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AN OVERVIEW INTO ALUMINUM TOXICITY IN MAIZE

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Summary. The physiological function of Al in plants is controversial. This metal injury or toxicity is often related to field crops grown in acid soils with high availability of Al. Acid soils having pH below 5 increases the solubility of Al, which in subsoils is particularly harmful because it causes shallow rooting, drought susceptibility, and poor use of subsoil nutrients lowering the production of maize crops. In this overview the modulation of excess Al in maize is analysed following an integrated approach that characterises the: (i) interaction of Al with root growth; (ii) threshold of Al toxicity; (iii) interactions with other nutrients accumulation; (iv) reduction mechanisms of nitrate into ammonia; (v) modulation of photosynthesis; (vi) senescence processes coupled to ethylene production. These parameters are also compared with the general knowledge of Al interactions in plant physiology.

***Key Words*:** Al toxicity; ethylene production; nitrate reduction; nutrients accumulation; photosynthesis; oxy radicals

***Abbreviations*:** ACC – 1-aminocyclopropane-1-carboxylic acid; CuZnSOD – Cu,Zn-superoxide dismutase; EFE – ethylene forming enzyme; ϕe – (=Fv/Fm.qP) estimation of quantum yield of non-cyclic electron transport; FBPase – fructose–1,6-bisphosphatase; Fm – maximum fluorescence; Fo – minimum or basal fluorescence; Fv – variable fluorescence; gs – stomatal conductance; LHC – light harvesting complex; NADP-ME – NADP-malic

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enzyme; NADP-MDH – NADP-malate dehydrogenase; OAA; PCR – photosynthetic carbon reduction; PEP – phosphoenolpyruvate; PEPc – phosphoenolpyruvate carboxylase; PN – net photosynthetic rate; PPDK – pyruvate Pi dikinase; PS – photosystem; qE – energy dependent quenching; qN – non-photochemical quenching; qP – photochemical quenching; SAM – S-adenosylmethionine; SOD – superoxide dismutase.

Introduction

Maize is a coarse annual grass that ranks second, following wheat, in the world production of cereal crops. In the world the total area devoted to maize is higher than 129 million hectares, corresponding to 470 million tonnes of maize grain. This high production per hectare reflects the widespread use of hybrids and improved crop management practices. However, the recognized adaptability of maize crop is clearly associated to a wide range of environmental conditions. This species is adapted to such a wide range of climates that this plant is now more extensively distributed over the earth than any other cereal crop. It grows from 48°N to about 40°S latitude all over the world. Similarly, it grows from below sea level to altitudes of about 4 000 metres. The ideal soil for maize is a deep, medium-textured, well-drained, fertile soil with a high water-holding capacity, but it also grows on a wide variety of soils giving high yields if the crop is well managed. This plant specie prefers soils having a pH ranging between 5.5 and 8.0, the optimum range being 5.5 to 7.0, but if it is cultivated in soils having a pH below 5 with high content of Al sulphate, the yield becomes sharply affected. As one of the main constituents of the earth's crust, Al in rocks commonly ranges from 0.45 to 10%. The total Al content in soils is inherited from parent rocks, however, only the fraction of Al that is easily mobile and exchangeable plays an important role in soil fertility. The available Al in acid soils can be taken up rapidly by plants resulting in a chemical stress. Indeed, Al toxicity is a severe impediment in production of many crops in acid soil. Toxicity can be reduced through lime application by raising soil pH, however this amendment does not remedy subsoil acidity, and liming may not always be practical or cost-effective. This review sums up the general knowledge of these nutrient interactions during growth.

Al toxicity and root growth

High Al concentrations are particularly difficult to interpret in terms of physiological responses. A high proportion of Al in the nutrient growth medium might become inert by precipitation (e.g., with phosphate) or by polymerisation and complexation. Thus, the concentration of free Al promoting toxicity in plant metabolism can be much lower

than that existing in the growth medium (Mengel and Kirkby, 1987). Low concentrations of Al can also lead to a stimulation of root growth in tolerant genotypes of *Zea mays* L. (Clark, 1977) due to an increasing activity of the apical meristem (Bennet and Breen, 1989).

In non-accumulators plant species the negative effects of Al on plant growth prevail in soils with low pH (Marschner, 1995), the reduction in root growth being the most serious consequence (Foy, 1983; Lidon and Barreiro, 1998; Calba et al., 1999; Tabuchi and Matsumoto, 2001). This symptom of Al toxicity (Kerridge and Kronstad, 1968; Henning, 1975; Lee and Pritchard, 1984; Simon et al., 1994) has been related to the linkage of Al to carboxylic groups of pectins in root cells (Klimashevsky and Dedov, 1975) or to the switching of cellulose synthesis into callose accumulation (Teraoka et al., 2002), to Al inhibition of mitosis in the root apex (Rengel, 1992; Delhaize and Ryan, 1995; Liu and Jiang, 2001) implicating blockage of DNA synthesis (Horst et al., 1983), aberration of chromosomal morphology and structure (Liu and Jiang, 2001) occurrence of anaphase bridges and chromosome stickness (Liu and Jiang, 2001) and to Al-induced programmed cell death in the root-tip triggered by reactive oxygen species (Pan et al., 2001).

Aluminum toxicity has also been associated with Al^{3+} and $AlOH^{2+}$ dominant ionic Al species (Moore, 1974). However, Al toxicity is greater at pH 4.5 than at pH 4.0 since $AlOH^{2+}$ concentration is about twice as high as that at pH 4.0 (Moore, 1974). Indeed, at pH 4.5 the dissolution of $Al(OH)_3$ into Al^{3+} and $AlOH^{2+}$ is higher because it gives rise to $AlOH^{2+}$, which is a soluble Al form highly toxic to plants. This process eventually is coupled to the inhibition of some protein kinases in *Coffea arabica* (Martinez-Estevez et al., 2001).

According to Comin et al. (1999) tolerant cultivars of *Zea mays* L. have different toxicity mechanisms, following monomeric or polymeric forms of Al supplied to the growth medium. Aluminum can easily polymerise, transforming the monomeric form (Al^{3+}) into a polymeric form (Al_{13}), which is much more phytotoxic in maize (Bell and Edwards, 1986). Yet, although Rayburn et al. (1993) had noticed Al nucleotypic effects on maize, a lack of nuclear DNA content variability was found among wheat isolines differing in Al response (Wetzel et al., 1999) as well as four genes that ameliorate Al toxicity (Ezaki et al., 2001). Indeed, the general responses to Al excess by tolerant genotypes deal with the varying ability of plants to modify the pH of the soil-root interface (Mengel and Kirkby, 1987; El-Shatnawi and Makhadmeh, 2001). Cation uptake might exceed anion absorption leading to the excretion of H^+ by the roots, lowering the pH in the surrounding environment. Al^{3+}-dependent efflux of malate from root apices is also a mechanism for Al^{3+} tolerance in wheat. The malate anions protect the sensitive root tips by chelating the toxic Al^{3+} cations in the rhizosphere to form non-toxic complexes (Zhang et al., 2001). Evidence exists that the difference in Al^{3+}-induced malate efflux between Al^{3+}-tolerant and Al^{3+}-sensitive genotypes lies in the differing

capacity for Al^{3+} to activate malate permeable channels and cation channels for sustained malate release (Zhang et al., 2001). Additionally, it has also been suggested (Osawa and Matsumoto, 2001) that protein phosphorylation is involved in the Al-responsive malate efflux in the wheat root apex and that the organic anion-specific channel might be a terminal target that responds to Al signalling mediated by phosphorylation. Tolerant mechanisms have further been related either to higher uptake rate of NO_3^- in the presence of additional supply NH_4^+, either to the exclusion of excess Al. The increase of the ionic strength of the growth medium as well as the Al activity, might stimulate the rate of this metal adsorption by roots and its precipitation at the outer surface of cortical cells (Pavan and Bingham, 1982). Thus, as Al has reduced mobility and a consequent low bioavailability in tolerant cultivars, higher Al concentrations are required in the growth medium. Prior to the development of toxicity symptoms, this metal penetration in the plasmalemma of meristematic root cells is blocked because of Al accumulation in the apoplasm. After inducing the alteration of plasma membrane permeability (Ishikawa et al., 2001), inside the cell Al is equally harmful. Aluminium detoxification by chelation can also be developed through excretion organic acids and polyphenols to the rhizosphere (Kayama, 2001; Tesfaye et al., 2001), eventually implicating the alteration of Mg and Ca levels (Silva et al., 2001; Yang et al., 2001). Yet, although secretion of organic acids and phosphate by root apices and alkalinization of the apical rhizosphere are commonly believed to be important mechanisms of Al resistance, it was found (Menosso et al., 2001; Wenzl et al., 2001) that root apices of signalgrass secreted only moderately large quantities of organic acids (being efflux from signalgrass apices three to 30 times smaller than from apices of Al-resistant genotypes of buckwheat and maize).

Threshold of Aluminum Toxicity and Interactions on Nutrient Accumulation

To accurately define the threshold toxicity of elements, a dose response curve relating growth to the concentration of nutrient solutions has long been recognised as an accurate criterion (Ulrich, 1952). The assumption of this model is that critical tissue concentrations of nutrients are usually nearly constant because of limited nutrient supply, but toxic concentrations may cause unlimited passive nutrient uptake even through normal growth may have stopped (Berry, 1977; Berry and Wallace, 1989). A dose response curve relating root elongation to Al concentration during maize growth has pointed out that Al concentrations higher then 9 mg/L triggers increasing toxicity (Lidon and Barreiro, 1998). Besides the application of a dose response curve, the characterisation of a nutrient accumulation curve, relating total nutrient accumulation in plant tissues to its concentration in an external growth solution has also been adopted as a final diagnostic criterion to determine potential phytotoxicity. In this context, Al concentration value of ca 13 µg/g (DW) has been reported for maize (Ulrich, 1952; Lidon and Barreiro, 1998).

Several authors reported that in maize roots tissues excess Al affects the distribution of other nutrients (Tanaka and Nasero, 1966; Sivasubramaniam and Talibuden, 1971; Foy, 1978; Dehaize and Ryan, 1995; Ozaki, 2001). In non stressed maize roots, the concentration values of N, P, K, Ca, Mg, Mn, Fe and Zn are (in mg/g[dw]) ca. 12.5, 6, 4, 2.5, 5.5, 0.03, 0.4 and 0.04, respectively. Moreover, Al toxicity triggers an increasing accumulation of K, Mn and Zn in the roots while the concentration of Fe decreases (Furlani and Clark, 1981; Simon et al., 1994; Lidon et al., 2000). This pattern is quite different from that found in xenopus because in this species Al enters in plant cells through a Ca^{2+} channel-like pathway and inhibits K^+ uptake by internally blocking K^+ channels (Liu and Luan, 2001). In maize, the concentration of N is only slightly affected (Lidon et al., 2000). Different plant species grown with high levels of Al usually have lower P, Ca and Mg contents but in maize a clear trend usually can not be found (Rengel, 1992; Simon et al., 1994; Lidon et al., 2000). This characteristic seems to implicate that gene encoding resistance to Al excess triggered by the breaking of Ca homeostasis found in wheat (Sasaki et al., 2002), yet this does not seem to occur in the maize. In non stressed maize shoots, the concentration values of N, P, K, Ca, Mg, Mn, Fe and Zn were (in mg/g [DW]) ca. 18, 5, 6, 3.5, 3.5, 0.04, 0.05 and 0.05, respectively, but toxic Al concentrations decreased significantly the concentrations of N, Mg, P and Fe (Clark et al., 1981; Furlani and Clark, 1981; Alva and Edwards, 1990; Simon et al., 1994; Lidon et al., 1999), whereas an opposite trend prevails with Mn (Lidon et al., 1999). A similar pattern can be found in triticale (Quartin et al., 2001) and *Quercus* (Akaya and Takenaka, 2001), being hypothesized that P deficiency specifically triggers the reduction of biomass production. Nevertheless, considering that in *Melastona malabathricum* high Al levels seem to be the primarily cause of growth (Watanabe and Osaki, 2001), some controversy remains on this subject.

Nitrate Uptake and Reduction

Depending on plant species, Al mediates the inhibition or stimulation of nitrate uptake, following a close link that implicates root acidification capacity and the chemical properties of membranes permeability (Keljens and van Ulden, 1987; Keljens, 1988; Klotz and Horst, 1988; Taylor, 1991; Nichol et al., 1993; Lazof et al., 1994; Lorenc-Plucinska and Ziegler, 1996). In Al stressed maize root acidification capacity and nitrate accumulation were inhibited while the electrolytic conductance was stimulated (Lidon et al., 1998; Ahn et al., 2001). Non-toxic root Al concentrations are associated with this metal binding in negatively charged sites of the cell walls and at the external surface of membranes (Rufty et al., 1995), increasing proton extrusion and net nitrate uptake rate (Kinraid, 1993) or diminishing nitrate efflux (Cakmak and Horst, 1991). Moreover, in Al treated maize the increasing membrane permeability of the roots is coupled to a sharp penetration of this metal into the root symplastic areas inhibiting the root acidification capacity (Durieux et al., 1993; Lazof et al., 1994) and, therefore,

the modulation of the H^+/NO_3^- co/transport. Indeed, excess Al resulting from increased membrane permeability limits the rate of nitrate uptake, possibly through the inhibition of the activity of nitrate transporters (Simon et al., 1994). In the leaves of Al treated maize the properties of membrane permeability remained unchanged but nitrate, nitrite and ammonia concentrations decreased (Lidon et al., 1998). Hence the decreasing accumulation of nitrate in the leaves is not due to plasma membrane degradation, which could inhibit nitrate translocation. The kinetics of leaf nitrate and nitrite reductases also increased significantly in Al treated maize (Lidon et al., 1998), which, according to Dinev and Stancheva (1993), indicated that *in vivo* reduction of nitrate to ammonia was not limited by these enzymes. Eventually, the modulation of nitrate reductase activity controls metabolic oscillations implicating concurrent alterations of nitrate concentrations (Sanchez and Heldt, 1990), which might restore the initial balance between nitrate reduction and accumulation. As in maize, the concentrations of nitrite are somewhat complementary to those of nitrate, Al mediated interactions coupled to nitrate accumulation also control nitrite into ammonia conversion. Indeed, the enzyme kinetics of nitrate to ammonia conversion follows a cascade inhibition because, as nitrate reductase activity is limited by a decreasing accumulation of substrate, the related decrease of nitrite concentrations also becomes a limitation for maximum nitrite reductase functioning and therefore affected the ammonia concentrations (Lidon et al., 1998). Thus, nitrite reductase activity parallels the Al mediated modulation of nitrate reductase, further limiting the imbalance between nitrite consumption and accumulation (Lidon et al., 1998).

Modulation of Photosynthesis

In Al treated maize the rate of the "light" and "dark" photosynthetic reactions are stimulated. This pattern is specific for maize because it is long recognized that this metal toxicity specifically inhibits the photosynthetic apparatus of many species (McLean, 1979; Cambraia and Calbo, 1980; Foy, 1984; Haug, 1984; Ohki, 1986; Moustakas and Ouzounidou, 1994; Lorenc-Pblucinska and Ziegler, 1996; Akaya and Takenaka, 2001). The structure and organisation of chloroplasts from Al treated maize are coupled to non-significant alterations of Fo and Fv/Fm, which indicates that the energy transfer from the LHCII was not inhibited (Lidon et al., 1999). This pattern is somewhat similar to that reported for other plants, namely for *Quercus* (Akaya and Takenaka, 2001). Thus, the yield of fluorescence emission of Chl a, before the excitons have migrated to the reactions centres (Krause and Weis, 1991), is independent of photochemical events (Krause and Weis, 1984) or of both the initial density of excitons within the PSII pigments (Lichtenthaler, 1988), and of structural conditions that could affect the probability of excitation energy transfer between antenna pigments and reaction centres of PSI and PSII (Prange, 1986). In fact, the maintenance of the Fv/Fm ratio combined with the stabilisation of Fo suggests the presence of regulatory mechanisms act-

ing in the antennae (Krause, 1988) that shield against impairments, implicating the inactivation of PSII (Franklin et al., 1992). Toxic Al concentrations also triggered only minor effects on qE, qN and qP of maize chloroplasts (Lidon et al., 1997b), indicating that the rate of utilisation/dissipation of excitation energy by PSII was not inhibited. Additionally, a similar tendency occurs with the proportion of excitation energy trapped in the open centres of PSII (Horton and Hague, 1988; Walker, 1988; Krause and Weis, 1991; Schafer and Schmidt, 1991). These global effects are well expressed by ϕe, which sharply increased (Lidon et al., 1998), indicating that the *in vivo* reducing power generated by the photosynthetic electron flow was stimulated. The confirmation of this trend can also be found in isolated thylakoids of Al treated maize because the rates of the photosynthetic electron transport associated with PSII were stimulated (Cambraia and Calbo, 1980; Haug, 1984; Lidon et al., 1997b; 1999) and, additionally, a similar trend was displayed by CO_2 assimilation. Moreover, the pattern displayed by CO_2 intake can be interpreted considering that the diffusion of the assimilated CO_2 in C_4 leaves between bundle sheath and mesophyll cells is highly restricted (Hatch and Osmond, 1976; Hattersley and Perry, 1984; Hatch, 1987; Weiner et al., 1988; Jenkins et al., 1989). Indeed, the development of relatively high CO_2 concentrations in the bundle sheath cells are required during photosynthesis (Hatch and Osmond, 1976; Edwards and Walker, 1983; Hatch, 1987; Furbank and Hatch, 1987; Furbank et al., 1989). Following a pattern quite different from the reported for *Quercus* (Akaya and Takenaka, 2001), PN and gs were stimulated in Al treated maize (Lidon et al., 1996, 1997b, 1999) yet, starch and soluble saccharide concentrations as well as the FBPase activity did not vary significantly (Lidon et al., 1997a). This indicates that the carbon flux necessary for the regeneration of ribulose bisphosphate was preserved. As FBPase activity is a limiting step in the PCR cycle further controlling saccharide synthesis (Lawlor, 1987) and hence the rate of saccharide synthesis was unaffected in Al treated maize, the additional assimilated carbon was exported to the cytosol via a phosphate translocator in the form of triose/P produced by the assimilatory segment of the PCR cycle (Geiger and Servaites, 1994). In the NADP-ME type enzymatic system coupled to mesophyll and bundle sheath cells of maize the developed in increasing Al concentrations, although inhibiting the activity of NADP-MDH, stimulated the kinetics of PPDK, NADP/ME and PEPc (Lidon et al., 1997a). The inhibition of NADP-MDH activity limits maximum activities of NADP-ME, PPDK and PEPc. As PPDK was activated by high ratios of ATP/ADP and pyruvate/PEP in the mesophyll cells (Edwards et al., 1985), the important regulatory role advanced for this enzyme kinetics in the regeneration of PEP (Andrews and Hatch, 1969) was also inhibited in maize. Nevertheless, the increased CO_2 uptake in the mesophyll cells indicates that, OAA and malate will not accumulate inhibiting PEPc (O´Leary, 1982). Furthermore, the reaction proceeding towards PEP synthesis will not be inhibited and this will contribute to maintaining a balance between PEP production and its utilisation in carboxylation reactions.

Oxy Radicals Production in the Chloroplasts

Non lethal Al toxicity in maize does not change significantly the relative proportions of the thylakoid acyl lipids and, additionally, the ratio between total galactolipids or phospholipids and total acyl lipids remains almost constant (Lidon et al., 1997a). Nevertheless, thylakoid lipid peroxidation and ethylene production coupled to the photosynthetic light reactions increase significantly (Lidon et al., 1999) which, as previously shown with other heavy metals (Rabinowitch and Fridovich, 1983; Gutteridge, 1987; Fernandes and Henriques, 1991; Lidon and Henriques, 1993), reveals an increasing rate for the peroxidative chain reactions in the chloroplasts. In Al-treated maize phenol concentration also increases, whereas the chloroplast quinone pool decreases (Lidon et al., 1999). An Al induced phenol accumulation has long been reported (Klimascevskii, 1972), which could be associated with an enhanced generation of active oxygen forms. Inhibition of SOD in Al-treated maize plants was reported (Lidon et al., 1999) which may result in an increased level of superoxide free radicals. In Al-treated maize ascorbate peroxidase activity increases significantly (Lidon et al., 1999). This is an observation quite similar to previous reports of the interacting effect of Al excess on peroxidase kinetics (Cakmak and Horst, 1991), yet, maximum activity of glutathione reductase becomes inhibited, whereas that of dehydroascorbate reductase was stimulated (Lidon et al., 1999). The Al mediated effect on glutathione reductase allows the assumption that *in vivo* this enzyme activity is the limiting step in the chloroplast peroxidase system of maize (Polle et al., 1980; Cross and Jones, 1991). Indeed, as the kinetics of glutathione reductase is inhibited, the peroxidase system has a lower efficiency due to substrate limitations for the required functioning of the enzymes ascorbate peroxidase and dehydroascorbate reductase. Additionally, catalase also decreases in Al treated maize (Lidon et al., 1999) and, therefore, the control of the concentrations of photosynthetically-generated hydrogen peroxide that diffuses out of the chloroplast also is less effective in the peroxisomes. This inhibition apparently is related to the decrease of SOD activity, as their synergistic function (Rabinowitch and Fridovich, 1983) is blocked, further allowing the production of hydroxyl radicals (Rabinowitch and Fridovich, 1983).

Ethylene Production Through the Methionine Pathway

There have been some conflicting reports about the extent of involvement of ethylene in leaf senescence. Foliar senescence triggered by ethylene has long been suggested (Aharoni et al., 1979a,b; Aharoni and Lieberman, 1979; Grodzinski et al. 1982; Kao and Yang, 1983; Lesham et al., 1986), however since the works of Thomas and Stoddart (1980) and Roberts et al. (1984) these results are increasingly being cosidered with caution. It has also been suggested that leaf senescence, involving ethylene production might be retarded by several ions, or by anaerobic conditions (Kao, 1978; Yu and Kao, 1980; Kao and Yu, 1981). In this context Al toxicity appears to be the

result of several interactions, and there is no consensus on its mechanisms of action in higher plants (Hampp and Schnabl, 1975; Jackson et al., 1990).

In Al treated maize ethylene production through the methionine pathway increases (Lidon et al., 1995), indicating, as previously suggested (Aharoni et al., 1979a,b; Aharoni and Lieberman, 1979; Grodzinski et al., 1982; Kao and Yang, 1983; Lesham et al., 1986), a positive correlation with the decreased leaf elongation and biomass production. ACC synthase activity also increased with non-lethal Al levels, with the concentration of endogenous ACC showing minimum values in the absence of Al (Lidon et al., 1995). Thus, in contrast with previous suggestions for different plant species (Adams and Yang, 1979; Yu et al. 1979; Konze and Kende, 1979; Yu and Yang, 1980; Kende and Boller, 1981) stating that the conversion of SAM to ACC is the step at which ethylene biosynthesis is regulated, in maize Al did not affect the kinetics of ACC synthase. The EFE activity showed increasing values with non-lethal Al concentrations (Lidon et al., 1995), which further supports that assumption. Nevertheless, although the participation of chemical elements in the opening of the cyclopropane ring has been suggested for the chemical oxidation of ACC (Boller et al., 1979; Baldwin et al., 1985), for the degradation of ACC by free-radical-producing enzymes (Vioque et al., 1981; Bousquet and Thimann, 1984) and for the production of ethylene by plant extracts *in vitro* (Konze and Kwiatowski, 1981), apparently the *in vivo* activity of this enzyme complex was not affected by Al treated maize.

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