Influences of Intermittent Anaerobic Exercise Program on Physical Fitness and Plasma Lactate, Oxidant and Anti-oxidant Status in Smokers and Non-smokers Judo Players.

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Abstract

Background: There is evidence of smoking’s negative impact and physical activity’s positive impact on long-term health. However, evidences regarding the association between smoking and exercise activity and the independent effects of these factors on antioxidant defense are lacking. The aim of this study is to investigate the association between smoking and the intermittent anaerobic exercise on the physical fitness, oxidant and antioxidant status in Judo player.

Methods: Twenty male Judo player student from Department of Physical Education Al-Azhar University were enrolled in the study. They were of 2 groups, Non-smokers (NS) and smokers (SM). Both groups were subjected to regular Judo training program (2 hours/day, 3 sessions/week for 12 weeks). Also, both groups were subjected to an effort test (running of submaximal intensity until exhaustion ) and record of the maximum oxygen capacity (VO$_2$max) using an ergometric bicycle. Heart rate (HR), mean arterial blood pressure (MBP) were recorded and blood samples were taken pre and post-effort test for determination of malondialdehyde (MDA),total antioxidant capacity (TAC), uric acid (Ua) and lactate (La) in the plasma. Also the time to exhaustion was recorded during the effort test. These measures were performed pre and post the training program.

Results: At rest the pre-program data of SM showed significantly higher H.R., and plasma MDA ,and significantly lower TAC with tendency to increase in MBP and La levels, and decrease in VO$_2$max, and Ua levels compared to NS. Before program and in response to acute exercise SM showed significantly higher H.R, MDA and La levels with significantly lower TAC, Ua and time to exhaustion compared to NS. On the other hand, after the program and in response to acute exercise, SM showed more pronounced significantly higher HR, MDA and La with significantly lower VO$_2$max, time to exhaustion and TAC with insignificant changes in the Ua compared to the NS that showed significant increase in the Ua levels with insignificant changes in the MDA, TAC and the La levels in response to acute exercise. In response to the training program, SM showed significantly higher HR, MBP, MDA and La levels, with significantly lower VO$_2$max, time to exhaustion, and TAC, and insignificant changes in the Ua levels compared to NS.

Conclusion: We conclude that cigarette smoking has a significant detrimental effects on cardiovascular fitness, physical endurance, TAC and lipid peroxidation that all worsened by the intermittent anaerobic exercise. On the contrary , this type of training is beneficial in nonsmokers. Also we demonstrated that smoking prevent the beneficial effects of exercise , so smoking should be avoided in Judo players. Smokers should be given strong encouragement to stop smoking and to improve physical fitness.

Introduction

Cigarette smoking is associated epidemiologically with a high risk for various types of chronic illnesses including vascular disease (McNamara and Fitz Geraled, 2001). The underlying mechanisms of smoke-related severe damage to
tissues and organs are still not completely understood; O-and N-derived free radicals are thought to play a major pathophysiological role (Polidori et al., 2003).

Cigarette smoke contains several types of toxic components, including carbon monoxide (CO), nicotine and benzo (a) pyrene (Raveendran et al., 2004). Super oxide, reactive oxygen species (ROS) and radicals are also component of cigarette smoke. One of the principal ROS produced in aerobic organism is $O_2^-$, which is highly cytotoxic. However it may be possible to reduce smoke-induced damage by enhancing the protective defense system i.e increased levels of antioxidant enzymes (Gilks et al., 1998).

Physical exercise is known to have many beneficial effects on health. However, there is also evidence that free radical production increases during exercise especially intense physical exercise in unconditioned individuals, and that oxidative damage may occur in the muscle, liver, blood, and perhaps other tissues (Margaritis et al., 2003). The free radicals produced during exhaustive exercise are very reactive chemical species and can readily lead to uncontrolled reactions, which may result in damage to DNA, proteins and lipids, causing molecular and cellular damage. So these individuals require stronger antioxidant defenses. Some of these defenses adapt with training (Groussard et al., 2003), or with proper antioxidant supplementation (Margaritis et al., 2003), but they can be overwhelmed by the higher production of ROS during intense physical exercise. There is evidence suggested that exercise intensity could influence the production of ROS and redox status in the body (Covas et al., 2002).

Judo training is a violent exercise which characterized by several series of 15-30 sec. duration efforts with 10-15 sec. intervals between them (Finaud et al., 2006). Because these efforts are very intense and intervals are not long enough, ATP resynthesis by oxidative ways are unviable, and the effort becomes dependent on the glycolytic pathway that cause very high lactate accumulation (Tabata, 1997).

Data concerning the acute effects of exercise on antioxidant enzyme defenses in human are controversial because of many types of exercise and experimental conditions adopted in previous researches, which do not allow for comparison between studies (Tauler et al., 2005).

However, evidences regarding the association between smoking and exercise activity and the independent effects of these factors on fitness and the antioxidant defense state are completely lacking, because in developed countries the athletes are less common to smoke. But in developing countries it is common to find an association between performing exercise and smoking specially in students of sport education faculties, who obliged to perform the physical activity in their practical lessons. This motivated us to study the effect of physical training Judo program, on lipid peroxidation represented by plasma level of MDA to reflect the oxidant status and the antioxidant state reflected by measurement of serum level of the TAC and uric acid in smokers and non-smokers Judo player students.

**Subjects and Methods**

The current study was conducted during the period of 3 months, between February and April 2006 on the 2nd year male students of Department of Physical Education, Faculty of Education Al- Azhar University. Twenty volunteer male student were enrolled in this study, their (mean ±SE) anthropometric measurements as age, height and weight are shown in table (1). They were selected and assigned as 2 groups:

- **Group (A):** They were non smokers (NS).
- **Group (B):** Smoker (SM), they smoke an average of 15 cigarettes /day for at least 3 years prior to the study.

All subjects were Judo practitioners had at least the brown belt, and all of them had to meet the following selected criteria

(a) In a good health, with no known disease including diabetes, cancer or heart disease;
(b) Not currently on a reducing diet regimen;
(C) Not using medications known to affect the immune system;
(d) Not using
anabolic, vitamins or mineral supplements; 
(e) Written consent was obtained from each subject. 
- Both groups were subjected to regular 
  Judo training program that consists of 
  sessions of 2 hours/day (from 5-7 pm) 
  for 3 sessions /week for 12 weeks. Each 
  session was consisted of warming up 
  for 15min., performing the training 
  program for 95 min. then cooling down 
  for 10 min. 
- The training program were designed and 
  supervised by a specialist. 

Experimental design 
At the beginning we recorded the 
height and weight for each subject, then 
each group was subjected to the following: 
Recording of the physical fitness param-
eters, represented by measuring of: 

a-Acute exercise, as an physical effort test, 
in the form of running of submaximal 
intensity until exhaustion, as it give the 
optimum reflection for acute aerobic 
exercise (Brunsgard, 2005). The time to 
exhaustion was recorded using stop 
watch. This test was done one day before 
beginning the training program (pre-
program) and then repeated one day after 
the program i.e after 12 weeks. 
b-Recording the H.R. from the mitral area 
,using stethoscope, for each subject 
after 10 min of supine rest. 
c-The systolic and diastolic blood 
pressures were measured using 
sphygmomano-meter. This was done 
for each subject after 5-10 min. of 
supine rest. 
d- \( \text{VO}_2 \text{ max} \): It was measured indirectly by 
application of Astrand test for aerobic 
fitness from the H.R and the work load 
performed on the ergometric bicycle for 
6 min. at submaximal (70%) of the 
work load. Then the \( \text{VO}_2 \text{max} \) was 
measured by extrapolation using special 
table (Monogram) and expressed as 
litters/min.(Astrand and Rodhal, 1988). 
This test was done one day before 
beginning the training program (pre-
program) and then repeated one day 
after the program. 

Blood Sampling 
Fasting blood samples from all 
subjects were withdrawn into EDTA 
containing tubes, from an antecubital vein 
with subjects in the seated position. Blood 
was centrifuged at 3000g for 15 min at 
4\(^\circ\)C., and the plasma was separated then 
stored at -80\(^\circ\)C until analysis. 
- The HR, ABP and Blood samples were 
performed between 8 and 9 am before 
and within 10 min. after the running test 
(i.e the pre and post-effort readings). This 
was done one day before beginning the 
Judo training program and repeated one 
day after finishing the program. 
- The smoker subjects had been instructed to 
stop smoking for 2 hours before both the 
effort test and before each training 
session (Turner and Mc Nicol, 2002). 

Biochemical analysis 
- Plasma malondialdehyde (MDA) was 
measured to reflect the degree of lipid 
peroxidation, according to the method 
of Suttnar et al., (1997). 
- Plasma uric acid (Ua) was measured 
according to the method of Williams et 
al. (1960). 
- Plasma lactate was determined according 
- The plasma total antioxidant capacity 
(TAC) was measured spectrophotome-
trically according to the method of 
Koracevic et al., (2001) that based on 
measuring the capacity of the biological 
fluids to inhibit the production of 
thiobarbituric acid reactive substances 
(TBARS) from sodium benzoate under 
the influence of the free oxygen 
radicals derived from fenton's reaction. 

Statistical analysis 
Data were expressed as mean ± SE, 
statistical paired comparison of sequential 
results were preformed with the paired t-
test for parametric comparisons of (pre) 
with post-effort data and (pre) with post-
program data of the same group. While, 
unpaired t-test was used when compared 
one group to the other group. P-value less 
than 0.05 was considered significant.
Results

Table (1) demonstrated the anthropometric characteristics of both groups at base line. There were no significant differences among the two groups in the age, height, weight and VO2 max.

Table (2) demonstrated the differences in all parameters between smokers and non smokers at the base line (before beginning the program) and in response to the acute exercise (post-effort). At base line the smokers showed significantly higher H.R, and MDA levels (P<0.01) and significantly lower TAC (P<0.01). While the mean arterial blood pressure (MBP) and Ua and La levels showed insignificant differences between the two groups. On the other hand, in response to the acute exercise, the smokers showed significantly higher HR, MDA and La and significantly lower TAC and Ua levels in comparison to the non-smokers.

Table (3) demonstrated the differences in the different parameters between smokers and non-smokers in response to 12 weeks of Judo training program. In nonsmoker group the training program could significantly elevate the VO2 max (22.78%), the time to exhaustion (66.5%) and the levels of TAC (45.96%) and Ua (17.9%), with tendency to lower the H.R (-4.54%), MBP (-1.83%) and plasma La levels (-4.85%). However the MDA levels showed insignificant changes compared to the resting preprogram values. On the other hand in the smokers the training program caused significant increase in MDA levels (9.85%), tendency to increase in MBP (2.62%) and insignificant changes in the remaining parameters compared to the resting pre-program values. In comparison to the non-smokers, the training program could aggrivate and increase the difference in smokers to show significantly higher HR (16.1%), MBP (8%), MDA (17.9%) and La (22.45%) and significantly lower VO2 max (-25%), time to exhaustion (52.9%) and the TAC (-42.9%) levels compared to the non-smokers.

Table (1): Anthropometric characteristics of both groups at base line.

<table>
<thead>
<tr>
<th></th>
<th>Non-smokers</th>
<th>Smokers</th>
<th>P-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Number</td>
<td>10</td>
<td>10</td>
<td></td>
</tr>
<tr>
<td>Age(years)</td>
<td>18.5±0.23</td>
<td>18.7±0.31</td>
<td>P&gt;0.05</td>
</tr>
<tr>
<td>Height (cm)</td>
<td>179.6±4.2</td>
<td>177.8±4.5</td>
<td>P&gt;0.05</td>
</tr>
<tr>
<td>Weight (Kg)</td>
<td>83.4±2.6</td>
<td>79.5±2.8</td>
<td>P&gt;0.05</td>
</tr>
<tr>
<td>VO2 max (l/min)</td>
<td>3.16±0.18</td>
<td>2.87±0.08</td>
<td>P&gt;0.05</td>
</tr>
</tbody>
</table>
Table (2): Comparison between the different parameters of smokers and nonsmokers in response to acute exercise before starting the program.

<table>
<thead>
<tr>
<th></th>
<th>Non-Smokers (A) n=10 (Mean ±SE)</th>
<th>Smokers (B) n=10 (Mean ±SE)</th>
<th>Difference between A&amp;B</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Pre-effort</td>
<td>Post-effort</td>
<td>Pre-effort</td>
</tr>
<tr>
<td></td>
<td>68.3±2.3</td>
<td>167.2±3.2</td>
<td>76.8±2.3</td>
</tr>
<tr>
<td>H.R (beat/min)</td>
<td>P&lt;0.0005</td>
<td>P&lt;0.0005</td>
<td>P&lt;0.01</td>
</tr>
<tr>
<td></td>
<td>92.2±2.91</td>
<td>109.8±4.2</td>
<td>95.3±2.12</td>
</tr>
<tr>
<td>MBP (mm Hg)</td>
<td>P&lt;0.0005</td>
<td>P&lt;0.0005</td>
<td>P&lt;0.05</td>
</tr>
<tr>
<td></td>
<td>34.1±1.8</td>
<td>39.2±2.1</td>
<td>39.6±1.2</td>
</tr>
<tr>
<td>MDA (µmol/l)</td>
<td>P&lt;0.01</td>
<td>16.13%</td>
<td>P&lt;0.01</td>
</tr>
<tr>
<td>T AC (m mol/l)</td>
<td>1.98±0.13</td>
<td>1.82±0.18</td>
<td>1.55±0.11</td>
</tr>
<tr>
<td></td>
<td>P&lt;0.0005</td>
<td>P&lt;0.0005</td>
<td>P&lt;0.05</td>
</tr>
<tr>
<td>Uric acid (mg/dl)</td>
<td>5.02±0.21</td>
<td>5.98±0.23</td>
<td>4.58±0.41</td>
</tr>
<tr>
<td></td>
<td>P&lt;0.0005</td>
<td>P&lt;0.0005</td>
<td>P&gt;0.05 - 8.76%</td>
</tr>
<tr>
<td>Lactate (m mol/l)</td>
<td>1.03±0.08</td>
<td>1.85±0.12</td>
<td>1.13±0.06</td>
</tr>
<tr>
<td></td>
<td>P&lt;0.0005</td>
<td>P&lt;0.0005</td>
<td>P&gt;0.05</td>
</tr>
</tbody>
</table>

-P value less than 0.05 is considered significant.

Table (3): Comparison between the different parameters of smokers and nonsmokers in response to acute exercise after performing the program.

<table>
<thead>
<tr>
<th></th>
<th>Non Smokers (A) n=10(Mean ±SE)</th>
<th>Smokes (B) n=10(Mean ±SE)</th>
<th>Difference between (A) &amp; (B)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Pre-effort</td>
<td>Post-effort</td>
<td>Pre-effort</td>
</tr>
<tr>
<td></td>
<td>65.2±2.4</td>
<td>148.5±3.8</td>
<td>75.7±2.1</td>
</tr>
<tr>
<td>H.R (beat/min)</td>
<td>P&lt;0.0005</td>
<td>P&lt;0.0005</td>
<td>P&lt;0.0005</td>
</tr>
<tr>
<td></td>
<td>90.5±3.1</td>
<td>105.6±4.2</td>
<td>97.8±2.32</td>
</tr>
<tr>
<td>MBP (mmHg)</td>
<td>P&lt;0.0005</td>
<td>P&lt;0.0005</td>
<td>P&lt;0.05</td>
</tr>
<tr>
<td></td>
<td>36.9±2.2</td>
<td>41.8±2.3</td>
<td>43.5±1.3</td>
</tr>
<tr>
<td>MDA (µmol/l)</td>
<td>P&lt;0.0005</td>
<td>P&lt;0.0005</td>
<td>P&lt;0.01</td>
</tr>
<tr>
<td>T AC (m mol/l)</td>
<td>2.89±0.22</td>
<td>2.69±0.21</td>
<td>1.65±0.16</td>
</tr>
<tr>
<td></td>
<td>P&lt;0.0005</td>
<td>P&lt;0.0005</td>
<td>P&lt;0.0005</td>
</tr>
<tr>
<td>Uric acid (mg/dl)</td>
<td>5.92±0.48</td>
<td>6.98±0.31</td>
<td>5.53±0.52</td>
</tr>
<tr>
<td></td>
<td>P&lt;0.0005</td>
<td>P&lt;0.0005</td>
<td>P&lt;0.05</td>
</tr>
<tr>
<td>Lactate (m mol/l)</td>
<td>0.98±0.08</td>
<td>1.16±0.11</td>
<td>1.20±0.12</td>
</tr>
<tr>
<td></td>
<td>P&lt;0.0005</td>
<td>P&lt;0.0005</td>
<td>P&lt;0.025</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>P&lt;0.0005</td>
</tr>
</tbody>
</table>

-P value less than 0.05 is considered significant.
Table (4): Comparison between the different parameters of smokers and nonsmokers in response to 12 weeks of Judo training program.

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Non-Smokers (A) n=10 (Mean ±SE)</th>
<th>Smokers (B) n=10 (Mean ±SE)</th>
<th>Difference Between A &amp; B</th>
<th>Pre-program</th>
<th>Post-program</th>
<th>P-value</th>
<th>%change</th>
</tr>
</thead>
<tbody>
<tr>
<td>H.R (beat/min)</td>
<td>68.3±2.3</td>
<td>76.8±2.3</td>
<td>P&lt;0.01</td>
<td>65.2±2.4</td>
<td>75.7±2.1</td>
<td>12.45%</td>
<td>4.54%</td>
</tr>
<tr>
<td>MBP (mmHg)</td>
<td>92.2±2.91</td>
<td>95.3±2.12</td>
<td>P&lt;0.05</td>
<td>90.5±3.1</td>
<td>97.8±2.32</td>
<td>4.54%</td>
<td>-1.83%</td>
</tr>
<tr>
<td>VO₂ max (l/min)</td>
<td>3.16±0.18</td>
<td>2.87±0.08</td>
<td>P&lt;0.01</td>
<td>3.88±0.21</td>
<td>2.91±0.18</td>
<td>12.45%</td>
<td>-22.78%</td>
</tr>
<tr>
<td>Exhaustion Time (min)</td>
<td>19.4±1.8</td>
<td>13.5±1.3</td>
<td>P&lt;0.0005</td>
<td>32.3±2.4</td>
<td>15.2±1.5</td>
<td>16.1%</td>
<td>-66.5%</td>
</tr>
<tr>
<td>MDA (µmol/l)</td>
<td>34.1±1.8</td>
<td>39.6±1.2</td>
<td>P&lt;0.025</td>
<td>36.9±2.2</td>
<td>43.5±1.3</td>
<td>16.1%</td>
<td>8.21%</td>
</tr>
<tr>
<td>TAC (mmol/l)</td>
<td>1.98±0.13</td>
<td>1.55±0.11</td>
<td>P&lt;0.01</td>
<td>2.89±0.22</td>
<td>1.65±0.16</td>
<td>30.4%</td>
<td>-45.96%</td>
</tr>
<tr>
<td>Uric acid (mg/dl)</td>
<td>5.02±0.21</td>
<td>4.58±0.41</td>
<td>P&lt;0.05</td>
<td>5.92±0.48</td>
<td>5.53±0.52</td>
<td>12.45%</td>
<td>-17.9%</td>
</tr>
<tr>
<td>Lactate (mmol/l)</td>
<td>1.03±0.08</td>
<td>1.13±0.06</td>
<td>P&lt;0.05</td>
<td>0.98±0.08</td>
<td>1.20±0.12</td>
<td>22.45%</td>
<td>-4.85%</td>
</tr>
</tbody>
</table>

-P value less than 0.05 is considered significant.
Fig. (1) : Comparison between the different parameters of smokers and nonsmokers in response to 12 weeks of Judo training program.
Discussion

Cigarette smoking is associated epidemiologically with a high risk for various types of chronic illnesses including atherosclerosis (McNamara and Fitz Gerald, 2001). The process of atherosclerosis is believed to be initiated by lipid peroxidation (Polidori et al., 2003).

Physical exercise is known to have many beneficial effects on health. However, data on the effects of acute and chronic exercise on antioxidant enzyme defences in humans are controversial and lacking in smokers.

Our results revealed that, at the base line (pre-program), during rest, smokers showed significantly higher H.R. and plasma MDA and significantly lower TAC levels with tendency to increase in MBP and lactate levels and decrease in VO$_2$ max and Ua levels compared to nonsmokers.

The increased H.R. in smokers may be related to the deficient oxygenation to the tissue that caused by carbon monoxide (CO) present in cigarette smoke (Polidori et al., 2003). Also the effect of nicotine is probably contributed to the significantly higher resting H.R. in smokers (Kobayashi et al., 2004). Turner and Mc Nicol (2002), also reported significant elevations in H.R and blood pressure after nicotine administration. Furthermore an animal study by Symons and Stebbins (1996) observed detrimental hemodynamic effect of nicotine on the cardiovascular system at rest, but during exercise these were minimized.

It has been reported that chronic smoking and acute exposure to cigarette smoke extract may alter endothelium dependent reactivity via the production of oxygen derived free radicals, due to the direct effect of nicotine on resistance arterioles, compliance arteries, smooth muscle cells, and ion channels in the cardiovascular system and in turn produce hypertension (Hanta et al., 2006). The smoker in the present study showed mild increase in the MBP compared to non-smokers. This may be due either to the short period of smoking, as the back years were only 3 years, or due to the stoppage of smoking 2 hours before the training as instructed in the study.

The observed decrease in VO$_2$ max and time to exhaustion in smokers are previously reported in several studies. Chatterjee et al., (1997) and Dyrstad et al., (2002) observed significantly lower VO$_2$ max only in young age group of the smokers. Tchissambou et al (2004), reported that the subjects who were heavily dependent on nicotine had lower VO$_2$ max values and aerobic capacity than those less dependent. This may explain the decrease in the time to exhaustion and slower running observed in our study. It has been shown that smoking also affects work performance, specially endurance exercise (Surmen-Gur et al., 1999). Kobayashi et al., (2004), reported that VO$_2$ max and anaerobic threshold were acutely reduced in apparently healthy adult smokers immediately after smoking, as compared to five hour after smoking. Presumably because of elevated carbon monoxide and nicotine. This may explain the tendency to increase in the lactate levels in smokers in this study.

The significantly higher MDA and lower TAC levels observed in smokers in this work, may be due to one or more of the following reasons: First of all, smokers are prone to oxidation from inhalation of large numbers of gas-phase and other radicals giving rise to increased oxidative damage (Hanta et al., 2006). Second, depletion of plasma antioxidants, otherwise protecting against oxidative damage such as lipid peroxidation, that has consistently been observed among smokers (Lykkesfeldt et al., 2000). Third, smokers have been shown to have a lower intake of fruits and vegetables (Serdula et al., 1996), that known to protect against oxidative damage (La Vecchia and Tavani, 1998).

In agreement with these findings, smokers have been shown to have higher levels of lipid peroxidation compared with non-smokers, as measured by increased plasma thiobarbituric acid-reactive substances (TBARS), plasma and urinary concentrations of MDA, F$_2$-isoprostanates and
conjugated dienes (Block et al., 2002). However, higher levels of lipid peroxidation among smokers in the study of Block et al. (2002) and several previous studies could in fact primarily be a secondary effect of the poorer antioxidant status also found in smokers. Block’s group also found an inverse correlation between plasma ascorbic acid and MDA (Block et al., 2002). This further supports the link between antioxidant status and MDA. However Lykkesfeldt et al. (2004), suggested that lipid peroxidation is induced by smoking per se. While poor antioxidant status presumably also affect lipid peroxidation, it is only partly responsible for the increased level found in smokers in general.

Goraca and Skibska (2005) studied the relationship between lipid peroxidation and total plasma antioxidant capacity (TAC) in healthy smoking and non-smoking subjects. They found that, smokers have significantly lower TAC and significantly higher TBARS than nonsmokers. They also found significantly lower plasma concentrations of non enzymatic antioxidants (alpha-tocopherol, beta-carotene and ascorbic acid) in chronic smokers compared to nonsmokers. In the same study, in vitro addition of ascorbic acid, or trolox to plasma samples from smokers, significantly increased their TAC. Also, it has previously been reported that plasma exposure to gas-phase cigarette smoke caused depletion of carotenoids, Vitamin C, uric acid and α-tocopherol, and induction of lipid peroxidation in vitro (Polidori et al., 2003).

In the present study and in response to acute exercise, both groups showed similar response but smokers showed more pronounced elevation of the HR and levels of MDA and lactate, and more pronounced reduction in TAC levels and the time taken to reach exhaustion compared to nonsmokers.

This similarity of the exercise HR responses in the nonsmokers and smokers at equivalent stress levels, in response to acute exercise both in pre-program and post-program periods, suggests that the cardiovascular system was fully and equally taxed in both groups in terms of stroke volume. This indicates that the arteriovenous difference was smaller and the O₂ extraction reduced in smokers. This in agreement with the study of Kobayashi et al. (2004) who found similarity in HR responses of smoker and non-smokers to sub-maximal and maximal exercise but the O₂ pulse was always lower in smokers. Also the return of HR to resting levels after exercise was slower in chronic smokers. This may be related to the chronotropic and inotropic effects of catecholamines mobilized by nicotine absorbed from cigarette smoke (Tchissambou et al., 2004). This may explain also the decreased time to exhaustion and the slower performance observed in smokers which may be also due to the progressive increase in lactate levels in smokers by exercise. However in response to the training program the nonsmokers showed lower resting H.R and MBP compared to smoker. This indicates the good adaptation response to training program in nonsmokers and failure of this response in smokers. Our results are in line with the study of Godsland et al. (1998).

The elevated lactate levels in response to acute exercise specially in smokers in the present study, agree with the results reported by many studies (Surmen-Gur et al., 1999 and kobayashi et al., 2004). According to kobayashi group, the more intense the exercise, the more lactate is produced and taken up by the working muscle. This was more pronounced in smokers. Smokers have been shown to have a reduced anaerobic threshold, as a consequence of increased anaerobiosis and reduced tissue PH and an increase in CO₂ output via the bicarbonate buffer system (Hemila et al., 2006).

The increased lipid peroxidation and decreased antioxidant activity in response to acute exercise in the pre-program period is reported by several studies Surmen-Gur et al., 1999, Groussard et al., 2003 and Demirbag et al., 2006). According to these data, the principal factor responsible for oxidative damage during exercise is the increase in the oxygen consumption. However, in the post-program period and in response to acute exercise, the resting (pre-effort) value showed insignificant changes
in non smokers but significantly elevated in smokers and progressively increased following acute exercise. This may be due to in addition to the increase in oxygen consumption already found in smokers, other theoretical factors such as acidosis, catecholamine auto-oxidation and ischemia-reperfusion syndrome, may also be operative during short-term supramaximal anaerobic exercise (Franchini et al., 2003) that is repeated during and throughout the period of Judo training in our study. This may explain the failure of adaptation of cardio-respiratory systems in smokers in the post-program period, compared to pre-program values, as the nonsmokers showed lower values of H.R and MBP in both resting and in response to acute exercise, but smokers still had higher values. This is in agreement with the study of Bernaards et al. (2003).

The slower performance and early exhaustion observed in smokers in our study in response to acute exercise in both the pre-program and post-program period, agree with the study of Hemila et al. (2006). They found that the young male smokers ran slower and the exercise endurance time on the treadmill protocol was significantly longer in the nonsmokers. But the aerobic performance (VO\textsubscript{2max}) was affected only during high-intensity aerobic exercise in smokers.

Groussard et al. (2003), hypothesized that short-term supramaximal anaerobic exercise (30 second Wingate test) could induce an oxidative stress. But they observed that, the recovery of exercise was associated with a significant increase in lipid radical production, as well as with decrease in the erythrocyte reduced glutathione (GSH) level and superoxide dismutase (SOD) activity. Also they suggested that, the paradoxical decrease in plasma TBARS which was correlated with the peak power developed during the Wingate test, strongly suggests that such exercise stimulate the elimination of MDA. This in line with the effect of 3 month training program in non-smokers in our result. However the progressive increase in MDA and decrease in TAC in smokers is probably due to the action of CO and nicotine and other superoxides and other reactive oxygen species (ROS) found in cigarette smoke, together with the oxidative stress caused by the acute exercise that cause oxidative damage and disturbance in the oxidant/antioxidant ratio. (Tsuchiya et al., 2002 and Ozbay and Dulger 2002). This is supported by the findings of Demirbag et al., (2006) who demonstrated that treadmill exercise test increases oxidants, decreases TAC and vitamin C, but this stress is not enough to produce DNA damage. While Covas et al. (2005), found that antioxidant enzyme activities in post-exercise values were significantly higher than those of pre-exercise values, in saliva of elite Judoists.

In response to the training program our findings regarding the increase in TAC in non-smokers were in accordance to the findings of (Chevion et al., 2003). Also, an animal study investigated the effect of intermittent anaerobic exercise program on the tissue antioxidant capacity and lipid peroxidation, found that this type of exercise led to a higher TAC in muscle, heart and brain in mouse (Qiao et al., 2006).

However the data about the acute and chronic effect of exercise on TAC and lipid peroxidation in smokers are completely lacking. But the decrease in TAC and the increase in MDA levels in response to acute exercise and non-improvement in their levels by the training program were expected in smokers, probably due to the oxidative stress caused by the gas phase in cigarette smoke together with the oxidative stress caused by the intermittent anaerobic exercise of Judo training. Both can cause oxidative damage, disturbance in the oxidant/antioxidant ratio and decrease in the anaerobic threshold (Ozbay and Dulger 2002, and Goraca and Skibska, 2005). This may also explain the decreased time to exhaustion and VO\textsubscript{2max} and the elevation in lactate levels in smokers in post-program readings.

Uric acid (Ua) has been identified as the most important non-enzymatic antioxidant present in human body fluids (Tsuzita et al., 2006).

In the present study, the plasma Ua showed significant increase in non smokers
in response to acute exercise both pre and post-program compared to the smokers that showed insignificant changes. Also, the training program caused significant elevation in the basal level (pre-effort) of plasma Ua in non-smokers compared to smokers. These findings are in agreement with that of Chevion et al. (2003) and Demirbag et al. (2006) who found significant increase in plasma uric acid after acute exercise due presumably to increases in the metabolic rate and consequent pyrimidine nucleotide metabolism. However Tsuzita et al. (2006) suggested that, the increase in adenine nucleotide degradation and lactic acid production, as well as a release of noradrenalin caused by exercise, contribute to increases in plasma level of urate, as well as decreases in urinary excretion of Ua. While the increase in Ua level by the training program may be due to the repeated effect of anaerobic exercise throughout the period of Judo training, as it has been reported that Wingate test (30 sec-sprint anaerobic exercise), led to increase in plasma Ua and ascorbic acid concentrations. (Groussard et al., 2003). On the other hand, the tendency to decrease in Ua observed in smokers in response to both acute and chronic exercise in the present study is in agreement with the study of Tsuchiya et al. (2002), who reported that smoking a single cigarette decreases plasma levels of Ua.

On the contrary, it has been suggested that there is no association between smoking and plasma uric acid levels (Stathis et al., 2005).

Frei et al. (1999), found that exposure of plasma to cigarette smoke caused oxidation of plasma protein thiols and albumin–bound bilirubin, where as uric acid is not consumed at significant rates.

**Conclusion**

We conclude that cigarette smoking has a significant detrimental effect on cardiovascular and physical fitness, indicated by decreased response to adaptation of HR, MBP, reduced VO₂ max and decreased time to exhaustion in response to both intermittent anaerobic Judo program and to acute aerobic exercise. This probably related to decreased O₂ carrying capacity and decreased plasma lactate threshold in smokers. Also smoking per se led to lipid peroxidation and lowered the total antioxidant capacity and exacerbated them in association to intermittent anaerobic exercise. On the contrary this type of exercise is beneficial for physical fitness and antioxidant defenses in nonsmokers. Thus our finding indicated that smoking is a detriment to physical fitness even among relatively young fit individuals, as it prevented the beneficial effects of exercise. So smoking should be avoided in Judo players and smokers should be given strong encouragement to stop smoking to improve their physical fitness.

**References**


تأثر برنامج التمرينات الرياضية اللاهوائية المنتقطة على اللياقة البدنية وحالات الالتهاب والمؤكسدات ومضادات الأكسدة في البلازما في لاعبي الجودو المدخنين وغير المدخنين

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توجد أدلة على الآثار السلبية للتدخين والأثار الإيجابية للنشاط البدني على الصحة على المدى الطويل ولكن الأدلة التي تخص مصاحبة التدخين مع النشاط الرياضي وتأثيرها على مضادات الأكسدة فإنها مفتقة. والهدف من هذا العمل هو دراسة تأثير مصاحبة التدخين للرياضة على اللياقة البدنية وحالات التأكسد ومضادات الأكسدة في لاعبي الجودو.

الطريقة: اشترك في هذا البحث عشرة طلابًا من قسم التربية الرياضية بكلية التربية بجامعة الأزهر وهم من لاعبي الجودو (عشرة مدخنين وعشرة غير مدخنين)، كل منهم تعرض لأداء برنامج تمريض الجودو (ساعتين يومياً. ثلاث مرات أسبوعياً لمدة ثلاثة شهور). أيضاً كل من المجموعتين تعرضت لأداء اختبار المجهود (الجري بسرعة تحت القصوى حتى الإجهاد) وتسلسل أقصى استخدام للأركسجين باستخدام المجلة الإيجورترية. وقد تم تسجيل معدل ضربات القلب باستخدام السماعة الطبية وقياس متوسط ضغط الدم باستخدام جهاز قياس الضغط وسحب عينة الدم لكل لاعب قبل وبعد أداء المجهود وذلك لتحديد مستوى المالونيدييهد والمعرج عن أكسدة الدهون وتحديد المقدرة الكلية لمضادات الأكسدة وحمض البوليك ومتوسط اللاكتيتي في البلازما. كما تم حساب الوقت الذي يصل به اللاعب للإرهاق أثناء أداء المجهود.

تم تحديد هذه القياسات قبل وبعد أداء البرنامج (أي بعد ثلاث شهور).
النتائج: في البداية وقبل أداء المجهود أظهرت النتائج عن زيادة معدل ضربات القلب ومستوى المالونيدالديهيد زيادة ذات دلالة إحصائية ونقص مستوى مضادات الأكسدة الكلية نقص ذو دلالة إحصائية مع ميول لزيادة متوسط ضغط الدم واللاكتيت ونقص القدرة القصوى لإستهلاك الأوكسجين وحمض البوليك في المدخنين مقارنة بغير المدخنين. كما أنه لوحظ زيادة ذو دلالة إحصائية في استجابة المدخنين للمجهود وذلك في معدل ضربات القلب ومستوى المالونيدالديهيد وحمض البوليك واللاكتيت ونقص في المضادات الكلية للأكسدة مقارنة بغير المدخنين سواء قبل أداء البرنامج أو بعد أداء البرنامج.

أما عن تأثير أداء البرنامج فقد لوحظ زيادة ذة دلالة إحصائية في معدل ضربات القلب ومستوى ضغط الدم ومستوى المالونيدالديهيد واللاكتيت ونقص ذو دلالة إحصائية في قدرة استهلاك الأكسدة الكلية وسرعة ظهور الإجهاد وعدم تغير حمض البوليك في المدخنين مقارنة بغير المدخنين. أما في غير المدخنين فقد أدى أداء البرنامج إلى زيادة اللياقة البدنية وتحسين قدرة الاستطالة عليها بنقص معدل ضربات القلب وضغط الدم والماليونيدالديهيد ومستوى اللاكتيت وزيادة الكفاءة الكلية لمضادات الأكسدة وحمض البوليك والقدرة القصوى لإستهلاك الأوكسجين ووقت الوصول للاجهاد.

وتنستخلص من ذلك أن التدخين تأثير سلبي على كفاءة الجهاز الدورى واللياقة البدنية. كما أثبتت الدراسة أن التدخين وحده قد أدى لزيادة أكسدة الدهون ونقص في القدرة الكلية لمضادات الأكسدة والتي أزدادت ميولاً بممارسة رياضة الجودو في المدخنين حيث أن التدخين قد منع التأثير المفيد للرياضة على كليهما. وعلى العكس فان هذا النوع من الرياضة مفيد كليهما في غير المدخنين. وذلك فالتدخين سيؤثر على اللياقة البدنية حتى في السن الصغير ذو اللياقة البدنية العالية ونصح لاعبي الجودو بعدم التدخين كما نصح بتشجيع المدخنين لإيقاف التدخين لتحسن اللياقة البدنية.