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Since patients with diabetes mellitus are more dependent on fat as a source of energy than the normal subjects. It is tempting to try to explain the hyperlipoidemia that is so common in this disease on the basis of some theory that assumes a relationship between the amount of fat in the diet and the percentage of lipoids in the blood. In the literature that has accumulated on this subject a tendency to assume this relationship is evident. In the Rockefeller monograph (1919) the authors stated that lipemia "is largely associated with the fat intake and with other diabetic symptoms". Ervin (1919) stated that the lipemia of a diabetic will disappear with the elimination of fat from the diet". Bang (1919) believed that lipemia was in part alimentary. Joslin (1921) suggested a relationship between high fat and protein diets of former days and the high degree of lipemia reported and stated that "with restricted diet, particularly of fat, the blood fat rapidly falls".

Bloor has supported a suggestion of Allens (1917) that there is lacking a pancreatic hormone which is necessary for proper removal of the fat from blood. Bloor continues by conceiving that the factor of over work must be taken into consideration in examining into the cause of diabetic lipemia and that the patient has a fat tolerance which can be raised or lowered according as the ingested fat is restricted or increased.
When large amount of fat are ingested the mechanism for the utilization of fat might be expected to break down and he reported a case in which he alleged that a high lipemia resulted from a dietetic indiscretion which consisted chiefly in ingestion of milk and cream.

In diabetes mellitus disturbances of serum lipoprotein concentrations may account for the increased frequency of atherosclerotic disease is affected patients (Bierman, 1978; Ganda, 1980). Increased level of very low density lipoprotein (VLDL) and low density lipoprotein (LDL) lipids and a decreased concentration of high density lipoprotein (HDL) cholesterol have been frequently described (Lopes-Virella, 1978; Taylor, 1981; and Briones, 1984).

The increase in VLDL has been attributed to the deficiency of lipoprotein lipase activity which is stimulated by insulin. The increase in LDL with concomitant decrease in HDL has not been satisfactorily explained but generally attributed to an increase in dietary fat and cholesterol.

How do diabetics dispose of a cholesterol load given with saturated fat is a question for which no satisfactory answer has yet been provided.

Although many researchers (Bierman, 1978; Ganda, 1980; and Lopes-Virella, 1978) have characterised basal serum lipoprotein abnormalities in diabetes. None have
performed metabolic studies to find out the lipid response to the exogenously administered cholesterol saturated fat load.

Marsh (1923) had noted the effect on the lipidemia of diabetic patients of high fat, low protein and low carbohydrate diets, but at that time various lipid fractions like LDL, HDL, VLDL and triglycerides were even not known. So the results and observations are inconclusive.

In the present study we have tried to fulfill their lacunas.