New

Title Exercise-induced neuroplasticity in human Parkinson's disease: What is the evidence telling us? Article Link


INTRODUCTION: Internationally, exercise is viewed by movement disorders clinicians as a key medicinal ingredient in treating patients at all stages of PD who do not display contraindications to becoming more physically active. Yet, movement disorders professionals' voices often invoke a fatalistic attitude toward exercise-induced neuroplasticity in PD. For example, it is still widely believed among movement scientists that neuroplasticity-based exercise strategies are lacking in PD. While animal models of exercise and PD have pushed the field forward, few studies have addressed exercise-induced neuroplasticity in human PD.

METHODS: A review of studies that examined exercise-induced neuroplasticity using the following markers of neuroplasticity: increases in maximal corticomotor excitability, exercise-induced changes in voxel-based gray matter volume changes and increases in exercise-induced serum levels of brain derived neurotrophic factor (BDNF).

RESULTS: Taken together, the studies highlighted suggest that exercise can be an important component of neuroplastic changes in human PD brain and supports the central hypothesis that self-produced activity is important in slowing, halting or reversing human PD. The studies have several limitations, including, selection bias, modest sample sizes, use of quasi-experimental designs, and enrollment of participants in the early PD stage. Possibly failure to detect correlations among BDNF levels and the PD functional outcome measures in several studies could indicate limitations in the data itself or may indicate a very minimal relation among those with early PD.

CONCLUSIONS and CLINICAL IMPLICATIONS: Evidence suggests exercise triggers several plasticity related events in the human PD brain including corticomotor excitation, increases and decreases in gray matter volume and changes in BDNF levels.