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Toxicity of Aluminium on Plants Physiological and Metabolic Functions

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ABSTRACT

Aluminium (Al) toxicity is one of the major factor that limit plant growth and development in acid soils. About 50% of the world's potentially arable lands are acidic in nature and Al toxicity occurs at pH <5.5 due to solubilisation of toxic forms of Al into the soil solution. In (µ) molar concentrations of Al inhibits root growth and impairment physiological and metabolic functions of plant. It is important to understand the mechanism of Al toxicity and resistance, for stable food production in future. This article examines our current understanding regarding impact of Al toxicity on plants physiological and metabolic functions.

It is estimated that around 30% of the world's total land area consists of acid soils, and about 50% of the world's arable lands are acidic. Moreover, up to 60% of the acid soils in the world occur in developing countries like South America, Central Africa and Southeast Asia due to which, crop production is a critical challenge in this region. Aluminium (Al) toxicity in acid soils affects the production of staple food crops, particularly grain crops, by decreasing their yield and vigor. Although the poor fertility of acid soils is due to some of the minerals toxicity (Al, Fe and Mn) and deficiencies (P, Ca, Mg and Mo), among them Al toxicity is the major constraint for crop production on 67% of the total acid soil area. Several plant species are susceptible to the µmolar concentration of Al and root growth inhibition is certainly the most easily recognizable trait of Al toxicity which can widely be marked as a measure of Al toxicity in plants (Schmitt *et al.,* 2016). However, the meticulous mechanism responsible for Al toxicity is still not well known. As Al^{3+} poses the capacity of interaction with a number of intra and extracellular components and various mechanisms of Al^{3+} toxicity have been proposed such as cell wall modification, interruption of signaling pathways, disruption and depolarization of the plasma membrane, modified transport processes and Al^{3+} binding to the DNA (Kochian *et al.*, 2005). In this article, the salient features of Al toxicity on plant physiological and metabolic functions are highlighted.

Aluminium toxicity on root

The most easily affected region of Al toxicity is the root in plant and root growth inhibited at lower dose of Al. Root elongations a process of cell division, but Al phyto-toxicity blocks the mechanism of cell division in the meristem and cell elongation in the elongation zone is inhibited (Verbelen *et al.,* 2006). As a result of this, root become stunted and brittle, root hair development is poor and the root apices become swollen and damage. The root apex *i.e.*, root cap, meristem and elongation zone is highly sensitive to Al and accumulates Al very easily. As a result it causes greater physical damage which ultimately leads to lower ion and water absorption (Barceló and Poschenrieder, 2002).

Aluminium toxicity on leaves

The symptoms of Al toxicity are not easily identifiable. In plants, the foliar symptoms resemble to Phosphorus (P) deficiency such as overall stunting, small, dark green leaves and late maturity, purpling of stems, leaves, and leaf veins, yellowing and death of leaf tips. In some cases, Al toxicity appears as an induced Calcium (Ca) deficiency which produces symptoms like curling or rolling of young leaves and collapse of growing points or petioles. Excess Al even induces Iron (Fe) deficiency symptoms in leaves of rice, sorghum and wheat (Foy and Fleming, 1982).

Aluminium toxicity on cell wall

The primary binding of Al^{3+} in the apoplast is probably the pectin matrix, with its negatively charged carboxylic groups. Aluminium absorption in the plant cell wall reduces the movement of water and solutes through the apoplasm which directly decreases nutrient acquisition by the root (Blamey, 2001). Aluminium crosses links with pectin and increases cell wall rigidity thus leading to decrease in the mechanical stability and ultimately decrease in cell growth. Cell wall cations are strongly

replaced by Al^{3+}, finally resulting in drastic change in cell wall structural and mechanical properties (Kochian *et al*, 2005).

Aluminium toxicity on plasma membrane

Depending on pH and other factors, Al can bind either to proteins or lipids of plasma membrane. As Al has greater affinity for the choline head of phosphotidyl choline, it displaced other cations like Ca that may form bridges between the phospholipid head groups of the membrane bi-layer (Akeson *et al.,* 1989). Aluminium interaction with plasma membrane could lead to depolarization of the trans-membrane potential and reduction of H^+-ATPase (Ahn *et al.,* 2002) which alter the activities of ions near the plasma membrane surface and impaired the formation and maintenance of the trans-membrane H^+ gradient (Kochian *et al.,* 2005). These changes are related to direct Al^{3+} interactions with plasma membrane ion channels and changes in membrane potential which finally leads to nutritional imbalances.

Aluminium toxicity on DNA/nuclear damage

Aluminium is known to have a genotoxic profile and its prolonged exposure would lead to DNA alteration in higher plants at very low concentration (0.2–1.0 mM). Aluminium restricts the replication of DNA by escalating the rigidity on double helix (Gupta *et al.,* 2013). Grabski and Schindler (1995) reported that Al shows greater susceptibility towards nucleoside triphosphates along with a coalition constant which is about 107 times as higher than Mg^{2+}. And therefore, Al prefers prior binding with DNA as compared to histone and non-histone proteins. In addition, Al restricts the movement of chromosome on mitotic spindle by inhibits tubulin polymerization which instigate delay in microtubular disassembly during mitosis.

Aluminium toxicity on mitochondria

Aluminium toxicity affected severely the mitochondrial respiratory functions and altered the redox status in-vitro and also the internal structure, which caused finally cell death. The Mg^{2+} are essential for normal functioning of mitochondria as their deficiency often results in mitochondrial disintegration, reactive oxygen species (ROS) production, and photo-oxidative damage in many plant species (Cakmak and Kirkby, 2008). It was presumed that Al toxicity causing Mg deficiency inside the mitochondria by substituting Mg^{2+} for Al^{3+} in Mg^{2+} dependent enzymes. It was well know that mitochondrial Mg porters could be the target site for Al^{3+} to cause toxicity (Rezabal *et al.,* 2006).

Aluminium toxicity on oxidative stress

Plant exposure to Al stress elicits the production of ROS in mitochondria, chloroplast and peroxisome which may instigate oxidative damage to cellular components if antioxidant machinery is overwhelmed (Sharma and Dubey, 2007). Aluminium itself is not a transition element, but exposure to Al could affect production of ROS in plants because Al induces the expression of several genes encoding antioxidative enzymes such as glutathione S-transferase, peroxidase and superoxide dismutase (SOD) (Gupta *et al.,* 2013). Moreover, Al^{3+} forms electrostatic bond with oxygen donor ligands such as carboxylate and pectin which further enhance ROS production.

Aluminium toxicity on signal transduction pathways

Aluminium influences signal transduction pathways by disrupting the intracellular Ca^{2+} and pH homeostasis. Aluminium can also interact with and inhibit the enzyme phospholipase C of the phosphoinositide pathway associated with Ca^{2+} signaling. Most probable sites for Al interactions are guanine nucleotide binding proteins (G proteins) and a phosphatidylinositol-4, 5-diphosphate (PIP2) specific phospholypase C (He *et al.,* 2015) and cause physiological and morphological alterations in plants. Despite this, it has been proved that Al plays a significant role in the regulation of protein phosphorylation (Matsumoto, 2000) which regulates signal transduction pathway for mediating extracellular stimuli into cells.

Aluminium toxicity on nutrient imbalance

Many physiological and morphological disorders imposed by Al are reviewed in this article; however one of the most obvious symptoms of Al toxicity is nutrient imbalance. Under Al stress, the uptake of many cations including Ca^{2+} (69%), $Mg^{2+,}$ K^+ (13%) and NH_4^+ (40%) is inhibited while the influx of the anions of NO_3^- (44%) and P (17%) get enhanced. Aluminium interfered with the binding of the cations in the cell wall but strongly enhanced the phosphate binding (Nichol *et al.,* 1993). It is also observed that Al toxicity is closely related to nitrogen metabolism and nitrate-reductase activity increases in presence of Al. In maize, Al showed a negative effect on the uptake of micro (Mn and Zn) and

macronutrients (K, Ca and Mg). In wheat Al content decreased K and Mg contents in roots whereas, Ca, Al and Si contents increased (Silva *et al.*, 2010). It was reported that NO_3^- uptake by soybean was decreased when Al concentration in solution increased from 10 to 50 µM (Rufty *et al.*, 1995). Excess Al induces Fe deficiency symptoms in rice, sorghum and wheat.

Conclusions

Aluminium toxicity is an important growth limiting factor for plants in acid soils which is comprised in a large area of fertile land, particularly at pH <5.5. The morphological and physiological symptoms of Al toxicity in plants are often clearly recognizable. Aluminium interferes with DNA replication, cell division, signal transduction pathways, water and nutrients uptake, root and leaves development, mitochondrial respiration etc. Therefore, intensive research and experiments is required in Al toxic soils for finding better comprehensive responses of plants towards Al exposure and the mechanism of Al resistance in agricultural crops.

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