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Review

Action and target sites of nitric oxide in chloroplasts

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ABSTRACT

Nitric oxide (NO) is an important signalling molecule in plants under physiological and stress conditions. Here we review the influence of NO on chloroplasts which can be directly induced by interaction with the photosynthetic apparatus by influencing photophosphorylation, electron transport activity and oxido-reduction state of the Mn clusters of the oxygen-evolving complex or by changes in gene expression. The influence of NO-induced changes in the photosynthetic apparatus on its functions and sensitivity to stress factors are discussed.

Contents

Abbreviations: Chl, chlorophyll; cPTIO, 2-(4-carboxyphenyl)-4,4,5,5-tetramethylimidazoline-l-oxyl-3-oxide; cyt b_6f, cytochrome b_6f; ETR, electron transport rate through PSII; Fm/Fo, ratio of the maximal to minimal fluorescence in dark adapted state; Fv/Fm, maximal quantum efficiency of PSII photochemistry in dark adapted state; Fv′/Fm′, efficiency of excitation energy capture by open PSII reaction centres; Φ_{PSII}, effective quantum yield (efficiency) of PSII photochemistry in light adapted state; GSNO, S-nitrosoglutathione; GSSG, glutathione disulphide; NO, nitric oxide; NOR2, (±)-(E)-4-methyl-2-[(E)-hydroxyimino]-5-nitro-3-hexenamide; NOS, nitric oxide synthase; NPQ, non-photochemical quenching; NR, nitrate reductase; OEC, oxygen evolving complex; PAM, pulse amplitude modulation; Pn, net photosynthetic rate; PPFD, photosynthetically active photon flux density; PSI, photosystem I; PSII, photosystem II; qL, coefficient of photochemical fluorescence quenching assuming interconnected PSII antennae; qN, non-photochemical quenching coefficient; qP, coefficient of photochemical fluorescence quenching assuming non-interconnected PSII antennae; ROS, reactive oxygen species; Rubisco, ribulose-1,5-bisphosphate carboxylase/oxygenase; SNAP, S-nitroso-N-acetylpenicillinamine; SIN-1, 3-morpholinosydnonimine; SNOC, S-nitrosocysteine; SNP, sodiumnitroprusside.

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1. Introduction

Nitric oxide (NO) is now established as an important signalling molecule regulating a wide range of physiological, biochemical and molecular processes in animals [1]. The pathway of NO synthesis in animals, mediated by isoforms of the enzyme nitric oxide synthase (NOS) from L-arginine, is well characterized. In animals, NO plays a vital role in maintaining blood pressure, host immune system, neural transmission and gene expression [2]. Nitric oxide is a gaseous free radical molecule which diffuses readily through biological membranes and has a biological half-life ranging from 5 to 13 s [3]. This short half-life reflects the highly reactive nature of the molecule: it reacts with metal complexes and other radicals, and with biomolecules such as nucleic acids, proteins and lipids [4]. It can also react with superoxide (O_2^-) to form peroxynitrite ($ONOO^-$), which can damage lipids, proteins and nucleic acids [5]. There is growing evidence that NO is also an important signalling molecule in plants [2,6–11] which has been obtained via the application of NO, usually in the form of donors, via the measurement of endogenous NO, and through the manipulation of content by chemical and genetic means [12]. As in animals, the roles of NO in plants may be equally diverse by having a role in cell signalling and regulation of plant growth and development [6–10,13]. These effects can be manifested directly by exogenous NO donors or through effector molecules that preferentially regulate the redox state of the cell [14]. The latter process occurs by the endogenous generation of NO in plants either by metabolic regulations, perturbations or modulations by developmental, environmental or genetic cues [6–9]. NO can modify the structure and function of haemoproteins or proteins by reversible iron-ligand binding to functional haem prosthetic groups or to thiol groups and its role as a radical cytotoxin or cytoprotectant through the redox homeostasis of the cellular environment in plants has also been reported [15–17]. These diverse effects of NO arise from concentration-dependent reactions in living cells and tissue: low (sub-μmolar concentrations) of exogenous NO donors stimulate plant growth, regulation of stomatal movement and retardation of programmed cell death and senescence [6–9,16,18–21] while higher (mmolar concentrations) disturb metabolic activities in plant cells including decreased photosynthetic electron transport, increased viscosity of isolated thylakoid membrane lipid monolayers, leaf expansion [18,22] and net photosynthesis [23].

Chloroplasts are very sensitive and susceptible to stressful climatic changes [24–26] and because NO is a signalling molecule in plants known to ameliorate stress effects, understanding its role on chloroplasts is crucial. In addition, chloroplasts are proposed as a site for the synthesis of endogenous nitric oxide [27–29]. Currently, despite many recent reviews on NO effects in plants [6–9,14,30–35], little is known of the effect of NO on the regulation of different physiological, biochemical and molecular processes in chloroplasts. This review summarizes the most recent studies on the effect of NO on chloroplasts with particular emphasis on the effect of NO on the efficiency of the photochemical-transduction- and carbon-fixation – pathways as well as its stress ameliorating effect on chloroplasts.

2. Chloroplasts generate NO

Chloroplasts are semi-autonomous organelles in green plants with diverse structure, function and environmental adaptability. They have developed a thylakoid membrane network, suspended in the stroma matrix: these membranes possess pigment protein complexes which transduce solar energy by converting it to electrical potentials that generate chemical energy (ATP) and reducing equivalents (protons) in the form of $NADPH^+$ generated by the photolysis of water. The ATP and reductants generated are utilized to reduce carbon dioxide to carbohydrates in the stroma.

The thylakoid membranes have four multi-subunit protein complexes: photosystem II (PSII), photosystem I (PSI), cytochrome b_6f (cyt b_6f) and ATP-synthase [36]. PSII and PSI are intrinsic pigment–protein complexes spatially separated in the stacked (grana) and unstacked (stromal lamellae) regions of the thylakoid membranes in chloroplasts. PSII catalyzes the oxidation of water and the reduction of plastoquinone [37,38]. Cyt b_6f complex accepts an electron from plastoquinol and donates it to PSI via plastocyanin. The electron is transferred ultimately to the $NADPH^+$ via PSI, and then released to the stroma to be utilized in the CO_2 reduction process. Concomitantly with the electron transfer, the accumulated protons in the lumen are used for ATP synthesis by ATPase.

Production of NO in plant cells arises from several different pathways and in different organelles [30,39,40] including mitochondria [40], peroxisomes [41] and the chloroplast [27,28]. Dependant on concentration, NO can provoke both beneficial and harmful effects which also depend on its location within the plant cell [42,43]. In plants, NO can be produced from nitrite [44], from L-arginine by NOS-like biochemical pathways [41] and from S-nitrosoglutathione by decomposition [45]. Endogenous NO generation in chloroplasts without any external donors has been recently reported [27,28]. It is notable that NO synthesis was first detected in chloroplasts in response to various stressors like high temperature, salinity, iron, elicitors or osmotic stress, using NO-sensitive diaminofluorescein probes [46–48].

Nitric oxide synthase (NOS) activity was first detected in fungi and a higher plant by Ninnemann and Maier [49]. Despite evidence of an Arg-dependent pathway for NO synthesis in higher plants, no NOS homologs have been identified in plants [50]. Guo et al. [51] described the identification of a plant NOS gene involved in hormonal signalling (Atnos1); however, since no NOS activity was detected in the purified AtNOS1 protein [52,53], it was renamed AtNOA1 (nitric oxide associated 1), because it appeared essential for NO generation in the cell. Another enzymatic source of NO in plants is the cytosolic nitrate reductase (NR) [12] and the NO so produced can readily diffuse into the chloroplast stroma [43]. Nitrite can also be reduced to NO non-enzymatically at acidic pH values [54]. Non-enzymatic, light-mediated conversion of nitrogen dioxide to nitric oxide by the plastid carotenoids has also been documented [55].

3. Target sites of NO action in chloroplasts

The effect of NO on target sites in the chloroplast and on its photosynthetic apparatus has mostly been accumulated by applying exogenous gaseous NO or by various NO donors on leaves, leaf discs, leaf extracts, intact chloroplasts or isolated chloroplast components. The results are often confusing and contradictory but, in this review, an effort is made to clarify such discrepancies.

3.1. Effects of NO on photosystem II

3.1.1. Electron transport chain

Several electron transport chain components of PSII have been identified as target sites for NO in chloroplasts. EPR and chlorophyll fluorescence analysis of isolated PSII-enriched membranes and chloroplasts treated with NO gas demonstrated NO binding to the non-haem iron of PSII between Q_A and Q_B quinone binding sites, namely, $Q_AFe^{2+}Q_B$ [56,57], at the catalytic Mn cluster of the oxygen evolving complex (OEC) [58] and at the Y_D tyrosine residue of D2 protein [59]. The proposed target sites of NO binding are illustrated in Fig. 1.