# LIFE STYLE RISK FACTORS FOR THE DEVELOPMENT OF CHRONIC DEGENERATIVE DISEASES IN AN INDUSTRIAL SET UP IN BARODA

SA Desai, UV Mani, SM Deshmukh, UM lyer, AK Sen, RP Patel

## ABSTRACT

Chronic degenerative diseases (CDD) such as obesity, diabetes, hypertension and chronic heart diseases (CHD) are rapidly increasing to epidemic proportions and gaining their hold in the developing countries. It has therefore become essential to know the prevalence of these diseases along with the lifestyle related risk factors, which lead to the development of these diseases. Thus the objective of this study was to find out the prevalence of CDD and to analyze the risk factors in relation to the lipid profile of the subjects. The results indicated the prevalence of overweight to be 42.2%, obesity 8.0%, diabetes 7.4%, hypertension 5.1% and CHD 0.6%. The nutrient composition of the diets of the male and female subjects showed significant differences in the proximate principles as well as in fibre, iron and potassium content. General habits of individuals such as smoking, chewing tobacco and alcohol intake showed significant aberrations in the lipid profile. Further alterations in the lipid profile were also noticed in case of obesity, diabetes, hypertension and CHD. Statistically significant differences were noticed in the fasting blood glucose (FBG), total cholesterol (TC), high density lipoprotein cholesterol (HDL-C), very low density lipoprotein cholesterol (VLDL-C), triglyceride (TG) and TC/LDL-C (TC/L) ratio of the obese subjects when compared with normal subjects (who were not suffering from any kind of diseases). Similar observations were seen when the lipid levels of diabetics and hypertensive subjects were analysed in comparison to the normal subjects. Thus, the present observations indicate that a variety of factors such as dietary and general habits, various diseases etc. influence the prevalence of CDD. Further, these studies indicate the need for large community based intervention programs, which lay emphasis on the modifiable risk factors in preventing the occurrence of these CDD.

**KEY WORDS:** Chronic degenerative diseases; Lifestyle risk factors; Prevalence; Dietary habits.

## **INTRODUCTION**

Chronic degenerative diseases have been one of the leading causes of mortality and morbidity in the present century. Amongst the chronic degenerative diseases, the one which has been identified as a priority area by World Health Organization (WHO) for research in the developing countries, is cardiovascular disease/chronic heart disease. It has been predicted that by the year 2015, CHD will be the most important cause of fatality in India (1). The prevalence of CHD appears to depend on combination of lifestyle, dietary, environmental and population specific risk factors. Studies have shown that elevated levels of blood lipids (TC and TG) are one of the major risk factors for heart disease. Numerous epidemiological studies (2,3,4) have shown that hypercholesterolemia when present for a longer duration leads to atherosclerosis, which in turn may precipitate cardiovascular disease. The incidence of CHD in adults is related positively to levels of low density lipoprotein cholesterol (LDL-C) and negatively to HDL-C (5,6). High levels of LDL-C and low HDL-C may be related more strongly to anatomic lesions of coronary atherosclerosis, in later life. In addition, studies have shown that HDL-C and LDL-C / HDL-C ratio (L/H) is more predictive of CHD than is either LDL-C or TC alone. Thus, serum lipid levels play an important role in the prediction of risk factors for CHD. Interventions for reducing the mortality and risk factors of CDD have been carried out in many countries, especially the Western nations viz. the Stanford Five City Study and the North Karelia Project, that showed reduction in the CHD risk factors by using mass media to educate the people (7). Also, the Malmo feasibility study and the Oslo diet and exercise study, demonstrated that dietary interventions and exercise resulted in a reduction in body mass index (BMI), a significant fall in insulin resistance etc. (8,9). Very few studies have been conducted in India concerning the same. As the geographical and ethnic diversity plays an important role in determining the lipid profile and that causative factors vary from region to region, the

Department of Foods and Nutrition, M S University of Baroda and Gujarat Refinery Hospital, Baroda 390 002, Gujarat, India.

present study was designed to study the effects of various risk factors in relation to the lipid profile of the subjects.

### MATERIALS AND METHODS

Eight hundred and thirty seven subjects in the age group of 20-60 years (mean age  $42 \pm 8$  years, 520 males and 317 females) were enrolled from an industrial set up in Baroda, Gujarat. The dietary information.of the subjects was collected using the 24-hour dietary recall method and the nutritive value of the foods consumed was calculated by using the values given in the 'Nutritive Value of Indian Foods' (10). Information regarding general habits and medical history was obtained through a structured questionnaire. Standing height, weight and circumferences of the waist and hip were measured without shoes. BMI and the waist to hip ratio (WHR) were calculated as a measure of fat distribution.

Venous blood sample was collected after an overnight fast of 12 hours and the serum was used for estimation of various parameters like fasting blood glucose (FBG), TC, TG and HDL-C. The analysis of the parameters was done using the Boehringer Mannheim enzymatic kits on a Boehringer Mannheim autotek instrument. LDL-C and VLDL-C were calculated using the Friedewald's formula (11).

*Statistical Analysis:* Students 't' test was used for finding out the significant difference between the two means.

### RESULTS

The clinical profile of the subjects shows an average age of  $42 \pm 8$  years. Mean height was 160.6 cms (males  $165.1 \pm 6.8$ , females  $152.9 \pm 5.7$  cms) and the mean weight was  $63.8 \pm 11.4$  Kg (males  $66.9 \pm 10.7$ , females  $58.5 \pm 10.7$  Kg). The mean BMI of male subjects was  $24.5 \pm 3.6$  m/kg<sup>2</sup> and that of females was  $25.0 \pm 4.3$  m/kg<sup>2</sup> (average  $24.7 \pm 3.9$  m/kg<sup>2</sup>). WHR of the male and female subjects was  $0.93 \pm 0.06$  and  $0.87 \pm 0.05$  respectively.

Table 1 presents the lipid profile and serum glucose levels of the enrolled subjects. The TG, TC, VLDL-C values were significantly higher in male subjects than in female subjects, while the HDL-C and LDL-C were almost similar in both the groups. Table 1: Fasting Blood Sugar and Lipid Profile ofMale and Female Subjects (mg/dl) (Mean ± SD)

Ν	MALES	FEMALES	TOTAL
n	392	235	627
FBS	$98.8\pm36.3$	87.4 ± 21.3 ***	$94.5\pm32.0$
TC	$200.8\pm39.5$	$189.6 \pm 34.3^{***}$	$196.6\pm38.0$
HDL-C	$38.7\pm8.1$	$37.6\pm8.3$	$38.3\pm8.2$
LDL-C	$124.8\pm42.8$	$127.34\pm29.9$	$124.6\pm40.3$
VLDL-C	$30.0\pm16.1$	$24.1 \pm 13.2^{***}$	$27.3 \pm 15.3$
TG	$151.8\pm79.9$	$120.6 \pm 65.4^{***}$	$140.1\pm75.6$
TC/H	$5.3\pm0.9$	$5.16\pm0.91$	$5.24\pm0.9$
L/H	$3.51\pm0.87$	$3.5\pm0.88$	$3.51\pm0.8$
TC/L	$1.57\pm0.35$	$1.52\pm0.3$	$1.5\pm0.33$
	C* 1 1.CC		0.001

\*\*\* Significantly different from males at P < 0.001

#### Table 2: Dietary Analysis of Subjects (Mean ± SD)

	MALES	FEMALES	TOTAL
ENERGY (Kcal)	1964 ± 582	1516 ± 333***	1792 ± 545
PROTEIN (g)	58.7 ± 17.1	$44.3 \pm 11.4^{**}$	$53.2 \pm 16.7$
FAT (g)	87.6 ± 65.4	$52.83 \pm 18.4^{**}$	74.2 ± 55.1
CARBOHYDRATE(g	) 252.4 ± 86.7	$208.7\pm54.6^{\star}$	$235.6\pm78.6$
FIBRE(g)	$7.3\pm2.4$	$5.8\pm2.3^{\star}$	$6.7\pm2.4$
IRON(mg)	17.8 ± 13.2	10.4 ±4.4*	$13.9\pm10.3$
SODIUM (mg)	$174.9\pm165.3$	$105.3\pm53.8$	$138.7\pm124.5$
POTASSIUM (mg)	$1598.1 \pm 801.3$	1122.2 ± 388*	$1350.2 \pm 659.6$
CAROTENE (mg)	1409 ± 1778.4	$937\pm1310$	$1163.2 \pm 553.6$
VITAMIN C (mg)	74.7 ± 57.1	59.2 ± 49.7	66.5 t 53.3
% CALORIES			
CARBOHYDRATE	51.3	5.1	52.8
PROTEIN	12.0	11.6	11.8
FAT	40.3	31.5	37.2

\* Significantly different from males at P < 0.05. \*\*Significantly different from males at P < 0.01. \*\*\* Significantly different from males at P < 0.001.

The dietary intake of the subjects is given in Table 2. The mean caloric intake of the male and female subjects was  $1964 \pm 582$  Kcal and  $1516 \pm 333$  Kcal respectively. The percent calories coming from carbohydrates, protein and fat was found to be 52.7 %, 11.8 % and 37.2 %. Significant differences were observed amongst the males and females in case of energy, protein, fat, carbohydrate, fibre, iron and potassium intake.

Since fat intake is known to influence the lipid parameters, the data on lipid profile were analysed looking specifically into the fat intake (Table 3). Table 4 depicts the lipid profile of the subjects based on the type of diet consumed, which indicated that the non-vegetarians had significantly higher levels of TG, LDL-C and VLDL-C in comparison to vegetarians.

# Table 3: Lipid Profile of the Subjects in Relation to the Fat Intake (mg/dl) (Mean $\pm$ SD)

	FAT INTAKE	
	<b>30-60 g/day</b>	> 60 g/day
FBG	$100.2\pm51.4$	$100.0 \pm 40.7$
TC	$179.3\pm35.2$	$188.6\pm39.5$
TG	$123.6\pm55.0$	$126.5\pm64.8$
HDL-C	$35.5\pm7.9$	$36.9\pm7.5$
LDL-C	$119.0\pm33.0$	$126.5\pm32.9$
VLDL-C	$24.7\pm11.0$	$25.3 \pm 14.0$
TC/H	$5.2\pm1.03$	$5.21\pm0.95$
L/H	$3.45 \pm 1.01$	$3.52\pm0.93$
TC/L	$1.57\pm0.35$	$1.53\pm0.27$

Table 4: Lipid Profile of the Subjects based on the Type of Diet Consumed (mg/dl) (Mean  $\pm$  SD)

	VEGETARIANS	NON-VEGETARIANS
n	365	118
FBG	$98.6\pm34.9$	$99.5{\pm}40.5$
TC	$201.8\pm38.6$	$198.6\pm41.7$
HDL-C	$38.7\pm8.1$	$38.8\pm8.2$
LDL-C	$97.4\pm60.9$	$118.9\pm44.3^{\ast}$
VLDL-C	$28.6 \pm 14.3$	$32.9 \pm 19.3 *$
TG	$144.9 \pm 69.6$	$165.9 \pm 95.7*$
TC/H	$5.32\pm0.93$	$5.22\pm0.82$
L/H	$3.57\pm0.88$	$3.36\pm0.85$
TC/L	$1.54\pm0.28$	$1.63\pm0.48$

\* Significantly different from vegetarians at P < 0.05.

The lipid profile of the subjects was also compared taking into consideration the smoking habits, tobacco chewing and alcohol intake. In the present study, none of the female subjects smoked, chewed tobacco or consumed alcohol. The impact of smoking on lipid profile revealed that the TG levels were significantly higher amongst smokers than the non-smokers (162.4  $\pm$  75.8 versus 137.2  $\pm$  69.3 mg/dl respectively, p < 0.05). The TC levels were lower in non-smokers but they were not statistically

significant (205.7  $\pm$  35.2 versus 199.9  $\pm$  40.2 mg/dl respectively). With regard to tobacco chewing habbit of the subjects, the TG levels were higher in those chewing tobacco, but the difference was not statistically significant (163.7  $\pm$  96.1 versus 148.9  $\pm$  73.9 mg/dl). The lipid profile of subjects consuming

Table 5: Prevalence of Chronic Degenerative Diseasesamongst the Subjects Enrolled from the IndustrialSetup (Figures in brackets are percentages).

	MALE	FEMALE	TOTAL
n	520	317	837
OVERWEIGHT	214 (41.1)	139 (43.8)	353 (42.2)
OBESE	30 (5.8)	37 (11.7)	67 (8.0)
DIABETES	48 (9.2)	14 (4.4)	62 (7.4)
HYPERTENSION	38 (7.3)	5 (1.5)	43 (5.1)
CHD	5 (1.0)	nil	5 (0.6)

Table 6: Lipid Profile of Normal and Overweight +Obese Subjects (Obese) (mg/dl) (Mean + SD)

n	Males Females Total	NORMALS 136 132 268	<b>OBESE</b> 62 63 125
FBG	Males Females Total	$\begin{array}{l} 91.0 \pm 20.5 \\ 83.5 \pm 16.8 \\ 87.3 \pm 19.2 \end{array}$	$\begin{array}{l} 107.9 \pm 47.1^{***} \\ 90.1 \pm 23.7^{**} \\ 98.9 \pm 38.1^{***} \end{array}$
TC	Males Females Total	$\begin{array}{c} 175.6 \pm 27.6 \\ 173.7 \pm 27.3 \\ 174.7 \pm 27.4 \end{array}$	$\begin{array}{c} 210.8 \pm 43.5^{***} \\ 199.8 \ t \ 36.1^{***} \\ 205.2 \pm 40.2^{***} \end{array}$
TG	Males Females Total	$\begin{array}{c} 118.9 \pm 46.3 \\ 103.4 \pm 52.4 \\ 111.3 \pm 49.9 \end{array}$	$\begin{array}{l} 172.1 \pm 55.4^{***} \\ 141.3 \pm 67.0^{***} \\ 156.6 \pm 89.2^{***} \end{array}$
HDL	Males Females Total	$\begin{array}{c} 34.4 \pm 7.6 \\ 34.7 \pm 7.0 \\ 34.6 \pm 7.3 \end{array}$	$\begin{array}{c} 39.6 \pm 9.8^{***} \\ 39.1 \pm 8.1^{***} \\ 38.3 \ t \ 9.0^{***} \end{array}$
LDL	Males Females Total	$\begin{array}{c} 117.7 \pm 21.2 \\ 117.5 \pm 23.8 \\ 117.6 \pm 22.5 \end{array}$	$\begin{array}{c} 136.8\pm 36.0^{***}\\ 132.6\pm 27.6^{***}\\ 134.7\ t\ 31.9^{***} \end{array}$
VLDL	Males Females Total	$\begin{array}{c} 23.8 \pm 9.3 \\ 20.7 \pm 10.5 \\ 22.5 \pm 10.0 \end{array}$	$\begin{array}{c} 34.4 \pm 21.1^{***} \\ 28.6 \pm 13.3^{***} \\ 31.5 \pm 17.8^{***} \end{array}$
TC/H	Males Females Total	$\begin{array}{c} 5.3 \pm 0.9 \\ 5.1 \pm 0.9 \\ 5.2 \pm 0.9 \end{array}$	$5.5 \pm 0.9$ 5.2 t 0.8 $5.3 \pm 0.9$
L/H	Males Females Total	$\begin{array}{c} 3.6 \pm 0.9 \\ 3.5 \pm 0.8 \\ 3.5 \pm 0.8 \end{array}$	$3.6 \pm 1.0$ $3.5 \pm 0.8$ 3.5 t 0.9
TC/L	Males Females Total	$\begin{array}{c} 1 \ .5 \pm 0.2 \\ 1.5 \pm 0.2 \\ 1.5 \pm 0.2 \\ \end{array}$	$1.6 \pm 0.5^{*}$ $1.5 \pm 0.2$ $1.6 \pm 0.3^{*}$ s at $P < 0.05^{*}$ ***
* Signi	ncantiv diffe	rent from normal	s at $P < 0.05$ . ***

\* Significantly different from normals at P < 0.05. \*\*\* Significantly different from normals at P < 0.001. alcohol showed a significant rise of TG (170.3  $\pm$  95 vs 145.8  $\pm$  72.1 mg/dl, p < 0.01 ), TC (205.6  $\pm$  41 .8 vs 196.1  $\pm$  38.6 mg/dl, p < 0.05) and VLDL-C (33.7  $\pm$  19.2 vs 28.8  $\pm$  14.7 mg/dl, p < 0.01 ) levels. The LDLC levels showed a statistically insignificant rise amongst alcohol consumers (129.4  $\pm$  36.9 vs 123.8  $\pm$  44.0 mg/dl). No significant difference was observed in the HDL-C. Lipid parameters were also analysed taking into consideration the exercise regime of the subjects. No alteration in the lipid profile was observed in those who exercised versus those who did not.

Diseases related to lifestyle such as obesity, diabetes, hypertension and chronic heart disease are rapidly increasing not only in the developed countries, but are also gaining their hold over the developing countries. The prevalence of these diseases in the present study is, 7.4% diabetics, 5.1% hypertensives and 8.0% are obese (Table 5). Significant differences were observed in the lipid profile when the normal subjects were compared HDL-C with overweight + obese, diabetics and hypertensives (Table 6-9).

 Table 7: Lipid Profile of Normal and Diabetic Subjects (mg/dl) (Mean t SD)

		NORMALS	DIABETICS
n	Males	136	39
	Females	132	12
	Total	268	51
FBG	Males	$91.0\pm20.5$	$167.9 \pm 58.6 ***$
	Females	$83.5\pm16.8$	$142.3 \pm 38.4 ***$
	Total	$87.3 \pm 19.2$	161.5 ± 55.6 ***
TC	Males	$175.6\pm27.6$	212.9 ± 53.0 **
	Females	$173.7 \pm 27.3$	$196.5 \pm 24.5 ***$
	Total	$174.7 \pm 27.4$	209.0 ± 48.2 ***
TG	Males	$118.9\pm46.3$	194.5 ± 116.7 ***
	Females	$103.4 \pm 52.4$	178.5 ± 128.0 ***
	Total	$111.3 \pm 49.9$	$190.8 \pm 118.3^{***}$
HDL-C	Males	$34.4\pm7.6$	39.4 ± 10.4 **
	Females	$34.7\pm7.0$	$37.6\pm8.0$
	Total	$34.6\pm7.3$	$39.0 \pm 9.9 ***$
L.DL-C	Males	$117.7 \pm 21.2$	$134.6 \pm 36.6^{***}$
	Females	$117.5 \pm 23.8$	$124.5\pm33.0$
	Total	$117.6 \pm 22.5$	$129.6 \pm 41.9 **$
VLDL-C	Males	$23.8\pm9.3$	$38.9 \pm 23.3^{***}$
	Females	$20.7\pm10.5$	$37.6 \pm 24.6^{***}$
	Total	$22.5\pm10.0$	$37.8 \pm 23.8 ***$
TC/H	Males	$5.3\pm0.9$	$5.5 \pm 1.0$
	Females	$5.1 \pm 0.9$	$5.4\pm0.9$
	Total	$5.2 \pm 0.9$	$5.5 \pm 1.0*$
L/H	Males	$3.6\pm0.9$	$3.5 \pm 1.0$
	Females	$3.5\pm0.8$	$3.3 \pm 1.1$
	Total	$3.5 \pm 0.8$	$3.5 \pm 1.0$
TC/L	Males	$1.5 \pm 0.15$	$1.6\pm0.4^{**}$
	Females	$1.5 \pm 0.2$	$1.8\pm0.9^{\ast\ast\ast}$
	Total	$1.5 \pm 0.2$	$1.7 \pm 0.6^{***}$

\* Significantly different from normals at at P < 0.05. \*\*Significantly different from normals at P < 0.01. \*\*\*Significantly different from normals at P < 0.001.

Table	8:	Lipid	Profile	of	Normal	and	Hypertensive
Subjec	ets	(mg/dl)	) (Mean	±S	<b>D</b> )		

	NORMALS	HYPERTI	ENSIVE
n	Males	136	31
	Females	132	4
	Total	268	35
FBG	Males	$91.0 \pm 20.5$	$90.5 \pm 15.6$
	Females	$83.5 \pm 16.8$	$88.8 \pm 16.3$
	Total	$87.3 \pm 19.2$	$90.3 \pm 15.5$
TC	Males	$175.6\pm27.6$	$200.0 \pm 34.5^{***}$
	Females	$173.7 \pm 27.3$	$175.5\pm28.0$
	Total	$174.7 \pm 27.4$	$197.2 \pm 34.4 ***$
TG	Males	$118.9\pm46.3$	$155.9 \pm 82.0 * * *$
	Females	$103.4\pm52.4$	$176.5 \pm 60.6^{**}$
	Total	$111.3 \pm 49.9$	$157.3 \pm 79.4 ***$
HDL-C	Males	$34.4\pm7.6$	$40.7 \pm 6.2^{***}$
	Females	$34.7 \pm 7.0$	$32.5 \pm 5.8$
	Total	$34.6 \pm 7.3$	$39.8 \pm 6.7 ***$
LDL-C	Males	$117.7 \pm 21.2$	$128.1 \pm 30.6*$
	Females	$117.5\pm23.8$	$107.7\pm31.4$
	Total	$117.6 \pm 22.5$	$125.8\pm31.0$
VLDL-C	Males	$23.8\pm9.3$	$31.2 \pm 16.4^{***}$
	Females	$20.7\pm10.5$	$35.4 \pm 12.1^{**}$
	Total	$22.5\pm10.0$	$31.7 \pm 15.9^{***}$
TC/H	Males	$5.3 \pm 0.9$	$4.9 \pm 0.7$
	Females	$5.1 \pm 0.9$	$5.4 \pm 0.4$
	Total	$5.2 \pm 0.9$	$5.0 \pm 0.7$
L/H	Males	$3.6\pm0.9$	$3.2 \pm 0.8*$
	Females	$3.5\pm0.8$	$3.3 \pm 0.6$
	Total	3.5 t 0.8	$3.2 \pm 0.8*$
TC/L	Males	$1.5 \pm 0.2$	$1.6 \pm 0.3^{**}$
	Females	$1.5 \pm 0.2$	$1.7 \pm 0.3*$
	Total	$1.5\pm0.2$	$1.6 \pm 0.3^{***}$

\* Significantly different from normals'at P < 0.05. " Significantly different from normals af P < 0.01. \*\*\* Significantly different from normals at P < 0.001.

# Table 9: Lipid Profile of Normal and Coronary HeartDisease (CHD) Subjects (mg/dl) (Mean ± SD)

NORMALS	CHD
136 (Males)	5 (Males)
$91.0\pm20.5$	$138.2 \pm 95.1 ***$
$175.6\pm27.6$	$214.4 \pm 60.5 **$
$118.9\pm46.3$	$226.4 \pm 133.5^{***}$
$34.4\pm7.6$	$42.4\pm15.1*$
$117.7\pm21.2$	$126.7\pm20.4$
$23.8 \pm 9.3$	$45.3 \pm 26.7 ***$
$5.3\pm0.9$	$5.2\pm0.7$
$3.6\pm0.9$	$3.2\pm0.9$
$1.5\pm0.2$	$1.7 \pm 0.2 *$
	136 (Males) $91.0 \pm 20.5$ $175.6 \pm 27.6$ $118.9 \pm 46.3$ $34.4 \pm 7.6$ $117.7 \pm 21.2$ $23.8 \pm 9.3$ $5.3 \pm 0.9$ $3.6 \pm 0.9$

\* Significantly different from normals at P < 0.05. \*\*Significantly different from normals at P < 0.01. \*\*\*Significantly different from normals at P < 0.001.

### **DISCUSSION**

Chronic degenerative diseases are fast emerging as major cause of mortality in developing countries. Cardiovascular disease was the foremost among the non-communicable diseases to be identified as a priority area for research in the developing countries, by the WHO Ad Hoc committee on Health Research (12). It is difficult to isolate any single risk factor for the development of CVD, but enough evidence has accumulated to suggest the high levels of cholesterol and its sub fractions, except HDL-C, can lead to atherosclerosis. Besides these, there are various exogenous and endogenous factors affecting lipid levels.

Various studies carried out have shown that gender differences are seen in the case of lipid profile, i.e. the male subjects tend to have higher lipid levels in comparison to females (13-15). Similar observations have also been noticed in this study, which might be attributable to the favourable effect of estrogen.

The dietary analysis revealed that the fat intake amongst male subjects was more in comparison to female subjects. This might be due to the consumption of fried snacks available in the industrial canteen, at highly subsidized rates. The lipid profile, when analysed in relation to the amount of fat consumed daily, did not show a significant rise in the lipid levels of those who consumed more than 60 gms of fat daily in comparison to those who consumed between 30-60gms daily. When the lipid levels of vegetarians and non-vegetarians were compared, it showed significant rise in the TG, LDLC and VLDL-C levels of the non-vegetarians. This indicates that the intake of non-vegetarian food, which is rich in fats, led to an increase in the lipid levels.

Cigarette smoke is an important risk factor for premature or accelerated peripheral, coronary and cerebral atherosclerotic vascular disease. Cigarette smoke contains large amounts of free radicals that could directly initiate and propagate the process of lipid peroxidation (16). Elevated lipid profile in smokers has been observed in various studies (17-19). The results of the present study showed significant differences in the TG levels, with a non significant rise in the TC levels and no appreciable changes in the LDL-C and HDL-C. This maybe due to the fact that the subjects consumed only 4-5 cigarettes per day, which is much less in comparison to most active smokers.

The subjects chewing tobacco showed a significant increase in TG levels, with no appreciable changes in other lipid parameters. This might be due to the fact that the subjects were consuming only two to three packets of *gutkha* per day.

Adverse effect of alcohol consumption was observed in the TC, VLDL-C and TG levels amongst the subjects consuming alcohol. However, the HDLC did not show any statistical significant change in the present study. The results regarding TC and VLDL-C correlate well with various studies which have shown that consistent intake of alcohol, even at low to moderate levels, increases serum levels of lipids and lipoproteins other than HDL-C in men (20-22).

No changes in the lipid profile were observed in the subjects who exercised, because of irregularity in the exercise regime and as the majority of the subjects had started doing exercise recently.

Aberrations in the lipid profile were observed in case of obesity, diabetes and hypertension. Obesity, which is said to be the mother of most important degenerative diseases, predisposes an individual to the risk of development of diabetes, hypertension and chronic heart disease. The lipid profile showed significant differences amongst the overweight and obese individuals, when compared with normal subjects. The difference was also observed in the TC/LDL-C (TC/L) ratio. These results thus validate the fact that the overweight and obese subjects have an altered lipid profile and that they are at a higher risk of developing other degenerative diseases.

Diabetes mellitus is associated with increased mortality and a high risk of developing cardiovascular and other complications leading to premature disability and death (23). Impairment in insulin secretion leads to an excessive and prolonged rise in plasma glucose concentration. Similar results were seen when the FBG of the normal subjects were compared with that of diabetics. The frequency of elevated plasma lipid levels in diabetic patients is between 20-90% depending on the degree of diabetic control and the type of diabetes (24, 25). In this study, elevated TG and TC and its sub fractions was observed.

Hypertension has been accepted as the most important risk factor for the development of cardiovascular morbidity. Hyperlipidemia amongst the hypertensive subjects was observed in the study population and significant differences were noted when compared to normal subjects. Also, the ratio of LDL-C/HDL-C (L/H) showed significant difference amongst the two groups, indicating that they were at an excess risk of developing coronary heart disease (26). The lipid profile of those who had already developed CHD was analysed and compared with those of normal subjects. Predictably, the results showed significant differences in the lipid profile, in spite of the number of subjects in the CHD group being small. Also amongst the subjects suffering from CHD (n=5), two were diabetics and two were hypertensives, which emphasizes the fact that one is at a higher risk of developing CHD, when one has any of the latter diseases. These diseases have been found to be the powerful contributors in the development of CHD in various studies (27, 28).

Thus, from the present study, it is clear that lipid levels are influenced by a variety of factors such as gender, dietary habits, general habits, various diseases etc. These risk factors are known to be interactive and any combination of risk factors has a greater impact on CHD risk, than the sum of their independent effects. Therefore, community based intervention approaches should lay emphasis on modifiable risk factors in preventing the aberrations in lipid profile, which in the long run would help in bringing down the prevalence of CDD in the population at large.

#### ACKNOWLEDGEMENTS

The data reported in this communication forms part of the PhD thesis of Ms. Swati Desai, to be submitted to M S University of Baroda. Part of the data communicated in this report has been presented at International Conference on Heart and Health, New Delhi, Oct 1999, World Congress on Clinical Nutrition, New Delhi, Oct 1999 and at National Conference of Indian Dietetic Association, Baroda Oct 2000.

### REFERENCES

- 1. Reddy KS. Why is preventive cardiology essential in the Indian context ? *In*: Preventive Cardiology: Introduction. Wasir HS. *Ed.* New Delhi, Vikas Publishing House, 1991; 1-14.
- Godon T, Fisher M, Ernst W, Rificind BM. The relation of diet to LDL, VLDL, total cholesterol and triglycerides in white adults. The lipid research clinic programme prevalence study. Arteriosclerosis 1982; 2: 502-12.
- 3. Kannel WB, Castelli WP, Gordon T, McNamara PM. Serum cholesterol, lipoproteins and the risk of coronary heart disease-The Framingham Study. Ann Intern Med 1971; 74:1-12.
- 4. Kahn HA, Medalie JH, Neufeld HN, Riss E, Balogh M, Groen JJ. Serum cholesterol: its distribution and association with dietary and other variables in a

survey of 10,000 men. Israel J Med Sci. 1969; 5:1117-27.

- 5. Gordan T, Castelli WP, Hjortland MC, Kannel WB, Dawber TR. High density lipoprotein as a protective factor against coronary heart disease -The Framingham Study. Am. J. Med. 1977; 62:707.
- 6. Miller GJ, Miller NE. Plasma high density lipoprotein and development of ischemic heart disease. Lancet 1975; 1:16-9.
- 7. The prevention and management of overweight and obesity in populations: A public health approach. *In* Obesity Prevention and Managing the Global Epidemic Report of a WHO Consultation on Obesity, Geneva: 1997; 3-5 June.
- 8. Eriksson KF, Lindgerde F. No excess 12 year mortality in men with IGT who participated in the Malmo preventive trial with diet and exercise. Diabetologia 198;41 (9) : 1010-6.
- 9. Torgesen PA, Birkeland KI, Anderson SA et al. Lifestyle changes may revert the development of insulin resistance syndrome. The Oslo diet and exercise study a randomized trial. Diabetes Care 1997; 1:20: 26-31.
- Gopalan C, Rama Sastri BV, Balasubramanian SC. Revised and updated by Narasinga Rao BS, Deosthale YG and Pant KC. Nutritive Value of Indian Foods. National Institute of Nutrition, Indian Council of Nutrition, Hyderabad, India.1989.
- 11. Friedewald WT, Levy RI, Fredrickson DS. Estimation of the concentration of low-density lipoprotein cholesterol in plasma, without use of the preparative ultracentrifuge. Clinical Chemistry. 1972; 18: 499-502.
- 12. Ad Hoc Committee on health research relating to future intervention options. Investing in health research and development. Geneva: World Health Organization 1996.
- Gandhi BM. Lipoprotein composition of normal healthy subjects in northern India. Ind. J. Med. Res. 1982; 75: 393-401.
- Haigh NZ, Katherine MS, Chase GA, Jenkins JA, Bachorik PS, Kwiterovich PO. The East Baltimore study: the relationship of lipids and lipoproteins to selected cardiovascular risk factors in an inner city black adult population. Am. J. Clin. Nutr. 1983; 38:320-6.
- 15. Mendlein JM, Freedman DS, Douglas PG, Allen B, Percy CA, Ballew C, Mokdad AH, White LL. Risk factors for coronary heart disease among Navajo

Indians. Findings form the Navajo Health and Nutrition Survey. J. Nutr. 1997;127:2099S-2105S.

- Marangon K, Herbeth B, Lecomte E, Dauphin AP, Grolier P, Chancerelle Y, Artur Y, Siest G. Diet, antioxidant status and smoking habits in French Men. Am. J. Clin. Nutr. 1998: 67:231-9.
- Whig J, Singh CB, Soni GL, Bansal AK. Serum lipids and lipoprotein profiles of cigarette smokers and passive smokers. Indian J. Med. Res.(B) 1992; 96:282-7.
- Castelli WP. Diet, smoking and alcohol influence on coronary heart disease risk. Am. J. Kidney Disease 1990; 16(54): 41-6.
- 19. Henry C., McGill Jr. The cardiovascular pathology of smoking. Am. Heart J. 1988; 115: 250-7.
- Taskinen MR, Nikkila EA, Valimaki M, Sane T, Kuusi T, Kesaniemi A, likahri R. Alcohol induced changes in serum lipoproteins and in their metabolism. Am. Heart J. 1987; 113: 458.
- Moorjani S, Lupien PJ. Alcohol consumption and plasma lipoproteins. Can. Med. Association J. 1990; 142:1089.

- 22. Barboriak JJ, Grunchow HW, Anderson AJ. Alcohol consumption and the diet heart controversy. Alcoholism 1983; 7 : 31.
- 23. Mani UV, Desai S, Iyer U. Studies on the long-term effect of spirulina supplementation on serum lipid profile and glycated proteins in NIDDM patients. Jour. Nutraceuticals, Functional & Medical Foods 2000; 2(3):25 -32.
- 24. Dunn FL. Hyperlipidemia and diabetes. Medical Clinics of North America 1982; 77: 1347-61.
- 25. Mani I Mani UV. Serum triglyceride, cholesterol, Iipoprotein cholesterol and urinary hydroxyproline levels in insulin dependent and non-insulin dependent diabetes. J. Clin. Biochem. Nutr. 1988; 4:241-8.
- 26. Kannel WB. Hypertension and other risk factors in CHD. Am. Heart. Jr. Oct 1987; Vol. 114 (4):918-25.
- 27. WHO Technical Report Series, No. 678, Prevention of coronary heart disease : a report of a WHO Expert Committee 1982.
- Kannel WB. Elevated systolic blood pressure as a cardiovascular risk factor. Am. J. Cardiol. 2000; 85:251-5.