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STUDY ON TRACE ELEMENTS IN HUMAN BRAIN SAMPLES

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Analyses of human brain tissues are becoming of great interest in the medical field since trace elements may influence in the cognitive functions as they are involved in metabolic processes and redox reactions. Besides, essential elements are required in the brain for development and maintenance of the central nervous system, and to play an important role in neurodegenerative disorders. The aim of this study was to investigate on trace elements in two regions (hippocampus and medium frontal cortex) of human brains from normal individuals, selected according to Clinical Dementia Rating score. This research was approved by the Ethic Committee and brain samples from 32 normal individuals (17 females and 15 males) aged 51—95 years were provided from the Brain Bank of the Brazilian Aging Study Group of São Paulo University, Medical School. The brain tissues were cut using a titanium knife, ground, freeze-dried and then analyzed by neutron activation analysis. Samples and element standards were irradiated at the IEA-R1 nuclear reactor under a thermal neutron flux for Br, Fe, K, Na, Rb, Se and Zn determinations. Student's t test ($p = 0.05$) was applied for comparison of results. Results indicated higher concentrations of Fe, Se and Zn in frontal cortexthan those found in hippocampus. No significant difference was found between the genders for frontal cortex tissues. However, the males presented higher Zn concentrations (69 ± 8 mg/kg) in hippocampus than those presented by females (62 ± 6 mg/kg). Comparative study based on two different age groups of individuals indicated that the element concentrations of hippocampus region from group aged 51 to 75 years showed significant difference for Fe from those for the group of 76 to 95 years. Most of our results agreed with the literature ones. It is our intention to extend the study to Alzheimer disease human brains. Biological certified reference materials were also analyzed for quality of the analytical results.

ZINC, A NOVEL IONIC MEDIATOR OF NEURONAL INJURY

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Glutamate is the most widespread neurotransmitter in the brain and when released in excessive amounts like during ischemia, epilepsy or Alzheimer's disease is potently neurotoxic. In the last thirty years many molecular mechanisms linked to glutamate dependent neurotoxicity, also called excitotoxicity, have been clarified. Key factors involved in the excitotoxic cascade are loss of intracellular homeostasis for ions such as Ca^{2+}, Na^{+}, and K^{+}, alterations in mitochondrial function, and generation of reactive oxygen species (ROS). However, recent findings indicate a more complex scenario. In recent years, a new ionic mediator of excitotoxicity has been proposed: Zn^{2+}. Zn^{2+} is present at synapses of many glutamatergic neurons and released during ischemia, brain trauma and epilepsy. Zn^{2+} enters neurons through three different routes (NMDA receptors, voltage sensitive $Ca2^{+}$ channels and Ca^{2+}-permeable AMPA receptors (Ca-ARs) in response to glutamate receptor activation. Among all the entry routes, Zn^{2+} preferentially fluxes through «Ca-ARs», and rapid (micromolar) Zn^{2+} accumulation causes mitochondria to undergo prolonged and irreversible alterations in their function with consequent increases in ROS production. Interestingly, even lower $[Zn^{2+}]_i$ rises potently activate mitochondrial cell death signaling pathways, triggering, with far greater potency than Ca^{2+}, induction of the mitochondrial permeability transition (mPT), mitochondrial swelling, and release of the mitochondrially sequestered pro-apoptotic factors, cytochrome C and apoptosis inducing factor. Moreover, recent findings indicate that Zn^{2+} is also a potent activator of autophagy. Finally, a major pathogenic role for Zn^{2+} has been suggested in the neurodegeneration associated with Alzheimer's disease (AD) as the cation is a key component of amyloid plaques and the cerebral amyloid angiopathy observed in AD. This lecture will describe a novel blueprint of Zn^{2+}-dependent death signaling pathways involved in the neuronal loss associated with major neurological conditions.