DISCUSSION
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Estimation of diet induced changes in lipid lipoprotein profile and its relevance in predicting an individual risk for future atherosclerosis still remains an unexplored field.

Drastic individual variability in assimilating a cholesterol fat test load is the main reason why till now no reasonable cholesterol tolerance test could be devised. Individual also respond differently to single feeding and prolong feeding, thus this still deepens the mystery, whether a single dose or a prolong feeding programme could be a better screening procedure.

The possibility that the particular response of an individual to single high cholesterol test load may be the determinant of the development of the atherogenic process in him, has formed the basis of the present work.

This work has been conducted on healthy male and female volunteers of age 20-60 years, healthy first degree relatives of patients of
coronary artery disease and diabetic patients.

**SERUM TOTAL CHOLESTEROL**

The basal serum total cholesterol was 164.4±36.98, 185.2±36.2, 178.7±30.17 and 206-37±64.75 mg/dl of group A, B, C and D respectively.

Group A, B and C had basal STC within normal limits as set by lipid research clinics, however diabetics (group D) subjects had a 10% more basal STC level than normal subjects of the same age group. In healthy males (group A) highest basal levels was found in young subjects of 20-30 years, this may be because of high fat consumed and belonging to better socioeconomic status than older age person. However in healthy females (Group B) basal STC increased with age till 50 years and then after it showed a slight fall, this may be because of hormonal changes and repeated pregnancies associated with increasing age.

On feeding wingle high cholesterol test lead consisting of 800 mg of egg cholesterol, a fall in STC after 1 hour was observed in majority
of subjects of group A, B, and type II diabetes, while in type I diabetes there was rise in STC level and in group C levels were more or less unaffected. After three hours, the levels of STC have started increasing, but still they were below basal levels in group A and B, while in group C and type I diabetes they were well above the fasting levels.

Thus on the basis of post prandial response the healthy population can be divided in three groups. The majority of the population shows a fall in serum total cholesterol and LDL levels, 1 hour after feeding, in a minority STC level start rising 1 hour after feeding, while in the remaining these in no change. In contrast to these results, majority of the subjects who were predisposed to the risk of CHD showed a rising trend of STC. Similarly all type I diabetes showed a rising trend of STC. In the past Niskila et al. 1962 and Richard S., Havel 1957 have also reported fall in STC level after feeding. The explanation for this fall could be related to the suppression of LDL receptors after over night fasting (Medical clinics of North America Vol. 66 No.2
March 1982 page 344). When fat cholesterol load is given LDL receptors are stimulated by as yet some undefined hormonal or neurogenic reflexes, in anticipation of the cholesterol load that will enter the circulation. Large amount of LDL from intra-vascular compartment shifts intracellularly, resulting in an acute fall in serum LDL and STC levels after 1 hour. The cholesterol levels slowly increase after 3 hours as a result of the absorbed cholesterol and the reverse intravascular movement of LDL that had entered the tissues earlier.

In diabetes, majority of healthy relatives of patients of CAD and minority of healthy populations the increase of STC and LDL could be explained by some inherent biochemical block in anticipating and assimilating a cholesterol load.

**HDL CHOLESTEROL**

The basal HDL level were 46.0±6.34, 47.3±15.0, 43.5±11.5, and 47.8±27 mg/dl of group A, B, C and D respectively. These level were all within normal limits set by lipids research clinics. However group C subjects, i.e. first degree relation of patients of coronary artery disease, had competitively lower level of HDL than their healthy counterpart subjects of group A. Age seems to have no effect on basal HDL level. Female sex had competitively higher level of fasting HDL. On feeding single high cholesterol test load, no appreciable changes in HDL were observed after one hour. After three
hours, diabetic subjects (Group D) showed a significant fall in HDL levels, in other no significant changes were observed. Feeding induced fall in HDL level in diabetics was associated with rise of STG and LDL.

**SERUM TRIGLYCERIDE**

The basal STG levels were 146.0±38.7, 189.5±56.35, 183.6±50.93 and 204.37±62.7 mg/dl respectively of group A, B, C and D. All the groups had much higher STG value than set by lipid research clinics. Diabetics had the highest level of basal STG.

Healthy females had a much higher value of STG than healthy males. Basal STG value tended to increase with rising age.

After feeding single high cholesterol test load, STG levels tended to increase 1 and 3 hours post prandially in all the groups, except first degree relatives of patients of coronary artery disease where in first hour there was actually a slight fall in mean STG level, followed by very insignificant rise in third hour. Maximum rise of STG levels was observed in diabetics.
The peak level of STG in our study was observed at the end of third hour.

Barritt (1956) Brown et al. (1961) and Angervall (1964) reported peak STG levels, four hours after feeding in healthy subjects.

Barritt 1956 reported peak of STG after seven hours in subjects of IHD, in our study first degree relating of patients of CAD had a delayed peak in fact the STG started rising after 3 hours. end of 3 hours. Sklarin et al. (1961) and Mehra et al. (1963) also reported delayed and sustained rise of STG levels in diabetic subjects.

**VLDL CHOLESTEROL**

Changes in VLDL were exactly similar to those observed in STG.

**LDL CHOLESTEROL**

Basal LDL values were 88.90±37.0, 99.7±29.7, 98.7±24.25 and 119.27±50.79 mg/dl of group A, B, C and D respectively. These values were within limits set by lipid research clinics. Healthy females and diabetic subjects had a higher level of basal LDL.
valves as compared to healthy males. First degree relatives of patients of CAD had a lower level of basal mean LDL levels than their healthy counterparts. Age seemed to have no appreciable effect on basal LDL levels.

Feeding single high cholesterol load resulted in decrease in LDL levels in healthy male, female and type II diabetic subjects, while no appreciable change was observed in first degree relatives of patients of coronary artery disease, in type I diabetics the levels actually started rising after feeding. After three hours the LDL levels were still declining in group A and B, levels were increasing in group D while they were unaffected in group C. The explanation for the observed phenomenon is the same as that for STC.