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Review Article

Aerobic Capacity and Regular Physical Exercise among Tobacco Smokers

Dr. Maha S. Almohannadi[1], Dr. Anees Alyafei[2]*

[1]Community Medicine Specialist, Hamad Medical Corporation, Doha, Qatar.

[2]Wellness program, Preventive medicine, Primary Health Care Corporation, Doha, Qatar.

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***Corresponding author:** Wellness In charge, Primary Health Care Corporation, Doha, Qatar. Mobile: +97455080077; E mail: qat900@yahoo.com

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Abstract

Background: The Aerobic capacity is a well-recognized indicator for cardiopulmonary fitness, which is affected by many factors, including tobacco use. Tobacco smoking is also known for its toxic effects on the cardiopulmonary system. Thus, aerobic capacity change is an exciting area for discussion among tobacco smokers, parallel to the cardiopulmonary effect. There is significant agreement that aerobic capacity is reduced in tobacco smokers and improves by regular physical exercise in a dose-related manner. Few scattered studies showed minimal or no effect of physical exercise on aerobic capacity. Further clinical trials need to assess the impact of physical exercise and related improving & suppresser factors on the aerobic capacity among tobacco smokers.

Abbreviations

AC Aerobic Capacity, BP Blood Pressure, CPS Cardiopulmonary System, CVD Cardiovascular Disease, PFT Pulmonary Function Test, PE Physical Exercise, RHR Resting Heart Rate.

Introduction

One major parameter for Cardiopulmonary System (CPS) fitness is Aerobic Capacity (AC). Aerobic Capacity is defined as the maximum Oxygen consumption during the maximum effort of the aerobic or cardio physical exercise [1]. The pathological effects of tobacco smoking on CPS are thoroughly studied [2]. Thus, parallel to the CPS effect, the change in AC is an exciting area for research and discussion.

This narrative review aims to gather all available information about the AC definition, assessment, and factors that affect it. Moreover, the evidence that could the regular Physical Exercise (PE) improve it.

Effect of Tobacco Smoking on Cardiopulmonary System

The effect of tobacco smoking on the cardiopulmonary system is illustrated in the medical literature. Tobacco smoking is the leading cause for cardiovascular disease (CVD), including coronary heart disease, which is the leading cause of mortality worldwide, an independent major risk factor for total

atherosclerotic CVD, cerebrovascular disease, peripheral vascular disease, cardiac failure, abnormal lipid profile, hypertension, and all-cause mortality, in dose-dependent relationship [3-8].

In addition to the CVD effects, there is a well-recognized spectrum of pulmonary diseases related to tobacco smoking. Chronic obstructive pulmonary diseases, chronic bronchitis, emphysema, and bronchial asthma are among the most common sequelae of tobacco smoking. Furthermore, it leads to significant disturbance in all the lung volumes [9]. The overall toxic effect of tobacco smoking will interrupt the average Oxygen consumption in the body.

Physical Exercise Effect on Cardiopulmonary System

Physical Exercise is defined as repeated and structured movements of muscles or groups of muscles that reach either health or skill-related benefits. The health-related benefits are change in; body composition, CPS fitness, i.e., AC or VO_2 max, muscle endurance & strength, and flexibility. In contrast, the skill-related benefits are agility, coordination, balance, power, reaction time, and speed. Physical exercise is part of physical activity, which includes all the movements of an individual throughout the day [10].

The consequences of the regular PE on the CPS are well recognized in the literature. It is not limited to the improvement in heart rate, systolic blood pressure, peripheral resistance, VO_2 max, lung capacity, anaerobic capacity, and peripheral blood flow. It includes improvement on the vascular walls, reduction in arterial stiffness, microcirculation on the deep tissues, reconstruction of the inner vascular compartment, and lowering the risk for micro

ischemic events at the peripheral circulation level. These effects are also supported by improving many metabolic parameters such as glycemic control, lipid profile, weight reduction, overall physical fitness, and loss of visceral fat [11, 12].

A possible explanation for the cardiopulmonary effect of PE could be the followings, as shown in (Figure (1): [13-15], (Figure 1): The mechanism of physical exercise on improving the cardiopulmonary system. The drop in resting cardiac rate is properly secondary to the heart muscles' training on more blood needs, which ultimately cause a relative physiological rise in the left ventricular muscle mass. Improvement in BP is due to increased sensitivity of aortic baroreceptors, contributing to the regulation of systolic blood pressure. Moreover, diastolic B.P is enhanced due to the peripheral resistance reduction which results from PE-associated vasodilatation effect. Reduced action of the sympathetic nervous system and the renin-angiotensin system were also documented.

There is a parallel improvement in overall pulmonary function due to the demands of Oxygen and cardiac function. All the related lung volumes are increased on patients following eight weeks PE program as studied by Saki et al., Where the people with diabetes had three sessions a week of moderate-intensity exercise (45-60) minutes. The improvement in the pulmonary function was due to an increase in forced vital capacity as well as forced expiratory volume [16].

Further evidence is going far more at the endothelial level. Many Authors agree on PE as one important factor for improving the endothelial function by a rise in NO activity, which exerts the anti-atherosclerosis effect [17].

Aerobic Capacity

It has many synonyms such as VO_2 max, which is widely used, peak O_2 uptake, maximum O_2 consumption, and maximum O_2 uptake. It is measured as ml/Kg/minutes by different physical tests in the gym or exercises physiology lab using a treadmill, cycling, rowing ergometers, or even steps by gradually increasing exercise intensity over certain period [18]. There is a wide range of tests to assess and estimate the AC. The most used are the Cooper running test, the differences in the maximal and resting heart rates multiplied by (15.3), Rockport Fitness Walking Test, the (1.6 km) walking test, and the bench step test [19].

Aerobic capacity is a valid representative of cardiopulmonary fitness and body ability to sustain prolonged PE in proportionate dose-response relation [20]. It is the outcome of the cardiac output and arteriovenous oxygen difference at exhaustion. A low AC is greatly linked to an increased probability for all-cause mortality, including CVD and other prevalent comorbidities [21, 22]. Many factors may alter the level of VO_2 max, as in (Figure 2). Genetics contributes up to (30%), while the advanced age reduces it by around (1%) yearly after the age of (25) years, the maximum could be at the age of (18) years [23, 24]. Generally, males have a higher AC value comparing to females [25]. The more critical influencer is, the more aerobic exercise the highest the AC value [26]. Moreover, the fatter percentage and sedentary lifestyle, the less AC value observed [27].

General health is crucial if the CPS affected through any pathology subsequently negatively affect the AC value. Tobacco

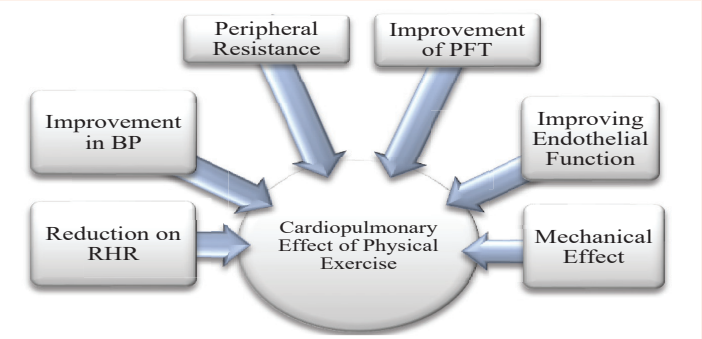

Figure 1: PFT Pulmonary Function Test, BP Blood Pressure, RHR Resting Heart Rate.

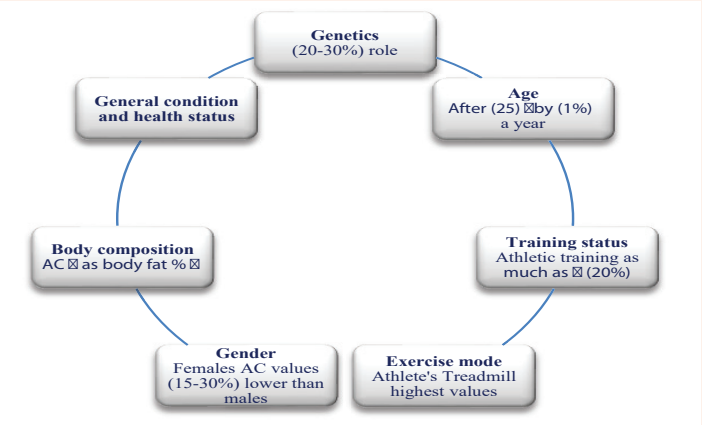

Figure 2: Factors Affecting the Aerobic Capacity.

smoking is a well-recognized risk factor that led to a broad spectrum of cardiopulmonary diseases, limiting the hearts and lungs' normal maximum physiological Oxygen consumption [28].

Evidence on the Effect of Regular Physical Exercise to Improve Aerobic Capacity among Tobacco Smokers

There is obvious agreement and almost consistent findings that AC is improving with PE among tobacco smokers. The AC was reduced due to tobacco use in a dose-related manner, as Putra and his team illustrated. The study among high school students demonstrated a higher AC level among non-tobacco smokers than more heavy smokers [29]. Similar findings were highlighted by Ricci *et al*. among the elderly population. The previous history of tobacco smoking, duration of smoking time, and history of obesity were crucially influencing the AC negatively [30]. Further confirmed data reported among smokers of asbestos workers by Wang and his team were more serious obstructive impairment illustrated in static lung function and more significant limitation during PE [31].

The fact of improved AC among x-smokers was concluded following PE program included in tobacco smoking cessation program as studied by Aparici and his colleagues [32]. Similar conclusion was described in the literature following cessation intervention or in compression between the active smokers and non-smokers [33, 34].

Repeated results were also seen with Tchissambou and his team, where the values of AC achieved by the football player smokers were significantly less than those achieved by the non-

smokers [35]. Consistent negative correlation between tobacco smoking and AC was also demonstrated by T Ingemann-Hansen. Likewise, the results of assessing the AC on chronic tobacco smokers in compression with non-smokers were found to be compatible with literature findings by others [36-39].

A few references found opposite results or minor PE effects on the AC among tobacco smokers. Wier et al. concluded that the effect of smoking on AC is minimal unless exceeding (20) cigarettes a day among both males and females [40]. Further, CHATTERJEE, Satipati, et al. suggested that smoking was not significantly influencing AC and only impaired it among the young age group [41].

Conclusion

The pathological effect of tobacco smoking on CPS is well presented. The accumulative evidence indicates a noticeable improvement of AC among smokers following a PE program. Structured PE could help significantly in a smoking cessation program. The recommended PE prescription of (150-300) minutes weekly is required to show up the effect of regular PE on the smokers AC. Aerobic PE training at moderate to vigorous intensity is more superior than resistant exercises on frequency of (2-3) times per week is recommended. The need for further clinical trials to evaluate the related factors that improve AC among tobacco smokers is vital.

Conflict of Interest

Authors have no financial interest, arrangement, or affiliation with anyone in relation to this narrative review that could be perceived as a real or apparent conflict of interest in the context of the subject of this study.

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