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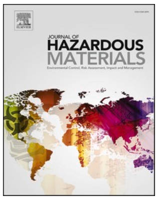

Involvement of nitric oxide (NO) in plant responses to metalloids

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A B S T R A C T

Plants respond to the limited or excess supply of metalloids, boron (B), silicon (Si), selenium (Se), arsenic (As), and antimony (Sb) *via* complex signaling pathways that are mainly regulated by nitric oxide (NO). The absorption of metalloids from the soil is facilitated by pathways that involve aquaporins, aquaglyceroporins, phosphate, and sulfate transporters; however, their regulation by NO is poorly understood. Using *in silico* software, we predicted the *S*-nitrosation of known metalloid transporters, proposing NO-dependent regulation of metalloid transport systems at the posttranslational level. NO intensifies the stress-mitigating effect of Si, whereas in the case of Se, As, and Sb, the accumulation of NO or reactive nitrogen species contributes to toxicity. NO promotes the beneficial effect of low Se concentrations and mitigates the damage caused by B deficiency. In addition, the exogenous application of NO donor, sodium nitroprusside, reduces B, Se, and As toxicity. The primary role of NO in metalloid stress response is to mitigate oxidative stress by activating antioxidant defense at the level of protein activity and gene expression. This review discusses the role of NO in plant responses to metalloids and suggests future research directions.

1. Introduction

The diverse group of metalloids includes boron (B), silicon (Si), arsenic (As), selenium (Se), antimony (Sb), germanium (Ge), tellurium (Te), and polonium (Po). While most metalloids are non-essential to land plants, metalloids such as As, Ge, and Sb are hazardous to plants and harmful to human health. However, B is an exception; it is an essential nutrient primarily because it stabilizes the cell wall matrix by cross-linking with the cell wall component rhamnogalacturonan II (O'Neill et al., 1996). B also positively influences plant reproductive development, seed quality, and antioxidant and polyphenol synthesis. Moreover, it is involved in the nucleic acid synthesis, phenol metabolism, and carbohydrate biosynthesis (Landi et al., 2019).

Recently, Lewis (2019) questioned the essential role of B in plants, arguing that the formation of B complexes in the cell wall is a detoxifying mechanism rather than the evidence of its essentiality. As a result, a scientific debate occurred (Wimmer et al., 2020; González-Fontes, 2020; Lewis, 2020). Excess B has been proven to cause growth impairment, necrosis in leaves and stems, and malformation of fruits as well as altered root growth (Landi et al., 2019). Si is recognized as a semi-essential element because in its absence, plants develop without symptoms. However, in silicified species, such as horsetail and rice, Si deficiency increases susceptibility to fungal infection (Law and Exley, 2011). In addition, Si added to the growth medium enhances plant fitness and resistance to (a)biotic stress (Azeem et al., 2015; Coskun et al., 2016; Guerriero et al., 2016) by, among other things, promoting efficient water use, activating defense enzymes and metabolites, and delaying senescence (Frew et al., 2018). In the plant kingdom, Se, like Si, is essential for algae but not for higher plants. This indicates that the capability of using Se as an essential nutrient has been evolutionarily lost (Pilon-Smits and Quinn, 2010; Schiavon and Pilon-Smits, 2017). At low concentrations, Se exerts positive effects on germination as well as vegetative and reproductive growth, delays senescence, and alleviates abiotic stresses (Kolbert et al., 2019a). Unlike Si, Se exerts a phytotoxic

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Abbreviations: Al, Aluminum; Sb, antimony; As, arsenic; AsA, ascorbate; APX, ascorbate peroxidase; B, boron; Cd, cadmium; cPTIO, 2-(4-carboxyphenyl)-4,4,5,5-tetramethylimidazoline-1-oxyl-3-oxide; CAT, catalase; CK, cytokinin; DHAR, dehydroascorbate reductase; ET, ethylene; GSH, glutathione; GPX, glutathione peroxidase; GST, glutathione-S-transferase; Hb, hemoglobin; H_2O_2, hydrogen peroxide; H_2S, hydrogen sulfide; $^\bullet OH$, hydroxyl radical; LPO, lipid peroxidation; MDHAR, monodehydroascorbate reductase; NR, nitrate reductase; L-NMMA, N^G-monomethyl-L-arginine; NO, nitric oxide; NOS, nitric oxide synthase; L-NAME, N_ω-Nitro-L-arginine methyl ester; $ONOO^-$, peroxynitrite; POD, peroxidase; PGPR, plant growth promoting rhizobacteria; P5CS, pyrroline-5-carboxylate synthase; RNS, reactive nitrogen species; ROS, reactive oxygen species; SA, salicylic acid; Se, selenium; SeCys, selenocysteine; SeMet, selenomethionine; Si, silicon; GSNO, S-nitrosoglutathione; GSNOR, S-nitrosoglutathione reductase; SNO, S-nitrosothiol; SNP, sodium nitroprusside; $O_2^{\bullet -}$, superoxide anion; SOD, superoxide dismutase.

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Table 1
The metalloid transporter proteins predicted to be modified by *S*-nitrosation. The amino acid sequences in the FASTA format were downloaded from the UniProt database and uploaded to the GPS-SNO 1.0 and iSNO-PseAAC software. The predictions were performed using a medium threshold. The prediction results on the positions and peptides were extracted into an Excel file for further analysis. The target Cys (C) are indicated in italics, and Cys predicted by both computational tools are labeled with asterisks.

		GPS-SNO 1.0		iSNO-PseAAC	
Transporter	Metalloid transported	Cys position of S-nitrosation	Amino acid sequence	Cys position of S-nitrosation	Amino acid sequence
ZmPIP1–1	$B(OH)_3$	141*	TRAVFYIIMQ**C**LGAICGRGVV	141*	TRAVFYIIMQ**C**LGAICGRGVV
ZmPIP1–2	$B(OH)_3$	143	LFYIIMQ**C**LGAVCGA	n.d.	
AtNIP5;1	$B(OH)_3$, As(III)	n.d.		116	GAETLIGNAA**C**AGLAVMIIIL
				168	YIAAQVSASI**C**ASFALKGVFH
AtNIP6,1	$B(OH)_3$, As(III)	n.d.		115	KTDGAETLIG**C**AASAGLAVMI
HvPIP1;3	$B(OH)_3$	146*	VFYIVMQCLGAICGA	146*	TRAVFYIVMQ**C**LGAICGAGVV
HvPIP1;4	$B(OH)_3$	146*	VFYIVMQ**C**LGAICGA	146*	VFYIVMQ**C**LGAICGA
AtBOR1	$B(OH)_3$	78	TLASTAI**C**GMIHSII	129	DLFLAWSGWV**C**VWTALMLFVL
				145	MLFVLAICGA**C**SIINRFTRVA
				477	NLLQSTMVGG**C**VAAMPILKMI
				557	TLFQTTYLLI**C**FGLTWIPIAG
AtBOR2	B(OH)3	78*	TLASTAI**C**GIIHSII	78*	AVQTLASTAI**C**GIIHSIIGGQ
		627	GSTASYP**C**DSEILDE	129	NLFLAWSGWV**C**VWTSLILFVL
		647*	RGEFRHT**C**SPKVTSS	477	NLLQAVMVGG**C**VAAMPLLKMI
				647*	TRSRGEFRHT**C**SPKVTSSTST
OsLsi1 (OsNIP2;1)	Si, Se(IV)	204	LAVGSAV**C**ITSIFAG	139	YWAAQFTGAI**C**ASFVLKAVIH
OsLsi2	Si	123	ALFTNDTC**C**VVLTEF	108	QGGRDLMCRV**C**VVTALASALF
		317	DFRDAEP**C**LDTVSYS	124	ASALFTNDTC**C**VVLTEFVLEL
				265	ITTKHPWFMQ**C**TEARRKLFLK
HvLsi1	Si	66*	FLLVFVT**C**GAAAISA	66*	VSTFLLVFVT**C**GAAAISAHDV
		204	LAVGSSV**C**ITSIFAG	139	YWAAQFTGAI**C**ASFVLKAVLH
ZmLsi1	Si	202	LAVGSAVCITSIFAG	n.d.	
ZmLsi6	Si	n.d.		n.d.	
AtSULTR1;2	Se(VI), SO_4^-	135	VPPLVYA**C**MGSSRDI	469	FKVDKLDFIA**C**IGAFFGVIFV
		406	AVNFMAG**C**QTAVSNI		
		645	VADAVEA**C**CPKLSNE		
AtSULTR2	Se(VI), SO_4^-	431	AVNFAAG**C**ETAMSNI	56	QPDRSKWLLD**C**PEPPSPWHEL
		450*	TVFVALE**C**LTRLLYY	450*	MAVTVFVALE**C**LTRLLYYTPI
		568*	RVKSALL**C**FANASSI	568*	LIFRVKSALL**C**FANASSIEER
				672	YLTIGEALDA**C**FGLKVXXXXX
AtPHT1;1	As(V)	n.d.		41	GFFTDAYDLF**C**VSLVTKLLGR
				111	YGLTLVMMIL**C**SVASGLSFGH
				131	HEAKGVMTTL**C**FFRFWLGFGI
AtPHT1;4	As(V)	111*	TLMVMVL**C**SIASGLS	41	GFFTDAYDLF**C**ISLVTKLLGR
				111*	YGMTLMVMVL**C**SIASGLSFGH
				131	HEPKAVMATL**C**FFRFWLGFGI
AtPHT1;7	As(V)	111*	TLMVMVL**C**SIASGLS	41	GFFTDAYDLF**C**ISLVTKLLGR
				111*	YGMTLMVMVL**C**SIASGLSFGS
				131	SNPKTVMTTL**C**FFRFWLGFGI
AtPHT1;8	As(V)	117	VCTTRRS**C**VMVSLGF	38	GLFTDAYDLF**C**IAPVMKMISH
				95	RVGRRRVYGL**C**LIIMILSSFG
				179	LVSSAVTMAV**C**VAFKRSGGGL
				482	RIAFLILGGV**C**IAGILVTYFF
AtPHT1;9	As(V)	118	VCTTRRS**C**VMVSLGF	39	GLFTDAYDLF**C**IAPIMKMISQ
				180	LMSSAVTMVV**C**LAFKNAGEGS
				488	RIAFLILGGV**C**IAGMIVTYLF
OsPHT1;1	As(V)	n.d.		41	GFFTDAYDLF**C**ISLVTKLLGR
				111	YGFTLILMVV**C**SVASGLSFGS
				131	SSAKGVVSTL**C**FFRFWLGFGI
OsPHT1;4	As(V)	110*	TLMLMVI**C**CLASGLS	110*	YGMTLMLMVI**C**CLASGLSFGS
		111	LMLMVI**C**CLASGLSF	130	SSAKGVMATL**C**FFRFWLGFGI
				489	VRNSLFFLAG**C**NVIGFFFTFL
OsPHT1;8	As(V)	115*	TLLMMVI**C**SIASGLS	45	GFFTDAYDLF**C**ISLVTKLLGR
				115*	YGMTLLMMVI**C**SIASGLSFSH
AtNIP1;1	As(III)	n.d.		161	RLLFGLDHDV**C**SGKHDVFIGS
AtNIP1;2	As(III)	n.d.		69	LGTYFLIFAG**C**AAVAVNTQHD
AtNIP3;1	As(III)	291*	EASQDEI**C**VLRVVDP	274	KSYSEIIRPN**C**NKVSSRDRQE
				291*	DRQEASQDEI**C**VLRVVDPANQ
				306	VDPANQNYFI**C**SSPTDINGKC
				316	CSSPTDINGK**C**NVTCKLAXXX
AtNIP7;1	As(III)	n.d.		35	DHPSRQRLFG**C**LPYDIDLNPL
				183	IVVFLASALH**C**GPHQNLGNLT
				262	YRSISLKTRP**C**PSPVSPSVSS
OsNIP1;1	As(III)	n.d.		65	FGTYFLIFAG**C**GAVTINQSKN
OsNIP2;2	As(III)	207	LAVGSAV**C**ITSIFAG	69	VATFLLVFVT**C**GAASIYGEDM
				142	YWAAQFTGAM**C**AAFVLRAVLY
OsNIP3;1	As(III)	54*	KSMPRCK**C**LPAAVAE	54*	YERKSMPRCK**C**LPAAVAEAWA
				70	AEAWAPSAHG**C**VVEIPAPDVS
				173	YVAVQVLGSI**C**AGFALKGVFH
OsNIP3;2	As(III)	n.d.		n.d.	

(*continued on next page*)

Table 1 (*continued*)

		GPS-SNO 1.0		iSNO-PseAAC	
Transporter	Metalloid transported	Cys position of S-nitrosation	Amino acid sequence	Cys position of S-nitrosation	Amino acid sequence
OsNIP3;3	As(III)	n.d.		n.d.	
OsPIP2;4	As(III)	135	LLYMAAQ**C**LGAICGV	n.d.	
OsPIP2;6	As(III)	128	VMYIVAQ**C**LGGIVGV	70	GYKVQSSADQ**C**GGVGTLGIAW
OsPIP2;7	As(III)	135*	VLYVVAQ**C**LGAIAGA	77	YKNQRATVDA**C**TGVGYLGVAW
				99	FGATIFVLVY**C**TGGVSGGHIN
				135*	VRTVLYVVAQ**C**LGAIAGAGIV
LjNIP5;1	As(III)	n.d.		168	AAQVSASICA**C**FALKYVYHPF
LjNIP6;1	As(III)	n.d.		52	GKKKKSLLKN**C**NCFTVEEWTI
				118	KTQGAETLIG**C**AASTGLAVMV
				173	YIIAQVMAGI**C**ASFGLKGVFN

effect at elevated concentrations due to molecular mechanisms such as protein selenization, oxidation, nitration (Kolbert et al., 2019b), and disturbance of the balance of hormones and nutrients (Kolbert et al., 2016). In the case of Se, B, and non-essential metalloids, such as As, Sb, Te, and Ge, phytotoxicity can be linked to the imbalance of reactive oxygen species (ROS) and the consequent oxidative stress. Contrarily, Si's stress-alleviating effects are realized mainly by reducing the ROS levels and the associated damages.

Beyond ROS, metalloids also modify the metabolism of reactive nitrogen species (RNS). Peroxynitrite ($ONOO^-$) and S-nitrosoglutathione (GSNO), both derived from the gaseous signal molecule nitric oxide (NO), can be considered as relevant RNS (Valderrama et al., 2007). In higher plants, NO can be synthesized by oxidative and reductive reactions that involve enzymes or occur spontaneously (Kolbert et al., 2019c). NO reacts with thiol (SH)-containing proteins and peptides, forming low-molecular-weight S-nitrosothiols (SNOs), such as GSNO (Hogg, 2000; Foster et al., 2003), which is the most abundant SNO that can liberate NO or be reduced by GSNO reductase (GSNOR) (Barroso et al., 2006; Corpas et al., 2008; Leterrier et al., 2011).

The perception and transduction of the NO signal are believed to be realized primarily through the tyrosine nitration, metal nitrosylation, and *S*-nitrosation of proteins (Umbreen et al., 2018). NO can indirectly influence protein activity through the formation of $ONOO^-$, leading to protein tyrosine nitration, which is an irreversible modification due to the formation of 3-nitrotyrosine-containing nitro-proteins (Souza et al., 2008). In plant cells, protein tyrosine nitration mainly inhibits enzyme activity (Kolbert et al., 2017) and may prevent or induce tyrosine phosphorylation, ultimately influencing cell signaling (Souza et al., 2008). *S*-nitrosation is a reversible reaction affecting Cys thiol groups, leading to the formation of *S*-nitroso-proteins with altered activity, subcellular localization, and interactions or binding activities (Lubega et al., 2021). There are many types of antioxidant enzymes, including superoxide dismutase (SOD), catalase (CAT), peroxiredoxins, and the enzymes of the ascorbate–glutathione cycle, the activity of which is positively or negatively regulated by NO-dependent *S*-nitrosation or tyrosine nitration, or both (Begara-Morales et al., 2016). In addition, it has been widely accepted that NO is involved in plant responses to heavy metals partly *via* the regulation of antioxidant activities (Terrón-Camero et al., 2019). In recent years, there is an increasing evidence that NO also participates in metalloid stress regulation.

This review collates, discusses, and evaluates the accumulated literature with regard to the metabolic, signaling, and physiological role of NO and its derivatives in the plants exposed to metalloids to suggest the research directions for future studies. Additionally, using *in silico* prediction tools, we examined the possible involvement of the NO-dependent *S*-nitrosation of proteins in plant metalloid uptake.

2. Metalloid uptake by plants and its putative regulation by NO

The hydrophilic nature of metalloids in aqueous solutions necessitates the help of transmembrane proteins driven by concentration gradients or pumps in facilitation of metalloid movement across the plasma membrane.

In case of limited B supply, aquaporins and aquaglyceroporins such as ZmPIP1, AtNIP5;1, AtNIP6;1, HvPIP1;3, and HvPIP1;4 have been demonstrated to facilitate B transport (Fitzpatrick and Reid, 2009; Dordas et al., 2000; Dordas and Brown, 2001a; Takano et al., 2002, 2006, 2008). However, no specific solute pumps have been identified for the active transport of B. In the presence of sufficient B, the passive diffusion of uncharged, small boric acid (H_3BO_3) without solute channel facilitators satisfies the demand of plants for B (Dordas and Brown, 2001b; Zangi and Filella, 2012). Conversely, efflux is an active transport catalyzed by BOR1 and BOR2 transporters in *Arabidopsis* (Yoshinari and Takano, 2017). BOR transporters regulate B uptake under B-deficient conditions (Tanaka et al., 2008), whereas they are degraded in case of excess B (Aibara et al., 2018; Hrmova et al., 2020).

Similar to B, Si uptake is possible in the form of uncharged $Si(OH)_4$ (Amo and Brzezinski, 1999). In rice, the low silicon rice 1 (*OsLsi1*) gene encodes a plasma membrane protein belonging to the Nodulin26-like intrinsic protein (NIP) subfamily of aquaporins (Ma et al., 2006). The expression level of *OsLsi1* is regulated by Si supplementation. *OsLsi1* homologs in barley (*HvLsi1*, Chiba et al., 2009) and maize (*ZmLsi1*, ZmLsi6 (Mitani et al., 2009)) have also been characterized. Silicon efflux is catalyzed by the plasma membrane protein encoded by the low silicon rice 2 (*OsLsi2*) gene. Additionally, active Si uptake in the form of anions has been observed in marine diatoms. Sodium-requiring channels are involved in this process (Hildebrand et al., 1998; Mandlik et al., 2020).

The uptake of Se in the form of Se(VI) is based on its similarity to sulfate and mediated through the activity of sulfate transporters (Trippe and Pilon-Smits, 2021). In *Arabidopsis thaliana*, *SULTR1;2* and *SULTR1* were found to be responsible for the internalization of selenate into the plant cell (El Kassis et al., 2007). However, it seems that *SULTR1;2* is the predominant transporter for absorbing selenate *via* the root system (Shibagaki et al., 2002; El Kassis et al., 2007). According to Li et al. (2008), the Se(IV) uptake in wheat, mediated by active transport, is significantly inhibited by the metabolic inhibitor carbonyl cyanide m-chlorophenyl hydrazone. Further analysis reveals that the phosphate transport system may be involved in the Se(IV) uptake (Zhang et al., 2014). Additionally, the Silicon Influx Transporter ***OsNIP2;1*** has been demonstrated to mediate Se(IV) uptake in rice (Zhao et al., 2010).

Owing to its similarity to phosphate (Pi), As(V) is taken up by plant cells *via* the Pi transport systems; several phosphate transporters have been identified for the As(V) uptake and translocation in various plant species, such as AtPHT1;1, AtPHT1;4 AtPHT1;7, AtPHT1;8, and AtPHT1;9 in *Arabidopsis* and OsPHT1;1, OsPHT1;4, and OsPHT1;8 in rice. Similarly, several aquaporins involved in As(III) transport have been identified. AtNIP1;1, AtNIP1;2, AtNIP3;1, AtNIP5;1, AtNIP6;1, and AtNIP7;1 in *Arabidopsis*; OsNIP1;1, OsNIP2;1 (*OsLsi1*), OsNIP2;2 (*OsLsi6*), OsNIP3;1, OsNIP3;2, OsNIP3;3, OsPIP2;4, OsPIP2;6, and OsPIP2;7 in rice; HvNIP1;2 in barley; and LjNIP5;1 and LjNIP6;1 in *Lotus japonicus* have been demonstrated to assist in the passive transport of As (III) (Garbinski et al., 2019; Tang and Zhao, 2020).

Contrary to the As(V) uptake, the mechanism of the Sb(V) uptake has not been identified, but it may not involve phosphate transporters

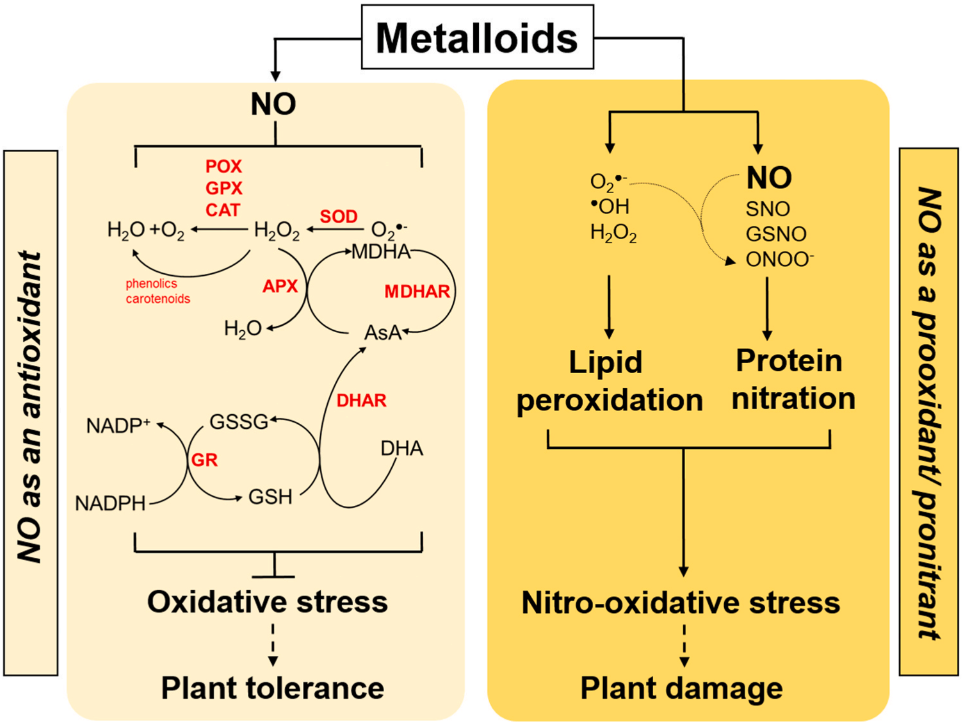

Fig. 1. The antioxidant and prooxidant/pronitrant roles of NO/RNS in metalloid-induced stress mitigation and damages in plants. Metalloid-induced NO upregulates antioxidant enzymes and non-enzymatic antioxidants, reducing oxidative stress and improving tolerance. The nitric oxide-regulated elements of the antioxidant system are indicated in red. Depending on their hazardous nature and concentration, metalloids induce accumulation of ROS and RNS, causing nitro-oxidative stress, as indicated by lipid peroxidation, protein nitration, and plant damage. Abbreviations: NO, nitric oxide; $O_2^{\bullet -}$, superoxide anion; H_2O_2, hydrogen peroxide; MDHA, monodehydroascorbate; MDHAR, monodehydroascorbate reductase; AsA, ascorbic acid; APX, ascorbate peroxidase; DHA, dehydroascorbate; DHAR, dehydroascorbate reductase; GSSG, oxidized glutathione; GSH, reduced glutathione; GR, glutathione reductase; OH, hydroxyl radical; SNO, *S*-nitrosothiol; GSNO, *S*-nitrosoglutathione; ONOO$^-$, peroxynitrite.

(Zangi and Filella, 2012). However, NIPs are involved not only in the As (III) uptake but also in the entry of Sb(III) into the cells (Pommerrenig et al., 2015; Zhu et al., 2020).

Since metalloid transporters are proteins and NO signaling is achieved mainly by posttranslational modifications, NO may regulate proteins *via S*-nitrosation, increasing or decreasing their metalloid transport activity. This hypothesis was tested *in silico*. The *S*-nitrosation of the most relevant metalloid transporters was predicted using two independent software tools (peptide sequences were extracted from UNIPROT (www.uniprot.org) and submitted to the prediction software GPS-SNO 1.0 (http://sno.biocuckoo.org, Xue et al., 2010) and iSNO-PseAAC (http://app.aporc.org, Xu et al., 2013). For most metalloid transporters, the *S*-nitrosation of one or more Cys in them was predicted by both tools (Table 1), supporting the theoretical possibility of protein-level regulation of metalloid transport by NO. However, it should be noted that the *S*-nitrosation of metalloid transporter proteins may also affect the uptake of essential elements, such as phosphate and sulfate, as well as the transport of water in plant cells.

3. The involvement of NO in regulating metalloid stress responses

3.1. Nitric oxide regulates plant responses to B deficiency and toxicity

Boron is a unique micronutrient for plants owing to its narrow range of beneficial and toxic concentrations. Most of the available literature focuses on excess B-induced phytotoxicity, and only a few studies involving NO examine plants' physiological and molecular responses to B deficiency.

According to Kobayashi et al. (2018), B deficiency results in rapid cell death within the root apex of *Arabidopsis*. Using pharmacological treatments, ROS have been shown to participate in the induction of cell death in response to B deprivation. The production of ROS is thought to be triggered by stretching of the plasma membrane and the influx of calcium (Ca) ions *via* the mechanosensitive channels. Furthermore, NO formation is increased in B-deficient roots. Decreasing the level of NO with 2-(4-carboxyphenyl)-4,4,5,5-tetramethylimidazoline-1-oxyl-3-oxide (cPTIO), or with the inhibitor of mammalian nitric oxide synthase (NOS), N^G-monomethyl-L-arginine (L-NMMA), mitigates cell death. These findings suggest that in addition to ROS, NO is required for B deficiency-induced programmed cell death. Another interesting result obtained from this study is that B limitation triggers transcriptome changes resembling pathogen-induced responses, suggesting that B deficiency induces ROS- and NO-associated hypersensitive response in *Arabidopsis* roots as a response to damages in the cell wall structures.

Recently, Kaya et al. (2019) observed that, similar to B deficiency, B toxicity promoted endogenous NO production in the leaves of two wheat cultivars. This finding was explained by the activation of a NOS-like enzyme by excess B. However, because no NOS enzyme has been identified in higher plants, these results should be treated with caution. The B toxicity-reducing effect of thiourea (TU) treatment, achieved by, among other things, the reduction of hydrogen peroxide (H_2O_2), electrolyte leakage, and lipid peroxidation (LPO), has also been presented. Contrarily, TU treatment further increases NO levels and NOS-like activity in the leaves of wheat cultivars. By examining several growth, oxidative, and antioxidant parameters, TU-induced tolerance against B toxicity was found to be eliminated by cPTIO-triggered NO depletion. These data confirm that NO is an essential component in the regulation of plant tolerance to excess B. The same research group revealed that thiamine-induced NO was essential in the amelioration of B toxicity in pepper plants (Kaya et al., 2020a).

The possibility that NO mitigates B toxicity was studied for the first time by Aftab et al. (2012), who observed that the addition of B or aluminum to soil significantly reduced the yield and growth of *Artemisia annua* and decreased the photosynthetic rate, stomatal conductance, intracellular CO_2 concentration, and total chlorophyll content. The application of the NO donor, sodium nitroprusside (SNP), improved the growth and photosynthetic performance of stressed and non-stressed plants. The artemisinin content was also increased in B-exposed *Artemisia annua* plants by the addition of NO. In the study by Kaya and Ashraf (2015), elevated B levels caused a significant decrease in dry biomass and fruit yield of tomato plants compared with the non-stressed plants. Excess B increased electrolyte leakage and LPO and promoted ROS production as well as antioxidant protection. The foliar application of SNP improved biomass production and fruit yield and decreased B-induced oxidative damages and antioxidant enzyme activities. The macroelement homeostasis in tomato leaves was improved. At the same time, the B concentration in the tissues was reduced by SNP treatment (Kaya and Ashraf, 2015). This indicates that NO downregulated the uptake of B. In another study, watermelon plants were exposed to 0 (deficiency), 0.5 (standard supplementation), and 10 mg/L (excess) B concentrations with or without SNP (Farag et al., 2017). B toxicity-triggered growth inhibition of the seedlings was associated with high B translocation to the shoot tissues, causing LPO and chlorophyll depletion. Contrarily, B deficiency accelerated ROS production, mainly hydroxyl radical ($^{\bullet}OH$), and induced oxidative injury. SNP enhanced chlorophyll content and photosynthesis, consequently inducing biomass production in B-deficient and B-excess watermelon seedlings through the reduction of B accumulation, LPO, and ROS generation. SNP also activated antioxidant enzymes, such as SOD, peroxidase (POD), and ascorbate peroxidase (APX), thus protecting the seedlings from ROS-induced oxidative damage.

The results discussed above indicate that both B deficiency and toxicity upregulate NO formation, causing its overproduction. Although NOS-like activity is assumed to be responsible for NO synthesis in both cases, the existence of a NOS in higher plants is uncertain. Therefore, the possibility that NO is synthesized *via* B supply-induced nitrate reduction should be closely examined (Eraslan et al., 2007). With regard to the role of NO, available data suggest that, depending on the amount of B, elevated NO production may contribute to the damage or increase tolerance (Fig. 1). In addition, a few studies suggest that exogenous NO mitigates the detrimental effects of B toxicity, improving the growth and yield partly by reducing B uptake. However, the molecular mechanism of NO's action on B transport remains unknown. NO administration may prevent B-induced ROS overproduction by activating antioxidant protection.

3.2. Dual participation of NO in Si-induced stress alleviation

The stress-relieving effects of Si in plants have been extensively studied, and recent studies have demonstrated NO as a regulator of Si's beneficial effects.

Pandey et al. (2016) examined the stress-mitigating effect of Si in hydroponically grown *Brassica juncea* treated with As. Si was found to improve root growth, increased the concentration of several micro- and macronutrients, decreased the total ROS and superoxide anion radical ($O_2^{\bullet -}$) levels, and enhanced the activity of antioxidant enzymes in the presence of As. As exposure increased the NO level in the root, the extent of which was moderated by Si. The authors concluded that the As-triggered accumulation of ROS and NO was reduced by Si, thereby protecting the tissues from oxidative damage. Similar effects have been observed in the treatment of young maize plants exposed to aluminum (Al) with 20-nm, spherical Si nanoparticles (NPs) (de Sousa et al., 2019). At higher Al concentrations, Si increased root and shoot biomass and improved photosynthetic parameters and pigment contents compared with the plants treated only with Al. Al mainly increased LPO, protein oxidation, and NO levels in the root, but supplementation with Si NP decreased these enhancements and promoted viability. In this comprehensive work, the activity of numerous antioxidant enzymes and the content of non-enzymatic antioxidants, such as flavonoids, polyphenols, and tocopherols, were analyzed. In most cases, the upregulating effect of Si was observed.

Also, Si was found to mitigate the stressor-induced accumulation of NO free radicals, which presumably enhanced stress tolerance. The protective effect of Si against salinity has also been demonstrated (Chung et al., 2020). Si application improved photosynthetic responses, such as transpiration rate and net photosynthesis rate in salinity-exposed soybean, along with reduced CAT, APX activities, and glutathione (GSH) content. Contrary to the previous works, this study analyzed the SNO content increased by Si as well as by salt stress. When Si was applied in salt-treated plants, the SNO content significantly decreased. Moreover, salt stress upregulated the soybean GSNOR genes *GmGSNOR1*, *GmGSNOR2*, and *GmGSNOR3*. The effect of Si on the expression of the *GSNOR* genes was time-dependent, with a noticeable induction within a shorter period of 3–6 h, whereas Si reduced the expression of these genes after 12 h. Thus, it could be concluded that Si reduced oxidative and nitrosative damage by reducing the SNO levels through the upregulation of metabolizing GSNOR genes, thus increasing salt tolerance. In drought-stressed lentil plants, Si treatment was found to maintain nitro-oxidative homeostasis by balancing reactive oxygen species ($O_2^{\bullet -}$, H_2O_2) and reactive nitrogen species (NO) levels thus contributing to better tolerance (Sajitha et al., 2021).

The beneficial effects of Si in relation to NO have been studied not only in plants exposed to abiotic stresses but also in the case of biotic stressors, such as the infestation of *Orobanche ramosa*, a holoparasitic root weed in tomato (Madany et al., 2020). In this work, seed priming was performed using 20-nm, spherical Si NPs. The stress-mitigating effect of Si NPs was demonstrated by the reduction in infection, the increment in biomass production, and the improvement of photosynthetic parameters. Furthermore, Si NPs reduced LPO, H_2O_2, and NO levels triggered by *Orobanche* infection in the root and shoot. In addition, the activity and amount of several ROS scavenging enzymes and non-enzymatic antioxidants were measured, and Si NP-induced increment was observed in almost all cases. Thus, the antioxidant defense was enhanced in the infected plants. Collectively, these works identify NO/SNO as damaging signal molecules induced by the stressors, including As, Al, salt, Cu and *Orobanche*, and Si or Si NP treatment alleviates stress by moderating NO/SNO overproduction.

In some other works, the opposite has been found. For instance, Tripathi et al. (2017) compared the effect of bulk Si on wheat subjected to UV-B stress with that of 20- to 95-nm, spherical Si NPs. The beneficial effect of Si NP manifested itself in the reduction of UVB-induced ROS production and the regulation of enzymes, including SOD, CAT, GPX, and APX, as well as non-enzymatic antioxidants, including proline (Pro), AsA, phenolics, and flavonoids. Contrarily, the level of UV-B-induced NO was enhanced by Si and Si NP. Therefore, the authors hypothesized that Si and Si NP regulate antioxidant defense through the upregulation of NO. However, direct evidence regarding the involvement of NO was not provided in this work. These observations were supported by the work of Kaya et al. (2020b), where the addition of Si was found to enhance pepper's cadmium (Cd) tolerance by lowering the leaf's Cd concentration and oxidative stress levels and promoting the antioxidant defense system, the leaf's Si content, photosynthetic traits, and plant growth as well as the NO, Pro, and hydrogen sulfide (H_2S) content. The diminution of Si-induced NO production by cPTIO reduced the activity and number of antioxidants, such as SOD, CAT POD, AsA, and GSH, and aggravated biomass and chlorophyll loss. These results provided direct evidence of the role of Si-induced NO in the upregulation of antioxidants and alleviation of Cd-triggered oxidative stress. Moreover, copper (Cu) exposure induced NO production in *Salvia officinalis* which was further increased by Si addition. Authors concluded that Si may improve Cu tolerance *via* inducing NO generation which triggers defense mechanisms in *Salvia* plants (Pirooz et al., 2021).

Researchers also examined the oxidative stress-mitigating effect of Si in *Brassica juncea* exposed to silver nanoparticles (AgNPs). Although Si's beneficial effect could not be demonstrated (Vishwakarma et al., 2020), Si further increased the NO level in the AgNP-treated plants. However, the co-administration of Si and plant growth promoting rhizobacteria (PGPR) significantly improved the plant's AgNP tolerance by reducing NO and ROS accumulation and activating the AsA–GSH cycle, which presumably positively affected photosynthesis and plant fitness. These data suggest that high NO levels are associated with toxicity and that decreased NO content is associated with AgNP tolerance. Conversely, in Liang et al. (2015), the effect of Si on cell death in tobacco BY-2 cells was examined. Si treatment in the presence of ethylene synthesis inhibitor silver nitrate increased the production of NO and H_2O_2, together contributing to cell death.

The stress-mitigating activities of both Si and exogenous NO donor, mainly SNP, are well known. Liu et al. (2020) explored the effect of the combined treatment of Si and SNP on the growth and Cd uptake of maize. Both Si and SNP improved the photosynthesis, pigment content, biomass production, and yield parameters of the plants exposed to Cd at 35 mg/kg $CdCl_2$. The treatments decreased the Cd content in the vegetative organs and the grains. Meanwhile, the combined treatment elicited the most significant effects, indicating that the stress-alleviating effects of Si and SNP were additive. These results indicate that it may be possible to develop and apply a combined treatment method in maize cultivation in the future. Similarly, the combined application of Si and NO ameliorated the Cd-triggered oxidative damage by upregulating the AsA-GSH cycle in wheat seedlings (Singh et al., 2020a). Moreover, the As(III) uptake and oxidative stress were mitigated by the combined application of Si and SNP to *Brassica juncea* plants (Ahmad et al., 2021). These results indicate the synergistic effect of Si and NO on stressed plants.

Most of the experiments discussed above also examined the effect of Si on healthy plants, but their results are quite diverse. The growth-stimulating effect of Si accompanied by increased tissue NO levels has been described in Indian mustard, wheat, and tomato grown under stress-free conditions (Vishwakarma et al., 2020; Tripathi et al., 2017; Madany et al., 2020). Si treatment was found to upregulate the synthesis of polyamines (Manivannan and Ahn, 2017), which theoretically may be a substrate for oxidative NO synthesis (Tun et al., 2006). Si-induced nitrate reductase (NR) (Gottardi et al., 2012) may also contribute to the elevated NO levels, but the mechanism by which Si increases the NO level requires further investigation. However, in other experiments, Si alone did not affect the growth or NO content of the non-stressed plants (Pandey et al., 2016; Kaya et al., 2020b). This suggests that the growth-inducing activity of Si in healthy plants may be related to the changes in the tissue NO content. This assumption is supported by the critical role of NO in growth regulation (Sanz et al., 2015); future experiments should focus on elucidating this issue. Depending on the type of the stress, abiotic such as Al, As, salt, Cu, Cd, and UV-B, or biotic such as *Orobanche* infection, Si modulates endogenous NO levels differently. In some experimental systems, NO acts as a stress/ROS scavenger and contributes to stress-induced damages in other systems (Fig. 1). Under stress conditions, NO may play protective or damaging roles depending on its local concentration (Fancy et al., 2017); this may also be the case with Si-induced stress mitigation.

3.3. Nitric oxide contributes to the beneficial effects and phytotoxicity of Se, and NO also regulates Se uptake and metabolism

The range is narrow between the beneficial and toxic concentrations of Se, similarly to other metalloids. Only a few studies have investigated the involvement of NO in plant responses to beneficial Se concentrations. For instance, Hajiboland et al. (2019) studied the well-known Se-delayed senescence (Xue et al., 2001; Djanaguiraman et al., 2004, 2005; Kolbert et al., 2019a) in *Brassica napus*. The foliar application of Se increased NO production in young and old leaves, and a correlation was observed between the NO levels and the improvement in the photosynthetic parameters in senescent leaves. These results indicate that Se delays leaf senescence in a ROS-independent but NO-related manner, upregulating the genes involved in photosynthesis while down-regulating the senescence-associated genes. The foliar application of low Se doses at 6, 8, and 10 mg/L increased the stem length and leaf number of *Stevia rebaudiana* plants without significantly inhibiting photosynthesis. The beneficial effects of Se were accompanied by the Se concentration-dependent regulation of GSNOR protein levels and protein tyrosine nitration, indicating that the beneficial doses of Se induces nitrosative signaling in *Stevia* leaves (Borbély et al., 2021). The indirect evidence for the involvement of NO in the beneficial effects of Se should be supported by further research as the low-dose Se may be used in crop production and biofortification practices. According to the recent results of the study by Abedi et al. (2021), the low concentration of nano Se at 4 mg/L exerted beneficial effects on the growth of chicory seedlings; the effect was further enhanced by the addition of NO (SNP). Moreover, secondary metabolism was synergistically enhanced by nano Se and NO. However, the higher dose of nano Se at 40 mg/L triggered negative effects on biomass and flowering; the adverse effects were ameliorated by the addition of NO (Abedi et al., 2021).

It has been demonstrated that Se exposure causes a disturbance in RNS metabolism. In the early stage of seedling development, NO levels are decreased by Se but increase in the longer term (Lehotai et al., 2012). In another experiment, Se-induced cytokinins (CK) were observed to reduce the NO content in *Arabidopsis* roots (Lehotai et al., 2016a). Recently, it was found that in the Se-stressed roots, the ethylene (ET) levels were increased, whereas NO generation was reduced. Since NO negatively influences the ET levels, Se exposure creates a feedback loop, resulting in lateral root outgrowth (Feigl et al., 2019).

Se toxicity also depends on the plant species, which can be categorized as non-accumulators, accumulators, and hyperaccumulators. Most research involving NO analyzes the effect of toxic Se concentrations on plant species with varying levels of tolerance. The NO/GSNO-overproducing *Arabidopsis* mutant, *gsnor1–3*, was more resistant, but the NO-deficient *nia1nia2* mutant exhibited a more pronounced sensitivity to Se stress than the wild type. These data suggest that endogenous NO may contribute to Se tolerance (Lehotai et al., 2012). However, Chen et al. (2014) associated Se-induced NO production with ROS formation and Se toxicity in *Brassica rapa* roots. These results were supported by Lehotai et al. (2016b), who demonstrated that Se treatment promoted the production of NO and $ONOO^-$ in pea organs and caused toxicity due to protein tyrosine nitration. In addition, selenite caused more severe protein nitration and toxicity than selenate in *Brassica juncea* (Molnár et al., 2018a). However, the Se-sensitive *Arabidopsis thaliana* suffered more severe oxidative stress but milder nitrosative stress compared with the Se-tolerant *B. juncea* (Molnár et al., 2018b). This suggests that selenite tolerance and sensitivity were more tightly associated with oxidative processes in these species. In a comparative study, Se seriously affected the metabolism of RNS *via* NO production, the $ONOO^-$ and GSNO levels, and the GSNOR activity and protein nitration in Se-sensitive *Astragalus membranaceus*, whereas no relevant Se-induced changes were observed in Se-hyperaccumulator *Astragalus bisulcatus,* indicating a correlation between RNS-induced nitrosative stress and Se tolerance (Kolbert et al., 2018, 2019b).

There is also evidence of the influence of exogenous NO (SNP) on the Se uptake and speciation in rice. According to Xiao et al. (2017), SNP stimulates GSH biosynthesis and triggers Se metabolism in rice seedlings exposed to a moderate Se concentration at 6 μM. In addition, SNP supplementation induced the expression of phosphate and sulfate transporter genes encoding OsPT2, OsSultr1;2, and OsSultr4;1, increasing the selenocysteine (SeCys) and selenomethionine (SeMet) contents in rice roots. Conversely, Dai et al. (2020) reported that SNP promoted growth and photosynthesis; enhanced antioxidant capacity; downregulated the expressions of *OsPT2, OsSAMS1,* and *OsSBP1* genes in the roots and *OsPT2, OsCS,* and *OsSBP1* genes in the shoots; and reduced the Se uptake in rice seedlings subjected to severe Se treatment at 25 μM. The seemingly contradictory results highlight the possibility of SNP exerting opposite effects on the Se uptake and metabolism depending on the magnitude of the Se exposure. Furthermore, NO seems to exert its regulatory role on the Se uptake at the gene expression level.

Collectively, beyond ROS imbalance and protein oxidation, toxic Se concentrations disrupt endogenous RNS metabolism in non-hyperaccumulator plant species, such as *Arabidopsis thaliana*, *Brassica rapa*, *Brassica juncea*, *Pisum sativum*, and *Astragalus membranaceus*, and increase protein tyrosine nitration, contributing to secondary nitro-oxidative stress and ultimately to Se phytotoxicity (Kolbert et al., 2019b). Furthermore, data suggest that Se-mediated NO antagonizes phytohormones, such as CK and ET, and their interplay regulates root development during Se stress. Not only does Se affect endogenous NO/RNS metabolism, but NO content increased by SNP also positively or negatively regulates the Se uptake and speciation within the plant tissues depending on the Se concentration.

3.4. *Arsenic modulates NO/RNS metabolism and exogenous NO mitigates As toxicity via numerous pathways*

Arsenic causes damage to all organisms, including plants; however, the rate of its toxicity depends on the form of As and the plant species. While most of the published experiments focus on the beneficial effect of exogenous NO donors on As-induced damages, other works analyze the As-induced changes in endogenous NO/RNS metabolism (Sharma et al., 2021).

The observed effects of As on endogenous NO and RNS levels are variable. As(III) exposure was found to increase NO levels in the rice root (Rao et al., 2011). Moreover, As(V) exposure in *Arabidopsis* (Leterrier et al., 2012), rice (Kushwaha et al., 2019; Solórzano et al., 2020), *Vicia faba* guard cells (Xue and Yi, 2017), and *Spirodela intermedia* (Da Silva et al., 2018) was demonstrated to increase NO levels. Contrarily, decreased NO levels were measured in As(V)-treated pea (Singh et al., 2015; Rodríguez-Ruiz et al., 2019) and maize seedlings (Kaya et al., 2020c) compared with the untreated plants. According to Leterrier et al. (2012), As(V) increased GSNOR activity, decreased GSNO levels and $O_2^{\bullet-}$ production, and increased protein nitration, supporting As-triggered nitro-oxidative stress in *Arabidopsis* (Fig. 1). Recently, Rodríguez-Ruiz et al. (2019) observed that protein oxidation and LPO increased in As(V)-treated pea. Still, the nitrosative parameters such as $ONOO^-$, NO levels, and GSNOR activity demonstrated As-induced diminution in this experimental system. Therefore, As likely regulates ROS and RNS signaling differently.

Further studies examine the stress-mitigating or damage-inducing effect of endogenous NO on plants. NO participates in the induction of As-triggered cell death, as was shown in the *Vicia faba* guard cells by Xue and Yi (2017). As treatments caused cell death in a concentration-dependent manner and concomitantly increased the levels of NO, ROS, and Ca as well as NR activity. Arsenic-induced cell death was blocked by cPTIO, NR inhibitor sodium azide, AsA, CAT, EGTA, and Ca channel blocker lanthanum chloride, demonstrating that NO, ROS, and Ca as signal components are required for the induction of As-induced cell death in guard cells. Moreover, this work demonstrated that As caused NO formation in guard cells through NR activation (Xue and Yi, 2017).

Many other studies have proven the stress-mitigating effect of endogenous NO production on plant cells. For instance, Singh et al. (2015) found that supplementing As-exposed pea plants with H_2S promoted As tolerance by improving photosynthesis and decreasing ROS accumulation due to the upregulation of the AsA–GSH cycle. These effects were associated with the increased NO level; therefore, the authors hypothesized that H_2S contributes to As tolerance by the NO-dependent upregulation of the antioxidant defense. Furthermore, the As tolerance of mustard seedlings could be enhanced by Ca treatment and aggravated by Ca chelation (Singh et al., 2020b). Using NO scavenger cPTIO with Ca, Ca's beneficial effect on biomass production, photosynthesis, and ROS diminution was remarkably reduced in the seedlings exposed to As. This result indicates that NO is an antioxidant molecule in the Ca-induced As tolerance. Similarly, Kaya et al. (2020c) revealed that NO was a regulatory intermediate in the salicylic acid (SA)-induced As tolerance of maize. The upregulating effect of SA on the AsA–GSH and glyoxalase systems was abolished by NO scavenging. This observation can be considered as an additional evidence of the antioxidant role of NO in As tolerance. Moreover, the As(V) treatment of rice seedlings promoted the formation of new adventitious roots (AR) but reduced the length and weight of the primary root (Kushwaha et al., 2019). L-NMMA could reverse the effect of As on AR, and the levels of $O_2^{\bullet-}$ and H_2O_2 were higher in the roots treated with As and L-NMMA than in the control. The effect of exogenous NO in this system was also examined. SNP was found to further increase the number of AR, decrease the level of ROS, and upregulate the dehydroascorbate reductase (DHAR) activity and AsA–GSH cycle in As-exposed plants. The inhibition of NOS-like activity-derived NO negatively influenced the cell-cycle dynamics, whereas SNP maintained it. Therefore, it can be concluded that NO is an endogenous regulator of As-induced AR formation that contributes to stress tolerance due to a more advanced root system.

In addition, the alleviation of As toxicity by exogenous NO donor (mostly SNP) treatments in various monocots and dicots has been uncovered (Bhat et al., 2021). Some studies apply both SNP and a NO scavenger, such as cPTIO, L-NAME, and Hb, to As-stressed plants in order to investigate the involvement of the endogenous and exogenous NO in As-triggered plant responses. In tall fescue leaves and bean seedlings, As-induced oxidative stress was characterized by the accumulation of $O_2^{\bullet-}$ and H_2O_2, the inactivation of antioxidant enzymes, and the intensified membrane damage (Jin et al., 2010; Talukdar, 2013). These adverse effects were mitigated by the SNP addition, whereas the damages were aggravated by the application of a NO scavenger such as cPTIO. These results indicate that endogenous NO upregulates antioxidant defense, thus reducing As-triggered oxidative stress. Recently, Souri et al. (2020) provided additional evidence for the beneficial role of endogenous NO in As tolerance in the hyperaccumulator species *Isatis cappadocica*. In this study, the application of bovine Hb as a NO scavenger and L-NAME as an inhibitor of mammalian NOS was found to worsen As-induced oxidative damages. Recently, the ameliorating effect of both exogenous and endogenous NO on arsenate toxicity in soybean has been demonstrated using pharmacological approach (Singh et al., 2021). Decreasing NO level by the application of L-NAME led to increased arsenate toxicity supporting that endogenous NO is involved in stress mitigation. Further results indicated that H_2O_2 may act downstream of NO signaling ameliorating arsenate toxicity (Singh et al., 2021).

Numerous studies utilized the standard experimental design, in which the control and As-exposed plants were supplemented with SNP as the exogenous NO donor at a concentration of 30–250 μM, most often 100 μM, primarily *via* the nutrient solution. Many results demonstrate that the use of SNP reduces As-induced ROS (mainly $O_2^{\bullet-}$, H_2O_2) overproduction and mitigates oxidative damages of lipids and DNA by upregulating a wide range of antioxidant components, such as SOD, APX, CAT, GPX, glutathione reductase (GR), POD, MDHAR, DHAR, glyoxalase I and II, AsA, GSH, phenols, and carotenoids (Singh et al., 2009, 2013, 2016, 2017a; Ismail, 2012; Farnese et al., 2013; Namdjoyan and Kermanian, 2013; Hasanuzzaman and Fujita, 2013; Saeid et al., 2014; Silveira et al., 2015; Mohamed et al., 2016; Andrade et al., 2016; Karam et al., 2017; Praveen and Gupta, 2018; Praveen et al., 2019; 2020; Chandrakar and Keshavkant, 2019; Ahmad et al., 2020). The reduction of the uptake, bioaccumulation, and translocation of As was demonstrated to be additional effects of SNP (Singh et al., 2009, 2013, 2016, 2017a, 2017b; Ismail, 2012; Namdjoyan and Kermanian, 2013; Silveira et al., 2015; Andrade et al., 2016; Praveen and Gupta, 2018; Praveen et al., 2019; Chandrakar and Keshavkant, 2019; Ahmad et al., 2020). SNP-derived NO was found to downregulate the expression of As transporters OsLis1 and OsLis2 and modulate metal transporters, particularly NIP, NRAMP, ABC, and iron transporters (Singh et al., 2016, 2017a, 2017b). Moreover, applying an NO donor activated intracellular As binding due to the promotion of heavy metal conjugating GST activity/expression and to the elevation of phytochelatin content (Singh et al., 2013, 2017b; Farnese et al., 2013). NO administration also improved the mineral nutrition of As-exposed plants by increasing the expression of genes and activity of enzymes that are involved in phosphorus, potassium, and, in particular, nitrogen and ammonium uptake and metabolism, *e.g.*, NR, nitrite reductase, glutamate dehydrogenase, and glutamine oxoglutarate aminotransferase (Mohamed et al., 2016; Praveen and Gupta, 2018; Praveen et al., 2019, 2020).

Another beneficial effect of SNP supplementation in As-treated

Table 2
The expression of NO-modified plant genes in metalloid-exposed plants. In all cases, NO treatment was applied in the form of sodium nitroprusside (SNP), and the gene expression was analyzed *via* RT-PCR.

Gene symbol	Function	Plant species	Treatments	Effect	References
OsSultr2;1 and *OsSultr4;1*	sulfate transport	*Oryza sativa*	6 µM Se(IV), 10 µM SNP, 6 µM Se(IV) plus 10 µM SNP for 4 days	all treatments cause upregulation	Xiao et al. (2017)
OsPT2	phosphate transport	*Oryza sativa*	6 µM Se(IV), 10 µM SNP, 6 µM Se(IV) plus 10 µM SNP for 4 days	all treatments cause upregulation	Xiao et al. (2017)
OsNIP2;1	silicon transport	*Oryza sativa*	6 µM Se(IV), 10 µM SNP, 6 µM Se(IV) plus 10 µM SNP for 4 days	all treatments cause downregulation	Xiao et al. (2017)
Osγ-ECS	glutathione synthesis	*Oryza sativa*	6 µM Se(IV), 10 µM SNP, 6 µM Se(IV) plus 10 µM SNP for 4 days	all treatments cause upregulation	Xiao et al. (2017)
OsGS	glutathione synthesis	*Oryza sativa*	6 µM Se(IV), 10 µM SNP, 6 µM Se(IV) plus 10 µM SNP for 4 days	SNP-induced upregulation in the absence and presence of Se(IV)	Xiao et al. (2017)
OsLsi1 and *OsLsi2*	arsenite transport	*Oryza sativa*	25 µM, 50 µM As(V), 100 µM SNP, 25 µM As (V) plus 100 µM SNP, 50 µM As (V) plus 100 µM SNP for 7 days	SNP decreases the As(V)-upregulated expressions	Singh et al. (2016)
OsYSL2, OsFRDL1, OsIRO2, OsIRT1	iron transport	*Oryza sativa*	25 µM, 50 µM As(V), 100 µM SNP, 25 µM As (V) plus 100 µM SNP, 50 µM As (V) plus 100 µM SNP for 7 days	SNP decreases the As(V)-upregulated expressions	Singh et al. (2016)
OsLsi2	arsenite transport	*Oryza sativa*	25 µM As(III), 30 µM SNP, 25 µM As(III) plus 30 µM SNP for 7 days	SNP-induced upregulation in the absence of As(III), and downregulation in the presence of As (III)	Singh et al. (2017a, 2017b)
OsNRAMP5	manganase transport	*Oryza sativa*	25 µM As(III), 30 µM SNP, 25 µM As(III) plus 30 µM SNP for 7 days	SNP-induced upregulation in the absence of As(III), and downregulation in the presence of As (III)	Singh et al. (2017a, 2017b)
OsIRT1, OsYSL2	iron transport	*Oryza sativa*	25 µM As(III), 30 µM SNP, 25 µM As(III) plus 30 µM SNP for 7 days	SNP induced-upregulation in the absence of As(III), and downregulation in the presence of As (III)	Singh et al. (2017a, 2017b)
OsLSi1	arsenite transport	*Oryza sativa*	25 µM As(III), 30 µM SNP, 25 µM As(III) plus 30 µM SNP for 7 days	SNP induced-downregulation in the absence of As(III) and in the presense of As(III)	Singh et al. (2017a, 2017b)
OsFRD1	iron transport	*Oryza sativa*	25 µM As(III), 30 µM SNP, 25 µM As(III) plus 30 µM SNP for 7 days	SNP induced-downregulation in the absence of As(III) and in the presense of As(III)	Singh et al. (2017a, 2017b)
OsIRO2	iron transport	*Oryza sativa*	25 µM As(III), 30 µM SNP, 25 µM As(III) plus 30 µM SNP for 7 days	SNP doesn't alter the expression in the absence of As(III) and it causes downregulation in the presense of As(III)	Singh et al. (2017a, 2017b)
OsPIN1a, OsPIN1b, OsPIN1c, OsPIN1d, OsPIN2, OsPIN5a, OsPIN5c, OsPIN8, OsPIN9, OSPIN10b	auxin transport	*Oryza sativa*	150 µM As(III), 100 µM SNP, 150 µM As(III) plus 100 µM SNP, for 2 days	SNP-induced upregulation in the absence and in the presence of As (III)	Praaven and Gupta (2018)
OsAMT, OSNIR, OsNTR	nitrogen uptake and metabolism	*Oryza sativa*	150 µM As(III), 100 µM SNP, 150 µM As(III) plus 100 µM SNP, for 2 days	SNP-induced upregulation in the absence and in the presence of As (III)	Praaven and Gupta (2018)
OsPHT	phosphate uptake	*Oryza sativa*	150 µM As(III), 100 µM SNP, 150 µM As(III) plus 100 µM SNP, for 2 days	SNP-induced downregulation in the absence of As(III) and upregulation in the presense of As (III)	Praaven and Gupta (2018)
OsKTP	potassium uptake	*Oryza sativa*	150 µM As(III), 100 µM SNP, 150 µM As(III) plus 100 µM SNP, for 2 days	SNP-induced downregulation in the absence of As(III) and upregulation in the presense of As (III)	Praaven and Gupta (2018)
BjAMT1;1, BjAMT2, BjNTR1;1, BjNTR1;2, BjNTR2;1, BjNTR2;7	nitrogen uptake	*Brassica juncea*	150 µM As(III), 100 µM SNP, 150 µM As(III) plus 100 µM SNP, for 2 days	SNP-induced downregulation in the presence of As(III)	Praaven and Gupta (2018)
BjNR1, BjNR2, BjNiR, BjGS1;1, BjGS2, BjGDH1, BjGDH2, BjASN2, BjFd-GOGAT, BjNADH-GOG	nitrogen metabolism	*Brassica juncea*	150 µM As(III), 100 µM SNP, 150 µM As(III) plus 100 µM SNP, for 2 days	SNP-induced downregulation in the presence of As(III)	Praaven and Gupta (2018)
BjPIN1a, BjPIN1b, BjPIN2c, BjPIN3, BjPIN5, BjPIN6,	auxin transport	*Brassica juncea*	150 µM As(III), 100 µM SNP, 150 µM As(III) plus 100 µM SNP, for 2 days	SNP-induced upregulation in the absence and presence of As(III)	Praaven and Gupta (2018)

(*continued on next page*)

Table 2 (*continued*)

Gene symbol	Function	Plant species	Treatments	Effect	References
BjPIN7b, BjPIN8a, BjPIN8b					
GmP5CS	proline synthesis	*Glycine max*	50 μM As(III), 75 μM SNP, 50 μM As(III) plus 75 μM SNP for 5 days	SNP-induced upregulation in the presence of As(III)	Chandrakar and Keshavkant (2019)

plants is the improvement of water balance by the reduction of transpiration and enhancement of sugar, glycine betaine, and Pro contents (Praveen and Gupta, 2018; Ahmad et al., 2020). Also, the production and activity of the Pro synthesis enzyme, pyrroline-5-carboxylate synthase (P5CS), was found to be induced by SNP (Chandrakar and Keshavkant, 2019). NO is also involved in the restoration of the hormone system disrupted by As since the application of SNP improved the level of the hormones, such as gibberellic acid, indole-3-acetic acid, kinetin, and benzyl adenine (Mohamed et al., 2016), and reduced the level of stress hormones, such as abscisic acid and jasmonic acid (Mohamed et al., 2016; Singh et al., 2017b). The expression of the PINOID (PIN) auxin efflux carrier gene was significantly enhanced in SNP-supplemented, As-exposed rice and mustard, possibly improving polar auxin transport and root growth (Praveen and Gupta, 2018; Praveen et al., 2019). In As-exposed *Arabidopsis*, the application of SNP enhances ROS formation, which is thought to cause IAA oxidation and consequently reduce IAA levels, thus equilibrating auxin homeostasis favoring lateral root induction (Piacentini et al., 2020a, 2020b).

A large amount of data indicates the diverse effects of NO in As-exposed plants. Beyond its antioxidant effect, NO reduces As uptake and accumulation as well as improves nutritional homeostasis, water balance, and hormonal status of plant tissues, resulting in enhanced stress endurance.

3.5. RNS contribute to Sb phytotoxicity via nitro-oxidative stress

Antimony (Sb) is a metalloid rarely studied by plant biologists, even though its accumulation in soils due to anthropogenic activity is toxic to all organisms, including plants (Feng et al., 2013).

Recently, besides ROS, the metabolism of RNS was studied in Sb-exposed sunflowers and tomatoes (Ortega et al., 2017; Espinosa-Vellarino et al., 2020). In both cases, the plants were treated in hydroponics with 0, 0.5, or 1 mM of Sb for similar periods. The accumulation of Sb was observed in the organs of both species, and it was found to reduce biomass production, chlorophyll content, and photosynthesis. Nutrient contents such as magnesium and iron were similarly decreased by Sb exposure, and most of the observed antioxidants, such as SOD, APX, GR, and POD, were induced in both species. In the root of Sb-exposed sunflowers and tomatoes, the SNO, $O_2^{\bullet -}$, and H_2O_2 levels significantly increased (Ortega et al., 2017; Espinosa-Vellarino et al., 2020), and the activity of GSNOR in sunflowers was induced mainly by the higher Sb concentration, indicating that Sb caused secondary nitro-oxidative stress (Ortega et al., 2017). In the study by Espinosa-Vellarino et al. (2020), Sb-exposed roots also exhibited elevated NO and $ONOO^-$ levels, further supporting the observation that Sb exposure disturbed RNS metabolism and induced nitro-oxidative stress in plants (Fig. 1). Exogenous NO may have a protective effect against Sb stress like it did to Se, but no experimental data regarding this issue is available.

4. Conclusion

In general, NO participates as a regulator in plant responses to excess metalloids and limited metalloid supply. NO plays diverse roles depending on the broad spectrum of actions of the metalloids. Moreover, it intensifies the stress-mitigating effect of Si, whereas in the case of Se, As, and Sb, the accumulation of NO/RNS contributes to toxicity. It promotes the positive effect of low concentrations of Se and enhances the damage caused by B deficiency. The participation of endogenous NO/RNS in metalloid-induced stress mitigation or damage is presented in Fig. 1. Additionally, the exogenous application of NO donor SNP reduces B, Se, and As toxicity. The current data suggest that the *general role of NO common for all metalloids* is to relieve secondary oxidative stress due to the activation of antioxidant defense at the level of protein activity and gene expression. Moreover, NO has been demonstrated to regulate the expression of genes involved in phytohormone metabolism, transport, nutrient transport, and nitrogen metabolism in metalloid-exposed plants. In some cases, such as Se, the NO-dependent regulation of metalloid-specific uptake systems at the gene expression level has been suggested; these can be considered as *metalloid-specific NO effects*. Also, the genes involved in the plant responses to metalloids affected by exogenous NO are summarized in Table 2.

5. Future perspectives of the research field

According to the cited publications, the research field on NO in plant responses to metalloids is relatively novel. So far, the studies have mainly described the effects of NO. However, in the near future, it is important to shift the research direction toward the examination of the molecular mechanisms of NO-dependent signaling. Protein targets of metalloid-induced *S*-nitrosation and tyrosine nitration should be characterized to explain the effects of NO.

Based on *in silico* prediction, we propose *S*-nitrosation as a putative mechanism for the NO-associated regulation of metalloid transporters; such a proposal needs to be supported by future experimental data. Furthermore, it is important to examine the expression of the NO-induced genes in plants exposed to metalloids and the genes involved in NO metabolism to further elucidate their molecular mechanisms. Methodological developments, such as the application of NO donors other than SNP such as GSNO and NO-releasing nanomaterials, are also necessary. In the case of SNP application, it is recommended to verify the NO-releasing capacity and investigate the reversibility of the effects using a NO scavenger. In several studies, inhibitors of mammalian NOS were used; such an approach should be treated with caution as the homolog of the enzyme in higher plants remains unidentified. At the same time, it is important to explore the unknown pathways of NO metabolism regulated by metalloids. Given the currently increasing environmental contamination worldwide, there is great potential in applying NO to mitigate metalloid stress. However, studying the regulatory role of NO is the requisite of its application.

CRediT authorship contribution statement

Zsuzsanna Kolbert: Conceptualization, Funding acquisition, Writing – original draft, Writing – review & editing. **Attila Ördög:** Software, Visualization, Writing – review & editing.

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Declaration of Competing Interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

References

Abedi, S., Iranbakhsh, A., Ardebili, Z.O., Ebadi, M., 2021. Nitric oxide and selenium nanoparticles confer changes in growth, metabolism, antioxidant machinery, gene expression, and flowering in chicory (*Cichorium intybus* L.): potential benefits and riskassessment. Environ. Sci. Pollut. Res. 28, 3136–3148.

Aftab, T., Khan, M.M.A., Naeem, M., Moinuddin, I.M., Teixeira da Silva, J.A., Ram, M., 2012. Exogenous nitric oxide donor protects *Artemisia annua* from oxidative stress generated by boron and aluminium toxicity. Ecotoxicol. Environ. Saf. 80, 60–68.

Ahmad, P., Alam, P., Balawi, T.H., Altalayan, F.H., Ahanger, M.A., Ashraf, M., 2020. Sodium nitroprusside (SNP) improves tolerance to arsenic (As) toxicity in *Vicia faba* through the modifications of biochemical attributes, antioxidants, ascorbate-glutathione cycle and glyoxalase cycle. Chemosphere 244, 125480. https://doi.org/10.1016/j.chemosphere.2019.125480.

Ahmad, A., Khan, W.U., Shah, A.A., Yasin, N.A., Naz, S., Ali, A., Tahir, A., Batool, A.I., 2021. Synergistic effects of nitric oxide and silicon on promoting plant growth, oxidative stress tolerance and reduction of arsenic uptake in *Brassica juncea*. Chemosphere 262, 128384. https://doi.org/10.1016/j.chemosphere.2020.128384.

Aibara, I., Hirai, T., Kasai, K., Takano, J., Onouchi, H., Naito, S., Fujiwara, T., Miwa, K., 2018. Boron-dependent translational suppression of the borate exporter BOR1 contributes to the avoidance of boron toxicity. Plant Physiol. 177, 759–774.

Amo, D.Y., Brzezinski, M.A., 1999. The chemical form of dissolved Si taken up by marine diatoms. J. Phycol. 35, 1162–1170.

Andrade, H.M., Oliveira, J.A., Farnese, F.S., Ribeiro, C., Silva, A.A., Campos, F.V., Neto, J.L., 2016. Arsenic toxicity: cell signalling and the attenuating effect of nitric oxide in *Eichhornia crassipes*. Biol. Plant. 60, 173–180.

Azeem, M., Iqbal, N., Kausar, S., Javed, M.T., Akram, M.S., Sajid, M.A., 2015. Efficacy of silicon priming and fertigation to modulate seedling's vigor and ion homeostasis of wheat (*Triticum aestivum* L.) under saline environment. Environ. Sci. Pollut. Res. 22, 14367–14371.

Barroso, J.B., Corpas, F.J., Carreras, A., Rodríguez-Serrano, M., Esteban, F.J., Fernández-Ocaña, A., Chaki, M., Romero-Puertas, M.C., Valderrama, R., Sandalio, L.M., del Río, L.A., 2006. Localization of S-nitrosoglutathione and expression of S-nitrosoglutathione reductase in pea plants under cadmium stress. J. Exp. Bot. 57, 1785–1793.

Begara-Morales, J.C., Sánchez-Calvo, B., Chaki, M., Valderrama, R., Mata-Pérez, C., Padilla, M.N., Corpas, F.J., Barroso, J.B., 2016. Antioxidant systems are regulated by nitric oxide-mediated post-translational modifications (NO-PTMs). Front. Plant Sci. 7, 152. https://doi.org/10.3389/fpls.2016.00152.

Bhat, J.A., Ahmad, P., Corpas, F.J., 2021. Main nitric oxide (NO) hallmarks to relieve arsenic stress in higher plants. J. Hazard. Mater. 406, 124289 https://doi.org/10.1016/j.jhazmat.2020.124289.

Borbély, P., Molnár, Á., Valyon, E., Ördög, A., Horváth-Boros, K., Csupor, D., Fehér, A., Kolbert, Zs, 2021. The effect of foliar selenium (Se) treatment on growth, photosynthesis, and oxidative-nitrosative signalling of *Stevia rebaudiana* leaves. Antioxidants 10, 72. https://doi.org/10.3390/antiox10010072.

Chandrakar, V., Keshavkant, S., 2019. Nitric oxide and dimethylthiourea up-regulates pyrroline-5-carboxylate synthetase expression to improve arsenic tolerance in *Glycine max* L. Environ. Prog. Sustain. Energy 38. https://doi.org/10.1002/ep.12978.

Chen, Y., Mo, H.Z., Hu, L.B., Li, Y.Q., Chen, J., Jang, L.F., 2014. The endogenous nitric oxide mediates selenium-induced phytotoxicity by promoting ROS generation in *Brassica rapa*. PLoS One 9, e110901. https://doi.org/10.1371/journal.pone.0110901.

Chiba, Y., Mitani, N., Yamaji, N., Ma, J.F., 2009. HvLsi1 is a silicon influx transporter in barley. Plant J. 57, 810–818.

Chung, Y.S., Kim, K.-S., Hamayun, M., Kim, Y., 2020. Silicon confers soybean resistance to salinity stress through regulation of reactive oxygen and reactive nitrogen species. Front. Plant Sci. 10, 1725. https://doi.org/10.3389/fpls.2019.01725.

Corpas, F.J., Chaki, M., Fernández-Ocaña, A., Valderrama, R., Palma, J.M., Carreras, A., Begara-Morales, J.C., Airaki, M., del Río, L.A., Barroso, J.B., 2008. Metabolism of reactive nitrogen species in pea plants under abiotic stress conditions. Plant Cell Physiol. 49, 1711–1722.

Coskun, D., Britto, D.T., Huynh, W.Q., Kronzucker, H.J., 2016. The role of silicon in higher plants under salinity and drought stress. Front. Plant Sci. 7, 1072. https://doi.org/10.3389/fpls.2016.01072.

Dai, Z., Rizwan, M., Gao, F., Yuan, Y., Huang, H., Hossain, M.M., Xiong, S., Cao, M., Liu, Y., Tu, S., 2020. Nitric oxide alleviates selenium toxicity in rice by regulating antioxidation, selenium uptake, speciation and gene expression. Environ. Pollut. 257, 113540 https://doi.org/10.1016/j.envpol.2019.113540.

Da Silva, C.J., Canatto, R.A., Cardoso, A.A., Ribeiro, C., Alves de Oliveira, J., 2018. Oxidative stress triggered by arsenic in a tropical macrophyte is alleviated by endogenous and exogenous nitric oxide. Braz. J. Bot. 41, 21–28.

de Sousa, A., Saleh, A.M., Habeeb, T.H., Hassang, Y.M., Zrieq, R., Wadaan, M.A.M., Hozzein, W.N., Selim, S., Matos, M., AbdElgawad, H., 2019. Silicon dioxide nanoparticles ameliorate the phytotoxic hazards of aluminum in maize grown on acidic soil. Sci. Total Environ. 693, 133636 https://doi.org/10.1016/j.scitotenv.2019.133636.

Djanaguiraman, M., Devi, D.D., Shanker, A.K., Sheeba, J.A., Bangarusamy, U., 2004. Impact of selenium spray on monocarpic senescence of soybean (*Glycine max* L.). Food Agric. Environ. 2, 44–47.

Djanaguiraman, M., Devi, D.D., Shanker, A.K., Sheeba, J.A., Bangarusamy, U., 2005. Selenium – an antioxidative protectant in soybean during senescence. Plant Soil 272, 77–86.

Dordas, C., Chrispeels, M.J., Brown, P.H., 2000. Permeability and channel-mediated transport of boric acid across membrane vesicles isolated from squash roots. Plant Physiol. 124, 1349–1361.

Dordas, C., Brown, P.H., 2001a. Permeability and the mechanism of transport of boric acid across the plasma membrane of *Xenopus laevis* oocytes. Biol. Trace Elem. Res. 81, 127–139.

Dordas, C., Brown, P.H., 2001b. Evidence for channel mediated transport of boric acid in squash (*Cucurbita pepo*). Plant Soil 235, 95–103.

El Kassis, E., Cathala, N., Rouached, H., Fourcroy, P., Berthomieu, P., Terry, N., Davidian, J.C., 2007. Characterization of a selenate-resistant *Arabidopsis* mutant. Root growth as a potential target for selenate toxicity. Plant Physiol. 143, 1231–1241.

Eraslan, F., Inal, A., Gunes, A., Alpaslan, M., 2007. Boron toxicity alters nitrate reductase activity, proline accumulation, membrane permeability, and mineral constituents of tomato and pepper plants. J. Plant Nutr. 30, 981–994.

Espinosa-Vellarino, F.L., Garrido, I., Ortega, A., Casimiro, I., Espinosa, F., 2020. Effects of antimony on reactive oxygen and nitrogen species (ROS and RNS) and antioxidant mechanisms in tomato plants. Front. Plant Sci. 11, 674. https://doi.org/10.3389/fpls.2020.00674.

Fancy, N.N., Bahlmann, A.-K., Loake, G.J., 2017. Nitric oxide function in plant abiotic stress. Plant Cell Environ. 40, 462–472.

Farag, M., Najeeb, U., Yang, J., Hu, Z., Fang, Z.M., 2017. Nitric oxide protects carbon assimilation process of watermelon from boron-induced oxidative injury. Plant Physiol. Biochem. 111, 166–173.

Farnese, F.S., de Oliveira, J.A., Gusman, G.S., Leão, G.A., Ribeiro, C., Siman, L.I., Cambraia, J., 2013. Plant responses to arsenic: the role of nitric oxide. Water Air Soil Pollut. 224, 1660. https://doi.org/10.1007/s11270-013-1660-8.

Feigl, G., Horváth, E., Molnár, Á., Oláh, D., Poór, P., Kolbert, Zs, 2019. Ethylene-nitric oxide interplay during selenium-induced lateral root emergence in *Arabidopsis*. J. Plant Growth Regul. 38, 1481–1488.

Feng, R., Wei, C., Tu, S., Ding, Y., Wang, R., Guo, J., 2013. The uptake and detoxification of antimony by plants: a review. Environ. Exp. Bot. 96, 28–34.

Fitzpatrick, K.L., Reid, R.J., 2009. The involvement of aquaglyceroporins in transport of boron in barley roots. Plant Cell Environ. 32, 1357–1365.

Foster, M.W., McMahon, T.J., Stamler, J., 2003. S-nitrosylation in health and disease. Trend Mol. Med. 9, 160–168.

Frew, A., Weston, L.A., Reynolds, O.L., Gurr, G.M., 2018. The role of silicon in plant biology: a paradigm shift in research approach. Ann. Bot. 121, 1265–1273.

Garbinski, L.D., Rosen, B.P., Chen, J., 2019. Pathways of arsenic uptake and efflux. Environ. Int. 126, 585–597.

González-Fontes, A., 2020. Why boron is an essential element for vascular plants. New Phytol. 226, 1228–1230.

Gottardi, S., Iacuzzo, F., Tomasi, N., Cortella, G., Manzocco, L., Pinton, R., Römheld, V., Mimmo, T., Scampicchio, S., Dalla Costa, L., Cesco, S., 2012. Beneficial effects of silicon on hydroponically grown corn salad (*Valerianella locusta* (L.) Laterr) plants. Plant Physiol. Biochem. 56, 14–23.

Guerriero, G., Hausman, J.F., Legay, S., 2016. Silicon and the plant extracellular matrix. Front. Plant Sci. 7, 463. https://doi.org/10.3389/fpls.2016.00463.

Hasanuzzaman, M., Fujita, M., 2013. Exogenous sodium nitroprusside alleviates arsenic-induced oxidative stress in wheat (*Triticum aestivum* L.) seedlings by enhancing antioxidant defense and glyoxalase system. Ecotoxicology 22, 584–596.

Hildebrand, M., Dahlin, K., Volcani, B., 1998. Characterization of a silicon transporter gene family in *Cylindrotheca fusiformis*: sequences, expression analysis, and identification of homologs in other diatoms. Mol. Gen. Genet. 260, 480–486.

Hogg, N., 2000. Biological chemistry and clinical potential of S-nitrosothiols. Free Radic. Biol. Med. 28, 1478–1486.

Hrmova, M., Gilliham, M., Tyerman, S.D., 2020. Plant transporters involved in combating boron toxicity: beyond 3D structures. Biochem. Soc. Trans. 48, 1683–1696.

Ismail, G.S.M., 2012. Protective role of nitric oxide against arsenic-induced damages in germinating mung bean seeds. Acta Physiol. Plant. 34, 1303–1311.

Jin, J.-W., Xu, Y.-F., Huang, Y.-F., 2010. Protective effect of nitric oxide against arsenic-induced oxidative damage in tall fescue leaves. Afr. J. Biotechnol. 9, 1619–1627.

Karam, E.A., Keramat, B., Asrar, Z., Mozafari, H., 2017. Study of interaction effect between triacontanol and nitric oxide on alleviating of oxidative stress arsenic toxicity in coriander seedlings. J. Plant Interact. 12, 14–20.

Kaya, C., Ashraf, M., 2015. Exogenous application of nitric oxide promotes growth and oxidative defense system in highly boron stressed tomato plants bearing fruit. Sci. Hortic. 185, 43–47.

Kaya, C., Sarioğlu, A., Akram, N.A., Ashraf, M., 2019. Thiourea-mediated nitric oxide production enhances tolerance to boron toxicity by reducing oxidative stress in bread wheat (*Triticum aestivum* L.) and durum wheat (*Triticum durum* Desf.) plants. J. Plant Growth Regul. 38, 1094–1109.

Kaya, C., Aslan, Mustafa, Uğurlar, M., Ashraf, M, F., 2020a. Thiamine-induced nitric oxide improves tolerance to boron toxicity in pepper plants by enhancing antioxidants. Turk. J. Agric. 44, 379–390.

Kaya, C., Akram, N.A., Ashraf, M., Alyemeni, M.N., Ahmad, P., 2020b. Exogenously supplied silicon (Si) improves cadmium tolerance in pepper (*Capsicum annuum* L.) by up-regulating the synthesis of nitric oxide and hydrogen sulfide. J. Biotechnol. 316, 35–45.

Kaya, C., Ashraf, M., Alyemeni, M.N., Corpas, F.J., Ahmad, P., 2020c. Salicylic acid-induced nitric oxide enhances arsenic toxicity tolerance in maize plants by upregulating the ascorbate-glutathione cycle and glyoxalase system. J. Hazard. Mater. 399, 123020 https://doi.org/10.1016/j.jhazmat.2020.123020.

Kobayashi, M., Miyamoto, M., Matoh, T., Kitajima, S., Hanano, S., Sumerta, I.N., Narise, T., Suzuki, H., Sakurai, N., Shibata, D., 2018. Mechanism underlying rapid responses to boron deprivation in *Arabidopsis* roots. Soil Sci. Plant Nutr. 64, 106–115.

Kolbert, Zs, Lehotai, N., Molnár, Á., Feigl, G., 2016. "The roots" of selenium toxicity: a new concept. Plant Signal. Behav. 11, e1241935 https://doi.org/10.1080/15592324.2016.1241935.

Kolbert, Zs, Feigl, G., Bordé, Á., Molnár, Á., Erdei, L., 2017. Protein tyrosine nitration in plants: present knowledge, computational prediction and future perspectives. Plant Physiol. Biochem. 113, 56–63.

Kolbert, Zs, Molnár, Á., Szőllősi, R., Feigl, G., Erdei, L., Ördög, A., 2018. Nitro-oxidative stress correlates with Se tolerance of *Astragalus* species. Plant Cell Physiol. 59, 1827–1843.

Kolbert, Zs., Szőllősi, R., Feigl, G., 2019a. Selenium-induced abiotic stress tolerance in plants. In: Mirza Hasanuzzaman, Masayuki Fujita, Hirosuke Oku, M. Tofazzal Islam (Eds), Plant Tolerance to Environmental Stress Role of Phytoprotectants. pp. 255–270.

Kolbert, Zs, Molnár, Á., Feigl, G., van Hoewyk, D., 2019b. Plant selenium toxicity: proteome in the crosshairs. J. Plant Physiol. 232, 291–300.

Kolbert, Z., Barroso, J.B., Brouquisse, R., Corpas, F.J., Gupta, K.J., Lindermayr, C., Loake, G.J., Palma, J.M., Petřivalský, M., Wendehenne, D., Hancock, J.T., 2019c. A forty-year journey: the generation and roles of NO in plants. Nitric Oxide 93, 53–70.

Kushwaha, B.K., Singh, S., Tripathi, D.K., Sharmad, S., Prasad, S.M., Chauhan, D.M., Kumar, V., Singh, V.P., 2019. New adventitious root formation and primary root biomass accumulation are regulated by nitric oxide and reactive oxygen species in rice seedlings under arsenate stress. J. Hazard. Mater. 361, 134–140.

Landi, M., Margaritopoulou, T., Papadakis, I.E., Araniti, F., 2019. Boron toxicity in higher plants: an update. Planta 250, 1011–1032.

Law, C., Exley, C., 2011. New insight into silica deposition in horsetail (*Equisetum arvense*). BMC Plant Biol. 11, 112. https://doi.org/10.1186/1471-2229-11-112.

Lehotai, N., Kolbert, Zs, Pető, A., Feigl, G., Ördög, A., Kumar, D., Tari, I., Erdei, L., 2012. Selenite-induced hormonal and signaling mechanisms during root growth of *Arabidopsis thaliana* L. J. Exp. Bot. 63, 5677–5687.

Lehotai, N., Feigl, G., Koós, Á., Molnár, Á., Ördög, A., Pető, A., Erdei, L., Kolbert, Zs, 2016a. Nitric oxide-cytokinin interplay influences selenite sensitivity in *Arabidopsis*. Plant Cell Rep. 35, 2181–2195.

Lehotai, N., Lyubenova, L., Schroeder, P., Feigl, G., Ördög, A., Szilagyi, K., Erdei, L., Kolbert, Zs, 2016b. Nitro-oxidative stress contributes to selenite toxicity in pea (*Pisum sativum* L.). Plant Soil 400, 107–122.

Leterrier, M., Chaki, M., Airaki, M., Valderrama, R., Palma, J.M., Barroso, J.B., Corpas, F. J., 2011. Function of S-nitrosoglutathione reductase (GSNOR) in plant development and under biotic/abiotic stress. Plant Signal. Behav. 6, 789–793.

Leterrier, M., Airaki, M., Palma, J.M., Chaki, M., Barroso, J.B., Corpas, F.J., 2012. Arsenic triggers the nitric oxide (NO) and S-nitrosoglutathione (GSNO) metabolism in *Arabidopsis*. Environ. Pollut. 166, 136–143.

Lewis, D.H., 2019. Boron: the essential element for vascular plants that never was. New Phytol. 221, 1685–1690.

Lewis, D.H., 2020. The status of boron as an essential element for vascular plants. New Phytol. 226, 1238–1239.

Li, H.-F., McGrath, S.P., Zhao, F.-J., 2008. Selenium uptake, translocation and speciation in wheat supplied with selenate or selenite. New Phytol. 178, 92–102.

Liang, X., Wang, H., Hu, Y., Mao, L., Sun, L., Dong, T., Nan, W., Bi, Y., 2015. Silicon does not mitigate cell death in cultured tobacco BY-2 cells subjected to salinity without ethylene emission. Plant Cell Rep. 34, 331–343.

Liu, X., Yin, L., Deng, X., Gong, D., Du, S., Wang, S., Zhang, Z., 2020. Combined application of silicon and nitric oxide jointly alleviated cadmium accumulation and toxicity in maize. J. Hazard. Mater. 395, 122679 https://doi.org/10.1016/j.jhazmat.2020.122679.

Lubega, J., Umbreen, S., Loake, G.J., 2021. Recent advances in the regulation of plant immunity by S-nitrosylation. J. Exp. Bot. 72, 864–872.

Ma, J.F., Tamai, K., Yamaji, N., Mitani, N., Konishi, S., Katsuhara, M., Ishiguro, M., Murata, Y., Yano, M., 2006. A silicon transporter in rice. Nature 440, 688–691.

Madany, M.M.Y., Saleh, A.M., Habeeb, T.H., Hozzein, W.N., AbdElgawad, H., 2020. Silicon dioxide nanoparticles alleviate the threats of broomrape infection in tomato by inducing cell wall fortification and modulating ROS homeostasis. Environ. Sci.: Nano 7, 1415. https://doi.org/10.1039/c9en01255a.

Mandlik, R., Thakral, V., Raturi, G., Shinde, S., Nikolić, M., Tripathi, D.K., Sonah, H., Deshmukh, R., 2020. Significance of silicon uptake, transport, and deposition in plants. J. Exp. Bot. 71, 6703–6718.

Manivannan, A., Ahn, Y.K., 2017. Silicon regulates potential genes involved in major physiological processes in plants to combat stress. Front. Plant Sci. 8, 1346. https://doi.org/10.3389/fpls.2017.01346.

Mitani, N., Yamaji, N., Ma, J.F., 2009. Identification of maize silicon influx transporters. Plant Cell Physiol. 50, 5–12.

Mohamed, H.I., Latif, H.H., Hanafy, R.S., 2016. influence of nitric oxide application on some biochemical aspects, endogenous hormones, minerals and phenolic compounds of *Vicia faba* plant grown under arsenic stress. Ges. Pflanz. 68, 99–107.

Molnár, Á., Feigl, G., Trifán, V., Ördög, A., Szőllősi, R., Erdei, L., Kolbert, Zs, 2018a. The intensity of tyrosine nitration is associated with selenite and selenate toxicity in *Brassica juncea* L. Ecotoxicol. Environ. Saf. 147, 93–101.

Molnár, Á., Kolbert, Zs, Kéri, K., Feigl, G., Ördög, A., Szőllősi, R., Erdei, L., 2018b. Selenite-induced nitro-oxidative stress processes in *Arabidopsis thaliana* and *Brassica juncea*. Ecotoxicol. Environ. Saf. 148, 664–674.

Namdjoyan, S., Kermanian, H., 2013. Exogenous nitric oxide (as sodium nitroprusside) ameliorates arsenic-induced oxidative stress in watercress (*Nasturtium officinale* R. Br.) plants. Sci. Hortic. 161, 350–356.

O'Neill, M.A., Warrenfeltz, K.K., Pellerin, P., Doco, T., Darvill, A.G., Albersheim, P., 1996. Rhamnogalacturonan-II: a pectic polysaccharide in the walls of growing plant cell, form a dimer that is covalently cross-linked by a borate ester: in vitro conditions for the formation and hydrolysis of the dimer. J. Biol. Chem. 271, 22923–22930.

Ortega, A., Garrido, I., Casimiro, I., Espinosa, F., 2017. Effects of antimony on redox activities and antioxidant defence systems in sunflower (*Helianthus annuus* L.) plants. PLoS One 12, e0183991. https://doi.org/10.1371/journal.pone.0183991.

Pandey, C., Khan, E., Panthri, M., Tripathi, R.D., Gupta, M., 2016. Impact of silicon on Indian mustard (*Brassica juncea* L.) root traits by regulating growth parameters, cellular antioxidants and stress modulators under arsenic stress. Plant Physiol. Biochem. 104, 216–225.

Piacentini, D., Corpas, F.J., D'Angeli, S., Altamura, M.M., Falasca, G., 2020a. Cadmium and arsenic-induced-stress differentially modulates Arabidopsis root architecture, peroxisome distribution, enzymatic activities and their nitric oxide content. Plant Physiol. Biochem. 148, 312–323.

Piacentini, D., Della Rovere, F., Sofo, A., Fattorini, L., Falasca, G., Altamura, M.M., 2020b. Nitric oxide cooperates with auxin to mitigate the alterations in the root system caused by cadmium and arsenic. Front. Plant Sci. 11, 1182 https://doi.org./10.3389/fpls.2020.01182.

Pirooz, P., Amooaghaie, R., Ahadi, A., Sharififar, F., 2021. Silicon- induced nitric oxide burst modulates systemic defensive responses of *Salvia officinalis* under copper toxicity. Plant Physiol. Biochem. 162, 752–761.

Pilon-Smits, E.A.H., Quinn, C.F., 2010. Selenium metabolism in plants. In: Hell, R., Mendell, R.R. (Eds.), Cell Biology of Metals and Nutrients, Plant Cell Monographs, 17. Springer-Verlag, Berlin Heidelberg, pp. 225–241.

Pommerrenig, B., Diehn, A.T., Bienert, G.P., 2015. Metalloido-porins: essentiality of Nodulin 26-like intrinsic proteins in metalloid transport. Plant Sci. 238, 212–227.

Praveen, A., Gupta, M., 2018. Nitric oxide confronts arsenic stimulated oxidative stress and root architecture through distinct gene expression of auxin transporters, nutrient related genes and modulates biochemical responses in *Oryza sativa* L. Environ. Pollut. 240, 950–962.

Praveen, A., Pandey, A., Gupta, M., 2019. Nitric oxide alters nitrogen metabolism and PIN gene expressions by playing protective role in arsenic challenged *Brassica juncea* L. Ecotoxicol. Environ. Saf. 176, 95–107.

Praveen, A., Pandey, A., Gupta, M., 2020. Protective role of nitric oxide on nitrogen-thiol metabolism and amino acids profiling during arsenic exposure in *Oryza sativa* L. Ecotoxicology 29, 825–836. https://doi.org/10.1007/s10646-020-02250-z.

Rao, K.P., Vani, G., Kumar, K., Wankhede, D.P., Misra, M., Gupta, M., Sinha, A.K., 2011. Arsenic stress activates MAP kinase in rice roots and leaves. Arch. Biochem. Biophys. 506, 73–82.

Rodríguez-Ruiz, M., Aparicio-Chacón, M.V., Palma, J.M., Corpas, F.J., 2019. Arsenate disrupts ion balance, sulfur and nitric oxide metabolisms in roots and leaves of pea (*Pisum sativum* L.) plants. Environ. Exp. Bot. 161, 143–156.

Saeid, Z.D., Zahra, A., Abdolhamid, N.S., 2014. Investigation of synergistic action between coronatine and nitric oxide in alleviating arsenic-induced toxicity in sweet basil seedlings. Plant Growth Regul. 74, 119–130.

Sajitha, B., Sigfredo, F., Dorin, G., 2021. Silicon modulates nitro-oxidative homeostasis along with the antioxidant metabolism to promote drought stress tolerance in lentil plants. Physiol. Plant. https://doi.org/10.1111/ppl.13437.

Sanz, L., Albertos, P., Mateos, I., Sánchez-Vicente, I., Lechón, T., Fernández-Marcos, M., Lorenzo, O., 2015. Nitric oxide (NO) and phytohormones crosstalk during early plant development. J. Exp. Bot. 66, 2857–2868.

Schiavon, M., Pilon-Smits, E.A.H., 2017. The fascinating facets of plant selenium accumulation – biochemistry, physiology, evolution and ecology. New Phytol. 213, 1582–1596.

Sharma, S.S., Kumar, V., Dietz, K.-J., 2021. Emerging trends in metalloid-dependent signaling in plants. Trend Plant Sci. 26, 452–471.

Shibagaki, N., Rose, A., McDermott, J.P., Fujiwara, T., Hayashi, H., Yoneyama, T., Davies, J.P., 2002. Selenate-resistant mutants of *Arabidopsis thaliana* identify Sultr1; 2, a sulfate transporter required for efficient transport of sulfate into roots. Plant J. 29, 475–486.

Silveira, N.M., de Oliveira, J.A., Ribeiro, C., Canatto, R.A., Siman, L., Cambraia, J., Farnese, F., 2015. Nitric oxide attenuates oxidative stress induced by arsenic in lettuce (*Lactuca sativa*) leaves. Water Air Soil Pollut. 226, 379. https://doi.org/10.1007/s11270-015-2630-0.

Singh, H.P., Kaur, S., Batish, D.R., Sharma, V.P., Sharma, N., Kohli, R.K., 2009. Nitric oxide alleviates arsenic toxicity by reducing oxidative damage in the roots of *Oryza sativa* (rice). Nitric Oxide 20, 289–297.

Singh, V.P., Srivastava, P.K., Prasad, S.M., 2013. Nitric oxide alleviates arsenic-induced toxic effects in ridged Luffa seedlings. Plant Physiol. Biochem. 71, 155–163.

Singh, V.P., Singh, S., Kumar, J., Prasad, S.M., 2015. Hydrogen sulfide alleviates toxic effects of arsenate in pea seedlings through up-regulation of the ascorbate–glutathione cycle: Possible involvement of nitric oxide. J. Plant Physiol. 181, 20–29.

Singh, A.P., Dixit, G., Kumar, A., Mishra, S., Singh, P.K., Dwivedi, S., Trivedi, P.K., Chakrabarty, D., Mallick, S., Pandey, V., Dhankher, O.P., Tripathi, R.D., 2016. Nitric oxide alleviated arsenic toxicity by modulation of antioxidants and thiol metabolism in rice (*Oryza sativa* L.). Front. Plant Sci. 6, 1272. https://doi.org/10.3389/fpls.2015.01272.

Singh, A.P., Dixit, G., Kumar, A., Mishra, S., Kumar, N., Dixit, S., Singh, P.K., Dwivedi, S., Trivedi, P.K., Pandey, V., Dhankher, O.P., Norton, G.J., Chakrabarty, D., Tripathi, R. D., 2017a. A protective role for nitric oxide and salicylic acid for arsenite phytotoxicity in rice (*Oryza sativa* L.). Plant Physiol. Biochem. 115, 163–173.

Singh, P.K., Indoliya, Y., Chauhan, A.S., Singh, S.P., Singh, A.P., Dwivedi, S., Tripathi, R. D., Chakrabarty, D., 2017. Nitric oxide mediated transcriptional modulation enhances plant adaptive responses to arsenic stress. Sci. Rep. 7, 3592. https://doi.org/10.1038/s41598-017-03923-2.

Singh, S., Prasad, S.M., Sharma, S., Dubey, N.K., Ramawat, N., Prasad, R., Singh, V.P., Tripathi, D.K., Chauhan, D.K., 2020a. Silicon and nitric oxide-mediated mechanisms of cadmium toxicity alleviation in wheat seedlings. Physiol. Plant. 13065. https://doi.org/10.1111/ppl.13065.

Singh, R., Parihar, P., Prasad, S.M., 2020b. Interplay of calcium and nitric oxide in improvement of growth and arsenic-induced toxicity in mustard seedlings. Sci. Rep. 10, 6900. https://doi.org/10.1038/s41598-020-62831-0.

Singh, S., Husain, T., Kushwaha, B.K., Suhel, M., Fatima, A., Mishra, V., Singh, S.K., Bhatt, J.A., Rai, M., Prasad, S.M., Dubey, N.K., Chauhan, D.K., Tripathi, D.K., Fotopoulos, V., Singh, V.P., 2021. Regulation of ascorbate-glutathione cycle by exogenous nitric oxide and hydrogen peroxide in soybean roots under arsenate stress. J. Hazard Mater. 409, 123686 https://doi.org/10.1016/j.jhazmat.2020.123686.

Solórzano, E., Corpas, F.J., González-Gordo, S., Palma, J.M., 2020. Reactive oxygen species (ROS) metabolism and nitric oxide (NO) content in roots and shoots of rice (*Oryza sativa* L.) plants under arsenic-induced stress. Agronomy 10, 1014. https://doi.org/10.3390/agronomy10071014.

Souri, Z., Karimi, N., Farooq, M.A., Sandalio, L.M., 2020. Nitric oxide improves tolerance to arsenic stress in *Isatis cappadocica* desv. shoots by enhancing antioxidant defenses. Chemosphere 239, 124523. https://doi.org/10.1016/j.chemosphere.2019.124523.

Souza, J.M., Peluffo, G., Radi, R., 2008. Protein tyrosine nitration - Functional alteration or just a biomarker? Free Radic. Biol. Med. 45, 357–366.

Takano, J., Noguchi, K., Yasumori, M., Kobayashi, M., Gajdos, Z., Miwa, K., Hayashi, H., Yoneyama, T., Fujiwara, T., 2002. Arabidopsis boron transporter for xylem loading. Nature 420, 337–340.

Takano, J., Wada, M., Ludewig, U., Schaaf, G., von Wirén, N., Fujiwara, T., 2006. The Arabidopsis major intrinsic protein nip5;1 is essential for efficient boron uptake and plant development under boron limitation. Plant Cell 18, 1498–1509.

Takano, J., Miwa, K., Fujiwara, T., 2008. Boron transport mechanisms: collaboration of channels and transporters. Trend Plant Sci. 13, 451–457.

Talukdar, D., 2013. Arsenic-induced oxidative stress in the common bean legume, *Phaseolus vulgaris* L. seedlings and its amelioration by exogenous nitric oxide. Physiol. Mol. Biol. Plants 19, 69–79.

Tanaka, M., Wallace, I.S., Takano, J., Roberts, D.M., Fujiwara, T., 2008. NIP6;1 is a boric acid channel for preferential transport of boron to growing shoot tissues in *Arabidopsis*. Plant Cell 20, 2860–2875.

Tang, Z., Zhao, F.J., 2020. The roles of membrane transporters in arsenic uptake, translocation and detoxification in plants. Crit. Rev. Environ. Sci. Technol. 1–36. https://doi.org/10.1080/10643389.2020.1795053.

Terrón-Camero, L.C., Peláez-Vico, M.Á., Del-Val, C., Sandalio, L.M., Romero-Puertas, M. C., 2019. Role of nitric oxide in plant responses to heavy metal stress: exogenous application versus endogenous production. J. Exp. Bot. 70, 4477–4488.

Tripathi, D.K., Singh, S., Singh, V.P., Prasad, S.M., Dubey, N.K., Chauhan, D.K., 2017. Silicon nanoparticles more effectively alleviated UV-B stress than silicon in wheat (*Triticum aestivum*) seedlings. Plant Physiol. Biochem. 110, 70–81.

Trippe, R.C., Pilon-Smits, E.A.H., 2021. Selenium transport and metabolism in plants: phytoremediation and biofortification implications. J. Hazard Mater. 404 (B), 124178 https://doi.org/10.1016/j.jhazmat.2020.124178.

Tun, N.N., Santa-Catarina, C., Begum, T., Silveira, V., Handro, W., Segal Floh, E.I., Scherer, G.F.E., 2006. Polyamines induce rapid biosynthesis of nitric oxide (NO) in *Arabidopsis thaliana* seedlings. Plant Cell Physiol. 47, 346–354.

Umbreen, S., Lubega, J., Cui, B., Pan, Q., Jiang, J., Loake, G.J., 2018. Specificity in nitric oxide signalling. J. Exp. Bot. 69, 3439–3448.

Valderrama, R., Corpas, F.J., Carreras, A., Fernández-Ocaña, A., Chaki, M., Luque, F., Gómez-Rodríguez, M.V., Colmenero-Varea, P., del Río, L.A., Barroso, J.B., 2007. Nitrosative stress in plants. FEBS Lett. 581, 453–461.

Vishwakarma, K., Singh, V.P., Prasad, S.M., Chauhand, D.K., Tripathi, D.K., Sharma, S., 2020. Silicon and plant growth promoting rhizobacteria differentially regulate AgNP-induced toxicity in *Brassica juncea*: implication of nitric oxide. J. Hazard. Mater. 390, 121806 https://doi.org/10.1016/j.jhazmat.2019.121806.

Wimmer, M.A., Abreu, I., Bell, R.W., et al., 2020. Boron: an essential element for vascular plants. New Phytol. 226, 1232–1237.

Xiao, Q., Li, X.-L., Gao, G.-F., Chen, J., Liu, X., Shen, Z.-J., Zhu, X.-Y., Zheng, H.-L., 2017. Nitric oxide enhances selenium concentration by promoting selenite uptake by rice roots. J. Plant Nutr. Soil Sci. 180, 788–799.

Xu, Y., Ding, J., Wu, L.-Y., Chou, K.-C., 2013. iSNO-PseAAC: predict cysteine S-nitrosylation sites in proteins by incorporating position specific amino acid propensity into pseudo amino acid composition. PLoS One 8, e55844. https://doi.org/10.1371/journal.pone.0055844.

Xue, M., Yi, H., 2017. Nitric oxide signaling is involved in arsenic-induced guard cell death in *Vicia faba* L. (*Fabaceae*). Braz. J. Bot. 40, 635–642.

Xue, T., Hartikainen, H., Piironen, V., 2001. Antioxidative and growth-promoting effect of selenium on senescing lettuce. Plant Soil 237, 55–61.

Xue, Y., Liu, Z., Gao, X., Jin, C., Wen, L., Yao, X., Ren, J., 2010. GPS-SNO: computational prediction of protein S-nitrosylation sites with a modified GPS algorithm. PLoS One 5, e11290. https://doi.org/10.1371/journal.pone.0011290.

Yoshinari, A., Takano, J., 2017. Insights into the mechanisms underlying boron homeostasis in plants. Front. Plant Sci. 8, 1951. https://doi.org/10.3389/fpls.2017.01951.

Zangi, R., Filella, M., 2012. Transport routes of metalloids into and out of the cell: A review of the current knowledge. Chem. Biol. Interact. 197, 47–57.

Zhang, L., Hu, B., Li, W., Che, R., Deng, K., Li, H., Yu, F., Ling, H., Li, Y., Chu, C., 2014. OsPT2, a phosphate transporter, is involved in the active uptake of selenite in rice. New Phytol. 201, 1183–1191.

Zhao, X.Q., Mitani, N., Yamaji, N., Shen, R.F., Ma, J.F., 2010. Involvement of silicon influx transporter OsNIP2;1 in selenite uptake in rice. Plant Physiol. 153, 1871–1877.

Zhu, Y.M., Yang, J.G., Wang, L.Z., Lin, Z.T., Dai, J.X., Wang, R.J., Yu, Y.S., Liu, H., Rensing, C., Feng, R.W., 2020. Factors influencing the uptake and speciation transformation of antimony in the soil-plant system, and the redistribution and toxicity of antimony in plants. Sci. Total Environ. 738, 140232 https://doi.org/10.1016/j.scitotenv.2020.140232.