Aluminium reduces sugar uptake in tobacco cell cultures: a potential cause of inhibited elongation but not of toxicity

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Abstract:

Aluminium is well known to inhibit plant elongation, but the role in this inhibition played by water relations remains unclear. To investigate this, tobacco (Nicotiana tabacum L.) suspension-cultured cells (line SL) was used, treating them with aluminium (50 mM) in a medium containing calcium, sucrose, and MES (pH 5.0). Over an 18 h treatment period, aluminium inhibited the increase in fresh weight almost completely and decreased cellular osmolality and internal soluble sugar content substantially; however, aluminium did not affect the concentrations of major inorganic ions. In aluminium-treated cultures, fresh weight, soluble sugar content, and osmolality decreased over the first 6 h and remained constant thereafter, contrasting with their continued increases in the untreated cultures. The rate of sucrose uptake, measured by radio-tracer, was reduced by approximately 60% within 3 h of treatment. Aluminium also inhibited glucose uptake. In an aluminium-tolerant cell line (ALT301) isogenic to SL, all of the above-mentioned changes in water relations occurred and tolerance emerged only after 6 h and appeared to involve the suppression of reactive oxygen species. Further separating the effects of aluminium on elongation and cell survival, sucrose starvation for 18 h inhibited elongation and caused similar changes in cellular osmolality but stimulated the production of neither reactive oxygen species nor callose and did not cause cell death. We propose that the inhibition of sucrose uptake is a mechanism whereby aluminium inhibits elongation, but does not account for the induction of cell death.

Keywords:

Aluminium toxicity, cell death, elongation, osmotic potential, reactive oxygen species, sugar uptake, water uptake.

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