Nitric oxide could allay arsenic phytotoxicity in tomato (*Solanum lycopersicum* L.) by modulating photosynthetic pigments, phytochelatin metabolism, molecular redox status and arsenic sequestration

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Plant Physiology and Biochemistry

Abstract

Plants do not always have the genetic capacity to tolerate high levels of arsenic (As), which may not only ~~may~~ arrest their growth but ~~commit~~ pose potential health risks ~~by~~ through dietary bioaccumulation. ~~However~~Meanwhile, the interplay between the tomato plants and As-NO-driven molecular cell dynamics is obscure. ~~Hence~~Accordingly, seedlings were treated with As (10 mg/L) alone or in combination with 100 μM sodium nitroprusside (SNP, NO donor) and 200 μM 2-(4-carboxyphenyl)-4,4,5,5-tetramethylimidazoline-1-oxyl-3-oxide (cPTIO, NO scavenger). Sodium nitroprusside immobilized As in the roots and reduced the shoot translocation by up-regulating the transcriptional expression of the *PCS*, *GSH1*, *MT2,* and *ABC1*. SNP further restored the growth retardation ~~by~~ through modulating the chlorophyll and proline metabolism, increasing NO accumulation and stomatal conductance along with clear crosstalk between the ~~activity of antioxidants~~antioxidant activity as well as glyoxalase I and II leading to endogenous H_2O_2 and MG ~~decrease~~reduction. Higher PCs and glutathione accumulation helped ~~to~~ protect photosynthetic apparatus; however, cPTIO reversed the protective effects of SNP, confirming the role of NO in the As toxicity alleviation.

Introduction