ONLINE ISSN : 1880-1404
PRINT ISSN : 0916-717X

Biomedical Research on Trace Elements
Vol. 15 (2004) , No. 3 275-277

[Image PDF (527K)] [References]

Effects of gadolinium and other metal on the neurotoxicity of immortalized hypothalamic neurons induced by zinc

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(Accepted: September 13, 2004)

Abstract:
Zinc is an essential trace element and crucial for numerous biological functions. In contrast, excessive zinc in the synapses plays central roles in the neurodegenerative processes following brain injury such as the transient global ischemia. We have previously demonstrated that zinc caused marked death of immortalized hypothalamic neuronal cells (GT1-7 cells) in a dose- and time-dependent manner. To investigate the molecular mechanism of zinc-induced neurotoxicity of GT1-7 cells, we applied various metal to GT1-7 cells prior to exposure to zinc, and observed the morphological and degenerative changes. Among tested, gadolinium (Gd^{3+}), a widely known channel blocker, significantly blocked zinc-induced cell death in a dose-dependent manner. Aluminum (Al^{3+}) also inhibited zinc neurotoxicity. Our results suggest that functions of Gd^{3+} such as the modification of calcium homeostasis are implicated in the mechanism of zinc-induced neurotoxicity.

Key words: zinc, GT1-7, gadolinium, aluminum, apoptosis, ischemia

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