

Active and Passive Smokers - A Haematobiochemical and Cytogenetic Study

K. Sasikala, F. Regina Rosalin, A.L. Calistus Jude, R. Ashok Kumar, S. Sudha, M. Vimala Devi, N. Balachandar, K.A. Sajeetha Beegam, N. Meenakshi and Asia Begum

Division of Human Genetics, Department of Zoology, Bharathiar University, Coimbatore 641 046, Tamil Nadu, India

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ABSTRACT Use of tobacco is now pandemic and it is probably the most important preventable causes of human morbidity and mortality. This study is aimed to find out the effect of smoking on RBC and WBC counts, mean corpuscular volume, haemoglobin content, lipid profile and chromosomal complements among Active and Passive smokers. In smokers, the RBC count and haemoglobin content were found to be decreased and the WBC count and mean corpuscular volume were elevated and statistically significant results were noted in lipid profile. A significant number of minor chromosomal aberrations were observed among the different groups of Active smokers

INTRODUCTION

The scenario of selecting this work was to find out the effect of smoking on the human chromosomes because, cigarette smoke is one of the important mutagenic factor which causes damage to human genetic material (Jin et al. 1997).

Every cigarette reduces the life span by about 5 minutes. Globally, smoking kills more than 4 million people every year and by 2020 it is likely to cause more premature deaths and disability than single diseases.

The chemical composition of burning tobacco and paper produce more than 4000 chemicals compounds in the form of gases, vapours and particulates. Some of these are carbon monoxide (CO), hydrogen cyanide, phenols, ammonia, formaldehyde, benzene (a) pyrene, nitrosoamines, nicotine and tar. According to Khan (1996) cigarette smoke also includes heavy metals, radio active products, poisons and at least 48 known cancer - producing substances. Among the gases produced by tobacco, CO is found to be more toxic. It binds to haemoglobin in red blood cells and interferes with the body's ability to transport and utilize oxygen (Joan Luckman 1990).

Tobacco smoking is the most important risk

factor associated with chronic bronchitis and emphysema (Flenley et al. 1980). Parental smoking is said to exacerbate respiratory diseases in children. Maternal smoking has been shown to be a leading cause of pediatric deaths from low birth weight, short gestation, respiratory distress syndrome and sudden infant death syndrome (Bhatia and Vijayan 1994). The frequency of stillbirth was found to be increased with paternal smoking habits (Bridges et al. 1974). Rincon et al. (1999) in his work found that cigarette smoking appears to have profound effects on glucose transport in skeletal muscle.

Cytogenetic studies by Chung-Hua-Liu-Hsing and Ping-Hseuh-Tsa-Chil (1999) showed that cigarette smoking is one of the important mutagenic factors which causes damage to human genetic material. Doll and Peto (1976) and Demarani (1983) demonstrated that variable chromosome change such as chromosomal aberrations and sister chromatid exchanges are known to be caused by cigarette smoking.

The present study was undertaken to correlate haematobiochemical and cytogenetic effects of cigarette smoke in active and passive smokers and to apply this information for better investigation and management.

METHODS

Subjects for the present study were selected using questionnaire from the general public residing in Coimbatore city, Tamilnadu, India. 150 Active smokers and 150 Passive smokers were selected for the study. Equal numbers of normal, healthy individuals were selected to serve as Controls.

The Experimental subjects were categorised as A (Active Smokers) and B (Passive Smokers). Further grouping was done based on their age group as Group I - 20 to 30 years; Group II - 31 to 40 years; Group III - 41 to 50 years and above. The fresh blood samples were utilized for estimating Total Erythrocyte (RBC) Count, Total

Leucocyte (WBC) Count, Total Haemoglobin content, Mean Corpuscular Volume, Estimation of serum High Density Lipoprotein (HDL) and Low Density Lipoprotein (LDL) and Chromosomal Analysis.

RESULTS

Among 300 Experimentals (150 Active smokers; 150 Passive smokers), 43 Active and 23 Passive smokers exhibited chronic bronchitis. Mild smoker's cough was found in 19 Active and 8 Passive smokers. Upper respiratory tract infection was detected in 22 Active and 9 Passive smokers. 16 smokers (13 Active and 3 Passive smokers) displayed Hyperactivity. Myalgia-chest was noted in 11 Active smokers.

Table 1 depicts the frequency distribution of different haematobiochemical parameters in the smokers and control subjects. The mean RBC content of the Controls of groups I to III were 5.94 ± 0.690 , 6.293 ± 0.603 and 6.41 ± 0.437 million cells / cu.mm. The Experimentals of A and B of groups II and III displayed statistically significant decrease with regard to mean RBC count [Group II (A): 4.013 ± 0.197 , (B): 4.83 ± 0.297 ; Group III (A): 4.056 ± 0.207 , (B): 4.936 ± 0.523 million cells/cu.mm] when compared with the Control groups.

Except Passive smokers of Group I, all the Active and Passive smokers of Groups I to III exhibited elevated mean WBC count when compared to that of their respective Controls and these increased values were statistically significant.

In contrast to the above parameters, the mean Haemoglobin level decreased significantly when compared to the Controls [Group I (A): 12.5 ± 0.507 ; (B): 14.43 ± 0.829 ; Group II (A): 12.6 ± 0.279 , (B): 14.6 ± 0.396 and Group III (A): 12.56 ± 1.124 , (B): 14.56 ± 1.107 gm/dl].

Distribution of Mean Corpuscular Volume (MCV) among Active and Passive smokers exhibited an elevated range than that of the Controls and all Experimentals of A and B exhibited statistically significant results.

All the Experimental samples of Active smokers of Group I, II and III recorded decreased mean HDL level which were statistically significant, whereas the decrease in HDL levels in samples of Passive smokers did not show statistically significant values. The mean LDL level of all the Active smokers of Group I, II, III and Passive smokers of Group II and III showed a statistically significant elevation when compared to Controls.

All the 150 Controls and 150 Experimentals of Active and Passive smokers were subjected to chromosomal analysis. Chromosomal aberrations such as chromatid gaps, chromatid breaks and dicentric chromosomes were observed among the Experimentals A and B (Table 2). The Control cultures of different Groups (I, II, III) displayed a total number of 4, 8 and 6 chromosomal aberrations, whereas the Active smokers of similar groups displayed 14, 24 and 28 chromosomal aberrations which were statistically significant over their Controls.

In contrast to the Active smokers, the Passive smokers did not show a statistically significant

Table 1: Distribution and serum levels of hematobiochemical parameters (RBC, WBC, Hb, MCV, HDL & LDL) among active and passive smokers and the controls.

S. No.	Particulars	No. of subjects studied	RBC (million cells /cu.mm)	WBC (1000 cells/ cu.mm)	Hg (g/dl)	MCV (fl)	HDL (mg/dl)	LDL (mg/dl)
1. Group I								
	Control	45	5.94 ± 0.690	9100 ± 179.9	15.53 ± 0.916	89.83 ± 1.491	62.58 ± 1.97	145.90 ± 7.92
	Experimental A	45	$4.316 \pm 0.207^*$	$10500 \pm 19.9^*$	$12.5 \pm 0.507^*$	$111.7 \pm 3.96^*$	$49.94 \pm 1.74^*$	$208.78 \pm 16.93^*$
	Experimental B	45	$4.54 \pm 0.303^*$	9800 ± 127.6	$14.43 \pm 0.829^*$	$104.43 \pm 2.76^*$	61.57 ± 1.96	161.78 ± 9.63
2. Group II								
	Control	53	6.293 ± 0.603	9633.3 ± 197.6	15.5 ± 0.697	89.96 ± 1.97	61.43 ± 1.17	158.62 ± 6.76
	Experimental A	53	$4.013 \pm 0.197^*$	$10866.6 \pm 201.3^*$	$12.6 \pm 0.279^*$	$115.13 \pm 2.97^*$	$37.62 \pm 1.96^*$	$214.83 \pm 11.34^*$
	Experimental B	53	$4.83 \pm 0.297^*$	$1016.66 \pm 159.9^*$	$14.6 \pm 0.396^*$	$102.33 \pm 2.76^*$	$59.96 \pm 2.27^*$	$172.04 \pm 13.72^*$
3. Group III								
	Control	52	6.41 ± 0.437	9700 ± 223.6	15.73 ± 1.366	89.76 ± 2.72	63.72 ± 1.93	145.21 ± 13.97
	Experimental A	52	$4.056 \pm 0.207^*$	$11100 \pm 106.6^*$	$12.56 \pm 1.124^*$	$116.46 \pm 2.63^*$	$39.7 \pm 1.89^*$	$224.82 \pm 10.93^*$
	Experimental B	52	$4.936 \pm 0.523^*$	$10266.6 \pm 112.9^*$	$14.56 \pm 1.107^*$	$104.33 \pm 2.37^*$	$59.74 \pm 1.66^*$	$165.50 \pm 9.67^*$

Group I : 20-30 years
 Group II : 31-40 years
 Group III : 41 years and above

* Significant at 5% levels
 Experimental A : Active smokers
 Experimental B : Passive smokers

Table 2: Frequency distribution of chromosomal aberrations among active and passive smokers and the controls.

S. No.	Particulars	No. of subjects studied	Total no. of metaphase plates examined	Chromatid type aberrations			Total no. of chromosomal aberration	
				Gaps	Breaks	Dicentric	Total	%
1	<i>Group I</i>							
	Control	45	50	2	-	-	2	4
	Experimental A	45	50	5	2	1	7	14 *
	Experimental B	45	50	2	1	-	3	6
2.	<i>Group II</i>							
	Control	53	50	2	1	-	4	8
	Experimental A	53	50	7	3	1	12	24 *
	Experimental B	53	50	3	2	1	5	10
3.	<i>Group III</i>							
	Control	52	50	2	1	-	3	6
	Experimental A	52	50	9	3	2	14	28 *
	Experimental B	52	50	4	2	1	7	14
Group I : 20-30 years				*Significant at 5% level.				
Group II : 30-40 years				Experimental A : Active smokers				
Group III : 41 years and above				Experimental B : Passive smokers				

frequency of Chromosomal aberrations (6,10 and 14 total chromosomal aberrations in Groups I, II and III) even though they displayed an increased percentage of aberrations over the Controls.

DISCUSSION

An elevated WBC count and decreased RBC count in the smokers with respect to non-smokers was reported earlier by Van Furth (1970) and Corre et al. (1971) examined 4,264 men and reported that smokers showed an increase in the number of leucocyte especially granulocyte and monocyte. The present study corroborates with the above studies with respect to the counts of RBC and WBC.

In the present study the mean haemoglobin content of the Active smokers showed a sharp decrease with respect to the Control subjects while in the Passive smokers the decrease was found to be meager. The MCV was found to increase in the Experimentals A and B of Group I, II and III with respect to their Controls and the values were found to be statistically significant.

The alterations in lipid profiles in Active and Passive smokers in the present study is supported by the following findings. Abdou-Azza-Sadd et al. (1998) demonstrated effect of long term smoking on lipid profile, erythrocyte function and antioxidant enzyme activities in female subjects and showed an increase in serum cholesterol, triglycerides, low density lipoprotein

and glutathionine peroxides in Active smokers. Increased levels of serum and tissue cholesterol and low levels of serum HDL cholesterol associated with cigarette smoking have been reported by several workers (Pollini et al. 1985; Arti and Rajeswari 1986; Padmini and Mortlage 1987).

Verma (1959), Obe and Herha (1978) and Sinus et al. (1990) found a greater frequency of chromosomal aberrations in lymphocyte chromosomes from smokers compared with non-smokers. Randerath et al. (1989) reported that cigarette smoke enhances the age dependent increase in chromosomal aberrations. On terms of biological activity, cigarette smoke and its conductors have been shown to form adducts with DNA protein (Perara et al. 1987) and to induce chromosome aberrations (Littlefield and Joinie 1986). Nakayama et al. (1985) reported DNA strand break in smokers due to the effect of electrophilic substances in tobacco such as catechol, methyl derivatives and hydroquinone. The "slower" response of peripheral lymphocytes of smokers as compared with non-smokers found in this analysis may be an expression of influence of cigarette smoke components on cellular immune response in man.

The peripheral blood leucocyte culture of Experimentals A and B, and the Controls showed chromatid type aberrations such as gaps, breaks and dicentrics. The percentage of total chromosomal aberrations of Active smokers was high

when compared to the Passive smokers. The Control cultures however displayed minimal percentage of chromosomal aberrations than the Experimental groups.

According to Evan and O' Riordan (1975) the types of chromosomal damage which can be distinguished at metaphase can be divided into two main groups, chromosome type and chromatid type. The circulating lymphocyte is in the G_0 and G_1 phases of mitosis and exposure to certain mutagenic agents during these stages produces chromosome type damage where the unit of breakage and revision is the whole chromosome (i.e., both chromatid within the same line). However, cells exposed to these while in S or G_2 stages of the cell cycles after the chromosome has divided into 2 sister chromatids, yield chromatid type aberrations and only the single chromatid is involved in breakage or exchange.

CONCLUSION

Tobacco is a poison and needs to be banned for several reasons. Eventhough the chromosomal aberrations recorded in this study are minimum they may contribute in a great way to linkage and affect the progenies.

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