

Physical Exercise Intervention in Autoimmune Disease

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Autoimmune diseases originate from the inappropriate responses, i.e. immune responses, originating from the body against agents and tissues that are under normal circumstances present in the body, i.e. autoimmunity. These conditions may be restricted to particular organs or involve particular tissues at different sites or regions. Several criteria are considered to determine the categorization of the disorder: (i) direct evidence arising from transfer of pathogenic antibody or pathogenic T cells, (ii) Indirect evidence derived from the observations of disorder in experimental animal models of autoimmune disease, and (iii) circumstantial evidence from clinical indications [1]. Treatments and interventions, typically, are designed to reduce the immune response. Autoimmune diseases include a huge variety of disorder conditions [2], not least of cardiovascular disease conditions, systemic lupus erythematosus and Multiple Sclerosis (MS).

Both in the general population and in patients presenting cardiovascular disease and hypertension, macronutrient and micronutrient

deficiencies/conditions, resulting from genetic/environmental/prescription drug use causes, these disorders have been observed to contribute major health hazards with significant impact upon cardiovascular health and outcome as well as medical costs and patients' quality-of-life. Thus, interventions that reduce blood pressure, improve vascular health, endothelial dysfunction and vascular biology, and decrease cardiovascular 'events' offer a necessary and much required benefit. Physical exercise, in addition to other lifestyle modifications, combined with the appropriate pharmacological therapies offers a plethora of advantages, not least the proliferation and consolidation of angiogenesis [3], for the attainment of acceptable blood pressure levels, the reduction of cardiovascular risk factors, improvement of vascular biology and vascular health, the reduction of cardiovascular target organ damage, and the eventual reduction of medical care expenditures [4]. Similarly, in Systemic Lupus Erythematosus (SLE), chronic autoimmune disorder characterized by production of autoantibodies directed against nuclear and cytoplasmic antigens which affects several organs, physical has provided benefits [5-7] carried out a prospective study where female SLE patients, available to perform physical exercise (walking at a

heart rate corresponding to the ventilatory 1 threshold obtained from ergospirometry and monitored by a frequency meter), were allocated to an Exercise Group (EG, $n = 18$) to perform supervised physical exercise for 1 h (3 times/week) for 16 weeks; patients unavailable were allocated to the Control Group (CG, $n = 20$). Patients were examined for brachial artery endothelial function after 16 weeks. There was an increase in flow-mediated dilation in the EG but not CG. Improvements were observed in exercise tolerance, maximum speed and threshold speed by the EG but not the CG. Thus, exercise improved endothelial function and aerobic capacity without compromising the health of SLE patients.

In an animal laboratory model of MS, [8] examined the experimental interventional effects of forced wheel running on rats induced with Experimental Autoimmune Encephalomyelitis (EAE). MS, an inflammatory disorder characterized by damage to the insulating covers of neurons in the brain and spinal cord, involves a multitude of neurologic signs and symptoms that include physical, psychological and psychiatric domains. They (ibid) assigned female Lewis rats randomly either one of four groups, prior to inoculation: (i) EAE Exercise (EAE-Ex), (ii) EAE Sedentary (EAE-Sed), (iii) Control Exercise (Con-Ex), or (iv) Control Sedentary (Con-Sed). Forced treadmill running at increasing intensity levels was presented over 10 consecutive days; there no significant differences in the level of induced disability or brain mass recorded in the EAE groups at the conclusion of this stage. Although brain tissue protein analysis indicated that Tumour Necrosis Factor- α (TNF- α) concentrations were higher in both EAE groups compared with the control groups ($p < 0.05$), no significant differences were seen between the EAE-Ex and EAE-Sed groups. Nevertheless, Nerve Growth Factor (NGF)

concentrations were greater in the EAE-Ex animals compared with both control groups but no differences in the concentrations of Brain-Derived Neurotrophic Factor (BDNF) were obtained. The authors suggested that that aerobic exercise may modulate the proteins associated with disability in EAE [9, 10], but it is likely that the 10-day exercise intervention was too short [11]. Have shown 'low level' beneficial evidence for physical activity programmes used in isolation or in combination with other interventions. Furthermore, [12] observed that self-efficacy and perceived benefits of exercise predicted propensity for the "health belief model" of physical activity in MS patients. Thus, patients presenting MS believe in the benefits of physical activity and are likely to remain healthy even in the disease context. Additionally, [13] have highlighted the putative influence of premorbid physical activity for 'slowing-down' disability progression over time in persons with relapsing-remitting MS. Finally, [14] have presented evidence, from clinical trials, implicating the beneficial effects of physical exercise training upon muscle strength, aerobic capacity and walking performance, and on fatigue, gait, balance and quality of life.

Psychosocial stress and negative affect are linked to elevations in several inflammatory biomarkers. Physical exercise diminishes inflammation and elevates agents and factors involved in immunomodulatory function, providing manifest anti-inflammatory benefits, mediated possibly by BDNF and a variety of anti-inflammatory cytokines, under both laboratory and clinical conditions, in autoimmune disorders and related conditions [15]. The effects of physical exercise training on systemic inflammation have been studied profusely within epidemiological research. It is implied currently that the strongest improvements in inflammatory profiles are

evidenced at higher intensities of exercise; combinations of aerobic and resistance exercise programs show greatest potential for provision of lasting anti-inflammatory benefits [16]. Not least, exercise offers a useful therapeutic modality to reduce risk factors associated with the pathogenesis of insulin-resistance in obese, sedentary males [17].

In conclusion, the experimental papers concerning this specific field did show some differences among the different sports and exercise period. Therefore, a clear conclusion is not applicable related to the benefit effect of the exercise to the improvement of the autoimmune diseases and further demonstrations of exercise efficacy are awaited.

References

1. Witebsky E, Rose NR, Terplan K, Paine JR, Egan RW (1957) Chronic thyroiditis and autoimmunization. *J Am Med Assoc* 164: 1439-1447.
2. Rose MR, Bona C (1993) Defining criteria for autoimmune diseases (Witebsky's postulates revisited). *Immunol Today* 14: 426-430.
3. Archer T (2012) Influence of physical exercise on traumatic brain injury deficits: scaffolding effect. *Neurotox Res* 21: 418-434.
4. Houston MC (2013) The role of nutrition, nutraceuticals, vitamins, antioxidants, and minerals in the prevention and treatment of hypertension. *Altern Ther Health Med*. 1: 32-49.
5. Barnes JN, Tanaka H (2012) Cardiovascular benefits of habitual exercise in systemic lupus erythematosus: a review. *PhysSportsmed* 40: 43-48.
6. Fortuna G, Brennan MT (2013) Systemic lupus erythematosus: epidemiology, pathophysiology, manifestations, and management. *Dent Clin North Am* 57: 631-655.
7. dos Reis-Neto ET, da Silva AE, Monteiro CM, de Camargo LM, Sato EI (2013) Supervised physical exercise improves endothelial function in patients with systemic lupus erythematosus. *Rheumatology (Oxford)* 52: 2187-2195.
8. Patel DI, White LJ (2013) Effect of 10-day forced treadmill training on neurotrophic factors in experimental autoimmune encephalomyelitis. *Appl Physiol Nutr Metab* 38; 194-199.
9. Maker-Clark G, Patel S (2013) Integrative therapies for multiple sclerosis. *Dis Mon* 59: 290-301.
10. Padgett PK, Kasser SL (2013) Exercise for managing the symptoms of multiple sclerosis. *PhysTher* 93: 723-728.
11. Amatya B, Khan F, La Mantia L, Demetrios M, Wade DT (2013) Non pharmacological interventions for spasticity in multiple sclerosis. *Cochrane Database Syst Rev* 2:CD009974. doi: 10.1002/14651858.CD009974.pub2.
12. Kasser SL, Kosma M (2012) Health beliefs and physical activity behavior in adults with multiple sclerosis. *Disabil Health J* 5: 261-268.
13. Motl RW, Dlugonski D, Pilutti L, Sandroff B, McAuley E (2012) Premorbid physical activity predicts disability progression in relapsing-remitting multiple sclerosis. *J NeurolSci* 323: 123-127.
14. Motl RW, Pilutti LA (2012) The benefits of exercise training in multiple sclerosis. *Nat Rev Neurol* 8: 487-497.

15. Archer T, Fredriksson A, Schütz E, Kostrzewa RM (2011) Influence of physical exercise on neuroimmunological functioning and health: aging and stress. *Neurotox Res* 20: 69-83.
16. Nimmo MA, Leggate M, Viana JL, King JA (2013) The effect of physical activity on mediators of inflammation. *Diabetes ObesMetab.* 3: 51-60.
17. Samjoo IA, Safdar A, Hamadeh MJ, Raha S, Tarnopolsky MA (2013) The effect of endurance exercise on both skeletal muscle and systemic oxidative stress in previously sedentary obese men. *Nutr Diabetes* 3:e88.