Environmental and Experimental Botany 100 (2014) 84–93

Contents lists available at ScienceDirect

Environmental and Experimental Botany

journal homepage: www.elsevier.com/locate/envexpbot

Review

# Cross-talk between nitric oxide and hydrogen peroxide in plant responses to abiotic stresses

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ARTICLE INFO

*Article history:*
Received 11 October 2013
Received in revised form 9 December 2013
Accepted 25 December 2013

*Keywords:*
Nitric oxide
Hydrogen peroxide
Stress response
Cross-talk

ABSTRACT

Nitric oxide (NO) and hydrogen peroxide ($H_2O_2$) are two signaling molecules, which play roles in diverse organisms. In the past two decades, evidence has been accumulating to address their involvements in stress responses in plants, but how these two molecules interact with each other and how the signals are integrated in biological processes remain fragmentary and far from clear in the literature. This review brings together the knowledge obtained so far on these two molecules and their cross-talk in plant stress responses, particularly abiotic stresses including drought, salinity, extreme temperatures, UV light, and heavy metals. We tentatively discuss, in the context of abiotic stresses, how NO and $H_2O_2$ interact with each other at two levels, biosynthesis, and regulation of gene expression or protein activities. The cross-talk between NO and $H_2O_2$ with other signaling pathways in the regulation of abiotic stress responses in plants is also discussed.



**Contents**



## 1. Introduction

As an important plant endogenous signaling molecule, nitric oxide (NO) mediates complex biological functions in plants. This is mainly due to its properties: free radical, small size, short-lived, and highly diffusible (Leshem et al., 1998). Previous studies have implicated the involvement of this molecule in almost all biological processes *in planta*, including plant maturation and senescence (Guo and Crawford, 2005; Mishina et al., 2007), seed germination or dormancy (Beligni and Lamattina, 2000; Bethke et al., 2006, 2007; Libourel et al., 2006), as well as ABA-mediated floral transition and stomatal movement (Neill et al., 2002a; Guo et al., 2003; He et al., 2004). Meanwhile, NO also mediates a range of resistance mechanisms in plants under stress conditions (Delledonne et al., 1998; Uchida et al., 2002; Zhao et al., 2004, 2006, 2007). The effects of NO depend on its location and concentration. NO at

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http://dx.doi.org/10.1016/j.envexpbot.2013.12.014

high levels has a series of negative effects, ranging from reductions in photosynthetic electron transport, inhibition of shoot and root development, to membrane damage and DNA fragmentation leading to cell death (Leshem et al., 1998; Pedroso et al., 2000). However, NO also promotes normal growth and development of plants at lower concentrations (Beligni and Lamattina, 2001). Both biotic and abiotic stresses alter (promote or suppress) NO production, and externally applied NO donors can enhance plant tolerance to abiotic stresses (Delledonne et al., 1998; García-Mata and Lamattina, 2002; Uchida et al., 2002; Zhao et al., 2007). More interestingly, numerous studies have discovered cross-talk between NO and other signaling molecules like hydrogen peroxide ($H_2O_2$) in plants upon exposure to environmental stimuli.

$H_2O_2$ is a form of reactive oxygen species (ROS) generated as a result of oxidative stresses. Oxidative stresses may provoke oxidative damage due to excessive accumulation of ROS under various abiotic stress conditions, and ultimately lead to cell death (Fotopoulos et al., 2006; Tanou et al., 2009a). Metabolic pathways in plant organelles are sensitive to changes in environmental conditions, and metabolic imbalances can induce oxidative stresses in cells by promoting the generation and accumulation of ROS, causing oxidation of cellular components, and interfering with metabolic activities and organelle integrity (Suzuki et al., 2012). ROS were initially thought to be toxic byproducts of aerobic metabolism, but now have been implicated as central players in a multitude of cellular signaling networks (Moller and Sweetlove, 2010). $H_2O_2$ is generated in chloroplasts, mitochondria and peroxisomes, and can be kept in homeostasis in plant cells by the complex and effective scavenging systems (de Carvalho, 2008). Moreover, accumulating evidence indicates that $H_2O_2$ acts as a local and systemic signal that directly regulates expression of numerous genes involved in plant biotic and abiotic responses (Desikan et al., 2001a; Zago et al., 2006). $H_2O_2$ generation is also induced in plants following exposure to a wide variety of environmental stimuli. It is apparent that $H_2O_2$ acts as a signal to induce a range of molecular, biochemical and physiological responses within cells and plants, and mediates cross-talk between signaling pathways (Neill et al., 2002b).

NO molecule itself possesses antioxidant properties (Karplus et al., 1991), regulates the level and toxicity of ROS by modulating ROS production and degradation (Palmieri et al., 2008). NO prevents oxidation damage by regulating cellular redox homeostasis, enhancing the $H_2O_2$-scavenging enzymes activities, and thereby decreases the levels of $H_2O_2$ and superoxide anions (Lamattina et al., 2003; Shi et al., 2007). Several lines of evidence support the presence of a strong cross-talk between oxidative and nitrosative signaling under stress conditions (Molassiotis and Fotopoulo, 2011). Cross-talk between the different signaling molecules results in synergetic or antagonistic interactions that play crucial roles in the responses of plants to abiotic stresses. In the past few years, the roles of NO and $H_2O_2$ and their cross-talk in mediating tolerance of plants to abiotic stresses have been largely established. In this review, we summarize the knowledge obtained on the possible cross-talk between NO and $H_2O_2$, and in particular, highlight the latest findings that support the connection between NO and $H_2O_2$ signaling networks modulating plant responses to abiotic stimuli.

## 2. Cross-talk between NO and $H_2O_2$ in tolerance of plants to abiotic stresses

As mentioned in Section 1, NO and $H_2O_2$ have been implicated to play vital roles in stress responses in plants. It is commonly observed that NO and ROS are generated in response to similar abiotic stress stimuli with similar kinetics (Desikan et al., 2004). Exposure of plants to various abiotic stresses usually induces the generation of both $H_2O_2$ and NO. Such stresses include dehydration, salinity, drought, atmospheric pollutants such as ozone, UV irradiation, temperature extremes, and mechanical wounding (Desikan et al., 2004). Almost all abiotic stresses generate free radicals and other oxidants, particularly from the chloroplasts, mitochondria and peroxisomes (Mano, 2002), resulting in oxidative stress in terms of an increased level of ROS in plant cells (Mittler, 2002). Nitrite-dependent NO production has been reported in mitochondria (Planchet et al., 2005), and arginine dependent NOS-like activity has also been detected in peroxisomes (Corpas et al., 2001) and chloroplasts in plant cells (Jasid et al., 2006). Given that the resistance to stressful conditions is intimately connected to the capacity of plants to tolerate oxidative stress, it is not surprising that the $H_2O_2$ and NO signaling pathways are closely linked and coordinated during the overall responses of plants to environmental stimuli (Molassiotis and Fotopoulo, 2011).

Both NO and ROS are known to exhibit either toxic or protective effects to the organisms, depending on the circumstances. When present at low levels, ROS, mostly $H_2O_2$, act as signals for the activation of defense responses. However, higher concentrations of ROS can cause severe injury. Thus, it is a survival response for plants to regulate the cellular concentrations of ROS. While toxicity is incurred predominantly from ROS, NO may act as a chain breaker and thus limit the damage (Lipton et al., 1993). Exogenous supplies of NO can protect plants from oxidative damage by eliminating superoxide anions ($O_2^{\bullet-}$) and lipid radical $R^-$ (Shi et al., 2007). The scavenging of $O_2^{\bullet-}$ by NO leads to the formation of peroxynitrite ($ONOO^-$), which is highly toxic in animal cells but not toxic to plant cells (Delledonne et al., 2001; Kopyra and Gwóźdź, 2004). NO can also decrease membrane permeability, production of ROS and malondialdehyde (MDA), and intracellular $CO_2$ concentration under abiotic stresses by promoting activities of ROS-scavenging enzymes like CAT, peroxidases (POD), superoxide dismutase (SOD), ascorbate peroxidase (APX), and proline accumulation (Kopyra and Gwóźdź, 2003; Shi et al., 2007; Sheokand et al., 2008; Lopez-Carrion et al., 2008; Guo et al., 2009).

In following subsections, we discuss the possible relationships between NO and $H_2O_2$ in plants under each individual abiotic stress.

### 2.1. Salt stress

Soil salinity is a major threat to global crop productivity and food security. Salinity affects plant growth and development in two ways: it imposes osmotic stress, and destroys ionic homeostasis and ultimately interferes with various metabolic processes. Plant responses to the osmotic and ionic components of salt stress are complicated and involve many gene networks and metabolic processes (Abogadallah, 2010). Altered production of both $H_2O_2$ and NO is amongst these responses of plants to salt stress (Avsian-Kretchmer et al., 2004; Zhang et al., 2006; Zhao et al., 2007). Salt stress often leads to increased production of ROS in plants, which include $H_2O_2$, whereas the effects of salt stress on NO production remain elusive. As reported by Zhao et al. (2007), NaCl treatment suppressed the expression of the *AtNOA1* gene, an Arabidopsis NO-associated gene, resulting in a reduction of NO level (Zhao et al., 2007). In contrast, the expression of *OsNOA1*, the rice homolog of *AtNOA1* was moderately promoted by salt stress (Qiao et al., 2009). This difference may reflect that different species may employ their own NO-synthetic or regulatory systems to respond to salt stress.

$Na^+/K^+$ homeostasis is an important salt tolerance mechanism, in which $H_2O_2$ and NO are involved. NO may serve as a signal in inducing salt tolerance by reducing the $Na^+/K^+$ ratio, which is dependent on the increased plasma membrane (PM) and vacuolar $H^+$-ATPase, as well as $H^+$-PPase activities, or on the NO-induced expression of plasma membrane $Na^+/H^+$ anti-porter and $H^+$-ATPase-related genes (Zhao et al., 2004; Zhang et al., 2006; Wang et al., 2009). Mazid et al. (2011) reported that NO regulated