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MINI-REVIEW

Microbial interactions with aluminium

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Although aluminium is the most abundant metal in the Earth's crust, it lacks biological functions and shows a low bioavailability. Acid rain, however, solubilizes aluminium to toxic levels. Most research on the biological effects of aluminium has been centred on the analysis of aluminium-tolerant plants as well as its possible relationship with neurological disorders in humans. Also, several studies have been reported concerning aluminium effects on microorganisms, with more interest directed to cyanobacteria, soil bacteria and mycorrhizal fungi. Competition with iron and magnesium, and binding to DNA, membranes or cell walls are considered the main toxic effects of aluminium in microbes.

Keywords: aluminium, interaction, microbe

Introduction

Microorganisms continuously interact with varied inorganic ions, some of which are essential for biological functions whereas others exert inhibitory effects that limit normal development. Aluminium is a metal lacking biological functions and in this respect pertains to the non-essential class of chemical elements. The presence of toxic ions in the environment may select for the appearance of tolerant microbial variants possessing genetic determinants which confer resistance to the poisonous compounds (Trevors *et al.* 1986, Belliveau *et al.* 1987, Trevors 1987, Silver & Walderhaugh 1992, Slawson *et al.* 1992).

In this article we review the current status of knowledge on the microbial interactions of aluminium that may contribute to the analysis of bacterial mechanisms of resistance to the metal ions. It must be noted that no information was found on the effects of aluminium on eukaryotic algae or protozoa cells.

Chemical properties of aluminium

Aluminium is located in group IIIa of the periodic table, generally having a valence of +3. Aluminium displays a high reactivity with oxygen at room temperature, and rapidly reacts with acids and bases to produce salts and release hydrogen. Commonly present in the form of oxides and silicates, aluminium is the most abundant metal in the Earth's crust, representing 8.8% of its weight (*The Merck Index*, 1989). Aluminium is a light metal with a density of 2.78 g cm^{-3}, and is an excellent conductor of heat and electricity.

Aluminium preferentially interacts with oxygen-donor ligands which, being anionic, counter the +3 charge of the cation. In biological environments, carboxylate and phosphate groups, inorganic phosphate, citrate, polyphosphate, nucleotides, and polynucleotides fill this requirement (Macdonald & Martin 1988).

Aluminium bioavailability

Bioavailability of a substance is defined as a measure of its potential to interact with biological systems and to cause a response (Exley & Birchall 1992). The bioavailability of aluminium in soils and its concentration in waters remains low because of its adsorption to mineral surfaces, its association with organic matter and the insolubility of the hydroxide complexes that form when the pH is near neutrality. In recent years, however, the presence of aluminium has been recognized as a serious pollution problem related to acidification of soil and water. Through acid rain aluminium is being released (solubilized) from its natural reservoirs (Figure 1) (Ganrot 1986, Martin 1986, Macdonald & Martin 1988, Gilmour 1992, Myrold & Nason 1992).

Martin (1986) and Macdonald & Martin (1988) indicate that the proportion of the different oxidation forms of

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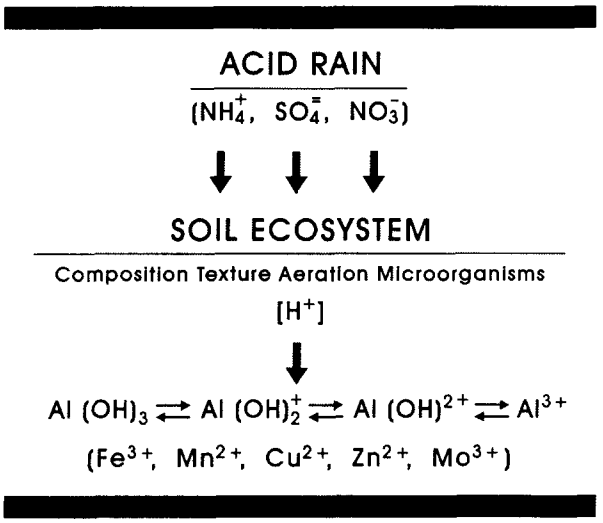

Figure 1. Interactions of acid precipitation with the soil ecosystem and factors affecting solubilization of aluminium ions from soil. Other released metal ions are also shown.

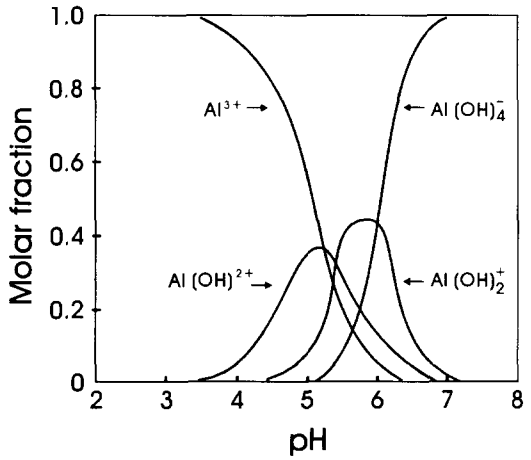

Figure 2. Distribution of the various aluminium species as a function of pH. The toxic Al^{3+} form predominates below pH 5.0; insoluble hydroxide species occur at or above neutrality. Adapted from Macdonald & Martin (1988) with permission.

aluminium is a function of the environmental pH and that small variations in acidity cause great changes in the concentration of each of those species (Figure 2). Disregarding the interactions of aluminium with other substances, in solutions with a pH lower than 5.0, most aluminium exists as an octahedral hexahydrate $Al(H_2O)_6^{3+}$ (usually referred to as Al^{3+} or free aluminium). As the solution becomes more alkaline, this complex deprotonates to successively yield $Al(OH)^{2+}$, $Al(OH)_2^+$ and, in neutral solutions, the precipitate $Al(OH)_3$, which redissolves at pH $\geqslant$ 7.4 due to the formation of tetrahedral $Al(OH)_4^-$, which shows a structural homology to phosphate (PO_4^{3-}).

Bruce *et al.* (1988) found that in soils with a pH of 5.8, the concentration of free aluminium is 6.3 μM. Lowering the pH to 4.77 increased the Al^{3+} level to 700 μM, whereas increasing the pH to 6.22 diminished the Al^{3+} concentration to 5 μM.

Aluminium bioavailability depends not only on the natural properties of soil and on changes in pH, but man's activities can also modify it through inadequate agricultural procedures or by disposal of industrial, mining and even domestic wastes (Foy *et al.* 1978).

Aluminium toxicity

It is well documented that the aluminium ion is toxic to living organisms, which is understandable since most organisms thrive in environments with pH ranges near 7.0 and, thus, have not evolved the ability to tolerate high aluminium activities (Driscoll 1985). With an increase in the concentration of aluminium in acidified waters, pathologies in man, animals and plants have been observed (Foy *et al.* 1978, Joshi 1990, Gagen *et al.* 1993, Stenson *et al.* 1993, Wilkinson & Campbell 1993). However, the causes of these disorders have not been assigned since the acidification of the environment may directly affect the organisms by increasing the concentration of H^+ or, indirectly, through changes in the levels of aluminium and other ions, such as phosphate (Pi) or heavy metals (Myrold & Nason 1992).

Numerous studies on variations in the pH of soil and water have shown that acidification is detrimental to most organisms (Keyser & Munns 1979, Munns 1986, Gilmour 1992, Myrold & Nason 1992, Gagen *et al.* 1993, Stenson *et al.* 1993). Since acidification alters the biogeochemical properties of many metals (Figure 1), Gilmour (1992) has suggested that the mobilization, or solubilization, of toxic metals may be the primary cause of the adverse effects attributed to acidification of soil and water. Thus, it is possible that the toxic effects of acidification are at least partially due to the action of released aluminium ions.

The mechanism of aluminium toxicity is not understood, but several possible targets have been advanced, mostly in plants and animals, given the metal's chemical properties. Aluminium ions may bind to the hydrophilic heads of cell membrane phospholipids, thus altering lipid–protein interactions and modifying transport activity (Suhayda & Haug 1986, Zambenedetti *et al.* 1994). Alternatively, aluminium may diminish the negative charges of phospholipids, or of amino acid residues on membrane proteins, blocking surface potential (Kinraide *et al.* 1992). Binding of aluminium to membrane transport proteins may also disrupt their function (Schroeder 1988). Aluminium ions are taken up by microbial cells via a still unknown pathway. Once inside the cells, aluminium may affect metabolism by binding to enzymes (e.g. phosphatases) or to enzyme substrates (Macdonald & Martin 1988).

The ability of aluminium to substitute for magnesium in biological systems is derived from a high association constant with diverse ligands. For example, aluminium binds almost 10^7 times more tightly to ATP than magnesium does, which means that aluminium concentrations lower than nanomolar are required to compete with milimolar

Table 1. Mechanisms of aluminium toxicity in microorganisms

Mechanism	Affected process
Acidification of the medium	ion homeostasis, macromolecule structure/function
Binding to membrane components	membrane transport
Binding to enzymes or substrates	enzymatic processes
Substitution for magnesium	metabolic processes
Inhibition of ion transport	ion homeostasis
DNA binding	DNA replication/transcription
ATP binding	energy-requiring processes
Inhibition of ATP synthesis	metabolic processes

magnesium concentrations (Macdonald & Martin 1988). Inhibition of magnesium-dependent activities may modify a variety of cellular processes, since magnesium plays diverse biochemical and regulatory functions in all living organisms. Table 1 summarizes the possible mechanisms of aluminium toxicity in microorganisms.

Effects of aluminium on bacteria

Inorganic ions that interact with bacteria can be divided into three types: (i) a group of essential ions (K, Mg, Pi, SO_4^-) required for normal metabolism; (ii) a second kind of essential micronutrient ions which are toxic when present in relatively high concentrations (Cu, Zn, Co); and (iii) a group of intrinsically toxic ions with no known biological function (Pb, Hg, Cd, Ag, Al) (Silver 1983). Most bacteria have evolved specific transport systems for essential ions but these pathways are generally lacking for toxic ions (Silver & Walderhaugh 1992).

Relatively little research has been reported about aluminium effects on microorganisms, when compared to plants. The complex chemistry of aluminium, which polymerizes, interacts with phosphates and organic acids, and acidifies culture media, frequently complicates the interpretation of experimental results.

Guida *et al.* (1992), when studying aluminium toxicity towards *Escherichia coli*, found that growth inhibition was markedly dependent on pH, recording sensitivity to 0.9 and 2.25 mM Al at pH 5.4 and 6.6, respectively; aluminium toxicity increased when iron was omitted from the medium, which suggests that aluminium uptake involves iron transport systems, as previously reported (Davis *et al.* 1971). Accordingly, Gascoyne *et al.* (1991) found that some siderophore-producing alkalophilic bacteria were able to accumulate aluminium, as well as iron and gallium, from culture media. Inhibitory aluminium concentrations increased porphyrin synthesis and excretion in *Arthrobacter aurescens* (Scharf *et al.* 1994); aluminium treatment also caused a significant decrease in intracellular haem in *A. aurescens*, thus confirming a role of aluminium toxicity in porphyrin metabolism.

Using *lux* transcriptional gene fusions, Guzzo *et al.* (1991) showed that aluminium induces the expression of the flagellin (*fliC*) gene in *E. coli*, suggesting that environmental metals may regulate bacterial motility properties. On the other hand, aluminium was found to stimulate the activity of the enzyme luciferase in *E. coli* transcriptional fusions in a pH-dependent fashion (Guzzo *et al.* 1992). This finding has been related to the widespread use of transcriptional luminescence gene fusions as biosensors for environmental pollutants (Guzzo *et al.* 1992).

Aluminium concentrations lower than 100 μM (Vargas & Graham 1988, Lesueur *et al.* 1993) or even 50 μM (Keyser & Munns 1979, Whelan & Alexander 1986, Wood *et al.* 1988) are inhibitory to the growth of *Bradyrhizobium* spp. Appanna *et al.* (1994) found that a *Pseudomonas fluorescens* strain was able to tolerate up to 50 mM Al when provided as a citrate–aluminium complex. The citrate moiety of aluminium–citrate complexes, supplied as the sole carbon source, is known to be utilized by *Pseudomonas* cells (Madsen & Alexander 1985, Appanna & St Pierre 1994). The growth of *P. fluorescens* in the presence of aluminium, as well as the fate of aluminium ions, appeared to be affected by the level of Pi in the culture medium (Appanna & St Pierre 1994).

Plant nodulation by bacteria appears to be a very aluminium-sensitive process (Munns 1986, Graham 1992). Johnson & Wood (1990) found that aluminium binds to DNA in *Rhizobium* cells, affecting DNA synthesis in aluminium-sensitive but not in aluminium-tolerant strains. Inhibition of nodulation was reported in *Stylosanthes* (Carvalho *et al.* 1981, 1982) and in other legumes (Murphy *et al.* 1984) by 25 μM Al. On the other hand, Richardson *et al.* (1988) found that as little as 7.5 μM Al prevented expression of nodulation genes in *Rhizobium*. Less than 5 μM Al can inhibit soybean nodulation (Brady *et al.* 1993).

Binding of aluminium to the cell wall has been demonstrated in *Staphylococcus aureus* (Bradley & Parker 1968) and the cyanobacterium *Anabaena cylindrica* (Pettersson *et al.* 1985b). Inhibitory effects of aluminium on growth, photosynthesis and nitrogen fixation by *A. cylindrica* were also reported (Pettersson *et al.* 1985a). By using X-ray microanalysis in *A. cylindrica*, Pettersson *et al.* (1985b) showed that aluminium is rapidly taken up and accumulated by cell walls and polyphosphate granules, and that increasing Pi concentration in the culture medium caused a higher accumulation of aluminium in both structures; uptake of aluminium took place mainly by passive diffusion and was independent of Pi consumption (Pettersson *et al.* 1985b, 1986). It is considered that accumulation of aluminium occurs due to an increase in the binding ability of the cell compartments and depends on the phosphorylation status (Pettersson *et al.* 1985b). Aluminium toxicity to *A. cylindrica* was found to be due to intracellular aluminium and not to interactions of the metal with nutrient medium components (Pettersson *et al.* 1986).

Husaini & Rai (1992) showed that aluminium toxicity to the cyanobacterium *Nostoc linckia* increased when pH was lowered from 7.5 to 4.5 and that ATP content was affected by a combination of aluminium and low pH. Moreover, electron transport was inhibited at pH 6.0 or 4.5 when combined with 0.8 and 0.6 mM Al, respectively. It was concluded that aluminium stops ATP synthesis at high pH levels but binds to ATP at acid pHs, hereby rendering it

metabolically unavailable (Husaini & Rai 1992). Toxic aluminium effects in *Sphaeronostoc* sp. cells were detected mainly at the morphological level, causing alterations in cell division patterns (El-Ayouty & Shaaban-Dessouki 1992).

Effects of aluminium on fungi

Soil fungi usually show an optimal pH for growth 1–2 pH units lower than bacteria from the same environment. This difference, which indicates that fungi have a higher tolerance to acidity, is mainly based on a distinct cell wall structure and to the presence of vacuoles in fungal cells (Myrold & Nason 1992). It has been found that mycorrhizal fungi, which establish a symbiotic relationship with certain plants, show a variable aluminium susceptibility but possess a higher aluminium tolerance than *Rhizobium* (Paulus & Bresinsky 1989, Myrold & Nason 1992).

Zel *et al.* (1993) reported that aluminium modifies membrane dynamics in the mycorrhizal fungus *Amanita muscaria* causing a decrease in the proportion of less-ordered membrane domains. This result was opposite to the one obtained in the fungus *Lactarius piperatus*, which coincided with the contrary effects of aluminium on these fungi: inhibiting *A. muscaria* and stimulating *L. piperatus* (Zel *et al.* 1993).

In experiments conducted at pH 4.5, the soil fungus *Neocosmospora vasinfecta* was inhibited by 50 μM Al but, after a lag of several hours, growth initiated in a similar fashion as the untreated control (Oluyedun & vanLoon, 1994). These authors also showed that aluminium was rapidly taken up by the cells of *N. vasinfecta* in an energy-dependent process. Accumulation of aluminium by vacuole polyphosphates has been found in the mycelium of the basidiomycete *Laccaria bicolor* grown with 0.5 mM $AlCl_3$ (Martin *et al.* 1994); even after a 9 day incubation in a medium lacking aluminium, the metal remained in the aluminium–polyphosphate complex.

Similar to what occurs in mammalian cells, aluminium inhibits the activity of glucose-6-phosphate dehydrogenase of *Saccharomyces cerevisiae* (Cho & Joshi 1989); no further studies on aluminium toxicity in yeasts have been reported.

Mechanisms of aluminium tolerance

Explanations of the mechanisms of aluminium tolerance in living organisms include those proposing that some cells are capable of extruding aluminium from cytoplasm or that cells excrete substances that chelate aluminium extracellularly.

The mycorrhizal symbiosis is an important relation for the plants which establish it, since it improves the acquisition of mineral nutrients and may provide the plant with a higher tolerance to toxic metals in polluted soils (Barea 1991). The ability of mycorrhizal fungi to protect plants from aluminium toxicity has been related to changes in the production of radicular exudates (i.e. aluminium-chelating compounds) or the uptake of ions able to precipitate (Pi) or to compete (Ca, Mg, Fe) with soil aluminium (Koslowsky & Boerner 1989).

The fungus *Suillus variegatus* was reported as being highly resistant to aluminium (Jones & Muehlchen 1994), tolerating up to 3.7 mM (Paulus & Bresinsky 1989), although the mechanism of resistance has not been determined. Another species, *S. luteus*, was able to tolerate up to 11 mM Al (Thompson & Medve 1984).

Bacterial resistance to inorganic toxic ions may be determined by genes located in the chromosome or in extrachromosomal elements called plasmids. Functions coded for by plasmids are usually dispensable but confer advantageous properties to host bacteria useful for survival under hostile conditions or increasing the cell's metabolic versatility in certain environments. The study of bacterial resistance to heavy metals (Hg, As, Cd, Cu, Zn) conferred by plasmids has acquired special importance in recent years for its potential use for bioremediation processes as well as for its possible utilization in the recovery of valuable metals (Silver 1994).

Mechanisms of bacterial resistance to inorganic toxic ions have been elucidated, in some cases to the molecular level (Silver & Walderhaugh 1992). Most research on these resistance systems has, however, focused on toxic ions showing a high natural bioavailability, whereas only a few studies have been carried out on bacterial interactions with poorly available ions such as aluminium.

Copper-resistance plant-associated *Pseudomonas* strains, which accumulate copper, were also shown to accumulate aluminium, but not other metals, when induced by copper (Cooksey & Azad 1992); in some of these *Pseudomonas* isolates, aluminium was not removed by treatment of cells with EDTA, suggesting an intracellular location of the metal.

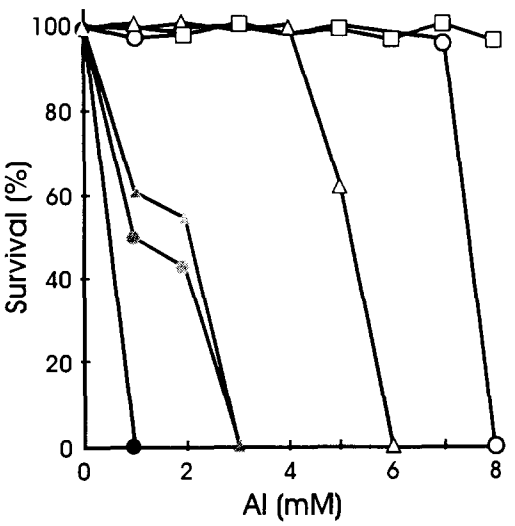

Figure 3. Susceptibility to aluminium by bacteria isolated from heavy-metal polluted zones. Cells were grown in agar plates with the indicated concentrations of aluminium (added as $AlCl_3$) and colony-forming units were recorded after a 24 h incubation at 30°C. Survival represents the percentage of colonies scored at each aluminium concentration as compared to the plates without aluminium (100%). Each symbol refers to a different bacterial isolate. Garcidueñas Piña & Cervantes (unpublished data).

Table 2. Potential mechanisms of bacterial resistance to aluminium.

Mechanisms	Effectors
Extracellular chelation	organic acids, proteins, lipids
Cell wall binding	lipopolysaccharides, peptidoglycan
Membrane binding	phospholipids, proteins
Intracellular chelation	proteins, organic acids
Aluminium extrusion	ATPases, membrane potential
Chemical transformation	reductases, methylases

Appanna *et al.* (1994) reported that an aluminium-tolerant strain of *Pseudomonas fluorescens* specifically sequesters and detoxifies aluminium by producing an extracellular lipid compound. As the complexing lipid residue is rich in phosphorus, a role for Pi in aluminium homeostasis has been proposed for *P. fluorescens* (Appanna & St Pierre 1994). A similar lipid residue was recently found to be produced by both aluminium-sensitive and aluminium-resistant bacterial strains (in preparation), thus disputing its possible role in aluminium tolerance. In a collection of environmental bacteria isolated from heavy-metal polluted zones (Vargas *et al.* 1995), we recently tested aluminium susceptibility; although a high variability in aluminium tolerance was found, we identified aluminium-resistant isolates (Figure 3), which were mostly Gram-negative bacteria (in preparation). The genetic basis for aluminium resistance in these strains is currently under study.

Potential strategies for bacterial resistance to aluminium are shown in Table 2. Similar systems have already been found to function for other toxic inorganic ions and may be of chromosomal or plasmid origin (Silver & Walderhaugh 1992, Cervantes & Gutiérrez-Corona 1994, Cervantes *et al.* 1994).

Concluding remarks

The continuing increase of bioavailable aluminium forms in soils and waters makes it interesting to analyse the impact of aluminium ions on living organisms. Microbial cells may be used as a model system to study aluminium toxic effects as well as aluminium tolerance mechanisms. Understanding the biological interactions of aluminium with microorganisms may be useful for both academic and applied purposes. For example, potential bacterial aluminium resistance genes may be transferred, by genetic engineering techniques, to aluminium-sensitive plants to improve their growth properties in acidic soils (L. Herrera-Estrella, personal communication). Also, as has occurred with other metals (Silver 1994), bioremediation procedures might be developed to alleviate aluminium pollution in specific settings.

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