Abstract

Alzheimer disease is a progressive neuro-degenerative disorder and the leading cause of dementia in older adults. Growing evidence suggests that regular physical exercise may help mitigate cognitive decline and delay the onset of Alzheimer-related symptoms. Clinical studies and research findings support the promotion of structured physical activity as a promising, non-pharmacological strategy for maintaining cognitive health and potentially lowering the risk of Alzheimer's disease.

Background

Alzheimer's dementia (AD) is a degenerative disease of the central nervous system characterized by progressive cognitive, memory, behavioral, and spatial impairments (Zhao, 2020). It mainly shows a decline in cognitive ability, behavioral disorder, and a gradual decline in activities of daily living (ADL) in the clinic (Yu et al., 2006). Among them, the decline in ADL is mainly concentrated in two aspects: the decline of instrumental activities of daily living (IADL) and basic activity of daily living (BADL).

Study suggests that the typical histopathology of AD includes the β-amyloid (Aβ) waterfall theory, tau protein theory, and neurovascular hypothesis (Fu and Shao, 2010). With the aging population, dementia has become one of the most serious health problems for the public and one of the main challenges for the health care system.

A decline in instrumental ADL generally occurs in the early stages of AD. With the deterioration of the disease, patients will not be able to independently carry out basic daily activities, Liu-Seifert et al. (2016) which is the key factor leading to the loss of independence in patients with AD. Many types of dementia exist, including AD, vascular dementia, Lewy body dementia, Parkinson's disease, mixed dementia, and frontotemporal dementia (Alzheimer's Association, 2018).

The fact that current treatments aren't doing enough to slow it down has pushed researchers to look at other options, like lifestyle changes. Exercise, in particular, is being looked at for its potential to protect our brains. There's a good reason to believe that staying active could postpone memory issues by improving how our brains work and tackling some of the risks tied to Alzheimer.

The American College of Sports Medicine et al. (2009) stated that exercise can treat potential cognitive and motor function deterioration (Forbes et al., 2014). Some studies have also found that

exercise may increase the release of protective agents such as neurotrophic factors, and affect the nervous system to improve cognitive ability (Tonoli et al., 2015).

Physical exercise can improve functional degradation, reduce depression and anxiety, the risk of falls, and improve the patients' independence and balance ability (Arcoverde et al., 2008).

Methodology

To comprehend the effects of exercise on individuals with Alzheimer's we must employ multiple research methods in order. The data source includes PubMed, frontier journals, science direct journals and the Journal of the Alzheimer's Association. The search terms used include "Alzheimer" or "Alzheimer's disease" or "AD"" or "dementia" and "exercise" or "physical exercise" or "aerobic exercise" or "resistance training" and "cognitive function".

Methodology to demonstrate the effectiveness of exercise interventions is through randomized controlled trials (RCTs), and comprehensive summaries of the evidence are provided by systematic reviews and meta-analyses. Real-world observational studies show how exercise affects people over the long term. Animal models help us understand the biological mechanisms at work, and neuroimaging studies can show how exercise changes the brain.

Results

Meta-analysis and systematic review indicated that exercise intervention might improve the cognitive function of AD or slow down the decline of cognition. Aerobic exercise enhanced cognitive function. Maintaining the integrity of mitochondria is an essential factor in promoting the practical function of cells. Mitochondrial dysfunction, defects in mitochondrial dynamics and impaired mitophagy can lead to increased oxidative stress, synaptic dysfunction, neuronal loss and cognitive impairment, thereby inducing the development and progression of AD. As an effective non-drug interventional strategy, exercise is beneficial to alleviate or delay the occurrence and development of AD. Exercise can enhance the efficiency of mitochondrial dynamics and activate mitophagy, thereby reconstructing the optimal mitochondrial network, improving the plasticity of nerves, and providing practical strategies for healthy aging and the prevention of AD.

Discussion

Alzheimer disease is a multidimensional disease, which requires a large number of experiments to evaluate the results. The number of studies included in this review was limited, therefore, it is difficult to evaluate the long-term efficacy of exercise intervention.

15 RCT studies were included in the meta-analysis. The results showed that physical exercise had a positive effect on the improvement of ADL in patients with AD [SMD = 0.312, 95% CI (0.039–0.585), $P = 0.02$] and had a better effect on the improvement of BADL [SMD = 0.564,95% CI (−0.186 to 1.134]. The degree of benefit depends on the type of physical exercise, exercise cycle, and exercise duration.

Brain derived neurotrophic factor (BDNF) is a neurotrophin that promotes the survival of neurons and is essential for regulating memory function (Loprinzi and Frith, 2019). It has been proved that the impact of exercise on brain plasticity is associated with the action of brain-derived neurotrophic factor (de Melo Coelho et al., 2014). Exercise can increase the concentration of brain-derived neurotrophic factor, promote nerve regeneration, improve nervous system, so as to improve the brain structure involved in cognition (Pereira et al., 2007; Gomez-Pinilla et al., 2011; Liu et al., 2011), reduce the degree of dementia in patients with AD and further improve their ability of daily living.

Exercise can increase the volume of gray matter and white matter in the prefrontal and temporal cortex of the hippocampus, enhance hippocampal neurogenesis, reduce the aggregation of tau protein, and improve the ability of daily living in patients with AD (Ahlskog et al., 2011).

Chronic inflammation and oxidative damage are important causes of AD. Studies have found that moderate intensity exercise can promote the reduction of NLRP3 inflammation (Khakroo Abkenar et al., 2019), improve mitochondrial function, promote redox balance, and slow down the development of AD (Bernardo et al., 2016).

Studies have shown that exercise can improve cerebral blood flow in the frontal and temporal lobes, increase the oxygen supply, and improve cerebral circulation (Lista and Sorrentino, 2010).

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Exercise creates an anti-inflammatory environment in the peripheral organs and brain, and by modulating neuroinflammation, it reduces cellular and cognitive damage in AD (Kelly, 2018).

Exercise can improve the brain function by increasing mitochondrial biogenesis, improving mitochondrial metabolic function and reducing neuronal Aβ deposition via the AMPK/SIRT1/PGC-1α signaling pathway in the brain of AD subjects. Study has demonstrated exercise training can improve mitochondrial function in the brain by improving the function of ETC and promoting a shift in the mitochondrial fission-fusion balance toward fission (increased Drp1) in aged mice (Gusdon et al., 2017). Mitophagy can selectively reduce the membrane potential and degrade mitochondrial progeny that exceeds their repairing capability. Exercise-induced autophagy caused by treadmill running can improve the learning and memory capacity, which is first discovered in the cerebral cortex of normal mice in 2012 (He et al., 2012).

To summarize, physical exercise has a positive effect on the ability to perform daily living in patients with AD. For patients with mild to moderate AD, 12–16 weeks of a moderate cycle and 30–45 min of anaerobic exercise, such as strength and balance training, are ideal for the improvement of basic daily living ability. Further studies are needed to investigate the specific details of exercise interventions and formulate a more accurate and effective exercise plan for patients with AD

Conclusion

Regular exercise is one of the best things you can do for your brain health. It lowers your risk of cognitive decline and may even delay the start or slow the progression of Alzheimer's disease. This method is a simple and safe way to keep our brains healthy and improve our overall health as we get older. A growing body of scientific research indicates that regular exercise is a powerful, non-pharmacological way to prevent cognitive decline and reduce the risk of Alzheimer's disease. Exercises that are aerobic and resistance-based have been shown to enhance neuroplasticity, cerebral blood flow, neuroinflammation, and the release of neurotrophic factors such as BDNF. These improvements have been shown to enhance memory, executive function, and overall brain health. More research, including long-term randomized controlled trials (RCTs), is needed to establish a clear causal link between exercise and dementia prevention.

References

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