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Differential response of oxidative stress and thiol metabolism in contrasting rice genotypes for arsenic tolerance

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The mechanism of arsenic (As) tolerance was investigated on two contrasting rice (*Oryza sativa* L.) genotypes, selected for As tolerance and accumulation. One tolerant (Triguna) and one sensitive (IET-4786) variety were exposed to various arsenate (0–50 μM) levels for 7d for biochemical analyses. Arsenic induced oxidative stress was more pronounced in IET-4786 than Triguna especially in terms of reactive oxygen species, lipid peroxidation, EC and pro-oxidant enzymes (NADPH oxidase and ascorbate oxidase). However, Triguna tolerated As stress through the enhanced enzymes activities particularly pertaining to thiol metabolism such as serine acetyl transferase (SAT), cysteine synthase (CS), γ-glutamyl cysteine synthase (γ-ECS), γ-glutamyl transpeptidase (γ-GT), and glutathione-*S*-transferase (GST) as well as arsenate reductase (AR). Besides maintaining the ratio of redox couples GSH/GSSG and ASC/DHA, the level of phytochelatins (PCs) and phytochelatin synthase (PCS) activity were more pronounced in Triguna, in which harmonized responses of thiol metabolism was responsible for As tolerance in contrast to IET-4786 showing its susceptible nature towards As exposure.

1. Introduction

Arsenic is a food chain contaminant and class *I* carcinogen (Zhao et al., 2010). Currently, a considerable concern exists globally about arsenic (As) in potable water extracted from contaminated aquifers (Smedley and Kinniburgh, 2002). The sources of As include both natural (through dissolution of As compounds adsorbed onto pyrite ores into the water by geochemical factors) and anthropogenic (e.g., through use of insecticides, herbicides, and phosphate fertilizers, and from the semi-conductor industry) processes (Mondal et al., 2006). Arsenic contamination is prevailing in China, Russia and Vietnam and the situation is at its worst in the Bengal Delta (Bangladesh & West Bengal, India), where over 80 million people living in zones, having groundwater As level above 50 $\mu g\,L^{-1}$ (Smedley and Kinniburgh, 2002). Continuous irrigation of paddy fields with As contaminated groundwater has led to its build-up in paddy soil, with subsequent elevation of As in rice grain (Meharg and Rahman, 2003) and further its consumption constitutes a health risk for ~50% world population which depends on rice as a staple food (Norton et al., 2009).

Arsenic predominantly exists as inorganic arsenite (As^{III}) and arsenate (As^{V}) in nature. Arsenate is taken up by plants through high-affinity phosphate transporters (Meharg and Hartley-Whitaker, 2002; Tripathi et al., 2007) and disrupts energy flows in cells via competing with phosphate uptake. Being a redox active metalloid, As exposure also induces the generation of reactive oxygen species (ROS) through its intraconversion from one form to other and causing lipid peroxidation and associated toxicity (Srivastava et al., 2007; Mishra et al., 2008). However, an unrestrained increase in ROS level upon exposure to any abiotic stress including As may lead to oxidative stress (Srivastava et al., 2007). Therefore, the production and dismutation of ROS need to be harmonically balanced to allow plants to survive and grow both under normal and stressed conditions. To keep ROS production under control, plants are equipped with various enzymes and antioxidant compounds such as ascorbate (ASC) and glutathione (GSH) which are required for proper maintenance of redox state (Foyer and Noctor, 2009). NADPH plays important part in maintaining both ASC and GSH in a highly reduced state as an ultimate reductant. Such ROS-induced changes in ASC and/or GSH status are often taken as indicative of "oxidative stress". GSH is also required as a substrate for the synthesis of metalloid chelating ligands, the phytochelatins (PCs) (Grill et al., 2006). GSH is also used as reductant for enzymatic or nonenzymatic reduction of As^{V} to As^{III} (Bleeker et al., 2006), and further As^{III} is known to be complexed by both GSH and PCs (Raab et al., 2004).

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