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THE EFFECT OF SMOKING ON RESPIRATORY FUNCTIONS, ARTERIAL BLOOD PRESSURE AND MELATONIN LEVELS OF TRAINED SPORTSMEN

DEMİRCİ NEVZAT[1], BEYTUT EBRU[2]

Abstract

The aim of the study was to investigate the negative effects of smoking upon respiratory function, blood pressure, pulse rate and the levels $MaXVO_2$ and melatonin in sportsmen, and to investigate the importance of exercise in respect of these valves.

The research subjects were 14 male basketball players. The research group has divided into two goups; one comprising smokers (n=7) and the other non-smoker (n=7). Druing the research, both groups followed a training programme consisting of worming up, exercise and restin for 120 minuts every day, three days a week for two weeks.

The systolic and diastolic blood presure and the pulse rate at smoker sportsmen before and after training were significantly different from those of the non-smokers ($p<0.05$, $p<0.01$ and $p<0.001$ respectively). The VC, $MaXVO_2$, FVC and FEV1 values of the smokers, before and after the training, were found to be significantly lower than those of the non-smokers ($p<0.001$ and $p<0.01$, respectively). The melatonin levels increased noticeably immediately after the training, but reterned to the pre-training levels 2 hours after training. These increases and decreases were found to be statistically significant ($p<0.001$). Moreover, the levels of melatonin in the smoker-sportsmen in the second week of training when compared to the first week, were also significantly higher than those at the non-smokers ($p<0.001$).

İn coclusion, it was determined that whereas smoking plays a role in increasing systolic and diastolic, blood presure and pulse rate in sportsmen, it plays a role in decreasin respiratory function, $MaXVO_2$ and melatonin levels. On the other hand, it has been shown that systematic exercise has a regulatory influence upon such negative effects of smoking as the reduction in sportive performance, resporatory capocity and melatonin levels.

Key words: Cigarette, Respiratory function test, blood presure, pulse, Melatonin.

Introduction

Smoking is one of the most important risk factors for cardiovascular, respiratory and malignant diseases, and is a cause of preventable disease and death. It has long been known that there is a direct relationship between smoking, cigarettes and smoking-related diseases. Epidemiological studies have suggested that cardiovascular diseases and cause of death are related to the amount, type of cigarette smoked and the age of smoking (AC. Guyton., JE. Hall, 1996).

Active or passive smoking are known to result in chronotropic incompetence of the heart, reduced heart-rate during exercise, atherosclerosis in veins, (T. Conwy., TA. Cronan, 1992) decrease in alveolar ventilation, deterioration in diffusion capacity, increase in respiratory resistance (G. Tortora., SR. Grabowski, 1996) and reduced oxygen-carrying capacity of the blood. All these changes reduce quality of life of smokers by directly affecting exercise capacity or physical activity level. It is indicated that smoking also limits physical activity by decreasing the exercise tolerance level (G. Metin et al, 2005). The heart has an adaptation mechanism, which can change its performance according to different temporal, physiologic and environmental conditions. In this adaptation, either peripheral or central source neuronal and hormonal factors, such as melatonin, play a role (A. Altun et al 2001). Melatonin is known to have a lowering effect on blood pressure, vascular tonus and norepinephrine levels. This lowering effect of melatonin on blood pressure can be associated with sympathetic nervous system inhibition and postsynaptic α_1 adrenergic receptor blockage (A. Laflamme., J.Wu L, de Champlain, 1998). Despite different experimental results, it is indicated that there can be differences between exercise and melatonin level according to light exposure and the time of exercise during the day (G. Atkinson et al, 2003).

Furthermore, melatonin has the characteristics of directly detoxifying free radicals which have oxygen in their structure and their related reactants. Harmful radicals in cigarette smoke increase the oxidative damage in cells and tissues by deteriorating the oxidant-antioxidant balance; can affect plasma lipid profile and antioxidant enzyme level by leading to catabolism of cellular membranes and the inadequacy of physiologic functions (A. Altun et al 2001). In brief, smoking can increase lipid peroxidation by exposing the liver tissue to oxidative damage in sportsmen. Considering all this information, the present study investigated the harmful effects of smoking on respiratory functions, arterial blood pressure and melatonin levels of sportsmen and whether or not these effects have an exercise limiting role.

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Materials and Methods

The present study included 14 volunteer licensed male basketball players with the following characteristics: age range of 19 to 24 years, weight range of 65 to 85 kg and height range of 1.75 to 1.92 group was composed of seven non-smokers. This study met the ethical standards suggested by Harriss and Atkinson (DJ. Harriss., G. Atkinson, 2009), and ethics approval was obtained from the local institutional Review Board.

Throughout the study, the two groups were administered 120-minutes training three days a week for two weeks. The training program was organized specifically for the groups, in order to acquire the most convenient physiologic effects. This included type, volume, duration and frequency of the exercise. The training program was composed of three sections: warming up, exercise and cooling down. Weight and height were measured precisely, according to the method of Tamer (K. Tamer, 2000).

From the start of the study, blood samples were taken into tubes with EDTA before, immediately after and two hours after training. The blood samples were centrifuged in a cooling centrifuge at 3000 rpm for ten minutes. The remaining plasma was taken into covered polypropylene tubes; the samples were kept at -20 °C until the melatonin level was tested. Melatonin hormone levels were measured as cited in ELISA DRG KIT.

Systolic and diastolic blood pressures of the sportsmen were measured from a. brachialis of the right arm before and immediately after the training.

Participants' heart-rates were measured during relaxation, before the training and after the training (following the relaxation) using the method described by Tamer (K. Tamer, 2000).

Vital capacity (VC), force vital capacity (FVC), forced expiratory volume (FEV_1) and average flow rate in the half of the forceful expiration rate (FEF 25-75%) parameters were measured with a spirometer before and after the training. Spirometric data were revised according to Body Temperature and Pressure Saturated (BTPS) values (K. Tamer, 2000). European Coal and Steel norms were used in calculating the target values for age, height, weight, gender and race criteria.

A 12-minute walk/run test (Cooper) was used as an indirect method to predict maximum volume of oxygen consumption ($MaXVO_2$). The results were determined by multiplying the number of tours and the distance of each tour (400m) and adding the distance of the completed tour (meter). $MaXVO_2$ values were determined using the Balke formula (B. Balke, 1961).
MaXVO2 ml/kg-min = 33.3+(X-150) x 0.178 ml/kg-min, where
X= the distance run in one minute.

Statistical Calculations

The MINITAB statistical package was used to calculate the average ($\overline{X}$) and standard deviation ($s_{\overline{X}}$) of the data (Minitab Inc. Pennsylvania, Version 12.1 USA). The General Linear Model function of the same program was used to detect the analyses in the study. Analysis of variance (ANOVA) was used to compare the melatonin levels of smoking and non-smoking groups in the first and second week, immediately following training. Tukey's test was administered to identify statistical differences between groups.

m. The subjects were asked to provide medical reports indicating that there is not any medical harm for them to exercise. Participants were classified into two groups, the first group was composed of seven sportsmen who had smoked a minimum of ten cigarettes per day for at least one year; the second

Findings

1. Systolic and diastolic blood pressure levels of smoking and non-smoking sportsmen before and immediately after training (Graphic 1):

The increase in the systolic and diastolic blood pressures of smoking sportsmen during the pre-training period and immediately after training was statistically significant when compared with the non-smoking group (respectively, $p<0.05$, $p<0,01$).

2. Pulse rates of smoking and non-smoking sportsmen before and immediately after two-week training (Graphic 2):

The increase in the pulse rates of smoking sportsmen during the pre-training period and immediately after training was statistically significant when compared with the non-smoking group ($p<0.001$).

3. VC levels of smoking and non-smoking sportsmen before and after two-week training (Graphic 3):

The decrease in VC values of smoking sportsmen during the pre-training period and immediately after training was statistically significant when compared with the non-smoking group ($p<0.001$).

4. FVC levels of smoking and non-smoking sportsmen before training and after two-week training and statistical differences between the levels (Graphic 4):

When the pre-training and post-training FVC levels were compared, they were significantly lower in the smoking group than the non-smoking group ($p< 0.01$).

5. FEV_1 levels of smoking and non-smoking sportsmen before and after two-week training and statistical differences between the levels (Graphic 5):

The decrease in FEV_1 levels of the smoking group was statistically significant when compared with the non-smoking group ($p< 0.01$).

6. FEF 25-75% levels of smoking and non-smoking sportsmen before and after two-week training and statistical differences between the levels (Graphic 6):

There was no statistically significant difference between the smoking and non-smoking groups, in terms of FEF 25-75% levels before and after training ($p>0.05$).

7. $MaXVO_2$ levels of smoking and non-smoking sportsmen before and after two-week training and statistical differences between the levels (Graphic 7):

The decrease in $MaXVO_2$ levels of smoking sportsmen was statistically significant when compared with the non-smoking group ($p<0.001$).

8. Melatonin levels of smoking and non-smoking sportsmen before two-week training, immediately after training and two hours after training and statistical differences between the levels (Graphic 8):

Although the melatonin levels of non-smoking sportsmen were higher before training when compared with smoking sportsmen, the differences between the levels were not statistically significant ($p>0.05$). Nevertheless, significantly increasing melatonin levels of the smoking and non-smoking sportsmen immediately after the training decreased to pre-training values after two hours. These increases and decreases were statistically significant ($p<0.001$).

9. Melatonin levels of smoking and non-smoking sportsmen immediately after the 1st and 2nd week of training and statistical significances between the levels (Graphic 9):

The increase in melatonin levels of smoking and non-smoking sportsmen immediately after the 2nd week of the training was on $p<0.001$ level in non-smoking group.

Discussion

Insufficient exercise and smoking are important risk factors for ischemic heart diseases. Furthermore, it is indicated that smoking limits physical activity by decreasing the exercise tolerance level. Smoking a cigarette can increase the recovery heart rate by 10-20 beats per minute. It can increase the systolic and diastolic blood pressures, resulting in reduced maximum respiratory capacity among smokers (N. Akgün, 1986).

A study by Akgün (1986), it was reported that smoking decreases the systolic and diastolic blood pressure by reducing sporting performance. Similarly, the present study detected a significant increase in the systolic and diastolic blood pressures of smoking sportsmen in the pre-training and post-training periods (Graphic 1). This increase can be associated with the increase of blood pressure due to catecholamine, which is released by the adrenal glands with the effect of nicotine in cigarettes.

Heart rate is one of the most important factors in sporting performance. The present study also found that the increase in the pulse rates of the smoking sportsmen in the pre-training and post-training periods was statistically significant when compared with non-smoking sportsmen ($p<0.001$) (Graphic 2). Similarly, Düzen (1996) observed that the pulse rates of non-smokers who were actively involved in sport were significantly lower than those who smoke and do not participate in sport. According to these results, regular and graded training not only gives rise to a physiologic dilatation and hypertrophy in the heart of the sportsmen, but it was also established that it regulates the circulation and reduces the pulse rate, which increases immediately after the training to normal. Furthermore, it can be indicated that smoking, which decreases the beta-adrenergic receptors, increases the pulse in relation to increasing catecholamine and causes a negative effect by repressing the sympathetic system and lowering the noradrenalin response during exercise.

Vital capacity measurement provides beneficial information on the strength of respiratory muscles and lung functions (PG. Burstyn, 1990, , FA. Wilson, 1985). In the present study, the decrease in the VC values of smoking sportsmen before and after training was significant when compared with non-smoking sportsmen ($p<0.001$) (Graphic 3). Özgüner et al. (1998) also found that passive smoking decreases VC volume. On the other hand, Pringle et al. (2005) reported significant increases in VC values before and after a 10-km run. Consequently, it can be concluded that there are some differences in the physical capacity, organ and system functions of trained and untrained people over time and these differences always occurs in favor of people engage in physical exercise.

Some researchers (M. Doherty., L.Dimitriov, 1996, T. Holmen et al. 2002), reported a significant decrease in the FVC levels of smokers when compared with non-smokers, and also found a positive correlation between physical activity, fitness and lung capacity. The present study also detected significant decreases in FVC levels of smoking sportsmen before and after training (Graphic 4). Consequently, it was established that these harmful effects of smoking on the respiratory system can result from toxic metabolites and the pharmacologic effects of nicotine in cigarettes. It is also known that the smoking lowers the respiratory and O_2 carrying capacity by reducing sporting performance. The present study found a significant reduction in the FEV_1 levels of smoking sportsmen before and after training (Graphic 5). The FEV_1 levels of sportsmen who regularly exercise and do not smoke were significantly higher than those of sportsmen who exercise but smoke. These results support the theory that smoking reduces sporting performance by reducing respiratory capacity. However, pre-training and post-training FEF 25-75% levels were not statistically significant in smoking and non-smoking sportsmen ($p>0.05$) (Graphic 6). In that case, it can be concluded that the respiratory superiority of sportsmen is generally related to the ability to use lung capacity and low respiration is related to insufficient exercise and smoking.

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$MaXVO_2$ value is accepted as one of the most important indicators of sporting performance. Previous studies have found this to be higher in non-smokers (PO. Astrand, 1988). These findings are consistent with the results of the present study (Graphic 7). The present study also found a significant decrease in pre-training and post-training $MaXVO_2$ levels of smoking sportsmen when compared with non-smoking sportsmen ($p<0.001$). Based on all these findings, it can be concluded that smoking lowers the maximum respiration capacity, reduces sporting performance, lowers the O_2 carrying capacity of blood, and increases the heart rate and flow per minute.

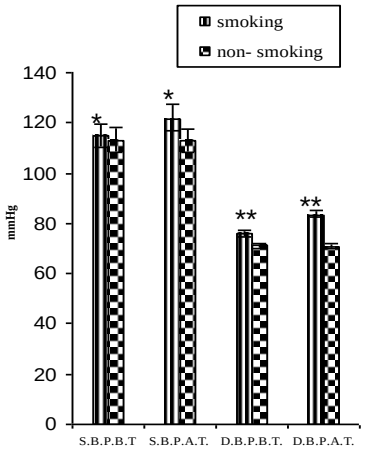
On the other hand, exercise can damage the balance between free radicals identified as oxidative stress and antioxidants. During exercise, O_2 consumption can be greater than during relaxation and, as a result, the free radical producing capacity of the mitochondria increases temporarily. A study by Buxton et al. (1997) reported that in gentle exercise minimum O_2 consumption was 40-60% in 3 hours and maximum O_2 consumption was 75% in 1 hour and that significant increases were detected in melatonin levels.

In the present study, although the pre-training melatonin levels of non-smoking sportsmen (Graphic 8) were higher than the smoking sportsmen, the differences between these levels were nor statistically significant ($p>0.05$). When the pre-training melatonin levels of smokers and non-smokers were compared, it was found that melatonin levels, which increased immediately after training, returned to normal levels two hours later. These increases and decreases were found to be statistically significant ($p<0.001$). A statistically significant increase was observed in the melatonin levels of smoking and non-smoking sportsmen after the 2nd week of training ($p<0.001$) (Graphic 9). Similarly, Özgüner et al. (2005) investigated the melatonin levels of female students who smoke 21 cigarettes a day and those who do not smoke. It was found that the melatonin levels of the smoking students were lower than those of non-smoking students. In another study, Pilaczynska et al. (2004) compared the melatonin levels of people who undertake intense physical activity and those who undertake light physical activity. They observed a negative correlation between plasma melatonin levels and light physical activity; a positive correlation between intensive exercise and plasma melatonin levels. The data indicates that regular physical activity increases melatonin level. These findings indicate that physical activity increases plasma melatonin level.

It is established that the production of oxidants increases during exercise. Excessive physical activity is one of the particular factors that induce the formation of reactive oxygen species (ROS) in the organism. Depending on increasing metabolic activity, O_2 consumption increases and, consequently, ROS emerges. In case of any problems during the removal of these oxidants, significant oxidative damage can occur in the cellular biomolecules. Nevertheless, regular exercise protects myocytes from the harmful effects of oxidants and enables the adaptation of skeletal muscles to antioxidant capacity in order to prevent cellular damage (T. Şinoforoğlu et al. 2006). Furthermore, it can be stated that smoking reduces the exercise tolerance level and limits physical activity and that regular exercise raises maximum respiratory capacity. In the light of all this information, the volume, duration and the time of exercise is very important in determining the effect of the exercise on melatonin; melatonin can also show a powerful antioxidant effect against oxidative damage in the lungs by reducing free radical damage caused by smoking.

Consequently, it was found that cigarette has an increasing effect on the systolic and diastolic blood pressure and pulse rate a decreasing effect on VC, FVC, FEV_1, FEF 25-75%, $MaXVO_2$ and melatonin levels. Nevertheless, regular and gradual training has been shown to have a regulating role on the negative effect of smoking on sporting performance, respiration capacity and melatonin.

Graphic 1. Systolic and diastolic blood pressure levels of smoking and non-smoking sportsmen before and after training
Sigara içen: Smoking
İçmeyen: Non-smoking



*:p<0.05 , **:p<0.01

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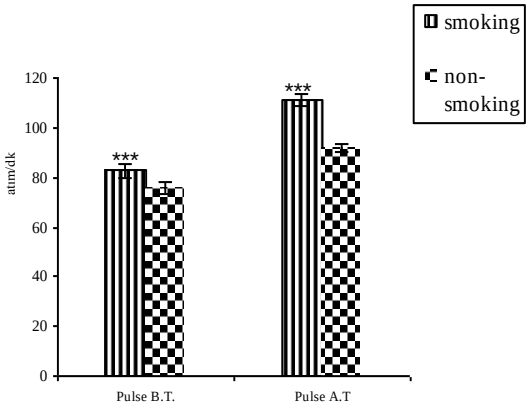
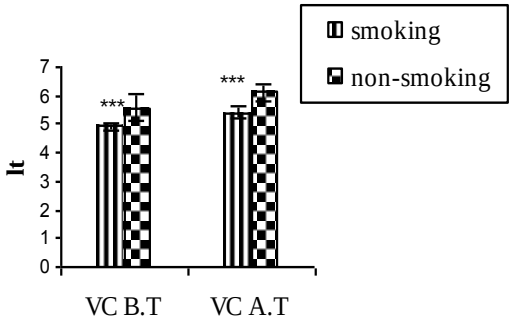
S.B.P.B.T: Systolic Blood Pressure before Training
S.B.P.A.T: Systolic Blood Pressure after Training
D.B.P.B.T: Diastolic Blood Pressure before Training
D.B.P.A.T: Diastolic Blood Pressure after Training

Graphic 2. Pulse rates of smoking and non-smoking sportsmen before and after training



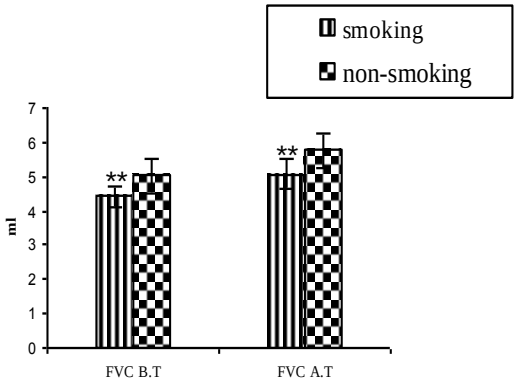
***: p<0.001
Pulse B.T: Pulse before training
Pulse A.T: Pulse after training

Graphic 3. VC levels of smoking and non-smoking sportsmen before and after training



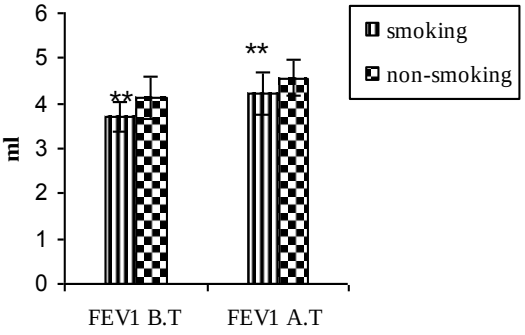
***: p<0.001
VC B.T : Vital capacity before training
VC A.T : Vital capacity after training

Graphic 4. FVC levels of smoking and non-smoking sportsmen before and after training



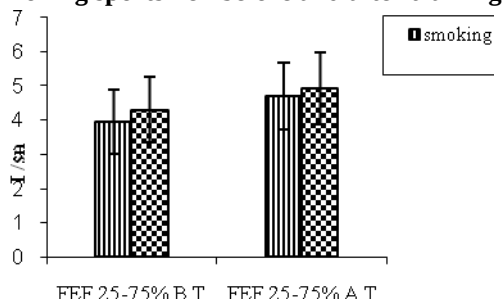
**:p<0.01
FVC B.T : Force vital capacity before training
FVC A.T : Force vital capacity after training

Graphic 5. FEV_1 levels of smoking and non-smoking sportsmen before and after training



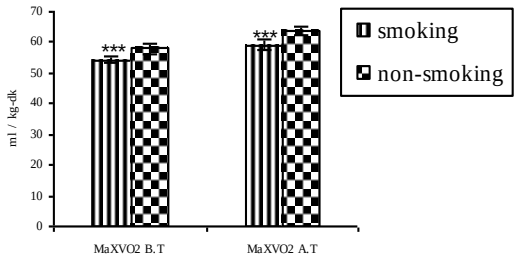
**:p<0.01
FEV1 B.T : Forced Expiratory Volume before training
FEV1 A.T : Forced Respiratory Volume after training

Graphic 6. FEF 25-75% levels of smoking and non-smoking sportsmen before and after training



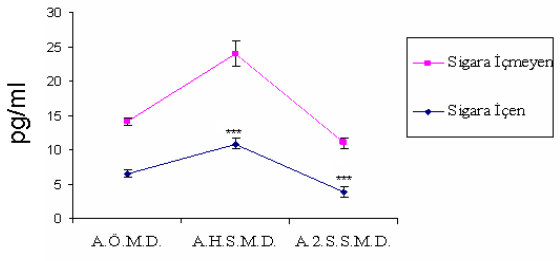
FEF 25-75% B.T: average flow rate in the half of the forceful expiration rate before training
FEF 25-75% A.T: average flow rate in the half of the forceful expiration rate after training

Graphic 7. $MaXVO_2$ levels of smoking and non-smoking sportsmen before and after training



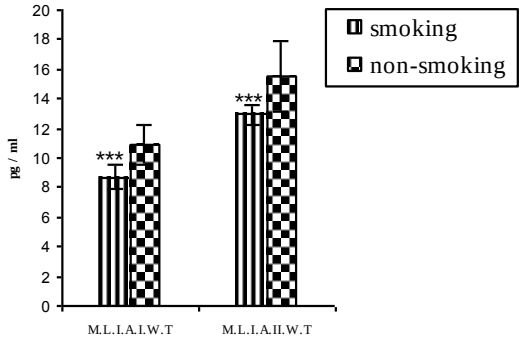
***:p<0.001
$MaXVO_2$ B.T: Maximum Oxygen consumption before training
$MaXVO_2$ A.T: Maximum Oxygen consumption after training

Graphic 8. Melatonin levels of smoking and non-smoking sportsmen before training, immediately after training and two hours after training



***:p<0.001
M.L.B.T: Melatonin level before training
M.L.I.A.T: Melatonin level immediately after training
M.L.2.A.T: Melatonin level two hours after training

Graphic 9. Melatonin levels of smoking and non-smoking sportsmen immediately after the 1st and 2nd week of the trainings



***:p<0.001
M.L.I.A.I.W.T: Melatonin level immediately after the 1st week of training
M.L.I.A.II.W.T.: Melatonin level immediately after the 2nd week of training

Acknowledgments

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