Original Article: The Main Determinant of Muscle Strength and Endurance with Endurance Training and Effectiveness

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ABSTRACT

Experimental studies show that if the weight loss program is done only through diet and calorie restriction, 35-45% of the total weight loss will be allocated to the reduction of lean tissue, which will also reduce the weight of the heart. However, if the diet is combined with endurance exercise, the size of the lean tissue will not change much, but body fat will be significantly reduced. Metabolic mechanisms through which exercise reduces or maintains body weight include increased energy expenditure, increased fat recall, increased adipose tissue activity, and a slight increase in resting metabolic rate after exercise. Exercise increases the likelihood of an exothermic response to food if exercise and eating time are close together, minimize net weight loss, expand mental function, delay basal metabolic rate reduction due to nutritional limitations, and probably better control the appetite. Exercise is important because it helps maintain a resting metabolic rate and lean mass. Regular exercise can help control your appetite or lose weight mentally.

Introduction

Such changes are attributed to exercise in response to the stimulation or mobilization of fat cells, and this effect is facilitated by increased activity of the autonomic sympathetic nervous system. The ability to reclaim fat mass will continue even after the end of exercise and will remain intact with the availability of fat stores of pure tissue. Exercise also slows down the synthesis of fatty acids in adipose tissue [1-3] (Figure 1).

In the first years of life after puberty, exercise plays an essential role in controlling and inhibiting the increase in adipose tissue mass of active people far less than sedentary people [4-7]. Significant changes in body composition occur following aerobic exercise. Body weight typically decreases over a long period of 3 months or more. But during the first months of exercise, minor changes in body weight are not uncommon [8-10]. This is due to changes in body composition. Decomposition of body fat changes very little with a similar increase in net weight, and at this time, weight loss is a true

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reflection of body fat changes [11-13]. The number of training sessions is a determining factor in changing body composition. Exercising for 3 and 4 days a week leads to significant changes in body composition. While the effect size of programs with a frequency of 2 days a week is very small [14-16]. It is possible to estimate a certain threshold for the minimum amount of energy expenditure in each exercise of daily or weekly exercise or for the duration and intensity of work, which causes major changes in net weight and fat mass. Increasing the number and size of mitochondria is probably the best adaptation of muscular endurance to endurance training [17-19]. After endurance training, the size and volume of the mitochondria increase, increasing the cross-sectional area of the mitochondria exposed to the cytosol. Increasing the volume of mitochondria increases the concentration of mitochondrial enzymes such as TCA and beta-oxidation cycle enzymes [20-22]. The electron transfer chain is coupled to ADP phosphorylation, increasing the cell concentrations of electron-containing molecules containing the electron transfer chain. It should be noted that increasing the concentration of the enzyme is proportional to its activity [23-25]. As a result, increasing the volume of mitochondria increases the ability of a skeletal muscle to maximize oxygen consumption. Endurance training increases plasma volume and thus blood volume, thereby increasing blood flow back to the heart, and increasing end-diastolic volume [26-28]. This effect will increase the contractile force of the heart during cardiac systole due to the Frank-Starling law of the heart and due to the stretching of the muscle fibers of the ventricular wall and the reaching of the sarcomeres to their normal length (2.2 μm) [29-31]. Endurance exercise causes the ventricular cavities of the heart to become larger and the free walls of the ventricles to increase proportionally [32-34]. Because with the increase in blood volume and the return of venous blood, the number of cardiac arrests increases, due to the increase in the volume of the diastolic end of the ventricles. As the heart works harder, it will become hypertrophic and expand in its ventricular cavities (Figure 1 and 2) [35-37].

Figure 1. Muscular strength, Endurance workout, Strength and conditioning

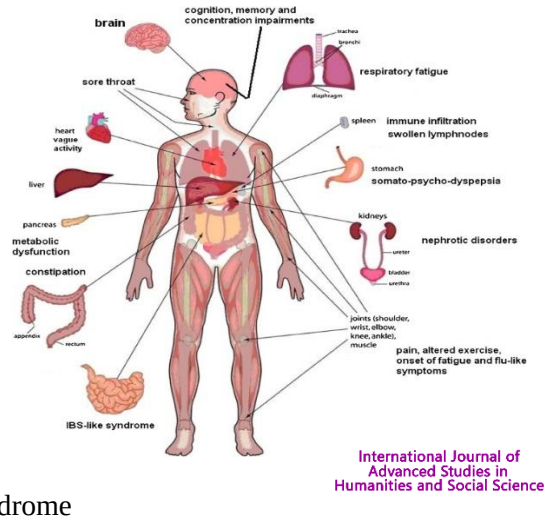

Figure 2. Chronic fatigue syndrome

The Effect of Endurance Training on Blood Pressure

Although the role of regular exercise in improving hypertensive conditions is not yet clear, it is concluded that an aerobic program can reduce systolic and diastolic blood pressure to a moderate extent. Such a result was obtained with subjects with normal blood pressure and hypertension at rest [38-40]. A decrease in mean arterial pressure has also been observed during sub-maximal exercise in healthy middle-aged men after endurance exercise [41-43].

Hogberg and Sales (2018) reported that in most research findings, exercise significantly has reduced systolic blood pressure and in other studies it has significantly reduced diastolic blood pressure, rest in patients with basal hypertension [44-46]. For example, the mean systolic blood pressure dropped to 153 mm Hg before exercise and to 142 mm Hg during the recovery period. On the other hand, throughout the training period, the diastolic pressure decreased from 94 to 86. Of course, part of this drop in blood pressure has coincided with a simultaneous decrease in body weight [47-49].

In people with normal blood pressure, regular endurance exercise has been shown to have a protective effect against the spread of high blood pressure later in life. The mechanisms involved in explaining the changes in blood pressure resulting from regular aerobic exercise are not very clear [50]. However, factors such as decreased sympathetic nerve tone during relaxation (decreased sensitivity of pressure receptors), changes in myogenic structures and tone, and decreased cardiac output have been shown to lower systolic blood pressure. There is ample evidence that endurance exercise reduces endurance cardiac output and peripheral vascular resistance in people with hypertension [51]. Decreased peripheral vascular resistance may be due to decreased light concentrations of epinephrine or changes in renal function due to exercise. On the other hand, the reason for lowering blood pressure due to endurance training can be the reduction of Catecholamines produced due to exercise [52-54]. This reaction contributes to the reduction of environmental resistance to blood flow and consequent lowering of blood pressure. Exercise can also facilitate the excretion of sodium from the kidneys, thereby reducing fluid volume and blood pressure [55-57]. Numerous studies have shown that regular exercise reduces Baroreflex control, reduces sympathetic nerve traffic, and thus lowers blood pressure [58-60]. During low- to moderate-intensity exercise (50-50% of maximum heart rate), there is an effect on diastolic blood pressure, which is likely to occur in low-intensity exercise before high working pressure leads to a significant increase. In the cardiac output, a decrease in total peripheral vascular resistance occurs, leading to a decrease in blood pressure [61-63]. Losing body fat is probably the key to losing weight. But this relationship is not necessary [64-66]. Because in the study of the treatment of hypertensive patients with exercise, patients who have lost weight have shown a reduction in blood pressure, and not all patients who have had a definite reduction in blood pressure have also lost weight [67-69]. The relaxation response, characterized by hypotension after exercise, is another possible mechanism. Blood pressure is regulated by a complex interaction between neurological, endocrine, renal, cardiovascular, and behavioral functions. Cardiovascular factors such as cardiac output and peripheral vascular resistance are the main determinants of blood pressure [70-72]. Cardiovascular control of blood pressure is primarily performed by neural regulation. This is followed by endocrine regulation and renal and behavioral factors. This response is important because neural regulation must intervene rapidly when hypertensive disorders carry the risk of immediate systemic changes in systemic blood pressure (Figure 3) [73].

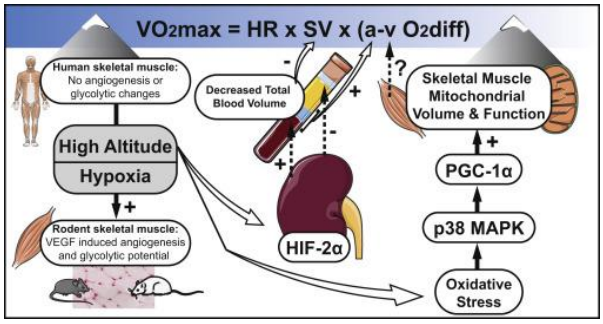

Figure 3. Maximizing Cellular Adaptation to Endurance Exercise in Skeletal Muscle

The Effect of Endurance Training on Heart Rate

Heart rate is one of the simplest and most alarming variables of the cardiovascular system. Endurance training reduces heart rate during rest. In normal healthy people who do not exercise, the resting heart rate may reach 60-100 beats per minute or more. Resting heart rate can be reduced to 35 beats or less with endurance training [74]. As a result, trained people are known to have a lower heart rate at rest and non-trained people are known to have a higher resting heart rate. If the inactive person has a resting heart rate of 80 beats per minute, in the first few weeks, for each week of exercise, his or her heart rate will decrease by approximately one beat per minute. Therefore, after ten weeks of moderate-intensity endurance training, the resting heart rate decreases from 80 to 70 beats per minute [75]. The exact mechanism of this reduction is not known. However, exercise seems to increase the activity of the parasympathetic nerve and on the other hand reduces the activity of the sympathetic nerve. Exercise creates an imbalance between the tonic activity of sympathetic and Neurosympathetic neurons in favor of greater vagal dominance. This is mainly due to an increase in parasympathetic activity and perhaps a decrease in sympathetic drainage. In addition, exercise may also reduce the intrinsic rate of stimulation of the S-A node. Such adaptations result from decreased heart rate, which is often seen in fully trained endurance athletes or inactive individuals following aerobic exercise [76-78]. The slowness of resting heart rate as a result of exercise is likely to consist of two main components:

a) Slow reduction of the intrinsic rate of Atrioventricular, node S-A. This in turn may be related to the increase in the amount of acetylcholine (a parasympathetic nerve transmitter) seen in the atrial tissue after exercise, as well as a decrease in the sensitivity of the ventricular tissue to Catecholamines, which are a group of chemicals including parasympathetic neurotransmitters, epinephrine, and light epinephrine [79].

b) Increased parasympathetic influence (Vagi) on the amount of productivity as a result of decreased sympathetic activity. In other words, it is thought that the increase in parasympathetic effect compared to the main decrease in sympathetic nervous system activity resulting from exercise in degree. The second is important.

Respiratory Adaptations with Endurance Training

Recognizing the structural adaptations of the lungs of a young adult is more difficult than endurance training. In general, lung volumes and capacities change slightly as a result of exercise. Vital capacity, i.e. the amount of air that can be expelled from the lungs after a deep breath, increases slightly. The remaining volume, i.e. the amount of air that cannot leave the lungs, is slightly reduced. The changes in these two volumes may be related. The total capacity of the lung remains essentially unchanged. After the endurance program, the resting volume does not change, but increases maximally during the performance of sports exercises. Endurance training reduces resting breathing rate, which is probably a reflection of the greater lung function resulting from exercise and may increase with maximal exercise. Also, athletes with adequate endurance training have a wider capillary contact surface with more pulmonary capacity and as a result have more gas exchange capacity at rest and exercise. However, research has shown that animals exposed to hypoxia develop about 20% more lung volumes than other animals. The increase in pulmonary volume appears to be mainly due to the enlargement of the existing

pulmonary vesicles. There have been reports of exercise causing dilatation of the pulmonary vesicles. It has also been reported that the bubble walls become thicker due to cell proliferation and consequently, the number of pulmonary bubbles increases. The changes that occur in the lungs as a result of exercise are gradual and occur over 4-6 weeks, leading to greater ventilation efficiency. More ventilation efficiency means that the amount of ventilated air at the same level of oxygen consumption is less than non-athletes. Because the cost of ventilation oxygen increases significantly with increasing ventilation, higher ventilation efficiency, especially during prolonged activity, will result in less oxygen distribution in the respiratory muscles and more oxygen in the active striated muscles [80].

The inability to predict the athletic performance of healthy individuals was obtained through practical lung parameters in large groups of adolescent girls and boys. When lung volume and capacity matched body size, no significant relationship was observed between lung function and different running functions, including endurance. In the study of marathon runners, there was no difference between values related to practical lung parameters such as vital capacity with pressure, total lung capacity, the volume of exhalation with pressure and its percentage, maximum voluntary ventilation, current volume, and respiratory rate. Athletes and inactive people were not the same sizes. When differences in body size are noted, there is no association between maximal oxygen consumption and vital capacity or maximal ventilation volume in healthy untrained subjects. It seems that the natural capacity of pulmonary ventilation does not limit exercise performance. Larger-than-normal lung volumes and respiratory capacity are usually attributed to differences in hereditary traits and may indicate stronger respiratory muscles as a result of special exercise. It has been well established that maximal voluntary ventilation can be increased by exercise, and it is not really surprising to see values of about 200-250 liters per minute for highly trained athletes, which is why they are able to move quickly [81].

The Effect of Endurance Training on Creatine Phosphate Reserves

Human muscle Phosphagen stores consist of approximately 15 mmol/kg (of muscle fresh weight), phosphocreatine, and 4.5 mmol/kg adenosine triphosphate (ATP), which is a very limited energy source. The usefulness of this device is its speed of operation and reconstruction. In general, Phosphagen stores in fast-twitch fibers are slightly richer than in slow-twitch fibers. Muscle Creatine phosphate stores are not as compatible with exercise as other energy stores, but any additional increase in Creatine phosphate stores can significantly increase exercise performance and strenuous exercise. It has been suggested that there is a type of Creatine phosphate metabolism and Creatine itself is a stimulus for improving Vo2 in exercise. The importance of the Phosphagen apparatus can be seen in powerful movements, rapid beginnings of speed champions, soccer players, high jumpers, weightlifters, and similar activities that take only a few seconds to complete. Without this device, fast and powerful movements will not take place, because these activities require rapid storage of ATP energy instead of large amounts of ATP energy. This device represents the fastest and most accessible source of muscle ATP. Because it does not depend on a series of long reactions, it does not depend on the transfer of respiratory oxygen to active muscles, and both ATP and PC are stored directly in muscle contractile proteins. Long-term endurance exercise mainly uses skeletal muscle Creatine phosphate to transport phosphate molecules from mitochondria to contractile proteins [82-84]. The resulting Creatine is then regenerated in the mitochondria to be reused. But the usefulness of such a shift in endurance sports seems to be small. The reduction and depletion of the Phosphagen apparatus for certain tasks are less in endurance trainees than in beginners. In addition, the amount of ATP and CP increases with endurance training in the muscles. Information on the short-term adaptations of Creatine phosphate during increased exercise shows that Creatine phosphate stores do not decrease at training intensities equal to or less than 60% max vo_2. This amount of exercise intensity does not stimulate the increase of Creatine phosphate reserves. In fatigue, the pattern of Phosphagen depletion is the same for endurance exercise that lasts between 2-20 minutes [85-87]. Phosphagen depletion during intermittent endurance exercise is greater than in continuous endurance exercise. In addition, the depletion of

Creatine phosphate in the initial minutes of endurance work is below its maximum at its maximum [88]. The decrease in CP is relatively greater than the decrease in ATP. It is sometimes seen that CP is depleted by up to 60% during exercise, but there is still no change in ATP. This is because CP is used for the rapid regeneration of ATP. Phosphagen depletion is variable and heterogeneous among FT species [89].

Conclusion

The main factor that determines the strength and power of the muscles is their size or, more scientifically, the cross-sectional area of the muscle itself. Nearly 50% of the observed changes and inequalities between the muscle strength of different individuals can be justified and defined on the basis of different and unequal sizes and cross-sectional areas of their muscles. Aerobic activities such as jogging and endurance cycling are largely dependent on slow-twitch activity. In response to exercise-induced stimuli, these fibers become 7-22% larger than fast-twitch fibers. Prolonged endurance training leads to muscle mass and is almost always associated with a capillary increase in the striated muscle. In addition, research has shown that in heroes, each muscle fiber is surrounded by an average of 5.9 capillaries, while in untrained individuals, it is surrounded by an average of only 4.4 capillaries. The number of capillaries surrounding each striated muscle fiber is related to two factors: The size or diameter of the muscle fiber, and the type of fiber or the number of mitochondria in each muscle fiber. The intensity of work in endurance sports can vary. The use of a wide range of intensity in endurance training can lead to different patterns of muscle fiber hypertrophy. At low intensities, the muscle does not develop hypertrophy, but the slow-twitch fibers are used gradually. When the intensity of work is absolutely increased and the time is still long, after working for 4 to 5 months, the slow-twitch muscle fibers selectively increase their mass and cross-section and suffer from sports hypertrophy. Prolonged training time in each session, without increasing the intensity of work cannot cause hypertrophy and increase the cross-sectional area of these muscle fibers although it uses slow-twitch fibers; therefore, it increases in muscle fiber mass. Hypertrophy is strongly related to exercise, not its duration. In addition, experiments performed on the muscles of inexperienced, inexperienced, and young people show that increasing the intensity of exercise in these individuals also utilizes rapidly contracting muscle fibers. In these young people, in addition to the slow contraction fibers increase in volume and cross-section, the subgroup of rapid contractions also develops hypertrophy.

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