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Tracking the antidepressant effects of physical exercise and its mechanisms

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Physical exercise is known to be beneficial for alleviating depression; however its underlying mechanisms are still unclear. Our previous animal studies have shown that physical exercise (voluntary wheel running) elicits it antidepressant effect and promotes hippocampal-dependent learning and memory via increasing adult neurogenesis and dendritic complexity in the dentate gyrus of the hippocampus. To further study how physical exercise enhances hippocampal plasticity, we targeted an adipocyte-secreted hormone: adiponectin which exhibits anti-inflammatory, insulin-sensitizing, and anti-diabetic properties just like exercise. Adiponectin can pass through the blood–brain barrier and its levels in the hippocampus can be increased by physical exercise. Administration of recombinant adiponectin into the brain decreases depression-like behaviors and promotes hippocampal neurogenesis, whereas genetic knockout of adiponectin diminishes the neurogenic and antidepressant effects of exercise. Recent studies reported that adiponectin has a rapid antidepressant effect independent to hippocampal neurogenesis. Our latest data have shown a novel role of adiponectin in promoting synaptic plasticity in the hippocampus by modulating N-Methyl-D-aspartate receptor function. These results not only reveal possible mechanisms by which exercise exerts its antidepressant effect, but also suggest a novel therapeutic treatment for depression by up-regulating adiponectin levels.