Differential response of plants to aluminum. A review

Respuesta diferencial de las plantas a aluminio. Una revisión

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ABSTRACT

Aluminum toxicity is a major limiting factor to the growth and development of plants in acidic soils worldwide, occurring in 40% of arable soils. The root seems to be the object of aluminum toxicity, particularly the apex, producing a rapid inhibition of cell division and elongation of the root. Fortunately, plants differ in their ability to tolerate aluminum and grow in acidic soils. Tolerance mechanisms have commonly been defined in genetic and physiological terms, however, tolerance mechanisms are not the same in all species, moreover, in certain species, mechanisms can operate simultaneously producing tolerance through their combined effects; the genetic control of tolerance can be very complex and involve many genes. The toxic action of aluminum, according to several studies, can be reduced by internal or external Al chelation with different organic compounds such as organic acids, proteins and polysaccharides, although this type of tolerance mechanism is very controversial and highly debated.

Key words: acidity, aluminum toxicity, aluminum tolerance.

RESUMEN

La toxicidad por aluminio es uno de los mayores limitantes para el crecimiento y desarrollo de las plantas en muchos suelos ácidos del mundo. El 40% de los suelos arables tiene este problema. La raíz parece ser el órgano de la planta objeto de la toxicidad de aluminio, particularmente el ápice, produciendo una rápida inhibición de la división celular y elongación de la raíz. Afortunadamente, las plantas difieren en su habilidad para tolerar aluminio y crecer en suelos ácidos. Los mecanismos de tolerancia comúnmente se han definido en genéticos y fisiológicos; sin embargo, el mecanismo de tolerancia no es igual en todas las especies, más aun, en ciertas especies pueden estar operando de manera simultánea una combinación de mecanismos para producir la tolerancia; el control genético de la tolerancia puede ser muy complejo e involucrar muchos genes. La acción tóxica del aluminio según varias investigaciones puede reducirse mediante procesos de quelatación interna o externa del Al con diferentes compuestos orgánicos como los ácidos orgánicos, proteínas y polisacáridos, aunque son muy controversiales y discutidos este tipo de mecanismos de tolerancia a aluminio.

Palabras clave: acidez, toxicidad por aluminio, tolerancia al aluminio.

Introduction

Aluminum toxicity is a major limiting factor for crop production in acid soils, present in more than 40% of the arable land in the world, particularly in the tropics and subtropics (Kochian, 1995); about 64% of the tropics in South America, 32% of the Asian tropics and 10% of Central America, the Caribbean and Mexico, are considered to have acidic soils (Salazar *et al.,* 2003). In South America, approximately 250 million hectares in the Neotropic savannas have Al toxicity problems, including the Colombian savannas of Orinoquia (Vera, 2000).

The Colombian Orinoquia region encompasses 23% of the country's total area, equivalent to 26 million hectares. In this region, two subregions have been defined: the piedmont plains and Altillanura/well-drained, of high interest for the development of sustainable agricultural. However, Altillanura/well-drained despite having conducive agroclimatic conditions, precipitation between 1,800 and 2,300 mm, average temperature of 26°C, easily managed topography, low relative cost of land and proximity to large cities as advantages over other regions of the country, its excess of aluminum in the soil presents one of its major limiting factors (Valencia, 2002a; Valencia and Ligarreto, 2010a), however, these soils are of great importance for current and future economic development because in recent decades these soils have been studied, which has contributed to the understanding of their genesis and how to better manage and optimize their productivity (Mejía, 1996; Rincón and Ligarreto, 2008). According to Inostroza *et al.* (2008), one option to reduce the toxic effect of Al is to neutralize the acidity with the use of liming; this practice is still very laborious, expensive and ineffective.

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Another alternative is to search for genetic variability for tolerance to Al in the genome of cultivated species and / or their wild relatives.

In general, the strategy for exploiting the savannas is based on the search for new technological patterns which utilize the use of adapted germplasm and integrated and efficient management of production resources, where the generation of new technologies allows for the improvement of the availability and productivity of the ecological capital of these regions.

As for adapted germplasm, plant varieties and species differ in their tolerance to aluminum and the variations are an important source for crop production in acid soils (Valencia and Ligarreto, 2010b). Considerable progress has occurred in identifying adapted germplasm to these soils in annuals, grasses and forage legumes in Colombia (Tab. 1), with little knowledge of the control mechanisms. Considerable research has been done to elucidate the mechanisms of toxicity and tolerance of aluminum in the last decade. The study of differential tolerance mechanisms will allow for an improved, faster, more accurate varietal (Valencia and Leal, 2004). Various methodologies for selecting Al-tolerant genotypes have been developed (Moustakas *et al.,* 1993; Horst *et al.,* 1997; Valencia *et al.,* 1999; Valencia, 2002b, Basu *et al*., 1999), to reduce the time and cost of research. This article briefly describes the effects of aluminum and some mechanisms that may produce differential tolerance in plants.

TABLE 1. Improved varieties from ICA, CIAT, Corpoica, CIMMYT, FFA, INTSORMIL and Alcaravan, released for cultivation in acid soils of Colombia.

Crop	Variety	Year
Rice	Oryzica Sabana 6	1991
	Oryzica Sabana 10	1995
	Soyica Altillanura 2	1994
	Orinoquia 3	1999
Soybean	Corpoica Libertad 4	2004
	Corpoica Taluma 5	2006
	Corpoica Superior 6	2009
	Sorghica Real 40	1991
Sorghum	Sorghica Real 60	1991
	Icaravan 1	1993
	Sikuani V-110	1994
Corn	Corpoica H-108	2000
Grass and forages		
Androgon gayanus	Carimagua 1	1981
Stylosanthes capitata	Capica	1983
Centrosema acutifolium	Centrosema Vichada	1987
Braquiaria dictyoneura	Pasto Llanero	1987
Arachis pintoi	Maní Forrajero	1992

Aluminum toxicity in plants

Aluminum toxicity is a major limiting factor for plants in acid soils with a pH below 5.0, but can occur at a pH as high as 5.5. This problem is particularly acute in extremely acid subsoils which are difficult to lime. These subsoils reduce the depth of plant roots, increase susceptibility to drought and decrease subsoil nutrient utilization (Foy *et al.,* 1978).

According to Wang *et al.* (2006), Al toxicity is considered a complex disorder of growth and development of plants, which can manifest as a deficiency of essential nutrients such as calcium, magnesium, iron or molybdenum, reduced phosphorus availability or manganese and hydrogen toxicity.

Watanabe *et al.* (2006) found that the absence of phosphate in the presence of aluminum reduced the weight of roots of the hybrid *Brachiaria* in a lesser proportion than in *Andropogon gayanus*. According to Mejía *et al.* (2009), plants respond to P deficiency by increasing the formation and elongation of lateral roots and reducing primary root elongation. Changes in morphology and root growth are proportional to the concentrations of growth regulators, particularly auxin, cytokinins and ethylene. Stimulated production of ethylene in plant roots with deficiencies of phosphorus (P) may be responsible for the formation of root hairs. Levels of cytokinins decrease in P deficient plants. Genes have been identified which influence expression of auxins and control the lateral development of the root (Hammond *et al.,* 2004).

Aluminum affects the plant from the standpoint of physiological and biochemical aspects as follows: structure and function of the cell membrane, by joining aluminum to the hydrophilic region of phospholipids and altering the natural interaction between lipids and proteins; DNA synthesis and mitotic processes, increasing the rigidity of the double helix and inhibiting nucleic acid synthesis; cell elongation, by joining the free carboxylic groups of pectin, reducing the elasticity of the cell wall; mineral absorption and metabolism by interfering with the absorption and transport of essential plant elements (Hamilton *et al*., 2001; Taylor, 1989; Wagatsuma *et al.,* 1987), resulting in poor growth and production (Wang *et al.,* 2006).

Symptoms of aluminum toxicity in plants are not easily identifiable and resemble phosphorus deficiency, or in some cases calcium (Ca) or iron (Fe) deficiencies, such as in rice (*Oryza sativa*), sorghum and wheat (Rout *et al.,* 2001). The main symptom of aluminum toxicity in plants is the

rapid inhibition of cell division and elongation of the root (Kochian, 1995 and Wang *et al.,* 2006), which is caused by different mechanisms, such as aluminum interaction with the cell wall, the plasma membrane and the root symplast. Delhaize and Ryan (1995) suggest that growth inhibition per se provides little information about the causes of stress that precede or coincide with changes in the growth and it is necessary to know the locations where the phytotoxicity occurs.

Cellular localization of aluminum in the roots

Electron microscopy and mass spectrometry have been used to provide direct evidence of quick Al absorption at the intracellular level in roots. Microanlytical techniques do not allow for the clear determination of whether Al accumulates in the apoplast or symplast (Silva *et al.,* 2002). Colorimetric methods based on fluorescence staining with Al unions are widely used; the lumogallion fluorescence method has the highest sensitivity to Al (3 - [2.4 dihidroxiphenilazo]-2-hydroxy-5-chlorobenzene sulfonic acid), which permits ion mapping at the cellular level through microscopy (Roos, 2000). Although Rangel (1996) reported that the highest amount of aluminum is found in the apoplast, this methodology is not only able to determine that Al accumulates in greater proportion in the cellular apoplast but also that a substantial amount of Al is found in the periphery symplast and the cellular nuclei in the root tissue of soybean (Silva *et al.,* 2002).

With the use of isotope ^{26}Al and mass spectrometry, Taylor *et al.* (2000), also showed that the cell wall of the species *Chara* sp. was the site of highest Al accumulation and its transport through the plasma membrane was fast, with Al detected in the symplast only 30 min after exposure.

The Al concentration in the root depends on the differential sensitivity of plants to aluminum and its effect on growth is related to the content in the root, since tolerant plants have exclusion mechanisms for this metal (Yamamoto *et al.,* 1994). Experiments conducted by the researchers Delhaize *et al.,* 1993, and Sasaki *et al.,* 1997 demonstrated that Al accumulates at the apices of the root, including the cap and meristematic and elongation zones by applying hematoxylin staining techniques in different wheat varieties.

Aluminum tolerance mechanisms of plants

The intra-and interspecific variability of species has provided a significant source of germplasm useful to breeding programs that aim to develop cultivars tolerant to aluminum (Delhaize and Ryan, 1995; Valencia and Ligarreto, 2010a). This variability has been studied from the genetic and physiological standpoint, searching for the mechanisms that control tolerance.

Genetic control of tolerance

Plants differ in their ability to grow in acid soils (Kochian, 1995) and the mechanism of tolerance is probably not the same in all species, moreover in some species a combination of mechanisms may be operating simultaneously to produce tolerance, hence the genetic control of tolerance can be very complex and involve many genes (Valencia and Leal, 2004), other reports indicate that by altering the synthesis of citrate, transgenic plants can produce aluminum tolerance (De la Fuente *et al.,* 1997; De la Fuente and Herrera, 1999).

Differential tolerance has been reported in rice, soybeans, corn, sorghum, wheat, potatoes, alfalfa, tomato, sunflower and other species; intraspecific differences were found. In some plants such as maize, additive effects explain to a greater degree the tolerance to Al than dominance or epistatic effects. Arcos *et al.* (2007), in studies to determine the genetic effects of callose formation in root tips of maize of resistant and susceptible lines, found that the effects of additivity and nonadditivity were important. To Mortvedt *et al.* (1983), this characteristic can be controlled by a single locus with multiple allelic series and there are no reports of cytoplasmic effects.

Major advances in genetics of tolerance have been achieved in wheat (*Triticum aestivum*) (Carver and Ownby, 1995). In this species, differential tolerance has been attributed to a single dominant gene, although complex inheritance has also been suggested, as in soybean, alfalfa, tobacco, rye, sorghum and maize. Tolerance in barley is controlled by a single locus on chromosome 4H. Molecular markers linked to Al tolerance loci have been identified and validated in a wide range of populations (Wang *et al.,* 2006).

The wheat gene that confers resistance to aluminum, ALMT1, has been cloned and identified as a gene encoding malate transporter activated by aluminum, and the expression of this gene in other genotypes increases malate exudation and improves resistance to aluminum (Delhaize *et al.,* 2004). According to Ma *et al.* (2000), organic acid release is stimulated by aluminum to express genes on the short arm of triticale chromosome 3R.

Physiological tolerance mechanisms

Tolerance mechanisms are commonly defined as internal and external mechanisms. Internal tolerance would

be the compartmentalization of Al in vacuoles such as organelles after being absorbed, reducing its toxic effect. Outer tolerance is related to the ability of plants to prevent absorption and transportation of aluminum into the plant. According to Silva *et al.* (2002) some of the physiological mechanisms are:

Apoplast properties and the cation exchange capacity (CEC) of the root cells. The roots have a net negative charge and therefore a cation exchange capacity (CEC). These negative charges come from the carboxylic groups associated with various components of the cell wall and outer face of the plasma membrane. Among these components, peptic substances (extensive complex of polygalacturonic acid in 35% of cell wall material) have been associated with the root CEC. Cell walls with a lower content of pectin and high degree of methylation (low CEC) bind less Al and therefore are more resistant to damage from Al. This low CEC could contribute to a lower accumulation of Al in the symplast because of a lower activity of Al^{3+} on the surface of the plasma membrane (Silva *et al.,* 2002). According to Blamey and Breem (1990), a poor root CEC characterizes plants adapted to soils with a high aluminum content.

Secretion of mucilage. Secretion products of the root or mucilage contain polysaccharides with a high molecular weight (Ray *et al.,* 1988). Mucilage can protect the root from Al damage through its ability to form bonds with polyvalent cations. There is evidence that mucilage plays an important role in protection against Al in soybean, cowpea, sorghum and wheat. Root mucilage contains substantial amounts of proteins that can also contribute to the reduction of Al toxicity. Differential tolerance may be associated with the amount and composition of mucilage. The presence of organic acids in mucilage may chelate aluminum before it comes in contact with the cell surface (Henderson and Ownby, 1991).

Composition and permeability of the plasma membrane. The plasma membrane has been suggested as the primary site of Al toxicity (Barceló *et al.,* 1996). The negative potential of the membrane has been suggested as a tolerance mechanism to Al, where a membrane with a reduced negative charge lowers Al activity and phytotoxicity, because of the lower amount of Al that may attach to the membrane. Tolerant genotypes can maintain membrane integrity and electrical balance through a net inflow of H^+ and K^+ flux compared to susceptible genotypes. It has been postulated that Al can be metabolically excluded from the roots of resistant genotypes by the selective flow of Al through the membrane by transport proteins.

Alkalinization of the rhizosphere. The change in pH induced by the rhizosphere of plants has been proposed as a mechanism for excluding Al. The increased pH decreases the solubility and toxicity of Al; plants that maintain a relatively high pH in the apoplast or the rhizosphere are exposed to a lower Al^{3+} activity.

Degenhardt *et al.* (1998) uncovered direct evidence of the effect of pH on the activity of the rhizosphere with a study that determined the pH in a wild genotype and an aluminum-tolerant mutant of *Arabidopsis thaliana* (alr-104). Although no differences were detected in the flow of H^+ between roots of the wild type and the mutant in the absence of Al, the pH of the rhizosphere of the mutant genotype was increased two-fold relative to the wild type when the plants were exposed to 300 uM Al.

Flow and concentration of organic acids anion of low molecular weight in the root. Plants have mechanisms to reduce aluminum toxicity through the formation of complex aluminum chelate of low toxicity. Tolerant plants contain or exude anions of organic acids or other ligands that can chelate aluminum at the root-soil interface or within the plant. It is contended that aluminum stimulates the exudation of organic acids such as citrate, malate and oxalate in the roots as an important aluminum tolerance mechanism.

Miyasaka *et al.* (1991) observed that tolerant varieties of bean plants released citrate 70 times higher in the presence of aluminum than in its absence; conversely, the more susceptible plants released citrate only 10 times higher in the presence of Al than the controls, in a medium without aluminum. Delhaize *et al.* (1993) obtained similar results in studies where tolerant wheat varieties exuded 5-10 times more malate than susceptible varieties. This exudation of malate was specific for aluminum and was produced in the root terminal, 5 to 10 mm from the apex. Silva *et al.* (2002) reported citric acid associated with aluminum tolerance in soybeans, sorghum, papaya and tobacco; malic acid in alfalfa; citrate and malate exudation in maize; and malic and oxalic acids in wheat.

Delhaize and Ryan (1995) suggest that the exudation of malate, present as divalent ions, from the cytoplasm to the external medium is achieved by an electrochemical gradient and could be mediated by channels in the plasma membrane. Although the apices of aluminum tolerant seedlings synthesize more malate than susceptible ones in response to aluminum, the apices of both genotypes show similar activity for PEP carboxylase (phosphoenolpyruvate carboxylase) and NADP-malate dehydrogenase, two

important enzymes in the synthesis of malate. Tolerant and susceptible genotypes have the same ability to synthesize malate, the difference lies in the ability to transport malate through the membrane in response to an aluminum inductor. For this, the ALMT1 gene in wheat is responsible for the tolerance; could be encoded to regulate the permeability of malate channels. According to Delhaize *et al.* (2004), barley transgenic plants with the gene ALMT1 were consistent with tolerance to aluminum and malate exudation. These results demonstrate that ALMT1 is an important gene that confers a high tolerance to aluminum and Basu *et al.* (2001) found that transgenic *Brassica napus* plants over-expressing a mitochondrial manganese superoxide dismutase cDNA are resistant to Al.

In sorghum, expression of the gene Alt_{SB} responsible for tolerance is associated with citrate exudation in the root (Magalhaes *et al.,* 2007). Silva *et al.* (2002) found a relationship between tolerance to aluminum and citrate release after four to six hours of exposure to aluminum stress in soybean; detected exudation of citric acid in 30 min. After exposure to Al, maximum induction occurs after 6 h. However, root growth and citrate release were similar in susceptible and resistant genotypes in the first six hours of exposure to Al and significant differences were only detected after 24 h of treatment.

In research by Delhaize *et al.* (2001) on transgenic tobacco with overexpression of the enzyme citrate (100 times more), the transformed plants showed no substantial improvement of root growth compared to the wild type when grown in the presence of aluminum. Therefore, it is unknown whether the amount of organic acids released by the plant is a reliable variable for classifying genotypes of aluminum tolerance. Nian *et al.* (2004) suggest that citrate secretion induced by aluminum stress may not be a differential tolerance mechanism of some soybean genotypes, finding susceptible soybean genotypes secreting more citrate than genotypes tolerant to aluminum.

Compartmentalization of aluminum in the vacuole. Plants with a high tolerance to aluminum can accumulate large amounts of Al. In many cases, high levels of Al are not toxic to the plant because it is present as non-toxic organometallic complexes, located in some instances in cellular vacuoles (Cuenca *et al.,* 1990). However, there is little evidence that the tolerance mechanism by sequestration of Al in the vacuole in the plant is occurring. In cells of the root apex at the primary site of Al toxicity, only small vacuoles are present and according to Silva *et al.* (2002), these small vacuoles accumulate significant amounts of Al-P and Al-Si, in maize genotypes tolerant to aluminum. The vacuolar compartmentalization was accompanied by a reduction in the apoplast Al and improved root elongation. However, in Australian tolerant and susceptible perennial grasses, Crawford *et al.* (1998) observed that most of the intracellular Al was associated with apical cellular nuclei without evidence of accumulation of precipitates of Al-P in the vacuole.

Despite the presence of barriers to Al absorption, Al sometimes enters the symplast, depending on the concentration of Al and the cultivar. Because of the strong affinity of aluminum for oxygen donor compounds such as inorganic phosphate, RNA, DNA, proteins, carboxylic acids, phospholipids, anthocyanins and other oxygen donor ligands, very low concentration of aluminum in the symplast is potentially toxic. Therefore, internal mechanisms related to plant aluminum tolerance are: chelation of Al in the cytoplasm, aluminum transport to the vacuole and aluminum complexes with proteins, which permit full or partial inactivation of toxic aluminum (Taylor, 1989).

Conclusions

The apex of the root is the primary site of toxicity and therefore the main effect of aluminum on plants is the inhibition of root growth and elongation. The aluminum binds to the cell wall, the plasma membrane, DNA and other cellular components of the cytoplasm.

Plants differ in their ability to tolerate aluminum in acid soils and genetic and physiological mechanisms are not the same in all species. Genetic control of tolerance can be very complex and involve many genes.

The physiological mechanisms of tolerance are associated with: the apoplast and CEC properties of the root cells, mucilage secretion of the root, composition and permeability of the plasma membrane, alkalization of the rhizosphere, concentration and flow of organic acid anion and compartmentalization of aluminum in the vacuole.

The toxic effects of aluminum can be reduced by chelation processes, internal or external, of Al with various cellular compounds such as organic acids, proteins and polysaccharides. However, there are still large discrepancies on the importance and value of these compounds to differential tolerance to aluminum.

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