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# Lower Total Antioxidant Capacity in Smokers Compare to Non-smokers

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ABSTRACT: Smoking is characterized by increased free radicals and stress oxidative. The objective of present study was to compare total antioxidant capacity between adult men with or without cigarette smoking. For this purpose, sixteen untrained adult smoker's men and the same number non-smokers matched for age  $40 \pm 5.6$  year, body weight  $93 \pm 11$  kg and height  $174 \pm 4$  cm was assigned to this study by accessible sampling. Venous blood was collected from subjects after an overnight fast between 8:00 a.m. and 9:00 a.m. Blood samples were used for evaluation serum total antioxidant capacity. Participants were non-athletes and non-alcoholics. Independent sample T-test was used to compare total antioxidant capacity between smoker and non-smokers. A P-value of < 0.05 was considered to be statistically significant. Data showed that total antioxidant capacity was significantly higher in smoker than non-smoker subject (p = 0.012). Based on these finding, we support that smoking is associated with decreased antioxidant capacity and stress oxidative. Future studies should examine the potential role of smoking on oxidant/antioxidant capacity balance.

Keywords: Antioxidant capacity, Smoking, Inflammation

## INTRODUCTION

Despite frequent notice of irreversible disadvantages and consequences of smoking in public media and by other forms of advertising, the consumption of cigarette is growing dramatically in both developed and developing countries. In that, based on the available statistics, even in previous years, almost one-third of population over age 30 years are smokers [1]. On the other hand, the more recent figures and statistics indicate that more than 700 million children are secondhand smokers [2, 3]. In addition, the consequences of smoking are not only temporary, rather according to the existing evidence, there is a direct correlation between cigarette smoking and many cardiovascular and respiratory, as well as atherosclerosis problems along with morphological changes of vessel walls [4].

It has been reported that the smokers are more prone to cardiovascular problems caused by increased production of free radicals as well as decreased level of anti-oxidants. Such conditions are in fact resulted from imbalance between the production of oxidants, as well as defense capacity and capability of anti-oxidants of immune system [6]. Decrease in protective systems of anti-oxidants, due to cigarette smoking, is reported as the cause of many pathological conditions [7]. Since, in addition to reduced antioxidant capacity, increased level of free radicals and several oxidant agents is among the outcomes of cigarette smoking [8]. However, in a relatively recent study, the activity level of super-oxidant as an enzymatic antioxidant has been reported higher in the smokers [9]. In another study, lower levels of superoxide dismutase activity have been reported lower in smokers than non-smokers [10]. In contrast, some other researches have reported higher activity of that enzyme in cigarette smokers [11, 12, and 13]. With respect to the research findings from the comparison of the levels of some antioxidants between cigarette smokers and non-smokers, as well as according to few number of comparative studies into the total capacity of antioxidants, as the representation of all existing antioxidants, the present study is conducted aiming for making comparison between a group of male smokers and non-smokers in terms of total capacity of antioxidants.

### MATERIAL AND METHODS

#### A. Human Subjects

We previous mentioned that this study aimed to compare total antioxidant capacity between adult men smokers and those with non-smoking. For this purpose, sixteen untrained adult smokers men and the same number non-smokers matched for sex (men), aged (40  $\pm$  5.6 year of old) and body weight (93  $\pm$  11 kg) were selected for participate in this study by accessible sampling.

## B. Inclusion and exclusion criteria

All participants were non-athletes and non-alcoholics. All subjects were non-smokers and had not participated in regular exercise/diet programs for the preceding 6 months. Inclusion criteria to study for smoker group were smoking history of At least 10 cigarettes a day for 5 years for smoker group [14]. Those with type II diabetes, respiratory and cardiovascular diseases, cancer, kidney dysfunction and other chronic diseases were excluded. Each participant received written and verbal explanations about the nature of the study before signing an informed consent form.

### C. Anthropometric measures

All participants of two groups matched for age and anthropometrical markers. For this purpose, each subject's body weight and height were measured. Body mass index (BMI) was calculated by dividing body mass (kg) by height in meters squared  $(m^2)$ . Weight was measured to the nearest 100 g using digital scales. Standing height was measured to the nearest 0.1 cm with the use of a wall-mounted stadiometer. The abdominal circumference was measured to the nearest 0.1 cm, using a non-extendable flexible tape applied above the iliac crest and parallel to the ground; with the subject standing erect with abdomen relaxed, arms along the body, and feet together. Hip circumference was measured at the maximum circumference between the iliac crest and the crotch while the participant was standing and was recorded to the nearest 0.1 cm.

### D. Laboratory Analyses

For measure total antioxidant capacity, fasting blood samples were collected after an overnight fast between 8:00 a.m. and 9:00 a.m. All participants refrained from any severe physical activity 48 h before measurements. Plasma total antioxidant capacity was measured by FRAP method (the sensitivity of method was 0.1 Units/ml).

## E. Statistical analyses

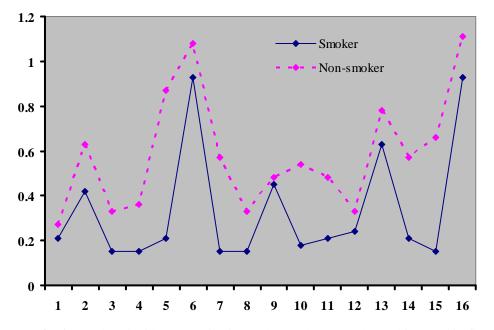
Statistical analysis was performed with the SPSS software version 15.0. An Independent sample T-test was used to compare the serum levels of all resist in between asthma and none-asthma subjects. The Kolmogorov-Smirnov test was applied to determine the variables with normal distribution. Significance was accepted at P < 0.05.

## RESULTS

Total antioxidant capacity was compared between nontrained adult men with or without smoking. Table 1 shows descriptive characteristics of anthropometrical markers and total antioxidant capacity of studied subjects. Data represent by mean and standard deviation. Based on independent samples T test, we observed that non-smokers subject have higher level of total antioxidant capacity when compared with smoker subjects (p = 0.012).

	Exercise group=1,	N	N4		Std. Error
	Control group=2	N	Mean	Std. Deviation	Mean
Age (year)	1	16	41.06	3.696	.924
	2	16	39.13	2.094	.523
Height (cm)	1	16	175.44	2.732	.683
	2	16	174.81	4.460	1.115
Weight (kg)	1	16	93.31	4.840	1.210
	2	16	94.25	7.353	1.838
Abdominal (cm)	1	16	101.81	5.394	1.349
	2	16	103.13	4.992	1.248
Hip (cm)	1	16	101.69	4.672	1.168
	2	16	103.06	5.310	1.327
АНО	1	16	1.0013	.02802	.00700
	2	16	1.0006	.03108	.00777
BMI (kg/m2)	1	16	30.345	1.9514	.4878
	2	16	30.813	1.4823	.3706
Total antioxidant	1	16	.3356	.26860	.06715
capacity (mmol/L)	2	16	.5869	.26038	.06509

 Table 1: Descriptive characteristics of anthropometrical markers and total antioxidant capacity of studied subjects



**Fig 1.** Total antioxidant capacity in smoker and non-smoker subjects. This fig shows that smoker subjects have lower antioxidant capacity than non-smokers.

#### DISCUSSION

According to the statistical findings of this study, male smokers had lower levels of total antioxidant capacity. These findings relatively support the devastating effects of cigarette smoking on antioxidant defense system, as well as the progress of oxidative stress in the presence of cigarette smoking. According to these findings, it can be clearly concluded that reduced antioxidant capacity in cigarette smokers is associated with increased production of oxidants and free radicals. Increase or improvement of antioxidant capacity is facilitated through regular exercise, good nutrition, and more importantly the use of antioxidant supplements, which under different conditions, such as exercising, each antioxidant system shows different immediate or chronic response based on biochemical and biomolecular regulatory mechanisms. These systems are weakened under some conditions. In other words, some internal or external stimuli contribute to decreased antioxidant capacity and consequently to increased production of oxidants or free radicals, based on the stimulation degree. For example, the devastating impacts of smoking, especially cigarette as the most common tobacco product, on the antioxidant system have been frequently discussed. Clinical studies have indicated that per puff of cigarette contains more than 1014 free radicals and is a complex mixture of 4700 chemical compounds [15].

Literature has mentioned that oxidants and free radical particles are the major constituents of cigarette smoking. Investigation on a large group of French people suggest that the plasma levels as well as the absorption of vitamins E and C, as strong antioxidants against free radicals, decrease in cigarette smokers, which is associated with the reduction of antioxidant capacity [16]. Broad studies into increased rate of cardiovascular diseases in the smokers have mostly indicated to the decreased level of anti-oxidants, increased level of oxidized lipid, and increased levels of lipoproteins [17, 18].

Literature has also mentioned that the level of lipid peroxidation is far higher in cigarette smokers than non-smokers, which result in Atherosclerosis due to the destruction of lipids [19]. On the other hand, it has been reported that tobacco decreases anti-oxidant capacity of salvia for unidentified reasons [20]. Researchers also put that the imbalance between the level of free radicals and reactive oxygen species with antioxidants, due to cigarette smoking, has a key role in the onset and development of oral inflammatory lesions [21, 22, and 23]. In this regard, literature has taken cigarette smoking, which contains oxidants and pre-oxidants, as a major resource of free radicals [24].

Under the mentioned conditions and despite increased level of oxidants and free radicals during smoking, it seems that the anti-oxidant system is more active in the smokers. In that, in a relatively recent study, superoxide activity as an enzymatic anti-oxidant agent was reported higher in the smokers, and this difference is indeed due to the higher levels of free radicals in such people [9]. In contrast, another investigation reported lower mean of superoxide dismutase, glutathione peroxidase, and salivary peroxidase in the smokers [25]. In general, scientific resources have indicated that in the smokers, total capacity of antioxidants decreases and the oxidative/anti I oxidative balance evolves towards oxidative [26]. On the other hand, the key role of cigarette smoking in developing oxidative stress and performance disruption of Beta cells have been reported in some researches in the past [27]. Clinical observations revealed that even the second-hand smokers may develop type II diabetes [28].

# REFERENCES

- Zenzes MT. (2000). Smoking and reproduction: gene damage to human gametes and embryos. *Human Reproduction Update*. **6**: 122-131.
- Ekerbicer HC, Celik M, Guler E, Davutoglu M, Kilinc M. (2007). Evaluating environmental tobacco smoke exposure in a group of Turkish primary school students and developing intervention methods for prevention. BMC Public Health. 7: 202.
- Hwang SH, Hwang JH, Moon JS, Lee DH. (2012). Environmental tobacco smoke and children's health. *Korean J Pediatr.* **55**: 35-41.
- Barua RS, Ambrose JA, Eales-Reynolds LJ, DeVoe MC, Zervas JG, Saha DC. (2001).
  Dysfunctional Endothelial Nitric Oxide Biosynthesis in Healthy Smokers with Impaired Endothelium-Dependent Vasodilatation. *Circulation*. 104: 1905-1910.
- Bloomer RJ. (2007). Decreased blood antioxidant capacity and increased lipid peroxidation in young cigarette smokers compared to nonsmokers: Impact of dietary intake. *Nutrition Journal.* **6**: 39.
- Ookawara T, Haga S, Ha S. (2003). Effects of endurance training on three superoxide dismutase isoenzymes in human plasma. *Free Radic Res.* **37**: 713-719.
- Rao GM, Sumita P, Roshni M, Ashtagimatt MN. (2005). Plasma antioxidant vitamins and lipid peroxidation products in pregnancy induced hypertension. *Indian Journal of Clinical Biochemistry*. **20**(1): 198-200.

- Rouzbahani R, Asgary S, Naderi GA, Dehghan Nejad M, Rezaei F. (2009). Comparision of plasma peroxidants, glycosilated hemoglobin, conjugated dienes and CRP level in smokers and non-smokers men. *Journal of Isfahan Medical School.* 27(93): 115-121.
- Baharvand M, Maghami AG, Azimi S, Bastani H, Ahmadieh A, Taghibakhsh M. (2010). Comparison of superoxide dismutase activity in saliva of smokers and nonsmokers. South Med J. 103(5): 425-7.
- Pasupathi P, Saravanan G and Farook J. (2009). Oxidative stress bio markers and antioxidant status in cigarette smokers compared to nonsmokers. *Pharmaceutical sciences and research.* 1: 55-62.
- Aruoma Oi, Kaur H and Halliwell B. (1991). Oxygen free radicals and human diseases. *JR Soc Health.* **111**: 172-177.
- Kocyigit A, Erel O and Gur S. (2001). Effects of tobacco smoking on plasma selenium, zinc, copper and iron concentrations and related antioxidative enzyme activities. *Clin. Biochem.* **34**(8): 629-633.
- Hulea SA, Olinescu R, Nitã S, Crocnan D and Kummerow FA. (1995). Cigarette smoking causes biochemical changes in blood that are suggestive of oxidative stress: a case-control study. *J Environ Pathol Toxicol Oncol.* 14(3-4): 173-180.
- Fahim A, Helke MF, Cindy L, Tracey M, Eric A, Gerald M. (2006). The Relationship between Plasma Adiponectin Concentration and Insulin Resistance Is Altered in Smokers. *The Journal of Clinical Endocrinology & Metabolism.* 91(12): 5002-5007.
- Axelsen M, Eliasson B, Joheim E, Lenner RA, Taskinen MR, Smith U. (1995). Lipid intolerance in smokers. J Intern Med. 237(5): 449-455.
- US Department of Health and Human Services. The Health Consequences of Involuntary Exposure to Tobacco Smoke: A Report of the Surgeon General. Washington DC: Public Health Service. Office on Smoking and Health; 2006.
- Miller ER, III, Appel LJ, Jiang L, Risby TH. (1997). Association between cigarette smoking and lipid peroxidation in a controlled feeding study. *Circulation.* **96**(4): 1097-101.
- Schooler C, Feighery E, Flora JA. (1996). Seventh graders' self-reported exposure to cigarette marketing and its relationship to their smoking behavior. *Am J Public Health*. **86**(9): 1216-21.

- Henriksen T, Mahoney EM, Steinberg D. (1983). Enhanced macrophage degradation of biologically modified low density lipoprotein. *Arteriosclerosis.* **3**(2): 149-59.
- Battino M, Ferreiro MS, Gallardo I, Newman HN, Bullon P. (2002). The antioxidant capacity of saliva. *J Clin Periodontol.* **29**(3): 189-94.
- Pasupathi P, Rao YY, Farook J, Saravanan G, Bakthavathsalam G. (2009). Effect of cigarette smoking on lipids and oxidative stress biomarkers in patients with acute myocardial infarction. *Res J Med Sci.* 4(2): 151-9.
- Preston AM. (1991). Cigarette smoking-nutritional implications. *Prog Food Nutr Sci.* **15**(4): 183-217.
- Osecika M, Erelb O, Sevincc E, Selekb S. (2005). Increased oxidative stress in children exposed to passive smoking. *Int J Cardiol*. **100**(1): 61-4.

- Yildiz L, Kayao<sup>°</sup>glu N, Aksoy H. (2002). The changes of superoxide dismutase, catalase and glutathione peroxidase activities in erythrocytes of active and passive smokers. *Clin Chem Lab Med.* **40**(6): 612-5.
- Abdolsamadi H, Goodarzi M, Mortazavi H, Robati M. (2011). Comparison of salivary antioxidants in healthy smoking and non-smoking men. *Chang Gung Med J.* **34**(6): 607-11.
- Nazeri S, Hedayati M, Ahmadvand H. (2013). The comparision of serum antioxidant capacity and superoxide dismutase and catalase activity of cigarette smokers to nonsmokers. *Yafteh.* **15**(3): 70-75.
- Ding EL, Hu FB. (2007). Smoking and type 2 diabetes: underrecognized risks and disease burden . JAMA. 298: 2675-6.
- Foy CP, Bell RA, Farmer DF, Goff DC, Wagenknecht LE. (2005). Smoking and Incidence of Diabetes Among U.S. Adults. Findings from the Insulin Resistance Atherosclerosis Study. *Diabetes Care.* **28**: 2501-7.