Nitric Oxide in Plants

A Molecule with Dual Roles

Edited by:
Mohammad Abass Ahanger
Parvaiz Ahmad

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Preface

Plant growth and development is significantly influenced by environmental factors. Due to rapid global climate change both biotic and abiotic stresses have intensified and so have the deleterious effects on normal plant growth and productivity. Environmental stress-mediated decline in plant productivity imparts significant pressure on global food security, and therefore threatens the likelihood of serious food crises in the near future for the increasing world population. Stresses result in oxidative damage through excessive generation of reactive oxygen species (ROS), thereby inducing the oxidation of lipids, proteins, and nucleic acids. Plants have evolved key mechanisms to counteract the damaging effects of stresses. Osmolyte and secondary metabolite accumulation, the antioxidant system, phytochelatin production, ion compartmentation and exclusion, etc. are some of the key mechanisms to counteract the stress factors. All these tolerance mechanisms are regulated at the gene and protein level. Plants exhibiting upregulation of the tolerance mechanisms show better performance in terms of photosynthesis, mineral uptake and assimilation, enzyme functioning, and hence yield productivity. However, it should be mentioned here that enhancing the tolerance potential by employing efficient management practices can be very effective in protecting the yield potential of plants. Every tolerance pathway mentioned above is a combination of many components, which could be organic molecules or enzymes or metabolites. From the past decade onward there has been increasing advocacy for exploiting these individual beneficial molecules to improve the tolerance pathways. In this respect exogenous usage of the key components of tolerance pathways means either foliar or through the roots. Nitric oxide (NO) is a gaseous signaling molecule that was considered toxic for plant metabolism; however, advances in research have confirmed its beneficial role in stress tolerance through its role in signaling and regulation of key developmental events including germination and programmed cell death. Fine-tuned mechanisms elicited by NO have been confirmed through metabolomic, transcriptomic, and proteomic studies. Identification of genes and other key molecules interacting in NO-mediated growth and developmental regulation under stressful conditions is being investigated. Modulations in the endogenous NO concentrations, either through stress exposure or by exogenous application of protectants, confirm the role of NO in plant stress management. In addition, physiological and biochemical studies have confirmed the vital role of optimal NO concentrations in regulation of photosynthesis, carbon assimilation, osmolyte synthesis, antioxidant and secondary metabolite metabolism, and nutrient uptake and assimilation. Interactions with phytohormones like abscisic acid, ethylene, salicylic acid, jasmonic acid, and cross-talk with other signaling molecules have been reported to play pivotal roles in

NO-mediated stress tolerance in plants. Keeping in view the above mentioned facts this book is compiled with the aim of providing the scientific community with the latest updates and future goals of NO research. *Nitric Oxide in Plants: A Molecule with Dual Roles* has 13 chapters, with every chapter having updated information about the relevant topic. The book aims to fill the existing knowledge gap in NO and plant metabolism regulation.

Chapter 1 provides an overview of the biosynthesis and regulation of NO synthesis in plants vis-à-vis its regulatory role in plant growth regulation. In addition the interaction of NO with certain key stress molecules is also discussed. Chapter 2 examines the enzymatic and nonenzymatic biosynthesis of NO, and the influence of different stress factors on NO synthesis. Moreover, modulations in NO synthesis due to osmolytes are considered, and a brief discussion about signaling components including transcription factors and phytohormones is also included. Chapter 3 discusses the reductive and oxidative pathways of NO synthesis, and the physiological, biochemical, and molecular modulations resulting from interactions of NO with auxin, gibberellic acid, cytokinin, ethylene, and abscisic acid. NO-mediated regulation of growth, photosynthesis, and tolerance mechanisms under different environmental stresses are also discussed. Chapter 4 reviews the molecular interventions for enhancing NO synthesis for its optimum exploitation for plant growth enhancement. NO-mediated regulation of phytohormones and fatty acids vis-à-vis signaling mechanisms is also the subject of this chapter. In addition, gene expression modulation and stress tolerance are examined. The role of nitrogen in production of NO and the subsequent regulation of major plant cellular pathways are described in Chapter 5. The role of beneficial microbes in NO production under normal and changing environmental conditions are dealt in Chapter 6, with a focus on physiological-, biochemical-, and molecular-level tolerance mechanisms. In addition, the interactive effects of ROS and NO in tolerance to biotic and abiotic stresses are mentioned. There is also a focus on the contribution of nitrifying and denitrifying bacteria in NO production and subsequent alterations in gene expression, as well as the role of polyamines. Chapter 7 discusses the synthesis, metabolism, and transport of ROS and reactive nitrogen species (RNS). The role of reactive oxygen, sulfur, and nitrogen species in stress signaling and the underlying cross-talk for tolerance against abiotic and biotic stresses are explained. Chapter 8 deals with understanding the role of endogenous and exogenous NO in modifying the key antioxidant pathway – the ascorbate–glutathione cycle. The role of NO-induced changes in enzymatic and nonenzymatic components of the ascorbate–glutathione cycle in plant growth regulation is examined. The authors also explain the posttranslational modifications of NO and their role in signaling and stress tolerance. In Chapter 9 the authors take the opportunity to explain the cross-talk mechanisms underlying environmental stress tolerance mediated by NO and phytohormones like auxins, cytokinins, gibberellins, abscisic acid, ethylene, jasmonic acid, salicylic acid, and brassinosteroids. The regulation (positive and negative) of several signaling pathways and the elicitation of tolerance mechanisms for improved plant performance are also discussed. Phytohormone–NO cross-talk for improved nutrient acquisition and photosynthetic regulation is the topic of Chapter 10. In addition the authors also examine the antagonistic and synergistic influence of NO on phytohormone synthesis, photosynthate partitioning, gene expression, and programmed cell death regulation. Chapter 11 covers the role of polyamines in NO synthesis and their interactive effects in alleviating the damaging effects of stresses. Polyamine metabolism-induced ROS production and

subsequent triggering of NO accumulation are also discussed. In Chapter 12 the authors consider the NO-mediated modulation of the synthesis and transport of auxins, abscisic acid, and brassinosteroids under normal and adverse environmental conditions. In addition the molecular mechanisms of the signaling events resulting from their interactions are discussed. Finally, Chapter 13 looks at the interactive effects of NO and salicylic acid, jasmonic acid, and ethylene, with a focus on the antioxidant functioning of plants with regard to stress tolerance. Posttranslational modifications by NO and its cross-talk with salicylic acid, jasmonic acid, and ethylene for regulation of several downstream targets are discussed.

This volume presents a wealth of knowledge on the current understandings of NO and growth regulation in plants, and provides insights into the abiotic and biotic stress tolerance mechanisms regulated by NO. The chapters included in this volume are the authors' own work and necessary editorial modifications were incorporated wherever the need was felt. We have tried our best to gather the most up-to-date information about NO research; however, some areas are inevitably missing and there is always the possibility of errors creeping into the book. Therefore, we seek the readers' indulgence, and suggestions are welcome for improvements in future editions. We are very thankful to the well-versed contributors who accepted our invitation and contributed chapters despite the hardships created by the pandemic. Moreover, we extend our sincere thanks to the entire team at Wiley, especially Kerry Powell, Rebecca Ralf, and Camille Bramall, for their invaluable suggestions and constant help in accomplishing this project and its publication.

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1

Nitric Oxide: A Dynamic Signaling Molecule Under Plant Stress

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1.1 Introduction

Nitrogen monoxide, often known as nitric oxide, is a physiologically active chemical that is widely used in animal and plant signaling mechanisms. In plants and animals, it is an intracellular and intercellular signaling molecule with a variety of regulatory roles. Its functions in the central nervous, cardiovascular, and immunological systems, platelet inhibition, programmed cell death, and host responses to infection, among other things, have been widely studied in animals.

Because of the presence of an unpaired electron, it is a highly reactive gaseous molecule that occurs with oxygen in a variety of reduced states such as nitroxyl ion (NO^-), nitric oxide free radical ($NO^\bullet$), and nitrosonium (NO^+). These NO-derived molecules are referred to as reactive nitrogen species (RNS). NO influences signaling in biological systems through a variety of mechanisms. The interaction of $NO^\bullet$ with O_2 results in the formation of several redox compounds (including $NO_2^\bullet$, N_2O_3, and N_2O_4), which may react with cellular amines and thiols or simply change to form the metabolites nitrogen dioxide radical (NO_2^-) and nitrate (NO_3^-) (Wendehenne et al. 2001). NO combines with dioxygen to form NO_2 or with reactive oxygen species (ROS) to form peroxynitrite ($ONOO^-$), which triggers cellular damage. $NO^\bullet$ facilitates electrophilic assault on reactive sulfur, oxygen, nitrogen, and aromatic carbon centers, with thiols being the most reactive of the reactive teams. Nitrosation is the name given to this natural process. Nitrosation of numerous enzymes or proteins results in chemical change, which may affect the function of those entities. These alterations are reversible, and supermolecule nitrosation–denitrosation might be a crucial mechanism for controlling signal transduction (Hayat et al. 2010).

In contrast to the mammalian system, the cellular/subcellular localization of NO production in plants is exceedingly diverse and contentious. The production of NO in plants is determined by the plant's physiological condition. This includes NO production during root development, stomatal movement control, blooming, plant component expansion, and leaf senescence (Neill et al. 2002; Mishina et al. 2007). NO is produced in plants through nonenzymatic and accelerator systems, depending on the plant species, organ, or tissue, as well as the plant's state and ever-changing environmental circumstances. The most effective recognized NO sources in plants are as a substrate by cytosolic (cNR) and

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membrane-specific nitrate enzyme (PM-NR), and NO synthesis by many arginine-dependent gas synthase-like activities (NOS).

According to studies, mitochondria are a major source of arginine- and nitrite-dependent NO synthesis in plants. Tischner et al. acquired the first evidence for mitochondrial NO synthesis in plants when they assessed NO production under anoxic conditions from the unicellular blue green alga *Chlorella sorokiniana* (Tischner et al. 2004). This green alga does not create NO when exposed to nitrate (NO_3), but it does create NO when exposed to nitrite (NO_2). NO generation was also inhibited by mitochondrial electron transport inhibitors. Shortly after, mitochondrial NO synthesis in higher plants was discovered. Gupta et al. discovered mitochondrial NO production in barley plants grown in anoxic conditions (Gupta and Kaiser 2010). Under anoxic circumstances, a tobacco Nia 1, 2 (nitrate reductase-deficient) cell suspension was able to manufacture NO from exogenous nitrite, despite the absence of nitrate reductase (which can also manufacture NO from nitrite) (Gupta et al. 2011). Other putative NO producers in plants include xanthine oxido-reductase, peroxidase, and cytochrome P450. NO is a ubiquitous chemical that is found in all eukaryotes. The NR system is by far the most effective and well-characterized mechanism for NO generation in plants. In this case, the cytosolic NR mostly catalyzes the reduction of nitrate to NADH as the predominant negatron donor. NR's NAD(P)H-dependent NO production has been demonstrated in vitro and in vivo (Rockel et al. 2002). The biological significance of NR activity as a source of NO was first shown in *Arabidopsis* guard cells by Desikan et al. (2002). The peroxisomal catalyst organic compound enzyme can also catalyze group reduction to NO (XOR). XOR activity in pea (*Pisum sativum*) leaves is linked to peroxisomes, and as a result, the possibility of interaction between the construction of reactive chemical elements and reactive gas species (ROS and RNS, respectively) has been suggested (del Río et al. 2004). NO production in animals was demonstrated by a chemical reaction of arginine transforming into citrulline mediated by the enzyme NO synthase (Palmer et al. 1987). Following the discovery of a purpose for NO in plants in 1998 (Delledonne et al. 1998; Durner et al. 1998), several researchers began to look for NOS activity in plants, despite the fact that the *Arabidopsis thaliana* ordering failed to reveal any factor with significant similarity to animal NOS (Moreau et al. 2010).

1.1.1 Historical Evidence and Biosynthesis of Nitric Oxide

Several studies have been conducted over the past several decades to investigate the presence and characteristics of NO gas in living beings. Gas, as a versatile molecule, has piqued curiosity and opened up new opportunities for research. NO gas is an atom gas with well-defined communication roles in mammalian systems, serving as a second messenger during vasorelaxation, neurotransmission, immunity, and toxicity. It is now clear that NO performs a critical role in animal physiology. Because of its extensive biological relevance, NO was designated "Molecule of the Year" in 1992 by *Science*, and Furchgott, Murad, and Ignarro were given the Nobel Prize in Physiology and Medicine in 1998. Furchgott discovered in 1980 that an unknown molecule found in animal tissue could relax smooth muscle cells, and he dubbed it EDRF (endothelium-derived reposeful factor). Murad discovered years ago that vasodilators activate guanylate cyclase (GC), which creates cyclic guanosine monophosphate and relaxes muscle fibers. This discovery begged the question of how a

vasodilator outside the cell could influence a catalyst inside the cell. The solution was that the vasodilator was tainted with NO residues. Murad then bubbled NO gas across swish muscle cells, activating gigacycles per second. Thus, even before eukaryotes were thought to produce NO, he postulated that hormones may regulate swish muscles via NO. Years later, Ignarro demonstrated that NO has comparable chemical behavior to EDRF and is, in fact, a twin of EDRF. NO release from plants was initially suspected by Klepper in soybeans in 1975, much earlier than in mammals (Klepper 1979). The vast biological importance of gas in plants was established in the 1990s (Gouvea et al. 1997; Leshem et al. 1998).

In animals, NO has since been recognized as an important signaling molecule in maintaining blood pressure within the circulatory system, stimulating host defenses within the system, controlling neural transmission within the brain, controlling organic phenomena, protoplasm aggregation, learning and memory, male sexual function, toxicity and cytoprotection, the development of artery hardening, and a variety of other functions. It functions as a secondary transmitter in mammalian systems during vasorelaxation, neurotransmission, immunity, and toxicity. As a result, they play critical roles in animal physiology.

However, unlike animal physiology, the physiology and chemical chemistry of NO in plants is less well known. NO has the potential to be a dynamic bioactive molecule that plays an important physiological role in plants and animals.

1.1.2 NO Biosynthesis in Plants

The process of NO production has been explored in a variety of organisms, including microorganisms, alga, lichens, gymnosperms, and angiosperms (Rőszer et al. 2014). NO synthesis utilizes both accelerator and nonaccelerator mechanisms. Body parts, plastids, mitochondria, and peroxisomes are important sites for NO production (Rőszer 2012a, 2012b). Furthermore, multiple organelles, including protoplasm, cell wall, endoplasmic reticulum, and apoplast, generate NO in higher plants (Fröhlich and Durner 2011). Chakraborty and Acharya (2017) distinguish between subtractive and aerobic NO production mechanisms. The protoplasm, mitochondria, plastid, peroxisomes, and apoplast are the primary sites of subtractive NO_X production, which is mediated by the nitrate enzyme or mitochondrial negatron transport chain and deoxygenated proteins containing heme. The aerobic route of NO production begins with L-arginine, which appears to include the enzyme NO synthase. Despite the fact that numerous genes and proteins coding for NOS enzymes are known in the class system, prokaryotes, and eukaryotes, the kingdom Plantae is still little characterized (Figure 1.1).

1.2 The Function of Nitric Oxide in Plants

Nitric oxide is a common chemical molecule that is important in plant physiological activities. NO sources are useful in breaking dormancy and boosting seed germination in a variety of crops (Bethke et al. 2007; Giba et al. 2007; Prado et al. 2008; Albertos et al. 2015; Sanz et al. 2015). Similar research has been done to show the impact of organic nitrates in enhancing light-dependent and phytochrome-regulated germination in *Pauwlonia tomentosa* and *Stellaria* medium (Grubisic et al. 1992; Jovanovic et al. 2005). NO is involved in the

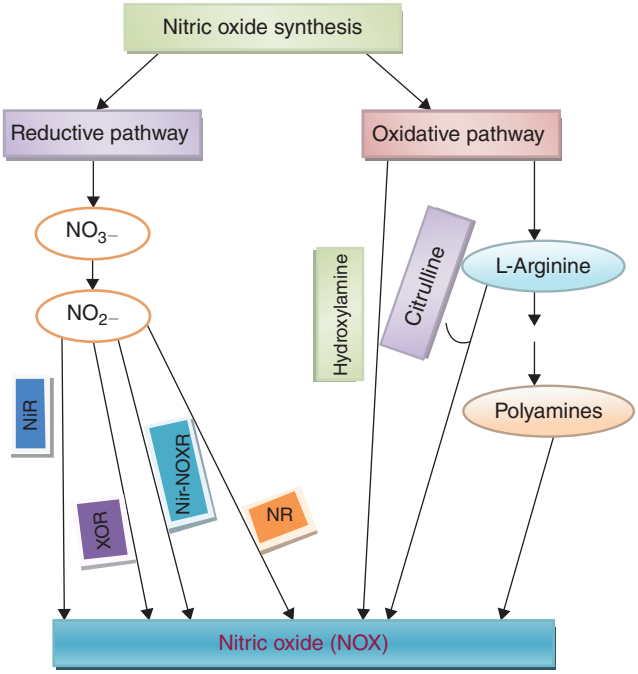

Figure 1.1 Diagram representing NO synthesis. NO_3–, nitrate; NiR, plastidial nitrate reductase; XOR, xanthine oxido-reductase; NiR-NOR, nitrite reductase; NR, nitrate reductase; PA, polyamines.

regulation of catalase, ascorbate peroxidase, and aconitase activities (Clarke et al. 2000; Navarre et al. 2000), in cell wall lignification (Ferrer and Ros Barcelo 1999), the regulation of ion channels of guard cells(Garcia et al. 2003), mitochondrial and chloroplastic functionality (Yamaski et al. 2001), cell death (Pedroso et al. 2000), senescence (Hung and Kao 2003), accumulation of ferritin (Murgia et al. 2002), wound signaling (Orozco-Cardenas and Ryan 2002), cytokinin-induced programmed cell death (Neill et al. 2003), and abscisic acid (ABA)-induced stomatal closure (Neill et al. 2002). NO mediates maturation and senescence, operates on ethylene antagonism (Lamattina et al. 2003), induces increase of flavonoid production in *Camellia sinensis* L., and endogenous NO stimulates brassinosteroid (Li et al. 2017). Nitric oxide has a visible part to play in the formation of plant roots and shoots. It promotes root and plant development in many plants (Corpas et al. 2006, 2015). Controlling morphogenesis, growth, and development in plants requires targeted NO control (Hebelstrup et al. 2013). NO is primarily necessary for the establishment of plant–microbe interactions, which regulate N_2-fixing symbiotic relationships and nodule senescence (Hichri et al. 2015). NO influences senescence in several plant species, including *Arabidopsis*, tobacco, pea, wheat, and others (Procházková et al. 2011), as well as flower formation in olive and *Arabidopsis* (Seligman et al. 2008; Zafra et al. 2010; Procházková et al. 2011). Nitric oxide is a crucial chemical in agriculture. It promotes seed germination, reduces postharvest losses by delaying fruit ripening, and improves the shelf life and quality of cut and detached flowers. NO also increases the activity of antioxidant enzymes in plants, which influences RNS and ROS metabolism (Corpas and Palma 2018).

1.3 NO's Role in Biotic Stress

Several studies conducted within the past decade have revealed that NO is engaged in communicating defense responses during plant–pathogen interactions. Pathogen challenges typically result in hypersensitization (HR). There is evidence that NO, in addition to forming reactive oxygen intermediates (ROIs) and salicylic acid (SA), plays a vital communication function throughout life (Delledonne et al. 1998; Durner and Klessig 1999).

Hypersensitized necrobiosis via NO is a prominent example of programmed cell death (PCD). NO-donor treatment of plant structure has been found to trigger chromatin granule condensation and dioxyribonucleic acid fragmentation (Clarke et al. 2000; Pedroso et al. 2000). Furthermore, NO-induced necrobiosis may be silenced by an animal caspase-1 molecule (Clarke et al. 2000). Despite studies demonstrating proteolytic enzyme activity in plants (D'Silva and Poirier 1998; Hatsugai et al. 2004; Rojo et al. 2004), associated transgenic plants with an overexpression of a proteolytic enzyme inhibitor – protein p35 and Op-IAP – show homologous recombination (HR) inhibition (Dickmann et al. 2001; Del Pozo and Lam 2003).

Plants have practical homologs of animal caspases, known as metacaspases (Bozhkov et al. 2005). Belenghi et al. (2007) recently demonstrated that *A. thaliana* metacaspase nine (AtMC9) is rendered inactive by *S*-nitrosylation of a cysteine residue in AtMC9. In turn, the mature form of this necrobiosis fiduciary is resistant to *S*-nitrosylation by NO. Exogenous NO promoted necrobiosis in *Arabidopsis* suspension cells in amounts comparable with those produced by cells challenged with an avirulent bacterium (Clarke et al. 2000). However, reactions between NO and H_2O_2 produce either singlet oxygen or free radicals (Noronha-Dutra et al. 1993), which might cause necrobiosis. A simultaneous rise in NO and H_2O_2 triggered necrobiosis in soybean and tobacco cell suspensions, but a rise in only one of the preceding variables promoted necrobiosis very modestly (Delledonne et al. 1998; de Pinto et al. 2002). Furthermore, microscopic anatomy studies revealed that either injection of NO donors or a change in H_2O_2 level has no effect on HR in infected oat cells, despite the fact that each molecule was required for the initiation of death in neighboring cells (Tada et al. 2004). The mechanism by which NO and H_2O_2 kill remains mostly unclear. The reaction of NO with O_2 creates peroxynitrite, an exceedingly poisonous chemical for animal cells that mediates necrobiosis. $ONOO^-$ is relatively nontoxic to plants (Delledonne et al. 2001). However, it has been shown that $ONOO^-$ stimulates pathogenesis-related protein (PR-1) accumulation in tobacco leaves (Durner and Klessig 1999) and supermolecule nitration modulates cell oxido-reduction status (Delledonne et al. 2001). Furthermore, it was demonstrated that whereas peroxynitrite was responsible for the death of most *Arabidopsis* cells in response to avirulent *Pseudomonas syringae*, scavenging of this ion did not result in efficient defense against avirulent bacterium (Alamillo and Garcia-Olmedo 2001).

NO, when rebuilt into a peroxynitrite particle, may join forces in killing microorganisms (Durner and Klessig 1999; Romero-Puertas et al. 2004), though it has not been determined if NO and its derivatives are directly hazardous to plant diseases (Garcia-Olmedo et al. 2001). In vitro, it was undeniable that the growth of virulent and avirulent *Pseudomonas* bacterium was stifled by both NO and the plant system producing peroxynitrite (sodium nitroprusside + hypoxanthine/xanthine oxidase) (Noronha-Dutra et al. 1993; Garcia-Mata and Lamattina 2002). Romero-Puertas et al. (2004) proposed that $ONOO^-$ might be

continuously synthesized in healthy cells, implying that plants may evolve certain detoxifying mechanisms. Ascorbates may have a significant role in the inactivation of $ONOO^-$ in animal cells (Arteel et al. 1999). Given that vitamin C (AsA) is a quantitatively dominant inhibitor in plant cells (Smirnoff 2000), it is possible that AsA also contributes to $ONOO^-$ breakdown in plant cells. The genetic composition of the plant (R genes) and the microbe (avr) seems to be strongly related to early NO production, referred to as NO burst (Mur et al. 2005; Bennett et al. 2005). Prompt gas generation (30–45 min after inoculation) was seen in noncompatible systems of *P. s*. pv. *phaseolicola*–tobacco and *P. s*. pv. *tomato–Arabidopsis*. This early NO burst happened six hours before the appearance of obvious HR-type death signs and directly preceded H_2O_2 production. As proof, plants injected with a mutant of the avirulent bacterium (hrp) incapable of supplying the avr supermolecule to the plant showed a lack of NO emission (Mur et al. 2005).

The use of fluorescent dyes in cytochemical methods has made it possible to present the mechanics of NO generation inside the stratum of tobacco leaves treated with cryptogein. After tissue treatment, a supermolecule elicitor derived from the morbific plant life *Phytophthora cryptogea* stimulated NO buildup at several minute intervals (Foissner et al. 2000). In turn, Prats et al. (2005) discovered a significant, transient rise in NO level before programmed death of barley dermal cells infected with *Blumeria graminis* f. sp. *Hordei* using DAF-2DA (5,6-diaminofluorescein diacetat) dye. Furthermore, Zeier et al. (2004) isolated an *Arabidopsis* transgenic line with overexpression of nitric oxide dioxygenase (NOD) gas, an accelerator catalyzing the twofold reaction of NO to nitrates. Transgenic plants treated with an avirulent strain of *P.s.* pv. *tomato* avr B showed decreased NO production and a significantly lower mortality rate, confirming that NO is required for HR stimulation. According to Modolo et al. (2006), *P. syringae* is diminished concurrently in NR-deficient double mutants (Nia1 and Nia2) of *Arabidopsis* because these plants lack L-arginine and NO_2, endogenous precursors for NO production.

Until recently, it was thought that HR is only seen in incompatible relationships, where the plant has a resistance gene encoding R and the microbe has a virulence gene, avr (Levine et al. 1994). However, it has recently been demonstrated that HR of host cells may also arise in plants to partially protect against a specific microbe and only in the situation of non-host-kind resistance (Vleehouwers et al. 2000).

The majority of the evidence demonstrating that NO works as a messenger in gene-for-gene defensive responses was gained by studying completely separate plant–biotrophic microbe systems (Delledonne 2005). It has yet to be confirmed what part NO plays inside the plant and the necrotrophic pathogen. In a review in 2004 Van Baarlen noted that the formation of endogenous NO and H_2O_2 was recorded in distinction to the compatible interaction, i.e. during disease development of liliaceous plant and *Botrytis elliptica*. Another event associated with plant resistance in which NO appears to be involved is phytoalexin buildup (Able 2003). Exogenous NO stimulated the accumulation of rishitin in potato tubers. Furthermore, the outcome of this compound's inhibited production was identified after the use of a NO scavenger (Noritake et al. 1996). Several times after NO treatment of soybean cotyledons, the production of particular phytoalexins was identified (Modolo et al. 2002). NO could also play a role in the emergence of general systemic acquired resistance (SAR). In tobacco, exogenous NO increases the accumulation of salicylic acid, which plays a key role in SAR (Durner et al. 1998). Activation of the PR-1 macromolecule, produced by

NO, occurs with the participation of SA, because an identical result was not seen in transgenic plants unable to accumulate SA (NahG). Furthermore, disease spots formed by tobacco mosaic virus (TMV) on leaves pretreated with NO were dramatically decreased as compared with transgenic plants. SAR was lowered by using inhibitors specific for animal NOS or NO scavengers (Song and Goodman 2001). As a result, our findings suggest that NO plays an important role in the development of a distal signal network, resulting in increased SAR in tobacco.

NO may be transferred to plants in the form of nitrosoglutathione (GSNO), much as in mammals' vascular systems (Durner and Klessig 1999). It is hypothesized that GSNO may act as both an intracellular and organismal NO carrier, and that it is distributed throughout the plant via vascular tissue bundles. Glutathione-dependent formaldehyde dehydrogenase (GS-FDH)/I-nitrosoglutathione reductase (GSNOR) may play an important role in turning off/on the NO or GSNO signal, as well as modifying the amount of intracellular thiols, which may cause nitrosative stress (Diaz et al. 2003). In *Arabidopsis*, Feechan et al. (2005) discovered that the deletion of AtGSNOR1, an *S*-nitrosoglutathione enzyme, resulted in an increase in cellular *S*-nitrosothiols, which was correlated with a decrease in resistance to microbial infection. Throughout the prevalence of avirulent microorganisms and the consequent hypersensitized response, NO demonstrates an unusually wide range of affinities to a variety of signaling chemicals. The popularity of an avirulent microbe is associated with a robust aerobic burst, during which there is a redoubled creation of ROS/RNS, primarily superoxide ($O_2^{\bullet}$), peroxide (H_2O_2), or gas (NO), and the commencement of an avirulent microbe. In soybean cell suspension infected with avirulent *P. syringae* pv. *glycinea*, treatment with NO donor, sodium nitroprusside, significantly increased the induction of death by exogenous H_2O_2 or ROS (Delledonne et al. 1998). This response was significantly inhibited not solely by the NO scavenger 2-4-carboxyphenyl-4,4,5,5-tetramethylimidazoline-1-oxyl-3-oxide (CPTIO), but additionally by diphenyleneiodonium, a substance in the leukocyte NADPH enzyme that inhibits the plant oxidative burst, and by an enzyme that destroys H_2O_2; NO reacted synergistically, resulting in a 10-fold increase in ROI-induced death (Delledonne et al. 1998).

NO production, along with H_2O_2 and O_2, was observed to be associated with elicitor-elicited mortality in Mexican cypress. The co-accumulation of ROS and NO, as well as the interplay between H_2O_2, O_2, and NO, mediated the death response. The use of protein scavengers/inhibitors was used to investigate the role of NO and O_2 in death. NO and H_2O_2 reciprocally promoted each other's assembly, whereas NO and O_2 reciprocally suppressed each other's production. The interaction between NO and O_2, but not between NO and H_2O_2, triggered PCD via peroxynitrite ($ONOO^-$) (Neill et al. 2002; Zhao 2005). The stress response in bacterially triggered PCD in soybean and *Arabidopsis* concerned reactions to NO and H_2O_2, with interactions between NO and H_2O_2 being synergistic and additive in different ways, which is consistent with previous findings. After exposing tobacco leaves to a high intensity of stress, NO-mediated death was seen in enzyme-deficient (CAT1AS) plants but not in wild types, revealing the interaction of NO with H_2O_2 throughout the NO-mediated death response (Zago et al. 2006).

Plants protect themselves from microorganisms by triggering elaborate defenses. Similarly, it was recently demonstrated that NO works synergistically with ROS to prolong host mortality in soybean suspension cells, and that NOS inhibitors impair the

hypersensitized resistance response in *Arabidopsis* and tobacco (Delledonne et al. 1998). It was discovered that the host plant has intracellular defense mechanisms to combat the oxidative stress caused by pathogens. Increased activity of the catalyst inhibitor system, total phenol content, and phenyl ammonia lyase activity slowed oxidative burst, which was presumably mediated by excessive chemical group content and hence involved gas signaling; this explained the protection against pathogen stress (Dwivedi et al. 2016). Following that, extensive studies revealed that NO could influence the expression of genes encoding a variety of plant effector and restriction proteins (Polverari et al. 2003; Huang et al. 2004; Parani et al. 2004; Zago et al. 2006). Furthermore, recent research on catalase-deficient tobacco plants discovered a small number of genes that are particularly controlled by NO or H_2O_2 (Zago et al. 2006). Interestingly, most of the notable genes were affected by both NO and H_2O_2, showing a significant overlap in these two separate chemical process signaling agents (Figure 1.2).

1.3.1 Interaction of NO with Other Molecules to Confer Biotic Stress Responses in Plants

Plants defend themselves against microorganism attack by activating elaborate defenses. Similarly, it has been recently demonstrated first that NO acts synergistically with ROS to extend host death in soybean suspension cells, and second that NOS inhibitors compromise the hypersensitized resistance response in *Arabidopsis* and tobacco (Delledonne et al. 1998). NO and ROS induce SA synthesis, and elevated SA may subsequently result in improved NO and ROS levels. SA additionally induces expression of genes that may be NO targets/sensors (e.g. pathogen-induced oxido-reductase [PIOX]). What is more, SA may enhance the results of NO activity through interaction with many NO_X-regulated enzymes (e.g. catalase and aconitase). On the other hand, SA, which is a very important antioxidant in mammals, may counter the effects of NO. As an example, NO blocks respiration via inhibition of cytochrome enzyme, while SA induces production of various NO-resistant enzymes and sensors/targets in plants (e.g. pathogen-induced oxygenase, PIOX). NO and SA have an effect on common targets (e.g. catalase, aconitase). SA counteracts NO action (e.g. NO inhibits cytochrome enzyme, whereas SA induces a different oxidase). The induction of necrobiosis and/or defense factor activation occurs via the action of NO, SA, and ROS. NO is not only thought to perform throughout the event of hypersensitive cell death but is also needed in the establishment of disease resistance. Melatonin also enhances pathogen resistance in plants by increasing the levels of SA and ethylene. Lee et al. (2015) reported that decreased level of melatonin content in *Arabidopsis* mutant lowers the SA content thereby reducing resistance to pathogen infection. It also stimulates elevation of endogenous NO levels and increased expression of defense-related genes, along with disease resistance against Pst DC3000 infection.

Melatonin confers plants with innate immunity via the NO- and H_2O_2-dependent pathway by stimulating mitogen-activated protein kinase kinase kinase 3 (MAPKKK3) and oxidative signal-inducible1 (OXI1). Melatonin stimulates accumulation of NO and SA and improved tomato TMV resistance (Zhao et al. 2019).

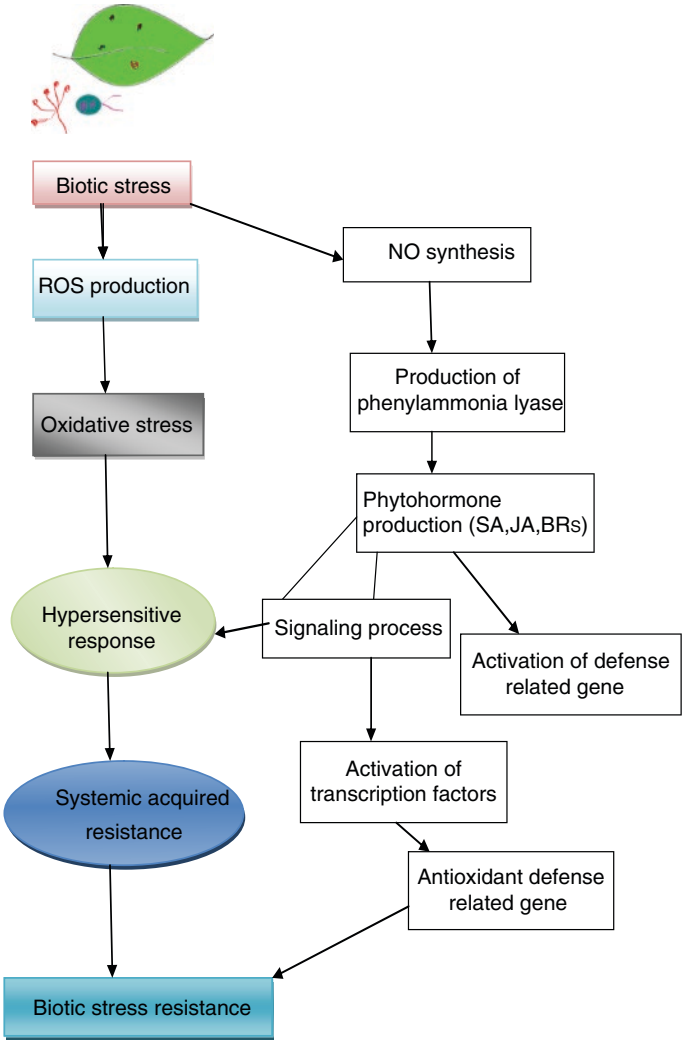

Figure 1.2 Schematic representation of NO signaling against biotic stress in plants.

Interaction between NO and glutathione is an important component to the NPR1-dependent defense signaling pathway in *A. thaliana* (Kovacs et al. 2015). Cao et al. (2019) found that brassinosteroids aid in stimulating the susceptibility of maize to maize chlorotic mottle virus infection in a NO-dependent manner. Nitric oxide further acts as a downstream signaling molecule in brassinosteroid-mediated virus susceptibility to maize chlorotic mottle virus in maize. The brassinosteroids provide systemic resistance against viruses along with H_2O_2 and NO in *Nicotiana benthamiana* (Deng et al. 2016). However, several studies have demonstrated the integrative role of brassinosteroids with NO in response to pathogen attack in various species (Hayat et al. 2010; Shi et al. 2015; Zou et al. 2018; Kohli et al. 2019).

1.4 Nitric Oxide's Role in Abiotic Stress

Existing climatic aberrations cause plants to be exposed to a variety of abiotic stresses such as drought, temperature increases, salinity, and oxidative damage. Because plants are sessile, they must deal with changing environmental conditions throughout their lives. Though plants develop various strategies to cope with adverse conditions, plant scientists and researchers are discovering new methods to learn about plants in stressful environments. In the presence of salt, substantial metals, drought, and oxidative stress, NO plays a critical role in abiotic stress management (Zhao et al. 2001, 2004; Uhida et al. 2002; Kopyra and Gwóźdź 2003). Abiotic stressors are a key stumbling block to agricultural growth while also jeopardizing food security (Figure 1.3). They also jointly

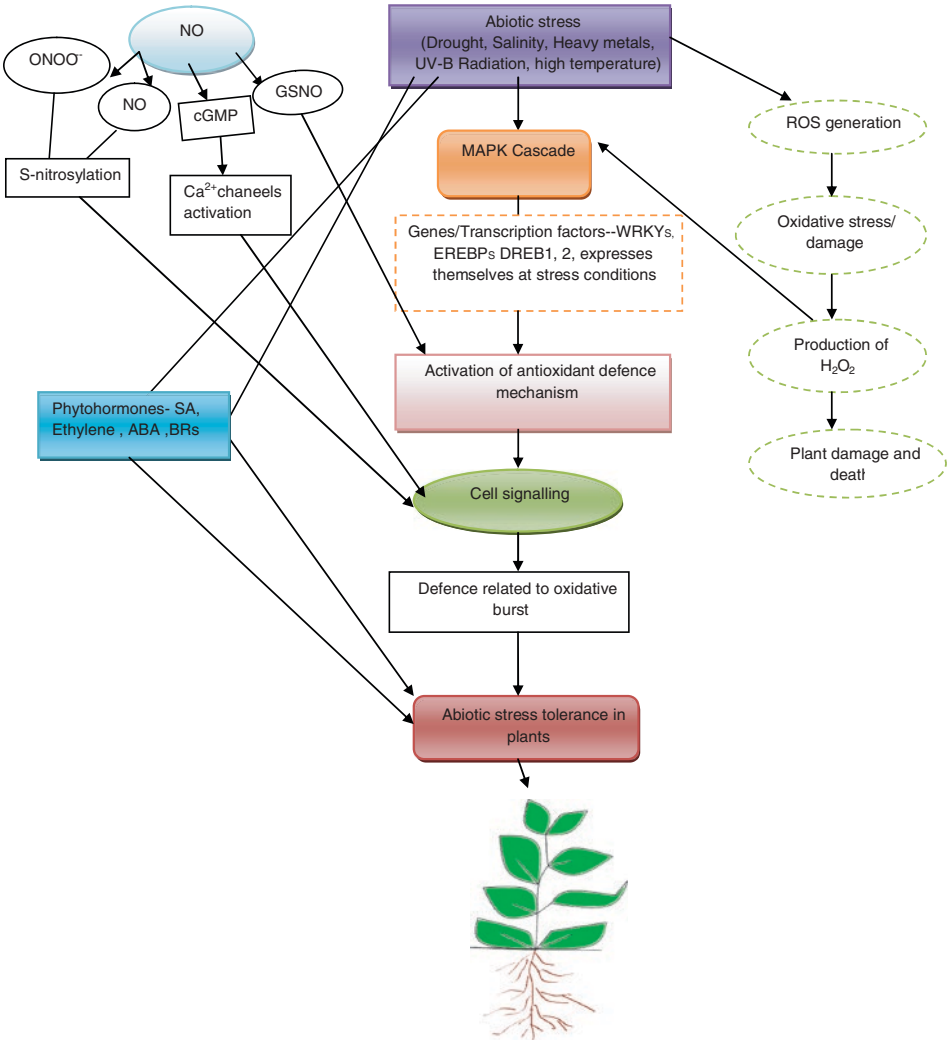

Figure 1.3 Schematic illustration of NO signaling in abiotic stress.

influence the metabolism of plants and the defense of inhibitors. Recent NO studies show that they play a serious role in plant growth and mitigate environmental stress (Aroca et al. 2015; Corpas et al. 2019). Drought tolerance of cut leaves and seedlings of wheat was improved by exogenous NO application (Garcia-Mata et al. 2001). NO also mediates drought tolerance by the activation of numerous enzymes and plant metabolism inhibitors (Filippou et al. 2014; Shi et al. 2014). In many species, NO mediates stomatal rotation and defense methods in water stress (Greco et al. 2012; Garcia-Mata and Lamattina 2013; Chen et al. 2016). ROS and NO work synergistically to address the stress in water that causes ABA synthesis in wheat roots (Zhao et al. 2001). As proof of this, ABA accumulation under stress was prevented by the administration of ROS scavengers. The accumulation of NO tested jointly is needed in broad bean throughout ABA-induced stomata closure (Garcia-Mata and Lamattina 2002). Heat treatment of alfalfa cells, for example, has led to an increase in NO synthesis, while the application of exogenous NO improved cold tolerance in tomato, wheat, and maize (Neill et al. 2003). The observed effects were most likely associated with NO's antioxidative action, which amplifies the negative effects caused by the intensification of peroxidative metabolism in high temperatures (Neill et al. 2002).

Mackerness et al. (2001) demonstrated the involvement of NO in plant response to UV-B radiation, demonstrating poststress induction of chalcone synthase expression, a rise in NOS-type protein activity, and an increase in NO levels. According to the findings of Shi et al. (2005), NO positively shields plants against UV-B radiation, most likely through increased activity of the antioxidative system. NO-donor treatment of potato tubers prior to UV-B irradiation resulted in the development of approximately 50% more healthy leaves than plants not subjected to NO treatment (Neill et al. 2003). Exogenous NO has been shown to reduce the deleterious effects of heavy metals, ethylene, and herbicides on plants in response to alternative abiotic stresses (Kopyra and Gwóźdź 2003). The authors explained the protective effect as a result of NO-donor treatment of plant materials by the effect of NO on the elevation of activity of antioxidative enzymes, particularly SOD (Kopyra and Gwóźdź 2003).

According to the cited authors, such a chain of events could effectively reduce the amount of ROS produced during stress, thereby limiting oxidative stress in plant cells. Similarly to salinity stress, NO-donor treatment of rice seedlings resulted in loss minimization (Uhida et al. 2002). When NO was used, plant growth was increased, along with the maintenance of acceptable photosystem II activity, an increase in antioxidative protein activity, and thus the expression of specific salinity stress resistance genes. On the other hand, prolonged stress situations may result in the production of NO and NO-derived products, resulting in specific responses, referred to as nitrosative stress. Valderrama et al. (2007) found that salinity stress elicited the assembly of RNS, i.e. NO, GSNO, and RSNO, as well as an increase in tyrosine-nitrated proteins, which are sensitive markers of nitrosative stress, in olive leaves. Furthermore, they demonstrated that vascular tissues may play an important role in the distribution of NO-derived forms during nitrosative stress and in signaling processes. Tissue damage, which is usually due to a microorganism invading a cell, frequently results in NO production and H_2O_2 accumulation (Delledonne et al. 1998). According to Orozco-Cardenas and Ryan (2002), injury does not induce NO synthesis; however, the use of exogenous NO inhibits the method of NO generation and expression of wound-inducing genes.

It is important to note that many types of abiotic stress (cold and heat stress, salt and drought stress) increase polyamine (PA) synthesis (Bouchereau et al. 1999). Tun et al. (2006) discovered that PAs significantly increase NO generation. Work on *Arabidopsis* seedlings confirms that NO acts as a channel between PA-mediated stress responses and an alternative stress mediator, with NO as a stepping stone.

By scavenging ROS, NO plays a significant role in inhibitor defense against many abiotic stresses. Salinity stress has a negative impact on plant morphological traits and diffusion balance. Additionally, it promotes membrane disintegration, DNA damage, particle discharge, and death. NO has been shown to have a potential effect on diffusion stress tolerance as well as increased spermatophyte growth in rice, lupin, and cucumber when subjected to salt stress (Uchida et al. 2002; Kopyra and Gwóźdź 2003; Fan et al. 2007, 2013; Barakat et al. 2012). Similar evidence has been reported demonstrating the potential effect of NO in mitigating salinity stress in alfalfa, barley, jatropha, chickpea, and sunflower (Nabi et al. 2019). In *Oryza sativa*, a NO donor SNP inhibited accumulation, reduced ROS generation, and improved root growth (Kushwaha et al. 2019). In various plant species, the potential role of gas in mitigating serious metal toxicity has been investigated (Ahmad et al. 2018; Yuanjie et al. 2019; Bhuyan et al. 2020; Wei et al. 2020; Khator et al. 2021). As a result, it is clear that NO gas could be a potential mitigating agent for abiotic stresses.

1.4.1 Crosstalk of Nitric Oxide with Other Phytohormones in Plants to Confer Abiotic Stress Tolerance

NO is involved in advanced signal mechanisms, as well as synergistic collaboration with phytohormones and alternative secondary signal molecules, to confer stress tolerance in plants. NO has been linked to a variety of phytohormones, including gibberellins, brassinosteroids, and ABA. The interaction of NO with phytohormones has been studied in a variety of ways. NO and plant hormones work together to regulate a wide range of physiological responses in plants. By activating Ca^{2+} and calcium-dependent protein kinase via downstream signals, NO and auxin promote root development (Pagnussat et al. 2002). Similarly, the interaction of NO and auxin promotes Cd tolerance in the rosid dicot genus *Truncatula* by reducing auxin degradation (Xu et al. 2010). Furthermore, there is a growing of evidence pointing to the effect of NO in reducing serious metal toxicity (He et al. 2012; Yuan and Huang 2016; Wei et al. 2020). Iron deficiency, on the other hand, stimulates the assembly of auxin and increases NO levels, thereby upregulating ferric-chelate enzyme activity in *Arabidopsis*.

Various studies have emphasized the role of cytokinins in plant organic process processes, cellular division, and leaf senescence. Cytokinins and NO interact in a variety of ways, including synergistic and antagonistic responses. NO and cytokinins interact synergistically in response to drought stress causing leaf senescence, cellular division, and photosynthetic activity (Mishina et al. 2007; Shao et al. 2010; Shen et al. 2013). NO signaling via gibberellins induces multiple physiological responses in plants, including seed germination, root growth (Lozano-Juste and León 2011; Sanz et al. 2015), photosynthetic activity, and nutrient use potency. NO and gibberellins also have an antagonistic relationship because NO suppresses gibberellic acid signal events and signal transduction by promoting the accumulation of DELLA proteins (Asgher et al. 2017). Wu et al. (2014) discovered that gibberellins work antagonistically with NO to manage root growth in *Arabidopsis* at low

and high P concentrations. Likewise, NO production is required for ABA-induced stomatal closure in guard cells (Neill et al. 2002). The role of NO–ABA interactions in drought stress and UV-B radiation stress has been well established in controlling stomatal closure and inhibitor defense machinery (Neill et al. 2008; Tossi et al. 2009).

Several studies have shown that ABA and NO interact in plant physiological responses and signaling mechanisms (Castillo et al. 2015; Asgher et al. 2017; Wang et al. 2020). Furthermore, NO and ethylene have an antagonistic interaction because NO inhibits ethylene synthesis and action by inhibiting leaf senescence and ripening (Leshem et al. 1998; Manjunatha et al. 2010). However, serious physiological responses to NO interactions with ethylene are being studied in *Arabidopsis*, *Cucumis*, and *Nicotiana* (Ederli et al. 2006). As a result, it is clear that the interaction of NO with phytohormones in abiotic stress tolerance is supported by a variety of evidence. Consider the interaction of NO with various hormones such as brassinosteroids, jasmonates, and polyamines (Liu et al. 2014; Lau et al. 2021; Nahar et al. 2016). The interaction of NO with hormones activates the advanced signal cascade, inducing a variety of responses to environmental stresses.

Despite the fact that there is conflicting evidence regarding the interference mechanism of NO with hormones, future research on the mechanisms of interaction of multiple hormones with NO and their potential pathways is required to be addressed in terms of stress responses and plant growth traits (Table 1.1).

Table 1.1 The physiological role of NO in plants under abiotic stress.

Plant species	Stressors	Physiological role	Reference
Wheat	Drought	Enhanced drought tolerance	Garcia-Mata and Lamattina 2001
Alianthus altissima	Drought	Enhanced antioxidant defense mechanism, proline and osmolyte metabolism	Filippou et al. 2014
Wheat	Drought	Enhanced seedling growth, high relative water content, mitigation of oxidative stress	Tian and Lei 2006
Crambe abyssinica	Drought	Enhanced NR activity and suppressed ROS and malondialdehyde content	Batista et al. 2018
Arabidopsis	Drought	Early drought responsive processes along with translational and transcriptional reprogramming	Ederli et al. 2019
Bean	UV-B radiation	Decreased H_2O_2 content, enhanced leaf growth, elevated antioxidant enzyme activity	Shi et al. 2005
Alfalfa	Salinity	Enhanced plant growth and seed germination	Wang and Han 2007
Chickpea	Salinity	Stimulates plant development and antioxidant enzyme activity	Ahmad et al. 2016
Avicennia marina	Salinity	Enhanced photosynthetic activity	Shen et al. 2018

(*Continued*)

Table 1.1 (Continued)

Plant species	Stressors	Physiological role	Reference
Zea mays	Salinity	Enhanced activity of tonoplast H^+- ATPase and gene for Na+/H+ antiporter	Zhang et al. 2006
Cucumis sativus	Salinity	Spermidine accumulation has increased	Fan et al. 2013a
Sunflower	Salinity	Seedling growth is improved, and antioxidant activity is increased and reduced ROS formation	Kaur and Bhatia 2016; Arora and Bhatia 2017
Jatropha	Salinity	Improved seedling growth with less oxidative stress and lower toxic ion deposition	Gadelha et al. 2017
Crocus sativus	Salinity	Increased growth due to osmolyte accumulation and antioxidant enzyme activity, as well as increased secondary metabolite synthesis	Babaei et al. 2020
Pea	Salinity	Enhanced chlorophyll content, nutrient uptake, and antioxidant enzyme activity	Dadasoghi et al. 2020
Mustard	Salinity	Increased synthesis of antioxidant enzymes, enzymes for N metabolism, photosynthesis and respiration, decreased H_2O_2, MDA content, and PCD	Sami et al. 2021
Vigna radiata	Salinity	Increased activity of proline, total amino acids, reducing sugars, modulates antioxidant enzyme activities, physiological traits	Roychoudhary et al. 2021
Tomato	Salinity	Enhanced activities of NO and ROS	Liu et al. 2015a
Spinach	Salinity	Enhanced secondary metabolites and activity of antioxidant enzymes	Du et al. 2015
Oryza sativa	As toxicity	Enhanced root growth and formation, reduced ROS generation, and As accumulation	Kushwaha et al. 2019
Arachis hypogea	Cd toxicity	Increased antioxidant enzyme activities, reduced ROS and Cd accumulation	Yuanjie et al. 2019
Arabidopsis	Cu toxicity	Improved cell viability	Peto et al. 2013
Tomato	Cd toxicity	Enhanced water uptake, reduced Cd uptake and oxidative damage	Ahmad et al. 2018
Cowpea, rye grass	Al toxicity	Enhanced chlorophyll content, antioxidant enzyme activities	Khairy et al. 2016; Sadeghipour 2016

(*Continued*)