OPEN ACCESS

GAVIN PUBLISHERS

Journal of Orthopedic Research and Therapy

Moustogiannis A and Theos A. J Orthop Res Ther 4: 1151.

Editorial

DOI: 10.29011/2575-8241.001151

How Exercise Benefits Your Health - The Cellular and Molecular Mechanisms

Athanasios Moustogiannis[1], Apostolos Theos[2*]

[1]Department of Physiology, National & Kapodistrian University of Athens, Greece

[2]Department of Community Medicine & Rehabilitation, Section of Sports Medicine, Umeå University, Sweden

[*]**Corresponding author:** Apostolos Theos, Department of Community Medicine & Rehabilitation, Section of Sports Medicine, Umeå University, Sweden.

Citation: Moustogiannis A, Theos A (2019) How Exercise Benefits Your Health - The Cellular and Molecular Mechanisms. J Orthop Res Ther 4: 1151. DOI: 10.29011/2575-8241.001151

Received Date: 12 December, 2019; **Accepted Date:** 23 December, 2019; **Published Date:** 27 December, 2019

The importance of physical fitness, regular exercise and physical activity has been acknowledged for over 7000 years, dating back to ancient Chinese and Greek civilizations [1]. However, scientific data documenting the essentiality of exercise for health did not emerge until the late 1800s and early 1900s when epidemiological studies clearly demonstrated that physically inactive individuals were more likely to have coronary heart disease than those who led active lifestyles [2]. In recent years' physical inactivity has been associated with many disorders such as obesity, cardiovascular disease, hypertension, type 2 diabetes, metabolic syndrome, insulin resistance, hyperlipidaemia, breast and colon cancer as well as depression and anxiety. Exercise on the other hand has been shown to be able to reverse the atrocious results of physical inactivity, promote cardiometabolic wellness, improve cognitive performance, and effectively aid in the prevention and treatment of non-communicable diseases. The biological mechanisms at a cellular and molecular level that are triggered by regular exercise will be briefly presented in the present editorial.

The cellular adaptation of exercise in heart can be associated with both endogenous and exogenous factors: 1) exercise induces cardiac growth via hypertrophy and renewal of cardiomyocytes, and 2) exercise induces endothelial progenitor cells to proliferate, migrate and differentiate into mature endothelial cells, giving rise to endothelial regeneration and angiogenesis. The cellular adaptations associated with exercise are due to the activation of several signaling pathways, in particular, the growth factor neuregulin1 (NRG1)-ErbB4-C/EBPβ and insulin-like Growth Factor (IGF)-1-PI3k-Akt signaling pathways [3,4]. In diabetes exercise improves mitochondrial function, increases mitochondrial biogenesis, and increases the expression of glucose transporter proteins and numerous metabolic genes. The key role of these actions is translocation of GLUT4. Muscle contraction involves changes in energy status (i.e., increased AMP/ATP), increases in intracellular Ca2+ concentration, Reactive Oxygen Species (ROS), and protein kinase C. These changes activate various signalling cascades, some of which seem to be involved in the phosphorylation of re-2/USP6, BUB2, cdc16 domain family member 1 (TBC1D1) and Akt substrate of 160 kDa (AS160) and activate GLUT4 translocation [5]. In addition, physical exercise has been shown to reduce cancer incidence and inhibit tumour growth through sex hormones, insulin/IGF, and inflammatory markers [6]. Exercise leads to increased blood flow, shears stress on the vascular bed, increases temperature, and activates sympathetic and endocrine regulation (release of catecholamines and exercise hormones, myokine secretion). All the above result in increased tumour perfusion, oxygen delivery, intra-tumoral metabolic stress, cellular damage, and ROS production. These changes are able to elicit signalling pathways that prevent metastasis. Moreover, exercise (especially strength and balance training) seems to be an effective preventive and therapeutic method that reduces the neurotoxic effects of chemotherapy (for example of platinum chemotherapies, commonly used for the treatment of colon cancer) and positively impacts the lives of patient. The mechanisms are not completely clear but they probably involve BDNF and GDNF, that promote muscle reinnervation and axon regeneration [7]. Current evidence indicates that moderate to high-intensity endurance exercise is superior to light exercise, when aiming to target tumour intrinsic factors. Furthermore, exercise has extensively been shown to regulate the cellular immune system, as cytotoxic immune cells are mobilized through mechanisms involving blood flow-induced shear stress and adrenergic signalling. Chronic training adaptations lead to systemic alterations that include improved immune function, reduced e systemic inflammation, and elevated metabolic health. Furthermore, enhanced blood perfusion, immunogenic profile, and immune cell infiltration seem also to be related to chronic training adaptations [8]. Last but no least, exercise can induce an upregulation of genes involved in leukocyte protein production rate and mitochondria biogenesis, as well as a downregulation of

inflammation [9]. This is due to the identified roles of sympathetic activation, catecholamine signalling, and mobilization of cytotoxic immune cells derived from high-intensity endurance training.

Regular exercise may be an especially effective intervention in treating and/or preventing a variety of comorbidity expressed conditions characterized by dysregulated neuroendocrine, inflammatory, metabolic and behavioural stress responsive pathways Further research is needed in order to promote in depth knowledge of the above mentioned molecular mechanisms that will in turn allow the prescription of the optimal dose, intensity, and mode of exercise training.

References

1. Silverman MN, Deuster PA (2004) Biological mechanisms underlying the role of physical fitness in health and resilience. Interface focus 4: 20140040.
2. MacAuley D (1994) A history of physical activity, health and medicine. Journal of the Royal Society of Medicine 87: 32-35.
3. Neufer PD, Bamman MM, Muoio DM, Bouchard C, Cooper DM, et al. (2015) Understanding the cellular and molecular mechanisms of physical activity-induced health benefits. Cell metabolism 22: 4-11.
4. Tao L, Bei Y, Zhang H, Xiao J, Li X (2015) Exercise for the heart: Signaling pathways. Oncotarget 6: 20773-20784.
5. Stanford KI, Goodyear LJ (2014) Exercise and type 2 diabetes: Molecular mechanisms regulating glucose uptake in skeletal muscle. Advances in physiology education 38: 308-314.
6. McTiernan A (2008) Mechanisms linking physical activity with cancer. Nature reviews. Cancer 8: 205-211.
7. Ferioli M, Zauli G, Martelli AM, Vitale M, McCubrey JA, et al. (2018) Impact of physical exercise in cancer survivors during and after antineoplastic treatments. Oncotarget 9: 14005-14034.
8. Hojman P, Gehl J, Christensen JF, Pedersen BK (2018) Molecular mechanisms linking exercise to cancer prevention and treatment. Cell metabolism 27: 10-21.
9. Cambell JP, Turner JE (2018) Debunking the myth of exercise-induced immune suppression: Redefining the impact of exercise on immunological health across the lifespan. Frontiers in Immunology 9: 648.