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Treating mitochondrial and memory impairments in aging and Alzheimer's disease

Alzheimer's disease (AD) is a progressive age-related neurodegenerative disease; where neuropathologic hallmarks include the deposition of amyloid beta (Aβ) plaques and the formation of neurofibrillary tangles (NFTs). However, investigators now consider AD to be a multifactorial condition given the increasing number of factors implicated in AD, where aging is the greatest risk factor. Interestingly, the earliest deficits in aging and in AD seem to be caused by impaired mitochondrial function and are associated with impairments in memory formation, which are manifested before the appearance of Aβ plaques and NFTs. In fact, current evidence suggests that there are several mechanisms that can affect mitochondrial function, which are linked to aging and AD.

However, recent data show that the nutritional supplement creatine (Cr) alleviates mitochondrial deficits and prevents hippocampal impairments in AD. In addition, recent data suggest that by stimulating the transcription factor, nuclear factor kappa B (NF κB) in neurons that mitochondrial function and memory function can be enhanced by molecular mechanisms involving both Cr and NF-κB signaling. To test these ideas, the author utilized experimental paradigms of neural changes associated with in vivo behavioral memory, in vitro methods involving long-term potentiation (LTP) and long-term depression (LTD), and other cell and molecular methods too. These methods were used in several transgenic mouse models using mice of various ages. In brief, it was observed that freshly isolated hippocampal mitochondria from mice lacking the p50 subunit of NF-κB resulted in deficits of mitochondrial function. It was also found that mitochondrial deficits existed in our transgenic mouse models of AD. Importantly, it was found that Cr treatment reversed NF-κB-induced deficits and other induced deficits in memory and mitochondrial function. It was suspected that these improvements in function occurred due to the stimulation of NF-κB neuroprotective pathways by Cr.

These data suggest critical roles for Cr/NF-κB signaling in memory and suggest that the regulation of cellular energy homeostasis may be one potential mechanism that becomes dysfunctional as we age and in AD. The data also suggest that the use of Cr might provide resilience to aging and may prevent AD by enhancing mitochondrial function in key areas of the brain involved in memory processing.

Biography

Albensi's background is diverse where he has received training in both basic and clinical research. He received a PhD in Neuroscience from the University of Utah's Medical School in 1995, where he developed novel MRI methods for characterizing neonatal hypoxic-ischemic injury. Subsequently, he was awarded a Postdoctoral Fellowship at the prestigious Georgetown University in Washington, DC, USA where he further developed novel MRI methods for investigating TBI and brain cognition. Following this, he went on to work as a Postdoctoral Scholar at the Sanders-Brown Center on Aging – University of Kentucky. He was appointed as Project Staff in the Department of Neurological Surgery – Cleveland Clinic Foundation and also as an Adjunct Assistant Professor of Biology at Case Western Reserve University in Ohio, where he conducted novel work on mechanisms of deep brain stimulation. He now has several appointments in Canada, which include serving as an Associate Professor of Pharmacology and Therapeutics at the University of Manitoba and as a Principal Investigator at St. Boniface Hospital Research. He is also an adjunct professor of Electrical & Computer Engineering at the University of Manitoba and the Everett Endowment Fund Chair. He has reviewed grants for numerous foundations worldwide including NIH, CIHR, NSERC, US DOD, FASEB, to name a few. He is also currently a member of the Board of Directors for the Alzheimer's Society of Manitoba.

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