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FNDC5/Irisin Pathway Correlation with Exercise and Neurodegenerative Disease

Ecler Jaqua*[1] and Jade Deschamps[2]

[1]*Loma Linda University Health – Family Medicine Department - Geriatric Medicine Division.*

[2]*Loma Linda University Health School of Medicine*

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***Corresponding author**: Ecler Jaqua, Loma Linda University Health – Family Medicine Department - Geriatric Medicine Division

1200 California Street, Suite 240, Redlands, CA 92374

Abstract

Many studies have shown that exercise boosts cognitive function and overall brain health, and thus is significant in slowing the onset of dementia. Neurodegenerative disease includes a realm of chronic disorders characterized by memory problems, impaired reasoning, and personality changes. In the past decade, there has been a growing body of research revealing the relationship between exercise and increased expression of the FNDC5/Irisin pathway, which appears to regulate BDNF, a key promoter of axonal regeneration. As the prevalence of Alzheimer's and neurogenerative disease rises in the United States' aging population, the positive relationship between exercise and FNDC5/Irisin deserves further study

Keywords: Neurodegenerative disease; Alzheimer's disease; Dementia; Irisin myokine; Memory loss.

Background

As we all know, physical exercise has many benefits to physical, emotional, social health and has been extensively studied and recommended for health promotion to all patients, especially older adults. Countless studies show that individuals who practice regular exercise have a lower risk of developing any type of neurocognitive impairment such as Alzheimer's disease (AD) [1,2]. Physical activity helps to increase brain chemicals that may protect the brain against memory loss and increase brain neuronal connections that are lost or diminished with aging [1,2]. Being physically active also helps prevent and improve many modifiable risk factors for neurocognitive impairment, including cardiovascular disease, diabetes mellitus type 2, metabolic syndrome, and hyperlipidemia [2].

Neurodegenerative Diseases

Alzheimer's disease is the most prevalent age-related neurodegenerative disorder, followed by Lewy body dementia, vascular and frontotemporal dementia, respectively [3]. AD is a progressive disease where cognitive abilities and functional status slowly worsen over many years; it causes progressive brain atrophy and memory loss leading to dementia, disability, and death. The most significant well-known risk factor for Alzheimer's disease is old age, with most of the cases affecting individuals 65 years and older [1-3]. Memory formation relies on the strengthening or weakening synaptic connections in response to changes in neuronal activity [1-3]. In AD brains, toxic proteins like beta-amyloid plaques and tau tangles impairs synaptic plasticity, preventing brain cells from communicating and, as a result, producing memory loss and other cognitive deficits [3].

Physical Activity and FNDC5/Irisin Pathway

Irisin is an exercise-induced myokine, a cleavage protein of fibronectin type III domain-containing protein 5 (FNDC5) first described by Bostrom et al. in 2012 [4]. Studies showed increased levels of irisin in the skeletal muscle after prolonged endurance exercise on both humans and mice [4,5]. Epidemiologic studies have shown exercise to help prevent or slow the progression of AD and other associated dementias. A possible mechanism is that the FNDC5/Irisin pathway is induced by physical activity and enters the brain, triggering the signaling cascade to change neuronal function [4-6]. Irisin also was described as a myokine that may cause "browning" of the white adipose tissue, which is subcuta neous fat-containing iron-rich mitochondria that promote fat metabolism, increase thermogenesis, and energy expenditure [5,6].

FNDC5/Irisin and Neurodegenerative Diseases

FNDC5/Irisin is an exercise-induced myokine that mediates the benefit of exercise in Alzheimer's disease models by regulating neurogenesis, neurobehavior, neuronal metabolism,

and enhancing synaptic plasticity and memory [6,7] Increased FNDC5/Irisin levels may induce brain-derived neurotrophic factor (BDNF) in the hippocampus, which consequently promotes neuronal cell survival, synaptic integrity, special memory, and is vital to learning development [6-8]. A few studies showed that the FNDC5/irisin levels are decreased in the hippocampus and cerebrospinal fluid of humans and mouse models with AD. Analysis revealed that increasing irisin in the brain of AD mice improves synaptic plasticity and memory [5,7,8]. Irisin reduces the expression of synapse-related genes that are induced by amyloid-β (Aβ) peptides and reactivates the AD-linked translational repression in hippocampus neurons [6-8].

Conclusion

AD is a neurodegenerative disease that predominantly affects memory, and unfortunately, there is no cure. Physical activity may prevent cognitive decline by increasing the expression of FNDC5/Irisin in the hippocampus [6-8]. Exercise-induced irisin protects the nervous system by promoting neurogenesis and suppressing Aβ accumulation [7,8]. Of all lifestyle changes, exercise has shown to be a very effective approach to decrease the risk of neurocognitive impairment. Start exercising to prevent memory loss.

References

1. Alzheimer's disease: Can exercise prevent memory loss? Mayo Clinic.
2. Benefits of Physical Activity (2021) Centers for Disease Control and Prevention.
3. What is Alzheimer's? Alzheimer's Disease and Dementia.
4. Wrann CD (2015) FNDC5/Irisin – Their Role in the Nervous System and as a Mediator for Beneficial Effects of Exercise on the Brain. Brain Plasticity 1(1): 55-61.
5. Jin Y, Sumsuzzman D, Choi J, Kang H, Lee SR, et al. (2018) Molecular and Functional Interaction of the Myokine Irisin with Physical Exercise and Alzheimer's Disease. Molecules 23(12): 3229.
6. Wrann CD, White JP, Salogiannnis J, Bogoslavski DL, Wu J, et al. (2013) Exercise Induces Hippocampal BDNF through a PGC-1α/FNDC5 Pathway. Cell Metabolism 18(5): 649-659.
7. Lourenco MV, Frozza RL, de Freitas GB, Zhang H, Kincheski GC, et al. (2019) Exercise-linked FNDC5/irisin rescues synaptic plasticity and memory defects in Alzheimer's models. Nature Medicine 25(1): 165-175.
8. Pignataro P, Dicarlo M, Zerlotin R, Zecca C, Dell Abate MT, et al. (2021) FNDC5/Irisin System in Neuroinflammation and Neurodegenerative Diseases: Update and Novel Perspective. International Journal of Molecular Sciences 22(4): 1605.

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