



Impact of smoking on aerobic capacity in young adult smokers

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ABSTRACT

Cigarette smoking is a worldwide public health challenge, Cigarette smoking is also a strong risk factor for musculoskeletal and cardiovascular disease, It is also well known that low and declining muscle strength is linked to increased smoking .[23]Aims of this study was to examine the chronic effects of smoking on cardiovascular fitness in young and healthy male smokers[13]. This study was carried out in university of hail ,physiotherapy lab, ,30male participant was recruited from university students of hail divided into two group 15 smoker (A) ,15 nonsmoker (B) .All subjects underwent a sub maximal Bruce treadmill test and their HR was recorded during, at peak, and after termination of exercise. Our study revealed that the resting HR was 5.3 bpm higher in smoker than in non smoker (P:0.0001), data indicated that there was a significant difference found between young smokers and non-smokers regarding their sub-maximal HR values (P:0.0063), where smokers had significantly higher HR values. also there was no difference between both groups regarding to recovery heart rate (P:0.56). Smoking was found to affect young smokers' increasing HR at rest, slowing of HR increase during exercise, and impairing their ability to reach the age predicted HRmax., Also smoking was associated with an attenuated HR. . also Smokers had a higher resting HR and showed a higher HR response during sub-maximal exercise compared to Non smokers .

Key words: aerobic capacity, exercise testing , smoking,

INTRODUCTION

Cigarette smoking has long been known as a serious topic in public health. and It has been increasing in many developing countries around the world.[1]Smoking is a major risk factor for cardiovascular morbidity and mortality, and was considered to be the leading preventable cause of death in the world. [2] Tobacco has more than 4,720 chemical products, nicotine was being the responsible for addiction, increase of heart rate (HR), blood pressure (BP) [3]and double product (DP)[4] which are alterations associated with the increase of cardiac work in smokers.[5],[6].The European Society of Cardiology reported recently that smoking causes 28% of cardiovascular deaths in men aged 35 to 69 years and 13% in women of the same age. [7]

Smoking either active or passive, can cause cardiovascular disease via a series of interdependent processes, such as enhanced oxidative stress, haemodynamic and autonomic alterations, endothelial dysfunction, thrombosis, inflammation, hyperlipidaemia, or other effects.[7]The prevalence of current smoking in Saudi Arabia ranges was from 2.4 -52.3%, among school student the prevalence of current smoking from 12-29.8%, among university student from 2.4-37% ,and among adults from11.6-52.3%. In elderly people the prevalence of current smoking is 25% The prevalence of current smoking in male ranges from 3-38% , while in female the ranges from 1-16% (median=9%).[8]Exercise tolerance, expressed as the peak workload achieved during a maximal treadmill exercise test, is a leading indicator of circulatory system capacity as it is strongly related with maximum O₂ uptake [9], which is an excellent noninvasive measure of maximum cardiac output and exercise capacity [10] [11].

It was well documented that smokers have tendencies to be less physically active than nonsmokers [12].so we hypothesises that there was a big relation between smoking and decrease in cardiovascular fitness

The purpose of this study was to examine the chronic effects of smoking on cardiovascular fitness in young and healthy male smokers[13].

MATERIALS AND METHODS

This study was done in university of hail ,physiotherapy lab,30 male participant were recruited from university students of hail divided into two group 15smoker (A) ,15nonsmoker (B) .

heart rate (HR) were non-invasively measured in young non-smoking and smoking subjects at rest, during the accomplishment of a submaximal exercise test and recovery period.

Measurements

- Heart rate measured manually
- Exercise test unite (treadmill and ECG monitor)

All subjects were underwent a sub maximal Bruce treadmill test and their HR was recorded during, at peak, and after termination of exercise.

Exercise test

1. All participants were exercised with the standard Bruce treadmill test. The sub maximal exercise test was used as an indirect measure of participants' exercise capacity.
2. Age-predicted target HRs were used as predetermined endpoints.
3. Testing was terminated at sub maximal effort or when symptoms such as intense exhaustion, fatigue, dyspnoea, or intense leg pain occurred.
4. All subjects were placed sitting immediately after termination of the exercise test for a 5-min recovery period.

RESULTS

Data analysis Statistical analysis was performed on the data obtained from 30 subjects . All statistical analysis was performed using SPSS for Windows 16.0; paired t-test was carried out to determine the significance of the outcome measurements in the two groups. P-value of less than 0.05 was used to determine the significance of the outcome measurements between the two groups .

In group A (smoker) was composed of 15subjects their mean ages ,weight and height were 20.2±1.11 years,75.8±7.2Kg ,173.9±5.2Cm. respectively ,In group B (nonsmoker) was composed of 15 subjects their mean ages ,weight and height were 20.4±1.2 years ,74.6±7.4 Kg ,174.4±5.4 Cm respectively.

There was no significant difference between both groups regarding to age, weight and height, with P value ; 0.75 ; 0.61 ; 0.75 respectively (table (1)and figure (1)

Table 1.Comparison between both groups regarding to age, weight and height

	Age	Weight	Height
Group A Smoker	20.2±1.11	75.8±7.2	173.9±5.2
Group B Non smoker	20.4±1.2	74.6±7.4	174.4±5.4
P value	0.75	0.61	0.75
Significance	N S	N S	N S

Table 2 and figure 2 showed the significant change between both groups regarding to resting heart rate in group A and B (75.7±4.06 versus 70.4±4.02)with P value;0.0001

Regarding to heart rate after exercise in group A and B, it was (134.4±6.5 versus 129.6±7.4)with P value; 0.0063) regarding to heart rate after 5 min recovery from exercise in group A and B, it was (84±7.7 versus 83±7.4)with P value;0.56

Regarding to time exercise in group A and B, it was (4.8±0.8 versus 6.4±0.9)with P value;0.0001

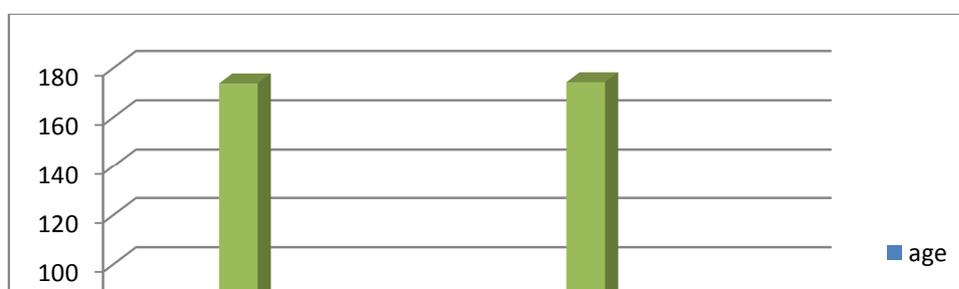


Figure 1.comparison between both groups regarding to age ,weight and height

Table 2.comparison between both groups regarding to HR rest, HRex, HR rec and EX duration

	HR rest	HR ex	HR rec 5min	Ex. Duration
Group A	75.7±4.06	134.4±6.5	84±7.7	4.8±0.8
Group B	70.4±4.02	129.6±7.4	83±7.4	6.4±0.9
P value	0.0001	0.0063	0.56	0.0001
Significance	S	S	N S	S

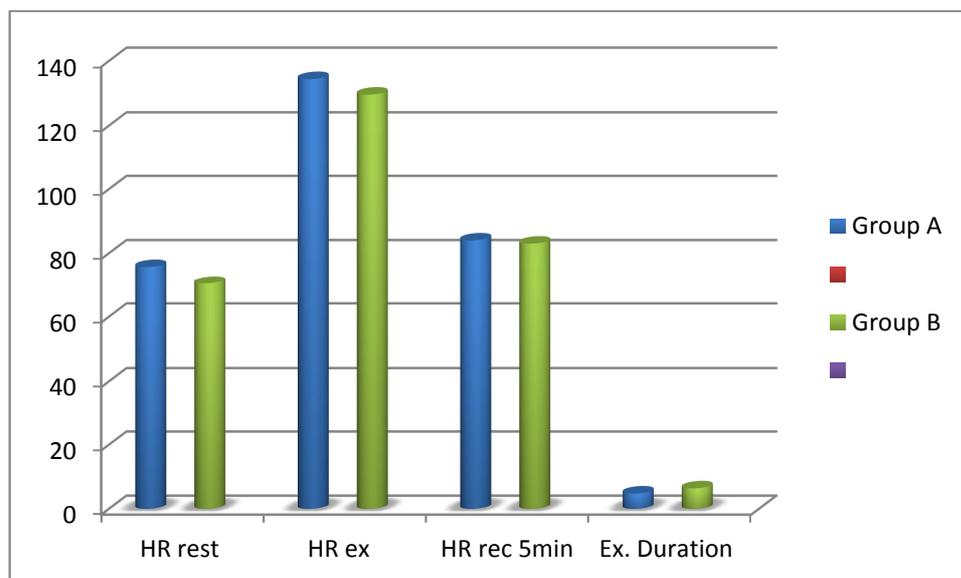


Figure 2. comparison between both groups regarding to HR rest ,HR ex ,HR rec and EX duration

DISCUSSION

Smoking is a major risk factor for cardiovascular morbidity and mortality, and is considered to be the leading preventable cause of death in the world. Based on WHO estimates, tobacco continues to kill nearly 6 million people each year, including more than 600,000 passive smokers, through heart disease, lung cancer, and other illnesses; that is one and a half million more than the corresponding estimate for 1990. If current trends continue, the death toll is projected to reach more than 8 million per year by 2030. [6]

Smoking is associated with an increased risk of all types of cardiovascular disease, including coronary heart disease, ischemic stroke, peripheral artery disease and abdominal aortic aneurysm. Internationally, 25% of middle-aged cardiovascular deaths are attributable to smoking.[21]

In the present study, smoking was found to affect the resting and exercise HR responses in young male smokers. Smokers had elevated HR rest, a slower HR increase during exercise, impaired ability to reach their age-predicted HR max when compared to non smokers .

Heart rate at rest

In our study, the resting HR was 5.3 bpm higher in smoker than in non smoker this agrees with the average resting HR difference found in most studies. These results are in line with previously published data from young populations, [13]and they are also in agreement with many HR-related studies of healthy middle aged populations, where smoking has been associated with increased resting HR values.[14] [15] [16]

Smoking is associated with autonomic dysfunction and with selective alterations in cardiac autonomic control. More specifically, smoking, acting at peripheral sympathetic sites, increases circulating levels of catecholamines, augments sympathetic outflow, and causes a long-term reduction in vagal drive. This sympathetic predominance, seen even in young heavy smokers, is also associated with impaired baroreflex function, leading to a marked increase in HRrest. [16]

Heart rate at sub-maximal workload

The HR value at a fixed sub-maximal aerobic workload (HRsubmax) is directly related with the increased metabolic demands imposed by the specific workload intensity. Thus, HRsubmax can be considered as an important marker of myocardial work, being inversely associated with exercise capacity ,and cardiovascular health. [18]

Our data indicated that there was a significant difference found between young smokers and non-smokers regarding their sub-maximal HR values, where smokers had significantly higher HR values.

There are few studies examining the effects of smoking on HRsubmax in healthy young adults. The results are confusing, since in some studies smoking was found to increase men's HR at a fixed sub-maximal workload, [14]

Whereas elsewhere it was suggested that smokers have lower HR at sub-maximal exercise, and others found no differences. Differences in methodology (e.g. definition of sub-maximal workload, HR evaluation protocol, selection criteria for smokers, etc.) might have contributed to these divergent findings. [19]

Heart rate decline during recovery

After the termination of exercise, sympathetic activity is withdrawn and vagal reactivation mediates the rate at which HR declines. HR decline during recovery is a useful marker of cardiac autonomic control, being directly associated with the intensity of post-exercise parasympathetic activity. [20]

In our study there was no difference between both groups regarding to recovery heart rate There are very few studies that have examined the association between smoking and HR rec in young adults. Kobayashi et al reported that young smokers had attenuated HR decline after sub-maximal exercise. [20]

CONCLUSION

Smoking was found to affect young smokers' HR, increasing HR at rest, slowing HR increase during exercise, and impairing their ability to reach the age predicted HRmax. In addition, smoking was associated with an attenuated HR decline during recovery. Smokers had a higher resting HR and showed a higher HR response during sub-maximal exercise compared to Non smokers .

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