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Neurotoxicity of Zinc: The Involvement of Calcium Homeostasis and Carnosin

Masahiro Kawahara[1)], Keiko Konoha[1)] and Yutaka Sadakane[1)]

1) Department of Analytical Chemistry, School of Pharmaceutical Sciences, Kyushu University of Health and Welfare

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Abstract:
Zinc is an essential trace element that is abundantly present in the brain. In spite of its importance for normal brain functions, it is widely recognized that excess zinc is neurotoxic. Numerous studies have indicated that zinc is crucial for neuronal injury after transient global ischemia and is linked with the pathogenesis of vascular type of dementia. We have investigated the molecular mechanisms of zinc-induced neurotoxicity in vitro and have explored substances that protect zinc-induced neurotoxicity. Pharmacological evidence based on results of our own and numerous other studies has indicated the significance of Ca^{2+} dyshomeostasis in the mechanism of zinc-induced neuronal injury. The introduction of zinc into neurons is reportedly mediated through several types of Ca^{2+}-permeable channels. Ca^{2+} channel blockers attenuate zinc-induced neurotoxicity. Furthermore, calcium overload attenuates zinc neurotoxicity, and vice versa. In this paper, we review the routes of zinc entry and mechanisms of zinc-induced neuronal death in relation with calcium homeostasis. The possible role of carnosine (β-alanyl histidine), a dipeptide that is present in the brain, as an endogenous protective substance for neuronal injury is also discussed.

Key words: Calcium homeostasis, vascular type of dementia, ischemia, excite toxicity

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