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Original article

Quitting smoking replenishes body antioxidant status

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ABSTRACT

The effects of smoking on human health are destructive and widespread. Cigarette smoke contains large amounts of free radicals which lead to increased oxidative stress in smokers than in non-smokers. The present study was done to evaluate the extent of oxidative damage caused by smoking. For this purpose 75 subjects were included in the study and divided into three groups: Group A consisted of 25 subjects who were non-smokers and served as controls, Group B (25 subjects) consisted of smokers and in Group C, 25 subjects were included who were smokers earlier but had quitted smoking. Levels of glutathione peroxidase (GPx), catalase (CAT), superoxide dismutase (SOD) and malondialdehyde (MDA) were measured in blood of all the groups and compared. It was observed that the antioxidant enzymes (GPx, CAT and SOD) were decreased ($p<0.001$) while lipid peroxidation products (MDA) were increased ($p<0.001$) in smokers as compared to non-smokers. Further it was observed that extent of oxidative damage increased proportionately with increase in amount and duration of smoking but oxidative stress decreased significantly in the subjects who had quitted smoking as compared to smokers.

1. Introduction

Cigarette smoking is probably the most addictive and dependence producing form of object-specific, self-administered gratification known to man. According to present estimates, tobacco is responsible for causing 3.5 million deaths every year. One million of these deaths occur in developing countries [1]. Numerous known and suspected tumorigenic agents have been identified in the gaseous and particulate phases of tobacco smoke [2]. Cigarette smoke contains two very different populations of free radicals, one in tar and one in the gas phase [3]. The most important free radicals in biological systems are radical derivatives of oxygen [4]. Oxidation of the Poly unsaturated fatty acids (PUFA) generates a fatty acid radical (L•) that rapidly adds oxygen to form a fatty acid peroxyl radical (L0O•). The peroxyl radicals are the carriers of the chain-reaction, they can oxidize further PUFA molecules and initiate new chains, producing lipid hydroperoxides (LOOH) that can breakdown to yet more radical species and to a wide range of compounds like alkanals, alkenals, hydroxyalkenals, malondialdehyde (MDA) and volatile hydrocarbons [5].

Antioxidant defences against the deleterious actions of free radicals are divided into two main categories: those whose role is to prevent the generation of free radicals and those that intercept any that are generated [6]. Some of these antioxidants are naturally produced in the body while others are taken exogenously. Decreased body antioxidant status as a result of excessive smoking leads to depletion of the antioxidant mechanisms of the body which predisposes various organs to free radical damage. Quitting smoking reduces the oxidative stress in human body thereby replenishing the antioxidant defense system.

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2.Materials and Methods

The present study was undertaken in the Department of Physiology, Adesh Institute of Medical Sciences and Research, Bathinda.

2.1.Study Design

This study was carried out on total number of 75 subjects and they were divided into the following groups:

Group A (Non-smokers):
In this group 25 healthy male non-smokers in the age group of 25-45 years were selected to serve as controls.
Group B (Smokers):

This group consisted of 25 male smokers in the same age group. Group C (Ex-Smokers): This group consisted of 25 subjects who smoked for more than 10 years but have now quitted smoking for more than 2 years.

2.2.Selection of Subjects:

1. The subjects were selected from general population and from amongst the attendants of the patients visiting the medical outpatient department in Adesh Institute of Medical Sciences and Research, Bathinda.
2. Persons smoking more than 10 cigarettes per day for more than 10 years were included in the study.
3. For control group, the non-smokers who were exposed to passive smoking were not included in the study.
4. Persons suffering from any disease like diabetes mellitus, rheumatoid arthritis, malignancy, tuberculosis, parkinsonism, Alzheimer's disease, CVA, ischaemic heart disease and persons taking antioxidant drugs were not included in the study.

2.3.Collection and processing of blood samples:

10 ml of venous blood was taken under fasting conditions with a dry, disposable syringe and needle (21 gauge) under all aseptic conditions by venepuncture in the antecubital vein in heparinized vials for biochemical investigations. The Erythrocytes were washed with cold isotonic saline and used for estimation of GPx and CAT while the plasma was used for estimation of SOD and MDA.

2.4.Methods of analysis:

The subjects in the study and control groups were screened for plasma SOD by the method of Marklund and Marklund [7]. CAT was measured colorimetrically by the method of Sinha [8] and Glutathione Peroxidase was assayed as described by Paglia and Valentine [9]. Malondialdehyde (MDA) was used as an indicator of lipid peroxidation and was estimated by the method of Satoh [10].

The case history was recorded and a complete clinical examination of the subjects was done.

Statistical analysis was carried out by Student's paired't'-test. The data were expressed as Mean ± SD and the p value < 0.05 was taken as significant.

3.Results

Table 1 shows the mean ± SD of the various parameters studied in controls, smokers and persons who had quitted smoking. There was statistically significant decrease in levels of antioxidant enzymes (SOD, CAT, GPx) in smokers as compared to non-smokers. The levels of MDA were significantly higher in smokers than in controls. On comparing these levels in persons who have quitted smoking, the antioxidant status in them was found to be similar as that in healthy controls. Table 2 shows that with increase in number of cigarettes smoked per day, the antioxidant status of the blood deteriorated as was evident from the progressive decrease in levels of antioxidant enzymes and increase in levels of MDA. According to table 3, the levels of SOD, CAT, GPx decreased while the levels of MDA increased with increase in duration of smoking from 10 to 25 years.

Table 1: Showing Mean + SD of levels of Superoxide dismutase, Catalase, Glutathione peroxidase and MDA in study and control groups

Group	No. of subjects	SOD (u/ml) Mean±S.D	Catalase (KU/g Hb) Mean±S.D	GPx (U/g Hb) Mean±S.D	MDA (nmol/ml) Mean±S.D
Group A (Controls)	25	3.76 ± 0.41	32.95 ± 3.04	47.14 ± 3.12	3.54 ± 0.82
Group B (Smokers)	25	2.15 ± 0.33**	16.43 ± 2.86**	29.66 ± 3.75**	4.78 ± 0.91**
Group C (Ex-Smokers)	25	3.65 ± 0.22**++	30.32 ± 2.31**++	35.23 ± 2.08*+	4.43 ± 1.02*+

Statistical comparison was done between: Group A and Group B; Group A and Group C; Group B and Group C.
* p<0.01, ** p<0.001, ***p>0.05 when Group B and C compared with Group A;
+ p< 0.01, ++ p<0.001 when Group C compared with Group B

Table 2: Showing relation of amount of smoking with Superoxide dismutase, Catalase, Glutathione peroxidase and MDA levels.

No. of cigar-ettes smoked per day	No. of subjects	SOD (u/ml) Mean±S.D	Catalase (KU/g Hb) Mean±S.D	GPx (U/g Hb) Mean±S.D	MDA (nmol/ml) Mean±S.D
Nil (Controls)	25	3.87±0.27	32.95 ± 3.04	47.14 ± 3.12	3.55±1.03
10-15	13	2.76±0.41**	24.28 ± 3.53*	29.66 ± 3.75**	4.61±0.87*
16-20	08	2.12±0.29**	15.04 ± 2.94**	18.93 ± 2.08**	4.98±0.65**
21-25	04	1.85±0.36**	19.97 ± 2.57**	17.40 ± 1.99**	6.12±1.26**

* p<0.01, ** p<0.001 when compared with Controls

Table 3: Showing relation of duration of smoking with Superoxide dismutase, Catalase, Glutathione peroxidase and MDA levels.

Duration of smoking (in years)	No. of subjects	SOD (u/ml) Mean±S.D	Catalase (KU/g Hb) Mean±S.D	GPx (U/g Hb) Mean±S.D	MDA (nmol/ml) Mean±S.D
Nil (Controls)	25	3.93±0.64	32.95 ± 3.04	47.14 ± 3.12	3.13±1.43
10-15	12	3.20±0.42**	16.03 ± 1.96**	37.38 ± 4.26*	4.83±1.19**
16-20	07	2.57±0.39**	17.15 ± 2.01**	14.29 ± 2.77**	4.45±1.63*
21-25	06	2.16±0.16**	15.77 ± 2.62**	14.40 ± 2.53**	5.21±1.49*

* p<0.01, ** p<0.001 when compared with Controls

4.Discussion

Cigarette smoke contains large number of oxidants that can be inhaled into the body. These oxidants lead to damage of all the constituents of cell including proteins, lipids, carbohydrates and DNA. Oxidation of amino acids in proteins leads to physical changes like fragmentation, aggregation and proteolytic degradation. Oxidized carbohydrates react with protein molecules to form glycated products which have been implicated in the pathogenesis of diabetes mellitus. DNA damage induced by oxidants includes both base alterations and strand breaks resulting in misreading by DNA polymerase [11]. These errors in DNA replication can eventually result in mutagenesis and carcinogenesis.

The increase in oxidative stress caused by cigarette smoking is evident from our study as we observed a significant lowering of antioxidant enzymes i.e. SOD, CAT, GPx in the plasma of smokers as compared to non-smokers. SODs are a family of metalloenzymes that convert superoxide anion to hydrogen peroxide. It is considered to be stress protein which is synthesized in response to oxidative stress. Catalase is a primary antioxidant defense component that works to catalyze the decomposition of hydrogen peroxide to water. Glutathione peroxidases are selenoenzymes which catalyze the reduction of hydroperoxides to water. The lipid peroxidation end product i.e. MDA is also raised in the plasma of smokers as the oxidants lead to damage of cellular lipids by causing a self perpetuating chain reaction called lipid peroxidation [12]. Peroxidation of membrane lipids disrupts the lipid bilayer and structural organization. Lipid peroxidation has been implicated in a wide range of tissue injuries and diseases e.g. atherosclerosis.

The decrease in levels of SOD, CAT, GPx and increase in levels of MDA with greater daily smoking quantity signifies that the extent of oxidative stress caused in an individual is directly proportional to the amount of smoking. Similar findings have been reported by other authors [13]. Chronic smokers who are addicted to cigarette smoking for a longer duration of time tend to have more deleterious effects of oxidants than those who are smoking for a relatively shorter duration of time. The oxidants continuously act and lead to more and more damage of body tissues leading to severe disorders like Bronchogenic carcinoma, coronary artery disease, hypertension, ageing, chronic bronchitis, etc. Additional studies have also suggested that smoking duration has a stronger effect in the prediction of lung cancer risk than number of cigarettes smoked per day [14].

Smokers should always be motivated to quit smoking as quitting smoking not only stops the oxidative damage to the body but also reverses the body antioxidant defense system and the benefits become more evident with the passage of time [15,16]. Our study supported the same fact as we observed that after quitting smoking the plasma antioxidant enzyme levels started rising and the lipid peroxidation levels started decreasing [17]. The chances of developing smoking related lung carcinomas and other disorders can be minimized through smoking cessation [18]. Indeed, cessation of smoking prior to middle age is associated with a more than 90% reduction in cancer risk attributed to tobacco [19] and the risk of death diminishes soon after cessation of smoking [20].

5.Conclusion

Smoking increases oxidative stress in the body which is a reason for various life threatening diseases. The oxidative damage to body tissues increases with increase in amount and duration of smoking. Cigarette smoking cessation is followed by a marked increase in plasma antioxidant concentrations which substantially improves plasma resistance towards oxidative challenge. Therefore quitting smoking represents an irreplaceable preventive strategy against tobacco-induced oxidative stress.

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