

RESULTS
&
DISCUSSION

● RESULTS & DISCUSSION

Chronic degenerative diseases such as obesity, diabetes mellitus, hypertension and coronary heart disease are increasing to epidemic proportions in the developing countries. The increasing prevalence of these diseases portray that the morbidity of these is on the rise, thus laying emphasis to study the risk factors associated with the same. The present study was thus planned with an objective to map the prevalence and to study the risk factors for the development of chronic degenerative diseases in an industrial set up in Vadodara.

The detailed findings of the present study have been presented under the following four sections:

SECTION I: Assessment of nutritional status

SECTION II: Prevalence and risk factor analysis of chronic degenerative diseases in an industrial set-up.

SECTION III: Metabolic profile of chronic degenerative diseases in an industrial set-up

SECTION IV: Apolipoproteins and total antioxidant activity of the subjects in an industrial set-up.

SECTION I

ASSESSMENT OF NUTRITIONAL STATUS

Dietary habits to a large extent define people's health, growth and development. The increasing westernization, urbanization and mechanization occurring in most countries around the world is associated with changes in diet towards one of high-fat, high-energy foods and a sedentary lifestyle. Thus, it becomes prudent to study the dietary habits of the population.

In the present study, 1025 subjects were enrolled from the Indian Oil Corporation, Vadodara. The background information (marital status, type of family, education, per capita income and designation) and clinical information (age, height, weight, body mass index, and waist hip ratio) was collected through a structured questionnaire. The nutrient composition of various foods supplied by the canteen and the dietary intake of the subjects were calculated.

The results of this section have been presented below:

BACKGROUND INFORMATION OF THE SUBJECTS

The background information of the subjects is depicted in **table 13**. Out of the total 1025 subjects enrolled, 63.1 % were males and 36.9 % were females.

TABLE 13

BACKGROUND INFORMATION OF THE SUBJECTS

	MALES	FEMALES	TOTAL
n	647 (63.1)	378 (36.9)	1025 (100)
MARITAL STATUS			
Married	610 (94.3)	366 (96.8)	979 (95.6)
Unmarried	15 (2.4)	1 (0.3)	16 (1.6)
Divorcee	1 (0.2)	0 (0.0)	1 (0.3)
Widow/Widower	2 (0.3)	1 (0.3)	3 (0.3)
EDUCATION PROFILE			
Elementary	56 (9.0)	66 (18.7)	122 (12.5)
High School	211 (33.9)	150 (42.5)	361 (37.0)
Dip /Graduate	190 (30.5)	93 (26.3)	283 (29.0)
Post Grad.	111 (17.8)	26 (7.4)	137 (14.1)
PhD	54 (8.7)	18 (5.1)	72 (7.4)
TYPE OF FAMILY			
Nuclear	-	-	333 (34.3)
Joint/Extended	-	-	639 (65.7)
PER CAPITA INCOME			
<=1000	-	-	219 (21.4)
1001-1500	-	-	178 (17.4)
1501-2000	-	-	220 (21.5)
2001-2500	-	-	123 (12.0)
2501-3000	-	-	80 (7.8)
3001-3500	-	-	49 (4.8)
3501-4000	-	-	71 (6.9)
4000+	-	-	84 (8.2)
DESIGNATION OF EMPLOYEES			
Staff	521 (80.5)	18 (4.8)	539 (52.6)
Officers	108 (16.7)	0 (0.0)	108 (10.5)
Spouses	18 (2.8)	359 (95.2)	377 (36.8)

Values in parenthesis indicate percentage

Majority of the female subjects were spouses of the employees and very few were employees, whereas, among the males majority were the employees. Ninety Six percent of the subjects were married whereas only 1.6 % were unmarried followed by 0.3 % divorcees and widow/widowers. The educational status of the subjects revealed that 42.5 % of the female and 33.9 % of male subjects had studied only till high school level. It was observed that 65.7 % of the subjects lived in joint or extended families. Data regarding the per capita income (PCI) showed that nearly half of the population studied had PCI up to 2000. There were 8.2 % who had PCI of 4000 and above.

CLINICAL INFORMATION OF THE SUBJECTS

The clinical information revealed that the mean age of the subjects was 42 years (Table 14). The average height of the subjects was 160.7 cms. The average weight of the male and females was 66.8 Kg and 58.6 Kg respectively. The average BMI of females was 25.1, and it was 24.5 for males, whereas, the average WHR was 0.9 for both the sexes.

NUTRIENT COMPOSITION OF FOODS PROVIDED BY THE CANTEEN

Out of the two canteens catering to the needs of the employees, one served lunch/dinner and the other provided snacks. These foods were provided at highly subsidized rates for the welfare of the employees.

TABLE 14
CLINICAL INFORMATION OF THE SUBJECTS
(Mean \pm SD)

	Males	Females	Total
Age (y)	43.4 \pm 8.2	39.6 \pm 7.8	42.0 \pm 8.2
Height (cm)	165.3 \pm 6.8	152.6 \pm 5.7	160.7 \pm 8.8
Weight (kg)	66.8 \pm 10.7	58.6 \pm 10.7	63.8 \pm 11.4
Waist (cm)	90.6 \pm 9.0	85.6 \pm 9.8	88.8 \pm 9.6
Hip (cm)	98.1 \pm 7.0	99.3 \pm 10.7	98.5 \pm 8.6
BMI *	24.5 \pm 3.6	25.1 \pm 4.2	24.7 \pm 3.9
WHR **	0.9 \pm 0.1	0.9 \pm 0.1	0.9 \pm 0.1

* BMI = $\frac{\text{Weight in Kg}}{\text{Height in (m}^2\text{)}}$

** WHR = $\frac{\text{Waist Measurement (cm)}}{\text{Hip Measurement (cm)}}$

The cost of the vegetarian lunch/dinner was 60 paise and the non-vegetarian lunch/dinner was served at one rupee. The nutritive value of the foods provided by the canteen, which served lunch and dinner is given in **table 15**. Puris, various pulses and pulav provided major portion of the calories. These items were either fried or prepared using appreciable quantity of fat.

The nutrient content of various snacks provided by the canteen is given in **table 16**. These snacks were mainly ready to eat ones in a packet size of 50g. These packets were given at a rate of 20-25 paise each. Each employee was provided with coupons to buy products up to Rs. 80/per month. Thus, the employee can purchase 400 packets in a month. The average caloric content of the snacks was 291 kcal and the protein, fat and carbohydrate content was found to be 8.3g, 23.2g and 18.4g respectively.

DIETARY INTAKE OF THE SUBJECTS

The dietary intake of the subjects based on the 24-hour dietary recall method is given in **table 17**. The subjects were able to meet approximately 90% of their RDA for energy. The fat intake of males was also found to be significantly higher in comparison to the female subjects (89.4 ± 52.6 g Vs 64.0 ± 13.9 g). However, for both the groups, 33-38% of calories came from fat, which is on the higher scale. The consumption of ascorbic acid (4mg to 198 mg) and β -carotene (249 μ g to 6916 μ g) were found to be of a wider range.

TABLE 15

**NUTRIENT COMPOSITION OF VARIOUS FOODS SUPPLIED
BY THE CANTEEN**

FOOD ITEM	SERVING	ENERGY (K cal)	PROTEIN (g)	FATS (g)	CHO (g)	FIBRE (g)
Pulav	1	208	2.7	10.2	26.6	0.4
Rice (Plain)	1	138	2.7	0.2	31.3	0.9
Dal	1	152	7.0	3.5	23.5	0.5
Kadhi	1	34	0.6	3.3	0.6	-
Puri	6	553	14.5	18.0	83.3	2.3
Mutton	1	189	16.3	12.1	2.6	0.3
Egg Curry	1	159	8.7	12.1	4.1	0.5
Chicken	1	182	19.7	10.5	2.2	0.1
Mix Veg	1	102	2.2	2.8	17.2	0.8
Cauliflower Sabzi	1	97	2.0	2.8	16.4	0.9
Bottle Gourd Sabzi	1	142	3.9	3.5	21.6	0.6
Kovar Sabzi	1	90	1.4	2.7	15.4	1.0
Cabbage Sabzi	1	101	1.6	2.7	18.1	0.7
Bringal Sabzi	1	125	1.4	6.2	16.5	1.0
Tomato Sabzi	1	109	1.3	2.7	20.4	0.6
Moong	1	261	14.4	3.4	44.0	2.5
Vatana Sabzi	1	305	12.0	10.7	40.8	2.8
Chana	1	283	10.4	9.2	39.7	2.4
Banana Raita	1	71	3.2	4.0	5.7	0.0
Bundi Raita	1	119	5.2	7.1	9.0	2.3

TABLE 16
NUTRIENT COMPOSITION OF ONE PACKET OF SNACKS
PROVIDED BY THE CANTEEN

FOOD ITEM	ENERGY (Kcal)	PROTEIN (g)	FATS (g)	CHO (g)	FIBRE (g)
Mori Sev	284	8.3	23.9	17.2	0.5
Ratlami Sev	352	8.3	23.9	24.7	0.5
Groundnut (Roasted)	285	13.1	13.4	19.9	1.6
Bhusu	333	6.9	28.4	21.3	0.5
Chewda	350	5.3	27.1	24.5	0.7
Dal Mooth	288	6.7	17.9	21.3	0.3
Phulwadi	369	8.3	33.8	22.2	0.5
Sweet Sakkarpara	291	2.8	33.4	16.2	0.1
Namkin Sakkarpara	231	2.8	18.5	16.2	0.1
Bundi Ladoo	261	5.2	29.8	13.4	0.3
Mix Bhajiya	214	3.5	14.1	15.8	0.4
Potato Vada	156	2.5	12.7	10.5	0.3
Puri Bhaji	344	4.3	30.9	22.5	0.9
Methi Bhajiya	293	7	17.4	21.8	0.7
Samosa	358	3.3	22.6	28.2	0.6
Uttapa	248	4	23.4	15.2	0.6
AVERAGE	291	8.3	23.2	18.4	0.5

1 Packet Size = 50 g

TABLE 17

DIETARY INTAKES OF MALES AND FEMALES (Mean \pm SD)

	MALES	FEMALES	TOTAL
Calories (Kcal)	2107 \pm 563	1727 \pm 419 ***	1942 \pm 538
Carbohydrates(g)	273.8 \pm 82.2	239.7 \pm 65.5 **	259.0 \pm 76.9
Proteins (g)	62.4 \pm 16.6	51.0 \pm 13.9 ***	57.4 \pm 16.4
Fats (g)	89.4 \pm 52.6	64.0 \pm 13.9 ***	80.6 \pm 45.0
Fibre (g)	7.6 \pm 2.3	6.57 \pm 2.5 *	7.15 \pm 2.4
Vitamin C (mg) (Range)	6.6 - 198.0	4.0 - 198.0	4.0 - 198.0
β -Carotene (μ g) (Range)	258.0 - 6916.0	249.0 - 6618.0	249.0 - 6916.0
Sodium (mg)	167.0 \pm 145.3	122.4 \pm 50.3	141.0 \pm 102.8
PERCENTAGE OF CALORIES COMING FROM:			
CHO	52	56	53
PROTEINS	12	12	12
FAT	38	33	37

Males * p<0.05, ** p<0.01, *** p<0.001 Vs Females

Among the non-vegetarians (about 29% of the population) few consumed non-vegetarian diet on daily basis where as the rest of them consumed occasionally or only once a week (**Figure 12**)

DISCUSSION

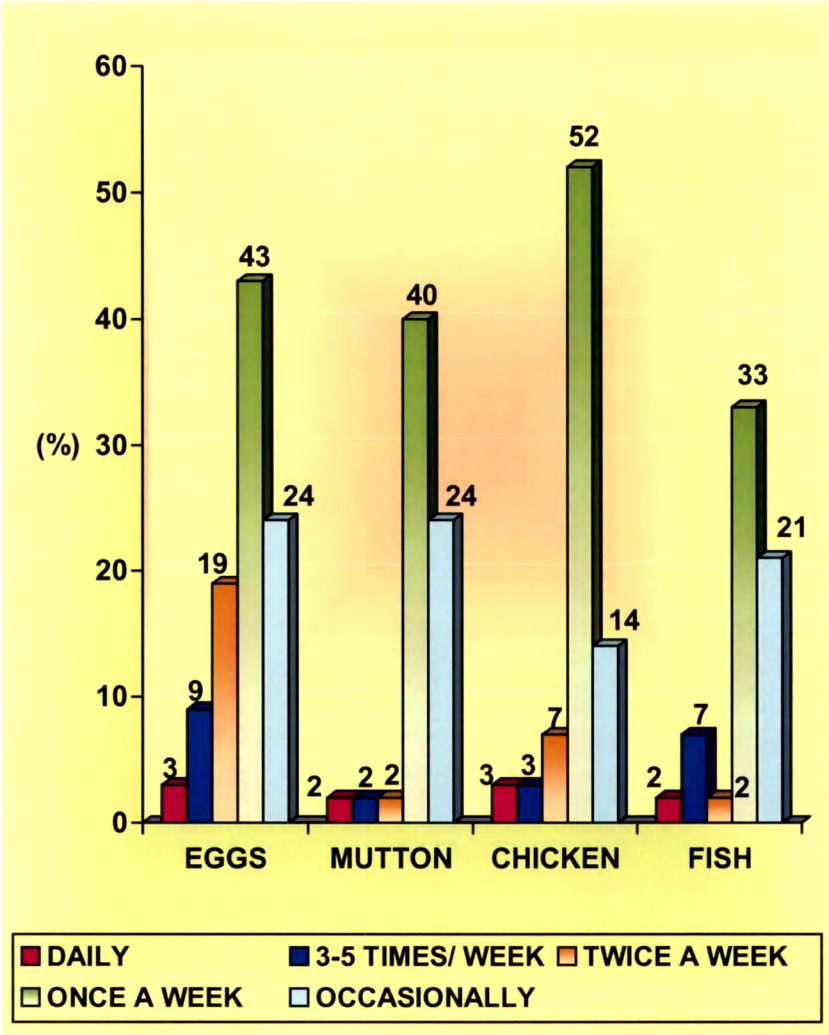
The growing epidemic of chronic degenerative diseases afflicting both developed and developing countries constitutes a concern for the health care planners predominantly because of the devastating effects of its chronic complications and the enormous burden on healthcare system. In the past decade rapid changes in the diets and lifestyles of the people have occurred because of industrialization, urbanization, economic development and market globalization resulting in a significant impact on the health and nutritional status of populations, particularly in developing countries in transition.

Further more, in the past decade, rapid expansion of the relevant scientific fields and of the available population-based epidemiologic evidence have helped to clarify the role of dietary component that increase the probability of occurrence of these diseases in individuals.

- With a view to understand the dietary pattern of the employees in the industrial set up the present section was planned to assess the nutritional status of the subjects

FIGURE 12

**FREQUENCY OF CONSUMPTION OF
NON VEGETARIAN FOODS (%)**



The background information of the subjects revealed that 37 % of the subjects had studied till high school and 29 % were diploma or graduate degree holders. The information regarding socio-economic status showed that only 21.4 % of the subjects had per capita income below Rs 1000, whereas the rest of the population had a per capita income of above 1000.

Chronic degenerative diseases are said to be the most ubiquitous, disabling and devastating disease of older people. Age is a risk factor for the development of these CDD (Manton et al 1997). In the present study it was observed that the mean age of the subjects was 42 years, and it ranged from 20 to 60 years. It was also observed that the prevalence of chronic degenerative diseases was more amongst those people who were above the age of 40 years, thus implying that advancing age is a risk factor for the development of chronic degenerative diseases. The body mass index, a tool used for the assessment of overweight or obesity, showed that the males had an average BMI of 24.5 and the females had a BMI of 25.1 indicating that many of the subjects may be overweight.

Diet and nutrition are important factors in the promotion and maintenance of good health throughout the entire course of life. Nutrition is coming to the fore as a major modifiable determinant of chronic diseases, with scientific evidences increasingly supporting the view that alterations in the diet have strong effects, both positive and negative, on health throughout life (WHO Report, 2003). At the global level great changes have swept the entire world

since the second half of the twentieth century, including major modification in diet, first in industrial regions and more recently in developing countries. Traditional diets, largely plant based, have been swiftly replaced by high fat, energy-dense diets with a substantial content of animal foods (WHO Report 2003)

In the present study the type of food consumed by the employees was dependent to a certain extent on the food served in the canteen. Apart from the food taken at home, employees were consuming snacks / lunch / dinner provided by the canteen, and these items are available at highly subsidized rate. As mentioned earlier the mean caloric content of lunch / dinner was approximately 1200 Kcal and that of snacks was 291 Kcal. The fat content was 23.2 g per packet of snack. These snacks were thus rich sources of calories and fats. It has been documented that diets rich in fat may lead to excess energy intake and obesity because of its palatability, high energy density and metabolic efficiency (Li-Ching Lynet et al 1994). Furthermore, when energy is in excess, the human body processes nutrients according to an oxidative hierarchy. Excessive carbohydrates and protein intakes are disposed of by increased oxidation. In contrast, excess fat intake does not promote its own oxidation in the short-and mid term and in the long-term, leads to an increase in fat stores (Ravussin and Tataranni 1997).

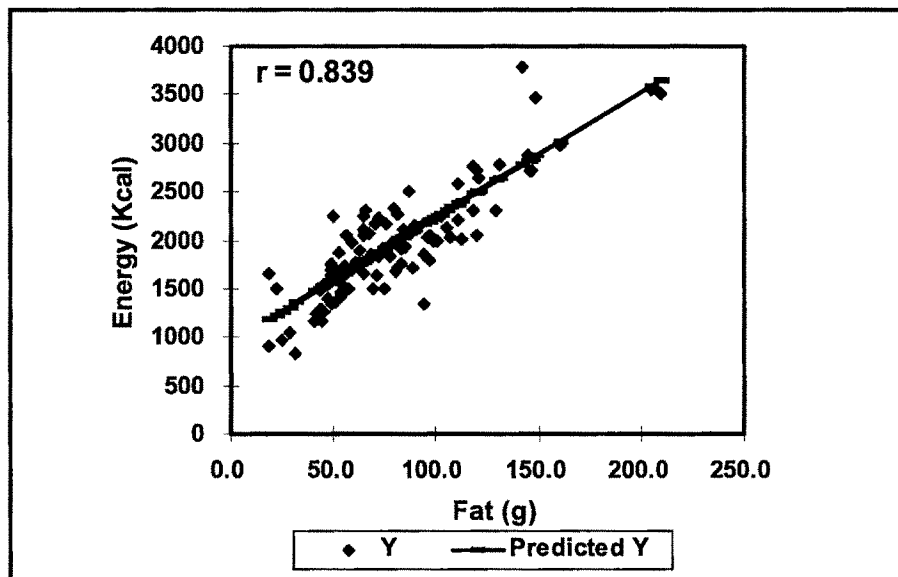
Regarding the nutrient intake of subjects, the fat energy ratio (FER) was greater than 30 % in both males and females. The energy intake of the

subjects highly correlated ($r= 0.83$) with fat as well as with carbohydrate ($r=0.83$) (Figure 13). This shows that the energy dense foods rich in fats may pave the way for the development of obesity in the population. Apart from the amount of fat, the type of fat is also an important causative factor of obesity related morbidities. In the present study it was seen that majority of the subjects consumed more of unsaturated fat in comparison to saturated fats. Based on the number of oil tins or ghee purchased for the whole year, it was observed that on an average one family used 4 tins (60 litres) of oil and 12 kg of ghee per year. This indicates that the intake of oil per individual was 41g/day and that of ghee was 8 g/day. This portrays that the average intake of visible fat at home was 49g per person. In addition to this, the consumption of fat rich food from the canteen would increase the fat intake of the individual. The high amounts of total fat consumed is of great concern and needs to be rectified in the subjects. Thus the low cost snacks provided in the canteen were energy dense and contributed to their fat intake along with their home diets.

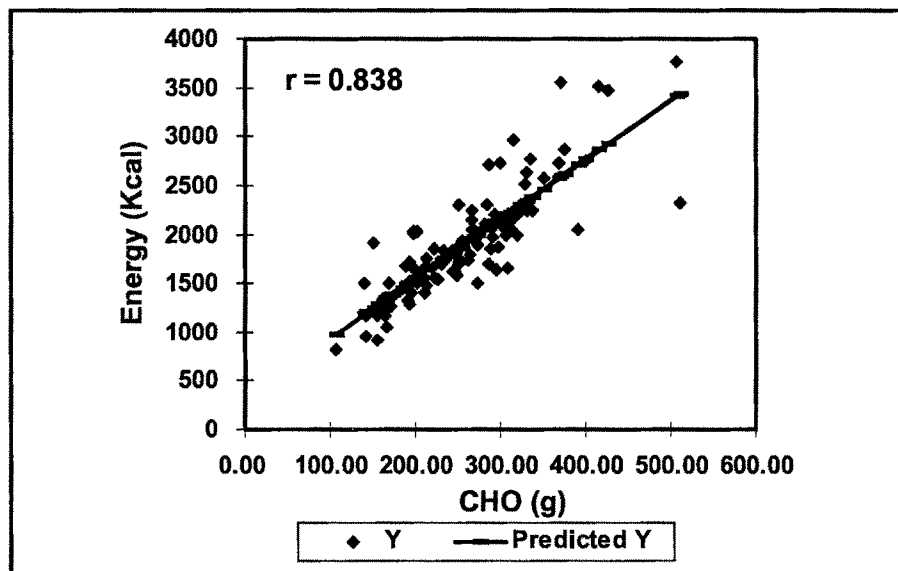
The quantity and quality of fat consumed by a person is known to influence the lipid profile. However, when the analysis of the lipid levels in relation to the quantity of fat consumed was analysed, it failed to show any significant rise in the atherogenic lipid parameters. There are studies, which show a positive correlation between the fat intake and the atherogenic rise in lipid parameters, however studies of individuals within the same population have generally either failed to find correlation between dietary variables and blood cholesterol

FIGURE 13
CORRELATION OF ENERGY WITH FAT
AND CARBOHYDRATE

CORRELATION BETWEEN ENERGY AND FAT



CORRELATION BETWEEN ENERGY AND CARBOHYDRATE



concentrations or found only a weak relation, and this has often been cited as evidence against the dietary fat-CAD hypothesis. A major disadvantage of 24 hour dietary recall method is that it is often difficult to obtain a wide range of nutrient intakes for individuals within an affluent population (Caggiula and Mustard 1997).

As reported by the canteen management, almost 800 employees eat subsidized lunch / dinner. In addition, snacks are also consumed from the canteen. The monthly sale of snacks by the canteen has been shown in Table 18. The data reveals that the average monthly sale of packets, irrespective of the food items, was found to be very high. This shows that the actual consumption of the snacks from the canteen is high. This would have been one of the causative factors for the high prevalence of obesity, which is presented in the section that follows.

The results of this section highlight that the industrial canteen provides energy dense foods at subsidized rates, tempting the employees to go for it. There is an urgent need to modify the food preparation and or to supply low calorie nutritious snacks for the welfare of employees.

TABLE 18
MONTHLY SALE OF SNACK PACKETS

Snacks	Number of Packets Sold
Bhusu	1,62,300
Dalmooth	89,600
Phulwadi	89,500
Roasted Groundnut	86,000
Mori Sev	7,75,500
Sakkarpara	86,100
Chewda	1,51,600
Bread Butter	4000/day
Bundi	30,000

- Total sale of packets irrespective of the food items = 7,93,200/month

At a glance

❧ Out of the total 1025 subjects enrolled from the industrial set up 63 % were males and 37 % were females

❧ The mean age of the subjects was 42 years. The mean Body Mass Index (BMI) was found to be 24.5 for males and it was 25.1 for females.

❧ The information regarding socio-economic status showed that only 21.4 % of the subjects had per capita income below Rs. 1000, whereas the remaining population had a per capita income above 1000.

❧ There were two industrial canteens catering to the needs of the employee's. One served lunch/dinner and the other provided snacks. These foods were provided at highly subsidized rates (20-25 paise/packet) for the welfare of the employees.

❧ The snacks served by the canteen provided 291 kcal and 23.2 g fat.

❧ The sale of snack packets was found to be 7,93,200 packets per month.

❧ The fat contributed 33-38 % of the total calories, which is on the higher side. A strong need is felt to modify and/or prepare low calorie nutritious food.

SECTION II

PREVALENCE AND RISK FACTOR ANALYSIS OF CHRONIC DEGENERATIVE DISEASES IN AN INDUSTRIAL SET-UP

Rapid changes in diets and lifestyles that have occurred with industrialization, urbanization, economic development and market globalization, have accelerated over the past decade. This is having a significant impact on the health and nutritional status of populations, particularly in developing countries and in countries in transition. While standards of living have improved, food availability has expanded and become more diversified, and access to services has increased, there have also been significant negative consequences in terms of inappropriate dietary patterns, decreased physical activities and increased tobacco use, and a corresponding increase in diet related chronic diseases. CDD such as obesity, diabetes mellitus, cardiovascular disease, hypertension and stroke – are increasingly significant causes of disability and premature deaths in both developing and newly developed countries, placing additional burdens on already overtaxed national budgets (WHO 2003).

The present section was planned with an objective to map the prevalence of chronic degenerative diseases and to carry out the risk factor analysis in the development of these diseases. The results pertaining to this are presented under the following headings

PREVALENCE OF CHRONIC DEGENERATIVE DISEASES IN AN INDUSTRIAL SETUP

The prevalence of chronic degenerative diseases in the industrial set up studied is depicted in Table 19 (a+b) The prevalence of overweight and obesity is given in **table 19 (a)** The overall prevalence of overweight and obesity ($BMI \geq 25$) in the industrial population studied was 41% The prevalence of overweight was found to be 33% and obesity to be 8% respectively. Further, it was seen that the prevalence of obesity was higher in female subjects than male subjects (12% Vs 5%).

Since overweight and obesity coexists with CDD, the prevalence of diabetes, hypertension and CHD in these subjects was looked into (**Table 19 b**) Of the total overweight & obese subjects, 82% of the subjects did not have any CDD i.e. diabetes, hypertension and CHD. But 9% of overweight & obese were diabetics, 8% of overweight & obese were hypertensives and 1% of overweight & obese were suffering from CHD Thus, about 18% of the overweight & obese subjects had diabetes, hypertension or CHD

Out of the total 1025 subjects enrolled, 325 subjects did not have any CDD. As seen in **table 20**, the prevalence of diabetes, hypertension and CHD was 8%, 6% and 1% respectively in the population studied The prevalence was higher in males than the female subjects viz., diabetes (10% Vs 5%), hypertension (8% Vs 2%) and CHD (1% Vs 0%). Primary hyperlipidemia was

TABLE 19(a)
PREVALENCE OF OVERWEIGHT AND OBESITY IN THE
INDUSTRIAL POPULATION

	MALES	FEMALES	TOTAL
n	648	377	1025
NORMAL WEIGHT	391 (60)	215 (57)	606 (59)
OVERWEIGHT	226 (35)	116 (31)	342 (33)
OBESE	31 (5)	46 (12)	77 (8)
OVERWEIGHT +OBESE	257 (40)	162 (43)	419 (41)

Values in parenthesis indicate percentage

TABLE 19(b)
PREVALENCE OF CHRONIC DEGENERATIVE DISEASES IN
OVERWEIGHT AND OBESE SUBJECTS

	MALES	FEMALES	TOTAL
No Complication			
OVERWEIGHT	173 (68)	106 (65)	279 (67)
OBESE	23 (9)	42 (26)	65 (16)
OVERWEIGHT +OBESE	196 (77)	148 (91)	344 (82)
With Complication			
DIABETES	27 (11)	9 (6)	36 (9)
HYPERTENSION	28 (11)	5 (3)	33 (8)
CHD	6 (2)	-	6 (1)
TOTAL	61 (24)	14 (9)	75 (18)

Values in parenthesis indicate percentage

TABLE 20

**PREVALENCE OF CHRONIC DEGENERATIVE DISEASES
AMONGST THE SUBJECTS ENROLLED FROM THE
INDUSTRIAL SETUP**

	PREVALENCE		
	MALE	FEMALE	TOTAL
n	648 (63)	377 (37)	1025 (100)
NORMAL	189 (29)	136 (36)	325 (32)
OVERWEIGHT AND OBESE (BMI \geq 25)	196 (30)	148 (39)	344 (33)
DIABETES	64 (10)	18 (5)	82 (8)
HYPERTENSION	52 (8)	9 (2)	61 (6)
CHD	6 (1)	-	6 (1)
HYPERLIPIDEMIC	61 (9)	11 (3)	72 (7)
HYPERCHOLESTEROLEMIC	73 (11)	51 (14)	124 (12)
HYPERTRIGLYCERIDEMIC	7 (1)	4 (1)	11 (1)

Values in parenthesis indicate percentage

present in 20% of the subjects viz 12% were hypercholesterolemic (HC), 1% was hypertriglyceridemic (HT) and 7% were hyperlipidemic (HL) Among the subjects suffering from diabetes, hypertension and CHD there were many who were overweight or obese This data is depicted in **figure 14**

After having studied the prevalence of CDD the analysis for lipid profile based on various risk factors is given below

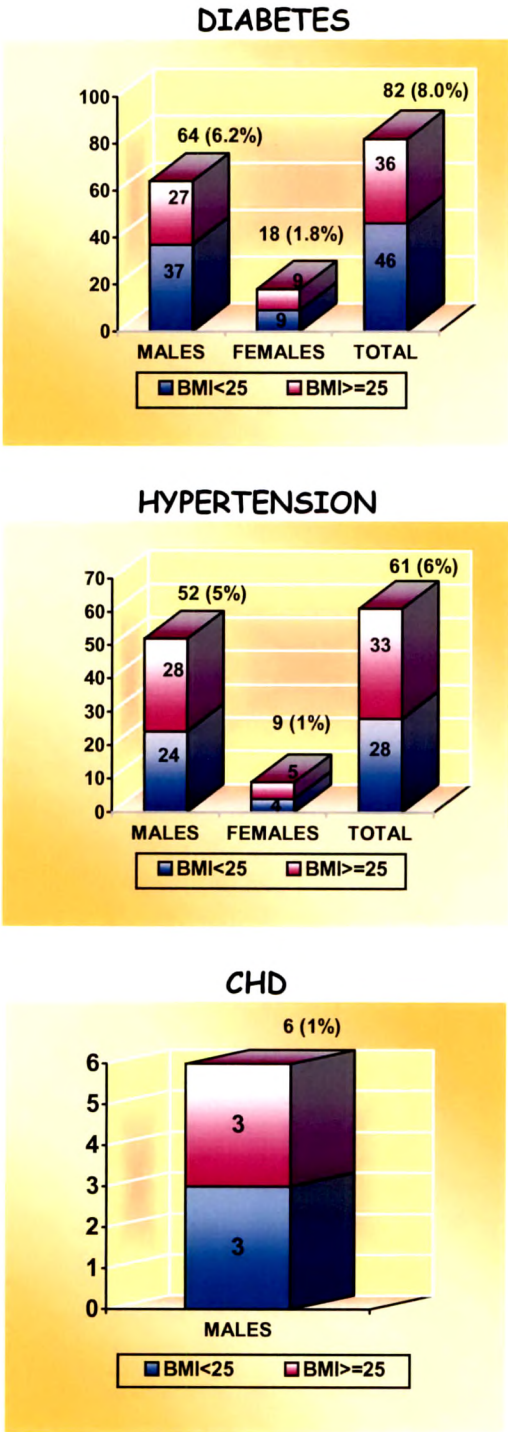
LIPID PROFILE OF MALE AND FEMALE SUBJECTS

The lipid profile of male and female subjects is presented in **table 21** The male subjects had significantly higher TC, TG, VLDL-C and Non HDL-C levels than the female subjects. The HDL-C levels were similar in both the groups and therefore, the TC/H ratio was higher in male subjects than female subjects (5.4 Vs 5.2, $p < 0.01$)

INFLUENCE OF AGE ON THE LIPID PROFILE OF THE SUBJECTS

It is well documented that as the age advances, the chances of one suffering from various CDD increases and aberrations in the atherogenic lipid parameters are observed (Kannel and Schatzkin 1983, Kritchevsky 1978) In this study a comparison was made in lipid profile in subjects greater than 40 years and those less than 40 years (**Table 22**) TC and LDL-C levels were elevated in both male and female subjects above 40 years than their

FIGURE 14
PREVALENCE OF DIABETES, HYPERTENSION AND CHD IN
RELATION TO BODY MASS INDEX



* Values in parenthesis indicate percentage

TABLE 21
LIPID PROFILE OF MALES & FEMALES
(mg/dl, Mean \pm SD)

	Males	Females
n	457	269
TC	200.6 \pm 38.7	190.0 \pm 34.1 ***
TG	147.8 \pm 76.4	118.6 \pm 64.9 ***
HDL-C	37.9 \pm 8.3	37.2 \pm 8.1
LDL-C	133.2 \pm 30.7	129.3 \pm 28.7
VLDL-C	29.6 \pm 13.3 **	23.7 \pm 13.0 ***
Non HDL-C	162.8 \pm 33.9	153.0 \pm 30.6 ***
TC/ H	5.4 \pm 1.0	5.2 \pm 0.9 **
L/H	3.6 \pm 0.9	3.6 \pm 0.9

Males ** P<0.01, *** P<0.001 Vs Females

TABLE 22

LIPID PROFILE OF THE SUBJECTS IN RELATION TO AGE (mg/dl, Mean \pm SD)

	AGE \leq 40 Years			AGE > 40 Years		
	Males	Females	Total	Males	Females	Total
n	134	147	281	309	113	421
TC	192.9 \pm 16.0	180.8 \pm 31.9	186.6 \pm 32.6	204.6 \pm 40.4 ^{**}	200.2 \pm 32.7	203.4 \pm 38.5 ^{***}
TG	147.1 \pm 91.0	105.0 \pm 49.6	125.1 \pm 75.2	148.4 \pm 70.2	132.1 \pm 69.5 ^{***}	144.0 \pm 70.3 ^{***}
HDL-C	37.5 \pm 8.0	35.1 \pm 7.2	36.3 \pm 7.6	38.5 \pm 8.1	39.9 \pm 8.6 ^{***}	38.9 \pm 8.2 ^{***}
LDL-C	126.3 \pm 27.7	125.1 \pm 27.7	125.6 \pm 37.7	136.4 \pm 31.5 ^{***}	133.9 \pm 29.4 ^{***}	135.8 \pm 30.9 ^{***}
VLDL-C	29.4 \pm 8.2	21.0 \pm 9.9	25.0 \pm 15.0	29.7 \pm 14.0	26.4 \pm 13.9 ^{***}	28.8 \pm 14.1 ^{***}
NON HDL-C	155.7 \pm 27.8	145.0 \pm 31.4	150.6 \pm 28.8	166.1 \pm 35.8 ^{***}	160.3 \pm 29.3 ^{***}	164.6 \pm 34.3 ^{***}
TC/H	5.3 \pm 0.9	5.3 \pm 1.0	5.3 \pm 0.9	5.4 \pm 0.9	5.1 \pm 0.9	5.3 \pm 0.9
L/H	3.5 \pm 0.9	3.7 \pm 0.9	3.6 \pm 0.9	3.6 \pm 0.9	3.5 \pm 0.9	3.6 \pm 0.9

Males (Age \leq 40 Years) ^{**} p<0.01, ^{***} p<0.001 Vs Males (Age >40 Years)Females (Age \leq 40 Years) ^{***} p<0.001 Vs Females (Age >40 Years)Total (Age \leq 40 Years) ^{***} p<0.001 Vs Total (Age >40 Years)

respective counterparts who were aged less than 40 years. The atherogenic lipoprotein levels in the form of Non HDL-C, was also significantly higher in subjects above 40 years than subjects below 40 years. Thus, placing them at risk for developing complications later in life.

LIPID PROFILE OF THE SUBJECTS IN RELATION TO SMOKING HABIT OF THE SUBJECTS

Smoking alters the lipid levels depending on the duration of smoking and number of cigarettes smoked per day. In this study none of the females either smoked or consumed tobacco or alcohol. The lipid profile of male subjects in relation to smoking is depicted in **table 23**. The TC and TG levels were higher among smokers. The HDL-C levels did not show any difference between the two groups and the Non - HDL-C levels showed a non-significant rise among smokers.

Further analysis among smokers according to their disease profile is given in **table 24**. It was noticed that there were more number of smokers amongst the diseased group, whereas number is less among the normals. The lipid levels of non-smokers among normal and diseased population showed significant differences. Among the smokers (normal Vs diseased) an increase in the TC, TG and Non HDL-C levels were seen.

TABLE 23

**LIPID PROFILE OF THE SUBJECTS IN RELATION TO
SMOKING HABIT (mg/dl, Mean \pm SD)**

	Smokers	Non-Smokers
n	69	392
TC	204.8 \pm 34.3	199.5 \pm 39.4
TG	161.6 \pm 90.8	145.5 \pm 73.6
HDL-C	37.7 \pm 7.9	37.9 \pm 8.4
LDL-C	134.7 \pm 29.2	132.9 \pm 31.0
VLDL-C	32.3 \pm 18.2	29.1 \pm 14.7
Non HDL-C	167.0 \pm 29.4	162.1 \pm 34.0
TC/H	5.5 \pm 1.0	5.4 \pm 1.1
L/H	3.7 \pm 0.8	3.6 \pm 0.9

TABLE 24

**ANALYSIS OF VARIANCE IN RELATION TO SMOKING HABIT AMONG NORMALS
AND SUBJECTS WITH CDD (mg/dl) (Mean \pm SD)**

	NORMALS		SUBJECTS WITH CDD		F Ratio	F Prob.	Significant Pairs
	Non Smokers	Smokers	Non Smokers	Smokers			
Groups	(1)	(2)	(3)	(4)			
n	193	25	199	44			
TC	189.4 \pm 39.4	189.6 \pm 33.4	210.1 \pm 36.7	211.8 \pm 32.7	12.010	0.000	(1,3), (1,4)
TG	132.9 \pm 68.5	154.1 \pm 117.2	157.7 \pm 76.4	165.1 \pm 76.4	4.479	0.004	(1,3)
HDL-C	36.3 \pm 8.7	35.7 \pm 9.6	39.5 \pm 7.9	38.6 \pm 6.9	5.416	0.001	(1,3)
LDL-C	126.7 \pm 30.0	123.1 \pm 23.4	139.1 \pm 30.8	140.2 \pm 30.3	7.162	0.000	(1,3)
VLDL-C	26.6 \pm 13.7	30.8 \pm 23.4	31.5 \pm 15.3	33.0 \pm 15.3	4.479	0.004	(1,3)
NON HDL-C	153.2 \pm 34.1	153.9 \pm 26.8	170.6 \pm 33.1	173.2 \pm 28.8	11.204	0.000	(1,3), (1,4)
TC/H	5.4 \pm 1.1	5.4 \pm 0.7	5.44 \pm 1.0	5.6 \pm 0.8	0.363	0.780	
L/H	3.6 \pm 1.0	3.6 \pm 0.8	3.6 \pm 1.0	3.7 \pm 0.8	0.053	0.984	

LIPID PROFILE OF THE SUBJECTS IN RELATION TO TOBACCO CONSUMPTION

The consumption of tobacco was mainly in the form of raw tobacco or in the form of padiki (gutka). The lipid profile of the subjects consuming tobacco is depicted in **table 25**. The lipid profile did not show any appreciable changes among the two groups i.e. tobacco consumers and non consumers. However, when the data was analysed based on the absence or presence of CDD (**Table 26**), a significant rise in TC with a concomitant significant elevated Non HDL-C levels in diseased subjects who consumed tobacco as compared to subjects who did not have any CDD was observed. Thus, the presence of disease i.e. CDD altered the lipid profile.

LIPID PROFILE IN RELATION TO ALCOHOL CONSUMPTION

The lipid profile of the subjects in relation to alcohol consumption is shown in **table 27**. A significant rise in the TG and VLDL-C was observed among subjects consuming alcohol as compared to non-alcoholics.

The lipid profile of the normal or diseased subjects in relation to alcohol consumption is depicted in **table 28**. The TC, LDL-C and Non HDL-C levels were significantly different between all the four groups. In line with the data observed for tobacco chewing, alcohol consumption also adversely altered

TABLE 25

LIPID PROFILE OF THE SUBJECTS IN RELATION TO
TOBACCO CONSUMPTION (mg/dl, Mean \pm SD)

	Tobacco Consumers	Non-Tobacco Consumers
n	85	372
TC	202.8 \pm 43.5	200.1 \pm 37.6
TG	159.3 \pm 145.2	145.2 \pm 71.6
HDL-C	38.8 \pm 8.4	37.7 \pm 8.3
LDL-C	132.6 \pm 33.3	133.4 \pm 30.1
VLDL-C	31.9 \pm 18.8	29.0 \pm 14.3
Non-HDL-C	164.5 \pm 37.8	162.4 \pm 33.1
TC/H	5.4 \pm 0.9	5.4 \pm 1.1
L/H	3.5 \pm 0.9	3.7 \pm 1.0

TABLE 26

ANALYSIS OF VARIANCE IN RELATION TO TOBACCO CONSUMPTION AMONG NORMALS AND SUBJECTS WITH CDD (mg/dl) (Mean \pm SD)

	NORMALS		SUBJECTS WITH CDD		F Ratio	F Prob.	Significant Pairs
	Non Tobacco Consumers	Tobacco Consumers	Non Tobacco Consumers	Tobacco Consumers			
Groups	(1)	(2)	(3)	(4)			
n	181	33	191	52			
TC	189.7 \pm 38.3	187.7 \pm 42.2	209.9 \pm 34.3	212.38 \pm 42.01	12.078	0.000	(1,3), (1,4), (2,3), (2,4),
TG	132.9 \pm 74.4	146.3 \pm 75.8	156.7 \pm 67.1	167.46 \pm 103.78	4.430	0.004	(1,3), (1,4)
HDL-C	36.2 \pm 8.9	37.0 \pm 7.8	39.2 \pm 7.5	39.94 \pm 8.60	5.444	0.001	(1,3), (1,4)
LDL-C	127.0 \pm 29.4	122.6 \pm 29.5	139.4 \pm 29.6	139.0 \pm 34.3	7.262	0.000	(1,3), (2,3)
VLDL-C	26.6 \pm 14.9	29.3 \pm 15.2	31.4 \pm 13.4	33.5 \pm 20.8	4.430	0.004	(1,3), (1,4)
NON HDL-C	153.6 \pm 32.9	151.9 \pm 36.3	170.7 \pm 31.0	172.4 \pm 36.9	11.187	0.000	(1,3), (1,4), (2,3), (2,4)
TC/H	54 \pm 11	52 \pm 0.9	55 \pm 1.0	54 \pm 1.0	0.461	0.710	
L/H	37 \pm 10	34 \pm 0.8	37 \pm 0.9	36 \pm 1.0	0.621	0.602	

TABLE 27

**LIPID PROFILE OF THE SUBJECTS IN RELATION TO
ALCOHOL CONSUMPTION (mg/dl, Mean \pm SD)**

	Alcoholics	Non-Alcoholics
n	106	351
TC	204.2 \pm 41.3	199.5 \pm 37.9
TG	164.8 \pm 93.2	142.7 \pm 69.9 **
HDL-C	39.1 \pm 8.5	37.6 \pm 8.3
LDL-C	132.2 \pm 31.2	133.5 \pm 30.6
VLDL-C	33.0 \pm 18.6	28.5 \pm 14.0 **
Non HDL-C	165.1 \pm 35.9	162.1 \pm 33.4
TC/H	5.3 \pm 0.9	5.5 \pm 1.1
L/H	3.5 \pm 0.9	3.7 \pm 0.9 *

Alcoholics * p<0.05, ** p<0.01 Vs Non-alcoholics

TABLE 28

ANALYSIS OF VARIANCE IN RELATION TO ALCOHOL CONSUMPTION AMONG NORMALS AND SUBJECTS WITH CDD (mg/dl) (Mean \pm SD)

	NORMALS		SUBJECTS WITH CDD		F Ratio	F Prob.	Significant Pairs
	Non Alcoholics	Alcoholics	Non Alcoholic	Alcoholics			
Groups	(1)	(2)	(3)	(4)			
n	171	43	180	63			
TC	190.4 \pm 38.9	185.5 \pm 38.5	208.1 \pm 34.9	216.98 \pm 38.5	13.146	0.000	(1,3), (1,4), (2,3), (2,4)
TG	131.9 \pm 67.0	147.0 \pm 99.1	152.8 \pm 71.1	177.0 \pm 87.7	5.968	0.000	(1,4)
HDL-C	36.1 \pm 8.8	36.8 \pm 8.6	38.9 \pm 7.5	40.6 \pm 8.1	6.053	0.000	(1,3), (1,4)
LDL-C	128.1 \pm 29.6	119.3 \pm 27.8	138.7 \pm 30.7	141.0 \pm 30.5	8.181	0.000	(1,3), (1,4), (2,3), (2,4)
VLDL-C	26.39 \pm 13.40	29.40 \pm 19.82	30.56 \pm 14.23	35.39 \pm 17.53	5.968	0.000	(1,4)
NON HDL-C	154.5 \pm 33.5	148.7 \pm 32.8	169.3 \pm 31.7	176.4 \pm 33.8	12.293	0.000	(1,3), (1,4), (2,3), (2,4)
TC/H	5.5 \pm 1.2	5.2 \pm 0.8	5.5 \pm 1.0	5.4 \pm 0.9	1.196	0.311	
L/H	3.7 \pm 1.0	3.4 \pm 0.8	3.7 \pm 1.0	3.6 \pm 0.9	1.731	0.160	

the lipid profile in the subjects who had CDD as compared to normal subjects, indicating the need to modify the lifestyle factors in these subjects

There was no marked difference in the lipid parameters between the subjects leading a totally sedentary life style or those with moderate activity in their routine life

RELATIVE RISK OF THE SUBJECTS IN RELATION TO GENERAL HABITS OF THE SUBJECTS

The relative risk of the subjects in relation to general habits of the subjects such as smoking or chewing tobacco and alcohol consumption is given in **figure 15**. The relative risk for smoking was found to be 1.6, indicating that the diseased people who were smoking were at a higher risk of developing complications in comparison to the normal subjects. Similarly the relative risk for tobacco consumers was 1.3 and that for alcohol consumption was 1.4 respectively.

The relative risk was also calculated taking into consideration the individual atherogenic lipid levels viz TC, TG, LDL-C and Non HDL-C among smokers, alcoholics and tobacco consumers (**Table 29**). The results clearly indicate that the risk for the elevation in lipid levels is more in case of smokers, tobacco chewers and alcoholics.

FIGURE 15

**RELATIVE RISK OF SUBJECTS WITH CHRONIC
DEGENERATIVE DISEASES IN RELATION TO
VARIOUS HABITS**

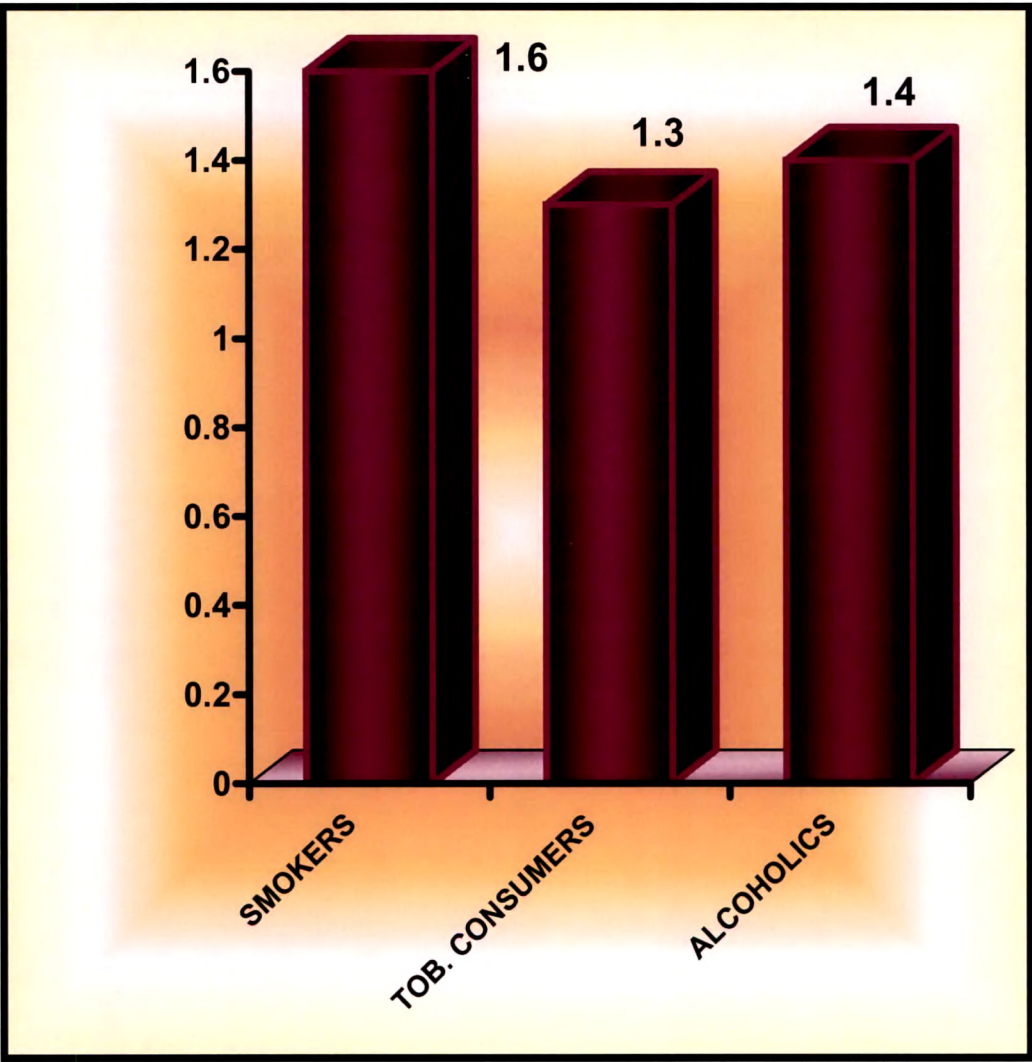


TABLE 29
RELATIVE RISK OF THE SUBJECTS WITH RESPECT TO LIPID
PROFILE AMONG SMOKERS, TOBACCO CONSUMERS AND
ALCOHOLICS

	RELATIVE RISK			
	TC	TG	NON HDL-C	LDL-C
SMOKERS	1 2	1 3	1 1	1 1
TOBACCO CONSUMERS	1 0	1 0	1 0	1 0
ALCOHOLICS	1 0	1 2	1 0	1 0

DISCUSSION

The prevalence of chronic degenerative diseases which is increasing to epidemic proportions around the world, especially in the developing nations has prompted us to find out the prevalence of these diseases in the developing countries and economies in transition. In addition to the prevalence of these CDD it is of utmost importance to study the risk factors that act synergistically in the development or progression of these diseases. Some of the risk factors are non-modifiable, whereas lifestyle related risk factors are modifiable.

In the present study the prevalence of CDD as overweight, obesity, diabetes, hypertension, CHD and HL was found to be 33%, 8%, 8%, 6% and 1% respectively. It is evident from the figure that among the prevalence of overweight and obesity was found to be more in comparison to other degenerative diseases. Out of 82 diabetics 44% were overweight and obese. Similarly among the hypertensives, 54% were found to be overweight and obese. The data clearly shows a high prevalence of CDD in the industrial setup. The magnitude of the problem is more with overweight and obesity, which is the mother of all CDD. Since overweight and obesity coexist with CDD, it becomes imperative to map the prevalence of CDD to develop strategies to tackle the problem. These results further strengthen the plausible role of obesity in predisposing an individual to the development of other CDD such as diabetes, hypertension and CHD. The prevalence data also reveals

that though in comparison to males, there were more number of females who were obese ($BMI \geq 30$), the prevalence of diabetes, hypertension and CHD was higher among the males than females. This implies that at any level of risk factors women have a distinct advantage over men (Veronique 2000)

The increasing prevalence of chronic degenerative diseases in this study portrays that the morbidity of chronic degenerative diseases is on the rise in urban cities of India and it lays emphasis to study the risk factors associated with the same

It is said that females have an advantage over men with respect to CDD until they reach menopause. The lipid levels of the male and female subjects showed that the males had significantly higher levels of TC ($p < 0.001$), TG ($p < 0.001$), VLDL-C ($p < 0.001$), Non HDL-C ($p < 0.001$) and TC/H ($p < 0.01$) ratio respectively (Table 20). The relative risk with respect to atherogenic lipid parameters showed that as compared to females the males showed 1.1 times more aberrations in TC. Similarly, the males were found to be 1.7 times more probable to show aberrations in TG, which is now considered to be an independent risk factor for the cardio vascular risk (Kannel and Schatzkin 1983). The relative risk for the atherogenic LDL-C was 1.1 and that for Non HDL-C was 1.1 when compared with female subjects. Thus gender appears to be the non-modifiable-risk factor for aberrations in lipid profile.

Another biological risk factor is age. As the age advances the risk of suffering

from any of the CDD increases. The influence of age on the lipid parameters of the male and female subjects is depicted in Table 22. It was observed that the males who were above the age of 40 years had significantly higher levels of TC ($p<0.01$), LDL-C ($p<0.001$) and Non HDL-C ($p<0.001$) in comparison to those below the age of 40 years. This is in line with the other published reports which has shown that as the age advances the aberrations in the lipid parameters also increases (Kannel and Schatzkin 1983, Kritchevsky 1978). It is also observed that the prevalence of CDD increases after the age of 40 years, which again emphasizes the fact that the chances of having atherogenic dyslipidaemia are more as age advances. In the present study, there were more subjects suffering from CDD above the age of 40 years.

The analysis of lipid parameters among females with respect to age showed that the atherogenic lipid parameters such as TG, LDL-C, VLDL-C and Non HDL-C was found to be significantly raised ($p<0.001$) among females who were above the age of 40 in comparison to those below the age of 40 years. The LDL-C levels start to accelerate between 40-50 years of age and that the HDL-C concentration remains constant in women throughout their lifetime (Leaf 1990). It has also been observed that the TG concentration increases progressively in men, reaching peak values, between 40-50 years of age and in case of women, the triglyceride concentration increases throughout out their lifetime. This might have been the underlying cause for getting similar TG concentrations among males in both the age groups, whereas the females showed significantly raised levels of TG. With advancing age the coronary risk

in menopausal women grows. The protective effect that the females have over men before menopause is lost and they are at the same level of risk as men to suffer from various CDD. Studies have shown that women share the same major coronary risk as men including serum cholesterol levels and other risk factors unique to their gender (Leaf 1990) Similar results have been observed here where it is evident that the atherogenic lipid parameters in male and female subjects above the age of 40 years is almost similar

When the lipid profile of the subjects with respect to educational status and per capita income was examined it did not show any appreciable changes in the lipid parameters. The subjects in this population had access to food items at a highly subsidized rate, which attracted even the more educated people to consume a higher quantity of energy rich foods. Thus, the perception of the employees was that it was their right as an employee to use the coupons provided to them for the purchase of snacks and many of them were taking it to their homes. Thus, in relation to dietary aspect the perception of the people was same whether they were educated less or more, which in turn might not have shown any difference in the lipid parameters with respect to educational status. The background information of the subjects revealed that majority of the subjects had PCI ≥ 1000 and even those below the 1000 mark had access to various facilities such as canteen foods and good housing which were provided by the company. Even the groceries were available at subsidized rate to them. Moreover, the lowest grade worker also got good pay so as to lead a satisfactory life in terms of monetary aspect. In view of this, the

difference in per capita income has not shown any major change in their food consumption pattern

The influence of habits of subjects such as smoking or chewing tobacco, alcohol consumption and physical inactivity of the individuals increases the chances of giving rise to complications among those subjects who are diseased as well as among the normal subjects. The risk of death from CAD is up to six times higher in smokers than in non-smokers (Whig et al 1992). Smoking acts adversely by altering the lipid profile and by enhancing the inflammatory response to high blood lipids leading to blood clots in the arteries. Smoking increases the level of stress hormones and platelet adhesiveness. Cigarette smoking has also shown to be associated with endothelial dysfunction. After smoking cigarettes, there is a doubling in the number of circulating endothelial cells in the peripheral blood vessels reflecting an increased turnover and desquamation of the endothelium (Davies et al 1985).

Cigarette smoking alters the serum lipids and lipoproteins and these changes are related to the duration and amount of smoking. Active smoking increases LDL-C and VLDL-C whereas HDL-C content is lowered, resulting in decreased ratio of HDL-C /TC and HDL-C / LDL-C (Whig et al 1992, Shah and Sadaria 2003). The effect of smoking habit of the subjects is depicted in Table 22. In the present study no appreciable changes in the lipid parameters were observed in relation to smoking habit. However, the TG concentration did

show a non significant rise among the smokers. The changes in the lipid levels are related to the amount and duration of smoking. In this population among the subjects who smoked (n=65) majority of them consumed less than 10 cigarettes per day, which in turn might not have shown a significant difference among the mean values. Further analysis of variance and the multiple range test was also carried in order to see if there was any difference in the lipid profile among the normal and diseased subjects (smokers versus non smokers) (Table 24). The TC and Non HDL-C showed significantly higher levels among the diseased smokers in comparison to the normals who were not smoking. The subjects suffering from CDD as such will have atherogenic dyslipidaemia, so it is necessary to know how much the habit of smoking contributes to the deterioration in the lipid parameters. Hence, the relative risk in relation to atherogenic lipid parameters was studied and the results showed that the risk of an increase in the TC levels because of smoking was found to be 1.2 times in comparison to normal subjects who smoked. The diseased subjects who smoked were 1.3 times more likely to develop aberration in the TG levels in comparison to normals who smoked. Similarly the relative risk for Non HDL-C and LDL-C was found to be 1.1 respectively which indicates that the diseased smokers were definitely at a higher risk in comparison to normal subjects to develop aberrations with respect to atherogenic lipid parameters. This also implies that the aberrations observed in the lipid parameters of the diseased smokers were to some extent attributable to the smoking habit.

In addition to smoking tobacco in the form of cigarettes or bidis, there is a

widespread prevalence among the population to chew tobacco, which again is hazardous or detrimental to the health. In Gujarat there are many people who are in the grip of this habit of chewing tobacco. The lipid profile of the tobacco chewers when compared with those who did not consume tobacco showed no significant difference. The rise in the lipid levels of the tobacco consumers are again dependent on the amount and form of tobacco consumed. The analysis of variance among the diseased and non-diseased subjects who either chewed or did not chew tobacco when studied showed that the diseased subjects had significantly higher levels of lipids in comparison to normal subjects not consuming and also with normal subjects consuming tobacco in the form of ghutka or padiki. Among the diseased subjects, those who chewed tobacco showed a rise in the TG levels when compared with non-tobacco consumers. The rise was non-significant as the number of padikis that the subjects said they consumed is either 2-3 per day which is less likely to bring about any changes in the lipid levels. However, the relative risk of the diseased subjects who chewed tobacco with respect to TG and TC was found to be 1.0, thus indicating that the diseased subjects who consumed tobacco were more likely to have aberrations in their TG and TC values in comparison to the normal subjects who chewed tobacco.

Many studies have shown an inverse relation between alcohol consumption and CHD, with a possible flattening at higher consumption levels (Rimm et al 1996). The results of some studies suggest that light to moderate alcohol consumption is associated with similar risk reductions in CHD (Ajani et al

2000) Further, studies have been carried out to assess whether the protective effect is confined to specific beverages (such as red wine) Bobak et al in 2000 showed that it is ethanol, which shows the protective effect, and not the specific substances present in different types of beverages

Light to moderate consumption of alcohol in some populations has shown some protective effect, but one cannot neglect the fact that excessive alcohol intake is a risk factor for CHD, hypertension and all other cardiovascular diseases Excess intake leads to increase in blood pressure because of increase in sympathetic nerve activity, which increase cardiac output and increase heart rate (Malhotra and Patel, 2003). Hart et al in 1999 showed a positive correlation between alcohol consumption and the risk of mortality from stroke in men drinking excessive alcohol. In addition to the amount of alcohol consumed there are studies which now show that not only the total amount consumed but the drinking style, may have an impact on the development of atherosclerotic disease and especially its progression (Kauhanen et al 1999)

The results of the present study show significantly raised levels of TG and VLDL-C among alcohol consumers in comparison to those not consuming alcohol. Though non significant, a rise in the level of HDL-C among alcohol consumers is observed The analysis of variance and multiple range test by Scheffe procedure showed significant rise in the level of TC, TG, HDL-C, LDL-C, VLDL-C and Non HDL-C concentrations among the diseased alcoholics in

comparison to the other groups (Table 28) The relative risk when calculated for various lipid parameters showed the risk to be highest with respect to TG levels among diseased subjects ($RR = 1.16$) Thus, indicating that the diseased subjects were 1.16 times at a higher risk than their normal counterparts to show aberrations in TG levels.

Physical inactivity as we are aware is also one of the risk factors for the development of complications related to various CDD, and that exercise or physical activity tends to lower the risk. However, the type of exercise and the amount of time spent for the same, will decide whether it is beneficial in relation to lipid parameters. Exercise by itself is beneficial, but how much a person needs to exercise in order to bring about positive changes in weight, management of diabetes or lowering the atherogenic lipid levels is of concern. There are various studies being carried out which shows the benefits of exercise, but these are based on following a strict regime for exercising for a specified time and duration (Bray 2000). The results of the present study did not show any appreciable changes in the lipid levels of those who exercised. Majority of the subjects exercised only two or three times a week which is less likely to bring about a reduction in the atherogenic lipid parameters. Furthermore, the subjects reported that they exercised even if they went for a stroll in the evening or even if they went for shopping in the township itself.

From the review available from various studies worldwide, and the risk factor analysis in this study, it is evident that the habits of the subjects such as

smoking or chewing tobacco and consumption of alcohol has a profound negative influence on lipid metabolism. In order to find out the contribution of each of these with respect to atherogenic lipid parameters the relative risk of the subjects who were smoking, eating tobacco and consuming alcohol was calculated (Table 29). As can be seen from the table the highest risk is observed with respect to TG among the smokers ($RR = 1.3$).

The relative risk of the diseased subjects who smoked, chewed tobacco or consumed alcohol was found out by making a comparison with the normal subjects who were addicts of such habits (Figure 15). The subjects who smoked showed a relative risk of 1.6 followed by 1.4 for alcoholics and 1.3 in case of tobacco consumers. This indicates that smokers who were suffering from any kind of CDD were 1.6 times more probable to develop complications related to the disease in comparison to normal subjects. Similarly the subjects who consumed alcohol were 1.4 times at a higher risk in comparison to normal subjects to worsen the clinical complications. And subjects chewing tobacco were found to be 1.3 times at a higher risk in comparison to normal subjects to develop complications related to alcohol consumption. These results indicate that the general habits of the subjects such as smoking or chewing tobacco and consumption of alcohol do play a role in worsening the disease profile of the subjects and that those who are not smoking, chewing tobacco or consuming alcohol are at a much lower risk in comparison to those having these habits.

The results of this study thus showed that the females had a lower level of atherogenic lipids, which implies that the females are at an advantage over male subjects with respect to lipid profile. It was also noticed that as age advances a profound increase in the atherogenic lipid parameters was observed. The results also emphasize the risk involved in developing complications among the normal and diseased subjects with respect to various habits of the subjects such as chewing or smoking tobacco, consumption of alcohol etc., which along with other risk factors act synergistically in the progression or development of various CDD.

At a glance

✎ The prevalence of overweight or obesity (BMI ≥ 25) was found to be 41%. About 18% of the overweight or obese subjects had either diabetes, hypertension or CHD. The prevalence of diabetes (8%), hypertension (6%) and CHD (1%) was 15%. Dyslipidemia was present in 20% of the subjects.

✎ Aberrations in the lipid profile were more pronounced in male subjects than female subjects. The male subjects had TC levels (200.6 ± 38.7 mg/dl) significantly higher than the females (190.0 ± 34.1 mg/dl). Similarly, the TG, VLDL-C, Non HDL-C and TC/H ratio were also found to be significantly higher among the males.

✎ The atherogenic lipid parameters were found to increase in both men and women above 40 years of age.

✎ Among the modifiable factors studied, the relative risk for aberrations in lipid profile was found to be highest for smoking (RR = 1.6), followed by alcohol consumption (RR = 1.4) and tobacco chewing (RR = 1.3).

SECTION III

METABOLIC PROFILE OF CHRONIC DEGENERATIVE DISEASES IN AN INDUSTRIAL SET-UP

Lifestyle related diseases such as obesity, diabetes, hypertension and CHD are rapidly increasing to epidemic proportions and gaining their hold over developing countries. Among the various etiological factors, elevated levels of the circulating lipids are of great importance. Therefore, it becomes prudent to study the atherogenic lipid parameters of the subjects suffering from various chronic degenerative diseases.

The present section was thus designed to study the atherogenic lipid parameters of the study population.

The study group comprised of a) normals, b) dyslipidemics (based on increased TC, increased TG, or increased TG and TC), c) overweight and obese subjects (based on BMI) and d) those subjects having any one of the CDD.

The results of this section are presented under the following headings

FASTING BLOOD SUGAR AND LIPID PROFILE OF THE STUDY GROUP

The FBS and lipid profile of the normal, overweight or obese, diabetic, hypertensive and CHD subjects is depicted in **Table 30**. The overweight and obese ($\text{BMI} \geq 25$), diabetic, hypertensive & CHD subjects showed significant elevations in the lipid profile as compared to normal subjects. Also, the atherogenic ratio of TC/H demonstrated an increase among the diabetics in comparison to normal subjects.

The diabetic subjects had significantly raised FBS (157.3 ± 50.7 mg/dl) in comparison to overweight and obese (87.5 ± 13.8 mg/dl) subjects. The diabetics also had raised TG (190.8 ± 106.7 mg/dl Vs 142.2 ± 72.1 mg/dl), VLDL-C (38.2 ± 21.3 mg/dl Vs 28.5 ± 14.4 mg/dl) and Non HDL-C (173.0 ± 36.8 mg/dl Vs 163.4 ± 32.4 mg/dl) levels in comparison to overweight and obese subjects.

When the comparison was done between overweight and obese and hypertensive subjects, higher levels of LDL-C ($p < 0.05$) observed among the overweight and obese subjects. The atherogenic indices of TC/H ($p < 0.05$) and L/H ($p < 0.01$) also demonstrated an increase among the overweight and obese subjects. But, the CHD subjects had significantly higher levels ($p < 0.05$) of TG and VLDL-C in comparison to overweight and obese subjects.

TABLE 30

FASTING BLOOD SUGAR AND LIPID PROFILE OF THE STUDY GROUP (mg/dl, Mean \pm SD)

FBS, which is the hallmark of diabetes was found to be significantly high among the diabetics (157.3 ± 50.7 mg/dl) in comparison to hypertensive (89.3 ± 10.9 mg/dl) subjects. The atherogenic lipid levels viz TC, TG, VLDL-C and Non HDL-C were increased significantly in comparison to hypertensive subjects. The TC/H ratio was also significantly high among diabetics in comparison to hypertensive subjects.

FASTING BLOOD SUGAR AND LIPID PROFILE OF MALES

The lipid profile of the male subjects suffering from any kind of chronic degenerative disease showed higher lipid levels compared to the normals (**Table 31**). The diabetics showed significant increment ($p < 0.001$) in the FBS level in comparison to overweight and obese subjects. The TG (190.3 ± 102.9 mg/dl Vs 154.8 ± 83.7 mg/dl) and VLDL-C (38.1 ± 20.6 mg/dl Vs 31.0 ± 16.7 mg/dl) levels also demonstrated a significant rise compared to overweight and obese subjects. The overweight and obese subjects also showed significantly raised TC/H ($p < 0.05$) and L/H ($p < 0.05$) ratios in comparison to hypertensives.

When a similar comparison was made between diabetics and hypertensives, apart from high FBS ($p < 0.001$), the TC ($p < 0.05$), TG ($p < 0.05$), VLDL-C ($p < 0.05$), Non HDL-C ($p < 0.01$) and TC/H ($p < 0.01$) levels also showed significant increase in diabetics.

TABLE 31
FASTING BLOOD SUGAR AND LIPID PROFILE OF MALES (mg/dl, Mean \pm SD)

VARIABLE	NORMALS	OVERWEIGHT & OBESE SUBJECTS	DIABETICS	HYPERTENSIVES	CHD SUBJECTS
n	88	126	55	42	6
FBS	86.1 \pm 17.5	89.3 \pm 15.0	161.3 \pm 53.7 ^{***} ●●● ^{ψψψ}	89.0 \pm 10.7	139.3 \pm 85.1 ^{***} ●●● ^{†††}
TC	164.1 \pm 20.6	207.1 \pm 38.8 ^{***}	214.1 \pm 46.5 ^{***} ^ψ	195.8 \pm 37.5 ^{***}	210.83 \pm 54.81 ^{***}
TG	106.6 \pm 46.5	154.8 \pm 83.7 ^{***}	190.3 \pm 102.9 ^{***} ● ^ψ	147.1 \pm 77.3 ^{***}	211.83 \pm 124.59 ^{***}
HDL-C	32.4 \pm 7.9	39.0 \pm 8.3 ^{***}	39.2 \pm 9.9 ^{***}	39.4 \pm 6.9 ^{***}	42.67 \pm 13.56 ^{**}
LDL-C	110.9 \pm 17.9	137.2 \pm 30.9 ^{***}	136.9 \pm 34.9 ^{***}	127.0 \pm 34.5 ^{***}	125.8 \pm 18.4
VLDL-C	21.3 \pm 9.3	31.0 \pm 16.7 ^{***}	38.1 \pm 20.6 ^{***} ● ^ψ	29.4 \pm 15.5 ^{***}	42.4 \pm 24.9 ^{***}
NON HDL-C	132.3 \pm 17.8	168.2 \pm 33.7 ^{***}	174.9 \pm 39.8 ^{***} ^ψ	156.4 \pm 35.1 ^{***}	168.2 \pm 42.3 ^{***}
TC/H	5.4 \pm 1.4	5.4 \pm 0.8 ^φ	5.6 \pm 1.0 ^{ψψ}	5.1 \pm 1.0	5.1 \pm 0.7
L/H	3.7 \pm 1.2	3.6 \pm 0.8 ^φ	3.6 \pm 1.0	3.3 \pm 1.0	3.2 \pm 0.8

All groups * p<0.05, ** p<0.01, ***p<0.001 Vs Normals
 Overweight & Obese Subjects ● p<0.05, ●● p<0.01, ●●● p<0.001 Vs Diabetics & CHD Subjects
 Overweight & Obese Subjects^φ p<0.05 Vs Hypertensives
 Diabetics ^ψ p<0.05, ^{ψψ} p<0.01, ^{ψψψ} p<0.001 Vs Hypertensives
 Hypertensives ^{†††} p<0.001 Vs CHD Subjects

FASTING BLOOD SUGAR AND LIPID PROFILE OF FEMALES

The FBS and lipid levels of the females who were normal and those suffering from various CDD are depicted in **Table 32**. The atherogenic lipid parameters showed a significant increase in females with any one of the CDD as compared to normals.

The diabetic females had significantly elevated levels of FBS ($p < 0.001$), TG ($p < 0.001$) and VLDL-C ($p < 0.001$) as compared to overweight and obese but clinically normal females. But the same overweight and obese clinically normal group exhibited a difference in lipid pattern (TC, $p < 0.05$), (LDL-C, $p < 0.01$), (Non HDL-C, $p < 0.05$) and (L/H, $p < 0.05$) as compared to subjects with hypertension.

FASTING BLOOD SUGAR AND LIPID PROFILE OF THE STUDY GROUP IN RELATION TO OVERWEIGHT & OBESE CONDITION

The comparison of lipid profile in the study group (males and females) based on the overweight and obese condition is presented in **Table 33**. The overweight and obese diabetics had significantly higher levels of FBS ($p < 0.001$), TG ($p < 0.01$), VLDL-C ($p < 0.01$) and Non HDL-C ($p < 0.05$) in comparison to overweight and obese subjects who were clinically normal. The atherogenic LDL-C, Non HDL-C, TC/H as well as the L/H ratio were found to be significantly higher among the overweight and obese subjects in comparison to overweight and obese hypertensives.

TABLE 32
FASTING BLOOD SUGAR AND LIPID PROFILE OF FEMALES (mg/dl, Mean \pm SD)

VARIABLE	NORMALS	OVERWEIGHT & OBESE SUBJECTS	DIABETICS	HYPERTENSIVES
n	76	105	16	6
FBS	80.4 \pm 11.0	85.3 \pm 12.0 ^{**}	143.5 \pm 37.2 ^{***} ●●● ^{ψψ}	91.2 \pm 13.6 [*]
TC	165.5 \pm 25.9	195.6 \pm 33.9 ^{***} ϕ	202.0 \pm 28.3 ^{***} ψ	165.8 \pm 33.8
TG	87.0 \pm 52.4	127.2 \pm 51.6 ^{***}	192.8 \pm 122.3 ^{***} ●●●	167.3 \pm 56.7 ^{**}
HDL-C	33.4 \pm 6.7	37.9 \pm 8.7 ^{***}	38.1 \pm 8.3 [*]	35.0 \pm 6.0
LDL-C	114.6 \pm 22.5	132.3 \pm 27.2 ^{***} ϕ ϕ	127.8 \pm 30.0 [*]	97.4 \pm 39.1
VLDL-C	17.4 \pm 10.5	25.4 \pm 10.3 ^{***}	38.6 \pm 24.5 ^{***} ●●●	33.5 \pm 11.3 ^{**}
NON HDL-C	132.1 \pm 24.1	157.7 \pm 30.1 ^{***} ϕ	166.3 \pm 23.7 ^{***} ψ	130.8 \pm 32.9
TC/H	5.1 \pm 1.0	5.3 \pm 0.9	5.5 \pm 0.9	4.8 \pm 1.1
L/H	3.6 \pm 1.0	3.6 \pm 0.9 ^ϕ	3.4 \pm 1.0	2.8 \pm 1.1

All groups * p<0.05, ** p<0.01, *** p<0.001 Vs Normals
 Overweight & Obese Subjects ● p<0.05, ●● p<0.01, ●●● p<0.001 Vs Diabetics
 Overweight & Obese Subjects ϕ p<0.05, ϕ ϕ p<0.01 Vs Hypertensives
 Diabetics ψ p<0.05, ψψ p<0.01, ψψψ p<0.001 Vs Hypertensives

TABLE 33

**FASTING BLOOD SUGAR AND LIPID PROFILE OF THE STUDY
GROUP IN RELATION TO OVERWEIGHT & OBESE
CONDITION (mg/dl, Mean \pm SD)**

VARIABLE	OVERWEIGHT & OBESE SUBJECTS	DIABETICS (OVERWEIGHT/OBESE)	HYPERTENSIVES (OVERWEIGHT/OBESE)
n	231	29	25
FBS	87.5 \pm 13.8	157.1 \pm 47.1 *** $\Psi\Psi\Psi$	90.0 \pm 12.6
TC	201.9 \pm 37.0	215.9 \pm 48.2 Ψ	187.6 \pm 35.8
TG	142.2 \pm 72.1	187.7 \pm 110.2 **	148.4 \pm 65.1
HDL-C	38.5 \pm 8.5	39.8 \pm 9.7	38.6 \pm 6.9
LDL-C	134.9 \pm 29.3 Ψ	139.9 \pm 39.1	119.3 \pm 36.6
VLDL-C	28.5 \pm 14.4	37.5 \pm 22.0 **	29.7 \pm 13.0
NON HDL-C	163.4 \pm 32.4 Ψ	177.5 \pm 41.4 * Ψ	149.0 \pm 33.7
TC/H	5.4 \pm 0.9 Ψ	5.6 \pm 1.1 Ψ	5.0 \pm 1.0
L/H	3.6 \pm 0.8 Ψ	3.6 \pm 1.1	3.2 \pm 1.0

Overweight & Obese Subjects * p<0.05, ** p<0.01, *** p<0.001 Vs Diabetics (Overweight/ Obese)

Overweight & Obese Subjects Ψ p<0.05 Vs Hypertensives (Overweight/ Obese)

Diabetics (Overweight/ Obese) Ψ p<0.05, $\Psi\Psi$ p<0.01, $\Psi\Psi\Psi$ p<0.001 Vs Hypertensives (Overweight /Obese)

FASTING BLOOD SUGAR AND LIPID PROFILE OF MALES IN RELATION TO OVERWEIGHT & OBESE CONDITION

When the comparison was done on gender basis, overweight and obese male diabetics had higher levels of FBS ($p < 0.001$) than the overweight and obese males without any clinical problems and significantly higher levels of FBS ($p < 0.001$), Non HDL-C ($p < 0.05$) and TC/H ratio ($p < 0.05$) in comparison to the overweight and obese hypertensives but the overweight and obese males had higher TC/h ratio as compared to hypertensive subjects (**Table 34**)

FASTING BLOOD SUGAR AND LIPID PROFILE OF FEMALES IN RELATION TO OVERWEIGHT AND OBESE CONDITION

When the overweight and obese conditions taken into consideration among females with and without any of the CDD, the diabetic population showed a raised FBS ($p < 0.001$), TG ($p < 0.05$) and VLDL-C ($p < 0.05$), and there is an increasing trend with respect to TC, Non HDL-C and LDL-C in comparison to overweight and obese who were clinically normal. On comparison with hypertensive group also the elevation in FBS ($p < 0.01$), TC ($p < 0.05$) and Non HDL-C ($p < 0.05$) were noticed in diabetic females (**Table 35**). Even though there is no known CDD present in the overweight and obese group there is an altered lipid profile pattern (TC, LDL-C and Non HDL-C) in this group as compared to hypertensive group.

TABLE 34

**FASTING BLOOD SUGAR AND LIPID PROFILE OF MALES IN
RELATION TO OVERWEIGHT & OBESE CONDITION
(mg/dl, Mean \pm SD)**

VARIABLE	OVERWEIGHT & OBESE SUBJECTS	DIABETICS (OVERWEIGHT/OBESE)	HYPERTENSIVES (OVERWEIGHT/OBESE)
n	126	22	21
FBS	89.3 \pm 15.0	161.1 \pm 51.6 ^{***$\Psi\Psi\Psi$}	89.1 \pm 12.3
TC	207.1 \pm 38.8	219.3 \pm 54.0	192.8 \pm 34.6
TG	154.8 \pm 83.7	190.1 \pm 109.9	147.7 \pm 67.0
HDL-C	39.0 \pm 8.3	39.5 \pm 10.0	39.2 \pm 7.0
LDL-C	137.2 \pm 30.9	141.8 \pm 43.6	124.0 \pm 34.3
VLDL-C	31.0 \pm 16.7	38.0 \pm 22.0	29.5 \pm 13.4
NON HDL-C	168.2 \pm 33.7	172.8 \pm 47.2 ^{Ψ}	153.6 \pm 32.3
TC/H	5.4 \pm 0.8 ^{Φ}	5.6 \pm 1.1 ^{Ψ}	5.0 \pm 0.9
L/H	3.6 \pm 0.8	3.7 \pm 1.1	3.3 \pm 1.0

Overweight & Obese Subjects • p<0.05, •• p<0.01, ••• p<0.001 Vs Diabetics (Overweight/Obese)

Overweight & Obese Subjects ^{Φ} p<0.05 Vs Hypertensives (Overweight/Obese)

Diabetics (Overweight/Obese) ^{Ψ} p<0.05, ^{$\Psi\Psi$} p<0.01, ^{$\Psi\Psi\Psi$} p<0.001 Vs Hypertensives (Overweight/Obese)

TABLE 35

**FASTING BLOOD SUGAR AND LIPID PROFILE OF FEMALES
IN RELATION TO OVERWEIGHT & OBESE CONDITION
(mg/dl, Mean \pm SD)**

VARIABLE	OVERWEIGHT & OBESE SUBJECTS	DIABETICS (OVERWEIGHT/OBESE)	HYPERTENSIVES (OVERWEIGHT/OBESE)
N	105	7	5
FBS	85.3 \pm 12.0	144.6 \pm 27.9 *** $\Psi\Psi$	84.8 \pm 14.9
TC	195.6 \pm 33.9 Φ	205.4 \pm 21.0 Ψ	160.3 \pm 33.1
TG	127.2 \pm 51.6	180.3 \pm 119.6 *	152.0 \pm 63.4
HDL-C	37.9 \pm 8.7	41.2 \pm 9.4	35.3 \pm 6.2
LDL-C	132.3 \pm 27.2 Φ	134.1 \pm 21.0	94.6 \pm 43.2
VLDL-C	25.4 \pm 10.3	36.1 \pm 23.9 *	30.4 \pm 12.7
NON HDL-C	157.7 \pm 30.1 Φ	170.1 \pm 10.5 Ψ	125.0 \pm 34.8
TC/H	5.3 \pm 0.9	5.3 \pm 0.9	4.7 \pm 1.4
L/H	3.6 \pm 0.9	3.3 \pm 1.2	2.8 \pm 1.3

Overweight & Obese Subjects • p<0.05, ** p<0.01, *** p<0.001 Vs Diabetics (Overweight/Obese)

Overweight & Obese Subjects Φ p<0.05 Vs Hypertensives (Overweight/Obese)

Diabetics (Overweight/Obese) Ψ p<0.05, $\Psi\Psi$ p<0.01 Vs Hypertensives (Overweight/Obese)

LIPID PROFILE OF NORMAL AND DYSLIPIDEMIC MALES

The comparison of the dyslipidemias viz. hyperlipidemias, hypercholesterolemias and hypertriglyceridemias with the normal males is depicted in **table 36**. It was observed that hyperlipidemias had significantly increased levels of all the atherogenic lipid parameters except the TC/H and L/H ratios in comparison to normal subjects. Similar observations were noticed on comparison of lipid parameters between normals and hypercholesterolemias. The hypertriglyceridemias had significantly higher TG ($p<0.001$), LDL-C ($p<0.01$) and VLDL-C ($p<0.001$) and L/H ratio ($p<0.05$) in comparison to the normals.

The TC ($p<0.01$), TG ($p<0.001$), VLDL-C ($p<0.001$) and Non HDL-C ($p<0.05$) levels in hyperlipidemias were found to be significantly higher than hypercholesterolemias. On the other hand, hyperlipidemias had significantly decreased L/H ratio ($p<0.001$) in comparison to hypercholesterolemias.

When compared with hypertriglyceridemias, the hyperlipidemic subjects displayed increased levels of TC ($p<0.001$), LDL-C ($p<0.001$), Non HDL-C ($p<0.001$), TC/H ($p<0.05$) and L/H ($p<0.01$) ratios, similarly the hypercholesterolemias also had higher levels of TC ($p<0.001$), LDL-C ($p<0.001$), Non HDL-C ($p<0.001$), TC/H ($p<0.05$) and L/H ($p<0.001$) in comparison to hypertriglyceridemias, whereas, the levels of TG ($p<0.001$) and VLDL-C ($p<0.001$) were raised among the hypertriglyceridemias.

TABLE 36

LIPID PROFILE OF NORMAL AND DYSLIPIDEMIC MALES (mg/dl, Mean \pm SD)

VARIABLE	NORMALS	HYPERLIPIDEMICS	HYPER CHOLESTEROLEMICS	HYPER TRIGLYCERIDEMICS
n	88	61	73	7
TC	164.1 \pm 20.6	222.6 \pm 31.8 *** ## $\varphi\varphi\varphi$	210.9 \pm 18.0 *** $\pi\pi\pi\pi$	158.6 \pm 24.0
TG	106.6 \pm 46.5	191.6 \pm 45.4 *** ###	107.9 \pm 27.6	188.7 \pm 23.9 *** $\pi\pi\pi$
HDL-C	32.4 \pm 7.9	40.9 \pm 6.4 *** # φ	38.3 \pm 6.4 ***	34.4 \pm 7.7
LDL-C	110.9 \pm 17.9	143.4 \pm 27.8 *** $\varphi\varphi\varphi$	151.0 \pm 17.4 *** $\pi\pi\pi\pi$	86.4 \pm 29.0 **
VLDL-C	21.3 \pm 9.3	38.3 \pm 9.1 *** ###	21.6 \pm 5.5	37.7 \pm 4.8 *** $\pi\pi\pi\pi$
NON HDL-C	132.3 \pm 17.8	181.7 \pm 28.9 *** # $\varphi\varphi\varphi$	172.5 \pm 17.4 *** $\pi\pi\pi\pi$	124.1 \pm 26.4
TC/H	5.4 \pm 1.4	5.5 \pm 0.8 φ	5.6 \pm 1.0 π	4.8 \pm 1.1
L/H	3.7 \pm 1.2	3.5 \pm 0.7 $\varphi\varphi$	4.1 \pm 0.9 * ### $\pi\pi\pi\pi$	2.7 \pm 1.0 *

All groups * p<0.05, ** p<0.01, ***p<0.001 Vs Normals

Hyperlipidemics # p<0.05, ## p<0.01, ### p<0.001 Vs Hypercholesterolemics

Hyperlipidemics φ p<0.05, φ φ p<0.01, φ φ φ p<0.001 Vs Hypertriglyceridemics

Hypercholesterolemics π p<0.05, $\pi\pi\pi$ p<0.001 Vs Hypertriglyceridemics

LIPID PROFILE OF NORMAL AND DYSLIPIDEMIC FEMALES

The assessment of lipid profile of normal females versus hyperlipidemics, hypercholesterolemics, and hypertriglyceridemics is represented in **table 37**. In comparison to the normals, the hyperlipidemics showed increments in all the lipid parameters, whereas, the hypercholesterolemics showed a rise ($p < 0.001$) in the levels of TC, HDL-C, LDL-C and Non HDL-C than the normals. On the other hand the hypertriglyceridemics had significantly higher levels of TG ($p < 0.001$), LDL-C ($p < 0.01$), VLDL-C ($p < 0.001$) and L/H ratio ($p < 0.01$) than the normals.

The assessment between hyperlipidemic and hypercholesterolemic subjects showed that the hyperlipidemics had significantly higher levels of TC ($p < 0.01$), TG ($p < 0.001$), VLDL-C ($p < 0.001$) and Non HDL-C ($p < 0.01$) in comparison to hypercholesterolemic subjects. The hyperlipidemic females also had significantly increased levels of TC ($p < 0.01$), LDL-C ($p < 0.01$), Non HDL-C ($p < 0.01$) and L/H ratio ($p < 0.05$) in comparison to hypertriglyceridemic subjects. A similar comparison between hypercholesterolemic and hypertriglyceridemic subjects showed that the hypercholesterolemic female subjects had significantly higher ($p < 0.001$) TC, LDL-C, Non HDL-C and L/H ratio than the hypertriglyceridemic subjects, whereas the hypertriglyceridemic subjects had significantly raised ($p < 0.001$) levels of TG and VLDL-C in comparison to hypercholesterolemic female subjects.

TABLE 37
LIPID PROFILE OF NORMAL AND DYSLIPIDEMIC FEMALES (mg/dl, Mean \pm SD)

VARIABLE	NORMALS	HYPERLIPIDEMICS	HYPER CHOLESTEROLEMICS	HYPER TRIGLYCERIDEMICS
N	76	11	51	4
TC	165.5 \pm 25.9	229.3 \pm 38.5 ^{*** ## qϕ}	208.4 \pm 17.5 ^{*** $\pi\pi\pi$}	157.0 \pm 11.2
TG	87.0 \pm 52.4	213.0 \pm 52.9 ^{*** ###}	93.0 \pm 25.9	197.5 \pm 23.5 ^{*** $\pi\pi\pi$}
HDL-C	33.4 \pm 6.7	42.6 \pm 8.4 ^{***}	40.0 \pm 6.8 ^{***}	38.3 \pm 8.3
LDL-C	114.6 \pm 22.5	114.0 \pm 35.6 ^{*** qϕ}	149.9 \pm 16.3 ^{*** $\pi\pi\pi$}	79.3 \pm 21.0 ^{**}
VLDL-C	17.4 \pm 10.5	42.6 \pm 10.6 ^{*** ###}	18.5 \pm 5.2	39.5 \pm 4.7 ^{*** $\pi\pi\pi$}
NON HDL-C	132.1 \pm 24.1	186.6 \pm 38.4 ^{*** ## qϕ}	168.4 \pm 16.1 ^{*** $\pi\pi\pi$}	118.8 \pm 18.9
TC/H	5.1 \pm 1.0	5.5 \pm 1.2	5.3 \pm 0.8	4.3 \pm 1.1 ^{π}
L/H	3.6 \pm 1.0	3.5 \pm 1.0 ^{ϕ}	3.9 \pm 0.8 ^{$\pi\pi\pi$}	2.2 \pm 0.9 ^{**}

All groups * p<0.05, ** p<0.01, ***p<0.001 Vs Normals
Hyperlipidemics # p<0.05, ## p<0.01, ### p<0.001 Vs Hypercholesterolemics
Hyperlipidemics ϕ p<0.05, $\phi\phi$ p<0.01 Vs Hypertriglyceridemics
Hypercholesterolemics π p<0.05, $\pi\pi\pi$ p<0.001 Vs Hypertriglyceridemics

LIPID PROFILE OF OVERWEIGHT AND OBESE AND DYSLIPIDEMIC MALES

The lipid profile of the overweight and obese males is compared with that of hyperlipidemic, hypercholesterolemic and hypertriglyceridemic male subjects and the same is shown in **table 38**. The hyperlipidemic males had raised ($p<0.01$) TC, TG, VLDL-C and Non HDL-C levels than the overweight and obese subjects. The hypercholesterolemics had significantly elevated level of ~~LDL-C~~ LDL-C ($p<0.05$) and the L/H ($p<0.001$) ratio in comparison to the overweight and obese subjects. In the case of hypertriglyceridemic subjects, TC ($p<0.01$), LDL-C ($p<0.001$) and Non HDL-C ($p<0.01$) along with the L/H ratio ($p<0.01$) were found to be significantly higher than the overweight or obese subjects.

LIPID PROFILE OF OVERWEIGHT AND OBESE AND DYSLIPIDEMIC FEMALES

The lipid levels of overweight and obese and dyslipidaemic females is depicted in **table 39**. The TC of the hyperlipidemic females was found to be 229.3 ± 38.5 mg/dl, which was significantly higher than that observed among overweight and obese subjects (195.6 ± 33.9 mg/dl). In addition to this, the TG ($p<0.001$), VLDL-C ($p<0.001$) and Non HDL-C ($p<0.01$) were also found to be significantly higher among the hyperlipidemic subjects. The hypercholesterolemic female subjects also had significantly raised TC, ~~LDL-C~~ LDL-C and Non HDL-C levels when compared with overweight and

TABLE 38

LIPID PROFILE OF OVERWEIGHT & OBESE AND DYSLIPIDEMIC MALES (mg/dl, Mean \pm SD)

VARIABLE	OVERWEIGHT & OBESE SUBJECTS	HYPERLIPIDEMICS	HYPER CHOLESTEROLEMICS	HYPER TRIGLYCERIDEMICS
n	126	61	73	7
TC	207.1 \pm 38.8 ^{ππ}	222.6 \pm 31.8 ^{**}	210.9 \pm 18.0	158.6 \pm 24.0
TG	154.8 \pm 83.7 ^{###}	191.6 \pm 45.4 ^{**}	107.9 \pm 27.6	188.7 \pm 23.9
HDL-C	39.0 \pm 8.3	40.9 \pm 6.4	38.3 \pm 6.4	34.4 \pm 7.7
LDL-C	137.2 \pm 30.9 ^{πππ}	143.4 \pm 27.8	151.0 \pm 17.4 ^{##}	86.4 \pm 29.0
VLDL-C	31.0 \pm 16.7 ^{###}	38.3 \pm 9.1 ^{**}	21.6 \pm 5.5	37.7 \pm 4.8
NON HDL-C	168.2 \pm 33.7 ^{ππ}	181.7 \pm 29.0 ^{**}	172.5 \pm 17.4	124.1 \pm 26.4
TC/H	5.4 \pm 0.8	5.5 \pm 0.8	5.6 \pm 1.0	4.8 \pm 1.1
L/H	3.6 \pm 0.8 ^{ππ}	3.5 \pm 0.7	4.1 \pm 0.9 ^{###}	2.7 \pm 1.0

Overweight & Obese Subjects ^{**} p<0.01 Vs Hyperlipidemics
 Overweight & Obese Subjects ^{##} p<0.01, ^{###} p<0.001 Vs Hypercholesterolemics
 Overweight & Obese Subjects ^{ππ} p<0.01, ^{πππ} p<0.001 Vs Hypertriglyceridemics

TABLE 39

LIPID PROFILE OF OVERWEIGHT & OBESE AND DYSLIPIDEMIC FEMALES (mg/dl, Mean \pm SD)

VARIABLE	OVERWEIGHT & OBESE SUBJECTS	HYPERLIPIDEMICS	HYPER CHOLESTEROLEMICS	HYPER TRIGLYCERIDEMICS
n	105	11	51	4
TC	195.6 \pm 33.9 ^{π}	229.3 \pm 38.5 ^{$\pi\pi$}	208.4 \pm 17.5 [#]	157.0 \pm 11.2
TG	127.2 \pm 51.6 ^{###}	213.0 \pm 52.9 ^{$\pi\pi\pi$}	93.0 \pm 25.9	197.5 \pm 23.5 ^{$\pi\pi$}
HDL-C	37.9 \pm 8.7	42.6 \pm 8.4	40.0 \pm 6.8	38.3 \pm 8.3
LDL-C	132.3 \pm 27.2 ^{$\pi\pi\pi$}	114.0 \pm 35.6	149.9 \pm 16.3 ^{###}	79.3 \pm 21.0
VLDL-C	25.4 \pm 10.3 ^{###}	42.6 \pm 10.6 ^{$\pi\pi\pi$}	18.5 \pm 5.2	39.5 \pm 4.7 ^{$\pi\pi$}
NON HDL-C	157.7 \pm 30.1 ^{π}	186.6 \pm 38.4 ^{$\pi\pi$}	168.4 \pm 16.1 [#]	118.8 \pm 18.9
TC/H	5.3 \pm 0.9 ^{π}	5.5 \pm 1.2	5.3 \pm 0.8	4.3 \pm 1.1
L/H	3.6 \pm 0.9 ^{$\pi\pi$}	3.5 \pm 1.0	3.9 \pm 0.8	2.2 \pm 0.9

Overweight & Obese Subjects ^{$\pi\pi$} p<0.01 Vs Hyperlipidemics

Overweight & Obese Subjects [#] p<0.05, ^{###} p<0.001 Vs Hypercholesterolemics

Overweight & Obese Subjects ^{π} p<0.05, ^{$\pi\pi$} p<0.01, ^{$\pi\pi\pi$} p<0.001 Vs Hypertriglyceridemics

obese subjects. A similar comparison with hypertriglyceridemics showed the TG and VLDL-C to be considerably higher in comparison to the overweight and obese subjects. However, the TC, Non HDL-C and TC/H and L/H and LDL-C showed an upward trend among overweight and obese subjects

LIPID PROFILE IN RELATION TO BODY MASS INDEX

The effect of body mass index on the lipid profile of the normal and overweight or obese subjects who were not suffering from any of the CDD is depicted in **table 40**. Aberrations in lipid parameters were observed with an increase in BMI. A significant increase in the levels of TC was noticed among the overweight (201.0 ± 37.8 mg/dl) and obese (205.6 ± 33.5 mg/dl) in comparison to the subjects having normal BMI (164.7 ± 23.2 mg/dl). Substantial increase in the Non HDL-C level was observed in the case of overweight (162.8 ± 33.2 mg/dl) and obese (166.0 ± 29.1 mg/dl) subjects as compared to subjects with a normal BMI (132.1 ± 32.2 mg/dl). Similarly the overweight and obese subjects had significantly higher concentrations of TG (141.2 ± 74.8 mg/dl and 146.6 ± 59.8 mg/dl Vs 97.5 ± 50.2 mg/dl), LDL-C (134.5 ± 30.2 mg/dl and 136.6 ± 25.5 mg/dl Vs 112.5 ± 20.2 mg/dl) and VLDL-C (28.2 ± 14.9 mg/dl and 29.3 ± 12.0 mg/dl Vs 19.5 ± 10.0 mg/dl) as compared to subjects with BMI ranging from 18-24.

TABLE 40

**LIPID PROFILE IN RELATION TO BODY MASS INDEX
(mg/dl, Mean \pm SD)**

	BODY MASS INDEX (BMI)			F Ratio	F Prob.	Signific ant Pairs
	18 - 24	25 - 29.99	≥ 30			
Groups	0	1	2			
n	164	187	44			
TC	164.7 \pm 23.2	201.0 \pm 37.8	205.6 \pm 33.5	64.9	0.000	(0,1) (0,2)
TG	97.5 \pm 50.2	141.2 \pm 74.8	146.6 \pm 59.8	23.6	0.000	(0,1) (0,2)
HDL-C	32.9 \pm 7.4	38.2 \pm 8.6	39.6 \pm 7.7	23.9	0.000	(0,1) (0,2)
LDL-C	112.5 \pm 20.2	134.5 \pm 30.2	136.6 \pm 25.5	35.8	0.000	(0,1) (0,2)
VLDL-C	19.5 \pm 10.0	28.2 \pm 14.9	29.3 \pm 12.0	23.6	0.000	(0,1) (0,2)
Non-HDL-C	132.1 \pm 32.2	162.8 \pm 33.2	166.0 \pm 29.1	59.1	0.000	(0,1) (0,2)
TC/H	5.2 \pm 1.2	5.4 \pm 0.9	5.3 \pm 0.8	0.8	0.455	
L/H	3.6 \pm 1.1	3.6 \pm 0.8	3.5 \pm 0.8	0.2	0.842	

LIPID PROFILE IN RELATION TO WAIST HIP RATIO

The assessment of lipid profile of the males in relation to WHR is given in **table 41**. Here, the subjects were categorized into low, moderate or high-risk group based on their WHR. The TC, TG, VLDL-C and Non HDL-C were found to be significantly higher among the subjects of high-risk group ($\text{WHR} > 1.0$) as compared to the subjects of low risk group ($\text{WHR} < 0.9$). In the case of females, subjects in the moderate ($\text{WHR} = 0.75-0.85$) and high-risk ($\text{WHR} > 0.85$) groups had higher levels of lipids in comparison to the low risk group ($\text{WHR} < 0.75$). However, the elevation observed was statistically non significant (**Table 42**).

LIPID PROFILE IN RELATION TO WAIST CIRCUMFERENCE

The analysis of variance and multiple range test was carried out in relation to the waist circumference in all the subjects (**Table 43 & 44**). On categorization, there was a significant increase in (TC, TG, VLDL-C and Non HDL-C) the lipid parameters of males belonging to moderate and high risk categories as compared to their counterparts in the low risk group (**Table 43**). The atherogenic indices, represented by TC/H and L/H are also on the upward trend in both moderate and high risk group but the statistical significance is noticed in the moderate risk group, as compared to the low risk group.

TABLE 41

LIPID PROFILE OF MALES IN RELATION TO WAIST HIP
RATIO (mg/dl, Mean \pm SD)

	WAIST HIP RATIO (WHR)			F - Ratio	F - Prob	Significant Pairs
	< 0.9 (Low Risk)	0.9 - 1.0 (Mod Risk)	> 1.0 (High Risk)			
Groups	0	1	2			
n	50	156	8			
TC	177.0 \pm 27.5	192.5 \pm 41.2	207.0 \pm 35.6	4.0	0.02	(0,1)
TG	105.4 \pm 54.5	143.2 \pm 78.9	158.3 \pm 47.0	5.5	0.00	(0,1)
HDL-C	36.3 \pm 8.7	36.2 \pm 8.9	41.6 \pm 10.9	1.7	0.19	
LDL-C	120.3 \pm 18.1	127.9 \pm 32.2	133.7 \pm 23.6	1.5	0.22	
VLDL-C	21.1 \pm 10.9	28.6 \pm 15.8	31.7 \pm 9.4	5.5	0.00	(0,1)
Non- HDL-C	141.4 \pm 23.1	156.5 \pm 35.6	165.4 \pm 27.0	4.6	0.01	(0,1)
TC/H	5.2 \pm 0.1	5.5 \pm 1.2	5.1 \pm 0.8	2.9	0.06	
L/H	3.5 \pm 0.8	3.7 \pm 1.1	3.3 \pm 0.7	1.1	0.32	

TABLE 42

**LIPID PROFILE OF FEMALES IN RELATION TO WAIST HIP
RATIO (mg/dl, Mean \pm SD)**

	WAIST HIP RATIO (WHR)			F - Ratio	F - Prob.	Significant Pairs
	< 0.75 (Low Risk)	0.75 - 0.85 (Mod. Risk)	> 0.85 (High Risk)			
Group	0	1	2			
n	3	71	107			
TC	162.7 \pm 27.0	184.2 \pm 31.3	182.7 \pm 36.2	0.6	0.56	
TG	97.3 \pm 4.0	105.3 \pm 58.1	114.0 \pm 54.4	0.6	0.55	
HDL-C	31.3 \pm 3.1	36.1 \pm 8.9	36.0 \pm 7.8	0.5	0.61	
LDL-C	111.9 \pm 28.8	127.0 \pm 26.3	123.8 \pm 27.1	0.7	0.52	
VLDL-C	19.5 \pm 0.8	21.1 \pm 11.6	22.8 \pm 10.9	0.6	0.56	
Non- HDL-C	131.3 \pm 28.2	148.1 \pm 28.9	146.6 \pm 31.6	0.5	0.64	
TC/H	5.2 \pm 1.1	5.3 \pm 1.1	5.2 \pm 0.9	0.4	0.70	
L/H	3.6 \pm 1.1	3.7 \pm 1.1	3.5 \pm 0.8	0.8	0.46	

TABLE 43

**LIPID PROFILE OF MALES IN RELATION
TO WAIST CIRCUMFERENCE (mg/dl, Mean \pm SD)**

	WAIST CIRCUMFERENCE (cm)			F- Ratio	F- Prob.	Significant Pairs
	<94 (Low Risk)	94 – 102 (Mod. Risk)	> 102 (High Risk)			
Groups	0	1	2			
n	112	84	18			
TC	175.8 \pm 31.5	203.1 \pm 41.3	210.1 \pm 38.1	16.9	0.00	(0,1) (0,2)
TG	112.8 \pm 50.4	157.2 \pm 94.2	168.9 \pm 53.6	11.6	0.00	(0,1) (0,2)
HDL-C	35.0 \pm 7.4	36.9 \pm 9.8	41.2 \pm 9.6	4.4	0.13	(0,2)
LDL-C	118.6 \pm 24.1	134.8 \pm 33.8	135.1 \pm 24.8	8.8	0.00	(0,1)
VLDL-C	22.6 \pm 10.1	31.5 \pm 18.8	33.8 \pm 10.7	11.6	0.00	(0,1) (0,2)
Non- HDL-C	141.1 \pm 27.5	166.2 \pm 34.1	168.0 \pm 30.6	18.3	0.00	(0,1) (0,2)
TC/H	5.2 \pm 0.9	5.2 \pm 1.3	5.7 \pm 0.7	7.0	0.01	(0,1)
L/H	3.4 \pm 0.8	3.5 \pm 1.2	3.9 \pm 0.6	3.8	0.02	(0,1)

In case of female subjects there was significant increase in TC, LDL-C and Non HDL-C in both moderate and high risk groups as compared to low risk group (**Table 44**). The high risk group also exhibited an elevated level of TG and VLDL-C as compared to the low risk group. With respect to TC, TG and VLDL-C, the rise was more prominent in the high risk group as compared to moderate risk group.

LIPID PROFILE OF OVERWEIGHT AND OBESE SUBJECTS HAVING DYSLIPIDEMIA

The analysis of the lipid profile among the overweight and obese subjects having some kind of dyslipidaemia were compared with those overweight or obese who were normolipidaemic (**Table 45**) portrayed that there were more number of dyslipidaemic (having either TG, TC or both the TG and TC raised) subjects in comparison to the normolipidemic subjects. The analysis of lipid profile amongst these two groups showed that the levels of TC, TG, LDL-C, VLDL-C and Non HDL-C were found to be significantly raised ($p<0.001$) among the dyslipidemic group. The ratio of TC/H was also found to be significantly raised ($p<0.01$) among the dyslipidemics.

TABLE 44

**LIPID PROFILE OF FEMALES IN RELATION
TO WAIST CIRCUMFERENCE (mg/dl, Mean \pm SD)**

	WAIST CIRCUMFERENCE (cm)			F- Ratio	F- Prob.	Significant Pairs
	< 80 (Low Risk)	80 – 88 (Mod. Risk)	> 88 (High Risk)			
Groups	0	1	2			
n	37	67	77			
TC	160.1 \pm 19.8	181.2 \pm 31.9	195.4 \pm 35.8	15.7	0.00	(0,1) (0,2) (1,2)
TG	83.5 \pm 32.7	103.6 \pm 59.5	129.0 \pm 54.4	10.2	0.00	(0,2) (1,2)
HDL-C	33.4 \pm 8.0	35.1 \pm 8.1	38.0 \pm 8.0	4.8	0.09	(0,2)
LDL-C	110.0 \pm 22.1	125.2 \pm 25.6	131.6 \pm 27.2	8.7	0.00	(0,1) (0,2)
VLDL-C	16.7 \pm 6.5	20.7 \pm 11.9	25.8 \pm 10.9	10.1	0.00	(0,2) (1,2)
Non- HDL-C	126.7 \pm 19.1	146.1 \pm 29.0	157.4 \pm 31.3	14.7	0.00	(0,1) (0,2)
TC/H	5.0 \pm 1.1	5.3 \pm 1.0	5.2 \pm 0.9	1.2	0.32	
L/H	3.5 \pm 1.1	3.7 \pm 0.9	3.6 \pm 0.8	0.7	0.52	



TABLE 45

LIPID PROFILE OF OVERWEIGHT & OBESE SUBJECTS
HAVING DYSLIPIDEMIA (mg/dl, Mean \pm SD)

	NORMOLIPIDEMICS	DYSLIPIDEMICS
n	59	172
FBS	86.4 \pm 10.7	87.9 \pm 14.8
TC	160.3 \pm 12.5	216.1 \pm 31.5 ***
TG	94.1 \pm 30.0	158.8 \pm 74.9 ***
HDL-C	32.3 \pm 6.4	40.60 \pm 8.1 ***
LDL-C	109.2 \pm 11.6	143.8 \pm 28.4 ***
VLDL-C	18.8 \pm 6.0	31.2 \pm 13.3 ***
NON HDL-C	128.0 \pm 11.0	175.5 \pm 28.2 ***
TC/H	5.1 \pm 0.8	5.4 \pm 0.9 **
L/H	3.5 \pm 0.7	3.7 \pm 0.9

p<0.01, *p<0.001 Significantly raised in comparison to normolipidemics

DISCUSSION

Aberrations in the lipid profile of the subjects suffering from various chronic degenerative diseases is well documented, however, the variation in the etiology of these dyslipidemias among different ethnic groups remains under investigation. Hence, in the present section the lipid profile of the subjects suffering from various chronic degenerative diseases were assessed.

Atherogenic dyslipidaemia was found to be present in subjects suffering from various CDD such as overweight & obesity, diabetes, hypertension and CHD. The overweight and obese subjects who were clinically normal but had abnormal BMI ($BMI \geq 25$) were found to have detrimental levels of atherogenic lipids viz. TC, TG, LDL-C, Non HDL-C, VLDL-C etc (Table 30,31,32). Numerous studies have shown that a BMI of above 25 increases the risk of early death, mainly from heart disease or cancer, and that a BMI of 30 dramatically increases the chances (Pi-Sunyer 1993, Higgins et al 1988).

Among the obese subjects, the highly lipolytic nature of adipocytes in general triggers a cascade of metabolic abnormalities resulting in dyslipidaemia (Rippe et al 1998). Obese subjects on an average have higher TC, TG, blood glucose and plasma insulin levels than lean persons (Jousilathi et al 1996). Similar results have been observed in the present study and also by those carried out in the department (Mani & Tiwari, 2002, Mani and Khan 2002).

Type 2 diabetes mellitus is characterized by four major metabolic abnormalities viz obesity, impaired insulin action, insulin secretory dysfunction and increased endogenous glucose output. Hyperglycemia is always the consequence of these abnormalities (Weyer et al 1999). The pathophysiological basis for hyperglycemia is the reduced insulin mediated uptake of glucose from muscle, exaggerated glucose production from liver and increased free fatty acid mobilization from adipose tissue. The result initially is post-prandial hyperglycemia, which later is followed by fasting hyperglycemia (Lebovitz 1999). The observations from the present study were in accordance with the literature and revealed that the fasting blood glucose of the diabetics was significantly higher in comparison to the other groups (Table 30,31,32). Furthermore, hyperglycemia and insulin resistance affect each lipid and lipoprotein fraction in patients with diabetes. Approximately 30 % of diabetic patients suffer from lipid abnormalities and these are presumed to be responsible for the increased risk of complication in patients with diabetes mellitus (Wolffenbuttael and Drzewoski 1999). Atherogenic dyslipidemia amongst diabetics was observed in present study.

Research has shown that two abnormalities characterize lipoprotein metabolism in type 2 patients: fasting and post prandial concentrations of TG-rich lipoproteins, especially VLDL-C are higher in the diabetic group (Mani et al 1986, Kannel 1985, Taskinen 1990, Howard 1987). Although many mechanisms contribute to hypertriglyceridemia in type 2 diabetes, insulin resistance seems to be the common basis. The insulin resistant state impairs

the normal suppression of fatty-acid release from adipose tissue in post prandial state (Syvanne and Taskinen 1997). Consequently the flux of FFA to the liver increases and over production of VLDL-C from these substrates occur when hyperinsulinaemia is present. The relationship between hypertriglyceridemia and abnormalities in LDL-C particle size and composition has also been extensively investigated in diabetes (Grey et al 1997, Lahdenpera et al 1996) The compositional abnormalities that occur in LDL-C in type 2 diabetes may account for some of the increased risk of atherosclerosis in the population. Diabetic dyslipidemia characterized by elevated levels of TG, VLDL-C, TC, Non HDL-C and LDL-C was observed among the subjects of the present study as well.

Another CDD where hyperlipidemia has been observed is hypertension. Various studies including the Framingham (Castelli and Anderson 1984) as well as the Multiple Risk Intervention Trial (NIH Report 1997) have shown that TC levels are high in hypertensive subjects. Various Indian studies have also shown that TC and LDL-C levels were higher amongst hypertensive subjects in comparison to normotensive subjects (Joglekar and Nanivadekar, 1996, Latheef et al 1998, Mani and Gujarathi 2002). In the current study the hypertensive subjects had significantly higher levels of atherogenic lipids in comparison to the normals (Table 30,31). As the number of females with hypertension is too small (n=6) no meaningful conclusion can be drawn.

The possibility of the subjects taking anti hypertensive drugs, which in turn

might have resulted in lowering the lipids is also evident when the comparison is made between the overweight and obese and hypertensive subjects irrespective of the gender difference. No significant differences are observed among the TC, TG etc. (Table 30) However, the LDL-C and TC/H and L/H are significantly higher among the overweight and obese subjects, where as among the female subjects TC, LDL-C, Non HDL-C, as well as the ratio of L/H is found to be higher among the overweight or obese subjects in comparison to the hypertensive subjects (Table 32) indicating the use of hypertensive drugs.

Dyslipidemia was also noticed with respect to TC, TG and VLDL-C of CHD subjects in comparison to normals (Table 30) A significant relationship has been demonstrated between hypercholesterolemia and CAD in the western world (Kannel and Schatzkin 1983, Kannel and Thomas 1982). However, Indian studies have shown that majority of the people with myocardial infarction have plasma cholesterol levels less than 200 mg/dl (Krishnaswami et al 1970, Chadhuri et al 1966, Bhatnagar et al 1995). This indicates that among the Indian subjects the occurrence of CHD is at a lower level of TC than that observed among the Western world. The results of the present study are in accordance with the literature that revealed the occurrence of CHD at lower levels of serum total cholesterol. In addition to cholesterol, recent studies also show that hypertriglyceridemia is an important cardiac risk factor (Assman et al 1996). One of the direct effects of hypertriglyceridemia is an increase in large VLDL-C particles enriched with Apo E. This high content of

Apo E enhances their uptake by macrophages to produce foam cells. Hence, these large VLDL particles are atherogenic.

Another effect of increase in TG is the induction of increased, small dense LDL-C particles. These are considered more atherogenic than normal LDL-C because they readily filter into the arterial walls (Assman et al 1996). The Framingham heart study also states TG to be an independent marker of CHD (Castelli 1986). Elevated plasma triglyceride levels increase the transfer of cholesterol esters from HDL-C particles to TG-rich lipoprotein, an action resulting in lowering HDL-C concentration (Manocha and Srivastava 2002). However, once triglycerides rise beyond a certain level, they do not further cause a transfer of cholesterol esters from HDL-C. Hence the inverse correlation between triglycerides and HDL-C is strong only up to a distinct elevated range beyond which any further rise in triglycerides is not associated with a further fall in HDL-C (Lechleitner et al 1990, Tato et al 1997). The results of this study are in line with the literature reviewed which reveals that in comparison to normals, the HDL-C were found to be elevated among the subjects suffering from chronic degenerative diseases. But, the cardio protective effect of this increased HDL-C cannot be determined unless we find out which fraction of HDL-C is raised because it is reported that increased levels of HDL₂ are partially responsible for the cardio protective action of HDL-C (James and Pometta 1990). Thus, HDL-C needs to be fractionated to understand the exact composition. In addition to this, the Non HDL-C and LDL-C levels were also found to be high, so the rise in HDL-C proves to be

insignificant

Research has shown that Non HDL-C is considered to be a better indicator than LDL-C as it reflects a combined risk of all lipoprotein changes observed in various CDD (Havel and Rapaport 1995, Frost and Havel 1998, Garg and Grundy 1990) In the present study the Non HDL-C and LDL-C were found to be significantly raised in the overweight and obese, diabetic and hypertensive subjects (Table 30,31,32) In case of CHD subjects it was observed that the LDL-C was not significantly increased when compared with normal subjects, however, the Non HDL-C was found to be significantly higher, thus suggesting that the Non HDL-C is a better indicator than LDL-C A recent study conducted in a cohort containing both diabetic and non diabetic individuals showed that Non HDL-C was a somewhat better indicator of CVD than LDL-C (Cui et al 2001). This is in line with the results obtained in the current study as half of the subjects suffering from CVD were diabetic in the population. The National Cholesterol Education Programme (NCEP) has also recommended using Non HDL-C in assessing CVD risk in patients with diabetic (Executive Summary of the NCEP 2001).

In addition to the comparisons made between the normals and subjects suffering from various CDD, the study also assessed the FBS and lipid profile between various chronic degenerative disease groups. One such comparison was made between the overweight and obese, diabetic, hypertensive and CHD subjects in the study population (Table 30) and among the male (Table

31) and female subjects (Table 32) The comparison of FBS and lipid profile among the overweight and diabetic subjects (Table 30) showed that the diabetics had had significantly higher levels of FBS, which is the hallmark of diabetes mellitus The diabetic subjects also had significantly higher levels of TG, VLDL-C and Non HDL-C respectively. This is supported by research over the years which has shown that hyperlipidemia is a common sign of diabetes, and recent research points out that hypetriglyceridenmia is more prominent among diabetic subjects (Kannel 1985, Taskinen 1990), which has been discussed earlier in the chapter

The relationship between obesity and type 2 diabetes is well established. Numerous studies have shown that overweight and obese individuals are at an increased risk of developing this disease (Lew and Finkel 1979, Albu and Pi-Sunyer 1993, Jung 1997). It is said that obesity leads to insulin resistance and to co-morbidities related to the insulin resistance syndrome In fact, evidence shows that BMI is directly and continuously related to the risk of type 2 diabetes (Colditz et al 1990). The initiating event is probably the positive energy balance, which eventually leads to increased fat mass. As fat mass increases and as fat cell size increases, the regulation of FFA metabolism becomes abnormal. This change leads to an increase in FFA production The increased FFA competes with glucose for oxygen in insulin sensitive tissues and stimulates endogenous glucose production, causing insulin resistance. Insulin resistance, in turn, results in increased secretion of insulin from the pancreas Hyperinsulinemia contributes to hypertension and can lead to type

2 diabetes because of the increased load on the beta cells to produce insulin. Thus, indicating that obesity and type 2 diabetes are both states of insulin resistance. In the present study this plausible mechanism might be true because out of the total 82 diabetics 44 % were found to be overweight or obese. This result implies that overweight and obesity among the subjects might have been one of the causes to take the subjects into true diabetic state.

Obesity also carries a risk for the development of hypertension. Both systolic and diastolic blood pressure increase with an increase in BMI, and obese individuals are at a higher risk of developing hypertension than their lean counterparts (Stalmer et al 1989). Data from NHANES II, report the prevalence of hypertension, to be 2.9 times higher among overweight subjects. A study carried out by Kodali (1997) found that hypertensives had significantly higher body weight, body fat, BMI and WHR as compared to controls in both men and women. In the present study 54 % of the hypertensives were overweight or obese.

When the overweight and obese subjects were compared with CHD subjects the FBS levels of the CHD subjects to be significantly higher (Table 30,31). This was due to the fact that half of the subjects suffering from CHD were diabetics. Majority of the CHD subjects were either diabetics, hypertensives or had more than one co-morbidity together. The lipid profile of these subjects showed significantly raised TG and VLDL-C levels in comparison to

overweight or obese (Table 30,31) There is a strong link between obesity and CVD (Stamler 1993), and a similar link between diabetes and CVD (Carey et al 1997) A further addition to the plausible mechanism explained earlier linking obesity to insulin resistance states that insulin resistance is also accompanied by a pro-coagulant state that predisposes to atherosclerosis (Goldberg 2001) This suggests that the causal relationship between obesity and insulin resistance, and the insulin resistant states and CVD is likely multifactorial Overall, numerous processes work together to help explain the strong link between obesity, insulin resistance and CVD This further strengthens the fact that obesity is the underlying causative factor for the development and progression of other CDD such as diabetes, hypertension and CVD.

Keeping this in mind the analysis amongst diseased subjects who were overweight and obese was also carried out so as to study the risk of weight gain on the lipid profile of these subjects. The normal subjects had lower FBS as well as lower lipid profile in comparison to other groups Similarly, as one moves from the normal to diseased state, atherogenic lipid parameters tend to increase. This is also evident from the significantly high lipid parameters (TG, VLDL-C and Non HDL-C) observed among diabetic subjects who were overweight and obese in comparison to overweight and obese subjects who were clinically normal (Table 33). This indicates that the atherogenic lipids show more detrimental changes among those who are carrying double burden of obesity and diabetes

The most common forms of primary hyperlipidemia result from the interaction of multiple genes with diet, body weight, exercise habits and endocrine factors, and are often termed multi-factorial hyperlipidemias. As depicted in Table 36 and 37, the male and female subjects of the hyperlipidemic group had significantly higher levels of atherogenic lipids. This observation is similar to various studies, which have shown that hyperlipidemia as a factor for CVD (Syvanne and Taskinen 1997, Kannel 1985).

When the overweight and obese subjects were compared with dyslipidemics (Table 38,39), it was noticed that the atherogenic LDL-C was found to be significantly higher among the hypercholesterolemic subjects, thus indicating that the subjects are at a higher risk than the overweight and obese male and female subjects though they are clinically normal subjects. Similarly, the TC, TG, VLDL-C and Non HDL-C were found to be significantly higher amongst the hyperlipidemic subjects in comparison to the overweight and obese males and females (Table 38,39). The ratios of TC/H and L/H also showed an increasing trend among the hyperlipidemic and hypercholesterolemic subjects. The Framingham data suggests that hypercholesterolemia precedes the onset of CHD, and the association is generally strong (Kannel and Schatzkin 1983). The ratio of TC/H is about as efficient as any other lipid profile in estimating the risk of CHD. A ratio of 5 indicates the average high risk in affluent industrialized Western populations (Kannel and Schatzkin 1983).

Obesity as mentioned earlier is associated with detrimental changes in the

lipid profile BMI, a tool to measure the degree of obesity, is found to bring about alteration in the lipid profile As BMI increases TG and LDL-C levels increase, while HDL-C levels decrease (Austin and Selby 1995) The comparison of lipid profile amongst overweight and obese with other diseased population who were either overweight or obese has already been discussed. The lipid profile of normal and overweight and obese subjects (who were clinically normal) was analysed in order to study the changes in lipid levels with changes in BMI As one moves from a lower to higher BMI, there is a significant rise in the atherogenic lipid levels (Table 40) This once again emphasizes the fact that overweight and obesity carry a risk for the occurrence of atherogenic dyslipidaemia, which in turn is a risk factor for CVD

It is not only the amount of weight that one is carrying which is important, but the pattern of fat distribution is also important Abdominal or android fat distribution in the central or upper body has been found in both cross-sectional and longitudinal studies to be significantly more risky for health than gluteal femoral or lower body or gynoid obesity (Higgins et al 1998, Garrison and Kannel 1993, Despres et al 2001, Bray et al 1992, Lemieux 1996).

The tools commonly used for measuring abdominal fat distribution are waist hip ratio (WHR) and waist circumference (WC) The analysis of atherogenic lipid parameters with respect to WHR showed the TC, TG, VLDL-C and Non HDL-C levels to be significantly raised among the males having higher WHR (Table 41) However, the lipids did not show significant rise with an increase

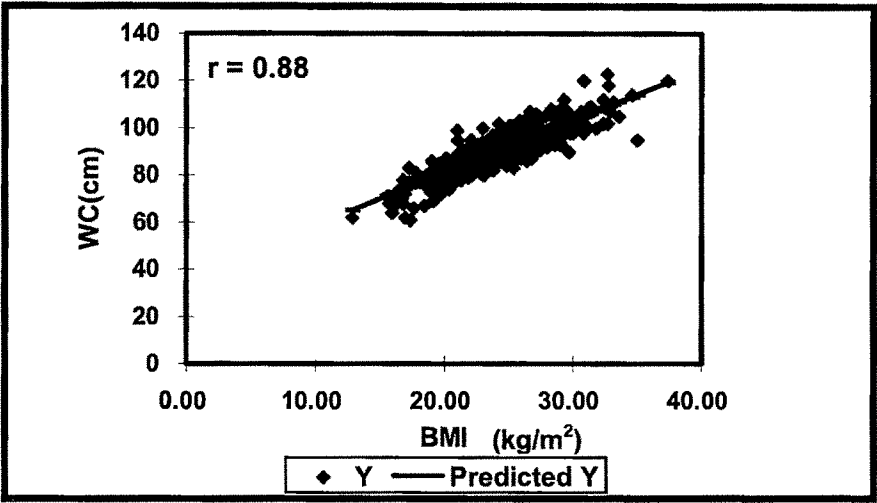
in the WHR of the females (Table 42) Significant rise in the atherogenic lipids were observed among the male and female subjects having a higher waist circumference Thus it can be concluded that WC has more prominent effect on the lipid parameters in comparison to the WHR These results are in accordance with the WHO (1998) and the National Heart Lung and Blood

Institute (NHLBI) recommendations, suggesting the use of BMI and WC for classifying abnormalities in body weight and body fat distribution

In order to find out whether WHR or WC is a better anthropometric indicator, the linear regression was carried out amongst male and female subjects irrespective of the presence/absence of CDD. The correlation coefficient 'r' was 0.88 and 0.77 among male and female subjects when BMI and WC were compared (**Figure 16a**) The correlation coefficient 'r' was 0.47 and 0.02 among the male and female subjects when BMI and WHR were considered (**Figure 16b**) This indicates that in the present study BMI correlates well with WC rather than with WHR. It emphasizes that BMI and WC are good indicators of body fat distribution rather than BMI and WHR. As mentioned above males showed a correlation of 0.47 when BMI and WHR were considered whereas among female subjects it was found to be 0.02. And on the other hand among the female subjects high degree of correlation between BMI and WC was seen This might be because among the Indian women, the hip size is usually large, and along with it a fairly large waist may not produce

FIGURE 16 a
CORRELATION BETWEEN BODY MASS INDEX
AND WAIST CIRCUMFERENCE

MALES



FEMALES

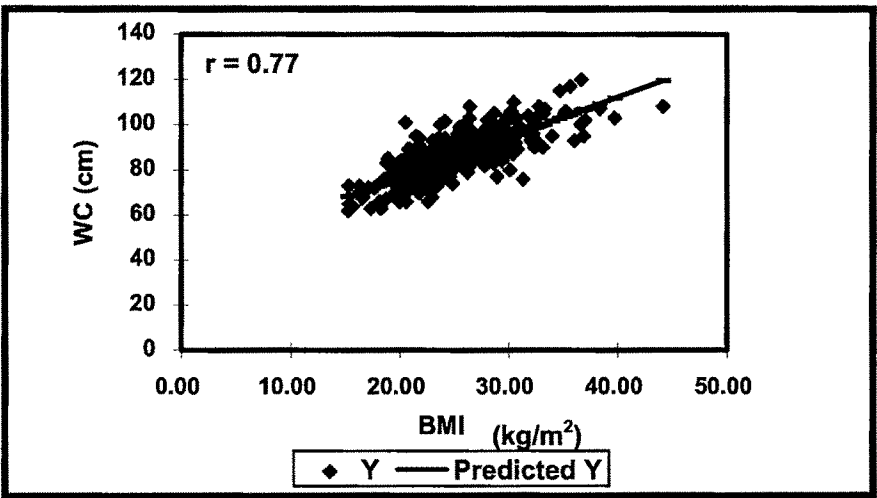
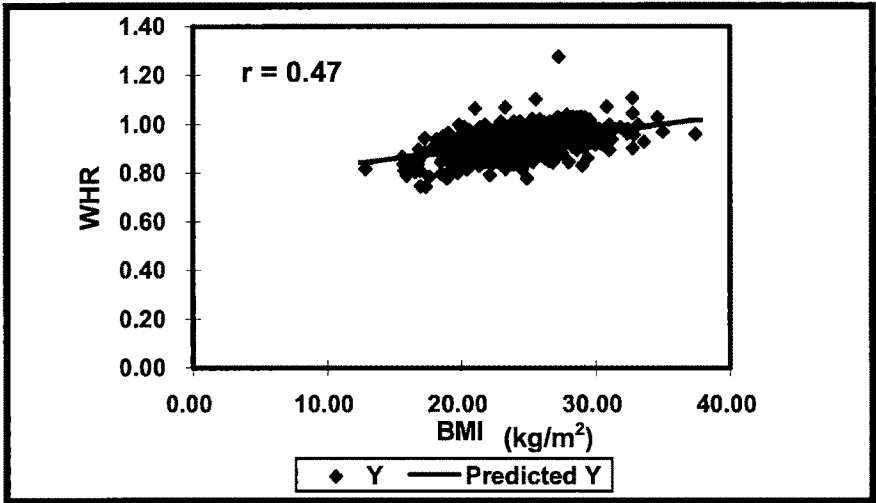


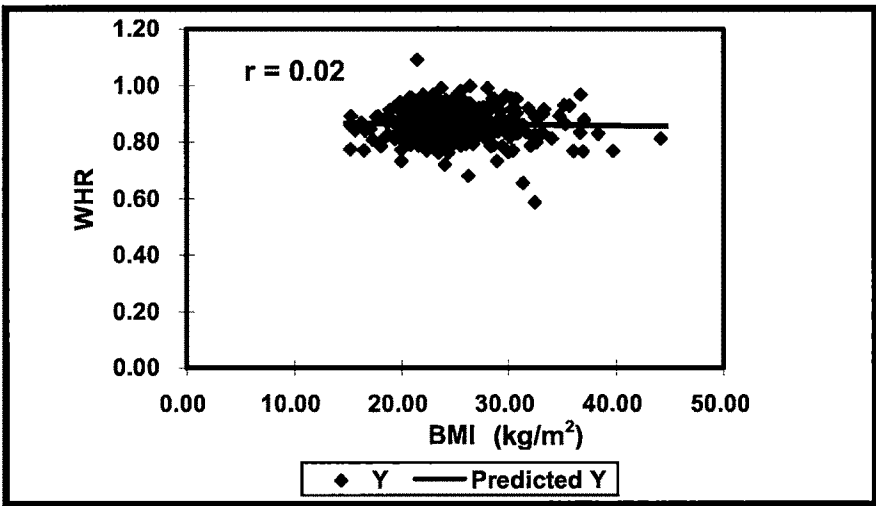
FIGURE 16 b

CORRELATION BETWEEN BODY MASS INDEX
AND WAIST HIP RATIO

MALES



FEMALES



an abnormal WHR. Hence, absolute waist measurement instead of WHR is considered a good parameter for the body fatness. Katzmarzyk et al in 1999 also advocated WC to be a good indicator of body fatness as WHR ratio may have a fallacy especially among Indian women. Taylor et al 1999 showed BMI and WC to provide simple yet sensitive methods for the estimation of total and central adiposity. Hence the use of both the methods, in combination help in establishing the degree of overweight and obesity.

The analysis among the overweight or obese when carried out by segregating them based on their lipid levels as normolipidemics and dyslipidemics (having TG, TC or both TG and TC raised) showed that the normolipidemics had significantly lower lipid values in comparison to dyslipidemics. It was also noticed that the numbers of normolipidemic overweight or obese were very few in comparison to dyslipidemics. This shows that as BMI increases detrimental changes are observed in the lipid profile. The overweight or obese normolipidemic subjects showed similar pattern of lipid profile as normal subjects (BMI <25). This was because the overweight and obese normolipidemics had a BMI much lower than the hyperlipidemic overweight and obese. This emphasizes the fact that as BMI increases the abnormalities in the lipid levels increase.

In addition to the comparison of lipid profile between various groups with respect to their disease profile, the analysis of relative risk of the subjects (Normals Vs Diseased) in relation to the lipid profile was also done so as to

study the risk of various atherogenic lipid parameters. As seen in **table 46** the TC was found to be highly dependable risk factor in comparison to other lipid parameters. It was observed that with respect to TC, the obese subjects were 4.2 times more prone to clinical complications than the normal subjects. With regard to TG, Non HDL-C and LDL-C the obese subjects were 3.4, 1.8 and 2.2 times at a higher risk of developing complications.

In comparison to normal subjects the diabetics were 4.4 times more prone to have aberration in TG. This observation is in line with data which shows that in type 2 diabetes, the abnormality that characterizes lipoprotein metabolism is the fasting and post prandial triglyceride rich lipoproteins (Kannel 1985, Taskinen 1999, Howard 1987 and Syvanne et al 1994). Among the diabetic subjects the relative risk for TC and LDL-C was found to be 4.2 and 2.0 respectively.

Similarly, the hypertensive subjects were three times at a higher risk to develop aberrations in TC (RR=3.4) and TG (RR=3.0) levels. Similar observations were noticed for the subjects suffering from CHD. These results indicate that the subjects suffering from any kind of CDD are at a higher risk to develop aberrations in atherogenic lipids in comparison to the normal subjects who are not suffering from any kind of CDD.

In addition to the relative risk of the subjects with respect to various lipid parameters the correlation analysis was done taking into consideration the biochemical, anthropometric and general habits of the subjects because it

TABLE 46

**RELATIVE RISK IN RELATION TO ATHEROGENIC LIPID
PARAMETERS AMONG SUBJECTS SUFFERING FROM
VARIOUS CHRONIC DEGENERATIVE DISEASES**

	RELATIVE RISK			
	TC	TG	NON HDL-C	LDL-C
OVERWEIGHT SUBJECTS	3.8	2.9	1.6	1.9
OBESE SUBJECTS	4.2	3.4	1.8	2.2
DIABETICS	4.2	4.4	1.7	2.0
HYPERTENSIVES	3.4	3.0	1.5	1.6
CHD SUBJECTS	3.6	3.3	1.6	1.9

is known that no single risk factor leads to the development of CDD, but it is always a combination of various risk factors. The correlation analysis when carried out for the entire population (i.e. irrespective of the clinical profile of the subjects) showed that the TC levels correlated positively with age and the habit of tobacco chewing at $p < 0.01$ level (Table 47). The correlation coefficient 'r' for TC was found to be highly significant with alcohol consumption. An association was also observed among various lipid parameters viz. between TC and TG, LDL-C, Non HDL-C etc, thus implying that as one level increased, the others were also affected. The TG, LDL-C and Non HDL-C showed positive association with alcohol consumption whereas LDL-C and Non HDL-C showed correlation with tobacco consumption. The anthropometric parameters such as WHR and WC strongly correlated with age whereas WC correlated positively with BMI and WHR.

When the correlation was done among normal subjects, BMI and WHR showed correlation with WC. The correlation coefficient 'r' was found to be 0.8 when BMI and WC were taken into consideration and the 'r' value was 0.6 between WHR and WC (Table 48). However, no correlation was observed between BMI and WHR. This suggests that BMI and WC are better indicators in this population as a measure of overweight and obesity.

Among the overweight or obese population, tobacco chewing habit of the subjects found correlation with various lipid parameters such as TC, LDL-C and Non HDL-C (Table 49). It indicates that tobacco chewing does bring about alteration in the lipid levels. Similarly the lipid parameters among

TABLE 47

CORRELATION BETWEEN VARIOUS RISK FACTORS

(ENTIRE POPULATION)

VARIABLE	TC	TG	HDL-C	LDL-C	NON HDL-C	WHR	WAIST
AGE	0 3 *				0 3 *	0.4 **	0 4 **
BMI							0 8 **
WHR							0 6 **
ALCOHOL	0.4 **	0 3 *		0 3 *	0.4 **		
TOBACCO	0.3 *			0 3*	0 3 *		
TG	0 3 *		0 3 *				
HDL-C	0 5 **	0 3 *		0 3*	0 4 **		
LDL-C	0 9 **		0 3 *		0.9**		
NON HDL-C	1 0 **	0 3 *	0 4 **				

1 TAILED SIGNIFICANCE * Significant at 0 01, ** Significant at 0 001

TABLE 48

CORRELATION ANALYSIS BETWEEN VARIOUS RISK

FACTORS (NORMAL POPULATION)

VARIABLE	TC	NON HDL-C	BMI	WHR
WAIST CIRCUM.			0 8 **	0 6 *
LDL-C	0 9 **	0 9 **		
NON HDL-C	0.9 **			

1 TAILED SIGNIFICANCE. * Significant at 0.01, ** Significant at 0.001

TABLE 49

CORRELATION ANALYSIS BETWEEN VARIOUS RISK
FACTORS (OVERWEIGHT & OBESE POPULATION)

VARIABLE	TC	HDL-C	LDL-C	NON HDL-C
TOBACCO	0.7 *		0.8 **	0.7 *
HDL-C	0.8 **			0.6 *
LDL-C	0.9 **			0.9 **
NON HDL-C	1.0 **	0.6 *	0.9 **	

1 TAILED SIGNIFICANCE * Significant at 0.01, ** Significant at 0.001

themselves showed correlation viz TC with HDL-C, LDL-C, Non HDL-C etc , suggesting that a change in one of the levels influences the other values Thus, the lipid parameters are interrelated with each other.

The TC of the diabetic population showed correlation with HDL-C, LDL-C and Non HDL-C. Non HDL-C correlated well with LDL-C ($r=0.9$, $p<0.001$) (Table 50). The correlation coefficient 'r' was found to be 0.7 ($p<0.01$) between BMI and age This leads to a conclusion that as the age advances, the chance of one becoming obese increases This may be due to reduction in the physical activity without any change in caloric intake The correlation coefficient 'r' was found to be 0.7 ($p<0.01$) between BMI and WHR, where as it was 0.8 ($p<0.001$) when BMI and WC were compared, indicating stronger relationship between BMI and WC

Among the hypertensive subjects TG and age showed significance at $p<0.01$ level (Table 51). Among the hypertensive subjects a good correlation was observed between BMI and WC and a poor correlation was noticed with BMI and WHR

From all the correlations studied, one single fact that emerges very strongly is that BMI correlates well with WC and poor correlation is noticed with WHR. Thus, BMI and WC are better anthropometric indicators for the measurement of overweight and obesity

TABLE 50

CORRELATION ANALYSIS BETWEEN VARIOUS RISK FACTORS (DIABETIC POPULATION)

VARIABLE	TC	NON HDL-C	BMI	WHR
AGE			0.7 *	0.8 *
BMI				0.7 *
WAIST			0.8 **	
HDL-C	0.7 *			
LDL-C	0.9 **	0.9 **		
NON HDL-C	1.0 *			

1 TAILED SIGNIFICANCE * Significant at 0.01, ** Significant at 0.001

TABLE 51

CORRELATION ANALYSIS BETWEEN VARIOUS RISK FACTORS (HYPERTENSIVE POPULATION)

VARIABLE	TC	TG	NON HDL-C	BMI	WHR
AGE		0.7 *			
WAIST				0.8 *	0.8 **
SMOKING					0.9 **
LDL-C	0.9 **		0.9 **		
NON HDL-C	1.0 **				

1 TAILED SIGNIFICANCE: * Significant at 0.01, ** Significant at 0.001

The results of this section indicate that as one moves from a lower BMI to higher BMI, the risk of suffering from various CDD increases and that obesity is emerging as the root cause of all the complications. Thus, it is aptly said that obesity is the mother of all CDD. The study also showed that BMI and WC are the two anthropometric indices, which show high correlation. The subjects suffering from various CDD showed aberrations in lipid profile, thus proving that atherogenic dyslipidemia is the hallmark of all these CDD.

At a glance

❧ Aberrations in the atherogenic lipid parameters of the overweight or obese, dyslipidemics, diabetics, hypertensives & CHD subjects when were noticed.

❧ Obese subjects on an average had higher total cholesterol, triglycerides, and other atherogenic lipids than lean persons. This might have been due to the highly lipolytic nature of adiposities, which in general triggers a cascade of metabolic abnormalities resulting in dyslipidaemia. This has also been observed in studies carried out in the department (Mani and Tiwari 2002, Mani and Khan 2002).

❧ The insulin resistant state seems to be the plausible cause of dyslipidemia noticed in diabetic population of the industrial set up. Furthermore, the compositional abnormalities that occur in LDL-C in type 2 diabetes may account for some of the increased risk of atherosclerosis in the population.

❧ Non HDL-C that reflects a combined risk of all lipoproteins, was found to be significantly high among the overweight and obese, diabetics and hypertensives, thus implying that they were at a higher risk for the development of cardiovascular disease.

❧ Out of the total number of diabetics (n=82), 44% were found to be overweight and obese, and out of the total hypertensives, 54% were overweight and obese, thus portraying the plausible role of obesity as a risk factor for the development of these disorders

❧ An aberration in the atherogenic lipid parameters was also observed as one climbed the ladder of Body Mass Index (BMI)

❧ The correlation coefficient 'r' was found to be 0.88 and 0.77 among the males and females when BMI and WC were compared. It was found to be 0.47 and 0.02 when the BMI and WHR were taken into account. This indicates that in this study the BMI was found to correlate well with WC rather than with WHR, thus emphasizing the use of BMI and WC as an indicator of body fat.

❧ The relative risk of the atherogenic lipid parameters among the subjects suffering from atherosclerotic cardiovascular disease (CVD) showed that the TC was found to be the highly dependable risk factor for taking a person into clinically uncontrolled state.

❧ The relative risk for triglyceride was found to be highest among the diabetic (RR=4.4) subjects in comparison to other diseases. This observation is in line with data, which shows that in type 2 diabetes, the abnormality that characterizes lipoprotein metabolism is the fasting, and postprandial triglyceride-rich lipoproteins (Kannel 1985, Taskinen 1999, Howard 1987 and Syvanne et al 1994).

SECTION IV

APOLIPOPROTEINS AND TOTAL ANTIOXIDANT ACTIVITY OF THE SUBJECTS IN AN INDUSTRIAL SET-UP

The alarmingly increasing rates of chronic degenerative diseases in the less developed countries and economies in transition is well documented and the World Health Organisation has warned of an epidemic of cardiovascular diseases in the developing nations

In profiling the risk of these non-communicable diseases it has been customary to utilize the measurement of FBS, TC, HDL-C, LDL-C, TG etc. But recent studies and reports emphasize the usage of additional biochemical markers such as the measurement of antioxidants and apolipoprotein A1 (ApoA1) and apolipoprotein B (ApoB), which are considered to be better indicators for the assessment of coronary risk

Therefore, the present section was planned to see the efficacy of these parameters in a subset of the group under study. One ninety three subjects, willing to participate were enrolled for the study. Venous blood sample was collected after an overnight fast of 12 hours and the serum was used for estimation of various parameters viz FBS, lipid profile, apolipoproteins (Apo A1 and Apo B) and total antioxidants

The results of this section are given under the following sub headings.

CLINICAL PROFILE OF THE SUBJECTS

The clinical profile of the subjects is given in **Table 52**. The mean age of the normal subjects was 44 years, that of dyslipidemic and overweight & obese subjects to be 40 years, and 42 years and 43 years for the diabetic and hypertensive subjects. The average BMI was found to be 23.5 for normal subjects, 22.9 for dyslipidemics, 27.4 for overweight or obese subjects, 25.0 and 24.2 in case of diabetic and hypertensive subjects. WHR of the subjects was found to be 0.9 for all the groups.

DIETARY INTAKE OF THE SUBJECTS

The dietary intake of the subjects is depicted in **Table 53**. The caloric intake of the overweight & obese (2400 Kcal), diabetic (2250 Kcal) and hypertensive (2215Kcal) subjects was significantly higher than the normal subjects (1900 Kcal). Significantly higher values of fat and lower values of vitamin C were observed amongst the diabetics, overweight & obese and hypertensive subjects.

FASTING BLOOD SUGAR AND LIPID PROFILE OF THE SUBJECTS

The FSB levels of the subjects as shown in **table 54** indicate that the diabetic subjects had significantly higher ($p < 0.001$) levels in comparison to the normal, dyslipidaemic, overweight & obese and hypertensive subjects.

TABLE 52

CLINICAL PROFILE OF THE SUBJECTS (Mean \pm SD)

	NORMALS	DYSLIPIDEMICS	OWERWEIGHT & OBESE SUBJECTS	DIABETICS	HYPERTENSIVES
n	31	21	54	48	39
AGE (y)	44 \pm 8	40 \pm 8	40 \pm 4	42 \pm 7	43 \pm 6
HEIGHT (m)	1.7 \pm 0.7	158.4 \pm 8.4	1.7 \pm 0.5	1.6 \pm 0.7	1.6 \pm 0.6
WEIGHT(Kg)	64.0 \pm 10.7	57.8 \pm 11.2	75.9 \pm 8.1	70.0 \pm 9.2	65.8 \pm 11.4
BMI *	23.5 \pm 3.6	22.9 \pm 2.6	27.4 \pm 4.3	25.0 \pm 4.5	24.2 \pm 3.9
WAIST (cm)	85.9 \pm 8.9	83.8 \pm 9.7	89.1 \pm 9.9	87.0 \pm 8.0	85.0 \pm 9.6
HIP (cm)	99.0 \pm 7.0	93.1 \pm 7.5	98.7 \pm 10.8	99.3 \pm 9.1	98.0 \pm 8.6
WHR **	0.9 \pm 0.1	0.9 \pm 0.1	0.9 \pm 0.1	0.9 \pm 0.1	0.9 \pm 0.1

* BMI = $\frac{\text{Weight in Kg}}{\text{Height in (m}^2\text{)}}$

** WHR = $\frac{\text{Waist Measurement (cm)}}{\text{Hip Measurement (cm)}}$

TABLE 53
DIETARY ANALYSIS OF THE SUBJECTS (Mean \pm SD)

	NORMALS	DYSLIPIDEMICS	OVERWEIGHT & OBESE SUBJECTS	DIABETICS	HYPERTENSIVES
Calories (Kcal)	1900 \pm 200	2100 \pm 323	2400 \pm 345**	2250 \pm 400**	2215 \pm 275**
Carbohydrates (g)	260 3 \pm 55 1	270 5 \pm 59 0	265 6 \pm 45 4	250 8 \pm 34 6	246 0 \pm 40 7
Proteins (g)	67 7 \pm 10 6	65 7 \pm 12 6	60 8 \pm 8 7	64 5 \pm 11 2	66 6 \pm 15 2
Fats (g)	65 1 \pm 20 0	67 1 \pm 20 5	98 9 \pm 25 3***	93 6 \pm 30 3***	90.2 \pm 28 9***
Fiber (g)	9 2 \pm 2 4	8 0 \pm 2 1	7.6 \pm 3 1	8 0 \pm 2 6	8 5 \pm 4 3
Vitamin C (mg)	78 4 \pm 12 7	68 0 \pm 58 2 ***	68 50 \pm 13 9***	59 6 \pm 13 2***	66 5 \pm 15 2***
β - Carotene (μ g)	1590 1 \pm 985.2	1200 1 \pm 1105 5	1409 3 \pm 785 5	1560 8 \pm 684 9	1115 7 \pm 1000 5

All groups ** p<0 01, *** p<0 001 Vs Normals

Dyslipidemics *** p<0 001 Vs Overweight or obese, diabetics and hypertensives

TABLE 54
LIPID PROFILE OF THE NORMAL, DYSLIPIDEMIC, OVERWEIGHT & OBESE, DIABETIC AND
HYPERTENSIVE SUBJECTS (mg/dl, Mean \pm SD)

VARIABLE	NORMALS	DYSLIPIDEMICS	OVERWEIGHT & OBESE SUBJECTS	DIABETICS	HYPERTENSIVES
n	31	21	54	48	39
FBS	84.8 \pm 12.3	85.9 \pm 10.1	88.0 \pm 12.6	168.2 \pm 44.0 ^{*** vvv} ###	87.21 \pm 9.56
TC	161.2 \pm 16.9	207.9 \pm 23.6 ^{***}	207.3 \pm 30.0 ^{***}	213.7 \pm 41.2 ^{***}	198.85 \pm 36.36 ^{***}
TG	92.2 \pm 31.5	144.9 \pm 50.0 ^{***}	141.0 \pm 53.0 ^{***}	171.2 \pm 60.6 ^{*** z}	164.79 \pm 72.44 ^{***}
HDL-C	35.2 \pm 7.0	37.3 \pm 7.5	36.9 \pm 7.2	37.3 \pm 7.1	39.03 \pm 7.40 [*]
LDL-C	107.6 \pm 16.5	141.5 \pm 23.1 ^{***}	142.3 \pm 24.4 ^{*** qq}	141.8 \pm 30.5 ^{*** #}	126.86 \pm 35.63 ^{**}
VLDL-C	18.4 \pm 6.3	29.0 \pm 10.0 ^{***}	28.2 \pm 10.6 ^{***}	33.9 \pm 11.9 ^{*** z}	33.0 \pm 14.5 ^{***}
NON HDL-C	126.0 \pm 16.4	170.5 \pm 21.9 ^{***}	170.5 \pm 25.9 ^{***}	175.2 \pm 36.3 ^{*** #}	159.8 \pm 33.3 ^{***}
TC/H	4.8 \pm 1.0	5.7 \pm 1.1 [*]	5.7 \pm 0.8 ^{*** qq}	5.8 \pm 0.8 ^{*** ##}	5.2 \pm 0.9
L/H	3.2 \pm 1.0	3.9 \pm 1.0 [*]	4.0 \pm 0.8 ^{*** qq}	3.9 \pm 0.8 ^{*** ##}	3.3 \pm 1.0

All groups * p<0.05, ** p<0.01, ***p<0.001 Vs Normals
Dyslipidemics * p<0.05 Vs Hypertensives
Dyslipidemics vvv p<0.001 Vs Diabetics
Overweight & Obese ** p<0.01, *** p<0.001 Vs Diabetics
Overweight & Obese *** p<0.01Vs Hypertensives
Diabetics # p<0.05, ## p<0.01, ### p<0.001 Vs Hypertensives

The lipid profile of the subjects as depicted in Table 54 indicate that all the groups showed significantly raised levels of TC, TG, LDL-C, VLDL-C and Non HDL-C in comparison to normal subjects

When the dyslipidemic group having either high TC, TG or both TG and TC was compared with diabetic subjects it was noticed that the diabetics showed a rise in the atherogenic lipids such as TC, TG and Non HDL-C. When the same group was compared with hypertensive subjects, the TC/H and L/H ratios were found to be significantly raised ($p < 0.05$) among the dyslipidemics in comparison to the hypertensive subjects.

The comparison between the overweight & obese and diabetic groups showed that the TG and VLDL-C was significantly high in diabetics. The TG level of the diabetics was found to be 171.2 ± 60.6 mg/dl in comparison to 141.0 ± 53.0 mg/dl of the overweight & obese subjects.

The LDL-C and the TC/H and L/H ratios were found to be significantly higher in the case of overweight & obese subjects in comparison to the hypertensive subjects.

The diabetics had significantly higher ($p < 0.05$) level of LDL-C in comparison to hypertensives. The Non HDL-C was also found to be raised at $p < 0.05$ level among the diabetic subjects. The ratios of TC/H and L/H were found to be significantly higher in diabetics.

APOLIPOPROTEIN LEVELS OF THE SUBJECTS

The apolipoproteins, which are considered to be better indicators of cardiovascular risk have been shown in **Figure 17**. No significant difference was observed among the normal and overweight & obese subjects in relation to the apolipoproteins. Whereas, the ratio of Apo A1 / Apo B was found to be lower in this group. The Apo A1 and Apo B levels were found to be raised (122.6 ± 32.3 mg/dl and 119.2 ± 24.4 mg/dl) in the diabetics, as compared to the normal subjects (118.2 ± 34.7 mg/dl and 94.1 ± 23.1 mg/dl). The Apo B levels of the diabetics were significantly higher than the normal, dyslipidemic and overweight & obese subjects. The Apo A1 and Apo B levels were found to be 117.6 ± 21.1 mg/dl and 113.1 ± 20.7 mg/dl in the hypertensives and the ratio of Apo A1 / Apo B was 1.04 ± 0.2 in the hypertensive subjects which is lower than the normals.

TOTAL ANTIOXIDANT ACTIVITY OF THE SUBJECTS

The Total antioxidant activity (TAA) of the subjects is shown in **Figure 18**. The normal subjects had significantly higher levels of antioxidants (1.81 ± 0.19 $\mu\text{mol/l}$) in comparison to overweight or obese (1.65 ± 0.17 $\mu\text{mol/l}$), diabetic (1.62 ± 0.15 $\mu\text{mol/l}$) and hypertensive subjects (1.64 ± 0.17 $\mu\text{mol/l}$).

FIGURE 17

APOLIPOPROTEIN LEVELS OF THE NORMAL, DYSLIPIDEMIC, OVERWEIGHT & OBESE, DIABETIC AND HYPERTENSIVE SUBJECTS (mg/dl)

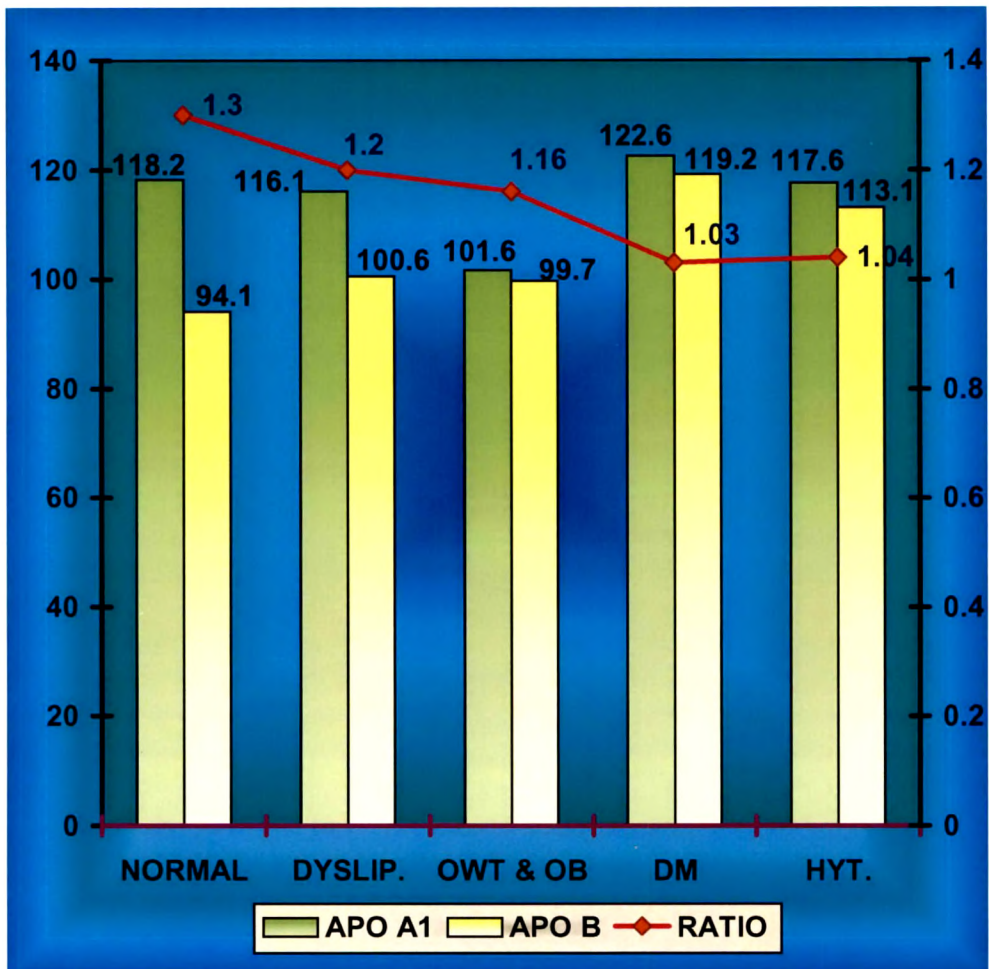
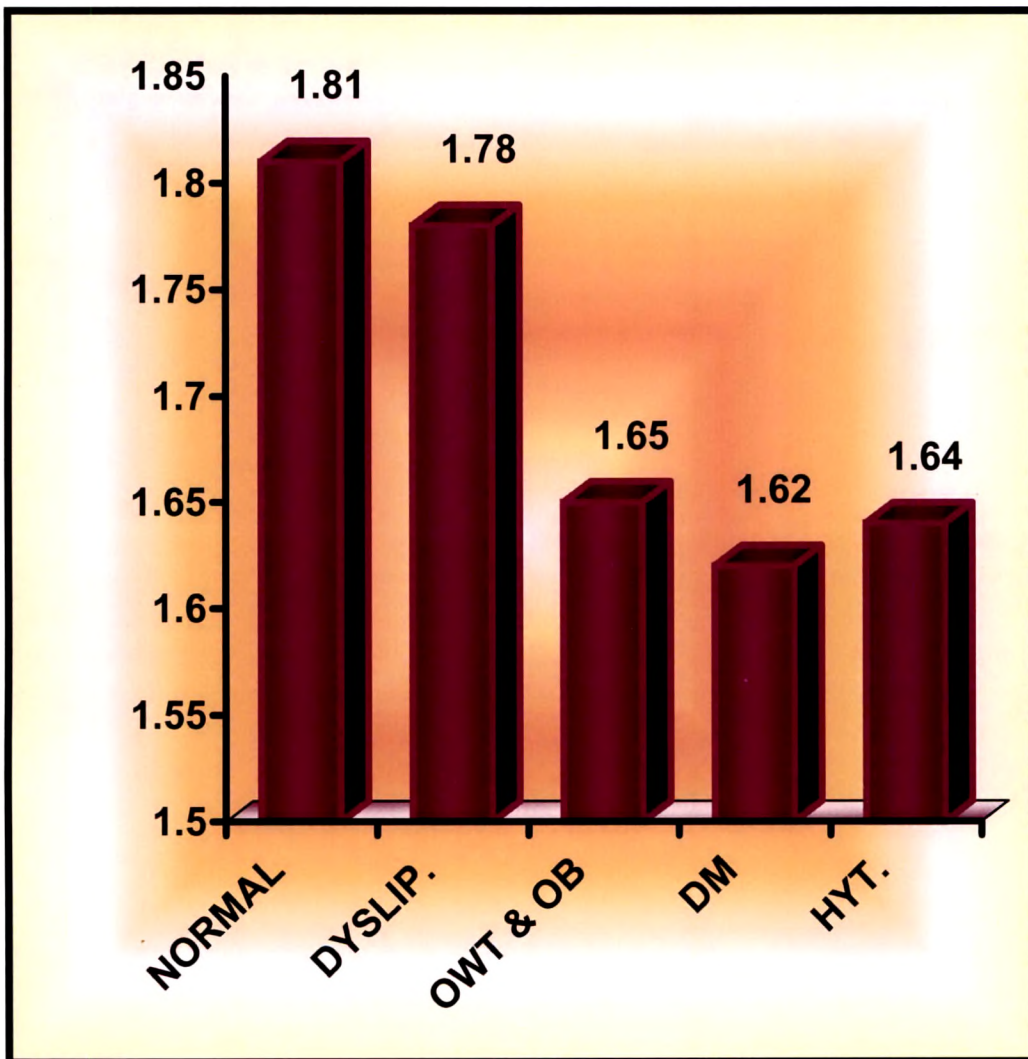


FIGURE 18

**TOTAL ANTIOXIDANT ACTIVITY OF THE NORMAL,
DYSLIPIDEMIC, OVERWEIGHT & OBESE, DIABETIC AND
HYPERTENSIVE SUBJECTS**



DISCUSSION

Coronary artery disease (CAD) is the single most important disease entity in terms of both mortality and morbidity and is a leading cause of death (Shahid 2000). Epidemiological studies have demonstrated that major risk factors such as dyslipidaemia, hypertension, diabetes mellitus and the use of tobacco products act in a synergistic manner (Kannel and Schatzkin 1983). Other risk factors include physical inactivity, obesity, age, gender, alcohol consumption etc. Raised serum concentration of cholesterol, LDL-C and low serum concentration of HDL-C are all associated with an increased risk of coronary atherosclerosis (Shestov et al 1993, Burke et al 1997). However, recent studies suggest that it is better to monitor levels of the apolipoproteins Apo B and Apo A1 which are considered to be better indicators of possible coronary artery disease. It is said that higher levels of Apo B may indicate increased risk of cardiovascular disease even when the total LDL-C is not high (Walldius et al 2001). It is also mentioned in the current research that antioxidants play a powerful role in the prevention of cardiovascular disease (Gale et al 2001, Luoma et al 1995, Esterbauer et al 1992, Steinberg 1992).

In addition to routine parameters of lipids, the estimation of apolipoproteins and total antioxidant activity would be able to predict the risk of CAD more accurately. Therefore, the present study was planned to assess the efficacy of these parameters along with lipid profile analysis in the study population.

The dietary analysis of the subjects in the present study revealed the intake of calories to be more among the overweight or obese, diabetic and hypertensive subjects. A significant increase in the fat was also observed among these subjects. This could be attributed to the consumption of fried foods available in the industrial canteen as explained in the previous sections

Aberrations in the lipid profile were observed in case of dyslipidemias, obese, diabetics and hypertensives. Obesity has been established as an independent risk factor for the development of CAD (Field et al 2001). Obesity has been aptly said to be the mother of all degenerative diseases, which predisposes an individual to the risk of development of diabetes, hypertension and CHD. The lipid profile of the overweight and obese subjects showed significantly higher levels of TC, LDL-C, VLDL-C, TG and Non HDL-C when compared with normal subjects. These results thus validate the fact that the overweight or obese subjects have an altered lipid profile and that they are at a higher risk of developing other degenerative diseases.

Diabetes mellitus has been shown to be associated with lipid abnormalities. About two to three times higher risk for coronary heart disease rate is seen in diabetic patients (Parikh et al 2001). Impairment in insulin secretion leads to an excessive and prolonged rise in glucose concentration. Similar results were seen when the blood glucose of the normal subjects were compared with diabetic subjects. The frequency of raised plasma lipid levels in diabetic subjects is between 20-90% depending on the degree of diabetic control and

the type of diabetes (Mani and Mahida 2002) The results of the present study also showed raised TC, LDL-C, VLDL-C, TG and Non HDL-C levels, which is in line with the findings obtained by Mani and Mani in 1988

Hypertension has also been marked as the most important risk factor for the development of cardiovascular morbidity (Castelli and Anderson 1986) Elevated lipid levels were observed among the hypertensive subjects and significant differences were noted when compared to normal subjects indicating their being at a greater risk of developing other secondary complications

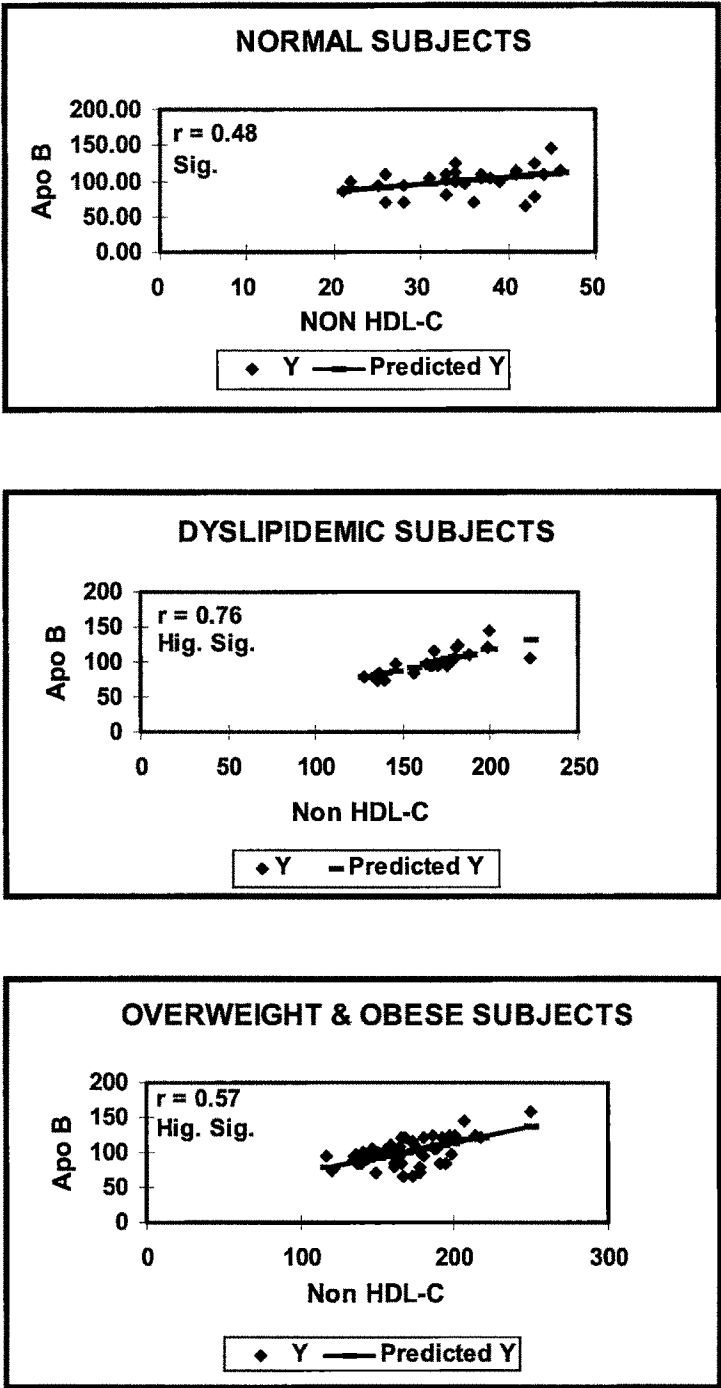
Plasma levels of Apo B and Apo A1 are important determinants of the risk of premature coronary artery disease In fact, Apo B and Apo A1 are thought to be better predictors of acute myocardial infarction than the TC and LDL-C (Walldius et al 2001). Apo B might be a good indicator because it is a major component of all lipoproteins (except HDL-C) that are associated with the development of atherosclerosis

Figure 17 shows the levels of Apo A1 and Apo B among the normal, dyslipidaemic, overweight and obese, diabetic and hypertensive subjects. The Apo B levels of the overweight and obese subjects were found to be higher than the normals. The ratio of Apo A1 / Apo B was found to be lower among the overweight and obese subjects thus indicating that they might be at a higher risk of developing other degenerative diseases.

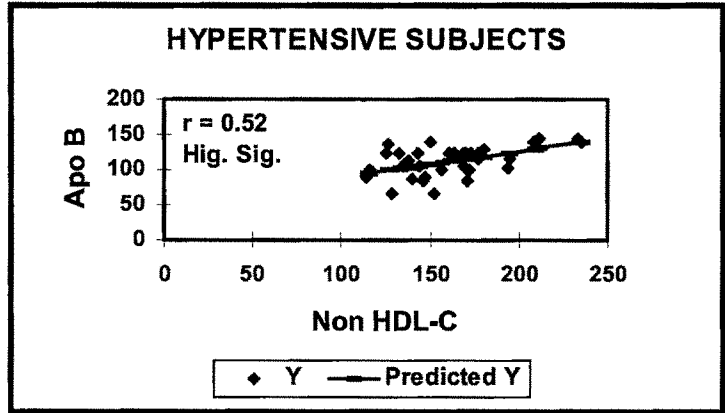
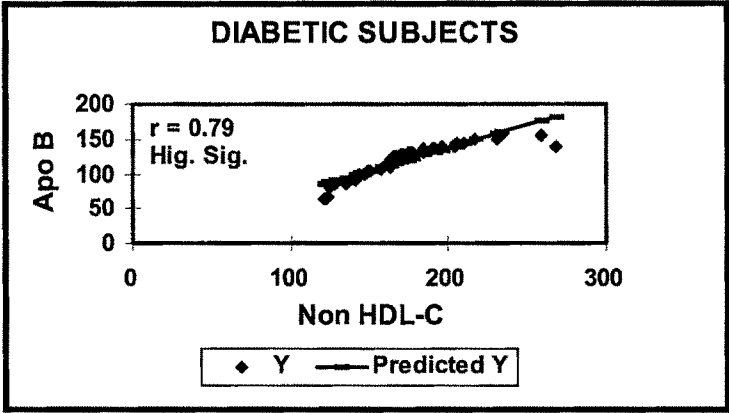
The comparison of the Apo B levels among the diabetic and normal subjects showed that they were found to be significantly higher among the diabetics. Similarly, the hypertensive subjects also showed higher levels of Apo B in comparison to the normal subjects. It is clearly evident from the figure that the ratios of Apo A1 / Apo B show a decreasing trend among the dyslipidaemic, overweight and obese, diabetic and hypertensive subjects thus indicating their being at a greater risk of developing CVD, as the ratio is considered to be a sensitive index for predicting the risk.

Some investigators have recently suggested that the levels of Non HDL-C might be a useful marker of increased CVD risk (Havel and Rapoport 1995, Frost and Havel 1998, Garg and Grundy 1990). A recent study conducted in a cohort containing both diabetic and non-diabetic individuals showed that Non HDL-C was a somewhat better predictor of CVD than LDL-C (Cui et al 2001). In the present study also, Non HDL-C, which is the component obtained after subtracting only HDL-C level from TC level was found to be significantly correlated with Apo B levels. Apo B is present in all atherogenic lipoproteins namely VLDL-C, LDL-C and IDL. In the normal subjects the correlation coefficient was found to be 0.48 as seen in **Figure 19**. The correlation coefficient among the dyslipidaemic, overweight or obese, diabetic and hypertensive subjects was 0.76, 0.57, 0.79 and 0.52 respectively and all were found to be statistically significant.

FIGURE 19
LINEAR REGRESSION BETWEEN NON HDL-C AND APO B



Contd.....



The evidence that antioxidants play a role in the prevalence of atherogenesis has been increasing rapidly in recent years (Esterbauer et al 1992, Steinberg 1992). Several studies have suggested that antioxidants such as alpha tocopherol, retinal, albumin and selenium may reduce cardiovascular mortality (Luoma et al 1995). They have also suggested that variations in serum antioxidant levels may explain the cross-cultural differences in the incidence of ischemic heart disease better than the classic risk factors such as raised serum cholesterol; high blood pressure and smoking (Luoma et al 1995).

Therefore, in the present study the total antioxidant activity was analysed. The results showed that the total antioxidant activity of the normal subjects ($1.81 \pm 0.20 \mu\text{moles/l}$) was significantly higher compared with overweight and obese ($1.65 \pm 0.17 \mu\text{moles/l}$), diabetic ($1.62 \pm 0.15 \mu\text{moles/l}$) and hypertensive ($1.64 \pm 0.17 \mu\text{moles/l}$) subjects thereby implicating a role for low levels of antioxidants in the development of IHD. However, the low intakes of vitamin C as determined by 24 hr dietary recall method did not go parallel with consumption of β -carotene. Various dietary surveys indicate a wider range in the intake of β -carotene (Gale et al 2001, Mani and Kannan, 2003, Mani and Kumar 2003, Mani and Tiwari 2002). In line with these observations, the present study also shows that a combination of methods (such as 24 hr dietary recall, food frequency method and a detailed interview) probably is necessary in order to get the dietary intake of various nutrients in a more meaningful way.

Hence, from the present study it is clear that aberrations in the lipid levels are observed as one develops various CDD. The apolipoproteins and the total antioxidant activity showed an adverse trend in the subjects suffering from various CDD thus suggesting that they can be used as additional tools for mapping the risk for the development of cardiovascular disease.

At a glance

✎ The caloric intake of the overweight or obese (2400 Kcal), diabetic (2250 Kcal) and hypertensive (2215Kcal) subjects was found to be significantly higher than the normal subjects (1900 Kcal) Furthermore, higher levels of fat and lower levels of vitamin C content in diet were observed in the diabetics, overweight or obese and hypertensive subjects

✎ The lipid profile of the diseased subjects showed increments in the atherogenic lipid parameters in comparison to normal subjects This shows that aberrations in the atherogenic lipid parameters are observed in subjects with CDD

✎ Appreciable rise in the Apo B levels of the diabetics was observed in comparison to the normals, dyslipidemics and overweight or obese subjects Similarly, in comparison to normals, the Apo B levels were found to be higher among the hypertensives (113.1 mg/dl)

✎ Aberrations in the lipid profile of the overweight or obese subjects showed significantly higher levels of TC, LDL-C, VLDL-C and TG in comparison to normals 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

❧ The frequency of raised plasma lipid levels in diabetic subjects is between 20-90% depending on the degree of diabetic control and the type of diabetes (Mani and Mani 1988) The results of the present study also showed raised TC, LDL-C, VLDL-C and TG levels, which is in line with the above cited findings

❧ Elevated lipid levels were observed among the hypertensive subjects and significant differences were noted when compared to normal subjects indicating they are at a greater risk of developing other secondary complications

❧ The normal subjects had significantly higher levels of antioxidants ($1.81 \pm 0.19 \mu\text{mol/l}$) in comparison to overweight or obese ($1.65 \pm 0.17 \mu\text{mol/l}$), diabetic ($1.62 \pm 0.15 \mu\text{mol/l}$) and hypertensive ($1.64 \pm 0.17 \mu\text{mol/l}$) subjects This adverse trend of antioxidants in subjects suffering from various CDD calls for dietary intervention strategies to improve antioxidant status.