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Effects of Physical Activity on Brain Energy Biomarkers in Alzheimer's Disease

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Abstract

The prevalence of dementia has substantially increased worldwide. Currently, there is no cure for dementia or Alzheimer's disease (AD), and care for affected patients is financially and psychologically costly. Of late, more attention has been given to preventive interventions—in particular, physical activity/exercise. In this review, examine the risk factors associated with AD and the effects physical activity may play in the prevention of the degenerative process of this disease, loss of memory and cognitive performance in the elderly. To date, research has shown that physical activity, especially aerobic exercise, has a protective effect on cognitive function and memory in the elderly and Alzheimer's patients. In comparison with aerobic exercise, several strength training studies have also shown positive effects, and the rare studies that compare the two different modalities show no difference.

1. Introduction

Alzheimer's disease (AD) is the most common cause of dementia and is an age-related neurodegenerative disease which leads to the loss of memory and learning in mid-to-late life [1].Globally, over 50 million people have dementia, Alzheimer's disease being the most common form and responsible for 60–70% of cases (WHO) [2]. Currently, about ten percent of the population over 65 years, more than 25 million people worldwide, are affected by this disease.

Sharp memory decline, brain atrophy, senile plaques (SPs, or neuritic plaque), and neurofibrillary tangles (NFTs) [3]. Senile plaques and NTFs consist of aberrant cumulative extracellular amyloid β-protein (Aβ) and the hyper-phosphorylation of intracellular microtubule-associated protein tau [4]. The most affected areas in the brains of AD patients include the olfactory bulb, neocortex, and hippocampus, [3] which plays a leading role in spatial memory and learning [5].

2. Risk Factors for Alzheimer's Disease

Alzheimer's disease is a complex disorder, which may be due to a combination of genetic, biochemical and environmental factors. The biggest risk factor for AD, age, is unmodifiable but the disease is not a normal part of the aging process.

3. Impaired Brain Creatine Kinase Activity and Cerebral Glucose Metabolism

Lowered levels of brain creatine kinase activity and reduced cerebral PCr levels are associated with AD [35] and could be considered a risk factor in diagnosis. Creatine kinase (CK) activity is vital for the energy reaction of every cell in the human body as a spatial energy shuttle and energy sensor [35] and is thus paramount in bioenergetics of the brain [35].

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The cerebral form of CK is known as cytosolic brain-type creatine kinase (BB-CK). AD patients manifest high levels of cytosolic brain-type creatine kinase (BB-CK) oxidative damage [35,36]. BB-CK activity in AD patients is reduced by to up to 86%, coinciding with a 14% reduction in CK Protein expression [36] Reduced BB-CK activity in AD results in decreased ATP stores in neural cells and synapses [35,36] BB-CK and the simultaneous expression of ubiquitous mitochondrial creatine kinase activity (uMtCK) is reduced in AD patients via high levels of oxidation, manifesting in BB-CK enzyme dysfunction and the progression of AD [35]. AD patients have mitochondrial DNA mutations manifesting in the dysfunctional cerebral bioenergetics of the brain [35] in essence, BB-CK activity is paramount in neuronal energetics, the facilitation of synaptic glutamate uptake and, ultimately, neurotransmitter uptake [35].

Another cerebral bioenergetic risk factor for AD is reduced cerebral glucose metabolism and mitochondrial DNA mutation [35,36].AD-associated mitochondrial DNA mutations result in perturbed energy metabolism in the brain and impaired central nervous system (CNS) function. Reduced acetyl-CoA production, cortical acetylcholine esterase activity, and oxidative phosphorylation are important risk factors in the onset of AD-associated senile dementia [35].

4. The Effect of Physical Activity on Alzheimer's Disease

Evidence from both human and animal studies suggests that physical activity, especially exercise that increases cardiorespiratory fitness, facilitates the neuroplasticity of certain brain structures related to cognitive function.

Keywords: Alzheimer's, physical activity, prevention and memory.

Biography

Navin H. Khan is the sports nutritionist, exercise physiologist, and Chief Scientific Officer for Immune Whey LLC.He has conducted over 1000 VO2 max and resting metabolic assesements. He is known for Determining client-specific nutrient and energy requirements, with consideration to specific lifestyles, physiology and medical concerns of all his clients. His research areas focus on the effect of dietary supplements on brain health and exercise performance. His passion is researching, innovating, and pushing the boundaries of human performance, sports nutrition, and exercise physiology. His career is dedicated to educating and motivating others to be optimal in their approach to naturopathic nutrition, training, and supplementation.

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