Involvement of nitric oxide in aluminium neurotoxity: effects of L-NAME are protective and dose-dependent

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Undoubtedly, aluminium is a very harmful substance when enters the human body, which happens primarily unintentionally from the environment. When accumulated in the brain, it is involved in severe damages found in chronic neurodegenerative diseases, including Alzheimer's disease. Knowing the pathogenetic mechanisms of these damages could improve prevention/treatment of aluminium-induced neurotoxicity. Since the important role of nitric oxide (NO) in these processes, in our research, just prior to aluminium chloride, a nonselective nitric oxide synthase inhibitor Nω-nitro-L-arginine methyl ester (L-NAME) was applied in the hippocampus of Wistar rats with three doses. Effects of both substances were examined clinically by the active avoidance test and biochemically by measuring cytochrome c oxidase and glucose-6-phosphate dehydrogenase activity in the forebrain cortex, basal forebrain, striatum and hippocampus. It was demonstrated that inhibition of NO synthesis protects animals against aluminium neurotoxicity. That was registered through improved behaviour, or even its reversion, i.e. the decreased number of active avoidance responses induced by aluminium chloride reached the values of control animals by the pre-treatment with L-NAME. Also, aluminium-induced disrupt of glucolysis and mitochondrial oxidative phosprorilation was statistically significantly improved with the highest dose of L-NAME. Neuroprotective effects of L-NAME against aluminium neurotoxicity was shown to be dose-dependent.