EFFECTS OF MODERATE SUGAR INTAKE ON GLYCAEMIC CONTROL OF PATIENTS WITH TYPE 2 DIABETES MELLITUS

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This dissertation is dedicated to Gary Hunter, and Baltie & Bettie Erasmus

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LIST OF ABBREVIATIONS

%	Percent
A1c	Glycosylated
ACE	Angiotensin converting enzyme
ADP	Adenosine diphosphate
ADSA	Association for Dietetics in South Africa
ADA	American Diabetes Association
ATP	Adenosine triphosphate
BMI	Body mass index
BUN	Blood urea nitrogen
CDA	Canadian Diabetes Association
CARMEN	Carbohydrate Ration Management in European National diets study
CDC	Centres for Disease Control
CHD	Coronary heart disease
CHO	Carbohydrate
CI	Confidence interval
CVD	Cardiovascular disease
DCCT	Diabetes Control and Complications Trial
DESSA	Diabetes Education Society in Southern Africa
DGAC	Dietary Guidelines Advisory Committee
DKA	Diabetic Ketoacidosis
G6PDPH	Glucose-6-phosphate dehydrogenase
GAD65	Glutamic acid decarboxylase
GAD-AB	Glutamic acid decarboxylase antibodies
GAR-PPT	Goat antirabit preprecipitated serum
GI	Glycaemic index
GI	Gastrointestinal
GLP-1	Glucagon-like peptide-1
Hb	Haemoglobin
HbA1c	Glycated Haenoglobin
HDL	High density lipoproteins
HK	Hexokinase
ISAK	International Society for the Advancement of Kinanthropometry

kD	kiloDalton
LADA	Late onset Auto-immune Diabetes Mellitus of the Adult
LCD	Liquid crystal display
LDL	Low density lipoproteins
Lt	Lieutenant
Max	Maximum
Med	Median
Min	Minimum
MNT	Medical Nutrition Therapy
MODY	Maturity onset diabetes of the young
MRFIT	Multiple Risk Factor Intervention Trial
NAD	Nicotinamide adenine dinucleotide
NADH	Reduced form of NAD
NHANES	National Health and Nutrition Examination Survey
NKHS	Nonketotic hyperosmolar state
PAI-I	Plasminogen activator inhibitor-1
PCOS	Polycystic ovary syndrome
PVD	Pheripheral vascular disease
RD	Registered dietician
RIA	Radioimmunoassay
SD	Standard deviation
SEMDSA	Society for Endocrinology, Metabolism and Diabetes of South Africa
SFD	Sugar Free Diet
SID	Sugar Inclusive diet
SMBG	Self-monitoring of blood glucose
TE	Total energy
UKPDS	United Kindom Prospective Diabetes Study
VLDL	Very Low Density lipoproteins

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1.1 INTRODUCTION AND MOTIVATION

The earliest signs of diabetes were recorded by the physician Hesy-Ra on Third Dynasty Egyptian papyrus in 1552 B.C. He mentioned polyuria (frequent urination) as a symptom (Canadian Diabetes Association, 2000). Diabetes mellitus is a growing global problem. About 10.3 million people in the United States have been diagnosed as having diabetes mellitus, and another 5.4 million have diabetes mellitus, but are presently undiagnosed (Franz, 2000, p.243). The diabetic population is expanding, with an estimated number of 200 million worldwide (Huddle & Kalk, 2000). The World Health Organization (WHO) estimates that the number of people with diabetes mellitus will reach an alarming 300 million by 2025 (Canadian Diabetes Association, 2000). Huddle and Kalk (2000) estimated that there were 2-3 million diabetics in South Africa at the beginning of the new millennium. By the year 2002, it was thought that 5% of the South African population were affected by type 2 diabetes mellitus (Servier Laboratories, 2002), with the highest incidence amongst Coloureds and Asians (Motala *et al.*, 2003).

The prevalence of diabetes in South African communities is increasing aggressively, due to population and lifestyle changes associated with rapid urbanization. Traditional rural communities still have a very low prevalence; at most one to two percent. One to thirteen percent or more adults in urban centres have diabetes. Type 2 diabetes is the predominant form, with a rate of 70-90%. Due to the high urban growth rate, dietary changes, reduction in physical activity, and increasing obesity, it is estimated that the prevalence of diabetes is due to triple within the next 25 years (Sobngwi *et al.*, 2001).

Two major types of diabetes are recognized, namely, type 1 and type 2 diabetes mellitus (Canadian Diabetes Association, 2000). Type 1 diabetes mellitus is characterized by beta cell destruction, usually leading to absolute

insulin deficiency, and may account for 5% to 10% of all diagnosed cases of diabetes. The peak incidence for developing type 1 diabetes is around ages 10 to 12 years in girls, and ages 12 to 14 years in boys, although it may occur at any age. These persons are dependent on exogenous insulin to prevent ketoacidosis and death (Franz, 2000, p.744). Type 2 diabetes is characterized by insulin resistance and relative insulin deficiency. People with type 2 diabetes can range from predominantly insulin-resistant to predominantly deficient in insulin secretion with insulin resistance. Endogenous levels of insulin may be normal, depressed, or elevated, but they are inadequate to overcome concomitant insulin resistance. Persons may or may not experience classic symptoms of uncontrolled diabetes (polydipsia, polyuria, polyphagia, and weight loss), and they are not prone to develop ketoacidosis, except during times of severe stress. Type 2 diabetes may account for 90-95% of all diagnosed cases of diabetes (Franz, 2000, p.745).

Dietary intervention is one of the most important elements in the management of type 2 diabetes mellitus. One of the main aims in dietary management of diabetes mellitus is to optimize blood glucose control. The risk of developing ophthalmologic, macrovascular, nephropathic and/or neuropathic infections as well as other complications is reduced by achieving optimal blood glucose control (Franz, 2000, pp.762-764). The restriction of sugar intake was often considered the only significant part of the diabetic diet (Wolever & Brand Miller, 1995).

During the 1970's and 1980's, official international diabetes associations compiling position statements around the world, started to revise their dietary recommendations, and advised a lowered fat consumption coupled with an increased carbohydrate intake for patients with diabetes mellitus. Advice regarding restriction of sugars (glucose, fructose, sugar and lactose) remained unchanged. There was a general consensus that harmful effects of sucrose (sugar) on people with diabetes had been exaggerated.

The rationale that banned sugar stems from Allen's work on dogs in the 1920's. He noted that dogs that had pancreatectomies showed greater glycosuria after glucose intake than after starch intake. He concluded that glucose, compared to complex carbohydrate, caused a more rapid rise in blood glucose concentrations and, therefore, greater glycosuria. Unfortunately, this conclusion was expanded to all simple sugars, including sucrose (sugar) (Wolever & Brand Miller, 1995).

It is common for diabetics to use sweetening agents. In one diabetic clinic population (Colagiuri *et al.*, 1989), 65% regularly used these products. These individuals were instructed to avoid added sucrose based on the assumption that refined carbohydrates, sugars included, have a deleterious effect on postprandial glycaemia (Colagiuri *et al.*, 1989). A study by Colagiuri and co-workers (1989) showed that although aspartame was an acceptable sugar substitute for diabetics, it had no specific advantage over sucrose.

The American Diabetes Association (ADA) stated in their 1994 guidelines (Gillespie, 1996), that scientific evidence has shown that the inclusion of sugar as part of a meal plan does not impair blood glucose control in patients with type 1 or type 2 diabetes mellitus. Sugar must, however, substitute other carbohydrate foods like starches, milk, and fruit, within the context of a healthy meal plan and may not just be added additionally (ADA, 2000a). The Diabetes Care and Education Practice Group of the American Dietetic Association conducted a survey on how its members implemented the 1994 diabetic recommendations. The overall response was positive, but more than 50% of the respondents felt that the liberalization of sugar was controversial. Several patients and members thought that liberalizing sugar intake was inappropriate, and therefore questioned the dieticians' credibility as healthcare professionals when recommending it. Some members of the group were concerned that patients would exchange more nutritious food like vegetables, fruit, grains and milk for sugary foods and sweets, and still be able to maintain a consistent carbohydrate intake. This is a valid concern and places great responsibility on

diabetes educators to ensure that these carbohydrate recommendations are interpreted correctly and that nutritional intake is not compromised (Gillespie, 1996). This concern is shared by Nadeau and co-workers (2001), who found that despite the strong evidence that sugar does not alter glycaemic control, health professionals often fear that teaching free-living patients the latest sugar guidelines will lead to a deterioration of eating habits and metabolic profile, if the guidelines are not applied.

The Society for Endocrinology, Metabolism and Diabetes in Southern Africa (SEMDSA) and the Diabetes Education Society in Southern Africa (DESSA), allows people with diabetes to include sugar as part of an appropriate energy controlled, low fat, high fibre eating plan (Workgroup: Diabetes and diet, 2000, p.11). One of the guidelines in the position paper of the Association for Dietetics in South Africa (ADSA) (ADSA, 1997) for patients with well-controlled diabetes, is to allow sugar (10% of the total energy intake) as part of a balanced diet. Foods containing sugar have a relatively low glycaemic index of 68 ± 5, based on ten studies using glucose as the standard, and are therefore included in the diabetic diet (Foster-Powell *et al.*, 2002). Health professionals are sceptical whether this may be advisable to all the patients they consult with since patients might opt for sweets and sugary foods in the place of more nutritious foods and still be able to maintain a consistent carbohydrate intake (Gillespie, 1996).

As sugar is found in so many foods, it is difficult to exclude it from the diet, even for the most dedicated diabetic patient. Most diabetic patients would welcome the possibility of selecting from a wider variety of foods, without feeling guilty or anxious about the choices they made. Few documented studies, in which free-living diabetic patients choose their own food and incorporate sugar in their diets are available (Peterson *et al.*, 1986). Out of ten studies done to compare glycaemic effects of isocaloric amounts of sugar and starch in patients with diabetes mellitus, all had a small number of subjects (n: 6-24). Furthermore, these studies were done over a relatively

short period of time, ranging from 6-24 weeks, or patients were given a single meal. In all these studies, prepared meals were provided for subjects by the investigators. The results of these studies have shown that the percentage of energy derived from sugar ranged from 7% to 38%, with no adverse effect on glycaemia (Franz *et al.*, 1994).

Considerable controversy exists about the potential effects of dietary sugar on lipaemia in diabetic patients. In a study done by Bantle and co-workers (1993), the sugar did not result in any significant changes in serum cholesterol or serum triglycerides. Coulston and co-workers (1985) reported that type 2 diabetic patients fed a high sugar diet and reference sugar-free diet for 15 days, demonstrated increasing fasting plasma cholesterol during the high sugar diet. In contrast, Abraira and Derler (1988) reported that type 2 diabetic patients fed high sugar or high complex carbohydrate diets for 1 month demonstrated no significant differences in fasting serum total or LDL-cholesterol. Colagiuri and co-workers (1989) found that fasting concentrations for total serum cholesterol, HDL-cholesterol and triglycerides of patients with type 2 diabetes mellitus were not significantly different at the end of sugar- or aspartame- supplemented periods compared with pre-treatment levels.

No data on sugar intake for the South African diabetic population exists. Furthermore, present data on the effect of moderate sugar intake in the diets of patients with type 2 diabetes in the free-living environment is scarce and needs further investigation. For the purpose of this study sugar will refer to sucrose.

1.2 HYPOTHESIS

Patients with Type 2 diabetes mellitus can safely include 15% of the total daily energy intake as sugar in their diet, without deleterious effects on glycaemic profile.

1.3 AIMS

The main aim of this study was to evaluate the effects of 15% of the total daily energy intake as sugar on the glycaemic control of patients with type 2 diabetes mellitus.

To accomplish this aim, the effects of the inclusion of 15% of the total daily energy intake as sugar were compared to the exclusion of sugar in the diets of free-living patients with type 2 diabetes mellitus on:

- glycaemic control (fasting plasma glucose concentrations, serum fructosamine, HbA_{1c})
- lipid profiles (serum cholesterol, serum HDL cholesterol, serum LDL cholesterol and serum triglycerides); and
- compliance with the prescribed diabetic diet.

1.4 SCOPE

This dissertation is divided into six chapters. Chapter one includes the introduction and motivation of the study. The hypothesis and aims of the study are also stated.

Chapter two is a literature study discussing classification, diagnosis, aetiology, complications, management and monitoring of type 2 diabetes and medical nutrition therapy (MNT). The effects of sugar intake on glycemic control in type 2 diabetics are of special interest.

Methods used to conduct the study are described in chapter three. The operational definitions, sample and study procedure are outlined. Furthermore, the selection and standardization of techniques, as a measure of validity and reliability, are found in this chapter. Statistical analysis of results is

described. Practical problems experienced while conducting the study, and how these problems were overcome are also discussed.

Chapter four describes the results of the study using, amongst other, tables.

A discussion of the results of this study is to be found in chapter five. The data is interpreted by comparing it to other studies in the scope of the topic. Possible explanations for results are given.

The conclusions are set out in chapter six. Recommendations for the inclusion of sugar in the diabetic diet are stated. A short summary captures the study.

2.1 INTRODUCTION

Chapter 2 discusses the prevalence of type 2 diabetes mellitus, normal physiology and metabolism of glucose, metabolic homeostasis in the non-diabetic human, pathophysiology and aetiology, as well as the clinical symptoms, signs, diagnosis and complications of type 2 diabetes mellitus. Medical nutritional management and medication are also discussed in this chapter.

2.2 PREVALENCE

Diabetes mellitus is a group of diseases characterized by high blood glucose concentrations resulting from defects in insulin secretion, insulin actions, or both. Abnormalities in the metabolism of carbohydrates, proteins and fats, are also present. People with diabetes do not produce or respond to insulin, a hormone produced by the beta cells of the pancreas that is necessary for the use or storage of body fuels. Without effective insulin, hyperglycaemia occurs, which can lead to both the short-term and long-term complications of diabetes mellitus (Franz, 2000, p.243).

Recent figures from the Centres for Disease Control and Prevention (CDC) (Henry, 2001), in the United States of America, confirm that the prevalence of diabetes is growing at an alarming rate. Among young people in their 30's, for example, there was a 70% increase in the incidence of diabetes between 1990 and 1998. In this same time period, the prevalence of diabetes increased with 33% among people of all ages and ethnic groups. In South Africa, the situation seems similar, with 5% of the population being affected by type 2 diabetes mellitus (Servier Laboratories, 2002). A ten year prospective population study by Motala *et al.* (2003) has shown that, especially among

South African Asians, there is an increased incidence of type 2 diabetes mellitus.

2.3 CLASSIFICATION

The two broad categories of diabetes mellitus are designated type 1 and type 2. Type 1 diabetes mellitus is characterized by beta-cell destruction, usually leading to absolute insulin deficiency, and may account for 5% to 10% of all diagnosed cases of diabetes. The peak incidence for developing type 1 diabetes is around ages 10 to 12 years in girls, and ages 12 to 14 years in boys; although it may occur at any age. These people are dependent on exogenous insulin to prevent ketoacidosis and death (Franz, 2000, p.744).

For the purpose of this study, type 2 diabetes will be the focus. Insulin resistance and relative insulin deficiency characterize type 2 diabetes. People with type 2 diabetes can range from predominant insulin-resistant to predominantly deficient in insulin secretion with insulin resistance. Endogenous concentrations of insulin may be normal, depressed, or elevated, but are inadequate to overcome concomitant insulin resistance. Persons may or may not experience classic symptoms of uncontrolled diabetes (polydipsia, polyuria, polyphagia, and weight loss), and are not prone to develop ketoacidosis, except during times of severe stress. Although people with type 2 diabetes do not require exogenous insulin for survival, approximately 40% will eventually require exogenous insulin for adequate blood glucose control. Insulin may also be required for control during periods of stress-induced hyperglycaemia. Type 2 diabetes may account for 90-95% of all diagnosed cases of diabetes (Whitney & Rolfes, 2002, p.257).

Other types of diabetes mellitus include specific genetic defects in insulin secretion or action, metabolic abnormalities that impair insulin secretion, and a host of conditions that impair glucose tolerance. Maturity onset diabetes of

the young is a subtype of diabetes mellitus, characterized by autosomal dominant inheritance, early onset hyperglycemia, and impairment in insulin secretion. Gestational diabetes mellitus may develop due to glucose intolerance during pregnancy. Insulin resistance related to the metablic changes of late pregnancies increases requirements and may lead to hyperglycaemia or impaired glucose tolerance (Powers, 2001, p.2109).

Risk factors for type 2 diabetes include (Whitney & Rolfes, 2002, p.257; Powers, 2001, p.2112):

- age,
- obesity,
- a family history of diabetes,
- a prior history of gestational diabetes,
- impaired glucose homeostasis,
- physical inactivity,
- HDL cholesterol concentration ≤ 0.90mmol/l and/or triglyceride concentrations ≥ 2.82 mmol/l,
- race or ethnicity
- and polycystic ovary syndrome.

Although approximately 80% of type 2 diabetics are obese, or have a history of obesity at the time of diagnosis, type 2 diabetes can occur in non-obese individuals as well, especially in the elderly (Franz, 2000, p.745; Whitney & Rolfes, 2002, p.127).

2.4 NORMAL PHYSIOLOGY AND METABOLISM OF GLUCOSE

When food is chewed, it is mixed with the saliva, which contains the enzyme ptyalin, mainly secreted by the parotid glands. This enzyme hydrolyzes starch into disaccharide maltose and other small polymers of glucose that contain three to nine glucose molecules. Food remains in the mouth only for a short time, and probably not more than five percent of all starches that are eaten

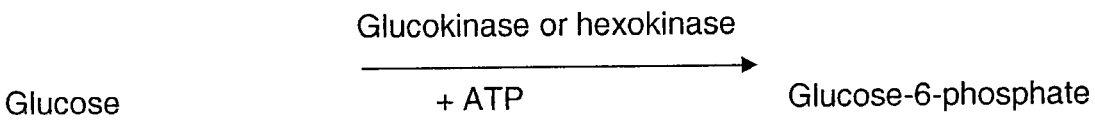
will have become hydrolyzed by the time the food is swallowed. Digestion continues in the body and fundus of the stomach for as long as one hour before the food becomes mixed with the stomach secretions. Then the acid of the gastric secretions blocks the activity of the salivary amylase, because it is essentially non-active as an enzyme once the pH of the medium falls below about 4.0. Nevertheless, on average, before the food becomes completely mixed with the gastric secretion, as much as 30 to 40 percent of the starches will have been hydrolyzed, mainly to maltose (Guyton & Hall, 1996, p.834; Beyer, 2000, p.12).

A great proportion of the chemical reactions in the cells are concerned with making the energy in foods available to various physiological systems of the cells. All the energy foods; carbohydrates, fats, and proteins, can be oxidized in the cells, and in this process, large amounts of energy are released in the form of adenosine triphosphate or ATP (Guyton & Hall, 1996, p.855; Ettinger, 2000, p.61). The final products of carbohydrate digestion in the alimentary tract are almost entirely glucose, fructose, and galactose – with glucose representing, on the average, about 80 percent of these. After absorption from the intestinal tract, much of the fructose and almost all the galactose are also rapidly converted in the liver into glucose. Therefore, little fructose and galactose are present in the circulating blood. Glucose, thus, becomes the final common pathway for the transport of almost all carbohydrates to the tissue cells.

In the liver cells, appropriate enzymes are available to promote interconversions among the monosaccharides. The dynamics of the reactions are such that when the liver releases the monosaccharides back into the blood, the final product is almost entirely glucose. The reason for this is that the liver cells contain large amounts of glucose phosphatase. Glucose-6-phophatase can be degraded back to glucose and phosphate, and the glucose can be transported through the liver cell membrane back into the blood. Once again, it is emphasized that usually more than 95 percent of all

monosaccharides that circulate in the blood are the final conversion product, glucose (Guyton & Hall, 1996, p.856; Beyer, 2000, p.12).

Immediately on entry to the cells, glucose combines with a phosphate radical in accordance with the following reaction:



This phosphorylation is promoted mainly by the enzyme glucokinase in the liver or hexokinase in most other cells. The phosphorylation of glucose is irreversible, except in the liver cells, the renal tubular epithelium, and the intestinal epithelial cells. In these cells, another enzyme, glucose phosphatase, is also available, and when this is activated, it can reverse the reaction. Therefore, in most tissues of the body, phosphorylation serves to capture the glucose in the cell. That is because of its almost instantaneous binding with phosphate. The glucose will not diffuse back out, except from those special cells, especially liver cells, that have the phosphatase. After absorption into the cells, glucose can either be used immediately for the release of energy to the cells, or be stored in the form of glycogen, which is a large polymer of glucose. All cells of the body are capable of storing at least some glycogen, but certain cells can store large amounts, especially the liver cells, which can store up to five to eight percent of their weight as glycogen. Glucose-6-phosphate first becomes glucose-1-phosphate; then this is converted to uridine diphosphate glucose, which is then converted into glucogen (Guyton & Hall, 1996, p.857; Ettinger, 2000, p.61).

Several specific enzymes are required to cause these conversions, and any monosaccharide that can be converted into glucose can enter into the reactions. Certain smaller compounds, including lactic acid, glycerol, pyruvic acid, and some deaminated amino acids, can also be converted into glucose or closely allied compounds and then into glycogen. Glycogenolysis means

the breakdown of the cell's stored glycogen to reform glucose in the cells. The glucose can then be used to provide energy. By far the most important means by which energy release from the glucose molecule is initiated, is glycolysis. Then the end products of glycolysis are mainly oxidized to provide the energy. Glycolysis means splitting of the glucose molecule to form two molecules of pyruvic acid (Guyton & Hall, 1996, p.857).

2.5 METABOLIC HOMEOSTASIS

The differentiation between non-diabetic and diabetic homeostasis, metabolic homeostasis in non-diabetics and diabetics, is discussed as follows.

2.5.1 Metabolic homeostasis in the non-diabetic person

During fuel homeostasis, the healthy human being maintains plasma glucose concentrations within narrow limits, independent of glucose flux through the plasma compartment; an adequate supply of emergency carbohydrate in the form of liver and muscle glycogen, diverting excess consumption to adipose tissue; an adequate supply of protein for body structure and enzyme functions, and body stores of protein in preference to fat during fasting (Powers, 1996, p.8). The normal blood glucose concentration in a person who has not eaten a meal within the past three to four hours is about 5,0 mmol/l. After a meal containing large amounts of carbohydrates, this concentration seldom rises above 7,7 mmol/l, unless the person has diabetes mellitus (Guyton & Hall, 1996, p.863). Immediately after a high carbohydrate meal, the glucose that is absorbed into the blood causes rapid secretion of insulin. The insulin in turn causes rapid uptake, storage, and use of glucose by almost all tissues of the body, but especially by the muscles, adipose tissue, and liver (Guyton & Hall, 1996, p.973). Glucagon, a hormone secreted by the alpha cells of the Islets of Langerhans when the blood glucose concentration falls,

has several functions that are diametrically opposed to those of insulin (Guyton & Hall, 1996, p.978).

The liver has a central role in the metabolic response to food ingestion. Increased insulin and decreased glucagon shift the liver to glycogen synthesis and curtail hepatic glucose release. In the normal fed state, the liver takes up about 60% of ingested glucose. The magnitude of this effect is determined by the portal vein insulin concentration (Powers, 1996, p.8). When the quantity of glucose entering the liver cells is more than can be stored as glycogen or be used for local hepatocyte metabolism, insulin promotes the conversion of all this excess glucose into fatty acids. These fatty acids are subsequently packaged as triglycerides in very low-density lipoproteins, transported in this form by way of the blood to the adipose tissue, and deposited as fat (Guyton & Hall, 1996, p.974).

Muscle cell sensitivity to insulin is related to how recently the muscle has been exercised and the quantity of glycogen already stored. Sensitivity to insulin is markedly increased after exercise. Muscle cell storage of amino acids as protein is also stimulated by insulin. In adipose tissue, glucose is primarily converted to glycerol, the backbone of stored triglycerides. Only a small fraction is converted to adipose tissue glycogen. Insulin stimulates adipose tissue uptake of circulating triglycerides by enhancing lipoprotein lipase activity, while simultaneously exerting an antilipolytic effect through inhibition of hormone sensitive lipases. In fasting, a catabolic state occurs in which the processes discussed above are reversed and glucose is diverted from most other tissues to maintain its supply to the central nervous system (Powers, 1996, p.9). The brain is quite different from most other tissues of the body, in that insulin has little or no effect on uptake or use of glucose. Instead, the brain cells are normally permeable to glucose and can use glucose without the intermediation of insulin. The brain cells are also quite different from most other cells in the body, in that they normally use only glucose for energy and can use other energy substrates, such as fats, only with difficulty. Therefore,

the maintenance of the blood glucose concentration above a critical concentration is essential, which is one of the most important functions of the blood glucose control system. When the blood glucose does fall too low, into the range of 1.11 to 2.78 mmol/l, symptoms of hypoglycaemic shock develop, characterized by progressive nervous irritability that leads to fainting, seizures, and even coma (Guyton & Hall, 1996, p.974).

The brain's constant glucose supply in fasting depends on a decline in circulating insulin concentrations to basal concentraions of 10 to 15 μU/ml, and the ability of glucagon to maintain hepatic glucose production at the rate that will prevent hypoglycaemia. This shift in hormone balance induces endogenous glucose production through glycogenolysis and gluconeogenesis (Powers, 1996, p.9; Anderson, 1999, p.1369). For the first 8 – 12 hours of fasting, plasma glucose concentrations are maintained by the steady release of hepatic glucose at 2 to 3 ml/kg per minute, about 75% of which derives from glycogenolysis and the remainder from gluconeogenesis. Most of this glucose supplies the central nervous system. Even though skeletal muscle constitutes 40 to 45% of total body mass, it uses less than 20% of fasting hepatic glucose production, because the fasting plasma insulin concentration is too low to enhance muscle glucose uptake. As fasting continues, hepatic glycogen stores are depleted, being essentially gone by 24 hours. Thus, the role of gluconeogenesis in maintaining hepatic glucose output becomes increasingly important (Powers, 1996, p.10).

For the first 3 to 7 days of continued fasting, protein catabolism is the principal supplier of substrate for hepatic glucose production. Beyond seven days, loss of amino acids from skeletal muscle protein is minimized by ketone production resulting from lipolysis. Ketones then take the place of glucose as the major fuel source for the central nervous system. As plasma insulin declines in the fasting state, adipose tissue escapes from insulin's anti-lipolytic effect and hormone-sensitive lypase activity increases. Lipolysis releases glycerol which is used by the liver as a substrate for gluconeogenesis, and free fatty acids, of

which some are used by the liver, but most are consumed by skeletal muscle. Protein is catabolized during fasting with release of skeletal muscle amino acids at the rate of 0,5 to 1,0 g/kg per day. Alanine and glutamine make up two thirds or more of the total α-amino-nitrogen loss. Alanine is a substrate for gluconeogenesis by the liver. Glutamine is distributed primarily to the kidney and gut (Powers, 1996, p.9; Anderson, 1999, p.1369; Cryer, 2001, p.2138).

2.5.2 Metabolic homeostasis in the diabetic person

In general, metabolism in diabetes can be described as a runaway fasting state. Both diabetes and fasting are characterized by reduced insulin activity. In diabetes, this results from an absolute or relative decrease in insulin concentration. Plasma glucagon concentrations are increased in both fasting and diabetes. As a result, both fasting and diabetes are associated with increased glycogenolysis, gluconeogenesis, and lipolysis. As an effective amount of insulin is not available, the balance between insulin and glucagon is lost. There is no longer co-ordination and equality between the amount of glucose leaving and entering the circulation, and hyperglycaemia results. Adequate tissue supply is maintained, but only in the presence of an elevated plasma glucose concentration. The degree to which the insulin effect is lost, is a determinant of the clinical presentation of the disorder. The process of fuel homeostasis is short circuited in diabetes, and the body attempts to rid itself of a troublesome by-product of that dysregulation (Powers, 1996, p.10).

2.6 PATHOPHYSIOLOGY

Type 2 diabetes mellitus describes a condition of fasting hyperglycemia that occurs despite the availability of insulin. The metabolic abnormalities that contribute to hyperglycaemia in people with type 2 diabetes mellitus include (Porth & Gaspard, 2003):

- impaired insulin secretion,
- peripheral insulin resistance,
- and increased hepatic glucose production.

2.6.1 Insulin secretion

Non-diabetic subjects exhibit biphasic insulin release. Early-phase insulin release is rapid and derives from pre-formed insulin pools. Late-phase insulin release is protracted and derives from both stored and newly synthesized sources. In type 2 diabetes, early-phase insulin secretion is markedly diminished or lost, and late-phase insulin secretions are prolonged. There is, thus, a failure of insulin concentrations to decrease appropriately in response to hypoglycaemia. There is a delay of insulin decline after meals. The plasma glucose eventually returns to normal, but only at the expense of insulin overproduction during the late secretory phase. In fact, the overall insulin response is lost. The reactive hypoglycaemia frequently found in mild type 2 diabetes or impaired glucose tolerance may also result from this delayed decline of postprandial insulin concentrations (Powers, 1996, p.12; Porth & Gaspard, 2003).

Insulin is derived from a single-chain precursor, pro-insulin. Within the Golgi apparatus of the pancreatic beta cell, pro-insulin is cleaved by convertases to form insulin, C-peptide, and two pairs of basic amino acids. Insulin is subsequently released into the circulation at concentrations equimolar with those of C-peptide. Unlike insulin, C-peptide is not extracted by the liver and is excreted almost exclusively by the kidneys. Therefore many investigators have used concentrations of C-peptide as a marker of beta cell function (Buse *et al.*, 2003, p.1443).

2.6.1.1 Physiological factors regulating insulin secretion

Factors regulating insulin secretion that will be discussed are carbohydrates, other macronutrients, hormonal factors, neural factors, insulin secretion in type 2 diabetes mellitus, and C-peptide loss after hyperglycaemia.

- **Carbohydrates**

The most important physiologic substance involved in the regulation of insulin release is glucose. The effect of glucose on the beta cell is dose-related. In addition to its acute secretagogue effects on insulin secretion, glucose has intermediate and longer term effects that are physiologically and clinically relevant. In the intermediate term, exposure of the pancreatic beta cell to a high concentration of glucose primes its response to a subsequent glucose stimulus, leading to a shift to the left in the dose-response curve relating glucose and insulin secretion. When pancreatic islets are exposed to high concentrations for prolonged periods, a reduction of insulin secretion is seen. There is evidence that long term exposure to high glucose concentrations reduces expression of a number of genes that are critical to normal beta cell function, including the insulin gene. These adverse effects have been termed "glucotoxicity" and the precise mechanisms are not known (Buse *et al.*, 2003, p.1444).

- **Other macronutrients**

Amino acids stimulate insulin release in the absence of glucose; the most important secretagogues being the essential amino acids leucine, arginine, and lysine. In contrast to amino acids, various lipids and their metabolites appear to have only minor effects on insulin release in vivo. Carbohydrate-rich fatty meals stimulate insulin secretion, but carbohydrate-free fatty meals have minimal effects on beta cell function. Ketone bodies and short- and

long-chain fatty acids have been shown to stimulate insulin secretion acutely both in islet cells and in humans. It appears that elevated free fatty acids may contribute to the the failure of beta cell compensation for insulin resistance (Buse *et al.*, 2003, p.1445).

- **Hormonal factors**

The release of insulin from the beta cell after a meal is facilitated by a number of gastrointestinal peptide hormones, including glucose-dependent insulinotropic peptide, cholecystokinin, and GLP-1. Their effects are evident only in the presence of hyperglycaemia. Other hormones that have a stimulatory effect on insulin secretion include growth hormone, glucocorticoids, prolactin, placental lactogen, and the sex steroids (Buse *et al.*, 2003, p.1445).

- **Neural factors**

The neural effects on beta cell function cannot be entirely dissociated from the hormonal, because some of the neurotransmitters of the autonomic nervous system are hormones (Buse *et al.*, 2003, p.1445).

- **Insulin secretion in type 2 diabetes mellitus**

Due to the presence of concomitant insulin resistance, patients with type 2 diabetes are often hyperinsulinaemic, but the degree of hyperinsulinaemia is inappropriately low for the prevailing glucose concentrations. Many of these patients have sufficient beta cell reserve to maintain a euglycaemic state by dietary restriction, with or without an oral agent. The beta cell defect in patients with type 2 diabetes mellitus is characterized by an absent first-phase insulin and C-peptide response to an intravenous glucose load and a reduced second-phase response (Buse *et al.*, 2003, p.1445).

- **C-peptide loss after hyperglycaemia**

Concentrations of C-peptide can be utilized to assess remaining beta cell function after diagnosis of diabetes mellitus. C-peptide concentrations are usually measured in the fasting state, after intravenous glucagons, or with a standard meal. Determination of C-peptides provides the best current measure for assessing the impact of new therapies (Eisenbarth *et al.*, 2003).

2.6.2 Insulin resistance

Basal and meal-stimulated plasma insulin concentrations in type 2 diabetes may be normal, reduced, or even increased. Patients become hyperinsulinemic when their pancreas attempts to overcome the underlying defect of insulin resistance by increasing insulin secretion. Hyperinsulinaemia is a marker for the underlying insulin resistance, which is a hallmark of the metabolic syndrome (Defronzo *et al.*, (1992) cited by Goldstein, 2002). Resistance to insulin action at cellular level is present in most type 2 diabetes patients, even in the absence of the increased insulin resistance associated with obesity. Their level of fasting hyperglycaemia appears to be directly proportional to the degree of insulin resistance. The resistance is manifested in both hepatic and muscle tissue. Hepatic resistance results in an inappropriately high level of glucose production in the fasted state, as well as an inappropriately low level of glucose uptake in the fed state. Muscle resistance impairs glucose uptake. Resistance may be attributed to defects in the insulin receptor, to reduced concentration of receptors on the cell surface, and/or post receptor defects within the cell. Insulin must first bind to the cell membrane receptor before glucose can be transported across the cell membrane. Insulin resistance, secondary to impaired insulin binding, is present in most individuals with impaired glucose tolerance, and in essentially all type 2 diabetic individuals whose fasting plasma glucose concentration exceeds 7,7 mmol/l. This reduced insulin binding appears to result from a decreased number of available receptors, rather than from decreased

receptor affinity. The higher the basal insulin concentration, the lower the receptor concentration, and this influences cell receptor levels inversely (Powers, 1996, p.12).

Any manipulation that will lower basal insulin secretion, such as diet and weight loss, will increase receptor concentration and decrease insulin resistance (Powers, 1996, p.12). The findings of Hu *et al.* (2001) indicated that a higher intake of polyunsaturated fat and possibly long-chain n-3 fatty acids could be beneficial, whereas a higher intake of saturated fat and trans-fat could adversely affect glucose metabolism and insulin resistance.

2.6.3 The role of increased hepatic glucose production in hyperglycaemia

The resistance of adipose tissue, especially visceral fat, to suppression of lipolysis by insulin, is responsible for part of the inability of insulin to suppress hepatic glucose production by the indirect route, resulting in enhanced gluconeogenesis. In addition, the suppression of glucagon concentrations in humans with insulin resistance may be impaired, again leading to an increase in endogenous glucose production (Porth & Gaspard, 2003).

2.7 ETIOLOGY

Genetic factors, MODY, fetal programming and the "thrifty gene hypothesis"; intrauterine growth retardation and low birth weight; family history, geographical location and the pathogenesis of diabetes mellitus; sex, age and ethnicity; as well as behavioural and lifestyle-related risk factors, all play a role in the etiology of diabetes.

2.7.1 Genetic factors

Inheritance of type 2 diabetes is clearly more common than is type 1 diabetes. When an identical twin has type 2 diabetes, the chances are 90 to 100% that type 2 diabetes will appear in the other twin as well (Powers, 1996, p.11; Powers, 2001, p.2114). Genetically, type 2 diabetes consists of monogenic and polygenic forms. The monogenic forms, although relatively uncommon, are nevertheless important and a number of genes involved have been identified and characterized. The genes involved in the common polygenic form or forms of the disorder have been far more difficult to identify and characterize. In the monogenic forms of diabetes, the gene involved is both necessary and sufficient to cause disease. Environmental factors play little or no role in determining whether or not a genetically predisposed individual develops clinical diabetes (Buse *et al.*, 2003, p.1430). However, environment plays such an important role in the pathogenesis of type 2 diabetes, that it has been called a disease of civilization. Its prevalence clearly increases as urbanization, working patterns, and dietary habits evolve from primitive to industrial modes, perhaps associated with more abundant food supply. The latter is especially an issue to the extent that it leads to increased obesity, insulin resistance, and impaired insulin secretion (Powers, 1996, p.11; Vorster *et al.*, 1999; Gaziano, 2001).

2.7.2 Maturity onset diabetes of the young

Type 2 diabetes mellitus has always been classified as a disease among older people; however, one monogenic form of type 2 diabetes mellitus is worth mentioning, due to its onset in the young. Maturity onset diabetes of the young (MODY), is a genetically and clinically heterogenous group of disorders characterized by nonketotic diabetes mellitus, an autosomal dominant mode of inheritance. The onset is usually before 25 years of age and frequent in childhood or adolescence. The primary defect is in pancreatic beta cell function (Buse *et al.*, 2003, p.1431). Nongenetic factors that affect insulin

sensitivity (infection, pregnancy, and rarely obesity) may trigger diabetes onset and affect the severity of hyperglycaemia in MODY, but do not play a significant role in the development of MODY (Buse *et al.*, 2003, p.1431).

2.7.3 Fetal programming and the "thrifty gene hypothesis"

Studies associating impaired glucose tolerance or type 2 diabetes in adults with lower birth weight, smaller head circumference, and thinness at birth, indicated that limited beta cell capacity and insulin resistance might be programmed in utero (Phillips and Barker, 1993). The reduced growth of the endocrine pancreas was thought to be a consequence of maternal undernutrition. It was subsequently demonstrated that glycaemic response to insulin was also reduced in individuals who had been thin at birth (Phillips & Barker, 1993). These findings of the effect of fetal undernutition have been confirmed in a large study in Sweden. Reduced birth weight for length was associated with a threefold increased risk for type 2 diabetes by age sixty, although at age fifty there was no evidence for decreased beta cell function. In a cohort of 23,000 healthy men in the United States, there was a nearly twofold increased risk for the development of diabetes mellitus in those with low birth weight (Curhan *et al.*, 1996). The "Thrifty Phenotype Hypothesis" was proposed by Hales and Barker (1992), to account for the epidemiological observations described above. This hypothesis postulates that type 2 diabetes mellitus and other features of the metabolic syndrome have a strong environmental basis. It suggests that fetal and early nutrition play an important role in determining the susceptibility of an individual to these diseases (Ozanne & Hales, 2002).

2.7.4 Intrauterine growth retardation and low birth weight

A number of studies, mostly in developing countries, have suggested that intrauterine growth retardation and low birth weight are associated with

subsequent development of insulin resistance (Stern *et al.*, 2000, cited by WHO, 2003). In those countries where there has been chronic undernutrition, insulin resistance may have been selectively advantageous in terms of surviving famine. In populations where energy intake has increased and lifestyles have become sedentary, however, insulin resistance and the consequent risk of type 2 diabetes have been enhanced. In particular, rapid postnatal catch-up growth appears to further increase the risk of type 2 diabetes later in life (WHO, 2003).

2.7.5 Family history

A family history of type 2 diabetes is a risk factor for insulin resistance, as suggested by the high frequency of such a history in newly diagnosed children with type 2 diabetes, from 85 to 100 percent in reported series (Dabelea *et al.*, 1999).

2.7.6 Geographical location and the pathogenesis of diabetes mellitus

The thin type 2 diabetic patients in Sub-Saharan Africa are often from rural areas, and the clinical picture is similar to that of type 1 diabetic patients. The BMI of the non-obese type 2 diabetic patients was approximately 22 kg/m^2 similar to that of their type 1 diabetic patients. In addition, the beta cell function in the non-obese type 2 diabetes patients is severely diminished at fasting and during glucose challenge. As expected, the serum C-peptide values were significantly lower in the lean than in the obese type 2 diabetic patients. In addition, the obese type 2 diabetic patients often reside in cities or urban areas. Thus, westernization, with its associated obesity and insulin resistance, tends to modify the metabolic characterization of type 2 diabetes in Sub-Saharan Africa (Papoz et al., cited by Osei et al, 2003).

2.7.7 Sex, age and ethnicity

Type 2 diabetes is associated with aging. This can be attributed to a loss of lean body mass and an increase in adipose tissue, especially in sedentary individuals. This results in less muscle tissue available for glucose disposal and relatively more adipose tissue, leading to insulin resistance in susceptible individuals (Goldstein, 2002).

2.7.8 Behavioural and lifestyle-related risk factors

Diet, fat intake, obesity, physical inactivity, alcohol intake and polycystic ovary syndrome are some of the risk factors.

2.7.8.1 Diet

It is estimated that by 2020 two-thirds of the global burden of disease will be attributable to chronic non-communicable diseases, most of them strongly associated with diet. The nutrition transition towards refined foods, foods of animal origin and increased fats, plays a major role in the current global epidemics of obesity, diabetes and cardiovascular diseases, among other non-communicable conditions (Chopra *et al.*, 2003).

2.7.8.2 Fat intake

The majority of studies in both animals and humans have suggested that higher concentrations of total dietary fat, regardless of fat type, produce greater insulin resistance. The Insulin Resistance Atherosclerosis Study (Mayer-Davis *et al.*, 1997) found a significant relationship between the percentage of energy from total fat and insulin sensitivity. In the San Luis

Valley Diabetes Study (Marshall *et al.*, 1994), an increase in fat intake of forty gram per day was associated with a 3.4-fold increase risk for type 2 diabetes, even after adjusting for obesity.

2.7.8.3 Obesity

Diabetes is more common in individuals who are overweight. The risk of developing type 2 diabetes mellitus increases with the amount of excess weight, the duration of the obesity and the central deposition of the fat. Women who are more overweight and gaining weight are strong predictors of diabetes. The prevalence of diabetes is almost three times as high in the obese as in the non-obese (WHO, 1998).

2.7.8.4 Physical inactivity

Physical inactivity increases the risk of diabetes, independent of obesity. In addition, physically active societies have a lower incidence of diabetes than less active societies; and cross-sectional studies have demonstrated an inverse association between the prevalence of type 2 diabetes and physical activity. This inverse association has been largely attributed to the fact that exercise increases insulin sensitivity, improves glucose tolerance, and promotes weight loss (Kelly and Goodpaster, 2001).

2.7.8.5 Alcohol intake

The French Paradox relates to the observation that mortality rates due to coronary heart disease are relatively low in France, despite a diet rich in saturated fats. Another paradox linked to alcohol is the diverse associations of acute and chronic use with respect to insulin resistance, incidence of type 2

diabetes and incidence of cardiovascular disease in type 2 diabetes (Zilkens and Puddey, 2003).

2.7.8.6 Polycystic ovary syndrome

Polycystic ovary syndrome (PCOS) is a common disorder that affects premenopausal women and is characterized by chronic anovulation and hyperandrogenism. Insulin resistance is seen in a significant subset of women with PCOS, and the disorder substantially increases the risk for type 2 diabetes mellitus, independent of the effects of obesity (Powers, 2001, p.2116).

2.7.8.7 Other predisposing factors

Other predisposing factors are as follows:

- Drugs (steroids and thaizides)
- Pancreatic diseases (chronic pancreatitic diseases, surgery with > 90% pancreas removed, haemochromatosis, cystic fibrosis)
- Endocrine diseases (Cushing's, acromegaly, phaeochromocytoma, thyrotoxicosis)
- Glycogen storage diseases (Hope *et al.*, 1993, p.528).

2.8 CLINICAL SYMPTOMS AND SIGNS

Two thirds of type 2 diabetic patients in the U.K. are obese (Williams & Monson, 1994, p.754). A prevalence of hyperglycaemic symptoms like polyuria, thirst and polydypsia, have usually been present for months or even years (Williams & Monson, 1994, p.754; Powers, 2001. p.2111). An acute presentation and sudden marked weight loss are unusual but may occur, especially with intercurrent illness, introduction of diabetogenic drugs or

carcinoma of the pancreas, which is associated with type 2 diabetes in older people. Women often have troublesome pruritis vulvae due to candidiasis. Type 2 diabetes often has a long sub-clinical cause, and many asymptomatic patients are diagnosed incidentally when screened routinely for glycosuria or hyperglycaemia. Others present with chronic diabetic complications, such as myocardial infarction, retinopathy (especially maculopathy), or foot ulceration (Williams & Monson, 1994, p.754).

Polyuria (excessive elimination of urine), polydypsia (excessive drinking of water), polyphagia (excessive eating), loss of weight, and asthenia (lack of energy), are the earlier symptoms of diabetes. Polyuria is due to the osmotic diuretic effects of glucose in the kidney tubules. In turn, polydypsia is due to the dehydration resulting from polyuria. The failure of glucose (and protein) metabolism by the body causes loss of weight and a tendency toward polyphagia. Asthenia is caused mainly by loss of body protein, but also by diminished utilization of carbohydrates for energy (Guyton & Hall, 1996, p.981).

2.9 DIAGNOSIS

In South Africa, the proposed criteria for diagnosis and management of diabetes mellitus are symptoms of diabetes, accompanied by a random plasma glucose concentration of ≥ 11.1 mmol/l; fasting plasma glucose concentration of ≥ 7 mmol/l, or a 2 hourly plasma glucose concentration of ≥ 11.1 mmol/l during an oral glucose tolerance test. Random is classified as any time of the day without considering the last meal. Fasting is defined as no energy intake for at least 8 hours (SEMDSA, 2003). It is imperative to achieve glycaemic control after diagnosis. SEMDSA's recommendations for glycaemic control (2003), is illustrated in Table 1.

Table 1: SEMDSA's recommendations for glycaemic control (2003)

Biochemical index	Optimal	Acceptable	Additional action suggested
Capillary blood glucose values (fingerprick)			
Fasting (mmol/l)	4-6	6-8	> 8
Two-hourly postprandial (mmol/l)	4-8	8-10	>10
HbA_{1c} (%)	< 7	7-8	> 8
Weight			
BMI (kg/m^2)	< 25		> 27*
Waist circumference (cm):			
Male	< 94		> 102
Female	< 82		> 88

* In the presence of diabetes mellitus, this level is 27 and not 30.

2.9.1 Laboratory tests

Laboratory tests include blood glucose concentration, fructosamine concentration, glycosylated haemoglobin, GAD 65 and C-Peptide.

2.9.1.1 Blood glucose concentration

Glucose reagent is used to measure the glucose concentration by a timed end-point method. In reaction with hexokinase (HK) catalysts, the transfer of a phosphate group from adenosine triphosphate (ATP) to glucose to form adenosine diphosphate (ADP) and glucose-6 phosphate occurs. The glucose-6 phosphate is then oxidized to 6-phosphogluconate, with the concominant reduction of ß-nicotinamide adenine dinucleotide (NAD) to reduced ß-nicotinamide adenine dinucleotide (NADH), by the catalytitc action of glucose-6- phosphate dehydrogenase (G6PDPH) (Glucose, 1998).

A blood glucose concentration is frequently obtained by physicians as part of routine blood chemistries that are monitored in patients with diabetes. As the test is actually performed on serum or plasma rather than whole blood, values will probably vary from the whole blood glucose determinants performed on capillary blood in the finger stick method. Laboratory values are usually 10 to 15% higher than capillary values. Fasting blood glucose measurements are performed after an overnight fast of 8 to 12 hours. The glycaemic excursions due to food consumed more than 8 hours earlier have resolved and, thus, do not influence the measurement. Patients are usually instructed to take their oral hypoglycaemic agent after the blood sample is obtained. In type 2 diabetics the fasting glucose concentration is a measure of the patient's own overnight insulin secretion, and can indicate the need for further improvement in metabolic control, by either weight reduction, initiation/adjustment of oral hypoglycaemic dose, or initiation of insulin (Powers, 1996, p.141).

- **Two-hourly Postprandial Plasma Glucose Measurements**

The two-hourly postprandial glucose measurements are often used in conjunction with the measurement of fasting plasma glucose. The patient is advised to consume a meal that contains approximately 100 grams of

carbohydrates. Two hours after eating, a blood sample is drawn for plasma glucose measurement. A glucose value greater than 11.1 mmol/l indicates diabetes mellitus. A variation of this test is to use a standardised load of glucose. A solution containing 75 grams of glucose is administered, and a specimen for plasma glucose measurement is drawn two hours later (Bishop *et al.*, 1996, p.307).

- **Oral Glucose Tolerance Test**

The National Diabetes Data Group of the United States recommends the test be performed on ambulatory individuals who are not on restricted diets. Approximately 150g of carbohydrates should be consumed on each of the preceding 3 days. A sample of the patient's blood is drawn after an overnight fast. The patient then consumes 75g of glucose solution and blood is drawn every 30 minutes for two hours. Plasma glucose greater or equal to 11.0 mmol/l at the 2-hourly time point and at one other time point, indicates diabetes mellitus (Bishop *et al.*, 1996, p.307).

2.9.1.2 Fructosamine

Fructosamine is a time averaged indicator of blood glucose concentrations and is used to assess the glycaemic status of diabetics (Fructosamine, 2000). Fructosamine refers to any glycated serum protein. Typically, the predominant glycated serum protein is albumin, and the degree of glycolasation of albumin is then a measure of the glucose control of the patient over a period of time related to the half-life of albumin. The principle for the analysis of fructosamine is the reduction of the dye nitroblue tetrezolium. The half-life of albumin is approximately 2½ weeks, and thus fructosamine reflects short-term glucose control (Bishop *et al.*, 1996, p.309). The concentration of glycated protein such as, glycohaemoglobin, glycoalbumin or glycated total protein, is generally recognized to be valuable in evaluating the glycaemic status of diabetic subjects (Fructosamine, 2000).

2.9.1.3 Glycosylated haemoglobin (HbA_{1c})

Glycosylated haemoglobin (HbA_{1c}) is produced when haemoglobin reacts with high concentrations of serum glucose. Because it increases with continued glucose exposure for the life of the red blood cell, it affords an assessment for long-term (60 to 120 days) blood glucose control. The normal ranges for glycosylated haemoglobin are method dependant, although a concentration of around 5 to 7% is within the normal range for most methods (Bishop *et al.*, 1996, p.308). The Department of Chemical Pathology, University of the Free State uses a range of between 4.4 and 6.4%. Values of 12% or greater can be expected in diabetics with poor control (Bishop *et al.*, 1996, p.308). Patients with poor control should be monitored 2-4 times per year. A range between 6-7% is regarded as ideal for diabetics (SEMDSA, 2003). These patients should be evaluated annually (Levy, 1999).

Although practice differs widely between clinics, insulin treatment should be considered for patients on the maximum dose of medication if the HbA_{1c} > 8%. Insulin treatment would be justified according to the UKPDS findings. Patients with percentages consistently > 9% after dietetic review and maximizing oral medication, carry a high risk of complications, and are likely to be associated with osmotic symptoms and lethargy. Acetaldehyde, a metabolite of ethanol, can reduce HbA_{1c} percentages in combination with haemoglobin. The effect of alcohol and the subsequent reduction in HbA1c should be taken into account when evaluating glycaemia of alcohol users (Levy, 1999). A 1% increase in HbA_{1c} increase the risk of developing retinopathy over 10 years by 60 to 70%, and the risk of death from ischemic heart disease by 10%. An HbA_{1c} of 10% at baseline predicted a 10-fold increase in the risk of microvascular end-points, and a 2.5 fold increase in the risk of myocardial infarction (Yki-Järvinen, 2000). Measurement of HbA_{1c} is accepted as a method to measure long-term glucose control in subjects with diabetes mellitus. Determination of HbA_{1c} provides an important diagnostic

tool for monitoring the efficiency of dietary control and therapy during the treatment of diabetes mellitus. Long term treatment of the disease emphasizes control of blood glucose concentrations in preventing the acute complications of ketosis and hyperglycaemia (Hemoglobin A_{1c}, 1998). A range between 7-8% is acceptable and additional action is suggested if the HbA_{1C} was >8% as recommended by SEMDSA (SEMDSA, 2003).

The United Kingdom Prospective Diabetes Study (UKPDS, 1998) showed a comparable degree of benefit in macrovascular complications in type 2 diabetes. Fasting glucose measurements correlate closely with HbA_{1c} in diet- and tablet-treated type 2 diabetes, which makes routine measurements of HbA_1C less critical than in type 1 or insulin-treated type 2 (Levy, 1999).

The Diabetes Control and Complications Trial Research Group reported the relationship between HbA_{1c} and average blood glucose during the preceding two to three months as 1% ΔHbA_{1c} = 1.6 mmol/L (30 mg/dL) Δaverage blood glucose (Glycated Hemoglobin, 1994). Table 2 illustrates the relationship between HbA_{1c} and average blood glucose.

Table 2: Relationship between HbA_{1c} and average blood glucose

(Glycated Hemoglobin, 1994)

% HbA_{1c}	Average Glucose (mmol/l)
4	3.3
5	5.0
6	6.6
7	8.3
8	10.0
9	11.6
10	13.3
11	15.0
12	16.6

2.9.1.4 GAD 65

Type 2 diabetes is a heterogenous and multiform disease due to peripheral resistance to insulin. Some subjects with type 2 diabetes gradually develop insulin deficiency, a condition referred to as "slowly progressive type 1 diabetes" (especially in non-obese subjects). The beta cell destruction seen in this case is thought to be induced by pancreatic autoimmunity. The presence of autoantibodies to glutamic acid decarboxylase (GAD-AB) may be useful to distinguish subjects with type 1 versus type 2 diabetes in adults. The reference range is between 0 - 1 U/ml. The result is positive in type 1 diabetes mellitus and LADA (late onset auto-immune diabetes mellitus of the adult). GAD exists in 2 major isoforms: 65 and 67 kD. It is established that antibodies against the GAD65 kD form are more specific in the above

purposes. This procedure specifically measures GAD65 antibodies (GAD-AB, 2003).

2.9.1.5 C-peptide

Human insulin and C-peptide originate as a single polypeptide chain known as pro-insulin, which is formed on the surface of the rough endoplasmic reticulum in the beta cells of the Islets of Langerhans. Proinsulin is then transported to the Golgi complex of the beta cells, where it is packed into granules. It is in these granules that pro-insulin is cleaved proteolytically into insulin and C-Peptide. Insulin and C-Peptide are stored in these beta cell granules until their secretion is stimulated, at which time approximately equimolar amounts of each are released into the portal vein. Because of differences in uptake in the liver and in clearance times of these peptides, peripheral concentrations of C-peptide are higher than insulin. While insulin has a pervasive influence on the body, affecting virtually every organ and biochemical component, C-peptide has no known physiologic function, other than possibly facilitating correct insulin conformation. Insulin and C-peptide do not share basic antigenic components and, therefore, antisera directed against insulin will not cross-react with C-peptide. It is a lack of immunoreactivity that makes C-peptide measurement quite valuable. C-peptide and insulin concentrations correlate strongly in most sera, and assessment of C-peptide immunoreactivity will provide sound evaluation of beta cell function, and often eliminate the effect from endogenous insulin antibodies. The reference range is 265 - 1324 pmol/l (C-Peptide of Insulin, 2000).

2.9.1.6 Blood lipids

Blood lipids that will be discussed are cholesterol, high-density lipoprotein cholesterol, low-density lipoprotein cholesterol and triglycerides

- **Cholesterol**

Cholesterol measurements are used in the diagnosis and treatment of atherosclerotic coronary artery disease. Cholesterol measurements are also used in the diagnosis of metabolic disorders involving lipids and lipoproteins, such as HDL cholesterol, LDL cholesterol and triglycerides. Total serum cholesterol concentrations depend on many factors including age, gender, diet, physical activity, liver disease, and other metabolic disorders (Cholesterol, 2000). The cholesterol reference range used in this study was < 5.0 mmol/l as recommended by SEMDSA (SEMDSA, 2003).

- **HDL (high-density lipoprotein) cholesterol**

HDL cholesterol is inversely related to the risk of developing coronary heart disease. A low HDL/LDL cholesterol ratio is directly related to the risk of developing coronary artery disease (Powers, 1996, p.145). High HDL cholesterol is associated with the "longevity" syndrome (HDL cholesterol, 2000). HDL's reference range is > 1.2 mmol/l, as recommended by SEMDSA (SEMDSA, 2003).

- **LDL (low-density lipoprotein) cholesterol**

LDL cholesterol contains 50% cholesterol by weight and is the most cholesterol-rich of the lipoproteins. Lipoproteins are synthesized in the liver and are responsible for transporting cholesterol from the liver to the peripheral tissues. LDL particles have diameters ranging from 18 to 30 nanometers. LDL particle-size is inversely related to serum triglyceride concentrations. Smaller, denser LDL particles are associated with a higher risk for CHD (Warnick *et al.*, 1996, p.318; Powers, 1996, p.145). The risk of atherosclerosis steadily increases with the increasing LDL cholesterol concentration (Powers, 1996, p.145). The reference range is ≤ 3.0 mmol/l, as recommended by SEMDSA (SEMDSA, 2003).

- **Triglycerides**

Another lipid that is often measured is serum triglyceride. Triglycerides are also transported in the blood in association with various lipoproteins. The very low-density lipoproteins (VLDL) carry most of the circulating triglycerides. Hypertriglyceridemia is often seen in uncontrolled diabetes. Values between 2.83 – 5.65 mmol/l are associated with increased cardiovascular risks (Powers, 1996, p.145). Triglyceride measurements are used in the diagnosis and treatment of subjects with diabetes mellitus, liver obstruction, other diseases involving lipid metabolism, or various endocrine disorders (Triglycerides, 2000). The reference range for triglycerides is < 1.5 mmol/l, as recommended by SEMDSA (SEMDSA, 2003).

2.9.1.7 Renal Function Tests

Patients with diabetes are at increased risk for development of renal insufficiency. Serum creatinine and blood urea nitrogen (BUN) are both routinely obtained as part of automated chemistry screens. Patients who develop renal failure are not able to excrete urea or creatinine efficiently, and thus the concentrations in the blood rise. The elevation of serum creatinine or BUN concentration occurs at a rather late phase. Urinary studies provide a better assessment of kidney dysfunction in its earlier stages. The creatinine clearance is a measure of the ability of the kidney glomerulus to filter creatinine, and thus is an indicator of the glomerular filtration rate. Microalbuminuria can be detected by sensitive radioimmunoassay. It is replacing previously described urinary protein measurements for the routine detection of early renal insufficiency in diabetes (Powers, 1996, p.145).

2.10 MANAGEMENT

The present philosophy in diabetes management is to maintain blood glucose concentrations as close to normal as possible, without causing hypoglycaemia. With the availability of equipment to self-monitor blood glucose, it has become possible to achieve euglycaemia.

2.10.1 Medical nutrition therapy

Medical nutrition therapy (MNT) is an integral component of diabetes management and diabetes self-management education. MNT for diabetes includes the process and the system by which nutritional care is provided for diabetic individuals and the specific lifestyle recommendations for that care. These recommendations should also take into consideration lifestyle changes the individuals can make and maintain. Cultural and ethnic preferences should be taken into account, and the person with diabetes should be involved in the decision-making process. Results from the Diabetes Control and Complications Trial (DCCT) and the U.K. Prospective Diabetes Study (UKPDS), convincingly demonstrated the importance of glycaemic control in preventing microvascular complications of diabetes. MNT in diabetes addresses not only glycaemic control, but other aspects of metabolic status as well, including dyslipidaemia and hypertension – major risk factors for cardiovascular disease. This is important, as macrovascular complications are the major contributors to the morbidity and mortality associated with diabetes (Franz *et al.*, 2002).

The health professional with the greatest expertise in providing MNT for diabetes is the registered dietician (RDSA); knowledgeable and skilled in diabetes management. Outcome studies have demonstrated that MNT provided by RDSA's results in a 2.0% decrease in HbA_{1c} in patients with newly diagnosed type 2 diabetes and a 1.0% decrease in HbA_{1c} in patients with an average 4-year duration of type 2 diabetes. The effectiveness of

dietician delivered MNT in improving dyslipidaemia has also been demonstrated. It is still essential that all team members involved in diabetes treatment and management are knowledgeable about MNT and supportive of the patient's need to make lifestyle changes (Franz *et al.*, 2002). The risk of complications in patients with type 2 diabetes is strongly associated with hyperglycaemia. Each 1% reduction in HbA_{1c} was associated with reductions in risk of 21% for any end point of diabetes, 21% for death related to diabetes, 14% for myocardial infarction, and 37% for microvascular complications. Any reduction in HbA_{1c} is likely to reduce risk of complications. Patients with HbA_{1c} values in the normal range have the lowest risk (Ousman & Sharma, 2001).

In a prospective seven day estimated weighed food record trial (Close *et al.*, 1992) 92 diabetic subjects' results were computer analysed and compared with non-diabetic British adults. Only three diabetic patients achieved the recommended 50-60% energy intake as carbohydrates, four achieved less than 30% as fat and 20 ate more than 30 g fibre per day. The overall results of these diabetic patients reflected those of non-diabetic subjects, except for the intake of protein and smaller intakes of sugar and alcohol. These findings affirm the problems currently faced in achieving the present recommendation for diabetic subjects (Close *et al.*, 1992). Food intake frequency – three meals or smaller meals and snacks – is not associated with long term differences in glucose, lipid, or insulin responses. Therefore, division of food intake should be based on individual preferences (Franz *et al.*, 2002).

2.10.1.1 Goals of medical nutrition therapy for diabetes

The following goals apply to all persons with diabetes according to Franz *et al.* (2002):

1. To attain and maintain optimal metabolic outcomes, including

   a) blood glucose concentrations in the normal range or as close to normal as is safely possible to prevent or reduce risk for complications of diabetes

   b) a lipid and lipoprotein profile that reduces the risk for macrovascular disease

   c) blood pressure levels that reduce the risk for vascular disease.

2. To prevent and treat chronic complications of diabetes; modify nutrient intake and lifestyle as appropriate for intervention and treatment of obesity, dyslipidemia, cardiovascular disease, hypertension, and nephropathy.

3. To improve health through healthy food choices and physical activity.

4. To address individual nutritional needs, taking into consideration personal and cultural preferences and lifestyle while respecting the individual's wishes and willingness to change.

- **Energy balance and obesity**

The prevalence of obesity has risen dramatically over the past three decades and is threatening to become a global epidemic. A substantial proportion of adults are at increased risk of morbidity and mortality as a result of increased body weight. As a consequence, total costs of obesity-associated diseases have been estimated to be four to seven percent of all healthcare

expenditures in several developed countries. Efforts to reduce the prevalence of obesity have focused on the fat content of the diet. High-fat diets are energy dense and usually lead to an increase in energy intake. This, together with the prevalence of low levels of physical activity, means a positive energy balance and weight gain is inevitable (Saris *et al.*, 2000). The rate of obesity and the number of "dieters" are increasing in parallel. Surveys consistently show that most adults are trying to lose or maintain weight. More than 54 million Americans are currently on a diet, yet the prevalence of overweight and obesity continues to increase. If dieting worked, obesity should be decreasing, or at least not be on the increase. It is true that many dieters succeed in losing weight, but very few—maybe just five percent, but at most ten percent—manage to keep the weight off over the long term (Franz, 2001a).

Because of the effects of obesity on insulin resistance, weight loss is an important therapeutic objective for obese individuals with type 2 diabetes. Short-term studies lasting 6 months or less have demonstrated that weight loss in type 2 diabetic patients is associated with decreased insulin resistance, improved measures of glycaemia, reduced serum lipids, and reduced blood pressure. Long-term data assessing the extent to which these improvements can be maintained in people with type 2 diabetes are not available (Franz *et al.*, 2002). Data from the general public suggest that long-term maintenance of weight loss is challenging. In two observational studies on weight maintenance after weight loss in non-diabetic subjects, one study reported that only six percent in the final study group maintained a five percent weight loss over 9-15 years, while in a random telephone survey 21% of 228 overweight subjects reported that they had intentionally lost weight and maintained a weight loss of ten percent for at least less or equal to five years. In studies of weight loss of type 2 diabetic subjects, the most successful long-term weight loss from a diet was reported in the Diabetes Treatment Study, with weight loss of nine kilograms maintained over a six-year study period (Franz *et al.*, 2002).

Statistics suggest that about half of the adult population in the United States is overweight (BMI: 25–30kg/m^2), and 16% are obese (BMI >30kg/m^2) (Franz, 2001a). In South-Africa, 29% of adult men are overweight and another 9% are obese. Overweight occurs in 55% women and 29% of all women in South-Africa are obese (Dept. of Health, 1999). It is not surprising that so many consumers are searching for the "magic bullet" that will allow them to lose weight quickly and effortlessly. Unfortunately, health professionals also contribute to this phenomenon by constantly warning the public and their patients about the perils of being overweight. Herein lies the quandary—how to solve the problem of increasing obesity and its related health risks without making the problem worse (Franz, 2001a). Early in the course of the disease when insulin resistance is present, energy restriction not related to weight loss and moderate weight loss (5–10% of body weight or 4.5–9.1kg) have been shown to improve glycaemia, but as the disease progresses and insulin deficiency becomes the central issue, it may be too late for weight loss to be helpful (Franz, 2001a). If individuals with type 2 diabetes must lose weight, all that is required is a 9.1kg loss to achieve glycaemia.

The challenge for health professionals is to convince overweight diabetic individuals that this initial 5-10% weight loss should be their goal (Franz, 2001a). Foster *et al.* (1997) reported that women participating in a weight-loss program expected to lose 34% of their body weight. However, after 48 weeks of treatment, they had lost only 16% of their initial weight, and they reported being unsatisfied with their weight loss. Katan and co-workers (1997) questioned the importance of low-fat diets in the prevention and treatment of obesity. Randomized controlled trials showed only very limited weight reduction and the so-called "fat-paradox" can be seen in several countries where there is a poor association between dietary fat intake and percentage of the population that is overweight. Also a direct relation between dietary fat and energy density has been questioned on the basis that many low-fat foods currently available are claimed to be based on sugar or highly refined carbohydrates, leading to energy density values similar to those of their high fat counter parts (Saris *et al.*, 2000). Many individuals with type 2 diabetes are

overweight, with ~36% having a BMI ≥ 30 kg/m^2, which would classify them as obese. The prevalence of obesity is higher in women and members of minority populations with type 2 diabetes. As body adiposity increases, so does insulin resistance. Obesity may also aggravate hyperlipidaemia and hypertension in type 2 diabetics (Franz *et al.*, 2002). Modest weight loss (4.5 to 9.1 kg), even if the person is still overweight, can improve control of diabetes and reduce the risk for heart disease by lowering blood pressure and blood cholesterol (Whitney & Rolfes, 2002, p.281).

- **Carbohydrates**

Carbohydrates should provide 50-60% of energy intake (Anderson, 1999, p.1375; Lank, 2000). Greater amounts, up to 70%, are tolerated in research studies and by highly committed individuals, but are not generally recommended for most people with diabetes. Under certain circumstances, especially with low fibre intake, high-carbohydrate, low-fat diets can worsen blood glucose control, increase serum triglyceride concentrations and lower HDL cholesterol concentrations (Anderson, 1999, p.1375). Of total energy, 60-70%, should be divided between carbohydrates and monounsaturated fat (Franz *et al.*, 2002). The main rationale for increased carbohydrate intake is the desire to reduce the content of fat, especially saturated fat, without a concomitant increase in dietary protein (Vessby, 1994).

Carbohydrate counting is a meal planning approach used with diabetic subjects that focuses on carbohydrate as the primary nutrient affecting postprandial response. The concept of carbohydrate counting was used as one of four meal planning approaches in the Diabetes Control and Complications Trial. In the trial, carbohydrate counting seemed to be effective in meeting outcome goals, and allowed flexibility in food choices with the potential of improving metabolic control (Gillespie *et al.*, 1998). In weight-maintaining diets for type 2 patients with diabetes, replacing carbohydrate with monounsaturated fat reduces postprandial glycaemia and triglyceridemia

(Whitney & Rolfes, 2002, p.115; Franz *et al.*, 2002), but there is concern that increased fat intake may promote weight gain and potentially contribute to insulin resistance. This should be individualised based on nutrition assessment, metabolic profiles, and weight and treatment goals (Franz *et al.*, 2002).

A study by Marshall and co-workers (1997), showed that dietary fibre and starch intake were inversely associated with fasting insulin concentrations and may have implications for attempting primary intervention of type 2 diabetes mellitus. In individuals with type 2 diabetes, postprandial glucose concentrations and insulin responses to a variety of starches and sucrose are similar, if the amount of carbohydrate is constant. This has been demonstrated in both controlled and free-living subjects. When studied, the effects of starches and sucrose on plasma lipids were similar and no adverse effects where observed (Franz *et al.*, 2002).

- **Glycaemic index**

The glycaemic index (GI) is a system of classifying foods that contain carbohydrate (CHO) based on their acute glycaemic response with the view that the slower the response, the better it may facilitate glycaemic control and lipid profiles in people with diabetes (Whitney & Rolfes, 2002; Workgroup: Diabetes and diet, 2000, p.9). Furthermore, foods with a low GI elicit a greater satiety effect.

The GI is defined as:

$$GI = \frac{\text{Incremental blood glucose area after test food}}{\text{Corresponding area after equicarbohydrate portions of standard CHO food}}$$

Some diabetics may have been taught to differentiate between complex carbohydrates (e.g., cereal) and simple carbohydrates (e.g., table sugar). These terms are no longer in favour, as they do not indicate the impact of the carbohydrate on blood glucose. Instead, dieticians now consider something known as the 'glycaemic index' (Lank, 2000). GI values obtained from various centres can differ due to varied methods used to determine the GI of test foods. Either glucose or white bread can be used as the reference food. Those scientists who use a 50g carbohydrate portion of white bread do so because it is more physiological – typical of what is actually eaten. On this scale, where the GI factor of white bread is set at 100, some foods will have a GI value of over 100, because their effect on blood sugar concentrations is higher than that of bread. The use of the two standards has caused some confusion, but it is possible to convert from one to the other using the factor of 1.4 (100/70 – white bread has a value of 70 when glucose is the reference food with a GI of 100). In any table with a list of foods and their respective GI values, the reference food should be stated and taken note of (Workgroup: Diabetes and diet, 2000, p.9).

Several factors affect the GI of food. Factors that affect the GI of a food are related to the chemical composition and physical form of the food, and include all factors that influence digestion of starch and absorption of glucose. The interaction of starch with other nutrients in a particular food or meal influences the GI, because fat and protein delays upper gastrointestinal transit. The GI of different sugars ranges from 20 for fructose, to 105 for maltose. Fructose elicits a low GI, probably because of the incomplete absorption and only partial conversion to glucose. Sucrose with an intermediate GI of 59 challenges the historical view that foods containing sugar cause an excessive rise in blood glucose concentrations, and that sugars elicit a higher glycaemic response than starches (Workgroup: Diabetes and diet, 2000, p.10). The amylose to amylopectin ratio of a starch, the type of heat applied (dry versus moist), temperature and time of cooking, influence the GI of starch. The cooling of cooked starch results in the retrogradation of the starch molecule

that is a more resistant type of starch. Therefore, cold maize porridge and cooled potato have a lower GI than their hot equivalents.

The GI is also affected by the degree of ripeness in fruits containing starch e.g. bananas. Addition of salt to bread and lentils increases the GI probably through the effects of amylase secretion or glucose absorption. The addition of vinegar or acid or "ting" lowers the GI of a food. The physical form of a food influences the GI. The higher the proportion of whole intact grain in a cereal results in a lower GI. Disruption to the grain increases the availability to enzyme digestion and starch gelatinization and, thus, elevates the GI. Processing like milling, grinding, puffing, canning, flaking and dry heating has been associated with higher GI's. Viscous, gel-forming and soluble fibre components are responsible for lowering the GI of foods. Anti-nutrients such as phytates, lectines, tannins and saponins, influence starch digestibility and thus the GI. The high concentrations in legumes could partly explain the low GI of legumes (Workgroup: Diabetes and diet, March 2000, p.11; Anderson, 1999, p.1375).

Jarvi and colleagues undertook a landmark controlled study in which individuals with type 2 diabetes were provided with fairly similar meals which, through the use of skilled food technology, differed only in glycameic index (Jarvi *et al*, 1999). Macronutrient composition and dietary fibre content were identical on the two diets. After 24 days, the low glycaemic index diet was associated with improved glycaemic control, reduced total and LDL cholesterol and reduced concentrations of plasminogen activator inhibitor-I (PAI-I) when compared with the high glycaemic index diet. This confirmed the ability of low GI foods to confer benefit, independent of dietary fibre in terms of not only blood glucose control and lipoprotein mediated cardiovascular risk reduction, but also a reduction in thrombogenic risk (Mann, 2001). The GI opens the way to greater flexibility of food choices for people with diabetes, because many traditionally taboo foods do not cause the unfavorable effects on blood sugar they were believed to have. The GI should never be used in

isolation, but incorporated along with current macronutrient and carbohydrate distribution recommendations. When educating people with diabetes the golden rule is to incorporate the GI into dietary advice e.g. to substitute high GI foods for low GI alternatives whenever possible or eat high GI and low GI within the same meal. It is important to distinguish between low GI carbohydrates and low GI fatty carbohydrates (Workgroup: Diabetes and diet, March 2000, p.11). The recommendation for subjects with diabetes mellitus concerning the glycaemic effect of carbohydrates, is that the total amount of carbohydrates in meals and snacks is more important than the source or type, and is the first priority in the planning of meals and snacks (Franz, 2001b).

In diabetic subjects, evidence from medium-term studies suggests replacing high-glycaemic-index carbohydrates with low-glycaemic–index carbohydrates, improves glycaemic control and, among persons treated with insulin, reduces hypoglycaemic effects (Willet *et al.*, 2002). The usefulness of the glycaemic index is surrounded by controversy as researchers' debate whether selecting foods based on the glycaemic index offers any health benefits. Those in favour of the using the glycaemic index in meal planning, claim that lowering the glycaemic index of the diets reduces insulin secretion and improves glucose and lipid metabolism, amongst others. Those opposing the use of the glycaemic index, argue that it is too complicated to teach: relatively few foods have had their glycaemic index determined, and this information is neither intuitively apparent, nor provided on food labels. Experts are concerned that instead of selecting a carbohydrate based on quality, consumers will reduce their carbohydrate quantity – that is, they will adopt a low-carbohydrate diet instead of a low glycaemic one (Whitney & Rolfes, 2002, p.108).

- **Fibre**

Originally fibre was defined as the components of plant cell walls that are indigestible in the human small intestine. Later, that definition was expanded to include storage polysaccharides within plant cells e.g. the gums in some

legumes (Mathers & Wolever, 2002). Soluble fibre from legumes, oats, fruits and some vegetables is capable of inhibiting glucose absorption from the small intestine, although the clinical significance is probably insignificant (Franz, 2000, p.753; Engel, 2003, p.1). Dietary fibre may be beneficial in treating or preventing several benign gastrointestinal disorders and colon cancer (Franz, 2000, p.753; Whitney & Rolfes, 2002, p.115). Insoluble fibres such as cellulose (as in cereal brans, fruits, and vegetables) enlarge stools, easing passage, and speed up transit time. In this way, the undigested fibres, together with the microbial growth they stimulate, help to alleviate or prevent constipation (Whitney & Rolfes, 2002, p.115; Engel, 2003, p.1).

Diets containing 20 g/day of soluble fibre may be capable of modestly reducing fasting circulating total and low-density lipoprotein cholesterol when administered in conjunction with a diet containing at least 50% carbohydrates. It is difficult to consume that level of soluble fibre in foods alone. Daily inclusion of a fibre containing breakfast cereal, whole-grain products, fruits, vegetables and legumes are useful (Franz, 2000, p.753). The ingestion of large amounts (24 to 50g) of fibre is necessary to confer metabolic benefit. It is not clear whether the palatability and gastrointestinal side effects of fibre in this amount would be acceptable to most people (Franz *et al.*, 2002). Engel (2003) suggested that to obtain 26 grams of fibre daily, one should have two fruits at breakfast time with whole grain cereal, fruit as an in-between snack, three to five servings of vegetables daily, and several grain servings. Mann (2001), stated that in order to achieve improved glycaemic control and reduced LDL without any increase in very low-density lipoproteins (VLDL) and triglycerides, or reduction in HDL, it was essential to increase dietary fibre, especially soluble fibre. Launching suddenly into a high-fibre diet can cause temporary bouts of abdominal discomfort, gas, and diarrhoea and, more seriously, can obstruct the GI tract (Whitney & Rolfes, 2002, p.115).

To prevent such complications, a person adopting a high-fibre diet is advised to:

- Increase fibre intake gradually over several weeks to give the GI tract time to adapt;
- Drink lots of fluids to soften the fibre as it moves through the GI tract;
- Select fibre-rich foods from a variety of sources like fruits, vegetables, legumes, and whole-grain breads and cereals (Whitney & Rolfes, 2002, p.115).

- **Nutritive sweeteners**

Nutritive sweeteners include sucrose, fructose and sugar replacers.

- **Sucrose**

Sucrose (table sugar, cane sugar, beet sugar, grape sugar) is a common, naturally occurring disaccharide composed of one glucose and one fructose molecule (Brand Miller *et al.*, 1995; Ettinger, 2000, p.46; Franz *et al.* 2002). Only half the glucose-equivalents are available compared with an equal carbohydrate portion of bread or glucose. The sweetness of fruit is determined by a mixture of glucose, fructose, sucrose and other sugars, which do not affect plasma glucose concentrations equally (Brand Miller *et al.*, 1995). For the general population, the average intake of total sugars is 18% of the total energy, which includes 11% from sucrose and other added sugars (Gillespie, 1996). The average per capita consumption of sucrose and other sugars in the U.S. increased since then to an estimated 94g/day, accounting for 22% of energy intake in 2002 (Franz *et al.*, 2002). Sugars are an important component of diets in developed countries, providing about 20% of the total energy consumed and nearly half the total carbohydrates. Approximately half the sugar is derived from added sugars and the other half from naturally-occurring sources, such as fruit and milk (Brand Miller *et al.*, 1995).

The American Diabetes Association (ADA) principles of 1994 state that sugars are an acceptable part of a healthy diet for people with diabetes, particularly sugars obtained from fruits, vegetables and dairy products. Published recommendations suggest limiting added sugars to 10% to 15% of the energy (Gillespie, 1996). Up to 10 percent of total daily calories can come from added sugars, such as table sugar and sugar-sweetened products, without negatively affecting blood glucose control in most people with diabetes. For example, if a subject consumed about 8,400 kilojoules in a day, the subject could have up to (but no more than) 840 kilojoules in the form of added sugar (Lank, 2000). The rationale that banned sugar for diabetics stems from Allen's work on dogs in the 1920's. He noted that dogs that had pancreatectomies showed greater glycosuria after glucose intake than after starch intake. He concluded that glucose, compared with low GI carbohydrates, caused a more rapid rise in blood glucose concentrations and, therefore, greater glycosuria. Unfortunately, this conclusion was expanded to all simple sugars, including sucrose (Wolever and Brand Miller, 1995).

Scientific evidence does not support this assumption (Abraira & Derler, 1988; Bantle *et al.*, 1993; Franz *et al.*, 2002). The American Diabetes Association (ADA) stated in their guidelines in 1994 (Gillespie, 1996), that scientific evidence proved that the inclusion of sucrose as part of a meal plan does not impair blood glucose control in patients with type 1 or type 2 diabetes mellitus. Along with other published research, Wheeler and co-workers' study supports the concept that sucrose-containing foods are not detrimental to persons with well-controlled type 1 diabetes mellitus, and can be safely included in meal planning for persons with diabetes. This conclusion does not imply, however, that persons with diabetes can eat unlimited amounts of sucrose containing foods. People with diabetes should be informed consumers who read labels and match serving size and nutrient information to individualized diabetic meal plans (Wheeler *et al.*, 1996; Cronier, 2003). Sucrose must substitute other carbohydrate-rich foods like starches, milk, and fruit within the context of a healthy meal plan and may not just be added additionally (ADA, 2000b).

The Diabetes Care and Education Practice Group of the American Dietetic Association conducted a survey on how its members implemented the 1994 diabetic recommendations. The overall response was positive, but more than 50% of the respondents felt that the liberalization of sucrose was controversial. Several patients and members thought that liberalizing sucrose intake was inappropriate and therefore questioned the dieticians' credibility as healthcare professional when recommending it. Some members of the group were concerned that patients would exchange more nutritious food like vegetables, fruit, grains and milk for sugary foods and sweets, and still be able to maintain a consistent carbohydrate intake. This is a valid concern and places great responsibility on diabetes educators to ensure that these carbohydrate recommendations are interpreted correctly, and that it does not compromise nutritional intake (Gillespie, 1996). This is shared by Nadeau and co-workers (2001) who found that despite the strong evidence that sucrose does not alter glycaemic control, health professionals often fear that teaching free-living patients the latest sugar guidelines will lead to a deterioration of eating habits and metabolic profile if the guidelines are not applied. Unfortunately, some health professionals and people with diabetes mellitus still believe that sucrose, as well as sucrose containing foods, should be avoided. Wheeler and co-workers' (1996) results, as well as others, refute the belief of rapidly absorbed sugars. They support nutrition recommendations that stress the importance of the quantity, rather than the source of carbohydrates in the management of diabetes mellitus (Wheeler *et al.*, 1996).

Many patients with diabetes appear unwilling to do without sweet-tasting foods, and it was reported that the fat restriction recommended for both diabetic and non-diabetic individuals becomes increasingly difficult once sucrose has been removed from the diet (sugar-fat see-saw). Even though there is not a specific nutritional need for added sugar, its exclusion from the diet may have important psychological and therapeutic consequences. Sugar is not just a source of "empty calories" if its use helps ipso facto to limit the intake of high GI starch and fatty foods that have been proven to have undesirable consequences on carbohydrate and lipid metabolism (Wolever & Brand Miller, 1995). As sucrose is found in so many foods, it is difficult to

exclude from the diet, even for the most dedicated diabetic patient. Most diabetic patients would welcome the possibility of selecting from a wider variety of foods without feeling guilty or anxious about the choices they made (Peterson *et al.*, 1986). Dieticians and healthcare professionals need to be willing and able to show clients how to appropriately use the carbohydrate treats that they probably will consume anyway, at least occasionally. On the other hand, some clients feel most comfortable simply avoiding sweets as they always have, and do not want the option of substituting sugar containing foods for other carbohydrate choices (Gillespie, 1996). Provision of such a diet will add to the quality of life, improve residential satisfaction and nutritional intake, thereby reducing the likelihood of malnutrition and nutritional deficiencies (Tariq *et al.*, 2001). Sparing use of sucrose taken during mixed meals might help well-controlled diabetic patients to comply with their daily dietary prescription while maintaining good blood glucose control (Slama *et al.*, 1984). Therefore, dieticians and health care providers should be encouraged to teach their patients how to incorporate the new "sugar guidelines", since doing so may increase patient awareness and understanding of the food exchange system, and consequently result in a better adherence to a healthy meal plan with more careful carbohydrate intake (Nadeau *et al.*, 2001). SEMDSA and DESSA, allow people with diabetes to include sucrose as part of an appropriate energy controlled, low fat, high fibre eating plan (Workgroup: Diabetes and diet, 2000, p.11). One of the guidelines in the position paper of the Association for Dietetics in South Africa (ADSA) (ADSA, 1997) for patients with well-controlled diabetes, was to allow sucrose (10% of the total energy intake) as part of a balanced diet. Foods containing sucrose have a relatively moderate glycaemic index and are, therefore, included in the diabetic diet. Health professionals are sceptical whether this may be advisable to all the patients they consult with (Daniels *et al.*, 2000). However, this position statement by SEMDSA and DESSA is in accordance with the American Diabetes Association (ADA) and the Canadian Diabetes Association (CDA) (Bantle, 1993; Nadeau *et al.*, 2001).

The American Diabetes Association stated in their dietary recommendations for people with diabetes that "In some individuals, modest amounts of sucrose

and other refined sugars may be acceptable, contingent on metabolic control and body weight". Bantle's study (1993) suggests that this recommendation is unnecessarily cautious, as a diet providing 19% of total energy as sucrose did not aggravate glycaemia in diabetic subjects. However, the subject sample size of the study was small and provided an 80% chance of detecting a difference between the study diets in mean plasma glucose of 1.5 mmol/l. Thus, small differences in glycaemia between the study diets might have been overlooked. Moreover, it is important to emphasize that the two study diets were iso-caloric. If individuals with diabetes add sucrose or sucrose-containing foods to their diets without reducing other sources of carbohydrate energy, increased glycaemia can be expected. Increased sucrose consumption would also increase the risk of dental caries (Bantle, 1993). Few documented studies in which free living diabetic patients choose their own food and incorporate sugar in their diets are available (Peterson *et al.*, 1986). Out of ten studies done to compare glycaemic effects of iso-caloric amounts of sucrose and starch in patients with diabetes mellitus, all had a small number of subjects (n: 6-24). Furthermore, these studies were done over a relatively short period of time, ranging from 6-24 weeks, or patients were given a single meal. In all these studies, prepared meals were provided to subjects by the investigators. The results of these studies have shown that the percentage energy derived from sucrose ranged from 7% up to 38%, with no adverse effect on glycaemia (Franz *et al.*, 1994). When sucrose was fed to diabetic subjects as a single nutrient, as part of a meal or as part of a snack, it did not cause a greater rise in blood glucose than iso-caloric amounts of starch-containing foods. Although several studies have attempted to assess the more chronic effects of dietary sucrose in diabetic subjects, only a few have established adequate control of study nutrients by providing subjects with meals prepared in a metabolic kitchen. Of these studies, one reported (Coulston *et al.*, 1985) that a diet containing 16% of energy as sucrose increased glycaemia relative to a reference diet nearly devoid of sucrose in type 2 diabetic subjects. However, two other studies (Abraira *et al.*, 1988; Slama *et al.*, 1984) found that dietary sucrose did not increase glycaemia relative to a high starch reference diet in either type 1 or type 2 diabetic subjects. Bantle and co-workers (1993) designed a study to further explore

this issue and assess the effects of sucrose on glycaemia and lipemia in diabetic subjects (Bantle *et al.*, 1993).

Whatever the mechanism, it is clear that sucrose in moderate amounts (17% of the total energy) can be exchanged gram for gram for complex carbohydrate in the context of a healthy meal plan without adversely affecting glucose control. Data from Sievenpiper and co-workers' (1998) study suggests that 20 to 35 g of sucrose, exchanged for starch, may actually improve glycaemic control between breakfast and lunch, at least when given to children with near normal fasting glucose concentrations. Theoretically, the lower glycaemic excursion with the 17% sucrose diet could suggest that more of the carbohydrate was stored either as glycogen or as fat. If so, sucrose-containing diets may maintain more normal blood glucose concentrations, but could, in time, facilitate excess weight gain. In clinical practice, excess weight gain occurs when energy intakes exceed energy needs, regardless of the source of excess energy, and may be prevented by good nutritional management and exercise (Sievenpiper *et al.*, 1998).

It is common practice for diabetics to use sweetening agents. In the diabetic clinic population of Colagiuri and co-workers (1989), 65% regularly used these products. These individuals were instructed to avoid added sucrose, based on the assumption that refined carbohydrates, sugars included, have a deleterious effect on postprandial glycaemia. However, the use of sweetening agents in South African clinics by diabetics is not typical practice (Taljaard *et al.*, 2003). The study by Colagiuri and co-workers (1989) showed that although aspartame was an acceptable sugar substitute for diabetics, it had no specific advantage over sucrose in the sense of glycaemic control, lipid levels, carbohydrate tolerance, or in vivo insulin action as a result of the daily addition of sucrose. Nadeau and co-workers determined the consequences of the inclusion of sucrose intake on dietary habits, metabolic control and perceived quality of life, by teaching free-living subjects with type 2 diabetes how to use and integrate the sugar guidelines. The hypothesis was that subjects who were taught how to incorporate the Canadian Diabetes Association (CDA) sugar choices, would forget to take into account the extra

carbohydrates and fat found in these foods, and consequently gain weight and/or deteriorate their metabolic control, while improving their perceived quality of life. Giving the subjects the freedom to eat added sugars and sweets did not result in any metabolic deterioration (Nadeau *et al.*, 2001). Nadeau and co-workers had hypothesized that "permitting" added sugars and sweets to people with type 2 diabetes would improve their perceived quality of life, by acknowledging the human desire for sweet tastes and minimizing the sense of deprivation and misplaced guilt of "cheating". The results in this study, however, did not indicate any significant impact on the subjects' perceived quality of life.

One could argue that the negligible change in quality of life may be attributable to the fact that the subjects were given the freedom to incorporate—or not to incorporate—the sugar guidelines, but in fact they chose to maintain a similar intake of sugar comparable to that of a non-diabetic population. This may be because of previous anti-sugar indoctrination and/or guilt (Nadeau *et al.*, 2001). Bantle (1993) found that the metabolic profile of their 12 subjects did not change significantly after consuming a diet containing 19% of calories as sucrose for 28 days. Abraira and Derler found similar results with 18 hospitalized subjects, who were fed a diet containing 38% of energy as sucrose (up to 120 g of sucrose) for 4 weeks (Abraira & Derler, 1988). The only study to evaluate the effect of dietary sucrose on free-living diabetic subjects demonstrated that a calculated menu (45 g sucrose compared with a diet consisting mainly of complex carbohydrates) prepared at home resulted in an increase in sucrose intake (from 10 to 18% of energy) without altering energy, protein, or fat intake. No deterioration was noted on the metabolic profile of these free-living individuals after a 6-week intervention period (Nadeau *et al.*, 2001).

A moderate amount of sucrose taken daily at mealtimes does not cause deterioration in metabolic control in diabetic patients following a high fibre/low fat diet. A diet containing approximately 45g of sucrose (around 18% total daily energy) can safely be taken by both type 1 and type 2 diabetic patients, regardless of their degree of glycaemic control (Peterson *et al.*, 1988).

Peterson and co-workers (1988) also found that subjects with long-standing diabetes initially had some difficulty adding sucrose to their diet, despite enjoying the freedom of eating a modest amount of sucrose (45 g/day).

In subjects with type 2 diabetes and poor blood glucose control, there were no significant differences in plasma glucose or insulin responses between test meals containing 24-25% sucrose, potato or wheat starch. In type 2 individuals with good control, Slama (1984), examined plasma glucose and insulin responses to a sucrose-sweetened rice cake and a saccharin-sweetened rice cake, taken at the end of a regular mixed meal; and Bornet and co-workers (1985), examined the effect of exchanging sucrose or honey for starch in a breakfast meal. In both studies there were no significant differences between sucrose- and starch-containing meals with respect to plasma glucose and insulin responses, whether in peak values, peaking times, or area under the curve. Sucrose will raise glycaemic responses if substituted for starchy foods, such as beans, that normally produce low blood glucose responses. Nevertheless, up to 20% of the starch from butter beans may be replaced by sucrose with minimal increase in glucose and insulin responses, but a large increase in palatability (Wolever and Brand Miller, 1995). According to Anderson (1999, p.1380), up to 25g of added sucrose may be allowed, provided it is part of a low-fat, high-fibre diet and that it substitutes for an iso-caloric amount of fat or high-glycaemic index food, or other nutritive sweeteners. There is no strong reason to recommend fructose in preference to sucrose within this limit. There appears to be no significant advantage of alternative nutritive sweeteners over sucrose (Franz, 2000, p.754). A study by Malerbi and co-workers (1996) demonstrated that a diet containing almost 80g sucrose/day; which comprised 19% of its total energy; did not cause any change in glycaemic control, serum lipid concentrations, or insulin and C-peptide secretion when compared with a diet deriving carbohydrate energy primarily from polysaccharides. Marchini and co-workers (1995) found that the glycaemic response of patients with diabetes who were fed added sucrose did not show worsening in glycaemic control. Bantle and co-workers reported no difference in the glycaemic response after ingesting different carbohydrates in both healthy individuals and patients with

diabetes. Coulston and co-workers (cited in Tariq *et al.*, 2001), found that short-term substitution of a regular diet for a diabetic diet did not result in deterioration of the glycaemic response in patients with type 2 diabetes at a chronic-care facility. The residents with diabetes in long-term-care facilities can be successfully managed with a regular diet, without a limitation on concentrated sweets, with the recommendation that glucose concentrations should be monitored and medication adjusted, rather than restricting the diet for these high-risk residents (Tariq *et al.*, 2001).

However, the data are not consistent with a report by Coulston *et al.* (1985) who fed type 2 diabetic subjects a diet containing 16% of energy as sucrose for 15 days and found that day-long plasma glucose concentration was higher than when the subjects were fed a sucrose-free reference diet. However, 57% of the carbohydrate energy in the sucrose-free reference diet was derived from sugars other than sucrose. These other sugars were not identified by the authors. If a significant percentage of the other sugars were fructose, then Coulston *et al.* (1985) may actually have compared a high sucrose diet with a high fructose diet. Because dietary fructose can reduce glycaemia in diabetic subjects, the higher day-long plasma glucose concentration during the sucrose diet may have been attributable to fructose present in the reference diet (Bantle, 1993).

Peters (1990) substituted chocolate cake for baked potato in a meal given to type 1 diabetics and reported no differences in the glucose response between the two meals. Loghmani *et al.* (1991), evaluated exchanging sucrose for starch in the diets of children with type I diabetes. They compared a 2% sucrose diet with a 10% (of the total energy) sucrose diet. The diets were followed for two days in a crossover design. This is limited by its short duration, but similar results in both groups were found (Franz *et al.*, 1993, pp.144-150). Patients with type 1 diabetes may substitute a sucrose-containing dessert for another carbohydrate in their diet without compromising their postprandial glucose response (Peters *et al.*, 1990). Wise *et al* (1989) studied the effect of sucrose containing snacks on blood glucose control in sixteen type 1 diabetics. One group ate sucrose sweetened snacks (7% of

total energy) twice a day, like 2 fudge brownies, 1½ iced cupcakes, or a slice of apple pie. The other group ate snacks (2% of the total energy) sweetened with aspartame. No significant differences were found between the control or the sucrose group on daily capillary blood glucose concentrations or fructosamine concentrations on days zero and five (Franz *et al.*, 1993). Equivalent gram amounts of carbohydrates as pre-sweetened breakfast cereals are not detrimental to person with type 1 diabetes mellitus, compared with unsweetened cereals. Therefore, pre-sweetened cereals can be used in the correct portion sizes and based on the number of carbohydrate or starch servings in a person's diabetic meal plan (Wheeler *et al.*, 1996). In adolescents with type I diabetes and fasting euglycaemia, the glycaemic responses of high sucrose and moderate sucrose did not differ, despite a two-fold increase in sucrose (17% to 35% energy). These data refute concerns of adverse glycaemic effects of sucrose and further support the use of higher sucrose-containing foods in mixed meals for adolescents with type I diabetes and fasting euglycaemia. (Rickard *et al.*, 2001).

Contrary to what might have been expected, but consistent with what is already found in type I and type 2 diabetic patients, exchanging an iso-glucidic iso-caloric amount of bread for sugar (sucrose) or honey (glucose and fructose), has no additional hyperglycaemic effect in type 2 diabetics, whether well- or uncontrolled. In acute conditions, substitution of part of a mixed meal by a reasonable amount of simple, currently available sugars, has no deleterious effect on blood glucose regulation and insulin secretion in type 2 diabetic patients. To what extent such sugars could be allowed in type 2 diabetic patients, very often obese, is to be discussed cautiously. They could at least be put to the test when negotiating with the patients to adopt a better commitment to the compulsory hypocaloric diet. The validity of these conclusions now has to be confirmed on long-term follow up studies (Bornet *et al.*, 1985). In a study by Brand Miller and co-workers (1995), the median GI of foods containing naturally occurring sugars was not significantly different from that of foods containing added sugars. In addition, they could find no significant difference between the responses to cakes, muffins and most cookies made with or without added sugars. Foods where added sugars

produced substantially higher values included canned peaches, milk, yoghurt, and soft drinks. However, even in these instances, the GI values of the foods containing added sugars were less than that of bread (Brand Miller *et al.*, 1995). Mbhenyane and co-workers (2001) determined the glycaemic index and insulin index of indigenous foods consumed in the Northern Province of South Africa. The addition of sugar to soft porridge made from sorghum or mealiemeal did not significantly influence the glycaemic and insulinaemic responses to these foods. Mieliemeal porridge without sugar produced a GI of 117, and with sugar 123 (Mbhenyane *et al.*, 2001). Restrictions on sucrose intake could be relaxed to correspond with recommendations on sugar intake for the normal, healthy population (Anderson, 1999, p.1380, Workgroup: Diabetes and diet, 2000, p.11). These results support the predictability of GI in the context of mixed meals. It is clear that the addition of sucrose (which has an intermediate GI) to a low GI food will increase the final GI, while the addition of sucrose to a high GI food will result in a low GI meal. The low mean GI of the foods studied is best explained by the composition of the individual sugars (Brand Miller *et al.*, 1995).

The available evidence from clinical studies demonstrates that dietary sucrose does not increase glycaemia more than iso-caloric amounts of starch. The intake of sucrose and sucrose-containing food in diabetic individuals need not be restricted, because of a concern of aggravating hyperglycaemia (Anderson, 1999, p.1380; Ludwig, 2000; Franz *et al.* 2002). If the sucrose is part of a balanced meal plan, it should be substituted for other carbohydrate sources or, if added, should be adequately covered with insulin or other glucose-lowering medication. The intake of other nutrients (such as fat) often ingested with sucrose-containing food should be taken into account (Franz *et al.* 2002). The total amount of carbohydrate is just as important as the source of the carbohydrate.

- Effects of sucrose on lipemia

Considerable controversy exists about the potential effects of dietary sucrose on lipemia in diabetic patients. In a study done by Bantle *et al.* (1993), the

sucrose did not result in any significant changes in serum cholesterol or serum triglycerides. Coulston *et al.* (1985) reported that type 2 diabetic patients fed a high sucrose diet and reference sucrose-free diet for 15 days, demonstrated increasing fasting plasma cholesterol and plasma triglyceride values during the high sucrose diet. In contrast Abraira and Derler (1988) reported that type 2 diabetic patients fed high sucrose (more than 75 fold difference in sucrose intake: 220g versus 3g) or high complex carbohydrate diets for 1 month, did not affect repeated fasting or postprandial glucose concentrations, glycohaemoglobin concentrations, or triglyceride and cholesterol concentrations in subjects with type 2 diabetes on no medication. Subjects with pre-existing high triglyceride concentrations actually had lower postprandial triglyceride concentrations when in the high sucrose group (Franz *et al.*, 1993). Colagiuri *et al.* (1989), found that fasting concentrations for total serum cholesterol, HDL cholesterol and triglycerides of patients with type 2 diabetes mellitus were not significantly different at the end of sucrose- or aspartame- supplemented periods, compared with pre-treatment concentrations (Gillespie, 1996). The first long term study of the effect of sucrose intake on glycaemia and lipemea was done by Peterson *et al.* (cited in Franz *et al.*, 1993), where diabetics followed a 45g sucrose replacement at each of three meals. At the end of the six weeks trial there was no difference between the diets in the day-long blood glucose concentrations. Cholesterol, triglyceride concentrations, medications and bodyweight remained unchanged. This finding suggested that substituting sucrose for carbohydrates in a meal does not cause deterioration in blood glucose or blood fat concentrations (Franz *et al.*, 1993). Thus, it is not certain whether dietary sucrose has adverse effects on serum lipids in diabetic subjects, and this probably should be considered an open issue.

- **Fructose**

Fructose is a common, naturally occurring monosaccharide (Anderson, 1999, p.1380) that accounts for ~ nine percent of the average energy intake in the U.S. (Franz *et al.*, 2002). Fructose is somewhat sweeter that sucrose.

Fructose produced a reduction in postprandial glycaemia when it replaced sucrose or starch as a carbohydrate source. Consumption of large amounts of fructose (15-20% of daily energy intake) has been shown to increase fasting total and LDL cholesterol in subjects with diabetes, and fasting total and LDL cholesterol and triglycerides in non-diabetic subjects, and therefore must be a concern (Franz *et al.*, 2002). Fructose provides 17 kJ/g, as do other carbohydrates, and it does have a lower glycaemic response than sucrose and other starches. There is no reason for people with type 2 diabetes to avoid fructose, which occurs naturally in fruits and vegetables, as well as in foods sweetened with fructose (Franz, 2000, p.754).

- Sugar replacers (previously called sugar alcohols)

Sorbitol, mannitol, and xylitol, as well as maltitol, isomalt and lactitol are common sugar alcohols that also have a lower glycaemic response and lower energy content than sucrose and other carbohydrates (Franz, 2000, p.754; Cronier, 2001; Whitney & Rolfes, 2002, p.127; Franz *et al.*, 2002). These sugar replacers add bulk and sweetness to cookies, hard candies, sugarless gums, jams, and jellies. Some products that may contain sugar replacers include hard candy, chocolate, table syrups, chewing gum, jams and jellies and some cough lozenges and syrups (Cronier, 2003). These products claim to be "sugar–free" on their labels, but in this case, "sugar-free" does not mean free of energy. Sugar replacers do provide energy, but fewer than their carbohydrate cousins, the sugars. Sugar alcohols occur naturally in fruit and vegetables; they are also used by manufacturers as a low-energy bulk ingredient in many products (Whitney & Rolfes, 2002, p.127). Because they are not soluble in water, they often are combined with fat. Therefore, foods sweetened with sugar alcohols may have an energy content similar to that of the foods they are replacing (Franz, 2000, p.754; Franz *et al*, 2002).

Some individuals report gastric discomfort (gas and abdominal discomfort), after eating foods sweetened by these products. Consuming large quantities may cause diarrhoea. For this reason, regulations require food labels to state that excess consumption may have a laxative effect. Reasonable

consumption could result in the daily ingestion of 50 grams of alcohol (Franz, 2000, p.754; Whitney & Rolfes, 2002, p.127; Franz *et al*, 2002). Starch hydrolysates are formed by the partial hydrolysis of edible starches. The reducing activity of starch hydrolysates can then be eliminated by hydrogenation, whereupon the product becomes a polyol (Franz, 2000, p.754; Franz *et al*, 2002).

- **Non-nutritive sweeteners**

Saccharin, aspartame, acesulfame K, and sucralose are non-caloric sweeteners currently approved for use in the United States. Alitame, cyclamates and neotame are awaiting approval from the Food and Drug Administration (FDA) (Franz *et al.*, 2002). Individuals with diabetes, including pregnant women, can use all FDA- approved non-nutritive sweeteners. Because saccharin crosses the placenta, other sweeteners are better options during pregnancy (Franz, 2000, p.754). It is not known if the use of non-nutritive sweeteners improves long-term glycaemic control or assists in weight loss (Franz *et al.*, 2002). Many people eat and drink products sweetened with artificial sweeteners to help them control weight. Ironically, a few studies have reported that intense sweeteners, such as aspartame, may stimulate appetite, which could lead to weight gain. Contradictory to these reports, most studies find no change in feelings of hunger, and no change in food intakes or body weight. Adding to the confusion, some studies report lower energy intakes and greater weight losses when people eat or drink artificially sweetened products (Whitney & Rolfes, 2002, p.126).

- **Protein**

Gluconeogenesis is accelerated in poorly controlled type 2 diabetes and may account for the majority of increased glucose production in the post absorptive state. The independent influence of dietary protein on glycaemia and insulin-sensitivity in well-controlled type 2 diabetes is minimal. Amino acids provide substrates for new hepatic glucose synthesis, but do not increase the rate of

hepatic glucose release (Franz, 2000, p754). The Recommended Dietary Allowance (RDA) for protein intake of 0.8g/kg/day for non-diabetic individuals is also appropriate for diabetics (Anderson, 1999, p1376; Franz, 2000, p.754). A protein intake of 10% to 20% of the daily energy intake is recommended (Franz, 2000, p.754). Protein accounts for 15 –20% of average adult energy intake in the U.S., a statistic that varied little from 1909 till the present. Protein intake in subjects with type 2 diabetes in the U.K. Prospective Diabetes Study was 21% of daily energy (Franz *et al.*, 2002). According to Anderson (1999, p.1376) diabetic individuals consume, on average, more protein than non-diabetic individuals do. People with type 2 diabetes have an increased need for protein during moderate hyperglycaemia, and an altered adaptive mechanism for protein sparing during weight loss. With energy restriction, the protein requirements of people with diabetes may be greater than the recommended dietary allowance (RDA) of 0,8 g protein/kg body weight, although not greater that usual intake, which is ~1.0 g protein/kg body weight or ~ 100g protein/day (Franz *et al.*, 2002). With the onset of nephropathy, protein intake should be no greater than the recommended dietary allowance of 0,8 g/kg. Then the percentage of the daily energy should be sufficiently restricted, and this is recommended for individuals with evidence of nephropathy (Anderson, 1999, p.1377).

- **Dietary fats**

The American Diabetes Association (2003) permits either a high-carbohydrate diet or a higher-fat diet enriched in polyunsaturated or monounsaturated fat. Yet, there is controversy as to the merits of either. As recently reviewed, the controversy revolves, to some extent around the fact that weight loss is more difficult to attain with a higher-fat diet; and a high-carbohydrate diet is associated with higher triglyceride and lower HDL concentrations than a higher-fat diet. Therefore, patients' degree of obesity should probably guide dietary choices. In addition, a diet high in fibre, particularly soluble fibre, may improve glycaemic control and concomitantly lower plasma lipid

concentrations in patients with type 2 diabetes (Henry, 2001). The proportion of saturated fat in the meal plan should be reduced. The ADA suggests an increase in either carbohydrate or monounsaturated fat to compensate for the reduction in saturated fat. Some (but not all) studies suggest that a high-monounsaturated fat diet may have better metabolic effects than a high-carbohydrate diet, although other experts have suggested that such a dietary modification may make weight loss more difficult in obese diabetic patients (American Diabetes Association, 2003).

- **Saturated fats and dietary cholesterol**

High total and saturated fat intake were associated with higher fasting insulin concentrations. These findings by Marshall and co-workers (1997), may have implications for studies attempting primary prevention of type 2 diabetes mellitus. The primary goal regarding dietary fat in patients with diabetes is to decrease intake of saturated fat and cholesterol. Saturated fat is the principal dietary determinant of LDL cholesterol. Compared to non-diabetic subjects, diabetic subjects have an increased risk of coronary heart disease with higher intakes of dietary cholesterol. The ADA and the National Cholesterol Education Program's Adult Treatment Panel III have recommended that food with a high content of saturated fatty acids and cholesterol be limited (Franz *et al.*, 2002). The goal for patients with diabetes remains the same as for the general population which is to reduce saturated fat to less than ten percent of the energy intake (Anderson, 1999, p.1377; Franz *et al.*, 2002). Individuals with LDL cholesterol ≥ 2,6 mmol/l may benefit by reducing saturated fat to less than seven percent of energy intake. The goal for dietary cholesterol intake is < 300 mg/day and for individuals with LDL cholesterol ≥ 2,6 mmol/l, < 200 mg/day (Franz *et al.*, 2002). The SEMDSA (2003) indicates that diabetic subjects should aim for a total-cholesterol concentration of < 5 mmol/l.

- **Monounsaturated fats**

Partial replacement of complex carbohydrates with monounsaturates in type 2 diabetics does not increase the concentration of LDL cholesterol and may improve glycaemic control, triglyceride, and HDL cholesterol concentrations. If triglycerides and VLDLs are elevated, a moderate increase in monounsaturated fat intake may be liberalized to include up to 20% of the energy with a more moderate intake of carbohydrates. Increased monounsaturated fat intake may enhance insulin resistance and, in obese individuals, may perpetuate or aggravate obesity (Anderson, 1999, p.1377). Diets high in cis-monounsaturated fatty acids or simply monounsaturated fat, or low in fat and high in carbohydrates, result in improvements in glucose tolerance and lipids, compared with diets high in saturated fat. Metabolic study diets in which energy intake is maintained, and which are high in either carbohydrates or monounsaturated fat, lower plasma LDL cholesterol equivalently. Low saturated fat, high-energy carbohydrate diets increase postprandial concentrations of plasma glucose and insulin, increase plasma triglycerides, and in some studies were shown to decrease plasma HDL cholesterol when compared in metabolic studies to isocaloric high-monounsaturated fat diets. High-monounsaturated fat diets have not been shown to improve fasting plasma glucose or HbA_{1c} (Franz *et al.*, 2002).

- **Polyunsaturated fats**

Only a few studies have evaluated the effects of polyunsaturated fats on plasma lipid concentrations and glycaemic control in subjects with diabetes. In one study (Anderson, 1999, p.1377) of type 2 subjects with diabetes, a diet high in total and polyunsaturated fat resulted in lower plasma totals and LDL cholesterol than a diet high in total saturated fat, but produced no difference in other plasma lipid concentrations (Anderson, 1999, p.1377; Franz *et al.*, 2002). A different study in type 2 diabetics compared a diet high in polyunsaturated fat with one high in monounsaturated fat, and reported higher

plasma totals and LDL cholesterol, fasting glucose, and insulin concentrations with the polyunsaturated fat diet (Franz *et al.*, 2002). Artificially high intakes and supplemental polyunsaturates are not advised, and the American Diabetes Association (ADA) recommends a restriction of below eight percent of energy. The World Health Organization (WHO) recommendation for the general population is three to seven percent of energy from polyunsaturates. High intakes of polyunsaturates have been suggested to be potentially damaging, relating to increased production of lipid peroxides (Anderson, 1999, p.1377).

- **Low fat diets**

There are potential benefits from low fat diets. Low fat diets are usually associated with modest loss of weight, which can be maintained as long as the diet is continued and if combined with aerobic exercise. Studies, which evaluated the effect of ad libitum energy intake as a function of dietary fat content, a low fat, high-carbohydrate intake are associated with a transient decrease in energy intake and modest weight loss, leading to a new equilibrium body weight. With this weight loss, a decrease in total cholesterol and plasma triglycerides and an increase in HDL cholesterol occur (Franz *et al.*, 2002).

- **Exercise**

Exercise should be an integral part of the treatment plan for patients with diabetes (Franz, 2000, p.758). The best approach to weight management combines diet and physical activity. People who combine diet and exercise are more likely to loose more fat, retain more muscle, and regain less weight than those only on diet. Even when people who include physical activity in their weight management program do not lose more weight, they seem to follow their diet plans more closely, and maintain their losses better than those who do not exercise. Exercise improves cardio respiratory fitness, regardless

of weight loss (Whitney & Rolfes, 2002, p.284). A minimum cumulative total energy expenditure of 4200 kilojoules/week from physical activities is recommended (Franz *et al.*, 2002). Exercise helps these patients to improve insulin sensitivity, reduce cardiovascular risk factors, control weight, and bring about a healthier outlook. Blood glucose control can improve with exercise because of decreased insulin resistance and increased insulin sensitivity, which results in the increased peripheral use of glucose not only during but also after the activity (Franz, 2000, p.758; American Diabetes Association, 2001).

Because enhanced insulin sensitivity is lost within 48 hours after exercise, repeated periods of exercise at regular intervals are needed to reduce the glucose intolerance associated with type 2 diabetes. This exercise-induced insulin sensitivity occurs without changes in body weight. Exercise also decreases the effect of counter regulatory hormones; and in turn, reduces hepatic glucose output, contributing to improved glucose control. Timing of the exercise can play a role. Exercise performed later in the day showed a reduced overnight hepatic glucose output and fasting glycaemia. Exercise after eating can reduce postprandial hyperglycaemia, common to type 2 diabetes (Franz, 2000, p.758). A complete exercise program includes warm-up and cool-down sessions. This prepares muscles for an aerobic workout and improves range of motion. Most people can undergo a walking program safely. The aerobic portion should last a minimum of 20 minutes, with a goal of 30 to 40 minutes. Even three sessions of 10 minutes of activity during the day can improve physical fitness (Franz, 2000, p.759; American Diabetes Association, 2001).

The insulin resistance syndrome in type 2 diabetics continues to gain support as an important risk factor for premature coronary heart disease, particularly with concomitant hypertension, hyperinsulinaemia, central obesity, and the overlap of abnormalities of hypertriglyceridemia, low HDL, altered LDL, and elevated free fatty acids. Most studies show that these patients have a low

level of fitness compared with control patients. Regular exercise has consistently been shown to be effective in reducing concentrations of triglyceride-rich VLDL. Effects of exercise on reducing blood pressure levels have been demonstrated most consistently in hyperinsulinaemic subjects. Accumulated data have indicated that exercise may enhance weight loss, and in particular weight maintenance, when used along with an appropriate energy controlled meal plan (American Diabetes Association, 2001).

- **Potential problems with exercise**

Hypoglycaemia is one potential problem associated with exercise; more after, than during exercise. The reason for this is that the repletion of liver and muscle glycogen can take 24 to 30 hours. Another problem is hyperglycaemia. Hyperglycaemia and worsening ketosis can result from insulin deficiency if exercise is started when blood glucose concentrations are higher than 13,88 to 16,65 mmol/l. With elevated fasting blood glucose and urine ketones, exercise should be postponed until control improves. The presence of peripheral neuropathy increases the risk of foot, soft tissue and joint injury. High quality footwear should be used. Self-monitoring of blood glucose pre- and post exercise is the safest way to understand how exercise affects diabetes control. Monitoring provides feedback to guide carbohydrate adjustments. In general, one hour of increased exercise requires an additional 15g of carbohydrates, either before or after exercise. Exercise that is more strenuous, requires 30g of carbohydrates per hour, and moderate exercise for less than 30 minutes rarely requires any additional carbohydrates. A small snack may be needed if the blood sugar concentration is < 5,5 mmol/l (Franz, 2000, p.759).

- **Alcohol**

According to the data for the periods 1989-1991, alcohol amounts for ~2.5% of energy intake in U.S. adults compared with the previous ~5% based on NHANES II data (1976-1980). It is not clear whether this can be attributable to decreased intake or to methodological differences in the measurement of alcohol intake. Nearly 67% of the adult U.S. population are reported to drink alcoholic beverages, whereas 33% claim to abstain from them. People with diabetes undoubtedly fall in both categories (Franz *et al.*, 2002). The alcohol in distilled spirits (hard liquor), wine, and beer is ethanol. It is a by-product of the oxidation of sugars by yeast enzymes. One drink or alcoholic beverage is commonly defined as 340 ml beer, a 142 ml glass of wine, or 1,5 glasses of distilled spirits, each which contains ~ 15g of alcohol (Franz *et al.*, 2002). From the moment an alcoholic beverage enters the body, it is treated as if it has special privileges. Unlike foods, which require time for ingestion, alcohol needs no digestion and is quickly absorbed. About 20% is absorbed directly across the walls of an empty stomach, and can reach the brain within a minute. Consequently, a person can immediately feel euphoric when drinking, especially on an empty stomach. Carbohydrate snacks, slow alcohol absorption, and high-fat snacks slow peristalsis, keeping alcohol in the stomach longer (Anderson, 1999, p.1381; Whitney & Rolfes, 2002, p.231). Alcohol consumption by type 2 diabetics should be limited to no more than two drinks per day for men and no more than one drink for women. In type 2 diabetics it is suggested than alcohol is best substituted as a fat exchange (one alcoholic beverage equals two fat exchanges) (Anderson, 1999, p.1381).

- **Alcohol and blood glucose concentrations**

Alcoholic beverages can have both hypo- and hyperglycaemic effects in patients with diabetes, depending on the amount of alcohol acutely ingested; if alcohol is consumed with or without food; and if alcohol use is chronic or excessive. Moderate or severe hypoglycaemia, no hypoglycaemia and

hyperglycaemia have all been reported in patients with diabetes after alcohol ingestion (Franz *et al.*, 2002). Alcohol cannot be converted to glucose and it blocks gluconeogenesis. It also increases the effects of insulin by interfering with the counter regulation response to insulin-induced hypoglycaemia. All these factors contribute to the development of hypoglycaemia when alcohol is consumed without food. Alcohol should always be consumed slowly and with food to lessen hypoglycaemic episodes. Identification should be worn indicating diabetic diagnosis, since the symptoms of insulin reaction and intoxication are similar (Anderson, 1999, p.1380; Franz, 2000, p755). Moderate amounts of alcohol can enhance the glucose-lowering action of exogenous insulin and certain glucose-lowering agents. Although alcohol does not affect the rate and degree of decline in plasma glucose, it appears to alter the phase of glucose recovery by interfering with hepatic gluconeogenesis. The hypoglycaemia induced by alcohol is not ameliorated by glucagon, because it is caused by indirect impairment of gluconeogenesis and is not associated with excessive insulin secretion (Franz *et al.*, 2002).

- **Relationship of alcohol to other health risks**

In people with type 2 diabetes, chronic alcohol ingestion (customary intake of ~ 45g/day) causes deterioration of long- and short- term glucose metabolism. Therefore, metabolic control should be carefully monitored if alcohol is an important component of a patient's diet. The effect induced by an excess of alcohol is reversed after abstinence from alcohol for 3 days. Alcohol ingestion increases the capacity for lipoprotein synthesis, especially of VLDL particles. This increase in synthesis is enhanced by a genetic predisposition, high-fat diet, and diabetes. Increased lipoprotein synthesis may be more an effect of chronic or excessive alcohol intake, as non-diabetic subjects with fasting hypertriglyceridaemia who, in one study, consumed the equivalent of two alcoholic beverages did not demonstrate an acute increase in triglycerides. This suggests that even people with hypertrygliceridaemia may occasionally use alcohol in moderation (Franz *et al.*, 2002). This differs from suggestions by Franz (2000) that persons whose blood glucose concentrations are out of

control and have elevated serum triglyceride concentrations, should avoid alcohol.

2.10.2 Medication

The two groups of medication used by diabetics are oral preparations and insulin preparations and will be discussed as follows.

2.10.2.1 Oral hypoglycaemic drugs

Oral hypoglycaemic agents are adjunct to the treatment of hypoglycaemia in type 2 diabetics, and not a substitute for diet and exercise. Weight gain may be observed because of fewer kilojoules lost through hyperglycosuria; the patient may feel better and have a better appetite; and compliance to diet and exercise is slacking. General contra-indications for oral hypoglycaemics are: type 1 diabetes mellitus; pregnant or lactating (relative) women; stressful concurrent conditions with significant hyperglycaemia like infections, trauma, myocardial infarction, sulphur allergy, significant renal impairment and use pre-operatively (Greeff, 2000). Table 3 indicates the types of oral hypoglycaemic drugs.

Table 3: Oral hypoglycaemic drugs

(GlaxoSmithKline, 2002; Greeff, 2000; Distiller, 2002).

Type		Action	Trade names
1. Insulin Sensitizers	Biguanides	Promotion of peripheral glucose uptake and utilisation in collaboration with insulin, suppression of hepatic glucose output. It also has some beneficial effects on lipids, it may reduce adipose-tissue mass and decreases platelet sensitivity to aggregating agents/ increased fibrinolytic activity.	Metformin (Glucophage®) Rolab Metformin®
	Thiazolidinediones	Reduces insulin resistance and improves pancreatic ß-cell function	Pioglitazone Rosiglitazone maleate (Avandia ®) Actos®
2. Insulin Secretogogues	Sulphonylureas	Stimulating endogenous insulin secretion. Decreases blood glucose by 20-30% (2.2-4.4 mmol/l)	Tolbutamide Chlorpropamide (Diabinese®) Glibenclamide (Daonil®) Gliclazide (Diamicron®) Glipizide (Minidiab®) Glimepiride (Amaryl®) Diabitex® Dimelor® Euglucon® Glucomed® Glycomin® Hypomide® Norton-glibenclamide® Rolab-Gliclazide® Rolab-Glibenclamide® Ziclin®
	Meglitinide Derivatives	Stimulator of endogenous insulin secretion. Shorter acting.	Repaglinide (Novonorm®) Meglitinide Nateglinide
3. Alpha-glycosidase inhibitors		Limits sugar absorption from the intestine	Acarbose (Glucobay®)

- **Combination therapy**

Combination therapy can be considered once the maximum dosage of one agent proves to be insufficient to adequately reduce sugar concentrations. Compliance in general, with life style modification and drug taking, as well as other factors that may influence the sugar concentrations, should always be borne in mind. If the second agent is initiated, it is always important that the lowest dosage is prescribed, and patients should be monitored and warned about hypoglycaemic episodes and the symptoms thereof. Any combination can be used and usually patients are started on either metformin or a sulphonylurea, and the alternative drug is added to the already maximum dosage of the initial agent (Greeff, 2000).

2.10.2.2 Insulin preparations

Insulin therapy is indicated in type 2 diabetic subjects who failed to achieve glycaemic control on diet plus optimal oral hypoglycaemic therapy. Type 2 subjects who are exposed to severe stress, like intercurrent illness, infections, or peri-operatively may need insulin. In type 2 subjects, who no longer respond to oral agents, once daily injections can be used. (Huddle & Kalk, 2000, p.17). Table 4 illustrates the different types of insulin.

Table 4: Types of insulin (Huddle & Kalk, 2000, p.53)

Type	Onset of action (hrs)	Peak of action (hrs)	Duration of action (hrs)	Trade names
Ultra short-acting	¼	½-1½	4-7	Humalog® (Lilly) (Lispro)
Rapid-acting	½-1	1-3	5-7	Actrapid® (HM) (Novo)
	½-1	1-3	5-7	Humulin® R (Lilly)
Intermediate	1-2	6-8	18-24	Humulin L® (Lilly) (Lente)
	1-2	4-12	18-24	Humulin N® (Lilly) (NPH)
	1-2	6-8	18-24	Monotard® (HM) (Novo) (Lente)
	1-2	4-12	18-24	Protophane® (Novo) (NPH)
Biphasic Insulins	½-1	4-12	24	Actraphane HM® (Novo)
	½-1	4-12	24	Humulin 30/70® (Lilly)
	¼	½-1½	24	Humalog (Mix25) ® (Lilly)
Dual-release insulin analogue	10-20 minutes	1-4	24	NovoMix®30 (Novo)

2.10.2.3 Medication interactions with alcohol and other drugs

Alcohol can potentiate the hypoglycaemic effect of sulfonylureas. Aspirin, phenylbutazone, sulfonamides, and monoamine oxidase inhibitors may have a similar effect, but not to the degree exerted by alcohol. Corticosteroids have a hyperglycaemic effect, as do oral contraceptives and thiazide diuretics. Hypoglycaemia appears more commonly with chlorpromide (Diabinese) and glyburide (Micronase, DiaBeta). This hypoglycaemia may last 24 to 48 hours and may be very severe (Mahan and Arlin, 1992, p.534).

2. 11 MONITORING

Monitoring includes self-monitoring of blood glucose, urine monitoring, blood pressure monitoring and the keeping of food and activity records.

2.11.1 Self-monitoring of blood glucose (SMBG)

SMBG using a finger prick of capillary blood with glucose-oxidase agent strips, read visually or from a reflectance meter (glucometer), can be used to achieve and maintain a target goal for glycaemic control, prevent and detect hypoglycaemia, and adjust regimens for lifestyle changes in persons taking hypoglycaemic agents (Powers, 1996, p.135). SMBG can be done up to seven times per day – before breakfast, lunch and dinner; at bedtime; one to two hours after meals; during the night (once a week); or to determine causes of hypoglycaemia or hyperglycaemia. Type 2 diabetics may perform glucose monitoring one to four times per day, but only three or four days per week. It is important that the results of SMBG be written in a record book, and that individuals are taught how to adjust their management program based on the results. In using blood glucose monitor records, it should be remembered that factors other than food affect blood glucose concentrations. An increase in blood glucose concentrations can be a result of insufficient oral medication, too much food, or increases in glucagon and other counterregulatory hormones because of stress, illness, or infection. Factors contributing to hypoglycaemia include too much oral medication, not enough food, unusual amounts of exercise, and skipped or delayed meals (Franz, 2000, p.760).

2.11.2 Urine monitoring

By assessing urine glucose concentrations, urine monitoring is a crude assessment tool of diabetes management and should not be used. However, urinary testing for ketones is the only practical way, especially if the blood glucose is greater than 13,3 mmol/l, or during illness. Persons with type 2 diabetes rarely have ketosis. It should be done in the presence of a serious illness and severe kilojoule restricted diets (Powers, 1996, p.140; Franz, 2000, p.760). Urine ketones have a negative reference range in type 2 diabetes mellitus. This test excludes type 1 diabetes mellitus if the reference range is negative (Combur[10] Test, 2001).

2.11.3 Blood pressure monitoring

Many patients with diabetes who are concerned about blood pressure may monitor their blood pressure at home. Dieticians are beginning to measure blood pressure as part of their routine care. Blood pressure changes continually throughout the day. Patients are instructed to measure their blood pressure soon after they arise, but this value may be considerably lower than the other values obtained during the day. Caffeine can increase catecholamine concentrations, and blood pressure may be higher for up to 1 hour after consuming coffee or other products high in caffeine (Powers, 1996, p.140). Modest weight loss, even if the person is still overweight, can improve control of diabetes and reduce the risk for heart disease by lowering blood pressure and blood cholesterol (Whitney & Rolfes, 2002, p.281).

2.11.4 Food and activity records

When combined with self-monitoring of blood glucose, food and activity records kept by the patient can provide valuable information on how meals, food choices and exercise affect the concentration of glucose. They are useful in identifying problem eating times, problem foods, and situations that trigger overeating in the obese (Powers, 1996, p.145).

A food record combined with a validated quantitative food frequency questionnaire (Vorster *et al.*, 1985) (Appendix 3), can be used to determine usual eating habits of each patient. With a food record the amount of food and beverages consumed are assessed by volume, that is, they are described in terms of cups, teaspoons, or other commonly used household measures, dimensions or units (Rutishauser & Black, 2002, p.233). When using a quantified food frequency questionnaire, the interviewer has a structured questionnaire, and the respondent recalls how often and in what quantities the foods listed were eaten over a specific period of time; for example, the last

week or month. This is a long interview which needs an experienced interviewer and can overestimate intake (Joubert, 1999, p.250). It is however a low burden on the respondent and usual patterns can be determined. This method can be used for large study populations (Joubert, 1999, p.249). The quantified food frequency questionnaire gives a better indication of usual food intake than the 24-hour recall. The researcher needs insight into the eating habits of the research population, to ensure that often-used foods are included in the questionnaire (Joubert, 1999, p.250).

2.12 COMPLICATIONS

The complications of diabetes mellitus can be illustrated as indicated in figure 1 (Powers, 2001, p.2117):

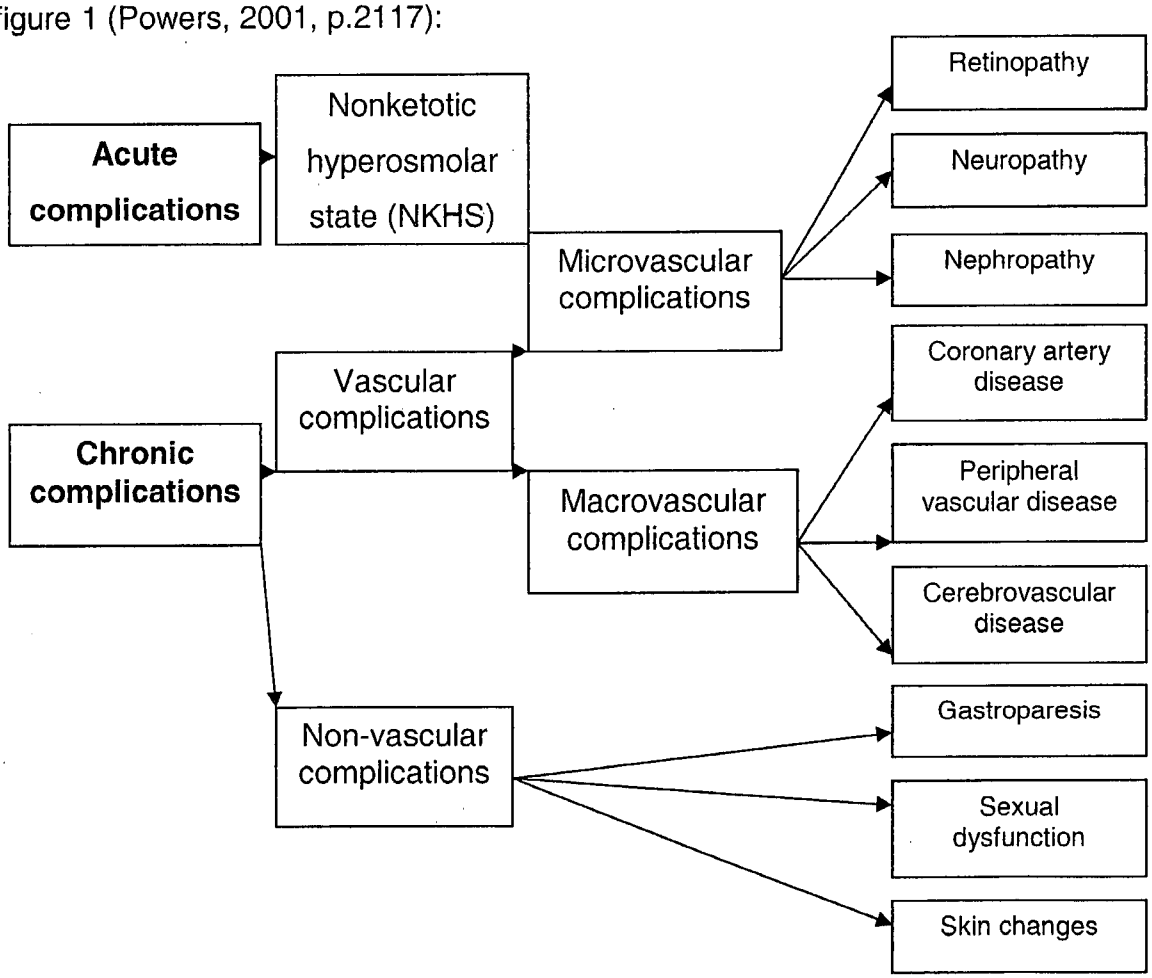


Figure 1: Complications of diabetes mellitus

2.12.1 Acute Complications

Diabetic ketoacidosis (DKA) and nonketotic hyperosmolar state (NKHS) are acute complications of diabetes. DKA is seen primarily in individuals with type 1 diabetes mellitus, and NKHS is seen in individuals with type 2 diabetes mellitus. Both disorders are associated with absolute or relative insulin deficiency, volume depletion, and altered mental status (Powers, 2001, p.2117).

2.12.1.1 Nonketotic hyperosmolar state

NKHS is most commonly seen in elderly individuals with type 2 diabetes mellitus. Its most prominent features include polyuria, orthostatic hypotension, and a variety of neurologic symptoms that include altered mental status, lethargy, obtundation, seizure, and possibly coma. The prototypical patient is a mildly diabetic, elderly individual with a several week history of polyuria, weight loss, and diminished oral intake that culminates in mental confusion, lethargy, or coma. Notably absent are symptoms of nausea, vomiting, and abdominal pain characteristic of DKA. NKHS is often precipitated by a serious, concurrent illness such as myocardial infarction or stroke. Sepsis, pneumonia, and other serious infections are frequent precipitants (Powers, 2001, p.2118).

2.12.2 Chronic complications

The chronic complications of diabetes mellitus affect many organ systems and are responsible for most of the morbidity and mortality rates associated with the disease. Chronic complications of diabetes can be divided into the vascular and non-vascular group of complications.

2.12.2.1 Vascular complications

Vascular complications are subdivided into microvascular and macrovascular complications.

(i) Microvascular disease

Microvascular disease includes retinopathy, neuropathy and nephropathy.

- **Retinopathy**

Diabetic eye disease, especially retinopathy, is the most common cause of blindness during the economically active years and, for most patients, is the most frightening complication of the disease. Fortunately, it is easily detected and now often treatable (Williams & Monson, 1994, p.764). Diabetic retinopathy is a leading cause of new blindness among adults. An estimated 5000 new cases of blindness related to diabetes are reported each year in the United States. All patients with type 2 diabetes should have annual eye examinations, including a visual acuity examination, and careful ophthalmoscopic examination with a dilated pupil, starting at the time of diagnosis (Franz, 2000, p.736).

- **Neuropathy**

Diabetic nerve damage involves the somatic sensory system, causing variable sensory and motor deficits, and the autonomic outflow to various organs. Pathological changes affect both the Schwann cells, with segmental peeling and loss of myelin, and the axons themselves (Williams & Monson, 1994, p.768). Chronic hyperglycaemia is also associated with nerve damage. Peripheral neuropathy usually affects the nerves controlling the sensation in

the feet and hands. Autonomic neuropathy affects nerve function controlling various organ systems (Franz, 2000, p.764).

- **Nephropathy**

Diabetic nephropathy is a specific microvascular disease affecting mainly the glomerulus, although tubular lesions also occur. In the United Kingdom, diabetic nephropathy accounts for 25% of patients with end-stage renal failure. Diabetes is a predisposition for urinary tract infections, especially in patients with incomplete bladder emptying, due to autonomic neuropathy (Williams & Monson, 1994, p.767). More that 20% of patients with both types of diabetes have overt nephropathy after 15 to 20 years of diabetes, and this may progress to end-stage renal disease requiring dialysis or renal transplantation. The earliest evidence of nephropathy is the appearance of low, but abnormal urine albumin concentrations (> 30 mg/day) called microalbuminuria. Without intervention, 20% to 40% of patients with type 2 diabetes and with microalbuminuria progress to overt nephropathy, but by 20 years after onset of overt nephropathy, only 20% will have progressed to end-stage renal disease. Angiotensin converting enzyme (ACE) inhibitors can reduce the amount of proteinuria and slow the progression of nephropathy (Franz, 2000, p.764).

(ii) Macrovascular disease

Macrovascular diseases can be divided into coronary artery disease (CHD), pheripheral vascular disease (PVD), and cerebrovascular disease (CVD). CHD, CVD, and PVD are not specific to diabetes, but they generally develop at an earlier age and are the major cause of death in patients with the disease (Franz, 2000, p.762). According to the Centres for Disease Control in the United States, 97% of adults with diabetes have one or more lipid abnormality (Henry, 2001). Macrovascular disease or atherosclerosis accounts for up to

80% of all mortality. About 75% of atherosclerotic diabetic mortality is the consequence of coronary heart disease (CHD); the remaining 25% results from a combination of accelerated cerebral vascular disease, peripheral vascular disease, or both. Most hospitalisations for complications are attributable to macrovascular disease (Raal, 2000).

- **Coronary heart disease**

Cardiovascular effects include postural hypotension and decreased responsiveness to cardiac nerve impulses, leading to painless or silent ischemic heart disease. When compared to non-diabetics, men with diabetes have twice the incidence and diabetic women four times the incidence of CHD. In addition, the cardio protection afforded to non-diabetic women, as compared to men, is lost in women with diabetes. Myocardial infarction and stroke are more extensive in diabetics and more likely to be rapidly fatal than in non-diabetics. Finally, up to 50% of patients with type 2 diabetes have pre-existing CHD at the time of diabetes diagnosis (Raal, 2000).

- **Peripheral vascular disease**

A common consequence of peripheral vascular disease is reduced circulation, particularly to the legs and feet. Other complications are coldness and fatigue in these areas. Wounds and infections can be dangerous because of poor healing due to poor blood supply. As a result gangrene can develop. In addition, there is reduced pain sensation and impairment of the inflammatory response to injury, so that the person with diabetes may not have the usual pain and redness in response to injury (Mahan & Arlin, 1992, p.529).

- **Cerebrovascular disease**

CVD is up to four times more common in people with diabetes than those without, and 50% of diabetic people have evidence of CVD at the time of their diagnosis (Henry, 2001). Patients with type 2 diabetes typically have smaller, denser LDL particles, which increases atherogenicity, even if the total LDL cholesterol concentration is not significantly increased. Raal (2000) confirmed this. Elevated plasma triglyceride and very low-density lipoprotein (VLDL) cholesterol concentrations, and lower HDL cholesterol concentrate ions are more common with type 2 diabetes (Franz, 2000, p.762).

- **Cardiovascular risk factors**

Cardiovascular risk factors include dyslipidaemia, elevated cholesterol, decreased HDL cholesterol, increased LDL cholesterol, increased triglycerides concentrations and hypertension.

- **Dyslipidemia**

An estimated 50% of all diabetics, whether type 1 or type 2, are dyslipidaemic. Both triglyceride and cholesterol abnormalities occur. It is important to recognize that type 2 diabetes is not solely a disorder of carbohydrate metabolism, but that lipid abnormalities are equally prevalent (Raal, 2000). Type 2 diabetes is associated with a two- to fourfold excess risk of coronary heart disease (CHD). Although the degree of glycaemia in diabetic patients is strongly related to the risk of microvascular complications (retinopathy and renal disease), the relation of glycaemia to macrovascular disease in type 2 diabetes is more modest. The finding of increased cardiovascular risk factors before the onset of type 2 diabetes also suggests that aggressive screening for diabetes, combined with improved glycaemic control alone will not be likely to completely eliminate excess risk of CHD in type 2 diabetic patients (ADA, 2003). There are two main types of lipid abnormalities in type 2 diabetes.

Firstly, there are changes in absolute concentrations of lipids and lipoproteins. The most frequently encountered disturbances in circulating lipids and lipoproteins in type 2 diabetes are an increase in serum triglyceride concentrations and a reduction in HDL cholesterol (usually by 15-25%). These changes tend to be more striking in women than in men. There are no consistent changes in LDL cholesterol, but concentrations tend to be mildly elevated. Elevated triglyceride concentrations and low HDL cholesterol concentrations are also seen in the metabolic syndrome prior to the onset of overt diabetes, and this may explain why CHD is often present at the time of diagnosis of type 2 diabetes (Raal, 2000).

Secondly, there are changes in the composition of lipids/lipoproteins. As mentioned earlier, LDL particles tend to be smaller and denser in type 2 diabetes, which may increase their atherogenicity. These small dense particles can penetrate the arterial wall more readily and bind more avidly to proteoglycans; this results in their being trapped within the arterial wall. In addition, chemical modification of lipoproteins, by glycation or oxidation, both of which are increased, may induce endothelial injury and accelerate foam cell formation by macrophages within the arterial wall (Raal, 2000). Diabetics should increase their regular physical activity to meet currently recommended levels, after undergoing an exercise test to assess the level of risk. Tobacco use and diabetes are synergistic risk factors for atherosclerotic disease. Weight loss and increased physical activity will lead to decreased triglyceride and increased HDL cholesterol concentrations, and also to modest lowering of LDL cholesterol concentrations. Diabetic patients who are overweight should be given a prescription for medical nutrition therapy and for increased physical activity. In addition to being at risk for microvascular disease, patients with diabetes are at very high risk of macrovascular disease, particularly CVD. Because diabetic patients without previous myocardial infarction have as high a risk of myocardial infarction as nondiabetic patients with previous myocardial infarction, all diabetic patients should be treated aggressively for the prevention of CVD (Henry, 2001).

Patients should be treated aggressively with diet, exercise, and glucose control. If these measures fail to achieve LDL cholesterol goals, the addition of cholesterol- and triglyceride lowering drugs, such as HMG-CoA reductase inhibitors (pravastin or simvastatin) or fibric acid derivatives (gemfibrozil) is warranted (Franz, 2000, p.763).

- **Cholesterol**

In the large Multiple Risk Factor Intervention Trial (MRFIT), total cholesterol as well as cigarette smoking and blood pressure predicted the development of cardiovascular disease in diabetic and nondiabetic subjects, suggesting that risk factors may be predictive in both groups (ADA, 2003).

- **HDL cholesterol**

The most common pattern of dyslipidaemia in type 2 diabetic patients is elevated triglyceride concentrations and decreased HDL cholesterol concentrations (Henry, 2001; ADA, 2003). This is also indicated in baseline data from the United Kingdom Prospective Diabetes Study (UKPDS) that showed that both decreased HDL and elevated LDL predicted CHD. In observational studies, HDL may be the most consistent predictor of CHD in type 2 diabetes subjects, followed by triglyceride and total cholesterol concentration (ADA, 2003). According to the American Diabetes Association (ADA), the presence of increased triglyceride and decreased HDL concentrations is the best predictor of CVD in patients with type 2 diabetes. Other predictive factors include a history of cigarette smoking and hypertension (Henry, 2001; ADA, 2003).

- **LDL cholesterol**

In diabetic patients, the concentration of LDL cholesterol is usually not significantly different from that seen in nondiabetic individuals. However, patients with type 2 diabetes typically have a preponderance of smaller, denser, oxidized LDL particles, which may increase atherogenicity, even if the absolute concentration of LDL cholesterol is not elevated (Henry, 2001; ADA, 2003). Since recommended LDL concentrations are considered to be < 2, 60 mmol/l, and since many diabetic patients have increased triglyceride concentrations, a large proportion of diabetic patients will have elevated concentrations of both LDL cholesterol and triglycerides. Aggressive therapy of diabetic dyslipidaemia will probably reduce the risk of CHD in patients with diabetes. Primary therapy should be directed first at lowering LDL concentrations. The goal is to reduce LDL concentrations to concentrations recommended for patients with pre-existing CHD (2,60 mmol/l). The initiation level for behavioral interventions is also an LDL cholesterol of > 2,60 mmol/l. The initial therapy should be to use statin therapy with the addition of a resin if necessary to reach the LDL goal. However, limited data are available from clinical trials, especially in diabetic patients without clinical cardiovascular disease. In the absence of such data, due to the high mortality rate for diabetic patients with first myocardial infarction, aggressive treatment of dyslipidemia is also indicated.

For patients without previous CHD, the goal for LDL cholesterol is 2,60 mmol/l; the initiation level for pharmacological therapy is set at an LDL concentration of 3,35 mmol/l. However, for patients with LDL concentrations between 2,60 and 3,35 mmol/l, a variety of treatment strategies are available, including more aggressive medical nutrition therapy (MNT) and pharmacological treatment with a statin. MNT should be attempted before starting pharmacological therapy. In addition, if the HDL is < 1,04 mg/dl, a fibric acid such as fenofibrate might be used in patients with LDL cholesterol between 2,60 and 3,35 mmol/l (ADA, 2003).

v) Triglycerides

According to the Centres for Disease Control in the United States, 97% of adults with diabetes have one or more lipid abnormalities. The central characteristic of dyslipidaemia in patients with type 2 diabetes is an elevated triglyceride concentration, particularly triglyceride-rich VLDL concentrations and decreased HDL cholesterol concentrations (Henry, 2001). The most common pattern of dyslipidaemia in type 2 diabetic patients is elevated triglyceride concentrations and decreased HDL cholesterol concentrations (Henry, 2001; ADA, 2003). According to the American Diabetes Association (ADA), the presence of increased triglyceride and decreased HDL concentrations is the best predictor of CVD in patients with type 2 diabetes. Other predictive factors include a history of cigarette smoking and hypertension (Henry, 2001; ADA, 2003). The use of alcohol and estrogen may also contribute to hypertriglyceridemia. The initial therapy for hypertriglyceridemia is behavioural modification with weight loss, increased physical activity, and moderation of alcohol consumption. In the case of severe hypertriglyceridemia (11,3 mmol/l), severe dietary fat restriction (<10% of energy; in addition to pharmacological therapy) is necessary to reduce the risk of pancreatitis (ADA, 2003). Lastly, as shown in the technical review, the median triglyceride concentration in type 2 diabetic patients is < 2,30 mmol/l, and 85–95% of patients have triglyceride concentrations below 4,5 mmol/l (ADA, 2003). In agreement with the earlier ADA consensus panel, increased triglyceride concentrations are recognized as a target for intervention. Since recommended LDL concentrations are considered to be < 2,60 mmol/l, and since many diabetic patients have increased triglyceride concentrations, a large proportion of diabetic patients will have elevated concentrations of both LDL cholesterol and triglycerides. The SEMDSA (2003) recommendation for serum triglycerides concentrations are < 1,5 mmol/l.

The initial therapy for hypertriglyceridemia is improved glycaemic control. Additional triglyceride lowering can be achieved with very high dose statins (for subjects with both high LDL and triglyceride concentrations) or fibric acid derivatives (gemfibrozil or fenofibrate) (ADA, 2003).

- **Hypertension**

In the sub-study of the United Kingdom Prospective Diabetes Study concerning the occurrence of hypertension in diabetes, the prevalence of hypertension (systolic pressure ≥ 160 mm Hg) and/or diastolic blood pressure ≥ 90 mm Hg was 39%. If one accepts the Joint National Committee VI recommendation that the treatment of hypertension at high-normal blood pressure levels is justified in a person with diabetes, at least 50% of patients with type 2 diabetes require antihypertension therapy (Yki-Järvinen, 2000). Treatment of hypertension in persons with diabetes should also be vigorous to reduce the risk of macrovascular and microvascular disease. The goal for blood pressure is less than 130/85 mm Hg. Sodium restriction, weight loss and restricted alcohol intake is effective in reducing hypertension (Franz, 2000, p.763).

(iii) Non-vascular complications

Non-vascular complications are gastroparesis, sexual dysfunction and skin changes.

- **Gastroparesis**

Gastroparesis affects about 25% of the type 2 diabetic population and is one of the most frustrating problems that dieticians and patients experience. It results in delayed or irregular contractions of the stomach, leading to various GI symptoms, such as feelings of fullness, bloating, nausea, vomiting,

diarrhoea, or constipation. It can cause detrimental effects on the blood glucose control. Minimizing abdominal stress should be the first option. Small, frequent meals may be better tolerated than three full meals per day. These meals should be low in fibre and fat. If solid foods are not well-tolerated, liquid meals should be recommended. Damage to nerves innervating the gastrointestinal (GI) tract can cause a variety of problems. Neuropathy can be manifested in the oesophagus as nausea and oesophagitis, in the stomach as unpredictable emptying, in the small bowel as loss of nutrients, and in the large bowel as diarrhoea or constipation (Franz, 2000, p.764).

- **Sexual dysfunction**

Sexual function may be affected; with impotence being the most common manifestation (Franz, 2000, p.764).

- **Skin changes**

The most common skin manifestations of diabetes mellitus are protracted wound healing and skin ulcerations. Diabetic dermopathy, sometimes termed pigmented pretibial papules or "diabetic skin spots", begins as an erythematous area and evolves into an area of circular hyperpigmentation. These lesions result from minor mechanical trauma in the pretibial region and are more common in elderly men with diabetes mellitus. Bullous diseases are also seen. Generalized or localized granuloma annulare and scleroderma are more prevalent in the diabetic population than in the general population. Xerosis and pruritus are very common and are relieved by skin moisturizers (Powers, 2001, p.2127).

2.13 SUMMARY

Nutrition recommendations for healthy lifestyles for the general public are also appropriate for individuals with type 2 diabetes. As most type 2 diabetes patients are overweight and resistant to insulin, medical nutrition therapy should emphasize lifestyle strategies that result in reduced energy intake, usually through reducing the fat content of the diet, and increased energy expenditure through exercise. Most patients with diabetes also have dislipidaemia and hypertension, making reductions in dietary intake of saturated fat, cholesterol, and sodium desirable. Therefore, the emphasis of nutrition therapies for type 2 diabetes is on lifestyle strategies to reduce glycaemia, dyslipidaemia, and blood pressure. These strategies should be implemented as soon as the diagnosis of diabetes is made (Franz *et al.*, 2002). A diagnosis of diabetes is no longer a life sentence of eating bland foods. Subjects can enjoy the foods they love if they understand how to fit them into a healthy meal plan. Diabetes will change as the subjects gets older and go through different life stages. They may need to adjust their eating habits from time to time. Diabetics should also be advised to keep their follow-up appointments with the dietician (Lank, 2000).

3.1 INTRODUCTION

This chapter describes the methodology and techniques used for the execution of the study. Study procedures, as well as methods used in statistical analysis of the study are included. Furthermore, this chapter delimits the context and presents the ethical approval for the performance of this study.

3.2 DEFINITION OF VARIABLES

Discussion of the different variables measured for the study as well as the techniques used to measure these variables, follows in greater detail. For the purposes of this study, the following were defined:

3.2.1 Indepenent variables

- A balanced diabetic diet refers to a diet with a total daily energy intake consisting of 50-60% carbohydrates, 10-20% protein and 30% fat. Each subject's diet was calculated individually according to his/her weight and activity level.
- Sugar inclusive diet refers to a balanced diabetic diet with 15% of the total energy added as sucrose (added to cereals, vegetables, tea or coffee).
- Sugar free diet refers to a balanced diabetic diet without added sucrose.
- Weight maintenance refers to maintenance of current weight during the 12 week stabilization period and 4 week trial of the study. The rationale

for weight loss, even in the overweight person, was that this may improve glycemic control (Whitney & Rolfes, 2002, p.281).

- Body mass index (BMI) refers to the relationship of weight to height, thus eliminating dependence on frame size. BMI is calculated as kg/m^2 (Quetelet index) (Laquatra, 2000, p493).

  Weight classifications based on BMI are as follows (Laquatra, 2000, p.493):

  BMI < 18.5 kg/m^2 = underweight

  BMI 18.5 to 24.9 kg/m^2 = healthy

  BMI 25.0 to 29.9 kg/m^2= overweight

  BMI 30.0 to 34.9 kg/m^2= obesity, class I

  BMI 35.0 to 39.9 kg/m^2 = obesity, class II

  BMI ≥ 40 kg/m^2 = extreme obesity, class III

- Study period refers to the 16 week study period starting at recruitment to the end of the study.
- Trial period refers to the 4 week trial period after randomization, during which dietary intervention took place.

3.2.2 Dependent variables

- Glycaemic control refers to optimal glucose concentrations in the blood in the fasting state. According to guidelines by The Society for Endocrinology, Metabolism and Diabetes of South Africa (SEMDSA), cut-off points for optimal fasting glucose control is between 4 - 6 mmol/l for nonpregnant adult diabetic subjects. Acceptable glycaemic control is between 6 - 8 mmol/l and additional action is suggested if the blood glucose concentration is > 8 mmol/l (SEMDSA, 2003).

- Total serum cholesterol refers to total serum cholesterol concentration. Optimum total serum cholesterol concentrations are < 5 mmol/l (SEMDSA, 2003).
- Serum HDL cholesterol refers to serum HDL cholesterol concentration. Optimal HDL cholesterol concentrations are a value of > 1.2 mmol/l (SEMDSA, 2003).
- Serum LDL cholesterol refers to serum LDL cholesterol concentration. Optimal serum LDL cholesterol concentrations are values of ≤ 3.0 mmol/l (SEMDSA, 2003).
- Serum triglyceride refers to serum triglyceride concentrations. Optimal serum triglyceride concentrations are < 1.5 mmol/l (SEMDSA, 2003).
- Compliance is the degree to which subjects adhered to their individual prescribed diets and was determined by serum fructosamine concentrations.
- Short term diabetic control refers to serum fructosamine concentrations of 205-285 µmol/l (Department of Chemical Pathology, University of the Free State), an indicator of subject's compliance over the preceding 21 days (Fructosamine, 2000).
- Glycated haemoglobin (HbA_{1c}) refers to long term diabetic control which provides an index of the average plasma glucose concentration over the preceding two or three months. Optimal HbA_{1C} values are < 7% (SEMDSA, 2003).

3.3 STUDY DESIGN

The study was a randomized controlled, single centre clinical trail.

3.4 SAMPLE

According to Leedy (1989, p.151) population parameter and sampling procedures are of paramount importance in the success of research. The size

and selection of the study population is determined by the type of the study, and according to the aims of the study. There are a number of factors that must be considered before a subject can participate in a study. These factors are referred to as the inclusion and exclusion criteria. The selection, inclusion criteria, exclusion criteria and size of the study population is discussed hereafter.

3.4.1 Sample size

Large samples used for intensive studies are time consuming and expensive, thus smaller samples are recommended for intensive studies (Leedy 1989, p159). In previous studies where the effects of sugar intake in glycaemic control were determined, 9-18 subjects were included (Bantle et al., 1983; Coulston et al., 1985; Peterson et al., 1986, Bantle et al., 1986; Abraira & Derler, 1988; Colagiuri et al., 1989; Bantle et al., 1993; Malerbi et al., 1996; Rickard et al., 1998). In order to obtain more reliable results, this study aimed at including more subjects to enable sub-group analyses, since randomisation is more effective in larger groups. As this was a randomized controlled study, the two groups had to be large enough to ensure statistically significant results. Originally, it was decided to recruit sixty subjects with type 2 diabetes mellitus, each group consisting of 30 subjects.

3.4.2 Sample selection

As subjects included in the study had to be available for the four month duration of the study and only type 2 diabetics were included, it was impossible to select a sample from the general population by means of probability sampling. Therefore, the study sample selected was deliberate and voluntary. The sample was selected in Bloemfontein from the diabetic clinics at Universitas and Pelonomi Hospitals, referrals from the local primary

health care clinics, private practitioners, private hospitals, as well as through the local media (Appendix 1).

3.4.3 Inclusion criteria

The sample included subjects who:

- had type 2 diabetes mellitus as determined by a GAD 65 and C-peptide value (see Appendix 4);
- volunteered to comply with a prescribed diet for the 16 week duration of the study;
- were willing and able to attend monitoring sessions fortnightly;
- were in the age range of 40 to 65 years.

3.4.4 Exclusion criteria

Subjects were excluded from the study if they:

- were non-diabetic;
- had type 1 diabetes mellitus;
- were pregnant;
- wanted to lose weight;
- had a chronic disease that required special dietary interventions, as this would deviate from the dietary prescription for the study;
- used medication that influenced glucose tolerance.

3.5 STUDY PROCEDURE

Figure 2 (p. 95) is a flow chart of the study procedure.

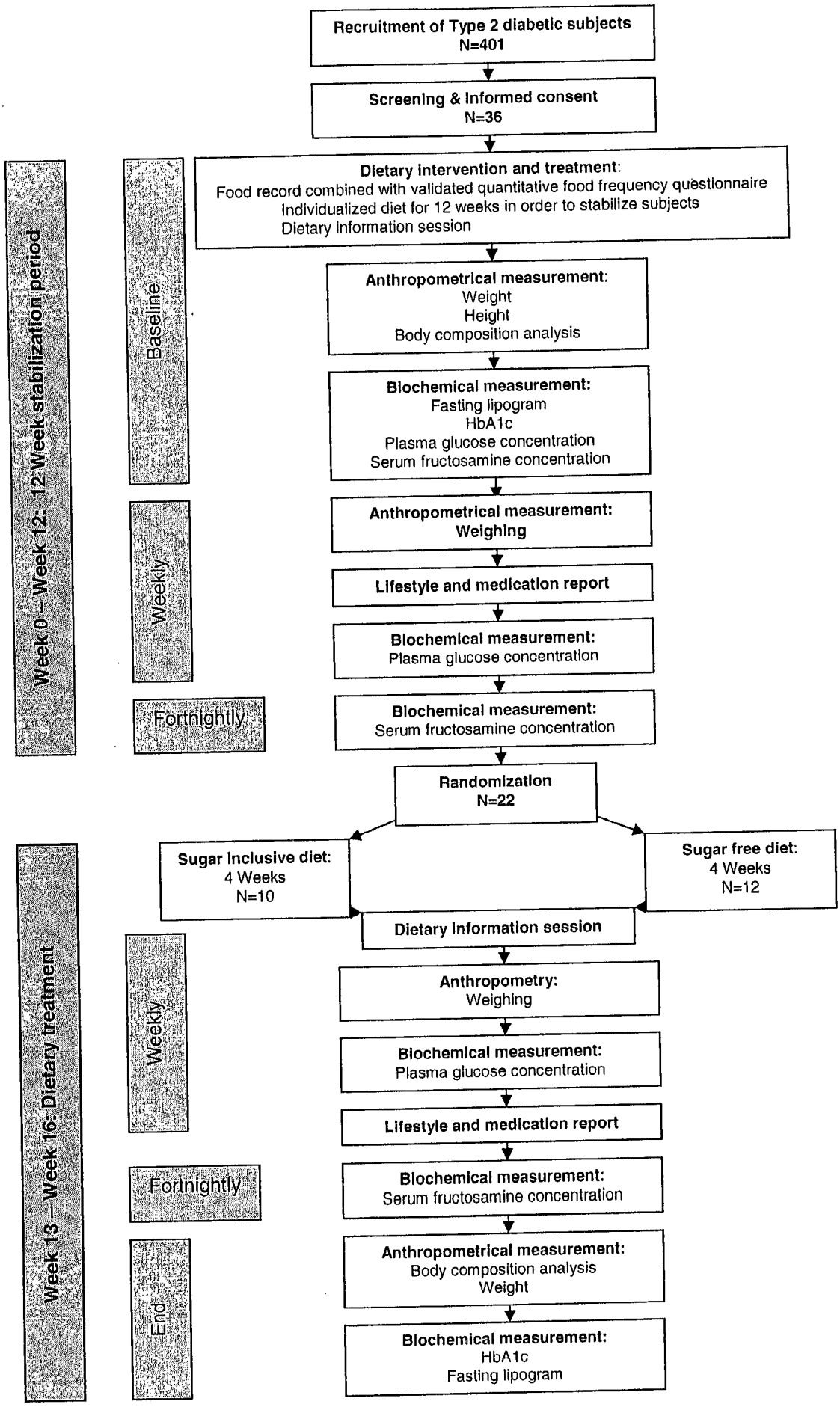


Figure 2: Flow chart of the study procedure

3.5.1 Recruiting, screening and informed consent

Volunteers were recruited by advertising in the local newspapers (Ons Stad and Die Volksblad), radio bulletins (Radio Rosestad), visits to hospitals, physicians, general practitioners, private practicing dieticians, diabetic clinics, the National Defence Force and screening days at shopping centres. After recruitment volunteers phoned the researcher (registered dietician) who screened them telephonically. The volunteers indicated their weight, height, age and medication used. In cases where subjects were referred by a general practitioner, dietician or registered nurse, the researcher phoned the subjects to do the screening. In both cases each subject's details were recorded on a screening form (Appendix 4) by the researcher. The following clinical criteria were used to confirm the presence of type 2 diabetes mellitus:

- Subjects had to be between 40 - 65 years. The onset of type 2 diabetes mellitus is usually between these ages (Franz, 2000, p.753).
- Subjects had to have a BMI within the reference range of above 18,5 kg/m^2. This range covers normal weight, overweight and obese subjects. Approximately 80% of type 2 diabetics are obese or have a history of obesity at the time of diagnosis, but type 2 diabetes can occur in nonobese individuals as well (Franz, 2000, p.754). A reference range of above 18,5 kg/m^2 was chosen as a criterion for this study, in order to represent a common sample population of type 2 diabetic subjects.
- The presence of ketones in a urine test will be an indicator of type 1 diabetes mellitus. No ketones had to be present in the subjects' urine. Fresh urine samples were obtained from all subjects and mixed thoroughly at room temperature. The required waiting period of 60 seconds was adhered to after the test had been briefly dipped into the urine, before comparison of the reaction colours of the test area to the colours on the label was made (Combur[10] Test, 2001). Urine ketones

have a negative reference range in type 2 diabetes mellitus. This test was necessary to exclude type 1 diabetes mellitus.

- Subjects were required to have C-peptide values above 265pmol/l. Assessment of C-peptide immunoreactivity will provide sound evaluation of beta cell function and, often eliminate the effect from endogenous insulin antibodies. C-peptide values below 265pmol/l indicates type 1 diabetes mellitus (C-peptide of Insulin, 2000).
- Negative GAD 65 antibody values (reference range: 0 – 1 U/ml) were required from subjects. The result is positive in type 1 diabetes mellitus (GAD-AB, 2003).

The researcher personally interviewed subjects who met all the inclusion criteria. During this interview, the aims of the study and all procedural details were explained to each subject individually at his/her home. Each subject had to sign a form where he/she gave informed consent (Appendix 2).

3.5.2 Randomization

Subjects were stabilized for a 12 week period, as recommended by Prof. Mollentze, Head of the Department of Internal Medicine, and endocrinologist at the Faculty of Medicine, University of the Free State. After the 12 week stabilization period, subjects were randomized into two groups, stratified by the type of control, namely, oral medication and diet alone. There was, thus, a separate computer-generated randomization list for each of these two strata, randomizing the subjects into a study and control group. The one group followed the sugar inclusive diet and the other followed the sugar free diet. Both groups sustained their particular diets for four weeks.

3.5.3 Dietary intervention and treatment

The researcher conducted individual interviews with all subjects who were screened, or who gave informed consent to participate in the study. At baseline, a food record and validated quantitative food frequency questionnaire (Appendix 3) was filled in by the researcher. The researcher calculated individual diets and explained each subject's diet to him/her individually. These contact sessions with the subjects took place at their homes. All subjects were instructed to follow a balanced diabetic diet for 12 weeks. After the 12 week period, subjects were randomized into a sugar inclusive diet and a sugar free diet for four weeks. At fortnightly contact sessions, the researcher motivated subjects to comply with their diet. The researcher gave a short informative talk at this contact session to motivate and encourage subjects to adhere to their diets and to gain insight into dietary aspects of type 2 diabetes mellitus.

The topics included were (Appendix 7):

- What is diabetes?
- Symptoms of hypo- and hyperglycaemia
- Meal distribution, carbohydrates, and fibre in the diabetic diet
- Fat in the diabetic diet
- Hints on cooking and food choices when eating away from home or in restaurants
- Alcohol intake
- Travel and diabetes
- Exercise

Increased activity levels improves glucose tolerance (Krummel, 2000, p.522). Subjects were encouraged to increase their activity levels as part of a healthy lifestyle.

3.5.4 Anthropometry

At baseline the following anthropometrical measurements were measured: weight, height, BMI and body-fat percentage (Appendix 5). The researcher used standardized methods and procedures at the subject's home. Weight was measured weekly thereafter by the nurse for the 12 week stabilization and four week trial of the study. At the end of the study the researcher measured each subject's body-fat percentage.

3.5.5 Biochemical measurements

A registered nurse collected fasting blood samples at each subject's home on a weekly basis. Blood samples were taken to the laboratory in closed test tubes in a vertical, stopper-up position on crushed ice in a cooler bag immediately after every collection. At baseline blood samples were analysed for: fasting lipogram, HbA_{1C}, plasma glucose concentration, and serum fructosamine concentration (see Appendix 5). The venous plasma glucose concentration of the subjects was measured weekly thereafter. Serum fructosamine was measured on a fortnightly basis thereafter. During the four week period after randomization, the subject's plasma glucose concentration was monitored weekly, while serum fructosamine was taken fortnightly by the nurse. At the end of the study, a fasting blood sample was drawn and analysed for lipogram and HbA_{1c}.

3.5.6 Lifestyle and medication

At baseline, and for the duration of the study, the registered nurse filled in a report regarding lifestyle and medication changes. This report included questions on weight changes, exercise, medication, smoking and alcohol concumption (Appendix 6).

3.6. SELECTION AND STANDARDIZATION OF APPARATUS AND TECHNIQUES

3.6.1 Apparatus

All measurements taken were according to standardized methods and techniques with standardized equipment.

3.6.1.1 Scale

The weight of each subjects was determined with a Seca Alpha digital electronic scale. The same scale was utilized throughout the study and was calibrated at regular intervals.

3.6.1.2 Stadiometer

Height was measured with a stadiometer.

3.6.1.3 Bodystat®

The Bodystat® 1500 unit (Bodystat® Limited, 1994) was used to determine body composition. Body-fat percentage was the only measurement used from this analysis. Using the scientifically validated principle of Bioelectrical Impedance Analysis, a complete analysis of body composition is displayed instantly on the liquid crystal display (LCD) screen of the hand-held unit, comprising fat and lean weight and body water levels. The unit has been precision electronically engineered to the highest quality standards offering

the user a safe and efficient means of measurement. It is able to calibrate itself prior to each measurement (Bodystat® Limited, 1994, p.1).

3.6.2 Methods and techniques

Anthropometrical measurements were done according to standardized methods and techniques by the researcher and the registered nurse (ISAK, 2001, pp.7-8, 54-55). The procedures were as follows:

3.6.2.1 Weight

The scale was placed on a hard surface to ensure an accurate reading. The scale reading was zero before subjects stood on the centre of the scale without support and with the weight distributed evenly on both feet. Subjects were barefoot and wore light clothing (ISAK, 2001, p.53). Weight was recorded in kilograms.

3.6.2.2 Height

The subject stood with the feet together and the heels, buttocks and upper part of the back touching the scale. The head, when placed in the Frankfort plane, did not need to touch the scale. Height was recorded in meters at the end of a deep inward breath (ISAK, 2001, p.55).

3.6.2.3 Body-fat percentage

Skinfold thickness measurement is the preferred method of assessing the amount of body-fat an individual has. Because of the practical use in clinical settings, validity depends on accuracy of the measuring technique and repetition of measurements over time. Accuracy decreases with increasing

obesity (Hammond, 2000, p.371). It was accepted that most of the study population would be overweight (80% of all type 2 diabetics are overweight). It was thus decided to use the Bodystat® 1500, because it is available, quick and user-friendly. For accurate and reproducible results of repeated tests, it is important that the subject is as normally hydrated as possible.

Subjects were instructed to do the following prior to the body composition determination:

- No eating or drinking 4 to 5 hours prior to the test
- No exercise 12 hours prior to the test
- No alcohol or caffeine consumption 24 hours prior to the test

Variations in body composition are likely to occur in some subjects using prescribed medications, like diuretics, and other conditions causing water retention or water loss. The above precautions are not specific to Bodystat®, but apply to all methods of body composition analysis. Prior to the measurement, the subject's height and weight was determined accurately. The subject removed the right shoe and sock or stocking, and lay flat on his/her back with the arms and legs spread slightly. The electrodes were placed after the subject was in a comfortable, relaxed position. The self-adhesive disposable electrodes were attached to the right hand and right foot. The Bodystat® 1500 has two main leads, which each divide at the ends into a red and black lead. The red leads were connected just behind the knuckle of the middle finger and behind the second toe next to the big toe. The black leads were then connected on the wrist next to the ulnar head and on the ankle at the level of and between the medial and lateral malleoli. The Bodystat® 1500 unit was then switched on and the following data were entered by the researcher:

- Subject number
- Gender
- Age (yrs)
- Height (cm)

- Weight (kg)
- Activity (Very low, Low/medium, Medium, Medium/High, Very high)

After the electrodes were connected, the results were displayed as selected by the researcher, after successful measurement and processing of the subject data (Bodystat® Limited, 1994, pp.13-21).

3.6.2.4 Dietary intakes

A food record, combined with a validated quantitative food frequency questionnaire (Vorster et al., 1985) (Appendix 3), was used to determine usual eating habits of each subject in this study. With a food record, the amount of food and beverages consumed are assessed by volume, that is, they are described in terms of cups, teaspoons, or other commonly used household measures, dimensions or units (Rutishauser & Black, 2002, p.233). When using a quantified food frequency questionnaire, the interviewer uses a structured questionnaire, and the respondent recalls how often and in what quantities the foods listed were eaten over a specific period of time; for example, the last week or month. This is a long interview which needs an experienced interviewer and can overestimate intake (Joubert, 1999, p.250). It is, however a low burden on the respondent and usual patterns can be determined (Joubert, 1999, p.249). The quantified food frequency questionnaire gives a better indication of usual food intake than the 24-hour recall. The researcher needs insight into the eating habits of the research population, to ensure that often used foods are included in the questionnaire (Joubert, 1999, p.250). The researcher analyzed individual food intakes to determine nutrient intakes with the Food Fundi® Computer Program (Joubert, 1999, p.251). Daily energy requirements were then calculated to maintain body weight. Each subject received his/her own individualized diet that he/she had to follow for 12 weeks prior to randomization. This was necessary to stabilize the subjects' biochemical and anthropometric measurements. The subjects were then randomized into a sugar inclusive diet or a sugar free diet.

These diets were then followed for four weeks. At contact sessions subjects was questioned about their food intake to check compliance.

3.6.2.5 Biochemical measurements

All blood samples were taken by a registered nurse at the subjects' home. Peripheral venous blood samples were drawn after an overnight fast of 10-12 hours. Standard laboratory techniques, apparatus and standard reference ranges were used by the Department of Chemical Pathology, University of the Free State, for the analyses of all blood samples according to standardized equipment and techniques. As the biochemical techniques applied in the study have been standardized for clinical use, further standardization was considered unnecessary.

a) Glucose

Plasma glucose concentrations were determined by the automated glucose oxidase method on the CX7 analyzer /or LX 20 analyzer (Glucose, 1998) with the reference range for fasting glucose: 4-6 mmol/l, recommended by SEMDSA (SEMDSA, 2003). Glucose Reagent, in conjunction with the SYNCHRON® Systems Multi Calibrator, determines the quantitative glucose concentration in serum, plasma, urine or cerebrospinal fluid on SYNCHRON LX systems. Glucose measurements are necessary for the diagnosis and treatment of carbohydrate metabolism disordersl including diabetes mellitus (Glucose, 1998). Glucose reagent measured the glucose concentration by a timed endpoint method. Hexokinase (HK) catalizes the transfer of a phosphate group from adenosine triphosphate (ATP) to glucose, to form adenosine diphosphate (ADP) and glucose-6 phosphate. The glucose-6 phosphate was then oxidized to 6-phosphogluconate, with the concominant reduction of ß-nicotinamide adenine dinucleotide (NAD) to reduced ß-nicotinamide adenine dinucleotide (NADH) by the catalytitc action of glucose-6- phosphate dehydrogenase (G6PDPH) (Glucose, 1998).

Method

The SYNCHORN LX System automatically delivered the appropriate sample and reagent volumes into the cuvette. The ratio used was one part sample to 100 parts reagent. The system monitored the change in absorbance at 340 nanometers. This change in absorbance is directly proportional to the concentration of glucose in the sample, and was used by the SYNCHRON LX System to calculate and express glucose concentration. Tubes of blood were kept closed at all times in a vertical, stopper-up position (Glucose, 1998).

b) Fructosamine

Fructosamine was determined by the automated method done on the CX7 analyzer (Beckman Coulter Inc., 2000) with the reference range 205-285μmol/l, used by the Department of Chemical Pathology, University of the Free State. Fructosamine is a time-averaged indicator of blood glucose concentrations and assesses the glycaemic status of diabetics. The concentration of glycated protein, such as glycohaemoglobin, glycoalbumin or glycated total protein, is valuable in evaluating the glycaemic status of diabetic subjects (Fructosamine, 2000).

Method

This assay is based on the nitrotetrazolium blue method, and yields a precise and easily automatable determination of the non-enzymatic glycation of serum proteins. The turnover of serum protein is less than that of haemoglobin, and therefore fructosamine determinations provide a means of monitoring subjects' blood glucose status over a shorter period (1-3 weeks) than glycohaemoglobin (6-8 weeks). As a result, changes in fructosamine concentrations decrease more quickly than HbA_{1c} when diabetic subjects are brought under better control. Roche/Hitachi systems automatically calculate the fructosamine concentration of each sample (Fructosamine, 2000).

c) Cholesterol

Cholesterol measurements are used in the diagnosis and treatment of atherosclerotic coronary artery disease. Cholesterol measurements are also used in the diagnosis of metabolic disorders involving lipids and lipoproteins. Total serum cholesterol concentrations depend on many factors including age, gender, diet, physical activity, liver disease, and other metabolic disorders (Cholesterol, 2000). The optimal cholesterol reference range used was < 5.0 mmol/l, as recommended by SEMDSA (SEMDSA, 2003).

Method

Cholesterol reagent is used to measure cholesterol concentration by a timed-endpoint method. In the reaction, cholesterol esterase hydrolyzes cholesterol esters to free cholesterol and fatty acids. Free cholesterol is oxidized to cholestene-3-one and hydrogen peroxide by cholesterol oxidase. Peroxidase catalyzes the reaction of hydrogen peroxide with 4-aminoantipyrine and phenol to produce a coloured quinoneimine product. The SYNCHRON LX System automatically proportions the appropriate sample and reagent volumes into the cuvette. The ratio used is one part sample to 100 parts agent. The system monitors the change in absorbance in 520 nanometers. This change in absorbance is directly proportional to the concentration of cholesterol in the sample, and is used by the SYNCHRON LX System to calculate and express cholesterol concentration (Cholesterol, 2000).

d) Triglycerides

Triglyceride measurements are used in the diagnosis and treatment of subjects with diabetes mellitus, liver obstruction, other diseases involving lipid metabolism, or various endocrine disorders (Triglycerides, 2000). The reference range for triglycerides used was < 1.5 mmol/l, as recommended by SEMDSA (SEMDSA, 2003).

Method

Triglyceride GPO reagent is used to measure the triglyceride concentration by a timed endpoint method. Triglycerides in the sample are hydrolyzed to glycerol and free fatty acids by the action of lipase. A sequence of three coupled enzymatic steps using glycerol kinase, glycerophosphate oxidase, and horseradish peroxidase causes the oxidative coupling of 3.5-dichloro-2-hydroxybenzenesulfonic acid with 4-aminoantipyrine to form a red quinoneimine dye. The SYNCHRON LX System automatically delivers the appropriate sample and reagent volumes into the cuvette. The ratio used is one part sample to 100 parts reagent. The system monitors the change in absorbance at 520 nanometers. This change in absorbance is directly proportional to the concentration of triglycerides in the sample, and is used by the SYNCHRON LX System to calculate and express the triglyceride concentration (Triglycerides, 2000).

e) HDL cholesterol

HDL cholesterol is inversely related to the risk of developing coronary heart disease. A low HDL/LDL cholesterol ratio is directly related to the risk of developing coronary artery disease. High HDL cholesterol is associated with the "longevity" syndrome (HDL cholesterol, 2000). HDL's reference range used was > 1.2 mmol/l, as recommended by SEMDSA (SEMDSA, 2003).

Method

This direct HDL cholesterol method is a homogenous assay, without the need for any offline pretreatment or centrifugation steps. The method depends on a unique detergent, which solubilizes only the HDL lipoprotein particles and releases HDL cholesterol to react with cholesterol esterase and cholesterol oxidase in the presence of chromogens, to produce a colour product. The same detergent also inhibits the reaction of the cholesterol enzymes with LDL, VLDL, and chylomicron lipoproteins, by adsorbing to their surfaces. A

polyanion contained in the reagent enhances the selectivity for HDL cholesterol assay by complexing LDL, VLDL, and chylomicron lipoproteins. HDL cholesterol reagent is used to measure the cholesterol concentration by a timed endpoint method. The SYNCHRON LX System automatically proportions the appropriate HDL cholesterol sample and reagent volumes into a cuvette. The ratio used is one part sample to 93 parts reagent. The system monitors the change in absorbance at 560 nanometers. This change is directly proportional to the concentration of cholesterol in the sample, and is used by the SYNCHRON LX System to calculate and express the HDL cholesterol concentration (Cholesterol, 2000).

f) LDL cholesterol

LDL contains 50% cholesterol by weight and is the most cholesterol-rich of the lipoproteins. They are synthesized in the liver and are responsible for transporting cholesterol from the liver to the peripheral tissues. LDL has diameters ranging from 18 to 30 nanometers. LDL size is inversely related to serum triglyceride concentrations. Smaller, denser LDL is associated with a higher risk for coronary heart disease (Warnick et al., 1996, p.318). The reference range used was ≤ 3.0 mmol/l as recommended by SEMDSA (SEMDSA, 2003).

Method

Friedewald's formula is used to calculate LDL cholesterol (Burtis, 2000, pp.843-844).

LDL cholesterol = (HDL + triglyceride/2.18)

g) HbA_{1c}

Measurement of HbA_{1c} is accepted as a method to measure long-term glucose control in subjects with diabetes mellitus. Determination of HbA_{1c} provides an important diagnostic tool for monitoring the efficiency of dietary

control and therapy during the treatment of diabetes mellitus. Long term treatment of the disease emphasizes control of blood glucose concentrations in preventing the acute complications of ketosis and hyperglycaemia (Hemoglobin A_{1c}, 1998). HbA_{1c} was determined by the LX-20 auto-analyser. The optimal value for HbA_{1c} is < 7 %. A range between 7-8% is acceptable and additional action is suggested if the HbA_{1c} was >8% as recommended by SEMDSA (SEMDSA, 2003). The Diabetes Control and Complications Trial Research Group reported the relationship between HbA_{1C} and average blood glucose during the preceding two to three months as 1% ΔHbA_{1c} = 1.6 mmol/l (30 mg/dl) Δ average blood glucose (Glycated Hemoglobin, 1994).

Method

The SYNCHRON LX Systems utilizes two unique cartridges, Hb and A_{1c}, to determine haemoglobin A_{1c} concentration as a percentage of total haemoglobin. Haemoglobin Reagent is used to measure total haemoglobin concentration by a colorimetric method. The SYNCHRON LX System automatically delivers the appropriate sample and reagent volumes into the cuvette. The ratio used is one part sample to 8.6 parts reagent. The system monitors the change in absorbance at 560 nanometers. This change in absorbance is directly proportional to the concentration of total haemoglobin in the sample, and is used by the SYNCHRON LX System to calculate and express total haemoglobin concentration. A_{1c} reagent is used to measure haemoglobin A_{1c} by the turbidimetric immuninhibition method. In the reaction, haemoglobin antibodies combine with HbA_{1c} from the sample to form soluble antigen-antibody complexes. Polyhaptens from the reagent then bind with the excess antibodies and the resulting agglutinated complex is measured turbidimetrically. The SYNCHRON LX System automatically delivers the appropriate sample and reagent volumes into the cuvette. The ratio used is one part sample to 31.6 parts reagent. The system monitors the change in absorbance at 340 nanometers. This change in absorbance is inversely proportional to the concentration of HbA_{1c} in the sample, and is used by the SYNCHRON LX System to calculate and express HbA_{1c} concentration as a

percentage of total haemoglobin by the following formula: % HbA_{1c} = A_{1c} (g/dl) / Hb(g/dl) x 100 (Hemoglobin A_{1c}, 1998).

h) GAD 65

GAD-AB is a radioimmunoassay (RIA) kit for the measurement of Glutamic Acid Decarboxilase (GAD) autoantibodies in human serum. Type 2 diabetes is a heterogenous and multiform disease, due to peripheral resistance of insulin. Some subjects with type 2 diabetes gradually develop insulin deficiency, a condition referred to as slowly progressive type 1 diabetes (especially in non obese subjects). The beta cell destruction seen in this case is thought to be induced by pancreatic autoimmunity. The presence of autoantibodies to glutamic acid decarboxylase (GAD-AB) may be useful to distinguish subjects with type 1 versus type 2 diabetes in adults. The reference range is between 0 - 1 U/ml. The result is positive in type 1 diabetes mellitus and LADA (late onset auto-immune diabetes mellitus of the adult). GAD exists in 2 major isoforms: 65 and 67 kD. It is established that antibodies against the GAD65 kD form are more specific in the above purposes. This procedure specifically measures GAD65 antibodies (GAD-AB, 2003).

Method

In the GAD auto-antibody assay, test serum samples are incubated, first with I-labelled human GAD. This is followed by the addition of solid phase protein A to precipitate the labelled GAD antibody complexes. After centrifugation, the precipitates are counted for 1 minute and the amount of radioactivity in the precipitate as proportional to the concentration of GAD antibody in the test sample (GAD-AB, 2003).

i) C-peptide

Human insulin and C-peptide originate as a single polypeptide chain known as pro-insulin, which is formed on the surface of the rough endoplasmic reticulum in the beta cells of the Islets of Langerhans. Pro-insulin is then transported to

the Golgi complex of the beta cells, where it is packed into granules. It is in these granules that pro-insulin is cleaved proteolytically into insulin and C-peptide. Insulin and C-peptide are stored in these beta cell granules until their secretion is stimulated, at which time approximately, equimolar amounts of each are released into the portal vein. Because of differences in uptake in the liver and in clearance times of these peptides, peripheral levels of C-peptide are higher than insulin. While insulin has a pervasive influence on the body, affecting virtually every organ and biochemical component, C-peptide has no known physiologic function, other than possibly facilitating correct insulin conformation. Insulin and C-peptide do not share basic antigenic components and, therefore, antisera directed against insulin will not cross-react with C-peptide. It is a lack of immunoreactivity that makes C-peptide measurement quite valuable. C-peptide and insulin levels correlate strongly in most sera and assessment of C-peptide immunoreactivity will provide sound evaluation of beta cell function, and often eliminate the effect from endogenous insulin antibodies. This test quantitatively analyzes human connecting peptide (C-peptide) of insulin by radioimmunoassay (RIA) in serum. C-peptide was detected with the disequilibrium assay in which sample and antibody are added and incubated for 16-24 hours at 2-8°C. Tracer is then added, followed by a second incubation for 16-24 hours at 2-8°C. A pre-precipitated second anti-body is added in a single step to separate bound from free antigen (C-peptide of Insulin, 2000). The reference range used by the Department of Chemical Pathology, University of the Free State for 12 hour fasting was 265 - 1324 pmol/l.

Method

Lyophilized reagents were reconstituted and any frozen reagents were allowed to thaw completely. Reagents did not reach temperatures above 25°C and were mixed gently before using. Disposable tubes were set up, labelled 12 x 75 mm in duplicate, according to the supplied protocol.

Reagents were added as follows:

a. Total count tubes were set aside until step 5
b. 0 standard - 100μL of 0 standard; 200μL of C-peptide of insulin Antiserum (green)
c. C-peptide of insulin standards - 100μL of C-peptide of insulin standard; 200μL of C-peptide of insulin Antiserum (green)
d. Control and unknown samples - 100μL of either serum or urine; 200μL of C-peptide of insulin Antiserum

Tubes were vortexed gently and incubated for 16-24 hours at 2-8°C. 200μL of 125I C-peptide of insulin were added to all tubes and mixed gently without foaming. Tubes were incubated for 16-24 hours at 2-8°C. The GAR-PPT was vigourously mixed; then 500μL were added to all the tubes, except the total count tubes. Tubes were vortexed without foaming and incubated for 15-25 minutes at 20-25°C. Tubes were centrifuged for 20 minutes at 760 x g(1118×10^{-8}) (radius in cm) at 20-25°C. The supernatant were immediately decanted from all the tubes, except the total count tubes, for a sufficient time to achieve statistical accuracy (C-peptide of Insulin, 2000).

j) Urinary ketones

Fresh urine samples were mixed thoroughly at room temperature. They did not stand for more than two hours. After the test strip had been dipped into the urine for one second, it was required to wait 60 seconds and then compare the reaction colours of the test area to the colours on the label. This test is based on the principle of Legal's test and is more sensitive to acetoacetic acid than to acetone. Phenylketones and phthalein compounds produce red colours on the test area. These are, however, quite different from the violet colours produced by ketone bodies. (Combur[10]Test, 2001). Urine ketones have a negative reference range in type 2 diabetes mellitus. This test was necessary to exclude type 1 diabetes mellitus.

3.7 STATISTICAL ANALYSIS

Statistical analyses were done by the Department of Biostatistics, University of the Free State. Results were summarized by group, using means and standard deviations, percentiles or frequencies and percentage, as appropriate. Two way analysis of variance was used to compare changes between groups, and 95% confidence intervals (95% CI) were calculated.

3.8 PROBLEMS ENCOUNTERED DURING THE EXECUTION OF THE STUDY

3.8.1 Subject recruitment

The researcher recruited a total number of 401subjects. In spite of all these recruitment efforts, only a total of 22 subjects completed the study, which means that 0.05 percent of the initial recruited subjects took part and completed the study. Subjects previously diagnosed with type 2 diabetes were included in the study if their HbA1c were ≤ 8% (SEMDSA, 2003), because there were so few newly diagnosed subjects. In order to recruit subjects for the study, the researcher visited nine physicians, 24 general practitioners, seven registered nurses who specializes in diabetic care, and 17 dieticians in Bloemfontein. The researcher contacted the local municipality with the aim of referring subjects from the surrounding municipal clinics. Fifteen pharmacies in Bloemfontein were approached by the researcher with the aim of possible referrals. The researcher communicated with the pharmacist or nurses in charge of the diabetic clinics at each pharmacy. Special permission was obtained from Lt. Colonel Kruger, from the National Defence Force, to use subjects from their clinics. In the latter recruitment effort only one subject was referred. and she did not wish to participate. Two general practitioners referred one subject each. After screening, both turned out to be non diabetic. One of these subjects showed undetected heart failure, so the researcher

referred the subject to a physician. According to SEMDSA guidelines for the oral glucose tolerance test, the other subject had glucose intolerance. Two private practising dieticians in Bloemfontein referred 23 subjects to the researcher. Only two subjects qualified for the study. The manager of Farmovs Parexel Clinic was approached to obtain lists of diabetics that successfully completed projects. A registered nurse from Farmovs Parexel referred 15 possible subjects. Four subjects out of the 15 screened were willing to participate. One registered nurse recruited seven subjects who complied with the inclusion criteria. Three subjects approached the researcher via word of mouth.

The researcher advertised in the local media and received many subjects who were interested in the study. An article on diabetes mellitus was published in the local Afrikaans daily newspaper (Volksblad). Furthermore, a local radio station, Radio Rosestad, broadcasted this article on diabetes mellitus, and the researcher received 40 phone calls from interested subjects. Only five subjects of those who approached the researcher after media advertisements could be included in the sample after screening. A screening day at Mimosa Mall, a local shopping mall, was planned to recruit possible subjects for the study. A letter of intent was written to the management, but the researcher did not receive any response. Permission to have a screening session at the Art-in-the-Park Market was declined by the authorities. Permission was obtained to recruit patients at Rosepark Hospital. The hospital manager referred the researcher to the hospital's practising dietician and nurse for diabetic education. The dietician made her office available for screening and follow-up of recruited subjects by the researcher. However, no subjects were referred. A diabetes awareness and screening day was initiated at The Pick and Pay, Fichardt Park, in Bloemfontein by the researcher with the aid of the registered nurse who was responsible for blood sampling. The nurse made arrangements with pharmaceutical companies that distribute glucometers to provide these meters plus glucose strips free of charge for all patients. This enabled the researcher and nurse to perform safe and accessible screening, free of charge. On this day, 45 subjects were screened. All subjects who met

the inclusion criteria did not wish to participate in the study. In association with the Mangaung Municipality, another screening day was organized at Checkers Hypermarket. Only one of the 60 subjects who were screened during the two consecutive days volunteered to participate in the study. This subject was supplied with a free glucometer and glucose strip starter pack. The researcher planned a Diabetes Open Day in association with the Diabetes Association of South Africa's Free State Branch at Grey College School. This diabetes day was advertised in the local media, as well as broadcasted on a local radio station. Invitations were also sent to all pharmacies, clinics, hospitals and shops. Speakers for the day included a physician, biokinethetist, podiatrist, psychologist and a dietician. Pharmaceutical Companies that sell diabetic products held exhibitions and awarded prizes to all participants. A fun run/walk took place, and all the hospitals in Bloemfontein were represented. None of the subjects screened during this day met all the inclusion criteria.

3.9 SUMMARY

The methodology of the study is decribed in this chapter. The study sample, as well as the selection of the sample, is also discussed. Special attention was given to the methods of suitable selection and standardization of apparatus, as well as techniques that were used in this study. The methods and techniques have been standardized by the use of standard apparatus and procedures, and were valid and reliable. The problems that were encountered during the execution of the study did not influence the results of the study in such a way that the goals of the study could not be achieved. The results of the study are described in chapter four.

4.1 INTRODUCTION

In order to recruit a significant number of subjects who met all the inclusion criteria, the study was carried out over a period of 17 months. Suitable subjects entered the study at different times. The study period for each subject was 12 weeks, to allow for the stabilization of the subjects. Randomization took place during week 12. The trial period started on week 13 and will be referred to as trial week one. The same principle is applied throughout for trial weeks two to four. Group 1 received a sugar-inclusive diet (SID), and Group 2 received a sugar free diet (SFD), for the duration of the trial period. The data were statistically analyzed to test for significant differences between the two dietary groups. The results of the study are presented in this chapter in the following order:

- Drop-outs that occurred during the study period and reasons for this.
- Description of characteristics and habitual dietary intake of subjects at baseline.
- Weight maintenance.
- Effects of the two diets on anthropometrical measurements during the trial period.
- Effects of the two diets on glycaemic control during the trial period.
- Effects of the two diets on blood lipid concentrations during the study period.
- Comparison between the effects of the two diets on biochemical parameters.
- Lifestyle and medication factors during the trial period.

4.2 DROP OUT OF SUBJECTS

Drop-outs occurred prior to baseline and randomization, as well as after randomization during the study period.

4.2.1 Drop-outs prior to randomization

Prior to randomization, thirteen drop-outs occurred. Table 5 shows the drop-outs prior to baseline measurements, and before randomization, as well as the reasons for this.

Table 5: Drop-outs prior to baseline measurements and before randomization, as well as reasons for this

Reason	Frequency	Study period
Died in shooting accident	1	4
Non-diabetic subject	2	Before baseline, 2
Chronic illness	1	13
Commencement of insulin therapy	2	Before baseline
Non-dietary compliance	3	Before baseline
Personal reasons	3	Before baseline
Cardiac arrest	1	9

4.2.2 Drop out of subject after randomization

The biochemical data of one subject in Group 2 (SFD), who completed the study period, could not be used, as his GAD antibodies indicated that he was

a type 1 diabetic. This test could only be done after completion of the study, since the analyzing kit contained 50 *in vitro* tests that required 50 subjects to be tested at once. This subject was referred to his physician.

4.3 DESCRIPTION OF CHARACTERISTICS AND HABITUAL DIETARY INTAKE OF SUBJECTS AT BASELINE

4.3.1 Description of characteristics of subjects at baseline

Five males and five females in Group 1 (SID), and six males and six females in Group 2 (SFD), completed the entire study. Table 6 shows the number of subjects that were diagnosed with type 2 diabetes mellitus prior to the study, and those who were newly diagnosed with type 2 diabetes mellitus during the screening period of the study. Group 1 had five previously diagnosed and five newly diagnosed subjects with type 2 diabetes mellitus, while Group 2 had five previously diagnosed and seven newly diagnosed subjects.

Table 6: Number of subjects previously diagnosed, and those who were newly diagnosed with type 2 diabetes mellitus

	Group 1 (SID) N=10	**Group 2 (SFD) N=12**		**Group 1 (SID) N=10**	**Group 2 (SFD) N=12**
Previously diagnosed			Newly diagnosed		
Male	3	2	Male	2	4
Female	2	3	Female	3	3

The mean, standard deviation (SD), as well as median of age; and the anthropometrical data of Groups 1 and 2 at baseline (week 0), are shown in Table 7. The two groups were similar regarding mean age and body-fat percentage.

Table 7: Age and anthropometrical data of Group 1 (SID) and Group 2 (SFD) at baseline (week 0)

	Group 1 (SID) N=10					Group 2 (SFD) N=12				
	Mean	**SD**	**Med**	**Min**	**Max**	**Mean**	**SD**	**Med**	**Min**	**Max**
Age (yrs)	55.3	6.7	56.5	40.5	62.9	56.1	7.2	57.0	39.7	63.9
Weight (kg)	94.0	17.6	96.7	66.0	115.0	98.4	16.4	93.5	82.0	135.0
BMI (kg/m^2)	34.0	6.3	33.6	22.1	43.4	31.9	5.61	29.9	27.4	43.6
Body-fat %	38.6	8.4	38.7	26.0	49.6	31.5	12.5	28.4	12.4	47.5

4.3.2 Habitual dietary intake of subjects at baseline.

A food record, combined with a validated quantitative food frequency questionnaire, were used to analyze habitual dietary intake. Table 8 shows the mean, standard deviation (SD), and median of energy, as well as dietary intake of proteins, carbohydrates and fats of the two groups.

Table 8: Dietary intake of Group 1 and Group 2 after recruitment

	Group 1 (SID) N=10					Group 2 (SFD) N=12				
	Mean	SD	Med	Min	Max	Mean	SD	Med	Min	Max
Energy (kJ)	10604.3	3362.9	8935.5	7413.5	17746.4	12106.2	3511.5	11698.8	6497.4	19079.0
Proteins (g)	89.1	26.2	81.0	56.6	139.5	107.8	35.0	107.0	50.3	182.3
Carbohydrates (g)	253.0	96.9	235.0	174.1	467.0	292.5	107.1	243.0	197.5	528.5
Fats (g)	113.9	57.5	112.1	39.0	225.8	126.5	42.2	130.5	42.6	182.3
Sucrose (g)	24.2	24.0	16.3	8.9	90.7	29.1	17.6	24.6	5.3	69.5

Table 9 shows the intake of carbohydrates, proteins and fats for Group 1 (SID) and 2 (SFD), as a percentage, after recruitment.

Table 9: Dietary intake of carbohydrates, proteins and fats for Group 1 (SID) and Group 2 (SFD), as a percentage, after recruitment.

	Group 1 (SID) N=10					Group 2 (SFD) N=12				
	Mean	**SD**	**Med**	**Min**	**Max**	**Mean**	**SD**	**Med**	**Min**	**Max**
Proteins (%)	14.5	2.1	15.0	12.0	17.0	15.4	4.2	14.0	10.0	24.0
Carbo-hydrates (%)	42.2	9.8	44.0	23.0	59.0	41.1	8.0	42.0	27.0	54.0
Fats (%)	39.8	13.0	37.0	20.0	66.0	39.1	6.2	39.0	25.0	51.0
Sucrose (%)	4.5	5.9	2.5	2.0	21.0	4.2	2.2	4.0	1.0	9.0

A balanced diet consists of a total daily energy intake of 50-60% carbo-hydrates, 10-20% proteins and 30% fats. The habitual intake after recruitment was as follows: The carbohydrate intake for Group 1 (SID) and Group 2 (SFD) was below 50% of the total daily energy intake. The protein intake for both groups was within the balanced diet range. Fat intake for Group 1 (SID) and Group 2 (SFD) was 39.8% and 39.1% respectively. Prior to the study period Group 1 (SID) showed a sugar intake of 4.5%, and Group 2 (SFD) 4.2% respectively.

4.4 WEIGHT MAINTENANCE DURING THE STUDY PERIOD

Subjects had to maintain their weight throughout the study period. The rationale for this was that weight loss might improve their glycaemic control. However, less than 5% weight change was allowed throughout the study period.

Table 10 shows the mean changes in weight from baseline to trial weeks 1, 2, 3 and 4. No statistically significant weight changes occurred within or between Group 1 (SID) and Group 2 (SFD) during the trial period.

Table 10: Mean differences in weight (kg) in Group 1 (SID) and Group 2 (SFD)

	Week	Group 1 (SID) N=10 Mean (95% CI)	Group 2 (SFD) N=12 Mean (95% CI)	95% CI for mean difference between groups
Weight (kg)	Trial wk 1 – Baseline (wk 12)	0.6 -0.6;1.8	-0.2 -0.9;0.5	-0.5;2.0
	Trial wk 2 – Baseline (wk 12)	0.5 -0.6;1.7	-0.2 -1.2;0.9	-0.7;2.1
	Trial wk 3 – Baseline (wk 12)	0.4 -0.7;1.6	-0.2 -1.1;0.6	-0.7;2.0
	Trial wk 4 – Baseline (wk 12)	0.1 -1.0;1.2	-0.6 -1.8;0.6	-0.8;2.3

Table 11 shows the BMI of the two groups at baseline (week 12) and during the four trial weeks.

Table 11: BMI (kg/m²) of Group 1 (SID) and Group 2 (SFD): weeks 12 and trial period

	Group 1 (SID) N=10					Group 2 (SFD) N=12				
	Mean	**SD**	**Med**	**Min**	**Max**	**Mean**	**SD**	**Med**	**Min**	**Max**
Week 12	33.3	6.0	32.9	21.6	43.4	31.3	6.0	29.5	26.6	44.2
Trial Wk 1	33.5	6.0	32.9	21.5	43.9	31.3	5.9	29.5	26.4	43.9
Trial Wk 2	33.5	6.2	33.1	21.4	44.1	31.3	5.9	29.5	27.0	43.9
Trial Wk 3	33.5	6.1	33.3	21.5	44.4	33.2	5.7	29.3	27.1	43.3
Trial Wk 4	33.4	6.0	33.0	21.7	43.9	31.1	5.5	29.4	27.2	42.5

Table 12 shows the mean changes in BMI at baseline, and trial weeks 1, 2, 3 and 4. No statistically significant changes occurred in BMI within or between the two study groups.

Table 12: Mean differences in BMI (kg/m²) in Group 1 (SID) and Group 2 (SFD)

	Week	Group 1 (SID) N=10 Mean (95% CI)	Group 2 (SFD) N=12 Mean (95% CI)	95% CI for mean difference between groups
BMI (kg/m²)	Trial wk 1 – Baseline	0.2 -0.2;0.7	-0.1 -0.3;0.2	-0.2;0.7
	Trial wk 2 – Baseline	0.2 -0.2;0.6	-0.0 -0.4;0.3	-0.2;0.7
	Trial wk 3 – Baseline	0.2 -0.3;0.6	-0.1 -0.3;0.2	-0.2;0.7
	Trial wk 4 – Baseline	0.0 -0.4;0.4	-0.2 -0.6;0.2	-0.3;0.8

Table 13 shows the baseline (week 0) and end (trial week 4) body-fat percentages of the two groups.

Table 13: Body-fat percentage of Group 1 (SID) and Group 2 (SFD): Baseline and end of the study

	Group 1 (SID) N=10					Group 2 (SFD) N=12				
	Mean	**SD**	**Med**	**Min**	**Max**	**Mean**	**SD**	**Med**	**Min**	**Max**
Baseline Wk 0	39.7	10.7	39.2	18.4	52.8	35.2	11.8	35.3	19.8	52.8
Trial wk 4	38.6	8.4	38.7	26.0	49.6	30.7	12.7	25.9	12.4	47.5

Table 14 shows the mean changes in body-fat percentage from baseline to trial week 4. Clinical significant body-fat changes occurred in Group 1, and statistically significant body-fat percentage changes occurred in Group 2. No statistical differences occured between the groups.

Table 14: Mean differences in body-fat percentage in Group 1 (SID) and Group 2 (SFD)

	Week	**Group 1 (SID) N=10 Mean (95% CI)**	**Group 2 (SFD) N=12 Mean (95% CI)**	**95% CI for mean difference between groups**
Body-fat %	End – baseline	-.07	-4.6	
	Trial Wk 4 – Wk 0	-7.2;5.7	-8.5;-0.6	-2.7;10.4

4.5 EFFECTS OF THE TWO DIETS ON GLYCAEMIC CONTROL DURING THE TRIAL PERIOD

The effects of the two diets on plasma glucose, serum fructosamine and HbA_{1c} are shown in tables 15 –20.

Table 15 shows the glucose concentrations of Group 1 (SID) and Group 2 (SFD) at baseline and for the four trial weeks. The mean values of both groups at all trial weeks were above the optimal fasting reference range of 4 – 6 mmol/l (SEMDSA, 2003) for diabetic subjects. The mean values of both groups at all trial weeks were within the acceptable glycaemic control reference range of 6 – 8 mmol/l (SEMDSA, 2003).

Table 15: Plasma glucose (mmol/l) of Group 1 (SID) and Group 2 (SFD): week 12 and trial period

	Group 1 (SID) n=10					Group 2 (SFD) n=12				
	Mean	SD	Med	Min	Max	Mean	SD	Med	Min	Max
Baseline	7.7	1.4	7.4	6.1	10.0	7.3	0.9	7.0	6.5	9.7
Trial Wk 1	8.0	1.4	8.0	6.0	10.2	7.4	1.2	7.1	5.6	9.4
Trial Wk 2	8.0	2.3	7.4	5.9	13.1	7.2	1.2	6.9	5.5	9.4
Trial Wk 3	8.0	1.5	7.9	6.3	11.2	7.7	1.6	7.1	6.1	10.9
Trial Wk 4	8.0	1.9	8.2	5.5	11.9	7.2	1.5	6.9	5.0	10.0

Table 16 shows the mean changes in plasma glucose concentrations from baseline and during the trial period. No statistically significant changes occurred in plasma glucose concentrations within or between the two groups.

Table 16: Mean differences in plasma glucose (mmol/l) concentration in Group 1 (SID) and Group 2 (SFD)

	Week	Group 1 (SID) N=10 Mean (95% CI)	Group 2 (SFD) N=12 Mean (95% CI)	95% CI for mean difference between groups
Plasma glucose (mmol/l)	Trial wk 1 – Baseline	0.3 -0.5;1.1	0.1 -0.5;0.7	-0.7;1.1
	Trial wk 2 – Baseline	0.3 -0.9;1.4	-0.1 -0.7;0.6	-0.8;1.5
	Trial wk 3 – Baseline	0.3 -0.4;0.9	0.4 -0.4;1.2	-1.2;0.8
	Trial wk 4 – Baseline	0.3 -0.6;1.2	-0.1 -1.0;0.9	-0.9;1.6

The fructosamine concentrations of Group 1 (SID) and Group 2 (SFD) at baseline (week 11) and trial weeks 2 and 4, are shown in Table 17. The mean values of both groups at all trial weeks were higher than the optimal fasting reference range of 205 – 285 μmol/l for diabetic subjects (SEMDSA, 2003).

Table 17: Serum fructosamine (μmol/l) concentrations of Group 1 (SID) and Group 2 (SFD): week 11, and trial period

	Group 1 (SID) N=10					Group 2 (SFD) N=12				
	Mean	**SD**	**Med**	**Min**	**Max**	**Mean**	**SD**	**Med**	**Min**	**Max**
Baseline (Wk 11)	300.8	40.0	298.5	245.0	387.0	303.0	40.9	295.0	252.0	386.0
Trial Wk 2	293.0	26.5	289.0	258.0	341.0	288.8	38.3	292.5	232.0	373
Trial Wk 4	297.2	21.8	298.0	268.0	341.0	294.2	38.3	294.0	231.0	355

Table 18 shows the mean changes in serum fructosamine concentrations at baseline and trial weeks 2 and 4. No statistically significant changes in serum fructosamine concentrations occurred within Group 1 (SID). The changes in serum fructosamine concentrations that occurred within Group 2 (SFD) were statistically significant. No statistically significant changes occurred between the two groups.

Table 18: Mean differences in serum fructosamine (μmol/l) concentrations in Group 1 (SID) and Group 2 (SFD)

	Week	Group 1 (SID) N=10 Mean (95% CI)	Group 2 (SFD) N=12 Mean (95% CI)	95% CI for mean difference between groups
Serum Fructos-amine (μmol/l)	Trial wk 1 – Baseline	-7.7 -35.6;20.2	-14.3 -25.3;-3.2	-19.5;32.6
	Trial wk 3 – Baseline	-3.6 -37.7;30.5	-8.8 -20.8;3.3	-26.0;36.3

Table 19 shows the baseline and end results of the HbA_{1c} of Group 1 (SID) and Group 2 (SFD). The mean percentage of both groups during all trial weeks was lower than the optimal fasting reference range of < 7% (SEMDSA, 2003) and within optimal control.

Table 19: HbA_{1c} (%) of Group 1 (SID) and Group 2 (SFD): baseline and end of the study

	Group 1 (SID) N=10					Group 2 (SFD) N=12				
	Mean	**SD**	**Med**	**Min**	**Max**	**Mean**	**SD**	**Med**	**Min**	**Max**
Baseline	6.8	0.7	6.7	5.6	8.1	7.4	2.2	7.1	4.2	11.0
End	6.3	0.6	6.2	5.4	7.6	6.0	1.1	6.0	4.4	7.9

The 95% CI for the difference in the HbA_{1c} between Group 1 (SID) and Group (SFD) 2 is shown in Table 20. Group 1 had an improvement very close to statistical significance. Group 2 showed a statistically significant change.

Table 20: Mean differences in HbA_{1c} % of Group 1 (SID) and Group 2 (SFD)

	Week	**Group 1 (SID) N=10 Mean (95% CI)**	**Group 2 (SFD) N=12 Mean (95% CI)**	**95% CI for mean difference between groups**
HbA_{1c} %	End – Baseline	-0.6 -1.2;0.1	-1.4 -2.6;-0.2	-0.5;2.2

4.6 EFFECTS OF THE TWO DIETS ON BLOOD LIPID CONCENTRATIONS DURING THE STUDY PERIOD

The effects of the two diets on serum cholesterol, serum triglycerides, serum HDL cholesterol and serum LDL cholesterol are shown in tables 21-28.

Table 21 shows the cholesterol concentrations for the two groups at baseline and at the end of the trial period. The mean concentrations of both groups at all trial weeks were higher than the optimal fasting reference range of < 5 mmol/l (SEMDSA, 2003).

Table 21: Serum cholesterol (mmol/l) concentrations of Group 1 (SID) and Group 2 (SFD): baseline (week 0) and end (trial week 4) of the study

	Group 1 (SID) N=10					Group 2 (SFD) N=12				
	Mean	**SD**	**Med**	**Min**	**Max**	**Mean**	**SD**	**Med**	**Min**	**Max**
Week 0	5.5	1.1	5.7	4.3	7.7	5.7	1.4	5.4	4.1	9.6
Trial Wk 4	5.5	0.6	5.6	4.2	6.5	5.1	0.9	5.3	3.8	6.4

Table 22 shows the mean changes in serum cholesterol concentrations from baseline to the end of trial week 4. No significant changes in serum cholesterol concentrations occurred within or between the two groups.

Table 22: Mean differences in serum cholesterol (mmol/l) concentrations in Group 1 (SID) and Group 2 (SFD)

	Week	Group 1 (SID) N=10 Mean (95% CI)	Group 2 (SFD) N=12 Mean (95% CI)	95% CI for mean difference between groups
Serum cholesterol (mmol/l)	End – baseline	0.0 -0.5;0.5	-0.6 -1.6;0.5	-0.7;1.8

Table 23 shows the triglyceride concentrations of Group 1 (SID) and Group 2 (SFD) at baseline and the end of the trial period. The mean values of both groups at all trial weeks were higher than the optimal fasting reference range of < 1,5 mmol/l (SEMDSA, 2003).

Table 23: Serum triglycerides (mmol/l) of Group 1 (SID) and Group 2 (SFD): baseline and end of the study

	Group 1 (SID) N=10					Group 2 (SFD) N=12				
	Mean	SD	Med	Min	Max	Mean	SD	Med	Min	Max
Baseline Week 0	1.8	1.2	1.6	0.6	4.1	2.1	1.2	1.6	0.9	4.6
End Trial wk 4	2.0	1.1	1.7	0.7	4.4	2.4	1.1	2.3	1.1	4.7

Table 24 shows the mean changes in serum triglyceride concentrations at baseline and trial week 4. No significant changes in serum triglyceride concentrations occurred within the two groups. No statistically significant changes occurred between the two groups.

Table 24: Mean differences in serum triglycerides (mmol/l) in Group 1 (SID) and Group 2 (SFD)

	Week	Group 1 (SID) N=10 Mean (95% CI)	Group 2 (SFD) N=12 Mean (95% CI)	95% CI for mean difference between groups
Serum Triglycerides (mmol/l)	End – baseline	0.1 -0.4;0.7	0.3 -0.3;0.9	-0.9;0.6

HDL cholesterol concentrations of Group 1 (SID) and Group 2 (SFD), at baseline and end of the trial period are shown in Table 25. The mean values of Group 1 (SID) were below the reference range of > 1,2 mmol/l (SEMDSA, 2003) in trial week 4. Group 2 (SFD) had mean values within the optimal fasting reference range at baseline. Group 2 (SFD) had a mean HDL cholesterol concentration below the reference range of > 1,2 mmol/l for trial week 4.

Table 25: Serum HDL cholesterol (mmol/l) concentrations of Group 1 (SID) and Group 2 (SFD): baseline and end of the study

	Group 1 (SID) N=10					Group 2 (SFD) N=12				
	Mean	**SD**	**Med**	**Min**	**Max**	**Mean**	**SD**	**Med**	**Min**	**Max**
Baseline										
Week 0	1.1	0.2	1.1	0.8	1.5	1.5	1.5	0.9	0.6	4.5
End										
Trial										
wk 4	1.0	0.3	1.0	0.7	1.5	0.90	0.2	0.9	0.6	1.1

The mean differences in HDL cholesterol concentrations of Group 1 and Group 2, at baseline and the end of the trial period, are shown in Table 26. No significant changes in HDL cholesterol concentrations occurred in the two groups. No statistically significant differences occurred between the groups.

Table 26: Mean differences in serum HDL cholesterol concentrations in Group 1 (SID) and Group 2 (SFD)

	Week	Group 1 (SID) N=10 Mean (95% CI)	Group 2 (SFD) N=12 Mean (95% CI)	95% CI for mean difference between groups
Serum HDL cholesterol (mmol/l)	End – baseline	-0.1 -0.3;0.1	-0.7 -1.6;0.3	-0.4;1.5

Table 27 shows the LDL cholesterol concentrations of Group 1 and Group 2, at baseline and the end of the trial period. The mean concentrations of both groups at all trial weeks were higher than the optimal fasting reference range of ≤ 3,0 mmol/l (SEMDSA, 2003).

Table 27: Serum LDL cholesterol (mmol/l) concentrations of Group 1 (SID) and Group 2 (SFD): baseline and end of the study

	Group 1 (SID) N=10					Group 2 (SFD) N=12				
	Mean	SD	Med	Min	Max	Mean	SD	Med	Min	Max
Baseline	3.5	1.0	3.5	2.3	5.5	3.2	1.0	3.0	1.1	4.9
End	3.6	0.7	3.9	2.3	4.5	3.2	1.0	3.5	1.2	4.3

Table 28 shows the mean differences in LDL cholesterol concentrations of Group 1 (SID) and Group 2 (SFD) at baseline and the end of the study. No statistically significant changes occurred in LDL cholesterol concentrations within the two groups. No statistical differences occured between the groups.

Table 28: Mean differences in serum LDL cholesterol (mmol/l) concentrations in Group 1 (SID) and Group 2 (SFD)

	Week	Group 1 (SID) N=10 Mean (95% CI)	Group 2 (SFD) N=12 Mean (95% CI)	95% CI for mean difference between groups
Serum LDL cholesterol (mmol/l)	End – baseline	0.1 -0.4;0.6	0.0 -0.7;0.7	-0.7;0.9

4.7 LIFESTYLE AND MEDICATION REPORT

The results of the lifestyle and medication report are discussed hereafter. The focus of the lifestyle report was smoking, alcohol intake and exercise patterns.

4.7.1 Smoking

At baseline one subject (10%) in Group 1 (SID) smoked 20 cigarettes per day, and one subject (8,3%) in Group 2 (SFD) smoked 10 cigarettes per day. A similar smoking pattern was maintained by the subjects throughout the four week trial period.

4.7.2 Alcohol

The habitual alcohol intake of subjects during the study period was documented on a weekly basis (Appendix 7). One subject in Goup 1 (SID) reduced his alcohol intake from nine servings per week to four servings per week during trial week 3. During trial week 3 and 4 one subject halved his alcohol intake. In Group 2 (SFD) one subject had six alcohol servings per day. Other alcohol intake patterns remained the same for both groups.

4.7.3 Exercise

In Group 1 (SID), 50% of the subjects exercised for approximately 2-6 times per week for 20-180 minutes for the duration of the trial period. In Group 2 (SFD) 83,3% exercised from 15-300 minutes per week, 1-7 times per week. Group 2 SFD) had a higher activity level than Group 1 (SID). Only one subject reduced his exercise routine in week four from 70 minutes twice a week to 20 minutes five times per week.

4.7.4 Medication

The medication intake for both groups remained the same during the four week trial period.

4.8 SUMMARY

Twenty three subjects out of the 401 that were initially recruited for the study, met the inclusion criteria, and took part in the study. One subject dropped out at the end of the study, because his GAD test indicated that he had type 1 diabetes mellitus.

The mean changes in weight that occurred were within the recommended range of less than five percent. This weight maintenance was important to exclude improvement of glycaemic control due to weight loss. Clinically significant mean

body-fat percentage changes occurred in Group 1 (SID). Group 2 (SFD) showed statistically significant improvements in mean body-fat percentage.

The mean plasma glucose concentrations for both groups were within the acceptable glycaemic control reference range of 6-8 mmol/l (SEMDSA, 2003) for baseline and the end of the trial period. The mean serum fructosamine concentrations of Group 1 (SID) remained unchanged during the trial period. The mean serum fructosamine concentrations of Group 2 (SFD) showed statistically significant improvement during the trial period. No changes occurred between the two groups.

No changes in mean serum cholesterol, HDL cholesterol, LDL cholesterol and serum triglyceride concentrations occurred within and between the two groups during the trial period.

Both groups maintained a mean HbA_{1c} percentage within the optimal fasting reference range of < 7% (SEMDSA, 2003). Group 1 (SID) showed an improvement in HbA_{1c} percentage that was close to statistical significance and is of clinical significance. The improvement of Group 2 (SFD) was of statistical significance.

Subjects' smoking habits, alcohol intake and exercise patterns remained constant throughout the trial period. The subjects maintained their medication usage during the trial period.

5.1 INTRODUCTION

In this chapter the effects of sucrose intake on glycaemic control and blood lipid concentrations in subjects with type 2 diabetes mellitus are discussed. Furthermore, the results of this study are compared with those of other studies and possible explanations for the results are given.

5.2 WEIGHT MAINTENANCE DURING THE STUDY PERIOD

5.2.1 Bodyweight and BMI

Table 29 shows trials that included sucrose in diabetic diets. The effects of the SID and SFD on body weight, BMI and body-fat percentage were as follows.

Although overweight and/or obesity were not inclusive criteria for this study, all subjects had class I obesity (BMI = 30 to 34.9 kg/m^2) (Laquatra, 2000, p.493), according to the World Health Organization classification. In other studies (Colagiuri *et al.*, 1989; Tariq *et al.*, 2001), in which subjects with type 2 diabetes mellitus were included, all subjects were overweight according to their BMI.

Obesity plays an important role in the etiology of type 2 diabetes mellitus. Approximately 36% of subjects with type 2 diabetes mellitus are obese (Franz *et al.*, 2002, p.20). Subjects who want to lose weight believe that sucrose restrictive diets will increase weight loss. Weight-loss studies with different types and amounts of carbohydrates, including a high and low sucrose content, did not show that weight loss is impaired by high-sucrose, energy restricted diets (Saris, 2003).

One of the most fundamental changes emphasized in the recent guidelines of the American Diabetes Association Expert Panel (Kelly, 2003), is the need for individualization of the nutritional prescription. This priority placed on total energy consumption and carbohydrate counting, is fundamental in the overall emphasis that metabolic control, in terms of hyperglycaemia in an overweight or obese person with type 2 diabetes mellitus, is highly sensitive to either positive or negative energy balance. Negative energy balance can promptly induce reductions in hyperglycaemia, even before the achievement of substantial weight loss, whereas consumption of excess energy has the opposite effect (Kelly, 2003).

All subjects had to maintain their bodyweight during the study period and their diets were individualized in order to achieve this. Therefore, glycaemic control, or the improvement thereof, could not be attributed to weight loss. In a similar study where Abraira and Derler (1988) added 120g sucrose to a balanced diet, and Peterson *et al.* (1986), added sucrose up to 18% of the total energy, the subjects also maintained their body weight throughout the respective study periods.

Table 29: Sucrose-inclusive diet trials

Reference	Study population	Sample size	Prescribed diet	Study period	Study design
Bantle *et al.* (1983)	- Healthy subjects or type 1 or type 2 subjects - No oral hypoglycaemic agents - Maintain weight - Metabolic kitchen prepared meals for the free living population	Group 1: N = 10 Group 2: N = 12 Group 3: N = 10	Identical amounts of carbohydrates, proteins and fat with different test carbohydrates, 24-25% of total energy. ± 42g glucose, fructose, sucrose, potato starch or wheat starch.	5 days	Randomized
Slama *et al.* (1984)	- Well-controlled subjects with type 1 or type 2 diabetes mellitus - Hospitalized subjects	type 1: N = 6 type 2: N = 12	20g Sucrose added to a standard diet	2 days	Comparative
Bantle *et al.* (1986)	- Subjects with type 1 or type 2 diabetes mellitus	type 1: N = 12 Type 2: N = 12	3 Diets 21% fructose 23% sucrose Almost all CHO energy as starch	8 days each	Cross-over

Reference	Study population	Sample size	Prescribed diet	Study period	Study design
Peterson *et al.* (1986)	- Subjects with type 1 or type 2 diabetes mellitus - BMI < 28 kg/m^2 - Free living	type 1: N = 12 Type 2: N = 11	1. 18% of total energy as sucrose (45g sucrose)	6 weeks each	Randomized cross-over
Abraira and Derler (1988)	- Subjects with type 2 diabetes mellitus - No oral hypoglycaemic drugs - Maintain weight - Inpatients for 40 days	18	120g sucrose added to a standard diet 1. 220g sucrose 2. < 3g sucrose	10 days and then 1 month	Uncertain, maybe cross-over
Colagiuri *et al.* (1989)	- Subjects with type 2 diabetes mellitus - Maintain weight - Sucrose and aspartame packed in sachets	9	45g Sucrose (9% TE) 162 mg Aspartame	≥ 3 months pre-study compliance check 6 weeks each	Randomized, double blind, cross-over
Wise *et al.* (1989)	- Subjects with type 1 diabetes mellitus - camp	16	7% sucrose 1% sucrose	5 days	Double blind
Loghmani *et al.* (1991)	- Subjects with type 1 diabetes mellitus - hospitalized	10	10% TE as sucrose 2% TE as sucrose	2 days	Cross-over

Reference	Study population	Sample size	Prescribed diet	Study period	Study design
Bantle *et al.* (1993)	- Subjects with type 2 diabetes mellitus - Maintain weight - Metabolic kitchen prepared food for free living population	12	19% TE sucrose < 3% TE sucrose	28 days	Cross-over
Marchini *et al.* (1994)	- Subjects with type 2 subjects diabetes mellitus - healthy subjects - hospitalized	N = 13 N = 6	30g/day sugar No sugar	22 hour	Comparative
Schwings-handl *et al.* (1994)	- Subjects with type 1 diabetes mellitus	Sugar: N = 11 Sucrose free: N = 13	5 % refined sugar	42-127 days (mean 83 days)	Comparative
Malerbi *et al.* (1996)	- Well-controlled subjects with type 2 diabetes mellitus - Maintain body weight - Free living, seen fortnightly	16	Standard diet with: 20% Fructose 19% Sucrose 5% Sugars (control diet)	28 days each with 14 day washout in between	

Reference	Study population	Sample size	Prescribed diet	Study period	Study design
Nadeau *et al.* (2001)	- Subjects with type 2 diabetes mellitus - Free living	48	10% of the total energy as added sugars	8 months	Randomized control trial
Tariq *et al.* (2001)	- Subjects with type 2 diabetes mellitus - Living in a nursing home	28	1. No-concentrated sweets diet 2. Standard diet	6 months	Comparative

5.2.2 Body-fat percentage

Both dietary groups in this study experienced improvement in mean body fat percentages, irrespective of weight maintenance. However, Group 2 (SFD) showed statistically significant improvement in body-fat percentage (4.5%), while the improvement observed in Group 1 (SID) is of clinical significance (1.1%). No differences occurred in body-fat percentage between the groups. Both diets included 30% fat, which was lower than the habitual dietary intake of the subjects prior to the study, ranging between 39.1 and 39.8 percent of the total energy respectively. Franz and co-workers (2002, p.19) state that higher fat intake is accompanied by higher fat deposits. During this study period, the fat intake was reduced with almost 10%. Thus, the daily fat deposits were lower, and this might be one of the reasons for the improvement in body fat percentage. Futhermore, both groups showed a mean habitual carbohydrate intake of 42.2 and 41.1 % respectively, prior to the study period. This carbohydrate intake was increased to 50-60% of the total daily energy intake during the study period. The fact that there was a change in body composition, without weight loss, may be attributed to the strict compliance and adherence of subjects to their dietary guidelines. The researcher also advised all subjects to increase their activity level, as this improves glycaemic control. Some of the subjects started with an exercise programme at the commencement of the study. These subjects increased their activity level prior to baseline and were able to maintain it throughout the study period. Exercise builds muscle and burns fat (Whitney & Rolfes, 2002, p.284). The improvement (reduction) in body-fat percentage might, thus, further be attributed to a change in a previously sedentary lifestyle to a more active lifestyle.

5.3 THE EFFECTS OF THE SID AND SFD ON GLYCAEMIC CONTROL

The effects of the inclusion of different percentage levels of sucrose (5-23% of the total energy intake) to the diabetic diet, were investigated by various authors and the effects of sucrose-inclusive diet trials on biochemical parameters are indicated in Table 30. The effects of the SID and the SFD on plasma glucose, serum fructosamine and HbA_{1c} percentages will be discussed hereafter.

5.3.1 Plasma glucose

The inclusion of 15% of the total daily energy intake as sucrose in the diet of Group1 (SID) had no deleterious effect on their plasma glucose concentrations. No significant changes occurred in plasma glucose concentrations within or between the two groups, respectively. This finding corresponds with studies where 5-23% of the total energy intake was included in the diabetic diet as sucrose (Bantle *et al.*, 1983; Slama *et al.*, 1985; Bornet *et al.*, 1985; Bantle *et al.*, 1986; Peterson *et al.*, 1986; Abraira *et al.*, 1988; Colagiuri *et al.*, 1989; Peters *et al.*, 1990; Bantle *et al.*, 1993; Malerbi *et al.*, 1996; Tariq *et al.*, 2001).

Recently the American Diabetes Association Expert Panel (Kelly, 2003), analyzed 22 studies addressing glycaemic control and sucrose intake as part of a diabetic diet. They concluded that when ingested in iso-caloric quantities, sucrose does not affect glycaemic control in diabetes mellitus significantly differently from other carbohydrates. They recommended that if sucrose is consumed, it should be substituted for other carbohydrates.

Table 30: Effects of sucrose-inclusive trials on biochemical parameters

	Means of incorporating sucrose in the diet	Plasma glucose concentration (mmol/l)		HbA_{1c} (%)		Serum cholesterol concentration (mmol/l)		Serum triglyceride concentration (mmol/l)		Serum HDL cholesterol concentration (mmol/l)		Serum LDL cholesterol concentration (mmol/l)	
		T	C	T	C	T	C	T	C	T	C	T	C
Slama *et al.* (1984)	20g to std. diet	5.8	5.8										
Peterson *et al.* (1986)	18% of the TE	8.9	9.1	9.0	9.3	5.3	5.1	1.7	1.8	1.4	1.5	3.4	3.0
Abraira and Derler (1988)	220g to std. diet	8.2				5.0		1.9		1.0		3.2	
Colagiuri *et al.* (1990)	9% of the TE	6.2	6.0	7.5	7.3	5.3	5.4	2.1	2.1	1.0	0.9		
Bantle *et al.* (1993)	19% of the TE	9.6	9.4			5.1	4.9	1.9	1.8	1.1	1.0	3.1	3.1
Tariq *et al.* (2001)	No-concentrated sweets diet	7.3	6.6	6.7	6.4								
Nadeau *et al.* (2001)	10% of the TE	8.0	8.4	7.2	7.7	5.1	4.9	2.0	1.7	1.3	1.2	2.8	2.9
This study	15% of the TE	8.0	7.2	6.3	6.0	5.5	5.1	2.0	2.4	1.0	0.9	3.6	3.2

T = Trial; C = Control; Std = Standard; TE = Total energy

In the U.S.A., the 2000 Dietary Guidelines Advisory Committee (DGAC) concluded that the only health problem with high dietary intakes of sugars (monosaccharides and disaccharides) that has been conclusively demonstrated was the association with dental caries (Murphy & Johnson, 2003). According to Kelly (2003), the available data do not clearly implicate risk of type 2 diabetes mellitus in relation to consumption of carbohydrates, monosaccharides and disaccharides specifically.

The American Heart Association concluded that no data suggest that to ingest foods high in sugars (monosaccharides and disaccharides) may be detrimental to the general population. Several studies (Murphy & Johnson, 2003) have indicated that high sucrose intake (> 20% of energy from sucrose) should be avoided, because sucrose has no nutritional value other than to provide energy. The most likely consequences of sucrose consumption beyond the levels described are over-consumption of energy and micronutrient inadequacies (Murphy & Johnson, 2003). Published recommendations suggest limiting added sugars to 10-15% of the energy (Gillespie, 1996; Lank, 2000; Sievenpiper *et al.*, 1998).

The inclusion of sucrose in the diabetic diet may have the advantage that it may enhance palatability of the overall diet, and decrease the sense of deprivation and restriction experienced by some subjects with diabetes mellitus. Subjects with type 2 diabetes mellitus may feel less guilty when they include this previous "forbidden" substance to a meal. If they are educated on how to exchange sucrose and other carbohydrates equally in a meal, their compliance will increase, since they no longer have to "cheat" while they are eating. Some subjects with type 2 diabetes mellitus feel excluded at social gatherings, like lunch or dinner parties, as friends and family often do not know what to serve to them. Subjects with type 2 diabetes mellitus can now eat wherever they want, with the knowledge that they can exchange some of their carbohydrate portions with sucrose, and still adhere to the dietary guidelines. However, it is imperative that subjects with diabetes mellitus should be educated about the inclusion of sucrose in their diets. Furthermore,

it is of vital importance to make them aware of foods containing sucrose that are also high in fat.

5.3.2 Serum fructosamine

Serum fructosamine is an indication of short term (the preceding 21 days) diabetic control (Fructosamine, 2000). Both groups had mean serum fructosamine concentrations that were higher than the optimal fasting reference range of 205-285 μmol/l for diabetic subjects, at baseline and at the end of the study period (Department of Chemical Pathology, University of the Free State). However, the mean serum fructosamine concentration of Group 2 (SFD) improved with statistical significance (95% CI: -25,3:-3,2) during the four week trial. The improvement in mean serum fructosamine concentration of Group 1 (SID) was not statistically significant, but is of clinical relevance. No significant differences were observed between the two groups. These data prove that the two groups complied with their diets on a short term basis. No similar studies that investigated the effect of sugar intake on glycaemic control, that determined serum fructosamine concentrations, could be found. This improvement in serum fructosamine concentrations shows that both dietary groups improved in compliance from baseline to the end of the study period.

Both groups were encouraged by the researcher on a fortnightly basis, and were also motivated continuously to adhere to their diets. Furthermore, the registered nurse who collected blood samples on a weekly basis also encouraged the subjects to comply with the dietary guidelines of the researcher. Thus, the reason for the improved compliance seems to be the strict control over dietary intake by the researcher. Compliance to dietary guidelines/ instructions remains a problem, as subjects at recruitment felt that a 16 week study period was too long and too restrictive for adherence.

5.3.3 HbA_{1c}

HbA_{1c} percentage refers to long term diabetic control over the preceding two or three months (Glycated haemoglobin, 1994). Both groups showed an improvement in HbA_{1c} percentage. Although Group 1 (SID) showed an HbA_{1C} percentage within the optimal range of < 7% (SEMDSA, 2003), there was an improvement in HbA_{1c}% that were very close to statistical significance. Group 2 (SFD) was within the acceptable range of 7-8 % at baseline, but was within the optimal range of < 7 % (SEMDSA, 2003) at the end of the study period. Group 2 (SFD) showed improvement of statistical significance (95% CI: -2.6;-0.2).

Several studies (Peterson *et al.,* 1986; Abraira and Derler, 1988; Colagiuri *et al.,* 1989; Malerbi *et al.*, 1996; Tariq *et al.*, 2001) had comparable results to this study. The strict control throughout the study period applied by the researcher may have ensured better compliance of subjects to their diets on the long term. The inclusion of sucrose in the diets of subjects with type 2 diabetes mellitus may improve their long term compliance to their diet and restrict their risk of developing complications. Complications due to diabetes can increase mortality and morbidity of these subjects. Thus, compliance to their diets may reduce mortality and morbidity due to complications during the course of the disease.

5.3.4 Summary

The inclusion of a moderate amount of sucrose (15% of the total energy) did not aggravate glycaemic control in subjects with type 2 diabetes mellitus. However, strict control was applied by the researcher throughout the study period and this might be a reason for the good compliance. However, in a 'real world' environment, subjects are not monitored by a registered dietician on a weekly basis, as this is not practical; especially if subjects live in remote areas. The question remains that if there is no supervision, will subjects still have optimal glycaemic control? If subjects with type 2 diabetes mellitus are

educated adequately and are less restricted with their diets, they might have better control.

5.4 THE EFFECTS OF THE SID AND SFD ON BLOOD LIPID CONCENTRATIONS

There is strong evidence in populations with type 2 diabetes mellitus, as well as healthy populations, that saturated fatty acids can worsen dyslipidaemia and insulin resistance, raise LDL cholesterol, and thereby increase the risk of cardiovascular disease (Kelly, 2003). A very low-fat diet, accompanied by a high intake of carbohydrates can precipitate metabolic changes that may result in atherogenic dyslipidaemia. The lipoprotein profile of atherogenic dyslipidaemia is characterized by elevated triglycerides; small, denseparticles of LDL cholesterol; and a low concentration of HDL cholesterol. High carbohydrate diets, especially high in sugars (monosaccharides and disaccharides), have been associated with the increased risk of cardiovascular disease. However, Murphy and Johnson (2003), emphasize that additional research is needed before these associations can be used to alter guidelines. On an iso-caloric basis, a high proportion of carbohydrates (relative to monounsaturated fats) causes plasma triglycerides to be elevated, and therefore might lead to deterioration of the dyslipidaemia, a characteristic of type 2 diabetes mellitus and insulin resistance.

A recent study (Kelly, 2003) has shown that replacing saturated fat with complex carbohydrates achieved beneficial effects on dyslipidaemia in type 2 diabetes mellitus; lowering LDL cholesterol independent of cholesterol intake, and without inducing adverse effects on HDL cholesterol and triglycerides. For many patients, for the public at large, and even within the medical community, the notion persists that persons with diabetes mellitus should avoid the ingestion of sugars (monosaccharides and disaccharides) (Kelly, 2003). Several studies (Fried & Rao, 2003, Schaefer *et al.*, 1995; and Kasim-Katakas *et al.*, 2000), demonstrated that, in highly compliant and motivated patients, low-fat, high-carbohydrate diets containing low or high amounts of

sucrose can be followed over the long term, without detrimental effects on fasting serum triglyceride or HDL concentrations. Considerable controversy exists about the potential effects of dietary sucrose on lipaemia in diabetic patients.

This study had a relatively short trial period (four weeks). From baseline of the study period to the end of the short trial period, no significant changes occurred between or within the groups regarding blood lipid concentrations. Thus, the inclusion of sucrose to the diet of subjects with type 2 diabetes mellitus had no short term aggravating effects on blood lipid concentrations. However, the effects of sucrose intake on long term blood lipid concentrations remains to be determined, as these subjects were not followed up after the trial period.

5.4.1 Serum cholesterol

Hyperlipidaemia is one of the complications of diabetes mellitus (Raal , 2000). In this study, the mean serum cholesterol concentrations of both groups at all trial weeks were higher than the optimal fasting reference range of < 5 mmol/l (SEMDSA, 2003). Although the mean serum cholesterol of Group 1 (SID) remained the same, there was a slight improvement (0,6 mmol/l) in the mean cholesterol concentration of Group 2 (SFD). However, Group 2 (SFD) had a higher baseline concentration than Group 1 (SID). No significant changes in serum cholesterol occurred within or between the two groups. These findings are in accordance with other studies (Peterson *et al.*, 1986; Abraira *et al.*, 1988; Colagiuri *et al.*, 1989; Bantle *et al.*, 1993; Malerbi *et al.*, 1996). In this study, both groups had high habitual fat intakes of ~39% of the total daily energy, but were advised to reduce fat intake to 30% of the total energy during the study period. The decrease in total fat intake might be a possible explanation for the slight improvement in serum cholesterol concentrations observed in Group 2 (SFD).

5.4.2 Serum triglycerides

In this study, the serum triglyceride concentrations of both dietary groups were higher than the recommendation of < 1.5 mmol/l (SEMDSA, 2003). However, no significant changes in serum triglyceride concentrations occurred within or between the two groups. Although there were no statistically significant changes in mean serum triglyceride concentrations in this study, there was a trend towards a slight increment in serum triglyceride concentrations after the study period. This deterioration in serum triglyceride concentrations is of clinical significance. Since the trial period was short (four weeks), the long term effect of sucrose on serum triglyceride concentrations could not have been evaluated. If this trend continued, it may lead to further increments in serum triglyceride concentrations which may have aggravated the long term glycaemic control of subjects with type 2 diabetes mellitus.

In similar studies that included sucrose (up to 23% of the total energy intake) (Bantle *et al.*, 1986; Peterson *et al.*, 1986; Colagiuri *et al.*, 1989; Bantle *et al.*, 1993; Malerbi *et al.*, 1996), no difference in the total triglycerides between the baseline and end concentrations, was observed. Abraira and Derler (1988) reported that repeated fasting or postprandial triglyceride concentrations were not affected when type 2 diabetic patients were fed 220g of sucrose for one month. Twelve of the subjects with pre-existing hypertriglyceridaemia had similar trends, but postprandial triglyceride concentrations were lower in the high-sucrose diet group of this sub-group.

On an iso-caloric basis, a high proportion of carbohydrates (relative to monounsaturated fats) causes plasma triglycerides to increase, and therefore might lead to the deterioration of dyslipidaemia, a characteristic of type 2 diabetes mellitus (Kelly, 2003). The long-term effects of high-sucrose and high-starch diets were tested in the Carbohydrate Ration Management in European National diet study (CARMEN). The low-fat, high-sucrose group increased their intake of sucrose by 33g (8%) to ~29% of the energy, and the

starch group decreased their intake of sucrose by 44 g (3.5%) to 19% of the energy, while increasing their starch intake by 8%. Both low-fat interventions resulted in a modest decline in food intake and body weight after six months, whereas the seasonal control group and high-fat groups tended to gain weight. Neither the high-sucrose, nor the high-starch diet, affected fasting serum triglyceride concentrations in these overweight subjects, but possible effects of the high-sucrose diet on post-prandial triglyceride concentrations cannot be ruled out (Fried & Rao, 2003).

Weight loss can promptly induce reductions in hypertriglyceridaemia, even before the achievement of substantial weight loss, whereas consumption of excess energy has the opposite effect (Kelly, 2003). As all subjects in this study were obese, the total energy intake of all subjects during the study period was very high, in order to maintain weight. Although the carbohydrate content of the diet was calculated according to the current diabetic guideline (ADSA, 1997) (50 – 60% of total energy), it was still relatively high. This high intake of carbohydrates might also have contributed to the slight increase in serum triglyceride concentrations during the study period. No attention was given to the source of fat intake during the trial period. Monounsaturated fats improve serum triglyceride concentrations, and perhaps the subjects did not include adequate amounts of monounsaturated fatty acids in their diets during the study period.

5.4.3 Serum HDL cholesterol

No significant changes in HDL cholesterol concentrations occurred. The mean serum HDL cholesterol concentration of Group 1 (SID) was below the reference range of > 1.2 mmol/l (SEMDSA, 2003), during the study period. However, the baseline serum HDL concentration of Group 2 (SFD) was above the reference range, but also decreased to below the reference range of > 1.2 mmol/l at the end of the study period. There was a trend in both groups towards decreased serum HDL cholesterol concentrations. Group 2 (SFD)

showed a greater decrease (from 1.5 to 0.9 mmol/l) which is of clinical significance.

Increased activity levels lessen cardiovascular risk by increasing HDL concentrations (Krummel, 2000, p.572). Despite a higher activity level, both groups had decreased HDL concentrations. Monounsaturated fat intake improves HDL concentrations (Anderson, 1999, p.1377). As the monounsaturated fat intake of subjects was not monitored during the study period, it is possible that the subjects' intake of monounsaturated fat was inadequate during the study period, and therefore decreased the serum HDL concentrations. If the monounsaturated fat intake was liberalized to include up to 20% of the energy with a more moderate intake of carbohydrates, the serum HDL concentrations might have increased. High carbohydrate diets are also associated with decreased serum HDL concentrations (American Diabetes Association, 2003). The high carbohydrate intake during the study period, might be a possible explanation for the slight decrease in HDL concentrations.

Peterson *et al.* (1986), Abraira *et al.* (1988), Colagiuri *et al.* (1989), Bantle *et al.* (1993) and Malerbi *et al.* (1996), found no difference in HDL cholesterol concentrations with the inclusion of sucrose up to 19% of the total energy intake.

5.4.4 Serum LDL cholesterol

The mean LDL concentrations of Group 1 (SID) and Group 2 (SFD) at all trial weeks were above the optimal fasting reference range of ≤ 3 mmol/l (SEMDSA, 2003). No significant changes occurred in LDL cholesterol concentrations within or between the groups. Similar sucrose inclusive studies found no difference in LDL concentrations (Peterson *et al.*, 1986; Abraira *et al.*, 1988; Bantle *et al.*, 1993; Malerbi *et al.*, 1996).

In the Insulin Resistance Atherosclerosis Study (Yki-Järvinen, 2000), the mean LDL cholesterol concentration in 594 patients with type 2 diabetes (30% White, 30% African-American, 40% Hispanic) was 3.6 mmol/l in patients with type 2 diabetes, which represented 92% of all participants, and 3.7 mmol/l in patients with insulin sensitive type 2 diabetes. Given that all guidelines recommend lipid-lowering therapy at LDL cholesterol levels greater than 3,4 mmol/l, this implies that at least 50% of patients with type 2 diabetes would need such treatment. In this study the mean serum LDL concentrations at the end of the study period was 3.6 and 3.2 mmol/l for Group 1 (SID) and Group 2 (SFD), respectively. This implies that Group 1 (SID) needed lipid-lowering therapy according to the Insulin Resistance Aterosclerosis Study. High saturated fat intake increases serum LDL cholesterol concentrations (Henry, 2001, p.118). A possible reason for the high serum LDL cholesterol concentrations might be that the saturated fat content of the diets was too high during the study period. Subjects were given practical advice on how to lower their fat intake, but no specific restrictions as to the percentage of the saturated fat to be ingested per day, were applied. In this study, there was a clinical significant change in body-fat percentage in Group 1 (SID) and a statistically significant body-fat percentage change in Group 2 (SFD). Usually, a decrease in body-fat percentage can be associated with a decrease in LDL cholesterol concentrations (Whitney and Rolfes, 2002, pp. 257;572). However, this was not evident in this study.

5.5 LIFESTYLE AND MEDICATION FACTORS DURING THE TRIAL PERIOD

Smoking, alcohol consumption, exercise patterns and medication usage are discussed.

5.5.1 Smoking

Only 1 subject in each dietary group smoked. Their smoking pattern was consistent throughout the study period. Other studies that investigated the

effect of sucrose on glycaemic control of subjects with type 2 diabetes mellitus, did not indicate the smoking pattern of their subjects.

5.5.2 Alcohol

Alcohol consumption in diabetics should be limited to two servings per day in men, and no more than one drink for women (Anderson, 1999, p.1381). Group 1 had an alcohol intake within this recommendation throughout the study period. One subject in Group 2 reported a high alcohol intake during the last two weeks of the trial period. The subject was advised to restrict his alcohol intake to two drinks per day. This subject had serum triglyceride concentrations of 4.6 mmol/l at baseline of the study period, and this reduced to 2.4 mmol/l at the end of the study period. No indication of alcohol consumption was found in Bantle *et al.*(1983), Slama *et al.* (1984), Bantle *et al.* (1986), Peterson *et al.* (1986), Abraira and Derler (1988), Colagiuri *et al.* (1989), Bantle *et al.* (1993), and Tariq *et al.* (2001).

5.5.3 Exercise

Only half of Group 1 (SID) exercised for 20-180 minutes for the duration of the study period. Group 2 (SFD) had an 83.3% exercise participation rate (15-300 minutes per week) of light to moderate intensity. Both dietary groups should be encouraged to exercise on a frequent basis. The aerobic part of an exercise programme should last a minimum of 20 minutes, with a goal of 30 to 40 minutes. People who combine diet and exercise are more likely to lose more fat, retain more muscle, and regain less weight than those who only diet (Whitney & Rolfes, 2002, p.284). Exercise helps to improve insulin sensitivity, reduce cardiovascular risk factors, control weight and bring about a healthier mind-set. Blood glucose concentrations can improve with exercise, because of decreased insulin resistance and increased insulin sensitivity, which results in increased use of peripheral use of glucose, not only during, but also after activity (Franz, 2000, p.758; American Diabetes Association, 2001). Sedentary subjects with upper-body and visceral obesity that have metabolic

syndrome, tend to be at higher risk for hypertriglyceridaemia in response to high-sucrose and high-carbohydrate diets: moderate weight loss mitigates the effect (Fried & Rao, 2003).

Abriara and Derler (1988) had a hospitalized, but ambulatory study population. Bantle *et al.* (1983), Bantle *et al.* (1986), Peterson *et al.* (1986), Abraira and Derler (1988), Colagiuri *et al.* (1989), Bantle *et al.* (1993), and Tariq *et al.* (2001) did not indicate the exercise pattern of their subjects for the duration of the study periods.

5.5.4 Medication

Both groups maintained their habitual medication usage throughout the trial period. Bantle *et al.* (1983), Abraira and Derler (1988) only included subjects on medical nutritional therapy. Other studies maintained medication usage throughout the study period (Slama *et al.*, 1984; Bantle *et al.*, 1986; Peterson *et al.*, 1986; and Colagiuri *et al.*, 1989).

5.6 SUMMARY

Although no statistically significant changes in blood lipid concentrations occurred after the inclusion of 15% sucrose to the diet of subjects type 2 diabetes mellitus, there was a trend towards increased serum triglyceride concentrations and decreased HDL concentrations after the study period. The fat content (saturated vs. monounsaturated fat) of the diet and the high carbohydrate intake, as a result of the high energy intake of the two groups, may be possible explanations for this. Subjects were not followed up after the short trial period (four weeks), and the long term effects of sucrose on blood lipid concentrations of subjects with type 2 diabetes mellitus needs to be clarified. If the subjects lose weight and increase their monounsaturated fat intake, their blood lipid concentrations will improve automatically.

This study showed that a moderate intake of sucrose did not aggravate glycaemic control of type 2 diabetes mellitus subjects. The compliance and long term glycaemic control of both groups improved, as a result of strict supervision and control applied by the researcher. This study also showed that a moderate intake of sucrose did not aggravate blood lipid concentrations during a short term trial of four weeks. The long term effects of sucrose intake on the blood lipid concentrations of subjects with type 2 diabetes mellitus still needs to be determined, as subjects were not followed up after the four week trial period.

The effect of sucrose intake on the glycaemic control of diabetic subjects is well documented and is included in diabetic diets. The majority of these studies regarding sucrose intake and glycaemic control in diabetics included dietary interventions based on weight maintenance, with dietary control alone, or with oral hypoglycaemic drugs. Exercise and lifestyle practices were not always clearly indicated in previous studies. The effects of these parameters could, therefore, not be compared to previous studies.

Chapter 6 Conclusions and recommendations

6.1 INTRODUCTION

The main aim of this study was to evaluate the effects of sucrose intake on the glycaemic control of type 2 diabetics. Two dietary groups were compared by adding 15% of the total energy intake as sucrose to the diet of one group, and excluding sucrose from the diet of the other group. This was the first study in South Africa to compare the effects of moderate sucrose intake on the glycaemic control of subjects with type 2 diabetes mellitus. Diets for both groups were individualized to maintain each subject's current weight. This was necessary to ensure that improvement in glycaemic control could not be accredited to weight loss.

Significant conclusions can be drawn after an experimental study only if the methods used were reliable and valid. In this study, standardised methods were used throughout the study period, and the researcher applied strict control over the dietetic compliance of all subjects, as discussed in Chapter 3. It can thus be concluded that the results of this study are reliable and valid.

6.2 CONCLUSIONS

The following conclusions are drawn.

- Subjects with type 2 diabetes mellitus can safely include a moderate amount (15% of the total energy) of sucrose in a balanced diet without deleterious effects on their glycaemic control.

- The long term glycaemic control (as measured by HbA_{1c}) improved with good dietary compliance in both diets that included/excluded sucrose. However, the diet excluding sucrose resulted in a statistically significant improvement during long-term glycaemic control.

- No differences in lipid concentrations, within or between Group 1 (SID) and Group 2 (SFD) occurred, suggesting that the moderate intake of sucrose (15% of total energy) had no aggravating effects on blood lipid concentrations of subjects with type 2 diabetes mellitus for a trial period of four weeks. However, the long term effects of sucrose on the blood lipid concentrations could not be assessed. A trend was observed that the exclusion of sucrose from the diet might result in decreased HDL cholesterol concentrations, which might be of clinical significance.

- Obesity is one of the major factors in the etiology of type 2 diabetes mellitus. Obesity aggravates glycaemia of subjects with type 2 diabetes mellitus. All the subjects in this study were classified as class I obese, according to their mean BMI.

- It is exceptionally difficult to find a large study population of subjects with type 2 diabetes mellitus who are willing to comply with all the inclusion criteria of this study. Certain subjects were diagnosed as type 2 diabetics by mistake. Other subjects found the 16 week study period too restrictive, without realising that the benefits inherent in the diet could actually enlarge their dietary choices and improve their glycaemic control on a long-term basis.

- Frequent home visits by the researcher resulted in a relationship of trust between the researcher and the subjects. The subjects were very open about their dietary intake and lifestyle patterns and compliance was good.

6.3 RECOMMENDATIONS

It is recommended that:

- Subjects with type 2 diabetes mellitus can include a moderate amount (15% of the total energy) of sucrose in a balanced diet. This modification may lead to improved adherence to their diets, as it minimizes the sense of deprivation. The inclusion of moderate sucrose in a balanced diet will enhance overall palatability of the diet and might improve long term compliance. Compliance to a balanced diet will improve diabetic control. Furthermore, fewer restrictions in the diet of subjects with type 2 diabetes mellitus may lead to better compliance and reduction in short and long term complications.

- Frequent consultation with a registered dietician is of the utmost importance to ensure that a trusting relationship is maintained and nurtured with each subject to maximize his/her compliance. If subjects with diabetes mellitus trust their dietician, they will be less susceptible to unqualified hearsay, opinions, misinformation and misinterpretations garnered from other less reliable sources and the resultant mythical belief in unscientific information/media.

- More research is needed to determine the long term effects of sucrose on blood lipid concentrations in subjects with type 2 diabetes mellitus.

- Education of subjects with diabetes mellitus concerning medical nutritional therapy remains one of the most important tasks of the registered dietician. Subjects should be educated about the incorporation of sucrose into a healthy eating pattern. Subjects must be able to distinguish between sucrose and a high fat sucrose diet to enable them to choose the lower fat sucrose options.

- Education of health workers including registered nurses, dieticians and general practitioners; as well as the public, on the inclusion of sucrose in the diets of subjects with type 2 diabetes mellitus is of the utmost importance. If health care workers continue to be reluctant to apply the inclusion of sucrose in the type 2 diabetic diet, because of personal prejudice or ignorance regarding the benefits of research such as this, it will create confusion and disbelief among diabetic patients concerning the efficacy of the diet. The colloquial concept of diabetes mellitus being merely a "sugar disease", and the misconception that sucrose causes diabetes mellitus, should be dispelled forthwith.

- Education of susceptible groups (lifestyle risk groups) on the prevention of type 2 diabetes mellitus by improvement of lifestyle is needed. Increased activity (in consultation with a physician and biokinethetist) can improve the glycaemic control of subjects with type 2 diabetes mellitus.

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APPENDIX 1

ADVERTISEMENT

DEPARTEMENT MENSLIKE VOEDING
UNIVERSITEIT VAN DIE VRYSTAAT
Kliniese navorsingstudie

Tipe 2 diabeet?

Is u

- Bereid om 'n voorgeskrewe dieet te volg?
- Bereid en instaat om aan weeklikse monitor sessies deel te neem?
- Tussen 40 en 65 jaar?

Indien u antwoord **JA** op al die bogenoemde vrae is, kan dit u interesseer om te weet dat u benodig word om aan 'n kliniese studie deel te neem.

Deelname aan hierdie studie sal in samewerking met u behandelende geneesheer geskied.

Skakel Elza Hunter by 082 481 4879 vir meer inligting gedurende kantoor ure

DEPARTMENT OF HUMAN NUTRITION
UNIVERSITY OF THE FREE STATE
Clinical research study

Type 2 diabetic?

Are you

- Willing to follow a prescribed diet?
- Willing and able to take part in weekly monitoring sessions?
- Between 40 and 65 years?

If your answer is YES on all the questions, you would be interested to know that we require your help to take part in a clincal study.

Participation will be in collaboration with your consulting doctor.

Contact Elza Hunter at 082 481 4879 during office hours for more information.

APPENDIX 2: INFORMED CONSENT FORM

TOESTEMMING / PERMISSION

Hiermee verklaar ek.....
dat ek my toestemming verleen tot die deelname aan die navorsingsprojek, soos aan my verduidelik.

Ek is tenvolle ingelig deur.....aangaande die waarskynlike voordelige, asook die waarskynlike nadelige gevolge wat die behandeling vir my kan voortspruit.

Die behandeling sal uitgevoer word deur:

- Personeel van die Departement Menslike Voeding.
- Personeel van die Departement Chemiese Patologie

Ek verleen ook my toestemming dat resultate van hierdie navorsingsprojek vir publikasie gebruik mag word, mits my anonimiteit te all tye beskerm word.

My toestemming word uit vrye wil verleen en ek besef ook dat ek my toestemming te enige tyd kan herroep.

* * *

I....., hereby declare
that I give my permission to take part in the research project as explained to me.

.....has made me completely aware of the possible advantages as well as negative results that may occur from the treatment.

The treatment will be performed by:

- Personnel of the Department of Human Nutrition and
- Personnel of the Department of Chemical Pathology

I give my permission that the results of this research project may be used for publication, as long as my anonymity is protected at all times.

My permission has been given out of own free will, and I also realise that my permission may be withdrawn at any time.

* * *

Ka hona ke tsebisa hora nna.....
ke fanna ka tumello ho nkeng karolo mosebetsing wa dipatlisiso jwalo ka ha o hlalositswe ho nna.

Ke tsebitsisi ka ho phethaliala ke .....mabapi le mohlomong thuso, esita le mohlomong thuso, esita le mohlomong ditshenyehelo tse ka bang tshebetsong ena ya ka.

Tshebetso tla phethwa Ke:

Batsamaisi ba tshebetso le barutwana ba selemo sa bona ba Lefapha la Phepo ya Batho le

- Batsamaisi le Lefapha la Chemiese Patologie
- Batsamaisi le Lefapha la Human Nutrition

Ke fana ka tumello horo diphetho tsa dipatlisiso tsena di ka sebedisetswa ho phatlalatswa ha feela ho ka sireletswa ho sa hlahe lebitso la ka ka dinako tsohle.

Ke fana ka tumello ya ka ho rata ha ka mme ke sa boetse ke lemoha hore nka hula tumello ena ya ka ka nako e nngwe le e nngwe.

Geteken te .....op.....(datum)

Signed at.....op.....(date)

E Saennwe.....ka la.....(letsatsi)

.....
RESPONDENT/ RESPONDENT/ MOKUDI

.....
PERSOON WAT DIE RESPONDENT INGELIG HET
PERSON WHO INFORMED THE RESPONDENT
MOTHO YA TSEBISITSENG MOKUDI

.....
PERSOON WAT DIE BEHANDELING UITVOER
PERSON CONDUCTING THE TREATMENT
MOTHO YA ENTSENG MOSEBETSI

APPENDIX 3

QUANTITATIVE FOOD FREQUENCY QUESTIONNAIRE

☐☐ 1-2

FREKWENSIE VRAELYS OM GEBRUIKLIKE VOEDSELINNAME TE MEET

Naam:_____ Geslag: M / V ☐ 3

Adres:_____

_____

Geboortedatum (dd/mm/j): _____ ☐☐☐☐☐☐☐☐ 4-11

Datum van opname:_____ ☐☐☐☐☐☐☐☐ 12-19

Etniese groep ☐

Blank ☐ Swart ☐ Kleurling ☐ Asiër ☐ Ander ☐ 20

BEANTWOORD ASSEBLIEF DIE VOLGENDE VRAE:

1. Gebruik u een van die volgende dieetaanvullings? Onderstreep/spesifiseer:
   Vitamiene [1], tonikums [2] Gesondheidskosse [3] liggaamsbou middels [4]
   dieetvesel [5] gewigsverliesmiddels [6] ander (spesifiseer) [7] ☐☐☐☐☐ 21-27

   _____

2. Sny u die vet van vleis af voor voorbereiding? J/N ☐ 28

3. Beskryf u oefen patroon in tyd (min) per keer
   kere per week
   per maand ☐☐☐☐☐☐☐ 29-35

4. Rook u ? J/N Hoeveel sigarette per dag? ☐☐☐ 36-38

5. Gebruik u alkohol? J/N ☐ 39

   Hoeveel sopies per dag/ per week/ per maand? ☐☐☐☐☐☐ 40-45

6. Gee 'n lys van u medikasie ☐☐☐☐☐☐ 46-51

   _____

   _____

HIERDIE VRAELYS HANDEL OOR DIE VOEDSEL WAT U GEWOONLIK EET.

Dui asseblief die hoeveelhede* en die hoeveelheid kere wat u 'n voedsel eet, aan. As daar mer as een opsie by 'n vraag op u van toepassing is, onderstreep dit wat van toepassing is, en merk die voedsel en sy hoeveelheid met 'n teken, byvoorbeeld: Boontjies #, ertjies $, spruitjies **, sodat die hoeveelhede maklik met die tipe voedsel in verband gebring kan word.

*Hoeveelhede word as volg aangedui (gebruik simbool in hakkies)
Gram (g), koppie (k), teelepel (t), eetlepel (E) milliliters (ml), snye (s), porsies (p).

Die dikte van snye brood is as volg:
Dun: 1cm en minder, Medium: tussen 1 en 2·cm, Dik tussen 2 cm en meer

VOEDSELITEM EN BESKRYWING	Hoev. per keer	Per dag	Per week	Per maand	Nooit/selde											
BROOD/BROODROLLE:																
Volgraan dun/med/dik												3	2	1	2	
Bruin dun/med/dik												3	2	1	1	
Wit dun/med/dik												3	2	1	0	
Brosbrood																
Provita												3	2	3	5	
Matzos												3	2	3	4	
Ander																
PAP: Hawermout												3	2	3	9	
Maltabella												3	4	3	7	
Mieliepap slap												3	3	9	9	
Styf												3	4	0	0	
Putu												3	4	0	1	
GRAANKOS: Pronutro												3	4	3	8	
Rice-Crispies												3	3	2	5	
Muesli												3	3	0	3	
All Bran												3	2	4	2	
Ander:																
Suikerbedekte (bv. Sugar Puffs)																
SUIKER: (saam met pap of graankos gebruik)												3	9	8	9	
GRAAN: Rys, Bruin												3	3	1	5	
Rys, Wit												3	2	4	7	
Koring																
Mielierys																
Stampmielies												3	2	5	0	
Heel mielies												4	1	3	3	

VOEDSELITEM EN BESKRYWING	Hoev. per keer	Per dag	Per week	Per maand	Nooit/selde											
Stampmielies en bone (gee die verhouding)													3	4	0	2
PASTA: Noem die tipe, asook gereg bv. Noedels																
Spaghetti bolognaise													3	2	6	0
Macaroni an kaas													3	3	0	1
Ander																
KOEK EN TERT:																
Soet skons													3	3	1	9
Pannekoek													3	3	4	4
Vetkoek													3	3	2	4
Laagkoek													3	2	8	7
Terte																
Ander																
Sout: Worsrolletjies																
Samosas Groente 3414 Lam 3355																
Ander																
BESKUIT: (noem tipe)																
SKYFIES: Aartappelskyfies													8	0	4	9
Springmielies													3	3	3	2
Ander																
NAGEREG: jellies en waterbasis nageregte													3	9	8	3
Melkbasis bv. Kitspoedings																
Vla													2	7	2	4
Gebakte/gestoomde poedings																
LEKKERS: Suiglekkers (noem soort)																
Toffies 'fudge' (noem soort)																
Sjokolade (stafies en blokke) (noem soort)																
KONFYT													3	9	8	5
HEUNING													3	9	8	4
STROOP (noem soort)													3	9	8	8
BROODSMEER: grondboontjiebotter,													3	4	8	5
Marmite													4	0	3	0
Vissmeer, ens													3	1	0	9
MELK: As 'n drank - Vars, Vol													2	7	1	8
Suur, Dik													2	7	8	7
Afgeroom													2	7	7	5
Ander:																

VOEDSELITEM EN BESKRYWING	Hoev. per keer	Per dag	Per week	Per maand	Nooit/selde											
MELKDRANKIE: Milo												0	0	2	3
Melkskommels												2	7	8	8
Nesquick												2	8	3	0
Ander															
MELK: (by tee of koffie, pap of ontbytkosse)															
SUIKER: (bygevoeg by tee/koffie)												3	9	8	9
POEIERMELK: Volroom												2	8	3	1
Afgeroom												2	7	1	8
MENGSELS VAN VARS EN POEIERMELK (noem verhouding)												2	7	7	1
INGEDAMPTE OF KONDENSMELK												2	7	1	4
NIE-SUIWEL KOFFIEVERROMERS: bv. 'Cremora'												2	7	5	1
KAAS: noem tipe bv. Cheddar												2	7	7	2
Soetmelk												2	7	2	3
Ander															
MAASKAAS/GEROOMDE KAAS: lae vet												2	7	2	9
Gewoon												2	7	5	9
ROOM: Vars												3	4	8	0
'orley whip'												3	4	9	2
ROOMYS: (noem soort)															
EIERS: Gekook,												2	8	6	7
Geposjeer												2	8	6	7
Gebak (vet of olie)												2	8	6	9
Omelet												2	8	7	1
Roereiers												2	8	8	9
Soufflès												2	8	9	9
Ander															
VLEIS: HOEVEEL KEER PER WEEK EET U VLEIS?															
ROOIVLEIS: spesifiseer gerooster, gebak, gebraai															
Gebraai; dit sluit gebraaide vleis in)															
Beesvleis (met vet / sonder vet)															
Skaapvleis (met vet 2947/ sonder vet)															
Varkvleis (met vet / sonder vet)															
Wors (spesifiseer soort)												2	9	3	1
PLUIMVEE: Spesifiseer gerooster, gebak, gekook, gebraai												2	9	2	5

VOEDSELITEM EN BESKRYWING	Hoev.per keer	Per dag	Per week	Per maand	Nooit/selde											
ORGAANVLEIS: Afval												3	0	0	3
Lewer, Bees 2920 Lam2955 Hoender 2970															
Niertjies, Bees 2923 Skaap 2956															
Ander															
KOUE EN GEPROSESEERDE VLEIS															
VLEISPASTEIE EN BREDIES (spesifiseer soort)															
SOUSE EN VLEISEKSTRAK (spesifiseer soort)															
SOUSE: Tamatiesous												3	1	3	9
Atjar												3	1	1	7
Bruinsous												3	1	2	2
Ander															
VIS: bv. Haring												3	0	8	2
Tuna (in water)												3	0	5	4
Visvingers												3	0	8	1
Ander															
Spesifiseer vars, gevries/geblik, gerooster, gebak gestoom															
SKULPVIS: (spesifiseer)															
GEDROOGDE PEULGROENTES: Bone												3	1	8	5
Erte												4	1	4	8
Lensies (heel)												3	2	0	3
NEUTE: (spesifiseer)															
PLANTPROTEïENPRODUKTE: Toppers, TVP, Sossies												3	1	9	6
Ander															
SOP: water - of melkbasis															
Geblik, Pakkies															
Tuisgemaak - Noem die soort bv. Ertjiesop															
GROENTE: Spesifiseer vars, geblik, gevries															
HOEVEEL PORSIES GROENTE EET U PER DAG?															
Rou slaaigroentes bv. Tamatie,												3	7	5	0
Kropslaai												3	7	2	3
Komkommer												3	7	1	8
Ander															
Gekookte slaaigroentes bv. beet												3	6	9	8

VOEDSELITEM EN BESKRYWING	Hoev.per keer	Per dag	Per week	Per maand	Nooit/selde											
Aspersies (geblik)													4	0	9	4
Ander																
Groen blaargroentes bv. spinasie ens.													3	7	8	6
Groen groentes soos ertjie													4	1	4	9
Groenbone (Aart+ Ui+ geen vet)													3	9	3	3
Ander																
Uie/tamaties (gekook)													3	9	2	5
Geelgroentes, bv. pampoen(soet/sout)													4	1	6	4
Wortels													3	8	1	8
Ander																
Wit groentes, bv. blomkool													3	7	1	6
Radyse													3	7	4	5
Ander																
Groenmielies													3	7	2	5
Gemengde groente (soet / sout)																
Eiervrug													3	7	0	0
Sampioene (gebraai in sonneblomolie)													3	8	4	1
Aartappels- gekook													4	1	5	5
Gebak													3	7	3	6
Fyn													3	8	7	5
Aartappelslaai													3	9	2	8
Witsous 3141/ kaassous (3128) by groente																
VRUGTE: Vars, geblik, gedroog																
HOEVEEL PORSIES VRUGTE EET U PER DAG?																
Appels en													3	5	3	2
pere													3	5	8	2
Steenvrugte - perskes													3	5	6	5
Pruime													4	2	3	0
Ander																
Sitrusvrugte bv. Lemoen													3	5	6	0
Piesangs													3	5	4	0
Koejawels													3	5	5	1
Ander (spesifiseer)																
Avokado's of													3	6	5	6
Olywe													3	6	5	8
Bessievrugte (geblik)													3	5	8	4
Gestoofde of geblikte vrugte (met / sonder suiker)													3	5	8	0
Vrugterolle bv. Guava of													3	6	9	4
Verglansde vrugte																
VETTE EN OLIES: Op brood, in slaaie, groente, ens.																
Botter bv. Butro													3	5	2	3
Margarien: (sag 3521/ hard 3484)																

VOEDSELITEM EN BESKRYWING	Hoev.per keer	Per dag	Per week	Per maand	Nooit/selde				
Vet						3	5	1	6
Olie (Sonneblom 3507, Olyf 3509, Canola									
Kaiings									
Ander									
Mayonnaise						3	4	8	8
Slaaisous						3	4	8	7
Ander									
DRANKE:									
Koffie						4	0	3	7
Tee						4	0	3	8
Suiker gebruik per koppie						3	9	8	9
Vrugtesap (vars, 'liquifruit', ens., noem soort, bv. appel)						3	6	0	6
Ander: koeldranke bv. Oros, ens.						3	9	8	2
Gaskoeldranke ('Coca Cola', Lemon Twist', ens.)						3	9	8	1
Maghou						4	0	5	6
ALKOHOLIESE DRANKE									
Bantoebier (Tlokwe ens.)						4	0	5	6
Bier						4	0	3	1
Sherrie, Vermoet (4043), Portwyn, ens. (soet(4032), medium, droog)									
Tafelwyn (soet, medium of droog)						4	0	3	3
Spiritualieë, bv. brandewyn, whisky, jenewer, ens.						4	0	3	4
Likeur (noem soort)									
Mengeldrankies (spesifiseer)									

OPSOMMING	Hoeveelheid per dag	Kode				
						52-56
						5761
						62-66
						67-71
						72-76
						1-5
						6-10
						11-15
						16-20
						21-25
						26-30
						31-35
						36-40
						41-45
						46-50
						51-55
						56-60
						61-65
						66-70
						71-75
						76-80
						1-5
						6-10
						11-15
						16-20
						21-25
						26-30
						31-35
						36-40
						41-45
						46-50
						51-55
						56-60
						61-65
						66-70
						71-75
						76-80
						1-5
						6-10
						11-15
						16-20
						21-25
						26-30
						31-35
						36-40
						41-45
						46-50

APPENDIX 4: Screening for type 2 diabetes mellitus

1-2

Subject name:_____

Datum:

3-10

Geboortedatum:

11-18

Physical address:_____

_____Tel nr:_____

Consulting doctor:_____ Tel nr:_____

Physical address:_____

_____

Surveyor: _____

Type 2 diabetic criteria	**Value**		**Accept Y/N**
> 40yrs	Age:		
Family history of type 2 diabetes mellitus	Y/N:		
No ketones in their urine test	Y/N		
C-peptide values > 265pmol/l			
Negative GAD 65 antibody value (reference range: 0 – 1 U/L)			
Inclusion criteria	**YES**	**NO**	**SUITABLE**
Subjects with type 2 diabetes mellitus			
Volunteer to comply to a a prescribed diet			
Volunteer to attend monitoring sessions fortnightly			
Between 40 and 65 years			
BMI > 18,5 kg/m^2			
Exclusion criteria	**YES**	**NO**	**SUITABLE**
Type 1 diabetes mellitus			
Pregnant			
Want to lose weight			
Chronic disease that requires special dietary intervention as this would deviate from the dietary prescription for the study group			
Use medication that influence glucose tolerance like oral glukocorticoids, β blockers, tiazide diuretics in high dosages and anti epileptics drugs like epanutin			

APPENDIX 5 : Schedule for blood measurements

Patient name: _____ Tel no:_____

Physical adress:_____

_____

_____

Name of Qualified nurse: _____

Week	Measurement	Volume of blood(ml)	Color of lid						
Baseline Date:	Weight (kg)						•		3-7
	Height (cm)						•		8-12
	Bodyfat percentage (%)					•			13-17
	Serum cholesterol (mmol/L)	2				•			18-22
	Serum HDL-cholesterol (mmol/L)					•			23-27
	Serum LDL-cholesterol (mmol/L)					•			28-32
	Serum Triglycerides (mmol/L)					•			33-37
	HbA1C	3							38-42
	GAD antibody- (U/L)	3				•			43-47
	Plasma glucose (mmol/L)	2					•		48-52
	Serum fructosamine (µmol/L)	2					•		53-57
Week 1 Date:	Weight (kg)						•		58-62
	Plasma glucose (mmol/L)	2					•		63-67
	Serum fructosamine (µmol/L)	2					•		68-72
Week 2 Date:	Weight (kg)						•		73-77
	Plasma glucose (mmol/L)	2					•		1-5
Week 3 Date:	Weight (kg)						•		6-10
	Plasma glucose (mmol/L)	2					•		11-15
	Serum fructosamine (µmol/L)	2					•		16-20
Week 4 Date:	Weight (kg)						•		21-25
	Plasma glucose (mmol/L)	2					•		26-30
Week 5 Date:	Weight (kg)						•		31-35
	Plasma glucose (mmol/L)	2					•		36-40
	Serum fructosamine (µmol/L)	2					•		41-45

Week	Measurement	Volume of blood(ml)	Color of lid						
Week 6 Date:	Weight (kg)						•		46-50
	Plasma glucose (mmol/L)	2					•		51-55
Week 7 Date:	Weight (kg)						•		56-60
	Plasma glucose (mmol/L)	2					•		61-65
	Serum fructosamine (µmol/L)	2					•		66-70
Week 8 Date:	Weight (kg)						•		71-75
	Plasma glucose (mmol/L)	2					•		76-80
Week 9 Date:	Weight (kg)						•		1-5
	Plasma glucose (mmol/L)	2					•		6-10
	Serum fructosamine (µmol/L)	2					•		11-15
Week 10 Date:	Weight (kg)						•		16-20
	Plasma glucose (mmol/L)	2					•		21-25
Week 11 Date:	Weight (kg)						•		26-30
	Plasma glucose (mmol/L)	2					•		31-35
	Serum fructosamine (µmol/L)	2					•		36-40
Week 12 Date:	Weight (kg)						•		41-45
	Plasma glucose (mmol/L)	2					•		46-50
Week 13 Date:	Weight (kg)						•		51-55
	Plasma glucose (mmol/L)	2					•		56-60
	Serum fructosamine (µmol/L)	2					•		61-65

Week	Measurement	Volume of blood(ml)	Color of lid						
Week 14 Date:	Weight (kg)						•		66-70
	Plasma glucose (mmol/L)	2					•		71-75
	Food record								
Week 15 Date:	Weight (kg)						•		76-80
	Plasma glucose (mmol/L)	2					•		1-5
	Serum fructosamine (µmol/L)	2					•		6-10
	Dietary information session 8								
	Food record								
Week 16 Date:	Weight (kg)						•		11-15
	Plasma glucose (mmol/L)	2					•		16-20
	Food record								
End Study Date:	HBA1C	3							21-25
	Serum cholesterol (mmol/L)	2				•			26-30
	Serum HDL-cholesterol (mmol/L)					•			31-35
	Serum LDL-cholesterol (mmol/L)					•			36-40
	Serum Triglycerides (mmol/L)					•			41-45

APPENDIX 6 : Schedule for measurements: Lifestyle and medication report

1-2

Patient name: _____ Tel no:_____

Physical adress:_____

_____

_____

Name of Qualified nurse: _____

Week	Measurement		
Baseline Date:	Bodyfat percentage%		3-6
	Height (cm)		7-12
Week 1 Date:	Exercise changes – Same Y/N		13
	More Time (min), frequency (/week,/month)		14-20
	LessTime (min), frequency (/week,/month		21-27
	Medication changes Y/N		28
	Specify		29-30
	Do you smoke? Y/N		*31*
	How many sigarettes per day?		*32-33*
	Do you use alcohol? Y/N		34
	How many servings /day/week/month?		35-41
Week 2 Date:	Exercise changes – Same Y/N		42
	More Time (min), frequency (/week,/month)		43-49
	LessTime (min), frequency (/week,/month		50-56
	Medication changes Y/N		57
	Specify		58-59
	Do you smoke? Y/N		*60*
	How many sigarettes per day?		*61-62*
	Do you use alcohol? Y/N		63
	How many servings /day/week/month?		64-69

Week 3 Date:	Exercise changes – Same Y/N		70
	More Time (min), frequency (/week,/month)		71-77
	LessTime (min), frequency (/week,/month		1-7
	Medication changes Y/N		8
	Specify		9-10
	Do you smoke? Y/N		*11*
	How many sigarettes per day?		*12-13*
	Do you use alcohol? Y/N		14
	How many servings /day/week/month?		15-20
Week 4 Date:	Exercise changes – Same Y/N		21
	More Time (min), frequency (/week,/month)		22-28
	LessTime (min), frequency (/week,/month		29-35
	Medication changes Y/N		36
	Specify		37-38
	Do you smoke? Y/N		*39*
	How many sigarettes per day?		*40-41*
	Do you use alcohol? Y/N		42
	How many servings /day/week/month?		43-48
Week 5 Date:	Exercise changes – Same Y/N		49
	More Time (min), frequency (/week,/month)		50-57
	LessTime (min), frequency (/week,/month		58-64
	Medication changes Y/N		65
	Specify		66-67
	Do you smoke? Y/N		*68*
	How many sigarettes per day?		*69-70*
	Do you use alcohol? Y/N		71
	How many servings /day/week/month?		72-77

Week 6 Date:	Exercise changes – Same Y/N		78
	More Time (min), frequency (/week,/month)		1-7
	LessTime (min), frequency (/week,/month		8-14
	Medication changes Y/N		15
	Specify		16-17
	Do you smoke? Y/N		*18*
	How many sigarettes per day?		*19-20*
	Do you use alcohol? Y/N		21
	How many servings /day/week/month?		22-27
Week 7 Date:	Exercise changes – Same Y/N		28
	More Time (min), frequency (/week,/month)		29-35
	LessTime (min), frequency (/week,/month		36-42
	Medication changes Y/N		43
	Specify		44-45
	Do you smoke? Y/N		*46*
	How many sigarettes per day?		*47-48*
	Do you use alcohol? Y/N		49
	How many servings /day/week/month?		50-55
Week 8 Date:	Exercise changes – Same Y/N		56
	More Time (min), frequency (/week,/month)		57-63
	LessTime (min), frequency (/week,/month		64-70
	Medication changes Y/N		71
	Specify		72-73
	Do you smoke? Y/N		*74*
	How many sigarettes per day?		*75-76*
	Do you use alcohol? Y/N		77
	How many servings day/week/month?		1-6

Week 9 Date:	Exercise changes – Same Y/N	7
	More Time (min), frequency (/week,/month)	8-14
	LessTime (min), frequency (/week,/month	15-21
	Medication changes Y/N	22
	Specify	23-24
	Do you smoke? Y/N	*25*
	How many sigarettes per day?	*26-27*
	Do you use alcohol? Y/N	28
	How many servings /day/week/month?	29-34
Week 10 Date:	Exercise changes – Same Y/N	35
	More Time (min), frequency (/week,/month)	36-42
	LessTime (min), frequency (/week,/month	43-49
	Medication changes Y/N	50
	Specify	51-52
	Do you smoke? Y/N	*53*
	How many sigarettes per day?	*54-55*
	Do you use alcohol? Y/N	56
	How many servings /day/week/month?	57-62
Week 11 Date:	Exercise changes – Same Y/N	63
	More Time (min), frequency (/week,/month)	64-70
	LessTime (min), frequency (/week,/month	71-77
	Medication changes Y/N	78
	Specify	79-80
	Do you smoke? Y/N	*1*
	How many sigarettes per day?	*2-3*
	Do you use alcohol? Y/N	4
	How many servings /day/week/month?	5-10

Week 12 Date:	Exercise changes – Same Y/N		11
	More Time (min), frequency (/week,/month)		12-18
	LessTime (min), frequency (/week,/month		19-25
	Medication changes Y/N		26
	Specify		27-28
	Do you smoke? Y/N		*29*
	How many sigarettes per day?		*30-31*
	Do you use alcohol? Y/N		32
	How many servings /day/week/month?		33-38
Week 13 Date:	Exercise changes – Same Y/N		39
	More Time (min), frequency (/week,/month)		40-46
	LessTime (min), frequency (/week,/month		47-53
	Medication changes Y/N		54
	Specify		55-56
	Do you smoke? Y/N		*57*
	How many sigarettes per day?		*58-59*
	Do you use alcohol? Y/N		60
	How many servings /day/week/month?		61-66
Week 14 Date:	Exercise changes – Same Y/N		67
	More Time (min), frequency (/week,/month)		68-74
	LessTime (min), frequency (/week,/month		1-7
	Medication changes Y/N		8
	Specify		9-10
	Do you smoke? Y/N		11
	How many sigarettes per day?		12-13
	Do you use alcohol? Y/N		14
	How many servings /day/week/month?		15-20

Week 15 Date:	Exercise changes – Same Y/N		21
	More Time (min), frequency (/week,/month)		22-28
	LessTime (min), frequency (/week,/month		29-35
	Medication changes Y/N		36
	Specify		37-38
	Do you smoke? Y/N		*39*
	How many sigarettes per day?		*40-41*
	Do you use alcohol? Y/N		42
	How many servings /day/week/month?		43-48
Week 16 Date:	Exercise changes – Same Y/N		49
	More Time (min), frequency (/week,/month)		50-56
	LessTime (min), frequency (/week,/month		57-63
	Medication changes Y/N		64
	Specify		65-66
	Do you smoke? Y/N		*67*
	How many sigarettes per day?		*68-69*
	Do you use alcohol? Y/N		70
	How many servings /day/week/month?		71-76
End study Date:	Bodyfat percentage (%)	.	77

APPENDIX 7: SHORT INFORMATIVE TALK SESSIONS

SESSIE 1 WAT IS DIABETES ?

DEFINISIE VAN TIPE 2 DIABETES MELLITUS

Diabetes is 'n toestand waar die liggaam nie instaat is om voedsel af te breek (verteer) en te gebruik (metaboliseer) soos dit moet nie. Die liggaam gebruik nie die insulien tot sy beskikking, optimaal nie.

HOE WERK DIT ?

By persone wat nie diabetes het nie, word voedsel afgebreek om voedingstowwe vry te stel in die spysverteringskanaal. Alle voedsel bestaan uit voedingstowwe. Hierdie voedingstowwe werk saam om die liggaam gesond en lewendig te hou.

Koolhidraatryke (styselryke) voedsel verskaf energie aan die liggaam. Dit word afgebreek na suiker molekules. Daarna word dit in die bloed opgeneem as bloedsuiker wat ook bloedglukose genoem word. Die hart pomp bloed na elke deel van die liggaam. So word die voedingstowwe deur die bloed vervoer tot by die verskillende selle waar dit benodig word. Elke sel het wande wat beheer uitoefen op dit wat die sel binnegaan of verlaat. Bloedsuiker word deur die selle gebruik vir energie. Wanneer dit nodig is, maak die pankreas ('n orgaan naby die maag) 'n hormoon genaamd insulien.

Insulien word na die bloedstroom gestuur om die selle te help om die bloed glukose op te neem in die sel. Sodra insulien sy werk gedoen het, word dit afgebreek. Die liggaam maak elke keer nuwe insulien soos dit nodig raak. Die oppervlakte van die selle het spesiale plekke waar insulien met die sel kan heg. Sodra insulien geheg het aan die sel, maak die sel oop soos 'n sleutel wat 'n deur oopsluit. Die bloedsuiker kan dan, vanaf die bloedstroom, die sel binne gaan. As 'n groot hoeveelheid voedsel ingeneem word, word 'n groot hoeveelheid insulien gestuur om die selle te help om al die bloedsuiker op te neem.

Bloedsuiker word vinnig omgesit in energie wat deur die selle gebruik kan word. Energie word benodig vir alle aktiwiteite soos byvoorbeeld loop, slaap, dink, werk en praat. Wanneer ernstige oefening of baie werk gedoen word, benodig die liggaam ekstra energie aangesien die selle meer bloedsuiker verskaf om vir die liggaam energie te kan gee. Sekere selle soos die brein en senuwee selle moet 'n gereeld voorsien word van energie in die vorm van bloedsuiker, om hul werk te doen.

Ekstra bloedsuiker word gestoor as 'n reserwe bron in die lewer, as glikogeen, of in die vetweefsel. Dit word vrygestel as die bloedsuiker te laag word, byvoorbeld as 'n persoon lank laas geëet het.

Afvalstowwe word terug gestuur na die bloedstroom. Soos bloed deur die niere gaan, word die afvalstowwe daar gefiltreer. Die afvalstowwe word dan uitgeskei uit die liggaam met uriene.

Die persoon met diabetes mellitus

Indien 'n persoon diabetes het, is daar nie genoeg insulien om die sel se deure oop te maak nie. Die selle kan daarom die bloedsuiker opneem nie. Die selle bou weerstand op teen insulien en dit kan nie heg aan die sel se wande nie. Die selle kan dan ook nie bloedsuiker omskakel in energie nie. Die bloedsuiker bly dan in die bloedstroom waar dit opbou. Sodra daar te veel bloedsuiker in die bloed is, word dit in die uriene uitgeskei. Hierdie suiker is nie net afkomstig van suikerbevattende voedsel soos tafelsuiker, konfyt of ander suikerdigte voedsel nie. Dit is die suiker wat afgebreek is van alle koolhidraatryke voedsels. Die bloedsuiker vlakke verhoog dan tot bokant normaal. Indien diabetes nie behandel word nie of buite kontrole is, is die bloedsuiker baie hoog. Suiker kan ook in 'n persoon met diabetes se uriene gevind word.

Die term diabetes mellitus beteken:

Diabetes - " om deur te loop"
Mellitus - " soet soos heuning" (bv. baie soet uriene)

Die selle benodig energie om te funksioneer. Indien hulle nie bloedsuiker kan kry om om te skakel na energie nie, word die liggaam se vetstore gebruik vir energie. Wanneer liggaamsvet afgebreek word vir energie, word ketoon liggaampies gevorm. Die selle kan hierdie ketone nie vinnig genoeg gebruik nie. Die ketone bou dan op in die bloed. Die ketone word uitgeskei uit die liggaam deur die longe en dit veroorsaak dan 'n "vrugte reuk" in die asem en uriene. Indien dit nie behandel word nie, kan dit lei tot moontlike dehidrasie, nier en hartversaking.

DIAGNOSE

Diabetes word gewoonlik ontdek sodra 'n persoon sleg voel. Indien diabetes vermoed word, word die persoon se uriene getoets vir bloedsuiker (glukose). Indien die uriene toets positief is, sal 'n klein hoeveelheid van 'n persoon se bloed getoets word. As die bloedtoets daarop wys dat daar 'n hoë hoeveelheid bloedsuiker (meer as 7 mmol) teenwoordig is as die persoon nie geëet het nie, is dit 'n aanduiding dat die persoon diabetes het. Die uriene en bloed kan ook vir ketone getoets word.

SIMPTOME

Die simptome van ongekontrolleerde diabetes is:

- Verhoogde aptyt
- Verhoogde frekwensie van urinering
- Verhoogde dors
- Onverklaarbare gewigsverlies
- Swakheid

OORSAKE

- Ouderdom - persone is gewoonlik bo 40 jaar
- Familie geskiedenis van diabetes
- Obesiteit - 80% van nuwe diabete is oorgewig
- Fisiologiese en sielkundige stres

KOMPLIKASIES

Die komplikasies van diabetes mellitus kan in twee kategoriëe ingedeel word:

- Akuut
- Chronies

Akute komplikasies:
Dit hou direk verband met die vinnige veranderinge in metabolisme soos baie hoë bloedsuiker vlakke by diagnose en /of 'n baie lae bloedsuiker vlak (hipoglukemiese reaksie). Hierdie komplikasies kan voorkom word en benodig onmiddelike intervensie.

Chroniese komplikasies:
Hierdie komplikasies sluit vaksulêre (hart) siekte, nefropatie (nier siekte), neuropatie (gewoonlik by voetsenuwee) en retinopatie (oog siekte) in. Die komplikasie kan verminder word deur terapie wat bloedsuiker vlakke naby aan die normale vlakke hou.

Die dieetkundige speel 'n belangrike rol in die voorkoming van kort termyn komplikasies en die uitstel van langtermyn komplikasies.

Wees altyd bedag om tekens van 'n hipoglukemiese (lae bloedsuiker) reaksie:

- Angstig
- Bewerig
- Bleek
- Deurmekaar
- Geïrriteerd
- Hardkoppig
- Hartseer
- Honger
- Kwaad
- Lighoofdig
- Lomp
- Moeg
- Naar
- Ongeduldig
- Senuweeagtig
- Slaperig
- Sweterig
- Swak

Algemene behandeling van 'n lae bloedsuiker (hipoglukemie)

Lae bloedsuike is maklik om te behandel. As dit laag is, maar nie dadelik getoets kan word nie, moet suikerbevattende voedsel geëet word. Indien daar twyfel is, is dit veiliger om die ekstra voedsel te eet as om 'n lae bloedsuiker reaksie te hê.

Indien die persoon nie instaat is om te sluk nie, moenie voedsel of vloeistof forseer nie

aangesien daar 'n risiko is dat die persoon kan verstik of die voedsel of vloeistof kan inasem.

Die volgende behandelings bevat 15g koolhidrate

- ❑ 3-4 glukose tablette
- ❑ ½ koppie koeldrank (nie suikervry)
- ❑ 4-5 klein harde suiker lekkers (gekou)
- ❑ ½ koppie vrugtesap (appel of lemoensap)
- ❑ 1 koppie afgeroomde melk
- ❑ 1 teelepel suiker

Herhaal elke 10-15 minute totdat simptome van lae bloedsuiker weg is.

SESSIE 2 SIMPTOME VAN HIPOGLUKEMIE EN HIPERGLUKEMIE

HIPOGLUKEMIE (lae bloedsuiker)	HIPERGLUKEMIE (hoë bloedsuiker)
SIMPTOME	**SIMPTOME**
Sweet Honger Dubbel visie Ongeduldig Bewerig Hoofpyn Flou gevoel	Koud en droë vel Dors Droë mond Naarheid en braking Blosende gelaat Hoofpyn Pyn in rug en bene Maagpyn Slaperig Dronk gevoel Erge swakheid
OORSAKE	**OORSAKE**
Oormatige insulin Oormatige oefening Uitstel van 'n ete Oorslaan van 'n ete	Tekort aan insulien Besering of infeksie Chirurgie Emosionele ontsteltenis
BEHANDELING	**BEHANDELING**
Gee halwe koppie vrugtesap OF een eetlepel heuning OF drie Super C lekkers OF 4 teelepels suiker Eet so gou moontlik daarna 'n koolhidraatryke ete.	Vloeistowwe Insulien

SESSIE 3 - MAALTYDVERSPREIDING, KOOLHIDRATE EN VESEL

MAALTYDVERSPREIDING

Dit is nie nodig vir 'n persoon met diabetes om verskillend te eet van die res van die gesin nie. Hulle moet eerder die voorbeeld stel vir die gesin om hul eetgewoontes te verbeter na 'n gesonde eetplan.

Die volgende moet onthou word met maaltydbeplanning:

- **3-6 Gereelde maaltye moet geëet word**
  Die maaltye moet
  - ewe groot
  - ewe ver uitmekaar
  - en op dieselfde tye van elke dag, geëet word.
- **'n Hoë vesel koolhidraatryke voedsel moet die basis van elke maaltyd vorm.**
- **Groente en vrugte moet by die koolhidraatryke voedsel gevoeg word.**
  Ten minste 5 porsies vrugte en groente moet per dag ingeneem word. Vette en olies moet nie bygevoeg word tydens die voorbereiding daarvan nie.
- **'n Klein, lae vet porsie proteïenryke voedsel kan by die maaltyd gevoeg word.**
  Probeer om 1-2 porsies per dag in te neem. Plant proteïen behoort ten minste 2 maal per week ingesluit te word. Gebruik so min moontlik vet in die voorbereiding daarvan.
- **Laataandvoedings behoort 'n klein hoeveelheid proteïen te bevat.**
  Dit laat die voedsel "langer hou" en voorkom dat die bloedsuiker vlakke te laag daal gedurende die nag.

  Goeie keuses vir laataandvoedings is bv.:
  Volgraan brood of beskuitjies met grondboontjiebotter of lae vet kaas
  Joghurt, melk of suurmelk
  Stamp en bone
  Oorskietkos van die hoofmaaltyd

KOOLHIDRATE

50-65% van die daaglikse energie moet as koolhidrate ingeneem word. Dit moet vanaf verskeie bronne ingeneem word. Koolhidrate moet die meeste van die energie in die dieet verskaf. Verhoog die hoeveelheid koolhidrate in maalltye en verlaag die inname van voedsel hoog in vet. Bronne van koolhidrate is brood, gekookte hawermoutpap, koring, hawer, semels, springmielies, kraakbeskuitjies, soutbeskuitjies, makaroni, spaghetti, noedels, rys en aartappels.

VESEL

Oplosbare vesel vorm 'n jel in water. 'n Voorbeeld is onder andere peule. Onoplosbare vesel kom voor in koringsemels en die eetbare skille en pitte van groente en vrugte. Voedsel hoog in oplosbare vesel soos hawer semels, peule, ontbytgrane, vrugte en groente moet die grootste deel van die totale vesel inname wees. Voedsel wat in die onverfynde vorm geëet word, verskaf die meeste vesel. Vesel moet geleidelik verhoog word in die dieet en vergesel word van genoegsame vloeistof inname. 'n Hoë vesel inname kan spysverterings ongemak by ouer persone met diabetes veroorsaak en moet aandag geniet.

Vesel het verskeie funksies:

- Dit voorkom spysverteringsprobleme soos hardlywigheid.
- Dit help met gewigsbeheer aangesien dit versadiging verhoog.
- Dit help beheer bloedsuiker en lipied (vet) vlakke deur die tempo waarteen hierdie substanse opgeneem word, te verlaag.

SESSIE 4 VET

VET

Een van die belangrikste riglyne vir 'n diabetiese dieet is dat die vet inname nie die aanbevole hoeveelheid moet oorskrei nie. Deur vet te beheer, word liggaamsmassa beheer en die risiko vir koronêre hartsiektes word verlaag.

'n Maksimum van een derde van die vet ingeneem per dag, kan van dierlike oorsprong wees (Versadigde vette).
'n Maksimum van een derde en verkieslik minder, kan afkomstig wees vanaf sonneblom olie en die meeste sagte margarien (Poli-onversadigde vette).
Die balans moet van mono-onversadigde vette kom soos olyf olie, canola olie en margarien, grondboontjiebotter en avokado.

'n Vermindering in versadigde vetsure word geassosieer met 'n verlaagde inname van dieetcholesterol.

Wenke om die tipe vet te varieer:

- Eet klein vleisporsies en probeer om vis (insluitende geblikte vis soos pilchards of tuna) en hoender meer gereeld te eet.
- Verminder die inname van dierlike vette soos botter, room en vet en verwyder alle sigbare vet van vleis voordat die gaarmaakproses begin word.
- Verminder die inname van sekere groente vette wat hoog is in versadigde vet soos klapper olie (gebruik in koffie verromers) en palm olie.
- Probeer om nie olie weer te gebruik nie en vermy oorverhitting van olie aangesien dit die chemiese struktuur verander en dit minder gesond maak.
- Indien vet bygevoeg word, varieer die tipe bv. as poli onversadigde margarien gebruik word as 'n smeer, behoort 'n mono onversadigde vet soos olyf of canola olie gebruik te word vir kookdoeleindes. Grondboontjiebotter of avokado kan ook as 'n bron van mono-onversadigde vet gebruik word.
- Persone met hoë cholesterol behoort die volgende voedsel matig te gebruik: orgaanvleis soos lewer, niertjies en brein, skulpvis soos garnale, kalamari en kreef. Beperk eiergele tot 3-4 per week, verkieslik nie meer as een op 'n keer nie (eierwit kan vrylik gebruik word.)

Wenke om die totale hoeveelheid dieetvet te verlaag:

- Beperk voedsel met 'n hoë vetinhoud - dit sluit bygevoegde vet soos volroom kase, volroommelk, geprosseseerde vleis, pasteitjies, koekies en koeke wat 'n botter versiersel het. Vetterige voedsel proe en lyk nie altyd vetterig nie. Beperk gebraaide voedsel soos aartappelskyfies, gebraaide vis en hoender, en vetkoek.
- Baie geriefsvoedsel het 'n hoë vetinhoud (pasteie, droë aartappelskyfies en neute). Moet dit nie gereeld eet nie.
- Kies lae vet gaarmaak metodes. Rooster, kook, bak of stoom voedsel eerder as om dit in olie of ander vette te braai.
- Kook maer vleissnitte en verwyder alle sigbare vet, en die vel van die hoender, voor dit gaargemaak word.
- Gebruik eerder kruie en speserye, suurlemoensap of krummels vir geur by groente, in plaas daarvan om margarien, botter of olie by groente te sit.
- Gebruik lae vet melkprodukte soos lae vet of afgeroomde melk, suurmelk, maas of joghurt gemaak van lae vet of afgeroomde melk; lae vet kase soos maaskaas in plaas van die volroom soorte. Gebruik lae vet afgeroomde melksoorte in plaas van koffie verromers aangesien laasgenoemde gemaak is van klapper olie en hoog is in versadigde vet. Kyk uit vir die egte suiwel merk op suiwelprodukte om seker te maak

dat dit slegs van melk gemaak is en nie mengsels van melk en koffie verromers is nie.

- Gebruik lae vet slaaisouse soos lae vet mayonnaise, of "geen olie" souse.
- Gebruik groente aftreksel en verdik met meel om sous van te maak in die plek van die vet wat oorgebly het in die pan of kastrol.
- Rooster voedsel op 'n rakkie om die vet te laat afdrup.
- Indien bredies of kerrie gemaak word, maak dit vooraf en laat dit afkoel. Die vet vorm 'n soliede lagie bo-op en kan dan maklik afgeskep word.
- Smeer margarien en ander smere baie dun, of beter nog, gebruik geen.
- Kyk na die verhouding van voedsel tot mekaar op jou bord. Proteïen behoort die kleinste porsie op die bord te wees met groente en stysel die grootste porsies. Bone en lensies is ryk in proteïen en koolhidrate en is 'n goeie keuse as proteïen bron.

SESSIE 5 GAARMAAK EN UITEETWENKE

WENKE VIR VOEDSELVOORBEREIDING

- Koop die maerste snitte vleis. Beperk geprosesseerde vleis, salami en spek.
- Smeer voedsel met suurlemoensap, joghurt, wyn of aftreksel terwyl dit gerooster word om uitdroging te voorkom.
- Eet ten minste twee vegetariese disse per week.
- Maak gebruik van kruie, speserye en ander geurmiddels om voedsel smaak te gee.
- Draai vis in foelie toe en bak in die oond.
- Gebruik 'n kleefwerende pan of kleefwerende sproei om te verhoed dat kos vassit.
- Roerbraai is 'n uitstekende manier om voedsel gaar te maak. Gebruik aftreksel in plaas van olie.
- Maer maalvleis kan verbruin op lae hitte sonder om vet by te voeg. Voeg water, wyn of aftreksel by indien dit begin vassit.
- Kasserol en bredies kan voorberei word sonder om die vleis vooraf te verbruin. Plaas vleis in kastrol, strooi die groente en vloeistof bo-oor en voeg geurmiddels by voordat dit op lae hitte gaargemaak word.
- Laat staan bredies oornag in 'n koel plek om die geur te versterk. Verwyder enige sigbare vet voordat dit herverhit en bedien word.
- Gooi 'n paar ysblokkies in die pan met uitgedrupte vet voordat die vleissous gemaak word. Die vet sal vinnig solied word en dan makliker uitgeskep kan word.
- Gebruik 'n mikrogolf waar moontlik. Voedsel behou hul geur en sap sonder om vet by te voeg.
- Kook, stoom, roerbraai of bak groente in die oond. Smeer met 'n klein bietjie olie en geurmiddels vooraf om geur te verbeter.
- Indien die resep 'n kaas bolaag voorstel, vervang dit met die helfte lae vet kaas en broodkrummels.
- Gebruik vetvrye suiwelprodukte waar moontlik en lae vet kaas.
- Gebruik lae vet of geen vet slaaisouse. Gebruik 'n basis van suurlemoensap of lae vet joghurt en voeg verskillende kruie en speserye of asyn by.
- Vervang room in resepte met vetvrye joghurt.
- Gebruik groente om toebroodjies vogtig te maak in die plek van margarien of botter.
- Bedien vars of gestoofde vrugte vir nagereg.
- Gebruik afgeroomde melk vir die bereiding van souse of vla.
- Voeg lae vet joghurt of geklitste lae vet ingedampte melk by jellie vir 'n heerlike nagereg.
- Gebruik so min sout moontlik, veral as hoë bloeddruk 'n probleem is.
- Gebruik genoeg veselryke voedsel, maar voeg dit geleidelik by. Volgraan brood, growwe mieliemeel, bruin rys, hawermout, droë bone en lensies, groente en vrugte met die pitte en skil aan is goeie voorbeelde.

MAALTYE WEG VAN DIE HUIS

Hoewel dit effens moeiliker is, is dit steeds moontlik om uit te eet en gesonde maaltye te nuttig indien die regte voedsel keuses gemaak word. Die ideaal is dat persone met diabetes hul voedsel wat tuis voorberei is saam met hulle neem. Op daardie manier is hulle altyd in beheer van wat hulle eet en hoe dit voorberei is. Indien dit nie moontlik is nie, is hier 'n paar maklike wenke om te volg:

Wegneem etes

- Geroosterde toebroodjies kan 'n goeie keuse wees indien dit nie met te veel margarien of botter voorberei word nie. Vra vir volgraan of bruinbrood. Kies 'n lae vet vulsel soos hoender, vis of tamatie.
- 'n Gewone bruinbrood toebroodjie is moontlik 'n beter opsie as 'n geroosterde broodjie omdat dit moeiliker is om die hoeveelheid margarien of botter te beheer.
- 'n Toebroodjie is 'n beter opsie as 'n pastei.
- Kies gebakte aartappels met tuna of hoender en sampioene.
- 'n Gewone of vegetariese pizza is 'n redelike keuse.

- Vermy diep gebraaide of soet suur disse indien Chinese kos op die spyskaart is. Kies geregte soos chop-suey of chow mein bedien met rys of noedels wat laer in vet is.
- Kies geroosterde hoender (verwyder die vel voordat dit geëet word) eerder as gebraaide hoender of hoender met 'n deeg om.
- Bestel 'n hamburger sonder aartappelskyfies.
- 'n Volgraan broodrolletjie of 'n gebakte aartappel met maaskaas of lae vet joghurt is goeie keuses om saam met 'n maaltyd te eet.
- Geniet 'n schwarma met ekstra slaai.
- Koop vrugte by 'n stalletjie.
- Koop maas of pap met 'n groente sous.

Restaurante

- Kies helder sop, en Parma ham met spanspek, oesters of 'n vars vrugtekelkie vir 'n voorgereg. Vermy brood of rolletjie as 'n voorgereg.
- Kies geroosterde vis, kalamari of hoender eerder as vleis. Vra die kelner om dit nie in ekstra olie of margarien gaar te maak nie.
- 'n Prego rol, tramezzini, sushi met rys of 'n geroosterde kebab kan as hoofgereg dien.
- Vra vir halwe porsies of dames porsies.
- Indien groente in 'n sous bedien word of gebraai word, vra vir 'n kleinbordie met slaai sonder sous of dat die slaaisous apart bedien word.
- Vermy gemarineerde groente en piekels.
- Vermy romerige souse. Kies eerder souse met 'n tamatie basis.
- Vermy romerige disse in Italiaanse restaurante. Kies die geregte met 'n tamatie basis.
- Vermy geregte met krummels, deeg (of verwyder die deeg voordat dit geëet word), aartappelskyfies en baie mayonnaise.
- Kies lae vet opsies waar moontlik.
- Kies 'n vrug na ete in die plek van nagereg.
- Drink water, mineraal water, dieet koeldrank of droë wyn en soda.

SESSIE 6 REIS EN DIABETES

Vakansie is 'n tyd om te ontspan en uit te rus. Hier is 'n paar wenke vir 'n kommervrye vakansie:

- Neem dubbel die hoeveelheid van die medikasie wat nodig is, toets material en batterye vir die glucose meter saam.
- Indien u in 'n hotel of gatehuis tuisgaan, reel vooraf vir geskikte kos met hulle.
- Hou diabetes mediaksie en toets apparate by u ten alle tye. Moet dit nie inpak saam met die bagasie en dan apart die vliegtuig in stuur nie.
- Dra u "Medic Alert" armband.
- Sorg dat u altyd 'n voorskruf van u medikasie en toetsstrokeis by u het.
- Indien u vreemde landstreke besoek, vermy kraanwater en ys gemaak van kraanwater.
- Ondersoek elke dag u voete vir blasé of ander skaafplekke en kry medies hulp indien daar tekens van infeksie is. Probeer om nie kaalvoet te loop nie.
- Hou vol met u oefenprogram tydens u vakansie.
- Hoe voedsel of vrugtesap byderhand ten alle tye.
- Onspan en geniet dit ! Verlaagde stress vlake help om die bloedsuiker vlakk normal te hou.

SESSIE 7 ALKOHOL

Praktiese riglyne vir die gebruik van alkohol:

1. Maak seker diabetes is goed gekontrolleerd.
2. Vermy alkohol indien u swanger is, hipertrigliseriedemie het, perifere neuropatie, pankreatitis of gastritis het.
3. Gebruik altyd alkohol saam met 'n maaltyd en nooit op 'n leë maag nie.
4. Alkohol se energie inhoud is 29kJ/kg. Moet nooit meer as 2 sopies per dag drink nie.
5. Indien oorgewig, moet die energie inhoud van die alkohol in berekening gebring word in u daaglikse inname van energie per dag. Die koolhidraat inhoud van die drankie hoef nie in berekening gebring te word as drankies soos soet wyn en sjerrie vermy word nie.
6. 'n Verlaging in vet inname word aanbeel, aangesien alkohol gemetaboliseer word soos vet en ook omdat dit so energie dig is.
7. Insulien afhanklike diabete moet nooit alkohol sonder voedsel inneem nie, aangesien dit kan lei tot alkohol geïnduseerde hipoglukemie (lae bloedsuiker).
8. Soet wyn, sjerrie en likeur behoort liefs vermy te word.
9. Mengeldrankies soos tonikum water behoort 'n lae koolhidraat inhoud te hê om die risiko vir hiperglukemie (hoë bloedglukose) te voorkom.
10. Moenie alkohol gebruik indien u moet bestuur nie.
11. Die verdowende effek van alkohol kan die simptome van hipoglukemie wegsteek.

Onderstaande tabel dui die samestelling en uitruilwaarde van sommige alkoholiese drankies aan.

Drankie	Sopie Volume (ml)	Alkohol (g/sopie)	Koolhidrate (g/sopie)	Energie (kJ(kcal)/ sopie)	Uitruiling Vir T2 DM
Bier, Gewone	337	13	13.7	634.2 (151)	1B*, 2F*
Bier, Lig	337	10.1	6	378 (90)	2F
Gin, rum, vodka, whiskey, Scotch	42	15.3	-	441 (105)	2F
Droë brandewyn of konjak	28	10.7	-	315 (75)	1½F
Rooi of Rosè wyn	112	11.6	1	357 (85)	2F
Droë wit wyn	112	11.3	0.4	336 (80)	2F
Soet wyn	112	11.8	4.9	428.4 (102)	½B, 2F
Ligte wyn	112	6.4	2	210 (50)	1F
Vonkel wyn	112	11.9	3.6	411.6 (98)	2F
Sjerrie	56	9.4	1.5	315 (75)	1½F
Soet sjerrie, port of muskadel	56	9.4	7	394.8 (94)	½B, F

*B = Brood
F = Vet

Moet nooit meer as 1-2 sopies per dag drink nie.

SESSIE 8 OEFENING

Gereelde oefening behoort deel te wees van die lewenstyl van persone met diabetes.

Oefening kan help om:

☺ Die medikasie dosis te verlaag

☺ Bloedglukose beheer te verbeter

☺ Kardiovaskulêre risikofaktore te verlaag

☺ Massaverlies te bevorder.

Sportskoene moet van 'n hoë gehalte wees. Oefening moet nie te lank of uitputtend wees nie. Dit kan vir kort tye (20-30 minute) elke dag of vir langer tye 3-5 keer per week gedoen word. Oefening kan massaverlies aanhelp, maar dit is moeilik om massa te verloor sonder om voedselinname te verlaag tesame met die oefenprogram. Gereelde oefening behoort 3-4 ure na maaltye te geskied. Wanneer onverwagte of ongewoon uitputtende oefening plaasvind, of indien oefening langer as 30 minute gaan duur, kan 'n bykomende voeding soos 'n appel of 'n volgraan toebroodjie benodig word.

Indien bloedglukose vlakke bokant 14mmol/l of onder 4mmol/l is, word oefening nie aanbeveel nie aangesien daar moontlik nie genoeg insulien in die liggaam sirkuleer om toe te laat dat die oefening die bloedglukose vlakke verlaag nie, of daar kan te veel insulien wees wat kan lei tot 'n hipoglukemiese koma.

Tabel 1: Aanbevole dieet koolhidraat innames voor en gedurende oefening.

Oefening intensiteit en duur	Bloed-Glukose (mmol/l)	Dieet koolhidrate	Aanbevole Voedsel
Kort, hoë intensiteit (<30minute) bv. Gewigoptel en naellope	6-10	Geen voedsel benodig	
Lig Bv. Loop 30 minute, matige pas aerobiese oefeninge 60 minute	<6 >6	15 Geen voedsel benodig	1 stysel uitruilling
Matig (<45minute) bv. Voetbal of fietsry	<6 6-10 10-14 14+	30-45 15 Geen voedsel benodig Oefening nie aanbeveel	2-3 stysel uitruilings or 3-4 vrugte uitruilings 1 Stysel uitruilling
Matig (> 60min) bv, voetbal, basket bal, tennis of fietsry	10-14 >13-14 +Ketone >17 (geen ketone)	10-15g/h Moenie oefen nie Oefening nie aanbeveel	1 Vrug of stysel uitruiling
Uitputtend (<60min) bv. Driekamp, maraton, kano, fietsry	<6 6-10 10-14 14+	45 30-45 15-30 Oefening nie aanbeveel	3 Stysel uitruilings 3-4 Stysel uitruilings 2-3 stysel uitruilings
Uitputtend (>60min) bv. Driekamp, maraton, kano, fietsry	<6 6-10 10-14	50g/h 25-30g/h 10-15g/h	500ml Kola of verdunde vrugtesap (2/3 sap + 1/3 water)/h 250-300ml Kola ofvedunde vrugtesap/h 100-150ml Kola of verdunde vrugtesap/h

SESSION1 IMPORTANCE OF GOOD CONTROL

DEFINITION OF TYPE 2 DIABETES MELLITUS:

Diabetes is a condition where the body is unable to break down (digest) and use (metabolise) food (especially starchy food) properly. The body does not use the insulin it has for this to happen.

HOW DOES IT WORK?

In people without diabetes, food is broken down to release nutrients into the gastrointestinal canal/tract (GUT)/ All food is made up of nutrients. These nutrients work together to keep the body alive and healthy.

Carbohydrate (starchy) foods provide energy. They are broken down to form sugar molecules. These are taken into the blood as blood sugar (which is also called (blood glucose). The heart pumps the blood to every part of the body. The nutrients are carried in the blood to the different cells where they are needed. The cells have cell walls, which control what enters and leaves the cell. The blood sugar us used by the cells for energy. Whenever it is needed, the pancreas (an organ inside the body near to the stomach) makes hormone insulin.

Insulin is sent into the bloodstream to help the cells take up the blood sugar. Insulin is broken down once it has done its work. The body makes new insulin whenever it is needed. The surfaces of the cell walls have special places on them to allow the insulin to join them. Once the insulin is joined onto the cell, it opens the cell wall just like a key opens a door. The blood sugar is then able to enter the cell from the bloodstream when a lot of food is eaten a large amount of insulin is sent to help the cells take up the extra blood sugar.

Blood sugar is changed very quickly into energy that can be used by the cells. Energy is used for all activities; such as walking, talking, thinking, working and sleeping. When a lot of exercise or physically heavy work is done, the body needs extra energy. The cells use more blood sugar to provide the extra energy for the body. Some cells e.g. brain and nerve cells, must have a regular supply of energy (provided by the blood sugar) in order to work.

Extra blood sugar is stored as a reserve (in the liver as glycogen or in fat tissue). It is later released when the blood sugar gets too low (e.g. if a person hasn't eaten for a long time).

Waste products are sent back into the blood stream. As the blood passes through the kidneys, it is filtered. The waste products are passed out of the body with the urine.

THE PERSON WITH DIABETES

When a person has diabetes, there is not enough insulin to "open the cell doors. Because of this, the cells cannot take up blood sugar. The cells are "resistant" to the insulin and don't allow it to attach itself to the cell wall. As a result, the cells cannot change the blood sugar into energy. The blood sugar remains in, and builds up into the bloodstream. When there is a large amount of blood sugar in the blood, it spills over to the urine. This blood sugar is not just sugar from eating cane sugar, jam or other sugar dense foods. It is the resulting glucose in the blood from the breakdown of all foods containing carbohydrates. The blood sugar level rises above normal. When diabetes is untreated or uncontrolled, the blood sugar levels are very high. Sugar can also be found in the diabetic person's urine. The term diabetes mellitus means:

Diabetes - "to run through"
Mellitus - "sweet like honey" (i.e. Very sweet urine).

The cells need energy to function. When they cannot get blood sugar to change into energy, the body's fat stores are used for energy. When body fat is broken down to produce energy, "ketone bodies" are formed. The cells cannot use the ketones fast enough. The ketones then build up in the blood. The ketones are removed from the body via the lungs causing a "fruity" breath, and in the urine. If untreated, this can lead to possible dehydration, kidney and heart failure.

DIAGNOSIS:

Diabetes is discovered when someone complains of not feeling well. If diabetes is suspected, a person's urine is tested for glucose (blood sugar). Sugar gives the body energy, but if the body does not have enough insulin, the blood glucose cannot be taken into the cells. Te blood glucose remains in the blood and spills into the urine. If the urine test is positive (shows there is glucose present), a test will be done on a small amount of a person's blood. If the blood test shows a high amount of glucose in someone who hasn't eaten (glucose greater than 7 mmol), it probably means the person has diabetes. The urine and blood may also be tested for ketones.

SYMPTOMS of uncontrolled diabetes is:

- increased appetite (poliphagia),
- increased frequency of passing urine (polyuria),
- increased thirst (polydipsia),
- unexplained weight loss
- weakness.

CAUSES

- Age - patients are usually over 40 years
- Family history of diabetes
- Obesity - 80% of new diabetics are overweight
- Physiologic and mental stress

COMPLICATIONS:

The complications of diabetes mellitus may be divided into two major categories:

1. Acute and
2. Chronic.

1. **Acute complications:**
   These are directly related to rapid changes in metabolism like a very high blood sugar level at diagnosis and/or a very low blood sugar (hypoglycaemic reaction).
   These complications are preventable and require immediate intervention

2. **Chronic complications:**
   These include vascular disease (heart), nephropathy (kidneys), neuropathy (feet) and retinopathy (eyes). They can be markedly reduced by therapy directed at regulating blood glucose levels close to the normal range.

The dietitian plays an important role in helping the person with diabetes achieve the control necessary to avoid short-term complications and to postpone long-term complications.

Always be aware of the signs of a hypoglycaemic reaction:

- Angry
- Anxious
- Clammy
- Clumsy
- Confused
- Hungry
- Impatient
- Irritable
- Light-headed
- Nausea
- Nervous
- Numb
- Pale
- Sad
- Shaky
- Sleepy
- Stubborn
- Sweaty
- Tense
- Tired
- Weak

General treatment of (hypoglycaemia (low blood sugar)

Low blood sugar is easy to treat. If it is low but cannot be tested at that time, something containing sugar should be eaten. When in doubt, it is safer to eat the extra food than to risk having a low blood glucose reaction.

If the person is unable to swallow, do not force feed any food or drink because of the danger of choking and aspiration.

The following treatments contain 15 grams of carbohydrate:

3-4 glucose tablets
½ cup cold drink (not sugar-free)
4-5 small hard sugar sweets (chewed)
½ cup juice (apple, orange)
1 cup skim milk
1 Tablespoon of sugar

- Repeat every 10-15 minutes until the symptoms of low blood sugar are gone.

SESSION 2 SYMPTOMS OF HYPOGLYCEMIA AND HYPERGLYCEMIA

HYPOGLYCEMIA (low blood sugar)	HYPERLYCEMIA (high blood sugar)
SYMPTOMS	**SYMPTOMS**
Sweating Hunger Double vision Impatience Trembling Headache Faintness "All gone" feeling	Cold and dry skin Thirst Dry mouth Nausea and vomiting Flushed face Deep sighing Acid breath Headache Pain in back and legs Abdominal pain Drowsiness Dizziness Extreme weakness
CAUSES	**CAUSES**
Excess insulin Excess exercise Delay eating Omission of a meal	Lack of insulin Injury and infection Surgery Emotional upset
TREATMENT	**TREATMENT**
Give half a cup of fruit juice OR one tablespoon of honey OR three Super C sweets OR four teaspoons of sugar Followed by a meal or snack	Fluids Insulin

SESSION 3 MEAL DISTRIBUTION, CARBOHYDRATES AND FIBRE

There is no need for the person to eat food different from the rest of the family. Rather, they should lead the way for all family members to improve their eating habits to follow a healthy eating plan.

The following points should be remembered when planning meals: -

- **3-6 Regular meals should be eaten.**
  They should be:
- evenly sized,
- evenly spaced and
- eaten at the same time every day.
-

- **A high-fibre carbohydrate food should form the basis of each meal.**

- **Some vegetables or fruit should be added to the carbohydrate food.** One should aim to have 5 servings every day if possible. Fats and oils should not be added when cooking or serving them.

- **To the meal a small, low-fat portion of protein food can be added.** Try to have 1-2 servings every day. Plant proteins should be included at least twice a week. As little added fat as possible, should be used.

- **Snacks eaten late at night should include a little protein.** This makes the food last longer and will prevent the blood glucose levels from dropping too low during the night. Good choices for late night snacks include:
- whole-wheat bread or biscuits with peanut butter or low-fat cheese
- yoghurt, milk or sour milk
- samp and beans
- left-over food from the main meal

CARBOHYDRATES:

The diet should provide 50 - 65% of the total daily energy intake as carbohydrate. This should come from a variety of sources.

Carbohydrate should supply most of the energy in the diet. Increase the amount of carbohydrate containing foods and decrease the intake of foods high in fat. Sources of carbohydrates is bread, cooked cereals with oats, whaet, dry cereals, wheat, oats, bran, popcorn, crackers, saltines, macaroni, spaghetti, noodles, rice and potatoes

FIBRE:

Soluble fibre: Forms a gel in water like for instance oats. Insoluble fibre: Edible skins and seeds of vegetables and fruit.

Food sources high in water-soluble fibre e.g. oat bran, legumes, cereals, fruit and vegetables, should be the biggest part of the total fibre intake. Foods eaten in the unrefined form supply the most fibre. Fibre should gradually be increased and accompanied by adequate fluid intake. A high fibre intake may cause gastrointestinal distress in the older patients with diabetes.

Fibre has several functions: -

- It prevents gastrointestinal problems e.g. constipation.
- It helps in weight control as it makes food more satisfying.
- It helps to control blood glucose and lipid levels by helping to slow down the rate at which these substances are absorbed.

SESSION 4 FAT

FAT:

One of the most important aspects of the diabetic eating plan is to ensure that fat intake does not exceed the recommended amounts. Controlling fat intake assists in weight control and helps minimize the risk of coronary heart disease.

People with diabetes should follow a low fat diet: - Total fat should compromise ≤30% of the total daily energy intake.

- A maximum of one third (≤10%) of the fat being supplied by saturated fat (fat from animal products)
- A maximum of one third, and preferably less (<10%, preferably 6 – 8%), from polyunsaturated fat (sunflower oil and most soft margarine); and
- The balance from monounsaturated fat (olive oil, canola oil and margarine, peanut butter and avocado).

A reduction in saturated fatty acids is usually associated with a reduction in intake of dietary cholesterol.

To comply with the recommendations regarding the correct types of fat in the diet:

- Eat small portions of meat and try to eat chicken and fish (including canned fish such as pilchards or tuna) more often.
- Minimize the use of animal fats such as butter, cream, lard, and ghee and remove visible fat from meat **before** cooking.
- Minimize the use of certain vegetable fats that are high in saturated fats such as coconut oil (used in coffee creamers) and palm oil.
- Try not to reuse oil and avoid overheating oil as this changes the chemical structure and makes it less healthy.
- When using added fats, vary the type used. For example, if a polyunsaturated margarine is used as a spread, a monounsaturated fat such as olive or canola oil should be used for cooking. Note: Peanut butter or avocado can also be used as a source of monounsaturated fat.
- People with a high level of cholesterol in their blood should use the following foods in moderation: organ meats (e.g. liver. Kidneys, brains) and shellfish (e.g. prawns, calamari, crayfish) and limit egg yolks to 3 - 4 per week, preferably not more than one at a time (egg whites can be used freely).

To reduce the total amount of fat in the diet, people with diabetes should be advised to:

- Try to cut down on foods with a high fat content - this includes both added fat such as oil, butter, cream, salad dressing; and "invisible" fat such as full fat cheeses, full cream milk, processed meats, pies and pastries, and many biscuits and cakes especially those with butter icing.
  - Fatty foods do not always either look or taste fatty.
  - Cut down on all fried foods e.g. chips, fried fish & chicken, vetkoek.
- Many convenience and fast foods have high fat content - these foods should not be eaten regularly e.g. pies, crisp, nuts.
- Choose low fat cooking methods - grill, boil, bake or steam foods rather than frying in oil or other fats.
- Buy lean cuts of meat and remove all visible fat, and skin from chicken, before cooking.
- Instead of adding fats such as margarine, oil or butter to vegetables, use herbs, spices, lemon juice or crumbs for added flavour.
- Use low fat dairy products such as low fat or skim milk; sour milk, maas or yoghurt made from low fat or skim milk; low fat cheeses such as cottage cheese instead of full cream varieties. Use low fat skim milk instead of coffee creamers as these are made from coconut oil and are high in saturated fat. Look for the dairy mark on dairy products to make sure they are made only from milk and are not blends or mixtures of milk plus coffee creamers.
- Use low fat salad dressings such as low oil mayonnaise or "no oil" dressing.
- Instead of using the drippings in the pan to make gravy, use a vegetables stock and thicken with flour.
- Grill foods on a rack to allow the fat to drip off.
- When making stews or curries, make them ahead of time and allow them to cool - the fat will form solid layer on top that can be easily removed.
- Spread margarine or other spreads very thinly, or, better still, use none at all.
- Look carefully at the ratio of foods on your plate, one to the other. Ideally, there should be a small portion of the protein rich food (e.g. meat / chicken / cheese) and larger portions of the starchy foods (e.g. bread / potatoes / rice / maize meal porridge / pasta) and vegetables *. Beans and lentils are rich in both protein and carbohydrate and therefore a good choice when choosing a protein source.
- See "plate model" under section on Meal Planning.

SESSION 5 COOKING HINTS AND EATING OUT

MEAL PREPARATION:

Key messages:

All foods should be prepared using as little added fat as possible (see section under "Fat").

Use high fibre foods frequently.

Following are more useful hints concerning food preparation: -

- Buy leanest cuts of meat. Cut down on processed meats, salami and bacon.
- When grilling, baste or marinate food in lemon juice, low-fat yoghurt, wine or stock to prevent drying out.
- Have at least 2 vegetarian main meals a week.
- Make use of herbs, spices and other flavouring agents to make food tasty.
- Wrap fish in foil and bake in the oven.
- Use a non-stick pan or use non-stick cooking spray to prevent food from sticking.
- Stir-frying is an excellent way of preparing meals, using stock rather than oil.
- Lean mince can be browned on low heat without adding any fat. Add water, stock or wine if it begins to stick.
- Casseroles, stews or bredies can be prepared without pre-browning meat. Place meat in a pot, cover with vegetables and liquid and add seasoning before cooking over low heat.
- Leave cooked stews overnight in a cool place to improve the flavour. Remove any visible fat before re-heating to serve.
- To make gravy, add a handful of ice cubes to the pan of meat dripping after roasting. This will cause the fat to solidify making it easy to remove.
- If available, use a microwave oven. Dishes can be prepared in a microwave without added fat and still keep their juices and flavour.
- Boil, steam, stir-fry or bake vegetables in the oven. Add seasoning and brush with a little oil beforehand to improve the flavour.
- When a recipe suggests a cheese topping replace it with half breadcrumbs or oats and half low-fat cheese.
- Use "low oil" or "no oil" salad dressing. Make salad dressing using lemon juice or low fat yoghurt as a base and adding different herbs, spices or various flavours of vinegar.
- Use fat-free dairy products when possible and low-fat cheese.
- Replace cream in recipes with fat-free yoghurt.
- Rather than using margarine or butter when preparing sandwiches or rolls, use vegetables to moisten them.
- Serve fresh fruit or stewed fruit as a dessert.
- Use skim milk when making sauces or custards.
- Add low-fat yoghurt or whipped low-fat evaporated milk to jelly to make a delicious dessert.
- Try to use as little salt as possible, especially if high blood pressure is a problem.
- Use plenty of high fibre foods, making sure they are introduced gradually. Examples include whole-wheat bread, coarse maize meal, brown rice, oats, dry beans and lentils, vegetables and fruit with the skin and peel on.

MEALS AWAY FROM HOME:

People often eat meals away from home. Although this makes it more difficult, it is still possible to eat out and have healthy meals, provided that the right food choices are made.

Obviously, the best thing for people with diabetes would be to take food, prepared at home, with them. That way, **they are in control** of what they are eating and how it is prepared.

If this is not possible, here are some hints that should help choose foods away from home.

Takeaways: -

- Toasted sandwiches can be a good choice provided that they are not prepared with too much margarine or butter. Ask for whole-wheat or brown bread. Use a low-fat filling e.g. chicken, fish or tomato. Ideally, no margarine or butter should be added.
- A plain brown bread sandwich is usually a healthier choice than a toasted one because it is difficult to control the amount of margarine added.
- A sandwich is a healthier choice rather than a pie.
- Try baked potatoes with fillings like tuna or chicken and mushroom.
- A plain or vegetarian pizza is a reasonable choice.
- If Chinese food is chosen, the deep fried or sweet & sour dishes should be avoided. Rather select dishes that are lower in fat e.g. chop-suey or chow-mein served with rice or noodles.
- Choose grilled chicken (remove the skin before eating) rather than deep fried or chicken cooked in batter.
- Order a hamburger without the chips.
- A whole-wheat roll or a baked potato topped with cottage cheese or low fat yogurt is good choices to eat with the meal.
- Enjoy a schwarma with extra salad.
- Eat fruit from a vendor.
- Buy maas or porridge with vegetable relish.

Meals in restaurants: -

- Choose clear soup, a plain salad, and Parma ham with melon, oysters or a fresh fruit cocktail as a starter. Avoid bread or rolls as a starter.
- Choose grilled fish, calamari or chicken rather than meat. Ask the waiter to have it prepared without extra fat (e.g. butter).
- A Prego roll, tramazzine, sushi with rice or a grilled kebab could be enjoyed as a main course.
- Ask for half portions or "ladies" portions.
- If vegetables are in a sauce or fried, ask for a side salad without dressing, or with dressing served separately.
- Avoid marinated vegetables and pickles.
- Avoid creamy sauces. Have tomato-based sauces instead.
- In Italian restaurants, avoid the rich, creamy dishes. Order dishes with a tomato based sauce instead.
- Avoid dishes with crumbs, batter (or remove crumbs or batter before eating), chips or lots of mayonnaise.
- Use low-fat options where possible.
- If you eat out often have fruit after a meal rather than pudding.
- Drink water, mineral water, diet cold drinks or dry wine and soda water.

SESSION 6 ALCOHOL

GUIDELINES FOR THE USE OF ALCOHOL:

In individuals with diabetes, like in those without it, alcoholic beverages form a part of their social lives. It is therefore important that the individual must be aware of the physiological effects of alcohol.

Alcohol is not metabolised to glucose; it could lead to hypoglycaemia if it is consumed without food.

Practical recommendations for the use of alcohol:

1. Be sure that the diabetes is well controlled.

2. Alcohol is contra-indicated in patients with Diabetes Mellitus with hypertriglyceridaemia, pregnancy, peripheral neuropathy, pancreatitis or gastritis.

3. Alcohol must always be ingested in combination with a meal and never on an empty stomach.

4. Alcohol has an energy content of 29 kJ/g. Alcohol should not exceed 6-10 % of the total daily energy intake (± 20-30 g alcohol per day). This is equivalent to 1-2 servings of alcohol per day.
   If drinks with a high carbohydrate content e.g. sweet wines and sherries are avoided, the carbohydrate content of the drink need not be counted in the daily carbohydrate allowance.

5. Because of the energy density of alcohol, patients with Diabetes Mellitus that are overweight should take the energy content of the alcohol into account. Therefore, the energy content of the alcohol should be included in the total energy intake of the day.

6. A reduction in fat intake is recommended because of the caloric density of alcohol and because alcohol is metabolized like fat.

7. Insulin dependent diabetics should not omit food, because of the possibility of alcohol-induced hypoglycaemia.

8. Drinks with a high carbohydrate content e.g. sweet sherry and wines, as well as liqueurs should preferably be avoided.

9. Mixers e.g. tonic waters should have a low carbohydrate content to avoid the risk of hyperglycaemia.

10. Alcohol should not be consumed before driving.

12. The cerebral effects of alcohol can mask the symptoms of hypoglycaemia.

Table 1: Indicates composition and exchange value of some alcoholic beverages.

BEVERAGE	SERVING SIZE (ml)	ALCOHOL (g/serving)	CARBOHYDRATE (g/serving)	ENERGY (kJ(kcal)/serving)	EXCHANGE FOR T2 DM
Beer					
Regular	337	13	13,7	634,2 (151)	1B*, 2F*
Light	337	10,1	6	378 (90)	2F
Liquor					
Distilled spirits (Gin, rum, vodka, whiskey, Scotch)	42	15,3	-	441 (105)	2F
Dry brandy or cognac	28	10,7	-	315 (75)	1½F
Wine, Table					
Red or rose	112	11,6	1	357 (85)	2F
Dry white	112	11,3	0,4	336 (80)	2F
Sweet	112	11,8	4,9	428,4 (102)	½B, 2F
Light	112	6,4	2	210 (50)	1F
Sparkling champagne	112	11,9	3,6	411,6 (98)	2F
Appetizer/dessert sherry	56	9,4	1,5	315 (75)	1½F
Sweet sherry, port, muscadel	56	9,4	7	394,8 (94)	½B, F

* : B = bread
F = fat

Never exceed 1-2 drinks per day.

SESSION 7 TRAVEL AND DIABETES

A few simple precautions can ensure a worry free trip.

- Take double your expected requirements of medication and testing equipment (including batteries).
- Organise diabetic meals on journeys and hotels
- Carry diabetes medication and testing supplies with you at all times. Do not leave it with the luggage in the hold of a plane.
- Wear your medic alert bracelet.
- Always carry a prescription for your medication and strips
- Avoid tap water and ice made from tap water.
- Avoid going barefoot. Check your feet each day for blisters or other abrasions and seek medical attention at the dirst sign of infection.
- Maintain your exercise program while you are on vacation
- Keep a food or a juicebox with you at all times
- Relax and have fun! Keeping the stress levels low helps keep blood glucose levels normal.

SESSION 8 EXERCISE

Regular exercise should be a part of the lifestyle of people with diabetes.
Exercise may help to:

- Lower the medication dose
- Improve blood sugar control
- Reduce cardiovascular risk factors
- Maintain muscle mass
- Promote fat loss

High quality footwear should be used. Exercise should not be too long or strenuous. It can be done for short periods (20-30 minutes) every day or for longer periods 3-5 times per week. Exercise can assist in weight loss, but it is difficult to lose weight without reducing food intake in addition to exercising regularly. Ideally, regular exercise should be doe 3-4 hours after meals. When unexpected or unusually strenuous exercise takes place, or if exercise is planned to last longer than 30 minutes, an additional snack (such as an apple or a wholewheat sandwich) may be needed.

If blood glucose levels are above 14mmol/L of below 4mmol/L exercise is not advised as, under these conditions, there may be enough insulin circulating in the body to allow for exercise to reduce the glucose levels, or alternatively, there may be too much insulin, which could lead to hypoglycaemic coma.

Table 1: Recommended dietary carbohydrate intakes before and during exercise

Exercise intensity and duration	Blood glucose (mmol.l)	Dietary carbohydrate (g)	Suggested food to use
Brief, high intensity (<30min) e.g. weight lifting,sprints	6-10	No food required	
Light e.g. walking 30min, easy pace aerobics 60min	<6 >6	15 No food required	1 bread exchange
Moderate (<45min) e.g. swimming, jogging, tennis, basketball	<6 6-10 10-14 14+	30-45 15 No food required Exercise not advised	2-3 bread exchanges or 3-4 fruit exchanges 1 bread exchange
Moderate (>60min) e.g. football,cycling	10-14 >13-14 +ketones >17(no ketones)	10-15g/h Exercise contraindicated Exercise not advised	1 fruit or bread exchange
Strenuous (<60min) e.g. triathlon, marathon, canoeing,cycling	<6 6-10 10-14 14+	45 30-45 15-30 Exercise not advised	3 bread exchanges 3-4 bread exchanges 2-3 bread exchanges
Strenuous (>60min) e.g. triathlon, marathon, canoeing,cycling	<6 6-10 10-14	50g/h 25-30g/h 10-15g/h	500ml Coke or fruit juice diluted (2/3 juice + 1/3 water)/h 250-300ml Coke or diluted fruit juice/h 100-150ml Coke or diluted fruit juice/h

Appendix 8: The alcohol intake pattern of the two groups at baseline and during the trial period.

	Alcohol intake pattern of Group 1 (SID) for the trial period (N=10)							Alcohol intake pattern of Group 2 (SFD) for the trial period (N=12)					
	Subjects who took alcohol	Servings per day	Subjects who took alcohol	Servings per week	Subjects who took alcohol	Servings per month		Subjects who took alcohol	Servings per day	Subjects who took alcohol	Servings per week	Subjects who took alcohol	Servings per month
Baseline			1	2	1	4				1	3		
Trial week 1			1	2	1	4				1	3		
			1	9									
Trial week 2			1	2	1	4				1	3		
			1	9									
Trial week 3	1	1	1	1	1	4		1	6	1	3		
			1	4									
Trial week 4			1	1	1	4				1	3		
			1	9									

ABSTRACT

The prevalence of diabetes mellitus in South African communities is increasing aggressively, due to population and lifestyle changes associated with rapid urbanization. It is estimated that the prevalence of diabetes is due to triple within the next 25 years. Currently 10% of the total energy intake as sucrose is allowed as part of a balanced diabetic diet, according to the Diabetes Education Society in Southern Africa. Health professionals are ignorant and/or sceptical about this guideline and are reluctant to advise the patients they consult with.

The aim of this study was to evaluate the effects of 15% of the total daily energy intake as sucrose on the glycaemic control of patients with type 2 diabetes mellitus. To accomplish this aim, the effects of the inclusion of 15% of the total daily energy intake as sucrose were compared to the exclusion of sucrose in the diets of free-living patients with type 2 diabetes mellitus on glycaemic control (fasting plasma glucose concentrations, serum fructosamine, HbA_{1c} percentages) and lipid profiles (serum cholesterol, serum HDL cholesterol, serum LDL cholesterol and serum triglycerides).

The study was a randomized, controlled, single-centre clinical trail. Only 22 of the possible 401 subjects screened, who had type 2 diabetes mellitus (determined by GAD 65 and C-peptide values), and who volunteered to comply with a prescribed diet for the 16 week study period, participated in the study. At baseline, a food record and validated quantitative food frequency questionnaire was filled in by the researcher. Anthropometrical measurements (weight, height, BMI and body-fat percentage) were measured, and blood samples were analysed. Prior to baseline, subjects were advised to increase their activity level as part of a healthy lifestyle. Lifestyle patterns (smoking, alcohol consumption, exercise and medication) had to be maintained throughout the study period. Individual diets were calculated for all subjects. After a 12 week period during which all subjects were stabilized on a

diabetic diet, subjects were randomized into two groups. Group 1, received a sucrose inclusive diet (SID) and Group 2, a sucrose free diet (SFD), for a four week trial period. The type of control, namely, oral medication and diet alone, stratified these groups. There was, thus, a separate computer-generated randomization list for each of these two strata; randomizing the subjects into a study and control group. During the entire 16 week study period the researcher and nurse had contact sessions with the subjects (fortnightly and weekly, respectively). A short informative talk to motivate and encourage subjects to adhere to, and gain insight into dietary aspects of type 2 diabetes mellitus, was given by the researcher. A registered nurse measured weight and venous plasma glucose concentrations of all subjects on a weekly basis. The registered nurse measured serum fructosamine concentrations on a fortnightly basis. At the end of the study each subject's body-fat percentage was measured and fasting blood samples (blood lipid concentrations and HbA_{1c} percentages) were analyzed statistically to test for significant differences between the two dietary groups.

The habitual dietary intake after recruitment showed that all subjects followed a low carbohydrate, high fat diet. The habitual sucrose intake in Group 1 (SID) showed a sugar intake of 4.5%, and Group 2 (SFD) of 4.2%, respectively. The mean BMI of subjects in both groups was within the class I, obese range (BMI= 30-34.9kg/m^2). Although all subjects in the study showed weight maintenance, both dietary groups experienced reduction in their body-fat percentage. However, Group 2 (SFD) showed statistically significant improvement (95% CI: -8.5;-0.6) in body-fat percentage (4.5%). The reduction in body-fat percentage of Group 1 (SID) could be considered as clinically significant (1.1%). No differences occurred in body-fat percentage between the groups. The fact that there was a change in body composition without weight loss may be attributed to the strict compliance and adherence of subjects to their dietary guidelines and exercise. The mean plasma glucose concentrations for both groups were within the acceptable glycaemic control reference range of 6-8 mmol/l throughout the study period. The mean serum fructosamine concentrations of Group 1 (SID) remained unchanged during the trial period. The mean serum fructosamine concentrations of Group 2 (SFD)

showed statistically significant improvement (95% CI: -25.3;-3.2) during the trial period. No significant differences were observed between the two groups. Both groups maintained a mean HbA_{1c} percentage within the optimal fasting reference range of < 7% throughout the study period. Group 1 (SID) showed an improvement (from 6.8% at baseline to 6.3% at the end of the study period) in HbA_{1c} percentage that were close to statistical significance and were clinically significant, while Group 2 (SFD) showed a statistically significant improvement (95% CI: -2.6;-0.2).

It can be concluded that subjects with type 2 diabetes mellitus can safely include a moderate amount (15% of the total energy) of sucrose in a balanced diet, without deleterious effects on their glycaemic control. The long term glycaemic control (as measured by the HbA_{1c} percentages) improved with good dietary compliance in both diets that included/excluded sucrose. Results of this study suggest that moderate intake of sucrose (15% of the total energy) had no aggravating effects on blood lipid concentrations of these subjects for a trial period of four weeks. However, the long term effects of sucrose on blood lipid concentrations could not be assessed. This sucrose modification in the diabetic diet may lead to improved adherence by subjects, as it minimizes the sense of deprivation. The inclusion of moderate sucrose in a balanced diet will enhance overall palatability and might improve long term compliance. Compliance to a balanced diet will improve diabetic control. Furthermore, fewer restrictions in the diet of subjects with type 2 diabetes mellitus may also lead to a reduction in short and long term complications. More research is needed to determine the long term effects of sucrose on blood lipid concentrations in subjects with type 2 diabetes mellitus.

If health care workers continue to be reluctant to advise the inclusion of sucrose in the type 2 diabetic diet, because of personal prejudice or ignorance regarding the benefits of research such as this, it may create confusion and disbelief among diabetic patients concerning the efficacy of the diet. The colloquial concept of diabetes mellitus being merely a "sugar disease", and the misconception that sucrose causes diabetes mellitus, should be dispelled forthwith.

Key words: type 2 diabetes mellitus, sucrose, glycaemic control, plasma glucose, serum fructosamine, HbA_{1c} percentages, compliance.

OPSOMMING

Die voorkoms van diabetes mellitus in die Suid-Afrikaanse gemeenskap neem geweldig toe a.g.v. veranderinge in die bevolking en lewenstyl geassosieer met vinnige verstedeliking. Die voorkoms van diabetes sal na verwagting drievoudig toeneem binne die volgende 25 jaar. Volgens die "Diabetes Education Society in Southern-Africa" word sukrose huidiglik as 10% van die totale energie inname per dag toegelaat as deel van 'n gebalanseerde diabetiese diet. Gesondheidswerkers is oningelig en/of skepties rakende hierdie riglyn en is teesinnig om pasiënte hieroor te konsulteer.

Die doel van hierdie studie was om die effek van 15% van die daaglikse energie inname as sukrose op die glukemiese beheer van pasiënte met tipe 2 diabetes mellitus, te evalueer. Om hierdie doel te verwesenlik is die effek van die insluiting van 15% van die daaglikse energie inname as sukrose vergelyk met die uitsluiting van sukrose in die diëte van vry lewende pasiënte met tipe 2 diabetes mellitus i.t.v. glukemiese beheer (vastende plasma glukose konsentrasies, serum fruktosamien, HbA_{1C} persentasies) en bloedlipiede (serum cholesterol, serum HDL cholesterol, serum LDL cholesterol en serum trigliseriede).

Die studie was 'n gerandomiseerde, gekontrolleerde, enkel-sentrum kliniese proef. Slegs 22 van die moontlike 401 gesifte proefpersone wat tipe 2 diabetes mellitus (bepaal deur GAD65 en C-peptied waardes) gehad het, en gewillig was om 'n voorgeskrewe dieet vir 'n 16 week studie periode te volg, het deelgeneem aan die studie. Basislyn data ingesamel sluit onder meer 'n voedselrekord en geldige gekwantifiseerde voedselfrekwensie vraelys deur die navorser ingevul in, sowel as antropometriese metings (massa, lengte, LMI en liggaamsvet persentasies) en bloed monsters wat ontleed is. Voor aanvang van die studie is proefpersone aangemoedig om hul aktiwiteit te verhoog as deel van 'n gesonde lewensstyl. Ander lewenstyl gewoontes (rook, alkohol inname, oefening en medikasie) moes gehandhaaf word gedurende die studie periode. Proefpersone se diëte is individueel uitgewerk. Na 'n 12 week periode waar proefpersone gestabiliseer is op 'n diabetiese dieet, is

hulle gerandomiseer. Groep 1, het 'n sukrose insluitende dieet (SID) ontvang en Groep 2, 'n sukrose uitsluitende dieet (SFD) vir 'n vier week proef tydperk. Daar was dus 'n aparte rekenaar ontwerpte randomisasie lys vir elk van die twee strata, sodat daar 'n kontrole en studie groep was. Die navorser het bondige praatjies gelewer om proefpersone te motiveer en aan te moedig om hul dieet te volg, sowel as om insig te verleen in die dieet aspekte van diabetes mellitus. Pasiënte is weekliks deur die suster geweeg. Proefpersone se veneuse plasma glukose konsentrasies is weekliks, en serum fruktosamien, twee weekliks bepaal. Aan die einde van die studie is elke proefpersoon se liggaamsvet persentasie en vastende bloed monsters (bloed lipied konsentrasies en HbA_{1c} persentasies) statisties geanaliseer om betekenisvolle verskille tussen die twee groepe te ondersoek.

Dit blyk uit die ontleding van gewoontelike diet inname dat proefpersone 'n lae koolhidraat, hoë vet dieet gevolg het. Die gewoontelike sukrose inname van Groep 1 (SID) was 4.5% en Groep 2 (SFD) s'n 4.2%. Die gemiddelde LMI van beide groepe was binne die reikwydte van klas I obesiteit (LMI = 30-34.9kg/m^2). Hoewel proefpersone hul massa gehandhaaf het tydens die studie, het beide groepe 'n verbetering in persentasie liggaamsvet getoon. Die verbetering (4.5%) in liggaamsvet persentasie van Groep 2 (SFD) was egter statisties betekenisvol (95% CI: -8.5;-0.6). Die verbetering van Groep 1 is egter van kliniese belang (1.1%). Geen verskille in liggaamsvet persentasie tussen die twee groepe is gevind nie. Die verandering in liggaamsvet persentasie met die instandhouding van liggaamsmassa kan moontlik toegeskryf word aan streng kontrole en dat proefpersone by hul dieetriglyne en oefen patroon gehou het. Die gemiddelde plasma glukose konsentrasies van beide groepe was deurentyd binne die aanvaarbare reikwydte vir glukemiese beheer (6-8 mmol/l). Die gemiddelde serum fruktosamien konsentrasies van Groep 2 (SFD) het statisties beduidend verbeter (95% CI: -25.3;-3.2) gedurende die proeftydperk. Geen betekenisvolle verskille tussen die twee groepe is opgemerk nie. Beide groepe se HbA_{1c} persentasies was binne die optimale vastende rykwydte van < 7% gedurende die studie tydperk. Groep 1 se verbetering (vanaf 6.8% met aanvang tot 6.3% teen die einde van die studie) in HbA1c was baie na aan statisties beduidend en van kliniese

belang, terwyl Groep 2 (SFD) 'n statisties beduidende verbetering (95% CI: -2.6;-0.2) getoon het.

Die gevolgtrekking kan gemaak word dat pasiënte met tipe 2 diabetes mellitus 'n matige hoeveelheid sukrose (15% van die totale energie) met veilighied in 'n gebalanseerde dieet kan insluit sonder nadelige effek op glukemiese beheer. Die langtermyn glukemiese beheer (soos gemeet deur die HbA_{1c} persentasies) het verbeter met goeie dieetkontrole in beide diëte wat sukrose ingesluit/uitgesluit het. Die resultate van die studie dui aan dat die matige insluiting van sukrose geen nadelige effek op hierdie pasiënte se bloed lipied konsentrasies gehad het nie. Die lang termyn effek van sukrose op bloed lipied konsentrasies kon egter nie bepaal word nie. Die sukrose modifikasie in die diabetiese dieet kan lei tot beter dieet kontrole aangesien dit die gevoel van uitsluiting verminder. Dit kan ook die smaaklikheid van die dieet verbeter en kan moontlik die lang termyn kontrole verhoog. Gebalanseerde dieet kontrole verbeter glukemiese beheer. Minder beperkings in die diëte van pasiënte met tipe 2 diabetes mellitus kan komplikasies op kort en lang termyn verminder. Meer navorsing is nodig om die lang termyn effekte van sukrose op bloed lipied konsentrasies in pasiënte met tipe 2 diabetes mellitus, te bepaal.

Indien gesondheidswerkers as 'n gevolg van persoonlike vooroordeel of oningeligtheid teësinnig bly om sukrose in te sluit in 'n tipe 2 diabetiese dieet, nadat voordele aangedui is uit navorsing soos die, kan dit onder diabete lei tot verwardheid en wantroue in die effektiwiteit van dieet. Die wanopvatting in alle daagse terme dat diabetes bloot 'n "suiker siekte" is en dat sukrose diabetes mellitus veroorsaak, moet gestaak word.

Sleutelwoorde: tipe 2 diabetes mellitus, sukrose, glukemiese beheer, plasma glukose, serum fruktosamien, HbA_{1c} persentasie, dieet kontrole.