ABSTRACT
Stress oxidative is recognized to be associated with metabolic syndrome and chronic diseases. To determine whether endurance exercise affects total antioxidant capacity in obesity. Twenty two inactive obese men were recruited through an advertisement in a local newspaper in this study and assigned to exercise (n=12) or control (n=12) groups matched for age (35-48 years) and body mass index (30-36 kg/m2). Anthropometrical markers and total antioxidant capacity were measured before and after 10 weeks endurance exercise for 3 times per weeks in exercise groups and control subjects (no training). Pre and post training variables were compared by statistical methods. Despite the signigicant decrease in body fat percentage and other anthropometrical markers (p < 0.01), Total antioxidant capacity was unchanged after 10 weeks of exercise intervention (p = 0.623). The unchanged total antioxidant capacity in our study may be rooted in lack of control diet during training program.

KEYWORDS: Endurance exercise, Obesity, Stress oxidative, Weight loss.

INTRODUCTION
The enzymatic and non-enzymatic antioxidants are activated to remove toxins and regulate oxidants in both internal and external cellular target environments. The very immune system of the body is set up such that it decreases the oxidative damages occurred during the increase of oxidants or free radicals. In other words, antioxidants protect the cells of body against oxidative damages during periods of increased production of oxidants (Powers et al., 2008; Powers et al., 2000). Several factors contribute to creation of oxidative stress and reduced antioxidant capacities. In this context, some studies have reported increased oxidative stress and decreased capacity of antioxidant system in patients with cardiovascular diseases (Amirkhizi et al., 2012). However, extensive studies have identified obesity as one of the initial risk factors of the prevalence of cardiovascular diseases, hypertension, metabolic syndrome and type 2 diabetes (Poirier, 2007).

The Stats are indicative of increased prevalence of obesity in Iran (Azizi et al., 2005). On the other hand, their major role in lipid peroxidation always plays an important role in the pathophysiology of cardiovascular diseases and diabetes that are among the consequences of obesity (Witztum, 1994). Today, determination and recognition of antioxidant properties along with inflammatory agents has been identified as an appropriate tool for diagnosis and treatment of many chronic diseases. However, it is still not fully understood that under the different conditions that increase the oxidants and free radicals or result in oxidative stress, to what extent does the anti-oxidant defense system contribute to the process? Increased oxidative stress and decreased total antioxidant capacity have been reported in fat individuals (Vincent et al., 1999; Olusi, 2002). Hence, creation of appropriate strategies to reduce oxidative stress and to increase total antioxidant capacity have been the focus of researchers in health sciences.

Among non-pharmacologic solutions and interventions, today exercise and physical activity play an important role as a non-drug therapy in reducing the severity of many chronic and metabolic diseases. The beneficial role of relatively long-term exercise training with anti-inflammatory and antioxidant properties in healthy and patient populations has been discussed many times (Naghizadeh et al., 2010; Onur et al., 2011; de Salles et al., 2010). Although some studies have referred to exercise training for the lack of response to the biochemical variables (Dekker et al., 2007; Hersoug et al., 2007), there are limited studies about the effects of exercise-induced weight loss on total antioxidant capacity or other factors affecting the oxidative stress. Hence, the present study was aimed to determine the effect of aerobic trainings on total antioxidant capacity levels in fat individuals.
MATERIALS AND METHODS

Participants were twenty two inactive obese men that recruited through an advertisement in a local newspaper in this study and assigned to exercise (n=12) or control (n=12) groups matched for age (35-48 years) and body mass index (30-36 kg/m^2). The study protocol was approved by ethics committee of Islamic Azad University, Iran. After the nature of the study was explained in detail, informed consent was obtained from all participants. A medical history to retrieve information about health status, current medications and a physical examination including height, weight, and waist circumference was performed of all participants.

Inclusion and exclusion
Inclusion criteria for the test group were: healthy, middle age, obesity (BMI ≥ 30). Neither the control or diabetic subjects had participated in regular exercise for the preceding 6 months, nor did all subjects have stable body weight. All subjects were non-smokers. The exclusion criteria were hepatic disorders, use of alcohol and a chronic disease such as diabetes, respiratory, kidney diseases or cancer.

Anthropometry
Anthropometric measurements (body height and weight, waist and hip circumference) were performed with the subjects wearing light underwear and without shoes. Subjects wore light clothing and removed all metal items which could interrupt the electronic current during the measurement. Body weight and height were measured with a standard physician’s scale when subjects were in a fasting state before the resting metabolism session. BMI was calculated as weight in kilograms divided by the square of height in meters (kg/m^2). Abdominal circumference and hip circumference were measured in the most condensed part using a non-elastic cloth meter. All anthropometrical markers were measured before and after exercise intervention of two groups.

Blood analysis and protocol
Blood samples were obtained before and after exercise intervention after an overnight fast between 8.00 and 9.00 am. Blood used to measure total antioxidant capacity by FRAP. Endurance exercise intervention was performed for 10 weeks involved 3 sessions per week. Each session involved warm up, main exercise and cool up. Main exercise of each session consisted of running without slope or cycle ergometry for 30-45 min at an intensity range between 55-75 % of Max heart rate. Participants wore heart rate monitors to ensure that they were reaching target heart rate levels. After the last session bout, subjects rested for 48 h before blood sampling for posttest.

Statistic
Data were analyzed by computer using the Statistical Package for Social Sciences (SPSS) for Windows, version 15.0. The Kolmogorov-Smirnov test was applied to determine the variables with normal distribution. Differences between groups were calculated using the independent samples t-test. Pre- and post-training of variables were compared using a paired-samples t-test. The relation between total antioxidant capacities with anthropometrical markers were assessed by Pearson’s correlation coefficients. A P value of less than 0.05 was considered statistically significant.

RESULTS
We previous mentioned that the current study was performed to assess effects of 10 weeks endurance training on total antioxidant capacity in obese men. According to data of independent T test and what shows table 1, we can see no significant differences in anthropometrical markers between two groups at baseline (p ≥ 0.05). No significant difference in total antioxidant capacity was also found between two groups at baseline (p ≥ 0.05).

Based on data from paired T test, aerobic training program resulted in significant decrease in body fat percentage in exercise group when compared with its value in pretest (p < 0.001, Fig 1). We also observed that other anthropometrical makers such as body weight, BMI and abdominal circumference decreased significantly by aerobic intervention (p < 0.05). In contrast, total antioxidant capacity did not change by aerobic intervention compared to pretest (p = 0.623, Fig 2). All variables remained without change in control groups (p ≥ 0.05). Total antioxidant capacity was not correlated with all anthropometrical markers after training program in exercise group (p ≥ 0.05).
Table 1: Mean and standard deviation of anthropometric characteristics and serum IL-6 of studied groups at baseline.

<table>
<thead>
<tr>
<th>Variables</th>
<th>Training group</th>
<th>Control group</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (year)</td>
<td>35.6 ± 2.84</td>
<td>35.8 ± 1.49</td>
</tr>
<tr>
<td>Weight (kg)</td>
<td>102 ± 3.9</td>
<td>101 ± 12.2</td>
</tr>
<tr>
<td>Height (cm)</td>
<td>175 ± 3.9</td>
<td>176 ± 2.8</td>
</tr>
<tr>
<td>Body Fat (%)</td>
<td>33.1 ± 3.7</td>
<td>32.1 ± 2.46</td>
</tr>
<tr>
<td>Body mass index (kg/m²)</td>
<td>33.2 ± 3.4</td>
<td>32.8 ± 3.29</td>
</tr>
<tr>
<td>Abdominal circumference (cm)</td>
<td>107 ± 11.2</td>
<td>106 ± 10.7</td>
</tr>
<tr>
<td>Hip Circumference (cm)</td>
<td>106 ± 10</td>
<td>104 ± 9.9</td>
</tr>
<tr>
<td>Total antioxidant capacity (mmol/L)</td>
<td>0.395 ± 0.054</td>
<td>0.385 ± 0.044</td>
</tr>
</tbody>
</table>

Table 1: Mean and standard deviation of anthropometric characteristics and serum IL-6 of studied groups after intervention.

<table>
<thead>
<tr>
<th>Variables</th>
<th>Training group</th>
<th>Control group</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (year)</td>
<td>35.6 ± 2.84</td>
<td>35.8 ± 1.49</td>
</tr>
<tr>
<td>Weight (kg)</td>
<td>97 ± 13.4</td>
<td>102 ± 12</td>
</tr>
<tr>
<td>Height (cm)</td>
<td>175 ± 3.9</td>
<td>176 ± 2.8</td>
</tr>
<tr>
<td>Body Fat (%)</td>
<td>29 ± 2.39</td>
<td>32.1 ± 2.73</td>
</tr>
<tr>
<td>Body mass index (kg/m²)</td>
<td>31.5 ± 3.31</td>
<td>32.9 ± 3.25</td>
</tr>
<tr>
<td>Abdominal circumference (cm)</td>
<td>100 ± 10.8</td>
<td>106 ± 10.7</td>
</tr>
<tr>
<td>Hip Circumference (cm)</td>
<td>100 ± 0.03</td>
<td>104 ± 9.9</td>
</tr>
<tr>
<td>Total antioxidant capacity (mmol/L)</td>
<td>0.388 ± 0.05</td>
<td>0.392 – 0.059</td>
</tr>
</tbody>
</table>

Fig 1: Pre and post training of Total antioxidant capacity: Lack significant change by exercise intervention.
DISCUSSION

The relationship between obesity and increased body fats and oxidative stress and reduced antioxidant capacity has been propounded. Hence, it is thought that weight loss and reduced body fats in fat individuals is associated with improvements in total antioxidant capacity as the resultant of all antioxidants of the body. But the findings of this study showed that although three months of aerobic exercise was associated with weight loss in fat men, it did not affect the total antioxidant capacity. In other words, the weight loss or reduction of body fat levels did not change the total antioxidant capacity in studied obese men in response to exercises program. However, some recent studies have supported the significant increase of total antioxidant capacity after similar training programs in fat people (Tofighi et al., 2010).

The antioxidant molecules have both enzymatic and non-enzymatic sources and the total antioxidant capacity is a resultant of all the antioxidants of the body (Serafini et al., 2004). Theory of association of obesity with increased oxidative stress and decreased antioxidant capacities is previously reported by other studies (Furukawa et al., 2004; Keaney et al., 2003; Ozata et al., 2002). Also in the present study, a significant inverse correlation was observed between total antioxidant capacity, body fats and body mass index in fat men. In fat people, due to increased fat accumulation in adipose tissue and blood, the lipids are targeted by free radicals (Furukawa et al., 2004). Studies have shown that the body's antioxidant levels such as beta-carotene and vitamins E and C in blood circulation are decreased compared to the people with normal weights (Kuno et al., 1998; Ohrvall et al., 1993). On the other hand, increased oxidative stress condition in fat individuals may be due to the more consumption of oxygen in the heart muscle and other active organs, the outcome of which is the generation of superoxide or hydrogen peroxide in the mitochondrial respiratory chain (Kukrajia et al., 1992). In this context, the studies of animal species have shown that the removal of electrons from the electron transport chain in mitochondria leads to reduction of oxygen molecules into superoxide radicals. Regardless of the discussed mechanisms, decreased activity of antioxidant enzymes such as glutathione peroxidase or superoxide dismutase also play a special role in increased oxidative stress in fat individuals (Olusi, 2002; Ozata et al., 2002).

Scientific studies have shown that after exercise training, the oxidative stress in fat people increases more than people with normal weight (Vincent et al., 2004). These studies support the increased oxidative stress following short-term
exercise activities, particularly, high intensity exercise. Although exercise activities are associated with increased free radicals or oxidants, but under these conditions, the antioxidant system comes into play with the same intensity. In other words, the antioxidant capacity of the mammalian organ systems is compatible with oxygen consumption and production of free radicals. Therefore, those tissues with the highest rate of oxygen consumption under different circumstances also have the highest antioxidant capacity. The same issue also applies to the skeletal muscles, somehow. So that the skeletal muscles with the highest antioxidant capacity has a higher oxidative capacity than the muscles with low oxidative capacity (Powers et al., 2000).

The unchanged total antioxidant capacity following the weight loss induced by exercise program was observed in fat people when the exercise program was accompanied by significant reduction in body weight and body fats. On the other hand, in confirmation of previous studies (Keaney et al., 2003; Ozata et al., 2002), there was a significant and inverse relationship between total antioxidant capacity and abdominal environment and body fat percentage in pre-training conditions that reflects the effective role of obesity on total antioxidant capacity. Hence, the lack of improvement of this antioxidant index following training program may be attributed to some effective non-controlled factors. The unchanged total antioxidant capacity in our study may be rooted in individual's diet during training program, because this study was merely aimed at investigating the effect of exercise program on total antioxidant capacity in the absence of dietary control.

REFERENCES
Olusi SO. (2002). Obesity is an independent risk factor for plasma lipid peroxidation and depletion of erythrocyte cytoprotective enzymes in humans. Int. J. Obes. Relat. Metab. Disord. 26:1159-1164.