THE CONSEQUENCES OF A SEDENTARY LIFESTYLE

НАСЛІДКИ МАЛОРУХЛИВОГО СПОСОБУ ЖИТТЯ

Seniuk I.V., Tkachenko O.V., Benarafa Ibrahim Amin
Сенюк І.В., Ткаченко О.В., Бенарафа Ібрахім Амін
National University of Pharmacy, Kharkiv, Ukraine
Національний фармацевтичний університет, м. Харків, Україна

Анотація. Недостатня фізична активність є основною причиною більшості хронічних захворювань. У статті розглядаються: визначення активності та профілактики; історичні докази того, що недостатня фізична активність шкодить здоров'ю та нормальній функціональній здатності органів; причина проти лікування; механізми фізичної активності та бездіяльності відрізняються; взаємодія генів і середовища; специфічність адаптації до типу тренувань. Далі фізична активність/вправи розглядаються як первинна профілактика понад 30 хронічних станів: прискорене біологічне старіння/передчасна смерть, низька кардіореспіраторна придатність, саркопенія, метаболічний синдром, ожиріння, інсулінорезистентність, переддіабет, діабет 2 типу, неалкогольна жирова хвороба печінки, ішемічна хвороба серця, захворювання периферичних артерій, гіпертонія, інсульт, застійна серцева недостатність, ендотеліальна дисфункція, артеріальна дисліпідемія, гемостаз, тромбоз глибоких вен, когнітивна дисфункція, депресія та тривога, остеопороз, остеоартрит, порушення рівноваги, переломи/падіння кісток, ревматоїдний артрит, рак товстої кишки, рак молочної залози, рак ендометрія, гестаційний діабет, прееклампсія, синдром полікістозних яєчників, еректильна дисфункція, больовий синдром, дивертикуліт, запор, захворювання жовчного міхура.

Ключові слова: малорухливість, фізична активність, метаболізм, дисфункція, хронічні захворювання.

Abstract. Physical inactivity is a primary cause of most chronic diseases. The article considers: activity and prevention definitions; historical evidence showing physical inactivity is detrimental to health and normal organ functional capacities; cause vs. treatment; physical activity and inactivity mechanisms differ; gene-environment interaction; and specificity of adaptations to type of training. Next, physical activity/exercise is examined as primary prevention against over 30 chronic conditions: Accelerated biological aging/premature death, low cardiorespiratory fitness, sarcopenia, metabolic syndrome, obesity, insulin resistance, prediabetes, type 2 diabetes, non-alcoholic fatty liver disease, coronary heart disease, peripheral artery disease, hypertension, stroke, congestive heart failure, endothelial dysfunction, arterial dyslipidemia, hemostasis, deep vein thrombosis, cognitive dysfunction, depression and anxiety, osteoporosis, osteoarthritis, balance, bone fracture/falls, rheumatoid arthritis, colon cancer, breast cancer, endometrial cancer, gestational diabetes, preeclampsia, polycystic ovary syndrome, erectile dysfunction, pain, diverticulitis, constipation, and gallbladder diseases.

Key words: inactivity, physical activity, metabolism, dysfunction, chronic diseases.

Introduction. We define "functional capacity" as the ability of a cell, organ, system, or body to maintain homeostasis within their narrow limits of survival in response to a specified stress. If an external stress disrupts homeostasis beyond an organism's functional capacity, life may not be sustained. Diminished ability to adapt to stressors increases the likelihood of death. Functional capacity is pliable; declining

rapidly with extreme physical inactivity or more slowly with aging, while preventing inactivity can increase functional capacity (considered in specific detail in the aging section). Importantly, a direct relationship between functional capacity and survival is a cornerstone of general medicine theory. A major predictor of functional capacity is Maximal Aerobic Capacity (VO_2max), which while directly testing cardiovascular fitness and integrity also represents a combination of other physiologic components. For instance, VO_2max also depends on pulmonary and muscle function, health status of other organ systems, nutritional status, medications, orthopaedic limitations, and others [1]. An aerobic functional capacity in patients under 4-Metabolic Equivalents (METs), a typical demand during normal daily activities, increases postoperative (time from admission to discharge from surgery) cardiac and long-term risks [2]. In another study, patients were grouped by MET capacity in relationship to complication prevalence after they underwent angiographically verified coronary artery disease and subsequent open abdominal nonvascular surgery [3]. Those from the group <4 METs had cardiologic complications in 64% of cases, the 4-7 METs group had 29%, and the 7-10 METs group had 8%. These remarkable findings can be extrapolated to other stresses where the probability of complications, and even survival, is dependent upon the functional capacity needed to maintain homeostasis.

The aim of the study. To study functional and biochemical processes in a sedentary lifestyle.

Materials and methods. A literature search was conducted in PubMed, Scopus and Web of Science databases using keywords.

Results. The term "diseasome of physical inactivity" was presented by Pedersen [4] to describe a clustering of diseases. Our article enlarges Pedersen's cluster to include over 35 diseases/conditions and death, which constitute most of the remaining article. Joyner and Pedersen [5] contend that it is a failure of regulation at multiple levels that causes many common diseases. They further argue that a lack of fluency to use key physiological concepts (like homeostasis, regulated systems and redundancy) as major intellectual tools to understand at multiple levels how whole animals adapt to exercise and maladapt to physical inactivity.

Less physical activity shortens years of life relative to average lifespan Healthy behavioural choices in Californian Adventists extend life expectancy by several years, even as much as a decade [6]. Various reports estimate that higher physical activity levels may extend life expectancy relative to average lifespan by 2.1 [7], 2.5 (395), 5.1 (men) [8], and 5.7 (women) [8] yrs for the physically active population.

Another example of lifetime physical activity shortening years lived is the increased risk of chronic diseases such as type 2 diabetes. Diagnosis of type 2 diabetes at the age of 20 yrs is associated with 17.2 and 17.9 yrs of life lost in males and females, respectively [9].

Less physical activity increases percentage of population that is disabled at the same age for death, the high physical activity group spent less time disabled than the overall population of men (2.5 vs. 3.0 years), while the low physical activity group actually spends more time disabled than all men (2.6 vs. 1.4 years) [8]. Thus, less lifetime physical activity shortens years of life.

While light physical activity is associated with rather low-intensity muscle contractions, it still has favorable improvements on plasma glucose in glucose tolerance tests [10], and differs substantially from the absence of muscle activity while sitting The detrimental effects of sitting have been hypothesized by Stamatakis et al. [11] to occur in the following sequence of events: excessive sitting lowers skeletal muscle blood flow, lowering shear stress on vascular endothelial cells, and decreasing endothelial nitric oxide synthase (NOS) expression. Rapid biochemical changes in a rodent models of sitting, hindlimb unloading, have demonstrated decreases in rat skeletal muscle protein synthesis rates within the first 6 hrs [12] and loss of insulin-stimulated glucose uptake into the mouse soleus muscle after 1 day. Sedentary lifestyle speeds secondary aging of skeletal muscle power by 24 yrs Low muscle strength has been inversely associated with all-cause-mortality in thirteen studies using subjects > 65 yrs of age [13].

According to Blair and co-authors [14], several possible biological mechanisms exist for the risk reduction of all-cause mortality in

individuals with higher Constant Rate Factor (CRF). Higher CRF is associated healthier values for risk factors including insulin sensitivity, blood lipid and lipoprotein profile, body composition, systemic inflammation, blood pressure and the autonomic nervous system functioning.

All risk factors for Metabolic Syndrome (MS) are exasperated by sedentary lifestyle. In other words, physical inactivity is a primary cause of MS risk factors by virtue of its being upstream to the common MS risk factors. Alternatively, risk factors for MS are secondary to sedentary lifestyle. Consequently, increased physical activity is primary prevention of MS. Several risk factors for MS are associated with physical inactivity, including low-grade inflammation and impaired metabolism. Conversely, prevention of physical inactivity through physical activity improves inflammatory markers by reducing resting CRP, interleukin-6 (IL-6), and tumor necrosis factor-α concentration. On potential mechanism is highlighted by Pedersen who has put forth the hypothesis that the muscle secretum (termed myokines) is involved in mediating some of the health effects of regular exercise, in particular chronic diseases associated with low-grade inflammation and impaired metabolism, as well as the brain. For example, contracting skeletal muscle during exercise produces interleukin-6, which has anti-inflammatory properties [15].

Sedentary lifestyle reveals an obesity phenotype that is primarily prevented by enhanced physical activity. Physical inactivity is a primary cause to Visceral Adipose Tissue (VAT) and whole-body obesities. Primary prevention of obesity is possible today for almost all able-bodied individuals able to exercise.

Physical inactivity is a primary cause of loss of insulin sensitivity in skeletal muscle, and thus whole-body. Primary prevention of almost all of insulin resistance by high levels of daily physical activity is possible for almost all humans up their seventh decade of life [16]. Continued long-term reductions in physical activity are a primary cause of insulin resistance.

Positive effects of chronic exercise on primary prevention of Coronary Heart Disease (CHD) could be explained by several mechanisms including: increased nitric oxide and antioxidants, decreased pro-inflammatory cytokine levels in blood by decreasing production from multiple tissues, and increased regenerative capacity of endothelium expressed by an increased number of circulating endothelial precursor cells [17].

Epidemiological, interventions, and mechanistic insights from human and rodent studies all suggest that physical inactivity can accelerate declines in cognitive function; a decline that be attenuated or potentially reversed by physical activity.

Physical inactivity causes up to 1/3rd of depression. Physical activity can primarily prevent 20-30% of depression.

Physical inactivity is a primary cause of bone loss in weight-bearing bones. Physical activity results in both gravitational and muscle-contraction loading of the skeleton and, therefore, is primary prevention of osteoporosis.

Conclusions. Physical activity, food, and reproduction are some of the minimal requirements for life. They evolved not as choices, but as requirements for individual and species survival. Modern humans have been able to engineer most physical activity out of daily life. Humans now have a choice not to be physically active. Conclusive and overwhelming scientific evidence, largely ignored and prioritized as low, exists for physical inactivity as a primary and actual cause of most chronic diseases. Thus, longer-term health was also engineered out with the successful removal of physical activity as a necessity for immediate survival. The comprehensive evidence herein clearly establishes that lack of physical activity affects almost every cell, organ, and system in the body causing sedentary dysfunction and accelerated death. The massive multifactorial nature of dysfunction caused by sedentarism means that just as food and reproduction remain as requirements for long-term continued human existence, physical activity is also a requirement to maximize health span and lifespan. The only valid scientific therapeutic approach to completely counter sedentary dysfunction is primary prevention with physical activity itself.

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Information about the Authors/Відомості про авторів

1. **Сенюк Ігор Валерійович**, кандидат фармацевтичних наук, доцент закладу вищої освіти, кафедра біологічної хімії, Національний фармацевтичний університет, м. Харків, Україна.

Seniuk Igor Valeriyovych, Candidate of Pharmaceutical Sciences (PhD), Associate Professor of Higher Education, Department of Biological Chemistry, National University of Pharmacy, Kharkiv, Ukraine.

e-mail: citochrom@gmail.com

ORCID: 0000-0003-3819-7331

2. **Ткаченко Оксана Володимирівна**, кандидат фармацевтичних наук, доцент закладу вищої освіти, кафедра біологічної хімії, Національний фармацевтичний університет, м. Харків, Україна.

Tkachenko Oksana Volodymyrivna, Candidate of Pharmaceutical Sciences (PhD), Associate Professor of Higher Education, Department Biological Chemistry, National University of Pharmacy, Kharkiv, Ukraine.

e-mail: kseniashovkova@gmail.com

ORCID: 0000-0002-9656-1165

3. **Бенарафа Ібрахім Амін**, випускник 2022 року, Магістр фармації.

Benarafa Ibrahim Amin, Graduated in 2022, Master of Pharmacy.