPs- and DAMPs-induced NO production has been shown to be under the control of Ca^{2+} influxes originating from the extracellular space. For instance, pharmacological inhibition of the influx of extracellular Ca^{2+} triggered by the elicitin cryptogein in tobacco cell suspensions (Lamotte et al., 2004), by endopolygalacturonase 1 from *Botrytis cinerea* in grape cells (Vandelle et al., 2006) and by LPS (lipopolysaccharide) or oligogalacturonides (OGs) in *A. thaliana* leaves (Ali et al., 2007; Rasul et al., 2012) led to a significant suppression of NO synthesis. Both genetic and pharmacological data point to CNGCs (Cyclic Nucleotides-Gated ion Channel) as plant cell channels mobilized in response to pathogenic invaders or MAMPs and mediating the Ca^{2+} fluxes required for NO production (Ma and Berkowitz, 2011). Notably, using the *dnd1* (*defense no death1*) *A. thaliana* mutant that has a null mutation in the *CNGC2* gene, in their pioneer work, Ali et al. (2007) linked a CNGC2-dependent plasma membrane Ca^{2+} conductance to downstream NO generation in *A. thaliana* cells responding to LPS and, more generally, undergoing HR. Further investigations favor the involvement of CaM (calmodulin) and protein kinases including MAPKs (Mitogen-Activated protein Kinases) and CDPKs (Ca^{2+}-Dependent Protein Kinases) in transducing the Ca^{2+} current to NO synthesis (reviews by Jeandroz et al., 2013; Ma and Berkowitz, 2011; see also Ma et al., 2013). More recently, a great deal of attention has been paid to the Ca^{2+} permeable channels glutamate receptors (GLR) as molecular components of the signaling processes related to plant defense responses (Kwaaitaal et al., 2011; Mousavi et al., 2013). Interestingly, animal GLR antagonists were shown to impair elicitors-induced NO synthesis in tobacco (Vatsa et al., 2011) and *A. thaliana* (Manzoor et al., 2013), thus providing evidences that GLRs could link the Ca^{2+} signal and NO generation. Manzoor et al. (2013) further demonstrated that AtGLR3.3, one of the 20 GLRs in *A. thaliana* playing a key role in resistance against *Hyaloperonospora arabidopsidis* and *Pseudomonas syringae* pv. *tomato* DC3000 (Li et al., 2013), partly controls the production of NO and ROS observed in response to OGs. Accordingly, the OG-induced NO synthesis was reduced of about 20–35% in leaves of three distinct *atglr3.3* mutant lines. Taken together, these data highlight that both CNGCs and

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