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Review

Neurophysiological and epigenetic effects of physical exercise on the aging process

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ABSTRACT

Aging is a gradual process during which molecular and cellular processes deteriorate progressively, often leading to such pathological conditions as vascular and metabolic disorders and cognitive decline. Although the mechanisms of aging are not yet fully understood, inflammation, oxidative damage, mitochondrial dysfunction, functional alterations in specific neuronal circuits and a restricted degree of apoptosis are involved. Physical exercise improves the efficiency of the capillary system and increases the oxygen supply to the brain, thus enhancing metabolic activity and oxygen intake in neurons, and increases neurotrophin levels and resistance to stress. Regular exercise and an active lifestyle during adulthood have been associated with reduced risk and protective effects for mild cognitive impairment and Alzheimer's disease. Similarly, studies in animal models show that physical activity has positive physiological and cognitive effects that correlate with changes in transcriptional profiles. According to numerous studies, epigenetic events that include changes in DNA methylation patterns, histone modification and alterations in microRNA profiles seem to be a signature of aging. Hence, insight into the epigenetic mechanisms involved in the aging process and their modulation through lifestyle interventions such as physical exercise might open new avenues for the development of preventive and therapeutic strategies to treat aging-related diseases.

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1. The aging process

The accumulation of molecular and cellular damage throughout life leads to a wide range of age-related pathological conditions such as vascular disorders, sarcopenia, osteoporosis, liver dysfunction, and cognitive decline. Loss of protein and bone mass and a concomitant increase in fat mass occur during aging, which results in a growing incidence of disorders such as inflammatory diseases, dyslipidemia, atherosclerosis, obesity, insulin resistance and type 2 diabetes mellitus. In humans such disorders are risk factors for cardiovascular disease or stroke in the later stages of life. In addition, some of these factors, e.g. dyslipidemia, are associated with increased incidence of neurodegenerative diseases characterized by deterioration of neurons and glial cells, the core components of the nervous system and frequently lead to cognitive decline and senile dementia (Deary et al., 2009; McNeill et al., 2006). Remarkably, two studies performed with 1624 Latino (Yaffe et al., 2007) and 2632 elders of African and Caucasian backgrounds (Yaffe et al., 2004) have reported that metabolic syndrome may be predictive of cognitive decline in a 3- and 5-year follow-up, respectively. These data suggest that factors associated with a lifestyle that reduces metabolic and cardiovascular risks might also diminish the risk for developing cognitive decline or Alzheimer's disease (AD).

Only a few decades ago, when life expectancy was shorter, neurodegenerative disorders were rarely manifested as the percentage of the population reaching ages commonly associated with central nervous system damage, progressive loss of personal independence and eventual disability, was much lower than nowadays. Therefore, understanding the mechanisms underlying aging-related alterations in brain structure and function has become critical for the identification of new therapeutic targets and the development of multimodal health-care strategies that meet the needs of an aging population. In this context, here we explore recent findings that give insight into the molecular and neurophysiological mechanisms elicited by physical exercise from the perspective of the aging process.

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