

**BDNF mediates the efficacy of exercise on synaptic plasticity and cognition.**[Vaynman S](#), [Ying Z](#), [Gomez-Pinilla F](#).

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We found that a short exercise period enhanced cognitive function on the Morris water maze (MWM), such that exercised animals were significantly better than sedentary controls at learning and recalling the location of the platform. The finding that exercise increased brain-derived neurotrophic factor (BDNF), a molecule important for synaptic plasticity and learning and memory, impelled us to examine whether a BDNF-mediated mechanism subserves the capacity of exercise to improve hippocampal-dependent learning. A specific immunoadhesin chimera (TrkB-IgG), that mimics the BDNF receptor, TrkB, to selectively bind BDNF molecules, was used to block BDNF in the hippocampus during a 1-week voluntary exercise period. After this, a 2-trial-per-day MWM was performed for 5 consecutive days, succeeded by a probe trial 2 days later. By inhibiting BDNF action we blocked the benefit of exercise on cognitive function, such that the learning and recall abilities of exercising animals receiving the BDNF blocker were reduced to sedentary control levels. Inhibiting BDNF action also blocked the effect of exercise on downstream systems regulated by BDNF and important for synaptic plasticity, cAMP response-element-binding protein (CREB) and synapsin I. Specific to exercise, we found an association between CREB and BDNF expression and cognitive function, such that animals who were the fastest learners and had the best recall showed the highest expression of BDNF and associated CREB mRNA levels. These findings suggest a functional role for CREB under the control of BDNF in mediating the exercise-induced enhancement in learning and memory. Our results indicate that synapsin I might also contribute to this BDNF-mediated mechanism.

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