GLYCEMIC RESPONSE TO SELECTED CARBOHYDRATE RICH FOODS IN DIABETICS

BY

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Dedicated to

my parents, brothers and sister

DECLARATION

I hereby declare that this thesis entitled "Glycemic response to selected carbohydrate rich foods in diabetics", is a bonafide record of research work done by me during the course of research and that the thesis has not previously formed the basis for associateship, fellowship or other similar title of any other university or society.

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CERTIFICATE

Certified that this thesis entitled **"Glycemic response to selected carbohydrate rich foods in diabetics"** is a record of research work done independently by **Miss.Kavita M.S.**, under my guidance and supervision and that it has not previously formed the basis for the award of any degree, fellowship or associateship to her.

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Sheila. M.S.

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INTRODUCTION

INTRODUCTION

Diabetes is a disease of complex etiology with a condition of chronic hyperglycemia and its prevalence varies greatly among countries and populations. Interpopulation variations are mainly attributable to dietary habits.

Indian diets are cereal based ones and diabetes is a common disease among the middle or higher income strata of the Indian population. During this century marked changes in disease profile have occured in developed and developing countries with Non Insulin Dependent Diabetes Mellitus (NIDDM) as one of the major causes of morbidity and mortality. In India 90 per cent of the patients are diagnosed to have NIDDM.

In the management of NIDDM, diet has been recognised as a corner stone of therapy. During the past few decades, dietary modification in the treatment of diabetes mellitus have advanced from alterations in nutrient constitution (mainly carbohydrate) of a meal to alteration in the whole meal itself. For many years, diabetic individuals were encouraged to consume diets high in fat and protein and low in carbohydrates (Anderson, 1980). But emphasis has been put

recently on a more liberal use of carbohydrates in the diet of diabetic patients. (Mann, 1980) so that this type of food represents 50 to 60 per cent of daily caloric intake.

Equivalent amounts of carbohydrate may give different responses, since the kind of carbohydrate, food form, nature of carbohydrate and method of cooking have a marked influence on postprandial glycemia. According to Christine et al (1994) unavailable carbohydrates act on intestinal motility and that these effects seem to have been evolved in the decreased postprandial glycemia.

Coulston et al (1981) showed that substitution of one starchy food for another in the course of a mixed meal elicited different glucose responses in glucose intolerant subjects. Hence the biological equivalent ie., the quantities of cooked food yielding the same effect on blood glucose or glycemic response of a food should be considered while planning a diet for diabetics.

Most of the earlier studies have not shown whether the concept of glycemic index persits when carbohydrate foods are incorporated in a mixed meal, the most common manner in which they are consumed. Since there is paucity of data on the glycemic response of food ingredients included in a

typical Kerala diet, the present study is attempted with a specific objective to assess the glycemic response to selected carbohydrate rich foods in Non-Insulin Dependent Diabetes Mellitus (NIDDM) subjects.

REVIEW OF LITERATURE

REVIEW OF LITERATURE

Literature pertaining to the study on "Glycemic response to selected carbohydrate rich foods", is presented under the following headings:

2.1 Prevalence of diabetes mellitus.

2.2 Major symptoms of diabetes mellitus

2.3 Factors influencing blood glucose profile and

2.4 Diet in diabetes

2.1 Prevalence of diabetes

Diabetes mellitus is the major health problem of modern society. It is characterised by an alteration in the system of regulating the blood sugar level and an imbalance of insulin and glucagon that leads to hyperglycemia. Welborn (1983) defined diabetes mellitus as a genetically determined disorder of carbohydrate metabolism characterised by glucosuria, fasting hyperglycemia and development of microvascular complications and accelerated atherogenesis. Acute cases of diabetes mellitus predisposes neuropathy, nephropathy and retinopathy to patients and finally diabetic coma and death.

Population based studies related to diabetes have led to a much better understanding of the social, behavioural and environmental components of the disease. The prevalence of this disease varies in different geographic regions and in different ethnic groups.

Diabetes mellitus is reported to affect 2 per cent of the world population (WHO, 1985). In United States diabetes mellitus is the fifth leading cause of death and approximately 4.2 million persons suffered from this disorder (Krall, 1984). Along with the control of various communicable diseases, diabetes mellitus has become a clinical entity even in developing countries (Dutta et al, 1987). According to Krall (1984) there are 50 to 75 million diabetics in the world. Ahuja (1979) found that diabetes mellitus was an important health problem in India with an overall prevalence of 1.8 per cent. Diabetes is prevalent among 1.2 per cent of rural population and 2.4 per cent of urban population in India (Geevargheese and Abraham, 1984). A greater incidence of diabetes is reported among men than women, the ratio being 2.6:1.6 in India (Ahuja, 1979).

According to Tattersall (1984) diabetes in the elderly is a major health problem. In old persons, the disease often comes to light incidentally while investigating heart

disease, arterial insufficiency or failing vision (WHO, 1985). Ramachandran (1993) found that the prevalence of diabetes in the urban population is 8.2 per cent higher than what had been reported earlier and by a rough estimate there are about 25 million diabetics in India today and by the year 2000, this number is expected to reach about 35 million.

Diabetes is a disease of complex etiology with several contributory factors. It is a condition of chronic hyperglycemia accompanied by related symptoms such as glucosuria. In a WHO study (1980), heredity, high birth weight, obesity, oral contraceptive use and mental stress are reported as various risk factors associated with diabetes. According to Welborn (1984), there is a positive association between excess energy consumption, obesity, dietary fat intake and urban factors in relation to prevalence of diabetes. Obesity is reported to be a major determinant in the etiology of non-insulin dependent diabetes mellitus (Lee, 1981 and Nuttal and Gannon, 1987). According to Hansen (1988) weight reduction and improvement in blood glucose control through dietary interconvention for obese person with non-insulin dependent diabetes is the greatest potential for reducing morbidity and mortality.

A cross sectional study was conducted on the prevalence of atherosclerotic vascular disease and its risk factors in the NIDDM and non-diabetic subjects in east and west Finland and concluded that in both areas and in both sexes the prevalence of coronary heart disease and stroke were higher in diabetic than in non-diabetic subjects. (Markulaakso et al., 1986)

Malnutrition diabetes is reported to be associated with specific dietary intake of cyanide from cassava and other foods (McMillian and Geevargheese, 1979). Certain types of diabetes related with malnutrition, are found to be prevalent in Kerala and Indonesia (WHO, 1980). Existence of malnutrition related diabetes is not confirmed among the rural population in Africa, whose diet is found to be 84 per cent carbohydrate and 8 per cent proteins (Tauscher et al., 1987).

2.2. Major symptoms of Diabetes mellitus

Diabetes mellitus is a metabolic disorder of energy balance with many contributory causes and forms (Sue, 1988).

Non-insulin dependent diabetic state is characterised by a combination of inadequate insulin secretion and resistance of peripheral tissues to its actions. (De Fronzo

et al., 1983). According to Pyke (1977) Diabetes mellitus is not a single disease but a syndrome with many nasological entities. Chronic hyperglycemia is reported to induce changes in the polyol and sorbitol pathways, damaging vulnerable tissues (Gabbay, 1973). The functions of connective tissues, lens of eyes and other important structural proteins may impair due to the increased glycosylation, which occured in them (Weiland, 1983).

Complications related to diabetes, are progressive change to the eyes, kidneys, nerves and arteries, represent the major threat to the health and life of diabetics. Evidence from clinical observations and experiments on animals strongly suggests that improved control of diabetic state can be attained by controlling normal level of blood glucose levels (WHO, 1985).

Anderson et al (1987) found out the positive effect of high protein and low fat diet versus a low protein and high fat diet on blood glucose and negative effect of serum lipoprotein and cholesterol metabolism in diabetic patients. As per the results the diabetic patients were hyperglycemic with very low density lipoprotein values.

In a report of American Diabetes Association (1987) it has been stated that hypertension and diabetes were commonly associated. Rosett (1988) reported that one third of the insulin dependent diabetics and one fifth of the non-insulin dependent diabetics developed neuropathy.

Individuals with an upper body form of obesity show greater association with higher glucose excretion, exacerbated insulin resistance, increased abnormality of lipoprotein profile and higher cardiovascular risk (Hansen, 1988). There was an excess number of deaths among diabetic subjects compared with normal subjects and subjects with impaired glucose tolerance (Zimmet et al, 1988).

According to Bachanan and Mc Carrol (1972) glucagon is a potent hypoglycemic hormone secreted by α cells (A) of pancreas. Plasma levels of immunoreactive glucagon (IRG) have been found elevated in NIDDM as well as in IDDM cases. Alpha cell dysfunction is further characterised by hyper-responsiveness to amino acids or protein meal and on an un-responsiveness to oral glucose and insulin (Gerich et al., 1973). Unger (1981) has observed that α cells appear to be un-responsive to changes in the levels of blood glucose in diabetes mellitus.

According to Ramachandran et al (1992) weight loss was directly correlated with the severity of hyperglycemia and symptoms had no association with original body mass index. The effect of hyperglycemia is reported to differ in obese and non-obese patients and the modulating effect of obesity in insulin secretion disappears with development of hyperglycemia (Snehalatha et al., 1992).

Any serious disturbance of the pancreas that interferes with the production and balance of insulin and glucagon predisposes a person to have clinical manifestation of diabetes. The synthesis of an abnormal, biologically less active, insulin molecule as a result of mutation of the insulin gene (mutant insulin) is demonstrated to be the cause of non-insulin dependent diabetes mellitus (NIDDM) (Shoelson et al., 1983) For unknown reasons, β cell mass in patients with NIDDM may be reduced at the time of diagnosis. Further more, insulin response to glucose challenge is diminished in many individuals (WHO, 1985).

Glycemic index is one of the valid ways to analyse blood glucose responses. However it is recognised that methodologic factors may influence profoundly the interpretation of glycemic response data (Gannon and Nuttal, 1987).

The ability of the food item to raise the blood sugar is measured in terms of glycemic index (GI).

$$GI = \frac{\text{Blood glucose area of test food}}{\text{Blood glucose area of reference food}} \times 100$$

Antia (1989)

According to Jenkins (1982) the following formula was used for computing the glycemic response (GR)

$$GR = \frac{\text{Area under 2 hour blood glucose response curve of test food}}{\text{Area under 2 hour blood glucose response curve for equivalent amount of glucose}}$$

Type II diabetes is diagnosed glucose clearance which is accompanied by compensatory hyperinsulinemia, suggesting that the primary defect is in peripheral tissue response to insulin and glucose, not in the pancreatic β cell (Warraun, et al., 1990).

According to Keen et al (1979) body mass and blood glucose levels were universely correlated with energy intake. Ketonuria accompanied by significant hyperglycemia was present in hospitalized diabetic patients with NIDDM (Papadakis and Grunfeild, 1986).

There is no correlation between HDLC and glycemic control as stated by Rao et al (1983). In this experiment

reduced HDL cholesterol and HDL_2 cholesterol are reported to occur in NIDDM, but patients with impaired glucose tolerance, were found to have no dramatic alterations in HDL levels (Falko et al., 1987). The degree of control of diabetes is based on the mean of the fasting blood glucose levels (Oli and Ikeakor, 1984)

Nielsen and Nielsen (1989) found that a pre-prandial blood glucose (PPBG) less than 13 mmol/L had no influence upon net glycemic response where as PPBGs greater than 13 mmol/L were significantly and negatively correlated to the net glycemic response.

Total area under the curve is a descriptive factor related to basal blood glucose value, whereas incremental and positive area under the curve more accurately describe glycemic response to foods (Le et al., 1990).

The peak area of the blood glucose curve after the consumption of a given food component is expressed as a percentage of the peak area obtained after the consumption of an equal amount of a standard food. Postprandial peak is found to be highly dependent on initial glucose concentration (Van et al., 1989). According to Van et al (1989) a better location with shorter intervals is better than frequent blood sampling to determine plasma glucose levels.

Thorburn et al (1986) found that the postprandial blood glucose concentrations reflect hepatic glucose as well as digestion and absorption of ingested food (American Diabetes Association, 1984). They concluded that insulin and counter regulatory responses to specific food and meals need to be considered as possible determinants of glucose concentrations.

In fasting patients with NIDDM, glucose is taken up by the skeletal muscle in normal amounts but preferentially used non-oxidatively with lactate formation, indicating that the muscle does not contribute directly to fasting hyperglycemia and may play an indirect role through an increased delivery of precursors of glucose and insulin induced normoglycemia which is not involved in the exchange of glucose and gluconeogenic substrates by the skeletal muscle (Cepaldo et al., 1990)

Glycemic responses in capillary blood are greater than those in venous blood or plasma and therefore may allow smaller differences in glycemic responses to different foods to be detected (Jackson et al., 1983).

According to Nishimune et al (1991) the stronger dependancy of glycemic index on soluble dietary fiber

suggests a major function of soluble dietary fiber in the total dietary fibre hypoglycemic effect. Thompson et al (1984) found that polyphenols especially the large polymeric type or condensed tannins, appear to be responsible in part for the reduced glycemic response to carbohydrate foods and in part to lower blood glucose response to legumes composed with cereal products.

According to Wolever (1990a) the reduced glycemic responses seen after soluble fiber enriched meals and low glycemic index foods can be explained by slow absorption and the reduction of insulin levels may be an important mechanism for the serum cholesterol lowering effect of soluble fibre and low glycemic index foods. Mclonor et al (1985) found when consumed with lunch as well as breakfast, high carbohydrate, high fibre (HCF) bars caused flattening of blood glucose responses during the postprandial period after breakfast and maintained flattened responses during the early and late postprandial periods after lunch and HCF bars can be used to blunt postprandial blood glucose responses with normal and abnormal carbohydrate metabolism.

Acarbose, an inhibiter of mucosal α - glucosidases (sucrase, maltase and glucoamylase) responsible for carbohydrate degradation, is now used in some countries to

treat diabetes (Caspary, 1978). According to Seshiah (1984), acarbose present in wheat is presumed to act as an inhibitor and hence prevented the absorption of carbohydrate and fats to such an extent that the rise in blood glucose following a meal could be restricted to as much as 50 per cent. Acarbose is reported to be a complex oligosaccharide which inversely inhibits glucosidase present in the small intestinal mucosa and hence delays the production of monosaccharides by digestion of ingested complex polysaccharides (Sephan and Clive, 1988). They suggested, acarbose as an effective oral hypoglycemic agent without side effects.

A reduced thermic effect of glucose causes or precipitates obesity in NIDDM because a low thermic response was the consequence of the increased insulin resistance and opposed by greater increases in resisting metabolic rate (Ravussin and Zawadski 1984).

According to Averna et al (1988) fructosamine is a good index for short term metabolic control, and if used in an integrated fashion with glycemia and glycosylated haemoglobin (HbAlc), can provide further information on the metabolic state of diabetes.

In type II diabetic patients the catabolism of leucine is accelerated even in the absence of ketosis and that the urinary β - hydroxyisovaleric acid concentration is a useful marker of short term metabolic control in these patients. (Yu et al., 1990).

Hyperglycemia on the long run seems so important for the development of microangiopathy at the base of the famous diabetic triad (nephropathy, retinopathy and neuropathy) (Ver donk, 1990).

Glycemic control was best achieved in mild to moderate diabetics by the use of methi and the acceptability of fenugreek seeds was reasonable and there were no side effects (Wahal et al., 1993).

The drug gliclazide showed a significant reduction on serum cholesterol and triglyceride and also showed an insignificant reduction of 1.5 kg of body weight after treatment. Thus gliclazide is an effective and safe oral antidiabetes agent in the treatment of obese NIDDM (Madhavan et al., 1992).

2.3 Factors influencing blood glucose profile

Gelatinization may be one of the factors that determine the rate of starch digestion and hence glycemic response (Susan et al., 1987). The factors proposed as starchy foods

include the nature of starch, the starch protein interaction, fiber content and the presence of antinutrients such as α amylase inhibitors, phytates and lectins (Liener, 1982 ; Coulston et al., 1981; Griffiths, 1979 ; Anderson and Chen, 1979 and Liener, 1969).

A close relationship existed between the degree of gelatinization, and certain variables like rate of starch hydrolysis in vitro and postprandial glucose and insulin responses in humans (Ross et al., 1987). Antia (1989) has also observed that cooked whole rice gives a lower blood sugar rise compared to rice flour.

As stated by Ross et al (1987), the greater the starch gelatinization, the easier is the access of α amylase to the starch and rate of digestion and the subsequent, glycemic response. Digestion of food starch by pancreatic amylase can be increased by a more disorganised state of the starch molecule, obtained by heating or stirring in excess water. This maximally disorganised state is a function of swelling temperature, which depends on the type of starch used. (Guilbot and Mercier, 1985). Van (1982) found that gelatinization improves the digestability of starches.

According to Gannon et al (1986) carbohydrate composition may be a more important determinant of glycemic response than the digestability of the food. Gaal et al (1988) showed that in patients with NIDDM, 50 g glucose or lactose, have less than one and a half of the glycemic response. The intake of 1.75 g fructose per kg body weight by normal subjects with a postprandial blood concentration of 1.0 g/kg body weight of glucose or corn starch also produced large and significant increase in the insulin response that could not be accounted for by a concurrent increase in the glucose response (Reiser et al, 1987).

Hagander (1987) and Hagander et al (1987) also had reported that a mixture of sugar beet fibre with soluble pectin and insoluble polysaccharides would reduce the postprandial reactive hypoglycemia. According to Bukar et al (1990) fructose is known to elicit a lower glycemic response than sucrose and high fructose desserts have been recommended for a diabetic diet.

Glucose tolerance decrease with age and the decrease affects postprandial values more than fasting plasma glucose concentrations (Paris - Bockel et al., 1987). Jackson et al (1988) found that age related glucose tolerance was

characterised by delays in peripheral glucose uptake, and was predominantly the result of impaired peripheral glucose utilization.

As stated in many experiments glucose content of the food is a major determinant of the glycemic response of any food. If a meal contains very little glucose, it is likely that other food components will be converted to glucose and increase the blood glucose concentration (Thomas et al.,1989 and Gannon et al., 1986). According to Hollenbeck et al (1988) the day long plasma glucose response may not vary substantially when patients with NIDDM consumed meals of widely different glycemic potency and they had provided further evidence on the clinical benefits to be gained by designing meals based on the glycemic index of carbohydrate rich foods. Therefore carbohydrate appears to be the food component responsible for blood glucose increase after a meal while protein and fat seem to augment the glycemic response to food very little (Malone et al., 1976 and Nuttal et al., 1984). Simson et al (1985a) found that fat and protein markedly reduced the glycemic response to oral carbohydrate intake in non diabetics but in NIDDM patients, the presence of protein and fat had no significant effect on the glycemic response.

Gaal et al (1988) demonstrated an association of excess abdominal fat, even without obesity, with worse diabetic metabolic control, cardiovascular complications and blood lipid levels.

According to Wolever et al (1987a) mean glycemic index values obtained for foods are very similar in IDDM and NIDDM patients. In another experiment Wolever et al (1989), found that glycemic index values for the same food do not vary significantly between different individuals. According to Rasmussen et al (1992a) a valid estimate of the glycemic response in a single patient is obtained after a single meal.

The area under the glycemic response curve for each food is expressed as a per cent of the mean response to the standard food taken by the same subject, and the resulting values are averaged to obtain the glycemic index value for the food (Thomas et al., 1991b). The important variables that affect the glycemic index value obtained include food portion size, choice of standard food, repeated testing, of the standard food, frequency and length of time and method of blood sampling, method of area calculation and subject characteristics such as age, sex, body fatness, glucose tolerance status, dose and timing of insulin or oral

hypoglycemic agent, the degree of diabetes control and the fasting blood glucose values on the day of the test (Thomas et al., 1991b).

It was reasoned that the knowledge of glycemic effects of individual foods might be of use in understanding the physiologic effects of whole diets, since factors such as food form, particle size, nature of starch, food processing and antinutrients may have large effects on the physiological properties of foods (Thomas et al., 1991b).

A number of parameters like the mode of cooking and processing, the form of food and the difference in food processing and the difference in food constituents, which affected the digestion, absorption and metabolism seem to influence the glycemic and insulin responses (Viswanathan et al., 1988).

According to Simpson et al (1985b) cooking led to no significant change in insulin response in non-diabetics but led to lesser insulin response in NIDDM. Chithra and Thilaga (1989) observed different glycemic responses when a single food was prepared in different ways.

Collings et al (1981) showed that plasma glucose and insulin responses depend on particle size and cooking time

as well as the type of cooking process. Brand et al (1985) found lower responses to home cooked foods compared with their processed equivalents. According to Francis et al (1990) starch susceptability to amylose increased after cooking. Crapo and Henry (1988) found that there is a negative relationship between particle size and glycemic responses and grinding of cereals enhances digestability and increases glycemic response.

Such factors as food form, dietary fibre and the nature of carbohydrate have been shown to have a marked influence on postprandial glycemia and allowances cannot be made for these, in lists which take into account only the available carbohydrate content of foods (Nuttal et al., 1983).

Food portion size has a major effect on the glycemic index value because glycemic responses are related to the carbohydrate load (Jenkins et al., 1981b and Gannon et al., 1989).

High carbohydrate diets either low or high fibre content improve blood glucose levels. (Anderson, 1978 and Riccardi et al., 1984).

At any glucose dose level the serum insulin response was approximately 40 per cent greater in men compared to women (Macdonald and Williams, 1988).

The glycemic index approach will be useful in planning diets for diabetic people (Chew et al., 1988). Per and Gunnar (1989) described a negative correlation between pre-prandial blood glucose levels and glucose response in healthy as well as in diabetic subjects. This may be due to an increased spilling of glucose into the urine when the final threshold for reabsorption of glucose is exceeded (Wolever et al, 1985).

2.4 Diet in diabetes

There are three methods of treatment of diabetes mellitus and each involves an obligation for the patient to adhere to a dietary regime for the reminder of his life. They are by diet alone, diet and oral hypoglycemic drugs and diet and insulin. Approximately 40 per cent of new diabetes can be controlled adequately by diet alone, about 30 per cent require insulin and another 30 per cent will need an oral hypoglycemic drug (Stanley et al., 1973). According to Beebe (1987) the primary goal of diabetes management was to maintain blood glucose, therefore the dieticians play an integral role in identifying patterns in blood glucose profile and assisting the patients in making intelligent dietary choices to improve diabetes control.

In a proper diabetic diet, carbohydrate in the diet has to be increased with a reduction in the total fat and particularly saturated fat intake (National Institute of Nutrition, 1989a). According to Sue (1988) in a well balanced diet carbohydrate provided about 50 to 55 per cent of total kilo calories. In most traditional Indian diets carbohydrate or starchy foods provide 60 to 65 per cent of the total calories (Umesh 1993).

There was no significant loss of protein content, in cereals, pulses and combination preparations, due to various methods of cooking, where as a significant loss was observed in the case of lysine, tryptophan and sugar contents (Madhu and Mathews, 1985). The proteins in the food may alter the glycemic response (Dibildose et al., 1985). Recent information on calcium binding protein and 1, 25 dihydroxy vitamin D_3 (1, 25 $(OH)_2$ D_3) receptors in pancreas has led to the proposition that vitamin D may have some effect on the endocrine function of pancreas. After vitamin D administration, there was an improvement in glucose tolerance test (GTT) (National Institute of Nutrition, 1989b).

In type II diabetics without over hyperglycemia, omega 3 fatty acid supplementation does not improve either the

glycemic control or serum lipids and it is associated with a potentially detrimental rise in serum apo B concentrations (Kasim et al., 1988). They further stated that until more information is available, use of such supplementation should be discouraged.

With respect to the pattern of food intake, WHO (1985) reported that, dietary fat should be limited to approximately 30 per cent of total dietary intake, and foods containing polyunsaturated vegetable oils (eg. margarines and cooking oils) should be substituted for those containing saturated fats (eg., dairy products) and proteins should account for approximately 15 to 20 per cent of the daily intake and carbohydrate rich in natural fibre should constitute the remaining food energy.

Dietary calcium supplementation causes reduced intracellular calcium in type II diabetic hypertensives (Zimmet et al., 1990).

The natural presence of antinutrients in the slow release of carbohydrate foods is partly beneficial in the management of diabetes and hyperlipidemia. Such antinutrients are represented by phytic acid, lectins, tannins or enzyme inhibitors such as amylose and disaccharide inhibitors (Thompson, 1988).

Diet programmes for diabetic patients must be tailored to the cultural frame work and traditional foods with desirable characteristics could be encouraged (Gohdes, 1988).

According to Swaran (1992), the diabetic diet is as close to the normal diet as possible so as to meet the nutritional needs and the treatment of the individual patient by keeping carbohydrates low and adequacy in other food principles. Swaran (1992) further stated that roots and tubers, sweets, puddings and chocolates, fried foods, dried fruits and nuts, sugar and fruits like bananas, sapotas and custard apple have no place in a diabetic diet.

Dietary recommendations for diabetic patient should be highly individualized, patient's food preferances and support system should be taken into consideration (Reed and Mooradian, 1990).

American Diabetes Association (1986) reported that the diet should be presented in an appropriate level of energy, including sufficient calories for normal growth and development. American Dietetic Association (1987) stated that diet for any person with diabetes is always based on the nutritional needs of that individual for positive

health. The consistency of food whether liquid, puree or solid, raw or cooked affected starch digestability and there by glucose response (Seshiah et al., 1986).

The diet prescription for diabetic obese patients should be implemented in stages, with caloric restriction as the first priority, since weight loss itself diminishes hyperglycemia to normal combinations of foods and different processing or cooking, of the same food are also reported to produce different glycemic responses (Wheeler et al., 1987).

Since atherosclerosis is a major cause of disability and death among diabetics, prudent diets being advised to reduce the risk of arterial disease (Mann, 1984).

The inclusion of low glycemic index foods in the diet of diabetic patients may be an additional measure which slightly but favourably influences carbohydrate and lipid metabolism, requires only small changes in nutritional habits and has no known deleterious effects (Fontvieille et al., 1992).

The carbohydrate exchange list that has regulated the diets of many diabetics for over three decades may not reflect the physiological effect of foods (Nuttal et al., 1983).

A diabetic diet has to supply 30 kilo calories per kilogram of desirable body weight of the patient, 12 to 20 per cent of the total calories from protein and 50 to 60 per cent from carbohydrate (Corinnie and Marilyn, 1982).

Calories especially those from carbohydrates, are to be distributed to coincide with the type of insulin being used and modified according to each patient's needs in order to achieve best possible regulation of carbohydrate utilization. Use of intermediate or long acting insulins, demands the distribution of carbohydrate (usually 20 to 40 g) for a mid afternoon or bed time feeding or both. This carbohydrate is expected to be in slowly available form and should be accompanied by a portion of the day's protein with a distribution of 1/7, 2/7, 1/7, 2/7, and 1/7 of the total carbohydrates and calories for breakfast, lunch, afternoon snack, dinner and evening snack. (Corinnie and Marilyn, 1982).

Walshe et al (1987) studied about the energy restriction in non-insulin dependent diabetes mellitus and the results indicated that 3 months of reduction of energy intake with weight loss in newly diagnosed NIDDM patients improved cell responsiveness to glucose, but had no effect on liver glucose output or on peripheral insulin action.

Red gram juice is hypoglycemic, hypocholesteremic and it may help in the control of diabetes. It can be used daily in diabetic diets (Giri et al., 1986). Wolever et al (1987b) examined the glycemic effect of canned beans and reported that it significantly lowered the glycemic indices than those of white bread.

To reduce the variability, the standard food (white bread) should be repeated at least three times in a subject (Wolever et al., 1985).

The glycemic response to wheat products is affected by the processing conditions used. The more severe the processing conditions, the more rapid the digestion of starch. In this connection the degree of starch gelatinization is one important factor (Holm et al., 1989).

White and wholemeal spaghetti are reported to produce significantly flatter blood glucose levels than white or whole meal bread equivalents by Jenkins and Wolever (1981). According to Kolata (1982) the more homogenised the food, the more rapid is the rise in blood glucose. Jenkins et al (1983a) have also reported that flour in the form of spaghetti raised the blood glucose level much less than when the same amount of carbohydrate eaten as bread.

Wolever et al (1986) found that different types of pasta may produce different glycemic response but that these are not necessarily related to differences in cooking or surface area. Cooking and gelatinization of starch (ie., swelling of granules in the presence of heat and water) increase its susceptability to enzymic degradation in vitro and its availability for digestion and absorption in the small intestine (Snow and O'Dea, 1981).

According to Jenkins et al (1984) different starchy foods have been shown repeatedly to elicit widely different metabolic responses. Various carbohydrate foods have different absorption patterns and these are reflected by differences in glycemic response (Jenkins et al., 1986).

Starches from different foods resulted in different postprandial blood glucose level (Crapo et al, 1977). According to Crapo et al, (1977) potatoes give large postprandial glucose and insulin increases almost comparable to those of glucose. Jenkins et al (1980) have reported that different starchy foods produce different glycemic responses when fed individually.

Crapo et al (1976) has shown that complex carbohydrates are digested slowly and therefore induce lower glucose responses.

According to Janette et al., (1990) slow digestion and absorption of starch in traditional foods were factors that helped to protect susceptable populations from developing diabetics. Garg et al., (1992) found that patients with mild NIDDM and in high carbohydrate diets neither improve glycemic control nor insulin sensitivity and they raise plasma triglyceride and VLDL cholesterol concentrations and reduce HDL cholesterol levels which may not be desirable.

High carbohydrate diets, composed primarily of simple sugars, will result in exaggerated glucose tolerance in the late afternoon and should be avoided by patients with maturity onset diabetics (Shade et al., 1980).

The assumption that isocaloric diets with similar carbohydrate, proteins and fat composition derived from a variety of foods have similar effects on blood glucose is reported, to be incorrect by Chithra and Thilaga (1989) However dietary fibre is reported to play a vital role in controlling diabetes since it may lead to decreased postprandial glucose response probably by retarding gastrointestinal absorption of carbohydrates (Jenkins et al., 1978). According to Jenkins and Jenkins (1985) addition of purified fibre to carbohydrate test meals has shown to flatten the glycemic response in both normal and diabetic

volunteers, and reduce the insulin requirements in patients on the artificial pancreas and in the longer term reduce urinary glucose loss and diabetes control. They found the mechanism of action appears in part to be due to the effect of fibre in slowing absorption rather than by increasing colonic losses of carbohydrate.

The addition of various types of dietary fibre to oral water loads has been shown to lower postprandial circulating glucose levels in healthy human subjects (Morgan et al., 1990 and Trowell, 1972). Fibre rich foods cause slower stomach emptying, delay intestinal transit time, reduce rate of glucose absorption, lower blood sugar rise and decrease urinary glucose excretion (Antia, 1989). Viswanathan et al (1984) suggested that high fibre diets are suitable and effective virtually for adults with diabetes mellitus. Boisen et al (1985) showed that dietary fibre, depending its nature, will influence in vivo digestability by reducing the enzyme activity in the lumen and by protecting the enzymes against degradation. Dried beans because of their high fibre content and low glycemic index is especially suitable for diabetic diets (Vorster et al., 1987). The digestability of rice and cassava, based diets were reported to be same

although the total crude fibre content was lower in rice than cassava in diet (Beryl, 1991 and Nicol and Philips, 1978).

Viscous fibres must be intimately, mixed with the food to have action, relate to a reduced rate of digestion rather than carbohydrate malabsorption (Wolever et al., 1991). According to Eggum (1991) fibre will, generally stimulate microbial activity in the digestive tract and reduce transit time of the digesta.

According to Collier et al., (1986) the relative glycemic effects of mixed meals can be predicted from the glycemic index of their carbohydrate components, again stressing the importance of the type of carbohydrate in regulating postprandial blood glucose levels. According to Wolever et al (1985), mixing carbohydrate foods of different glycemic indices have resulted in the observed glycemic index of the mixed meal to be within 2 per cent of the expected value. In an another experiment Wolever et al (1988) have observed that the glycemic responses to breakfast were significantly lower on mornings after low glycemic index dinners than after high glycemic index dinners. Coulston et al (1987) showed that the plasma glucose response to mixed meals did not vary as a function

of the calculated glycemic potencies and concluded that glycemic response to a mixed meal cannot be predicted on the basis of the published values of the glycemic index of the individual carbohydrate foods included in the meal.

Torsdottier and Anderson (1989) have reported a significant increase in blood glucose during the initial 60 minutes when a mixed meal was taken with 300 ml of tap water compared with a meal without water intake. However Soren et al (1990) found that the volume of tap water taken with a meal as well as the eating time have no major impact on the glycemic and insulinemic response in NIDDM subjects.

Jenkins et al., (1988a) identified potentially and clinically useful starchy foods producing relatively flat glycemic responses and included legumes pasta ; grains such as barley, parboiled rice and bulgar (cracked) wheat and whole grain breads; which were associated with reduction in low density lipoprotein, cholesterol and triglyceride levels in hyperglycemia and with improved blood glucose control in insulin dependent patients. Low glycemic index starchy foods may be beneficial in the treatment of type 2 diabetics (Wolever et al., 1992).

Rasmussen et al (1992b) found that the blood glucose response areas to white bread were significantly higher than those of rice. An earlier study conducted by Crapo et al (1980) demonstrated that dextrose and potato elicited similar plasma glucose responses where as rice and bread elicited lower responses and corn an intermediate response. The glycemic index of fructose has been reported to be 20 per cent, much lower than most carbohydrate foods (Hung, 1989).

Ragi is reported to produce more postprandial glucose response than phulkas which is a refined wheat preparation (Chithra and Thilaga 1989). Addition of guar gum to either glucose drinks or carbohydrate rich foods is reported to reduce postprandial rise in blood glucose and plasma insulin in healthy diabetic subjects as reported by Ellis et al (1988).

According to Ranga Rao and Seshiah (1989) salt does not influence the absorption of glucose in diabetics.

Addition of moderate amounts of sucrose to a low glycemic index food may improve palatability without impairing the favourable effect on blood glucose and insulin response (Vorster et al., 1987). Jenkins et al (1988b)

suggested that inclusion of low glycemic index foods may be an additional measure that favourably influences carbohydrate metabolism without increasing insulin demand. One of the consequences of hyperglycemia in human diabetes mellitus is increased metabolism of glucose by the sorbitol pathway. This involves the reduction of glucose to sorbitol, catalysed by aldose reductase and the oxidation of sorbitol to fructose by sorbitol dehydrogenase (Taylor and Agius 1988).

Lower glycemic response was found in parboiled rice, whereas higher results were obtained in regular rice (Jenkins et al., 1983b).

Diabetics have been advised to increase the amount of whole meal and grained cereal products and reduce intake of more refined products (Kostudvalget and Landsforeningen, 1982).

According to Liener (1969) ten to twenty per cent of the starch in white wheat flour may be malabsorbed as judged by breath hydrogen production. Removal of gluten from wheat flour resulted in an increased rate of amylotic digestion in vitro and an enhanced glycemic response in vivo (Jenkins et al., 1987).

The activity of acarbose is attennuated when whole wheat is milled (Jenkins, 1979). Less carbohydrate was malabsorbed from breads made from flours in which natural starch protein interaction had been disrupted (Anderson et al., 1981).

Tapioca is composed of type 1 starch with 16 per cent amylose and a negligible quantity of protein (0.03 per cent). It is a highly digestible starch in vitro in humans. Tapioca express was used to supply manioc starch because it is a well characterised compound (Hood et al., 1978 and Colonna and Mercier 1983).

The relative area under the insulin response curve was greatest following the ingestion of meal containing cottage cheese and was least with egg white compared with that following glucose alone (Gannon et al., 1988).

Ingestion of low glycemic index meal revealed excellent inhibitory effects upon postprandial glycemic elevation in the elderly diabetics. This implies the clinical usefulness of a low glycemic index meal (bean) for postprandial glycemic regulation in elderly diabetes mellitus subjects (Goriya et al., 1990).

A low glycemic index programme meal may prolong endurance during strenuous exercise by inducing less postprandial hyperglycemia and hyperinsulinemia, lower levels of plasma lactate before and during exercise and by maintaining plasma glucose and free fatty acid at higher levels during critical periods of exercise (Thomas et al., 1991a).

Fenugreek diet increased the metabolic clearance rate and as a result, area under the curve (AUC) and half life of glucose were significantly reduced and there was an increase in the molar insulin binding sites with fenugreek diet (National Institute of Nutrition, 1990).

An ideal diet for diabetes is expected to consist predominantly of cereals like rice and wheat combined with pulses and two vegetables providing a sufficient amount of carbohydrate and fibre resulting in the reduction of cholesterol, triglycerides, weight of the individual, blood glucose levels and insulin doses and in the increase of sensitivity of tissues to insulin and level of glucose in the urine (Umesh, 1993).

MATERIALS AND METHODS

MATERIALS AND METHODS

A study was designed to assess the glycemic response to selected carbohydrate rich foods such as rice, wheat, tapioca and ragi, used in South Indian homes.

3.1 Selection of subjects

Twenty Non-Insulin Dependent Diabetes Mellitus (NIDDM) or type 2 diabetes subjects were selected for the study. The criteria followed for selection were:

1. Only men in the age group of 40 to 51 years.
2. An Oral Glucose Tolerance Test (OGTT) after 75 g glucose load was administered to assess their diabetic condition. Only those subjects with a fasting blood sugar level greater than 140 mg/dl and 200 mg/dl after 2 hours of glucose ingestion were selected.
3. Diabetic subjects familiar with Keralite diets were selected to eliminate body's adaptation to a food as a variable.
4. Type 2 diabetes patients who were healthy and were free from any other complication but using only oral drugs.

3.2 Plan of action

The plan of action of the present study consists of the following stages:

1. A pilot study among the hospital records to prepare a list of the patients with the desired characteristics.
2. Conduct of an oral glucose tolerance test among the patients listed and to select those who showed the desired blood sugar levels.
3. Formulating the experimental diets.
4. Administering four types of experimental diets formulated.
5. Recording the fasting and postprandial blood sugar levels at half an hour intervals upto two hours, and
6. Analysing the glycemic response of each diet.

3.3 Conduct of the study

3.3.1 Screening patients for the study

A pilot study among the hospital records was conducted and 120 patients were located and tested for blood sugar levels. Oral glucose tolerance test was given to 63 patients. From this 20 patients were selected for the experiment.

3.3.2 Formulation of experimental lunches

Four experimental lunches were planned for the study. For this purpose a model diet was planned, following the standard procedures (National Institute of Nutrition, 1993). Nutritive value of the model diet planned is given in Appendix III

The experimental lunches planned were vegetarian diets. Rice, wheat, tapioca and ragi were served as the staple foods in these experimental lunches. The remaining dietary constituents were common in all the four experimental lunches. Methods of preparation were also uniform. Ingredients of each lunch are presented in Table.1.

Table.1 Composition of experimental lunches

Food stuffs	Quantity (gms)
a. Staple food items *	
Rice	96.90
Wheat	105.00
Ragi	104.00
Tapioca	196.80
b. Pulses and legumes	
Green gram	40.00
Red gram dhal	10.00
c. Leafy vegetables	
Amaranth	50.00
d. Roots and Tubers	
Onion	20.00
e. Other vegetables	
Cucumber	10.00
Bittergourd	30.00
f. Nuts and oil seeds	
Coconut	15.00
g. Fruits	
Tomato	20.00
Guava	100.00
h. Milk and milk products	
Curd	120.00
i. Cooking oil	2.00

* Any one of the staple foods was included in the experimental lunches. Other constituents were common in all the four lunches.

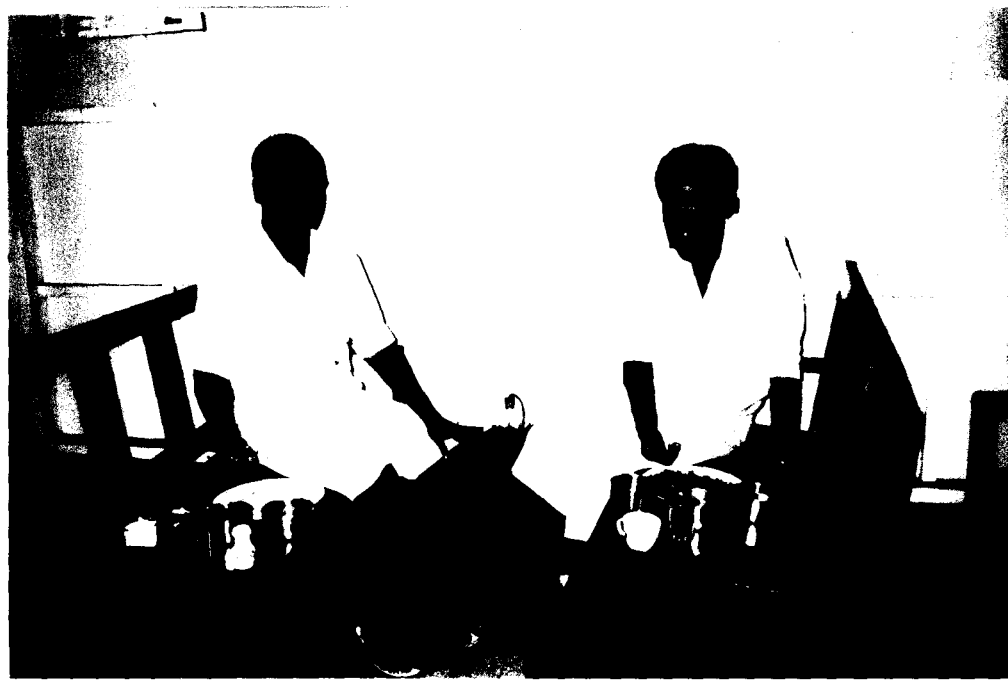
The experimental lunches planned were almost isocaloric (771 Kilo calories) and contained 75 g equivalent of carbohydrate load (as glycemic response was determined in comparison with glucose load) in the form of staple foods. Each experimental lunch supplied 3/7 of the daily caloric requirement of an adult diabetes patient weighing 60 kg of body weight. Nutritive values of the experimental lunches were given in Appendix IV.

Each lunch was prepared independently for each subject, after accurately weighing the raw food ingredients. Care was taken to prepare the lunch under hygienic conditions. Methods of preparation administered is presented in Appendix V.

3.3.3 Administration of glucose and experimental lunches

The subjects were asked to avoid drugs for 24 hours and to fast overnight (12 hours) and report at the clinic at 9.00 am without taking any solid food. They were allowed to take only one or two cups of tea or coffee without sugar, before administering the lunches. Blood samples were collected intravenously to determine their fasting blood glucose level. After this the diets were administered.

Fig.1. ADMINISTRATION OF EXPERIMENTAL LUNCHES BASED ON RICE AND WHEAT



The subjects were requested to consume the entire meal. Time alloted for consuming the entire meal was 30 minutes. They were also requested to remain non-active and non-smoking during the test period of 2 hours.

The administration of glucose (75 g) load served as the control of the experiment. An interval of at least two days was given between the successive lunches.

3.3.4 Collection and analysis of blood samples

Blood samples were drawn intravenously before the administration of the lunches and every 30 minutes for 120 minutes ie., at ½ hr, 1 hr, 1½ hr and 2 hr after the administration of each lunch. The samples with a pinch of NaF were centrifuged and stored at 4 ^{o}C.

Blood plasma glucose concentration of the collected samples were assayed by using a colorimetre (Boehringer Mannheim blood sugar testing reagent kit - GOD-PAP method). The method is presented in Appendix VI.

3.3.5 Calculation of glycemic response

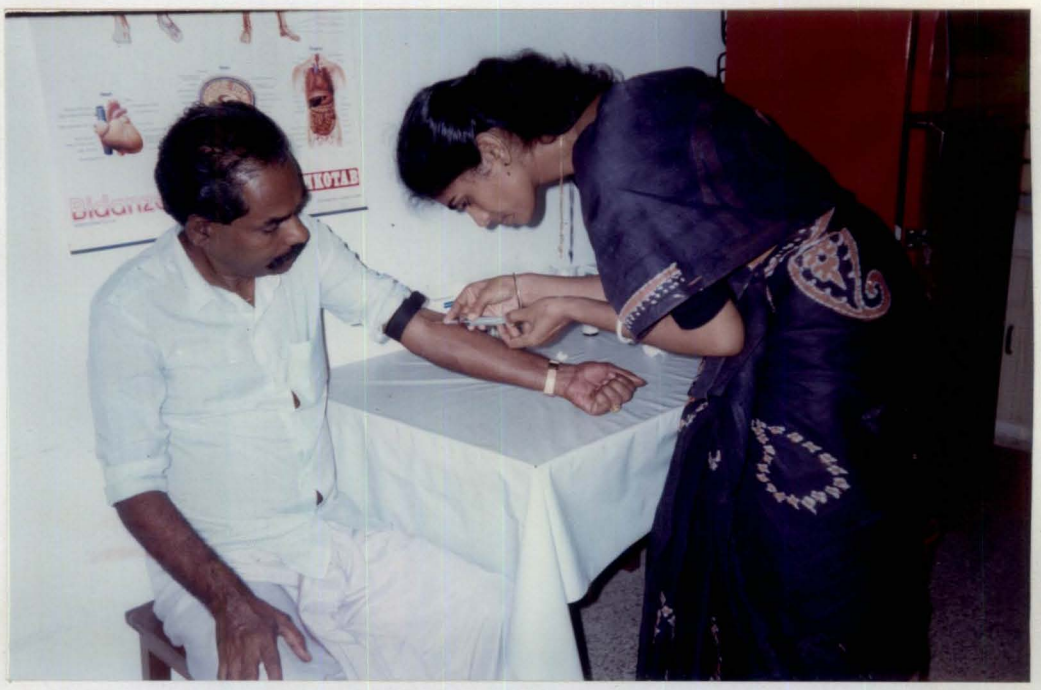
The area under the 2 hr glucose stimulation curve (AUC) of each lunch was calculated statistically after fitting the quadratic regression equation $y = a + bx + cx^2$ where, a, b

Fig.1. ADMINISTRATION OF EXPERIMENTAL LUNCHES BASED ON RICE AND WHEAT

Fig.2. ADMINISTRATION OF EXPERIMENTAL LUNCH BASED ON TAPIOCA

Fig.3. ADMINISTRATION OF EXPERIMENTAL LUNCH BASED ON RAGI

Fig.4. COLLECTION OF BLOOD SAMPLES



and c were the blood sugar levels at different time intervals and x the time at which the blood sugar level was found to decrease.

Glycemic Response (GR) of each lunch was calculated by using the formula:

$$GR = \frac{\text{AUC of test food}}{\text{AUC of reference food (glucose)}}$$

3.3.6 Statistical analysis

Results of the study were subjected to analysis of variance (ANOVA) to compare the plasma glucose concentrations at different time intervals after each lunch and also to compare the peak rise over the fasting blood plasma glucose levels after each lunch. Means were tested for significance by critical difference.

Correlation studies were done to find out the relationship between glycemic response and peak rise over the fasting blood plasma glucose concentrations after each lunch.

Relationship between glycemic response (y) and plasma glucose concentrations (x) were analysed by using the simple linear regression equation, "y = a + bx" where b is the rate of change of `y' for unit change in `x'.

RESULTS

RESULTS

The study entitled "Glycemic response to selected carbohydrate rich foods in diabetics", is related to the information regarding glycemic response to selected staple foods included in standard lunches. This was assessed in twenty Non-Insulin Dependent Diabetes Mellitus (NIDDM) subjects. Data collected were analysed and the major findings of the study are presented under the following headings:

4.1 Personal characteristics of the selected subjects and Oral Glucose Tolerance Test (OGTT)

4.2 Experimental lunches

4.3 Effect of experimental lunches on plasma glucose response.

4.3.1 Lunch with rice as staple food

4.3.2 Lunch with wheat as staple food

4.3.3 Lunch with tapioca as staple food and

4.3.4 Lunch with ragi as staple food

4.3.5 Mean plasma glucose values of the subjects

4.3.6 Mean peak rise over the fasting levels after the administration of glucose and experimental lunches.

4.3.7 Comparative changes of each lunch.

4.4 Glycemic response of the experimental lunches and

4.5 Relationship of glycemic response to plasma glucose concentration.

4.1 Personal characteristics of the subjects selected and oral glucose tolerance test

The personal characteristics of the selected subjects are listed in table 2. The selected subjects had a mean age of 45.7 ± 3.25 years ranging from 41 to 50 years. Forty five per cent of the subjects come under the age group of 40 to 45 years and 55 per cent of them were from the age group of 46 to 50 years.

Information related to the duration of the disease in selected subjects revealed that 50 per cent of the subjects were suffering from this disease for the past 4 to 6 years, 35 per cent for 7 to 10 years and only 15 per cent for the past 1 to 3 years.

Obesity and diabetes are expected to have the most frequent pathological association. The obese subjects have reduced glucose tolerance. Body mass index (wt/ht^2) helps to diagnose whether a diabetic is obese or not. In this study, from the data pertaining to body mass index, it was found that 30 per cent were found to be obese (BMI≥ 27) and the remaining 70 per cent of them were normal. The mean body mass index of the subjects was 25.4 ± 4.5.

Table 2 Personal characteristics of the selected Non Insulin Dependent Diabetes Mellitus subjects

Characteristics of the subjects	Details of the subjects	Per cent of the subjects
a. **Age (Years)**		
40-45	9	45
46-50	11	55
Total	20	100
b. **Duration of the disease (years)**		
1-3	7	35
4-6	10	50
7-10	3	15
Total	20	100
c. **Body Mass Index (kg/m^2)**		
< 27	14	70
≥ 27	6	30
Total	20	100

Table 3 details the chemical nature and common names of the oral hypoglycemic agents used by the subjects. These oral hypoglycemic agents include sulphonylurea with active principle of glibenclamide such as euglucon, glynase and daonil and biguanides with an active principle of phenformin and metformin, such as DBI-TD and glyciphage respectively.

Table 3 Oral hypoglycemic agents used by the subjects

Name of the drug	Chemical nature	Per cent of subjects
Euglucon	Sulphonylurea (Active principle-glibenclamide)	45
Glynase	Sulphonylurea	
Daonil	Sulphonylurea	
DBI - TD	Biguanide (Active principle - Phenformin)	5
Glyciphage	Biguanide (Active principle - Metformin)	
	A combination of sulphonylurea and biguanides	50
	Total	100

Majority of the subjects (50 per cent) used a combination of sulphonylurea and biguanides, 45 per cent of the subjects depended on sulphonylurea alone and only 5 per cent used biguanides as oral hypoglycemic agents. None of the subjects had reported the usage of diet as a therapeutic measure to control hyperglycemia.

Prior to the actual experiment an oral glucose tolerance test (75 g) was conducted on all the subjects to assess their metabolic response to glucose load and for its comparison with the glycemic response to the selected staple foods.

The table 4 presents the mean plasma glucose values of the subjects after oral glucose tolerance test (OGTT) and individual particulars are presented in Appendix VII.

From the table it was found that fasting blood sugar level before oral glucose tolerance test ranged from 140 to 287 mg/dl with a mean fasting blood sugar level of 193.5 ± 36.94 mg/dl. After ½ hr the range of blood sugar level was found to be from 202 to 400 mg/dl with a mean blood sugar level of 284.4 ± 47.92 mg/dl indicating a rise. There was a further increase in the blood sugar level which was in the range of 281 to 415 mg/dl with a mean level of 346.4 ± 35.28

Table 4 Mean plasma glucose values of the subjects after oral glucose tolerance test.

Interval (hrs)	Range of blood sugar level (mg/dl)	Mean blood sugar level (mg/dl)	Standard Error (SE) (mg/dl)
	Fasting blood sugar level		
	140 - 287	193.5	36.94
	Blood Sugar levels at intervals (hrs)		
½	202 - 400	284.4	47.92
1	281 - 415	346.4	35.28
1 ½	262 - 410	341.1	38.07
2	245 - 400	324.1	44.44
	Peak rise over the fasting level		
	100 - 223	164.5	31.55

mg/dl. However a trend to decrease was observed since after. At 1½ hr the blood sugar levels were observed to range from 262 to 410 mg/dl with a mean blood sugar level of 341.1 ± 38.07 mg/dl. The blood sugar values after 2 hrs were further lowered to 245 to 400 mg/dl with a mean level of 324.1 ± 44.4 mg/dl. The peak rise of blood sugar over the fasting blood sugar level was in the range of 100 to 223 mg/dl. The mean peak rise over the fasting blood sugar at this time was 164.5 ± 31.55 mg/dl.

Table 5 indicates the time of least glucose tolerance during oral glucose tolerance test (OGTT). The mean peak blood plasma glucose rise of 65 per cent the subjects at 1 hr was 163.15 ± 29.7 mg/dl. It was also found that 5 per cent, 25 per cent and 5 per cent of the subjects showed the

Table 5 Time of least glucose tolerance during oral glucose tolerance test (OGTT)

Time (hrs)	Per cent of subjects	Peak rise of blood sugar level over the fasting level (mg/dl)(mean±SE)
½	5	113.00 ± 0
1	65	163.15 ± 29.7
1 ½	25	183.00 ± 22.52
2	5	141.00 ± 0
Total	100	

peak rise at ½, 1½ and 2 hrs respectively and their mean peak rise in blood sugar level was 113, 183 ± 22.5 and 141 mg/dl respectively.

Table 6 shows the influence of glucose (of OGTT) on the peak rise of blood sugar over the fasting level. Fifty five per cent of the subjects showed the peak rise of 100 to 150 mg/dl and 35 per cent and 10 per cent of them showed 150

Table 6 Influence of glucose (of OGTT) on the peak rise of blood sugar over the fasting level.

Peak rise over the fasting blood sugar level (mg/dl)	Per cent of subjects
100 - 150	55
151 - 200	35
201 - 250	10
Total	100

to 200 mg/dl and 200 to 250 mg/dl respectively over the fasting blood plasma glucose levels.

The relationship between blood sugar levels(y) at various time intervals(x), explained by the quadratic regression equation $y = 194.31 + 3.69x - 0.0217x^2$ was found to be highly significant and gave an optimum time of 85.21 minutes, after which the blood sugar level decreased considerably. Sixty six per cent of the variation in blood sugar level can be attributed to the above fitted regression relationship between x and y.

4.2 Experimental lunches

Four experimental lunches planned for the study were isocaloric and similar, and supplied 75 gm of glucose in the form of rice, wheat, tapioca and ragi.

4.3 Effect of experimental lunches on plasma glucose response

4.3.1 Lunch with rice as staple food

Table 7 presents the mean plasma glucose values of the subjects after the rice based experimental lunch and individual particulars are presented in Appendix VIII.

Table 7 Mean plasma glucose values of the subjects after the rice based lunch

Interval (hrs)	Range of blood sugar level (mg/dl)	Mean blood sugar level (mg/dl)	Standard Error (SE) (mg/dl)
	Fasting blood sugar level		
	140 - 304	195.40	44.65
	Blood Sugar levels at intervals (hrs)		
½	183 - 552	296.05	74.10
1	244 - 537	345.05	72.45
1 ½	233 - 537	328.20	81.11
2	175 - 528	316.05	87.38
	Peak rise over the fasting blood sugar level		
	98 - 266	159.10	42.32

From the table it was found that fasting blood sugar level, before the administration of experimental lunch with rice as staple food, was observed to range from 140 to 287

mg/dl with a mean blood sugar level of 193.50 ± 36.94 mg/dl. Half an hour after the administration of the lunch the blood sugar level elevated to a range of 183 to 552 mg/dl with a mean blood sugar level of 296.05 ± 74.10 mg/dl and that after 1 hr from 244 to 537 mg/dl with a mean blood sugar level of 345.05 ± 72.45 mg/dl. A decline in blood glucose level was observed after this period with a range of blood sugar levels from 233 to 537 mg/dl with a mean blood sugar level of 328.20 ± 81.11 mg/dl after 1 ½ hr and from 175 to 528 mg/dl with mean of 316.05 ± 87.38 mg/dl after 2 hrs. It was found that the peak rise over the fasting blood sugar level ranged from 98 to 266 mg/dl with a mean of 159.10 ± 42.22 mg/dl.

Table 8 indicates the time of least glucose tolerance after the administration of rice based experimental lunch. The table revealed that 15 per cent of the subjects showed the peak rise of blood sugar level at ½ hr postprandially. Their mean peak rise over fasting blood sugar level was 149 ± 73.08 mg/dl. The majority of the subjects (75 per cent) indicated the peak rise at 1 hour postprandially and their mean plasma glucose level at this stage was 153.13 ± 33.58 mg/dl. Only five per cent of the subjects reached the peak level at 1 1/2hours and another 5 per cent of the subjects

had this at 2 hours. Their changes in plasma glucose level over the fasting were 266 mg/dl and 171 mg/dl respectively.

Table 8 Time of least glucose tolerance after the administration of rice based experimental lunch

Time (hrs)	Per cent of subjects	Peak rise of blood sugar level over the fasting level (mg/dl) (mean ± SE)
½	15	145.00 ± 73.08
1	75	153.13 ± 33.58
1 ½	5	266.00 ± 0
2	5	171.00 ± 0
Total	100	

Table 9 shows the peak rise of blood sugar level when the lunch contained rice as staple. The results reveal that only 5 per cent of the subjects had a peak rise of blood sugar level below 100 mg/dl. However, that of 40 per cent,35 per cent and 25 per cent were on an average peak rise of blood sugar level of 100 to 150, 151 to 200 and 201 to 250 mg/dl respectively.

Table 9 Influence of rice based lunch on the peak rise of blood sugar over the fasting level

Peak rise over the fasting blood sugar level (mg/dl)	Per cent of subjects
<100	5
100-150	40
151-200	35
201-250	20
Total	100

Table 10 indicates the relationship between the blood sugar level at various time intervals after the consumption of the experimental lunches and glucose (of OGTT).

The relationship between the blood sugar level (y) at various time intervals (x) has been explained by the quadratic regression equation $y = 199.85 + 3.68x - 0.31x^2$ which was found to be highly significant and gave an optimum time of 79.77 minutes, beyond which the blood sugar level decreased. Thirty four per cent of the variation in blood sugar level was attributed to the above fitted regression equation (Table 10).

Table 10 Relationship between the blood sugar level (y) at various time intervals (x)

Food	Regression equation	Optimum time(mins)	Coefficient of determination (per cent)
Glucose	$y = 194.31 + 3.69x - 0.0217x^2$	85.21	66
Rice	$y = 199.85 + 3.68x - 0.0231x^2$	79.77	34
Wheat	$y = 203.14 + 2.44x - 0.0167x^2$	72.92	17
Tapioca	$y = 203.02 + 4.47x - 0.0306x^2$	73.02	52
Ragi	$y = 204.55 + 2.88x - 0.0196x^2$	73.32	19

4.3.2 Lunch with wheat as staple food

Table 11 presents the mean plasma glucose values of the subjects after the consumption of wheat based experimental lunch and individual particulars are presented in Appendix IX.

Table 11 Mean plasma glucose value of the subjects after the wheat based experimental lunch.

Interval (hrs)	Range of blood sugar level (mg/dl)	Mean blood sugar level (mg/dl)	Standard Error (SE) (mg/dl)
	Fasting blood sugar level		
	140 - 363	200.15	55.39
	Blood sugar levels at intervals (hrs)		
½	193 - 436	266.35	68.10
1	211 - 440	291.90	69.85
1 ½	152 - 429	278.55	73.79
2	158 - 402	258.60	71.80
	Peak rise over the fasting blood sugar level		
	41 - 196	103.65	39.38

From the table it was found that the fasting blood sugar level before the administration of experimental lunch with wheat as staple food ranged from 140 to 363 mg/dl. The subjects had a mean fasting blood sugar level of 200.15 ±

55.39 mg/dl. Half an hour after the administration of the lunch, the blood sugar levels were observed to be in the range of 193 to 436 mg/dl with a mean blood sugar level of 266.35 ± 68.10 mg/dl. After 1 hr there was increase in the blood sugar level since the range was from 211 to 440 mg/dl with a mean blood sugar level of 291.9 ± 69.85 mg/dl. However after 1½ hr the blood sugar levels observed to decline as the blood sugar levels ranged from 152 to 429 mg/dl with a mean of 278.55 ± 73.79 mg/dl. The blood sugar level at 2 hr after the administration of lunch with wheat as staple food further declined and the range was observed to be from 158 to 402 mg/dl with a mean blood sugar level of 258.60 ± 71.80 mg/dl. It was also found that the peak rise over the fasting blood sugar level ranged from 41 to 196 mg/dl with a mean blood sugar level of 103.65 ± 39.38 mg/dl.

Table 12 indicates the time of least glucose tolerance after the consumption of wheat based experimental lunch. The peak blood sugar level (100.43 ± 34.09) over the fasting level was indicated at 1 hour in the majority (70 per cent) of the subjects. Ten per cent of them, however, reached this point (69.5 ± 28.5 mg/dl) at the first half hour itself while a peak level of 132 ± 38.34 mg/dl, of the remaining 20 per cent was observed after 1½ hours.

Table 12 Time of least glucose tolerance after the administration of wheat based experimental lunch

Time (hrs)	Per cent of subjects	Peak rise of blood sugar level over the fasting level (mg/dl) (mean±SE)
½	10	69.50 ± 28.50
1	70	100.43 ± 34.09
1½	20	132.00 ± 38.34
Total	100	

It was also found that when compared to glucose the wheat intake recorded a lower blood sugar rise and after 2 hours, wheat consumption recorded in much lower blood sugar level than after the consumption of glucose.

Table 13 shows the influence of wheat based lunch on the peak rise of blood sugar level over the fasting level. The peak rise of blood sugar levels over the fasting level showed that 60 per cent of the subjects experienced a peak rise of plasma glucose level over the fasting level below 100 mg/dl. Twenty five per cent of the subjects showed a difference between the peak rise and fasting level between 100 to 150 mg/dl and 15 per cent of the cases showed a difference between 151 and 200 mg/dl.

Table 13 Influence of wheat based lunch on the peak rise of blood sugar over the fasting level

Peak rise over the fasting blood sugar level (mg/dl)	Per cent of subjects
<100	60
100-150	25
151-200	15
Total	100

The relationship between blood sugar level (y) at various time intervals (x) after the consumption of wheat based lunch, explained by the quadratic regression equation $y = 203.14 + 2.44x - 0.0167x^2$ which was found to be highly significant and gave an optimum time of 72.92 minutes beyond which the blood sugar level was found to decrease. Eighteen per cent of the variation in blood sugar level was attributed to the above fitted regression equation (Table 10).

4.3.3 Lunch with tapioca as staple food

Table 14 shows the mean plasma glucose value of the subjects after the consumption of tapioca based experimental lunch and individual particulars are presented in Appendix X.

Table 14 Mean plasma glucose values of the subjects after the tapioca based experimental lunch.

Interval (hrs)	Range of blood sugar level (mg/dl)	Mean blood sugar level (mg/dl)	Standard Error (SE) (mg/dl)
	Fasting blood sugar level		
	142 - 296	199.45	43.73
	Blood sugar levels at intervals (hrs)		
½	212 - 412	312.10	51.24
1	292 - 488	375.35	53.54
1 ½	257 - 452	336.25	60.93
2	216 - 428	307.05	62.50
	Peak rise over the fasting blood sugar level		
	110 - 238	181.05	36.21

As indicated in the table, the blood sugar level before the administration of experimental lunch with wheat as staple food ranged from 142 to 296 mg/dl with a mean blood sugar level of 199.45 ± 47.73 mg/dl. Half an hour after the administration of the lunch, the blood sugar level ranged from 212 to 412 mg/dl with a mean of 312.1 ± 51.24 mg/dl. After 1 hr there was an increase in blood sugar which ranged from 292 to 488 mg/dl with a mean blood sugar level of 375.35 ± 53.54 mg/dl. At 1½ hour the blood sugar levels showed a trend to decrease as they ranged from 257 to 452

mg/dl with a mean blood sugar level of 336.25 ± 60.93 mg/dl. An observation after 2 hrs revealed that blood sugar level ranged from 216 to 428 mg/dl with a mean blood sugar level of 307.05 ± 62.50 mg/dl. The difference between the fasting and peak values for individual subjects ranged from 110 mg/dl to 238 mg/dl and on an average the peak rise over the fasting levels was over 181.05 ± 36.21 mg/dl.

Table 15 indicates the time of least glucose tolerance after the administration of tapioca based experimental lunch. The majority (85 per cent) of the subjects reached the peak level (183.23 ± 37.58 mg/dl) of blood sugar at 1 hr after the lunch. The remaining 5 per cent and 10 per cent of

Table 15 Time of least glucose tolerance after the administration of tapioca based experimental lunch.

Time (hrs)	Per cent of subjects	Peak rise of blood sugar level over the fasting level (mg/dl) (mean ± SE)
½	5	136.00 ± 0
1	85	183.23 ± 37.58
1½	10	161.00 ± 0
Total	100	

the subjects reached this maximum (136 and 161 mg/dl)level at ½ hr and 1½ hr respectively after the consumption of the lunch.

Table 16 indicates the peak rise of blood sugar level over the fasting blood sugar level after consuming the experimental lunch on tapioca. Among the subjects, 45 per cent showed a peak rise of blood sugar level over the fasting blood sugar level between 201 and 250 mg/dl. While 35 per cent showed a peak rise of blood sugar level over the fasting level between 151 and 200 mg/dl and 20 per cent between 100 and 150 mg/dl.

Table 16 Influence of tapioca based lunch on the peak rise of blood sugar over the fasting level.

Peak rise over the fasting blood sugar level (mg/dl)	Per cent of subjects
100-150	20
151-200	35
201-250	45
Total	100

Tapioca and glucose showed similar trends of change in blood plasma glucose levels, the sudden increase in the

blood glucose levels upto 1 hour was higher in tapioca than that of feeding glucose.

The relationship between blood sugar level at various time intervals, explained by the quadratic regression equation $y = 203.02 + 4.47x - 0.0306x^2$ was found to be highly significant and gave an optimum time of 73.02 minutes beyond which the blood sugar level was found to decrease. Fifty two per cent of the variation in blood sugar level was attributed to the above fitted regression equation (Table 10).

4.3.4 Lunch with ragi as staple food

Table 17 shows the mean plasma glucose values of the subjects after the consumption of ragi based experimental lunch and individual particulars are presented in Appendix XI.

From the table it was found that the blood sugar level,before the administration of experimental lunch with ragi as staple food, ranged from 155 to 334 mg/dl with a mean blood sugar level of 205.6 ± 58.09 mg/dl. Half an hour after the administration of the lunch, the range of blood sugar level was found to be from 195 to 498 mg/dl with a mean blood sugar level of 281.7 ± 73.26 mg/dl indicating a

Table 17 Mean plasma glucose values of the subjects after the ragi based experimental lunch

Interval (hrs)	Range of blood sugar level (mg/dl)	Mean blood sugar level (mg/dl)	Standard Error (SE) (mg/dl)
	Fasting blood sugar level		
	155 - 334	205.56	58.09
	Blood sugar levels at intervals (hrs)		
½	195 - 498	281.70	73.26
1	233 - 561	313.80	84.92
1½	205 - 548	299.45	90.43
2	140 - 517	268.65	91.29
	Peak rise over the fasting blood sugar level		
	66 - 284	114.55	59.64

rise. There was a further increase in the blood sugar level at 1 hr after the administration of lunch which was in the range of 233 to 561 mg/dl, then the mean blood sugar level was 313.8 ± 84.92 mg/dl. At 1½ hrs the blood sugar levels showed a trend to decrease. Then the blood sugar levels ranged from 205 to 548 mg/dl with a mean blood sugar level of 299.45 ± 91.29 mg/dl. The blood sugar level at 2 hr after the administration of lunch with ragi as staple food ranged from 140 to 517 mg/dl with a mean blood sugar level of 268.65 ± 91.29 mg/dl. The change in plasma glucose levels

between the fasting and peak values for individual subjects ranged from 66 mg/dl to 284 mg/dl and on an average the peak rise over the fasting levels was 144.55 ± 58.13 mg/dl.

Table 18 indicates the time of least glucose tolerance after the administration of ragi based experimental lunch. After the lunch, the majority of the subjects (80 per cent) reached the peak level over the fasting level at 1 hr. Only 10 per cent of the subjects reached the peak rise over the fasting blood sugar level at the first 30 minutes of the experiment with a mean peak rise of 81 ± 1.41 mg/dl

Table 18 Time of least glucose tolerance after the administration of ragi based experimental lunch.

Time (hrs)	Per cent of subjects	Peak rise of blood sugar level over the fasting level (mg/dl) (Mean ± SE)
½	10	81.00 ± 1.41
1	80	111.50 ± 60.18
1½	10	172.50 ± 64.35
Total	100	

over the fasting level while another 10 per cent of the subjects showed the same level at 90 minutes after the

consumption of the food with a mean peak rise of blood sugar level of 172.5 ± 64.35 mg/dl. After 2 hours the blood sugar level was found to decrease.

Table 19 reveals the peak rise of blood sugar level over fasting blood sugar level after consuming the experimental lunch on ragi. It was found that 70 per cent of the subjects showed a peak rise of blood sugar level over the fasting values below 100 mg/dl. Only 10 per cent showed it between 101 and 150 mg/dl. Five per cent of the subjects depicted this value varying between 200 and 250 mg/dl and that of another 5 per cent was between 251 and 300 mg/dl.

Table 19 Influence of ragi based lunch on the peak rise of blood sugar over the fasting level

Peak rise over the fasting blood sugar level (mg/dl)	Per cent of subjects
⩽100	70
101-150	10
151-200	10
201-250	5
251-300	5
TOTAL	100

The relationship between blood sugar levels at various time intervals explained by the quadratic regression equation $y = 204.55 + 2.88x - 0.0196x^2$ was found to be highly significant and gave an optimum time of 73.32 minutes beyond which the blood sugar level was found to be decreased. Nineteen per cent of the variation in blood sugar level was attributed to the above fitted regression equation (Table 10).

It was found that eventhough the experiment was started with almost the same fasting blood plasma glucose levels, after 2 hrs. ragi showed much lower value of plasma glucose level when compared to that of feeding glucose.

4.3.5 Mean plasma glucose values of the subjects

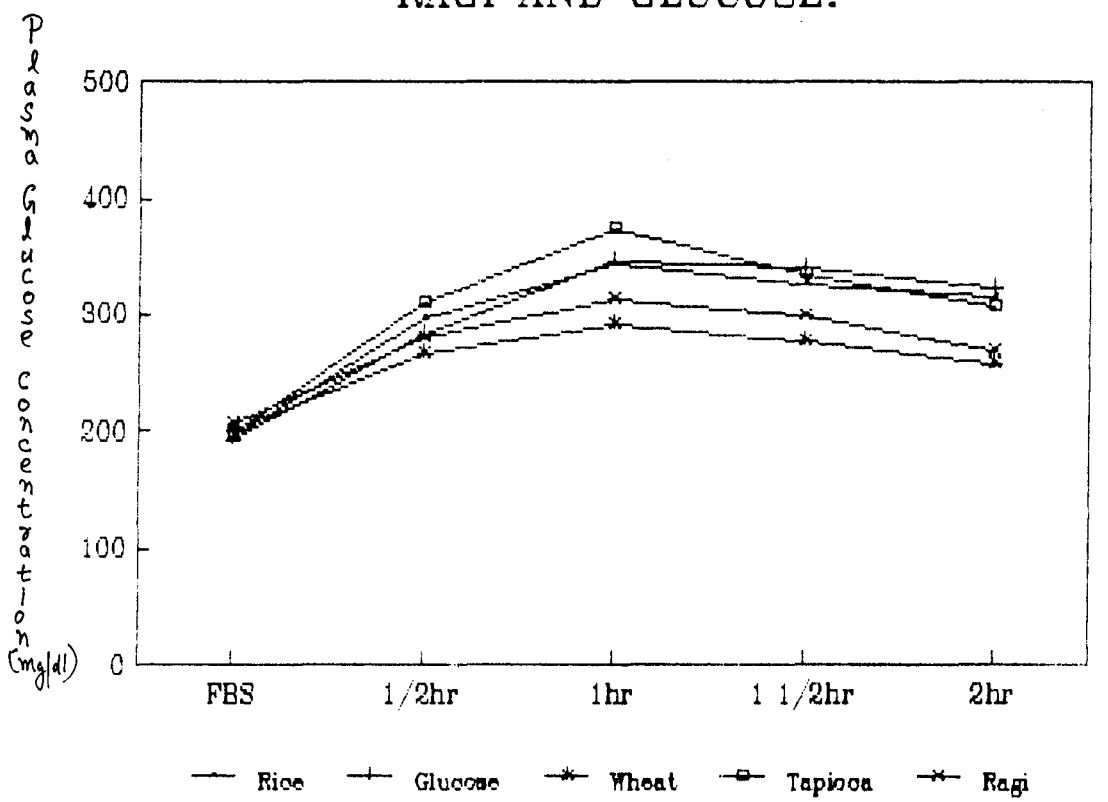
The mean plasma glucose response of the subjects with different lunches are presented in table 20 and is illustrated in figure 5.

The mean plasma glucose values of the subjects after consuming rice based lunch were 296.05 ± 74.1, 345.05 ± 72.5, 328.2 ± 81.1 and 316.1 ± 87.4 mg/dl at ½ hr, 1 hr, 1½ hr and 2 hr postprandially with 195.4 ± 44.7 mg/dl as fasting blood sugar level. While, for wheat it was 266.35 ± 68.1, 291.5 ± 69.9, 278.6 ± 73.8, 258.6 ± 71.8 mg/dl at the

Table 20 Plasma glucose values of the subjects to the different experimental lunches

Lunches tested	Plasma glucose (mg/dl) (mean ± SE)				
	Fasting blood sugar level	½ hr	1hr	1½ hr	2 hr
Glucose	193.5 ± 36.9	284.4 ± 46.9	346.4 ± 35.3	341.1 ± 38.1	324.1 ± 44.4
Rice	195.4 ± 44.7	296.1 ± 74.1	345.1 ± 72.5	328.2 ± 81.1	316.1 ± 87.4
Wheat	200.2 ± 55.4	266.4 ± 68.1	291.5 ± 69.9	278.6 ± 73.8	258.6 ± 71.8
Tapioca	199.5 ± 47.7	312.1 ± 51.2	375.4 ± 53.5	336.3 ± 60.9	307.1 ± 62.5
Ragi	205.6 ± 58.1	281.7 ± 73.3	313.8 ± 84.9	299.5 ± 90.4	268.7 ± 91.3

FIG.5.2 HOUR PLASMA GLUCOSE TOLERANCE CURVE OF RICE, WHEAT, TAPIOCA RAGI AND GLUCOSE.



same time intervals postprandially with 200.2 ± 55.4 mg/dl as fasting blood sugar level. It was also observed from the table, that the fasting blood sugar level for rice was lower than that for wheat and the peak rise over the fasting blood sugar level of the former was much higher than that of wheat. This trend was also noted in the case of tapioca. When the experiment on tapioca was started, the subjects had a mean fasting blood sugar level of 199.5 ± 47.7 mg/dl which increased to 312.1 ± 51.2 and 375.35 ± 53.5 mg/dl at the first 30 and 60 minutes respectively. At 90 and 120 minutes postprandially the blood sugar levels decreased to 336.25 ± 60.9 and 307.1 ± 62.5 mg/dl respectively.

The experimental lunch with ragi as the staple food showed a trend almost similar to that with wheat. The experiment was started with a mean fasting blood sugar level of 205.6 ± 58.1 mg/dl which increased to 281.7 ± 73.3 and 313.8 ± 84.9 mg/dl at ½ hr and 1 hr after the consumption of lunch. Then there was a decrease in blood sugar level from 299.5 ± 90.4 mg/dl at 1½ hr to 268.7 ± 91.3 mg/dl at 2 hour postprandially.

Figure 5 indicates the 2 hour plasma glucose stimulation curve of all the lunches. It indicates that tapioca showed the maximum peak rise of blood sugar levels

followed by glucose and rice. These two lunches and glucose showed a similar trend in increasing or decreasing the blood sugar levels during the experiment, Ragi and wheat showed similar effect on blood plasma glucose levels when compared to that of the other two lunches and glucose.

4.3.6 Mean peak rise over the fasting levels after the administration of glucose and experimental lunches

Table 21 shows the mean peak rise over the fasting plasma glucose values after the consumption of glucose (75 g) and the four experimental lunches, prepared from rice, wheat, tapioca and ragi. Comparison of the means by analysis of variance showed that the values significantly differed from each other (Appendix XII). The difference between the fasting and peak plasma glucose concentration was significantly lower after the administration of wheat when compared to that of glucose. The diet based on ragi also showed significantly lower rise in plasma glucose concentration than that of glucose but not significantly ($p = 5$ per cent and 2 per cent) different from that of wheat. Tapioca showed the highest rise in plasma glucose concentration which was greater than that of glucose. In general, plasma glucose concentration was higher in subjects given tapioca diet in comparison to the others except rice.

The peak rise in plasma glucose concentration was less in subjects given wheat and ragi based lunches.

Table 21 Comparison of mean peak rise over the fasting blood sugar level

Food	Mean Peak rise (mg/dl)
Glucose	164.5 ± 31.55
Rice	159.1 ± 42.22
Wheat	103.65 ± 39.38
Tapioca	181.05 ± 36.21
Ragi	114.55 ± 59.64

CD = 27.37 S.E. = 9.726

4.3.7 Comparative changes of each lunch

Average blood sugar levels at different periods during the experimental lunches showed that at fasting there was no significant difference in blood sugar levels of the subjects. However, postprandial blood sugar levels revealed different trends in the change in blood sugar levels with time, for various diets. Table 22 shows the comparison of mean plasma glucose values of the subjects served with experimental lunches.

Table 22 Comparison of mean plasma glucose values of the subjects

Item tested	Plasma glucose values (mg/dl)				
	Fasting blood sugar level	Postprandial blood sugar level			
		½ hr	1 hr	1½ hr	2 hr
Glucose (75 g)	193.5	284.4	346.4	341.1	329.1
Experimental lunches					
Rice	195.4	296.1	345.1	328.2	316.1
Wheat	200.1	266.1	291.9	278.6	258.9
Tapioca	199.5	312.1	375.4	336.3	307.1
Ragi	205.6	268.7	313.8	299.5	268.7

CD = 19.63, SE = 7.08

A statistical analysis of the data on postprandial blood glucose levels at ½ hr intervals for 2 hrs after the experimental diet on wheat, rice, ragi and tapioca showed significant differences between the diets, between the periods and between diets and periods. (Appendix XIII) The experimental lunches with wheat and ragi as staples did not raise the postprandial blood glucose levels as high as either tapioca or rice based lunches.

At 1 hr the postprandial blood sugar level also showed a differential trend in the blood sugar level from that of ½ hr. Wheat and ragi were found to raise the blood sugar level to the least extent from ½ hr postprandial level, when compared with that after the consumption of lunches based on rice and glucose. Lunch with tapioca showed the greatest rise of blood plasma glucose levels at 1 hour postprandially.

Ninety minutes after the consumption of the experimental lunches, it was found that lunch based on wheat showed much lower blood sugar level than ragi, eventhough lunches based on rice, tapioca and glucose (of OGTT) showed a decreasing trend in blood sugar concentration but not as low as that of wheat or ragi.

After two hours the blood glucose levels in subjects who had taken the lunches with wheat and ragi were much lower than when the lunches were with tapioca and rice as staples. Glucose (75 g) when fed, after two hours, gave a blood glucose level much lower than that obtained when lunch with rice as staple was fed.

4.4 Glycemic response of experimental lunches

Table 23 shows the mean area under the plasma glucose response curve (AUC) for the four experimental lunches. The table shows that, tapioca had the highest area under the curve followed by rice, ragi and wheat. The area under the curve obtained after the consumption of experimental lunch I (Rice) was more or less similar to that obtained after oral glucose tolerance test (OGTT). Wheat had the least area under the curve.

Table 23 Mean area under the curve of experimental lunches

Food	Area under the curve (square units) (mm^2)
Glucose	324.00
Rice	321.34
Wheat	273.44
Tapioca	332.68
Ragi	290.90

Table 24 shows the mean glycemic response of the various lunches. Tapioca had the highest glycemic response which was 2.68 per cent higher than that of glucose. Rice showed one per cent decrease in its glycemic response when compared to glucose. Ragi and wheat showed the lowest

glycemic response of 0.898 and 0.84 respectively which was 11 per cent and 16 per cent less than that of glucose.

Table 24 Mean Glycemic response to the lunches

Food	Glycemic response
Glucose	1.0000
Rice	0.9900
Wheat	0.8400
Tapioca	1.0268
Ragi	0.8980

The correlation studies of glycemic response and mean peak rise over the fasting blood sugar level showed a highly significant positive correlation ($r = 0.98$).

4.5 Relationship of glycemic response to plasma glucose concentration

Table 25 shows the regression relationship of glycemic response to plasma glucose concentration. Over the entire period of experiment the values differed significantly from each other.

FIG. 6. MEAN GLYCEMIC RESPONSE OF THE LUNCHES

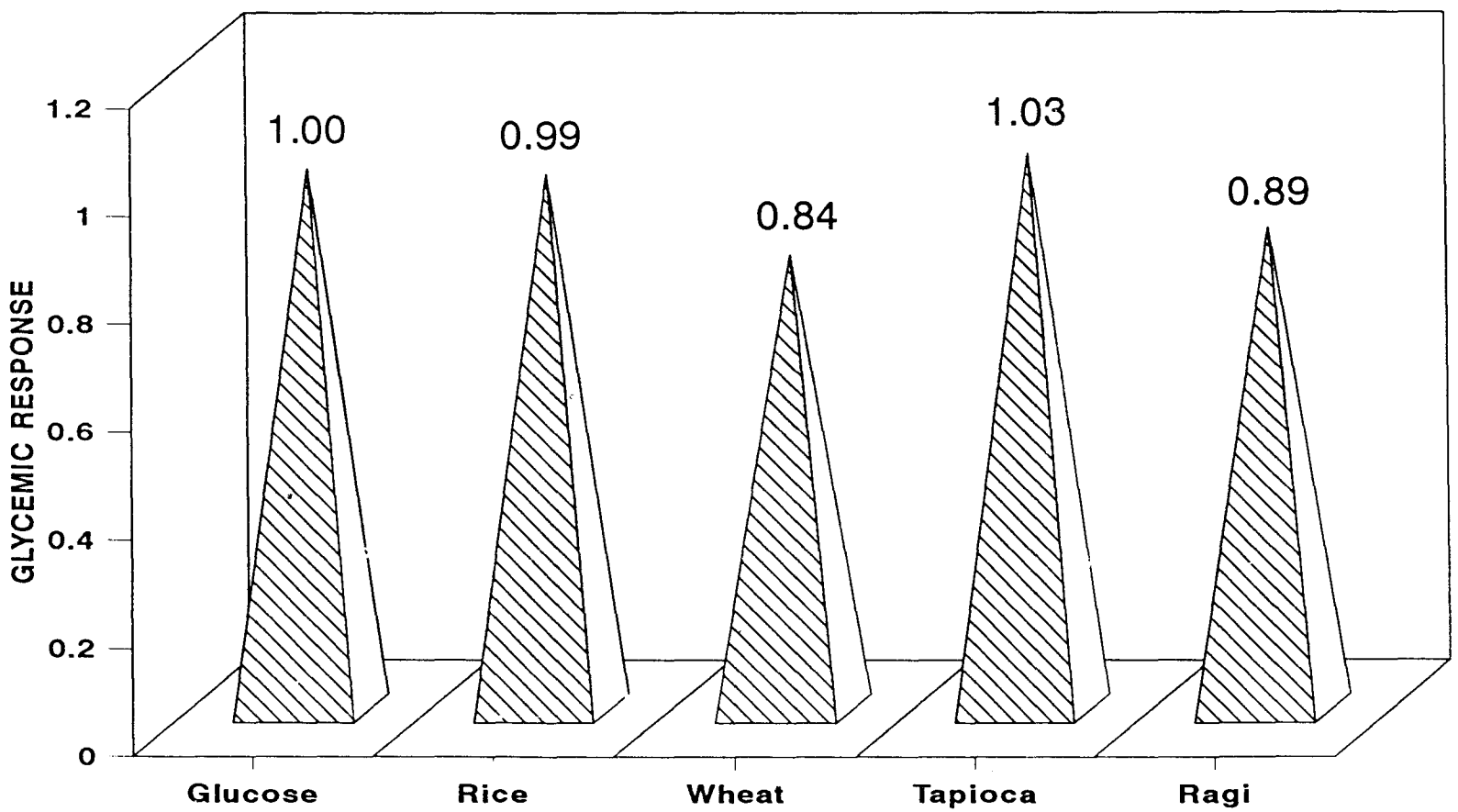


Table 25 Regression relationship of glycemic response (y) to plasma glucose concentration (x)

Time (Hr.)	Regression equation	F (1,18)	Coefficient of determination (per cent)
½	y = 107.23 + 190.02x**	10.39*	77.60
1	y = -45.31 + 398.96x**	69.31**	95.85
1 ½	y = 5.997 + 326.59x**	71.51**	95.97
2	y = -42.73+ 354.08x**	18.58*	86.10

* Significant at 5 per cent level

** Significant at 1 per cent level

After the first ½ hr of the experiment, about 77.6 per cent of the glycemic response was due to increase in plasma glucose concentration and for every unit rise in glycemic response there was 190.02 unit rise in plasma glucose concentration. At 1 hr. and 1½ hrs 95.85 and 95.97 per cent respectively of the glycemic response was due to increase in plasma glucose concentration. At these time periods, increase in plasma glucose concentration per unit rise in glycemic response was 398.96 and 326.59 respectively. Two hour after starting the experiment the influence of plasma glucose

concentration on glycemic response was found to decrease to 86.1 per cent and for every unit rise in glycemic response there was 354.08 unit rise in plasma glucose concentration.

The regression relationship thus brings out the importance of postprandial glycemic response and plasma glucose levels as best brought out at sampling after 1 to 1½ hrs when 95 per cent of the glycemic response is accounted by the plasma glucose concentration.

DISCUSSION

DISCUSSION

Diabetes mellitus is a complex metabolic disorder and is the third commonest disease in the world next to the cardiovascular and oncological disorders. Every fifth person in the world suffers directly or indirectly from Diabetes mellitus. In India diabetes is a common disease affecting 2 to 4 per cent of the population and the majority (90 per cent) are diagnosed as non insulin dependent diabetics. Recent studies have suggested that not just the carbohydrates ingested but the biological equivalents (ie, quantities of food yeilding the same effect on blood glucose) or the glycemic response of a food should be considered while planning a diet for diabetics. Hence the present study entitled, "Glycemic response to selected carbohydrate rich foods in diabetics," was aimed to assess the glycemic response of selected carbohydrate rich staple foods such as rice, wheat, tapioca and ragi in the form of Kerala type lunches given to twenty Non-Insulin Dependant Diabetes Mellitus (NIDDM) subjects.

The salient findings of the present study were discussed under the following headings:

5.1 Personal characteristics of the subjects selected and oral glucose tolerance test (OGTT)

5.2 Experimental lunches.

5.3 Effect of experimental lunches on plasma glucose response.

5.4 Glycemic response of the experimental lunches and

5.5 Relationship of glycemic response to plasma glucose concentration.

Twenty non-insulin dependant diabetic men in the age range of 40 to 51 years were served seperately the experimental lunch items viz., rice, wheat, tapioca and ragi supplying 75 g of glucose along with vegetables like onion, cucumber, tomato, bittergourd and amaranth, pulses like red gram dhal and green gram, nuts and oilseeds like coconut, fruits like guava, milk products like curd and cooking oil, and a 75 g glucose load in a random order after an overnight fast.

5.1 Personal characteristics of the subjects selected and oral glucose tolerance test (OGTT)

The characteristics of the subjects selected for the study such as age, sex, body fatness, glucose tolerance status, use of oral hypoglycemic agents, the degree of diabetes control and the fasting blood glucose values on the day of test are some of the important variables that affect

the glycemic index value of the foods consumed (Thomas et al., 1991b).

In this experiment the 20 subjects selected were adult males, since in India, there was a greater incidence of diabetes among men than women, the ratio being 2.6:1.6 (Ahuja, 1979). Majority of the epidemological surveys show a male preponderance among the Indian diabetics, both within the country and abroad (Ramachandran et al., 1993). The selected subjects had a mean age of 45.7 (±3.25) and their age ranged from 40 to 51 years. According to Cahil (1975), the incidence of Diabetes mellitus increased markedly with ageing. Paris-Bockel et al (1987) found that the glucose tolerance decreased with age and the decrease affected more postprandial glucose values than fasting plasma glucose concentrations.

From the results it was found that about 50 per cent of the subjects were suffering from this disease for the past 4 to 6 years. Thirty five per cent for 7 to 10 years and only 15 per cent for the past 1 to 3 years. Earlier studies invariably recommended to conduct such experiments on early detected diabetics since they were devoid of the signs of neuropathy and retinopathy [Ole and Kjeld (1991), Winnie and Parvathi (1987) and Bornet et al (1987)].

Body mass index (wt/ht2) of the selected subjects were calculated and found that 30 per cent of the subjects were obese. Ian (1994) found that obese people are more prone to diabetes because they are resistant to the effect of insulin which converts blood sugar to energy. Hansen (1988) suggested that weight reduction and improvement in blood glucose control through dietary intervention for obese person with non-insulin dependant diabetes was the greatest potential for reducing morbidity and mortality. The mean body mass index of the subjects was 25.4 ± 4.5. According to Keen et al (1979) body mass and blood glucose levels were universely correlated with energy intake.

All the subjects rely on oral hypoglycemic agents such as sulphonylurea and biguanides. Majority of the subjects used a combination of sulphonylureas and biguanides. This indicated that the subjects may either on primary treatment or there was secondary failure occuring with sulphonylureas alone (Asok,1993). None of the subjects used dietetics alone as a therapeutic measure.

Tolerance to glucose was a determinant of the characteristics of the subjects. Prior to the start of the experiment an oral glucose (75g) tolerance test was conducted for all the subjects to assess their metabolic

response to glucose load and for comparison with the glycemic response to the staple foods selected for the study. The fasting blood glucose levels ranged from 140 to 287 mg/dl. According to Sharad (1985) fasting blood glucose is important and ideal parameter in assessing NIDDM.

For oral glucose tolerance test the highest mean value in postprandial curves was observed at 60 minutes after the start of the experiment. The glucose concentration at the next measurement ie., at 90 minutes was slightly lower. The glucose (75g) was administered in the form of a solution with 200 ml water. According to Antia (1989) glucose ingested as a drink, does not require digestion and can, to a limited extent, be absorbed by the stomach and easily utilised. The peak rise over the fasting blood sugar level was found to be 164.5 ± 31.55 mg/dl.

5.2 Experimental lunches

Recent studies have demonstrated that equivalent amounts of carbohydrates give a different response depending on the kind of food consumed mainly because of varying rate of absorption pattern. The processing methods involved in the preparation of the four lunches were decided to make the amount of heat used in the preparation as it affects

digestability. Since boiling method alone was used, all the preparations were completely gelatinized, thereby increasing the digestability and eliciting the maximum glycemic response.

The experimental lunches were isocaloric and similar and supplied 75 g of glucose in the form of rice, wheat, ragi and tapioca. The lunches, were planned according to the principles of meal planning and dietetics (National Institute of Nutrition, 1991), and were balanced. According to Sue (1989) the basic principle guiding daily energy needs for a diabetic patient is sufficient kilocalories to meet growth and development needs, physical activities and maintenance of lean body weight.

The lunches planned supplied 3/7 of the daily requirement of an adult diabetic weighing 60 kg of body weight. The lunches supplied 771 kcals with 128 g carbohydrate, 28 g protein, 15 g fat and 10 g fibre.

According to Sue (1989) the nutrient allocation of total calories for a diabetic should be mainly from carbohydrate, [55 per cent to 60 per cent of the total calories, with the main portion 50 per cent as complex carbohydrate] followed by protein [0.8 g/kg body weight for

adults] and fats [30 per cent or less of the total calories].

Attempt was made to increase the protein content of the experimental lunches, since diabetics, in general, are in negative nitrogen balance, they should receive about twice as much proteins as normal subjects and the proteins should provide about 20 to 25 per cent of the calories in the diet (Swaminathan, 1990).

5.3 Effect of experimental lunches on plasma glucose response

The results of the study showed that the glucose concentrations before the meals (fasting blood sugar levels) do not differ systematically. This indicated that the volunteers had followed more or less the similar consumption pattern in the 24 to 48 hours before each meal (Van, et al., 1990). This supported the reliability of any difference found in postprandial blood glucose levels. For all the meals the highest mean value in the postprandial curves were observed at 60 minutes after the consumption of meal. The glucose concentration at the next measurement was always considerably lower. The plasma glucose concentration at 60 minutes was therefore most probably not the highest value

occuring postprandialy in blood. The mean time at which the plasma glucose concentration at the highest value, could be obtained by using the quadratic regression equation. It was found that glucose, rice, wheat, tapioca and ragi showed the peak rise of plasma glucose concentrations at 85.21, 79.77, 72.92, 73.02 and 73.32 minutes respectively.

The results indicated that all the subjects reached the peak at different time intervals after the consumption of different experimental lunches. This is contradictory to the findings of Van et al (1989) since in this experiment, a linear relationship was found to exist between the carbohydrate content of the meal and the postprandial glucose peak maximum in the four lunches because of the uniform carbohydrate and energy content. However Van et al (1989) had found that the type of carbohydrate in the meal appeared to have no effect on the peak maximum.

The present study utilized boiling as the method of cooking in the case of rice, wheat and tapioca and steaming for ragi.

The rice given to the subjects was parboiled and hand pounded. The method of cooking was boiling until there was complete gelatinization and swelling and draining out the

excess water. According to O'Dea et al (1980) the physical form of the rice was of particular importance in determining the postprandial glucose and insulin responses to rice and that fibre, per se, played a relatively minor role in this instance. Also the ratio of low surface area: starch limits the access of the hydrolytic enzymes to the ingested starch would be expected to slow the rate of glucose absorption and thereby reduce insulin secretion. The constituents of a diet are available only if digestive and absorptive functions are not impaired. The mean peak rise of blood plasma glucose concentration over the fasting levels were 159.1 mg/dl after the consumption of experimental lunch based on rice. All subjects reached the peak value of blood plasma glucose conentration at 60 minutes or more precisely 79.77 minutes after the consumption of lunch.

Tapioca showed the highest mean value of peak rise over the fasting blood sugar concentrations followed by glucose (75 gm) and rice. Tapioca was the only tuber included in the experiment. In 1977, Crapo et al found that potatoes give large postprandial glucose and insulin increases, almost comparable to those of glucose. The present study also supported the fact that starches from different foods resulted in different postprandial blood sugar level (Crapo et al, 1977 and Jenkins et al, 1988a).

The highest rise in postprandial blood glucose concentration can be attributed to the fact that starch in tapioca was easily available to the individuals. According to Ross et al (1987), a close relationship exists between the degree of gelatinization, rate of starch hydrolysis in vitro and postprandial glucose and insulin responses in humans. Tapioca was prepared by boiling and draining the excess water. Snow and O'Dea (1981) found that cooking and gelatinization of starch (ie., swelling of granules in the presence of heat and water) increase its susceptability to enzymic degradation in vitro and its availability for digestion and absorption in the small intestine.

According to Hood and Mercier (1978) and Colonna and Mercier (1983), tapioca is composed of type I starch with 16 per cent amylose, a negligible quantity of protein can usually be detected (0.03 per cent) and it is a highly digestible starch in vitro in humans.

Eventhough, tapioca has the fiber content greater than that of rice, it has shown the highest rise in blood sugar levels than rice and the rest of the experimental lunches. Beryl (1991) and Nicol and Philips (1978) found that the digestability of rice and cassava diets were the same in young Nigerian men, although the total crude fiber content

was lower in rice than in cassava diet. According to Kamalu (1991) rice fibre was more digestible than tapioca fibre and in a nutritionally balanced cassava diet starch was readily absorbed.

The low rise in blood sugar levels, after the consumption of experimental lunch on rice, compared to that of tapioca can be partly attributed to the protein content of rice. Seshiah et al (1993) found that the quality of proteins in rice is better than the quality of proteins in other cereals. There was no significant loss of protein content of cereals due to various methods of cooking, where as a significant loss was observed in the case of lysine, tryptophan and sugar contents (Madhu and Mathews, 1985). More over rice has the fat content double to that of tapioca which helped to reduce the rise in blood sugar concentration by decreasing the rate of absorption. Fat usually blunt the rate of increase in blood glucose by reducing the rates of absorption (Nuttal et al., 1984).

Wheat and ragi showed comparatively low peak rise over the fasting level in all the subjects when compared to that of tapioca and rice. This reduction in plasma glucose concentration may be due to the high protein and dietary fibre content of wheat and ragi. Dietary fibre has a blood

glucose reducing effect (Teusher, 1986). But the results showed that instead of low dietary fibre content than that of ragi, wheat showed the lowest rise in plasma glucose concentration. Acarbose present in wheat is presumed to act as an inhibitor and has prevented the absorption of carbohydrate and fat to such an extent that the rise in blood glucose following a meal could be restricted to as much as 50 per cent (Seshiah, 1984).

Whole wheat was cooked by boiling until complete gelatinization and swelling and draining out the excess water. The digestability of wheat increases with the processing conditions, the more the processing conditions, the more rapid the absorption of starch (Holm et al., 1989). According to Jenkins (1979), the activity of acarbose is attenuated when whole wheat is milled. Since in the study, the wheat was not milled, the acarbose may play an active role in reducing the postprandial blood sugar rise. Acarbose is an inhibitor of mucosal α glucosidases (sucrase, maltase and glucoamylase) responsible for carbohydrate degradation (Caspary, 1978).

The protein content of wheat is also higher than that of ragi. According to Dibildose et al (1985) proteins in the foods may alter the plasma glucose response. Gluten, present

in wheat can cause reduction in postprandial glucose response. Jenkins et al (1987) found that removal of gluten from wheat flour resulted in an increased rate of amylotic digestion.

Ragi was ground and steamed to increase the palatability. So the particle size was lower than that of the other staple food items for the experiment. Ragi showed a peak rise over the fasting blood sugar levels slightly greater than that of wheat. Crapo and Henry (1988) found that, grinding of cereals enhances digestability and increased postprandial plasma glucose reponses.

The fibre content of ragi was greater and twice as high as that of wheat and other experimental foods. Since fibre had a negative relationship with the absorption rate, the ability of wheat to decrease the postprandial blood glucose values may be mainly due to the effect of gluten and acarbose. The same results are obtained in earlier studies (Chithra and Thilaga, 1989). However the peak rise over the fasting blood sugar levels due to the consumption of wheat and ragi are not significantly different.

5.4 Glycemic response of lunches

The two hour incremental area under the curve for each experimental lunches were calculated by the quadratic regression equation. The accurate assessment of area under the curve is important since glycemic response is mathematically represented by the following formula:

$$\text{Glycemic response} = \frac{\text{Area under 2 hr blood glucose response curve of test food}}{\text{Area under 2 hr blood glucose response curve for equivalent amount of glucose}}$$

(Jenkins, 1982)

The method of area calculation affects the glycemic index value (Thomas et al., 1991 b). Also Gannon and Nuttal (1987) recognised that methodologic factors may influence the glycemic response data.

The highest area under the curve was shown by tapioca followed by glucose (of OGTT) and rice. Wheat and ragi showed the least area under the curve. According to Thomas et al (1991b), the area under the glycemic response curve for each food is expressed as a per cent of the mean response to the standard food taken by the same subject and the resulting values are averaged to obtain glycemic index value for the food.

The glycemic response of each food item tested were calculated according to earlier studies (Jenkins, 1982). From the results it was found that wheat had the least glycemic response followed by ragi, rice and tapioca. Tapioca showed the highest glycemic response which was higher than that of glucose (75 g of OGTT). This may be due to some metabolic error since glucose has the highest glycemic response than other foods. According to Wolever (1990a) variability of glycemic responses arises from day to day variation in the subject and variation between different subjects and there is less variability between glycemic index values of the different subjects than there is within the same subject from day to day. Therefore the mean glycemic index values are independent of glucose tolerance status of the subjects being tested.

As rice is a staple food in many parts of the world, it is important to study the factors that may help to predict its glycemic response (Leonora et al., 1991). In the present study rice based experimental lunch elicited a glycemic response nearer to that of glucose. The difference in the rates at which they elicited blood glucose and insulin responses had been attributed to numerous factors. The form of rice used in this experiment was parboiled and hand

pounded. According to Jenkins et al (1983 b) low glycemic response was found in parboiled rice, whereas higher results was obtained in regular rice.

Swaminathan (1979) found that amylose content of normal rice's starch was 25 per cent, and that of wheat and tapioca were 27 and 17 per cent respectively (Bornet et al., 1989).

Rice varieties whether white, brown or parboiled can be classified as high glycemic index foods based on their amylose content (Miller et al., 1992) Unlike non-glutinous and normal rice, glutinous (waxy) rice varieties are reported to have higher glycemic index values (Juliano et al., 1990).

Owing to the linear structure of amylose, starch granules rich in amylose are thought to have more extensive hydrogen bonding and hence more crystallinity in their structure than starch granules with least amylose content. Consequently they do not swell or gelatinize as readily upon cooking and therefore, are digested more slowly and resulting in lower blood glucose and insulin responses than those with low amylose content. For this reason, the intake of high amylose foods have been considered more desirable for individuals with impaired carbohydrate and lipid metabolism (Behall et al., 1988 and 1989).

The results of the study showed that the experimental lunch with tapioca as the staple food item showed the highest glycemic response. The role of amylose content as a factor influencing the glycemic response to starch was emphasised by Goddard et al (1984). It was noted that tapioca has the least amylose content (16 per cent) when compared to other three staple food items.

Wheat was found to have the highest amylose (27 per cent) content and the lowest glycemic response. According to Thorne et al (1983) the amylose-amylopectin ratio in starch is an important determinant of rate of digestion. Francis et al (1990) found that starch susceptability to amylose increases after cooking, eventhough overcooking was found to have only a slight effect.

Glycemic response of ragi comes next to the experimental lunch with wheat. This increased glycemic response of ragi when compared with that of wheat was also found in earlier studies (Chithra and Thilaga, 1989).

According to Leonora et al (1991), amylose content alone is not a good predictor of starch digestability and glycemic response. Proteins in food may also alter the glycemic response (Dibildose et al., 1985). Among the four

staple food items tested wheat was found to have the highest protein (11.8 per cent) content. The protein content of the other staple food items were 7.3 per cent, 6.4 per cent and 0.7per cent for ragi, rice and tapioca respectively. Results of the study demonstrated the fact that a negative relationship existed between the protein content of food and their glycemic response.

The lowest glycemic response of wheat may partly be due to the high gluten content of wheat. Jenkins et al (1987) demonstrated that removal of gluten from wheat flour resulted in an increased rate of amylotic digestion in vitro and enhanced glycemic response in vivo. Holm et al (1985) suggested that wheat strand is a large fraction of the starch, encapsulated in a protein matrix. Pagani et al (1986) had done scanning electron microscopy of wheat with freeze - fracturing and thin sectioning and confirmed that proteins coagulate to form a continuous network around each starch granule during cooking, thereby providing a structural explanation for the low plasma glucose responses. The natural presence of antinutrients in the slow release carbohydrate foods is partly beneficial in the management of diabetes and hyperlipidemia. Such antinutrients are represented by phytic acid, lectins, tannins or enzyme inhibitors of amylose and disaccharides (Thomson, 1988).

Among the four staple foods tested, phytic acid was absent in tapioca and hence elicited a higher glycemic response. Wheat was found to have the greatest phytic acid content followed by ragi. Rice contained a very small quantity of phytic acid when compared to that of wheat and ragi. The glycemic index was found to correlate negatively with the phytic acid content of the food (Yoon et al., 1983). Jenkins et al (1984) found that phytic acid caused greater reduction in blood sugar rise than fibre and the glucose response becomes less with increasing content of phytic acid in food items.

The glycemic response of ragi was greater than that of wheat but less than that of rice. Ragi had a protein content which was almost similar to that of rice but elicited a glycemic response less than that of rice. This may be due to the high phytic acid and fiber content of ragi. Ragi had the highest dietary fibre content among the four staple food items tested followed by wheat and tapioca. Dietary fibre has a blood glucose reducing effect, as is manifested by a diminished glycemic index. (Teusher, 1986). Eventhough the fibre content of tapioca was higher than that of rice, it produced the highest glycemic response, even higher than that of glucose (75 g, of OGTT). This may be due to some

metabolic error. The comparatively high amylose and phytic acid content of rice may cause a glycemic response lower than that of tapioca.

5.5 Relationship of glycemic response to plasma glucose concentration

The relationship of glycemic response to plasma glucose concentrations were studied at different time intervals. The values differed significantly from each other during the entire period of experiment. Influence of postprandial plasma glucose concentration on glycemic response was increased from ½ hr value to 1½ hr. This may be due to the fact that major part of digestion and absorption takes place at the first 1½ hrs and hence increased postprandial blood glucose concentrations. The results of the present study supported the above statement that after 1½ hrs, the influence of plasma glucose concentration on glycemic response was found to be reduced. Thus glucose or food ingestion over a prolonged period of time, mimicking slow absorption results in flat blood glucose responses (Wolever, 1990b).

This data provides a rationale for designing diabetic diets containing complex carbohydrates in a form which is

slowly digested and absorbed. Thus the differences in glycemic potencies of different starch containing foods are not due to variations in the starch molecule per se but rather the way it is presented to the gastrointestinal, digestive and absorptive processes. The physical access of digestive enzymes to starch molecules of native foodstuffs differs and blending of food alters this difference so that all starch molecules now have equal availability to digestive enzymes. Blending also leads to dispersion and hydration of starch which could be an important factor in absorption. Thus the particle size of food, becomes a critical factor since digestion of food, its absorption and as also its glycemic response depends to a large extent on its particle size. In summary among the four staple foods tested wheat is reported to have the lowest glycemic response followed by ragi, rice and tapioca.

SUMMARY AND CONCLUSION

SUMMARY AND CONCLUSION

Considerable research has been undertaken on how the control of non-insulin dependent diabetes mellitus can be improved by altering the nature and quantity of carbohydrate containing foods eaten. Current dietary recommendations, for treatment, prescribe a diet rich in complex carbohydrates and low in fat with the aim of improving glycemic control. The rise of blood sugar after a meal depend upon the amount of carbohydrates ingested and on the rapidity of absorption. Starchy foods are known to differ in the rates at which they are digested and absorbed and at which they elicit blood glucose responses. This led to the introduction of the glycemic response to rank carbohydrate foods. However, before the glycemic response can be employed to construct diabetic diets aimed at long term blood glucose control, the applicability to mixed meals must be ascertained. The present study was aimed to find out the glycemic response of selected carbohydrate rich foods to diabetics.

For this study, twenty NIDDM patients were selected based on sex, age and blood glucose level. Healthy men in the age group of 40 to 51 years, with a fasting blood sugar

level greater than 140 mg/dl and 200 mg/dl after 2 hours of glucose ingestion, and using only oral hypoglycemic agents were considered.

The personal characteristics of the selected subjects indicated that their age ranged from 41 to 50 years, with this disease for the past 4 to 6 years (50 per cent). Thirty per cent of the patients were found to be obese. However the mean body mass index of the selected subjects was 25.4 ± 4.5.

A combination of sulphonylurea and biguanides (50 per cent) sulphonylurea (45 per cent) and biguanides (5 per cent) were used as oral hypoglycemic agents.

Prior to the experiment as a criteria for selection, an oral glucose tolerance test was conducted with 75 g glucose load to assess the metabolic response of the patients to glucose load. The fasting plasma glucose level was in the range of 140 to 287 mg/dl. Many patients (60 per cent) showed the peak rise over the fasting level at 1 hr after starting the experiment. By 2 hours, the glucose level was found to fall in 95 per cent of the patients. The mean peak rise over the fasting blood sugar level was 164.5 ± 30.81 mg/dl. The relationship between blood sugar levels at

various time intervals was highly significant and gave an optimum time of 85.21 minutes after which the blood sugar level decreased considerably.

Four experimental lunches, which were isocaloric (771 kcals), balanced and vegetarian were formulated with variation only in the staple items. All the four lunches supplied 75 g of carbohydrates in the form of staple foods such as rice, wheat, ragi and tapioca.

The lunches were administered to the selected subjects who were after an overnight fast and who avoided drugs for 24 hours, prior to the experiment. The blood samples were collected intravenously before and after the administration of lunches with 30 minutes intervals for 120 minutes and analysed for plasma glucose concentration. Area under the 2 hour glucose stimulation curve (AUC) of the experimental lunches were calculated by using the quadratic regression equation $y = a + bx + cx^2$ and glycemic response was worked out, using the ratio AUC of test food : AUC of reference food (glucose).

Effect of experimental lunches on plasma glucose response was studied and the results indicated that the mean peak rise of blood sugar over the fasting level after

consuming the rice based experimental lunch was 159.1 ± 42.2 mg/dl. The lunch was administered when the subjects had a fasting blood sugar level of 195.4 ± 44.7 mg/dl. While the experimental lunch with wheat as staple food elicited a least rise of blood sugar over the fasting blood sugar among the four lunches. The mean peak rise over the fasting blood sugar level was 103.7 ± 38.4 mg/dl. The mean fasting blood sugar levels before the administration of wheat based lunch was 200.2 ± 53.9 mg/dl. The experimental lunch based on tapioca elicited the highest rise of blood sugar over the fasting blood sugar levels among the four lunches. The lunch resulted in a mean peak rise of 181.1 ± 35.3 mg/dl over the fasting blood sugar level (199.5 ± 46.5 mg/dl). While experimental lunch based on ragi comes next to wheat. It raised the blood sugar level upto 114.6 ± 58.1 mg/dl, when the fasting blood sugar level was 205.6 ± 56.6 mg/dl which was the highest fasting blood sugar level observed before administering the four lunches.

During the four experiments it was found that the majority of the subjects (80 per cent for rice, 70 per cent for wheat, 85 per cent for tapioca and 80 per cent for ragi) reached the peak maximum at 1 hr postprandially or more

accurately 79.77 minutes for rice, 72.92 minutes for wheat, 73.02 minutes for tapioca and 73.32 minutes for ragi.

Comparison of mean peak rise over the fasting blood sugar level by analysis of variance showed that blood sugar levels significantly differed among each other. Lunches with wheat and ragi were found to result in a decreased blood sugar level when compared to tapioca and rice.

The mean area under the 2 hr glucose stimulation curve showed that tapioca has got the highest area under the curve (332.7 square units) in comparison with the other three lunches and glucose (75 g). In the case of glycemic response also tapioca has the highest glycemic response (1.0268) followed by rice (0.9900), ragi (0.8980) and wheat (0.8400).

Correlation studies of glycemic response and mean peak rise over the fasting blood sugar level showed a highly significant positive correlation ($r = 0.98$). The relationship of glycemic response to plasma glucose concentration was found to be significant over the entire period of the experiment.

The findings of the study had indicated the necessity for taking up elaborate studies on these lines. Glycemic index of various carbohydrate foods commonly used in India

needs to be worked out. There is a need to conduct similar experiments on more subjects and extent to other food proportions and also to the various mixed meals consumed in a day in normals and diabetics to elucidate the factors affecting glycemic response to a food.

Similar experiments can also be carried out by substituting major cereal item by millets or minor roots and tubers or pulses in the commonly consumed meal items.

Effect of other hypoglycemic agents to the meal items in influencing their glycemic response, and influence of different cooking and processing methods applied on food items, on their glycemic response are to be investigated. Rice based traditional meals can be improved by substituting rice with other cereals. Investigation on the acceptability and glycemic response to these products may give sufficient information to introduce variety in the diets of diabetics.

Standardisation of rice based preparations by replacing rice with wheat and other millets and the effect of addition of hypoglycemic agents present in certain food crops should also be investigated.

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APPENDICES

APPENDIX I

ORAL GLUCOSE TOLERANCE TEST (OGTT)

Oral glucose tolerance test is carried out after 12 hrs of overnight fasting. Glucose 75 gm in adults and 1.75 gm/kg of body weight in children is orally administered. Before the glucose load and 30 minutes for 120 minutes after the administration of glucose, blood samples were collected and glucose levels were estimated.

The diagnostic criteria for diabetes and impaired glucose tolerance are given in the table. Generally in normal persons without diabetes or impaired glucose, the fasting blood sugar levels vary between 80-110 mg/100 ml. The blood sugar levels increase after the glucose load and come down to basal level within 2 hrs.

WHO Criteria with glucose load 75 gm in 250-150 ml water for adult OR 1.75 gm/kg body weight (To a maximum of 75 gm).

	Glucose Concentration		
	Venous whole blood (mg/dl)	Capillary whole blood (mg/dl)	Venous Plasma (mg/dl)
	Normal	**Diabetes mellitus confirmed**	
Fasting	120	120	140
2 hours after glucose load	180	200	200
		Impaired glucose tolerance	
Fasting	120	120	140
2 hours after glucose load	120 - 180	140-200	140-200

WHO (1980)

APPENDIX II

CALCULATION OF DIABETIC DIET PRESCRIPTION

(a) Calories

Recommended Dietary Allowance(RDA) = 30 kcal/kg of body weight

Assuming that body wieght of the patient is 60 kg.

Energy requirement per day = 60 x 30 = 1800 kcal.

(b) Carbohydrates

RDA = 60 to 65 per cent of total calories

$$\text{Carbohydrate requirement per day} = \frac{1800 \times 60}{100 \times 4} \text{ to } \frac{1800 \times 65}{100 \times 4}$$

$$= 270 \text{ gm to } 293 \text{ gms.}$$

(c) Proteins

RDA = 15 to 20 per cent of total calories

$$\text{Protein requirement per day} = \frac{1800 \times 15}{100 \times 4} \text{ to } \frac{1800 \times 20}{100 \times 4}$$

$$= 67.5 \text{ gm to } 90 \text{ gms.}$$

(d) Fat

RDA = 15 to 25 per cent of total calories

$$\text{Fat requirement per day} = \frac{1800 \times 15}{100 \times 9} \text{ to } \frac{1800 \times 25}{100 \times 9}$$

$$= 30 \text{ gm to } 50 \text{ gms.}$$

In other words, fat requirement per day

= Total calories-(calories from Carbohydrate + calories from protien)

Ref: Corinne and Merilyn (1982)

APPENDIX III

COMPOSITION AND NUTRITIVE VALUE OF INGREDIENTS OF THE MODEL DIET PLANNED

Preparation	Ingredients	Qty (gm)	Energy (kcals)	CHO (gm)	Protein (gm)	Fat (gm)	Fibre (gm)
Break fast							
Tea	Milk	120.00	80.40	5.28	3.84	4.92	-
Bread (Brown)	4 slices	60.00	146.40	29.40	5.28	8.24	0.72
Plantain	1	100.00	116.00	27.20	1.20	0.30	0.40
Cooked Bengal gram (whole)		42.00	151.20	25.58	7.20	2.23	1.64
Total		--	494.00	87.46	17.52	25.69	2.76
Lunch							
Rice (Parboiled & hand pounded)		96.50	338.18	75.00	8.20	0.58	--
Veg.Salad	Onion	10.00	5.00	1.11	0.12	0.01	0.06
	Cucumber	10.00	1.30	0.25	0.04	0.01	0.04
	Tomato	10.00	2.00	0.36	0.09	0.02	0.08
	Curd	120.00	72.00	3.60	3.72	4.80	--
Bitter-gourd Poriyal	Bittergourd	30.00	7.50	1.26	0.48	0.06	0.24
	Coconut	5.00	22.20	0.65	0.23	2.08	0.18

Contd...

Amaranth Greengram Pugath	Amaranth	50.00	22.50	3.05	2.00	0.25	0.50
	Greengram	40.00	133.60	22.68	9.60	0.52	1.64
	Coconut	10.00	44.40	1.30	0.46	4.16	0.36
Redgram dhal curry	Redgram dhal	10.00	33.50	5.76	2.23	0.17	0.19
	Onion	10.00	5.00	1.11	0.12	0.01	0.06
	Tomato	10.00	2.00	0.36	0.09	0.02	0.08
	Oil	2.00	18.00	--	--	2.00	--
Guava	Guava	100.00	51.00	11.20	0.90	0.30	5.20
	Total	--	758.18	127.69	28.28	14.99	8.63
Evening	**Tea**						
Tea	Milk	120.00	80.40	5.28	3.84	4.92	--
Dinner							
Chapathi	Wheat flour	100.00	341.00	69.40	12.10	1.70	1.90
Veg.Stew	Carrot	10.00	4.80	1.06	0.09	0.02	0.12
	Green peas	10.00	9.30	1.59	0.72	0.01	0.40
	Potato	10.00	9.70	2.26	0.16	0.01	0.04
Cucumber salad	Cucumber	100.00	13.00	2.50	0.40	0.10	0.40
Cooking oil	Oil	10.00	90.00	--	--	10.00	--
	TOTAL	--	548.20	82.09	17.31	16.76	2.86
GRAND	TOTAL	--	1800.40	296.58	63.10	47.44	14.25
	RDA	--	1800.00	270-293	68-90	30-50	--

APPENDIX IV (a)

COMPOSITION AND NUTRITIVE VALUE OF THE INGREDIENTS OF EXPERIMENTAL LUNCH I

Preparations	Ingredients	Qty (gm)	Energy (kcals)	CHO (gm)	Protein (gm)	Fat (gm)	Fibre (gm)
Rice (Parboiled & hand pounded)	Rice	96.90	338.18	75.00	8.20	0.58	---
Veg. Salad	Onion	10.00	5.00	1.11	0.12	0.01	0.06
	Cucumber	10.00	1.30	0.25	0.04	0.01	0.04
	Tomato	10.00	2.00	0.36	0.09	0.02	0.08
	Curd	120.00	72.00	3.60	3.72	4.80	--
Bittergourd Poriyal	Bittergourd	30.00	7.50	1.26	0.48	0.06	0.24
	Coconut	5.00	22.20	0.65	0.23	2.08	0.18
Amaranth	Amaranth	50.00	22.50	3.05	2.00	0.25	0.50
Green gram Pugath	Greengram	40.00	133.60	22.68	9.60	0.52	1.64
	Coconut	10.00	44.40	1.30	0.46	4.16	0.36
Redgram dhal curry	Redgram dhal	10.00	33.50	5.76	2.23	0.17	0.19
	Onion	10.00	5.00	1.11	0.12	0.01	0.06
	Tomato	10.00	2.00	0.36	0.09	0.02	0.08
	Oil	2.00	18.00	--	--	2.00	--
Guava	Guava	100.00	51.00	11.20	0.90	0.30	5.20
	TOTAL	--	758.18	127.69	28.28	14.99	8.63

APPENDIX IV (b)

COMPOSITION AND NUTRITINE VALUE OF THE INGREDIENTS OF EXPERIMENTAL LUNCH II

Preparations	Ingredients	Qty (gm)	Energy (kcals)	CHO (gm)	Protein (gm)	Fat (gm)	Fibre (gm)
Boiled Tapioca	Tapioca	196.80	308.97	75.00	1.38	0.39	1.12
Veg.Salad	Onion	10.00	5.00	1.11	0.12	0.01	0.06
	Cucumber	10.00	1.30	0.25	0.04	0.01	0.04
	Tomato	10.00	2.00	0.36	0.09	0.02	0.08
	Curd	120.00	72.00	3.60	3.72	4.80	--
Bittergourd Poriyal	Bittergourd	30.00	7.50	1.26	0.48	0.06	0.24
	Coconut	5.00	22.20	0.65	0.23	2.08	0.18
Amaranth Greengram Pugath	Amaranth	50.00	22.50	3.05	2.00	0.25	0.50
	Greengram	40.00	133.60	22.68	9.60	0.52	1.64
	Coconut	10.00	44.44	1.30	0.46	4.16	0.36
Redgram dhal curry	Redgram dhal	10.00	33.50	5.76	2.23	0.17	0.19
	Onion	10.00	5.00	1.11	0.12	0.01	0.06
	Tomato	10.00	2.00	0.36	0.09	0.02	0.08
	Oil	2.00	18.00	--	--	2.00	--
Guava	Guava	100.00	51.00	11.20	0.9	0.30	5.20
	TOTAL	--	728.97	127.69	21.46	14.80	9.81

APPENDIX IV (c)

COMPOSITION AND NUTRITIVE VALUE OF THE INGREDIENTS OF EXPERIMENTAL LUNCH III

Preparations	Ingredients	Qty (gm)	Energy (kcals)	CHO (gm)	Protein (gm)	Fat (gm)	Fibre (gm)
Cooked long wheat	Long wheat	105.00	363.30	75.00	12.39	1.60	1.30
Veg.Salad	Onion	10.00	5.00	1.11	0.12	0.01	0.06
	Cucumber	10.00	1.30	0.25	0.04	0.01	0.04
	Tomato	10.00	2.30	0.36	0.09	0.02	0.08
	Curd	120.00	72.00	3.60	3.72	4.80	--
Bittergourd Poriyal	Bittergourd	30.00	7.50	1.26	0.48	0.06	0.24
	Coconut	5.00	22.00	0.65	0.23	2.08	0.18
Amaranth Greengram Pugath	Amaranth	50.00	22.50	3.05	2.00	0.25	0.50
	Greengram	40.00	133.60	22.68	9.60	0.52	1.64
	Coconut	10.00	44.40	1.30	0.46	4.16	0.36
Redgram dhal Curry	Redgram dhal	10.00	33.50	5.76	2.23	0.17	0.19
	Onion	10.00	5.00	1.11	0.12	0.01	0.06
	Tomato	10.00	2.00	0.36	0.09	0.02	0.08
	Oil	2.00	18.00	--	--	2.00	--
Guava	Guava	100.00	51.00	11.20	0.90	0.30	5.20
	TOTAL	--	783.30	127.60	32.47	16.01	9.93

APPENDIX IV (d)

COMPOSITION AND NUTRITINE VALUE OF THE INGREDIENTS OF

EXPERIMENTAL LUNCH IV

Preparations	Ingredients	Qty (gm)	Energy (kcals)	CHO (gm)	Protein (gm)	Fat (gm)	Fibre (gm)
Ragi	Ragi	104.00	341.12	75.00	7.59	1.40	3.70
Veg.Salad	Onion	10.00	5.00	1.11	0.12	0.01	0.06
	Cucumber	10.00	1.30	0.25	0.04	0.01	0.04
	Tomato	10.00	2.00	0.36	0.09	0.02	0.08
	Curd	120.00	72.00	3.60	3.72	4.80	--
Bittergourd Poriyal	Bittergourd	30.00	7.50	1.26	0.48	0.06	0.24
	Coconut	5.00	22.20	0.65	0.23	2.08	0.18
Amaranth	Amaranth	50.00	22.50	3.05	2.00	0.25	0.50
Green gram Pugath	Green gram	40.00	133.60	22.60	9.60	0.52	1.64
	Coconut	10.00	44.40	1.30	0.46	4.16	0.36
Redgram dhal Curry	Redgram dhal	10.00	33.50	5.76	2.23	0.17	0.19
	Onion	10.00	5.00	1.11	0.12	0.01	0.06
	Tomato	10.00	2.00	0.36	0.09	0.02	0.08
	Oil	2.00	18.00	--	--	2.00	--
Guava	Guava	100.00	51.00	11.20	0.90	0.30	5.20
	TOTAL	--	783.30	27.60	32.47	16.01	9.93

APPENDIX V

METHOD OF PREPARATION OF LUNCHES

(a) Methods of preparation of staple food items

1. **Rice**

The parboiled and hand pounded rice was washed and then boiled until it was well cooked and strained.

2. **Wheat**

The long wheat was washed and then boiled until it was well cooked and strained.

3. **Ragi**

Ragi was given in the form of ragi puttu. For this it was soaked, powdered and seived and then moisturised and steamed to make the puttu.

4. **Tapioca**

The skin was peeled, cut into small pieces, washed and boiled to cook. After it was well done the water was strained out.

(b) Preparation of side dishes

1. Bittergourd poriyal

Bittergourd was washed and then cut into small pieces. It was then steam cooked. After it was well done the coconut and a pinch of masala and salt in the form of paste was added to it and again cooked in simmer for one or two minutes.

2. Amaranth green gram pugath

Green gram was soaked and cooked. Amaranth was chopped and then boiled with a little water. When it was well done the green gram along with coconut in the form of a paste with a pinch of masala and salt was added. The mixture was again cooked for one or two minutes.

3. Redgram dhal curry

Redgram dhal was soaked and cooked. The onion was fried in the oil. When it becomes golden brown in colour, a pinch of masala was added with chopped tomato pieces. To this the cooked red gram dhal was added along with ½ a cup of water and salt to taste and again cooked in simmer for 2 or 3 minutes.

4. Vegetable salad

Onion, cucumber and Tomato were cut into small pieces. To this salt to taste was added and mixed with curd.

5. Guava

Guava was cut into pieces and served as such.

APPENDIX VI

METHOD OF ESTIMATION OF BLOOD SUGAR

Enzymatic colorimetric method (GOD - PAP)

Test principle

$$\text{Glucose} + O_2 + H_2O \xrightarrow{GOD} \text{gluconate} + H_2O_2$$

$$2H_2O_2 + 4\ \text{aminophenazone} + \text{phenol} \xrightarrow{POD}$$

$$\text{4-(P-benzoquinone-mono-imino) phenazone} + 4H_2O$$

Sample material

Blood, serum, heparinised plasma, EDTA plasma

Reagents

1. Buffer/Enzymes/4 - Aminophenazone
2. Phenol
3. Standard Glucose 100 mg/100ml

Initial concentrations of solutions:

Phosphate buffer : 100 mmol/l, pH 7.0;

GOD ≥ 18 U/ml

POD ≥ 1.1 U/ml;

4 - aminophenazone : 0.77 mmol/l;

Phenol : 11 mmol/l

Preparation of solutions

1. **Reagent mixture**

Dissolve contents of Reagent one bottle of Reagent 1 in 200 ml redist, water and add contents of one bottle of Reagent 2 (phenol). Store in a dark bottle.

2. Phenol

Use contents undiluted to prepare the reagent mixture.

3. **Standard**

Use solution undiluted.

Sample preparation

Serum or plasma should be separated from cells immediately if possible, and certainly not later than one hour after collecting the blood specimen.

The sample can be stored up to 24 hours at + 15 to + 25 °C after addtion of a glycolysis inhibitor (NaF, KF), or up to seven days in a closed vessel at + 4 °C.

Procedure

Wavelength	:	Hg 546 nm (470-560 nm)
Spectrophotometer	:	510 nm
Cuvette	:	1 cm light path
Incubation temperature	:	20-25 °C

Measure against reagent blank. One standard and one blank are sufficient for each assay series.

Pipette into test tubes:

	blank (ml)	standard (ml)	sample (ml)
Standard	--	0.02	--
Sample	--	--	0.02
Reagent solution	2.00	2.00	2.00

Mix and incubate at 20-25 °C. Avoid exposure to direct sunlight. After 30-90 min, read absorbances of sample (A sample) and standard (A standard) against the blank.

If the glucose concentration exceeds 400 mg/100 ml (= 22.2 mmol/l), dilute sample 1+2 with 0.9% NaCl solution and repeat assay (result x 2).

Calculation

Of the concentration (c) of glucose in blood, serum, or plasma:

$$C = 100 \times \frac{\text{A sample}}{\text{A standard}} \quad \text{(mg/100 ml)}$$

$$C = 5.55 \times \frac{\text{A sample}}{\text{A standard}} \quad \text{(mmol/l)}$$

APPENDIX VII
PLASMA GLUCOSE VALUES BASED ON ORAL GLUCOSE TOLERANCE TEST (OGTT)

	Plasma glucose values in mg/dl					
Subject's No.	FBS*	Postprandial blood sugar level 1/2hr	1hr	1 1/2hr	2hr	PROF**
1	164	292	349	341	332	185
2	227	315	415	410	400	188
3	209	302	373	376	370	167
4	187	293	322	410	400	223
5	189	216	344	328	301	155
6	140	326	329	301	263	189
7	196	259	364	389	365	193
8	158	202	282	262	252	124
9	178	312	367	330	245	189
10	173	266	362	342	324	189
11	225	287	360	391	386	166
12	157	214	320	323	318	166
13	209	239	281	316	350	141
14	287	400	287	298	307	113
15	140	268	340	336	320	100
16	189	277	359	328	290	170
17	210	294	356	324	320	146
18	205	269	348	320	308	143
19	255	363	396	345	316	141
20	172	294	374	352	315	202
Mean	193.50	284.40	346.40	341.10	324.10	164.50
S.E.	36.94	47.92	35.28	38.07	44.44	31.55

* Fasting blood sugar level.
** Peak rise over the fasting level.

APPENDIX VIII

PLASMA GLUCOSE VALUES OF THE SUBJECTS AFTER THE RICE BASED EXPERIMENTAL LUNCH

Subject's No.	Plasma glucose values in mg/dl					
	FBS*	Postprandial blood sugar level				PROF**
		½hr	1hr	1½hr	2hr	
1	140	242	311	274	292	171
2	304	552	537	537	528	233
3	200	281	319	325	371	171
4	177	261	296	265	252	119
5	200	295	340	335	320	140
6	167	284	318	289	284	151
7	170	258	288	267	253	118
8	162	183	304	262	253	142
9	162	267	315	298	272	153
10	195	303	358	320	308	163
11	256	337	489	522	507	266
12	140	234	295	273	228	155
13	175	273	244	233	175	98
14	283	400	315	313	345	117
15	158	280	268	341	311	210
16	196	288	323	320	305	127
17	205	296	334	328	321	119
18	217	302	454	423	415	237
19	233	323	394	367	340	161
20	168	262	299	272	244	131
Mean	195.40	296.05	345.05	328.20	316.05	159.10
S.E.	44.65	74.10	72.45	81.11	87.38	42.22

* Fasting Blood Sugar level.
** Peak rise over the fasting level.

APPENDIX IX
PLASMA GLUCOSE VALUES OF THE SUBJECTS AFTER THE WHEAT BASED EXPERIMENTAL LUNCH

Subject's No.	Plasma glucose values in mg/dl					
	FBS*	Postprandial blood sugar level ½hr	1hr	1 ½hr	2hr	PROF**
1	216	313	385	344	336	169
2	304	436	440	429	400	136
3	363	412	428	411	402	65
4.	190	280	295	240	215	105
5	140	193	215	209	200	75
6	140	203	215	260	215	120
7	177	218	256	222	216	79
8	145	215	285	272	265	140
9	180	230	236	192	174	56
10	170	200	254	245	215	85
11	224	327	388	420	388	196
12	185	246	211	152	158	98
13	220	261	234	229	222	41
14	173	201	244	291	261	118
15	146	233	301	260	232	155
16	195	257	276	265	236	81
17	220	279	300	281	271	80
18	216	289	315	303	290	99
19	238	319	320	291	250	82
20	161	215	240	255	236	94
Mean	200.15	266.35	291.90	278.55	258.60	130.65
S.E.	55.39	68.10	36.85	73.79	71.80	39.38

* Fasting blood sugar level.
** Peak rise over the fasting level.

APPENDIX X
PLASMA GLUCOSE VALUES OF THE SUBJECTS AFTER THE TAPIOCA BASED EXPERIMENTAL LUNCH

Subejct's No.	Plasma glucose values (mg/dl)					
	FBS*	Post prandial blood sugar level ½ hr	1hr	1½ hr	2hr	PROF**
1	142	253	344	259	241	202
2	296	386	460	452	361	164
3	282	412	488	439	428	206
4	162	282	384	290	275	222
5	185	300	402	308	288	217
6	178	326	338	313	262	160
7	215	345	421	372	351	206
8	156	278	386	267	239	230
9	194	324	400	354	330	206
10	181	264	313	295	258	132
11	296	378	406	388	383	110
12	142	212	296	257	206	154
13	223	243	292	384	288	161
14	174	330	310	259	216	136
15	149	276	387	312	304	238
16	179	300	374	360	354	195
17	213	343	419	370	350	206
18	230	352	400	392	380	170
19	222	346	372	383	365	161
20	170	292	315	271	262	145
Mean	199.45	312.10	375.35	336.25	307.05	181.05
S.E.	47.73	51.24	53.54	60.93	62.50	36.21

* Fasting blood sugar level.
** Peak rise over the fasting level.

APPENDIX XI
PLASMA GLUCOSE VALUES OF THE SUBJECTS AFTER THE RAGI BASED EXPERIMENTAL LUNCH

Subject's No.	Plasma glucose values (mg/dl)					
	FBS*	Post Prandial Blood Sugar level ½ hr	1 hr	1½ hr	2 hr	PROF**
1	140	297	335	317	249	195
2	277	498	561	548	517	284
3	334	390	420	395	381	86
4	170	283	250	235	210	80
5	190	249	283	258	240	93
6	148	216	233	205	192	85
7	241	317	392	459	405	218
8	159	227	248	216	195	89
9	159	218	235	228	216	76
10	178	250	276	259	234	98
11	349	385	446	418	405	97
12	178	195	245	305	304	127
13	221	296	352	341	286	131
14	171	270	253	241	140	82
15	155	314	342	294	236	187
16	188	209	263	243	212	75
17	228	274	297	248	227	66
18	208	240	285	253	233	77
19	230	264	299	275	261	69
20	188	242	264	251	230	76
Mean	205.60	281.70	313.80	299.45	268.65	114.55
S.E.	58.09	73.26	84.92	90.43	91.29	59.64

* Fasting Blood Sugar level.
** Peak rise over the fasting level.

APPENDIX XII

ANALYSIS OF VARIANCE (ANOVA) TABLE

Source	D.F.	M.S.S.	F
Treatment	4	22573.75	11.93*
Error	95	1891.83	
Total	99		

SE = 9.726 CD = 27.37

* Significant at 5 per cent level.

APPENDIX XIII

ANALYSIS OF VARIANCE (ANOVA) TABLE

Source	D.F	M.SS.	F
Diet (A)	4	40245	2.397 *
Error I	95	16792	
Periods (B)	4	274882	274.100 **
Diet x Periods (A x B)	16	5366.75	5.350 **
Error II	380	1002.84	
TOTAL	499		

* Significant at 5 per cent level.

** Significant at 1 per cent level.

CD Values	SE Values
A = 36.47	12.96
B = 8.78	3.17
AB = 19.63	7.08

GLYCEMIC RESPONSE TO SELECTED CARBOHYDRATE RICH FOODS IN DIABETICS

BY

KAVITA M. S.

ABSTRACT OF THE THESIS
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ABSTRACT

A study was conducted to assess the glycemic response of selected carbohydrate rich foods in twenty NIDDM patients. The selected patients were adult males in the age group of 40 to 51 years, having a fasting blood sugar level of 140 mg/dl and 200 mg/dl at 2 hour after the consumption of food and relying on oral hypoglycemic agents.

Results of the personal characteristics of the selected patients showed that 50 per cent of them were suffering from diabetes for the past 10 years. Seventy per cent of them had normal body weight and all of them depend on sulphonylurea and biguanides or a combination of these two drugs.

An oral glucose tolerance test was done prior to the experiment. Glucose was administered to the patients with a fasting blood sugar level of 193.5 ± 36.0 mg/dl. The peak rise over the fasting blood sugar level was found to be 164.5 ± 30.8 mg/dl, sixty five per cent of the patients reached the peak at 1 hour postprandially. The relationship between blood sugar level at various time intervals showed an optimum time of 85.21 minutes after which the blood sugar level decreased considerably.

Experimental lunches planned for the study were isocaloric and similar except for the staple foods such as rice, wheat, ragi and tapioca which supplied 75 g of carbohydrate. Lunch with rice as a staple food was given to the patients with a fasting blood sugar level of 195.4 ± 44.7 mg/dl which gave the peak rise of 159.1 ± 42.2 mg/dl over the fasting blood glucose level after lunch. The peak was observed 1 hour after the consumption of lunch in 80 per cent of the patients. In this case the optimum time beyond which the blood sugar level decreased was found to be 79.77 minutes.

The patients had a fasting blood sugar level of 200.2 ± 53.9 mg/dl when wheat based lunch was administered. The peak rise over the fasting blood sugar level was found to be 103.7 ± 38.4 mg/dl, 70 per cent reached the peak at 1 hour after the lunch. The time at which the blood sugar level decreased was found to be at 72.92 minutes postprandially.

The lunch with tapioca was given to the patients with a fasting blood sugar level of 199.5 ± 46.5 mg/dl. After the lunch, the peak rise over the fasting blood sugar level was found to be 181.1 ± 35.3 mg/dl. At 1 hour after the lunch

the peak was observed among 85 per cent of the patients. It was at 73.02 minutes, the blood sugar level found to decrease.

Lunch with ragi as staple food was given to the patients when they had the fasting blood sugar level of 205.6 ± 56.6 mg/dl. After the lunch, majority of the patients (80 per cent) reached the peak level over the fasting blood sugar level at 1 hour. The peak rise over the fasting blood sugar level was found to be 114.6 ± 58.1 mg/dl. The time at which the blood sugar level found to decrease was 73.32 minutes.

From the results of area under the 2 hour glucose stimulation curve, it was found that wheat has the least area under the curve followed by ragi, rice and tapioca.

The glycemic response was analysed and found that wheat had the least glycemic response followed by ragi, rice and tapioca. Tapioca showed a glycemic response which was found to be higher than that of glucose. It may be due to some metabolic error on the part of the subjects.

The correlation studies of glycemic response and mean peak rise over the fasting blood sugar level showed a highly significant positive correlation. Relationship of glycemic

response to plasma glucose concentration was found to be highly significant.

The present study points out the need to conduct similar experiments with a variety of foods on large number of patients. Such data will enable to modify the diabetic diet to include locally available and low cost foods as hypoglycemic agents.