REVIEW

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Cadmium stress tolerance in crop plants
Probing the role of sulfur

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Plants can't move away and are therefore continuously confronted with unfavorable environmental conditions (such as soil salinity, drought, heat, cold, flooding and heavy metal contamination). Among heavy metals, cadmium (Cd) is a non-essential and toxic metal, rapidly taken up by roots and accumulated in various plant tissues which hamper the crop growth and productivity worldwide. Plants employ various strategies to counteract the inhibitory effect of Cd, among which nutrient management is one of a possible way to overcome Cd toxicity. Sulfur (S) uptake and assimilation are crucial for determining crop yield and resistance to Cd stress. Cd affects S assimilation pathway which leads to the activation of pathway responsible for the synthesis of cysteine (Cys), a precursor of glutathione (GSH) biosynthesis. GSH, a non-protein thiol acts as an important antioxidant in mitigating Cd-induced oxidative stress. It also plays an important role in phytochelatins (PCs) synthesis, which has a proven role in Cd detoxification. Therefore, S assimilation is considered a crucial step for plant survival under Cd stress. The aim of this review is to discuss the regulatory mechanism of S uptake and assimilation, GSH and PC synthesis for Cd stress tolerance in crop plants.

Introduction

Abiotic stress is the main factor negatively affecting crop growth and productivity worldwide. Plants are continuously confronted with the harsh environmental conditions (such as soil salinity, drought, heat, cold, flooding and heavy metal contamination). The heavy metal, Cd is commonly released into the arable soil from industrial processes and farming practices[1] and has been ranked No. 7 among the top 20 toxins.[2] Even at low concentrations, Cd is toxic for most of the plants at concentrations greater than 5–10 μg Cd g^{-1} leaf dry weight,[3] except Cd-hyperaccumulators which can tolerate Cd concentrations of 100 μg Cd g^{-1} leaf dry weight.[4-6] In spite of its high phytotoxicity, Cd is easily taken up by plant roots and transported to above-ground tissues[7-9] and enters into the food chain where it may pose serious threats to human health.[10,11] The International Agency for Research on Cancer in 1993[12,13] classified Cd as a human carcinogen and, interestingly, it has also been suggested that crops are the main source of Cd intake by humans.[14,15] Being highly mobile in phloem,[16] Cd can be accumulated in all plant parts which causes stunted growth, chlorosis, leaf epinasty, alters the chloroplast ultrastructure, inhibits photosynthesis, inactivates enzymes in CO_2 fixation, induces lipid peroxidation, inhibits pollen germination and tube growth, and also disturbs the nitrogen (N) and sulfur (S) metabolism and antioxidant machinery.[17-23] Cd can also inhibit the activity of several groups of enzymes such as those of the Calvin cycle,[24] carbohydrate metabolism[25,26] and phosphorus metabolism.[27,28] The effect of Cd on nitrate and S assimilation has been studied in several plants showing an inhibition of the nitrate uptake rate and the activity of the enzymes involved in the N assimilation pathway.[11,29-35] Alternatively, Cd-caused induction of enzymes of S assimilation pathway has been reported in many plants.[19,36,37] Therefore, the regulation of S assimilation may be necessary to ensure an adequate supply of S compounds required for heavy metal tolerance. However, Cd is a non-redox active metal, but it induces the generation of reactive oxygen species (ROS) including superoxide radical ($O_2^{\bullet-}$), hydrogen peroxide (H_2O_2) and hydroxyl radical ($OH^{\bullet}$) (**Fig. 1**),[38,39] which has to be kept under tight control because the presence of Cd lead to excessive production of ROS causing cell death due to oxidative stress such as membrane lipid peroxidation, protein oxidation, enzyme inhibition and damage to nucleic acid.[11,39-42] To repair the Cd-induced inhibitory effects of ROS, plants employ ROS-detoxifying antioxidant defense machinery which includes non-enzymatic (glutathione, GSH; ascorbic acid, AsA; α-tocopherol and carotenoids) and enzymatic (superoxide dismutase, SOD; catalase, CAT; ascorbate peroxidase, APX; glutathione reductase, GR; monodehydroascorbate reductase, MDHAR; dehydroascorbate reductase, DHAR; glutathione peroxidase, GPX; guaicol peroxidase, GOPX and glutathione-*S*-transferase, GST) antioxidants.[11,39,43,44] Gao et al.[45] reported that Arabidopsis lysophospholipase 2 (lysoPL2) binds acyl-CoA-binding protein 2 (ACBP2) to mediate Cd(II) tolerance in transgenic *Arabidopsis thaliana*. ACBP2 shows protein-protein interactions with an ethylene-responsive element binding protein (AtEBP) and a farnesylated protein 6 (AtFP6). Overexpression of ACBP2 (acyl-CoA-binding protein 2), lysoPL2 (lysophospholipase 2) and AtFP6 (farnesylated protein 6) in Arabidopsis resulted in tolerance to Cd(II) when compared with wild type. ACBP2 can mediate tolerance to Cd(II)-induced oxidative stress by interacting with two protein partners, AtFP6 and lysoPL2.[46]

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