Identification of the Gene by Which Physical Exercise Protects Brain Injury Against Stroke

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Purpose: Stroke is a fatal neurological disease causing severe long-term functional disability in patients. The incident of stroke is rising exponentially, irrespective of age. The beneficial effect of physical exercise in preventing the cause of human diseases is broadly accepted while, “Which components of the exercise program are therapeutic?” remain unknown. Hypothesis: Exercise protects the stroke-induced brain injury by turning on the protein deacetylase Sirtuin 1 (SirT1) gene expression in the brain. Procedure: We analyzed sham and stroke brains from pre-exercised WT (wild type) and SIRT1+/+ (transgenic) mice. We placed the brains on brain matrices and sliced them into coronal cross-sections for TTC (Tetrazolium Chloride) staining. We also used western blot and immunohistochemistry. Results: The pre-exercised WT and SirT1+/+ stroke mice displayed lower (1) brain infarct volume, (2) neurological score and (3) brain edema, and higher (4) neural progenitor cell numbers and (5) SirT1 protein in the brain compared to non-exercised WT stroke mice. Conclusions: We discovered exercise dramatically reduces the stroke-induced brain damage. Because exercise increases SirT1 gene expression in the brain and the SirT1+/+ mice are more resistant to the stroke induced brain injury even without exercise, the exercise-mediated protective effect could be mediated via the increased SirT1 function in the brain. We identified the epigenetic mechanism by which exercise protects brain injury caused by stroke. We propose early activation of SirT1 protein in the stroke brain by SirT1 activators would be a potential therapeutic strategy to prevent/reduce the brain damage and associated functional disability in stroke patients

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