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Review Article

TOXIC EFFECTS OF ALUMINIUM IN PLANTS

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ABSTRACT
Aluminium occurs in the form of oxides and silicates and is the most abundant metal in the earth's crust. Although it is abundant in earth, its impact on plants and other organisms was not a cause of major concern. However, over the course of time, it amount is steadily increasing, primarily in acidic soils. At pH 5 or below that, toxic effects of Al are more aggravated. Since over 50% of the worlds arable soils are acidic, Al toxicity is becoming an important limiting factor worldwide to crop productivity. In plants the major site of toxic effect of Al is the apical part of the root. It has been reported that the ultrastructure of root cap cells is affected by Al toxicity. However, the toxic effects are seen in both apoplast and the symplast of many plant species. Even short exposure to Al are reported to cause reduction in the root elongation, thereby limiting the acquisition of water and nutrients from the soil.

Keywords: Aluminium toxicity, Acidic soil, Apical root, Symplasm

INTRODUCTION
In the periodic table, Aluminium (Al) is in group IIIa and has a valency of +3. It shows high reactivity with oxygen at normal temperature. Along with this characteristic, it also reacts strongly with acids and bases to form salts and releases hydrogen. It commonly occurs in the form of oxides and silicates, and is the most abundant metal in the earth's crust. This metal is the most abundant in the Earth's crust, naturally absorbed from the soil by plants and foodstuffs. In the form of salts, it has properties that make it a versatile and useful additive. Al sulphate is added to water to improve clarity, all foods that need raising agents or additives, such as cakes and biscuits, contain Al. Children's sweets contain Al-enhanced food colouring. It is in tea, cocoa and malt drinks, in some wines and fizzy drinks and in most processed foods. It is also part of cosmetics, sunscreens and antiperspirants, and used also as a buffering agent in medications like aspirin and antacids. It is even used in vaccines. Over the course of time however, the amount of Al is steadily increasing. Aluminum toxicity is the primary factor that limits crop production on strongly acidic soils. At soil pH values at or below 5, toxic forms of Al are solubilized into the soil solution, and inhibit root growth and function, and thus reduce the crop yields. It has been estimated that over 50% of the world's potentially arable lands are acidic (Bot *et al*., 2000); hence, Al toxicity is a very important worldwide limitation to crop production. Furthermore, since up to 60% of the acid soils in the world occur in developing countries, where food production is critical. Toxicity effects of Al on crops are becoming a major cause of concern for the farmers.

Breeding of crops with increased Al resistance could be considered a way out, however, the underlying molecular, genetic and physiological bases are still not well understood. Because of the agronomic importance of this problem, understanding the mechanism of Al toxicity in plants is very important. The present review is a brief survey of studies related to the toxic effects of Al on the plants.

Bioavailability of Al
According to Exley and Birchall (1992), the bioavailability of a substance is defined as a measure of its potential to interact with biological systems and also the capacity to cause a response. Due to its adsorption to mineral surfaces, bioavailability of Al in soil and water remains very low. At pH near neutral, it forms associations with organic matter and also due to the insolubility of hydroxide complexes of Al, its bioavailability is considered to be very low. However, due to the acidification of soil and water, the presence of Al is being recognized as a major pollution problem. With acid rains also Al is released from its natural reservoirs (Myrold and Nason, 1992). When the pH of the solution is near or lower than

5.0, most of the Al exists as an octahedral hexahydrate $AI(H_20)_6^{3+}$ (referred to as Al^{3+} or free Al), and at neutral pH it precipitates as $Al(OH)_3$.

According to Martin (1986), the proportion of different oxidation forms of Al is function of the environmental pH, and even small variations in the acidity of the environment could cause great changes in the concentration of these species. Bruce *et al.,* (1988), a soil with pH ~5.8 has 6.3µM Al. And, when the pH is further lowered to 4.77, the Al level rises to 700 µM. On the contrary, increase in pH to 6.22 reduces the concentration to 5µM. The bioavailability of Al therefore is determined not only by the natural conditions of the soil and pH changes, but also by human activities which can modify the environment through inadequate agricultural procedures or by disposal of wastes in the environment.

Toxicity Effects of Al in Plants

Aluminium is very toxic to living organisms. One of the reason why this is so is that most of the organisms live in a pH range around 7.0, and therefore, have not developed the mechanisms to tolerate high levels of Al. As a consequence of this, when the concentration of Al rises in acidified waters, various ailments in human beings, animals and plants are observed.

Most easily recognized symptoms of Al toxicity in plants is the inhibition of root growth, which is considered to be the most widely accepted measure of Al stress in plants. In nutrient solutions even micromolar concentration of Al begins to inhibit root growth within a short time (~60min). As stated earlier, the forms of Al change rapidly with change in the pH of the soil or water, the form of Al which exerts the toxic effect is difficult to identify. A1 rapidly hydrolyzes in solution, as a result the trivalent A1 species $A1^{3+}$, dominate in acidic conditions (pH < 5), whereas the $A1(OH)^{2+}$ and $Al(OH)^{2+}$ species are formed as the pH increases. Since, many trivalent cations are toxic to plants and, because A1 toxicity is largely restricted to acid conditions, it is generally believed that $A1^{3+}$ is the major phytotoxic species of Al, however, it could not be concluded with certainty. Kinraide (1991) reviewed that nearly all of the monomeric A1 species have been considered toxic in one study or the other.

Site of Al Toxicity in Plants

The apical part of the root which includes root cap, meristem and the zone of elongation are reported to accumulate more Al and as a result suffer greater physical damage than the mature tissues in the root. Ryan *et al.,* (1993) reported that only the apical 2-3 mm of the maize root needed to be exposed to Al when it started to inhibit the root growth. Further, their study made an interesting observation that selective application of Al to the elongation zone or the entire root except the root apex did not cause any reduction in growth. In another study, Bennet and Breen (1991) observed a number of changes in the ultrastructure of the cap cells in maize roots when Al treatment was extended for 2 hours. They concluded that in such situations Al could inhibit root growth indirectly through signal response pathway, which involved root cap, hormones and secondary messengers. This hypothesis thus considers the involvement of root cap in signal perception and hormone distribution. But, it was also recorded by Ryan *et al.,* (1993) that the inhibition of root growth in maize was the same in intact and decapped roots. This points to the important role played by the root meristem in Al toxicity in maize.

Where does the Al start to exert its effect is difficult to prove. Since polyvalent ions (such as Al^{3+}) are insoluble in lipid bilayers, the plasma membrane is a barrier to Al entry. Even than it has been observed that some Al crosses the plasma membrane (probably as neutral Al ligand, or by endocytosis, or through membrane bound protein, or due to lesions caused by stress). But an interesting report by Tice *et al.,* (1992) has shown that half of the total Al present in the root apex was located in the symplasm. The absorption of Al has been linked to susceptibility of certain plant species to Al. It has been seen that root apices of Al tolerant wheat (*Triticum aestivum*) accumulated less Al than Al sensitive wheat genotypes. As reported earlier, exposure to Al for short duration (<60min) could inhibit root growth. One important question that needs to be answered is how quickly Al moves into the symplasm and that too in sufficient quantity to cause the effect. This question was partly resolved when Lazof *et al.,* (1994) detected Al in symplasm of soybean (*Glycine max*) roots after exposure to 30min only. This proves that Al could enter before the root growth is inhibited, and further that symplasm is probable site of Al toxicity. What needs to be remembered here is that after entry into the symplasm, the prevailing pH there (6.5to 7.5) and also

the large numbers of potential ligands will probably maintain a very low concentration of Al^{3+}. Therefore, at such low concentration Al could hardly cause significant damage in the symplasm. With this information in the background, it has been suggested that the primary cause of toxicity in the symplasm is the formation of Al-ligand complex. Once this association is formed, Al either inhibits the vital functions earlier performed by that ligand (the ligand could be binding to enzymes, calmodulin, tubulin, ATP, GTP, DNA) or the Al-ligand complex itself could now poison some metabolic process.

Apart from entry into the symplasm and the toxicity effects there, Al has very easy and quick access to apoplasm. This way, interaction with cell wall and membrane will preced transport into the symplasm, and interactions here (in the apoplast) could be possibly harmful. In the apoplast, association of Al with pectic residues and/or proteins in the cell wall could decrease the extensibility of cell wall, displace other ions from critical sites on the cell wall or membranes, bind to the lipid bilayer or membrane bound proteins and inhibit nutrient transport, or could disrupt intracellular metabolism from the apoplast itself by triggering secondary messenger pathways as suggested by several workers (Haug, 1984, Taylor, 1988, Haug *et al.*, 1994).

The importance of apoplast as site of activity of Al is further proved by X-ray microanalysis and secondary ion mass spectroanalysis studies, which indicate that a significant fraction of Al in roots is associated with apoplastic binding sites, predominantly in walls of cells of the root periphery (Vazquez *et al.*, 1999). Since the net negative charge of the cell wall determines its cation exchange capacity (CEC). Consequently it determines the degree to which Al interacts with the cell wall. Tabuchi and Matsumoto (2001) reported that Al interactions lead to the displacement of other cations (e.g., Ca^{2+}) fundamental for cell-wall stability. As an outcome of this, the strong and rapid binding of Al alters cell wall structural and mechanical properties, making it more rigid, leading to a decrease in the mechanical extensibility of the cell wall which is required for normal cell expansion.

Kinraide *et al.*, (1998) reported that $A1^{3+}$ interacts very strongly with the negatively charged plasma-membrane surface. Since Al has a more than 500-fold greater affinity for the choline head of Phosphatildylcholine (a-lipid constituent of the plasma membrane), than other cations such as Ca^{2+} have, $A1^{3+}$ can displace other cations that may form bridges between the phospholipid head groups of the membrane bilayer. The result is the altered phospholipid packing and fluidity of the membrane.

Besides, interaction of Al with the plasma membrane leads to screening and neutralization of the charges at the surface of the plasma membrane. This can alter the activities of ions near the plasma-membrane surface. In conclusion, the interactions of Al at the plasma membrane can modify the structure of the plasma membrane as well as the ionic environment near the surface of the cell; both can lead to disturbances of ion-transport processes, which ultimately perturb cellular homeostasis.

Another measure of Al toxicity is the callose accumulation in the apoplast, which is an early symptom of Al toxicity (Massot *et al.*, 1999). Callose synthesis depends on the presence of Ca^{2+}, hence, it is argued that displacement of Ca^{2+} by Al from the membrane surface increases the pool of Ca^{2+} in the apoplast, which is required to stimulate the synthesis of callose. Sivaguru *et al.*, (2000) reported that under Al stress, callose accumulation aggravates cellular damage by inhibiting intercellular transport through plasmodesmatal connections.

Thus Al has many effects at the apoplast and symplast levels that disturb the normal physiology the cell and functioning of the cell membrane and cell wall. These result in reduced plant growth and development, and ultimately reduced yield of crop plants.

Conclusion

With the decrease in pH of arable soils worldwide, due to indiscriminate use of fertilizers, the problem of Al toxicity in plants is likely to increase, and become a major limiting factor worldwide, more so in the developing countries. Since short term exposure to Al also are causing noticeable changes in the root apical meristem, the understanding of the mechanisms of Al toxicity in different plant species are important for reducing the toxicity symptoms in the plants of agronomic importance. Easiest solution to this problem is reducing the pace of acidifying the soil, which could in part be done reducing the dependence of chemical fertilizers.

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