**Aluminium toxicity: mechanism and effects on plant growth**

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**Abstract**

Aluminum (Al) is the third most abundant element in our earth crust and becomes major growth limiting factors to crop plant under acidic conditions when soil pH drops below pH 5.5. Under acidic conditions Al+3 ions are dissolved from clay minerals and become phytotoxic. Previous research and reports have revealed several Al toxicity to crop plants including decrease in crop production and inhibition of root growth. Here in the current review, we discuss the possible mechanism behind Al-toxicity in plants. Additionally, we also discussed phyto-toxicity of Aluminum on plant growth in the current review.

**Keywords: Aluminum, Phyto-toxic, Plant growth, Tolerant cultivar**

**Introduction**

Soil pH is one of the important factors affecting agricultural production (Kochian et al., 2004). Acidic soils (pH < 5.5) are limiting agricultural production globally. These soils constitute around 30% of the total land area and approximately 50% of the arable lands globally (Von and Mutert, 1995). Even though acidic soils present multiple stresses to plants including mineral toxicity (Al, Mn) and nutrient deficiency (P, Ca, Mg), Aluminium (Al) toxicity is one of the major growth-limiting factors for crop production on 67% of total acidic soil (Eswaran et al., 1997). Although Al is the third most abundant elements in the earth’s crust comprising 8% on elemental basis and most of it occurs in mineral forms which are harmless to plants however under acidic conditions Al+3 ions are dissolved from clay minerals and becomes phytotoxic impeding root growth and function (May and Nordstrom, 1991). Since most of the third world countries are located in acidic soil regions thus Al toxicity is adversely affecting the crop production in those parts of world where food security is critical.

To develop better and improved Al tolerant plant understanding the mechanism behind Al toxicity is also needed. Numerous literatures proposed mechanism behind Al toxicity like reductions and irregularity in cell division, alterations in root cell patterns (Doncheva et al., 2005), changes in cell shape and vacuolization (Vazquez et al., 1999; Ciamporova, 2000), cell wall thickening and callose deposition (Jones et al., 2006). Al+3 can block the function of ion channels involved in calcium (Ca+2) and potassium (K+1) influx and hence directly inhibit the nutrient uptake (Gassmann and Schroeder, 1994). Furthermore, the interference of Al+3 with signal transduction pathway involving 1, 4, 5-trisphosphate (Jones and Kochian, 1995) as well as actin and tubulin stability is also reported in the previous studies (Sivaguru et al., 2003).

Here in the present review key points related to our understanding of mechanism of Al toxicity has been addressed. Brief discussion on the effect of Aluminum toxicity on plant growth has been illustrated in this review.

1. **THE MECHANISM OF AL TOXICITY IN PLANTS**

Understanding the mechanism of Al toxicity is necessary for elaborating the Al resistance and tolerance mechanism. Numerous researchers have proposed mechanism of Al toxicity which has been summarized in many reviews (Kochian, 1995).

1. **Al+3 under acidic condition**

Under acidic condition Al+3 is solubilized in to the soil and becomes highly reactive and phyto-toxic and target mainly to the cell wall, plasma membrane surface, cytoskeleton and the nucleus (Kochian *et al.,* 2004). Al+3 has the affinity to bind pectin in cell walls and causes alterations in the synthesis or deposition of polysaccharides, hardened the cell walls resulting impairment of normal cell wall growth and inhibition of cell elongation (Gunsé *et al.,* 2000). The physicochemical properties of Al+3 allow it to bind with more strong affinity to negatively charged plasma membrane. Furthermore its interaction with plasma membrane could lead to depolarization of the trans-membrane potential (Kinraide and Parker., 1990) or reduction of H+/ATPase (Kinraide, 1991). These changes induced in plasma membrane can alter the uptake of several cations (e.g., Ca+2, Mg+2, K+, NH+4) (Samac and Tesfaye, 2003). Ultimately these changes are relevant to direct Al+3 interactions with plasma membrane ion channels (Pineros and Kochian, 2001) and changes in membrane potential. Al can block the function of ion channels in the root cell plasma membrane (Gassmann and Schroeder, 1994, Huang et al., 1994), thus Al might cross the plasma membrane using certain mechanism either via endocytosis or through ion transport proteins. Direct inhibition of nutrient uptake also occurs due to blockage of the function of ion channels involved in Ca+2 and K+1 influx (Gassmann and Schroeder 1994). Impairment of cytoplasmic Ca+2 homeostasis is also one of the primary target of Al+3 toxicity (Pineros and Tester, 1993) and may be cause inhibition of the cell division or root elongation by causing disruptions of Ca+2 dependent biochemical and physiological processes (Pineros and Tester 1993). Membrane depolarization and cytosolic Ca+2 also gets increased in response to Al+3 toxicity (Panda et al., 2009). May be this impairment in Ca+2 homeostasis is involved in the disorganization of the cytoskeleton and hence inhibiting division and root elongation (Ma et al., 2002). This increase in Ca+2 may be responsible for callose synthesis (Horst et al., 2010). Numerous reports have suggested Al inhibiting the root K+ uptake (Miyasaka et al., 1989).

1. **Mechanism of Al toxicity in apoplasmic or symplasmic sites in roots**

For better understanding the mechanistic basis for Al+3 toxicity in roots, knowledge about apoplasmic or symplasmic sites where injury is arising is also important. Most of the Al+3 in root is in the apoplast and only small fraction of the Al+3 rapidly enters the symplasm and interacts with symplastic targets (Silva et al., 2010,). Most of the Al+3 are located in the apoplast inside the root and probably the primary binding site of Al+3 is the pectin matrix (Chang et al., 1999). Studies also hypothesized that may be this binding is positively correlated to callose formation and hence to Al+3 sensitivity (Horst et al., 1999). Callose synthesis triggered after Al injury on plants leads to cellular damage by inhibiting intercellular transport through plasmodesmatal connections (Horst et al., 2010). Callose deposition is also used as reliable parameter for screening Al resistance genotypes (Tahara et al., 2005). Recent research has implicated that cellular mechanism of Al toxicity could involve interaction between Al and components of the phospho-inositide signal transduction pathway. This interference of Al+3 with signal transduction pathways and signaling also has some role in Al toxicity (Zhang and Rengel et al., 2014).

1. **Cell death mechanism induced by Al+**

Yamamoto et al. (2019) revealed three types of cell death mechanism induced by Al+3, first one is enhancement of iron mediated lipid per-oxidation leading to a loss of plasma membrane integrity (plasma membrane pathway), second one is dysfunction of mitochondria accompanied by ROS production and thirdly up-regulation of NtVPE1 encoding a vacuolar processing enzyme (VPE) leading to vacuolar collapse and the loss of plasma membrane integrity (vacuole pathway). Plants also vary in their response to Al+3 toxicity. *Threshold for toxicity* response has been observed in some plants root growth that means it remains unaffected at low concentrations of Al but declines once a threshold is reached (Barcelo and Poschenrieder, 2002). In other species hormesis-type response has been observed which means at low concentrations of Al root growth is stimulated and then become toxic at higher concentrations. *Threshold for tolerance* model which is an acclimation response also occurs in some species which shows growth inhibition at low concentrations of Al but little or no effect at higher concentrations.

1. **EFFECT OF ALUMINIUM TOXICITY ON PLANT GROWTH**

Severe damage to plants occurs as a result of Al toxicity.

1. **Morphological disruption**

Morphologically it causes several symptoms on different plant parts including stunting, curling of young leaves, and death of leaf tips, chlorosis and inhibition of root growth (Rout et al., 2001). Among site of injury the root apex is the most sensitive site of Al+3 toxicity. The first and most significant morphological symptom is inhibition and reduction of root growth (Foy, 1984). Root growth inhibition is also referred as a marker to screen sensitivity of plants to Al+3. Al+3 begin to inhibit root rapidly within minutes or hours. Numerous literatures providing evidences that reduction in cell division is the major cause behind Al+3 induced root growth inhibition (Bhalerao et al 2013). These studies confirming that Al+3 inhibition of root growth is followed or preceded with reduction in cell division. Since Al+3  induced root growth inhibition occurs very rapidly and within minutes certain physiological processes get altered led to hypothesis that Al+3 toxicity occurs via ion transporters or signal-transduction events initiated at the plasma membrane surface or root cap (Cumming et al.,2017). Further longer exposure results in thickened roots, impaired root cap and injury in the epidermal and cortical tissues near the apices. The root system becomes more and more shortened and hence limits the water and nutrient acquisition resulting poorly developed root system, nutrient deficiencies and finally reduced grain yields (Foy 1988). Decrease in K, Mg, Ca and P contents and uptake in rice and maize has been observed (Silva., 2012). Similarly in tomato cultivars, Al exposure decreases the content of Ca, K, Mg, Fe and Zn in roots, stems and leaves (Tian-Rong et al., 2007). Nutrient deficiency again results several morphological alterations. Ca deficiency in the presence of Al can be observed as curling or rolling of young leaves and collapse of growing points or petioles (Foy., 1988). Thus Al induced stress in roots also causes inhibition of leaf development (Haug et al., 1994).

1. **Physiological & Biochemical disruption**

Physiologically it causes inhibition of DNA synthesis, blockage of cell division, disjunction of cell walls, disruption of plasma membrane integrity, inhibition of signal transduction pathways and changes in cytoskeleton structure (Grauer and Horst, 1992). Functional disorders induced under Al+3 has been studied in maize (Horst et al. (1997), wheat (Silva et al. (2010), Norway spruce seedlings (Nagy et al., 2004), barley (Gruber et al 2010). In white clover, under Al+3 the number of root hairs get decreased and with further increase in Al+3 concentration root hairs get disappeared and root growth get stunted (Care 1995). Al+3 induce oxidative stress in root cells by prompting the production of ROS (Yamamoto et al. 2001). Activities of anti-oxidative enzymes such as peroxidase, superoxide and dismutase get elevated in the presence of Al+3. Hydrolytic enzymes like phosphatase, glucosidase and esterase were strongly inhibited at high Al concentration (Jones et al., 2006). The activities of different enzymes during seed imbibition and early growth of barley seedlings were also affected by Al. Literatures supported that Al treatment altered lipid composition on cell membranes (Zhang et al. 2014). Stiffening of bio-membrane occurs as a result of Al binding promoting the radical chain reactions by iron ions and increases the peroxidation of lipids (Matsumoto, H. and Motoda, H., 2012). This enhancement in lipid peroxidation has been reported for some species including barley (Guo et al., 2004), sorghum (Peixoto et al., 1999), triticale (Liu et al 2008), rice (Kuo and Kao, 2003), greengram (Panda et al., 2003), wheat (Hossain et al 2005). In conclusion, Al+3 disrupt many cellular functions in plants (**Fig1**).

1. **CONCLUSION AND FUTURE WORK**

The toxic effect of aluminum ions has been studied and reported by several researchers. This element becomes major limiting factors for plant growth under acidic condition and causes several toxicity in plants. Several morphological and physiological symptoms are observed in plant under Aluminim toxicity. Thus, Development of cultivars tolerant to Aluminium toxicity is the need of hour. Understanding the mechanism behind Aluminium toxicity in plants is necessary to go for development of Aluminium ions tolerant cultivars. The current advanced genomic technology will boost in best possible way to deal with this abiotic stress.



**Figure1: Morphological, physiological and biochemical effects of aluminum under acidic condition**

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