Neural Basis of How Exercise Promote Mental Well Being

Abstract

Physical exercise has long been recognized to benefit locomotor and cardiovascular systems. Although an increasing body of evidence also suggests it to be an effective non-medicinal remedy for mental disorders such as depression, the underlying mechanisms remain elusive. It has demonstrated in animals that increases of the adipocyte-secreted hormone adiponectin in the central nervous system following exercise may be responsible for these neuropsychological changes, including enhanced generation of neurons in the adult hippocampus, as well as mitigation of depressive severity.

In order to find out if these results can be translated to humans, it has investigated the effects of Baduanjin Qigong exercise on adiponectin and to evaluate whether adiponectin is involved in the antidepressive effects of Qigong exercise on chronic fatigue syndrome (CFS)-like illness. It has shown that Baduanjin Qigong significantly increased adiponectin levels in females with CFS-like illness. Decreases in depression symptoms were associated with increases in adiponectin levels following Qigong exercise, indicating that the potential contribution of adiponectin to Qigong exercise elicited antidepressive effects in human subjects.

Stress-related memory deficit is correlated with dendritic spine loss. Physical exercise improves memory function and promotes spinogenesis. However, no studies have been performed to directly observe exercise-related effects on spine dynamics, in association with memory function. It has utilized transcranial two-photon in vivo microscopy to investigate dendritic spine formation and elimination in barrel cortex of mice under physical constrain or naive conditions, followed by memory performance in a whisker-dependent novel texture discrimination task. It found that stressed mice had elevated spine elimination rate in mouse barrel cortex plus deficits in memory retrieval, both of which can be rescued by chronic exercise on treadmill. Exercise also elevated brain-derived neurotrophic factor (BDNF) expression in barrel cortex.