

Zinc Alters Excitatory Amino Acid Neurotoxicity on Cortical Neurons

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Recent studies have suggested that large amounts of free zinc may be coreleased during excitatory synaptic transmission at glutamatergic synapses, and may act postsynaptically to decrease actions mediated by *N*-methyl-D-aspartate (NMDA) receptors, while often increasing neuroexcitation mediated by quisqualate receptors. The present study examined the ability of zinc to alter excitatory amino acid (EAA) neurotoxicity. Murine cortical cell cultures were exposed to EAAs for 5 min in defined solutions, and neuronal cell injury was examined the following day both morphologically and by lactate dehydrogenase assay. Inclusion of 30–500 μ M zinc in the exposure solution produced a zinc concentration-dependent, noncompetitive attenuation of NMDA-induced neuronal injury, with an ED_{50} of about 80 μ M. In contrast, zinc produced the same concentration-dependent potentiation of quisqualate neurotoxicity; and with 500 μ M zinc, a small potentiation of kainate neurotoxicity was suggested. The effect of zinc on the neurotoxicity of the broad-spectrum agonist glutamate was consistent with these effects on specific agonists, as well as with a previous study showing that glutamate neurotoxicity normally depends predominantly on NMDA-receptor activation. Zinc produced a concentration-dependent reduction in glutamate-induced neuronal injury in a fashion similar to that seen with NMDA, but less effectively. In addition, despite this overall protective effect, zinc paradoxically increased the glutamate-induced destruction of nicotinamide adenine dinucleotide phosphate diaphorase (NADPH-d)-containing neurons, a subpopulation that was shown in the preceding paper (Koh and Choi, 1988) to exhibit resistance to NMDA receptor-mediated neurotoxicity, and vulnerability to non-NMDA receptor-mediated neurotoxicity. This selective destruction was not produced by several other NMDA antagonists, including magnesium.

These data indicate that endogenous zinc, at concentrations that may be realized during pathophysiological conditions, could importantly alter the neurotoxicity of endogenous glutamate or related EAAs, reducing NMDA receptor-mediated injury while increasing quisqualate (and perhaps kainate) receptor-mediated injury.

Several investigators have now postulated that the release of glutamate (and related compounds) at central excitatory synapses may commonly be accompanied by the corelease of substantial amounts of free zinc (Zn). Mammalian brain, especially

neocortex, pineal, and hippocampus (Wong and Fritze, 1969; Donaldson et al., 1973; Frederickson et al., 1982), contains considerable amounts of Zn. While Zn is likely a cofactor in myriad metabolic processes (Vallee, 1959), histochemical methods have revealed foci of chelatable Zn (Damscher et al., 1985), specifically in forebrain neuropil (Haug, 1973), and ultrastructural studies have further suggested that much of this Zn is located within certain synaptic vesicles in excitatory synaptic boutons (Ibata and Otsuka, 1969; Perez-Clausell and Damscher, 1985). Release of endogenous Zn to the extracellular space appears to occur spontaneously (Charlton et al., 1985; Perez-Clausell and Damscher, 1986), and has been shown to be increased in a calcium-dependent fashion by either high-potassium or electrical stimulation (Assaf and Chung, 1984; Howell et al., 1984; Anikstejn et al., 1987).

The function of such synaptically released Zn is at present unknown, although some participation in excitatory neurotransmission seems probable. Recently, we reported that Zn could selectively attenuate *N*-methyl-D-aspartate (NMDA) receptor-mediated excitation of cortical neurons, while producing some potentiation of quisqualate receptor-mediated excitation (Peters et al., 1987). On the basis of this finding, we proposed that the corelease of Zn with glutamate at central excitatory synapses may normally serve to modulate the distribution of receptor subtypes activated by glutamate, decreasing the activation of NMDA receptors while increasing the activation of quisqualate receptors.

If indeed Zn is coreleased with synaptically released glutamate, it might not only modify the postsynaptic effects of glutamate during the brief exposures associated with normal synaptic transmission, but it could also importantly modify the neurotoxicity of extracellular glutamate during the prolonged exposures thought to occur in certain disease states (Rothman and Olney, 1987). Glutamate neurotoxicity, as modified by Zn, could be highly relevant to the pathophysiology of neuronal loss in these disease states—in some brain regions, perhaps more directly than glutamate neurotoxicity alone.

We have previously reported that Zn could selectively attenuate NMDA-induced cortical neuronal damage (Peters et al., 1987). The purpose of the present study was to extend that observation, characterizing in greater detail the effect of Zn on EAA neurotoxicity. On the basis of the preceding study of the selective vulnerability of NADPH-d-containing [NADPH-d(+)] neurons (Koh and Choi, 1988), we utilized the fate of this special subpopulation as an assay for distinguishing NMDA receptor-mediated neurotoxicity from non-NMDA receptor-mediated neurotoxicity.

Methods

Methods for cortical cell culture, 5 min exposure to EAAs, NADPH-d histology, and assessment of neuronal cell injury were as described in the preceding paper (Koh and Choi, 1988).

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Christian Drosten



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Amino Acids G. Lubec, Gerald A. Rosenthal, 2012-12-06 There is little wonder in the fact that the investigation of amino acids is of fundamental interest to scientists from so many diversified fields If amino acids were only basic constituents of enzymes as well as structural and other proteins this property alone would elevate them to real scientific importance Added to this role however is their ability to serve as building blocks for the production of many classes of secondary metabolites They can support the biosynthesis of a myriad of natural products including nonprotein amino acids cyanogenic glycosides pharmacologically active alkaloids certain phenols purines and pyrimidines nucleic acids condensed tannins lignins and other metabolites The approximately twenty or so amino and imino acids that comprise proteins are well known less familiar are what is now approaching 600 nonprotein amino acids that have been isolated and characterized from plant fungal or animal sources Investigations of the protein amino acids have proven of outstanding value in enhancing our understanding of a variety of physiological and neurological topics that affect human health and well being Amino acids are used to probe inhibitory and excitatory transmission receptors in the brain They contribute to our understanding of epilepsy development of anti epileptic drugs production of novel γ aminobutyric acid uptake inhibitors and acute and chronic neurodegenerative disorders

Neuroprotective Agents and Cerebral Ischaemia, 1996-11-26 Since 1959 International Review of Neurobiology has been a well known series appealing to neuroscientists clinicians psychologists physiologists and pharmacologists This important serial is now being combined with Neuroscience Perspectives and Methods in Neurosciences This combination results in a series that reaches a wider audience and publishes a greater number of thematic volumes Stroke is the third major cause of death in the western world and recent data provide hope that treatments may soon be available Written by world experts on the mechanisms involved in neurodegeneration Neuroprotective Agents and Cerebral Ischaemia presents an up to date review of current research and developing therapies This book is essential reading for all clinicians and researchers searching for neuroprotective drugs Describes the mechanisms involved in cell death and the biochemical changes occurring during an ischaemic episode Presents the different factors affecting the neurodegenerative process and how compounds acting on these systems may lead to novel neuroprotective agents Reviews in vitro and in vivo models of stroke and the activity of putative neuroprotective drugs Discusses the application of preclinical data to formulate approaches to a clinical problem Provides detailed reviews of both completed clinical trials and those currently underway

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