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Physical exercise as an intervention for brain disorders

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Physical exercise, whether endurance or resistance type, and independent of a large range of parameters, has been shown repeatedly to evoke a wide variety of benefits both for healthy individuals and for individuals afflicted over a broad spectrum of neurologic and neuropsychiatric disorders and neuroimmune conditions. Physical exercise, which implies all activity that generates a force through muscular activity that disrupts a homeostatic state, presents inestimable benefits for general measures of fitness and function, quality-of-life, physical strength and endurance and may be characterized on the basis of type, intensity, frequency and duration. It has been defined as a planned, structured physical activity with the purpose of improving one or more aspects of physical fitness and functional capacity. Systematic, regular exercise offers a nonpharmacologic, noninvasive and available intervention with manifest advantages for cerebral integrity during aging, restoration of motor, emotional and cognitive functional domains and alleviation of symptom profiles thereby enhancing brain health and plasticity. Regular exercise/exertion promotes neuroimmune functioning and facilitates prevention of heart conditions, cardiovascular diseases, type II diabetes and obesity, and psychological health improvements, such as in depressiveness, all of which may exacerbate the brain disorder condition. Long-term exercise benefits brain functioning through increasing cerebral blood flow and oxygenation, mobilizing growth factors and synaptic plasticity, and the facilitation of performance through neurotransmitter release and turn-over. In controlled clinical studies, the implementation of exercise programs for patients presenting neurodegenerative disorders has improved daily activity, motor performance, ambulation, overall functional independence and care-giver burden. In the laboratory, regular aerobic physical exercise (e.g. running-wheel) induces plasticity-related changes in the brain that include synaptogenesis, neuronal arborization, enhanced glucose utilization, angiogenesis and neurogenesis.

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Skeletal muscle and motor neurons in development and disease

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As a medical doctor and developmental biologist, I perform studies in order to determine if voluntary (skeletal or striated) muscle plays an active role in the etiology and pathogenesis of Motor Neuron Diseases, such as Amyotrophic Lateral Sclerosis (ALS). Because of various pieces of information, I think that an initially asymptomatic muscle either sends a trigger(s) or/and does not send a trophic factor(s) to the lower motor neuron in the spinal cord or/and brain. This initiates a cascade of events, such as peripheral axonopathy and peripheral inflammatory response, followed by the motor neuron death, central inflammatory response and subsequent muscle atrophy. Within the field, I am not the only one with an interest in muscle as one of the earliest, and potentially primary, sites for the initiation of ALS-mediated degeneration of the body's muscle and nervous tissues. With my laboratory members and collaborators, we are preparing a publication to reveal some of our cDNA microarray analysis data. Here, I elaborate on my next immediate step to deploy a Systems Biology approach, employing: StarNet, BioBricks, Registry Biological Parts, Database of Interacting Proteins, Protein Data Bank, ProtFun, STRING, EmbryoNet, BioCreAtIvE, in order to design next best experiments and therapeutic strategies targeting muscle for the treatment and cure of ALS.

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