Abstract

The aluminium (in the form of Al^{3+} ions) toxicity being one of the most important crop growth-limiting factor around the world, this work is a part of the research on the mechanism of the general effect and specifically the toxic impact of the aluminium on the plant cells.

During the course of the previous works, the phosphoinositide signal system has been pointed out as one of the most significant target being affected by this toxicity, with the mechanism itself not yet clarified. Also, a link between the effect of the Al^{3+} on the cellular cytoskeletal system, namely tubulin, and the altered functionality of the phosphoinositide signal system has been suggested, with the suggested cause being the direct interaction of the free cytoplasmatic tubulin and formation of binary or higher complexes with the cellular phosphatidylinositol-phospholipase C (PI-PLC).

For the first part, the effect of the extracellular Al^{3+} in different concentrations and for different durations of exposition was quantified. The correlation between the concentration of the Al^{3+} and the relative inhibition of the PI-PLC was a 42% decrease in the enzyme activity per fifty-fold increase of Al^{3+} in the *in vitro* setting within the range of 20-1000 μM Al^{3+} concentrations. The inhibitory effect became apparent within 15 minutes and reached a fair peak in an hour.

When the PI-PLC (in plasma membrane) interacted directly with the tubulin, within the range of 1-333 nM tubulin concentration the results were unclear and chaotic, only with the concentrations of 1 and 2 μM tubulin a 130% and 150% activation was observed, although with a high error.

In the membranes obtained from the cells treated during lifetime with a range of Al^{3+} concentrations, a fairly linear correlation between the decreasing activity of the PI-PLC and the increasing amount of the tubulin bound to the membranes was found, suggesting a possible link between the two phenomena.