Recent Advances in Aluminum Phytotoxicity

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1 Introduction

Aluminum (in American English, aluminium in British English) is the third most abundant element following oxygen and silicon, while its oxide is the fourth among the most common compounds in the earth's crust. Aluminum (Al) is also the most abundant metal on the planet. Al is dense in the outer 16 km of earth's cortex constituting about 8.1% by mass. Naturally, Al never occurs in the metallic form because of its chemical activity; it is found in chemical compounds with other elements like bauxite. To remove Al from natural ores, it must first be reduced. Al is considered as an active metal reacting with concentrated acids and alkalis (Sade et al. 2016; Li et al. 2016).

The trivalent Al has three oxidation states. The most common oxidation state of Al is +3 and it reacts rapidly with the oxygen in the moist air to form aluminum oxide (Al_2O_3-alumina). Al_2O_3 is the refractory oxide of Al existing in bauxite. Occasionally, the oxidation state of +2 and +1 exists as aluminum monoxide (AlO) and aluminum hydride (AlH_3), respectively. The Al^{3+} ion can be stabilized by hydration, and the octahedral ion $[Al(H_2O)_6]^{3+}$ occurs both in aqueous solution and in several salts (Roesky and Kumar 2005; Li et al. 2016).

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It has been known that Al is also the most widely used metal in the industrial world after iron (Table 1). The large-scale (28%) use of Al is in the transportation industry. Packaging follows it by 23%. Because Al can be melted and reused, or recycled, it is ideal for foil, beer and soft drink cans, paint tubes, and containers for home products such as aerosol sprays. 14% of Al goes into building and construction such as windows and door frames, screens, roofing, and siding, as well as the construction of mobile homes and structural parts. The remaining 35% of Al is used in electrical wires and appliances due to being an excellent conductor, automobile engines, heating and cooling systems, bridges, vacuum cleaners, kitchen utensils, garden furniture, heavy machinery, and specialized chemical equipment (http://www.chemistryexplained.com/elements/A-C/Aluminum.html).

The widespread presence in earth crust and prevalent use of bioavailable Al may have immense and far-reaching implications for the health of humans and animals.

Table 1 Aluminum complexes used in industries (http://www.chemistryexplained.com/elements/A-C/Aluminum.html)

Al complex	Chemical formula	Area of usage
Aluminum ammonium sulfate	$Al(NH_4)(SO_4)_2$	• Mordant • Water purification and sewage treatment • Paper production • Food additive • Leather tanning
Aluminum borate	$Al_2O_3B_2O_3$	• Production of glass and ceramics
Aluminum borohydride	$Al(BH_4)_3$	• Additive in jet fuels
Aluminum chloride	$AlCl_3$	• Paint manufacture • Antiperspirant • Petroleum refining • Production of synthetic rubber
Aluminum fluorosilicate	$Al_2(SiF_6)_3$	• Production of synthetic gemstones, glass, and ceramics
Aluminum hydroxide	$Al(OH)_3$	• Antacid • Mordant • Water purification • Manufacture of glass and ceramics • Waterproofing of fabrics
Aluminum phosphate	$AlPO_4$	• Manufacture of glass, ceramics, pulp and paper products • Cosmetics • Paints and varnishes • In making dental cement
Aluminum sulfate, or alum	$Al_2(SO_4)_3$	• Manufacture of paper • Mordant • Fire extinguisher system • Water purification and sewage treatment • Food additive • Fireproofing and fire retardant • Leather tanning

In fact, much evidence shows that Al seems to be toxic to all forms of life on earth, and where it also appears in terrestrial biochemistry, it is invariably deleterious (Exley 2009; Shaw and Tomljenovic 2013).

2 Aluminum Toxicity in Plants

Considering the plants, Al is one of the abiotic stress factors. It is definite that anthropogenically released and/or naturally existing Al can solubilize and be absorbed by plants at low pH (acid) soils constituting one of the major plant growth-limiting factors. It has been known that potential farmable lands (approx. 67%) have acid soil worldwide (Abate et al. 2013; Ma et al. 2014). Al exists as a nontoxic complex in neutral or weakly acidic soils; however, when the complex Al is solubilized it turns to phytotoxic forms in acid soils. The most phytotoxic and dominant form is $Al(H_2O)_6^{3+}$ and dissolves to Al^{3+} which can be absorbed by plant roots (Matsumoto 2000; Vardar and Ünal 2007). It has been stated that solubilized Al presents in the range of 10–100 μM in acid soils affecting adversely the plant growth and development within a few minutes (Ciamporová 2002; Vitorello et al. 2005; Abate et al. 2013). Absorbed Al interacts with apoplasmic (cell wall), plasma membrane, and symplasmic (cytosol) targets. Al ions are penetrated from roots primarily and only a small proportion may be taken up through leaves (Kochian et al. 2005; Singh et al. 2017).

At the whole plant level, toxic Al affects adversely their anatomical and physiological structure such as chlorosis, reduction in leaf number, reduced photosynthesis, necrosis, and retardation of root growth. It has been widely known that roots are the first target of Al toxicity which have direct contact with rhizosphere. Al-induced root growth inhibition restrains the uptake of soil water and essential minerals leading to reduction in crop quality (Delhaize et al. 2004; Singh et al. 2017; Vardar et al. 2018). Root apex is the foremost region with regard to Al toxicity. As a first target, root apex plays a critical role in Al penetration and accumulation. This region absorbs more Al than the upper parts of root resulting in morphological alterations of root apices such as swelling, cracking, and appearing to be stubby and stiff (Matsumoto 2000; Vardar et al. 2006). It has also been visible that branching and root hair occurrence reduce significantly (Ciamporová 2002; Vardar et al. 2011). Several researches revealed that Al has detrimental effects reacting with different subcellular regions performed in different species and also varieties.

2.1 Cell Wall

Considering the cell structure, cell wall is the primary target of Al ions. It has been identified that Al binds and accumulates in the apoplasm in the range of 30–90% in root cortex cells (Rengel and Reid 1997; Vardar et al. 2011). Pectin matrix which has

negatively charged carboxylic groups is the first Al-binding site in the cell wall (Chang et al. 1999; Singh et al. 2017). It has also been known that Al reacts with apoplasmic face of plasma membrane. After Al-cell wall interaction, Al translocates to plasma membrane and symplasm (Schmohl and Horst 2000). Researchers suggested that Al accumulation degree is in direct correlation with pectin content and dissociation of carboxylic and hydroxylic groups of the pectin (Godbold and Jentschke 1998; Ahn and Matsumoto 2006). Strong binding and accumulation of Al alter the structural and mechanical properties of cell wall causing reduction in mechanical extensibility causing cracked and unoriented root growth (Kochian et al. 2005).

Al accumulation in cell wall discomposes the stability of other cations such as Ca^{2+} which is responsible for the strength of cell wall. This disturbance causes callose (β-1,3-glucan) synthesis and accumulation between cell wall and plasma membrane being one of the significant markers of Al toxicity (Tabuchi and Matsumoto 2001; Vardar et al. 2011; Ünal et al. 2013). Although callose may collaborate root to cope with Al toxicity by blocking the plasmodesmata, it also blocks the movement of water and minerals causing reduction of nutrient uptake (Singh et al. 2017; Vardar et al. 2018).

Eventually, Al reaction in cell walls causes reduced extensibility, disrupted growth orientation, callose formation, and accordingly restriction of water and mineral nutrient uptake across the plasma membrane (Kochian et al. 2005).

2.2 Plasma Membrane

Plasma membrane is the external barrier of the cell and it regulates the ion traffic. Negatively charged membrane displays strong interaction with $A1^{3+}$ (Kinraide et al. 1998). Plasma membrane-Al reaction alters the structure and function of membrane causing disruption in the cellular homeostasis (Kochian et al. 2005). It has been revealed that $A1^{3+}$ may interact with both phospholipids and proteins leading to lipid peroxidation in plasma membrane. Researchers revealed that the severity of Al toxicity causes to break the plasma membrane integrity (Vitorello et al. 2005; Panda et al. 2009; Singh et al. 2017). Lipid peroxidation also causes highly toxic free radical generation and accumulation (Panda et al. 2009).

Al has greater affinity than other cations such as Ca^{2+} and Mg^{2+} during competing for the choline head of phosphatidylcholine. This situation culminates in Al-induced positively charged bridges between head groups of the phospholipid layer and displacement of other cations (Bhalerao and Prabhu 2013; Singh et al. 2017). The positively charged layer restricts cation motion, but increases anion movement altering membrane electrochemical potential (Nichol et al. 1993). As we have stated above Al-induced cation alteration, principally Ca^{2+} displacement, also triggers callose synthesis (Gupta et al. 2013). Callose also inhibits intercellular transport through plasmodesmatal plugs (Sivaguru et al. 2000). Alterations in cation uptake of essential ions such as Ca^{2+}, K^{+}, Mg^{2+}, and NH_4^{+} also cause nutrient imbalances (Pineros and Kochian 2001; Singh et al. 2017).

2.3 Cell Signaling and Cytoskeleton

Several researchers stated that Al stress affected signal transduction pathway adversely mediated by secondary messengers due to imbalance of Ca^{2+} and pH homeostasis (Jones and Kochian 1997; Ma et al. 2002; Singh et al. 2017). In plasma membrane Al prefers to react with specific lipids which are important signaling molecules such as G proteins (guanine nucleotide-binding proteins) and a phosphatidylinositol-4,5-diphosphate (PIP2)-specific phospholipase C commonly (He et al. 2015). Besides, Al stress decreases inositol-1,4,5-triphosphate (IP3) amount in the plasma membrane (Rengel and Elliott 1992). After Al reaction, signaling pathways are interrupted in the cell.

Cell cytoskeleton including microtubules, microfilaments, and intermediate filaments is also one of the potential targets of Al ions. Al causes disruption in cytoskeletal dynamics which has a critical importance during cell-wall biosynthesis, cell growth, and cell division. It has been revealed that Al-induced disruption of microtubule and actin filament results in lateral cell swelling (Frantzios et al. 2001; Sivaguru et al. 2003). It has been suggested that Al disruption in cytoskeleton occurs either through direct interaction with cytoskeletal elements or through alteration in signaling pathway (Sivaguru et al. 1999). Protein phosphorylation-dephosphorylation and mitogen-activated protein kinase (MAPK) cascade which take charge during signal transduction are also reorganized by Al ions (Matsumoto 2000; Osawa and Matsumoto 2001; Singh et al. 2017). This interaction impairs the signal transduction pathway causing chaos in the cell.

2.4 Genotoxicity

Several researches reveal that Al has genotoxic impact and long-term Al exposure causes adverse effects on DNA composition and replication due to more rigid double-helix and chromatin structure (Vitorello et al. 2005; Panda et al. 2009; Gupta et al. 2013). It has been observed that Al ions decrease cell viability and mitotic index and increases chromosomal aberrations which are associated with Al-induced disturbance in tubulin polymerization-depolymerization. Tubulin disturbance limits the movement of chromosome on mitotic spindle causing chromosome laggards, bridges, micronuclei, and c-mitosis under Al stress (Frantzios et al. 2000; Vardar et al. 2011). It can also be considered that Al exposure may decrease the frequency of S-phase cells inducing delay in M phase (mitotic division) (Jaskowiak et al. 2018). Grabski and Schindler (1995) showed that Al has greater affinity to nucleoside triphosphates much more than Mg^{2+}. Hence, Al prefers to interact with DNA than histone proteins at first. Besides, several researches revealed that Al exposure may cause double-strand DNA breaks even at 15 min (Vardar et al. 2015, 2016). Recent studies also revealed that Al ions cause DNA methylation and polymorphism of LTR retrotransposons (Guo et al. 2018; Taspinar et al. 2018).

2.5 Oxidative Stress and Programmed Cell Death

Al toxicity stimulates generation of reactive oxygen species (ROS) leading to oxidative stress in plants. Lower concentrations of ROS have a role as signaling molecules; however, higher concentrations regress the balance between antioxidant machinery and ROS detoxification. Overproduction of ROS ($^{\cdot}O_2^-$, $^{\cdot}OH$, HO^-, H_2O_2) is generated in mitochondria, chloroplast, and peroxisomes causing imbalance of antioxidant enzyme, lipid peroxidation, protein denaturation, carbohydrate oxidation, pigment breakdown, and DNA damage (Sharma and Dubey 2007; Gupta et al. 2013; Vardar et al. 2018).

Phytotoxic levels of ROS also trigger programmed cell death (PCD) in plants. It has been suggested that ROS weakens the binding strength of cytochrome c (cyt c) through oxidation of cardiolipins in the inner mitochondrial membrane and reduces mitochondrial membrane potential ($\Delta\Psi m$) inducing cytochrome c release to the cytoplasm (Williams et al. 2014). Besides cytochrome c release amplifies more ROS generation and triggers vacuolar processing enzyme (VPE) activity. Although there are some studies concerning Al toxicity and PCD (Table 2), more detailed studies are needed to clarify the Al-induced PCD mechanism.

3 Al Tolerance Mechanisms

Al has the ability to make stable complexes with oxygen donor ligands; thus Al chelating with root exudates plays a critical role in the prevention of phytotoxic Al uptake by roots (Barceló and Poschenrieder 2002). It has been evidenced that Al chelating mechanism is performed by mucilage formation, organic anion efflux, phosphate secretion, and secondary metabolite production from tolerant root apices (Miyasaka and Hawes 2001; Ma et al. 2001; Ofei-Manu et al. 2001; Vardar and Ünal 2007; Singh et al. 2017). Whereas tolerant plants may use different types of Al exclusion strategies, organic anion efflux plays a central role in the exclusion of Al. Several genetic and molecular approaches concerning organic acid release were reported in different plant species (Ma et al. 2001). Al chelation by organic acids decreases or prevents its uptake through apoplasm and symplasm. Type of organic acids secreted by roots varies depending on Al-tolerant plant species. It has been reported that malate, citrate, and oxalate are the most commonly encountered organic secretions (Magalhães et al. 2007; Ryan et al. 2009). Researches revealed that organic acid exudation is activated by Al exposure rapidly suggesting a transporter located in the plasma membrane of tolerant roots (Kochian et al. 2005).

Whereas organic acid exclusion from roots and Al chelation in the rhizosphere appear to be the most common, several species tolerate Al toxicity by internal or symplastic detoxification after Al uptake into the root or shoot cells. This situation was first attained in Al-accumulating plant root, shoot, and leaf such as tea (*Camelia sinensis*), buckwheat (*Fagopyrum esculentum*), and Hydrangea (*Hydrangea macrophylla*).

Table 2 Recent studies concerning Al-induced PCD

Plant material	Al concentration	PCD signs	References
Hordeum vulgare	0.1–50 mM	DNA fragmentation (0.1–1 mM) Necrotic DNA smear (10–50 mM)	Pan et al. (2001)
Allium cepa	1–200 μM	Breaks in DNA	Achary et al. (2008)
A. cepa	200–800 μM	DNA damage	Achary and Panda (2009)
Arabidopsis thaliana	0.5 mM	Caspase-3-like activity Loss of MTP Swelling of mitochondria ROS generation	Li and Xing (2011)
H. vulgare	2.5, 5, 10 mM	DNA damage	Achary et al. (2012)
Nicotiana tabacum	0–150 μM	VPE activity Alterations in vacuole	Kariya et al. (2013)
Arachis hypogaea	100 μM	ROS burst Upregulation of *Rboh* and *COX* expression MPTP opening Decreased ΔΨm Cyt c release Caspase-3-like protease activity DNA fragmentation	Huang et al. (2014)
A. hypogaea	20, 100, 400 μM	ROS production MDA increase Reduction of mitochondrial Ca concentration Opening of MPTP Collapse of ΔΨm Cyt c release	Zhan et al. (2014)
H. vulgare *Secale cereale* *Triticosecale wittmack* *Avena sativa*	100 μM	Caspase-3, -8, and -9-like activities	Aytürk and Vardar (2015)
Triticum aestivum *S. cereale* *T. wittmack*	100 μM	DNA damage	Vardar et al. (2015)
H. vulgare *S. cereale* *T. wittmack* *A. sativa*	100 μM	DNA fragmentation	Vardar et al. (2016)
A. hypogaea	100 μM $AlCl_3$	Caspase-1, -2, -3, -4, -5, -6, -8, and -9 activities	Yao et al. (2016)
Nicotiana tabacum	50 μM	Increase in gene expression of VPE1a and VPE1b	Kariya et al. (2018)
H. vulgare	5–60 μM	DNA fragmentation	Jaskowiak et al. (2018)

MTP mitochondrial transmembrane potential, *MPTP* mitochondrial permeability transition pore

Internal detoxification consists of Al chelation with organic ligands in cytosol and their transfer to the vacuole for deposition (Kochian et al. 2004; Delhaize et al. 2012). Although most of the plants prefer only organic acid exudation or internal detoxification, some species such as *Pinus taeda* make use of both of the mechanisms to protect itself from Al toxicity (Nguyen et al. 2003; Nowak and Friend 2005).

4 Aluminum Tolerance Genes in Plants

Many plant species vary considerably in their ability to tolerate the toxic Al concentrations via efflux of organic anions such as malate, citrate, and oxalate from roots. Al tolerance has a strong correlation with genotype-dependent efflux capacity of organic anion and exclusion of Al once it enters cytosol (Kochian et al. 2004; Hiradate et al. 2007; Delhaize et al. 2012). Sasaki et al. (2004) isolated a gene controlling the Al-dependent efflux of malate from *Triticum aestivum* (wheat) named *TaALMT1* (*Triticum aestivum* aluminum-activated malate transporter 1). *TaALMT1* (formerly named *ALMT1*) encodes a hydrophobic protein (anion channel) localizing in the plasma membrane of root cells (Yamaguchi et al. 2005; Ligaba et al. 2006). ALMT protein family has 5–7 membrane-spanning regions in the N-terminal half of the protein and a long C-terminal tail (Delhaize et al. 2004, 2012). Researchers revealed that *TaALMT1* expression in Al-tolerant genotypes of wheat is 5- to 10-fold higher than in Al-sensitive genotypes (Sasaki et al. 2004; Raman et al. 2005). Subsequent analyses revealed that specific variations in diverse bread wheat genotypes could be classified into seven patterns, type I to type VII (Sasaki et al. 2006; Garcia-Oliveira et al. 2014). After the discovery of *ALMT1* in wheat, *Arabidopsis ALMT1* members were identified as *AtALMT1*, and similarly their homologs characterized in rape (*BnALMT1* and *BnALMT2*), soybean (*GmALMT1*), and rye (*ScALMT1*). All of them share similar functional characteristics that induce malate exudation in Al tolerance (Hoekenga et al. 2006; Ligaba et al. 2006).

Further studies revealed that another gene responsible for citrate exudation in response to Al toxicity exists in barley (*HvAACT1-Hordeum vulgare* aluminum-activated citrate transporter 1) which belongs to *MATE* (multidrug and toxic compound extrusion) gene family (Furukawa et al. 2007). Besides, *SbMATE* gene was also identified in *Sorghum bicolor* responsible for citrate transporter in response to Al toxicity (Magalhães et al. 2007).

It has been known that tolerant genotypes within species have significantly much more organic acid expression than sensitive genotypes. The extra expression is due to a series of *cis* mutations in the promoter of *TaALMT1* in wheat (Sasaki et al. 2006; Ryan et al. 2010). Raman et al. (2008) revealed that the promoter region is more polymorphic than coding region in *TaALMT1* and several alleles have accurate tandem repeats (Ryan et al. 2010). Besides, several examples indicated that transposable elements are able to alter the level and localization of gene expression during enhancing Al tolerance (Morgante et al. 2007; Delhaize et al. 2012).

In wheat the major Al tolerance locus was identified on chromosome 4DL (Luo and Dvořák 1996; Raman et al. 2008) and subsequently on chromosome 4BL responsible for phenotypic variation in citrate efflux (Ryan et al. 2009) suggesting that citrate is the secondary organic acid after malate in Al tolerance. Following molecular studies in different cultivars of wheat revealed that multiple genetic loci on the chromosome arms of 2DL, 3DL, 4BL, 4DL, 5AS, 6AL, 7AS, and 7D are very critical in Al tolerance mechanism (Aniol and Gustafson 1984; Aniol 1990; Papernik et al. 2001). However, it is still not clear that whether all of these loci are included in Al tolerance. Recently Al tolerance-related loci have been identified on different chromosomes in different plant species (Ryan et al. 2009; Boff et al. 2019).

Early studies suggested that Al resistance in wheat is driven by a single major genetic locus with different alleles inducing different degrees of Al tolerance (Campbell and Lafever 1981). Monogenic inheritance with multiple alleles was also identified in barley, maize, sorghum, pea, chickpea, and oat (Singh and Choudhary 2010; Singh and Raje 2011; Castilhos et al. 2011; Delhaize et al. 2012). However, subsequent microarray studies revealed the complexity of the genetic control of Al tolerance. Besides, most of the identified genes probably express response to Al stress rather than Al tolerance (Goodwin and Sutter 2009; Delhaize et al. 2012). According to the several researches in wheat root tips different genes expressed high amounts correlating with Al tolerance such as *ALMT1*, ent-kaurenoic, β-glucosidase, lectin, histidine kinase, pyruvate dehydrogenase, alternative oxidase, galactonolactone oxidase, and phosphoenolpyruvate carboxylate. These results suggest that Al tolerance can be co-regulated by multiple genes with diverse functions in plants in addition to *ALMT1* (Guo et al. 2007; Houde and Oury 2008).

In conclusion, Al toxicity is a widespread problem in industrial regions and acidic soils limiting crop productivity in the world. It has been known that the severity of Al toxicity is due to plant genotype, cell/tissue type, types of chelators, concentrations of other cations, and pH (Kinraide and Parker 1987). Since Al toxicity and tolerance mechanism and also Al-detoxifying mechanisms need to be clarified with more detailed studies, in this chapter, we reviewed recent information concerning physiological and molecular effects of Al toxicity and Al tolerance mechanism. The intensive researches on gene-based mechanisms of Al toxicity and tolerance may help to develop Al-tolerant varieties or transgenic to enhance the crop quality under Al toxicity.

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