EC Cardiology Editor's Column - 2019

Healthy Life Expectancy and Efficient Exercise for Older Adults

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COLUMN ARTICLE

Medical care has dramatically increased life expectancy in many industrialized countries. Consequently, the drastically higher proportion of elderly individuals evokes new socioeconomic and public health problems. The number of fitness gyms has increased, reflecting the increasing awareness and commitment to healthier lifestyles in Japan. In our fitness gym, which is affiliated and located in Sankuro Hospital, the most popular age groups of the members are the sixties (36.3%) and seventies (24.7%). About two-thirds of the members either have chronic diseases such as cardiovascular and lifestyle-related diseases or are cancer survivors. Healthy life expectancy is a commonplace but very important theme in clinical medicine.

Accumulated knowledge over the last 50 years undoubtedly proves that regular exercise lowers all-cause and cardiovascular mortality risks and enhances health and longevity and that an inactive lifestyle is inherently unsafe. Both major types of exercise, aerobic and resistance training, grant multi-systemic benefits and protection against the major features of aging, including mitochondrial dysfunction, recycling deficiency, impaired quality control, and systemic inflammation, thereby suggesting that exercise is a front-line modality to decelerate the aging process.

Human aging is divided into primary aging and secondary aging [1]. Primary aging is defined as the progressive decline in physiological homeostasis, physical performance, and fitness with increasing age (ultimately causing death) driven by energy flux (depending on the individual metabolic rate and high energy expenditure). With higher energy expenditure, more reactive oxygen species (ROS) are generated, which can lead to increased oxidative damage to mitochondrial DNA, proteins, and lipids that are essential for normal cellular function. Secondary aging is defined as any untoward change in body composition and metabolic function that reduces health and longevity through external factors such as overeating or low physical activity that can accelerate the aging process. In contrast, calorie restriction and exercise both decrease fat mass together with muscle strength and quality decelerating the aging process. .

Citation: Shigenori Ito. "Healthy Life Expectancy and Efficient Exercise for Older Adults". EC Cardiology ECO.02 (2019): 08-10.

The panaceas to increase healthy life expectancy include calorie restriction associated with efficient exercise [1]. The importance of exercise for older adults to prevent sarcopenia and cognitive disorders has been indicated, but many national and individual attempts have not necessarily obtained success. Calorie restriction exerts its effect on primary aging via a reduction in energy intake and subsequently reduced flux of macronutrients through the mitochondria. In turn, mitochondria release less ROS yielding decreased oxidative damage to DNA. Exercise increases energy expenditure, which increases the energy influx through the mitochondria and leads to greater generation of ROS and oxidative damage. In contrast, exercise has been shown to improve biomarkers associated with cardiovascular disease, reduce adiposity and maintain fat-free mass, leading to lower morbidity and all-cause mortality in epidemiological studies.

It is now widely accepted that the ability of the cardiorespiratory system and skeletal muscles to deliver and utilize oxygen is a strong determinant of health and longevity in modern humans. Increased maximal oxygen uptake (VO_{2max}) is associated with numerous cardiovascular and non-cardiovascular benefits, including reduced risk of coronary artery disease, hypertension, diabetes, stroke, and cancer [2-4], which decrease with aging [5]. For example, runners have an approximately 45 - 70% and 30 - 50% reduced risk of mortality due to cardiovascular disease and cancer, respectively, and live 3 - 10% (2 - 8 years) longer than non-runners live [4]. Aging is characterized by a progressive impairment of all body organs, including those that regulate VO_{2max} and locomotion, resulting in an approximately 10% decline in aerobic capacity per decade in both men and women after around 30 years of age. Because maintaining and improving VO_{2max} would be indispensable for health life expectancy, healthcare professionals should encourage patients and older adults to achieve and maintain high levels of fitness.

Mitochondria have a profound role in the evolution of aerobic life. They hold a central position in cellular homeostasis and drive many aspects of the biological aging process. Short., *et al.* [6] found that mitochondrial protein content and activity of a key oxidative enzyme like citrate synthase are reduced in skeletal muscle in older individuals. There was also a continuous decline in mitochondrial capacity for oxidative phosphorylation (i.e. adenosine triphosphate production) with advancing age in skeletal muscle samples from many healthy men and women between the ages of 18 and 87 years. On the other hand, in the endurance-trained muscle, most signaling pathways that are activated subsequent to motor neuron activity and mechanical stress converge on the peroxisome proliferator-activated receptor γ coactivator-1α (PGC-1α), which is a transcriptional coactivator that binds to a number of transcription factors, thereby potently inducing gene expression as a strong promoter of mitochondrial biogenesis and function [7]. High-intensity interval training (HIIT), which increases PGC-1α more than that by moderate-intensity endurance training in patients with chronic heart failure [8], might counteract mitochondrial dysfunction with aging.

Myokines, which are muscle-derived proteins analogous to the adipokines secreted from adipose tissue, are produced and secreted by skeletal muscle and exert subsequent auto-, para-, and endocrine effects and thereby have major implications on metabolic and other properties of muscle, as well as distal organs. There are data showing the multi-system benefits of endurance exercise training via myokines such as interleukin (IL)-6, IL-8, and IL-15 [7]. For example, IL-6 induces glucose uptake and fatty acid β-oxidation in muscle, stimulates hepatic gluconeogenesis, and induces lipolysis in fat. Similarly, IL-15 seems to be involved in muscle-adipose tissue crosstalk. High local IL-8 concentrations might be involved in exercise-induced angiogenesis and hence increased capillarization of skeletal muscle. These functions of exercise-induced myokines might also contribute to healthy life expectancy.

Recently, HIIT has been adapted for healthy old adults [9] and old adults with congestive heart failure [8]. HIIT improves cardio-respiratory fitness, insulin sensitivity, mitochondrial respiration, and fat-free mass more effectively than that by moderate-intensity continuous training, which has been popularly recommended as an exercise protocol for a wide range of subjects in many guidelines from academic societies. Furthermore, the more time-efficient and strenuous sprint-interval training (SIT)

protocol [10] has also been evaluated for older adults. To the best of my knowledge, the least dose and most time-efficient SIT could be reduced-exertion high-intensity interval training (REHIT) [11,12]. In the REHIT protocol, two sets of 20s all-out sprints interspersed with 3 - 4 minutes recovery performed 2 - 3 times a week improve VO_{2max} and insulin sensitivity. Older adults without physical disability or serious cardiovascular disease could be potential candidates of this protocol [13].

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