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Review

Nitric oxide (NO) in alleviation of heavy metal induced phytotoxicity and its role in protein nitration

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ABSTRACT

Nitric oxide (NO) is recognized as a biological messenger in various tissues to regulate diverse range of physiological process including growth, development and response to abiotic and biotic factors. The NO emission from plants is known since the 1970s, and there is copious information on the multiple effects of exogenously applied NO on different physiological and biochemical processes of plants. Heavy metal toxicity is one of the major abiotic stresses leading to hazardous effects in plants and its toxicity is based on chemical and physical property. A common consequence of heavy metal toxicity is the uncontrolled and excessive accumulation of reactive oxygen species (ROS) which leads to peroxidation of lipids, oxidation of protein, inactivation of enzymes, DNA damage and/or interact with other vital constituents of plant cells. Recently, an increasing number of articles have reported the effects of exogenous NO on alleviating heavy metal toxicity in plants but knowledge of physiological mechanisms of NO in alleviating heavy metal toxicity is quite limited, and some results contradict one another. Therefore, to help clarify the roles of NO in heavy metal tolerance, it is important to review and discuss the recent advances on this area of research. NO can provoke both beneficial and harmful effects, which depend on the concentration and location of NO in the plant cells. NO alleviates the harmfulness of the ROS, and reacts with other target molecules, and regulates the expression of stress responsive genes under various stress conditions. This manuscript includes, the latest advances in understanding the effects of endogenous NO on heavy metal toxicity and the mechanisms and role of NO as an antioxidant as well as in protein nitration are highlighted.

Contents

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Introduction

Nitric oxide (common name) or nitrogen monoxide (systematic name) is a chemical compound with chemical formula NO. Joseph Priestley in 1772 described NO a highly toxic compound as it is a component of pollutant gasses released by industrial waste, vehicle exhaust and cigarette smoke etc. Before the discovery of NO as a signal molecule, free radicals were considered as toxic by-products of oxidative metabolism but today's research is focused on the role of this free radical in various cellular functions. Nitric oxide is highly reactive (having a lifetime of a few seconds), yet diffuses freely across membranes. NO can exist in interchangeable structures namely the nitrosonium cation (NO^+), nitroxyl anion (NO^-)and the nitroxyl radical ($NO^{\cdot}$). These attributes make nitric oxide ideal for a transient paracrine (between adjacent cells) and autocrine (within a single cell) signaling molecule [1]. NO does not bind to a single receptor due to its chemistry and diffusible behavior. NO and its derivatives performs post translation modification signaling which is the main cellular activity of all NO targeted protein. Nitric oxide a bioactive molecule plays a key role in diverse physiological process in plants [2]. NO can be produced in plants by non-enzymatic and enzymatic systems [3–6] (biosynthesis of NO shown in Fig. 1). Keppler in 1979 reported that plants can generate this molecule too [7]. Intensive research is being done on the physiological function of NO under normal and stress conditions in plants [8–10]. Since past decade various articles have reported the effect of exogenous NO on alleviating heavy metal toxicity but our knowledge about physiological mechanism of NO involvement in alleviating heavy metal toxicity is quite limited. At present, it is well established that plant cells can generate NO, but there is some controversy regarding the specific source of NO in a defined physiological process, as well as about how NO levels are modulated under normal and adverse conditions. In this review, we discuss recent progress in understanding the function of NO in alleviating heavy metal toxicity in plants.

Heavy metal and mechanism of heavy metal toxicity

Heavy metals are those metals which have density higher than 5 g cm^{-3}. 53 out of 90 naturally occurring elements are heavy metals [11] but all of them are not biologically important. Only 17 heavy metals based on their solubility under physiological conditions are predicted to be available for living cells and of importance for organism and ecosystem. Among these metals Fe, Mo and Mn are important as micronutrients. Zn, Ni, Cu, V, Co, W and Cr are toxic elements with high or low importance as trace elements. As, Hg, Ag, Sb, Cd, Pb and U have no known function as nutrients and seem to be more or less toxic to plants [12–14] Heavy metal plays both the role of essential components for the maintenance of normal biological functions and toxic agents with damaging consequences when present in inappropriate amounts. Metal induced toxicity is very well reported in the literature. Previous reports suggest that metals are capable of interacting with nuclear proteins and DNA causing oxidative deterioration of biological macromolecules [15]. It has also been reported that heavy metals alter both the activities of antioxidative enzymes and the content of soluble antioxidants in plants, accompanied by an increase of oxidative stresses [16,17]. Oxidative stress includes hydroxylation, DNA fragmentation, lipid peroxidation and haem protein oxidation.

Heavy metal action can be understood on the basis of chemical properties. Mostly heavy metals are transition metals with an incompletely filled δ-orbital present as cations under physiological conditions. Heavy metal can be divided into two categories redox active and inactive metals. Metals having redox potential lower than biological molecules cannot be involved in redox reactions such as Zn^{2+}, Cd^{2+}, Ni^{2+}, Pb^{2+} etc. In Fenton-type reaction (metallooxo-oxidants and hydroxyl radical ($OH^{\cdot}$) produced by the reaction between ferrous iron and hydrogen (or alkyl) peroxide) redox active metals such as Fe^{2+} or Cu^{+} autooxidize to form superoxide anion (O_2^-) with subsequent production of hydrogen peroxide (H_2O_2) and $OH^{\cdot}$. Another mechanism is their ability to bind strongly to oxygen, nitrogen and sulfur atoms [18]. This binding affinity is related to free enthalpy of the formation of the product of metal and ligand. Due to these feature heavy metal can inactivate enzymes by binding to cystein residues.

Heavy metal leads to induction of lipid peroxidation which may be due to heavy metal induced increase in lipoxygenase (LOX) activity. Many enzymes need cofactor to work properly for both heavy metal ions (such as heam, biotin, Flavin adenine dinucleotide (FAD), Nicotinamide adenine dinucleotide (NAD) or coenzyme A). Displacement of divalent cation such as Co^{2+}, Ni^{2+} and Zn^{2+} by Mg^{2+} in ribulose-1, 5-biphosphate carboxylase/oxygenase (RuBisCO) results in loss of enzyme activity [19,20]. Membrane damage due to heavy metal toxicity involves various mechanisms such as the oxidation of and cross linking with protein thiols, inhibition of key membrane protein such as H^+ ATPase or causing changes in the composition and fluidity of membrane lipids [21].

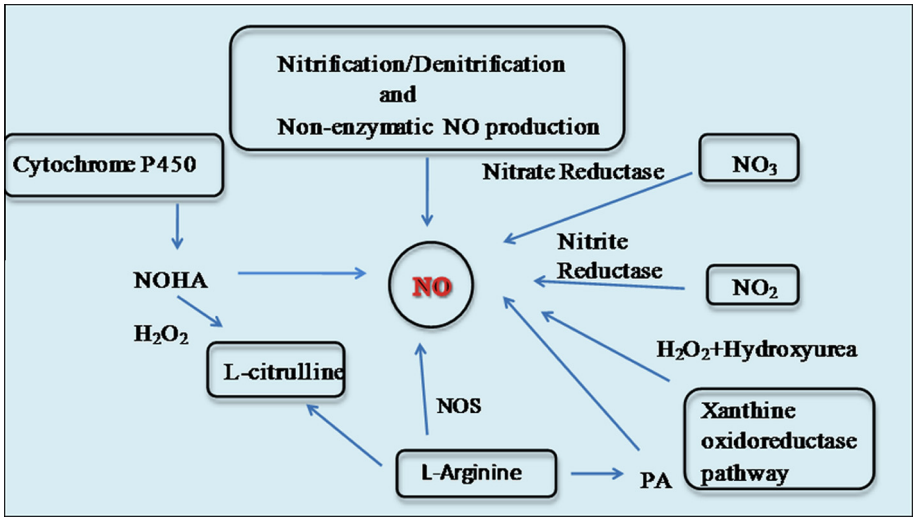

Fig. 1. Biosynthesis of NO in plant cell.