MODULATION OF SP1 AND NFκB BINDING ACTIVITY BY ALUMINUM IN HELA CELLS

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Aluminum is considered as a potentially toxic metal which has been linked to various neurological diseases such as amyotrophic lateral sclerosis, the Parkinsonism dementia complex of Guam and Alzheimer disease.

Although aluminum is not an redox metal, different studies have show that aluminum's cytotoxicity in neurological diseases would be link to an oxydative stress. However, the relationship between aluminum and free radicals still remains unclear.

On the other hand, it is now well established that the DNA binding activity of two transcription factors: NFκB and SP1 is regulated by redox control mechanisms.

The aim of our study was to investigate if aluminum is able to modulate the DNA binding activity of these two transcription factors. By electrophoretic mobility shifft assay we have shown that an 3 hours and 24 hours incubation of HeLa cells with various concentrations of aluminum sulfate led to an activation fo the DNA binding activity of NFκB in a dose dependant way, while DNA SP1 binding activity decrease.

We have also shown, by the same method, that aluminum is able to decrease DNA SP1 binding activity in an cellular system. So, concerning SP1, the date support the hypothesis that the effect of aluminum on this transcription factor may be due to a direct interation of aluminum with the protein SP1.

Further experiments are needed to determine if these effect of aluminum on NFκB and SP1 transcription factors result or not from an involment of this metal in an intracellular oxidative process.