Nitric oxide function in plant abiotic stress

Table 2: Differential S-nitrosylation of individual proteins related to abiotic stress.

S-nitrosylated proteins related to abiotic stress	Activity change after S-nitrosylation	Cellular localization of the protein	Reference
AHb1	ND	Cytoplasm	(Perazzolli *et al.* 2004)
APX	+	Cytoplasm	(Bai *et al.* 2011; Begara-Morales *e*
GAPDH (*in vitro* only)	-	Cytoplasm	(Wawer *et al.* 2010; Lin *et al.*
OST1/SnRK2.6	-	Cytoplasm	(Wang *et al.* 2015)
PAL	-	Cytoplasm	(Vanzo *et al.* 2014)
Trx	ND	Cytoplasm	(Lin *et al.* 2012)
GR	+/=	Cytoplasm & Chloroplast	(Bai *et al.* 2011; Begara-Morales *e*
Rubisco	-	Chloroplast	(Abat & Deswal 2009)
PrxII F	-	Mitochondria	(Camejo *et al.* 2013)
CAT	-	Peroxisome	(Ortega-Galisteo *et al.* 20
DHAR	+	Peroxisome	(Bai *et al.* 2011)
GOX	-	Peroxisome	(Ortega-Galisteo *et al.* 20
MDAR	-	Peroxisome	(Begara-Morales *et al.* 20
MDH	-	Peroxisome	(Ortega-Galisteo *et al.* 20

Key: ND, Not Detected; +, increased; -, decreased; =, not changed.

Figure Legends

Figure 1. Regulation of S-nitrosylation. S-nitrosylation is the attachment of an NO group to a cysteine (Cys) thiol (SH) to form an S-nitrosothiol (SNO). The SNO bond is reversible due to its redox sensitivity: i.e. ascorbate, reduced metal ions and GSH can reduce SNO to SH. S-nitrosoglutathione (GSNO), which acts as a stable NO reservoir, can transfer NO to SH group via transnitrosylation. Turnover of GSNO occurs via the enzyme GSNO reductase (GSNOR), generating oxidized glutathione (GSSG) and ammonia (NH_3).

Figure 2. Regulation of ascorbate-glutathione cycle by S-nitrosylation during salt stress. During salt stress the ascorbate–glutathione cycle is modulated via S-nitrosylation of APX, MDAR and GR proteins. MDAR activity is reduced after S-nitrosylation while GR activity is not significantly affected by this modification. However, APX activity is enhanced by S-nitrosylation. This regulation suggests that APX works to detoxify hydrogen peroxide while GR supports the regeneration of GSH, thereby maintaining the antioxidant capability of the ascorbate-glutathione cycle under stress conditions. APX, ascorbate peroxidase; GR, Glutathione reductase; MDAR, monodehydroascorbate reductase.

Figure 3. Model showing the role of NO and S-nitrosylation during drought stress signalling. Drought stress triggers ABA accumulation which in turn can induce the production of H_2O_2 leading to the generation of NO, which results in stomatal closure via the activation of a MAPK pathway. On the other hand, OST1/SnRK2.6 is a central component that regulates ABA signalling via a phosphorylation cascade. NO can regulate ABA signaling through S-nitrosylation of OST1/SnRK2.6, which inhibits its kinase activity, thus acting as a negative regulator of ABA signalling.

Figure 4. S-nitrosylation regulates different nodes of abiotic stress signalling. During abiotic stress, proteins related to different nodes of stress signalling become targets for S-nitrosylation/denitrosylation. These proteins include antioxidant proteins, proteins involved in RNS/ROS, cellular metabolism and also proteins that regulate programmed cell death (PCD). These proteins change their S-nitrosylation pattern during stress to control cell fate in two ways: either

stress acclimation or PCD. Rubisco, ribulose-1,5-bisphosphate carboxylase/oxygenase; GAPDH, glyceraldehyde 3-phosphate dehydrogenase; Trx, thioredoxin; AHb1, haemoglobin 1; APX, ascorbate peroxidase; GR, Glutathione reductase; DHAR, dehydroascobate reductase; GOX, glycolate oxidase; PrxII F, peroxiredoxin II F; MDAR, monodehydroascorbate reductase; CAT, catalase.

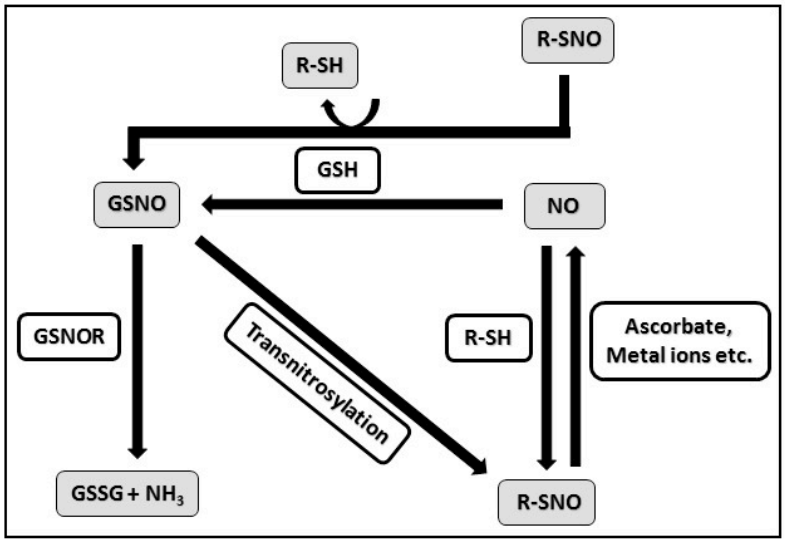

Figure 1. Regulation of S-nitrosylation.

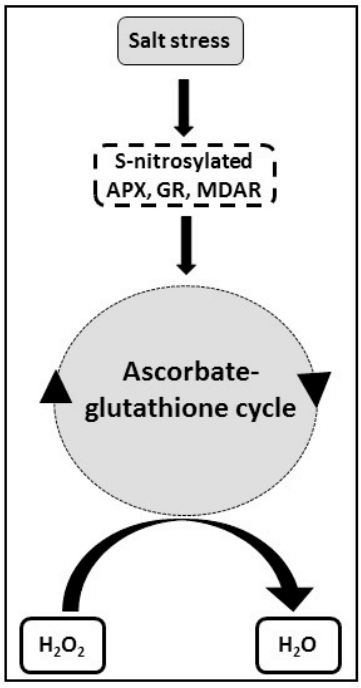

Figure 2. Regulation of ascorbate-glutathione cycle by S-nitrosylation during salt stress.

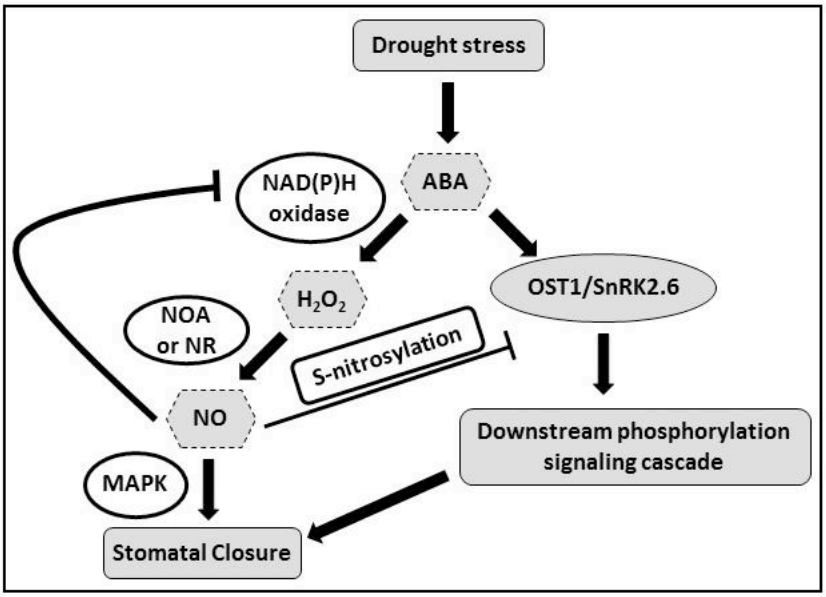

Figure 3. Model showing the role of NO and S-nitrosylation during drought stress signalling.

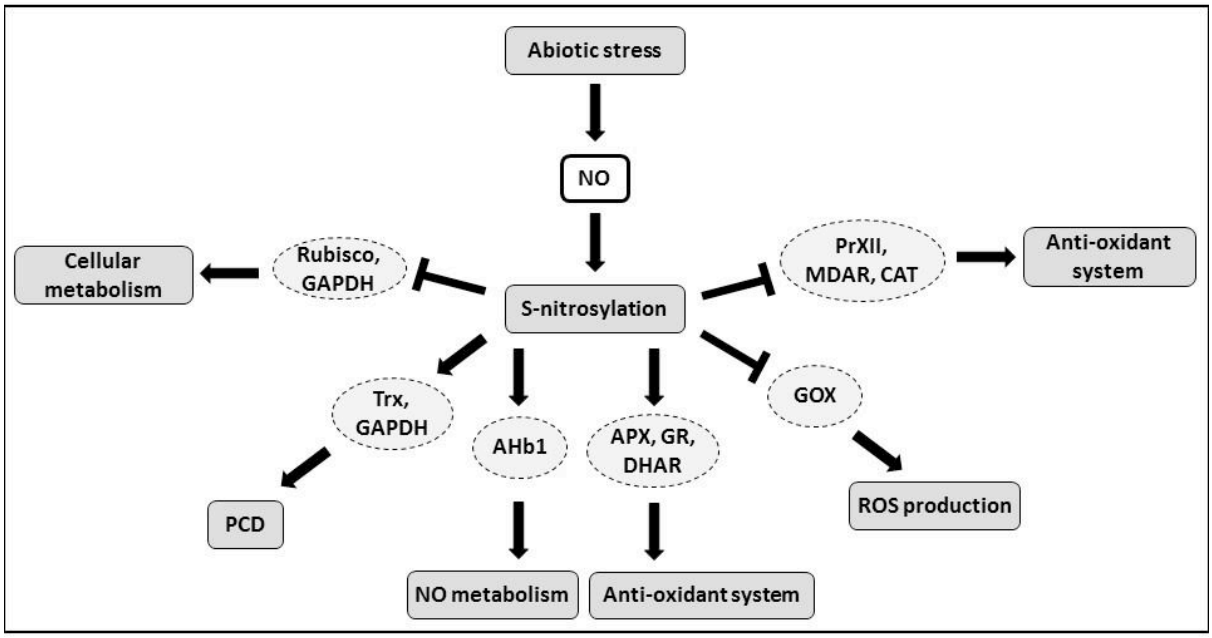

Figure 4. S-nitrosylation regulates different nodes of abiotic stress signalling.