

# The Effects of Exercise on the Brain

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Exercise has been touted to do everything from treat depression to improve memory, with the power to cure a host of problems while preventing even more. In particular, exercise leads to the release of certain neurotransmitters in the brain that alleviate pain, both physical and mental. Additionally, it is one of the few ways scientists have found to generate new neurons. Much of the research done in this area has focused on running, but all types of aerobic exercise provide benefits. Although the exact nature of these benefits is still being determined, enough research has been done to provide even skeptics with a motivation to take up exercise. Exercise exerts its effects on the brain through several mechanisms, including neurogenesis, mood enhancement, and endorphin release. This paper not only examines how these mechanisms improve cognitive functioning and elevate mood states, but also proposes potential directions for future research. Furthermore, it provides an explanation for exercise's generally non-habit forming nature, despite effects on the reward centers of the brain that mimic those of highly addictive drugs like morphine.

One of the most exciting changes that exercise causes is neurogenesis, or the creation of new neurons. The new neurons are created in the hippocampus, the center of [learning](#) and memory in the brain [\(1\)](#), however the exact mechanism behind this neurogenesis is still being explored. At a cellular level, it is possible that the mild stress generated by exercise stimulates an influx of calcium, which activates transcription factors in existing hippocampus neurons. The transcription factors initiate the expression of the BDNF (Brain-Derived Neurotrophic Factor) gene, creating BDNF proteins that act to promote neurogenesis [\(17\)](#). Thus the generation of BDNF is a protective response to stress, and BDNF acts not only to generate new neurons, but also to protect existing neurons and to promote synaptic plasticity (the efficiency of signal transmission across the synaptic cleft between neurons, generally considered the basis of learning and memory) [\(1, 3, 17\)](#). However, BDNF's effects are more than protective, they are reparative. For example, in a comparison between sedentary and active mice, scientists found that active mice regenerated more sciatic axons post-injury than sedentary mice. This effect was not observed when the active mice were injected with a neurotrophin-blocking agent, indicating that exercise stimulates injured neurons to regenerate axons via neurotrophin-signaling mechanisms [\(3\)](#).

This reparative effect is particularly relevant to humans because the brain starts to lose nerve tissue beginning at age 30. Aerobic exercise reinforces neural connections by increasing the number of dendrite connections between neurons, creating a denser network, which is then better able to process and store information [\(4\)](#). This suggests possible preventative and therapeutic effects for diseases such as Alzheimer's and Parkinson's that progress via the loss of neurons. Indeed, a correlation between lifestyle and Alzheimer's has already been demonstrated [\(6\)](#). In addition, exercise has been shown

to decrease the loss of dopamine-containing neurons in mice with Parkinson's (2).

There is a limit to the positive effects of neurotrophic factors, however. Mice bred to overexercise actually showed an inability to learn. A possible cause for this inability is the disruption of cognitive function by a preoccupation with exercise. The overexercising mice had elevated BDNF and neurogenesis, but the levels reached a plateau that did not increase with more exercise (14). This limitation is further illustrated by a study of exercise effects on a group of 60- to 75-year-olds versus a group of 18- to 24-year-olds. Sedentary 60- to 75-year-olds who began aerobic exercise demonstrated an improvement in executive cognitive functions, e.g. planning, scheduling, and working memory, while the group of 18- 24-year-olds did not. Brain-wave analysis showed a 35-millisecond faster brain response time post-exercise versus pre-exercise in the 18- to 24-year-olds. Essentially, less cognitive function was lost in 18- to 24-year-olds than in 60- to 75-year-olds, so there is less room for improvement, and that improvement will be less obvious (4). Apparently it is not possible to exercise to brilliance.

Fortunately, it may be possible to exercise to happiness. It has been shown that physically active people recover from mild depression more quickly, and physical activity is strongly correlated with good mental health as people age (7). Depression is related to low levels of certain neurotransmitters like serotonin and norepinephrine. Exercise increases concentrations of these neurotransmitters by stimulating the sympathetic nervous system (12). In addition, serotonin has a reciprocal relationship with BDNF, i.e. BDNF boosts serotonin production and serotonergic signaling stimulates BDNF expression (17). Since exercise also increases BDNF production directly, there is a reinforcement of the serotonin-BDNF loop, indicating exercise's significant potential as a mood-enhancer.

In fact, a combination of exercise and antidepressants (which increase BDNF via the serotonin-BDNF loop) has been particularly effective in treating depressive behaviors in rats. The BDNF gene can be expressed in multiple forms, and physical activity increases the expression of two forms: one with fast but short antidepressive effects, and one with slow but longer antidepressive effects. By combining exercise with antidepressants (which increase the expression of the long-lasting form), scientists were able to both increase and accelerate the production of BDNF. The rats showed a decrease in depressive behaviors in two days instead of the two weeks experienced by those given antidepressants alone, indicating a potential therapy for depressed patients that produces almost immediate results (13).

There also seems to be a role for neurogenesis in the treatment of depression. Studies show that the hippocampus of depressed women can be up to 15% smaller than normal. In addition, there is a correlation between the decrease in size and the length of the depression. This damage may be reversed by BDNF-stimulated neurogenesis. Interestingly, the time it took for antidepressants to take effect is equal to the time needed to induce neurogenesis (16). All of these facts seem to point back to BDNF as the key chemical underlying exercise's impact on the brain. Perhaps it is not exercise that has the curative power, but rather BDNF, and exercise is only the trigger.

Another factor to consider is endorphins, the chemicals released by the pituitary gland in response to stress or pain. They bind to opioid receptors in neurons, blocking the release of neurotransmitters and thus interfering with the transmission of pain impulses to the brain (12). Exercise stimulates the release of endorphins within approximately 30 minutes from the start of activity. These endorphins tend to minimize the discomfort of exercise and are even associated with a feeling of euphoria. There is some uncertainty around the cause of this euphoria since it's not clear if endorphins are directly responsible for it, or if they just block pain and allow the pleasure associated with neurotransmitters such as serotonin and dopamine to be more apparent (15). If the latter is true, this would indicate a connection to BDNF via the serotonin-BDNF loop. In this case, BDNF is again the underlying chemical providing the benefits of exercise, and endorphins act in a supporting role by blocking pain and reducing the cost associated with acquiring the benefits of exercise. The release of endorphins has an addictive effect, and more exercise is needed to achieve the same level of euphoria over time. In fact, endorphins attach to the same neuron receptors as opiates such as morphine and heroin (12). Yet, exercise is not nearly as addictive as these opiates; it's not even as addictive as milder substances such as nicotine. It seems strange that an activity as beneficial as exercise, with a built-in mechanism for addiction, is so easy to give up. According to some polls, only about 15% of Americans say they exercise regularly (18).

The key to this seeming contradiction may lie in the delayed gratification experienced during exercise. Exercise differs from other addictions in that there is an initial amount of pain to endure before the euphoric payoff. The approximate 30-minute delay in the release of endorphins requires a certain level of fortitude that has not been cultivated by the American culture of video games, 30-second commercials, and various timesaving devices. In addition, exercising is made up of several tasks— putting on correct clothing, deciding on a form of exercise, maintaining adequate hydration, etc. Though each task may be mundane enough to form a habit, putting all the tasks together requires too much attention for exercise to be experienced entirely as a habit, which associates the reward or pleasure of completing a particular task with the first step of that task. In addition, the subconscious brain may use the feeling of fatigue as a regulated, anticipatory response to exercise in order to preserve homeostasis (8), possibly discouraging the continuance of exercise before the addictive euphoria is attained. If future research could find a way to trigger the release of endorphins at the start of physical activity, exercise might become more popular. Another possibility would be research around the synthesis of BDNF. If it really is the underlying chemical for all of exercise's nervous system benefits, then making it safely and readily accessible could allow people to circumvent exercise altogether, at least in terms of the nervous system.

While exercise is attractive in theory, it can often be rather painful in actuality, and the discomfort of exercise is more immediately felt than its benefits. The delayed release of endorphins creates a lapse between the pain and the pleasure elements of physical activity. The next area for research could be finding ways to make the benefits of exercise more apparent while the exercise is actually occurring, thus satisfying the need for instant gratification and tipping the scales in favor of exercise.

## References

Note that starred (\*) sources are accessible only to Bryn Mawr, Haverford, and Swarthmore students through Tripod and double-starred (\*\*) sources are informational, but not directly cited resources

- 1) [Modie, Jonathan. \(2003\). "'Good' Chemical, Neurons in Brain Elevated Among Exercise Addicts." OHSU, online.](#)
- 2) ["Exercise protects brain cells affected by Parkinson's." \(2004\). Medical Research News, online.](#)
- 3) ["Exercise can help brain healing process." \(2004\). Medical Research News, online.](#)
- 4) [Chaudhry, Laura. \(2004\). "Brain Workout." South China Morning Post, online.](#)
- 5) ["Controlling Brain Wiring With the Flick of a Chemical Switch." \(2005\). AScribe Newswire, online.\\*\\*](#)
- 6) [Kotulak, Ronald. \(2005\). "Exercise, education found to supercharge genes, reduce Alzheimer's." Chicago Tribune, online.](#)
- 7) [McKimmie, Marnie. \(2005\). "Walk away from depression." The West Australian \(Perth\), online.](#)
- 8) ["Exercise fatigue may be part of a response coordinated in the subconscious brain." \(2004\). Obesity, Fitness & Wellness Week, online.](#)
- 9) ["Keep Your Noggin Fit With Brain Exercise." \(2003\). Southern Illinois Healthcare, online.\\*\\*](#)
- 10) [Francis, Lori. "The Biology of Pleasure." online.\\*\\*](#)
- 11) ["How to Maintain Brain Power." \(2005\). Help the Aged, online.\\*\\*](#)
- 12) ["How Does Exercise Affect Our Mood?" online.](#)
- 13) Russo-Neustadt, A.A., R.C. Beard, Y.M. Huang, and C.W. Cotman. (2000). "Physical Activity and Antidepressant Treatment Potentiate the Expression of Specific Brain-Derived Neurotrophic Factor Transcripts in the Rat Hippocampus." *Neuroscience*, 101, 305-312.\*
- 14) Rhodes, Justin S., Susan Jeffrey, Isabelle Girard, Gordon S. Mitchell, Henriette van Praag, Theodore Garland, Jr., and Fred H. Gage. (2003). "Exercise Increases

Hippocampal Neurogenesis to High Levels but Does Not Improve Spatial Learning in Mice Bred for Increased Voluntary Wheel Running." Behavioral Neuroscience, 117, 1006-1016.\*

15) ["The Antidepressive Effects of Exercise." online.](#)

16) [Sanders, Jenny. "Brain Physiology." online.](#)

17) [Mattson, Mark P., Wenzhen Duan, Ruqian Wan, and Zhihong Guo. \(2004\). "Prophylactic Activation of Neuroprotective Stress Response Pathways by Dietary and Behavioral Manipulations." NeuroRx, 111-116, online.](#)

18) [Farley, Tom, and Deborah Cohen. \(2001\). "Fixing a Fat Nation." Washington Monthly, online.](#)

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