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Respiratory Chain Senescence and Its Relation to Physical Activity

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A B S T R A C T

This article is a scientific review on the effects of senescence of the respiratory chain and it aims to provide an overview of what role exercise has on the way the aging of the respiratory chain occurs. There are several reasons that explain the lack of exercise in senescent, less need, less interest, different goals, and also deficiencies that make these decisions strongest, as mild equilibrium, coordination, strength, or vision abnormalities. The use of the whole respiratory chain from the mithocondria to the the lungs, its solicitation through exercise and a proper nutrition are basic to limit the senescence of the system. The capacity for renewal and adaptation to processes of time, environment exposition and use, will define a faster, difficult and hopeless or slower and comfortable aging. The absence of movement, the lack of function of any organ or system leads to the inefficiency, atrophy and death.

***Key words**: senescence, respiratory chain, exercise*

Introduction

The concept of aging, in spite of its complex biological nuances that nourish various hypotheses[1,2], lies in the individual's knowledge since childhood. The toy, the bicycle, the car, and ultimately any object, and any being, is modified according to three magnitudes of exposure: time, environment exposition and use. The capacity for renewal and adaptation to that process, will define a faster, difficult and hopeless or slower and comfortable aging. This last can be assumed as longevity.

With aging, the structure material loses its rigidity, containment capacity of support or protection. From a certain point the physical qualities are lost and the structure disappears. The machinery that governs its functionality, can alter their efficacy and safety resulting in a separate application for which it was designed, altering the rest of the structure and functioning. The electrical and mechanical components, if they exist, are denatured by time and environment; humidity, heat, cold, cause structural disorders, driving and fragility of the system, while allowing a function, define a known destination, the attic, storage room or the junkyard. Anyone can recognize that if there is a proper care of objects, keeping them in good condition, changing the altered parts over time, keeping them lubricated, »well nourished«, and above all, using them, will make the objects imperishable. These three aspects, use, proper use and care, so obvious and practical for objects, is forgottten when it comes to ourselves or our fellows. The anti-aging therapy, as fashion, has a moderate level of scientific evidence that we can observe related to the respiratory chain at structural and functional level. This article aims to provide an overview of what role exercise has on the way the aging of the respiratory chain occurs.

We are designed for movement. In fact, we are a collection of cells, extremely well organized, that move and work in order to reach the food to obtain energy and substrates to keep the system and the species alive. To keep on moving is fundamental to staying alive. The absence of this movement, the lack of function of any organ or system leads to the inefficiency, atrophy and death.

As time goes on, the amount and quality of movement decreases. At our society, the essential needs are covered without big effort. We do not need to hunt, gather or fight, at least manually or with a high physical effort. Long before the job retirement, the energy expenditure for which we are prepared was abandoned decades ago, even when it was done for professional purposes. By contrast, the diet, the amount of caloric intake, doesn't

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change. Usually high poor quality caloric intakes remain accompanied by a lack of essential nutrients in that time of life[1]. There are several reasons that explain the lack of exercise in senescent, less need, less interest, different goals, and also deficiencies that make these decisions strongest, as mild equilibrium, coordination, strength, or vision abnormalities. Even major athletes fall into a lower level of training due to, the lack of time, quality of training, objectives and the modification of its density and impact in the evolutionary process[1,2]. Thus, aging is associated with a significant decrease of solicitation, neuromuscular function, and nourishment. As we get older this decline brings the organism to an inevitable reduction in muscle mass and strength loss associated with it. This process is known as sarcopenia, from the Greek *sarx* flesh and *penia* loss[1]. Today the term sarcopenia is used to describe the loss of muscle mass and all the changes that accompany and condition its establishment, the innervation of the central and peripheral nervous system, changes in hormone levels with age, and the modification of nutritional intake and assimilation of nutrients due to inflammatory process[2] (Figure 1).

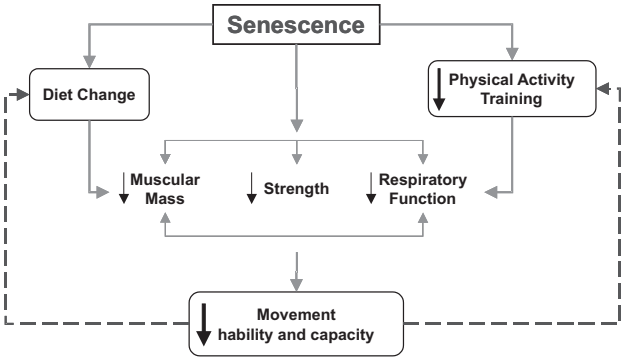

Fig. 1 Algorithm of the impact of aging, diet and exercise

Sarcopenia defines the relationship between the levels of strength and muscle mass. In this sense, it is known that the amount of peripheral muscle strength is closely related to lung function[2,3] and mortality in patients with respiratory disease[4]. We assume that the limbs strength depends to some extent on the quality and condition of the muscles of the respiratory system, whose application depends on the state and quality of the respiratory function[2]. If we consider that the absence of use leads to atrophy, it is easy to assume that the person who decides voluntarily to reduce their activity or a pathology brings him to rest, enters into a vortex of denaturation effect of the skeletal muscles, reducing the ability and the motivation to move, and thus accelerating senescence. It is helpful to know those studies where aging individuals with a certain presence of low levels of limbs strength, the mortality does not increase when the variables age, sex, education, nutrition level and muscle mass are introduced as relevant variables. Furthermore, the association of respiratory muscle strength and mortality decreases two quartiles when it is taken into account also the pulmonary function, irrespective of whether is associated a aging disease as chronic bronchial disease, Alzheimer, Parkinson symptoms, vascular or inflammatory disease, etc[2]... Sumarizing, pulmonary function is an important factor in relation to senescence as there is a very close relationship between the levels of muscle mass, and periferal and respiratory strength and function. The value and importance of this concept is unknown, but certainly encourages us to work on those aspects that maintain the respiratory function under optimal condition throughout exercise. The use of the whole respiratory chain from the mithocondria to the lungs, its solicitation through exercise and a proper nutrition are basic to limit the senescence of the system[2,3].

Discussion

The respiratory system senescent process compromises their different perspectives depending on the extent that affects various organs and their relationship to each other.

The osteoporotic process, the vertebral collapses, calcification of the costal and chondrosternal cartilages and the increased dorsal kyphosis associated with age, decreases the ability to expand the rib cage during inspiration and brings the diaphragm in a bad position to generate effective contraction. Moreover, the decrease in type II fibers and impaired mitochondrial function causes a 12–25% lower levels of force measured by transdiafragmatic pressure when compared healthy young and old subjects, maximum inspiratory pressure, maximum voluntary ventilation or is also altered according to studies. Otherwhise, the mechanical disruption compliance and increased residual volume, also suggests an impediment to fully empty the lungs in those individuals.

The residual volume increase occurs by an alteration of the lung parenchyma due to degeneration of the elastic fibers that surround the alveolar duct. This process, which starts at 50's, results in a premature closure of small airways during basal breathing creating an air trapping and increased airspace of a hyperinflation widely known as »senile emphysema«.

When we measure the lung function capacity to generate volume, flow or gas exchange, we observe that the aging process acts on each of these categories under different magnitudes. Most of the transverse studies are intended to provide the normal reference levels. The lung volumes and strength, regarding FEV1, are estimated to decrease at a rate of 25–30 mL/year from age 35 and 60 mL/year from 70 in healthy subjects. Although it should be noted that it is not easy to establish a healthy respiratory condition levels in seniors, who in one way or another are affected by this variable.

The respiratory chain begins at the mithocondria, a complex energy system of production and supply, and ends at the multicellular system, the lungs, which serves to provide the energy substrate and remove the key residue, oxygen and carbon dioxide respectively. In the middle of the chain, the transport system, the blood, the

muscle, the nutrition and the coupled systems and organs that maintain everything alive are a central and peripheral factors that as time passes are modified by senescence, or pathologies.

As some advanced age is reached the cardiac output decreases[15,16] due to the difficulty to achieve a high maximum frequency as the stroke volume is smaller. In that sense, it is estimated that in healthy individuals, and in active athletes, maximum heart rate is reduced 2 beats per 3 years of age from adolescence[17] while stroke volume only from 10 to 20%[15].

The supply of energy by aerobic cells occurs predominantly through oxidative phosphorylation. This complex mechanism summarized in the donation of electrons from the Krebs cycle, which reduces the water and oxygen and produce a potential generated in the mitochondrial intermembrane which is used by the ATPsintetasa to phosphorylate ADP and ATP yield. Aging seriously impacts mitochondrial function by altering both the capacity and the control of oxidative phosphorylation. In turn there is an age-accumulation of DNA damage, undoubtedly produced by exposure to free radicals, which alters protein synthesis, especially myosin heavy chain. This makes the effectiveness and efficiency of the respiratory chain smaller as time passes. The response to a workload is more or less tolerated as a perspective of molecular adaptation[18].

Besides this molecular bioenergetic aspect, undoubtedly the most striking effect in limiting the activity is sarcopenia, a notion that was discussed previously. The destructuring muscular depends on the high reduction of the glycolytic muscle fibers, the type IIa and IIb, due to the reduction of the voluntary inactivity solicitation, to the decrease of high intensity stimuli, the decreased con-

TABLE 1
SYSTEMIC ADAPTATIONS TO AGE, TRAINING AND DIET IN THE SENESCENCE

Variable	Aging	Training	CR + Ex*
Platelet aggregation	Increases	Decreases	Decreases
Plasma viscosity	Increases	Decreases	Increases
Arterial pressure	Increases	Decreases	Increases
Herat rate variability	Decreases	Increases	Increases
ltArrhythmia risk	Increases	Decreases or remains unchanged	Decreases
Triglycerides level	Increases	Decreases	Decreases
HDL level	Decreases	Increases	Increases
Omega 6/3 Index	Increases or remains unchanged	Decreases	Decreases
Vascular stiffness	Increases	Decreases or remains unchanged	Decreases
Bone Mineral Density	Decreases	Decreases or remains unchanged	Increases
Imflamatory markers	Increases	Decreases or remains unchanged	Decreases

*RC+Ex caloric restriction (specific diet) plus exercise

TABLE 2
MUSCULOSKELETAL ADAPTATIONS TO TRAINING AND SENESCENCE

	Aging	Training	CR + Ex*
Muscular Mass	Decreases	Decreases or remains unchanged	Decreases or remains unchanged
Abdominal fat	Increases	Decreases or remains unchanged	Decreases
Fiber Type I (%)	Increases	Remains unchanged	Remains unchanged
Fiber Type II (%)	Decreases	Remains unchanged	Remains unchanged
Fiber Type I (area)	Remains unchanged	Increases	Increases
Fiber Type II (area)	Decreases	Increases	Increases
Mitocondrial density	Decreases	Increases or remains unchanged	Increases
Oxidative capacity	Decreases	Increases	Increases
Glycolitic capacity	Decreases or remains unchanged	Remains unchanged	Remains unchanged
Capilar density	Decreases	Increases	Increases
Contraction time	Increases	Decreases or remains unchanged	Decreases or remains unchanged
Relaxation time	Increases	Decreases or remains unchanged	Decreases or remains unchanged
Shortness velocity	Remains unchanged	Increases	Increases
Tendinous stiffness	Decreases	Decreases or remains unchanged	Decreases or remains unchanged

*CR+Ex Caloric Restriction (specific diet) plus exercise

centration of neuromuscular hormonal disturbance, decreased recruitment of motor units fibers attached to the type II[19]. The changes of the myofibrillar population rates are the reason why the respiratory muscles of older individuals have a much higher redox potential than those from youngest individuals[20]. It should be argued that, unlike popular belief, the response to a work intensity responds with a concentration similar to testosterone producing a young subject to the same relative intensity[21] even that levels of anabolic hormones in plasma are lower as we get older[22]. This explains why the response of muscle mass, strength and architecture really works and persists when the stimulus is enough and maintained with age.

Senescence changes also occur in the connective tissue and the most characteristic is the loss of tendon tone and stiffness, which reduces their efficiency by limiting the transference of force from the contractile segment to the skeletal union. Another aspect that affects peripheral muscle tissue is the arteriovenous oxygen difference, which clearly decreases in the none active subject through time, and is consistent with the decrease in capillary density and mitochondrial enzyme activity.

Other peripheral issues involved in the process of aging are related to arteriovenous system rigidity and inability to adapt to certain requirements, such as those related to exercise, increased fat mass and decreased lean mass that, in turn, determines the change in volume of blood.

Conclusion

The progressive deterioration of most of the biological functions by aging of the body are somewhat less pronounced in people who practice a continuous and structured exercise compared to those who are sedentary. The subject who usually practices physical activity lives under the appropriate physiological environment for which we are prepared, keeping it moving to maintain the living organism in the broadest meaning of the word.

The exercise influences in the respiratory chain not only as a therapeutic tool, but also preventing sarcopenia as stimulates positive adaptations in skeletal muscle in adults of any age, increasing muscle strength, mass, protein synthesis and thus muscle composition, improving efficiency and recovery of injured muscle[23]. Exercising adult nutrition improves in quality and decrease in quantity[24], facilitating a process of energy efficiency and optimization of aging acts to increase the longevity and quality of life. When we measure the subjects ability by the reference standard, and we observe the maximum oxygen consumption, we note that, in absolute terms, the ability to generate response by mitochondrial substrate is not affected by age, when youth and elderly groups maintain similar oxygen consumption levels. The reason for this similarity is that seniors who exercise and maintain a good condition, have a higher content of mitochondria than younger subjects[25]. »More and more«, namely more number and smaller. That is why senior exercisers, with the muscular capillarization and the mitochondrial density, are capable of extracting a rate of oxygen from the blood to the same extent and efficiency as younger athletes[26].

The elderly skeletal muscle adapts well to endurance works, long duration and low or moderate intensity. But while this work model is good and desirable, it is not enough to maintain muscle mass and function as it grows older. To this end it is advisable to make a work of power, using free weights, closed chain systems, working with the body, etc... This type of stimulus, high intensity, short duration and repeated on a same session, maintains muscle mass levels, the different strength manifestations, shrinkage characteristics, histology and muscle architecture of individuals over 65 years, in a way equivalent to that of inactive young subjects[27,28]. The follow up of senior athletes that introduce power working session under their schedule allows us to observe that they achieve a senescence with a better quality of life and less involvement of different organ systems when there is a coupling disease that had to accelerate the process. Therefore it is appropriate to practice an endurance workout, adding the enough intense stimulus power to keep cell turnover

TABLE 3
RECORDS OF VETERAN ATHLETES BY AGE RANGE IN THE CURRENT REFERENCE AND THE TEST AT THE FIRST OLYMPICS

Event	Age range (years)				Olympic Games 1896		Olympic Record
	60–65	65–69	70–74	75–80	Record	Age	July 2012
100 m	11.70	12.00	12.70	13.50	12.00	21	9.70
200 m	24.00	24.73	26.48	27.97			19.32
400 m	53.90	56.40	59.30	01:05.0	54.20	21	43.20
800 m	02:08.6	02:14.3	02:20.5	02:32.5	02:11.0	23	01:41.1
1500 m	04:24.0	04:39.9	04:52.9	05:20.0	04:33.2	23	03:26.0
Marathon	2:36:30	2:41:57	2:54:58	3:04:53	02:58.5	23	02:04.3
Long Jump	6.1	5.5	5.2	4.8	6.4	22	9.0
Triple Jump	12.7	11.9	10.7	10.1	13.7	28	18.3

and ultimately a degree of hyperplasia and hypertrophy. The moderately high intensity work requests improves the quality and facilitates the transfer of tendon strength and physical sufficiency in these subjects[29].

It is assumed that the practice of sports like rowing, kayaking or swimming, where the movements of the muscles of the upper limbs are fundamental to request mobility, makes the ribcage under it widest range of movement during the training sessions that will prevent the immobility of senescence, and maintain paravertebral and accesory muscles more stimulatie that would be by a sedentary subject. Those effects are observed in anthropometric and functional aspects[30,31], but it would be desirable a cross-sectional study to evaluate the hypothesis on the structural and elastic component of the ribcage in these senior athletes which requests that upper trunk muscles, and supposedly bring the respiratory cage to a better functionallity and less age related changes. Senior athletes are the best model of aging the skeletal system on better conditions and optimal physical performance. Perhaps anecdotal, the scope of sports performance training even though the age is stimulating. At table 3 it is shown the results of the current record as the age range of Masters athletes compared to those made in the first Olympic Games in 1896 and the present time.

Finally, we must learn about the the impact of nutrition on senescence. This is not the objective of this article but is mentioned as is a relevant part of the process. In front of a maintained diet with a high energy intake, with elevated level of fat and protein, the decrease of the caloric intake, decreasing the intake of certain carbohydrates and fat, facilitating increased omega 3 fatty acids and decreasing the intake of omega 6, adapting some quantity and animal protein intake and increasing intake clearly fruits, vegetables and legumes, will make the aging process under the healthier regimen[32,33] and helping the of energy from the respiratory chain during exercising and living.

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STARENJE RESPIRATORNOG SUSTAVA I NJEGOVA POVEZANOST SA TJELESNOM AKTIVNOŠĆU

S A Ž E T A K

Rad je pregledni znanstveni članak koji istražuje učinke starenja respiratornog sustava i cilj mu je pružiti pregled učinaka vježbanja na način na koji se odvija starenje tog sustava. Nekoliko je razloga koji objašnjavaju nedostatak vježbanja u starosti, smanjena potreba, smanjen interes, drugačiji ciljevi, ali i deficijencije koje podupiru ove odluke, kao što je umjerena ravnoteža, koordinacija, snaga ili poremećaji vida. Korištenje cijelog respiratornog sustava od mitohondrija do pluća, njegovo poticanje kroz vježbu i pravilnu prehranu temelj su u ograničavanju starenja sustava. Kapacitet za obnavljenje i adaptaciju procesima vremena, izloženosti okolini i korištenja definirat će brže, teže i beznandno ili sporije i ugodnije starenje. Nedostatak kretanja, smanjenje funkcije bilo kojeg organa ili sustava dovodi do neučinkovitosti, atrofije i smrti.