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Protective effect of Omega-3 fatty acids on blood lipid profile of cigarette smoke exposed rats

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Abstract : *The present study is undertaken to investigate the role of Omega-3 fatty acids on blood lipid profile viz serum cholesterol, serum triglyceride, high density lipoprotein (HDL), low density lipoprotein (LDL), and very low density lipoprotein (VLDL) in cigarette smoke exposed albino rats. Experimental rats (100-158g) were kept in standard laboratory conditions and grouped into three sets – control set A exposed to ambient air, experimental set (B) was exposed to cigarette smoke for one hour per day for 28 days and experimental set (C) was exposed to cigarette smoke with pre-exposure supplementation of Omega-3 fatty acids. A significant decrease in serum cholesterol ($p<0.05$), serum triglyceride ($p<0.05$), LDL ($p<0.05$) and VLDL ($p<0.05$) with a significant increase in HDL level ($P<0.05$) after supplementation of Omega-3 fatty acids in comparison to cigarette smoke exposed albino rats.*

Key Words: Cigarette smoke, Albino rat, Blood lipid profile, Omega-3 fatty acids

Introduction

Now a days, cigarette smoke has become a great curse of indoor air pollution. It is the dominant risk factor for cardiovascular and cerebrovascular diseases and it is an escalating public health problem (Lakier, 1992; Heitzer and Munzel, 1996). Cigarette smoke is the known source of oxidant and contains more than 4,000 identified constituents which are harmful for the health (Kamisaki *et al.*, 1997). It produced by incomplete combustion of tobacco and generates a high free radical load *in vivo*. Inhaled smoke delivers toxic chemicals to the blood stream of the smokers through lungs. Increased production of free radicals in gas phase and tar phase of cigarette smoke produces oxidative stress and can result in the oxidation of lipids, consequently perturbs the antioxidant defense system (Ambrose and Barua, 2004). Blood lipids are well known factors associated with the development of cardiovascular disease. Fat builds up more easily on arterial wall damage by cigarette smoke and alters the level of lipoprotein (Garrison, *et al.*,1978; Stubbe, *et al.*,1982). Omega-3 fatty acids are essential fatty acids which can help in reducing the toxic effects of cigarette smoking.

Materials and Methods

Adult healthy wistar albino rats of weight ranging from 100-158g of both the sexes were kept in polypropylene cages. Inbred colony of albino rats were maintained at animal house of Zoology Department in standard condition of temperature 21±0.5°C and relative humidity 60±5% with a photoperiod 12 hours/day. The rats were fed on Goldmohar rat and mice feed, pellet and water *ad libitum*. Experimental animals are acclimatized for one month prior to experiment.

Selection of cigarette : Cavanders Gold leaf non-filtered cigarette, Godfrey Phillips India Ltd., Chakala, Andheri East, Mumbai was selected for the present study.

Selection of antioxidant : Mega-3 gelatin capsules, Dr. Reddy's Laboratories Ltd., Hyderabad was used as an antioxidant. 30mg/100g body weight Omega-3 fatty acids were given to each rat/day for four weeks by gavaging.

Experimental protocol : The albino rats were

Table 1. Blood lipid profile (mg/dl) after cigarette smoke exposure and pre-exposure supplementation with Omega-3 fatty acids in albino rats

Set	Exposure	Time period	Serum cholesterol (mg/dl)	Serum triglyceride (mg/dl)	HDL (mg/dl)	LDL (mg/dl)	VLDL (mg/dl)
			Range (Mean ± S.Em)	Range (Mean±S.Em)	Range (Mean ± S.Em)	Range (Mean ± S.Em)	Range (Mean ±S.Em)
Control Set-A(5)	Ambient air	28 days	82.00-84.00 (82.08±0.36)	81.00-86.21 (83.66±0.84)	22.00-35.00 (28.70±2.58)	40.13-60.20 (49.87±3.65)	19.10-28.39 (23.73±1.64)
Experimental Set-B(5)	Cigarette smoke	28 days	85.00-96.12 (89.67±1.81)***↑	85.00-92.50 (88.22±1.27)**↑	18.92-25.42 (21.83±1.25)**↓	49.50-71.25 (63.35±3.68)**↑	23.50-33.52 (29.18±1.66)**↑
Experimental Set-C(5)	Cigarette smoke + Omega-3 fatty acids	28 days	82.00-85.21 (84.03±0.79)*↑	78.69-87.90 (83.10±1.68)*↑	24.00-31.14 (26.97±1.39)*↓	42.21-60.22 (50.52±3.21)*↑	19.50-31.19 (26.20±2.05)*↑

S.Em= Standard Error
(5) = No. of rats

* - Non-significant
** - Significant
*** - Highly significant

↑ - Increase
↓ - Decrease

grouped into three sets- Control set A and experimental sets (B and C) contain six rats each.

Control set-A: Exposed to ambient air for one hour per day for four weeks.

Experimental set-B: Exposed to cigarette smoke one hour per day for four weeks.

Experimental set-C: Exposed to cigarette smoke with pre-exposure supplementation of Omega-3 fatty acids for one hour per day for four weeks.

Exposure to cigarette smoke : Experimental rats were kept in an isolated smoke chamber with their cages for the exposure to cigarette smoke. The rats were subjected to the whole body exposure of cigarette smoke of a non-filter cigarette (delivered as 3 cigarettes, 3 times) per day for four weeks at the bottom of smoke chamber by slow suction.

Collection of blood sample : At the end of exposure period rats were dissected and blood samples were collected from the ventricle of heart in sterilized plain centrifuge tubes for separation of serum.

Estimation of blood lipid profile : Serum cholesterol by Roeschalu *et al.* (1974). Serum triglyceride by Scheltter and Nussel (1975) method. HDL by Wybenga and Pileggi (1970) method. LDL and VLDL by Friedwald *et al.* (1972) method.

Statistical analysis : Analysis of variance (ANOVA) has been applied to the data obtained for statistical analysis with help of computer software (KpKy plot).

Results and Discussion

Data obtained for blood lipid profile in control and experimental sets are given in (Table1 and Fig 1). In the present study, a significant increase in serum cholesterol ($p<0.01$), serum triglyceride ($p<0.05$), LDL ($p<0.05$) and VLDL ($p<0.05$) with corresponding decrease in HDL level ($p<0.05$) have been observed after exposure to cigarette smoke in relation to control set (A).

Cigarette smoke immediately increases markers of oxidative stress. It leads to the uptake

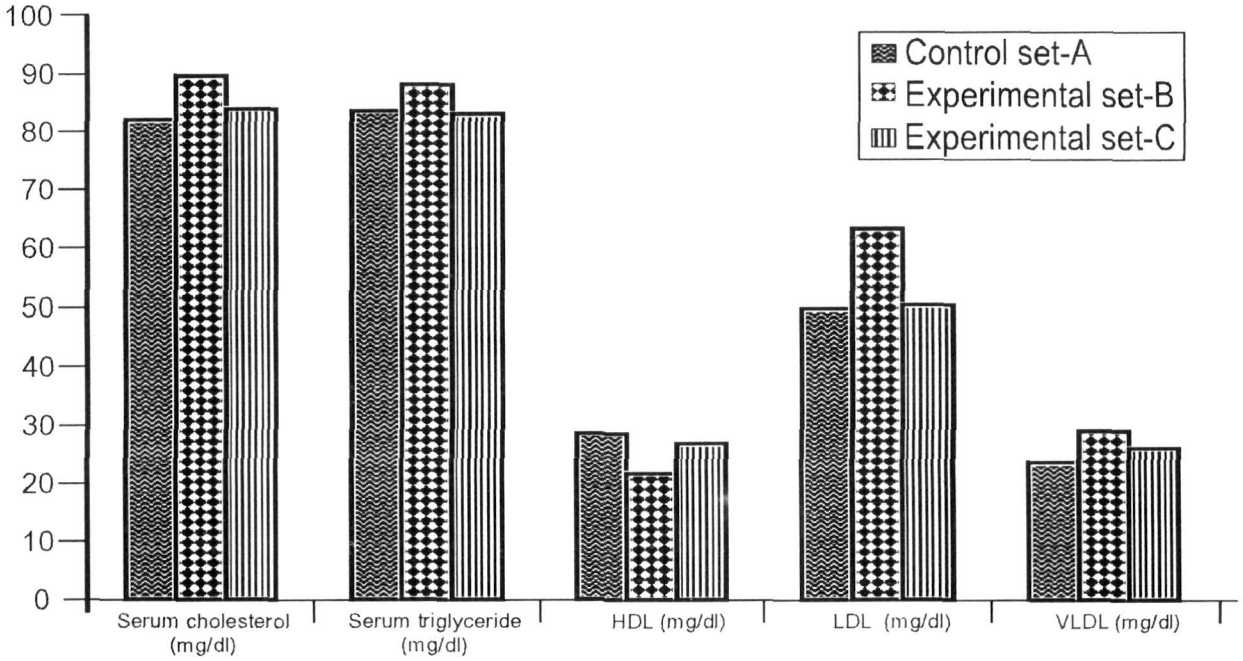

Fig.1 Graph showing blood lipid profile (mg/dl) after cigarette smoke exposure and pre-exposure supplementation with Omega-3 fatty acids in albino rats

of many hazardous compounds, such compounds or their metabolites may be electrophilic and thereby able to react with biological macromolecules, or they may give rise to oxidative stress by formation of reactive species or the initiation of radical chain reactions (Orhan *et al.*, 2005). Free radicals in cigarette smoke mediated lipid peroxidation (Anbarasi *et al.*, 2005). Lipid peroxidation is frequently used as an indication of tissue oxidative stress as a result free radicals attack on the cell membrane, leading to increased membrane permeability and cellular damage (Halliwell and Gutteridge, 1989). Increased oxidative stress and generation of free radicals can result in the modification of LDL to oxidized LDL that could lead to atherosclerotic lesion (Kharb and Singh, 2000). The role of free radicals in pathogenesis of atherosclerosis via oxidation of LDL that damage the arterial wall (Harman, 1992). Cigarette smoke also decrease the activity of paraxonase, an enzyme that protect against LDL oxidation (Nishio and Watanabe, 1997). After supplementation of Omega-3 fatty acids the blood lipid profile viz. serum cholesterol, serum triglyceride, LDL and VLDL significantly decreases with increase in HDL level. Omega-3 fatty acids are essential fatty acids which contain EPA and DHA which help in reducing inflammation and protect cell membrane damage due to lipid peroxidation by suppressing oxygen free radicals and reduce lipolysis (Pant, 2004). It has an anti-inflammatory action which inhibits synthesis of cytokines and mitogens that augment the inflammation and promote plague formation (Uauy and Valenzuela, 2000).

Present study suggests that supplementation of Omega-3 fatty acids mitigate the toxic effect of cigarette smoke on blood lipid profile and can reduce the factors of cardiovascular diseases.

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