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RESEARCH ARTICLE

EFFECTS OF CIGARETTE SMOKINGON OXIDATIVE STRESS AND SERUM MINERAL CONCENTRATIONS IN MALE SMOKERS OF KURNOOL DISTRICT

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Abstract:
Smoking has been accepted as a risk factor for many chronic diseases, including cardiovascular diseases, respiratory diseases, cancer, ulcers and osteoporosis. Tobacco smoke contains many oxidants and free radicals that can cause damage to lipids, proteins, DNA, carbohydrates and other biomolecules The purpose of this study is to examine the effect of cigarette smoking on serumoxidative damage, antioxidant status, and mineral concentrations in male subjects.Subjects were randomly chosen from different work places in Kurnool city. Serum oxidative defense enzyme activities (CAT andglutathione-s- transferase) and serum mineral (Zn, Cu and I) were evaluated. Serum zincand copper concentrations were similar in the two groups. Thesefindings show that there is depletion in the non enzymatic antioxidant change dependingon their co factor concentrations in tobacco smokers.

Keywords: Smoking, free radicals, , damage biomolecules, minerals , enzymes , cofactors, smokers .

Introduction

Smoking has been accepted as a risk factor for many chronic diseases, including cardiovascular diseases, respiratory diseases, cancer, ulcers and osteoporosis. Tobacco smoke contains many oxidants and free radicals that can cause damage to lipids, proteins, DNA, carbohydrates and other biomolecules.

Smoking is a widely accepted practice in India men and is associated with socialising, sharing, and male identity. Although smoking is a recognized risk factor for several diseases including emphysema, chronic bronchitis, cardiovascular diseases, and cancer .very little is known about the nutritional consequences of smoking.Each puff of a tobacco contains 104 oxidants in the tar phase and 105 in the gas phase. It has been demonstrated that one of the prominent risk factors for increased lipid peroxidation is smoking (Kocyigit A, Erel O, Gur S et al. 2001).Smoking may enhance oxidative stress not only through the production of reactive oxygen radicals in smoke but also through a weak of the antioxidant defense systems. Cigarette smoke may promote atherogenesis by producing oxygen – derived free radicals that damage lipids(Palanisamy pasupathi, G. Saravanan and Farook;et al. 2009).smoking produces Reactive oxygen species (ROS) which involved in many cellular metabolic and signaling processes and are thought to have a role in aging and smoking. Therefore, their detoxification and elimination are necessary for normal physiologic cell activity and survival. To defend themselves against these free radical attacks, cells have developed various antioxidant systems. Several enzymatic systems can detoxify free radicals, copper/zinc superoxide dismutase (SOD) catalyzes the conversion of the superoxide anion to hydrogen peroxide and works concomitantly with hydroperoxide, removing enzymes such as catalase and aselenoprotein , glutathione peroxidase (GPX) .

The present study was conducted to determine the effect of cigarette smoking on change in antioxidant status (glutathione , superoxide dismutase and catalase) in smoker healthy subjects and compared with non smokers healthy subjects.The potential damage that can be caused by free radicals is normally minimized by a combination of biological antioxidant systems including enzymatic and non-enzymatic reactions. Important antioxidant enzymes include copper and zinc (CuZn SOD) and manganese (Mn SOD) superoxide dismutase, catalase, selenium glutathione reductase (GSH-Red). Ascorbic acid, α-tocopherol, and urate can also act to reduce the concentration of free radicals.

Materials and Methods :

The patients who had a history of smoking 10 or more cigarettes per day were considered smokers, and those who never smoked were controls. The study did not include females in this study because smoking is not a norm among females inIndian society. This study included only heavy smokers who smoked at least 10 sticks per day, and excluded mild or casual smokers to leave a buffer zone of comparison between smokers and non-smokers.

Forty healthy, males ranging in age of 19-35 years, from Kurnool, volunteered to participate in this study. Thirty six of the volunteers had never smoked.. None of the volunteers had any history of cardiovascular, endocrine or gastrointestinal disorders, and none were receiving medication or taking any nutritional supplement.

The procedures of the study were approved by a research committee, and a written informed consent was signed from all volunteers after careful explanation of the purpose and procedures of the study.

Blood pressure and anthropometric measurements (weight and height) were done by well-trained staff. Blood pressure (mm Hg) was measured on the same arm with a standard cuff while the participant was sitting and in a relaxed position. Three separate measurements were taken and the average was recorded. All anthropometric measurements were taken with the participant wearing light clothing, standing relaxed and looking straight ahead, with arms at the sides, feet together and with weight equally distributed over both legs (14.) The weighing scale was zeroed before and after every measurement and standardized with a certified weight every day.

Volunteers were asked not to smoke for more than 10 hours before sampling to exclude the effects of acute smoking on the blood parameters studied. Two overnight fasting blood samples were collected from all volunteers. The first blood samples were centrifuged at 3000 *g*for ten minutes at room temperature and then serum was stored at -80°C until analyzed for the, zinc, copper and selenium. The second blood samples were centrifuged at 1700*g* for twenty minutes at 4°C, then serum was stored at -80°C until analyzed for serum vitamin C. Hemolyzed samples were excluded from the analysis. Serum concentrations of trace elements were measured by an atomic absorption spectrophotometer. Zinc and copper concentrations were determined after dilution with 6% 1-butanol solution by using flame atomic absorption spectrophotometer (20). Serum selenium determination was preformed after (1:4) dilution with 0.05% TritonX 100 in 0.125% (v/v) nitric acid by atomic absorption spectrophotometer with a graphite furnace (21)

Data analysis: Analysis of data was performed using the Statistical Package for Social Sciences version 11.0 (SPSS) computer software.

Results and Discussion

Some of the chemicals in cigarette smoke generate a large number of free radicals, 22. Recent studies have demonstrated that antioxidants, including vitamin C and some trace elements such as selenium, zinc and copper, protect the body against reactive oxygen species (ROS).

1. In thepresent study no statistical differences in body weight, height were observed between smokers and nonsmokers. These results are similar to the results of Kim *et al.* 2003, who found that the slightly lower body weight of smokers was probably secondary to a lower calorie intake in the smoking group compared to the non-smoking group. Kim *et al.* reported that cigarette smoking was not associated with a reduction in height.
2. Regarding the effect of smoking on hypertension, the average blood pressure in smokers and non-smokers was in normal range; however, the diastolic and systolic blood pressures were significantly ($P<0.05$) higher in smokers than in non-smokers and ranged to the upper limit of the normal range. This observation suggests that the hypertension, typically reported in smokers, reflects the effects of chronic and long-term vascular damage as mentioned by Kim *et al.*
3. These results were confirmed in the present study for the effect of cigarette smoking on trace elements as shown in Table 3 which shows that smoking decreased serum zinc and selenium, but increased copper concentrationsattributed to the hormonal changes induced by cigarette smoking, i.e., the increased release of corticosteroids and catecholamins.(Dubick *etal.* 1991 and Kim *et al.* 2003, have reported increased zinc. concentrations in smokers)
4. The lower serum vitamin C concentrations due to cigarette smoking are consistent with several investigators who have pointed out that smokers

Table : 1 Characteristics of the study subjects

Subjects (n)		Age (year)		Number of cigarette smoked per day	Duration of smoking (year)
Smokers	Non-Smokers	Smokers	Non- Smokers		
40	36	19 ± 16	21 ± 15	11.5 ± 1.9	10.2 ± 4.1

* Data are given as mean ± SD.

Table – 2

Comparison of height, body weight, blood pressure, and daily nutrient intakes between smokers and non-smokers*		
Variable	Smokers	Non-smokers
Height (cm)	166±2||	168 ±3
Body weight (kg)	59±4||	62±5
Blood pressure(mmHg)		
Diastolic	67± 2§	79±2
Systolic	101±3‡	107±2
Reported daily nutrient intake		
Energy (kcl/d)	1850±80§	2141±100
Ascorbic acid(mg/d)	27.2±3§	71.1±7
Ascorbic acid intake/energy intake	0.02	0.03

*Data are presented as mean ± standard error of the mean.
§P<0.001. ‖ Not significant by t test at ∝ = 0.05. ‡ P<0.05. † n = 19/group.

Table 3 : Comparison of plasma parameters of tobacco smokers and non-smokers

Variable	**Smokers (n= 40)**	**Non-smokers (n=36)**	***P***
Cu µg/dl	98.4 ± 21	92.1 ± 14.1	0.103
Zn µg/dl	68.4 ± 12.4	72.0 ± 12	0.484
Se µg/l	60.3 ± 12.9	88.9 ± 18.1	000
Fe µg/dl	106.2 ± 35	115.2 ± 34.6	0.443

Table : 4 Levels of serum lipids and antioxidant status in non smokers and smokers subjects

Parameters	Smokers	Non-smokers
MDA (u mol/L)	2.11± 0.7*	1.13± 0.45
GSH (u mol / L)	39.18 ± 16.38**	66.64± 18.61
SOD (u mol / L)	27.0 ± 10.63**	75.54 ± 11.34
CAT (u mol / L)	39.84 ± 20.79**	75.54 ± 11.34

possess a lower level of plasma antioxidants, especially vitamin C. (Mezzetti, A., Lapenna, D., Pierodomenico, S.D., Calafiore, A.M et al 1995, Halliwell, B. and Gutteridge, J.M. et al. 1990).

5. The serum MDA levels were significantly higher ($p < 0.05$)in smokers compared with non-smokers which indicate the oxidative damage of cigarette smoking . SOD along with CAT preventive antioxidants , plays a very important role in protection against lipid peroxidation . In this study, SOD and CAT activities were significantly lower smokers than in non smokers. CAT has been suggested to play an important role in the protection against oxidative stress (Valco, M., Leibfritz, D., Moncol, J. ,et al. 2007). Serum glutathione levels are believed to be predicators of morbidity and mortality (Venkatesan, A., Hemalatha, A., Bobby, Z., et al. 2006) . GSH plays a key role in protecting cells against electrophiles and free radicals.GSH can act directly as a free radical scavenger by neutralizing hydroxyl radicals, or indirectly by repairing initial damage to macromolecules inflicted by hydroxyl radicals. It is essential in the maintenance of protein and non protein SH group in reduced form (Goraca A., Skibska B et al. 2005).

Conclusion

Antioxidant defensces act as balanced and coordinated system and each relies on the action of the other.(Evans, P and Halliwell, B. et al. 2001) The oxidative damage observed in smokers can be associated with the direct effects of oxidants in cigarette smoke and the consequences of lower antioxidant nutrition status associated with smoking. Although the best medical advice for this population is to stop smoking, the present data also suggest that this population has an increased requirement for dietary antioxidants. Consistent with this concept, vitamin C supplements have been reported to reduce the extent of oxidative damage in smokers. The potential value of antioxidant supplements for smokers is underscored by the observation that dietary antioxidant intakes tend to be lower in smokers than in non-smokers. Campaigns aimed at improving the anti-oxidant status of this group should be mounted in conjunction with antismoking campaigns.

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