The Effect of Weight Training on Brain Function

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Abstract

The benefits of weight training on physical health are widely known, but this can also affect the structure and function of the brain. From small-scale studies done on rats, monkeys, and humans, there have been results showing lower levels of atrophy of cortical white matter, an increase in grey matter density in the cerebellum, an increase in neural plasticity, and an increase in hippocampal volume BDNF expression - all of which result in higher cognitive function. In rats, this resulted in those with mild cognitive impairment to perform even better on a cognitive test than those without any impairment. Further research could strengthen the preventative potential (in some cases, interventional potential) of resistance training for dementia, depression, and other neurodegenerative diseases.

Resistance training has been emphasized as part of healthy living for many years not only for its bodily benefits, but also prevention of disease and mood enhancement. However, the exact neurobiology and mechanics that take place which lead to these benefits, or any relationship between muscular strength and the brain, have not been explained as extensively. Studies thus far have unanimously shown that weight lifting has a positive effect on both cognitive function and memory through the preservation of white matter, the increase in the density of grey matter, an increased ability to make new neural connections, and an increase in hippocampal volume. Further research could also point towards weight training as a preventative method against aging of the brain, neurodegenerative disorders, and mood disorders such as depression.

Because the brain ages with the person, an outward presentation of forgetfulness, slowed processing of information, or reduced attention capacity are all occurring as we grow older (Filley, 2005). Inwardly, this aging is the result of the loss of white matter - the area in the brain consisting of axons, wrapped in a fatty layer of insulation called myelin. Myelin speeds up signal transmission, so a myelinated axon will carry information much quicker than an unmyelinated axon. In other words, decreased myelination will result in lower cognitive performance and can lead to neurodegenerative disorders and dementia. Studies have shown that resistance training specifically can slow the atrophy of this white matter as the brain ages. One study looked at older adults, in which one group was assigned resistance training and the other was assigned balance and toning (Herold, 2019). After fifty two weeks, the group that performed resistance exercises had a lower level of white matter atrophy and lesions in comparison to the group that performed toning exercises. This suggests that the repetitive motor movement requiring maximal force is more effective in slowing aging of the brain and induces different changes in the brain in comparison to other forms of exercise. In addition, older adults with mild cognitive impairment that performed resistance training twice a week for 26 weeks exhibited increased cortical thickness of grey matter in the posterior cingulate gyrus and better cognitive performance (Herold, 2019). These results are significant as a form of late intervention for MCI and the diseases associated with it, especially in the older population and over a period of only 6 months. Mild cognitive impairment (MCI) is associated with aging and presents itself through memory problems. MCI is also a marker for Alzheimer's, a disease characterized by the decline of grey matter. The results in this study of improved cognitive performance and an increase in grey matter show promise in the prevention of neurodegenerative diseases such as Alzheimer's just through resistance training.

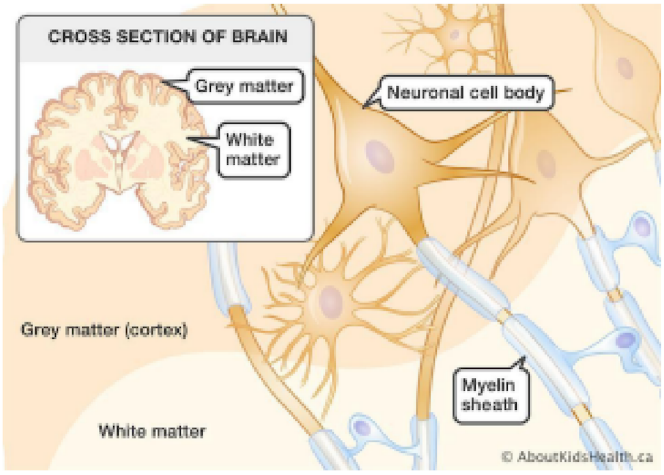

Figure 1: A schematic of the white and grey matter in the brain, composed of partly axons wrapped in myelin and neuronal cell bodies respectively.

Prevention is only possible in the time before diagnosis; after diagnosis, it's often much more difficult to slow, stop, or show improvements from many neurological diseases such as forms of dementia. However, one study has shown promise for improved cognitive function in patients with dementia through resistance training (Kelty, 2019). A group of rats were given an injection to induce inflammation in their brains, similar to dementia in the human brain. Half of those rats began a form of strength training with a progressive increase of weights. When placed into a maze as a cognitive test, the rats with the inflammation performed significantly worse than the rats without the inflammation. However, after six weeks, the rats with the inflammation on the strength training regime

improved and performed equal or better than the rats without the inflammation. In the brain tissues of these rats, there were increased signaling proteins and genetic markers like IGF-1, which is an insulin-like growth factor. IGF-1 acts as a signaling molecule to activate the conical protein kinase B and ERK1/2 pathways which leads to increased signaling of the downstream protein, AKT. Resistance training induced IGF-1 activation of AKT signaling is associated with increased neuronal proliferation and survival. This indicates the creation of new neurons and greater persistence of the neurons, suggesting that the resistance training allowed the rats' brains to remodel themselves and make new connections - significantly increasing cognitive function. The creation of new neurons and new learning connections exemplifies the increased neural and synaptic plasticity of the brain, which shows promise in reversing the effects of the inflammation. Neuroplasticity is the brain's ability to form or reorganize neural connections and therefore learn new skills or store memories. In this case, it may allow the brain to relearn previously forgotten information in early-stage Alzheimer's disease (Hill, 2011). Although neurodegenerative changes like hippocampal atrophy occur in this early stage, cognitive plasticity can still be maintained; however, the neurobiological mechanisms of these plasticity-related events are unclear at this time. With further research, this can be paralleled in humans with hopes of possibly reversing the effects or improving cognitive function in various forms of dementia.

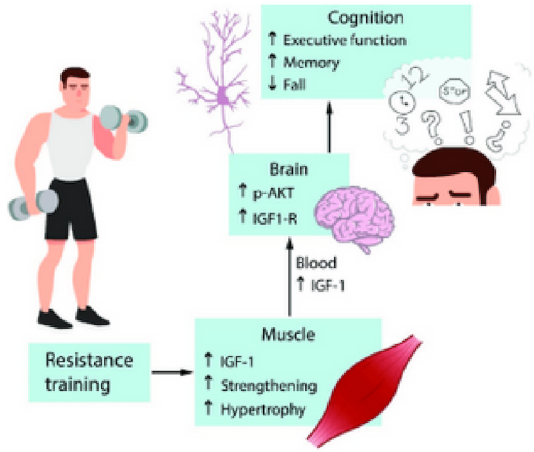

Figure 2: Resistance training can induce IGF-1 production, which acts as a signaling molecule to activate the conical protein kinase B and ERK1/2 pathways. This leads to increased signaling of the downstream protein, AKT, which is associated with greater neuronal proliferation and survival.

Thus far, it's been shown that resistance training is far more effective in preserving both white and grey matter and improving cognitive function than other forms of exercise. However, there is some ambiguity in what variable or mechanism associated with resistance training is responsible for this neural relationship. One study, done on female macaque monkeys, suggests that lifting weights results in changes in the brain before there is any increase in strength or muscle mass. From 50 trials of the monkeys pulling a weighted handle with progressive overload over a period of 9 weeks, it was concluded that the monkeys' brains experienced neural adaptations at the cortical level weeks before their muscles showed signs of hypertrophy (Glover, 2020). This suggests that the effects of strength training on brain function may not come directly from the muscle mass gained, but from the physical training regime itself. Another study supports the theory that these neural adaptations are associated with muscular strength: a group of young, healthy adult humans were studied, in which half of them followed a strength training regime. After four weeks, the individuals who gained the most strength, as measured by the increase in their maximum voluntary isometric contraction, also showed larger increases in white matter (Palmer, 2013). Because the participants' strength and white matter density showed correlation, it can be concluded that the intensity, consistency, or the duration of exercise is correlated with neural changes, since muscular strength can be a confounding variable (which also has a positive correlation with the intensity and consistency of the weight training).

The improvement in cognitive function and prevention of neurodegenerative diseases from physical exercise is partially due to the hippocampus. The hippocampus is a brain structure that resides in the medial temporal lobe and is responsible for important functions such as learning and memory, but is also highly susceptible to damage by an array of stimuli such as stress and aging. It is also part of the limbic system, which regulates emotion and motivation. The atrophy of the hippocampus is associated with many conditions such as Alzheimer's disease, major depressive disorder, schizophrenia, PTSD, and epilepsy. Fortunately, its atrophy is preventable through strength training, largely because of its induction of neurogenesis. One study done on 120 older adults over one year found that hippocampal volume increased by 2% (compared to the 1-2% annual shrinkage of the hippocampus) and increased hippocampal blood flow.

It was also found that BDNF, a neurotrophic factor, could be the link between exercise and increased hippocampal volume (Erickson, 2011). Exercise upregulates BDNF gene expression and therefore increases BDNF serum concentration, which also promotes synaptic plasticity. This translates to the prevention of the discussed diseases associated with the atrophy of the hippocampus, improved overall cognitive function, and increased or conserved functions of the structure such as spatial memory, decision-making, character judgements, and empathy (Rubin, 2014). One study also found that BDNF can have antidepressant-like effects for those with major depressive disorder. Using the Western Blot, the study found that the hippocampi of suicidal patients with depression had lower levels of BDNF compared to non-suicidal controls (Karege, 2005). Therefore, exercise inducing BDNF expression shows promise to decrease depressive symptoms. Depression is common and on the rise, impacting roughly 40 million adults in the US, and is associated with mild cognitive impairment and an acceleration of brain aging, which translates to accelerated cognitive decline. The evidence showing the relationship between exercise, BDNF expression, hippocampal volume, and depression is present and significant, but is limited. Further research could strengthen the preventative potential

depression, as well as exercise-induced BDNF as a form of an antidepressant.

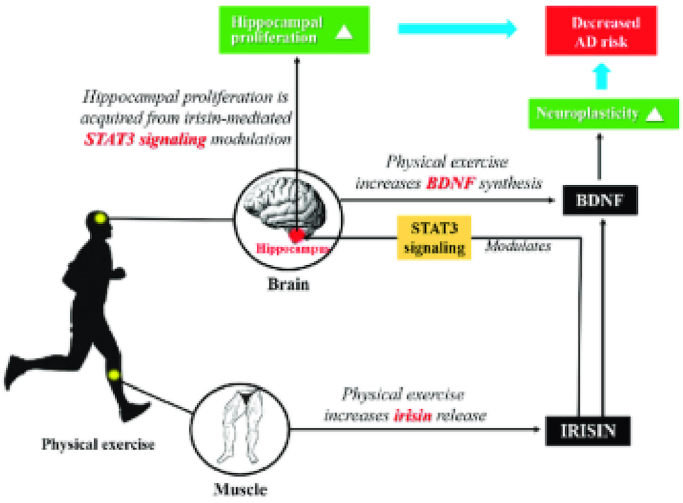

Figure 3: Exercise upregulates BDNF gene expression and therefore increases BDNF serum concentration, which also promotes synaptic plasticity. This translates to the prevention of the diseases associated with the atrophy of the hippocampus, such as AD, and improved overall cognitive function.

Although the studies available right now are small-scale and limited, weight training could be a form of preventative and mitigative medicine for neurodegenerative and neuroinflammatory diseases with further research. This is important even for a population with no cognitive impairment, for which regular resistance training could prevent cognitive decline and improve brain function. Many of the studies on this topic are done on a population with some form of mild cognitive impairment, so further study can be done on populations with no cognitive impairment to see if there are still effects on brain and cognitive function. Further research can also be done on the aspect of weight lifting correlated with improved cognitive function, such as the upper or lower limbs since these deteriorate at different rates, and whether it is the increased muscle mass or repeated motor movement requiring significant force which produces the results. Resistance training is a fundamental facet of healthy living, but also has strong preventative potential for an array of conditions, such as but not limited to: depression, other mood disorders, Alzheimer's disease, other forms of dementia, mild cognitive impairment, and forms of neurodegenerative disorders. With further research, it is possible for resistance training to possibly even have interventional potential in a clinical setting.

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