INTRODUCTION
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Diabetes Mellitus (DM) is a complex syndrome characterized by (i) hyperglycemia, secondary to deranged secretion and/or action of insulin, (ii) specific micro-vascular complications including thickening of capillary basement membranes (BM), retinopathy and nephropathy, (iii) macro-vascular disease i.e. accelerated atherosclerosis and (iv) a variety of other complications including neuropathy, complicated pregnancy and increased tendency to infection. In addition, in the untreated state there is accelerated catabolism of both fat and protein (Kahn 1987).

Primarily, diabetes is still a heterogeneous disease. There are two common types of diabetes:

Type I or Insulin dependent diabetes mellitus (IDDM)
formerly called Juvenile onset diabetes

Type II or Non-insulin dependent diabetes mellitus (NIDDM)
formerly called maturity onset diabetes.

These differ in both clinical presentation and etiology. IDDM is clearly associated with an absolute deficiency of insulin secretion, which appears to result from an autoimmune process triggered by some environmental factors such as viral infections or toxins. This autoimmune process is directed at
the insulin producing beta cells of the pancreas and ultimately leads to their destruction (Kahn 1987).

NIDDM is not associated with any specific HLA antigens, but it is clearly genetically influenced since it occurs in identical twins with almost total concordance (Taylor 1989). NIDDM is also found to be associated with obesity in more than 80% of the patients suggesting the possibility that the Type II DM may be due to a disordered mechanism of appetite regulation or energy expenditure. Further Type II diabetics have considerable preservation of the beta cell mass and often secrete substantial quantities of insulin into the circulation, indicating resistance of the peripheral tissues to respond to insulin (Kahn 1987). Thus, the pathogenesis of Type II diabetes is multifactorial and which factor constitutes the primary defect remains uncertain.

Insulin deficiency in DM affects the carbohydrate (CHO) protein and fat metabolism. Adipose tissue, muscles and liver are the primary contributors to the imbalance of metabolites in blood and to many metabolic alterations which frequently accompany the diabetic syndrome. This is due to:

(i) Impaired entry and oxidation of glucose into the muscles and other tissues resulting in an increased glycogenolysis, glycolysis and proteolysis. During
this process aminoacids are released into the blood stream.

(ii) Poor utilization of glucose for synthesis of fat in adipose tissue and liver. As a result, lypolysis occurs and free fatty acid (FFA) and glycerol are released into the blood.

(iii) Impairment in the synthesis of glycogen in the liver. As there is increased flux of aminoacids and FFA, the aminoacids are metabolised to pyruvate and FFA are used for the synthesis of triglyceride (TG) and cholesterol. These are released into the blood in unusually large amounts resulting in Hyperlipidemia.

In most of the studies of diabetic population the prevalence of hyperlipidemia has been found to be increased and abnormal lipid metabolism is now considered as a link between diabetic and vascular disease (Mani and Mani 1988, Goldstein and Brown 1977).

The most common abnormality of lipid metabolism in patients with DM is hypertriglyceridemia. An increase in plasma TG concentrations results from either elevated levels of very low density lipoproteins (VLDL) and/or chylomicron. In patients with IDDM, who are in extremely poor metabolic control, severe hypertriglyceridemia develops primarily due
to an accumulation of chylomicrons. However, this is relatively rare and plasma TG concentrations are normal in most insulin treated patients with IDDM, even when their hyperglycemia is relatively poorly controlled. In contrast, plasma TG concentrations are often elevated in patients with NIDDM and this is primarily due to an increase in the plasma VLDL concentrations (Reaven 1987).

Although TG is the major lipid constituent of VLDL, it also contains significant amounts of cholesterol. As a consequence, hypercholesterolemia may also develop secondary to increase in VLDL levels, though changes in low density lipoprotein cholesterol (LDL-C) and high density lipoprotein cholesterol (HDL-C) levels also occur with variable degrees of diabetic control. Since VLDL is the major precursor for LDL in the plasma, increased synthesis of VLDL might lead to increased formation of LDL. On the other hand plasma HDL-C concentration was found to be low in patients with NIDDM. Theoretically a low plasma HDL-C concentration could result from a decrease in its synthetic rate and/or an increase in its catabolic rate (Reaven 1987).

Thus defects in lipoprotein metabolism are a prominent feature in the diabetic syndrome and the regulation of plasma LDL-C levels is of critical importance, as LDL-C levels is strongly and positively related to coronary heart
disease (CHD).

Interaction of certain serum lipoproteins with arterial connective tissue macro-molecules is considered, one of the mechanisms of cholesterol accumulation in human atherosclerotic lesions (Srinivasan et al 1980). Observations based on \textit{in vitro} and \textit{in vivo} experiments imply an association of lipoproteins with glycosaminoglycans (GAG) or proteoglycans (the native form of which GAG occur) in the arterial lesions (Berenson et al 1984). Further in a rabbit model it has been shown that sustained hypercholesterolemia causes atherosclerosis. This is associated with a simultaneous increase in LDL uptake by aorta, alterations in GAG metabolism and formation of LDL complexes of GAG in combination with collagen and elastin (Srinivasan et al 1984). Thus GAG may play an important role in remodelling the arterial wall structure during regression of atherosclerotic lesions.

The complications of DM are thought to arise in part from the interaction of insulin deficiency, hyperglycemia or both with independent environmental or genetic factors that together alter the structure and function of the tissues involved. Tissues involved in diabetic complications generally do not require insulin for the uptake and metabolism of glucose and since the concentration of glucose in these tissues parallels that in plasma, hyperglycemia has been speculated
to produce direct adverse effects on these tissues through the non-enzymatic glycosylation (NEG) process (Greene and Lattimer 1986).

NEG of proteins arising from ambient blood glucose in serum is used for assessing the metabolic control of DM. Glucose can combine with proteins at a number of sites, thus NEG of proteins is not specific and a number of body proteins can be glycated in this manner. Proteins that are glycated in this fashion are those which have longevity and exposed to relatively high concentration of sugar (Bunn 1981).

Experience with the use of glycated haemoglobin throughout 1980's have confirmed its uniqueness and usefulness as an objective index of long term glycemia in DM and has enabled the definition of realistic and achievable targets for outpatient management (Kennedy and Lyons 1989). On the other hand measurement of glycated serum proteins (GSP), yields information over a much shorter time scale which may be particularly useful in diabetic pregnancy and short term therapeutic studies (Mani et al 1987; Kennedy and Lyons 1989).

The most intriguing aspect of NEG is that it may form the basis of a logical link between hyperglycemia and the chronic complications of diabetes (Brownlee et al 1985). NEG may involve a site which is crucial for the specific function
of the protein or its normal metabolism thereby inhibiting their function or metabolism. This is particularly relevant for short lived proteins with highly specialized functions. Well known example is the glycation of LDL, which leads to atheromatous lesions in diabetics (Mani et al 1987, Kennedy and Lyons 1989).

NEG can also alter the structure or physical properties of a protein. This has been best characterized in collagen. Collagen is a single long lived protein in the body, and therefore forms an excellent candidate for NEG due to its cross-linking ability. The glycoadducts formed on long-lived proteins can undergo a series of further reactions, rearrangements and dehydration to form complex structures called Advanced glycosylated end products (AGEP). The AGEP's are characterized by their brown colour, specific fluoresence and participation in protein to protein cross-linking. The AGEP are irreversibly attached to proteins and continue to accumulate indefinitely on long lived proteins - collagen. It is this continual accumulation of cross-linking moieties on proteins that is believed to play an important role in the development of some of the complications associated with diabetes and ageing (Cerami et al 1988).

The structural alterations of BM collagen due to over-glycosylation are found to be involved in diabetic
microangiopathy which is unique to diabetes. Further the secondary complications which causes most of the morbidity and mortality are due to the thickening of DM. This process alters the composition of DM components.

Collagen, a glycoprotein is unique in its aminoacid composition as it contains unusual aminoacid such as 3 and 4 hydroxyproline (HP). The excretion of HP in urine provides a convenient tool for studies on the collagen metabolism since HP is almost exclusively present in collagen and therefore forms an *in vivo* label for this protein. Recent studies by Mani and Mani (1986) indicated a higher excretion of HP in the case of diabetics in general and IDDM in particular. Thus, hyperglycemia also leads to altered collagen metabolism.

From the above literature it is clear that DM is not a single disease but a group of disorders characterized by hyperglycemia, abnormal lipid and GAG metabolism altered collagen metabolism and excessive accumulation of glycoproteins in tissues. These alterations brings about a series of complications that significantly reduces the quality and extent of life. Therefore appropriate nutrition management is essential for restoring and maintaining a normal metabolic state.

A prudent nutrition plan reduces the exaggerated risk
for atherosclerotic heart disease and metabolic complications of diabetes by improving lipid and glycemic control. The current consensus diabetic diet recommends 55-60% of energy as CHO, 12-20% as protein and less than 30% fat (Anderson and Geil 1988).

The hypoglycemic effect of CHO is attributed to their fibre contents. The pioneering work of Anderson et al (1978, 1979) demonstrated that it is possible to withdraw insulin therapy in diabetics with low doses of insulin and to reduce the insulin requirement in patients on higher doses by providing high carbohydrate high fibre diet.

Viscous polysaccharides such as guar gum and pectin have been shown to reduce postprandial glycemia substantially both in healthy and diabetic individuals (Jenkins et al 1976, Jenkins et al 1977, Jenkins et al 1978, Utasitupa et al 1989). Guar was successfully incorporated into crispbread (Jenkins et al 1978) and bread (Ellis et al 1981). However, these polysaccharides have not been widely used because of their low palatability.

Feeding soy dietary fibre to NIDDM patients have resulted in decreased Fasting blood sugar (FBS) levels and improved glucose tolerance curve (Madar 1988). Rice fibre has shown only minor effect as it is composed of cellulose and hemicellulose of low viscosity (Madar and Thorne 1987). Other
particulate fibre - wheat bran has shown only a marginal fall in FBS levels in diabetic patients (Mani et al 1987).

Legume seeds are one food group that have elicited low postprandial glucose and insulin responses (Jenkins et al 1980a, b, Dilawari et al 1981, Fleming and Shaheen 1988). Pea fibre has also been found to reduce postprandial blood glucose response (Hamberg et al 1989). Similarly oat-fibre have shown hypocholesterolemic activity (Shinnick et al 1988). Studies with colocasia leaves on serum and tissue lipids in hypercholesterolemic rats showed that colocasia is lipogenic in nature (Mani et al 1989).

Thus in a nutshell the above studies indicate that dietary fibre (DF) contributes significantly to the prevention of metabolic disease DM. Physiological effects of DF appear to depend heavily on the source and composition of the fibre. A variety of fibre from different sources may prove to be ideal in optimizing the benefits.

There are virtually no studies on the effect of fibres from dry Sundakai (Solanum Torvum) and curry leaves (Murraya Koenigii). As the population of South India consume a large amount of curry leaves and Sundakai the studies would be quite useful and applicable in forming a meaningful therapeutic dietary intervention to control the metabolic disorder DM.
Keeping all the above points in mind a study was planned with the following objectives:

1) To study the biochemical changes in IDDM and NIDDM patients and their role in the secondary complications.

2) To study the effect of dry Sundakai powder supplementation on serum and tissue lipids in diabetic rats.

3) To study the effect of dry Sundakai powder supplementation on lipid profile, glycated proteins and total aminoacids in NIDDM patients.

4) To study the effect of curry leaves supplementation on serum and tissue lipids in diabetic rats.

5) To study the effect of curry leaves supplementation on lipid profile, glycated proteins and total aminoacids in NIDDM patients.