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Cigratte Smoking Induced Oxidative Stress in Human

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Today millions of people die due to cigarette smoking in the world. Cigarette smoking is one major consequence to develop oxidative stress will damage the lung lead to death. The aim of the study is to evaluate the oxidative stress by novel markers like SOD and Catalase between young chronic smokers and non- smokers in the fasting state.

Key word: SOD, Catalase, TBARS, LPx , ROS, Smokers and Non – smokers.

Tobacco smoking is the act of burning the dried or cured leaves of tobacco plant and inhaling the smoke for pleasure, for ritualistic or social purposes, self medication or to satisfy physical dependence or addiction. *[Burns DM et.al (2001*)] .Tobacco smoke contains nicotine , an addictive stimulant. The effect of nicotine is first time or irregular uses is an increase in alertness and memory & mild euphoria. *[Kandel DB et.al (2000)]*.In chronic users, nicotine simply relieves the symptoms of confusion, restlessness, insomnia, anxiety and dysphoria. Nicotine also disturbs metabolism and suppress appetite. This is because nicotine act as stimulant which increase blood sugar level. Nicotine acts as an agonist that binds to nicotine acetylcholine receptor sites in the brain and body and effect the body by lowering levels of acetylcholine receptor stimulation can affect respiration, heart rate, memory alertness and muscle movement until the receptors are resensitized or restimulated *[Balfour DJ et.al (2002)]*. The different forms of tobacco smoke contains several chemicals and carcinogens which increase free radical and may leads to oxidative stress.

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MATERIALS AND METHODS

Fifty healthy male non-smokers in the age group of 20-35 years were placed in the group I. Fifty male smokers in the age group of 20-35 years were placed in the age group II. Blood samples were collected from smoking volunteers were smoke since past 15 years. Blood samples were collected from each of the subject for 90 days and analyzed for the following parameters :

TBARS,(Nichans and Samuelson *et.al*) Lipid hydroperoxide (Jiang *et.al)* Superoxide dismutase (Kakkar *et.al.*(1984) Catalase, (Sinha *et.al)*.

RESULTS

The level of the lipid peroxide was lowest in non-smokers (89.10 ± 3.60 in blood) and highest in smokers (143 ± 56.65 in blood).The increase was statistically significant in group II than group I. The Total thiol level were also higher in smokers than the non-smokers (2.16 ± 0.60 in smokers & 0.89 ± 0.26 in non –smokers) blood sample. The superoxide dismutase (SOD) level was higher in non – smokers (9.32 ± 0.70 in blood) than smokers (4.54 ± 0.84 in blood).The catalase showed a similar trend, where the levels is increased in non-smokers than smokers (196.51± 3.58 in group I blood and 86.03 ± 2.98 in group II blood). The significant increment in the levels of serum TBARS and lipid hydroxide by 142.5% and 60.62% respectively and decrement in the level of superoxide dismutase, catalase by 56.47% was recorded in smokers when compared to that of non- smokers.

Table 1. Levels of serum lipidhydroperoxides &TBARS in Non-smokers and smokers

Lipid profile	Non-smokers	Smokers	p value
Lipidhydroperoxides (mg/dl)	89.10 ± 3.60 (n =50)	143 ± 56.65 (n =50)	< 0.001*
TBARS (mg/dl)	0.89 ± 0.26 (n =50)	2.16 ± 0.60 (n =50)	< 0.001*

Table 2. Levels of Antioxidants in non-smokers and smokers

Lipid profile	Non-smokers	Smokers	p value
SOD (Unit /mg protein)	9.32 ± 0.70 (n = 50)	4.54 ± 0.84 (n = 50)	< 0.001*
CAT (Unit /mg protein)	196.51 ± 3.58 (n = 50)	86.03 ± 2.98 (n = 50)	< 0.001*

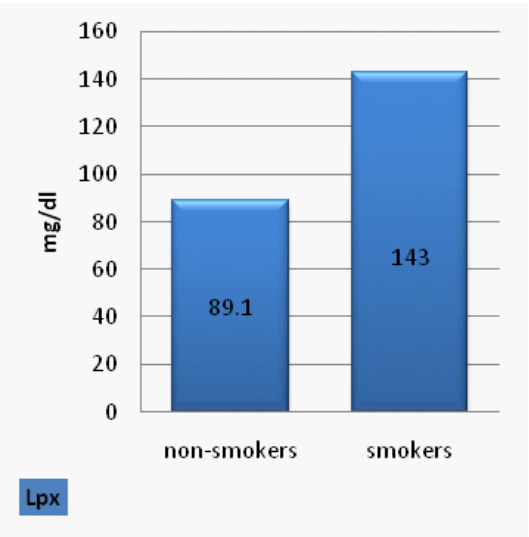

Fig. 1. Levels of serum Lipid hydroperoxide in non-smokers and smokers

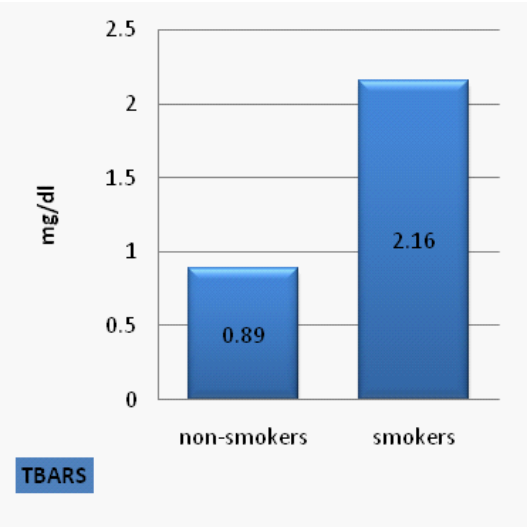

Fig. 2. Levels of serum TBARS in non-smokers and smokers

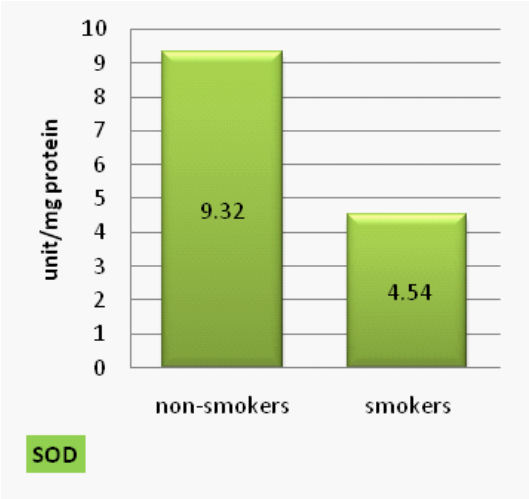

Fig. 3. Levels of serum SOD in non-smokers and smokers

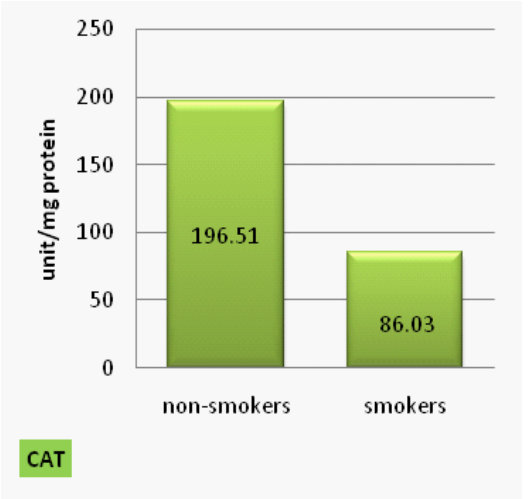

Fig. 4. Levels of serum catalase in non-smokers and smokers

DISCUSSION

Nicotine of cigarette results in excessive generation of free radicals[*Mjos OD et.al(1988), Yang Q et.al(1993) , Cryer PE et.al(1981), Pittilo et.al(1982)*]. Free radicals are the reactive oxygen species (ROS) are known to cause oxidative damage to number of molecules in cell; including membrane, lipids, proteins, and nucleic acids[*Woolf N et.al(1981), Mathew et.al(1987), comporti et.al(1987), Isayama et.al(2003)*]. The level of lipid per oxidation in the smokers serum was assessed by measuring the levels of thiobarbituric acid reactive substances (TBARS) and hydroperoxide [*Oteiza PI et.al(1995), Oteiza PI et.al(1996) Frie B et.al(1991)*] . The increased TBARS and hydro peroxide levels in the serum of smoking people indicate enhanced lipid per oxidation leading to tissue injury.(Table I)The cellular antioxidant defense mechanism, which include scavenging activities of enzymes Via superoxide dismutase and catalase play an important role in scavenging toxic intermediates of Reactive oxygen species . superoxide dismutase is metalloproteins catalyzing the dismutation of Superoxide anion to hydrogen and oxygen. [Fridovich J *et.al* (2001), Marlund *et. al.,* (1984)] . Numerous studies have shown the importance of SOD in protecting cells against oxidative stress.[*Carlson EJ et.al(1997)*] The decrease in SOD activity in blood implies, the activity of SOD in tissue decreased during alcohol ingestion(Table-II). This decrease could be due to the feedback inhibition or oxidative inactivation of enzyme protein due to excess ROS generation. [*Michiels Cet.al(1997*)] Catalase is a heme protein which catalyzes the reduction of hydrogen peroxides which act as preventive antioxidant plays an important role in protection against the deleterious effect of lipid per oxidation.[*Dinkova-Kostova AT et.al(2002*)] . The activity of levels of tissue catalase was decreased in smokers when compared to that of non-smokers, the inhibition of CAT activity is due to enhanced synthesis of singlet oxygen(O 2*) during the smoking of cigarette since singlet oxygen (O2*) is a powerful inhibitor of catalase.

The cellular antioxidant defense enzymes SOD & CAT were significantly reduced in smokers, this might have let decreased antioxidant defense and increased oxidative stress and thereby it may leads to tissue injury.

CONCLUSION

The study results show that smoking increases the level of free radicals (i.e.) significant increment in the levels of TBARS and lipid hydroperoxide indicate that the oxidative stress induced in human by smoking.

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