Effects of Cadmium Stress on Plants

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ABSTRACT

Cadmium (Cd) is a non-essential element readily taken up by the plants. Cd negatively affects plant metabolism affecting growth and development. It is released into the environment by a number of sources including power stations, metal working industries, heating systems, batteries, urban traffic, etc. Cd is recognized as an extremely deleterious pollutant due to its high toxicity and large solubility in water, and has been ranked number seven among the top 20 toxins. Having significant concern with plant and human health, Cd has been widely studied for its impacts on plant at various levels including metabolism. The objective of this review is to introduce the cadmium and provide an account of Cd toxicity in plants with main emphasis on photosynthetic parameters.

Keywords: Cadmium, Oxidative stress, Plants, Photosynthesis

1. INTRODUCTION

Cadmium (Cd) is a toxic heavy metal pollutant in the environment with a long biological half-life, originating mainly from industrial processes and phosphate fertilizers (Gill et al., 2013). Heavy metal refers to all chemical elements with a density greater than 5 g ml^{-1}. Cadmium is a heavy metal which classified as class C metals base of reactivity with the functional groups of biomolecules. Cadmium element with the symbol Cd has atomic number of 48 and atomic mass of 112.411 g. It has been located in the d-block and 12 group of the periodic table possessing and its electron configuration is [Kr] $4d^{10}$ $5s^2$.

Cadmium (Cd), released into agricultural lands not only induces numerous changes in plant growth and physiology but is also posing threats to human and other components of Ecosystem. In human, Cd is associated with numerous health hazards. Once absorbed, Cd is efficiently retained in the human body and accumulates for entire life. Cd is primarily toxic to the kidney, especially to the proximal tubular cells, the main site of accumulation. Cd can also cause bone demineralization, either through direct bone damage or indirectly as a result of renal dysfunction. In the industry, excessive exposures to airborne Cd may impair lung function and increase the risk of lung cancer (Bernard, 2008). In plants, the symptoms of Cd toxicity can be identified in form slight injury to lethality resulting into a crop failure. The main known mechanisms of Cd toxicity include its

affinity for sulfhydryl groups in proteins and its ability to replace some essential metals in active sites of enzymes, thus causing inhibition of enzyme activities and protein denaturation (Garg and Bhandari, 2013). Cd alters the levels of metabolic enzymes and indirectly induces oxidative stress by generating reactive oxygen species (ROS) (Romero-Puertas et al., 2004). The ROS react with lipids, proteins, pigments and nucleic acids and cause oxidative damage including lipid peroxidation leading to membrane damage (Chien et al., 2001).

Occurrence of toxicity in plants induced by heavy metals, particularly in agricultural and economic crops, poses a challenge for plant researchers associated with yield and quality in crops. On the other hand, mechanism of heavy metal accumulation potential and its detoxification (change of transitions state or binding to peptides) in plant may also provide us with the opportunities to remediate heavy metal contaminated soils biologically through growing metal accumulating species. Different perspectives and methods including potential use of phytoremediation process to clean-up contaminated soils have emerged among researchers. Some have examined the naturally occurring metal hyperaccumulators (Qadir et al., 2004), while others have developed transgenic plants (Zhu et al., 1999; Song et al., 2003; Kumar and Sinha, 2013).

Extensive progress has been made in characterizing the soil chemistry needed for phytoremediation and physiology of plant that hyperaccumulate and hypertolerate metals. High rates of uptake and translocation are observed in hyperaccumulator plants. Obviously metal tolerant plant must be able to prevent the absorption of excess Cd and, if taken up, detoxify the Cd after being absorbed but molecular events altered in moderately tolerant plants should also be understood at concentration of heavy metals occurring in nature. Thus, it becomes of utmost importance to clean Cd from environment and reduce the risk of Cd that causes toxicities. Traditional clean up processes of heavy metals pollution are expensive and environmentally destructive (Meagher, 2000). At present, scientists and engineers are emphasizing on generation of cost effective technologies that include the use of micro organisms, biomass and live plants in cleaning process of polluted areas (Boyajian and Carreira, 1997; Wasay et al., 1998), so as to reduce the risk of cadmium being incorporated into food chain. These strategies would help in cleanup of contaminated soils via bioaccumulation and may be of particular interest for agricultural soils as these will help in restoration of soil fertility and the possible re-use of agricultural land (Frey et al., 2000; Perronnet et al., 2000).

In response to the presence of Cd, cell rapidly induces or upregulates the synthesis of phytochelatins (PCs) which are thiol-based complex forming substances (Bashir et al., 2013). This becomes possible through the up-regulation of glutathione biosynthesis (Xiang and Oliver, 1998). As PCs are glutamate- and cysteine-rich peptides, sulfate and nitrate assimilation pathways are most likely involved in their synthesis: enzymes of the sulfur metabolism are required for the

synthesis of cysteine, enzymes of nitrogen metabolism are required for the synthesis of glutamate (Astolfi et al., 2004).

According to the assessment of data produced in response of Cd toxicity, exposure of plants results in so many physiological breakdowns that it becomes nearly impossible to determine which effects are primary and which are secondary (Prasad, 1995). However, numerous impact of Cd stress have been worked out varying from induction of oxidative stress to stunted growth (Table 1).

Table 1: Symptoms induced in plants exposed to cadmium stress

Symptoms	References (from research and review articles)
Oxidative stress and damaged thylakoids	Qureshi et al., 2010
Stunted growth	Schützendübel et al., 2001; Qadir et al., 2004; Marquez-Garcia et al., 2011; Bagheri et al., 2013
Chlorosis	Sanità di Toppi and Gabrielli 1999; Mishra et al., 2006; Marquez-Garcia et al., 2011; Gill and Tuteja, 2011
Leaf epinasty	Mishra et al., 2006; Marquez-Garcia et al., 2011; Gill and Tuteja, 2011
Alters the chloroplast ultrastructure	Mishra et al., 2006; Marquez-Garcia et al., 2011; Gill and Tuteja, 2011
Reduction in the transpiration and photosynthesis rate	Schützendübel et al., 2001; Sanitá di Toppi and Gabbrie 1999; Marquez-Garcia et al., 2011
Inactivates enzymes in CO_2 fixation	Mishra et al., 2006; Marquez-Garcia et al., 2011; Gill and Tuteja, 2011
Inhibition the activity of several groups of enzymes	Sandalio et al., 2001; Gill and Tuteja, 2011;
Inhibition of the enzymes in carbohydrate metabolism	Sanita di Toppi et al., 1999; Verma and Dubey et al., 2001; Gill and Tuteja, 2011
Inhibition of the enzymes in phosphorus metabolism	Shah et al., 1998; Sharma et al., 2006; Gill and Tuteja, 2011

Inhibition of the nitrate uptake rate	Gill and Tuteja, 2011
Inhibition the activity of the enzymes involved the N assimilation pathway.	Hasan et al., 2005; Gill et al., 2011
Reduction in chlorophyll content of leaves	Schützendübel et al., 2001
Lipid peroxidation	OMishra et al., 2006; Cuypers et al., 2010; Skórzyńska-Polit et al., 2010; Marquez-Garcia et al., 2011
Inhibits pollen germination and tube growth	Mishra et al., 2006; Marquez-Garcia et al., 2011; Gill and Tuteja, 2011
Disturbs the nitrogen (N) metabolism	Mishra et al., 2006; Marquez-Garcia et al., 2011; Gill and Tuteja, 2011
Disturbs the sulfur (S) metabolism	Mishra et al., 2006; Marquez-Garcia et al., 2011; Gill and Tuteja, 2011
Disturbs antioxidant machinery	Schützendübel et al., 2001; Mishra et al., 2006; Marquez-Garcia et al., 2011; Gill and Tuteja, 2011
Cell death	Nocito et al., 2007; Bagheri et al., 2013

Despite recent progress in understanding individual aspects of metal toxicity and resistance mechanisms, little is known about the coordination of cellular sequestration mechanisms with adaptation of plant growth. Plant uptake of Cd from soil solution is dependent in a system that is largely metabolically mediated and competitive with the uptake system for Zn, Fe and possibly other metals. This idea comes from a number of common symptoms of Cd toxicity, Zn and Fe-deficiency. Thus not only causing toxicity at individual level, Cd is also posing a competition to other ions which might be nutrients and their scarcity resulting into mineral nutrient deficiencies. A careful look up for a potential site of sufferings due to Cd, the photosynthetic apparatus appears to be particularly susceptible. Substantial inhibition of PSII activities caused by Cd has been reported and is accompanied or followed by such phenomena as the disappearance of granal stacks, degradation of thylakoid acyl lipids, release of some polypeptides associated with the oxygen evolving complex and disorganization of light harvesting complex II antenna system (Tukendorf and Baszynski, 1991). Photosystem II is essential for the regulation of photosynthesis, because it

catalyses the oxidation of water and supports electron transport. It consists of a core, a light harvesting antenna and an oxygen evolving system. The core of this photosystem is composed of reaction centre proteins known as D1 and D2, cytochrome CP47, the 33 kDa manganese stabilizing protein and several minor proteins. All pigments and prosthetic groups necessary for charge separation and stabilization are bound to D1 and D2 proteins. Heavy metal involves the breakdown of PSII core proteins like D1, which is particularly vulnerable component of the chloroplast. This damage may be further amplified in a destruction of positive feedback, damage to D1 induces changes in redox-potential itself and causing additional damage to D1 (Öquist et al., 1992).

At the basic cause of the problem might be the fact that the photosynthesis is coupled with a series of electron transport pigment-protein complexes forming an organized system and metal ions are suggested to block the electron transport pathway, thus in principle, causes inactivation of PSII as a result of Cd induced imbalance between changes in energy supply and energy consumption (Qureshi et al., 2010). The initial effects of environmental changes which affect redox chemistry are most clearly seen in the chloroplasts and mitochondria. Considering all the above facts, photosynthetic carbon fixation is the primary target of Cd toxicity. CO_2 fixation by the photosynthetic carbon fixation cycle acts as a sink for the product of photosynthetic electron transport.

The major part of reducing equivalents (NADPH and ATP) generated by the primary light reaction is utilized by carbon metabolism. Oxidation of NADPH continuously restores the terminal electron acceptor and permits a steady state of photochemical de-excitation of reaction centres. It is also an essential feature of the coupling of electron transport to ATP synthesis that the proton motive forces can bring about feedback inhibition of electron flow. The significance of such control mechanism serves as a dissipation mechanism for excessive excitation energy when the rate of ATP and NADPH synthesis exceeds demands.

Therefore, any factor which diminishes the utilization of photosynthetic energy in carbon metabolism and affects high-energy state non-photochemical quenching e.g. Cd ions will modify the rate of electron transport through PSII. It may influence the quantum yield of linear electron transport. Further, heavy metal tends to accumulate in the chloroplasts resulting in an ion imbalance, which can have very deleterious consequences on chloroplast activity. It is believed that this may be the reason for the observed photosynthetic multi-proteins disassembling (Qureshi et al., 2010).

Thus, Cd primarily seems targeting photosynthetic assembly and ultimately affecting photosynthetic efficiency of plants resulting into poor health and stunted growth.

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