EFFECTS OF A TRADITIONAL AFRICAN DIET ON THE METABOLIC CONTROL OF BLACK PATIENTS WITH TYPE II DIABETES MELLITUS

ANNELIE GRESSE, M Sc, R.D.

Thesis accepted for the degree Doctor Scientiae (Dietetics) in the Faculty of Natural Sciences of the Potchefstroomse Universiteit vir Christelike Hoër Onderwys

Promoter: Prof. H.H. Vorster, Department of Dietetics
Co-promoter: Prof. P.H. Joubert, Department of Clinical Research, Hoffmann-LaRoche, Basil, Switzerland

Potchefstroom
1991

Dedicated to

my husband, Johan,

for his encouragement and assistance

and my parents

for their support

ACKNOWLEDGEMENTS

This study could not have been completed without the assistance and support of many people. I, therefore, would like to thank the following:

* my promoter, Prof. H.H. Vorster, who was always ready with advice and assistance. Her encouragement, sacrifices and very competent conduct as promoter made the study a learning and enjoyable experience;

* Prof. P. Joubert of the Department of Pharmacology at MEDUNSA, who was willing to be co-promoter;

* Prof. I.I. Glatthaar of the Department of Human Nutrition at MEDUNSA, who was always willing to give advice and assistance;

* all the patients who took part in the study. Their patience and friendliness made clinic days a pleasure;

* all the persons who were involved in the clinic procedure, and specifically Hazel Saaiman, Marietjie du Plessis, sister Rosinah Lebina, sisters May Bokaba and Christina Segoe of the Ga-Rankuwa hospital diabetic clinic, Leonie Steyn and doctors Nelly Silvis and Christine Venter from the Department of Home Economics and Dietetics of the PU vir CHO;

* all the staff of the different laboratories and departments where samples were analyzed, namely the Departments of Clinical Pathology of MEDUNSA and Pretoria University and the Department of Haematology of MEDUNSA;

* Gretel Crowter and dr. Reinach of the Medical Research Council for the statistical analysis of a large part of the data;

* the staff of the Ferdinand Postma Library, PU vir CHO, for their assistance and services;

* MEDUNSA for permission to undertake the study;

* Prof. Annette Combrink for her services as language expert;

* all my family, friends and colleagues for their interest and encouragement;

* my husband, Johan, for all his sacrifices, assistance and advice; and

* above all, Soli Deo Gloria!

I want to thank the following organizations for their financial assistance and for products donated:

* The South African Sugar Association

* Robertsons (Pty) Limited and specifically Mr. Brain Jakins

* Ruto Mills and specifically Mr. McConnell and Mr. Beukes

* The Maize Board

LIST OF CONTENTS

CHAPTER 4: EATING HABITS AND NUTRIENT INTAKES OF BLACK PATIENTS WITH DIABETES MELLITUS IN THE GA-RANKUWA AREA

LIST OF TABLES AND FIGURES

LIST OF ABBREVIATIONS

%	percent
ADP	adenosine di-phosphate
AP	serum alkaline phosphatase
AST	serum aspartate aminotransferase
ALT	serum alanine aminotransferase
BMI	body mass index
BUN	blood ureum nitrogen
DF	dietary fibre
DM	diabetes mellitus
EDTA	ethylene-diamine tetra acetic acid
FFQ	food frequency questionnaire
GGT	serum gamma-glutamyltransferase
GI	glycaemic index
GIP	gastric inhibitory polypeptide
HbA_{1c}	glycated haemoglobin
HDL	high-density lipoprotein
ICA	islet-cell antibodies
IDDM	insulin-dependent diabetes mellitus
IGT	impaired glucose tolerance
LDH	serum lactate dehydrogenase
LDL	low-density lipoprotein
NIDDM	non-insulin-dependent diabetes mellitus
NSP	non-starch polysaccharides
PA ratio	protein-albumin ratio
P/S ratio	polyunsaturated fatty acids to saturated fatty acids ratio
RDA	Recommended Dietary Allowances from the USA
SMR	second meal response
USA	United States of America
VLDL	very-low-density lipoprotein
WHO	World Health Organisation
WHR	waist-to-hip circumference ratio

NB. All numbers up to 12 are written out
The decimal point (.) and not comma (,) is used.

SUMMARY

Diabetes mellitus has become more prevalent in developing cultures and takes an enormous human and monetary toll each year. Previous research has indicated that the traditional African diet may possibly be the optimal dietary treatment of westernised black non-insulin-dependent diabetes mellitus (NIDDM) subjects. The main hypothesis tested in this study was therefore that the traditional African diet, compared to the westernised diabetic diet as followed by local black people with NIDDM, improves metabolic control, reduces weight, and minimises the risk for macro-vascular complications. The study consisted of three phases.

Firstly, a pilot study was undertaken to develop a suitable food frequency questionnaire and to determine the food and nutrient intakes of the local black NIDDM population. It was found that these patients followed a three-meal-per-day pattern. The distribution of dietary energy was 17 % from protein, 35 % from fat and 48 % from carbohydrate. It became clear that the eating habits of these NIDDM patients were in a process of westernisation. Vitamin and mineral intakes were relatively low. Nutrition education and dietary counselling were recommended.

In the second phase, the acute or short-term effects of a traditional African meal on blood glucose responses were examined. The glycaemic index (GI) and second meal response of a traditional African meal, consisting of maize meal porridge, soya mince and "morogo" (cooked green leaves) were measured in 14 black NIDDM subjects. A standard reference meal of white bread plus tea, was used as control. The GI of the traditional meal was significantly lower in the women than in the men. In the women, it resembled predicted values based on published GI values of individual foods obtained in healthy subjects. A true Staub-Traugott effect (a facilitated glucose disposal during the second meal) was present in both men and women. A second meal response was only observed when the GI of the first meal was low.

Lastly, the long-term effects of a traditional African diet on the metabolic control of black patients with NIDDM were determined. A control group of eight men and 13 women followed an adapted, westernised diabetic diet and a test group of eleven men and 19 women followed a low GI African diet, rich in maize meal porridge, soya and green leafy vegetables for a period of five months.

Results showed that the patients could follow the diet success= fully. The test diet resulted in statistically significant, but not clinically significant weight loss. It did not, however, influence either glycated haemoglobin, nor fructosamine values. It was concluded that dietary intervention will improve glycaemic control possibly only if accompanied by substantial weight loss in these obese NIDDM subjects. Lipid profiles were normal to slightly high, in contrast with the high values repor= ted for white NIDDM patients. The weight loss in the test group was accompanied by small but statistically significant decreases in plasma triglycerides, apolipoprotein B, fibrinogen and total cholesterol (in men). It is possible that the lower GI of the test diet contributed to the improvement in lipoprotein profiles.

It is recommended that:

* a reducing diet with a low energy content as well as a low GI combined with moderate exercise, should be prescribed for overweight black NIDDM patients;
* the high prevalence of hypertension and overweight in these patients should get attention; and
* the energy needs of black obese NIDDM patients, and the relationship between weight and glycaemic control should be investigated further.

From the results of this study it is clear that the dietitian should be an integral part of the medical team and that he/she can play an important role in improving and maintaining quality of life for the NIDDM patient. The insight of the dietitian in the nutritional problems of the DM patient should enable him/her to provide education, motivation and attention, form diagnosis of diabetes, throughout the course of the disease.

OPSOMMING

Die voorkoms van diabetes mellitus onder ontwikkelende volke neem jaarliks toe en het verreikende finansiële implikasies. Vorige navorsing het getoon dat die tradisionale Afrika-dieet moontlik die ideale dieetbehandeling vir swart, verwesterse nie-insulien afhandlike diabetes mellitus (NIADM) pasiënte is. Die belangrikste hipotese van hierdie studie was dan dat die tradisionele Afrika-dieet, in vergelyking met die westerse diabetiese dieet soos wat dit deur die swart NIADM pasiënte in die omtrek gevolg word, metaboliese kontrole sal verbeter, tot gewigsverlies sal lei en die risiko vir makrovaskulêre komplikasies sal verminder.

Eerstens is 'n loodstudie gedoen om 'n geskikte voedselfrekwensie vraelys te ontwikkel waarmee die voedingstofinnames van die plaaslike swart NIADM populasie gemeet kon word. Daar is getoon dat die pasiënte drie maaltye per dag neem. Die energie-verspreiding van die dieet was 17 % vanaf proteïene, 35 % vanaf vet en 48 % vanaf koolhidrate. Dit was duidelik dat die eetgewoontes van die NIADM pasiënte 'n proses van verwestering ondergaan. Die inname van vitamiene en minerale was relatief laag. Voedingonderrig en voorligting in verband met die korrekte dieet is aanbeveel.

Die akute of korttermyn gevolge van 'n tradisionele Afrika-dieet op bloedglukoserespons is tydens die tweede fase getoets. Die invloed van 'n tradisionele Afrika-dieet, bestaande uit mieliemeelpap, gemaalde soja en "morogo" (gekookte groen blare), op die glukemiese indeks (GI) en die glukemiese respons tydens die volgende maaltyd is in 14 swart NIADM pasiënte gemeet. As kontrole is 'n standaard verwysingsmaaltyd van wit brood en tee gebruik. Die GI van die tradisionele maaltyd was betekenisvol laer in vroue as in mans. Die waardes het die voorspelde waardes, gebaseer op gepubliseerde glukemiese indekse vir die individuele voedselitems soos gemeet in gesonde individue, in die vroue weerspieël. 'n Ware Staub-Traugott effek ('n gefasiliteerde glukoseverbruik tydens die tweede maaltyd) was in

mans en vroue aanwesig. Die respons op die tweede maaltyd is alleenlik na 'n eerste maaltyd met 'n lae glukemiese indeks waargeneem.

Laastens is die langtermyn gevolge van 'n tradisionele Afrika-dieet op die metaboliese kontrole van swart pasiënte met NIADM ondersoek. 'n Kontrolegroep van agt mans en 13 vroue het 'n aangepaste, westerse diabetiese dieet gevolg. 'n Toetsgroep bestaande uit elf mans en 19 vroue het 'n Afrika-dieet met 'n lae glukemiese indeks, bestaande uit mieliemeelpap, soja en groen blaargroente vir 'n periode van vyf maande gevolg.

Die resultate het getoon dat die pasiënte suksesvol by die dieet kon hou. Die toetsdieet het tot 'n statisties betekenisvolle, maar nie klinies betekenisvolle, gewigsverlies gelei. Die dieet het egter nie 'n invloed op geglukeerde hemoglobien of fruktosamien waardes gehad nie. Die gevolgtrekking is gemaak dat 'n dieetingryping moontlik net glukemiese kontrole sal verbeter indien dit gepaard gaan met 'n groot afname in gewig. Lipiedvlakke was normaal tot effens hoog, in teenstelling met die hoë waardes wat vir blanke NIADM pasiënte gerapporteer word. Die gewigsverlies in die toetsgroep het gepaard gegaan met klein maar statisties betekenisvolle verlagings in plasma trigliseriede, apolipoproteïen B, fibrinogeen en totale cholesterol (in die mans). Die laer GI van die toetsdieet het moontlik bygedra tot die verbetering in die lipoproteïenwaardes.

Daar is aanbeveel dat:

* 'n verslankingsdieet met 'n lae energie-inhoud, sowel as 'n lae GI, gekombineer met matige oefening, vir oorgewig swart NIADM pasiënte voorgeskryf word;
* die hoë voorkoms van hipertensie en oorgewig in hierdie pasiënte aandag moet geniet; en
* dat die energiebehoeftes van swart vetsugtige NIADM pasiënte, sowel as die verband tussen gewig en glukemiese kontrole verder ondersoek behoort te word.

Die resultate van hierdie studie toon duidelik dat die dieetkundige 'n integrale deel van die mediese span behoort te wees. Die dieetkundige kan 'n belangrike rol speel in die verbetering en behoud van lewenskwaliteit vir die NIADM pasiënt. Die insig van die dieetkundige in die voedingsprobleme van die pasiënt met diabetes mellitus behoort hom/haar instaat te stel om die nodige voorligting, motivering en aandag tydens die verloop van die siekte, aan die pasiënt te gee.

CHAPTER 1

INTRODUCTION

1.1 BACKGROUND

Diabetes mellitus (DM) takes an enormous human and monetary toll each year (Anderson, et al., 1987). It is a common disease in affluent societies, affecting from one to three percent (%) of populations, and often five to ten percent of those over 40 years of age. It is estimated that 60 million people worldwide suffer from DM (Brownlee, 1985:185). In the developing nations of the world alone, there are probably a total of between 25 and 50 million diabetics (Bennet, 1983:55).

Non-insulin-dependent diabetes mellitus (NIDDM) is currently more common in lower socioeconomic groups of affluent societies (Hamman, 1983). With progressive westernisation, it has become more prevalent in developing cultures. NIDDM contributes to 85 - 90 % of diabetics in both developed and developing countries (Zimmet, 1982:400).

Information on the incidence of DM in Africa is scarce (Królewski & Warram, 1985), but there are indications that the incidence of NIDDM is rising among blacks (Zimmet, 1982). At present, one in 33 South Africans has diabetes. Out of the more than 690 000 diagnosed NIDDM cases in this country, 400 000 are urban blacks (Seedat, 1989a:18). The incidence of diabetes is unknown in rural areas, which means that the number of NIDDM cases may be drastically underestimated.

Most of the metabolic abnormalities of diabetes are associated with the long-term vascular complications of the disease (Banga & Sixma, 1986; Kostner & Karadi, 1988). Deaths from coronary heart disease among individuals with diabetes are two to three times more frequent than among those who do not have diabetes (Steiner, 1981). Progress in diabetes management into a sphere

beyond that of simple survival from ketoacidosis, together with an increase in longevity, has led to increased emphasis on research in the prevention of these long-term complications (Crapo & Vinik, 1987). Since the atherosclerotic process is accelerated in diabetes, maintenance of desirable serum lipid levels should be one of the primary goals in diabetes management. These atherosclerotic abnormalities are probably reversible with the correction of hyperglycaemia, known to be associated with raised serum lipid levels (Fehily et al., 1982; Jones & Peterson, 1981; Reaven, 1987).

Hyperglycaemia in most NIDDM subjects should first be treated with a diabetic diet alone (Cooppan, 1987). Current treatment too often revolves around insulin and drug therapy, neglecting diet and exercise. It is especially the oral hypoglycaemic agents that are abused. They are too often used as a substitute rather than a support for diet and exercise therapy (Anderson, et al., 1987). The modern diabetic diet, as prescribed by various expert organisations throughout the world, has a nutrient composition very similar to the typical traditional diet of the rural blacks in South Africa (Silvis, 1989). The diet is usually generous in dietary fibre (DF) and complex carbohydrates and restricted in fat. This type of diet has been shown to lower insulin requirements, decrease serum cholesterol and triglyceride levels (Anderson, 1986), increase peripheral tissue insulin sensitivity (Hjollund, et al., 1983), aid in weight control (Anderson & Sieling, 1981) and lower blood pressure (Anderson, 1983). Therefore, Silvis (1989) suggests that this traditional diet may possibly be the optimal dietary treatment of westernised black NIDDM subjects.

1.2 HYPOTHESIS AND OBJECTIVES

The main hypothesis to be tested in this study was whether the traditional African diet, compared to the westernised diabetic diet as followed by local black people with NIDDM, improves metabolic control, reduces weight, and minimises the

biochemical risk markers of the macro-vascular complications of the disease.

The following hypotheses were investigated to support the main hypothesis:

* The current diet of black NIDDM patients in the Ga-Rankuwa area does not comply with the recommendations for DM, except for a restriction in sugar intake.
* The glycaemic index (GI) of the traditional African diet is low.
* It is possible for black diabetics in the region of Ga-Rankuwa (some urbanised, some rural) to follow the traditional African diet.
* Nutrition intervention and attention, even to a small extent, will contribute to improvements in weight, metabolic control and risk markers of macro-vascular complications in black diabetics who visit an outpatient clinic.

The main objective of this study was therefore to investigate the ability of the traditional African diet to improve metabolic control and minimise the risk markers of macro-vascular complications in a group of black NIDDM patients.

In order to meet this objective, the following were examined:

* The current diet eaten by the general diabetic population in the Ga-Rankuwa area.
* The nutrient composition of the diets currently eaten by the NIDDM patients in this area.
* The adaptation of the traditional African diet to be more convenient, quicker to prepare, and to improve acceptability by the local diabetic population.
* The GI and nutrient composition of the adapted traditional African diet.
* The status of metabolic control of NIDDM patients in the Ga-Rankuwa area.
* The introduction of the adapted traditional African diet to

the NIDDM patients and their motivation to comply.

1.3. STRUCTURE OF THE THESIS AND STUDY

* Literature review

The second chapter of the thesis consists of a review of the relevant literature. A brief summary of the metabolism and biochemistry of NIDDM and a discussion of complications, abnormalities and risk markers of complications are given. The treatment of NIDDM, and more specifically the dietary treatment, is discussed. The use of the GI in the diabetic diet is reviewed extensively because the diet used in this study has been designed according to the GI of individual food items. The traditional African diet and nutrient intakes of South African blacks are reviewed. Background information on anthropometrical measurements used is given. The questionnaire applied to determine the eating habits of the NIDDM subjects is an important part of the study and is discussed in more detail.

In Chapter 3, materials and different methods that were used during the study are discussed in detail.

The study consisted of three phases. The general methodology, results, discussion and conclusions of each phase are presented in the following three chapters in a format of three separate publications.

* Development of a questionnaire and survey of the usual nutrient intake of a random sample of black non-insulin-dependent diabetes mellitus patients

The most practical way to determine what people eat is to interview them, using a structured food frequency questionnaire (FFQ) (Krall & Dwyer, 1987; Medlin & Skinner, 1988; Mullen et al., 1984). It was necessary for the planning of the main study to determine the regional meal pattern, food and nutrient intake

of the black diabetic and how it differs from the traditional rural diet and standard westernised diabetic diet. A FFQ was therefore drawn up, standardised and used to determine the usual nutrient intake of a random sample of NIDDM patients from the diabetic clinic at Ga-Rankuwa Hospital. The results of this phase of the study are reported and discussed in Chapter 4.

* Determination of the GI of a meal in the traditional diet in a random sample of non-insulin-dependent diabetes mellitus patients

One of the major aims of diabetes therapy is to maintain euglycaemia (American Diabetes Association, 1987). Many studies have found that different foods produce different glycaemic responses despite having the same nutrient composition (Jenkins, et al., 1983a). The GI has been proposed as a method of classifying the blood glucose responses to food (Jenkins, et al., 1983a). The use of low-GI foods may help to improve blood glucose control in diabetics and reduce insulin secretion and circulating blood lipids. For these reasons it was necessary to determine the GI of the meals that were to be used in the main study to ensure that the adapted traditional African diet has a lower GI than the standard diabetic diet used at Ga-Rankuwa Hospital that was eaten by the control group. The GI as well as the "second meal response" (SMR) of the traditional African meal in these subjects is discussed in Chapter 5.

* Measurement of long-term effects of the traditional diet on metabolic control and risk markers of macro-vascular complications in non-insulin-dependent diabetes mellitus patients

For the third phase of the study a random sample was drawn from the patients that regularly visit the Ga-Rankuwa diabetic clinic. They were seen each month during visits to the clinic. The ingredients of the adapted traditional African diet were given to the test or experimental group. The control group only

received some of the ingredients of their habitual diabetic diet, but was treated exactly the same as the test group. Motivation to comply with dietary instructions was done during each visit and the dietary intakes checked monthly.

In addition, anthropometry, blood pressures, metabolic control (blood glucose, glycated haemoglobin (HbA_{1c}), fructosamine) and levels of biochemical risk markers of macro-vascular complications (serum lipids and lipoproteins, plasma coagulation factors, serum enzymes, proteins, minerals, etc.) were monitored at baseline and after one and five months on the diets.

Results of this third phase of the study are reported and discussed in Chapter 6.

* In Chapter 7, the salient observations of all three phases of the study are discussed, conclusions are drawn and recommendations for further research are made.

CHAPTER 2

LITERATURE REVIEW

2.1 INTRODUCTION

DM is a chronic disease in which the course develops over many years or even decades. It therefore has a tremendous influence on the quality of life and life-style of people who suffer from the disease. In this chapter current knowledge on DM is reviewed in order to determine what could be done to improve the quality of life and longevity of DM patients.

The background of DM (definition, classification, epidemiology, pathophysiology) will be discussed in section 2.2. Complications of DM and risk markers of these complications will then be discussed in section 2.3. Thirdly, current concepts in the treatment of DM will get attention in section 2.4. The GI is the main concept used in the design of the study and section 2.5 will deal with various aspects of the GI. In section 2.6 the present diet and nutrient intake of the black population in South Africa are reviewed. Anthropometric measurements, an important aspect of nutrition research, will be reviewed in section 2.7 Lastly, the development of a FFQ is discussed in section 2.8, because the dietary intake of the participating patients forms an important part of the study.

2.2 DEFINITION, CLASSIFICATION, EPIDEMIOLOGY AND PATHOPHYSIOLOGY OF DIABETES MELLITUS

2.2.1 Definition

Recent reviews (Cahill, 1985:3; Kahn, 1985) define diabetes as a complex, heterogeneous syndrome characterized by:

* hyperglycaemia secondary to deranged secretion and/or action of insulin, for example a relative insulin deficiency;

* specific micro-vascular complications, including thickening of capillary basement membranes, retinopathy, neuropathy and nephropathy;

* macro-vascular complications, for example accelerated atherosclerosis, and a variety of other complications - it doubles the risk for stroke, increases the risk for heart attacks two-fold to three-fold and 50-fold for peripheral vascular problems, particularly in the feet;

* in the untreated state there is accelerated catabolism of both fat and protein;

* it tends to run in families.

2.2.2 Diagnostic criteria

Various sets of diagnostic criteria have been proposed for diabetes but there is still controversy regarding the lack of comparability between criteria. The diagnostic criteria as suggested by the World Health Organisation (WHO) Expert Committee for DM are presented in Table 2.1.

The criteria of the American Diabetes Association are presented in Table 2.2

Table 2.1 World Health Organization diagnostic criteria for diabetes mellitus

	Glucose concentration		
	Venous whole blood	Capillary whole blood	Venous plasma
Diabetes mellitus			
A. Fasting	≥ 120 mg/dl (6.7 mmol/l)	≥ 120 mg/dl (6.7 mmol/l)	≥ 140 mg/dl (7.8 mmol/l)
B. 2 h after glucose load	≥ 180 mg/dl (10.0 mmol/l)	≥ 200 mg/dl (11.1 mmol/l)	≥ 200 mg/dl (11.1 mmol/l)
Impaired glucose tolerance (IGT)			
A. Fasting	< 120 mg/dl	< 120 mg/dl	< 140 mg/dl
B. In between	≥ 180 mg/dl	≥ 200 mg/dl	≥ 200 mg/dl
C. 2 h after glucose load	>120,<180 mg/dl	>140,<200 mg/dl	>140,<200mg/dl

(WHO Study Group on Diabetes, 1985:15).

Table 2.2 Diagnostic criteria for Diabetes Mellitus of the American Diabetes Association

Diabetes mellitus - adults

1. Unequivocal elevation of plasma glucose ≥ 200 mg/dl and classic symptoms
2. Fasting plasma glucose ≥ 140 mg/dl on two occasions
3. Fasting plasma glucose < 140 mg/dl and two-hour plasma glucose of ≥ 200 mg/dl with one intervening value of ≥ 200 mg/dl after a 75 g glucose load

Impaired glucose tolerance

1. Fasting plasma glucose of < 140 mg/dl and a two hour plasma glucose of ≥ 140 mg/dl and < 200 mg/dl with one intervening value of ≥ 200 mg/dl after a 75 g glucose load.

(American Diabetes Association, 1990c:3).

General agreement has been reached that a 75 g oral glucose load should be used for diagnosis in adults (in a minimum of 250 ml of water) with loads of 1.75 g/kg ideal body weight in children. A maximum of 75 g is advised (Marble & Ferguson, 1985: 334).

2.2.3 Classification

Correct classification of diabetic subjects at the time of diagnosis is often difficult but clearly of importance in the decision about the correct treatment (Hother-Nielsen et al., 1988). Various classification schemes have been proposed. Most of the literature that has accumulated regarding the epidemiology of diabetes has used only one manifestational criterion, namely age at onset (Królewski & Warram, 1985).

According to current concepts, there are two broad groups of DM. Insulin-dependent diabetes mellitus (IDDM) or type I (with abrupt onset of classic symptoms such as polydipsia, polyuria, polyphagia and weight loss, ketosis proneness, insulin deficiency), almost always ends in total insulin deficiency. NIDDM or type II (by far the more common type with an insidious onset with relatively few or no symptoms) does not always have correlations with histocompatibility genes, viruses or auto-immunity and usually has some remaining beta-cell function. NIDDM patients often require insulin but are not dependent on insulin for life. In the latter type there is a much greater tendency to obesity and it is usually controllable with a restricted diet with or without oral hypoglycaemic agents. The two types differ both in clinical presentation and presumed etiology and both can be subdivided into smaller subgroups which may or may not be variations of the two general types (American Diabetes Association, 1990c; Cahill, 1985; Hother-Nielsen et al., 1988; Kahn, 1985).

The above-mentioned guidelines are vague and may be difficult to apply in practice. More precise criteria are needed. However, up to 75 % of patients may be classified correctly by experienced

endocrinologists on the basis of age of onset and percentage of the desirable body weight (Hother-Nielsen, 1988:536).

The most reliable parameter in classifying diabetes into IDDM or NIDDM and determining whether the NIDDM patient is insulin-requiring, is evaluation of beta-cell function. This can be done either fasting or by way of the glucagon-stimulated C-peptide concentration measurement. A low C-peptide level suggests that a patient is insulin-requiring. A high C-peptide level in a patient treated with diet and oral hypoglycaemic agents and displaying poor metabolic control, suggests that diet instructions are not followed and that compliance should be reinforced (Sarlund et al., 1987). In IDDM 100 % of patients have residual beta-cell function in the first year after diagnosis, declining to approximately 15 % after five years, after which the beta-cell function is minimal. In NIDDM no consistent impairment of beta-cell function is seen in relation to the duration of diabetes. However, a progressive deterioration of beta-cell function can in time be observed in subgroups of NIDDM patients (Hother-Nielsen, 1988; Madsbad, 1990:95).

Kahn (1985) mentions that the simple binary classification is useful, includes most diabetics, provides a comfortable concept to use for describing these complex syndromes and can be used as the only classification in everyday practice. The condition of many patients, however, appears to lie between these two extremes and may be difficult to classify.

The recommendations of the WHO (1985:15), the National Diabetes Data Group (1979: 3) of the American Diabetes Association, and other diabetic associations have resulted in a proposed classification of DM and other degrees of glucose intolerance. Table 2.3 is a summary of this proposed classification of DM as well as adaptations by Hother-Nielsen et al. (1988:534), Kahn (1985:43), Marble & Ferguson (1985:333, 348, 349) and Tuomilehto & Wolf (1987).

Table 2.3 Classification of Diabetes Mellitus and other states of glucose intolerance

---

I. Clinical types
   1. Diabetes mellitus
      A. IDDM or Type I (formerly juvenile-onset diabetes)
      B. NIDDM or Type II (formerly maturity-onset diabetes)
         a. Non-obese
         b. Obese
         c. NIDDM of the young (MODY)
      C. Other types (intermediate types)
      D. Secondary diabetes
         a. Pancreatic disease (chronic pancreatitis, haemochromatosis, pancreatectomy, etc.)
         b. Disease of hormonal aetiology (Cushing's syndrome, acromegaly, pheochromocytoma)
         c. Drug or chemically induced conditions (chlorothiazide, phenytoin etc.)
         d. Insulin receptor abnormalities (acanthosis nigricans, lipodystrophy, etc.)
         e. Certain genetic syndromes (ataxia telangiectasia, progeria, Laurence-Moon-Biedl syndrome, myotonic dystrophy)
         f. Miscellaneous
   2. Impaired glucose tolerance (formerly chemical diabetes)
      A. Non-obese
      B. Obese
      C. Impaired glucose tolerance associated with certain conditions and syndromes
   3. Gestational diabetes

II. Normal glucose tolerance but substantially increased risk of developing diabetes
   1. Previous abnormality of glucose tolerance (formerly "latent" diabetes)

Table 2.3 continued

2. Potential abnormality of glucose tolerance (formerly "pre-diabetes"

III. Non-diabetic meliturias

1. Glucosuria
   - A. Renal glucosuria
   - B. Due to hyperactivity of endocrine glands other than pancreas
   - C. Due to stimulation of intracranial centres
   - D. Alimentary glucosuria
   - E. Due to infections etc.
   - F. Due to chronic or degenerative conditions
   - G. Due to chemical agents
2. Chronic essential pentosuria
3. Fructosuria
   - A. Essential
   - B. Hereditary intolerance
     - a. Fructose-1-phosphate aldolase deficiency
     - b. Fructose-1-6-diphosphatase deficiency
4. Lactosuria
5. Galactosuria
6. Sucrosuria

For the purpose of this study, diabetes which occurs in adults will be considered as equivalent to NIDDM because other cases are rare in adult populations of 30 years and older (Cahill, 1985). According to Asmal et al. (1981) 1.6 % of blacks with NIDDM in their study actually have this disease before 30 years of age and although it is a milder form than real NIDDM, it can be classified with NIDDM.

2.2.4 The epidemiology of non-insulin-dependent diabetes mellitus

Data are scarce on the incidence of NIDDM in populations below the age of 30. The extent to which undiagnosed NIDDM exists in

this age group is also unknown, as well as how long such cases would remain undiagnosed (Królewski & Warram, 1985).

Brownlee (1985:185) estimates that 60 million people worldwide have DM. Approximately 1.5 to two million persons in the United States of America (USA) are treated with insulin, one to 1.5 million are treated with oral hypoglycaemic agents and possibly another three million are treated with diet alone. According to Brownlee (1985:185) there may be an additional four or more million with varying degrees of asymptomatic glucose intolerance in addition to these six to seven million mentioned above. In the developing nations of the world, there are probably a total of between 25 and 50 million diabetics (Bennet, 1983:55).

NIDDM contributes to 85 - 90 % of diabetics in both developed and developing countries (Zimmet, 1982:400). There are marked differences in prevalence between different ethnic groups in the same country and between people of the same ethnic group undergoing internal or external migration (Hamman, 1983). It seems as if NIDDM was rare in many populations in the past, particularly for those having a traditional non-western life-style. With progressive modernization NIDDM has become more prevalent, especially where significant changes in life-style have taken place during a relatively short time-span (Bennet, 1983). Thus, there may be an underlying genetic susceptibility to NIDDM in some populations which is unmasked by environmental factors (Zimmet, 1982), which operate independently of obesity. Possible factors that may play a role are, *inter alia*, exercise and diet (Królewski & Warram, 1985).

NIDDM is a common disease in affluent societies, affecting from one to three percent of populations, and often five to ten percent of those over 40 years of age. NIDDM and its associated mortality are currently more common in lower socioeconomic groups in affluent societies (Hamman, 1983). Prevalence rates are lower in people with higher education levels, regardless of sex or obesity, and the difference is more evident in whites than in

blacks in the USA. Low levels of education or income are also associated with higher incidence and prevalence rates for hypertension. There is no obvious explanation for these associations (Królewski & Warram, 1985).

The number of subjects suffering from NIDDM varies by as much as five-fold to ten-fold between societies. Methodological differences, however, make precise estimates difficult (Hamman, 1983:39). For example, incidence rates can be as low as two percent in countries such as Greenland and as high as 35 % in the American Pima Indians (Zimmet, 1982:401). These Indians, together with the South African Indians, have the highest diabetic prevalence in the world (Anon, 1963). Incidence rates of NIDDM in various countries are summarised in Table 2.4.

Table 2.4 Incidence rates per 1 000 per year of non-insulin-dependent diabetes mellitus in other countries

Population		Age group	
		40 - 49	50 - 59
Whites, USA	1960's	2.5	4.5
	1970's	3.1	5.8
Blacks, USA	1960's	3.5	7.0
	1970's	7.6	8.5
United Kingdom	1960's	0.8	1.6
Norway	1960's	0.3	0.7
Sweden	1960's	-	2.8
Israel	1960's	6.0	10.0
Nauru, Micronesia	1970's	15.1	40.5
Pima Indians, Arizona	1960's	57.0	49.9

Adapted from Królewski & Warram (1985:30,32)

The data for the USA population as given above indicate a steady increase in the prevalence of NIDDM in all age groups from the 1960's to the 1970's. The largest increases were for diabetes treated with diet or no treatment at all. These increases may

partly be due to a more widespread and sensitive ascertainment of asymptomatic cases which had been overlooked previously, or from a broadening of the criteria for the diagnosis to include lesser degrees of glucose intolerance. Doubts can, therefore, be raised about the conclusiveness of the evidence for an increase in the disease itself (Królewski & Warram, 1985).

The increasing number of cases of NIDDM can possibly also be ascribed to an increasingly older population (Hamman, 1983:39). It is clear that the incidence rates increase markedly with age. However, the substantial variability in the occurrence of NIDDM among Caucasians points to an important environmental component in the development of the disease (Hamman, 1983).

Other factors which may influence incidence rates are race, sex and obesity. Available data are not sufficient to draw any conclusions, but it may seem that NIDDM is significantly more frequent in women than in men (Jackson & Huskisson, 1965).

It also seems as if newly diagnosed NIDDM patients have a higher prevalence of obesity than non-diabetics. For example, in the well-known Framingham study, individuals with a relative weight of 140 % or higher than ideal body weight, according to the 1959 Metropolitan Life Insurance Tables, had a risk of diabetes 2.3 times higher than individuals with ideal body weight (Królewski & Warram, 1985). The finding that obesity is an important risk factor for NIDDM suggests that some of the variation in the occurrence of NIDDM according to age, race, nationality and age of onset might be due to variation in the occurrence of obesity, although these other factors may also cause an increase quite independently of obesity (Cahill, 1985).

There may also be seasonal differences in diagnosis and severety with fewer cases diagnosed and less severe symptoms in the summer (Christau et al., 1977).

2.2.5 Diabetes in black South Africans

DM is a common metabolic disorder in the South African population. It has been estimated that in excess of four percent of the population have DM (Huddle, 1989:11). That means that one in 33 people have the disease (Seedat, 1989a:18). All population groups are affected, although to different degrees (Huddle, 1989). Table 2.5 indicates the size of the problem in South Africa.

Table 2.5 Incidence of impaired glucose tolerance and diabetes mellitus in South Africa

Population group	Impaired glucose tolerance 1970's		Diabetes mellitus 1989		1970's	1960's
	%	Numbers	%	Numbers	%	%
Indians	6	31 200	10	52 000	16.7	10.4
Whites	?	?	4	105 000	3.6	
Mixed group	?	?	8.7	135 000	10.7	6.6
Blacks						
Urban	7	700 000	4.2	400 000	4.1	3.6
Rural	?	?	?	?	?	

(Jackson, 1978a:111; Marine et al., 1969:854; Seedat, 1989a:18).

The true prevalence of DM in the South African black population is, therefore, unknown. However, it seems as if DM was uncommon in rural blacks in the sixties and seventies (Seftel, Keeley & Walker, 1963), except in affluent sub-sections such as the royal families (Jackson, 1978a). It is not clear what the situation is at present.

In contrast with rural areas, the urban black population have at least the same incidence, if not higher, than whites (there were approximately 400 000 black diabetics in 1989 in South Africa (Seedat, 1989a:18), which may indicate a genetic potentiality for

NIDDM in blacks (Jackson, 1978b). According to Seftel, Keeley and Walker (1963), twice as many black women than men have NIDDM and the majority are overweight with a relatively mild NIDDM, infrequent ketosis and a tendency to insulin resistance. Ketosis is still the major cause of death in the African diabetic. The main reason for the high mortality rate is the late presentation and frequency of associated complications such as infection or liver disease.

Not all black diabetics are diagnosed as having diabetes in an early stage of the disease. It would appear that, at least among some black groups, hyperglycaemia may be frequent, but without symptoms and without general awareness of the condition of diabetes. Glucosuria is also not present in many of black diabetics (Jackson, 1978a).

2.2.6 Pathogenesis of non-insulin-dependent diabetes mellitus

2.2.6.1 **Risk factors for non-insulin-dependent diabetes mellitus**

Three main groups of risk factors for NIDDM have been identified:

* Factors involving fundamental biology, for example family history of NIDDM;

* factors involving biochemical and physiological mechanisms that may also be influenced by environmental factors, such as blood pressure, blood glucose, glutamic pyruvic transaminase, bilirubin, uric acid and lactate at rest; and

* factors involving the social environment and life-style such as obesity (Ohlson et al., 1988).

Other factors like smoking habits, alcohol consumption, physical fitness and experience of stress may be aggravating factors but do not predict NIDDM (Ohlson et al., 1988).

It has been shown, however, that risk factors may be heterogeneous in their effect upon different population and ethnic groups and between the sexes within groups. An assessment of risk variables operating in a given target community may thus be of value in the initial phase of a NIDDM prevention or control programme (King et al., 1984). This fact should be taken into consideration in the discussion of risk factors for NIDDM.

2.2.6.2 Differences in pathogenesis of insulin-dependent and non-insulin-dependent diabetes mellitus

The pathogenesis of NIDDM is less clear and more controversial than that of IDDM. It is genetically influenced since it occurs in identical twins with almost total concordance (Brownlee, 1985). It is also associated with obesity in more than 80 % of patients, suggesting the possibility that this type of diabetes may be due to a disordered mechanism of appetite regulation or energy expenditure (Kahn, 1985). Most importantly, in contrast to patients with IDDM, NIDDM patients have considerable preservation of the beta-cell mass (about 50 % of the normal level) and often secrete substantial quantities of insulin (Kahn, 1985). There may thus be a resistance of the peripheral tissues to respond to insulin. There is still considerable controversy as to which of the two factors, decreased insulin secretion or insulin resistance, is the major one in the pathogenesis of NIDDM. It seems likely that there is an interplay of both factors, as can be seen in Figure 2.1 (Kahn, 1985).

NIDDM seems to be a slowly developing disease with a long sub-clinical phase. There is no agreement as to the nature of the lesion responsible for the development of NIDDM. The sub-clinical manifestation is nonspecific since a substantial number of such individuals revert to normoglycaemia or remain for many years without progression to NIDDM (Królewski, Warram & Christlieb, 1985).

NIDDM is often seen as a disease of the rich. In many developing

nations, the disease has been reported to be more common in the ruling or upper-class families (Zimmet, 1982). It seems as if there is a spectrum of interaction between genetic susceptibility and environmental factors to produce NIDDM. In certain situations, the genetic role is much more powerful and environmental factor input probably negligible, or *vice versa* (Zimmet, 1982).

Zimmet (1982) described the Neel theory that people with a hereditary tendency to NIDDM are better able to store energy efficiently, permitting survival under conditions of alternating feast and famine. It is possible that this hereditary factor was necessary for preserving population numbers in response to varying food availability. At present this factor is of little use and the introduction of enough food on a regular basis leads to clinical NIDDM. One of the mechanisms suggested for these factors is an over-responsive beta-cell which promotes fat and glycogen deposition for more efficient utilization of foodstuffs. With continual over-stimulation, the beta-cells may lose their capacity to respond, resulting in NIDDM. Another hypothesis puts the emphasis on a genetically determined 'down regulation' of insulin receptors in response to repeatedly high levels of circulating insulin. With persistent hyperinsulinaemia, a vicious circle is created, whereby the beta-cells eventually fail in the face of loss of insulin sensitivity and the production of insulin resistance.

Some researchers feel that, when the mean prevalence of diabetes is high in a population group, some apparently normal spouses of affected family members might be contributing diabetogenic genes to the family. They feel, therefore, that it is crucially important that all members of the family, including spouses, are tested for impaired glucose tolerance (IGT) in such groups (O'Rahilly, Wainscoat & Turner, 1988).

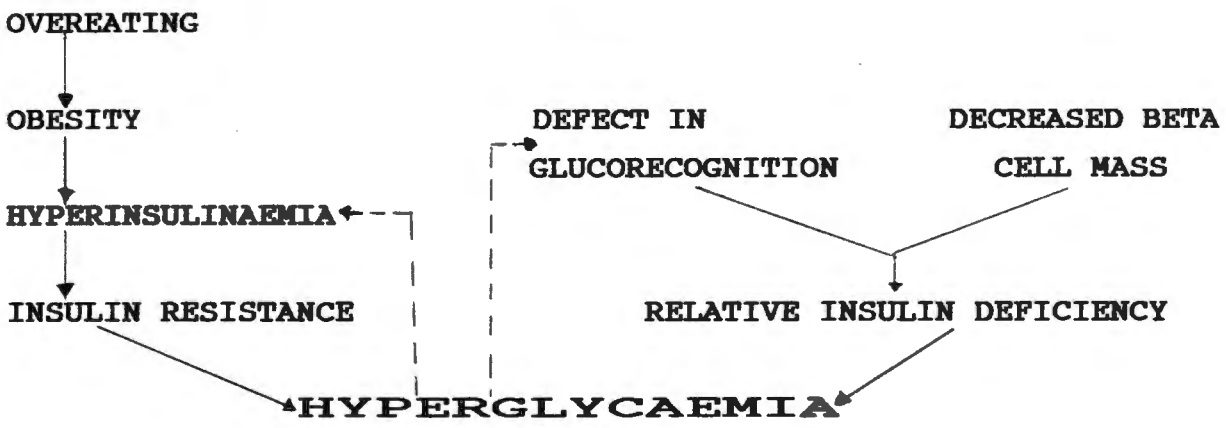


Figure 2.1 Possible pathogenesis of NIDDM (Kahn, 1985:47).

There is controversy concerning the relationship of the various complications of diabetes with hyperglycaemia. Whatever the exact order of events, the most striking early abnormality in both IDDM and NIDDM is the development of hyperglycaemia, which may be considered the result of a relative deficiency, an absolute lack of insulin or resistance to insulin action at the target-cell level (Kahn, 1985).

There may also be differences in pathogenesis between different population groups. According to Thorburn, Brand and Truswell (1987), some indigenous populations such as the Aborigines in Australia may have evolved without the ability to cope with the fast-release carbohydrate loads typical of contemporary western diets. Such responses may result from a "thrifty gene", which once enabled hunter-gatherers and other populations to survive food shortages by efficiently depositing fat during times of food abundance (Zimmet, 1982). The "thrifty" metabolism tends to promote hyperinsulinaemia, obesity and eventually NIDDM with the constant abundance of food in urbanised populations. People with a "thrifty" metabolism have an efficient system for converting dietary protein into glucose and fat as readily available forms of energy in both the short and long term and an efficient system for fat accumulation to take advantage of any feast periods (O'Dea et al., 1988). NIDDM may take years to develop in an urban environment, but O'Dea (1984) has shown that the disease

is markedly improved in diabetic aborigines if they revert to a traditional diet and life-style, even for as few as seven weeks. Although weight loss, exercise, and the low fat content of the traditional diet were important in these improvements, the slow-release nature ("lente") of the carbohydrates probably also played a role. This "thrifty gene" hypothesis may possibly also apply to the South African blacks.

2.2.6.3 **Insulin secretion**

There is little understanding of the defect in islet-cell function in NIDDM. Basal insulin levels in NIDDM are normal, or even elevated. Several different varieties of abnormalities have been identified in insulin secretion after stimulation of the beta-cell. Usually there is a loss of acute phase insulin release but not in other stages, a fact that suggests a specific defect in glucorecognition. The lack of glucorecognition is linked to the level of hyperglycaemia. It is probably a reversible phenomenon in the early phases of the disease - if blood glucose is lowered by treatment with a diet, oral hypoglycaemic agents or insulin, beta-cell function improves and insulin secretion is increased (Kahn, 1985).

The decrease in insulin secretion in obese people is noteworthy. In obese people there are decreased insulin responsiveness and post-receptor, intracellular lesions of glucose metabolism. In addition there is decreased insulin sensitivity associated with decreased insulin binding, especially in obese people with NIDDM (Berger & Berchtold, 1985).

2.2.6.4 **Insulin resistance**

It is clear that NIDDM is associated with a decrease in response to exogenous or endogenous insulin. This is most marked in patients with obesity, but also occurs in non-obese NIDDM patients.

A summary of possible causes of insulin resistance is given in Table 2.6

Table 2.6 Possible causes of insulin resistance

1.	*Target tissue defects*	
	Insulin receptor defects:	decreased number of receptors (with increase in receptor turnover)
		decreased affinity of the receptor for insulin
	Post-receptor defects:	abnormal tyrosine kinase activity
		abnormal receptor units
		abnormality in receptor transduction function
		abnormality in any of the intracellular steps of insulin action
2.	*Circulating antagonists*	
	Increased concentrations of catabolic hormones (glucagon, cortisol, growth hormone, catecholamines)	
	Increased concentrations of free fatty acids and 'ketone bodies'	
	Anti-insulin antibodies	
	Anti-insulin receptor antibodies	

(Adapted from Alberti & Hockaday, 1987: 9:64; Kahn, 1983:326 - 335)

The resistance can occur as a result of defects at several levels in the action of insulin. There may be a decrease in the number of insulin receptors (known as receptor down-regulation) in the presence of increased circulating insulin, with the functionality of the remaining receptors unaltered (Berger & Berchtold, 1985; Kahn, 1983). This defect appears to be due to a regulatory

effect of insulin on its own receptor concentration. Loss of protein-tyrosine kinase activity may play a role in this defect (Russel et al., 1987).

There may also be post-receptor defects in one of the intracellular steps of insulin action. However, the exact nature of these possible defects remains unclear. One possibility is a defect at the level of the receptor, which involves signal transduction rather than binding capacity. The defect appears to be related to the degree of metabolic abnormality. Treatment with diet or insulin will substantially reverse this defect, even when the receptor abnormality persists (Kahn, 1985). Weight reduction programmes, improvement of metabolic control and certain drugs usually enhance insulin sensitivity, restore beta-cell responsiveness to glucose and improve insulin binding (Berger & Berchtold, 1985).

2.2.6.5 **The role of hormones other than insulin**

Several hormones, recently reviewed by Ganda (1985a), may contribute to the pathophysiology of DM.

* Alpha-cell dysfunction exists in diabetes. It is, however, not clear whether the alpha-cell dysfunction is a primary or a secondary defect. Considerable controversy also exists on the questions as to whether glucagon is essential in the pathogenesis of diabetes and whether the suppression of glucagon might help improve control of diabetes.

* Circulating growth hormone levels are elevated in uncontrolled diabetics. The effects of long-term elevation of growth hormone levels may have a role in the pathogenesis of the macro-vascular complications of DM.

* The role of catecholamines in the regulation of insulin secretion and in the pathogenesis of diabetes remains controversial.

* A number of reports have shown abnormalities of gastric inhibitory polypeptide (GIP) secretion in patients with obesity and/or NIDDM.

* Other hormones that may play a role in diabetes and which are under investigation are somatostatin, pancreatic polypeptide, secretin, cholecystokinin, gastrin, the endorphin family, neurotensin, corticotrophin, glucocorticoids, prolactin and the prostaglandins.

2.2.6.6 **The immune system**

The presence of islet cell antibodies (ICA) in diabetics has been the focus of widespread interest. Kaldany, Busick & Eisenbarth (1985) mention that the prevalence of NIDDM varies from six to eight percent. It appears that NIDDM with circulating ICA represents an earlier stage of a disease process; culminating in IDDM. Circulating ICA in patients with NIDDM could therefore predict future insulin dependence.

Significant positive associations with NIDDM have been reported for various histocompatibility antigens in certain ethnic groups such as the Xhosas in South Africa (Briggs et al., 1980).

2.2.6.7 **Other pathogenic factors**

It is very difficult to assess what contribution stress might make in precipitating diabetes in people with a genetic susceptibility to NIDDM. However, the possibility that stress may be a diabetogenic factor cannot be ignored and is perhaps one of a number of factors which in varying degrees of magnitude have a role in causing the high prevalence of the disease in some migrant ethnic groups, for example (Zimmet, 1982).

Many black diabetics in South Africa suffer from diabetes caused by an acquired form of haemochromatosis (50 % of the haemochromatosis cases) (Jackson, 1978b). Apart from the liver

and spleen, the heaviest deposits of iron in these people are found in the pancreas. The haemochromatosis usually develops from iron deposited from locally brewed alcoholic concoctions such as kaffir beer and its numerous variants. The brews are markedly acid in reaction and corrode the crude iron containers in which they are usually prepared. Most of these diabetics are males, over 40 years of age, who are thin. They are all consumers of alcoholic brews, all have firm hepatomegaly and the majority show evidence of portal hypertension or liver failure (Seftel, Keeley & Walker, 1963). Their prognosis is poor, mainly because of the severity and rapid progression of the hepatic cirrhosis. Additional factors are a background of alcoholism, the high incidence of tuberculosis (Mollentze, Pansegrouw & Steyn, 1990) and the fact that in half the cases the diabetes is severe and difficult to control.

2.2.6.8 **Summary**

The pathogenesis of NIDDM is multifactorial. In the obese patient, the primary lesion may be overeating which leads to an increased insulin secretion, insulin receptor down-regulation, insulin resistance and ultimately, glucose intolerance. In non-obese patients the primary lesion may be at the level of the target cell with some form of post-receptor defect in insulin action leading to hyperglycaemia, hyperinsulinaemia and secondary changes in insulin receptor function. There may also be some lesion in the beta-cell which limits insulin output and leads to clinical NIDDM (Berger & Berchtold, 1985).

2.2.7 Prognosis

Diabetes is ranked seventh among the leading causes of death in the USA. It is implicated as a contributory cause in a substantial proportion of deaths from other diseases, particularly those of the cardiovascular-renal system. It is especially associated with deaths in the elderly population (Entmacher, Krall & Kranczer, 1985).

According to Panzram (1987), the prognosis of a patient with NIDDM varies considerably, *inter alia* for the following reasons:

* The possibility that NIDDM in a given patient represents a clinically inconsequential metabolic disorder without any sequelae for length and quality of life;

* the occurrence of severe, life-shortening vascular complications in association with NIDDM; and

* the development of acute life-threatening events such as coma or hypoglycaemia.

There is a clear pattern of progressively poorer prognosis as age at diagnosis increases. This indicates that the effects of NIDDM on survival are not strong enough to overwhelm the significant effect of age itself on prognosis (Panzram, 1987). With onset in the elderly over 75 years of age, NIDDM has little or no effect on longevity.

It is also interesting that the survival patterns for males and females are nearly identical, in contrast to the striking difference between the sexes seen in the general population. In the USA it was found that in people with NIDDM survival for both sexes declines rapidly after the age of 50. Median survival is reached at 65 for women and 66 for men. This is twelve years earlier than that of the general USA population for women and five years earlier for men (Panzram, 1987:124,125). The higher crude death rates among females possibly reflect the greater prevalence of NIDDM in females, their greater longevity and the fact that most deaths among NIDDM patients occur at older ages.

In the USA, the mortality of blacks is significantly higher than that of persons of all other races (Entmacher, Krall & Kranczer, 1985). The mortality from diabetes has a well-defined seasonal pattern. Deaths from diabetes are more frequently reported in the winter months (Entmacher, Krall & Kranczer, 1985).

Billingham et al. (1989) suggest that excess mortality is not closely associated with glycaemic control and only partly attributable to standard risk factors for coronary heart disease such as hypertension and smoking. Arterial disease, however, accounts for approximately 50 - 68 % of all deaths in NIDDM (Panzram, 1987:125). Coronary heart disease represents the most prominent cause of death in NIDDM subjects due to higher incidence of myocardial infarction, increased acute case fatality and more unfavourable long-term prognosis. The second major cause of death is cerebrovascular disease (15 % of all deaths) with a close relationship to the frequently co-existing hypertension (Panzram, 1987:126). There is overwhelming evidence for a high prevalence of already present atherosclerotic manifestations at the time of diagnosis, irrespective of age at diagnosis. Panzram (1987:128) reported that nearly half the patients (45 %) in their study had died within four years after diagnosis. Death due to renal failure is low in comparison with IDDM and has only a two-fold increase compared to healthy subjects.

Hyperglycaemia increases in many patients with NIDDM to the point that treatment with insulin is required (Królewski, Warram & Christlieb, 1985). There are few data on mechanisms responsible for the worsening of hyperglycaemia. It may be an intrinsic characteristic in some types of diabetes, determined genetically and expressed as part of the aging process. Behaviour may alternatively be responsible through diminishing attention to planning and/or executing treatment. Another possibility is that hyperglycaemia itself may initiate a vicious circle of increasing insulin resistance and diminishing insulin responsiveness to glucose. If true, deterioration could be prevented only by early diagnosis and normalization of glycaemia (Królewski, Warram & Christlieb, 1985).

2.2.8 Pathology

DM is a disease with an extraordinarily diverse pathology.

Although there is no consistent anatomic lesion found at autopsy, there are some distinctive abnormalities which provide a solid basis for the pathophysiology and clinical features of diabetes. The pathophysiology of DM has been reviewed by Warren, LeCompte & Legg (1966) and Legg and Harawi (1985) and will be briefly summarised here. Where available, information on black patients in South Africa will be added.

2.2.8.1 Changes in the pancreas and islets of Langerhans

The longest known and most frequently found lesions of the islets is hyaliniation, seen in about 30 % of diabetic subjects at autopsies in comparison with the ten percent prevalence in other subjects. It is particularly likely to be present in older NIDDM subjects with diabetes of long duration. It appears to be more closely related to the age of the patient than to duration or severity of the disease. It may be related to the production of excessive or abnormal insulin or to a local reaction to insulin or other hormones.

A reduction in beta-cell granulation in the absence of other apparent changes in the islets is a frequent finding in diabetes and is usually referred to as "degranulation of beta-cells". Fibrosis is another long-recognized lesion of the islets in the human diabetic pancreas. Hypertrophy is seen in about five percent of autopsies.

Fatty infiltration is rather common in diabetic individuals, especially in obese subjects. Chronic pancreatitis seems to be followed by an increased incidence of diabetes. Carcinoma of the pancreas is more frequently observed in diabetic autopsies. The average weight of the pancreas is lower than normal.

2.2.8.2 Changes in the kidney

Certain renal glomerular lesions in diabetic patients are among the most specific pathologic changes in the disease. There are

three common glomerular lesions, namely nodular glomerulo= sclerosis (Kimmelsteil-Wilson lesion; in 25 % of diabetic autopsies, infrequently in NIDDM), diffuse glomerulosclerosis (with basement membrane thickening as the initial lesion due to altered carbohydrate metabolism) and the "exudative" lesion. The latter lesion is non-specific for diabetes.

Both atherosclerosis and arteriosclerosis are prominent in the renal changes of diabetes, even in the absence of hypertension. Thickening of the basement membrane of the renal tubules is often seen as well as a vacuolization or the Armanni-Ebstein lesion. Pyelonephritis and renal papillary necrosis are common.

2.2.8.3 **Changes in the eyes**

Although a variety of changes in different parts of the eye have been related to diabetes, by far the most significant lesions occur in the retina. Diabetic retinopathy is one of the most important causes of loss of vision. Retinal microaneurysms progress to develop a thick wall, often with a large amount of lipids. Loss of retinal neurons occurs later on, as well as extensive haemorrhage and even retinal separation. Cataracts also develop.

2.2.8.4 **Changes in the nervous system**

Nearly all diabetic patients exhibit neural dysfunction of some degree, especially with uncontrolled DM. Peripheral neuropathy and diabetic amyotrophy are common. Lesions of single nerves due to ischemic lesions are also often seen.

2.2.8.5 **Changes in the cardiovascular system**

The quantitative association of atherosclerosis and arterio= losclerosis with DM has been firmly established. Coronary heart disease is the most important cause of death in DM patients. Gangrene is a major problem in the diabetic population.

Cardiovascular disease is not the most important cause of death in black South African diabetic patients. In fact, vascular disease, and especially cardiovascular disease, is very uncommon (Jackson, 1978b).

2.2.8.6 **Changes in the liver and digestive tract**

Hepatic glycogen is often considerably decreased. The liver is often found to be enlarged due to fat infiltration. There is an increased frequency of both cholecystitis and cholelithiasis in the biliary tract. Most of the stones are composed predominantly of cholesterol.

2.3 COMPLICATIONS, ABNORMALITIES AND MARKERS OF RISK FACTORS OF COMPLICATIONS IN NON-INSULIN-DEPENDENT DIABETES MELLITUS

2.3.1 Introduction

The chronic or long-term complications of DM include the microvascular and nervous system complications, and the macrovascular complications which lead to coronary heart disease.

Few clinical data on NIDDM and the complications of the disease in South African blacks (Morley et al., 1977) are available and therefore data from other communities have to be reviewed.

2.3.2 Micro-vascular disease and related abnormalities

The recent revival of interest in attempts to maintain strict glucose control has given renewed hope that the maintenance of normoglycaemia (insofar as safe and practicable), may prevent, postpone, or minimize chronic complications affecting the vascular and nervous systems.

According to Brownlee (1985) retinal capillary damage results in blindness which is 25 times more prevalent in diabetics than in the normal population. Cataracts appear earlier and seem to progress more rapidly. Chronic renal failure is 17 times more prevalent and there is a high incidence of nervous system impairments. Amputations due to gangrene are several times more frequent in diabetics and their expected average life-span is only two-thirds that of non-diabetics (Brownlee, 1985:185).

2.3.2.1 **Haematological abnormalities**

Several haematologic abnormalities have been defined in patients with DM. Relative tissue hypoxia may play a role in the pathogenesis of several diabetic complications.

Common haematologic abnormalities associated with DM are

summarised in Table 2.7.

Table 2.7 Haematologic abnormalities associated with non-insulin-dependent diabetes mellitus

---

White blood cells

1. Decreased chemotaxis
2. Decreased diapedesis
3. Decreased phagocytosis
4. Decreased bactericidal activity
5. Decreased cell-mediated immunity
6. Abnormalities in adherence

Red blood cells

1. Increased aggregation and microviscosity
2. Decreased deformability
3. Increased non-enzymatically glycosylated haemoglobins
4. Altered oxygen affinity
5. Decreased concentration of inorganic phosphorus
6. Glycosylation of 2,3-diphosphoglycerate binding site

Platelets

1. Increased platelet adhesion
2. Increased platelet aggregation (adenosine-di-phosphate (ADP), epinephrine, arachidonic acid)
3. Increased PGE_2-like material in platelets (ADP, epinephrine, collagen, arachidonic acid)
4. Decreased survival
5. Lower platelet count

Whole blood

1. Increased whole blood and plasma viscosity
2. Increased fibrinogen, haptoglobin
3. Increased von Willebrand Factor activity (other clotting factors, e.g. factor V)

Table 2.7 Continued

4. Increased alpha-2-macroglobulin, alpha-1-antiprotease
5. Decreased antithrombin III (AOD)
6. Increased CH50, C_1S, C_3, C_4, (complement system)
7. Increased fibrinogen, alpha-2-macroglobulin turnover
8. Decreased fibrinolysis
    a. decreased spontaneous blood fibrinolytic activity
    b. decreased plasminogen activator release
    c. decreased fibrinolytic response to venous occlusion

(Brownlee, 1985:187; Jones & Peterson, 1981:339).

The major function of the erythrocyte is to serve as a transport vehicle between various tissues, most importantly to deliver oxygen from the lungs to the rest of the body. Abnormalities of the oxygen-carrying system in diabetes may, therefore, have serious negative effects (Jones & Peterson, 1981). The increased aggregation of red blood cells may contribute to obliterative micro-vascular changes in the retina. The decreased deformability of the erythrocytes might seriously hamper rapid and homogeneous perfusion within the microcirculation (Schmid-Schönbein & Volger, 1976).

Patients with inadequately controlled diabetes may exhibit two-fold to three-fold elevations in haemoglobins HbA_0, HbA_{1a}, HbA_{1b}, HbA_{1c1} and HbA_{1c2}. In diabetes, the combination of a relative deficiency of 2,3-diphosphoglycerate and elevated glycohaemoglobin levels during periods of change in blood glucose level, may result in decreased oxygen delivery to critical tissues. It has also been reported that pronounced hyperlipoproteinaemia accentuates the impairment of oxygen delivery to tissues. The elevations in haemoglobins could, therefore, play a role in various micro-vascular complications (Brownlee, 1985).

Numerous functional abnormalities in the polymorphonuclear leucocytes have been demonstrated in diabetic patients (Bagdade,

Stewart & Walters, 1978). Changes in the cell surface may, in part, be responsible for alterations in a variety of cellular properties that have been observed, which may play a role in the development of chronic complications including microangiopathy (Brownlee, 1985). More research in this field is urgently needed. There has been little study of basophils and eosinophils in patients with diabetes. More is known about the neutrophils.

Jones & Peterson (1981) summarised the variety of metabolic abnormalities detected in lymphocytes of diabetics with poor metabolic control. There seems to be a selective vulnerability of sub-population of cells, such as the T-cells and to some extent the B-cells in animal systems. This appears to be true for humans, too. Most of the abnormalities are corrected when the serum glucose concentration is returned to normal.

Abnormalities in platelet behaviour (lower platelet counts and higher platelet adhesiveness) may play a role in the development of obliterative micro- and macro-vascular disease in diabetes (Fuller et al., 1979; Harrison, Reece & Johnson, 1980; Meade et al., 1986). It is possible that platelet abnormalities are secondary to the vascular complications, but that the abnormalities are too subtle to be detected before vascular disease is diagnosed (Jones & Petersen, 1981). Increased second-phase aggregation has been observed in diabetics in response to ADP, epinephrine, arachidonic acid, and collagen, as well as increased release of platelet factors III and IV. Levels of plasma beta-thromboglobulin, released during the aggregation process, are also elevated in diabetics. It was suggested that increased synthesis of prostaglandins may, in part, mediate the abnormal aggregation response of diabetic platelets. In addition, marked decreases in prostacyclin activity have been observed in tissues of diabetics (Harrison, Reece & Johnson, 1978). Increased amounts of lipid phosphorus, together with alterations in the fatty acid pattern of various phospholipids have been reported (Brownlee, 1985). Ulutin (1986) suggests that platelets in patients with atherosclerosis and thrombo-embolisms

are circulating in an activated form.

Significant associations have been noted between the diabetic state and several factors that could interfere with microcirculatory flow (Brownlee, 1985; Kannel et al., 1987). Increased levels of fibrinogen, beta-lipoprotein, ceruloplasmin and other glucoproteins have been shown. These glycoproteins cause an increased plasma viscosity and flow resistance and distortability of red blood cells. According to these authors, glycoprotein levels are most significantly correlated with the presence or absence of recent hypoglycaemic symptoms and developing cardiovascular lesions.

Fibrinogen concentrations in diabetic patients are significantly increased in comparison with levels in healthy people (Coller et al., 1978). Hyperfibrinogenaemia is most probably associated with the level of glycaemia and may be attributed to a compensatory overproduction by the liver (Fuller et al., 1979; Ulutin, 1986). Fibrinogen survival is also reduced in accordance with the level of glycaemia (Brownlee, Vlassara & Cerami, 1983). This phenomenon is, however, reversed when better glycaemic control is achieved (Jones & Peterson, 1979).

Increases in coagulation factors V (Egeberg, 1963) and VIII (Coller et al., 1978) have been reported. Increased thrombin coagulation factors II, IX, X, XI and alpha-globulin antithrombin III may be involved in decreasing fibrinogen survival in diabetics (Brownlee, 1985; Meade et al., 1986). Deficiencies of antithrombin III may contribute to the hypercoagulable state reported for NIDDM patients (Gandolfo, De Angelis & Torresi, 1980). A raised Von Willebrand factor may play an important role in platelet abnormalities since elevated levels of this glycoprotein plasma cofactor of platelet function correlate with the degree of platelet aggregation (Lufkin et al., 1979).

There have only been a few reports of factor VII activity in diabetic patients and the results are inconclusive. Some reports

show increased concentrations (Jones & Peterson, 1981; Meade et al., 1986). In the Northwick Park Heart Study an increased number of cardiovascular deaths were found in people with higher factor VII and VIII levels and it was suggested that these factors could be seen as markers of cardiovascular disease (Meade et al., 1986). Lower factor VII levels were reported in blacks than in whites in the above-mentioned study. Higher factor VII values were found in diabetic patients with retinopathy and proteinuria (Fuller et al., 1979). The fibrinolytic activity of diabetic blood is significantly below normal and the response to venous occlusion is also diminished, probably due to decreased release of plasminogen activator (Almér & Pandolfi, 1976). These abnormalities are most marked in diabetics who have retinopathy. Recent studies have suggested that fibrin deposition may play a role in the development of micro-vascular damage (Brownlee, 1985).

2.3.2.2 **Diabetic nephropathy**

Renal disease associated with chronic diabetes is manifested clinically by continuous proteinuria and a decreasing glomerular filtration rate (Brownlee, 1985). There is a correlation between the severity of these clinical features and the degree of glomerular basement membrane thickening, which causes progressive occlusion of glomerular capillaries, leading to chronic renal failure (Brownlee, 1985).

Structural changes in the kidneys occur at a high rate early in the course of diabetes, probably due to the accumulation of serum proteins as a result of an increased glomerular permeability. There are also other histological protein abnormalities. The amount of albumin excreted becomes progressively larger with increasing duration of the disease. Non-enzymatically glycosylated plasma proteins may play a particularly important role in these changes, especially if enhanced glycosylation decreases the susceptibility of these proteins to proteolysis (Mogensen, Osterby & Gundersen, 1979).

Due to hyperglycaemia there is an elevated glucose concentration within the cells of kidney tissues of diabetic subjects. The high glucose concentration influences the synthesis of various basement membrane substances to further promote the thickening of the membranes (Brownlee, 1985).

Although clinical diabetic nephropathy seems to be an infrequent complication of NIDDM in most populations, it may contribute to the health problems of NIDDM subjects of some populations (Królewski, Warram & Christlieb, 1985).

2.3.2.3 **Diabetic retinopathy**

Diabetic retinopathy remains one of the most difficult ophthalmological diseases to treat (Mouton & Gill, 1988). Although regional ischaemia appears to be a central mechanism in the pathogenesis of diabetic retinopathy, it is a multifactorial condition. According to Kohner (1976), there is an initial increase in both the volume of retinal blood and segmental retinal blood flow, accompanied by auto-regulatory dilatation of retinal blood vessels. As the retinopathy progresses, occlusion of capillaries limits auto-regulation of flow, after which retinal hypoxia, inadequately compensated for by increased blood flow, results in the formation of microaneurysms. Eventually increased retinal capillary permeability results in the clinical manifestations of intraretinal oedema and hard exudate formation (Kohner, 1976). Removal of oedema fluid from the retina may also be incomplete, since thickening of the basal membrane creates a greater barrier to diffusion at the pigment epithelial cell layer. Haematological abnormalities, such as hyperviscosity, elevated fibrinogen levels, increased platelet aggregability and decreased erythrocyte deformability, as discussed earlier in this chapter, may all play a role in the progress of diabetic retinopathy (Brownlee, 1985).

Cataract or opacification of the ocular lens is more prevalent and occurs at an earlier age in diabetics than in the normal

population (Brownlee, 1985). This author suggests that the increased tendency of lens proteins to undergo sulphydryl oxidation may facilitate the formation of aggregates. Hyperglycaemia increases glucose concentration within the lens. Subsequently, lens proteins are altered to form disulphide bonds more readily. A combination of these factors then contribute to the formation of the cataract (Brownlee, 1985).

Background retinopathy appears to be a frequent complication of NIDDM. It is closely related to the severity and duration of diabetes and the degree of hyperglycaemia (Klein et al., 1988). With increase in age, retinopathy in NIDDM becomes more severe (Mouton & Gill, 1988). Among older-onset persons not taking insulin, the relative risk of developing retinopathy was found to be 4.0 and the risk for progression 6.2 (Klein et al., 1988:2864). In a study of Mouton and Gill (1988), patients treated by diet alone had a much lower prevalence of retinopathy than those who received insulin or oral hypoglycaemic agents. A higher percentage of patients on oral treatment than those on insulin had serious retinopathy. The same study showed that hypertensive diabetics had a significantly higher prevalence of serious retinopathy than those who were normotensive. Patients with raised serum cholesterol levels also had a more severe degree of retinopathy than those with normal serum cholesterol values. The same applied to patients who smoked compared to the non-smokers.

2.3.2.4 **Diabetic neuropathy**

According to Jakobsen (1978) peripheral neuropathy affects somatic and autonomic neurones in their axonal processes. It is extremely common and highly symptomatic, although more often annoying than life-threatening. Numbness and paresthesias are the most common symptoms of peripheral neuropathy in the diabetic person. These symptoms are believed to be the clinical manifestations of changes in nerve electrophysiology and to reflect altered morphologic and biochemical properties of the

axon and the myelin sheath. Loss of large and small myelinated and unmyelinated fibres is one of the factors that contribute to the decreased sensory and motor nerve conduction velocities. Another morphologic alteration which develops is endoneurial oedema with resultant shrinkage of axons and Schwann-cells (Jakobsen, 1978).

Nerve fibres of diabetics also show a thickening of basement membranes and hyperplasia of the endothelial cell in small vessels. In some cases these excess cells completely occlude the vessel lumen. These abnormalities in the micro-vascular system of peripheral nerves may diminish blood flow through small vessels which, in turn, may result in regional ischaemia or micro-infarcts. This could contribute to neuronal dysfunction and degeneration (Timperly et al., 1977).

Increased activity of the sorbitol pathway has been implicated in the pathogenesis of neuropathy in acute diabetic cataracts, diabetic macro-vascular disease and in diabetic peripheral neuropathy (Brownlee, 1985). Hyperglycaemia and the resultant increased sorbitol pathway activity are associated with decreased oxygen uptake in the peripheral nerves and this may contribute to the development of neurologic dysfunction. Additionally, alterations in the metabolism of myoinositol may play a role in the functional abnormalities of peripheral neuropathy in diabetics. There is a marked increase in urinary myoinositol excretion and an apparent decrease in intracellular transport of myoinositol in uncontrolled diabetics (Clements & Reynertson, 1977).

2.3.3 Macro-vascular disease related abnormalities

2.3.3.1 **Introduction**

Although patients with NIDDM are less likely to experience the acute metabolic complications of IDDM, their vulnerability to vascular complications is not correspondingly reduced (Królewski,

Warram & Christlieb, 1985). Many of the variables predicting future NIDDM are also known to be risk factors or markers for macro-vascular disease (Brownlee, 1985:185). Therefore, it seems possible that both diabetes and macro-vascular disease have common antecedents and that some factors or clusters of factors may induce both diabetes and macro-vascular disease (Ohlson et al., 1988). The macro-vascular complications, originating from atherosclerosis, account for the majority of fatalities in the diabetic population and it is more frequently observed in NIDDM patients over 50 years of age (Marble, 1976). Diabetics have a two-fold greater risk of coronary artery disease and stroke than the normal population and a three-fold to four-fold greater risk of peripheral arterial disease. South Africa, specifically, has a very high prevalence of coronary hart disease in whites and lags behind other first-world communities with intervention programmes (Vermaak et al., 1988). It is, therefore, an aggravating factor in NIDDM. Cardiovascular disease is usually already present at the diagnosis of NIDDM. After clinically manifest diabetes develops, risk factors for cardiovascular disease are further increased (Wingard et al., 1983).

In addition to hyperlipidaemia, hypertension and obesity (which will be discussed), other risk factors play a role in the high incidence of macro-vascular complications in diabetes. Smoking, for example, may lead to increased hypoxia that leads to an increase in lipid uptake and increased platelet adhesiveness (Topping, 1977). Hyperglycaemia, especially if uncontrolled, may also play a role in tissue hypoxia (Keen et al., 1981). The NIDDM patient, with a constellation of increased adiposity, hyperlipidaemia and insulin-insensitivity, can benefit immensely from an exercise programme, since the capacity for carbohydrate storage in the sedentary state is clearly limited (Björntorp & Sjöström, 1978). The significance of behaviour patterns deserves emphasis as an additional factor in the diabetic patient. Deficiencies in copper, zinc and vitamin B-6 have also been linked to atherosclerosis (Ganda, 1985b).

2.3.3.2 **Hyperlipidaemia**

Although DM is by definition a state of abnormal carbohydrate metabolism, defects in lipoprotein metabolism are prominent features of the diabetic syndrome. This is particularly true of patients with NIDDM, and it is quite likely that the abnormal lipid metabolism contributes significantly to the increased morbidity and mortality from coronary artery disease (Reaven, 1987).

The changes in plasma lipid and lipoprotein concentrations commonly seen in NIDDM are summarised in Table 2.8. It should be taken into consideration that all the other factors, both genetic and environmental, which regulate lipoprotein metabolism are operative in patients with NIDDM. These factors modulate the effects of NIDDM on the concentrations of the lipids given in Table 2.8. Glycaemic control will also influence plasma lipids (Kostner & Karádi, 1988).

Table 2.8 Changes in plasma lipid and lipoprotein concentrations in non-insulin-dependent diabetes mellitus

Triglyceride	↑
Cholesterol	↑
Very low-density lipoprotein (VLDL)-triglyceride	↑
VLDL-cholesterol	↑
High-density-lipoprotein (HDL)-cholesterol	↓
Free fatty acid	↑

(Adapted from Kostner & Karadi, 1988; Laakso et al., 1987; Reaven, 1987).

The abnormal energy metabolism in diabetics, particularly fat, leads to higher circulating lipid levels, especially of low-density - lipoprotein (LDL)-cholesterol. Hyperlipidaemia, especially hypertriglyceridaemia with or without associated

increase in plasma cholesterol, is a metabolic abnormality frequently associated with diabetes (Ganda, 1980). Hypertriglyceridaemia in NIDDM is primarily due to an increase in the plasma very-low-density-lipoprotein (VLDL) concentration and is related to the degree of hyperinsulinaemia (Kostner & Karádi, 1988; Laakso et al., 1987; Reaven, 1987). The most common forms of hypertriglyceridaemia in diabetics are, in descending order, Type IV, Type IIb, Type V and Type IIa (Cohn, Gabbay & Weglicki, 1976). Disturbances of triglyceride metabolism may be partly explained by the high prevalence of obesity in these patients (Laakso et al., 1987).

Two lines of evidence might explain the increased atherogenicity of diabetic lipoproteins. First, diets high in cholesterol and saturated fat induce the synthesis of cholesterol-rich apolipoproteins with beta-VLDL which interact with macrophages and transform them into lipid-laden foam cells in the arterial wall (Mahley, 1981). Secondly, chemical modifications of the LDL particle, e.g. enzymatic glycosylation, might result in increased precipitation of LDL on the arterial walls (Kahn, 1985). In NIDDM subjects, VLDL synthesis is particularly enhanced in the presence of obesity and specifically with untreated NIDDM, abnormalities of triglyceride clearance have been found (Abrams, Ginsberg & Grundy, 1982). Micro-vascular complications leading to increased blood vessel wall leakage of proteins and fats, may result in more severe atherosclerosis (Cahill, 1985).

DM is occurring with increasing frequency in the black population in South Africa. However, in studies on black diabetics in South Africa it seems as if they are relatively free from macro-vascular complications. For example, in the study of Morley et al. (1977), only 20 % of the patients had hypercholesterolaemia and 21 % had hypertriglyceridaemia. This apparent immunity may be related to many factors, such as lower plasma lipid levels and an attenuated insulin response to oral glucose (Asmal & Leary, 1975). However, Silvis (1989:87) found a high incidence of hypertension (65 %), obesity and in women hypercholesterolaemia

(mean 6.77 mmol/l). Hypercoagulability, characterised by raised coagulation factors and low activities of inhibitors of coagulation, did not occur in these patients to the same extent as in whites.

The inverse relationship of HDL with atherosclerosis, independent of other lipid abnormalities, is well established (Avogaro et al., 1979; Kostner & Karádi, 1988; Laakso et al., 1987). Moderate lipoprotein lipase deficiency is also often found in NIDDM (Laakso et al., 1987). Fasting plasma insulin correlates negatively with HDL-levels (Laakso et al., 1987). Some studies have shown that it is specifically patients treated with oral hypoglycaemic agents who have lower HDL-levels. There is, however, still some controversy about this point (Ganda, 1985b). Despite controversy, most researchers believe that differences in cholesterol levels are at least partly explained by differences in diet and life-style (Shekelle, et al., 1981). Many other factors may have an influence on the lower HDL-levels in NIDDM patients, such as poor diet, not enough exercise and the habit of smoking (Henry, Bell & Glithero, 1979). Diabetic women usually show a greater decrease in HDL-cholesterol levels than men (Ganda 1985b). This may partly explain the relative increase in coronary heart disease in diabetic women compared to men (Reaven, 1987).

In black girls studied by Walker et al. (1979) in rural and urban areas, mean values for HDL-cholesterol were not significantly different in obese compared with non-obese girls. This is not the case in white obese girls. There may, therefore, also be a difference in HDL profiles of NIDDM blacks. Silvis (1989:94) found that HDL-levels are relatively high (mean 1.11 mmol/l, 19.2 %) in black NIDDM patients. It is therefore possible that, together with the normal coagulation profiles as observed by Silvis (1989) that the high HDL-levels could protect black diabetic patients against coronary heart disease.

Kubisz, Parizek and Cronberg (1986) found increased serum levels

of apolipoprotein B in diabetic patients. They also found a relationship between these increased levels and platelet hyperactivity. Apolipoprotein A levels were also increased. Apolipoprotein A and especially the A-IV fraction is very sensitive to diet; a low-fat diet can lead to the immediate reduction of apolipoprotein A (Weinberg, Dantzker & Patton, 1990).

The following conclusions regarding lipid profiles of DM patients can be drawn from the literature (American Diabetes Association, 1990a:21; D'Antonio et al., 1989; Laakso & Pyörälä, 1988):

* In untreated DM, the serum concentration of LDL-cholesterol is within normal limits or higher than normal. Hyperlipidaemia involves an increase in serum triglyceride, VLDL-triglyceride and VLDL-cholesterol concentrations. HDL-cholesterol levels may be decreased, particularly in NIDDM.

* DM and familial hyperlipoproteinaemias are not genetically co-inherited. Their frequent coexistence could result from their independent association with other metabolic disorders such as obesity.

* The mechanism of the lipoprotein disorder in diabetes affects the metabolism of plasma VLDL, LDL and HDL. The disorder greatly relates to the metabolic milieu of the diabetic syndrome, although the mechanisms of increased apolipoprotein B production and decreased HDL-cholesterol concentration observed in some diabetic individuals remain unknown.

* Treatment of hyperglycaemia is associated with improvement in plasma VLDL and LDL concentrations and can be accompanied by improvement in plasma HDL levels, particularly when associated conditions such as obesity are simultaneously treated.

* Epidemiological surveys, dietary intervention trials, and

studies in experimental animals, provide strong evidence that fat and cholesterol restriction could exert favourable influences on plasma lipid and lipoprotein levels as well as on cardiovascular risk.

Aspects concerning the dietary treatment and treatment of lipid abnormalities will be discussed in section 2.4.

2.3.3.3 Genetic factors in non-insulin-dependent diabetes mellitus

A family history of NIDDM is twice as common among those becoming diabetic than among the non-diabetic subjects (Ohlson et al., 1988). Genetic factors in atherosclerotic disease are intimately intertwined with the genetic control of some of the key risk factors such as diabetes (Ganda, 1985b). Apart from genetic factors, certain racial characteristics in the pathogenesis of atherosclerosis are noteworthy. In the black people of the Cape Town area (they have as much diabetes as the white people) the incidence of ischaemic heart disease is low compared to the incidence in whites. This is not entirely explained by differences in diet and obesity (Jackson, 1978b) and genetics may, therefore, be an important factor.

2.3.3.4 Haemostatic defects

As already mentioned in section 2.3.2.1, several lines of evidence indicate disorders of blood coagulability, viscosity and other rheologic disturbances in the diabetic. NIDDM patients often have increased plasma fibrinogen levels that may sometimes predict subsequent atherosclerotic events. The fibrinogen levels have been found to correlate with degree of diabetes control, as indicated by the glycosylated haemoglobin levels (Coller et al., 1978). Abnormalities in coagulation factors V, VII and X have also been demonstrated (Ganda, 1980). There is a positive correlation between initial plasma fibrinogen levels and the subsequent incidence of heart attacks, skinfold thickness,

systolic blood pressure and accelerated atherosclerosis. Plasma fibrinogen levels are important coronary risk factors (Kannel et al., 1987; Sgammato, 1987; Stone & Thorpe, 1985; Wilhelmsen et al., 1984). Stone and Thorp (1985) found that in men with high cholesterol or high systolic blood pressure levels, the incidence of heart attacks was respectively six and twelve times higher in those with high plasma fibrinogen levels than in those with lower levels. High fibrinogen levels in diabetics are therefore of great importance.

Smith (1986) summarised the evidence that enhanced blood coagulation is a risk factor not only for thrombotic occlusion, but also for atherogenesis and coronary heart disease. Possible mechanisms include stimulation of cell proliferation, migration, adhesion, mitogenesis, collagen synthesis, leukocyte attraction, endothelial permeability and tight binding of LDL by fibrin and fibrin degradation products.

2.3.3.5 **Hormonal alterations**

Considerable controversy exists concerning the role of insulin in the development of atherosclerosis. High growth hormone and other associated hormone levels may also play a role. The catecholamines probably play a significant role in the progression of both atherosclerosis and hypertension (Ganda, 1985b).

2.3.4 Hypertension

2.3.4.1 **Circumstances in South Africa**

Many studies have shown that hypertension in the black population of Southern Africa is common and severe (Isaacson, Milne & Van Niekerk, 1989). Hypertensive causes of death are approximately four times more common in all age groups and in both sexes among blacks than among whites. It occurs at a younger age than in whites and behaves in an explosive manner, with death occurring

mainly from cerebral haemorrhage, uraemia or congestive cardiac failure. It has also been demonstrated that hypertension is not a major problem in the rural black but becomes prevalent in the urban environment (Seedat, Seedat & Reddy, 1978). For black South African girls, Walker et al. (1979) found that the diastolic values in urban dwellers were higher than those in rural dwellers. Isaacson, Milne & Van Niekerk (1989) have shown that there are some clinical and pathological differences between black and white hypertensives.

It is still a matter of controversy whether there is an increased probability of hypertension in diabetics (Rosendorff, 1989). High blood pressure, however, is commonly found in patients with abnormalities of glucose and insulin metabolism and is a principal predictor of cardiovascular disease (Ohlson et al., 1988). Abrahamson (1988) found, for example, an almost four-fold increase in the prevalence of hypertension among NIDDM patients in the South African Indian community and a three times greater prevalence of hypertension among those with IGT. He has also found that obesity, although it is an aggravating factor, does not have to be present for a diabetic person to be hypertensive.

2.3.4.2 **Hyperinsulinaemia and hypertension**

Abrahamson (1988) mentions that there is mounting evidence that insulin resistance and hyperinsulinaemia occur in some people with hypertension, even in the absence of obesity and abnormal glucose tolerance. It has been suggested that insulin may somehow be responsible for the high prevalence of hypertension in IGT and NIDDM. If insulin does play a pivotal role in the pathogenesis of hypertension in these conditions, a number of important questions need to be answered. Firstly, is hyperinsulinaemia *per se* responsible for the rise in blood pressure or is the hypertension caused by the state of insulin resistance? Do similar defects in insulin dynamics occur in non-obese hypertensives with normal glucose tolerance? And, finally, can improvements in insulin resistance and hyperinsulinaemia

reduce blood pressure in affected individuals?

Two effects of insulin suggest that hyperinsulinaemia may play a role in causing hypertension. Firstly, insulin enhances renal tubular reabsorption of sodium and this results in an increased intravascular volume, increased cardiac output and enhanced arterial pressure (DeFronzo, 1981). Secondly, increases in plasma insulin concentration are associated with significant increases in plasma catecholamine cencentration. Insulin has been shown to stimulate sympathetic activity in the absence of hypoglycaemia with the result that blood pressure is increased (Rosendorff, 1989). There is also some preliminary evidence to suggest that improvements in insulin resistance and hyperinsulinaemia may improve blood pressure control. Weight loss and improvement in insulin sensitivity in certain obese individuals improve blood pressure more effectively than some antihypertensive agents and somatostatin (Abrahamson, 1988).

Blood viscosity is higher in hypertensive patients, due to higher plasma viscosity, elevated fibrinogen levels and increased haematocrit values (Letcher et al., 1981).

It is therefore likely that the increased risk of coronary artery disease in hypertension is due, at least in part, to the risk factors for vascular disease associated with insulin resistance such as hyperinsulinaemia, IGT, increased plasma triglyceride concentration, and decreased HDL concentration (Rosendorff, 1989) and possibly also increased plasma fibrinogen.

2.3.5 Obesity

Many studies, such as the study of Ohlson et al. (1988), show that people who become diabetic are significantly more obese at baseline as indicated by body mass index (BMI), had more pronounced central adipose tissue distribution according to the waist-hip-ratio (WHR), and have elevated systolic and diastolic blood pressures compared to non-diabetic subjects.

2.3.5.1 What is obesity?

A weight of 20 % or more above standard is frequently cited as reflecting obesity, while a weight of 20 % or more below standard is frequently equated with nutritional risk (Krause & Mahan, 1984; 208). A BMI between 24 (for females) and 25 (for males) and 27 is defined as overweight, not obesity. A person with a BMI over 30 is usually classified as obese (Krause & Mahan, 1984:518; Garn, Leonard & Rosenberg, 1986).

2.3.5.2 Different types of obesity

Fat distribution differs according to sex. In men it tends to be either predominantly in the abdominal region or in the upper trunk (android obesity); in women gluteal-femoral fat accumulation is more common (gynoid obesity). An elevated frequency of cardiovascular risk factors has been found in grossly obese subjects with a predominant distribution of fat in the abdominal or upper trunk region (Despres et al., 1990). Complications such as glucose intolerance (also higher glycated haemoglobin values), hyperinsulinaemia, hypertriglyceridaemia, low HDL-cholesterol, coronary and peripheral arterial disease, hypertension and possible gout are associated with a high WHR. Predominant abdominal and upper trunk fat distribution is an important risk factor for NIDDM as well (Evers, McCracken & Deagle, 1989; Freedman et al., 1990; Lundgren et al., 1989; Ostlund et al., 1990; Van Gaal et al., 1988). It has been proposed that the higher incidence of these health problems might be related to differences in metabolic behaviour of fat cells of different regions. Unbound androgens probably have an important role in this phenomenon (Van Gaal, VanSant & De Leeuw, 1989).

2.3.5.3 Obesity as a risk factor for non-insulin-dependent diabetes mellitus

With obesity there usually is an increase in insulin resistance which can lead to NIDDM. The high energy intake decreases

insulin sensitivity and beta-cell responsiveness to glucose as well as insulin binding decrease (Abrahamson, 1988).

IDDM and NIDDM subjects belong to different anthropometric types, a fact which probably reflects their different pathogenesis. NIDDM is characterized by more central and upper-fat deposition. Because body-fat deposition is largely genetically determined, it is highly probable that the two types of diabetes develop in patients with different body-fat distributions from the beginning. Increased metabolism of fat around the waist, the products of which flow directly into the portal system, can change the balance of hormones and finally result in higher peripheral insulinaemia and increased risk of the development of NIDDM and atherosclerosis (Lev-Ran & Hill, 1987). Results of Van Gaal et al. (1988) showed that metabolic deteriorations in diabetic subjects can be found independently of the degree of obesity, as long as abdominal fat-mass excess is present.

Differences were reported in fat patterning and fat distribution between black and white adults in the USA (Zillikens & Conway, 1990). Blacks have more upper-body fat and possibly more deep-body fat deposition than whites. Black females also have a relatively greater fat deposition on the trunk than on the extremities in comparison with white females, thus showing a more male, or centralized type of fat patterning. It has also been shown that diabetic women are more obese and have more abdominal fat than non-diabetic women (Lundgren et al., 1989).

2.3.5.4 Obesity as risk factor for the complications of non-insulin-dependent diabetes mellitus

Obesity is also frequently associated with hyperlipidaemia, low HDL-cholesterol levels and increased concentrations of LDL-cholesterol and triglycerides and hypertension due to insulin resistance (Streja, Boyko & Rabkin, 1980). Streja, Boyko & Rabkin (1980) found that in grossly obese subjects, a weight loss of 15 % of the body weight was necessary for a significant

increase in HDL-cholesterol concentration. Weight in their study had to fall by more than seven percent of body weight before concentrations of HDL-cholesterol rose. They also found that weight loss leads to a decrease in triglyceride and fasting blood sugar concentrations but no significant changes in total cholesterol, LDL-cholesterol or urate concentrations.

2.3.5.5 **Obesity among black South Africans**

A South African study of black diabetics (Morley et al., 1977:216) found that twelve percent of the men in the study were markedly obese and 31 % moderately obese. Of the women, 67 % were obese and 46 % markedly so. In total obesity was found in 65 % of patients with NIDDM with 38 % of them severely obese. No less that 76 % of the females with NIDDM were overweight. This is, however, less than the 91 % prevalence found in white women NIDDM patients.

Obesity is also common in non-diabetic black South African women (Walker et al., 1979), but despite this fact, serum HDL-cholesterol remains high in blacks from adolescence to old age and also with obesity. In black girls it has been found that blood lipid levels, serum uric acid levels, glucose tolerance values and blood pressure values remain low, even in the presence of obesity. It is therefore suggested that obesity by itself is not a major risk factor for coronary heart disease in NIDDM patients, but it is an important underlying factor in relation to other risk factors, although there may be less of a risk in the black South African population.

NIDDM may have a lower prevalence in traditional living rural communities as compared to their urbanised counterparts (King et al., 1984). Crude prevalence also increases with increasing relative weight. Whether urbanisation directly increases risk for NIDDM, whether findings relating to urbanisation represent the effect of increased obesity in the urban population, or whether both obesity and urbanisation are merely markers for some

other causal factors, has not yet been determined (King et al., 1984). In all different populations, examined by the above-mentioned author, mean BMI was higher in the urban than the rural groups and it is unlikely that the inactive urban residents would be more muscular than their rural counterparts. It was suggested that higher levels of habitual physical activity in the rural people may play a role (King et al., 1984). Another factor that may play a role is a change in dietary habits from traditional home-grown foods to imported processed products.

2.3.6 Impaired glucose tolerance

The prevalence of IGT in the USA adult population is 11.2 % compared to the 6.6 % for diabetes and may be the strongest factor for development of the disease among whites because one to five percent per year proceed to NIDDM (Harris, 1989: 471). The accepted risk factors for NIDDM (age, plasma glucose, obesity, family history of diabetes and physical inactivity) indicate that IGT is in an intermediate position. This suggests that IGT and NIDDM may have similar natural histories and may reflect a continuum of declining glucose tolerance from IGT to overt NIDDM. It is likely that many of the key events that precede NIDDM and are involved in its pathogenesis occur during the period of IGT (Harris, 1989).

There are also differences in races in glucose tolerance. Plasma glucose levels at all points of a glucose tolerance test are higher in Blacks than in Indians in South Africa (Asmal & Leary, 1975). The differences are more marked in females. Plasma insulin concentrations are greater in the Indian than in the Black population and the differences are again more marked in females (Asmal & Leary, 1975).

2.3.7 Measurement of metabolic control and of risk markers of complications in non-insulin-dependent diabetes mellitus subjects

2.3.7.1 **Measurement of glycaemic control**

Fingerprick blood glucose measurements at home is the obvious test for the patient to assess glycaemic control. It is, however, relatively pointless to measure this routinely at a clinic because of all the factors that may have an influence, for example the time of day. Fasting blood glucose estimation is useful at clinics for NIDDM subjects. It reflects overall control if it is done regularly (Alberti & Hockaday, 1987).

Home blood glucose monitoring is quite practicable nowadays and can give valuable information if it is done routinely (Clarke et al., 1987).

2.3.7.2 **Glycosylated protein**

The measurement of HbA_{1c} may provide a better answer to adequate assessment of overall glycaemic control and has become the standard (Cefalu, Parker & Johnson, 1988). It gives an integrated picture of the mean blood glucose level during the average life-span of the red blood cell (120 days), providing the stable irreversibly glycosylated form is separated from the unstable intermediate.

Glycosylated haemoglobin is, however, not suited to monitor glycaemic control over weeks because of the relatively long half-life of red blood cells. An alternative is to measure serum fructosamine which represents the glycosylated component of all serum proteins. It refers to a period of about three to six weeks. The test is based on the ability of glucose bound to protein with a ketamine linkage (fructosamine). The values of this assay correlate well with glycosylated haemoglobin, glycosylated albumin levels and fasting plasma glucose

concentration (Cefalu, Parker & Johnson, 1988; Smart et al., 1988). Some researchers feel that correlations improve if they are expressed with reference to the plasma albumin concentration (McCance et al., 1987), but Smart et al. (1988) believe that the resulting improvement does not seem to significantly alter the discrepancies and is probably not worth undertaking. Dominiczak et al. (1988) did not find good correlations between fructosamine and HbA_{1c} in all patients. Factors other than protein concentration, such as lipid content and sampling time also influence results of fructosamine determination (Flückiger, Woodtli & Berger, 1987).

Although there are difficulties in the standardisation of the method (Ashby & Frier, 1988), it is relatively inexpensive, suitable for automation and allows multiple assays with minimal effort (Ashby & Frier, 1988; Cefalu, Parker & Johnson, 1988).

It is important to recognise limitations in interpreting these techniques. For instance, a normal glycosylated haemoglobin value may depend upon frequent and dangerous episodes of hypoglycaemia (Alberti & Hockaday, 1987). Caution should be taken in interpreting glycaemic control from infrequent use of fructosamine assays, because it may not provide a reliable overview of glycaemic control over a period such as two to four months as can be done with glycosylated haemoglobin (Cefalu, Parker & Johnson, 1988). Some researchers, such as Ashby and Frier (1987) and Dominiczak et al., (1988) believe that both methods should be used together to complement each other in the measurement of glycaemic control.

2.3.7.3 **Other risk markers**

Because hyperlipidaemia and hyperfibrinogenaemia are associated with coronary heart disease (see 2.3.3), these variables should be measured routinely in NIDDM patients.

2.4 TREATMENT OF PATIENTS WITH NON-INSULIN-DEPENDENT DIABETES MELLITUS

2.4.1 Introduction

The main objectives in the treatment of DM should be to control blood glucose levels al well as all risk markers of the long term micro-vascular and macro-vascular complications of the disease. Treatment should be based on a combination of exercise and diet, with or without oral hypoglycaemic agents (and insulin in some instances) (American Diabetes Association, 1990b; Anderson & Geil, 1988; Mann, 1989).

Many conventional diabetes treatment programmes fall short of these main objectives. Insulin or drug therapy often constitutes the primary treatment, with only token mention of dietary change and no recommendations for exercise. This approach is less than optimal. Supplying exogenous insulin in excess can suppress endogenous insulin secretion and worsen insulin resistance (DeFronzo, Ferrannini & Koivisto, 1983). Although oral sulphonylurea agents facilitate insulin secretion and increase insulin sensitivity in certain individuals with NIDDM, they are too often used as a substitute rather than a support for diet and exercise therapy (Anderson et al., 1987).

There are a number of factors which complicate diabetes control. Even highly motivated patients experience difficulties which may seem to be beyond their control, but which simply reflect the realities of daily living, such as occupation, social events, changing energy needs, economic conditions and ethnic habits. Emotional problems such as depression, as well as an attitude failure may also complicate treatment (Flood et al., 1985).

2.4.2 Dietary treatment

Diet remains the cornerstone in the management of diabetes - as it has for hundreds of years (Nuttall, 1988). Intelligent

adherence to a suitable nutrition plan not only reduces risk for metabolic complications as a result of enhanced glycaemic control, but also reduces risk for atherosclerotic cardiovascular disease, usually the major risk factor for decreased quality of life and premature death (Anderson & Geil, 1988). It may also reduce the amount used and costs of oral hypoglycaemic agents (Mann, 1989).

Researchers in South Africa predict that urbanisation and westernisation of the black community may lead to an increasing prevalence of typical western degenerative diseases, including DM and the complications of this disease. Proof for this is found in the prevalence of conventional risk factors for coronary heart disease observed in black diabetics (Silvis et al., 1990). Furthermore, glycaemic control in blacks is usually very poor (Morley et al., 1977; Silvis, 1989). The traditional diet of blacks is in the process of westernisation (see section 2.6) and the dietary treatment of the black diabetic patient should, therefore, get special attention.

Several new considerations regarding the diabetic diet have been introduced during the last decade, especially concerning the amount and type of carbohydrate in the diet (Nuttall, 1988; Mann, 1989). Due to the new and emerging information, the role of nutrition continues to be examined.

2.4.2.1 Objectives of dietary treatment

Objectives and goals in the dietary treatment of DM should not be seen as fixed rules, but as guidelines that may change and should be continuously evaluated as new information about the disease emerges (Crapo & Vinik, 1987).

Nutritional recommendations for subjects with DM are very similar to recommendations to prevent cancer and heart disease (American Diabetes Association, 1990a). Recommendations should be based on achieving one primary objective - a healthy patient with a

full life-style and normal longevity. Other goals and objectives should aid in accomplishing this aim.

As already mentioned, the progress in diabetes management into a sphere beyond that of simple survival from ketoacidosis, to an increase in longevity, has resulted in researchers now being concerned with the long-term complications of DM. Another objective of nutrition therapy is therefore to reduce the frequency of specific diabetic complications such as retinopathy, nephropathy, neuropathy and macro-vascular disease (Crapo & Vinik, 1987). To reach these objectives, the key goals for a diabetes nutrition plan should be good glycaemic control, desirable serum lipid and lipoprotein levels, and the reduction of risk markers for metabolic, micro-vascular and macro-vascular complications (Anderson & Geil, 1988; Mann, 1989).

To obtain euglycaemia and low levels of the risk markers of the complications of DM, dietary planning should concentrate not only on the nutrient content of the diet, but also on factors such as meal frequency, individual life-styles, weight management, flexibility and institution of behavioural changes to optimise compliance with the diet (American Diabetes Association, 1990a; Crapo & Vinik, 1987).

Nutritional strategies are fundamental in the overall management and prevention of lipid abnormalities in people with NIDDM (American Diabetes Association, 1990a). Diet therapy should always be the first treatment for hyperlipidaemia and should continue throughout the pursuit of drug therapy (American Medical Association, 1983).

Elevated triglyceride levels are often the result of poorly controlled blood glucose values. Strict blood glucose control is therefore recommended if hyperlipidaemia is to be prevented, delayed or treated. Elevated triglycerides are often associated with obesity or alcohol consumption and weight reduction and/or alcohol restriction can be beneficial (Hagan & Wylie-Rosett,

1989).

2.4.2.2 Specific nutrients

All nutrients that may affect metabolic control need to be considered in the dietary prescription (Hagan & Wylie-Rosett, 1989). The following aspects of these nutrients should get special consideration:

* Energy

One of the important goals of diet therapy in the management of NIDDM is to assist the person in attaining and maintaining desirable body weight. The amount of energy consumed should be prescribed with this goal in mind (Beebe et al., 1991).

The potential benefits of weight reduction for obese diabetic persons include reduction or elimination of exogenous insulin requirements and increase in life expectancy and improved health. Any person following a weight-loss programme should be closely supervised by health professionals. Ideally, such a programme would be nutritionally balanced with an energy reduction of 2 000 to 4 200 Kj below normal daily levels. The key to sustaining successful weight loss is a long-term behavioural approach (Anderson & Geil, 1988; National Institutes of Health, 1987:640). Weight reduction will be discussed further as an additional consideration under 2.4.2.3.

* Carbohydrate

Diets high in complex carbohydrates and fibre, and low in fat, improve glycaemic control and lower serum cholesterol and triglyceride concentrations (Anderson, 1986; Anderson & Geil, 1988; O'Dea et al., 1989; Rivellese et al., 1980; Simpson et al., 1981), lower serum insulin levels (Anderson, 1986), increase peripheral tissue insulin sensitivity (DeFronzo, Ferrannini & Koivisto, 1983), aid in weight control, and may lower blood

pressure (Anderson et al., 1987; American Dietetic Association, 1988). Some researchers have found, however, that a high-carbohydrate diet, without an increase in the DF, does not affect the plasma cholesterol levels, while plasma triglyceride and apolipoprotein C levels are increased in NIDDM subjects (Rivellese et al., 1990). High carbohydrate, high fibre diets are practical and palatable for the motivated individual (Story et al., 1985).

The 1988 consensus diabetes diet of the American Diabetes Association (Beebe et al., 1991) recommends 55 to 60 % of the energy intake to be carbohydrate. Anderson et al. (1987:1193) suggest that an intake of 70 % of the energy as carbohydrates offers the greatest health benefits for individuals with diabetes, but agree that such a diet may be impractical as a life-style for many individuals.

Recently there has been increasing interest in the liberali= zation of sucrose in the diets of individuals with NIDDM. However, there is evidence from several well-controlled studies to demonstrate that the consumption of moderate amounts of sucrose may result in hyperglycaemia, hyperinsulinaemia, hypertriglyceridaemia, hypercholesterolaemia and reduced HDL-cholesterol concentrations (Hollenbeck, Coulston & Reaven, 1989). Coulston et al. (1985:963) found that the addition of sucrose in amounts comparable to those typically consumed by the general population results in significantly elevated day-long glucose and triglyceride responses, as well as elevated fasting total plasma cholesterol, triglyceride, VLDL-cholesterol and VLDL-triglyceride concentrations, while LDL and HDL-cholesterol remain unchanged. Hollenbeck, Coulston and Reaven (1989) suggested that the continuation of advice to patients with NIDDM at least to limit sucrose consumption until available data would allow the prediction of levels of sucrose consumption where adverse metabolic effects would not be present, seems reasonable. However, some researchers have found that sucrose does not adversely affect metabolic control in NIDDM patients and they

feel that moderate amounts of sucrose should be allowed (Colagiuri, Miller & Edwards, 1989).

Although concentrated carbohydrate may worsen glycaemic control and promote weight gain, the American Diabetes Association Task Force on Nutrition of 1985 suggests that "modest amounts of sucrose and other refined sugars may be acceptable, contingent on metabolic control and body weight" (American Diabetes Association, 1990a:18). The American Dietetic Association (1987:1691) advised a sucrose intake of not more than five percent of the total energy intake.

Riccardi et al. (1984) demonstrated that, compared with the old low-fibre, low-carbohydrate diet, a simple increase of digestible carbohydrates gives no benefit at all in terms of blood glucose control. However, other researchers experienced the opposite with complex digestible carbohydrates (Simpson et al., 1981). Vinik & Jenkins (1988) submit that the predominant effect of a high-carbohydrate, high-fibre diet results from the increase in DF, but that carbohydrates give additional benefits, especially if incorporated with viscous fibres. It has been stated that fibre supplementation appears to provide benefit only if given with a diet comprising at least 50 % of its calories in carbohydrate form (Simpson et al., 1981). Research has shown, however, that an increase in DF alone may produce a significant improvement in blood glucose control in both IDDM and NIDDM (Simpson et al., 1981). Silvis (1989) found that black diabetic patients who received a DF concentrate had worsened glycaemic control but improved serum lipid profiles. This supports the suggestion of Simpson et al. (1981) that there may be two separate mechanisms at work, and that the most marked improvement may be obtained by using both mechanisms in introducing a high complex-carbohydrate, high-fibre diet. The glycaemic response to carbohydrates from different food sources differs. This aspect of carbohydrates will be discussed in detail in section 2.5.

* Dietary fibre

Englyst et al. (1987) suggest that the term non-starch polysaccharides (NSP) should be used for dietary fibre. However, most analysis methods used for DF values in South African food composition tables do not extract the resistant starch before DF is determined (Gouws & Langenhoven, 1986). For the purpose of this study, the term DF will be used.

In addition to the effect of DF on glycaemic control, the incorporation of DF into the diabetic diet has other specific advantages. DF has a more pronounced reduction than carbohydrate on the LDL-cholesterol fraction. The ratio of LDL and VLDL to HDL-cholesterol is also reduced with a high-fibre, high-carbohydrate diet compared to a low-carbohydrate or high-carbohydrate, low-fibre diet (Hagander et al., 1988; Hagander et al., 1989; Ray et al., 1983; Riccardi et al., 1984; Rivellese et al., 1980). In contrast with high carbohydrate diets which may increase hypertriglyceridaemia, DF does not have such an influence (Abbot et al., 1989; Anderson et al., 1987). Fibre also leads to less hunger and greater satiety after meals and delays gastric emptying which has distinct benefits, especially in low-energy diets (Ray et al., 1983). It may also contribute to lower blood pressure and enhance weight loss (Hagander et al., 1989; Vinik & Jenkins, 1988). Other benefits, especially for obese NIDDM subjects, are decreased insulin requirements and increased insulin sensitivity (Anderson et al., 1987; Anderson & Geil, 1988; Kiehm, Anderson & Ward, 1976; Rivellese et al., 1980; Wigand et al., 1979), lower fibrinogen levels from cereal fibre (Fehily et al., 1982), and possible protection against hypercoagulability (Vorster et al., 1988a). However, some researchers found that blood glucose control is not always improved by a high-fibre diet (Hagander et al., 1989).

There are many speculations regarding the mechanisms through which DF works. It may decrease blood glucose levels because of a decreased carbohydrate absorption rather than increased total

glucose utilization, or suppression of the hepatic glucose production (Jenkins et al., 1980b). The possible effects of fibre within the intestine include changes in mixing, motility, convection, intraluminal digestion rates, thickness of the unstirred layer, inhibition of maximum transport capacity, altered pH-profile and, in the long-term, altered intestinal morphology (Vinik & Jenkins, 1988). There is also speculation that fibre may insulate carbohydrate from the digestive enzymes in the intestine and decrease access to the intestinal wall and thereby decreasing digestion rates. Certain fibres may even have anti-enzyme activity (Jenkins et al., 1980a). DF has also been shown to influence the release of gut hormones (Vinik & Jenkins, 1988). The mechanisms by which high fibre diets lower blood lipoproteins and insulin response is also not known. The importance of viscosity in causing delayed gastric emptying time and slowing of carbohydrate absorption has been suggested, as well as an enhanced insulin sensitivity following the high fibre meal, through unknown mechanisms (Potter et al., 1981). It has recently been shown (Venter et al., 1990) that the production of the short-chain fatty acids from fibre in the large intestine may contribute to its metabolic effects.

An increase in DF should focus on both soluble and insoluble forms of fibre-rich foods rather than fibre supplements. It seems as if soluble fibre has most advantages, especially on postprandial plasma glucose and insulin levels (Del Toma et al., 1988). This may be because of a slowing down of gastric emptying and limited diffusion of digestive products (Vinik & Jenkins, 1988). Recent work also indicates that soluble fibre may influence cholesterol and lipid metabolism at hepatic or peripheral sites. Soluble fibres are almost completely fermented in the colon to short-chain fatty acids, primarily acetate, propionate, and butyrate. These fatty acids are absorbed into the portal vein and may inhibit hepatic and peripheral cholesterol synthesis and increase LDL-cholesterol clearance (Anderson et al., 1987). The role of short-chain fatty acids in enhanced glucose utilization, insulin secretion and hepatic

glucose utilization needs further research (Vinik & Jenkins, 1988). Soluble fibre does not only lower total serum cholesterol, but may also raise HDL-cholesterol (American Dietetic Association, 1988). The main metabolic effects of DF are shown in Table 2.9.

Table 2.9 Main metabolic effects of dietary fibre

Soluble dietary fibre	Insoluble dietary fibre
↓ Cholesterol (VLDL, LDL) ↑ HDL-cholesterol ↓ Insulin ↑ Insulin sensitivity ↓ Postprandial blood glucose ↑ Short-chain fatty acid production to some extend	* Binds water - bulking effect, therefore softer, more frequent stools, prevents constipation and constipation-related diseases * Change morphology of digestive tract * Binds minerals

(Adapted from Vorster, 1987b: 15).

Because fibre has so many distinct benefits for people with DM, a goal of an increased intake up to 40 g per day or 15 to 25 g/ 4 200 kJ should be beneficial (American Diabetes Association, 1990a:20). Vorster et al. (1988a:292) recommend four to six grams of DF per 1 000 kJ as a practical level of intake with no detrimental side-effects and no mineral malabsorption. Mann (1989:1535) suggests a minimum intake of total DF around 35 g or 20 g/4184 kJ. Higher levels may be unacceptable in some individuals because of gastro-intestinal side-effects. The level of maximum benefit has not been determined, but a maximum intake of 50 g seems reasonable (American Diabetes Association, 1990a:20). According to Anderson et al. (1987:1193), diabetics should consume 50 g or more total plant fibre (25 g/ 4 200 kJ) with an intake up to 70 g for the greatest health benefits. The American Dietetic Association (1988:216) cautions that some

adverse effects may occur with the consumption of more than 50 g per day, including decreased bioavailability of certain vitamins and minerals. A practical goal would be to establish the level of intake and to increase it gradually with the goal of doubling the intake for most individuals (American Diabetes Association, 1990a:20). The gradual increase will prevent abdominal cramping, discomfort and flatulence (Vinik & Jenkins, 1988).

To be effective the DF has to be incorporated into food (Vinik & Jenkins, 1988). Probably the best approach to the incorporation of DF into the diet is supplementation of the usual diet with a variety of ordinary foods high in DF (Anderson et al., 1987; Rivellese et al., 1980; Simpson et al., 1981; Story et al., 1985). Fruits and vegetables should, where possible, be eaten raw (Vinik & Jenkins, 1988).

The incorporation of legumes into the high-fibre, high-carbohydrate diet was shown to be particularly useful in decreasing blood glucose responses, HbA_{1c}, plasma triglyceride concentration, total serum, LDL- and VLDL-cholesterol and insulin response, compared with other high-fibre foods (Erdman & Fordyce, 1989; Potter et al., 1981; Simpson et al., 1981). The beneficial effects of legumes are thought to be derived from various components including its high carbohydrate content, slow starch digestion and fibre content, especially soluble fibre (Erdman & Fordyce, 1989; Vinik & Jenkins, 1988).

It is important to note that the potential impact of high-fibre diets on mineral metabolism and vitamins needs to be more closely investigated, especially the use of commonly available foods (Story et al., 1985). Several long-term studies showed no negative effect (Anderson, 1980; Anderson et al, 1987). It is possible that people at risk of mineral deficiencies, such as the elderly, may require supplements of calcium and trace minerals (American Diabetic Association, 1990a). Silvis (1989) found no adverse effects on serum mineral status of black diabetic

patients who took a DF concentrate.

Adequate fluid intake should accompany increased DF intake in order to derive the beneficial effects of DF, and to avoid possible side-effects such as flatulence and intestinal distention, as well as the small potential risk of gastro-intestinal obstruction (American Dietetic Association, 1988; Anderson & Geil, 1988). People with upper gastro-intestinal dysfunction risk bezoar formation and are cautioned against diets high in fibre, especially the leafy vegetable type (American Diabetes Association, 1990a; Anderson et al., 1987; Vinik & Jenkins, 1988).

Because high-fibre diets lower blood glucose levels, urinary glucose loss and insulin requirements in the short term (Jenkins et al., 1980a), as well as in the long term (Simpson et al., 1981), careful attention must be given to the insulin or hypoglycaemic agent dose to avoid hypoglycaemia when fibre intake is increased (Anderson et al., 1987). Careful home glucose monitoring is, therefore, important.

It may sometimes be necessary to increase fibre intakes by prescribing a fibre supplement to the diabetic patient. Jenkins et al., (1980b) found that supplements such as guar gum can effectively reduce urinary glucose loss in diabetics who have relatively high carbohydrate intakes. When carbohydrate formed less than 40 % of the energy intake, this effect was less pronounced and more variable; the proportion of carbohydrate in the diet (and especially starchy carbohydrates with very little monosaccharides and disaccharides), seems an important factor if fibre supplements are taken. Many highly purified soluble fibre supplements produce nausea, flatulence, feelings of fullness, and abdominal discomfort in some individuals (Anderson et al., 1987). However, foodstuffs high in soluble fibre without an excessive increase of total DF causes limited discomfort and represents a satisfactory compromise between efficacy and tolerance (Del Toma et al., 1988). It was shown that increased soluble fibre

intakes of konjac glucomannan increase HDL-cholesterol in diabetics (Vorster et al., 1988b).

* Proteins

Traditionally, diabetic dietary recommendations emphasized protein intake (Nuttal, 1988). Newer evidence proves that high intakes are not necessary. The primary function of protein in the diet is for growth and tissue maintenance (American Diabetes Association, 1990a). There are various stages of decrease in insulin sensitivity and insulin resistance in NIDDM which may lead to a relative insulin deficiency. After the ingestion of protein meals, insulin deficiency in NIDDM patients results in greater arterial concentrations of the branched chain amino acids and longer clearance periods of these amino acids from muscle. This reflects ongoing protein catabolism and branched chain amino acid oxidation. The greater concentration of branched amino acids is directly due to insulin deficiency and in the case of alanine, also from elevated glucose concentrations (Aoki, 1985).

Although the nutritional recommendation for protein in the diabetic diet is 12 - 20 % of total energy, this amount exceeds the Recommended Dietary Allowance (RDA) for adults of 0.8g/kg body weight (American Diabetes Association, 1990a:18; Anderson & Geil, 1988:160; Food and Nutrition Board, 1989). The RDA more closely matches the actual protein needs of adults with NIDDM (Anderson & Geil, 1988:160). However, elderly subjects and people in an acute catabolic state may require more than the RDA (American Diabetes Association, 1990a). Anderson et al. (1987:1193) recommend a protein intake of 15 % to 20 % of energy intake, with a minimum of 45 g of protein daily.

There is some evidence that the progression of diabetic nephropathy is delayed by early protein restriction. A low-protein diet has also been the standard approach for treating end-stage renal disease associated with diabetes (Wylie-Rosett, 1988). However, it has been suggested that a higher-protein diet

may benefit individuals with NIDDM by enhancing insulin secretion and that individual preference should determine protein intake. A 1.5 g protein/kg diet to facilitate weight loss while protecting lean body tissue has been recommended for obese individuals with NIDDM (Anderson et al., 1987). Further research into this area is required.

The type of protein (animal *versus* plant food source) may be of importance. Traditionally, soy protein has not formed a significant component of the Western diet. The relative contribution of soy protein to human nutrition is bound to increase because of the protein's overall positive nutritional profile, low cost, high availability, excellent functional properties in food systems, and continued innovative food-product development. Moderate substitutions of cereals and legumes in the diet can improve nutritional status with regard to desirable weight, and intakes of total fat, saturated fatty acids, cholesterol, starch and DF (Erdman & Fordyce, 1989).

The protein quality of legume protein is 62 % in comparison with the 92 % of casein (Erdman & Fordyce, 1989:726). Only the combined amount of the essential sulphur amino acids, methionine and cystine, falls below recommended patterns of amino acid in most soy proteins. The arginine content of soy protein is relatively high and the lysine concentration is particularly high. Even without supplementation with methionine, soy protein as sole source of protein is capable of meeting the amino acid requirements of adults. Complementation with cereals, such as maize and wheat improves protein status. Heat processing is necessary for soy protein to optimize digestibility (92 - 100 %) of the protein and reduce heat-labile enzyme inhibitors such as protease (Erdman & Fordyce, 1989:727).

The hypolipidaemic effect of soy protein is well- known (Simpson et al., 1982). The substitution of soy protein for beef protein usually results in striking cholesterol and triglyceride lowering effects, primarily in the LDL-fraction, with no reduction in the

HDL-fraction in especially hyperlipidaemic patients (Simpson et al., 1982). A number of mechanisms of action of soy bean protein in contrast to animal protein have been suggested (Erdman & Fordyce, 1989). These include the fact that dietary soy protein in contrast to casein may improve the catabolism of cholesterol-rich VLDL; the absorption of lipids from the gastro-intestinal tract may also be more rapid with casein than with soy protein; soy protein may also increase biliary cholesterol excretion compared with casein; soy protein may reduce cholesterol biosynthesis, increase faecal steroid excretion; there may also be a hormonal effect: the potential effects of a soy protein-induced increased insulin release on cholesterol metabolism is unclear, but elevation of insulin may affect cholesterol synthesis or lipoprotein clearance rates. The component of soy bean products responsible for the cholesterol lowering is generally presumed to be the protein.

* Fat

Almost all the risk markers of atherosclerosis occur more frequently in subjects with DM. Hyperlipidaemia, glycosylation of lipoproteins, platelet dysfunction, hypercoagulability, arterial wall changes, hyperinsulinaemia, hypertension and obesity all combine in diabetes to accelerate atherosclerosis. The maintenance of desirable serum lipid levels is, therefore, a primary management goal (Brownlee, 1985; Anderson & Geil, 1988) and is closely related to the amount and type of fat in the diabetic diet.

The metabolic disadvantages of a high-fat diet are numerous and have been reviewed by Fehily (1982), Anderson & Geil (1988) and Vorster et al., (1988a). In addition to impairing intracellular glucose metabolism, high-fat diets cause insulin resistance, decrease the number of insulin receptors in several tissues, and stimulate rates of glyconeogenesis. A high fat-intake also produces hyperlipidaemia and influences the activity of factor VII in plasma; therefore, new emphasis is being placed on

limiting fat intake.

A fat intake of less than 30 % is recommended (American Diabetes Association, 1990a:18). It may even be as low as 20 % to 25 % (Anderson et al., 1987:1193) of which less than ten percent should be saturated fat, preferably between six and eight but not more than ten percent polyunsaturated fat, and the remaining four to 14 % of the energy from mono-unsaturated fat. Total cholesterol intake should be less than 300 mg per day and preferably even less than 200 mg (100 mg/ 4 200 kJ) (Anderson & Geil, 1988:161; American Diabetes Association, 1990a:18). Kinsella (1986a:89) suggests that eight percent of energy should be derived from saturated fatty acids, twelve percent from mono-unsaturated fatty acids and ten percent from n-6 and n-3 poly-unsaturated fatty acids.

The permissible amount of cholesterol in a hypolipidaemic diet is controversial. Some workers have suggested that humans are basically resistant to dietary cholesterol and that restriction of dietary cholesterol would not reduce risk for coronary heart disease (Grundy et al., 1988). In addition to this, Edington, et al. (1989:62) implied that changes in dietary cholesterol (for example reducing intake below 400 mg per day) have little overall influence on plasma total and LDL cholesterol when cholesterol intake and total dietary saturated fatty acid intake are relatively low. People may differ in their response to the intake of cholesterol, which has led to the documentation of hypo-responders and hyper-responders to dietary cholesterol (Edington, et al., 1989; Katan et al., 1986). There are many factors, such as genetic factors, variations in absorption of 25 - 75 %, and exogenous factors, which may lead to these differences in response (Grundy et al., 1988:97).

However, carefully controlled, metabolic-ward studies leave little doubt that increasing dietary cholesterol induces a rise in the plasma total cholesterol in the majority of people. In free-living conditions, the effect of cholesterol may be

influenced by fat intake. People who eat high cholesterol diets usually tend to eat high saturated fat diets (American Heart Association, 1990; American Medical Association, 1983; Grundy et al., 1988). These high-cholesterol, high-fat diets lead to the intake of excess energy, obesity and atherosclerotic cardiovascular disease (American Heart Association, 1990).

There is evidence that impaired essential fatty acid metabolism is a feature of DM. Levels of essential fatty acids, as well as the activity of delta-6-desaturase, the enzyme which is rate-limiting for conversion of linoleic acid to prostaglandin precursors, are reduced (McCarty & Rubin, 1984). A high intake of polyunsaturated vegetable oils which contain n-6 fatty acids, will reduce total plasma and LDL-cholesterol levels *via* multiple pathways (Goodnight et al., 1982). There may, however, be some harmful effects, such as increased formation of cholesterol gallstones and promotion of obesity that should be investigated more completely.

The use of n-3 fatty acids may be more effective (Goodnight et al., 1982). Recent research has shown that certain essential fatty acids of the n-3 class lower serum cholesterol moderately and serum triglyceride levels markedly. These n-3 fatty acids, a component of fish oils, also decrease platelet aggregation, tend to decrease platelet number, increase bleeding time and may potentially reduce cardiovascular disease risk in diabetes (Kinsella, 1986b; Anderson & Geil, 1988). There are also other advantages of n-3 fatty acids, such as decreased VLDL and LDL-cholesterol, and increased HDL-cholesterol, as well as decreased blood pressure (Goodnight et al., 1982; Kinsella, 1986b). However, more research on the effect of n-3 fatty acids on total cholesterol, blood pressure, platelet aggregation, blood viscosity, membrane fluidity and blood coagulation, optimum intake of n-3 and n-6 poly-unsaturated fatty acids, optimal vitamin E intake, supplementation with fish-oil and possible side-effects is necessary (Kinsella, 1986b; Norum & Drevon, 1986; Silvis, 1988). Silvis (1989) found, for example, that essential

fatty acid supplementation in black diabetic patients caused haemolysis of red blood cells, despite supplementation with vitamin E.

The use of fish oil supplements should be investigated more thoroughly before it can be recommended to DM patients.
A prudent approach suggests eating fatty fish at least once or twice weekly (Anderson & Geil, 1988; Kinsella, 1986a).

In an effort to lower the lipid content of the diet, new fat substitutes have been developed and are being tested for safety and efficacy. *Olestra*, or sucrose polyester (a non-digestible fat from sucrose bonded with eight long-chain fatty acids into a molecule too large to be hydrolysed in the small intestine) contains no energy or cholesterol and may be of benefit in the diabetic diet. Another fat substitute is *Simplese* (a protein derivative with a creamy taste sensation), that contains only 6.1 kJ/g instead of the 37.8 kJ/g of fat, may be effective in assisting persons in staging dietary changes to reach realistic nutrition goals (Anderson & Geil, 1988). These products are not yet available in South Africa.

* Vitamins, minerals and trace elements

One of the goals of the nutritional therapy of DM is to provide optimal amounts of all vitamins, minerals and trace elements. Generally, the vitamin and mineral status of diabetic patients is not well studied, but evidence that DM subjects have unique needs that warrant vitamin or mineral supplementation has not been consistently found. Therefore, recommendations in this area are the same as for the general population (Anderson & Geil, 1988).

However, experimental evidence has suggested that deficiencies in many of the trace elements including zinc, chromium, magnesium, copper, manganese as well as vitamin B-6 may lead to glucose intolerance, but adequately controlled studies are not

available. Serum or tissue content of certain elements such as copper, manganese, iron and selenium can be higher in diabetic patients than in non-diabetic controls (Mooradian & Morley, 1987:878). Table 2.10 summarises the possible micronutrient status of diabetic subjects reported in various studies and reviewed by Mooradian and Morley (1987).

Table 2.10 Micro-nutrient status of diabetic patients

Micro-nutrient	Status in diabetic patients	
	IDDM	NIDDM
Trace elements		
Zinc	↓	↓
Chromium	↑	No difference
Calcium	↓	No difference
Magnesium	↓	↓
Copper	No difference	No difference or ↑
Manganese	No difference or ↓	↑?
Iron	No difference	No difference
Selenium	↑	?
Vitamins		
Vitamin A	?	No difference
Thiamin	No difference	No difference or ↑
Vitamin B-6	No difference or ↓	No difference of ↓
Vitamin B-12	No difference or ↓	No difference
Vitamin C	No difference or ↓	No difference of ↓
Vitamin D	↓	No difference
Vitamin E	↑	↑

(Mooradian & Morley, 1987:878).

- Specific mineral requirements

Diabetics appear to be at increased risk of osteoporosis, presumably owing to an increased urinary calcium loss (McCarty & Rubin, 1984). Dietary calcium competitively inhibits magnesium

absorption. Calcium supplementation may therefore enhance a magnesium deficiency and should not be given without careful consideration (McCarty & Rubin, 1984). Calcium supplements may, however, be necessary under special circumstances (American Diabetes Association, 1990a).

Mooradian and Morley (1987) mention that there is some evidence to suggest that a zinc deficiency may enhance IGT, and that it may play a role in the development of some forms of glucose intolerance. The effect of a zinc deficiency on insulin secretion from the pancreas is controversial - it may induce a minor impairment of insulin secretion and increased insulin resistance. However, presently available data indicate that the role of a zinc deficiency in the pathogenesis of DM is not proven (Mooradian & Morley, 1987). There is accumulating evidence that the development of DM may lead to a zinc deficiency which may lead to impaired T-cell function (with a negative effect on the immune system) and wound healing (Niewoehner et al., 1986). The bioavailability of zinc from soy protein is an issue of concern and is known to be poor and due, *inter alia*, to the calcium and phytic acid in the soy protein foods. Complexes that are poorly soluble, poorly digested, and poorly absorbed may be formed (Erdman & Fordyce, 1989).

A deficiency of chromium or its biologically active form, the putative glucose-tolerance factor, has been implicated in the pathogenesis of some forms of IGT and DM (Anderson, 1982). Urberg et al. (1986) showed that a combination of chromium and nicotinic acid improve glucose tolerance. Chromium also appears to have an important role in lipid metabolism (Rabinowitz et al., 1983), especially in raising HDL-levels and in the insulin sensitivity of tissues (McCarty & Rubin, 1984). However, prevalence of a chromium deficiency among diabetic patients has not been well established and it seems as if chromium supplementation may only be beneficial in people who have a clinical chromium deficiency (Mooradian & Morley, 1987). A high-carbohydrate, high-fibre diet usually has a high chromium content

and may have the extra benefit of enhancing chromium status in diabetics (Anderson, 1982).

DM may be associated with increased urinary loss of magnesium especially when hyperglycaemia is poorly controlled, and that may lead to hypomagnesemia (Jackson & Meier, 1968). Magnesium may be an important determinant of insulin sensitivity in NIDDM and may play a role as a second messenger for insulin action (Paolisso et al., 1990). Magnesium deficiency has been linked to three common complications of diabetes, namely retinopathy, ischaemic heart disease (Mooradian & Morley, 1987), and high blood pressure (Joffres, Reed & Yano, 1987). Tests for a magnesium deficiency should, therefore, be done on NIDDM patients (Mooradian & Morley, 1987). McCarty & Rubin (1984) suggest that, since diabetics are at increased risk for coronary disease, it is prudent to offer them the potential protection of supplementary magnesium - particularly in the light of evidence that a relative magnesium deficiency is a feature of the diabetic syndrome.

Serum concentrations of copper and ceruloplasmin are usually elevated in NIDDM patients, but the implications of these observations are not known (Mooradian & Morley, 1987). Manganese status in diabetics is controversial. Elevated serum manganese levels were reported in patients with myocardial infarction and atherosclerosis (Mooradian & Morley, 1987).

The diabetic state is not associated with iron deficiency and there appears to be no evidence of major alterations in the iron status of diabetic patients who do not have renal failure or gastro-intestinal neuropathy with malabsorption. However, iron overload, as in haemochromatosis, can cause glucose intolerance secondary to pancreatic tissue injury (Bern & Busick, 1985). Leguminous products have mainly non-heme iron. When adequate ascorbic acid is taken with the legumes, absorption is enhanced (Erdman & Fordyce, 1989).

- Specific vitamin requirements

A decreased status of thiamin, vitamin B-6 and vitamin B-12 (only IDDM) has been associated with carbohydrate intolerance or DM. The thiamin status of diabetic patients is controversial. At present there are no data to suggest that thiamin supplementation should be recommended for diabetic patients. Although studies indicate that the vitamin B-6 levels in patients with the disease are significantly reduced, supplementation of the diabetic diet with this vitamin cannot be justified, except for some of the patients with severe neuropathy (Mooradian & Morley, 1987).

Numerous reports have suggested an intimate interrelationship between vitamin C and glucose homeostasis. Together with selenium and vitamin E, ascorbic acid has been shown to enhance a number of aspects of the immune function (McCarty & Rubin, 1984). DM appears to influence tissue ascorbic acid content. NIDDM subjects have a higher turnover of ascorbic acid. At present, minimum daily vitamin C requirements for the diabetic population are not known. In the absence of poor dietary vitamin C intake there seems to be no justification for high-dose vitamin C therapy (Mooradian & Morley, 1987).

In view of the known ability of vitamin E to inhibit platelet aggregation and the enhanced platelet aggregation in patients with diabetes, it seems likely that the reported increase in vitamin E in diabetics may be an attempt to compensate for the increased adhesiveness of diabetic platelets. Further study of this issue is indicated (Vatassery, Morley & Kuskowski, 1983).

- Vitamin and mineral supplementation in special circumstances

Special consideration for vitamin and mineral supplementation should be given to NIDDM patients on very low-energy diets for weight reduction (American Diabetes Association, 1990a).

High fibre diets, especially when rich in insoluble fibre and

phytate, may lead to a decrease in the absorption of calcium, iron and zinc and could result in deficiencies if maintained for a long time in susceptible individuals, although these deficiencies may have little significance in terms of energy balance. Theoretically vitamin B-12 and iron deficiency could result if the high-fibre diet is low in animal protein, but the abundance of epidemiological data point away from this. It is, therefore prudent to caution against the unmonitored long-term use of fibre in individuals at risk, such as the elderly, although there is no evidence that soluble fibre supplements or moderate increases in fibre intake from mixed sources cause malabsorption of micro-nutrients and macro-nutrients (Vinik & Jenkins, 1988).

There are inconsistencies in responses of diabetic patients to mineral and vitamin supplementation that may be secondary to the heterogeneity of this patient population. It is also likely that the response of patients to supplements is determined by their nutritional status. Judicious replacement of micronutrients in diabetic patients with demonstrated deficiencies may therefore well be necessary (Mooradian & Morley, 1987). Further research on the vitamin and mineral status of diabetic patients is definitely needed.

2.4.2.3 **Additional considerations**

* Alternative sweeteners

A continuing area of controversy in the nutrition management of DM is the use of various nutritive and non-nutritive sweeteners. More information is needed about the diabetic palate to determine actual needs for alternative sweeteners. The American Diabetes Association (1990a) states that the use of various nutritive and non-nutritive sweeteners is acceptable in the management of the disease. The American Dietetic Association (1987:1689) emphasises the point that individuals should "use moderation in their consumption of both nutritive and non-nutritive

sweeteners".

- Nutritive sweeteners

According to the American Diabetes Association (1990a), only individuals in whom diabetes is reasonably well-controlled should use nutritive sweeteners such as fructose or sorbitol. Xylitol has voluntarily been removed from the USA market because of possible safety concerns. The metabolic effects of chronic ingestion of diets containing fructose, mannitol, xylitol and/or sorbitol need further study to establish whether their use as a part of diet management is beneficial.

Fructose offers advantages over sucrose for diabetic persons because it tastes sweeter, is metabolized without insulin, and does not produce as rapid a rise in blood glucose levels as other simple carbohydrates (Anderson & Geil, 1988). However, glucose newly formed from fructose in the liver requires insulin in the same way as dietary-derived glucose in order to be metabolised. It can contribute to an increase in blood glucose rather than be stored as glycogen in the absence of enough insulin (Woods & Bax, 1982; American Dietetic Association, 1987).

The sugar alcohols are absorbed slowly from the gastro-intestinal tract and have less influence on blood glucose and insulin levels than glucose, sucrose or fructose (Anderson & Geil, 1988). However, sorbitol is rapidly converted to fructose and then to glucose in the liver. Initially it is not directly dependent on insulin, but it then follows the same metabolic pathways as fructose (American Dietetic Association, 1987). Possible side-effects of sorbitol and mannitol include osmotic diarrhoea, abdominal discomfort, gas formation and malabsorption, even with relatively low oral doses (Anderson & Geil, 1988). An intake of not more than 75 g of fructose, 30 - 50 g of sorbitol and not more than 30 - 40 g of xylitol per day is recommended (Olefsky & Crapo, 1980:393; Woods & Bax, 1982:214).

There is a lack of research demonstrating a definite benefit of nutritive sweeteners in improving dietary adherence of weight reduction (Anderson & Geil, 1988). Actually, the use of nutritive sweeteners such as fructose and sorbitol, in the belief that their energy contribution is not significant while it contributes exactly the same amount of energy as sucrose, may undermine efforts to lose weight and could even lead to weight gain (American Diabetes Association, 1990a).

- Non-nutritive sweeteners

Saccharin, aspartame and cyclamate comprise the non-nutritive sweeteners. The Food and Drug Adminstration of the USA has banned the use of cyclamate and has recommended safe intake levels of up to a 1 000 mg saccharin, and 50 mg aspartame per day (American Dietetic Association, 1987:1692; Woods & Bax, 1982:214). Saccharine may leave a bitter after-taste and does not bake well. The safety of the product is also currently under study. A disadvantage is that it may be packed with lactose or dextrose. Aspartame has a good sweetening capability and is available in a wide variety of products. It was extensively tested, also by the Food and Drug Administration, and found save at recommended intake levels, which is much higher than the usual intake. It is, however, expensive and cannot be used for baking (Heins, Wylie-Rosett & Green-Davis, 1987).

If sweeteners are used, the use of various sweeteners, each with its particular advantages, is recommended to disperse any potential risks. Individual ingestion of sweeteners should be limited to the established safe levels when available. Sweetener intake should only be considered as part of an otherwise balanced diet (American Diabetes Association, 1990a; American Dietetic Association, 1987). The use of speciality foods for diabetics, with alternative sweeteners, but usually with a high fat content, should be discouraged (Woods & Bax, 1982).

* Salt intake

Control of hypertension is one of the initial steps in the management of micro-vascular and macro-vascular complications associated with DM. The recommended sodium intake is 1 000 mg/ 4 200 kJ, not to exceed 3 000 mg/day (American Diabetes Association, 1990a:21). A moderate restriction in dietary sodium intake is believed to be sensible, with a more severe restriction required in some cases of hypertension (Anderson & Geil, 1988). Sodium restriction could also be harmful for certain individuals with poorly controlled diabetes, postural hypotension and fluid imbalance (American Diabetes Association, 1990a).

* Alcohol

Alcohol in moderation poses no greater health risk for diabetic than for non-diabetic persons, provided sensible guidelines are followed (Anderson & Geil, 1988). However, it is not an essential part of the diet and the patient could go without it. If alcohol is to be included in the diet, its effect on blood glucose and blood lipids must be considered (Hagan & Wylie-Rosett, 1989). The energy contribution should not exceed six percent of daily intake (which translates into no more than two glasses of dry wine or two beers daily) (Anderson et al., 1987:1193) and alcohol should only be taken by people with well-controlled glucose levels.

Specific concerns for persons with diabetes include the habit-forming and toxic effects of alcohol, errors in insulin or oral hypoglycaemic use, missed meals, hypoglycaemia due to alcohol intake, the possible unpleasant reaction of alcohol with oral sulphonylurea agents and stimulation of hypertriglyceridaemia (Anderson & Geil, 1988). Hypertriglyceridaemia may result from the increased esterification of fatty acids and synthesis of VLDL during ethanol metabolism. The effect of ethanol may be enhanced if it is accompanied or followed by a high-fat meal (Hagan & Wylie-Rosett, 1989).

* Weight reduction

- Reasons for and objectives of weight reduction

The National Institutes of Health (1987) state that individuals who are 20 - 30 % overweight are at an increased risk for NIDDM. Data suggest that in addition to the degree of obesity, increasing duration of obesity increases the risk. The specific fat distribution is also important; upper-body or android obesity appears to be more strongly associated with diabetes than lower-body or gynoid obesity (National Institutes of Health, 1987). Upper-body obesity, even when mild, is associated with higher glucose levels, exacerbated insulin resistance, greater abnormality of the lipoprotein profile, and increased cardiovascular risk (American Diabetes Association, 1990a). The WHR, as an indication of type of obesity, is discussed in section 2.7.3.

Dietary interventions directed toward weight reduction and improvement in blood glucose and lipids of the obese person with NIDDM have the greatest potential for a significant positive effect on morbidity and mortality. The metabolic improvements achieved with weight reduction of obese diabetic patients are indisputable and include reduction in hyperlipidaemia, hyperglycaemia, hypertension, proteinuria and the cardiovascular risk profile (American Heart Association, 1990; Uusitupa et al., 1990). Weight reduction also leads to more general benefits, including improved pulmonary function, reduced risks during operations and reduction of musculoskeletal problems. In addition, the improvement in glucose tolerance with reduction of energy intake may reduce or eliminate the need for oral hypoglycaemic agents or insulin. The timing of weight loss relative to the progression of NIDDM may be of critical importance to the long-term prognosis and possibly to the delay of onset or prevention of the development of complications. The objectives of therapy should be to induce weight loss, preserve lean body mass and to reduce WHR. Individualized and reasonable

weight goals need to be negotiated (American Diabetes Association, 1990a).

- Prescription

The diet should be nutritionally balanced, providing a variety of foods, and the energy level restricted (American Diabetes Association, 1990a). A reasonable weight-reducing diet for mild to moderately overweight adults should provide 3 400 - 5 000 kJ for women and 4 200 - 6 700 kJ for men. For morbid obesity a severely restricted high-carbohydrate, high-fibre diet, providing 2 100 - 3 400kJ daily, with careful supervision and intensive behaviour modification, may prove effective (Anderson et al., 1987:1194). Uusitupa et al. (1990) reported that a very low-energy diet of 2 100 kJ may be beneficial in patients who still have some insulin-secretion capacity but who have failed to be controlled with oral glycaemic agents. Very low-energy diets have also been studied in a small number of persons with NIDDM with reportedly safe and effective results in the short term by Henry, Wiest-Kent & Scheaffer (1986). Vitamin and mineral supplementation should then be used (American Diabetes Association, 1990a). Concern remains, however, about the long-term use of very low-energy diets, particularly in the area of endogenous protein metabolism (Anderson & Geil, 1988).

Although normalization of body weight is a desirable goal, even modest energy restriction, *per se*, may be beneficial due to the positive effects on blood glucose and requirements for insulin and oral anti-diabetic agents (American Diabetes Association, 1990a).

- Use of dietary fibre

Several studies have demonstrated that high-fibre diets and fibre supplements increase satiety and promote weight loss (Anderson et al., 1987). Delayed gastric emptying and release of certain gut hormones may contribute to the greater satiety of the high-

fibre diets. Leeds and Judd (1986) mention that nutrients such as starch, fatty acids, and nitrogen may be less well absorbed from high-fibre foods, providing slightly fewer calories than from comparable low-fibre foods. High-fibre foods usually take longer to eat too.

- Behavioural modification

The results achieved in most weight-control programmes are modest, and the outcome is extremely variable. To develop more effective weight-loss programmes, it would be helpful to identify the variables related to successful outcome. Changes in behaviour made by patients during and after treatment have been more strongly associated with outcome (Guare et al., 1989). There has been little research on the behaviour changes associated with weight loss in NIDDM patients. Four eating behaviours were found to be significantly related to weight reduction (Guare et al., 1989):

- If people can be persuaded to eat foods that they believe will aid them in losing weight;
- if they record the type and quantity of food that they eat;
- if they can be persuaded to refuse food offered to them by others; and
- if they could be persuaded to eat small amounts of food.

By simplifying treatment programmes and stressing the behaviours most clearly associated with weight-loss, long-term use of weight-loss programmes may be more successful (Guare et al., 1989).

- Maintenance diet

After successful weight loss, there is often great difficulty in sustaining the reduced body weight. It appears that stabilization of body weight at a reduced level requires the

continued ingestion of a restricted energy level and a lifelong commitment to sustaining the reduced weight. The prevention of regaining of lost weight requires greater attention if the benefits of reducing body weight of the obese are to be realized (American Diabetes Association, 1990a).

* Hypertension

- Associations with other factors

The association between hypertension and obesity is quite strong. Weight loss, energy restriction and exercise are associated with a fall in blood pressure but the mechanism of the antihypertensive effect of weight loss is undetermined. These nonpharmacological modalities should, however, be fully pursued in all cases (American Diabetes Association, 1990a; Seedat, 1989b; Simopoulos, 1985).

Hypertension is also associated with NIDDM independently of obesity (Rosendorff, 1989).

Patients with obesity, NIDDM and hypertension often have hyperinsulinaemia and/or are insulin resistant, although proof of insulin resistance in hypertension is not well established (Rosendorff, 1989). It was also shown that a daily intake of gamma-linolenic acid can significantly reduce blood pressure in moderate hypertension and may lead to spontaneous weight loss in some obese subjects (McCarty & Rubin, 1984).

- Salt intake

It is well-known that not everyone who has a high salt intake develops hypertension and that there are no known means at present to detect those who are salt-sensitive. However, persons prone to hypertension should restrict their salt intake, especially those with chronic renal disease, those with hypertensive parents, certain racial groups such as black men,

and people older than 50 years of age (Simopoulos, 1985). The advantages of sodium restriction over diuretic therapy include maintenance of normal body potassium stores and avoidance of the metabolic complications associated with administration of diuretics (Tuck, 1988).

The advocacy of dietary restriction of sodium is not without reservations because only 50 - 67 % of hypertensives on sodium restriction will respond with a measurable drop in blood pressure (Seedat, 1989b). A diet with too little or no sodium, besides being difficult to achieve, may be counterproductive. By activating both the sympathetic nervous system and the renin-aldosterone mechanism a sodium intake of less than 50 mmol/day may minimise or reverse the reduction of blood pressure and decrease the effects of concomitant diuretic therapy.

Despite reservations, a moderate dietary salt restriction seems to be practical and useful. There is even hope that reduction of sodium intake, beginning during infancy, will prevent the development of hypertension. The easiest and safest plan is simply to avoid adding salt to food in cooking and at table. Patients should select foods which have low amounts of sodium added during processing and fresh foods that are naturally low in sodium. The success of a diet high in DF in the lowering of hypertension may also be attributed to the fact that DF may lower sodium absorption (Seedat, 1989b).

Whenever dietary sodium is reduced by eating fresh foods low in sodium, the relative intake of potassium will increase because these two cations predominate in most foods, the less of the one, the more of the other. For most hypertensive patients, the relatively greater intake of potassium may prove beneficial because it can lower blood pressure. An increase in potassium intake can be achieved with a potassium-based salt substitute and a moderate increase in vegetable and fruit consumption (Seedat, 1989b).

- Other nutrients

Lacto-ovovegetarian diets successfully reduce blood pressure because of their high potassium content, but they also contain significant DF, vitamin C, vitamin E, magnesium, calcium and less total protein, saturated fat, mono-unsaturated fat and vitamin B-12 (Seedat, 1989b). Magnesium, calcium, phosphorus, potassium, vitamin C and vitamin D intakes are significant variables that show inverse associations with blood pressure. It is not that easy to separate the effect of different substances, such as magnesium, from that of other variables on blood pressure, because of the problem of high intercorrelation among many nutrients (Joffres, Reed & Yano, 1987).

Several types of epidemiological studies, dietary surveys and clinical data support the positive relationship of calcium with blood pressure. Oral calcium and calcium channel blockers therapy can be used for treatment but dietary calcium should remain the preferred treatment because of the cost and inherent risk of side-effects of pharmaceutical compounds. Dietary sources also provide other important nutrients. An intake of 800 - 1500 mg of calcium per day is recommended at the moment, although more research is necessary (Henry et al., 1985).

A reduction of total dietary fat and an increase in the ratio of polyunsaturated to saturated fat (P/S ratio), as in the Mediterranean diet, may lower blood pressure by increasing the synthesis of vasodilatory prostaglandin (Seedat, 1989b).

Hypertensive patients may also benefit from a reduction of dietary protein intake, particularly those with underlying renal disease as is often the case in diabetic hypertensive patients. A high-protein intake is known to increase the glomerular pressure and flow. This presumably accelerates the progress of glomerular sclerosis (Meyer, Anderson & Brenner, 1983).

- Alcohol

Heavy alcohol consumption may either cause or aggravate hypertension. There may be a deleterious effect on blood pressure with any degree of alcohol consumption (Seedat, 1989b).

In summary, dietary modification is suitable only for certain individuals, especially those with mild hypertension. However, nutritional therapy may substitute for drugs in a sizeable proportion of hypertensive patients or, if drugs are still needed, can lessen some unwanted biochemical effects of drug treatment (Seedat, 1989b).

* Hyperlipidaemia

Hyperlipidaemia has already been discussed in section 2.3.3.2. Only the nutritional or dietary recommendations to treat hyperlipidaemia will be discussed here.

- Weight reduction is essential if obesity is present (American Diabetes Association, 1990a).

- The risk of cardiovascular disease is high among subjects with diabetes. There is a known association between abnormalities in plasma lipid, lipoprotein concentrations and increased risk for cardiovascular disease. A fat-modified diet has established favourable effects on plasma lipids and lipoprotein concentrations. Because of all these facts, a fat-modified diet should be prescribed and hyperlipidaemia (American Diabetes Association, 1990a:21). That means that total fat intake, saturated fatty acids and cholesterol intake should be reduced, with small increases in polyunsaturated and mono-unsaturated fatty acids. The use of n-3 fatty acids may be considered.

- Changing the source of protein in the diet may have the additional benefit of decreasing total protein intake, a

change that may delay or prevent the potential renal complications and increase carbohydrates and DF. The DF will also increase satiety and assist in weight reduction (American Diabetes Association, 1990a:21; Hagan & Wylie-Rosett, 1989). This is most easily accomplished by decreasing animal foods and increasing vegetable foods (Hagan & Wylie-Rosett, 1989).

- Normo-lipaemia may not be achieved with optimal anti-diabetic therapy in some subjects, presumably due to co-existence of diabetes and a disorder of lipoprotein metabolism. A stricter fat-modified diet of 25 % of the energy from fat and cholesterol of 200 - 250 mg/ day may be necessary in such cases. In a second step fat restricted to 20 % of the energy intake and cholesterol to 100 - 150 mg per day, should be prescribed for those individuals who still have hyperlipidaemia. Under these circumstances protein intake should be liberalized. Additional hypolipidaemic agents may also be necessary. If individuals continue to exhibit an increase in VLDL-triglycerides and cholesterol concentrations, they are not likely to benefit from the stricter fat-modified diets, probably due to a disorder of the lipoproteins. The usual diabetic diet should then be followed.

- In the unusual event of massive hypertriglyceridaemia (plasma triglycerides 1 000 - 2 000 mg/dl), the danger of acute pancreatitis is high. The dietary fat intake should then initially be restricted to 10 - 20 % of total energy. Appropriate anti-diabetic therapy, management of accompanying causes of secondary hyperlipidaemia, and possibly a fibric acid derivative, will also be necessary (American Diabetes Association, 1990a:21).

2.4.2.4 **Practical applications**

Translating the diet prescription and all the aspects mentioned above into life-style changes and monitoring metabolic changes,

present a major challenge (Hagan & Wylie-Rosett, 1989). Quality of live implications should be considered when implementing any dietary prescription. Dietary manipulations should be made sequentially rather than simultaneously (Wheeler, Delahanty & Wylie-Rosett, 1987).

* High-carbohydrate, high-fibre, low-fat diet

A high-carbohydrate, high-fibre, low-fat diet, coupled with regular exercise and self-monitoring, should serve as the primary intervention strategy in diabetes management, with insulin and oral agent therapy supplementation when necessary (Anderson et al., 1987). However, there are researchers who suggest that such a diet can lead to changes in carbohydrate and lipid metabolism (such as elevated triglyceride levels) associated with an increased risk of coronary heart disease and that the recommendations should be reconsidered (Coulston et al., 1989).

Putting high-carbohydrate, high-fibre, low-fat diets into practice requires coordination between dietitians and other health care providers. Most individuals can increase their intake of fibre-rich foods with little difficulty, if they do so gradually. This diet usually requires individuals to decrease their intake of meat, high-fat dairy products and added fats and increase intake of natural carbohydrate foods such as whole grains, fruits, vegetables and definitely legumes (Anderson et al., 1987). The other family members should also be involved in the dietary changes and counselling; the diet can be a healthy way of eating for the entire family (American Diabetes Association, 1987).

* Individualization

A critical element in the effectiveness of the diet in the long term is individualization. Flexibility within the guidelines previously outlined should be encouraged to promote adherence (Wheeler, Delahanty & Wylie-Rosett, 1987). The best nutrition

plans are not effective if people do not follow them. Current eating habits and desirable weight estimates should be taken into consideration. An education strategy suited to the individual patient should be followed (Anderson & Geil, 1988).

Large differences in day to day management of diabetics should be eliminated because they may put extra strain on insulin requirements and may have a negative influence on metabolic control. The best meal pattern for the NIDDM may be six small meals that are equally distributed during the day and that can keep the day to day distribution of nutrients the same (Vorster, 1986).

* Special dietary modifications

Other disease and physiological states, coexisting with diabetes, require special dietary modifications. Individuals with hypercholestrolaemia should decrease cholesterol intake to 200 mg or less daily, and increase soluble fibre intake. Hypertriglyceridaemic individuals should restrict fat to a minimum, avoid alcohol and limit energy intake, if necessary. Overweight individuals should lower energy intake and start a regular exercise programme. A multivitamin and mineral supplement may be necessary for persons on a low-energy, high-fibre diet (Anderson et al., 1987).

2.4.2.5 **Summary of dietary treatment**

Recent developments in the rapidly changing field of nutrition have raised questions about the optimal carbohydrate, protein, and fat intake for persons with DM, as well as the use of fibre and the role of the GI of foods. Considerable clinical and experimental data have emerged to support recommendations of a diet generous in complex carbohydrates and fibre and restricted in fat and cholesterol. It is clear that both high DF diets and low dietary fat diets contribute independently to improved metabolic control in NIDDM patients (Nuttall, 1988; O'Dea et al.,

1989). Diets high in complex carbohydrate and fibre do not only improve glycaemic control, but also reduce insulin requirements and lower serum cholesterol concentrations. DF, therefore, offers distinct health advantages for persons with diabetes. Because almost all risk factors for atherosclerosis occur more frequently in persons with diabetes, it is even more important that emphasis be placed on limiting fat intake. Current recommendations for persons with diabetes include all the nutrition principles of a prudent or health-promoting diet. Individual energy needs should be evaluated to assist the patient in attaining and maintaining desirable body weight (Anderson & Geil, 1988). A short summary of the most important dietary recommendations for NIDDM is given in Table 2.11.

2.4.3 Medication

2.4.3.1 **Oral hypoglycaemic agents**

* Introduction

It is not easy to choose medication for the diabetic patient. Factors such as severity of the disease, ability to secrete insulin, potency of the drug, duration of action and patient compliance all play a role in the choice of the correct drug (Krall, 1985a).

Krall (1985a) gives rough guidelines for choosing for those who may benefit from treatment with oral agents:

- Onset of diabetes after the age of 40
- Duration of diabetes of 10 years of less
- Use of daily doses of insulin of 20 - 30 units or less.

There are, however, many exceptions to these guidelines.

Before starting treatment with oral agents, a trial treatment with diet alone is essential for at least two to three weeks. If this fails, low dosages of oral agents should be introduced and increased until good glycaemic control is achieved.

Successful treatment can often be reached with the use of sulphonylurea and biguanide agents together. With their combined effectiveness, the use of oral agents is prolonged, sometimes for a number of years, in cases that would have required treatment with insulin otherwise (Hermann, 1990).

Table 2.11 Summary of the nutritional recommendation for non-insulin-dependent diabetes mellitus

Energy	Normal intake except in the case of obesity. With obesity, weight reduction important, restrict energy intake.
Carbohydrates	Diet high in complex carbohydrates (55 - 60 % of energy). Restrict the intake of sugar and nutritive sweeteners, especially in the case of obesity.
DF	Diet high in DF (± 40 g to maximum of 50 g/ day). Soluble and natural sources important.
Protein	Normal protein intake with emphasis on plant protein.
Fat	Low-fat diet with a fat intake of less than 30 % of energy. Saturated and polyunsaturated sources should each be less than ten percent of energy intake. Polyunsaturated n-3 sources from fish should be included. Cholesterol intake less than 300 mg/day.
Vitamins	Normal intake
Minerals	Normal intake Restrict sodium intake to less than 3000 mg/day.
Non-nutritive sweeteners	Moderation and variety necessary.
Individualisa= tion	Important
Exercise	Important

* Sulphonylurea agents

Reviewed by Cooppan (1987), Kolterman & Olefsky (1984) and Krall (1985a), it seems as if the primary action of the sulphonylurea agents may be to stimulate insulin release, because functioning pancreatic islets are needed for the effectiveness of these compounds. The most direct effects of the sulphonylureas are related to the release of some insulin from viable beta-cells. Along with inhibiting glyconeogenesis and a decrease of hepatic glucose production, they possibly have effects on glucagon, inhibition of prostaglandin secretion, and on the sensitivity and actual increased number of insulin receptor binding sites (Krall, 1985a).

Newer sulphonylurea agents, such as glibenclamide, are more precise in their targeted action, have a longer action, have a shorter half-life and have no antidiuretic effects. Side-effects are fewer and they reduce the potential for hypoglycaemia when used in combination with other medication. They also have extra beta-cell actions, such as the stimulation of somatostatin and the inhibition of glucagon as well as its effect on receptor cells (Krall, 1985a).

Sulphonylurea agents only have minor side-effects, including gastro-intestinal distress, anorexia, malaise, fever, skin eruptions, muscular weakness, lethargy and dizziness. The most important adverse effect is long-lasting hypoglycaemia, usually caused by the wrong dosage or by drug interactions (Melander, Lebovitz & Faber, 1990). Acute flushing of the skin surface and other vascular phenomena in patients who use alcohol while under treatment with sulphonylurea agents may occur. Some researchers believe that such a reaction in non-diabetic persons may indicate a pre-diabetic state (Krall, 1985a). No negative side-effects in the long-term could be found by Faber et al. (1990).

There is, however, not much proof of the benefits of these drugs to reduce the risk of long-term complications of DM. There is

clearly a need for more information in specific groups such as the elderly (Halter & Morrow, 1990).

The documented toxicity of the sulphonylurea compounds has been low, considering the wide use of the agents. Hepatic damage may occur but only a few cases have been reported up to now. Haematologic and dermatologic toxicity is rare (Krall, 1985a).

Hypoglycaemia may occur with any oral hypoglycaemic agent and usually because of an overdosage or inadequate food intake. Combinations with other medication (especially ethanol, corticosteroids and some hypertensive agents) should be done with care. Surgery, pregnancy, old age and other stress situations may also lead to hypoglycaemia or hyperglycaemia when a patient is on sulphonylurea agents (Krall, 1985a).

* Biguanides

Agents such as metformin are indicated for the treatment of NIDDM patients in whom satisfactory control of blood glucose cannot be obtained by diet alone. Combined with sulphonylureas they are most effective (Cooppan, 1987; Vigneri & Goldfine, 1987). Biguanides lead to a decrease in plasma triglycerides, VLDL-triglyceride, LDL-cholesterol, and an increase in plasma HDL-cholesterol (Wu et al., 1990). They also cause weight loss (Vigneri & Goldfine, 1987).

Many mechanisms have been proposed to explain the action of the biguanide agents such as metformin (Krall, 1985a; Nosadini et al., 1987; Vigneri & Goldfine, 1987). It is clear a combination of mechanisms is involved. The agents are active only in the presence of endogenous insulin, but have no effect on the beta-cells. Insulin availability, initiated by sulphonylureas enhances the effect of biguanides to increase insulin effectiveness and cell binding sensitivity and therefore, increases glucose disposal rate. Increase in insulin binding by biguanides such as metformin may be secondary to the decrease in

insulin concentration (Wu et al., 1990). Research has indicated that insulin action is influenced at the post-receptor level (Nosadini et al., 1987). These agents do not interact with the liver and have a short half-life (Vigneri & Goldfine, 1987).

Biguanides may be responsible for isolated cases of lactic acidosis, when misused (Vigneri & Goldfine, 1987) and was, therefore, discontinued in certain countries, but not in South Africa.

* Other agents

Many herbal drugs have been used to treat diabetes. More than 400 traditional plant treatments for DM have been recorded, but scientific proof of their efficiency is very scarce. An example of herbal drugs used in South Africa for NIDDM is *Gymnema sylvestre* or Gurmar, a leaf with guanidine as the active substance (Bailey & Day, 1989: 559). It is, however, the mainstay of diabetic treatment in underdeveloped regions and is also used by black NIDDM patients in South Africa, often on the recommendation of witch doctors.

Alpha-glucosidase inhibitors such as acarbose, part of the group called starch blockers, can also be used in combination with other hypoglycaemic agents but more research is necessary on their effectiveness and side-effects (Reaven et al., 1990).

2.4.3.2 **Insulin therapy**

In cases of primary failure of treatment with oral hypoglycaemic agents, despite strict dietary and exercise regimens, insulin treatment may be necessary for some NIDDM subjects. Intercurrent infection or stress, or a gradual progression in the diabetic state, may also lead to the ineffectiveness of oral hypoglycaemic agents and the necessity of insulin therapy. Sometimes insulin therapy can be combined with oral hypoglycaemic agents in the above-mentioned cases. Some patients who change to insulin in

these instances will be able to change back to oral hypoglycaemic agents. However, in most cases, once having become insulin-requiring, they will need to remain on insulin (Distiller, 1989).

The dose required can be predicted from the level of the fasting blood glucose and the degree of obesity. When monitored by fasting blood glucose concentration, there is little risk of hypoglycaemia, and the patient can continue a normal life-style (Turner & Holman, 1990).

2.4.3.3 **Hypolipidaemic agents**

Five different classes of hypolipidaemic drugs are currently available, namely bile acid-binding resins, nicotinic acid, fibric acid derivatives, HMG CoA reductase inhibitors, and probucol. Because of the side-effects of the different drugs and the lack of information on the dosage for diabetics, it is recommended that bile acid-binding resins should be used as initial treatment (American Diabetes Association, 1990a). Attention has recently been focussed on *Acipimox,* a second generation nicotinic acid analogue for lipid lowering in diabetic patients (Sheperd, 1990), *inter alia,* because of its hypotriglyceridaemic effect.

2.4.3.4 **Antihypertensive agents**

The selection of an appropriate drug regimen for the treatment of hypertension in diabetic subjects entails special consideration (American Diabetes Association, 1990a).

Thiazide diuretics are recommended when renal function is normal (Tuck, 1988a). However, thiazide diuretics may have adverse effects on glucose and potassium levels, and thiazide diuretics as well as beta-blockers may effect lipid profiles (American Diabetes Association, 1990a). Diuretic agents produce elevations in LDL and VLDL-cholesterol but the long-term significance of these elevations has not been tested yet. A potassium-sparing

diuretic should be used, if at all (Tuck, 1988).

Beta-blockers which act on the renin-angiotensin system are not very effective in the NIDDM population. They may impair insulin release and worsen glucose tolerance. The centrally acting agents such as methyldopa and alpha-adrenergic-blocking agents such as prazosin also have side-effects which make their use less practical (Tuck, 1988).

Angiotensin-converting enzyme inhibitors and calcium channel blocking agents may be a better choice (American Diabetes Association, 1990a). The angiotensin-converting enzyme inhibitors have many benefits in the presence of renal disease and may be considered as the best antihypertensive agents in diabetic patients, especially in combination with a diuretic (Tuck, 1988).

2.4.4 Exercise

Exercise should be part of the life-style changes of the NIDDM patient and should be integrated into dietary programmes of the patients (Franz, 1987).

2.4.4.1 **Advantages**

Exercise as an adjunct to low-energy diets can help the obese diabetic patient to lose weight and normalise blood glucose and lipid levels (Barnard et al., 1982; National Institutes of Health, 1987; Williams et al., 1990) and reduce the medication dosage, especially in combination with a high-fibre, high-carbohydrate, low-energy, low-fat diet. Patients with the highest level of exercise show the best long-term weight loss and improvement in glycaemic control (Barnard et al., 1982; Wing et al., 1988). Vigorous exercise appears to blunt the rise in blood glucose that follows carbohydrate ingestion (National Institutes of Health, 1987).

Intensive exercise may lead to lower triglyceride and VLDL concentrations that might be explained in part by high lipoprotein lipase activity causing chylomicron and VLDL particles to be catabolized and cleared more rapidly as well as reduced hepatic lipase activity (Williams et al., 1990).

In addition, exercise training may increase insulin sensitivity. This change appears to be an acute effect associated with recent exercise and is reversed within two to three days by physical inactivity. Regular exercise may also increase the number of insulin receptors (Barnard et al., 1982).

Exercise also has psychological benefits. It decreases anxiety, improves mood and self-esteem, increases the sense of well-being and enhances quality of life (American Diabetic Association, 1990b).

The effect of regular physical exercise alone on metabolic control in NIDDM is variable and frequently of small magnitude. Greater improvement in glucose homeostasis can usually be obtained by weight loss supplemented with exercise. The specific benefits of exercise, as summarised by Vignati and Cunningham (1985) and general benefits as summarised by Franz (1987), are given in Table 2.12

2.4.4.2 Potential risks

The potential complications of exercise need consideration in patients with NIDDM. Potential risks are summarised in Table 2.13. The risk of these complications can be minimized if patients are screened before embarking on an exercise programme, if the exercise is correctly prescribed and if the patient is carefully monitored.

Table 2.12 Advantages of regular endurance exercise for the diabetic patient

General effects
Improved fitness (flexibility, muscle strength, cardiorespiratory endurance)
Improved psychological state (copeing with stress, better self-confidence, self-image)
Change in body composition (more lean tissue)
Weight control
Improved physical work capacity
Metabolic effects
Increased insulin sensitivity (decreased requirements)
Normalization of fuel oxidation rates
Increased oxidative enzymes
Increased storage of glycogen
Increased amino acid uptake
Increased maximal oxygen uptake
Cardiovascular effects
Decreased HbA_{1c}
Decreased triglycerides
Increased HDL-cholesterol
Lower resting blood pressure
Improved peripheral circulatory characteristics
Increased oxygen transport (increase 2,3 diphosphoglycerate, decrease blood viscosity)
Increased cardiac dynamics (stroke volume and cardiac output)

Franz, 1987:875; Vignati & Cunningham, 1985:454.

Table 2.13 Potential adverse effects of exercise on NIDDM

Cardiovascular
Cardiac dysfunction and arrhythmias due to ischemic heart disease
Excessive increments in blood pressure during exercise
Post-exercise orthostatic hypotension
Micro-vascular
Retinal haemorrhage
Increased proteinuria
Acceleration of micro-vascular lesions
Metabolic
Worsening of hyperglycaemia and ketosis
Hypoglycaemia in patients on insulin or sulphonylurea therapy
Musculoskeletal and traumatic
Foot ulcers (especially in the presence of neuropathy)
Orthopaedic injury related to neuropathy
Accelerated degenerative joint disease
Eye injuries and retinal haemorrhage

(American Diabetes Association, 1990b:786).

2.4.4.3 Conditions for exercise

NIDDM patients should undergo a thorough medical evaluation before increasing physical activity. The components of the evaluation will vary depending upon the severity and duration of the disease, the presence of complications, the likelihood of symptomatic coronary heart disease and the intensity of the activity (National Institutes of Health, 1987).

All NIDDM subjects who start with an exercise programme should have a medical examination with an exercise-stress electrocardiogram if the subject is older than 35 years of age

(American Diabetes Association, 1990b).

2.4.4.4 **The exercise programme**

Although exercise is a central therapeutic modality in diabetic patients, its prescription and expected effects have remained far less precise than those of diet and medication (Caron et al., 1982). In planning and recommending an exercise programme for NIDDM patients, health professionals should be aware of several factors. The diabetic should be well-controlled before an intensive regular exercise programme is started (Franz, 1987). The threshold of energy expenditure required to reduce postprandial hyperglycaemia and enhance insulin sensitivity has not been defined. The same holds true for the use of physical activity in lowering the incidence of coronary heart disease. Exercise should be individualised, frequent and continuously (National Institutes of Health, 1987). An effective programme should thus take type of activity, intensity of activity, duration and frequency into account (Franz, 1987). Motivation is also very important, because up to now, the success of weight loss and exercise programmes has been limited (Cooppan, 1987).

The American Diabetes Association (1990b:804) recommends an exercise programme that includes aerobic exercise at 50 - 70 % of an individual's maximum oxygen uptake. It should last 20 - 45 minutes and should be repeated at least three days per week. Low-intensity warm-up and cool-down exercises are also necessary.

Exercise alone, without concurrent energy restrictions, rarely results in significant weight loss. When the energy content of the diet is severely restricted (less than 4 000 kJ per day), carbohydrate intake should be maintained to preserve normal muscle glycogen stores and therefore the capacity for vigorous exercise and endurance. Energy requirements of special situations, such as hypermetabolic conditions, should also be considered (American Diabetes Association, 1990b).

Energy requirements for exercise in NIDDM individuals are not significantly different from those of non-diabetic people. Supplemental food before and during exercise is not needed by NIDDM patients to prevent hypoglycaemia and is not recommended except under conditions of severe, prolonged exercise such as endurance sports. In patients taking sulphonylureas there is a slight increased risk of hypoglycaemia during exercise and supplemental food intake may be required in some cases. This may be determined by glucose self-monitoring. NIDDM individuals taking insulin should usually decrease their insulin dosage before exercise and may take supplemental food if needed to prevent hypoglycaemia during or after exercise (American Diabetes Association, 1990b).

2.4.5 Education

Education of the diabetic patient could lead to a longer life (treating and preventing complications which may lead to death), improve quality of life and decrease the cost of diabetic care. Objectives should be to increase the patient's knowledge in order to change the attitude of the patient, and ultimately, to change behaviour and compliance (Krall, 1985b). Continued instruction regarding the condition, diet and drug therapy, exercise and behaviour modification should be given to the patient. Regular follow-up visits at a clinic for a medical checkup and education are, therefore, necessary. A team approach to education is always the best and in no other disease as important as in diabetes. The patient should take an active part in the education. Emotional aspects should also be considered (Marble, 1985).

Diabetic care in black diabetic clinics is often poor, with mainly medication prescriptions and the principal objective the control of raised blood glucose levels. The patients often also have language problems and a lack of understanding of their disease and its complications. There is, therefore, a desperate need for more comprehensive care and education for black diabetics (Morley et al., 1977).

2.5 THE GLYCAEMIC INDEX OF FOODS

2.5.1 Introduction

Recently, there has been a surge of interest in how food affects blood glucose levels and particularly which food components contribute to the effect of a food on blood glucose (Hughes, et al.. 1989). Modification of the rate of gastro-intestinal absorption was then proposed as a new therapeutic principle in the field of nutrition. Mechanisms by which this may be achieved were proposed and these were shown to produce a range of physiological effects, including reduction in serum lipids and improvement in carbohydrate metabolism (Jenkins, et al., 1987a). One of the mechanisms to reduce the rate of nutrient absorption in man chronically, is to select foods with slow rates of digestion and flat blood glucose responses specifically. This means that foods should systematically be classified according to the glycemic response they produce when indexed to a standard food. This enables the creation of diets composed of foods with either high or low glycaemic indices (Wolever et al., 1985).

2.5.2 The concept "glycaemic index"

When the diabetic food exchange lists for meal planning were compiled, the assumption was made that foods with similar nutrient content would have similar effects on postprandial plasma glucose concentration (Laine, et al. 1987). Crapo, Reaven & Olefsky (1976) were among the first to question the validity of this assumption when they demonstrated that feeding healthy subjects glucose, sucrose and various starches alone, or in combination with other foods, resulted in significantly different increases in blood glucose. The exchange lists are also not very accurate in other respects. For example, in the exchanges for fruit, convenient portion sizes are used, although sizes of fruit could differ considerably and cause significantly higher or lower intakes of carbohydrates.

Protein and fat are now known not to have a significant influence on the rate of increase in blood glucose (American Diabetes Association, 1986). Although carbohydrate appears to be the food component which produces most of the blood glucose increase after a meal, equal amounts of dietary carbohydrate from different sources or from the same food in different forms, can have widely different acute effects on blood glucose levels (Hoover-Plow, Savesky & Dailey, 1987). Jenkins et al. (1984a) presented evidence that the major reason for differences in the glycaemic responses of foods is a difference in rates of food digestion and absorption. Factors that influence the rate of digestion are not adequately listed in food tables; it is therefore not possible to predict the glycaemic effect of a food based on its chemical composition alone (Jenkins, et al., 1988; Parillo et al., 1985). The term GI was then proposed by Jenkins et al. (1984a) to describe these physiological differences. The GI is a measure of the extent to which the carbohydrate in a food can raise the blood glucose concentration or a measure of the relative glycaemic response of a food, with each subject's response indexed against his own response to a standard food, such as glucose or white bread (Wolever, et al., 1989). The GI helps to identify those foods which may be beneficial and produce low rises in blood glucose levels and those that are usually undesirable and produce large rises in blood glucose levels (American Diabetes Association, 1986). It is, therefore, a tool to allow diets to be designed on the physiological basis of the glycaemic impact of foods, rather than on the chemical basis of the carbohydrate content of foods. Gannon and Nuttal (1987) prefer to use the term relative glucose area (RGA) for the same concept, indicating that the time over which relative response is determined is variable.

For the purpose of this study, the GI is defined as the area under the postprandial blood glucose curve for a food expressed as a percentage of the area after the consumption of a reference food (American Diabetes Association, 1986). This means that a food with a low GI will cause a small glycaemic response after

consumption (Krause & Mahan, 1984). The available carbohydrate content of the test meal and the reference meal is the same (American Diabetes Association, 1986).

2.5.3 Determination of the glycaemic index

Test meals to determine the GI of a food are usually given after an overnight fast. Diabetic patients should take their insulin or oral hypoglycaemic agents five to ten minutes before the meal (Jenkins et al., 1983a). Meals are eaten within a specific time-limit, usually ten minutes, to avoid the influence of time as a factor in the glycaemic response. Capillary or venous blood samples are obtained for measurement of blood glucose, starting just before the intake of insulin or oral hypoglycaemic agents in the case of diabetic patients. The next sample is taken just before the ingestion of the first bite of food, and then regularly, with 30-minute intervals, usually for two or three hours (Jenkins et al., 1984b).

The test meal and the reference food should have exactly the same amount of carbohydrate. This is usually 50 g, but may be more or less, according to the foods that are tested and the amount of food that contains 50 g of carbohydrate (Jenkins et al., 1984b). Jenkins et al. (1981) found that the GI measured with 25 g carbohydrate food portions may have artificially high glycaemic indices in comparison with 50 g carbohydrate food portions, while glycaemic curves flatten with portions higher that 50 g.

It is important that when the GI of a food is determined, it should be compared with a reference food to allow the calculation of the index. The reference food can be either glucose or bread (Jenkins et al., 1984a). Bread is more palatable and avoids the possible problem of delayed gastric emptying resulting from the higher tonicity of glucose solutions; it may prove a better physiological reference standard than glucose (Jenkins et al., 1984a).

Since many patients also find the intense sweetness of a glucose solution unpleasant or even nauseating, bread is more acceptable (Jenkins et al., 1984b). However, Bornet et al. (1987) feel that bread is not an acceptable reference food. They found in their research, in contrast with other researchers, that bread does not have the same insulinemic index as other starchy foods. They speculate that the difference could be due to differences of wheat origin and bread processing and that this could invalidate the choice of bread as an internationally accepted standard for GI calculations.

It should be noted that bread contains some resistant starch which escapes alpha-amylase digestion in the small intestine (Englyst & Cummings, 1985). This proportion of starch is fermented, in the same way as DF, to short chain fatty acids in the colon. The amount of resistant starch in a food is influenced by various factors such as availability of starch granules to amylase digestion, physical form of the food, particle size, amylose-to-amylopectin ratio of the particular starch, etc. (Vorster, Venter and Silvis, 1990). Vorster, Venter and Silvis (1990) suggest that a standardised pure starch product should be developed for use as reference food in diabetic subjects.

Meals are usually taken with a beverage such as tea according to preference, but the same amount, type and composition of beverage should be used for every meal.

The main source of error in determining the GI of a food is the way in which areas under the curves are calculated. There are at least three different methods used by different groups to calculate the areas under the curves. These methods are reviewed by Wolever (1990). Different methods give significantly different results, although there are strong correlations between the different methods (LeFloch et al. 1990). There is agreement in the literature that total area alone may give false results, and incremental areas are therefore used to describe glycaemic

response to foods more accurately (Jenkins et al., 1981; LeFloch et al., 1990; Mourot et al., 1988). To exclude the problem of negative areas because of hypoglycaemia in healthy subjects, Vorster, Venter and Silvis (1990) suggest that the incremental area with the lowest glucose value as baseline should be used to calculate the GI. This method results in areas which reflect the actual curves as well as the maximum glucose increment. Blood glucose response areas are therefore calculated geometrically as the area above the fasting value and the GI of the meal is expressed as a percentage of the mean area of the reference meal:

$$GI = \frac{\text{blood glucose area of the food/meal}}{\text{blood glucose area of equivalent carbohydrate in reference food}} \times 100$$

Peak rises or the maximum glucose increment are given as the difference between the highest post-prandial blood glucose value minus the fasting value (Jenkins et al., 1983a).

2.5.4 Factors that influence glycaemic index

Several factors may influence the glucose and insulin response to a food. These factors can be divided into physiological factors, which will be responsible for individual variations in GI obtained with a specific food in different individuals or in the same individual over a period of time, and physical factors which will explain variations in GI of different foods in spite of the same carbohydrate load in the same individual (Vorster, Venter & Silvis, 1990).

2.5.4.1 **Physiological factors**

* Physiological and nutritional state

Caron et al. (1982) found that glycaemic responses to breakfast were improved by exercise performed half an hour after the breakfast was started in IDDM subjects. This could result from

a change in blood flow with the onset of exercise so that the absorption of food was slowed and/or the use of the absorbed glucose for metabolic fuel in the muscles.

In addition to effects of exercise and physical fitness on glucose tolerance, variations in background diet may play an important role in inter-individual variations in GI. The carbohydrate, fat and protein content of diets up to three days before a glucose tolerance test may also have an influence on the test. Actually, any factor that influences the circulating levels of free fatty acids prior to the test, may have an influence on the glucose response to a meal. Even factors which will influence insulin receptor number, affinity or function such as DF, will affect the glucose response (Vorster, Venter & Silvis, 1990).

The GI obtained in healthy subjects may differ from that obtained in DM patients. Variability in glucose tolerance tends to increase in persons who approach abnormal glucose tolerance. The coefficients of variation found by different researchers are given in Table 2.14:

Table 2.14 Coefficients of variation for the glycaemic index as found by various researchers

Researcher	IDDM	NIDDM	Healthy subjects
Jenkins et al., 1984c	38 %	25 %	-
Wolever et al., 1985	29 %	15 %	-
Coulston et al., 1987	-	24 %	9 %

According to Wolever et al. (1987b) the difference between the glycaemic responses on 20 foods which they tested was 36 % between IDDM and NIDDM patients. For a given subject, the variation in the GI of a particular food is, however, small.

These variations can, however, be reduced by 50 % if the results are expressed as GI and not coefficient of variability (Wolever et al., 1989).

This means that differences in glycaemic response can produce standard errors of only ten percent in the mean GI. Factors such as type and duration of the disease, severity and medication may also influence the variability of the glycaemic response to foods. These variations often make comparisons among glycaemic indices of different foods not statistically significant (Jenkins, et al., 1984c; Thorburn, Brand & Truswell, 1986a), but if more foods are taken into consideration and not single foods alone, the between-individual variation is less or even zero, according to a model that was used by Wolever et al. (1987b). Despite these differences Wolever et al. (1989) suggest that the GI is an effective way of standardising the glycaemic responses of different individuals.

* Rate of digestion

Recently it has been indicated that the glycaemic response may be predicted from the rate at which a food is digested *in vitro*. In general, slowly digested foods produce flatter glycaemic responses and such foods have been termed "lente carbohydrate foods" (Jenkins et al., 1984a). Therefore, foods that are slowly digested *in vitro* are likely to show low glycaemic and insulin responses as well (Thorburn, Brand & Truswell, 1987). For example, cooked legumes are digested less rapidly than other carbohydrate foods, and have a very low GI. Carbohydrate malabsorption resulting from slow digestion *in vivo* also contributes to the lower blood glucose levels seen after feeding low-GI foods. Thorburn, Brand and Truswell (1987) found that 23 out of 30 traditional bush foods of the Australian aborigines that they studied were digested significantly more slowly than western foods.

Jenkins et al. (1984b) found a significant relationship between

digestive indices and glycaemic indices of foods. It was shown that the initial absorption of a food appears to be related to the blood glucose rise. Therefore, if carbohydrate absorption time could be prolonged, the incremental blood glucose area would be smaller . More evidence for this is the fact that the glucose tolerance of a second meal in normal volunteers is improved after low-GI foods are taken during the preceding meal (Jenkins, et al., 1984a). Assessed by breath hydrogen excretion, however, substantial carbohydrate malabsorption has been reported after consumption of, for example, legumes (Jenkins et al., 1983b).

2.5.4.2 **"Food" factors which will influence glycaemic index**

* Sugar content

It appears that the GI of a sugar can be predicted approximately from the proportion of the sugar molecule that is glucose (Thorburn, Brand & Truswell, 1986a) and the fact that non-glucose sugars are neither rapidly nor completely converted to glucose (Jenkins et al., 1984a). Hughes et al. (1989) suggest that the amount of glucose in food is more important than the availability of the glucose in determining the GI. However, the blood glucose level before a meal is also very important (Erkelens, 1985). The glycaemic indices of whole food items are not as easy to predict, since the blood glucose response appears to depend on many factors that will alter the rate of digestion and/or absorption of carbohydrate (American Diabetes Association, 1986).

The non-direct relationship discussed above is presumably due to the very small rise produced by fructose and sucrose (Jenkins et al., 1981). Small amounts or sucrose consumed with fibre-rich products such as whole-grain breakfast cereals of whole-meal bread may have no more or even less glycaemic effect than their low-fibre sugar-free equivalents (Erkelens et al., 1985; Jenkins, et al., 1984a). A restricted addition of sugar as part of nutritionally balanced meals for non-obese diabetic patients may enhance palatability and so increase overall dietary compliance.

In a study by Vorster et al. (1987b) it was found that the GI of bean dishes did not change significantly with a 10 and 20 % sucrose addition while a 30 % addition increased the GI from 28.8 to 53.7. Slama et al. (1984) came to the same conclusion. Samanta, Burden and Jones (1985) also showed that honey consistently produces a lower glycaemic effect when compared with glucose and even sucrose and they suggest that honey can be usefully substituted for sucrose. However, some researchers, such as Hollenbeck, Coulston and Reaven (1989) feel that it seem reasonable to continue to advise patients with NIDDM at least to limit sucrose consumption until available data would allow the prediction of levels of sucrose consumption where adverse metabolic effects would not be present. In the case of obese NIDDM subjects, restriction is definitely necessary.

* Dietary fibre

There is a significant relationship between the GI and the DF content of a food (Jenkins et al., 1982a), although no simple correlation exists. Different types of DF have different effects. Viscous fibres, such as pectin, which is found in fruit and vegetables, and galactomannan, which is found in some legumes, can lower the blood glucose response, presumably by slowing the digestion of carbohydrates and limiting the diffusion of sugars towards the absorptive mucosal surface (Jenkins et al., 1978; Thorburn, Brand & Truswell, 1986a). The removal of fibre form cereal products has little effect on the glycaemic response (Jenkins, et al., 1981).

The influence of DF in the "trapping" of glucose by the food on digestion also plays a role in the GI. Trapping is most marked in leguminous products (Jenkins et al., 1982b).

* Fat

Fat that is associated with a food is likely to reduce the glycaemic response by delaying gastric emptying (Thorburn, Brand

and Truswell, 1986a). This is an important factor because foods rich in fat can appear in a falsely favourable light if the acute glucose response to a single meal is the only criterion for including the food in the diet.

* Energy content

Jenkins et al. (1983a), as well as Mourot et al. (1988) found no correlation between energy content and GI of foods.

* Type of starch

The nature of starch in itself may be of major importance in determining the glucose and insulin response and may be part of the reason for differences seen in the glycaemic indices of cereals and legumes (Jenkins et al., 1981). The latter has a higher content of amylose (30 - 40 %) as opposed to the higher content of amylopectin in cereal-starch (Jenkins et al., 1984a). There is usually a reduction in glucose and insulin responses after consumption of amylose compared to amylopectin (Behall, Scholfield & Canary, 1988:430). The linear amylose in starch may be less easily hydrated and therefore be less accessible to digestion than the branched amylopectin (Jenkins et al., 1984b). Retrograded amylopectin is classified by Englyst and Cummings (1985) as partially resistant starch and retrograded amylose as resistant starch. The development of a more rigid gel after the starches are cooked and cooled may make the amylose less accessible to hydrolytic enzymes. Gelation of amylopectin occurs at a slower rate and forms softer gels. Less retrogradation appears to occur with starches containing high levels of amylopectin. The branched structure may prevent hydrogen bonding between straight chains that can occur with amylose (Behall, Scholfield & Canary, 1988:430). It is reported that the amylose fraction of maize starch has particular gel-forming ability upon cooling. Reheating of the amylopectin fraction of starch partly reverses retrogradation. Differences in blood glucose and insulin responses observed with cooled and reheated maize

porridge may be ascribed to the presence of partially resistant and resistant starch (Venter et al., 1990).

The unique type of starch in maize meal may have important consequences for the use of the GI in diets for black South African diabetics who use maize meal porridge as staple food. The amylose content of maize starch varies from 22 - 36 % in common varieties to 70 % in varieties genetically manipulated to increase the amylose content. An example of maize meal that was analysed in Potchefstroom (South Africa) had 26 % amylose and 74 % amylopectin (Venter et al., 1990:4). In contrast, wheat starch usually has 17 - 30 % amylose (Behall, Scholfield & Canary, 1988:430). Thorburn, Brand and Truswell (1987) mentioned that traditional bush foods may contain more amylose which make them inherently more resistant to digestion, although carbohydrate malabsorption may also play a role. Differences in particle sizes and surface areas of starch may also play a role because of differences in the availability to the hydrolytic enzymes (Behall, Scholfield & Canary, 1988).

The degree of gelatinisation itself may also play a role in the differences in GI for different starch-containing foods. During gelatinisation the starch granules absorb water and swell. The extent of degree of starch gelatinisation is dependent on moisture availability, time, temperature and pressure. The method of processing, therefore, plays an important role in gelatinisation (Ross et al., 1987).

The reason for the low GI of pastas is unknown but may be due to the very hard wheat (*Triticum durum*) used in making pasta (Jenkins et al., 1983a). Pasta also does not react like other processed starchy foods. Usually starchy foods that are processed more have higher glycaemic indices. Processing reduces the glycaemic impact of pasta. Wheat starch may, therefore, behave differently from other starches - it has been shown that wheat starch swells (gelatinises) in a mode which differs from other starches (Brand et al., 1985). There is also a theory that

additional mechanical barriers such as protein matrix (for example gluten) may reduce GI. The matrix encapsulates gelatinised starch granules and limits access to amylase and reduce the starch availability. The high gluten content of pastas may explain its GI being lower than that of wheat starches (Bornet et al., 1987).

* Protein-starch and lipid-starch interactions

Although protein and fat do not show an effect as marked as that of carbohydrate, the protein and fat content of a food shows a significant negative correlation with GI of the food (Laine et al., 1987). Fat is known to delay gastric emptying and protein stimulates insulin secretion. However, it is not clear whether these actions or a direct effect of fat and protein in reducing the digestibility of food are responsible for the negative correlation (Jenkins et al., 1981).

It was shown that about 20 % of the starch in wheat bread is malabsorbed, but if the protein is removed from the bread, the starch is completely digested in the small intestine. This suggests that protein-starch interactions may retard the digestion of starch (Jenkins et al., 1987c; Thorburn, Brand & Truswell, 1986a). Lipid-starch interactions may have a similar effect. The similarity seen between the GI of whole and skim milk suggests that the action of fat may not be simple (Jenkins et al., 1981).

* Salt

It was shown that a moderate addition of salt increases the post-prandial plasma glucose and insulin responses to foods. The two most likely ways that salt increases the post-prandial plasma glucose and insulin responses are by accelerating the digestion of starch or accelerating the absorption of glucose, or both. Chloride, rather than sodium, may be the responsible factor as chloride ions are potent activators of amylase. Salt may

therefore have an effect on starch digestion by increasing both the amount of salivary amylase and its activity and the activity of pancreatic amylase. Sodium facilitates the absorption of glucose in the small intestine. Salt may increase the glycaemic response to starches as well as to sugars. That implies that variations in the glycaemic indices in various studies may be due to the salt content of some foods or meals (Thorburn, Brand & Truswell, 1986b).

* Anti-nutrients

A reduced rate of starch digestion occurs when substances that have a negative influence on the digestion of nutrients are added to food (Jenkins et al., 1982b). Many of these are found in legumes, which have a low GI. Examples of anti-nutrients are phytic acid, which is found mainly in the outer husks of cereal grains, tannic acid, which is found in tea, lectins that are also found in legumes, saponins and enzyme inhibitors (Jenkins et al., 1984a). These compounds may decrease the amylytic digestion of starch by complexing with proteins that are associated with food starch or by complexing with metal ions that are necessary for digestive enzyme activity (Thorburn, Brand & Truswell, 1986a).

Lectins and phytic acid have the strongest influence on the GI of foods. The effect of phytic acid could be due to its binding with proteins which are closely associated with starch, its association with the digestive enzymes which are themselves proteins, its chelation of calcium required for the activity of amylase, its direct binding with the starch, its influence on gastric emptying or its effect on starch gelatinisation during cooking or processing. The effect of phytic acid can be modified by the addition of a mineral such as calcium, which binds to the phytic acid (Thompson, 1988). High temperatures and pressures used to cook, for example canned legumes, could also alter the anti-nutrient content such as lectins and phytate, and the phytate may also leach out of foods in small concentrations (Wolever et al., 1987a). The mechanics of a tannin effect are

probably the same. Lectins affect the luminal phase of digestion. The ability of lectins to bind to intestinal mucosal cells and cause malfunction and interference in the absorption of nutrients is probably primarily responsible for the reduced glycaemic response in the presence of food lectins (Thompson, 1988). Lectins also inhibit amylase activity, possibly by inhibiting the access of starch to the active site of amylase due to binding of the lectin either to the enzyme or to the starch. The lectin concancavalin A has no effect on GI (Wolever, 1990).

* Food form

The physical form of carbohydrate-containing food and particle size of food molecules have a major impact in determining post-prandial glucose and insulin responses (Golay et al., 1986; Heaton et al., 1988). A much greater blood glucose response occurs after the consumption of pureed compared with whole foods (Brand et al., 1985). Ground rice produces a higher blood glucose peak level than does whole rice (O'Dea, Nestel & Antoff, 1980). Ground carrots also produce a more prolonged glucose response than do diced carrots (Thorburn, Brand & Truswell, 1986a). The way in which foods are processed, can therefore, be of great clinical importance. Processing methods where the integrity of the legume cells of the cotyledon filled with starch is maintained, were seen to be much more advantageous than the traditional milling processing of legumes before, for example extrusion. Insulin responses improve in particular. This is important because of the already low GI of legumes, which results not only from the malabsorption of carbohydrate, but mainly because of a decrease in the rate of absorption (Golay et al., 1986).

* Cooking and processing

Differences in the glycaemic response to carbohydrate meals can be brought about by cooking. A much greater blood glucose response occurs after the consumption of cooked compared with raw

starch (Brand et al., 1985). Cooking increases the accessibility of starch to amylase (Collings, Williams & Macdonald, 1981). Cooked carrots produce a larger blood glucose response than do raw carrots (Thorburn, Brand & Truswell, 1986a).

Commercially processed foods, processed with methods such as extrusion cooking, explosion puffing, and instantiation make use of extreme temperatures and pressure or repeated wetting and drying, and tend to have an even higher GI than do their home-cooked equivalents, because the high temperature and pressure cause disruption of starch granules. In contrast, conventional cooking methods such as boiling involve less physical disruption and only moderate heat and are, therefore, less likely to cause starch damage or complete gelatinisation (Brand et al., 1985).

Canned legumes however, are more acceptable and convenient that home-cooked legumes, and although the canning process will lead to a higher GI, the index will still be low enough to give canned legumes the advantage (Wolever et al., 1987a). During the processing of baked beans tomato sauce is added. The tomato sauce has a relatively high sucrose content which causes a higher GI. The baked beans are, however, more palatable and, therefore, more acceptable, while the GI is still lower than the index of many other foods (Wolever et al., 1987a)

* Mixed meals

Coulston et al. (1984) reported that the glycaemic responses to mixed meals containing different types of carbohydrate sources did not differ significantly and concluded that the GI approach would have little clinical utility. Chew et al. (1988) differ from Coulston et al. (1984); they found significant differences in the glycaemic and insulin responses of healthy individuals to different mixed meals. Moreover, the glycaemic indices of the mixed meals could be predicted from the glycaemic indices of the component carbohydrate foods. There are important differences

between the two studies. In the study of Coulston et al. (1984) individuals with NIDDM participated. They were given standardised meals containing 30 g of test carbohydrate. The study of Chew et al. (1988) was done on healthy individuals and the test carbohydrate portions used in the mixed meals were approximately 40 - 50 grams. The fat-content of the meals in the latter study was also higher and the meals were not given on the same time of the day. The latter study is, however, in agreement with many others (Collier et al., 1986; Wolever, et al., 1985; Wolever, et al. 1990), and shows that the GI approach is successful in predicting the glycaemic responses of healthy individuals and especially NIDDM, but also of IDDM subjects to mixed meals. Wolever et al. (1990) mentioned that there is no evidence to maintain that adding the same amount and type of protein or fat to different carbohydrate-containing foods abolishes the difference between their glycaemic responses. However, there may be different effects of different types of protein and of n-3 compared to n-6 fatty acids on insulin secretion and the glycaemic response. They recommended further research in this field.

The results of the retrospective analysis of data in the literature suggest that differences between the glycaemic responses of single foods are maintained in the setting of a mixed meal. If a low-GI food is added to a meal containing several other carbohydrate foods, the overall effect on meal GI may be less than the predictive difference in GI and if so, there may be no effect on the glycaemic response when measured once in a single individual. Nevertheless, as the number of times the test is repeated increases, the predictive difference in GI diminishes according to the square root of the number. Thus a smaller difference in GI may be important if it is maintained in the diet for a long period of time. Wolever et al. (1989) suggested that the GI will predict the ranking of glycaemic responses of two mixed meals with 95 % certainty if the difference in the GI is less than 34 for NIDDM and less than 50 for IDDM subjects. They called the GI difference associated with

a 95 % chance of correct prediction the *predictive difference*.

The GI difference between two meals is only one factor which influences the probability that their glycaemic responses will be correctly ranked by their glycaemic indices. Differences in carbohydrate, fat, and protein content of the meals will affect their glycaemic responses. Thus, accurate predictions of the glycaemic responses of mixed meals containing large amounts of fruit, or other foods with regional variation in composition or processing, are likely to be obtained only if the GI for the specific foods fed has been determined (Wolever et al., 1989). Parillo et al. (1985) also found that the properties of the foods which influence their digestibility and hence their physiological effects *in vivo* are preserved when the foods are consumed within the context of a meal.

* Influence of previous meals or the second meal response

Jenkins et al. (1982b) found that the glucose response to a second meal was lower after a mixed-food-low-GI meal than after a high-GI meal. They concluded that their results indicate that slow absorption of carbohydrate from the gastro-intestinal tract after one meal may facilitate the disposal of glucose absorbed from a subsequent meal. Wolever et al. (1988) found that the glycaemic responses to breakfasts were significantly lower on mornings after low-GI dinners than after high-GI dinners. Dinners with different fibre contents but the same GI had no effect on post-breakfast glycaemia. Therefore, even foods eaten the previous night can have an influence on the GI of foods eaten the following morning.

Shaneen and Fleming (1987) also found a similar reaction, but the differences were not statistically significant and they concluded that food eaten at breakfast does not appear to influence the glucose response to lunch.

* Other factors

There are many other factors which may alter the chemical composition and the glycaemic response of a food, such as the the stage of ripeness and the length of storage. For manufactured foods such as bread, breakfast cereals, or instant potatoes, the glycaemic values appear to be relatively constant from region to region. For rice, differences in glycaemic responses in different studies may be explained by differences in processing, for example, parboiled as compared to polished. For foods such as fruits and root vegetables regional variation in food composition and hence glycaemic response may be expected (Wolever et al., 1989). Bananas have, for example, different quantities of starch in different degrees of ripeness and the starch may even vary in indigestibility during different degrees of ripeness (Hoover-Plow, Savesky & Dailey, 1987).

The rate of gastric emptying also has an influence on the GI. It is unclear whether physical factors such as the particle size of the food molecules and composition of foods are the only determinants of rate of gastric emptying (Mourot et al., 1988).

2.5.5 Advantages of using the glycaemic index

2.5.5.1 **Better glucose control**

Very good blood glucose control has up to now been advocated for diabetics to reduce the incidence of long term complications (Canadian Diabetes Association, 1981). One of the important factors to maintain good blood glucose control is to prevent large fluctuations in blood glucose by selecting carbohydrate foods that minimise the post-prandial blood glucose excursions (Jenkins et al., 1984a). Lists which only take into account the available carbohydrate content of foods and not the effect on blood glucose response, are therefore not good enough; more information is necessary to supplement tables based solely on chemical analysis, such as lists with the GI of foods (Jenkins

et al., 1981)

It is not only cholesterol and triglyceride values which can be reduced by a low-GI diet (Jenkins et al., 1985) - glycosylated serum protein (fructosamine) levels and even HbA_{1c} levels fall progressively over a period when a low-GI diet is followed. That means that mean glucose levels over a longer period are lower on such a diet. The urinary C-peptide excretion is also lower on a low-GI diet (Jenkins et al., 1987a; Jenkins et al., 1988). Walker & Walker (1984) mentioned that rural blacks in South Africa eat a low-GI diet and also have a very low incidence of DM.

2.5.5.2 **Lower blood lipid levels**

In addition to their effect in long-term diabetic studies a further possible use for dried legumes and perhaps other low-GI foods is to reduce the serum cholesterol level of hypercholes= terolaemic individuals. A study of Jenkins et al. (1985) shows that there is a significant fall in serum triglycerides (16 %) and total serum cholesterol (nine percent) during a period in which a low-GI diet is followed, after and before a period on a control diet. Jenkins et al. (1987b) found a fall in serum triglycerides of 15.3 % and total serum cholesterol of 7.7 % in the same type of study. There was also a small reduction in LDL-cholesterol of ten percent during the low-GI period with no change in HDL-choles= terol levels (Jenkins et al., 1985) and a reduction of 8.5 % in LDL-cholesterol and no change in HDL-cholesterol levels (Jenkins et al., 1987b). They also found no significant changes in HDL-cholesterol with a low-GI diet. There was, however, no direct relationship between the individual reductions in dietary GI and falls observed in serum triglyceride levels, although those individuals who were able to lower their dietary GI by more than 13 % and took more than 50 % of their carbohydrate kilojoules as low-GI foods, tended to show the best results in terms of triglyceride lowering. On the other hand, the reduction in GI related significantly to the falls in both

total and LDL-cholesterol. There is therefore a growing body of evidence to suggest that high fibre starchy foods such as legumes could be usefully incorporated into the diabetic diet. Food selected on the basis of low glycaemic indices may also have a use as adjuncts in the treatment of hyperlipidaemia and especially hypertriglyceridaemia (Jenkins et al., 1985; Jenkins et al., 1987b).

2.5.5.3 **Higher dietary fibre content**

A diet with a low-GI usually also has a higher DF content. The advantages of a high fibre diet are well-known (Gresse, 1987). In the study of Jenkins et al. (1985), significant relationships were seen between the higher fibre content of the low-GI diet and the HDL-cholesterol, P/S ratio and total and LDL-cholesterol levels of participants, despite the fact that the difference in mean fibre intake between the control diet and the low-GI diet was small (4.1 g).

The effect of weight loss may be enhanced by using low-GI starchy foods (Jenkins et al., 1988).

In general, results of studies in young adult, normal weight, non-diabetic volunteers agree well with those in middle-aged and elderly, overweight, diabetic patients. That means that values of GI tables can be used widely (Jenkins et al., 1984a).

2.5.6 Possible problems with the glycaemic index

Before determining the GI of a food, all the factors that may have an influence should be identified and controlled. The reference food should also be considered, as well as the method to be followed (carbohydrate content, duration of test, calculation of areas and index, standardisation, etc.).

2.5.6.1 **Experimental subjects**

The experimental subjects and exclusion criteria should be chosen well. The number of experimental subjects and whether each subject should act as his or her own control will depend on the homogeneity of subjects regarding glucose tolerance. Diabetic subjects should be well described and homogeneous regarding type of diabetes, duration and severity of the disease and medication. Age, sex, and race can also play a role and should be controlled.

Carbohydrate intake three days prior to the determination of GI should be 250 - 300 g and the meal preceding the test and fasting period should be standardised in order to obviate variability. Tests should be done in the mornings after an overnight fasting period of ten to twelve hours (Vorster, Venter & Silvis, 1990).

2.5.6.2 **Reference food and determination of glycaemic index**

The carbohydrate load of the test meal and reference food, as well as the type of reference food, was discussed in section 2.5.3. The usefulness of the GI approach also depends on the size of the test meals. A mixed meal with a carbohydrate content higher than 50 g may provide a greater stimulus to insulin secretion and other homeostatic mechanisms than the usual test portions (Laine et al., 1987). Jenkins et al. (1981) found that the glucose-response curve flattens with carbohydrate portions much larger than 50 g, while a smaller carbohydrate portion, like 25 g, may give an unrealistically high GI. A portion that contains 50 g of carbohydrate may sometimes be an impractical amount of food, for example 500 g of peas. Such a large quantity of food is likely to affect gastric emptying.

Some researchers have used glucose as standard food and others have used bread, as discussed previously. In order to compare the results of studies with different standard foods, the GI obtained with glucose should be multiplied by 1.38 to get the same GI obtained with bread, since the glycaemic response of

glucose is, on average, 38 % greater than that of bread (Wolever, 1990:144).

Different groups use different methods of blood sampling. The GI was originally based upon measurement of glucose in whole capillary finger-prick blood. Many researchers use venous plasma or serum, which gives higher values (Wolever, 1990).

There are also differences in the way research groups may interpret the area under the curve to calculate the GI. Some use the area under the lowest blood glucose value and others the area above the fasting value. In some cases the peak blood glucose value may give a better indication of the true glycaemic effect and some researchers such as Samanta, Burden and Jones (1985) feel that the addition of a peak incremental index to the GI would be valuable. Such issues need to be resolved because the resulting GI values can vary considerably depending on the food that is ingested (Thorburn, Brand & Truswell, 1986a).

2.5.6.3 **Restrictions of the glycaemic index**

The GI gives no information about the energy density or the satiety value of a food. This has resulted in the misinterpretation of GI results by the popular press (Thorburn, Brand & Truswell, 1986a). Foods which are rich in fat, such as potato crisps and ice-cream, give low glycaemic indices but are undesirable in large amounts for a number of reasons. They also do not necessarily produce equivalent low insulin responses (Collier & O'Dea, 1983). The high fat content contributes to increased energy intakes and raises plasma LDL-cholesterol levels.

The concept of GI neglects the insulin secretion factor, which might be of major clinical significance in the non-diabetic as well as in the NIDDM population, since peripheral hyperinsulinism could be a risk factor for atherosclerosis (Bornet et al., 1987). However, Bornet et al. (1987) found that, with the exception of

bread, the mean insulinemic index significantly correlates with the GI of all the foods that were tested in a mixed meal.

2.5.6.4 **Food mixtures and long term studies**

The usefulness of the GI approach will depend on whether the GI of a mixture of foods in a meal can be predicted and on whether a low-GI is beneficial in the long term (Wolever et al., 1985). Bornet et al. (1987) found, for example, that the differences in GI between different foods in a mixed meal compared to those foods alone were significant, but the differences were not significant if the whole meal were taken into consideration, even with relatively high percentages of fat (37 %) and protein (20 %) in the meal. Calle-Pascual et al. (1986) found that the glycaemic response of foods with a high carbohydrate content when eaten as part of a standard meal is different from that expected on the basis of their GI alone. This effect seems to be due to an unspecific interaction with other foods present in the meal since the glycaemic response obtained in the study was independent of fat, protein or energy content of the diet. The glycaemic response of the whole meal should therefore be taken into account and not the responses of single items.

2.5.6.5 **Variability in responses**

The utility of the GI concept has been questioned because of variability in glycaemic responses. Differences exist between different individuals with respect to the absolute level of blood glucose achieved after meals. The variation among individuals is large, in spite of the fact that the calculation incorporates controls for differences in glucose tolerance status and body weight with the use of glucose as the reference food in each person. Other factors such as the degree of insulinization of the patients and different somatotypes of IDDM and NIDDM patients may contribute to the differences between subjects. In addition, the presence of autonomic neuropathy relating to the gastro-intestinal tract may influence blood glucose responses

(Wolever et al., 1989). The ingestion of glucose during different phases of the normal fasting activity cycle of the upper gut may also play a role (Thompson et al., 1982).

There are also differences in reported glycaemic indices of the same food items tested by different researchers. This may be caused by various factors such as differences in methods of assessing the glycaemic response area, differences in previous meals and differences in the glycaemic response of foods with the same name but different recipes, processing methods, etc. (Wolever et al., 1988). The coefficient of variation of mean glycaemic indices reported by different authors is discussed under section 2.5.4.1.

2.5.6.6 Viability

The average GI values of small groups of young, non-diabetic volunteers of normal weight, agree well with those of middle-aged persons with NIDDM (Jenkins, et al., 1983a). In general the agreement between mean values for the GI of various foods reported by different laboratories is good. Some differences may arise because laboratories differ in their estimates of available carbohydrate, in cooking methods, in the use of different cultivars of the food, and in their use of extras such as tea, coffee, added salt and even tinned tomatoes (Jenkins et al., 1981). It has been maintained that a consideration of average GI values is not valid since it conceals large differences between individuals. However, the GI is still an effective way of standardising the glycaemic responses of different individuals. This only means that the larger the difference in GI, the greater the probability that the meal with the larger GI will have the larger glycaemic response. If a prediction as to which meal will have the larger glycaemic response on any one occasion is to be correct at least 95 % of the time, then a difference in GI of approximately 34 for NIDDM patients and 50 for IDDM patients is required. These differences are large in the context of the differences between the GI values of common

foods. For NIDDM patients the difference is similar to that between many common foods and therefore not large (Wolever et al., 1989).

2.5.6.7 The glycaemic index in diabetic patients

Most of the work on GI was carried out in normal volunteers or patients with "chemical diabetes" and has involved the testing of a limited range of foods. Therefore more research is needed on the GI in diabetic patients, the GI of a wider variety of foods and also of mixed meals (Jenkins, et al., 1984c).

Certain differences exist in the conventional dietary advice given to IDDM and NIDDM subjects. Emphasis has been placed on evenness of carbohydrate distribution throughout the day for the former and caloric restriction for the overweight majority of the latter (Nuttall, 1979). The question therefore arises as to whether different dietary advice should be given to these two groups of diabetics and whether the GI is equally beneficial to use in both groups (Jenkins, et al., 1984c). Jenkins et al. (1984c) found no significant difference in GI between repeated white bread test meals in both NIDDM and IDDM patients, but no significant reduction in GI was seen in IDDM patients and a significant reduction in NIDDM patients with white pea beans. These results might suggest quantitative differences, even if major qualitative differences do not exist between the responses of NIDDM and IDDM patients to specific carbohydrate foods. It also appears likely that this difference is a reflection of the greater degree of variability in the results of the IDDM compared with the NIDDM patients. Part of the reason for this may be the more prolonged glucose tolerance test in IDDM patients and the raised blood glucose levels still seen at three hours. Similarly the GI for beans in the IDDM may have been underestimated. Nevertheless, if corrections were made for the lower fasting glucose levels of IDDM patients, then great similarity might have been seen between IDDM and NIDDM in terms of their responses to the beans. It is also possible that another factor predisposing

to alterations in carbohydrate tolerance was the degree of insulinization of the patients, but no evidence for that has been found. Insulin doses were held constant over the course of the study. It is also possible that the different somatotype of the IDDM and NIDDM patients may have been a factor in the different responses, but no such relationships were found.

2.5.6.8 **Complications**

Jenkins et al. (1988) unexpectedly found increased renal perfusion on the low-GI diet for non-diabetic volunteers. Further study is necessary to determine whether this negative aspect really results from a low-GI diet.

Increase in flatulence was noted by some subjects on low-GI diets (Jenkins et al., 1987a)

Some researchers do not believe in using the GI in the design of diabetic diets. Laine et al. (1987) concluded that the exchange lists for meal planning of diabetic diets more accurately predict the post-prandial response to a mixed meal than does the GI of foods. They believe that additional studies are necessary to determine whether clinically important differences in the physiological responses to carbohydrate-containing foods really exist. According to them, it is not optimal to analyse post-prandial increments in plasma glucose, because it is the absolute plasma glucose level rather than the increment in plasma glucose that is biologically important. They found that significant differences in post-prandial responses exist only in healthy subjects. Coulston et al. (1987) found that meals which vary substantially in predicted glycaemic potencies produce similar plasma glucose and insulin responses when total carbohydrate, fat, protein and energy are held constant. They suggest that one cannot use a mean GI for a number of people, especially diabetics, because of the difference between individuals in coefficient of variation, and that it is premature to use current glycaemic indices as a clinical tool to reduce

day-long glycaemia. However, several authors (Canadian Diabetes Association, 1981; Jenkins et al., 1985; Jenkins et al., 1987b) have reported long-term benefits of low-GI diets.

2.5.7 Glycaemic index values of different food sources

Most root vegetables, breakfast cereals, grain and cereal products produce large rises in blood sugar and have glycaemic indices of over 50. Whole-meal pasta and rye bread are the exceptions, and have glycaemic indices close to 40. Fruit, some vegetables, dairy products and legumes in particular, have low glycaemic indices (Thorburn, Brand & Truswell, 1986a).

Jenkins et al. (1981) found the following mean values for food groups: legumes 31 %, dairy products 35 %, fruit 50 %, cereals 60 % , breakfast cereals 65 %, vegetables 65 %, root vegetables 72 %, sugars 71 % and biscuits 60 %. However, great variations exist between different foods, also within most of the food groups. The exception is dairy products.

Many of the glycaemic indices for healthy volunteers and diabetic volunteers are significantly related (Jenkins et al., 1983a).

2.5.8 How to use the glycaemic index to formulate diets

The GI provides new information about how different carbohydrates influence blood glucose level and of the factors in food which can alter this response. Recommendations made are that diabetic patients should eat more legumes, which have a low GI and are also low in simple sugars and fat (Thorburn, Brand & Truswell, 1986a) and also more pasta. The relative contribution of especially soy protein to human nutrition is bound to increase because of the protein's overall positive nutritional profile, low cost, high availability, excellent functional properties in food systems, and continued innovative food-product development

(Erdman & Fordyce, 1989). There is also good evidence that leguminous diets improve glucose and insulin profiles, which suggests that slow-release carbohydrates may be involved in the amelioration of the disease (Mann, 1984). On the other hand it is also important to take the acceptability of the food into consideration, because not all diabetic patients are sufficiently motivated to comply with strict dietary regimens (Vorster et al., 1987a). Therefore, taste, health belief about the food and to a lesser extent cost and ease of preparation should also be considered. The formulation of diets with a low GI is a challenge, because foods with higher glycaemic indices are usually more palatable, take less time to prepare and are eaten more often (Jenkins et al 1984b).

Many feel that more research is needed before diets for diabetic persons are designed around the GI approach, but that it could have broad clinical application (Jenkins et al., 1987a; Thorburn, Brand & Truswell, 1986a; Wolever, 1990), although some feel that it will never be practical to use (Laine et al, 1987). More extensive classification of foods will be required to cater for individual tastes and differences in life-style (Jenkins et al., 1985). However, the information available can already be used with discretion.

When a low-GI diabetic diet is designed, the general rules for compiling a diabetic diet should still be considered, as discussed in section 2.4.1.

2.5.9 Conclusion

According to the American Diabetes Association (1984) and Weyman-Daum et al. (1987) there is a great need for increased investigative efforts aimed at defining and quantifying the nutritional factors involved in blood glucose regulation. Questions concerning the following issues remain to be answered:

- Difference in diets for IDDM and NIDDM and various degrees of glycaemic control

- Diets for individuals with both diabetes and hyperlipidaemia
- Variability in metabolic response in different individuals to carbohydrate-containing foods
- Effect of particular food combinations or meal compositions on glycaemic response
- The practical use of the GI
- The use of low-GI diets for children

It is necessary to study more foods, the accompanying insulin responses, mixed meals, and the long-term effects on carbohydrate and lipid metabolism. It is especially studies of the GI of foods for diabetic patients that are needed. Every food of interest will need to be tested in patients because all foods have unique structural properties that can influence their digestion. Foods will also have to be tested in a variety of combinations because different mixtures may alter digestion rates (Hughes et al., 1989). It is reasoned, since NIDDM patients form the greater part of the diabetic population (a large proportion of whom are treated with diet alone) that their subjective reactions to different foods are of special importance (Jenkins et al., 1984b). Studies of IDDM patients are also important, because they are the patients that are most in need of precise information. They have little endogenous secretion of insulin, which can have a different influence on the GI of foods (Hughes et al., 1989).

However, despite the fact that more research is needed, there is at present some evidence that an increase in the proportion or absolute amount of starchy, low-fat, low-GI foods may be of benefit in relation to carbohydrate and lipid metabolism (Jenkins et al., 1984a). The GI approach may prove to be a good predictor of glycaemic responses to mixed meals and be useful in planning optimal diets, not only for diabetic individuals, but also for those with carbohydrate-induced hyperlipidaemia and post-gastrectomy syndromes. It may even be useful in devising diets for hyperlipidaemia, weight reduction, exercise, athletic events and the prevention of dental caries (Jenkins et al., 1985;

Thorburn, Brand & Truswell, 1986a). On the other hand, patients with the reduced absorptive capacity of diabetics on the brink of insulin-induced hypoglycaemia may benefit from foods with a higher GI (Jenkins et al., 1981). The GI also seems a satisfactory tool to evaluate effects of additions of other dietary substances to a particular food (Vorster et al., 1987b).

The approach is likely to apply well with respect to planning dietary therapy for 80 to 90 % of the NIDDM population. With IDDM patients, although the approach is still warranted, the predictability of the results is minimized by the greater variability of IDDM responses. These, however, may also reflect other variables of importance in their diabetic management (Jenkins et al., 1984c). Some researchers believe that the approach is still more beneficial for poorly controlled individuals (Weyman-Daum et al., 1987).

Once identified, incorporation of foods with a low GI into the diabetic diet may be greatly aided by belief in positive health benefits with the logical implication of education as a means for change. In addition, the provisions of rapidly prepared food forms by industry and advice on easy to prepare recipes may enhance the use of these foods in diets of middle-aged and elderly diabetics (Jenkins et al., 1984b).

The use of the GI can also be simplified by classifying foods into ten percent GI ranges, as was done by Gericke and Muller (1987). This classification could be further developed to classify foods into high, medium, low or in-between-GI foods.

An open mind is therefore necessary in the dietary management of the diabetic patient.

2.6 THE DIETS AND NUTRIENT INTAKES OF THE SOUTH AFRICAN BLACK POPULATION

2.6.1 Introduction

Because of westernisation the South African black population does not eat according to traditional rural African dietary patterns any more (Du Plessis, 1963). These changing eating habits of the black population may play a role in the incidence of NUDDM and other nutrition-related diseases (Silvis 1989). It is, therefore, necessary to investigate these changes in order to design practical educational programmes to improve eating habits and to control and/or prevent the development of western diseases. Literature on the current diet of the black South African population is scarce. The present diet of blacks, as eaten in the rural and urban areas, will therefore be discussed with reference to the available literature.

2.6.2 Diet in the rural areas

Different ethnic groups of the black population have different eating habits. However, the basic rural diet is fundamentally the same for all the ethnic groups (Gelfand, 1973) and is reasonably well-balanced (Vorster *et al.*, 1990).

2.6.2.1 **Meal pattern and preparation of meals**

Usually there are two main meals with only one course per meal (Gelfand, 1973). The first meal is usually taken late in the morning and the second meal after dark in the evening. The meals consist of a cereal, often maize meal porridge, of which large quantities are eaten, with a relish (Du Plessis, 1963; Lubbe, 1971). No liquids are taken during or after the meal, although a little water is sometimes drunk afterwards. The same meals are eaten during the week and over week-ends (Mönnig, 1978). In families with children who go to school, the children eat an extra early morning meal of cold porridge (Crous & Borchardt, 1986).

Food is served in specific portion sizes and usually more food than required is prepared to make provision for unexpected visitors; that implies that there are usually left-overs which are kept as early breakfast for younger children (Kuzwayo, 1990). During times when food is scarce, just enough food is cooked, according to the portion sizes, availability and number of people who have to eat. A portion is a specific amount of cereal with relish, as given to the head of the household (Du Plessis, 1963). According to Bembridge (1987) food is often scarce among the Xhosas in the Transkei and therefore malnutrition is often seen. It is probably also the case in other rural areas.

The most important cooking methods are boiling, braising, roasting and to some extent, steaming and frying (Gelfand, 1973).

2.6.2.2 Cereals

Thick porridges, made from the different types of grain produced (mainly maize meal and sorghum), and which are cooked in various ways, form the staple food of the rural black population (Crous & Borchard, 1986). The Xhosas in the Transkei often use samp in the place of porridge (Kirsten, 1975). Rice and maize rice are used very seldom and then usually on Sundays. Types and descriptions of cereal foods that are used, are summarised in Table 2.15.

A portion of approximately 550 g of cooked cereal food is usually the main dish of a meal for the adult (Kirsten, 1975:18). The fat which is sometimes used in the preparation of the cereal dishes, is usually animal fat drippings, gathered when meat was fried.

Samp prepared at home is usually without the hull but with the germ. The nutritional value of the fat, thiamin and niacin is therefore higher than the commercially prepared product, although the DF content is very low. Maize meal is nowadays rather bought from the local shop than milled at home by the Xhosas (Kirsten,

1975). In some Venda areas *luvhele* (manna) is also cultured and used to make porridge (Crous & Borchardt, 1986:45).

Table 2.15 Types and descriptions of cereal foods commonly used in the rural areas

Name of dish	Tribe	Description
Isitshwala/	Xhosa	
Umqa	Xhosa	
Magohe	Pedi	Unfermented stiff porridge
Isigwampa	Xhosa	Unfermented stiff porridge with greens
		Unfermented stiff porridge mixed with fruit
Umkhupha	Xhosa	Steamed maize bread
Imbila	Xhosa	Fermented thin porridge
Indengani/	Xhosa	
Isidudu	Xhosa	Unfermented thin porridge
Uphutu/	Xhosa	
Umphokoqo	Xhosa	Dry crumbed porridge
		Dumplings
Umcuku	Xhosa	Fermented, moist crumbed porridge
Isitshwalapishi/	Xhosa	Samp and legumes with or without
Umngqusho	Xhosa	fat, sometimes curried
Tshidzimba	Venda	Samp and peanuts
		Samp with onions
		Samp with curry and fat
		Samp as such
Lewa	Pedi	Samp stew, sometimes with meat
Iinkobe	Xhosa	Cooked whole maize on the cob
Umphothulo	Xhosa	Milled cooked whole maize
Isophu	Xhosa	Soup of whole mealie kernels and dried beans

(Beyers, Hammer & Groenewald, 1979:96; Kirsten, 1975:17,18; Quin, 1964:970).

Home-baked and commercially prepared bread are luxuries and used as a relish, not as a main dish. However, when available, bread is eaten daily (Vorster et al., 1990). It is usually taken without fat or margarine, with soup, sour milk, coffee or tea. *Vetkoek*, a ball of a stiff flour mixture, fried in fat, is also used as a relish (Beyers, Hammer & Groenewald, 1979).

2.6.2.3 **Relishes**

The relish can be made from cultivated or indigenous plants, insects or domestic animals (Quin, 1964). Variety in relishes is only seasonal, when certain foodstuffs become available. Throughout much of the year the relishes are very stereotyped (Mönnig, 1978). Because of this, nutritional deficiencies are often experienced during winter and the early rainy season (Bembridge, 1987).

The plant relishes include cooked vegetables (for example pumpkin, cabbage, spinach), fruit, peanuts in some or other form, or legumes, and have a definite seasonal character (Crous & Borchard, 1986). Edible green leaves (*ilaxa, imifino or marogo*) play by far the most important role in the vegetable group. Whereas wild green leaves (approximately 30 different kinds are eaten seasonally) are regarded as common food suitable for women and children, the cultivated greens enjoy more status and are eaten during weekends. Wild green leaves are often consumed in the form of *isigwamba,* a thick green porridge of cooked leaves and maize meal (Kirsten, 1977:21). Other maize meal porridges mixed with vegetables, for example pumpkin (*umqa wethanga*), are also common (Beyers, Hammer & Groenewald, 1979:99). The green leaves are dried for the months when they are not available. Cabbage is the most popular cultivated vegetable, followed by potatoes and then spinach, carrots, onions, peas and tomatoes. Wild-growing roots are also consumed by a few people (Kirsten, 1977). Vegetables are usually consumed in the cooked form. Tomato-and-onion gravy on the cereal dishes is very popular (Beyers, Hammer & Groenewald, 1979).

On the whole, fruit can be regarded as a scarce item in the diet, except in the diet of the Pedi-tribe where wild fruit such as wild figs, prickly pear and the marula are still very important items (Mönnig, 1978; Quin, 1964). The types of fruit that are eaten differ from region to region, but apples, oranges, peaches, paw-paws and bananas are the most common kinds of fruit. Wild-growing fruit are mainly eaten by boys herding flocks and over weekends (Kirsten, 1977).

Apart from the small portion of dried beans which usually forms part of the samp dish, *isitshwalapishi*, dried beans are not optimally utilised. The two main bean dishes, namely *iinkobe* soup (thick soup of dried beans and the cooked whole grains of maize or sorghum) and *umqa* (thick porridge of cooked dried beans and maize meal), are excellent examples of low protein cereal improved by a protein-rich plant food (Kirsten, 1977:21).

Meat is sometimes used as a relish, if affordable, but it is a scarce commodity and not used on a regular basis, only once or twice a week, often on Sundays (Kirsten, 1977). The rural population still measures their wealth according to the number of cattle or other domestic animals they have, and do not slaughter the animals unless there is something to celebrate (Du Plessis, 1963). The Xhosas of Transkei use chicken most frequently, followed by pork, beef, mutton and goat. Canned meat is also used nowadays. Fish in the usual dietary pattern is mainly in the canned form and is rarely caught in streams (Kirsten, 1977). The Pedi tribe regard fish in the same class as snakes and do not eat fish, except in the canned form (Mönnig, 1978).

The black population also use other protein sources such as locusts, termites, crickets and birds. The Pedi classify their edible insects into *ditsie* (locusts and grasshoppers), *ditshoswane* (ants and termites), *diboko* (caterpillars), and *nkhwane* (beetles). They roast or stew the insects and then dry them (Mönnig, 1978:180). Mopani worms *(mashonza)* are popular

with all the black ethnic groups and considered a delicacy. In Venda it is not eaten more than once a week (Vorster et al., 1990). Eggs are not used as an ingredient in dishes and only a small amount is eaten as such and then always in the fried form (Kirsten, 1977). Milk is scarce and apart from the milk in tea and on porridge, it is used as relish by only a few (often as sour milk (*amasi*)). Whereas cow's, goat's and in some cases sheep's milk is used in tea, cow's milk is preferred for sour milk. If milk is not available from the people's own livestock, full cream milk powder, condensed milk and in a few cases, skim milk powder, are bought (Kirsten, 1977). Coffee creamers are also gaining popularity.

The black population do not habitually can fruit or cook jam. Canned fruit, sweets and biscuits are, however, bought occasionally (Kirsten, 1977).

A great variety of snacks are consumed during the day, for example, peanuts, roasted pumpkin seeds, wild fruit, honey and *macheu, metogo* or *amarewu,* non-alcoholic fermented cereal drinks which are used in large quantities (Du Plessis, 1963; Mönnig, 1978:189).

Tea is a favourite beverage and almost exclusively taken with milk and sugar (Kirsten, 1977). Sweetened condensed milk is preferred (Lubbe, 1971). Tea is at present used at least once a day (Vorster et al. 1990). Traditionally the black population do not differentiate between hot beverages - all are named tea (Kuzwayo, 1990), therefore, other hot beverages may be consumed in larger quantities, but are reported by researchers as tea. However, most people only take tea once a day at one of the two meals.

A fermented cereal beer (*utywala or bjalwa or mabundu or halwa*), brewed with any available ingredients is also popular and used in large amounts by adults on a daily basis and often more than once a day, especially during celebrations (Crous & Borchardt,

1986:46; Gelfand, 1973; Vorster et al., 1990). Sorghum or maize meal is usually used for the brewing, as well as wild fruit such as the morula (Quin, 1964). According to Kirsten (1977) the consumption of alcoholic drinks by the Xhosas in Transkei is negligible and apparently restricted to the festive season at the end of the year.

Salt is no longer gathered as it is much easier to buy. Sugar has become very popular and is used fairly often (Mönnig, 1978). *Lunonya* (a seed of a flower with a sharp taste) is often used as flavouring ingredient (Crous & Borchardt, 1984:43).

2.6.3 Urban diet

Food is much more freely available and money is not as scarce as in the rural areas. Blacks in the urban areas can, therefore, buy food and this fact has a profound influence on eating patterns and food choices.

2.6.3.1 **Meal pattern and preparation of meals**

Three meals, instead of the traditional two, are eaten by most urban blacks. However, some of the people who stay at home during the day still eat only two meals (Crous & Borchardt, 1984).

More than half of the people who work outside the home, take food to work with them, usually bread, porridge and meat and sometimes fruit, eggs, polony, cheese and a fermented non-alcoholic beverage (Crous & Borchardt, 1982).

Lunch usually consists of stiff maize meal porridge with a home-made tomato-and-onion sauce; sometimes cooked beef with bones is added. In that case the tomato-and-onion sauce is replaced by a gravy made from the water in which the meat was cooked. For supper maize meal porridge again forms the bulk of the meal. With it cooked green leafy vegetables and meat, with gravy or

tomato-and-onion sauce is popular (Oudkerk, 1965). Meat is eaten at least once a day by most but vegetable intake is low (Lubbe, 1971).

The traditional hospitality of the blacks is still seen in the urban Sunday lunch, where a large variety of food, everything edible in the house, is available for everyone, including the unexpected visitors. Therefore, the main meal on Sundays differs from the meals of week days; rice (or sometimes samp) is eaten in the place of porridge. A wide variety of cooked vegetables and salads is served and pudding, with jelly and custard by far the more popular, is often eaten on Sundays (Crous & Borchardt, 1982). Breakfast is often skipped on Sundays. Fish and chips are sometimes eaten for Saturday lunch and/or supper (Oudkerk, 1965).

The most prevalent preparation method is still boiling.

2.6.3.2 **Cereals**

Large quantities of stiff porridge is still eaten for the two main meals of the day. Oudkerk (1965:1149) found, for example that urban school children ate an average of 900 g stiff maize meal porridge per day, with no age and sex differences in the quantities eaten. Bread (some eat brown but many prefer white) with tea is an important cereal food for breakfast. However, soft porridge with sugar is eaten for breakfast during weekends and by some during the week (Crous & Borchardt, 1982; Lubbe, 1971).

School children usually eat bread for lunch (Crous & Borchardt, 1982). A variety of spreads, such as achar, jam, peanut butter, margarine, meat, fish or tomatoes are used (Crous & Borchardt, 1984). During the school break a "pickled burger" (a thick slab of bread of at least 150 g and a small amount of mango pickles or polony and atchar) and cold drinks are usually consumed.

The types of porridge that are prepared are not of such a variety as in the rural areas. Mealie rice and samp are used more often than in the rural areas, but rice is preferred (Crous & Borchardt, 1982). Bread is usually eaten dry, without fat, butter or margarine, but if affordable, jam is used (Oudkerk, 1965).

2.6.3.3 **Relishes**

In contrast to the rural areas, more than one relish is often added to the porridge for lunch and supper.

Cooked green leaves such as *marogo,* spinach and cabbage, are still the most popular vegetable dish (Crous & Borchardt, 1982). *Marogo* is used in dried form if it is not available in the fresh form and if the wild varieties are not available, leaves such as those of pumpkin and beetroot are used. Peanuts, in ground form, are often cooked with the vegetables (Oudkerk, 1965). The urban blacks also use tomatoes, onions, beetroot, pumpkin, carrots, green beans, sweet potatoes, mealies, squashes, fat, oil, peanut butter, legumes, and red peppers. Potatoes are cooked with the meat practically every day and often in the form of *sesebo* (stew of potatoes, onions and tomatoes) (Crous & Borchardt, 1984:42). Fat in the form of cooking oil or margarine is usually added to cooked vegetables, although the fat intake of the urban blacks is low (Oudkerk, 1965).

The use of fruit is determined by what is seasonably available but fruit is not usually taken regularly (Lubbe, 1971). Apples and oranges are the most popular choices. Fruit is eaten by the family as snacks and not part of meals. Wild fruit is no longer used generally (Oudkerk, 1965).

Meat, in small portions, is more available and is used frequently, mostly daily. Types of meat are used in the following order of frequency: beef (often shank), chicken, beef tripe, liver, sausages (Boerewors specifically), polony, mutton.

Pork is used by only some ethnic groups. Canned meat is also used frequently. Some blacks do not eat fish at all, but most do, and especially in the canned and fried in batter forms (Crous & Borchardt, 1982; Oudkerk, 1965).

Milk and eggs are used more frequently and are more readily available than in urban areas, although portion sizes of the relishes can be small. Protein sources such as locusts, termites, crickets and birds are not that popular in the urban diet, possibly due to unavailability; however, a few still eat mopani-worms that can be bought in the dried form (Crous & Borchardt, 1982).

Peanuts are a very popular snack and consumed at least once a week. Biscuits and sweets are consumed more often. The Vendas also use a snack called *mugumu* (roasted maize kernels and peanuts, milled together) (Crous & Borchardt, 1984).

Non-alcoholic beers are still made or bought and consumed frequently. Tea is very popular. Sweetened condensed milk and sugar are often used with the tea. Sometimes a mixture called starch water, is made with hot water, sugar and condensed milk. The families who can afford it use cacoa and other chocolate drinks fairly regularly (Oudkerk, 1965). Cold drinks are consumed more and more often (Crous and Borchardt, 1982). Commercially prepared alcoholic beer, the common type as well as the bantu beer and a home-made variety (*halwa*), are popular thirst quenchers for men (Crous & Borchardt, 1984).

Spices such as salt, curry powder, pepper, ginger, cinnamon, mustard powder, barbecue spices and chili powder are used freely (Crous & Borchardt, 1982).

2.6.4 Summary

The diet of the black South African has been westernised to some extent, even in the more remote rural areas. The diet of the

urban black South African is basically the same as that of the rural black, especially the composition of the meals (cereal staple and relish). However, the urban black uses more westernised foods, especially as snacks, and has adapted to a more westernised meal pattern (Oudkerk, 1965), resulting in an increase in the variety and intake of fat and animal protein and decrease in the intake of DF.

2.6.5 Nutrient intake

Lubbe (1971) mentioned that the difference between the traditional and the urban diets of blacks can be seen in the contrasting result obtained in respect of nutrient intakes. In his study of Vendas, rural and urban, he found that urban subjects had remarkably higher intakes of animal protein, animal fat, retinol and sugar, while their intakes of calcium, carotene and ascorbic acid were far below the intake levels of their rural counterparts.

A study done among rural Vendas (Vorster et al., 1990), has confirmed the association between a low-fat diet and low levels of coronary heart disease risk factors. The present rural diet is a low-fat diet with a high P/S ratio. The protein intake is often from plant foods. If at all, sunflower oil and margarine are used as fat in food preparation and these practices cause the high P/S ratio in the diet. Total mean protein intakes were adequate with the intake of plant protein almost double that of the animal protein. Approximately 16 % of the people were vegetarians. The bulk of energy intake comes from carbohydrates, especially the polysaccharides, with sugar and sugar-containing food items only as luxuries. Mean intakes of DF were not high at the time of the study.

Intakes of calcium and iron were high, vitamin A intake was adequate and intakes of vitamin D, B-6, folic and ascorbic acid were low with the intake of vitamin B-12 in women also low (Vorster et al., 1990). Most of the findings mentioned above

were also found by Lubbe (1971).

Circulating levels of retinol binding protein and several vitamins were relatively normal and vitamin B-12 serum levels were high. The haemoglobin level in most subjects was low, most probably because of the low vitamin B-6 and ascorbic intakes. Iron availability in the Venda diet could also be responsible (Vorster et al., 1990). The mean nutrient composition of the Venda diet as found by the above-mentioned researchers and some of the mean levels of blood constituents are given in Table 2.16.

Silvis (1989) studied a diabetic population from semi-urban areas in and around the Orange Free State (OFS). The energy intakes of the women were higher than those of the men. Protein intakes were relatively high (1.3 g/kg for men and 1.5 g/kg for women). Carbohydrate and DF intakes were lower than those recommended for diabetics. Zinc, vitamin B-6 and folic acid intakes were marginal if compared to the RDA (Food and Nutrition Board, 1989). Some of the results of Silvis (1989) are also shown in Table 2.16, but it should be remembered that the specific study was done on diabetic patients.

Table 2.16 Mean nutrient composition and correlating blood values in the rural and semi-urban black population

Variable	Vorster *et al.* 1990 Rural		Lubbe 1971 Rural	Silvis 1989 Semi-urban	
	Men (n=20)	Women (n=41)	(n = 226)	Men (n=18)	Women (n=22)
Nutrients					
Energy (kJ)	8 500	8 100	15389	8160.0	9160.0
% E from protein	13.2	13.2	-	18.3	19.9
% E from anim prot	4.4	3.4	-	12.3	14.4
Total protein (g)	67.5	66.1	117.7	89.8	111.2
Plant protein (g)	45.6	49.8	100.0	27.4	28.3
Animal protein (g)	21.9	16.3	17.7	62.0	82.7
% E from fat	16.0	23.3	-	27.6	31.6
P/S ratio	1.2	1.3	-	0.6	0.7
Total fat (g)	35.8	49.7	55.0	60.1	77.8
SFA (g)	8.6	11.3	-	20.6	26.3
MFA (g)	11.4	15.4	-	20.5	25.9
PFA (g)	9.9	14.5	-	11.0	16.5
Cholesterol (mg)	111.6	111.4	-	407.2	455.0
% E from CHO	60.8	55.5	-	48.5	46.4
% E from sugar	4.3	5.6	-	3.3	2.8
Total CHO (g)	303.6	265.3	583.3	225.2	242.5
Added sugar (g)	21.7	25.6	0.4	14.5	16.3
Total DF (g)	21.6	24.3	24.8	24.5	26.1
Calcium (mg)	634.0	633.0	1614.2	1057.2	1427.4
Iron (mg)	21.4	21.6	446.6	13.2	13.7
Magnesium (mg)	474.0	380.0	-	368.8	408.5
Phosphorus (mg)	1312.0	1197.0	2315.1	1497.7	1821.4
Potassium (mg)	2479.0	2515.0	5331.2	3275.9	3926.4
Sodium (mg)	1391.0	1624.0	3539.6	1786.3	2068.6
Zinc (mg)	12.2	11.7	-	13.4	14.9
Copper (mg)	1.3	1.7	-	1.4	1.6

Table 2.16 continued

Variable	Vorster et al.		Lubbe	Silvis	
	Men	Women		Men	Women
Selenium (mg)	2.8	5.0	-	32.9	32.7
Vitamin A (RE)	1400.0	2192.0	-	2373.4	2317.5
Vitamin D (µg)	1.7	1.6	-	2.7	4.6
Vitamin E (µg)	9.5	14.2	-	9.6	15.6
Thiamin (mg)	1.7	1.5	3.4	1.5	1.7
Riboflavin (mg)	1.7	1.3	2.5	2.1	2.6
Niacin (mg)	17.8	18.9	31.1	16.7	20.6
Vitamin B-6 (mg	0.5	0.7	-	1.5	1.7
Folic acid (µg)	77.7	126.0	-	286.2	309.6
Vitamin B-12 (µ	3.0	2.3	-	12.1	11.0
Vitamin C (mg)	38.6	54.6	57.7	109.1	139.3
Pantothenic acid (mg)	1.6	2.2	-	5.4	6.8
Biotin (µg)	7.2	8.3	-	33.6	39.2
Plasma and serum levels					
Vitamin A (µg/dl)	62.3	51.0			
Retinol b prot (mg/l)	4.9	4.3			
Vitamin E (mg/l)	7.8	7.6			
Vit B-12 (µg/ml)	76.0	347.0			
Folate (ng/ml)	5.6	4.8			
Tot lipids (mg/dl)	690.0	610.0			
Haemoglobin (mmol/l)	7.6	6.7		12.5	11.7
Tot cholest (mmol/l)	4.7	4.4		5.4	6.8

(Lubbe, 1971:1291-1296; Silvis, 1989; Vorster et al., 1990:19-23).

The study of Lubbe (1971) showed that urban subjects have a higher energy intake, and the intake increases with age, in contrast to the rural intake that decreases. The same trend could be seen in the weights and percentage body fat of the subjects.

Difference in protein quality is one of the most striking

differences between the rural and urban diets. The animal protein intake is much higher in urban subjects while the plant protein intake is higher in the rural subjects. The same pattern is naturally followed by fat intake (Lubbe, 1971), although the fat intakes of the urban subjects are still low (Kruger, 1987).

Total carbohydrate intakes of rural subjects are higher than those of their urban counterparts (Lubbe, 1971). Kruger (1987) found in urban blacks in Potchefstroom that the intake of DF and sugar was higher than in rural areas. The same was found by Lubbe (1971).

Calcium, phosphorus and iron intakes in rural areas were found to be higher. Iron intakes, especially in rural areas, were exceptionally high. It was speculated that the iron cooking utensils and acid content of the beer brewed in these utensils may further increase iron intake. Both groups have relatively high thiamin values. Riboflavin, nicotinic acid and ascorbic acid intakes of rural subjects were higher (Lubbe, 1971).

Mean nutrient intakes of the populations studied by Kruger (1987) and Lubbe (1971) are shown in Table 2.17.

Table 2.17 Mean nutrient intake of blacks in the Potchefstroom area and by urban Vendas

Nutrient	Kruger-Potchefstroom		Lubbe-Vendas
	Men (n=38)	Women (n=46)	(n = 241)
Nutrients			
Energy (kJ)	14246	11069	15904.0
Total protein (g)	114.4	78.4	126.0
Plant protein (g)	44.5	27.8	59.6
Animal protein (g)	69.4	50.5	66.4
Total fat (g)	102.6	99.7	126.7
% E from fat	27	34	30

Table 2.17 continued

Nutrient	Kruger-Potchefstroom		Lubbe-Vendas
	Men (n=38)	Women (n=46)	(n = 241)
SFA (g)	34.0	30.5	-
MFA (g)	33.7	30.8	-
PFA (g)	24.3	30.7	-
Cholesterol (mg)	481.7	414.4	-
Total CHO (g)	477.7	350.6	150.6
Added sugar (g)	151.6	127.2	62.0
Total DF (g)	33.8	20.4	5.7
Calcium (mg)	1115.9	800.9	420.8
Iron (mg)	20.2	12.7	34.7
Magnesium (mg)	561.2	365.5	-
Phosphorus (mg)	1960.3	1309.7	683.6
Potassium (mg)	4587.1	3143.2	2553.7
Sodium (mg)	2450.1	1814.2	4444.3
Zinc (mg)	16.1	10.6	-
Vitamin A (RE)	2437.5	1500.2	-
Vitamin D (µg)	2.6	1.9	-
Vitamin E (µg)	21.2	22.0	-
Thiamin (mg)	2.2	1.4	1.8
Riboflavin (mg)	2.9	1.7	1.5
Niacin (mg)	28.0	16.8	19.1
Vitamin B-6 (mg)	2.1	1.3	-
Folic acid (µg)	347.9	229.7	-
Vitamin C (mg)	129.6	93.2	20.3

(Kruger, 1987:29,30; Lubbe, 1971:1291 - 1296).

In general, the mean nutrient intake of the urban group is more than that of the rural group. Reasons are that more money is available, food is more abundant, there is a larger variety of food available and people learn to eat many new food items that are not always available in the rural areas.

2.7 ANTHROPOMETRIC MEASUREMENTS

Physical measurements reflect the total nutritional status over a lifetime. Some measurements, such as height, reflect past nutrition or chronic nutritional status. Others, such as weight, reflect present nutritional status. There is an increasing interest in the accurate estimation of body size and body composition due to the recognition of their associations with various chronic diseases.

It is, therefore, important that anthropometric measurements be included in a nutritional survey, as was done in this study.

2.7.1 Height and weight

To determine whether an adult's weight is appropriate for height, the weight is usually compared with one of two sets of charts that give the weights for heights for males and females. The first of these, and certainly the most commonly used, are the Metropolitan Life Insurance Tables (Appendix A). The tables give weight ranges for men and women at 1-inch increments of height for three body frame sizes. The tables presently in use were compiled in 1983. Krause & Mahan (1984) summarise the disadvantages of using these tables as follows:

* The given weight ranges merely reflect the weights of those with lowest mortality rates of insured persons in the USA.

* Weight ranges for lowest mortality do not necessarily reflect optimal weight for height for health or of the population.

* It is often not clear what the body frame size of a person is.

* It was compiled from data gathered from American subjects and are not necessarily applicable to other population groups such as black South Africans.

It is, however, the best available standard at present. The 1983 Metropolitan Height and Weight Tables are shown in Appendix A.

The other set of tables gives one weight for each height for men and women. It was designed according to the median weight for the medium frame size from the 1959 Metropolitan Life Insurance Tables. There are many disadvantages in using a single number as an ideal weight for height, but the advantage is that the person's present weight in relation to this given weight can be expressed as a percentage of the given weight. This allows for rapid calculation of differences from standard weights (Krause & Mahan, 1984).

A weight of 20 % or more above standard is frequently cited as reflecting obesity, while a weight of 20 % or more below standard is frequently equated with nutritional risk (Krause & Mahan, 1984; 208).

Differences in skeletal size and weight can contribute to variations in body weight among individuals of similar height. The same applies to lean body mass. Increased muscular development can increase body weight above the standard weight without the individual being obese. Several such differences in body composition between whites and blacks have been reported in the USA. The best documented difference is an increased density of the lean body mass in blacks because of a heavier and denser skeletal mass and even perhaps an increased and denser muscle mass (Zillikens & Conway, 1990).

However, in most cases, people who are overweight according to the above-mentioned tables are obese, and the excess weight is usually fat relative to lean body mass.

2.7.2 Body mass index

There has been considerable interest in identification of an index of obesity that is independent from stature, correlated

with weight, and that accurately reflects various aspects of body size and body composition. Such an index should also be applicable to the elderly as well as the young, and not only to the obese, but also to the lean (Micozzi et al., 1986) and should indicate over- or underweight, if present (Dias et al., 1989). It becomes apparent that weight *per se* is a poor reflector of body composition.

To account for differences in body composition and more accurately delineate the level of adiposity, a relationship of body weight to height is used. The BMI or Quetelet's index has been proposed for determining ideal body weight for height. The index, of weight in kg/(height in m)^2, has been found to have the least correlation with body height and the highest correlation with independent measures of body fatness. BMI also reflects frame size. A BMI between 24 (for females) and 25 (for males) and 27 is defined as overweight, not obesity. A person with a BMI over 30 is usually classified as obese (Garn, Leonard & Rosenberg, 1986; Krause & Mahan, 1984:518).

Some researchers believe that, on the basis of statistics, the BMI formula for women should not be the same as for men. For women the BMI formula of weight/height$^{1.5}$ was developed during the first of the USA National Health and Nutrition Examination Surveys (Micozzi et al., 1986). This is not, however, widely used.

BMI is not quite independent of height, however. BMI is also influenced by body proportions (relative leg length or relative sitting height). Short-legged individuals have higher BMI-values than long-legged individuals, even by as much as five units. BMI is also influenced nearly to an equal degree by the lean and the fat compartments of the human body (Garn, Leonard & Hawthorne, 1986:997). It is, therefore, advisable not to use BMI as the only determinant of obesity.

2.7.3 Waist-to-hip cicumference ratio

The WHR is an important anthropometric measurement to use in nutritional surveys because it is a risk factor of several degenerative diseases and is discussed in detail in section 2.3.5.

Some researchers use a WHR of more than one as an indication of abdominal adiposity and a WHR of less than one as an indication of gluteofemoral adiposity (Peeples et al., 1989), while other researchers use 0.85 as cut-off points.

Because treatment of obesity is of primary importance, especially in diabetic subjects, a measurement of type of obesity is needed. Both BMI and WHR are easy to measure and might be used as routine examinations at health centres and hospitals, especially in areas where people are prone to the consequences and complications of central obesity. Intervention studies with respect to these factors seem urgent and may be worthwhile. More information on classification (cut-off points of WHR) of the two types of obesity is necessary.

In summary, various methods exist for obtaining anthropometric data. Most of them have some limitations and it is therefore advisable to use different methods in combination to obtain the most accurate data.

2.8 MEASUREMENT OF DIETARY INTAKES

2.8.1 Introduction

In order to determine the habitual food intake of black NIDDM patients at Ga-Rankuwa hospital, a valid, reliable measuring instrument (in the form of a semi-quantitative FFQ) was developed for this study. The background and the process and criteria for development of the questionnaire are discussed in this chapter.

The measurement of human dietary intake is complicated. Two of the most important reasons for this complication are that no current method of dietary assessment is able to yield precise and accurate quantitative amounts of dietary intake (Borrelli et al., 1989) and that there is no ultimate criterion-measuring device that can be used for calibration of other devices (Reshef & Epstein, 1972). The uncertainty about the role of diet in the aetiology of many chronic diseases is then also due, to a great extent, to the problems inherent in the lack of accurate measurement of food intake (Moller-Jensen et al., 1984). Many widely accepted theories are based on methods that should have had the most limited of interpretations. Data from questionnaires should, therefore, be carefully evaluated before statements are made. The choice of dietary measurement must clearly be appropriate to the stated objectives of the study (Karkeck, 1987).

A variety of techniques for characterizing the eating habits of individuals have been developed and utilized. Some methods, such as seven-day diaries or records of foods that have been weighed, require subject training and sustained cooperation whereas others, such as the diet history, are based on lengthy interviews by professional personnel. Recall methods, more readily used with large numbers of subjects, may not provide sufficiently stable estimates of intake for individuals (Samet, Humble & Skipper, 1984).

The FFQ has been widely applied because of ease and speed of data collection and handling. Another reason for its wide use is the fact that it represents a compromise between the more exact methods such as the time-consuming weighed food intake and the representativeness of, for example, the food intake diary (Stuff et al., 1983). A food frequency interview can be focused on a subject's usual diet or on specific nutrients. In addition to frequency of consumption, a FFQ may assess amounts consumed, often in relation to standard portions, and the stability of dietary habits (Samet, Humble & Skipper, 1984). Although the validity of this and other methods remains to some extent unestablished, the FFQ is a feasible approach for determining relative intakes of specific foods or nutrients or a wide variety of nutrients (Willet et al., 1987).

Therefore, for the purposes of this study, it was decided to use an adapted semi-quantitative FFQ for the following reasons:

* Several 24-hour recalls or other shorter methods for each person will be necessary to determine the habitual food intake; time-wise this is not practical (Morgan et al., 1987).

* The other methods, such as weighed intakes, are also time-consuming and not practical in a situation where an overall habitual intake pattern is sought after - the FFQ was developed to determine habitual food intake (Hankin, 1986) and is, therefore, the best choice for this study.

* For this study, the FFQ was adapted to include amounts of food items consumed, so that a more reliable nutrient analysis could be done and because of the fact that amounts correlate positively with frequency. The data should then give more reliable information (Samet, Humble & Skipper, 1984).

* Variability of the diet does not affect the reliability of the FFQ (Reshef & Epstein, 1972).

* It imposes less burden on the respondent and is more reflective of long-term intake or usual food intake than a recall of foods eaten during the past 24 hours where day-to-day variation may lead to inaccurate data (Axelson & Csernus, 1983; Musgrave et al., 1989).

* It can afterwards form the basis for nutrition education, allowing the dietitian and respondent to relate individual eating patterns to specific foods or food groupings.

* The FFQ is independent of client ability or inclination to maintain a diary.

* It provides immediate feedback (Musgrave et al., 1989).

* It is a low-cost method and simple to administer (Axelson & Csernus, 1983; Mullen et al., 1984).

* It is a well-known fact that the variation between intake of individuals is less than the variation in the day to day food consumption of any single person. The FFQ will prevent the variation from having a major impact, because it gives an estimation of habitual intake (Stuff et al., 1983).

Validity and reliability are essential characteristics of a FFQ as a measuring instrument and contribute to its usefulness, but these characteristics do not ensure the use of the instrument in all circumstances; it is still only valid and reliable for the specific circumstances it was developed for. It is, therefore, necessary to determine first whether the purpose of the exercise that the questionnaire is going to be used for, justifies the effort of standardising the questionnaire (Talmage & Rasher, 1981; Talmage and Rasher, 1982).

2.8.2 Measurement with a questionnaire

Several studies have established to some extent the reliability

and validity of food frequency data and have measured these against other dietary intake data-gathering methods (Mullen et al., 1984; Musgrave et al., 1989; Samet, Humble & Skipper, 1984; Willet et al., 1985; Willet et al., 1987). Comparison with other methods is an important part of scientific evaluation and objective judgement (Kerlinger, 1986; Stanley & Hopkins, 1972).

2.8.2.1 **Validity of the questionnaire**

To be valid the instrument must measure what it is supposed to measure (Kerlinger, 1986). It should measure the goal(s) of the specific instrument accurately. To be valid the instrument must also be comprehensible to the person who will be subjected to it (Bailey, 1982).

There are different classes of validity: validity of the contents, validity of criteria and validity of construction of a questionnaire (Kerlinger, 1986).

Validity of contents is not statistical and has relation with the contents of the questionnaire. It refers to the critical evaluation of the capability or representativeness of the items that are part of the questionnaire, the purpose and structure of the items and the spectrum they cover (Smit, 1983). If the content is valid, it also implies that the questions in the questionnaire are clear and understandable and important enough to be included (Bailey, 1982; Kerlinger, 1986).

Validity of criteria can be measured by using another measuring instrument that measures the same objectives and by comparing the results of the two measuring instruments (Louw, 1982). In the validation of nutritional assessment methods, the reference measurement should be as accurate and as precise as possible and any errors associated with the two methods should be independent (Willet et al., 1985).

Validity of construction shows the extent to which a

questionnaire can be used to measure a theoretical concept (Louw, 1982). The relationship between the results of a measurement and the theory can thus be determined (Kerlinger, 1986). That implies that the questions in the questionnaire should be based on valid theoretical concepts or hypotheses. The hypotheses are tested by implementing the questionnaire.

2.8.2.2 **Reliability**

Synonyms for the reliability of a measuring instrument are stability, accuracy, dependability, predictability and consistency. Talmage and Rasher (1981) describe a reliable measuring instrument as one that is relatively free from error (the accuracy or precision of the measuring instrument) and that can give the same results with repetition, if all the other factors are kept constant. That implies that a questionnaire should be tested to determine the reliability before it is used for actual experimental work (Guilford & Fruchter, 1978). The construction process of this test should be designed to minimize the mistake factors in the instrument. Any factor that may lead to correct but untruthful answers, should be minimized. Factors that may lead to correct but untruthful answers are:

* Ambiguity of questions

* Questions that are difficult to answer so that the respondent starts to guess

* The respondent does not have enough time to answer the question or does not understand it correctly

* Answers that are interpreted incorrectly

* The interview is interrupted or the procedure unacceptable

* Large individual differences

* Length of the questionnaire
  (Guilford & Fruchter, 1978; Smit, 1983).

It is not always possible to control such errors. Bailey (1982) feels that the influence of uncontrollable errors can be cancelled out by other uncontrollable errors, if the sample is large enough. Nevertheless, if such errors are detected, they should be excluded from the questionnaire or else a correction factor should be used.

A problem that may have a large influence on reliability is the memory of the respondent. Recall often varies in completeness - individuals sometimes fail to report consumption of some foods that were actually eaten or recall foods that were never eaten. People often eat without devoting full attention to the type and amount of foods they are eating. Reported estimates of amounts and kinds of foods are often inaccurate too. Another factor that may influence reliability is that individuals often will tell the interviewer what they think that they should tell and not what was actually eaten. Other factors such as the diet of the day before or memories of old food habits, and inability to correctly judge the portion size at the time it was consumed, may also have an influence (Moller-Jensen, 1984; Dwyer, Krall & Coleman, 1987). Stimuli that may help the subject to remember what was eaten, are therefore important in improving the reliability of the questionnaire.

According to Wu, Whittemore & Jung (1986) the FFQ is consistently more reproduceable than either the dietary history (because the FFQ does not need such a long memory) or the 24-hour recall (which only measures one day's intake). The dietary history also tends to give higher estimates of nutrient intake and the 24-hour recall lower estimates, compared to a more reproduceable method such as the FFQ (Borrelli et al., 1989).

The questionnaire should also be objective. Objectivity in a questionnaire means that the results must be independent of any

subjective elements or any personal desires that the researcher may have (Bailey, 1982).

2.8.2.3 **Development of a measurement test**

The steps that are followed during the construction of a measuring instrument such as a questionnaire ought to focus primarily on accuracy, reliability and validity. Smit (1983) formulated the following steps to be taken during the construction of a measuring instrument:

* Specify the population on which the questionnaire will be used as accurately as possible

* Specify the way in which the questionnaire will be applied

* Specify the goal(s) of the instrument

* Write down the items that will be used

* Analyse the items to see whether they ask what they ought to, whether they are comprehensible and whether the researcher will get the intended answers

* Choose the needed items and arrange them in logical order

* Standardise the application procedures, instructions, time it will take and evaluation of responses

* Do the technical analysis - validity, reliability, norms

* Revise the measuring instrument

When the goals of the instrument are specified, it should also include the possibilities of application, not only by specifying the way in which the questionnaire will be applied in the specific study, but also in other circumstances (Smit, 1983).

The technical analysis should not be left to be done later during the construction phase; it forms part of the total development of the instrument from the beginning (Anastasi, 1983).

In order to analyse the items and to standardise the application procedures, the questionnaire should be tested on a sample of respondents with the same characteristics as those of the respondents who will be used in the study. According to Bailey (1982) the number of people for the pre-test sample will be determined by factors such as similarities between the pre-test sample and the sample of the study, the type of research and practical implications, for example randomization and the time factor. Kerlinger (1986) recommends that the sample be as large as possible. The application procedures and the evaluation of the responses should also be standardised and kept constant to ensure comparable results (Smit, 1983).

Revision of the instrument will only be necessary after a certain period if the questionnaire has been used for a period of years or if it is used in other studies (Smit, 1983).

2.8.2.4 **Problems with data obtained with a questionnaire**

Data obtained with a questionnaire may have several shortcomings. Morgan et al. (1987) showed that older women had reported lower intake levels than younger women, women residing in households of smaller size had reported higher intake levels than those residing in larger households, and that under-reporting had occurred within the group of women who have lower education levels. Subjects usually over-report when food intake is low and under-report when food intake is high (Stunkard & Waxman, 1981). Men tend to over-estimate food amounts (Karvetti & Knuts, 1985).

2.8.3 Twenty-four hour dietary recall

The 24-hour dietary recall is a useful epidemiological tool for measuring food intake in groups of individuals (Frank et al.,

1984). It was used in this study to measure the reliability and validity of the FFQ and to improve the accuracy of responses. It was impractical to use a more extensive method than the FFQ for standardisation, as was done by Willet et al. (1985).

The 24-hour dietary recall method is one of the most effective and least costly methods for quickly assessing mean intakes for groups of individuals, but can only give information comparable with that collected with more extensive methods such as the FFQ if it is done repeatedly and in combination with another quick method. It is, however, a far better method than mailed questionnaires or self-reported dietary intake questionnaires (Morgan et al., 1987), but may not be representative of the usual dietary intake (Stunkard & Waxman, 1981). It should also be remembered that the FFQ gives higher mean estimates than dietary recalls (Larkin et al., 1989).

Some researchers feel that the 24-hour dietary recall method does not provide useful information on the distribution of individual nutrient intakes since much of the apparent variation represents within-person variability (Willet et al., 1985). Other have found that very few subjects are able to accurately remember the types and amounts of food they had consumed in the previous 24 hours if the recall is done only once, especially because of large day-to-day variation (Todd, Hudes & Calloway, 1983). Foods eaten regularly show the highest recalled accuracy while foods less frequently consumed are often omitted (Karvetti & Knuts, 1985). Omissions, additions, and misidentifications are thus major sources of error in recalled food items. However, in the study of Karvetti & Knuts (1985) mean nutrient intakes did not differ much from observed intakes and they believe that the 24-hour recall can still be used to assess means and trends in food and nutrient intakes.

2.8.4 The food frequency questionnaire

The FFQ is used to obtain information on the habitual intake and

diet history of the population that is studied (Hankin, 1986). There are many advantages to this method, of which some were discussed under 2.8.1, including high response rates and minimal burden to respondents. If they are trained properly, the basic adminstration can be done by non-professionals (if coding is done by experienced nutritionists). The method also allows analysis by food groups, food components or nutrients (Medlin & Skinner, 1988). Detailed information, such as cooking methods and recipes, can easily be obtained with a FFQ to ensure the correct measurement of especially fat intake.

2.8.4.1 Criteria for the use of a food frequency questionnaire

Although trained dietitians or nutritionists are necessary for coding of the data, the implementation of a FFQ is often done by non-professionals and the following criteria should therefore be met in compiling the questionnaire:

* Foods and nutrients selected should be suitable to test the hypotheses concerning the etiology of the disease studied.

* The food items in the questionnaire should be representative of the eating patterns of the population.

* The data should provide a valid picture of the usual diet of each person during a particular time period.

* The method should be reproducible and objective (Hankin, 1986:870).

2.8.4.2 Development of the food frequency questionnaire to obtain diet history

After specification of the population, the application method and goals, as discussed under 8.2.3, data should first be collected on the foods typically consumed by a representative sample of the population under study. This data should then be interpreted and

coded for computer use or another analysis method. Thereafter the appropriate food items for the questionnaire should be selected and the appropriate serving sizes of the selected items identified. Enough cues should be given to respondents to ensure that they remember as many of the consumed food items as possible (Musgrave et al., 1989). The rest of the steps as discussed under 2.8.2.3, will then follow.

Weight estimation is one of the most important problems in gathering dietary intake data (Hankin, 1986). A visible technique such as photographs of prepared foods, plastic food models, available household measures such as cups and spoons, or geometric shapes can be used for showing different food items and portion sizes to subjects. Training of the respondents also makes a difference; estimates do not only become more accurate for some food items, but also more consistent (Bolland, Yuhas & Bolland, 1988). However, it should be taken into consideration that visual aids such as photographs may contribute to variation, because they are not three-dimensional. Photographs may lead to over-estimation because of this. Three-dimensional models together with photographs may improve recall of certain foods, better weight estimation, and will make the interview more interesting to the respondent (Boeijen, Brummer & Cramwinckel, 1985). Any procedure that offers a choice of serving sizes lends itself to a structured, objective questionnaire that minimizes subjective interpretation. It also saves time because it permits the interviewer to record only frequencies and serving sizes measured against the visual aids (Hankin, 1986; Bolland, Yuhas & Bolland, 1988).

A FFQ should be ethnic and culture-specific and should take factors such as geographic location, education and socioeconomic status into consideration (Musgrave et al., 1989).

Up to now, limited attention has been given to determining adequate training of interviewers and to measuring the effect of interviewers' skills on estimating nutrient intake. For economic

reasons, selection and training of interviewers are often not priorities but these can make a big difference to the quality of the data obtained (Frank et al., 1984). Not all researchers even use an interviewer for a FFQ; some questionnaires are filled in by the respondent himself, but the use of a skilled interviewer may improve the quality of the data obtained. Careful probing by an interviewer, particularly on the more general items, will increase the accuracy with respect to nutrient content of the total diet (Krall & Dwyer, 1987). Frank et al. (1984) found that more experienced interviewers can get more information on snacks, unique foods and unusual dietary patterns from subjects. Food items are also more correctly identified and consistently classified and named. Trained interviewers also have less difficulty in quantifying problem foods such as liquids, meats and sweets. The above-mentioned researchers also suggest that duplicate recalls should become a routine and is a manageable quality control technique for dietary recall collection.

The interviewers should also be trained to use the specific FFQ and understand the customs of the specific population (Hankin, 1986).

Coding of items is another problem area and should only be done by an experienced, specially trained dietitian or nutritionist. The nutrient analysis base size is very important. It should allow for all the typical foods of the population or should have space for additional entries of typical foods (Frank et al., 1984). Translation of food consumption to nutrient intake requires a suitable nutrient data base. The interpretation of nutrient intake data is influenced by the quality of the food composition data used to calculate it. The data should also consider the bioavailability of nutrients and missing values should be indicated (Guthrie, 1989; Karkeck, 1987).

Problems as discussed in 8.2.4 and inherent weaknesses of the FFQ should, however, be recognised and compensated for. Individuals may, for example be unaware of their "usual" food intakes,

although they can provide fairly accurate and reliable data on intakes for short, specific periods. A 24-hour recall method to accompany the food frequency may help to compensate for this. However, in most Western cultures, the diet varies considerably from day to day, so that data from short, specific periods rarely represent individuals' usual patterns. The ever-increasing complexity of the food supply also magnifies problems (Medlin & Skinner, 1988). Individuals may not be able to adequately describe food items eaten, because foods that look and taste nearly identical may differ in nutrient composition. Inaccurate data may result from incomplete recall, since the process of eating is often automatic (Dwyer, Krall & Coleman, 1987). Many people eat differently on weekends than on weekdays and that should be taken into consideration (Medlin & Skinner, 1988).

2.8.5 Interpretation of results

Interpretation of the findings of any dietary survey assumes the careful and complete collection of records of food intake over a prescribed period of time and an analysis of its nutrient equivalent using tables of food composition. The assessment of nutrient adequacy, however, depends on the availability of a standard, usually age and sex-specific, against which to compare the intake. Some of the standards currently used represent a generous intake with a wide margin of safety above the needs of most people. But, if the basis for the standard is not clearly identified and understood, it is very difficult to interpret the findings and to make comparisons from one study to another. The RDA of the USA (Food and Nutrition Board, 1989) represents a level of intake sufficient to meet the needs of essentially all healthy people, and will be used as standard in this study because South African standards are not available. For most nutrients, as for most biological data, the coefficient of variation of the requirements in the RDA is assumed to be 15 % of the mean. Thus, the RDA is set at 130 % of the mean for most of the nutrients. Therefore, an intake of 77 % of the RDA meets the needs of one half of that particular population (Guthrie,

1989:34). In the discussion of nutrient intakes, this will also be taken as the cut-off point for possible deficiencies, unless otherwise specified.

In spite of the many unresolved issues relating to dietary standards and the interpretation of dietary intake data, it is still possible to use them to make a reasonable assessment of dietary adequacy of groups and individuals (Guthrie, 1989).

2.8.6 Conclusion

Although considerable progress has been made in the development of dietary data collection methods currently in use, there is still an urgent need for improved, accurate dietary assessment methodology with significant refinements and creative approaches (Stunkard & Waxman, 1981). Techniques that are

* less time-consuming to ensure better cooperation,

* appropriate for different population segments,

* give precise guidelines for determination of appropriate sample sizes and

* that give accurate, quantitative data on individuals' usual food intake

are still necessary. The steps in the estimation process that lead to mis-estimation should also be identified and controlled (Larkin et al., 1989). It should always be remembered that members of a population do not maintain the same dietary habits over an indefinite period of time (Guthrie, 1989).

We also need clinical indices to validate accuracy of dietary data and ways to decrease the time and cost of initial data processing. Improved laboratory techniques for analysis of certain nutrients in foods to increase accuracy of nutrient data

bases and comparability of data bases are other fields where research is needed (Medlin & Skinner, 1988).

CHAPTER 3

METHODS AND MATERIALS

3.1 INTRODUCTION

The study consisted of three phases. During the first phase (Chapter 4) the eating habits and nutrient intakes of 94 black DM subjects in the Ga-Rankuwa area were examined with the use of a semi-quantitative FFQ, designed and tested for this population. In the second phase (Chapter 5) the GI of a traditional African meal and the effect of this meal on glucose disposal during a second meal were examined in 14 NIDDM patients. In the third phase (Chapter 6) the long-term effects of the African diet on metabolic control of black NIDDM patients were monitored. The study design and general methodology of each phase are given in Chapters 4, 5 and 6 respectively. In this chapter, methods and material used for anthropometrical measurements and biochemical analysis, as well as the statistical methods employed, will be discussed in detail.

3.2 ANTHROPOMETRIC MEASUREMENTS

During visits to the clinic the following measurements were made:

3.2.1 Weight

Patients were weighed in light, indoor clothing. The same electronic Soehnle® scale was verified each morning with a 10 kg weight, and was used for all the patients and for all the visits. Half a kilogram was subtracted from measured weight for clothing.

3.2.2 Height

During the first, second and last visit the heights of the patients were measured. They had to stand erect, without shoes, feet together against a beam with a fixed measuring tape and a bar resting flat on the top of the head. The height was then taken by a trained interviewer with the patient looking straight

ahead, without tipping the head up or down, and the tip of the ear and outer corner of the eye in a line parallel to the floor.

3.2.3 Body mass index

The BMI was then calculated as weight (kg)/(height (m))^2.

3.2.4 Waist-to-hip circumference ratio

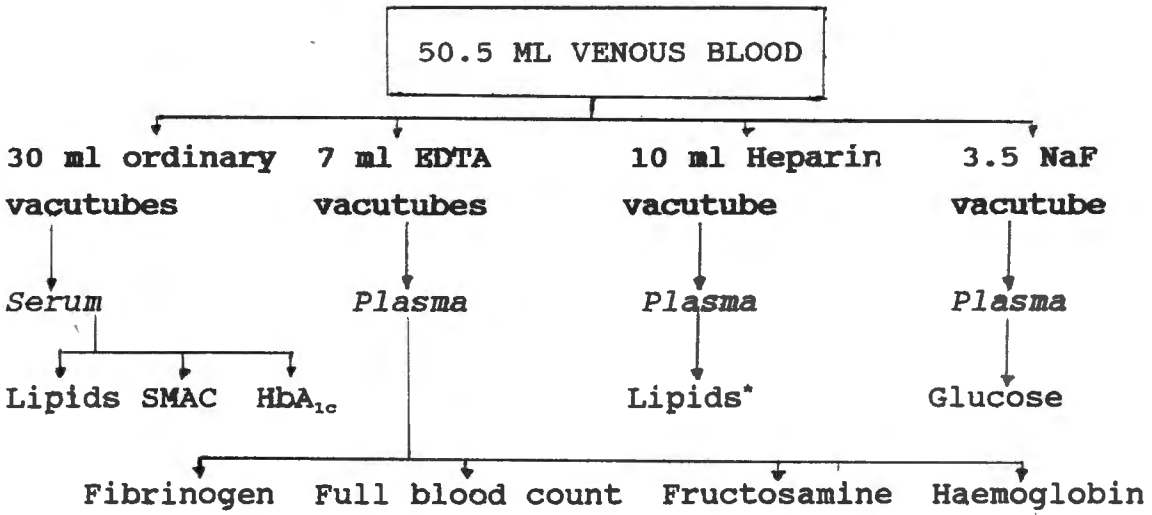
The circumference of the waist and hip was measured with a non-elastic, marked measuring tape, to the nearest 0.1 cm, in a standing position. Waist circumference was measured as the minimal girth between the xiphoid and the umbilicus and the hip circumference as maximal hip girth.

3.3 BLOOD PRESSURE

The blood pressure of patients was measured once during each visit. It was measured with a sphygmomanometer with a standard cuff placed about the midpoint on the left upper arm in a sitting position. Systolic blood pressure was recorded at the point of appearance of sounds (first Korotkoff sound) and diastolic blood pressure at the point of disappearance of sounds (fifth Korotkoff sound).

3.4 BLOOD SAMPLING

Antecubital venous blood samples (50.5 ml) were taken after an overnight fast, using a 21-gauge needle with a vacutainer directly into the vacutubes. Some of the patients were very obese and it was therefore necessary to put pressure on the vein. The allocation of the blood samples was as follows:



* Lipids of patients who did not give enough blood, control for patients with very high lipid values

Figure 3.1 Allocation of blood samples taken from the NIDDM patients

Except for the 30 ml blood drawn into the ordinary vacutubes, all the other samples were gently turned three times. The samples were kept on ice until the end of the clinic (not longer than one-and-a-half hour) and then taken to the laboratories for immediate analysis, except in the case of the SMAC-analysis. The centrifuged serum for the SMAC-analysis was stored at -20 °C until the end of the study when all the SMAC-analyses were done simultaneously.

3.5 BLOOD ANALYSIS

The blood analysis was done by different laboratories. In the discussion of the analysis of the different blood consitutents, the laboratory where the amounts of the specific constituents were determined, will be marked as follows:
MEDUNSA clinical pathology laboratory:^{cm}
Pretoria University clinical pathology laboratory:^{cp}
MEDUNSA haematology laboratory:^{hm}

At the clinical pathology laboratory of MEDUNSA, a control sample was run before each batch of ten samples and the analysis of

samples who gave strange values, was repeated after a control sample was run. A control sample was run once each morning by the haematology laboratory of MEDUNSA. When strange values for a particular consituent were obtained in the above-mentioned laboratory, the analysis of the sample was repeated on another, identical analyser. At the clinical pathology laboratory of Pretoria University, two control samples were run after every eighteenth sample that was done and calibration was done after every thirty-sixth sample. The analysis of samples with strange values was repeated.

3.5.1 Lipids

3.5.1.2 **Total serum cholesterol**[cm]

An enzymatic colorimetric test with cholesterol esterase, cholesterol oxidase and 4-aminophenazone (Uni-Kit Cholesterol-PAP method of Roche®) was used to determine total serum cholesterol. In the presence of peroxidase, the hydrogen peroxide formed affects the oxidative coupling of phenol and 4-aminophenazone to form a red-coloured quinoneimine derivate. The colour intensity is proportional to the cholesterol concentration and is determined by monitoring the absorbance in the range of 480 to 550 nm.

3.5.1.2 **Serum high-density lipoprotein cholesterol**[cm]

Chylomicrons, VLDL and LDL are precipitated by adding phosphotungstic acid and magnesium ions to the blood serum. Centrifugation leaves only the HDL in the supernatant. The cholesterol content is determined enzymatically with Monotest® (Boehringer Mannheim) on wavelength 470 - 560 nm.

3.5.1.3 **Serum low-density lipoprotein cholesterol**

LDL-cholesterol is determined with the following formula:
LDL-chol. = Total chol. - Triglycerides - HDL-cholesterol
2.2

3.5.1.4 **Serum triglycerides[cm]**

An enzymatic colorimetric test with glycerol phosphate oxidase and 4-aminophenazone was used to determine total triglycerides. In the presence of peroxidase, the hydrogen peroxide formed affects the oxidative coupling of 4-chlorophenol and 4-aminophenazone to form a red-coloured quinoneimine derivate. The colour intensity is proportional to the triglyceride concentration and is determined by monitoring the absorbance in the range of 490 to 550 nm. Uni-Kit reagents of Roche[R] for the Triglycerides-PAP test were used.

3.5.1.5 **Serum apolipoprotein A[cm]**

In an immunochemical reaction the apolipoproteins contained in human serum form immune complexes with their corresponding antibodies. The antibodies are obtained from antiserum prepared from rabbits or sheep that were immunized with purified human apolipoprotein. The concentrations present can be determined quantitatively by turbidity assays. The results are evaluated using a reference curve prepared with the aid of dilutions of the standard.

3.5.1.6 **Serum apolipoprotein B[cm]**

In an immunochemical reaction the apolipoproteins contained in human serum form immune complexes with their corresponding antibodies. The antibodies are obtained from antiserum prepared from rabbits that had been immunized with purified human LDL-cholesterol. The concentrations present can be quantatively determined by turbidity assays. The results are evaluated using a reference curve prepared with the aid of dilutions of the standard.

3.5.1.7 **Plasma fibrinogen[cm]**

Fibrinogen from ethylene-diamine-tetra-acetic acid (EDTA)-plasma

forms immune complexes in an immunochemical reaction with specific antibodies. The antibodies are formed from antiserum which has been manufactured by immunizing rabbits with the fibrinogen from human plasma. The concentrations present can be ascertained quantitatively by turbidimetry. Evaluation is effected by means of a reference curve prepared with the aid of dilutions of standard plasma.

3.5.2 Glucose control

3.5.2.1 **Capillary blood glucose**

Blood glucose was monitored after an overnight fast, using a Glucometer® II Reflectance Photometer (Ames Division, Miles Laboratories, Elkhart, Indiana, USA) and Glucostix® reagent strips (Ames Division, Miles Laboratories, Slough, England). The second drop of blood from a sterilised, pricked fingertip was used to cover both test pads of the reagent strip. Glucose oxidase catalyses the oxidation of glucose in the blood by oxygen in the atmosphere, producing gluconic acid and hydrogen peroxide. In the presence of hydrogen peroxidase, gluconic acid reacts with the chromogenic reagent strip. A shade of colour is produced with an intensity proportionate to the glucose concentration. The light reflected from the reacted test pad is measured electronically, and a direct readout of the glucose concentration is displayed on the digital display screen.

3.5.2.2 **Fructosamine**[cm]

The fructosamine concentration was measured on EDTA-plasma with a colorimetric test. The test is based on the ability of ketoamines to reduce nitroblue tetrazolium in an alkaline medium. The rate of formation of formazane is directly proportional to the fructosamine concentration and is measured photometrically. Measurement was made against a Roche® Fructosamine Calibrator (article 0728322) which was standardised via glycated polylysine and human serum glycated with ^{14}C-glucose.

3.5.2.3 Glycosylated haemoglobin[cm]

Glycosylated haemoglobin in whole blood was determined with an affinity chromatographic method based on the separation and quantification of glycosylated haemoglobin from red blood cell hemolysates. The blood sample containing a mixture of glycosylated and non-glycosylated variants is put in a chromatographic column with the reagents of the Pierce GlycoTest® kit (Cat. No. 4,269,605). The aldehyde group of glucose forms a reversible Schiff base linkage with the N-terminus of the α- and ß-chains and certain lysine residues of the heamoglobin molecule. This labile adduct then undergoes an Amadori rearrangement to the stable keoamine, commonly referred to as glycosylated haemoglobin. The resultant coplanar, *cis*-diol groups of glycosylated heamoglobins, can interact with the reagents to form a reversible five-member ring complex which can be dissociated by a sorbitol-containing buffer. Relative ratios of glycosylated and non-glycosylated fractions are then quantified by measuring the absorbance at an appropriate wavelength (414 nm).

3.5.2.4 Haemoglobin[hm]

Total haemoglobin was determined with a colorimetric method from Boehringer Mannheim (BM: Cat. no. 124 729) from EDTA whole blood. Cyanmethaemoglobin is formed from haemoglobin, cyanide and ferricyanide. Cyanmethaemoglobin Standard Set (BM: Cat. No> 125 482) was used to plot out a standard curve.

3.5.2.5 Haematocrit[hm]

Blood was collected into an EDTA vacutube and centrifuged in a heamatocrit centrifuge. The percentage of heamatocrit was read from a graduated haematocrit frame.

3.5.2.6 Serum glucose[cm]

The glucose concentration in serum was determined with a

colorimetric method. σ-toluidine reagent is added to serum and placed in a 100 °C water bath for ten minutes. The mixture is then cooled for a few minutes and remixed. The absorbance is then measured against a water blank at 630 nm and 480 nm. The difference in absorbance is subtracted to get the absorbance from glucose. The glucose concentration is calculated by comparing the absorbance from samples to that of a standard glucose solution.

3.5.3 Electrolytes

3.5.3.1 **Sodium**[CP]

Sodium is measured with a direct potentiometric procedure using a sodium ion-selective glass electrode. The sodium sample is mixed with an air-segmented stream of sodium buffer. The buffered sample flows past the glass membrane of the sodium ion-selective electrode. A reference stream flows in the opposite direction to form a liquid junction that permits electrical contact between the electrodes, and due to changes in electrical potentials that exist between the ionically variable outer surface and the ionically constant inner surface of the electrode, the sodium is automatically measured against the potential of the reference electrode.

3.5.3.2 **Potassium**[CP]

Potassium is measured with a direct potentiometric procedure that uses a potassium-selective valinomycin-based electrode. The potassium and sodium electrodes are combined in the same channel. Potassium is, therefore, measured in the same way as the sodium.

3.5.3.3 **Chloride**[CP]

A colorimetric procedure was used for the quantitative measurement of chloride in serum. The chloride sample is added to an air-segmented stream of chloride sample diluent. The

stream is then dialysed against an air-segmented stream of chloride recipient solution to remove the chloride sample from protein and serum pigment interferences. The chloride colour reagent is added to the recipient stream and the following reactions occur:

$Hg(SCN)_2 + 2Cl^- \quad \text{-----} > \quad HgCl_2 + 2(SCN)^-$

$3(SCN)^- + Fe^{3+} \quad \text{-----} > \quad Fe(SCN)_3$

The absorbance of the analytical stream is measured at 480 nm.

3.5.3.4 **Phosphorus**[CP]

To measure inorganic phosphorus from serum, a sample was mixed with inorganic phosphorus sample diluent and dialysed into an inorganic phosphorus recipient diluent. Following dialysis, ammonium molybdate is added and the phosphomolybdate complex formed passes to the colorimeter where the ultraviolet light that it absorbs is measured at 340 nm.

3.5.3.5 **Calcium**[CP]

Serum is added to a diluted solution of hydrochloric acid containing 8-hydroxyquinoline. The hydrochloric acid releases the protein-bound calcium and the 8-hydroxyquinoline binds the free magnesium ions present in the serum. The free, ionized calcium ions are dialysed into the analytical stream of cresolphthalein complexone, containing additional 8-hydroxyquinoline across a semipermeable membrane. Upon the addition of diethylamine to the analytical stream, a coloured complex is formed between the calcium and the dye. The absorbance of the reaction product is measured at 570 nm.

3.5.4 Proteins, enzymes and metabolites

3.5.4.1 **Carbon dioxide**[CP]

Carbon dioxide is released from the serum by acid and then

absorbed by an alkaline buffer solution containing phenolphthalein over a silicone rubber dialyser membrane. The decrease in colour, when measured at 550 nm, is proportional to the carbon dioxide content of the sample.

3.5.4.2 Serum ureum nitrogen (BUN)[CP]

The blood serum sample is diluted in a air-segmented stream of BUN sample diluent. Diacetyl monoxime is hydrolysed to diacetyl in a relatively weak acid solution, which reacts directly with the urea, which is highly soluble in water, in the presence of acidic ferric ions and sulphuric acid. The presence of thiosemicarbazide intensifies the colour of the reaction. The absorbance of the analytical stream is measured at 520 nm.

3.5.4.3 Uric acid[CP]

Uric acid is determined enzymatically with a chemical oxidizing agent by measuring hydrogen peroxide production. A serum sample is diluted with uric acid-2 sample diluent, which contains ascorbate oxidase. The serum sample is then mixed and dialysed into a buffered recipient stream consisting of the uric acid colour concentrate and the enzyme reagent (free uricase and peroxidase). The dialysate containing the uric acid diffusate undergoes the following reactions:

$2H_2O$ + Uric acid + O_2 $\xrightarrow{\text{Uricase}}$ H_2O_2 + CO_2 + Allantoin

H_2O_2 + 4-aminoantipyrine $\xrightarrow{\text{Peroxidase}}$ Chromophore

+ 3 N,N-(Dimethylamine) (benzoic acid) + H_2O

The coloured complex formed by the reactions is measured at 550 nm.

3.5.4.4 Creatinine[CP]

A serum sample is diluted with creatinine sample diluent. The diluted sample is dialysed against the creatinine recipient solution to remove the creatinine in the sample from protein and

other endogenous serum interferences. Sodium hydroxide solution and creatinine colour reagent are added to the recipient stream to form the red-coloured chromogen in an alkaline medium. The absorbance of the analytical stream is measured at 505 nm.

3.5.4.5 Total bilirubin[CP]

Two independent but interrelated test channels, a sample channel and a blank channel, are used to measure total bilirubin in serum. In the sample channel the serum sample is added to a stream of caffeine diluent. This stream reacts with the diazo reagent to form an azobilirubin complex. A strongly alkaline sodium potassium tartrate buffer is added, which stabilizes protein and eliminates the effect of variation in sample pH. Upon the addition of the alkaline buffer reagent, a conversion in colour takes place from a neutral pink to an alkaline blue azobilirubin. The final colour appears green since the blue alkaline azobilirubin complex is mixed with the yellow pigments derived from the reaction of caffeine with sulphanilic acid.

In the blank channel the sample is added to the caffeine diluent. Subsequent additions of sulphanilic acid and sodium potassium tartrate are made to provide a chemical environment similar to that present in the sample channel. However, missing from the blank channel and present in the sample channel is the diazo reagent that combines with the bilirubin in the serum sample to form the azobilirubin complex. As a result the absorbance determined in this channel is predominantly that of endogenous serum pigments.

The absorbance of each channel is measured at 600 nm. Blank subtraction is accomplished automatically by differential colorimetry.

3.5.4.6 Direct bilirubin[CP]

Two independent but interrelated test channels, a sample channel

and a blank channel, are used to measure direct bilirubin in serum. In the sample channel the serum sample is added to a stream of direct bilirubin sample diluent. This stream reacts with the diazo reagent to form an azobilirubin complex. Since the direct reaction is time-dependent, the reaction is stopped within one minute, by the addition of the ascorbic acid reagent which inactivates the diazo reagent. After the reaction has been quenched, a strongly alkaline sodium potassium tartrate buffer is added which solubilizes the protein and eliminates the effect of variation in sample pH. Upon addition of the alkaline buffer to the serum sample, a conversion in colour takes place from the neutral pink to the alkaline blue azobilirubin.

In the blank channel the sample is added to the direct bilirubin sample diluent. Subsequent additions of sulphanilic acid, ascorbic acid reagent and sodium potassium tartrate are made to provide a chemical environment similar to that present in the sample channel. The diazo reagent that combines with the bilirubin in the serum sample to form the azobilirubin complex is used in the sample channel but not in the blank channel. As a result the absorbance determined in the blank channel is produced predominantly by endogenous serum pigments.

The absorbance of each stream is measured at 600 nm. Blank subtraction is accomplished automatically.

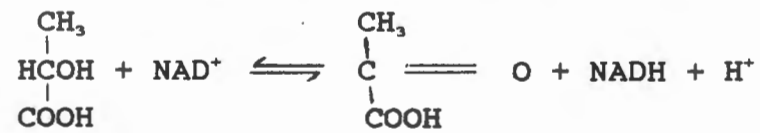
3.5.4.7 Serum aspartate aminotransferase (AST)[CP]

AST activity is determined by a three-point rate reaction. The choice of measurement time intervals is based on well-defined reaction conditions during which the AST obeys zero-order kinetics. A MDH/LDH/NADH reagent and sample diluent are combined with the sample and incubated for 1.7 minutes at 37 °C to remove endogenous serum interferences. Following the addition of α-ketoglutarate reagent, the transamination and dehydrogenase reactions proceed with additional incubation of 1.1 minutes (also at 37 °C). The absorbance is then measured at the first point.

After removal of endogenous serum interferences in the first heating bath the decrease in the absorbance of the analytical stream (determined at 340 nm) is also measured at two other points. The results are compared by computer for linearity. If the slopes agree, the absorbance difference between point one and three is used to calculate the AST activity.

3.5.4.8 **Serum lactate dehydrogenase (LDH)**[CP]

LDH is an enzyme that catalyses the following reaction:



(L-lactic acid) (Pyruvic acid)

Nicotinamide-adenine dinucleotide (NAD) has no absorption at 340 nm, but NADH has an absorption peak at this wavelength and the enzymatic activity is proportional to the amount of NADH produced. LDH is measured by a two-point rate reaction. The choice of measurement times is based on well-defined reaction conditions during which the enzyme obeys zero-kinetics.

The serum sample is added to the LDH substrate (buffered lactic acid solution). NAD is then added to initiate the enzymatic reaction. After a brief incubation period (20 to 25 seconds) at 37 °C, the absorbance is measured at the first point. The reaction mixture is further incubated in a second 37 °C incubation bath and the final absorbance is measured. The increase in absorbance with time is proportional to the LDH activity.

3.5.4.9 **Serum alanine aminotransferase (ALT)**[CP]

ALT activity is determined by a three-point rate reaction. The choice of measurement time intervals is based on well-defined reaction conditions during which the enzyme obeys zero-order kinetics. LDH/NADH reagents and sample diluents are combined

with a serum sample and incubated for 1.7 minutes at 37 °C. α-ketoglutarate reagent is added and transamination and dehydrogenase reactions proceed with the additional incubation of 1.1 minutes at 37 °C. The absorbance is then measured at the first point. After removal of endogenous serum interferences in the first heating bath the decrease in the absorbance of the analytical stream (determined at 340 nm) is also measured at two other points. The results are compared by computer for linearity. If the slopes agree, the absorbance difference between point one and three is used to calculate the ALT activity.

3.5.4.10 **Serum gamma-glutamyltransferase (GGT)**[cp]

GGT is measured by a two-point rate reaction. The catalytic action of GGT can be measured according to the following reaction:

L-γ-glutamyl-σ-nitroanilide (GNA)+ glycylglycine $\xrightarrow{\text{GGT}}$ L-γ-glutamyl-glycylglycine + p-nitroaniline

GGT activity is directly proportional to the amount of p-nitroaniline produced.

A stream of GGT buffer is added to an air-segmented stream of GNA substrate. A prediluted serum sample is then added and after passing through a mixing coil, the analytical stream enters the first flowcell where the absorbance is measured at 410 nm. After an incubation period (four minutes) at 37 °C the analytical stream enters the second flowcell and the final absorbance measurement is made at 410 nm. The enzyme activity of the sample is proportional to the increase in absorbance between the two flowcells.

3.5.4.11 **Serum alkaline phosphatase (AP)**[cp]

The AP method is based on the enzymatic hydrolysis of p-nitrophenyl phosphate (PNPP). The serum sample is added to a stream of PNPP reagent. The reaction stream enters an incubator

where the following colour-producing reaction proceeds at a temperature of 37 °C and at a pH of 9.9:

PNPP (colourless) $\xrightarrow[Mg^{2+}]{\text{Alkaline phosphatase}}$ p-nitrophenol + H_3PO_4 + H_2O

Following incubation, the free p-nitrophenol is dialysed into a buffered AP recipient diluent. The dialysis is performed to separate the p-nitrophenol from the bile pigment bilirubin. Since bilirubin absorbs light at approximately the same wavelength as p-nitrophenol, the added absorbance of bilirubin would cause a positive error in the determination of AP-activity. The absorbance of the analytical stream is measured at 410 nm.

3.5.4.12 Total protein[cp]

The total protein method requires the use of two independent but interrelated channels: a sample channel and a blank channel. In the sample channel, the serum sample is added to an air-segmented stream of biuret reagent. During the ensuing reaction, the protein in the sample combines with copper in the biuret reagent to form a purple complex. Sodium potassium tartrate acts as a complexing agent, and potassium iodide prevents autoreduction.

In the blank channel, the sample is added to an air-segmented stream of total protein blank solution, containing all of the constituents of the biuret reagent except copper sulphate and sodium tartrate. These two reagents normally react with the protein in the sample to produce the final reaction colour. The absence of these reagents from the blank channel prevents any colour development in the channel. As a result the absorbance determined in the blank channel is predominantly due to endogenous serum pigments. The absorbance of each channel is determined at 550 nm. Blank subtraction is performed by differential colorimetry.

3.5.4.13 **Albumin**[cp]

A serum sample is added to bromocresol green buffer reagent to form the stable albumin-bromocresol green complex at pH 7. The analytical stream is mixed at ambient temperature for approximately one minute, and the absorbance is measured at 630 nm.

3.5.5 Full blood count

3.5.5.1 **Leucocyte count**[hm]

A dextran solution was added to EDTA-plasma. After standing for 45 minutes for sedimentation, centrifugation at low centrifugal force takes place; leucocytes are separated from erythrocytes and the supernatant. Sodium chloride is added to the leucocyte-containing sample and it is shock-treated with water to remove the erythrocytes still present. After 30 - 40 minutes at 4 °C, an isotonic reagent is added. After centrifugation the volume of leucocytes is used to count the number of cells per liter.

3.5.5.2 **Erythrocyte count**[hm]

The erythrocyte-containing sample obtained with centrifugation to separate the leucocytes, is suspended and washed in sodium chloride. The erythrocytes are then spectrophotometrically determined by determining haemoglobin with a glucose-6-phosphate solution.

3.5.5.3 **Other**

Mean corpuscular haemoglobin[hm] and mean corpuscular haemoglobin concentration[hm] were determined by the method discussed under haemoglobin.

Platelet count[hm], mean plasma volume[hm] and mean corpuscular volume[hm] were determined on a Technicon analyser by high and low

angle scattering.

3.6 STATISTICAL ANALYSIS

3.6.1 Phase 1

Nutrient analysis was done with the Dietary Manager® Programme of Programme Management (Scharf, 1989, Scharf, 1990). Statistical analysis of the nutrient intake data was done with the EPI INFO® package (Dean, 1986).

3.6.2 Phase 2

Significant differences ($p < 0.05$) between mean areas under the glucose curve, maximum glucose increase, decrease, and glycaemic indices were calculated with the Student-Newman-Keuls test (Snedecor & Cochran, 1976). In addition the significant effect of the first meal on the response to the second meal was examined with a non-parametric test (Wilcoxin's ranked test for matched pairs) and correlation coefficients determined with the Spearman non-parametrical test using the BMDP3D® programme (Hill, 1987). The relationship between fasting and t_o values and the maximum increment was examined by calculating Pearson correlation coefficients, using the SAS® package (SAS® User's Guide, 1985).

3.6.3 Phase 3

Statistical analysis of the anthropometrical and blood analysis data was done at the Medical Research Council in Pretoria with the BMPD® package (Hill, 1987). Probability values for differences between visits were calculated with the non-parametric Wilcoxin ranked test for matched pairs and probability values for differences between the control and test groups were calculated with the Mann-Whitney test. The Spearman test was used to determine significance of correlations between variables.

CHAPTER 4

EATING HABITS AND NUTRIENT INTAKES OF BLACK PATIENTS WITH DIABETES MELLITUS IN THE GA-RANKUWA AREA

4.1 SUMMARY

A pilot study was undertaken with the purpose of developing a food frequency questionnaire (FFQ). Thirty-one black insulin-dependent diabetes mellitus (IDDM) patients were involved in this study. Fourteen of these patients also completed a 24-hour dietary recall.

The results of the pilot study showed that a three-meal-per-day-pattern, constant during the week, but not during week-ends, was followed. The most popular food items were, in descending order of portion size, maize meal porridge, full-cream milk, carbonated diet cold drinks, bread, apples, oranges, beef and chicken. The mean energy intake was 9380 (SD = ± 3345) kJ for the men and 9054 (SD = ± 4488) kJ for the women. Protein provided 17.9 % of the total energy, fat 33.7 % and carbohydrate 47.7 %. Mean daily cholesterol intake was 398 (± 207) mg and mean dietary fibre intake 32 (± 12) g. Values obtained with the 24-hour recall were slightly lower than those obtained with the FFQ.

The FFQ was then used to determine food and nutrient intakes of 63 black, obese non-insulin-dependent diabetes mellitus (NIDDM) patients. Meal patterns were basically the same as those followed by the IDDM patients. The most popular food items were, in descending order of portion size, maize meal porridge, full-cream milk, bread, apples, oranges, beef, carbonated dietary cold drinks, tomato-and-onion sauce, cabbage and chicken. The mean energy intake was 11305 (± 3788) kJ for the men and 9064 (± 2939) kJ for the women. Protein provided 17 % of the total energy, fat 35 % and carbohydrate 48 %. The mean daily cholesterol intake was 347 (± 207) mg and mean dietary fibre intake 31.1 (± 11.6) g. The mean calcium intake was 922 (± 624) mg, iron 16,4 (± 5)

mg, thiamine 1.8 (± 0.59) mg and ascorbic acid 99 (± 65) mg. It became clear that the eating habits of these NIDDM patients were in a process of westernisation. Vitamin and mineral intakes were relatively low. Nutrition education and dietary counselling targeted at the restriction of energy, fat and animal protein intakes and higher intakes of legumes, fruit and vegetables, were recommended.

4.2 INTRODUCTION

Information on the incidence of both IDDM and NIDDM in Africa is scarce (Krolewski & Warram, 1985). However, there are indications that the incidence of NIDDM is increasing (Zimmet, 1982). The changing eating habits of the black population from a traditional rural diet to a westernised urban diet may play a role in the incidence of diabetes and other nutrition-related diseases (Silvis, 1989). It is, therefore, necessary to investigate these changes in order to anticipate problems that may arise in designing practical educational programmes to improve eating habits, to control the disease, and to implement a prevention strategy.

A pilot study was, therefore, done to design and standardise a FFQ which could be used in determining the eating habits of the black DM population in the area. Thereafter, the FFQ was used to determine the eating habits and nutrient intakes of NIDDM patients in order to compile a low-GI meal that they would comply to. The use of the low-GI meal to measure its post-prandial and long-term effects is discussed in Chapters 4 and 5.

The results of the pilot study that was undertaken among IDDM patients to design the FFQ and the results found with the implementation of the questionnaire among NIDDM patients, are reported and discussed separately in sections 4.4.1 and 4.4.2 because both studies gave valuable information on the food and nutrient intake of black IDDM and NIDDM patients respectively.

4.3 STUDY DESIGN, METHODS AND MATERIALS

Background information on the current eating habits of black diabetic patients was obtained in a pilot study. A semi-quantitative FFQ was designed and tested on a random sample of 31 IDDM patients who attended a diabetic clinic at the Department of Pharmacology of MEDUNSA.

4.3.1 Collection of questions

In order to draw up and standardise the questionnaire, interviews were held with ten black people who work at MEDUNSA and live in Ga-Rankuwa. A 24-hour dietary recall was obtained and the subjects were asked to compare this with their habitual intake. Then they were asked to name foods which they liked and disliked. They were also asked to name the ten foods which they use most often and to state reasons for their answers. Lastly they were asked to mention foods that they use on special days and on specific occasions.

A provisional questionnaire was then drawn up. The data in section 2.6 of this thesis were also used in the questionnaire. To test the validity and reliability of the questionnaire (Guilford & Fruchter, 1978; Kerlinger, 1986) it was used during interviews with ten patients from the NIDDM-clinic at Ga-Rankuwa hospital, and changes were made as more information became available. Plastic food models, as well as utensils and cutlery used at home were used to help the patients to remember what they had eaten, in order to save time and to determine the exact quantities of food items consumed (Bolland, Yuhas & Bolland, 1988; Hankin, 1986). Different visual aids were used because of the problems with visual aids (as discussed in section 2.8) (Boeijen, Brummer & Cramwinckel, 1985).

4.3.2 Composition of the questionnaire

The questions were then analysed and changed, added to or omitted to simplify, improve interpretation, and shorten the questionnaire (Guilford & Fruchter, 1978; Smit, 1983). The semi-

quantitative FFQ was then drawn up. The questionnaire was divided into three main sections. With the first section, basic background information was obtained to find out more about the population.

The second section contained the open question: "What do you usually eat ....." and the different times of the day were then filled in to obtain information on the habitual meal pattern of the patient. If it was too difficult for the patient to supply this information, he was asked to tell the interviewer what he had eaten the day before, to make it more understandable (Bailey, 1982). After the information had been supplied, the interviewer went through all the information again, to ask probing questions to help the patient to give information that he had forgotten (Dwyer, Krall & Coleman, 1987) and to fill in the amounts of food items that had been consumed.

The last section was a food frequency list divided into the following categories:

Milk and dairy products
Cereal and cereal products
Other vegetables
Fats and oils
Miscellaneous
Meat and protein dishes
Starchy vegetables
Fruit
Sugars and sweets
Beverages

The patients were asked to give information of their weekly consumption of the foods listed under the categories. Specific attention was given to food items which were found to have been consumed in large quantities, for example stiff maize meal porridge and food items that were unusual, but eaten regularly, for example chicken feet. Many examples were also mentioned in each category to help the patient remember. The plastic food models, utensils and cutlery, as mentioned above, were used again, as well as pictures of different food items pasted into a scrap-book (Weiss, Kien & Clark, 1988).

4.3.3 Evaluation of the questionnaire

This questionnaire was then used to interview 31 patients from the IDDM-clinic at the Department of Pharmacology of MEDUNSA. The patients were randomly chosen from all patients older than 15 years of age who visited the clinic, excluding the 20 already questioned. Fourteen of the 31 patients were females and their ages varied between 15 and 63 years. Insulin was used to control the patients reasonably well.

All the patients interviewed visited the clinic for longer than one year and had changed their eating habits upon being informed of their illness, and according to the diet counselling they received. However, diet counselling was not given on a regular intensive basis afterwards. The interviews took place on the days when the patients came for their regular monthly check-ups.

To serve as control or standardisation comparison (Kerlinger, 1986), 14 of the 31 patients were also asked to complete a 24-hour dietary recall during their visit the month after they had completed the FFQ. The 24-hour dietary recall is seen as a useful epidemiological tool for measuring food intake in groups or individuals (Frank et al., 1984; Morgan et al., 1987), although it gives lower mean estimates than the FFQ (Larkin et al., 1989) and does not include food items less frequently consumed (Karvetti & Knuts, 1985).

The consumption of the different food items, meal plans and nutrient contents of the diets were then analyased by computer, by using the Dietary Manager Programme of Programme Management (Scharf, 1989), based on the RIND Food Composition Tables (Gouws & Langenhoven, 1986).

Out of all the information obtained with this FFQ, a final semi-quantitative FFQ was then drawn up (see Appendix B).

4.3.4 Implementation of the questionniare that was designed in the pilot study

4.3.4.1 **Training of the interviewer**

An interviewer (a qualified home economist who teaches Food Science to black students) was trained to help the researcher with the completion of the questionnaires. Training of the interviewer can markedly improve the accuracy of questionnaires (Hankin 1986; Medlin & Skinner, 1988).

Firstly, a discussion was held on the reasons for obtaining the data, as well as on the characteristics of the local black population. The questionnaire was worked through then. Examples of food items that are usually eaten or may be forgotten were given, as well as general trade names that are used by the black population (for example "Rama" if they mean margarine and "Cremora" to ascertain whether they used milk or creamer). The use of the audiovisual aids (food models, household measure items such as jugs and spoons, and pictures in a scrap-book) was discussed, as well as preparation methods. Lastly portion sizes and amounts of food items consumed were discussed. General portion sizes as used by the local black population were shown, as well as standardised portion sizes as described by Langenhoven et al. (1986). Enough cues should be given to respondents to ensure that they remember as many of the consumed items as possible (Musgrave et al., 1989).

4.3.4.2 **Implementation of the questionnaire**

Sixty-three NIDDM patients at the clinic of Ga-Rankuwa were chosen to fill in the FFQ. The patients were randomly chosen from all the patients who visited the clinic on Mondays and Thursdays (the only two clinic days of the week), who had a BMI between 27 and 35 and who were between 40 and 65 years of age. It was decided to use patients with a BMI of 27 and 35 because most of the patients at the clinic are overweight and weight

could have an influence on the results of the different phases of the study and the effect of weight should therefore be limited. Most of the patients at the clinic are also between the ages of 40 and 65 and the study was therefore done on this age group to limit the effect of age. Patients who completed the questionnaire for the standardisation and had been interviewed during the first month were not asked again. To randomise, the name and number of every sixth patient that was entered into the attendance book for the specific day was written down and checked against the criteria mentioned above. If not suitable, the next patient was taken. The interviewer (either the trained interviewer or the researcher) then went to the patient and asked the patient whether he/she would be willing to participate. There were no patients who refused. Where language was a problem, a qualified black dietitian was asked to act as interpreter.

The interviews were held while the patients waited for their monthly medical checkup. A separate consulting room in the clinic was used to ensure privacy and to exclude interruptions. The interviewers first just talked to the patients to put them at ease, and then the purpose of the interview was discussed. Thereafter the same procedure was followed as with the IDDM-patients during the pilot study.

Special care was taken to get an objective overall picture (Bailey, 1982). As it was impossible to interview all the patients on one day, the questionnaires were completed through a period of one month. It was then found that the patients gave more information at the end of the month, after they had received their salaries, when they bought and ate more food than during the rest of the month. They did not eat the same on week days and weekend days (Medlin & Skinner, 1988).

The interviewers evaluated the questionnaires together, directly after the interviews, to ensure that the information obtained was complete, and to integrate the usual eating pattern, asked about

on the first page, into the food frequency section.

4.3.4.3 Analysis of the data

All the food items and the amounts eaten per week were written down and codes (according to the RIND Food Composition Tables (Gouws & Langenhoven, 1986)) and grams were filled in by the researcher to ensure consistency and accuracy (Frank et al., 1984). The data were then analysed by the updated computer programme mentioned before (Scharf, 1990), by entering all the food items as a meal and dividing this by seven to get the mean daily nutrient intake.

To determine the weight of the popular food item portions, the food items were prepared and portion sizes as described by the IDDM patients of the pilot study were dished up. These portions were then weighed. A summary of the results can be seen in Table 4.1. Weights of other portions were taken from the Food Quantities Manual of the RIND (Langenhoven et al., 1986).

The type and quantities of food were recorded on a large sheet of paper to work out frequency of use and mean portion sizes. This information was used to determine the general meal pattern and peculiarities of the diet of the local black population.

Table 4.1 Summary of weights of portions of food items commonly consumed in the Ga-Rankuwa area.

Bread	1 slice	60 g
Stiff porridge	1 mug	310 g
Rice	1 mug	230 g
Mashed potatoes	1 cup	230 g
Cheese	1 slice	30 g
Beef without bones	small hand palm	90 g
Beef with bones	small hand size	120 g
Chicken,fried	small breast	100 g
Boerewors	length of pen	80 g
Spinach, cooked	heaped big spoon	90 g
Cabbage, cooked	heaped big spoon	70 g
Beetroot salad	big spoon	50 g
Tomato-and-onion sauce	half a mug	120 g

4.4 RESULTS AND DISCUSSION

4.4.1 Design of the food frequency questionnaire (pilot study)

4.4.1.1 Background data of patients used in the pilot study

Background information on the IDDM patients who took part in the pilot study is given in Table 4.2. Information was obtained for the specific day on which the FFQ was filled in.

The mean weight of the women was more than that of the men. The mean BMI of the women was 30.0 kg/m² and that of the men 24.6 kg/m². The blood glucose values of these patients were not exceptionally high for subjects with DM. However, the higher HbA_{1c} values indicated that overall control was worse than expected. Blood pressure was normal in most cases - many of the patients took medication for high blood pressure and they are monitored carefully on a monthly basis.

Table 4.2 Background information on patients who took part in the pilot study

Mean values	Men (n=17)	±SD	Women (n=14)	±SD	Total (n=31)	±SD
Age (years)	32	12.9	42	13.8	35	13.6
Weight (kg)	71	15.6	73	19.5	72	16.4
Height (cm)	170	9.4	156	4.2	160	8.1
Blood pressure (mmHg)	127/80	13/12	131/80	34/13	128/80	20/12
Blood glucose (mmol/l)	10.1	4.7	8.3	5.6	9.6	4.9
HbA_{1c} (mmol/l)	13.2	3.9	12.7	4.8	13.6	4.1

4.4.1.2 Current eating habits of the IDDM patients

*** Meal pattern**

The patients interviewed in this study ate three meals a day instead of the traditional two. For breakfast most of them (88 %) ate soft porridge with milk and/or bread. The porridge was usually made from maize meal but sometimes from grain sorghum. Brown bread was usually eaten and in 60 % of the cases margarine was spread on the bread. Nobody seemed to drink coffee, while tea was very popular. Traditionally the black population do not differentiate between hot beverages - all are named tea (Kuzwayo, 1990), and that could be the case in this study too. A few patients ate bread and tea as a snack in the morning at tea-time.

For lunch approximately 45 % of the patients ate stiff maize meal porridge, meat (usually cooked lean beef or chicken without the skin) and one or more portions of cooked vegetables. Forty percent of the patients ate bread in various combinations with milk, cheese, eggs and tea. The rest of the patients ate

different combinations of the food items already mentioned.

For supper 59 % of the patients ate maize meal porridge with meat and two or more portions of vegetables. Some of the other patients ate porridge and meat only, or porridge and vegetables only, while 12 % of them ate bread and vegetables with or without fruit. Stewed tomato-and-onions was a favourite dish with the stiff porridge but the porridge was also eaten with meat gravy or on its one.

The eating pattern was very constant during the week but varied during week-ends. Patients who did not work on Saturdays had a more substantial breakfast (they usually included eggs), but roughly the same lunch and supper as during the week. The traditional hospitality of the blacks was still seen in the Sunday lunch where a large variety of foods had been available for everyone, including visitors (Crous and Borchardt, 1982). To some extent the traditional diet was followed for Sunday lunch, but rice or another starch was substituted for stiff porridge. On Sundays the breakfast might be smaller or non-existent to compensate for the more substantial lunch. Meat was served and a much wider variety of vegetables was included. Some also had dessert in the form of fruit or diabetic jelly and/or custard.

* **Intake of specific food items**

The consumption of the most popular food items as obtained by the food frequency questionnaire is summarised in Table 4.3.

Fresh full-cream milk was used by 71 % of the patients. Only seven patients used skimmed milk and 38 % used non-dairy creamers alone or supplementary to fresh full-cream milk. Cheddar and/or Gouda and/or in a few cases, Feta cheese, was used by 54 % of the patients. The mean intake of milk products (excluding the cheese) was 420 ml per day.

Table 4.3: Popular food item consumption of a group of IDDM patients obtained with a food frequency questionnaire

Food item	% of patients who regularly eat the item	Mean weekly intake (edible portion) Mean	±SD
Dairy products			
Fresh whole milk	71	1 579 ml	2374.7 ml
Cheese	54	69 g	82.2 g
Meat, etcetera			
Beef	100	489 g	405.7 g
Chicken	100	350 g	95.1 g
Fish	42	55 g	33.7 g
Eggs	100	250 g	155.3 g
Cereals			
Bread, brown	100	1 190 g	791.6 g
Maize meal porridge	97	3 717 g	3244.0 g
Rice	77	145 g	155.4 g
Vegetables			
Potatoes	79	299 g	231.6 g
Cabbage	79	226 g	200.5 g
Spinach	67	275 g	195.1 g
Tomatoes	67	196 g	266.9 g
Tom-and-onion sauce	68	353 g	155.3 g
Beetroot	63	84 g	149.3 g
Fruit			
Apples	94	706 g	730.6 g
Oranges	71	600 g	652.9 g
Bananas	46	108 g	188.8 g
Miscellaneous			
Diet cold drinks	84	1 518 ml	1766.8 ml
Margarine	87	51 g	49.9 g

An average of three to five types of meat was used during a week. Apart from beef, chicken and fish, 29 % of the patients ate

mutton. Meat was most often boiled. Traditionally offal (chicken feet or giblets or beef intestines or tripe) is eaten regularly (Kuzwayo, 1990), but in this study only two people mentioned offal in the form of tripe as part of their habitual meat intake. Processed meat (especially polony, vienna sausages and tinned meat), eaten on a regular basis by the black population (Kuzwayo, 1990), was only eaten by six of the patients in this study and only on an irregular basis. They all said that they tried to avoid these food items because of the high fat and salt content of most processed meat products.

Traditional protein sources such as locusts, termites, crickets and birds were not eaten, possibly due to unavailability and the fact that these are not well-known as food sources in the urban areas anymore (Du Plessis, 1963). Usually eggs had not been eaten by women and children in the traditional diet (Crous and Borchardt, 1982). They were, however, very popular in the diet of all the patients at the clinic and were eaten by everyone. The mean intake of eggs was five per week. Most patients (71 %) ate boiled eggs to avoid the use of fat in preparation. The eggs were eaten at breakfast, lunch, supper or in between as a snack. Baked beans that had been mentioned by other researchers as a popular item (Crous & Borchardt, 1984) were not very popular under these patients, and the same applied to dried beans and other legumes.

Only one patient did not eat maize meal porridge with an intake range of 55 g per week to 11 700 g. Items like rusks, scones, cakes and even cereals such as mealie rice and samp were not used regularly. Only seven people did not eat rice and nearly every= body mentioned that they only eat rice during week-ends and usually in the place of maize meal porridge. Everybody ate brown bread and only two people mentioned that they sometimes got white bread at work, but the intake varied from 147 g per week to 3650 g.

The mean weekly intake of raw vegetables was 180,4 g edible mass,

usually tomatoes, lettuce, cucumbers and carrots. The mean weekly intake of cooked vegetables was 730 g edible mass and a mean of five types of vegetables was eaten per week with one or two types per meal. The mean vegetable intake per day was 135 g edible mass. Potatoes were usually eaten in the cooked form but chips and mashed potatoes were also favourites. The vegetables mentioned by the patients as regularly consumed had been freely available during the time of the study and they all mentioned that their consumption varied according to the availability of the different vegetables. The consumption pattern may, therefore, differ during other seasons of the year.

Apples, oranges and bananas were the most popular fruits. Only two patients did not eat apples. Oranges were eaten by 71 % of the patients and bananas were eaten by 46 % of the patients, although almost everybody remarked that it was not a good fruit for a diabetic person to eat. Avocado pears were also popular and used by 33 %. Other kinds of fruit, dried fruit and canned fruit were not so popular. Apples, oranges and bananas had been freely available during the time of the study and usually are throughout the year in this area. Patients said that they did not eat other types of fruit on a regular basis, although during different seasons other varieties of fruit might also be consumed. Patients mentioned that they did not drink fruit juices regularly because of the high sugar content of the juices. The mean intake of fruit was 133,5 g per day.

Although most of the patients said that they used butter, it was clear that just one really used butter when pictures of different wrappers for margarine and butter were shown to them. Only four patients used no margarine at all, while only three people used poly-unsaturated margarine. Only a few patients (four) used the margarine on their bread; it was mainly used in the preparation of vegetables, cereals other than porridge, and meat. The mean fat intake from sources like margarine, oil, fat and butter was 50,5 g per day.

All the subjects denied the use of sugar; saccharine, that is regularly provided by the clinic, free of charge, was used instead. Half of the patients mentioned that they kept hard boiled sweets with them for times when they felt hypoglycaemic and all of them could correctly describe the symptoms of hypoglycaemia. Just two mentioned that they sometimes ate diabetic chocolates and another two patients mentioned the use of diabetic jams and low-fat ice cream.

Only a few patients mentioned the occasional use of miscellaneous items such as low-fat mayonnaise, tomato sauce and atchar.

With the exception of five people, all the patients drank carbonated diet cold drinks weekly. (The study was done from August to October. More cold drinks might be consumed during the hotter months). Only three patients admitted that they sometimes drank beer, but the three of them consumed an average of 1 800 ml of beer per week.

4.4.1.3 Nutrient intake of the IDDM patients according to the present diet

The mean nutrient distribution and intake of the patients who took part in the study are given in Table 4.4. It should be taken into consideration that the RIND Food Tables (Gouws & Langenhoven, 1986) were used and that some values, especially for minerals and vitamins, were not available and were considered as zero values.

The ranges and standard deviations for all nutrients are very wide. One of the reasons was that these patients did not receive intensive dietary counselling and eat more or less the same as the rest of the family. Other factors that might have played a role were the differences in age and sex (Goodhart & Shils, 1980), although if the data were stratified for differences in sex and age, it was only the total protein and vitamin C intake that differed much between the males and females, as can be seen

in Table 4.4. The fact that a food frequency list and other types of recall methods for the gathering of data on food consumption are just crude methods of obtaining information, may also play a role (Krall & Dwyer, 1987).

Differences in the energy-intake between men and women were small. The use of slimming diets for most of the women should be investigated if weight loss forms part of the treatment strategy. Most of the patients (20) had a cholesterol intake of more than 300 mg/day. The mean saturated fat content of the diet was 31 %. The P/S ratio was also low and counselling should, therefore, be aimed at the encouragement of the intake of plant protein products and the restriction of animal protein products. Only three of the male patients had a protein intake lower than the 1989 USA RDA's (Food and Nutrition Board, 1989). Dietary fibre intake was generally acceptable, although it could be increased (American Dietetic Association, 1988). For an ideal diabetic diet the ratio of energy contribution between carbohydrates, fat and protein can change from the present 48:34:18 towards approximately 60 % carbohydrates (mostly unrefined), less than 30 % fat and the rest protein (American Diabetes Association, 1990a).

The mean calcium intake compares well with the RDA, but eleven men and three women had a calcium intake lower than 800 mg. Eleven women and four men (48 %) had an iron intake lower than the RDA. Although the mean intake of magnesium is far above the RDA, seven men had lower intakes. Nine men and five women (45 %) had lower zinc intakes than the RDA.

The mean vitamin A intake was relatively high, but much of it was from vegetable carotene sources like spinach, that are biologically less available and not toxic (Goodhart & Shils, 1980). The standard deviation was also large and eight men and four women had intakes lower than the RDA. Vitamin D intake was very low - all the patients had intakes below the RDA. Missing values in the Food Composition Tables may, however, play an

Table 4.4 Mean energy distribution and nutrient intake of a group of 31 IDDM patients

NUTRIENT	FOOD FREQUENCY QUESTIONNAIRE Men Mean (n = 18)	SD	Women Mean (n = 13)	SD	Total Mean (n = 31)	SD	24-HOUR RECALL Total Mean (n = 14)	SD	RDA 25 - 50 YEARS Men	Women
Energy (kJ)	9379.6	3345.0	9053.6	4487.7	9306.2	3553.2	8680.3	2969.6	12200.0	9200.0
Protein %	18.2	3.2	17.0	3.5	17.9	3.3	19.7	4.5		
Fat %	33.6	11.2	34.0	11.3	33.7	11.0	33.3	12.0		
Carbohydrate %	47.5	11.1	48.7	8.7	47.7	10.5	46.5	10.1		
Total prot. (g)	104.7	45.7	87.2	38.9	100.7	44.3	98.3	25.1	63.0	50.0
Animal prot.(g)	62.8	33.7	52.2	25.7	60.4	32.0	63.7	20.2		
Plant prot. (g)	42.2	21.3	34.9	15.4	40.5	20.1	34.2	11.0		
Total fat (g)	84.8	45.4	87.4	57.7	85.4	47.4	82.3	55.4		
P/S ratio	0.57		0.61		0.58		0.41			
Chol. (mg)	407.3	219.4	364.4	164.7	397.6	206.6	300.5	198.7		
Total CHO (g)	257.8	93.1	252.9	116.8	256.7	96.9	230.5	72.7		
DF (g)	31.6	12.3	30.8	13.7	31.5	12.4	25.6	7.2		
Calcium (mg)	806.5	517.7	844.9	506.2	815.1	507.0	471.3	285.1	800.0	800.0
Iron (mg)	17.1	6.5	13.9	5.5	16.4	6.3	16.4	3.9	10.0	15.0
Magnesium (mg)	440.5	195.9	365.0	169.7	423.5	190.3	381.0	110.9	350.0	280.0
Phosphorus (mg)	1515.5	652.9	1349.1	623.3	1478.0	639.9	1203.3	341.6	800.0	800.0
Potassium (mg)	3288.9	1267.6	3444.9	1806.6	3324.1	1374.4	2753.7	1073.5		
Sodium (mg)	1897.4	834.0	2044.9	1236.6	1930.7	918.2	1474.1	478.5		
Zinc (mg)	16.4	7.4	13.4	6.4	15.7	7.2	16.8	6.2	15.0	12.0
Copper (mg)	1.67	0.92	1.50	0.71	1.64	0.87	1.23	0.35		
Vitamin A (RE)	2014.6	2128.6	2321.7	1459.0	2084.0	1979.0	1391.3	1452.4	1000.0	800.0
Vitamin D (ug)	1.47	0.95	1.83	1.44	1.55	1.06	0.90	0.78	5.00	5.00
Vitamin E (mg)	12.8	6.7	17.0	17.9	13.8	10.1	9.9	7.2	10.0	8.0
Thiamine (mg)	1.66	0.66	1.66	0.84	1.74	0.69	1.53	0.48	1.50	1.10
Riboflavin (mg)	1.83	0.86	1.77	0.78	1.82	0.83	1.46	0.50	1.70	1.30
Niacin (mg)	22.2	12.4	17.0	7.4	21.1	11.5	18.9	6.8	19.0	15.0
Vit. B-6 (mg)	1.57	0.75	1.53	0.75	1.56	0.74	1.22	0.47	2.00	1.60
Folacin (ug)	316.7	174.6	293.0	155.8	311.4	168.3	230.9	110.2	200.0	180.0
Vit. B-12 (ug)	5.5	7.3	7.9	5.3	6.0	6.9	3.5	1.9	2.0	2.0
Pan. acid (mg)	5.3	2.4	5.1	2.0	5.3	2.3	3.7	1.9		
Biotin (ug)	29.2	13.9	29.0	9.7	29.1	12.9	17.3	14.9		
Vit. C (mg)	101.6	69.8	175.6	138.4	118.3	92.5	45.9	32.2	60.0	60.0

* RDA = Recommended daily allowances (National Research Council, 1989), RADA = Recommendations of the American Diabetic Association (American Diabetes Association, 1990).

Prot = Protein
P/S ratio = Polyunsaturated to saturated fatty acid ratio
Chol = Cholesterol
CHO = Carbohydrates
DF = Dietary fibre
Vit = Vitamin
Pan = Pantothenic acid

important role, as discussed previously. Although the mean intake of vitamin E was adequate, 39 % of the patients had intakes lower than the RDA.

The mean thiamin intake was quite high, but 35 % (seven men and four women) had intakes lower than the RDA. The same applied to nicotinic acid (48 % had lower intakes), folic acid (six men and two women had lower intakes) and ascorbic acid where five men and three women had lower intakes. The mean vitamin B-6 intake was lower than the RDA and 15 men and 9 women (77 %) had lower intakes.

The relatively low vitamin intake could be caused by the low intake of vegetables and fruit (see Table 4.4). Counselling should, therefore be aimed at promoting the intake of vegetables and fruit, especially in the raw form, to increase the dietary fibre intake simultaneously.

The 24-hour recall information compared well with the FFQ information. In most cases the values of the 24-hour recall were lower but not significant as was also found by other researchers such as Morgan et al. (1987). In the case of calcium and vitamin C the 24-hour recall values are much lower. This may be atributed to the fact that milk as such, cheese and fruit are not used daily and were therefore not reported as often on a daily basis as on a weekly basis. However, it can be concluded that the FFQ used in this study measured the food intake of the specific patients well enough, if not better, when compared to other possible measuring instruments as discussed in section 2.8.

From the results obtained with the FFQ, it could, therefore, be concluded that the questionnaire designed in this pilot study is a valid and reliable method of obtaining food intake data from black DM patients in this area.

4.4.2 Implementation of the food frequency questionnaire in black NIDDM subjects

4.4.2.1 Introduction

In order to design a low-GI diet that could be followed by the patients who took part in the second and third phase of the study, as well as other NIDDM patients in the Ga-Rankuwa area, information on their current diet was necessary. The low-GI diet could then be based on current eating patterns and well-known and liked food items to ensure compliance. Knowledge of the current diet could also help to explain baseline values for markers of risk factors of complications of the disease. In addition, it could be used in a motivation strategy for following the low-GI diet and used in future nutrition education and counselling.

It was, therefore, necessary to obtain information on the current diet of NIDDM patients with the help of the above-mentioned FFQ.

4.4.2.2 Background data of patients who completed the food frequency questionnaire

Background information on the 63 NIDDM subjects who were randomly selected to complete the questionnaire is given in Table 4.5.

Glycated haemoglobin values were not available. All patients were overweight or obese. The fasting blood glucose values were relatively high, which may be an indication of poor control in these patients. All the patients had a few short nutrition education sessions with a dietitian during previous years but not during this specific year, and none had had any intensive nutrition education and counselling. All were treated with oral hypoglycaemic agents (mostly metformin and glybenclamide in combination) and some took anti-hypertensive drugs.

Table 4.5 Mean background information of NIDDM subjects in the Ga-Rankuwa area

	Men (n=21)	Women (n=42)	Total (n=63)
Age (years)	52.2	54.4	53.5
Height (cm)	169.5	155.8	160.2
Weight (kg)	82.6	81.7	81.9
BMI (kg/m^2)	28.7	33.6	32.0
WHR	0.95	0.86	0.89
Blood glucose (mmol/l)	10.55	11.72	11.34

4.4.2.3 Current eating habits of NIDDM patients

*** Meal pattern**

The NIDDM patients had basically the same meal pattern as those interviewed in the pilot study. Most of them ate three meals a day instead of the traditional two. Breakfast was usually a cup of tea or coffee early in the morning (before departure for work) and then a more substantial breakfast at work at about 10h00. Many ate soft porridge with milk, but others ate bread. The porridge was usually made from maize meal. A few used grain sorghum or oats. Sour milk was popular with the porridge.

During the day, brown bread was eaten in all cases and in 84 % of the cases margarine was spread on the bread. In contrast with the group in the pilot study, tea and coffee were mentioned as drinks with breakfast and during the day and far fewer cold drinks were consumed. Originally, milk was not popular in the traditional African diet (Kirsten, 1977) and a high percentage of the black population have lactose intolerance and could therefore not tolerate large quantities of milk (Walker & Walker, 1984). In this study, the patients used milk frequently.

However, milk is cheaply and abundantly available in this area.

Lunch was basically the same as that of the IDDM patients but more (70 %) ate stiff maize meal porridge. The other patients ate bread in various combinations with milk, cheese, eggs and a drink. Many mentioned that they got lunch at work. In contrast with the patients in the pilot study, four patients often bought meat pies, chips or processed meat and fruit for lunch and did not know that the fat-content of these products was high, or that fat-intake should be restricted.

For supper, 65 % of the patients ate the same meal as with lunch, but more mentioned vegetables as part of the meal. Stewed tomato-and-onions with added oil was a favourite dish with the stiff porridge. A vegetables-and-meat-stew was also popular as a relish with the porridge. Meat was eaten more often in the evening.

Fruit, cheese, peanuts and cold drinks are not seen as food but snacks and used in-between. The patients could often not remember accurately how many of these items they had consumed, while some patients mentioned up to seven fruit portions per day.

The eating pattern was fairly constant during the week but varied during week-ends. A more substantial breakfast was eaten on Saturdays (they usually included eggs), but roughly the same lunch and supper as during the week. Fish and chips and half-*zwi* (bread with a type of meat-filling) are popular items for Saturday lunches. The Sunday meal pattern and lunches were mainly the same as those reported by the IDDM patients. Chicken was most often (80 %) mentioned as the meat for Sunday lunch and beetroot and/or green beans were very often (63 %) part of the vegetables. Only a few had dessert in the form of fruit or jelly and/or custard or ice cream.

*** Intake of specific food items**

The consumption of the most popular food items, as obtained by the food frequency questionnaire, is summarised in Table 4.6.

It was clear that the NIDDM patients had much less nutrition education than the IDDM patients. Although all of them were overweight, they used many more items containing sugar and high in fat and did not even mention that they knew it was not recommended, as was done by the IDDM patients.

Table 4.6: Popular food item consumption of a group of NIDDM patients in the Ga-Rankuwa area.

Food item	% of patients	Mean weekly intake (edible portion)			
		Men (n = 21)	Women (n = 42)	Total (n = 63)	
		Mean	Mean	Mean	±SD
Dairy products					
Fresh whole milk	95	3377 ml	1949 ml	2473 ml	2341 ml
Skim milk	6	0 ml	582 ml		1823 ml
Cheese	62	23 g	71 g	54 g	83 g
Meat, etc.					
Beef, stew	68	386 g	142 g	232 g	263 g
Beef, fried	22	163 g	41 g	86 g	215 g
Chicken, boiled	59	219 g	183 g	196 g	260 g
Chicken, fried	52	176 g	122 g	142 g	210 g
Fish, fried	56	188 g	72 g	115 g	202 g
Fish, canned	27	26 g	27 g	27 g	52 g
Boerewors	62	108 g	102 g	104 g	148 g
Minced meat	49	130 g	51 g	80 g	109 g
Eggs, boiled	55	154 g	76 g	105 g	139 g
Eggs, fried	65	59 g	32 g	42 g	93 g
Cereals					
Bread, brown	100	1156 g	1202 g	1184 g	770 g
Maize meal porridge	100	6314 g	5131 g	5564 g	2749 g

Table 4.6 (continued)

Food item	% of patients	Mean weekly intake (edible portion) Men Mean	Women Mean	Total Mean	±SD
Rice	90	335 g	149 g	217 g	228 g
Vegetables					
Potatoes, mashed	71	153 g	138 g	144 g	152 g
Potatoes, cooked	59	276 g	131 g	184 g	253 g
Cabbage	84	147 g	274 g	227 g	209 g
Carrots	11	102 g	111 g	103 g	84 g
Spinach	78	208 g	207 g	207 g	212 g
Tomatoes	40	45 g	94 g	76 g	130 g
Tom-and-onion sauce	60	385 g	195 g	255 g	280 g
Beetroot	56	32 g	31 g	31 g	40 g
Green beans	59	80 g	67 g	72 g	92 g
Pumpkin	65	92 g	103 g	99 g	133 g
Mealies	40	63 g	47 g	53 g	92 g
Mixed salad	30	40 g	92 g	73 g	231 g
Fruit					
Apples	87	577 g	409 g	471 g	449 g
Oranges	84	447 g	655	579 g	644 g
Bananas	49	218 g	92 g	138 g	267 g
Miscellaneous					
Diet cold drinks	56	320 ml	430 ml	375 ml	238 ml
Margarine, hard	68	78 g	130 g	111 g	110 g
Margarine, soft	16	79 g	13 g	37 g	105 g

Fresh full-cream milk was used by nearly all the patients (95 %) with a range from 500 to 8500 ml per week. The four patients who used skimmed milk, used it because of availability and not because of the lower fat content. Only two patients used non-dairy creamers. Cheddar and/or Gouda were used by 62 % of the patients. The mean intake of milk products (excluding the cheese) was 440 ml per day.

Beef and chicken were the most popular types of meat. Meat was eaten daily by 78 % of the patients. Most patients did not cut off fat from the meat or remove the skin of the chicken. Minced meat (usually stewed with oil, tomato and onions) and mutton were also popular. Meat was either cooked or fried. Twelve patients mentioned the use of tripe and six mentioned the use of liver. Processed meat (especially polony) was only eaten by eight of the patients in this study and only on an irregular basis because of economy. Eggs were very popular. Seventy-four percent of the patients at the clinic ate eggs on a weekly basis. The mean intake of eggs was three per week. Fried eggs were more popular than boiled eggs. Baked beans (used by 13 people only with a mean intake of 30 g per week) were not very popular, and the same applied to dried beans and other legumes (used by 16 patients, usually in soup, with a mean intake of 58 g per week). Only 19 patients used peanut butter on a regular basis and twelve patients ate peanuts regularly.

All the patients ate maize meal porridge, with an intake range of 400 to 13 250 g per week. Only six people did not eat rice, but most patients only ate rice during week-ends and usually in the place of maize meal porridge. Everybody ate brown bread and the intake varied from 210 g per week to 3360 g. Rusks, biscuits (usually Marie®), and vetkoek were eaten by a few patients.

Only small portions of vegetables were eaten. The mean weekly intake of raw vegetables was 122 g edible mass, usually tomatoes and carrots. The mean weekly intake of cooked vegetables was 743 g edible mass and a mean of three types of vegetables was eaten per week with one or two types per meal. Vegetables mostly mentioned were cabbage, spinach/marogo, mealies and pumpkin. The mean vegetable intake per day was 122 g edible mass. Vegetables were mostly used as part of a stew, or cooked with onions and/or potatoes and/or tomatoes. Nearly half mentioned that they often added fats and/or sugar to the vegetables. A few patients restricted salt-intake because of high blood pressure. Potatoes were usually eaten in the cooked form with other vegetables, as

mentioned above, but chips and potato salad were also eaten. Mashed potatoes were a favourite. The vegetables mentioned by the patients as regularly consumed had been freely available during the time of the study and they all mentioned that their consumption varied according to the availability of the different vegetables. The consumption pattern may, therefore, differ during other seasons of the year.

Only eight patients did not eat apples. Oranges were eaten by 84 % of the patients and bananas were eaten by 49 % of the patients. Apples, oranges and bananas are usually freely available in this area throughout the year, although during different seasons other varieties of fruit might also be consumed regularly. Pawpaws, naartjies and guavas were specifically mentioned as fruit that are used in season. The mean intake of fruit was 180 g or one fruit per day.

Hard margarine was the fat that was most often mentioned but sunflower oil was also often used. The fat was used as a spread on bread, as well as in the preparation of vegetables, cereals other than porridge, and meat. Only a few mentioned that they used the drippings from meat as a fat source. The mean fat intake from visible fat sources was only 23 g per day.

Some subjects mentioned that they sometimes used sugar, but none used it on a regular basis; saccharine was used instead. Only a few patients (four) kept hard boiled sweets with them for times when they feel hypoglycaemic, but these patients had a mean sweet intake of 70 g per week. Diabetic chocolate was sometimes eaten by one patient and another three patients mentioned the use of diabetic jams and low-fat ice cream.

Fifty-six percent of the patients drank carbonated diet cold drinks weekly. (The study was done from May to October. More cold drinks might be consumed during the hotter months). Only six patients admitted that they sometimes drank beer, but between them, they consumed an average of 3520 ml of beer per week. Two

patients mentioned that they drink whisky on a weekly basis.

4.4.2.4 Nutrient intake of the NIDDM patients according to the present diet

The mean nutrient distribution and intake of the patients who took part in the study, are given in Table 4.7.

The macro-nutrient content of the diets of the men and women did not differ much, but there were larger differences in the micro-nutrient content.

Differences in the energy-intake between RDA and those of the men as well as the women were small, but the ranges were wide (2889 to 22453 kJ). However, the use of slimming diets for most of the patients should be investigated if the BMI of the patients is taken into consideration.

Many patients (31) had a cholesterol intake of more than 300 mg/day. The P/S ratio was also low and there were no large differences between the men and the women. The low P/S ratio is reflected in fatty acid composition of the diets. The intake of the C16:0 (palmitic acid) and C18:0 (stearic acid) fatty acids were high. These long-chain fatty acids are more abundant in the animal protein sources and restriction of these sources could be recommended. The mean intake of linoleic acid (C18:2) was approximately eight percent, which should be sufficient, according to Kinsella (1986a).

Only one of the males and four females had a protein intake lower than the 1989 USA RDA's (Food and Nutrition Board, 1989). The essential amino acid patterns of all the patients showed that the correct type of protein sources were used. The essential amino acids are supplied well above the minimum recommendations, and the restriction of animal protein sources in favour of plant protein sources should, therefore, not cause a deficiency in the intake of some of the essential amino acids.

Table 4.7 Mean nutrient intake of the NIDDM patients at Ga-Rankuwa Hospital outpatient clinic compared to the RDA and RADA*

		Men Mean n = 21	SD	Women Mean n = 42	SD	Total Mean n = 63	SD	RDA 24-50 Men	RADA* years Women
Energy	(kJ)	11305	3788	9064	2939	9787	3375	12200	9250
Protein	(% of E)	18	3.8	17	2.9	17.00	3.2		
	Total (g)	119.37	49.5	89.41	29.0	99.10	39.1	63	46
	Plant (g)	41.5	11.8	36.0	12.2	37.80	12.2		
	Animal(g)	77.3	44.6	53.1	25.2	60.90	34.3		
	g/kg	1.50		1.07		1.21		0.8	0.8
Fat	(% of E)	35	8.0	35	7.7	35.00	7.7	<30	<30
	Total (g)	107.3	50.3	84.4	37.8	91.80	43.2		
	% Sat	30	5.5	30	4.8	30	5.0		
	% MU	34	3.8	36	5.4	34	4.9		
	% PU	23	5.5	21	6.5	22	6.2		
	P/S ratio	0.77		0.70		0.73		1	1
	Chol (mg)	408	210.7	317	201.9	347	207.4	<300	<300
Carbohydrates									
	(% of E)	47	7.5	47	8.5	48.00	8.2	55	55
	Total (g)	304.3	88.7	256.3	88.0	271.6	90.4		
	Fibre (g)	33.9	10.5	29.7	12.0	31.1	11.6	40	40
	Sugar (g)	4.5		4.6		4.55			
Minerals	Ca (mg)	1065	966.1	854	363.1	922	623.9	800	800
	Fe (mg)	18.5	5.0	15.4	4.6	16.4	5.0	10	15
	Mg (mg)	481.2	132.0	410.0	120.0	433.3	127.3	350	280
	P (mg)	1802	791.1	1401	423.8	1530	592.8	800	800
	K (mg)	3789	1673.3	3033	889.2	3277	1237.0	2000	2000
	Na (mg)	2242	796.2	1708	720.6	1880	780.8	750	750
	Zn (mg)	18.8	7.1	14	4.6	15.60	5.9	15	12
	Cu (mg)	1.76	0.65	1.45	0.65	1.56	0.66	2.25	2.25
	Se (mg)	25.6	12.4	8.8	3.6	18.0	12.6		
Vitamins	A (RE)	2561	1975.5	1877	1626.2	2179	1750.3	1000	800
	D (ug)	3.05	6.05	2.19	2.2	2.47	3.8	5	5
	E (mg)	25.2	17.7	15.8	9.7	18.8	13.4	10	8
	Thiam(mg)	2.03	0.65	1.69	0.53	1.80	0.59	1.5	1.1
	Ribof(mg)	2.29	1.33	1.74	0.6	1.95	0.96	1.7	1.3
	Niac (mg)	23.02	9.79	17.80	7.2	19.45	8.4	19	15
	B-6 (mg)	1.84	0.67	1.37	0.55	1.52	0.63	2	1.6
	Folac(mg)	306.4	112.6	273.0	135.2	283.7	128.4	200	180
	B-12 (ug)	11.1	12.2	6.5	6.1	8.0	8.7	2	2
	Panto(mg)	6.14	3.3	4.53	1.7	5.04	2.42	5.5	5.5
	Biot (ug)	32.45	20.98	23.0	10.7	26.08	15.3	65	65
	C (mg)	103	70.9	97	62.9	99	65.0	60	60
Amino acids (g)									
Histidine		3.58	1.93	2.26	0.59	2.98	1.57		
Isoleucine		5.82	2.86	3.78	0.92	4.89	2.36		
Leucine		10.27	5.06	6.78	1.43	8.68	4.11		
Lysine		8.95	5.09	5.52	1.72	7.39	4.17		
Metionine		2.95	1.59	1.84	0.44	2.45	1.29		
Phenylalanine		5.33	2.62	3.52	0.74	4.51	2.13		
Threonine		5.18	2.77	3.22	0.70	4.29	2.25		
Tryptophan		1.53	0.75	1.10	0.41	1.34	0.63		
Valine		5.82	2.93	3.64	1.10	4.83	2.47		
Arginine		6.72	3.81	4.36	1.04	5.65	3.03		
Fatty acids (g)									
	C4:0	0.63	0.45	0.48	0.22	0.56	0.36		
	C6:0	0.37	0.27	0.20	0.07	0.29	0.21		
	C8:0	0.25	0.14	0.16	0.05	0.21	0.11		
	C10:0	0.48	0.30	0.34	0.13	0.42	0.24		
	C12:0	0.72	0.46	0.40	0.10	0.57	0.37		
	C14:0	3.03	1.39	2.26	1.13	2.68	1.28		
	C16:0	17.52	9.64	14.98	4.58	16.36	7.52		
	C18:0	8.73	6.18	7.00	2.78	7.95	4.80		
	C20:0	0.13	0.14	0.24	0.17	0.18	0.15		
	C22:0	0.23	0.23	0.36	0.29	0.29	0.25		
	C24:0	0.08	0.10	0.12	0.13	0.10	0.11		
	C14:1	0.43	0.24	0.28	0.20	0.36	0.23		
	C16:1	2.07	1.07	1.58	0.86	1.85	0.97		
	C18:1	30.63	20.16	30.36	8.63	30.51	15.26		
	C20:1	0.17	0.14	0.18	0.08	0.17	0.11		
	C22:1	0.02	0.04	0.02	0.04	0.02	0.04		
	C18:2	22.12	19.27	17.38	8.77	19.96	14.92		
	C18:3	0.95	0.57	0.64	0.44	0.81	0.51		
	C18:4	0.00	0.00	0.00	0.00	0.00	0.00		
	C20:3	0.05	0.05	0.00	0.00	0.03	0.05		
	C20:4	0.23	0.12	0.16	0.09	0.20	0.11		
	C20:5	0.07	0.08	0.04	0.05	0.05	0.07		
	C22:5	0.03	0.05	0.00	0.00	0.02	0.04		
	C22:6	0.20	0.25	0.08	0.11	0.15	0.20		

* RDA = Recommended daily allowances (National Research Council, 1989), RADA = Recommendations of the American Diabetic Association (American Diabetes Association, 1990).

Ribof = Riboflavin
Niac = Niacin
Folac = Folacin
Panto = Pantothenic aci
Biot = Biotin

% of Energy = percentage of energy
Sat = Saturated fatty acids
MU = Mono-unsaturated fatty acids
PU = Polyunsaturated fatty acids
P/S ratio = Polyunsaturated to saturated fatty acid ratio
Chol = Cholesterol
Thiam = Thiamin

Counselling should, therefore, in the case of the NIDDM patients too, be aimed at the encouragement of the intake of plant protein products and the restriction of animal protein products as also mentioned by Erdman and Fordyce (1989). Dietary fibre intake was generally good and in one case very high (83.1 g). Therefore, the mean intake should be increased, because only 10 patients had an intake of 40 g or more as recommended by the American Diabetes Association (1990a). For an ideal diabetic diet the ratio of the energy distribution between the carbohydrates, fat and protein can change from the present 48:35:17 towards approximately 60 % of energy from carbohydrates (mostly unrefined), less than 30 % from fat and the rest from protein (American Diabetes Association, 1990a). The lower carbohydrate intake and higher fat intake show, as previously reported for black South Africans (Kruger, 1987), that these patients are in the process of westernisation. They are moving away from their traditional high carbohydrate intake (Vorster et al., 1990) to the high fat intake of the western diet.

The mean calcium intake compares well with the RDA, but 54 % of the patients, eleven men and twenty-three women, had a calcium intake lower than 800 mg. McCarthy & Rubin (1984) found a possible increased risk for osteoporosis in diabetic patients. The calcium intake and prevalence of osteoporosis among these black NIDDM patients, therefore, warrant further investigation. All the men had adequate iron intakes, but 50 % of the women had intakes lower than the RDA, with one women with an intake of only 6.4 mg. Although the mean intake of magnesium is far above the RDA, 38 % of the patients had lower intakes. Magnesium deficiency has been linked to various complications that are often present in diabetics (Joffres, Reed & Yano, 1987) and the patients should therefore be encouraged to prevent magnesium deficiency. The range for phosphorus was from 503 to 4682 mg and for potassium from 1014 to 8865 mg. A few patients tried to restrict sodium-intake and the range were from 341 to 4268 mg. Eight men and thirteen women (33 %) of all subjects had lower zinc intakes than the RDA. Zinc deficiency may enhance glucose

intolerance (Mooradian & Morley, 1987) and the patients should therefore be encouraged to make use of good sources of zinc. The intake for copper varied from 0.3 to 6.1 mg, 86 % of the women and 90 % of the men had an intake under the recommended minimum safe intake. The copper content of most of the items in the Food Composition Tables that were used are not given and these missing values were registered as zero intakes. It would be reasonable to accept that the copper intake is in reality not lower than the intake of the other nutrients, given the composition of the diet.

The mean vitamin A intake was relatively high, but much of it was from vegetable carotene sources like spinach. The standard deviation was also large and the intake ranged from only 133 RE to 8416 RE. Three men and eight women had intakes lower than the RDA. The vitamin D intake was very low - only five patients had intakes above the RDA. Missing values in the Food Composition Tables may, however, have played an important role, as discussed previously. These patients often get sunlight on their skins. An apparently low vitamin D intake is, therefore, not of great concern. The mean intake of vitamin E was adequate, and only 11 % of the patients had intakes lower than the RDA.

The mean thiamin intake was high, and only six patients had intakes lower than the RDA. The mean riboflavin intake was adequate for both groups but nearly 30 % of the patients had lower intakes than the RDA. The same applies to nicotinic acid where 35 % of the patients had intakes lower than the RDA. The mean vitamin B-6 was lower than the RDA and eight men and thirty women (60 %) had lower intakes. This may cause problems and more research on the influence of DM on vitamin B-6 values are necessary, as discussed in Chapter 2. Low intakes, together with a higher need, may accelerate the start of diabetic complications and although some researchers feel that supplementation is not necessary (Mooradian & Morley, 1987), it may be needed in this case. Fourteen percent of the patients had folic acid intakes lower than the RDA and 13 % had lower vitamin B-12 intakes. The mean pantothenic acid intake of the women is lower than the RDA

and 74 % of them had lower intakes, compared to the 43 % of the men. Biotin intake was also low - all the women had intakes lower than the RDA and only two men had intakes above the RDA. The mean ascorbic acid intake was good, but 33 % of the women had intakes below the RDA. NIDDM subjects may have a higher turnover of ascorbic acid (Mooradian & Morley, 1987) and the intake of fruit, especially those rich in ascorbic acid, should be encouraged.

4.5 CONCLUSION

The nutrient analysis and energy distribution of their diet showed that both the black IDDM and NIDDM patients were moving towards a more westernised diet with a high fat content and a lower carbohydrate intake and they should be encouraged to increase their intake of more complex carbohydrates, especially in the form of legumes, fruit and vegetables and to restrict their fat intake. Although maize meal porridge was still the staple food, a refined maize meal was used.

The meal pattern and food items used by both groups were very constant and did not differ much between patients. However, amounts of food items consumed do differ largely. Although energy intakes appear to be a little lower than the RDA, all the NIDDM patients and the women in the IDDM group were overweight and energy restriction should be recommended for them. It is also possible that the patients did not mention everything that had been eaten. The FFQ is, like many other dietary data gathering methods, only a crude method and may not always reflect the full extent of dietary intakes.

The mean intakes of the micronutrients were sufficient in many cases, but many patients had intakes below the RDA. The relatively low vitamin and mineral intake compared to the energy intake could be caused by the low intake of vegetables and fruit (see Tables 4.4 and 4.7 and discussions). Therefore, although total energy intake may look sufficient, the wrong types of foods

were eaten.

More nutrition education and dietary counselling aimed at reducing energy and fat intake and increasing carbohydrate intake, reducing animal protein intake and increasing legume, vegetable and fruit intake are also suggested among this group of patients. A weight-monitoring programme and necessary guidance for obtaining the correct weight should also be introduced.

CHAPTER 5

GLYCAEMIC INDEX AND SECOND MEAL EFFECT OF A TRADITIONAL AFRICAN MEAL IN BLACK NON-INSULIN-DEPENDENT DIABETIC SUBJECTS

5.1 SUMMARY

The glycaemic index (GI) and second meal response (SMR) of a traditional African meal, consisting of maize meal porridge, soya mince and "morogo" with tea, were measured in 14 (7 men and 7 women) black NIDDM subjects using the standard reference meal of white bread plus tea. The GI of the traditional meal was significantly lower in the women than in the men and resembled predicted values based on published GI values of individual foods obtained in healthy subjects in the women. All areas under the glucose response curves were consistently lower after the second meal which was given three hours after the first meal. The effect of the test meal as first meal on the mean percentage reduction in area of the reference meal as second meal was, however, substantially higher in the women. Conclusions were that:

- the GI of a mixed meal in female black NIDDM subjects can be predicted from the GI's of the individual foods;
- a true Staub-Traugott effect is present in NIDDM patients of both sexes if the second carbohydrate load is taken within three hours after the first load; and
- a SMR is only observed when the GI of the first meal is low.

The reasons for the differences in response in the men and women should be further examined.

5.2 INTRODUCTION

The prevalence of NIDDM amongst black Americans is higher than in white Americans (Krolewski & Warram, 1985). The number of black patients attending diabetes clinics in several large hospitals throughout South Africa indicates that the incidence of the disease is increasing among South African blacks. The

current diabetic diet recommended for optimal metabolic control and prevention of the long-term complications of the disease (American Diabetes Association, 1990a; and as discussed in section 2.4) is similar in energy distribution and nutrient content to the traditional diet which has been eaten by rural blacks in the past (Lubbe, 1971). It has recently been suggested that westernised blacks suffering from NIDDM should revert to their traditional diet (Silvis, 1989), which will ensure increased intakes of complex carbohydrate and DF and decreased intakes of fat. In Australia it was found that when Aborigines reverted to their traditional diet and life-style, diabetic control improved markedly (O'Dea, 1984; Thorburn, Brand & Truswell, 1987).

Before the traditional African diet can be advocated for use as a therapeutic diet in black diabetics, both its short-term (acute), and long-term (chronic) effects on diabetic control should be evaluated. The purpose of the present study was to examine the short-term effects of a traditional African meal on post-prandial blood glucose responses in 14 black NIDDM patients.

The influence of a food or a meal on short-term glycaemic control is reflected by blood glucose fluctuations after ingestion of the food or meal, defined as the glycaemic index (GI) of the food or meal (Jenkins et al., 1981; see section 2.5), as well as the effect of the food or meal on disposal of blood glucose during succeeding meals, referred to as the second meal response (SMR) (Wolever, 1990).

The SMR is defined as the carry-over effect of a first meal, during which carbohydrate is slowly digested and absorbed on the glucose disposal during and after a subsequent meal (Wolever, 1990). The SMR has been demonstrated between breakfast and lunch (Jenkins et al., 1982a) and between dinner and breakfast (Wolever et al., 1988). It seems as if this effect is dependent on the GI of the first meal. Shaheen and Fleming (1987) could not demonstrate a SMR after meals with the same GI. Based on

observations of lower circulating free fatty acid levels, and studies in which guar gum was used to slow down absorption of carbohydrate (Jenkins et al., 1980b), Wolever (1990) presents the hypothesis that suppression of free fatty acid release after slower but prolonged absorption of carbohydrate is responsible for the SMR. Slower absorption of carbohydrate prevents functional hypoglycaemia (undershoot of baseline glucose levels) and the resultant counterregulatory release of insulin-antagonistic hormones and free fatty acids.

The SMR should not be confused with the Staub-Traugott effect, defined as improved or facilitated glucose disposal or utilization of a second glucose (carbohydrate) load taken within one half to three hours after a first glucose load. The effect was first described in 1919 by Hamman and Hirschman (as quoted by Metz and Friedenberg, 1970). It seems as if the effect is not dependent on plasma free fatty acid levels or insulin release, and it has been described for treated diabetic patients (Metz & Friedenberg, 1970).

The glycaemic indices of maize meal porridge, "imifino" or "morogo" (cooked green leafy vegetable stew) and cooked dried beans, constituents of the traditional African diet, have been measured individually in healthy black children (Walker & Walker, 1984). No information on the GI of these foods or combination of foods in adult black NIDDM patients, nor on the SMR of these foods, is available. In the present study the GI as well as the SMR of a traditional African meal has been measured in 14 treated black NIDDM patients in order to evaluate the potential of this meal for use in the dietary treatment of these patients.

5.3 STUDY DESIGN AND METHODS

5.3.1 Selection of subjects

A random sample of 14 subjects (7 men and 7 women), selected from a sub-population of all black NIDDM patients attending the out-

patient clinic of the Ga-Rankuwa hospital in the Pretoria district, voluntarily participated with informed consent. The sub-population consisted of patients between 40 and 60 years of age and with a BMI between 27 and 35. The study was approved by the joint Ethics Committee of the Medical University of Southern Africa and the Transvaal Provincial Administration. The characteristics and personal details of the subjects are given in Table 5.1.

Table 5.1 Individual and mean characteristics of the 14 NIDDM subjects taking part in the study

Subject	Age (yrs)	BMI# (kg/m^2)	Duration of NIDDM (yrs)	Medication*	Glucose control (HbA_{1c})
Men					
1	59	28.5	4	Diet alone	7.4
2	45	27.8	3	G + M	14.9
3	50	27.5	4	G + M	10.2
4	53	34.0	9	M + D + R	12.7
5	41	31.8	5	G + M + A	8.7
6	60	33.9	5	G + M + A	12.3
7	50	28.7	9	G + M	12.3
Mean	51.1	30.3	5.6		11.2
SD	± 6.4	± 2.6	±2.3		± 2.4
Women					
1	56	30.7	10	G + M + P	15.8
2	40	27.9	6	G + M	7.5
3	42	34.1	7	G + M + P	15.3
4	60	35.2	4	G + M + A	12.1
5	41	35.6	9	G + I	10.8
6	59	29.2	4	G + M	8.3
7	51	32.3	1	G + M	12.7
Mean	49.9	32.1	5.9		11.8
SD	± 8.1	± 2.7	±2.8		± 2.9

BMI = Body mass index

* G = Glibenclamide® M = Metformin® P = Paracetamol®
A = Amyloride® I = Indomethacine® R = Remitet®

5.3.2 Study design

5.3.2.1 **Experimental design**

Each subject visited the laboratory twice, one week apart, for measurement of post-prandial blood glucose levels over six hours

after intake of a test meal (traditional African meal and tea) and a reference meal (white bread and tea). The composition and nutrient analysis of the test and reference meals are given in Table 5.2.

Table 5.2 Composition and nutrient analysis* of test and reference meals

Composition and nutrients	Test meal		Reference meal
	SA tables	Englyst	
Maize meal, raw (g)	54		-
Soya mince, raw (g)	25		-
"Morogo", cooked (g)	90		-
Milk (in tea) (ml)	50		50
White bread (g)	-		100.5
Volume (ml)	670		485
Total energy (kJ)	1372		1178
Carbohydrate: Total (g)	52.5		52.5
% of energy	65.7		76.5
DF (g): Total	5.8		2.8
Maize meal	1.7		
"Morogo"	2.5	2.3	
Soya mince	1.6	trace	
Protein: Total (g)	17.2		9.8
% of energy	21		14
Fat: Total (g)	5.2		4.5
% of energy	14		11

* Analysis based on South African food tables (Gouws & Langenhoven, 1986) and DF or NSP analysis done for this study with the Englyst method (Englyst et al., 1988; Englyst et al., 1989).

The effect of the test meal on glucose disposal during the reference meal three hours later, and *vice versa*, was determined in a random manner. During the first visit one half of the

subjects (named subjects A) ate the reference meal first and the other half (named subjects B) the test meal first. During the second visit, the procedure on a particular subject was reversed (see Figure 5.1).

Glucose (meal) tolerance tests		
Visit	First three hours	Second three hours
First visit:		
Subjects A (n = 7)	Test meal (T_1)	Reference meal (R_2)
Subjects B (n = 7)	Reference meal (R_1)	Test meal (T_2)
Second visit:		
Subjects A (n = 7)	Reference meal (R_1)	Test meal (T_2)
Subjects B (n = 7)	Test meal (T_1)	Reference meal (R_2)

Figure 5.1 Experimental design of the study

5.3.2.2 Preparation of meals

Realistic portion sizes, as chosen by diabetic patients interviewed before the final composition of the meal, were used in the test meal. The meals were prepared as follows:
The test meal consisted of maize meal porridge, made with 54 g vitamin enriched special maize meal, cooked in 125 g of water for 180 minutes; 0.1 g of salt per 300 g of porridge was added. A portion of 25 g dried, flavoured minced soya product was cooked in 100 g of water for 20 minutes. For "morogo", 120 g fresh, raw spinach was washed and chopped and added to 25 g fresh, raw, chopped tomato (with skin) and 6.6 g fresh, raw, chopped onion. The vegetables were cooked in 40 g boiling water for 25 minutes to give a 90 g vegetable portion. The meal was cooled after preparation and reheated before serving, because this is the way in which the black population eat their meals. It was previously shown that cooled, reheated maize meal porridge has a lower GI than porridge that was eaten hot, immediately after cooking (Venter et al., 1990). The reference meal consisted of a 53 g

carbohydrate portion of white bread (standard government type) as recommended by Jenkins et al. (1984a). One cup of tea with 50 g fresh, low-fat milk was given with both meals.

According to data that was obtained with food frequency questionnaires (see Chapter 4), the diets of all the patients at the Ga-Rankuwa diabetic clinic are very similar. Dietary variation prior to the determination of the index of the test meals was probably very small and would not have had a marked influence on the GI. The same applies to the amount of carbohydrate that was taken for three days prior to the determination. The patients ate a high carbohydrate diet and intakes are usually above 250 g, the amount suggested for standardisation by Vorster, Venter and Silvis (1990).

The subjects reported after an overnight fast (eight to twelve hours); fasting blood glucose levels were measured and subjects then took their oral hypoglycaemic medication as usual, prior to the ingestion of the first meal. All meals were consumed within ten minutes. Capillary blood glucose measurements were made fasting, every 15 minutes during the first hour and every 30 minutes during the next two hours after ingestion of the two meals. Glucose concentrations were measured with the Glucometer II® reflectance photometer Model 5529 (Ames Division, Miles Laboratories, Elkhart, Indiana, USA) with Glucostix® reagent strips. The accuracy of the glucometer readings was tested with "Dextro Check Calibrator" solutions and checked against the enzymatic colorimetric method for serum glucose of Boehringer Mannheim, West Germany (Cat. no. 676543).

Areas under the glucose response curve were calculated with a simple integration programme as:

- incremental area with the lowest blood glucose value as baseline (no negative areas),
- incremental area with the fasting and/or t_o value as baseline (negative areas being subtracted),
- total area with zero as baseline.

Glycaemic indices were calculated using similar areas, expressing the reponse on the test meal as first meal as a percentage of the response on the reference meal as first meal. The maximum glucose increase was calculated by subtracting the fasting (first meal) or t_o (second meal) value from the maximum value. The maximum decrease was calculated as the difference between the t_o and t_{iso} value for each meal.

5.3.3 Statistical analysis

Significant differences ($p < 0.05$) between mean areas under the glucose curve, maximum glucose increase, decrease and glycaemic indices were calculated with the Student-Newman-Keuls test (Snedecor & Cochran, 1976). In addition, the significant effect of the first meal on the response to the second meal was examined with a non-parametric test (Wilcoxin's ranked test for matched pairs) and correlation coefficients determined with the Spearman non-parametrical test using the BMDP3D programme (Hill, 1987). The relationship between fasting and t_o values and the maximum increment was examined by calculating Pearson correlation coefficients, using the SAS^R package (SAS^R User's Guide, 1985).

5.4 RESULTS

Table 5.1 shows that there were no significant differences between the mean age, BMI, duration of the disease or the long-term glycaemic control (HbA_{1c}) of the men and women.

Table 5.2 indicates that the carbohydrate content of the test and reference meals was similar. The test meal had a slightly higher protein and fat content, and therefore also energy content than the reference meal. Total dietary fibre content, although relatively low in both meals, was approximately double in the test meal in comparison with the reference meal.

Table 5.3 gives the calculated mean areas under glucose response curves, illustrated in Figures 5.2, 5.3 and 5.4, for men and

Table 5.3 Mean (± SD) areas (mmol/l/l) under glucose response curves and maximum glucose increases and decreases after reference and test meals

Variable	Men		Women		Total	
	Mean	±SD	Mean	±SD	Mean	±SD
Incremental area[1]						
Reference first	86.5	44.8	117.2	40.8	101.8[a,b]	45.5
Reference second	55.3	32.2	43.6	17.4	49.5[a,c]	26.5
Test first	96.5	30.8	76.0	30.4	86.3[c]	32.3
Test second	62.7	12.1	60.9	12.9	61.8[b]	12.5
Incremental area[2]						
Reference first	83.3		119.5		100.5	
Reference second	29.6		23.2		27.6	
Test first	84.2		61.1		71.6	
Test second	9.9		-13.6		-1.0	
Total area[3]						
Reference first	2397	1198	2889	797	2643	1047
Reference second	1915	564	2625	910	2270	836
Test first	2030	633	2912	1158	2471	1032
Test second	1896	652	1921	1219	2130	866
Maximum glucose increase (mmol/l)						
Reference first	8.1	5.3	10.4	2.6	9.2[d,f]	4.3
Reference second	4.3	2.6	3.9	2.1	4.1[d,g]	2.3
Test first	7.9	2.2	6.9	1.5	7.4[e,g]	1.9
Test second	3.9	1.0	4.3	2.1	4.1[e,f]	1.7
Maximum glucose decrease (mmol/l)						
Reference first	6.6	5.9	5.6	2.5	6.1	4.5
Reference second	3.2	2.0	4.4	1.8	3.8	2.0
Test first	5.4	1.6	6.6	3.2	6.0	2.6
Test second	6.6	2.8	5.9	1.9	6.3	2.4

[1] = Area calculated with lowest blood glucose as baseline

[2] = Area calculated with t_0 blood glucose as baseline: mean blood glucose levels were used because of individual negative areas

[3] = Total area calculated with zero level as baseline

[a - g] = Means with the same symbol differs significantly ($p < 0.05$)

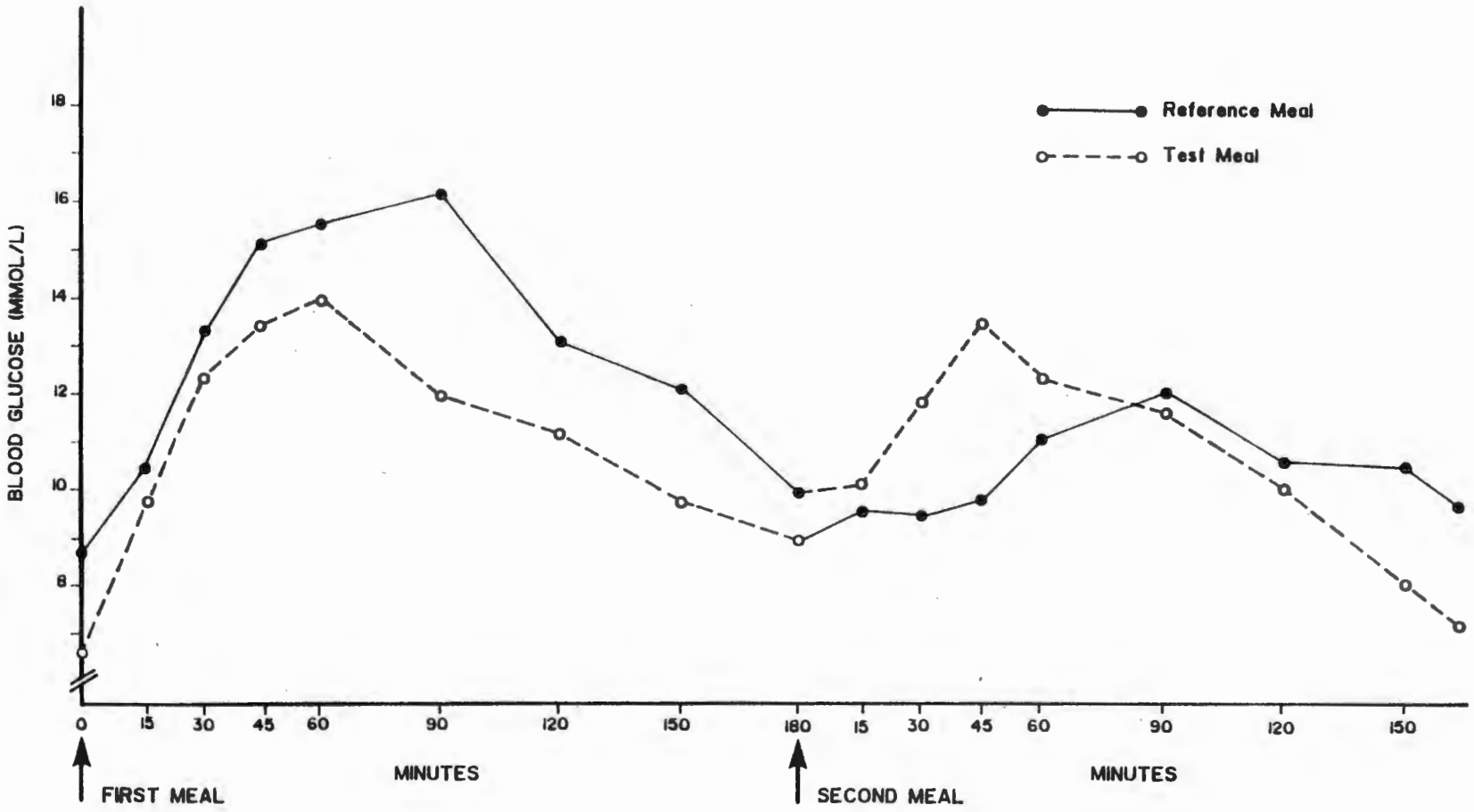


Figure 5.2 Mean glucose response curves of the men for the reference and test meals for both visits

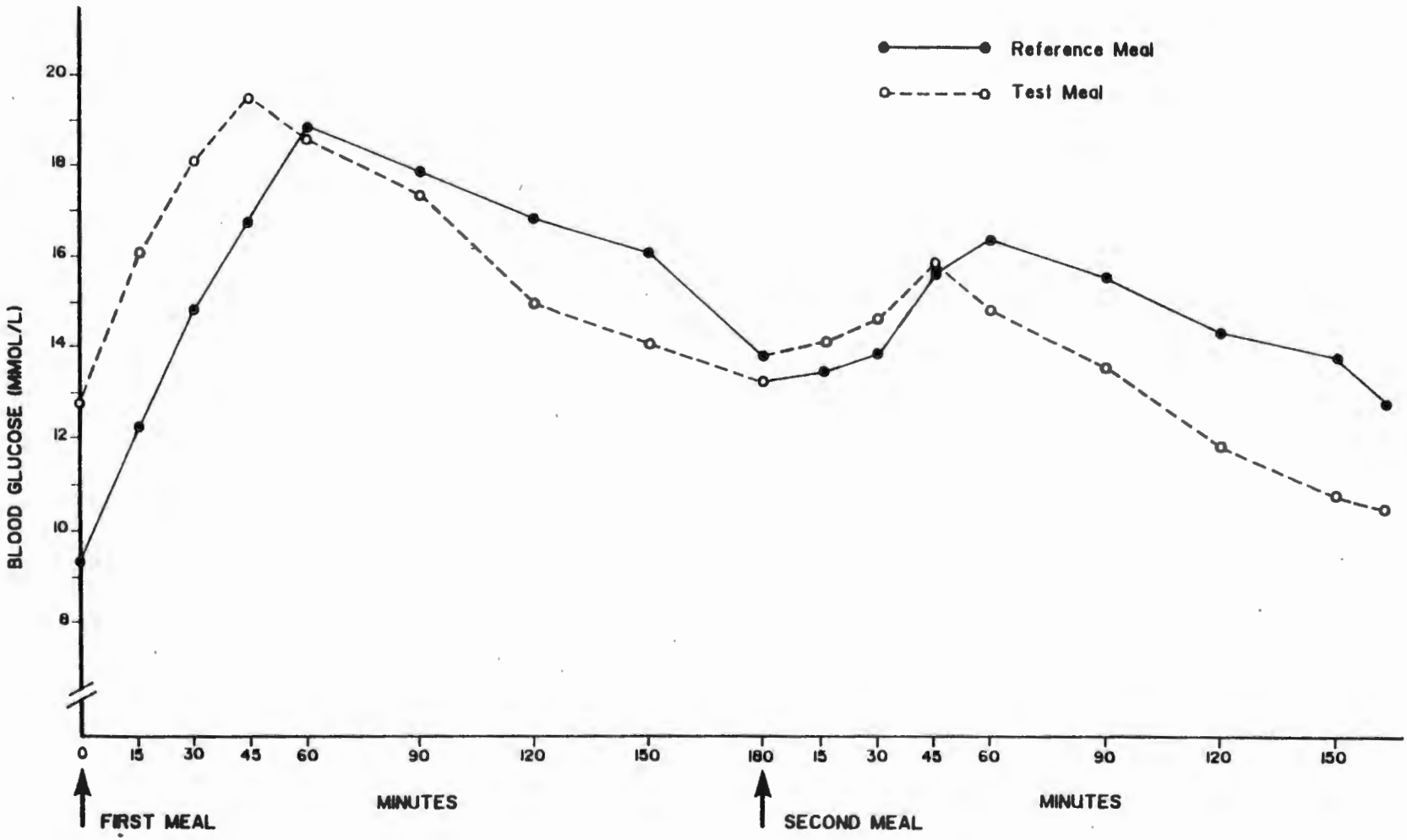


Figure 5.3 Mean glucose response curves of the women for the reference and test meals for both visits

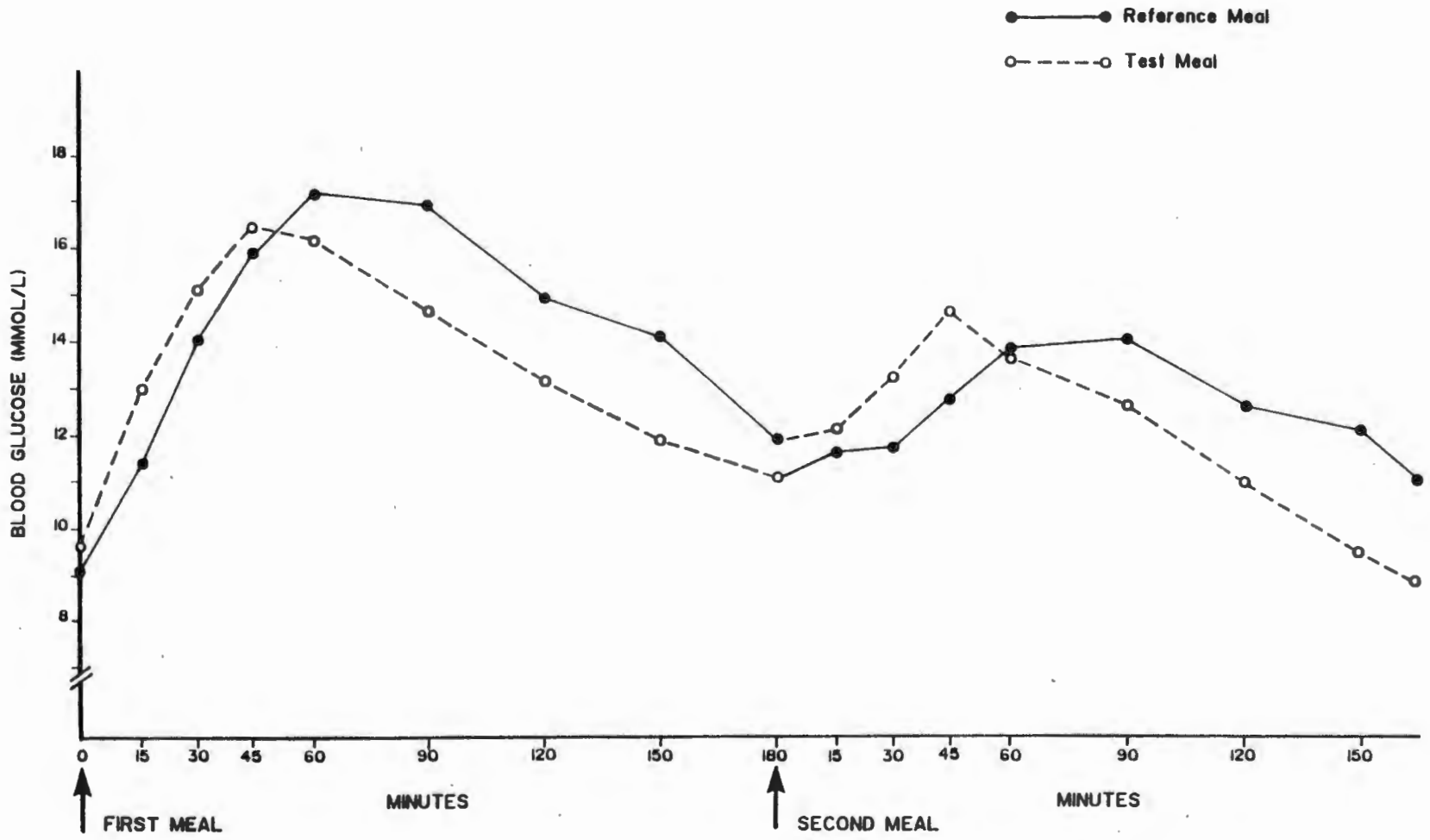


Figure 5.4 Mean glucose response curves of the all the patients for the reference and test meals for both visits

women separately and combined. Incremental areas calculated with the lowest blood glucose as baseline were similar to mean maximum glucose increases and seem to be a better reflection of the actual curves (Figures 5.2, 5.3 and 5.4), a finding previously reported (Vorster, Venter & Silvis, 1990).

However, in contrast to healthy subjects, a decrease of blood glucose below initial high fasting levels could represent a favourable effect in diabetic subjects. Therefore, areas calculated with fasting or t_o values as baseline, subtracting negative areas under this baseline, should be considered as the more accurate reflection of the meal-effect. The problem is that negative areas found in some individuals complicate the calculation of means and standard deviations. The mean blood glucose levels were therefore used to calculate these areas. Because of the extremely high glucose values of some of these subjects, total areas calculated with zero as baseline give little information and will not be used in evaluating the results.

Table 5.3 shows significant differences in incremental areas between the first and second meals, except between test first and test second, which only approached significance ($p < 0.06$). However, mean maximum glucose increases showed significant differences between all first and second meals. Figures 5.2, 5.3 and 5.4 illustrate the reduced responses during the second meal.

It is noteworthy that the GI, calculated with the first meals, was lower for women than for men (Table 5.4). Differences were significant when maximum glucose increments were used in the calculations.

Table 5.4 Mean glycaemic indices (± SD) of test meal*

Areas used in calculation	Glycaemic index (%)		
	Men (n = 7)	Women (n = 7)	Total (n = 14)
Incremental area with lowest blood glucose as baseline	131[a] ±50	71[a] ±35	101 ±53
Incremental area with t_0 as baseline	101	51	71
Total area with zero as baseline	95 ±30	99 ±16	97 ±24
Maximum glucose increase	123[b] ±45	71[b] ±19	97 ±43

* = Area/increment obtained with test meal as first meal x 100
Area/increment obtained with reference meal as first meal

[a] = Difference approached significance ($p < 0.06$)

[b] = Difference statistically significant ($p < 0.05$)

To calculate the predicted GI of the test meal (Table 5.5), available data on GI obtained from healthy black subjects (Walker & Walker, 1984), healthy subjects (Jenkins et al., 1981) and NIDDM subjects (Jenkins et al., 1984b) were used. The predicted GI of the test meal (Table 5.5) resembled the experimental GI (Table 4.5) in the women but not in the men.

Table 5.5 Predicted glycaemic index (GI) of test meal*

Contents of meal	Mass (g)	GI*	GI x mass
Maize meal as porridge	50	74	3915
Soya mince	25	15' (16$)	375 (1525)
"Morogo"	90	68	6120
Milk	50	3	150
Total	215		10560 (11710)
Predicted GI	$\frac{\text{Total (mass x GI)}}{\text{Total mass}}$		= 49.1 (54.5)

* = Based on GI determined in healthy black subjects (Walker & Walker, 1984)

' = GI of soya beans determined in healthy subjects (Jenkins et al., 1981)

$ = GI of kidney beans determined in NIDDM subjects treated with oral hypoglycaemic agents (Jenkins et al., 1984b)

Table 5.6 expresses the effects of the first meal on the SMR as a percentage reduction in the mean areas from the first to the second meal. The women always showed the larger percentage reduction, regardless of whether the first meal was the reference or test meal.

The influence of the fasting (t_{o1}) or starting (t_{o2}) glucose values on the glycaemic response is demonstrated in Table 5.7. There were no significant relationships between t_o values and the maximum increments, indicating that the t_o value did not influence the response. This is in contrast with the results reported by Nielsen and Nielsen (1989) which showed a significant negative correlation between preprandial blood glucose values greater than 13 mmol/l with glucose response.

Table 5.6 Percentage differences in mean areas and maximum glucose increase between first and second meals

First meal	Percentage reduction in mean area between first and second meal		
	Men	Women	Total
Test meal	$T_1 - R_2$	$T_1 - R_2$	$T_1 - R_2$
Area[1]	42.7	42.6	42.6
Area[2]	64.8	62.0	61.5
Maximum increase	51.9	58.6	55.4
Reference meal	$R_1 - T_2$	$R_1 - T_2$	$R_1 - T_2$
Area[1]	27.5	48.0	39.6
Area[2]	88.1	111.4	101.0
Maximum increase	51.9	58.6	55.4
Test meal	$R_1 - R_2$	$R_1 - R_2$	$R_1 - R_2$
Area[1]	36.1	62.8	51.4
Area[2]	64.4	80.5	72.5
Maximum increase	46.9	62.5	55.4
Reference meal	$T_1 - T_2$	$T_1 - T_2$	$T_1 - T_2$[a]
Area[1]	35.0	19.9	28.4
Area[2]	88.2	122.2	101.4
Maximum increase	50.6	37.6	44.5

R_1 = Reference meal as first meal

R_2 = Reference meal as second meal

T_1 = Test meal as first meal

T_2 = Test meal as second meal

Area[1] = Incremental area with lowest blood glucose as baseline

Area[2] = Incremental area with t_o glucose as baseline

[a] = Reduction statistically significant between T_1 and T_2 ($p = 0.0313$ with Wilcoxin ranked test for matched pairs with the area of maximum increase as value)

The only statistically significant difference according to the Wilcoxin ranked test for matched pairs was between the test meal as first and the test meal as second meal. The

difference between the reference meal as first and the reference meal as second meal approached significance (p = 0.0625). There were no significant correlations between any of the meals according to the non-parametric Spearman correlation coefficients.

Table 5.7 The relationship between fasting and starting glucose values (t_o) and maximum glucose increase (max incr)

Variables correlated	Pearson correlation coefficients	
	r	P
t_o x max incr : all 4 meals (n = 56)	0.33	0.26
t_o x max incr : R_1	0.32	0.27
t_o x max incr : R_2	-0.18	0.53
t_o x max incr : T_1	0.01	0.97
t_o x max incr : T_2	-0.39	0.17
max incr x mac incr:		
R_1 x T_2	0.20	0.48
T_1 x T_2^a	-0.74	0.003
R_1 x R_2	0.40	0.16
T_1 x T_2^b	0.02	0.95
t_{o1} x t^{o2}		
R_1 x T_2	0.86	0.0001
T_1 x R_2	0.85	0.0001

R_1 = Reference meal as first meal
R_2 = Reference meal as second meal
T_1 = Test meal as first meal
T_2 = Test meal as second meal
^{a} = separate days
^{b} = same day
t_{o1} = fasting blood glucose value (before first meal)
t_{o2} = starting blood glucose value (before second meal)

However, there were high and significant correlations between the

fasting glucose value (t_{o1}) and the starting value (t_{o2}) of the second meal on the same day.

5.5 DISCUSSION AND CONCLUSIONS

The study was designed to examine the short-term effects of a traditional African meal on acute blood glucose responses in black NIDDM patients by
- obtaining the GI of the African meal
- and examining the SMR after this meal.

A possible criticism of the design of the study could be that the second meal was not the standard bread meal in both instances. However, it was attempted to examine the effect of the bread meal on the African meal. Furthermore, each patient was his/her own control as recommended by Vorster, Venter and Silvis (1990).

The second meal was given to the patients three hours after the first meal was eaten. The effect of the second meal qualifies by definition for a possible Staub-Traugott effect (Metz & Friedenberg, 1970). The Staub-Traugott effect is not dependent on insulin secretion or circulating free fatty acid levels (Metz & Friedenberg, 1970). It seems as if it may be caused by a nutrient induction of activity of enzymes involved in carbohydrate metabolism. The question therefore arises whether the lower glycaemic response observed during the second meal could be classified as a true second meal effect as defined by Jenkins et al. (1982b) and Wolever (1990), or whether it was only a Staub-Traugott effect?

The predicted GI, based on the glycaemic indices of the individual food items (see Table 5.5), correlates well with the GI of the traditional African meal in the women but not in the men. Another question which therefore arises concerns the possible reasons for this phenomenon.

The GI of the test meal was significantly lower in the women than

in the men. According to Wolever (1990) and Jenkins et al (1982b), the SMR is observed only after a low GI meal. If this is true, the women should have shown a larger reduction in area during the second meal when the test meal was the first meal. The effect of the test meal as first meal on the mean percentage reduction in area of the reference meal was indeed substantially higher in the women.

This suggests that when the GI of the first meal is low, a SMR is observed.

Table 5.6 and Figures 5.2, 5.3 and 5.4 also show that the mean percentage reduction in area was similar in men and women when the areas of the first and second meals are compared, with either the test or the reference meal as first meal. This suggests that both the men and the women showed the Staub-Traugott effect.

The reasons why the men and the women had different glycaemic indices for the test meal are not clear. The mean age, duration of the disease, medication and control of the disease (HbA_{1c} levels) were similar. The women were slightly more obese than the men (see Table 5.1) but there was no clinically significant difference in BMI. In contrast with some other populations, the WHR of the black NIDDM patients who visit the Ga-Rankuwa hospital out-patient clinic does not differ much between the women and the men (see Chapter 6). Weight and fat distribution could, therefore, also not be the cause for the difference.

From the above it is clear that more research is needed to illuminate possible mechanisms through which the second meal effect is mediated as well as reasons why the effect differs in the different sexes. If the reasons for this effect are clear, it may be used in the design of diabetic diets - especially in the frequency of meals to ensure a slower, more even absorption of carbohydrates during the first meal.

CHAPTER 6

LONG-TERM EFFECTS OF A TRADITIONAL AFRICAN DIET ON THE METABOLIC CONTROL OF BLACK PATIENTS WITH NON-INSULIN-DEPENDENT DIABETES MELLITUS

6.1 SUMMARY

To determine the long-term effects of a traditional African diet on the metabolic control of black patients with NIDDM, 51 patients were divided into a control (8 men, 13 women) and test group (11 men, 19 women). The control group followed an adapted, westernised diabetic diet and the test group a low-GI African diet, rich in maize meal porridge, soya and green leafy vegetables, for a period of five months. Compliance of the test group to the experimental diet, which had a macro-nutrient distribution of 23 % of total energy as protein, 23 % as fat and 53 % as carbohydrates, was 71 %.

The following measurements were taken monthly:
Anthropometric data, dietary intakes and blood samples for determining lipids, electrolytes and minerals, proteins and enzymes, excretion products and full blood counts. Glycaemic control was determined by measurements of capillary and serum glucose, glycated haemoglobin and fructosamine.

The mean weight and BMI of both groups were above normal ranges and no significant change took place during the study. The mean WHR indicated that the obesity was mainly of the android or central type. Glycaemic control was poor and worsened during the study. Slight improvements were observed in the serum lipid and the plasma fibrinogen values of both groups, but more so in the test group. Electrolytes, minerals, proteins, enzymes and full blood counts were near or within normal ranges and did not show any significant change during the study.

It was concluded that:

- the traditional African diet with a low GI but generous energy allowance, as prescribed for these patients, did result in statistically significant but not clinically significant weight loss. A reducing diet with a low energy content as well as a low GI should be prescribed for overweight black NIDDM patients;
- in addition to overweight, the high prevalence of hypertension and high WHR's of these subjects should get attention;
- the black NIDDM patient can follow the traditional African diet successfully. With proper motivation and attention, the patients will probably be able to reduce energy, fat and animal protein intake and increase dietary fibre intake;
- lipid profiles were normal to slightly high in contrast with the high values reported for white NIDDM patients and are possibly not major contributors to macro-vascular complications;
- weight loss in these patients was accompanied by small but statistically significant decreases in plasma triglycerides, apolipoprotein B, fibrinogen and total cholesterol (in men);
- the weight loss was probably partly due to the low GI of the diet;
- glycaemic control in the black NIDDM patient can possibly not be improved without substantial weight loss;
- high chloride levels in these patients might indicate macro-vascular and renal damage.

More research to investigate the energy needs of black NIDDM patients, and the relationship between weight and glycaemic control, should be undertaken.

6.2 INTRODUCTION

It is estimated that 60 million people worldwide have DM (Brownlee, 1985:185). In the developing nations of the world alone, there are probably a total of between 25 and 50 million diabetics (Bennet, 1983:55). DM is also a common disorder in the

South African population. It has been estimated that in excess of four percent of the population have the disease (Huddle, 1989:11). Approximately 400 000 urban South African blacks have DM and the incidence of the disease in the rural areas is unknown (Seedat, 1989a:18). NIDDM contributes to 85 - 90 % of diabetics in both developed and developing countries (Zimmet, 1982:400).

The prevention of NIDDM or the prevention of the development of complications of the disease is, therefore, of utmost importance. Although patients with NIDDM are less likely to experience the acute metabolic complications of diabetes than patients with IDDM, their vulnerability to vascular complications is not correspondingly reduced (Krowlewski, Warram & Christlieb, 1985). Many of the variables predicting future diabetes are also known to be risk factors for macro-vascular disease. After clinically manifest diabetes develops, risk factors for cardiovascular disease are further increased (Wingard et al., 1983).

The main objectives in the treatment of DM should therefore be to control blood glucose levels as well as all risk markers of the long-term micro and macro-vascular complications of the disease. With the process of westernisation the prevalence of conventional risk factors for coronary heart disease is increasing in black South Africans. Furthermore, glycaemic control in blacks with NIDDM is poor (Silvis, 1989). Diet remains the cornerstone in the management of diabetes (Nuttal, 1988) and the dietary treatment of the black NIDDM patient should therefore get special attention.

The modern diabetic diet, as prescribed by various expert organisations throughout the world (American Diabetes Association, 1990a), has a nutrient composition very similar to the typical traditional diet of the rural blacks in South Africa (Silvis, 1989). The traditional diet also has a relatively low glycaemic index (see Chapter 5). This traditional diet can therefore possibly be the optimal dietary treatment of the westernised black NIDDM patient.

The main hypothesis tested in this part of the study was that the traditional, low GI African diet, compared to the westernised diabetic diet as eaten by the local black people with NIDDM, improves metabolic control and reduces weight, as well as minimising the risk markers of vascular complications. Other hypotheses in support of the main hypothesis were that it is possible for the black diabetic in the Ga-Rankuwa region to follow the traditional African diet and that nutrition intervention and attention, even to a small extent, will make a positive difference in the weight, metabolic control and risk markers of vascular complications in black diabetics who visit an outpatient clinic. In this chapter, the compliance of the NIDDM patients to the diet in the long-term, and the influence of the diet on glycaemic control and markers of risk factors for coronary heart disease, were examined.

6.3 METHODOLOGY

6.3.1 Study design

A summary of the study design can be seen in Figure 6.1. The

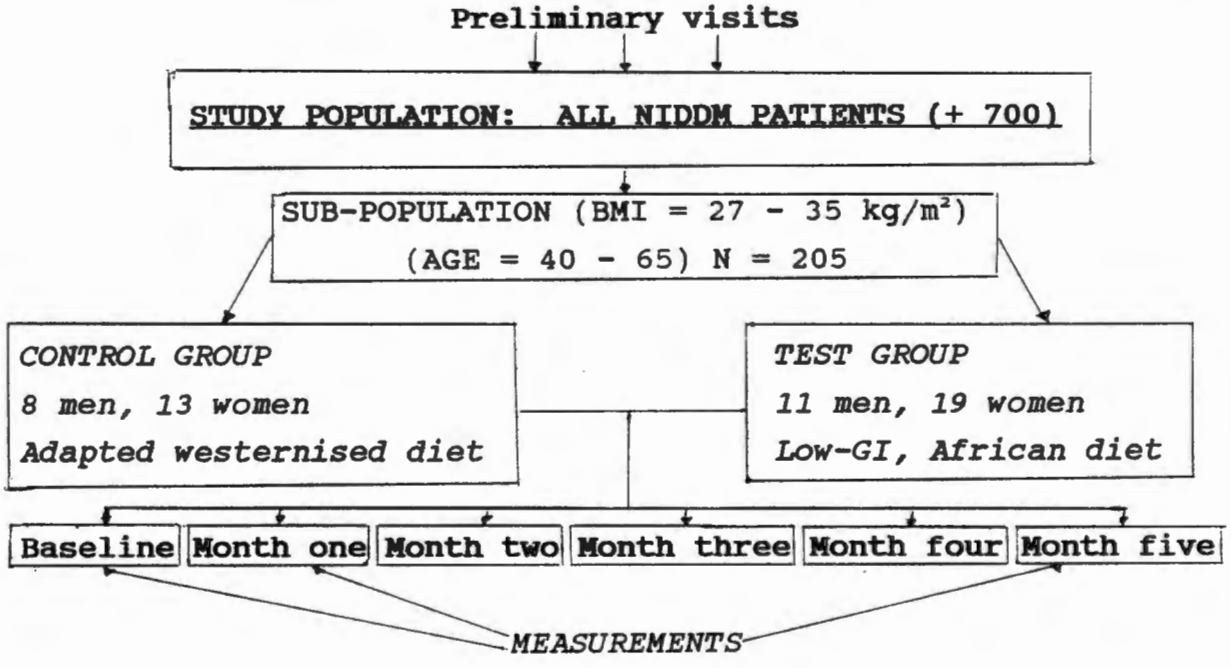


Figure 6.1 Design of the third phase of the study.

participating subjects visited a clinic in the Department of Human Nutrition at MEDUNSA once a month for five months, on the same day as their monthly check-up at the diabetic clinic of Ga-Rankuwa hospital. The subjects were randomized into a control group of which eight men and 13 women completed the study and a test group of which eleven men and 19 women completed the study. The control group followed a reference diet and the test group a test diet as indicated below. The diets were followed for the whole duration of the five months and all measurements were determined at baseline, after one month and after five months.

The study was approved by the Joint Ethical Committee of MEDUNSA and the Transvaal Provincial Administration.

6.3.2 Selection of patients

All patients attending the Ga-Rankuwa outpatient NIDDM clinic formed the population from which a sample was drawn. The patients who took part in the pilot studies were included in the sample. A sub-population of all the patients that were from 40 to 65 years of age in January 1990, who had a BMI of 27 to 35 kg/m^2, who were relatively inactive except for walking some distances some days during the week, and who took oral hypoglycaemic agents and not insulin as medicine in January 1990, was then chosen. Out of this sub-population of 205, 65 patients were randomly selected and they agreed to take part in the study.

The patients were interviewed at the Ga-Rankuwa clinic during their monthly visit to inform them of the aims, procedures and possible effects of the study. Procedures for possible emergencies were explained. The consent of the patient for these procedures was asked (consent form in Appendix C) and dates on which they should come to MEDUNSA for the first visit were determined.

Only 57 patients arrived for their appointments. It was difficult to obtain blood from five of the patients and blood

values of one patient were totally different from the rest. It was decided not to include these patients in the study.

6.3.3 Test diet, reference diet, motivation and measurement of compliance

From the data obtained with the methods discussed in section 3.1 and 3.2, a diet, including two typical African meals with a low GI, was compiled as the test diet. The reference diet was compiled to have the same pattern and food items as the westernised African diet currently followed by the NIDDM patients in the area. As the study was done on patients under free-living conditions and not in a metabolic unit, the meal plan with exact food items and portion sizes was given to patients on the test diet. A meal plan with the type of food items allowed and portion sizes was given to the patients on the reference diet. The diet sheets for these diets are given in Appendix D and E. The nutrient composition of the test and reference diets is shown in Table 6.1. Every third patient was asked to follow the reference diet and the rest were asked to follow the test diet, resulting in 21 subjects in the control group and 30 subjects in the test group.

The patients received a diet sheet at the beginning of the study and the diet was then discussed with them in detail. The patients received most of the food prescribed in the diet sheet. The patients monthly received enough maize meal (A1® super fine, enriched) and soya product (Knorrox® soya mince, various flavours) in the case of the test diet and maize meal only in the case of the reference diet. With each follow-up visit, the patients were motivated to follow the prescribed diet and a score was given for compliance. At the last visit (fifth month) the mean score was converted into a percentage to express compliance.

Table 6.1 Comparison of the nutrient composition of the recommended test and control diet with the RDA and RADA*

		CONTROL GROUP		TEST GROUP		RDA/ RADA*	
		Men n = 8	Women n = 13	Men SD n = 9	Women SD n = 19	Men	Women
Energy	(kJ)	14574	10738	11497	8496	12200	9250
Protein	(% of E)	19	19	13	14		
	Total (g)	161.2	121.6	134.5	114.3	63	46
	Plant (g)	64.4	45	121.3	101.1		
	Animal(g)	96.8	76.6	13.2	13.2		
	g/kg	1.97	1.48	1.64	1.39	0.8	0.8
Fat	(% of E)	26	28	13	14	<30	<30
	Total (g)	101.3	80.8	38.9	32.5		
	% Sat	33	35	27	31		
	% MU	38	38	23	23		
	% PU	14	14	18	16		
	P/S ratio	0.42	0.4	0.67	0.52	1	1
	Chol (mg)	302	243	56	56	<300	<300
Carbohydrates							
	(% of E)	55	52	67	62	55	55
	Total (g)	474	331.8	460.2	316.1		
	Fibre (g)	46.3	34.7	48.7	36.3	40	40
	Sugar (g)	0	0	0	0		
Minerals	Ca (mg)	907	842	1102	1052	800	800
	Fe (mg)	27.2	20.5	50.8	45	10	15
	Mg (mg)	673	514	768	610	350	280
	P (mg)	2095	1610	1989	1606	800	800
	K (mg)	3823	3168	3284	2724	2000	2000
	Na (mg)	3304	2460	1240	932	750	750
	Zn (mg)	27.3	20.4	12.3	8.88	15	12
	Cu (mg)	1.61	1.26	1.15	0.92	2.25	2.25
Vitamins	A (RE)	1063	1049	1007	1007	1000	800
	D (ug)	0.24	0.24	0.24	0.24	5	5
	E (mg)	9.67	8.06	9.4	7.65	10	8
	Thiam(mg)	3.16	2.32	2.74	1.92	1.5	1.1
	Ribof(mg)	2.42	2.02	1.67	1.44	1.7	1.3
	Niac (mg)	31.8	23.5	13.4	9.4	19	15
	B-6 (mg)	1.85	1.52	0.87	0.79	2	1.6
	Folac(mg)	360	318	326	292	200	180
	B-12 (ug)	4.8	4	1.6	1.6	2	2
	Panto(mg)	5.77	4.71	2.7	2.42	5.5	5.5
	Biot (ug)	19.8	16.3	17.8	15	65	65
	C (mg)	93	93	93	93	60	60

* RDA = Recommended daily allowances (National Research Council, 1989), RADA = Recommendations of the American Diabetic Association (American Diabetes Association, 1990).

% of Energy = percentage of energy

P/S ratio = Polyunsaturated to saturated fatty acid ratio

Sat = Saturated fatty acids

MU = Mono-unsaturated fatty acids

PU = Polyunsaturated fatty acids

Chol = Cholesterol

Thiam = Thiamin

Ribof = Riboflavin

Niac = Niacin

Folac = Folacin

Panto = Pantothenic acid

Biot = Biotin

6.3.4 Clinic procedure

A home economist and social worker were trained to obtain the data on dietary intakes, compliance and life-style changes. Two qualified nurses drew venous blood samples, measured blood glucose and took blood pressure.

6.3.4.1 **Measurements**

All measurements are discussed in detail in Chapter 3. Briefly, the following measurements were taken during each visit to the clinic:

* Weight was measured with an electronic Soehnle® scale, verified each morning with a 10 kg weight. Half a kilogram was subtracted from the measured weight for clothing.

* Height was taken by a trained interviewer with the patient looking straight ahead, without tipping the head up or down, and the tip of the ear and outer corner of the eye in a line parallel to the floor.

* The BMI was calculated as weight/(height(m))^2.

* Waist-to-hip circumference ratio was calculated as the smallest circumference of the waist divided by the largest circumference of the hip.

* Blood pressure was measured once during each visit. The first and fifth Korotkoff sounds were noted.

* Capillary blood glucose was monitored during every visit after an overnight fast, using a Glucometer® II Reflectance Photometer (Ames Division, Miles Laboratories, Elkhart, Indiana, USA) and Glucostix® reagent strips (Ames Division, Miles Laboratories, Slough, England). In addition, serum glucose was measured as part of the venous blood analysis.

6.3.4.2 **Measurement of dietary intakes**

At the baseline of the study a short list was used to determine the number of meals per day, the number of portions of cereal staple foods, vegetables and meat and the sizes of the portions.

During the visit one month later, the complete FFQ that was discussed in Chapter 4 was filled in to determine the habitual food intake before the study was started and notes were made of changes that took place during the previous month. The habitual intake was then compared with the short list of the previous visit and if there were discrepancies, the patient was asked to explain.

During the third visit the complete FFQ that was discussed in Chapter 4, was filled in again to determine the habitual food intake during the study. The habitual intake was then compared with the changes that were noted from baseline to the visit one month later. If there were discrepancies, the patient was asked to explain.

During the fourth visit a 24-hour recall was done for the previous Sunday, as it was clear from the results of section 3.1 that the Sunday meal pattern was different from the meal pattern during the rest of the week. This information was then checked against the information of the FFQ of the previous month and data that were not given in the FFQ were queried.

During the fifth month a 24-hour recall was done for the previous day (Wednesday because clinics were always on Thursday) and the information was checked against the information of the FFQ of the third month.

Some patients did not attend all the follow-up sessions. In their cases it was ensured that they had at least completed the FFQ of the second and the third month.

6.3.5 Blood sampling and analysis

Antecubital venous blood samples (50.5 ml) were taken after an overnight fast, using a 21-gauge needle with a vacutainer directly into vacutubes, for the preparation of plasma and serum as indicated in Chapter 3, Figure 3.1.

Variables that were measured in the plasma and serum are summarised in Table 6.2, and are discussed in detail in Chapter 3.

It is advised that a laboratory should determine normal ranges of blood values for the specific population it serves (Vermaak et al., 1988). Normal ranges for measured blood constituents given in the Tables are, therefore, the ranges used by the specific laboratories where the blood was analysed.

Table 6.2 Variables measured in blood samples

Department Chemical Pathology, University of Pretoria	Department Chemical Pathology, MEDUNSA	Department Haematology, MEDUNSA
Electrolytes (serum)	*Lipids (serum)*	*Full blood count*
Sodium	Total cholesterol	Leucocytes
Potassium	HDL-cholesterol	Erythrocytes
Chloride	Triglycerides	Mean corpuscular haemoglobin
Phosphorus	Apolipoprotein A	Mean corpuscular haemoglobin concentration
Calcium	Apolipoprotein A	Platelet count
	Fibrinogen (plasma)	Mean plasma volume
		Mean corpuscular volume
Proteins and excretion products (serum)	*Glucose control*	Haemoglobin
		Haematocrit
Blood urea nitrogen	Fructosamine (serum)	
Uric acid	Glycated haemoglobin (whole blood)	
Creatinine	Serum glucose	
Total bilirubin		
Direct bilirubin		
Aspartate aminotransferase		
Lactate dehydrogenase		
Alanine aminotransferase		
Gamma-glutamyltransferase		
Alkaline phosphatase		
Total protein		
Albumin		
Carbon dioxide		

6.3.6 Statistical analysis

The dietary data were analysed with Dietary Manager® of Programme Management (Scharf, 1990) and the RIND Food Composition Tables (Gouws & Langenhoven, 1986). Statistical analysis of the nutrient intake data was done with the EPI INFO® package (Dean, 1986).

Statistical analysis of the anthropometric and blood variables was done at the Medical Research Council in Pretoria with the BMPD® package (Hill, 1987). Probability values for differences between visits were calculated with the non-parametric Wilcoxin ranked test and probability values for differences between the control and test groups were calculated with the Mann-Whitney test. The Spearman test was used to determine significance of correlations between variables.

Significance of differences was taken as $p \leq 0.05$ in all cases.

6.4 RESULTS

6.4.1 Subject details

The information obtained from 49 of the patients selected for the study was complete and could be used. The characteristics of the subjects at the baseline of the study can be seen in Table 6.3. Significant differences in mean values between the test and control groups are given in Table 6.22 and indicated with an "x" on Table 6.3 as well as all other tables. Subjects were grouped according to sex and type of diet used at the time of the study. Although all the subjects were on oral hypoglycaemic agents when they agreed to participate, six of the patients were on insulin (three in the test group and three in the control group) when the study started, as can be seen in Table 6.6. New medical staff started to work at the diabetic clinic just before the study began and they believed that some of the patients could be better controlled with insulin. The characteristics of the patients on

Table 6.3 Baseline characteristics of subjects

	CONTROL GROUP						TEST GROUP						ORAL AGENTS		INSULIN	
	Men		Women		Total group		Men		Women		Total group					
	Mean	SD	Mean	SD	Mean	SD	Mean	SD	Mean	SD	Mean	SD	Mean	SD	Mean	SD
	n = 7		n = 12		n = 19		n = 10		n = 20		n = 30		n = 43		n = 6	
Age (years)	53.00	7.4	55.17	6.8	54.40	6.90	51.20	4.8	54.40	5.8	53.30	5.6	53.60	6.3	54.70	4.5
Height (cm)	168.29	5.0	157.33	2.9	161.40	6.50	170.80	7.7	155.30	4.8	160.50	9.4	161.50	8.5	156.00	5.1
Weight (kg)	79.59	14.4	82.98	16.8	81.70	15.60	84.39 ab	9.9	81.11 ab	16.1	82.20	14.2	81.80	14.7	83.70	15.6
BMI	28.10	4.9	33.43	6.3	31.50	6.30	28.93 b	3.2	33.65 ab	6.8	32.10	6.2	31.50	6.2	34.40	6.1
Blood Pressure (mmHg)	130.90	20.18	133.89	19.14	132.89	19.15	134.85	23.12	136.89	22.11	135.87	22.11	132.88		148.91	
WHR x	0.97	0.1	0.81	0.1	0.87	0.11	0.96	0.0	0.87	0.1	0.90	0.1	0.89	0.1	0.85	0.1
PA ratio	1.67	0.1	1.78	0.1	1.74	0.12	1.62	0.1	1.70	0.1	1.67	0.1	1.69	0.1	1.79	0.1
N Hypertensive					9.00						10.00		15.00		4.00	

Table 6.4 Characteristics of subjects after one month

	CONTROL GROUP						TEST GROUP						ORAL AGENTS		INSULIN	
	Men		Women		Total group		Men		Women		Total group					
	Mean	SD	Mean	SD	Mean	SD	Mean	SD	Mean	SD	Mean	SD	Mean	SD	Mean	SD
	n = 5		n = 12		n = 17		n = 10		n = 20		n = 30		n = 41		n = 6	
Age (years)	53.40	7.0	55.17	6.80	54.60	6.7	51.20	4.8	54.40	5.8	53.30	5.6	53.70	6.2	54.70	4.5
Weight (kg)	76.30	17.1	83.53	17.20	81.40	17.0	83.58 a	9.7	80.85 a	15.7	81.80	13.8	81.30	15.0	83.60	15.5
BMI	27.24	5.5	33.66	6.50	31.80	6.7	28.69	3.1	33.56 a	6.7	31.90	6.1	31.50	6.3	34.40	6.1
Blood Pressure (mmHg)	144.99	43.34	133.84	17.08	136.89	26.21	131.83	24.14	136.89	22.12	134.87	23.13	133.88	24.16	147.87	12.12
PA ratio	1.69	0.1	1.78	0.10	1.75	0.1	1.62	0.1	1.70	0.1	1.68	0.1	1.70	0.1	1.76	0.1
N Hypertensive					5.00						13.00		13.00		5.00	

Table 6.5 Characteristics of subjects after five months, at the end of the study

	CONTROL GROUP						TEST GROUP						ORAL AGENTS		INSULIN	
	Men		Women		Total group		Men		Women		Total group					
	Mean	SD	Mean	SD	Mean	SD	Mean	SD	Mean	SD	Mean	SD	Mean	SD	Mean	SD
	n = 6		n = 10		n = 16		n = 10		n = 19		n = 29		n = 39		n = 6	
Age (years)	51.83	7.3	55.70	7.30	54.30	7.3	51.20	4.8	54.16	5.8	53.10	5.6	53.40	6.5	54.70	4.5
Weight (kg)	80.22	16.8	83.51	17.90	82.30	17.0	82.81 b	9.4	80.00 b	16.4	81.00	14.2	80.90	15.0	84.70	16.8
BMI	28.28	5.7	33.75	6.80	31.70	6.8	28.39 b	3.2	33.17 b	7.0	31.50	6.3	31.10	6.3	34.80	6.6
Blood Pressure x (mmHg)	132.88	17.1	130.85	12.70	131.86	13.90	131.85	21.10	134.87	25.13	133.86	23.12	131.86	21.11	137.85	16.50
WHR	0.95	0.1	0.83	0.10	0.88	0.1	0.97	0.1	0.85	0.0	0.89	0.1	0.90	0.1	0.85	0.1
PA ratio	1.69	0.1	1.76	0.10	1.73	0.1	1.61	0.1	1.69	0.1	1.67	0.1	1.68	0.1	1.73	0.1
N Hypertensive					4.00						16.00		18.00		2.00	

BMI = Body mass index

WHR = Waist-to-hip circumference ratio

PA ratio = Protein to albumin ratio

N hypertensive = Number of subjects who are hypertensive (Blood pressure > 140/90 (Working group on hypertension, 1987))

a = good correlation between both test and control groups

b = good correlations during baseline and after one month

c = good correlations during baseline and after five months

d = good correlations after one and five months

x = correlation ≥ 0.6, p ≤ 0.5

oral hypoglycaemic agents and insulin are, therefore, given separately in the last two columns.

There were no statistically significant differences in age, height, weight, BMI-values, blood pressure and protein/albumin ratio between the patients in the test and control groups at baseline. Comparison of the BMI-values to existing standards (Krause & Mahan, 1984), shows that the men in both groups could be classified as being overweight and the women obese. If mean weights and heights of the women in both groups are compared to those of the men, it is clear why their mean BMI's were higher. Their weights were nearly the same as those of the men, but they were much shorter. Because obesity is one of the main risk factors for precipitating NIDDM, and for the development of the complications of the disease, a weight reduction strategy should be a main objective in the treatment of these subjects (Streja, Boyko & Rabkin, 1980). The mean weight and BMI values of the six insulin-treated patients were higher than those of the patients on oral hypoglycaemic agents.

The BMI values did not show consistently significant correlations with any of the other measured variables (see Tables 6.11 and 6.12).

The mean blood pressure for the males was 132/88, which is near the 134/78 of younger healthy rural black males (mean age 28 years) who took part in the study of Vorster et al. (1987a:53). Thirty-nine percent of the subjects were hypertensive. Some subjects took anti-hypertensive medication which lowered blood pressure effectively, while others still had high blood pressure despite medication (Table 6.6). If all patients who took anti-hypertensive drugs are classified as hypertensive, 59 % of the patients had high blood pressure. Proportionally the insulin-treated patients had a greater incidence of hypertension than those on oral hypoglycaemic agents. Their mean blood pressure (148/91) was also higher than the cut-off point for high blood pressure (140/90) while the mean blood pressure of the patients

who used oral hypoglycaemic agents was below this cut-off point.

Table 6.6 Medication of patients who took part in the study

Type of Medication	Number of subjects
Insulin (mainly Actraphane®) and anti-hypertensive medication	6
Aldomed® (ß-blocker)	4
Amiloride® and Methyldopa®	1
Capoten®	1
Oral hypoglycaemic agents (OHA) only	28
Glibenclamide® only	4
Metformin® and Glibenclamide®	24
OHA and anti-hypertensive medication	15
Aldomed®	7
Amiloride® and Methyldopa®	4
Capoten®	4
Hypertensive patients on medication for hypertension	7
Hypertensive patients not on medication for hypertension	9
Hypertensive patients on insulin and medication for hypertension	3

Hypertension is common in South African urban blacks (Seedat, Seedat & Reddy, 1978) and among diabetics (Ohlson et al., 1988), and obesity is an aggravating factor (Abrahamson, 1988). Weight reduction should therefore contribute to lower blood pressure. More attention should be given to hypertensive patients who do not receive anti-hypertensive drugs.

The mean WHR of the test group was significantly higher than that of the control group because of the higher mean WHR of the women in the test group compared to the control group. If a cut-off point of 0.85 is taken as an indication of android (> 0.85) and

gynoid (< 0.85) obesity and between high and low risk for coronary heart disease (Kaplan, 1989), both groups had a high risk, with only the women in the control group below the cut-off point. The mean WHR of the six insulin-treated patients was 0.85 (± 0.1) while that of the oral hypoglycaemic agent treated patients was 0.89 (± 0.1). Zillikens and Conway (1990) found that blacks, and especially black females in the USA, have higher WHR's than whites. It has also been shown that diabetic women are more obese and have more abdominal fat than non-diabetic women (Lundgren et al., 1989). The high WHR's found in this study support these observations.

A protein/albumin (PA) ratio of 1.7 to 2.5, with the albumin content of the blood between 40 - 60 % of the total protein, is regarded as normal (Silverman et al., 1986:586). At baseline the mean PA ratio of the control group was just above 1.7, while the mean PA ratio of the test group was 1.67. The mean PA ratio of the men in both groups were below 1.7. The blood albumin values of these patients were marginally higher than normal, probably indicating that they did not suffer from malnutrition.

The characteristics of the patients one month after the study was started, can be seen in Table 6.4. Significance of differences in variables between the baseline and after one month can be seen in Table 6.23 and is indicated with an "a" in Tables 6.3 and 6.4, as well as in all the other Tables. The patients were asked to give their age with every visit and some of them could not always remember their age correctly. Differences in age can, therefore, be ignored.

A small reduction in weight took place in both the control and test groups. The weight reduction in the men was more than that of the women and smaller in the insulin-treated patients than the other patients. In the test group the mean weight reduction of both the men and women were significant. The weight reduction had the same influence on the BMI of both groups, but the change was only significant in the women of the test group.

The mean blood pressure of the control group was slightly higher after one month because of the higher mean blood pressure of the men in the group. The mean blood pressure of the test group was slightly lower, but not significantly so. The weight reduction in the test group could explain the lower blood pressure, although the diet might also have had an influence. The WHR was not calculated for the visit after one month.

The mean PA ratios were slightly higher (0.01) in both the control and test groups after one month. This could possibly be attributed to the lower protein-content of the prescribed diets, but could also be coincidental.

The characteristics of the patients after five months can be seen in Table 6.5. Significance of differences between the baseline and after five months can be seen in Table 6.23 and is indicated with a "b" in Tables 6.3 and 6.5, as well as in all the other Tables. Significance of differences between the visits after one month and after five months can also be seen in Table 6.23 and is indicated with a "c" in Tables 6.4 and 6.5, as well as in all the other Tables.

The mean weight and BMI of the control group increased from baseline to the last (fifth) visit but continued to decrease in the test group. This can probably be attributed to the test diet and good compliance and motivation in the test group. The differences in weight and BMI were statistically significant in both the men and women of the test group from baseline to the fifth visit. Although significant, the mean reductions in weight were small (1.58 and 1.11 kg in men and women respectively). The small reductions in weight of the test group had been expected. Table 6.1 shows that the prescribed energy intake of the test group was lower for men and women than that of the control group. The prescribed diets were not aimed at weight reduction as such, because the main objective of the study was to examine the effect of the traditional African diet. There were no statistically or clinically significant differences between the WHR's during

baseline and after five months. More attention should probably be paid to weight reduction in the NIDDM patients. These results indicate that attention and motivation can make a difference and that patients will comply to a given diet.

The mean blood pressures of both groups decreased from baseline to one and five months, although not significantly so. The difference in blood pressure between the control and test group was statistically significant. The blood pressures for both groups during all the visits were lower than the cut-off point for high blood pressure, although higher than normal.

There were no statistically or clinically significant differences between the PA ratios at baseline, after one or five months.

6.4.2 Nutrient intake

The mean habitual nutrient intake of the patients at baseline is compared with the 1989 RDA and recommendations of the American Diabetes Association (1990a) in Table 6.7. The nutrient intake compares very well with the nutrient intake of the random sample of NIDDM patients at the Ga-Rankuwa diabetic clinic as reported in Table 4.6. It is, therefore, reasonable to believe that the information during baseline is valid and reliable. The mean intakes of the patients in the test and control groups were very similar at baseline. The energy intake was slightly lower than the RDA, but these patients are not very active. The mean values for energy intake compare well with the values found by Silvis (1989:89) (8160 kJ for men and 9160 kJ for women) in a semi-urban diabetic population in the Orange Free State. However, the high BMI's of the patients indicate that the reported intakes were either lower than their real intakes, or that their energy needs are lower than the RDA. If the last assumption is true, it may be further proof of the "thrifty gene" of the black population as speculated by O'Dea *et al*. (1988). This means that the energy needs of the black population could be lower because of genetic adaptation to famine. It is clear that to accomplish

Table 6.7 Habitual mean daily nutrient intakes of subjects at the baseline of the study and comparison with the RDA and RADA*

		CONTROL GROUP						TEST GROUP						RDA/RADA*	
		Men n = 8		Women n = 13		Total n = 21		Men n = 9		Women n = 19		Total n = 28		Men	Women
		Mean	SD	Mean	SD	Mean	SD	Mean	SD	Mean	SD	Mean	SD		
Energy	(kJ)	11257	4340.1	8911	2951.5	9805	3630.8	11037	2868.5	8538	2132.3	9611	3258.6	12200	9250
Protein	(% of E)	17	2.7	17	3.8	17	3.4	20	4.2	17	2.8	18	3.5		
	Total (g)	108.5	39.9	90.2	36.4	97.2	37.9	131.5	47.6	84.5	22.7	101.9	43.7	63	46
	Plant (g)	61.6	36.8	37.9	12.4	46.9	26.6	38.9	14.9	34.5	9.8	36.7	12.5		
	Animal(g)	46.7	18.0	51.7	34.1	49.8	28.6	92.4	45.4	50.0	20.9	65.0	35.5		
	g/kg	1.36		1.08		1.20		1.56		1.03		1.24		0.8	0.8
Fat	(% of E)	26	7.1	34	8.4	31	8.7	35	12.6	32	9.3	33	10.0	<30	<30
	Total (g)	73.3	20.5	81.0	32.9	78.1	28.5	102.9	51.0	73.0	30.9	85.20	43.6		
	% Sat	27	4.9	28	5.4	28	5.2	30	6.6	29	5.5	29	5.6		
	% MU	33	6.1	34	7.0	34	6.6	35	4.0	34	3.4	34	3.7		
	% PU	18	5.5	21	7.5	20	6.9	22	7.6	23	6.7	23	6.9		
	P/S ratio	0.67		0.75		0.71		0.73		0.79		0.79		1	1
	Chol (mg)	262	147.5	333	171.0	306	162.6	442	231.0	305	212.3	352	227.9	<300	<300
Carbohydrates															
	(% of E)	57	8.6	48	10.5	52	10.6	44	10.9	51	10.0	49	10.3	55	55
	Total (g)	394.5	200.4	252.6	93.2	306.7	155.7	289.1	98.2	256.8	74.9	273.9	93.0		
	Fibre (g)	42.9	21.9	29.0	10.4	34.3	16.7	30.3	10.8	29.3	9.5	30.1	10.1	40	40
	Sugar (g)	1.1		5.6		3.35		5.3		3.7		4.50			
Minerals	Ca (mg)	673.1	234.9	787.9	355.7	744.2	313.8	1175.3	1439.0	765.8	387.9	926.20	862.8	800	800
	Fe (mg)	22.0	8.3	15.1	3.9	17.7	6.71	18.3	6.02	15.0	3.7	16.30	5.3	10	15
	Mg (mg)	565.1	207.2	403.2	111.3	457.2	165.7	470.9	153.2	393	94.5	426.90	137.7	350	280
	P (mg)	1617	628.6	1381	454.4	1471	525.3	1950	1057.4	1310	340.8	1553	757.8	800	800
	K (mg)	3057	872.0	2936	1010.0	2982	939.1	3120	792.3	2810	793.3	3202	1483.0	2000	2000
	Na (mg)	2501	1565.6	1828	683.0	2084	1117.9	2025	690.2	1368	542.3	1702	781.6	750	750
	Zn (mg)	17.8	5.4	14.3	7.0	15.6	6.6	21.9	8.1	13.0	4.7	16.1	7.6	15	12
	Cu (mg)	1.74	1.12	1.54	0.58	1.61	0.8	1.71	0.42	0.41	0.41	1.38	0.62	2.25	2.25
Vitamins	A (RE)	1122	264.4	1659	1610.6	1454	1285.4	3114	2269.7	1377	825.4	1911	1580.4	1000	800
	D (ug)	1.36	1.06	2.31	2.35	1.95	1.94	4.58	8.8	2.08	3.0	2.87	5.3	5	5
	E (mg)	10.79	3.6	14.79	7.2	13.27	6.3	23.89	17.6	17.27	12.3	20.0	15.0	10	8
	Thiam(mg)	2.52	1.3	1.75	0.6	2.05	1.0	2.00	0.65	1.68	0.41	1.82	0.6	1.5	1.1
	Ribof(mg)	1.56	0.38	1.62	0.6	1.60	0.5	2.61	1.75	1.58	0.58	1.95	1.2	1.7	1.3
	Niac (mg)	23.3	12.6	19.2	7.4	20.8	9.6	23.64	8.2	16.47	5.2	19.3	8.2	19	15
	B-6 (mg)	1.30	0.5	1.24	0.6	1.26	0.5	1.86	0.58	1.23	0.36	1.46	0.6	2	1.6
	Folac(mg)	297	158.1	272	65.4	281	107.1	297	124.7	235	88.2	260	108.3	200	180
	B-12 (ug)	3.61	2.1	6.3	6.7	5.26	5.5	14.4	16.5	4.1	3.6	8	10.5	2	2
	Panto(mg)	3.99	1.6	4.66	1.9	4.40	1.8	7.18	3.9	3.94	1.3	5.05	2.9	5.5	5.5
	Biot (ug)	23.7	16.4	24.8	13.0	24.4	14.0	38.3	26.2	20.2	9.2	26.7	18.8	65	65
	C (mg)	57	34.9	78	45.9	70	42.5	88	42.8	101	80.6	96	68.3	60	60

* RDA = Recommended daily allowances (National Research Council, 1989), RADA = Recommendations of the American Diabetic Association (American Diabetes Association, 1990).

% of Energy = percentage of energy

P/S ratio = Polyunsaturated to saturated fatty acid ratio

Sat = Saturated fatty acids

MU = Mono-unsaturated fatty acids

PU = Polyunsaturated fatty acids

Chol = Cholesterol

Thiam = Thiamin

Ribof = Riboflavin

Niac = Niacin

Folac = Folacin

Panto = Pantothenic acid

Biot = Biotin

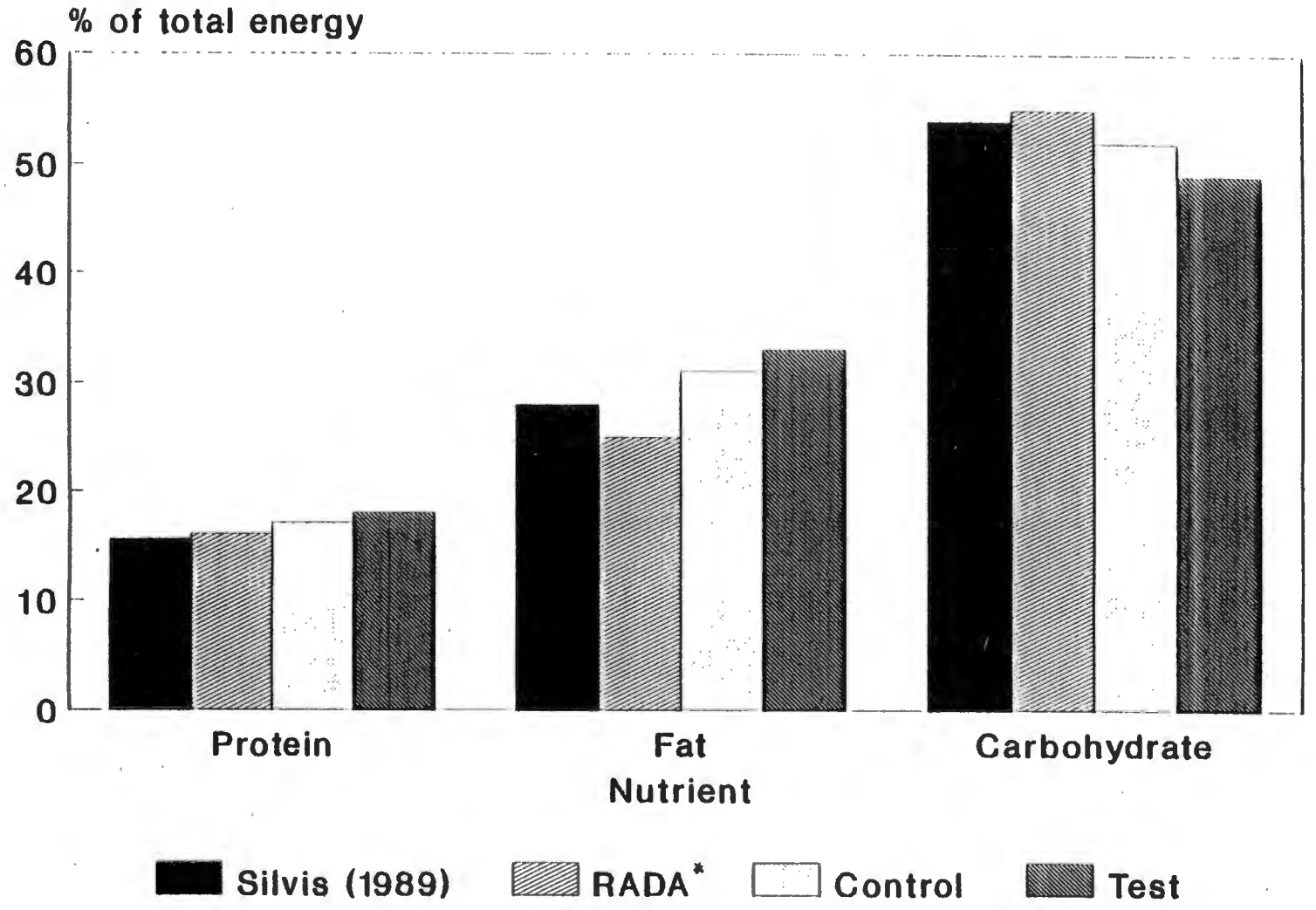
weight loss, a much lower energy intake will be necessary.

The energy distribution between protein, fat and carbohydrate was very similar to that reported for black NIDDM subjects by Silvis (1989) and is also similar to the recommendations for the diabetic diet of the American Diabetes Association (1990a) (Figure 6.2). The ratios of the control group were near those of the prudent diet for diabetics advocated by different organisations (American Diabetes Association, 1990a; Beebe et al., 1991). The ratios were slightly better than those of the diabetics at the Ga-Rankuwa clinic (Table 4.6).

The plant protein intake of the men in the control group was very high in comparison with the other patients, due to three of the men who ate a lot of baked beans in tomato sauce and legume soup. Except in their case, the animal protein intake was high. The amount of protein per kilogram weight was more than the RDA in all subjects, which indicates further, as does the PA ratio, that these diabetics were well-nourished. The recommendations for protein intakes for diabetics (12 - 20 % of energy) (Anderson & Geil, 1988:160) are also higher. The intake of this group of diabetic subjects complies well with the above-mentioned recommendations.

The percentage of fat in the diet was much higher than the fat-content of the rural African diet of approximately 16 % and 23 % in men and women respectively, as found by Vorster et al. (1990). The polyunsaturated to saturated fatty acid (P/S) ratio was much lower in all cases than the recommendations (Beebe et al., 1991). The reason is probably the high intake of animal protein products. The mean P/S ratio for both groups was higher than the ratios of 0.62 and 0.69 found by Silvis (1989:89,108).

Except in the case of the men in the test group, the cholesterol intake was near that recommended for the prudent diet but higher than the cholesterol content of the rural diet (111.5 mg) found by Vorster et al (1990). The patients did not regard food items



* Recommendations of the American Diabetes Association (1990)

Figure 6.2 Energy distribution between macro-nutrients for diabetic patients

such as eggs as a food, but rather as snacks. Two of the men in the test group had very high meat intakes, reflected in the animal protein intake, cholesterol intake and P/S ratio, as well as their lower carbohydrate intake. The fat intake indicates that these diabetic patients are in the process of westernisation. Their fat intake was not as high as that of the western diet, but much higher than that of the rural black population.

Except for the men in the test group, the carbohydrate intake of the patients was satisfactory if compared to the prudent diet, although it could be increased if compared with recommendations for the diabetic diet (Beebe et al., 1991). The carbohydrate intake also compared well with the intake of 52 - 55 % found by Silvis (1989:108). Except for the men in the control group, the DF intake was too low for a diabetic diet (American Diabetes Association, 1990a). Silvis (1989) reported a fibre intake similar to that found in this study. The DF intake of the patients should get attention because of the distinct benefits of DF (Vorster et al., 1988b). An increase in soluble as well as insoluble forms is necessary (Del Toma et al., 1988). The mean sugar intake was relatively low. Recently there has been increasing interest toward the liberalisation of sucrose in the diets of individuals with NIDDM (Hollenbeck, Coulston & Reaven, 1989). However, the black population in South Africa are not used to high sucrose intakes (Kirsten, 1977) and there is, therefore, no reason why it should be increased in the diet of this diabetic population.

The mean intakes of minerals were satisfactory, except for copper in all patients. Calcium intake was slightly too low for the patients in the control group and the women of the test group. Diabetics appear to be at increased risk of osteoporosis, presumably owing to an increased urinary calcium loss (McCarthy & Rubin, 1984). The calcium intake of the patients at the Ga-Rankuwa diabetic clinic (Table 4.6) was, however, satisfactory and the low intakes in this group could be incidental. The

copper content of most of the food items in the RIND Food Composition Tables is not given. The copper intake was therefore probably much higher than indicated in the Table. The mineral intakes of these two groups are compared with the RDA in Figure 6.3.

The mean intakes of vitamins were also satisfactory, except in the cases of vitamin D, vitamin B-6 and biotin, as found for the diabetics at the Ga-Rankuwa clinic (Table 4.6). These patients consumed relatively high amounts of animal products, which are good sources of the vitamins mentioned above. As with copper, these values are not given for many food items in the RIND Food Composition Tables. The intakes may, therefore, be higher. There may be an increased need for vitamin B-6 in diabetic patients (Mooradian & Morley, 1987). Vitamin D, which could also be classified as a hormone (Krause & Mahan, 1984), is partly obtained from synthesis when ultra-violet rays from sunlight on the skin convert 7-dehydroxycholesterol to vitamin D. These patients are often exposed to the sun and were probably not vitamin D deficient. The mean vitamin intakes of these two groups are compared with the RDA in Figure 6.4.

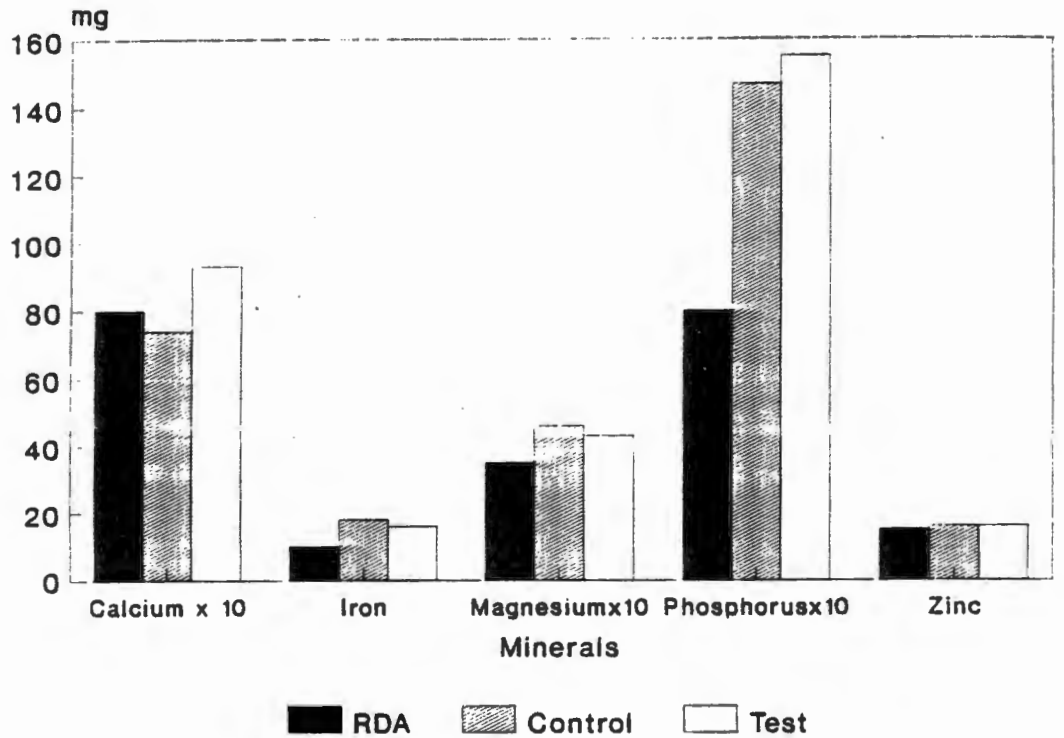


Figure 6.3 Mineral intake of patients before the study compared to the Recommended Dietary Allowances (RDA)

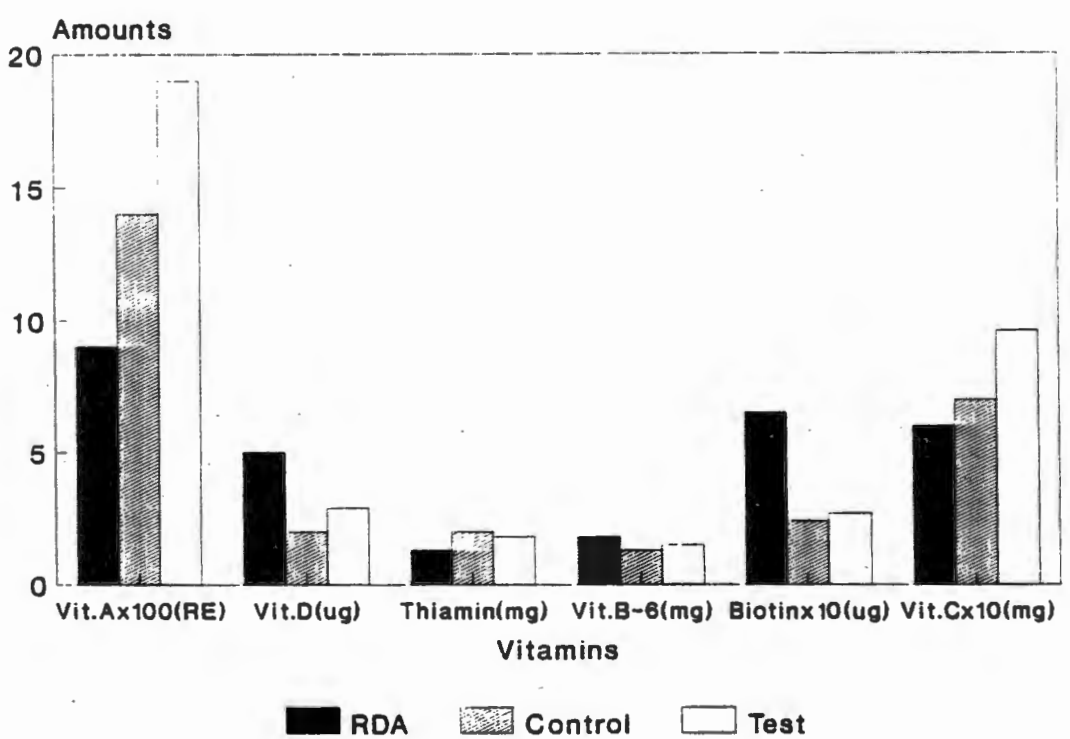


Figure 6.4 Vitamin intake of patients before the study compared to the Recommended Dietary Allowances (RDA)

The habitual mean intakes reported by the subjects during the study are given in Table 6.8. The energy intake was lower than before the study for both groups (Table 6.7), as well as the prescribed energy intake (Table 6.1), especially in the men. As could be expected, both groups also lost some weight. The weight loss was, however, not clinically significant, and did not match the expected weight loss, as will be mentioned in the discussion (Table 6.24). It was not intended in this study to test the effect of a reduced energy intake on weight loss, because none of the diets were slimming diets. The effect of a low GI diet on weight control was, however, one of the hypotheses tested in the study.

The contribution to total energy by protein, fat and carbohydrate did not change much for the control group, but improved for the test group to a mean ratio of 23:23:53. However, the ratios did not compare well with those of the prescribed diets. These ratios are compared to the prescribed ratios (Table 6.1) in Figure 6.5. This could have influenced the effect on other measured variables.

There was not a large difference between plant and animal protein intake of the control group before and during the study, but the intake of plant and animal protein of the test group changed dramatically. The plant protein intake of the men increased by 100 % and that of the women by 150 %, while the animal protein intake of the men decreased to only 38 % of the original intake and that of the women decreased to 43 % of the original intake. The protein intake per kilogram weight decreased, except in the case of the women in the test group, but both groups were still above the recommended 0.8 g/kg. The differences in animal and plant protein intakes before and during the study are compared with the prescribed intakes (Table 6.1) in Figure 6.6.

The standard deviations of the fat intakes were high because of the large variation in meat intake of both groups, probably due to large differences in the socio-economic status of the

Table 6.8 Habitual mean daily nutrient intakes of subjects during the study and comparison with the RDA and RADA*

		CONTROL GROUP						TEST GROUP						RDA/RADA*	
		Men n = 8		Women n = 13	SD	Total n = 21	SD	Men n = 9		Women n = 19	SD	Total n = 28		Men	Women
		Mean	SD	Mean	SD	Mean	SD	Mean	SD	Mean	SD	Mean	SD		
Energy	(kJ)	9156	2054	7681	3061	8129	2749	9076	2481	7809	1934	8231	2171	12200	9250
Protein	(% of E)	19	3.9	17	4.5	18	4.1	21	1.8	25	5.2	23	4.7		
	Total (g)	102	26.0	78.4	34.3	86.2	32.6	112.9	31.3	110.7	18.3	111.4	22.8	63	46
	Plant (g)	49.1	22.2	36.6	21.5	40.4	22.0	77.7	38.2	89.1	20.2	85.3	27.3		
	Animal(g)	52.8	15.8	41.3	21.4	45.5	19.4	35.1	27.8	21.6	10.5	26.1	18.8		
	g/kg	1.28		0.97		1.06		1.34		1.36		1.36		0.8	0.8
Fat	(% of E)	29	6.1	35	11.2	30	9.3	24	9.3	23	7.8	23	8.2	<30	<30
	Total (g)	70.0	20.5	71.0	35.0	69.9	29.2	57.8	28.5	47.5	21.4	51.0	23.8		
	% Sat	30	3.7	27	13.0	30	10.2	26	8.1	26	7.3	26	7.4		
	% MU	34	4.5	36	6.5	34	5.6	30	7.4	30	3.7	30	5.1		
	% PU	16	4.0	22	10.6	21	9.3	24	6.8	21	8.1	22	7.7		
	P/S ratio	0.53		0.81		0.70		0.92		0.81		0.85		1	1
	Chol (mg)	248	113	280	176	262	151	175	161.7	142	109	153	126.6	<300	<300
Carbohydrates															
	(% of E)	52	5.6	47	8.6	49	7.6	54	9.2	52	6.6	53	7.4	55	55
	Total (g)	283.1	80.2	213.2	80.3	236.2	85.3	258.6	141.1	245.3	76.7	249.70	100.1		
	Fibre (g)	32.2	10.4	26.4	9.5	28.3	9.9	34.7	13.5	28.6	8.9	30.60	10.8	40	40
	Sugar (g)	2.2		1.8		2.0		1.0		2.0		1.5			
Minerals	Ca (mg)	673	84.8	690	288.7	668	235.3	879	233.7	904	297.6	895	273.6	800	800
	Fe (mg)	20.7	9.3	15.1	9.4	17.0	9.4	33.9	14.6	40.0	8.1	38.0	10.8	10	15
	Mg (mg)	433	87.5	363	124.6	383	116.1	514	136.1	488	107.1	497	115.6	350	280
	P (mg)	1383	250.6	1196	476.8	1241	418.0	1570	410.1	1442	285.8	1485	330.1	800	800
	K (mg)	2724	283.1	2520	722.3	2570	694.8	2805	606.9	2270	632.6	2449	664.0	2000	2000
	Na (mg)	2211	1214.0	1545	722	1752	970.9	1328	538.2	982	420.3	1097	481.9	750	750
	Zn (mg)	15.9	4.7	11.0	4.6	12.7	5.1	11.9	6.2	7.7	2.2	9.1	4.3	15	12
	Cu (mg)	1.39	0.39	1.31	0.53	1.31	0.47	1.19	0.41	0.87	0.33	0.97	0.38	2.25	2.25
Vitamins	A (RE)	1222	327.0	1914	1638.7	1516	1307.3	2243	792.3	1056	654.0	1452	893.5	1000	800
	D (ug)	1.09	1.08	2.23	2.39	1.78	1.99	1.46	1.67	1.61	1.79	1.56	1.72	5	5
	E (mg)	9.69	3.99	14.27	6.96	12.72	6.22	14.62	9.24	11.23	9.36	12.36	9.28	10	8
	Thiam(mg)	1.76	0.59	1.47	0.51	1.55	0.54	1.80	0.54	1.46	0.48	1.57	0.52	1.5	1.1
	Ribof(mg)	1.44	0.24	1.37	0.55	1.36	0.46	1.43	0.50	1.25	0.47	1.31	0.48	1.7	1.3
	Niac (mg)	19.3	5.68	16.8	6.93	17.5	6.38	14.6	4.8	11.1	4.18	12.2	4.62	19	15
	B-6 (mg)	1.35	0.36	1.16	0.49	1.22	0.44	1.22	0.32	0.82	0.29	0.95	0.35	2	1.6
	Folac(mg)	246	81	245	69	244	71	234	63	203	77	213	73.3	200	180
	B-12 (ug)	3.6	1.77	5.4	6.85	4.7	5.34	3.2	3.56	2.1	2.08	2.5	2.65	2	2
	Panto(mg)	3.72	0.89	3.95	1.77	3.78	1.48	3.73	1.33	2.75	0.90	3.08	1.14	5.5	5.5
	Biot (ug)	17.1	3.71	20.3	10.50	18.5	8.79	17.44	7.50	15.6	6.67	16.21	6.89	65	65
	C (mg)	52	26.2	81	48.2	73	44.2	92	39.4	94	74.7	93	64.3	60	60

* RDA = Recommended daily allowances (National Research Council, 1989), RADA = Recommendations of the American Diabetic Association (American Diabetes Association, 1990).

% of Energy = percentage of energy

P/S ratio = Polyunsaturated to saturated fatty acid ratio

Sat = Saturated fatty acids

MU = Mono-unsaturated fatty acids

PU = Polyunsaturated fatty acids

Chol = Cholesterol

Thiam = Thiamin

Ribof = Riboflavin

Niac = Niacin

Folac = Folacin

Panto = Pantothenic acid

Biot = Biotin

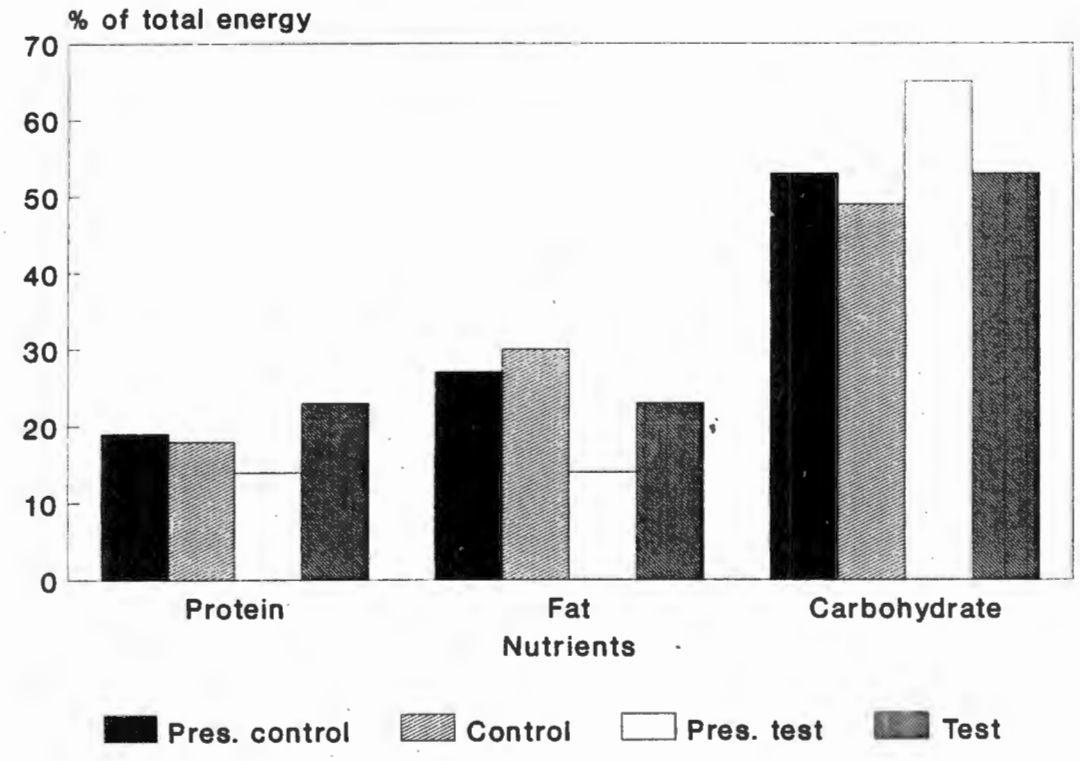


Figure 6.5 Prescribed (pres.) macro-nutrient distribution (percentage of total energy) and distribution during the study

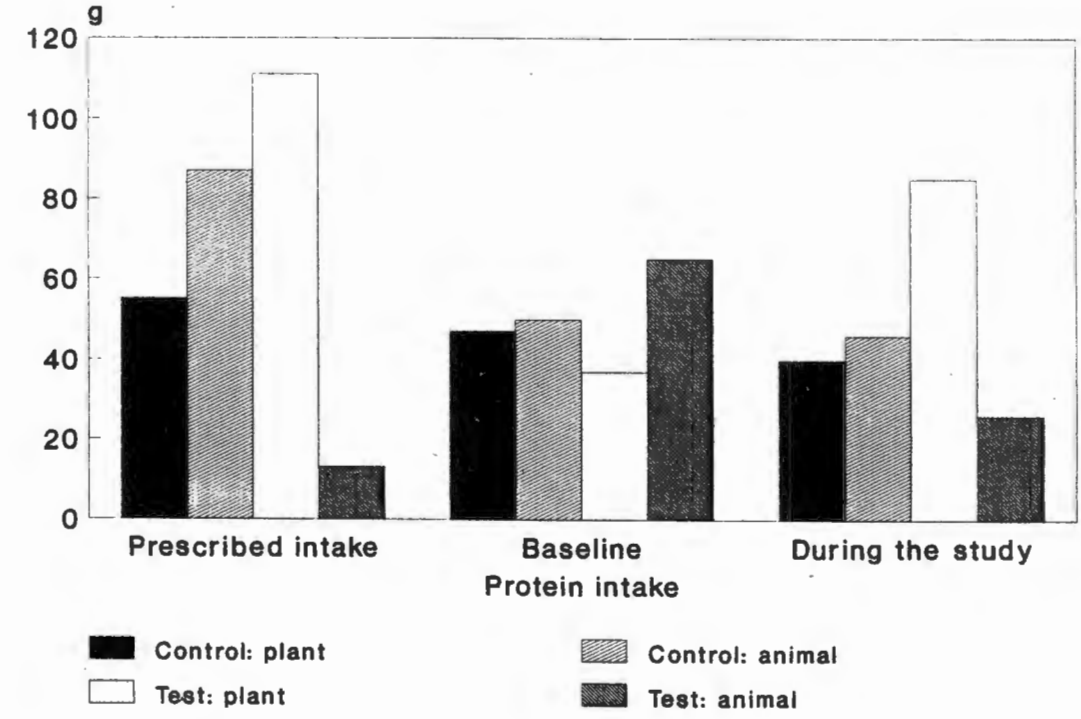


Figure 6.6 Prescribed distribution of protein intake and intakes before and during the study

patients. However, all subjects ate meat at least once a day at the baseline of the study. The ratio of polyunsaturated to saturated fat did not change much during the study and was better than the ratio of the prescribed diets. The cholesterol intake decreased, especially in the test group, but was not as low as that of the prescribed diet. The patients traditionally eat chicken every Wednesday and Sunday. Most of the patients in the test group could eat the soya products all the other days of the week, but still ate the chicken on Wednesdays and Sundays. This habit also explains the discrepancies between the protein and total fat intake of the test diet group and the prescribed test diet. The mean intake of fat of the control group was 86 % of the initial intake during the study and the mean intake of the test group was only 43 % of initial intake. The higher plant protein and lower animal protein intake of the test group was probably the main reason.

The DF intake of both groups decreased minimally, and was less than prescribed amounts. The lower energy intake might have played a role. Although the test group took in more plant protein products, analysis of the NSP (DF) of the soya product with the Englyst method (Englyst et al., 1988) showed that most of the DF was removed, probably during processing. Therefore, the soya product did not increase the fibre intake as had been expected.

Sugar intake also decreased slightly. The attention given to the patients might have made them more aware of their diets and provided them with more information. They might, therefore, have been more careful with products containing sugar, or failed to report the use of sugar-containing products, because they knew that it was expected of them not to use the products.

The RIND Food Composition Tables only give the values of a few minerals (calcium, iron, magnesium and phosphorus) and no vitamins for the soya product used in the test diet. Values given in Table 6.8 are probably underestimations. The calcium

intake of both groups decreased further during the study and was far below the prescribed intakes which were aimed at rectifying low nutrient intakes at baseline (Table 6.7). Attention should be given to the calcium intake of especially the women. The intake of the other minerals was generally higher than the RDA (especially mean iron intake of the test group because of the iron content of the soya product, although it might not be as absorbable). Zinc intakes were lower than the RDA. The prescribed diet had a zinc intake which compared well with the RDA and both groups could have had a better zinc intake if they had complied better with the prescribed diets. However, the soya product probably has a high zinc-content (Erdman & Fordyce, 1989) and the zinc intake should not be a concern. The lower meat intake of the test group could also explain the lower zinc intake, but reasons for the same trend in the control group are unclear. Their meat intake also decreased slightly as can be seen in the lower total protein intake. The low intake of copper was discussed previously. A comparison of intakes before and during the study with the intake of the prescribed diets (Table 6.1) is given in Figure 6.7.

The vitamin intakes of both groups were satisfactory, although the intake tended to be lower than before the study. A comparison of intakes before and during the study with the intake of the prescribed diets (Table 6.1) is given in Figure 6.8.

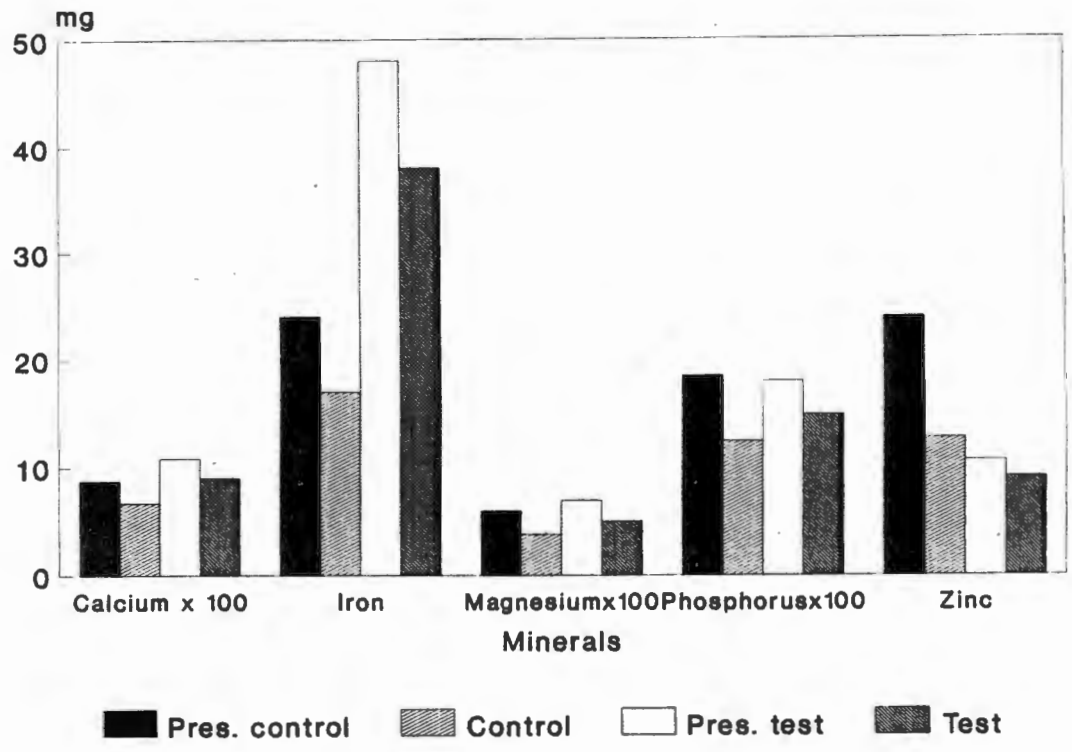


Figure 6.7 Mineral intake of patients during the study compared to the prescribed (pres.) intake (Table 6.1)

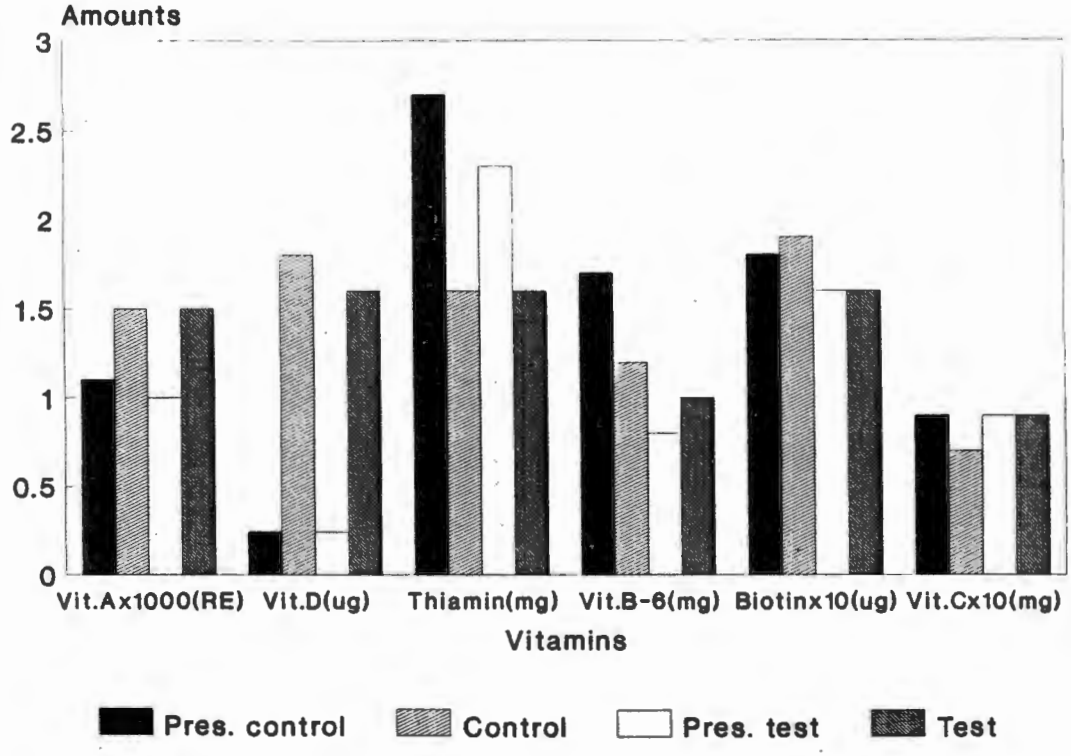


Figure 6.8 Vitamin intake of patients during the study compared to the prescribed (pres.) intake (Table 6.1)

6.4.3 Glycaemic control

For most of the variables measured in the blood (serum/plasma) there are no fixed limits with which to compare a patient's value. Different laboratories, using essentially the same methods and equipment, can for example differ in upper limits by as much as 50 % (Vermaak et al., 1988). Conventional wisdom suggests, however, that laboratories should develop reference ranges based on their own patient and disease-free populations. In practice this is usually accomplished by determining the mean ± two standard deviations on an appropriate healthy group of subjects. Many factors may also have an influence on determined values (Vermaak et al., 1988:486).

Variables measured to determine glycaemic control can be seen in Table 6.9. The standard deviations for most of the values are large due to the large differences between subjects. If only the data in this and the other tables are taken into consideration, statistically significant differences may seem illogical. However, it should be remembered that non-parametrical statistical tests were used for analysis and ranked values were therefore used to determine statistical significance of differences and correlations.

Although both variables indicate long-term glucose control, fructosamine as well as HbA_{1c} was measured because fructosamine gives an indication of control over three to six weeks, while HbA_{1c} gives an indication of control over three months (Cefalu, Parker & Johnson, 1988).

Baseline fructosamine values for the NIDDM patients in this study were much higher than those found by Silvis (1989) for black NIDDM subjects. Values for various parameters found in this study are compared with those reported by Silvis in Table 6.10. The HbA_{1c} values were also higher. Billingham et al. (1989) found a mean fructosamine value of 2.9 mmol/l and a mean HbA_{1c} value of 9.7 % in American diabetics on glibenclamide in

Table 6.9 Glycaemic control and haematological variables at baseline, after one month and at the end of the study (after five months)

	CONTROL GROUP						TEST GROUP						ORAL AGENT		INSULIN		TOTAL		Normal
	Men		Women		Total group		Men		Women		Total group								
	Mean	SD	Mean	SD	Mean	SD	Mean	SD	Mean	SD	Mean	SD	Mean	SD	Mean	SD	Mean	SD	Ranges
Fructosamine µmol/l																			<285
Baseline n = 49	446.43	131	375.42	76.0	401.60	102	357.40	67.2	389.00	93.7	378.50	86.0	384.80	96.9	406.20	49.3	387.40	92.3	
One month n = 47	405.00	135	353.75	46.7	368.80	81.5	343.80	75.7	375.80	117	365.10	105	363.60	101	386.30	49.3	366.50	95.9	
Five months x n = 45	338.17	130	374.20	46.9	360.70	85.4	331.00	107	397.63	144	374.70	134	367.10	125	386.50	59.7	369.70	118	
Protein g/l																			66.00 79.00
Baseline	74.86	8.30	74.50	4.20	74.63	5.8	74.80	4.8	74.50	4.4	74.60	4.5	74.26	4.9	77.17	4.9	74.61	5.0	
One month	75.80	3.60	72.50	6.80	73.47	6.1	73.80	4.7	74.40	4.0	74.20	4.1	73.44	4.8	77.33	4.2	73.94	4.9	
Five months	75.33	3.20	73.80	4.0	74.38	3.7	72.70	4.4	74.58	4.6	73.93	4.5	73.77	4.4	76.17	2.1	74.09	4.2	
Fruc/Prot ratio																			
Baseline	5.96		5.04		5.38		4.78		5.22		5.07		5.18		5.26		5.19		
One month	5.34		4.88		5.02		4.66		5.05		4.92		4.95		5.00		4.96		
Five months	4.49		5.07		4.85		4.55		5.33		5.07		4.98		5.07		4.99		
HbA_{1c} mmol/l																			3.50 6.00
Baseline	9.70	3.50	11.13	3.50	10.61	3.5	10.83	2.9	11.73	4.5	11.43	4.0	10.96	3.9	12.17	3.3	11.11	3.8	
One month	11.58	4.60	11.32	2.40	11.39	3.0	9.62	3.6	11.20	4.1	10.67 c	4.0	10.73	3.8	12.30	2.2	10.93	3.6	
Five months x	11.63	1.30	11.69	2.60	11.67	2.2	10.66	3.1	12.35	3.9	11.77 c	3.7	11.64	3.3	12.33	2.8	11.73	3.2	
Haemoglobin g/dl																			M 13.50 17.50
Baseline	14.30	2.0	12.76 a	0.90	13.33	1.6	14.00	1.6	13.63	1.0	13.76	1.2	13.57	1.4	13.72	1.1	13.59	1.4	F 12.00
One month	14.36	1.50	12.28 a	0.90	12.89	1.5	13.94	1.7	13.44	1.4	13.61	1.5	13.31	1.5	13.55	1.6	13.34	1.5	16.00
Five months	13.97	2.20	12.67	0.80	13.16	1.5	13.72	1.5	13.59	0.9	13.63	1.1	13.48	1.3	13.38	1.0	13.46	1.3	
Haematocrit %																			M 41.00 53.00
Baseline	43.10	5.90	39.41 b	2.40	40.77	4.3	43.58	5.3	41.01	2.9	41.93 b	4.0	41.34	4.2	42.48	3.3	41.56	4.1	F 36.00
One month	42.90	5.30	38.37	2.20	39.70	3.9	41.96	4.1	40.83	3.8	41.22	3.9	40.61	4.0	40.95	3.3	40.66	3.9	46.00
Five months	41.13	5.60	38.16 b	2.10	39.28	3.9	41.69	3.9	40.55	3.0	40.94 b	3.3	40.27	3.7	40.88	2.6	40.35	3.6	
Blood glucose mmol/l																			3.90 6.10
Baseline	11.73	5.20	11.83	4.30	11.79	4.5	9.99	3.1	10.84	3.8	10.56	3.6	10.97	4.1	11.48	2.5	11.04	4.0	
One month	10.48	3.50	10.81	3.30	10.71	3.2	9.18	3.3	10.42	4.4	10.00	4.1	10.48	3.8	8.73	3.6	10.26	3.8	
Five months x	12.42	2.90	10.37	2.80	11.14	2.9	7.71	3.4	11.80	5.8	10.39	5.4	11.08	4.7	7.90	3.5	10.66	4.6	

Fruc/Prot. ratio = fructosamine to protein ratio

HbA_{1c} = glycated haemoglobin

Blood glucose = capillary blood glucose

a = significant differences ($p \leq 0.05$) between visit 1 and 2

b = significant differences ($p \leq 0.05$) between visit 1 and 5

c = significant differences ($p \leq 0.05$) between visit 2 and 5

x = significant differences ($p \leq 0.05$) between control and test groups

comparison with the mean fructosamine value of 3.8 mmol/l and HbA_{1c} of 11.0 % of patients on oral hypoglycaemic agents in this study. It seems, therefore, that the glycaemic control of the patients in this study was poor and it emphasizes the need for more motivation and education of patients at the diabetic clinic.

There were no significant differences in mean fructosamine values for any of the two groups from baseline to one and five months. There were, however, slight reductions in fructosamine values from baseline to five months for the men but not the women in both the control and test groups. There were also no statistically significant differences between the two groups at baseline and after one month. The reduction in fructosamine was greater in the control group than in the test group at five months. If the patients who were treated with insulin are compared with those who were treated with oral hypoglycaemic agents, there were no significant differences between the two groups.

There were also no statistically significant differences between the HbA_{1c} values from baseline to one and five months, except for the total test group between one and five months. As with fructosamine, the only statistically significant difference between the two groups was during the fifth month where the increase in HbA_{1c} was more in the control than the test group. However, none of these statistically significant differences were clinically significant.

According to the HbA_{1c} values, control in the test group improved slightly initially, but in both groups control worsened over the five months. Therefore, where month to month control improved slightly according to the fructosamine values, long-term control did not improve.

The overall control according to the HbA_{1c} values is compared with those of Silvis (1989) (diabetic blacks in South Africa) and O'Connor et al., (1987) (Navajo Indians, USA) in Figure 6.9.

Table 6.10 Comparison of mean values for various variables measured at the baseline in this study and values reported by Silvis (1989) for black NIDDM subjects

	THIS STUDY						STUDY OF SLIVIS		
	Control group			Test group					
	Men	Women	Total	Men	Women	Total	Men	Women	Total
Fructosamine (mmol/l)	446.43	375.42	401.60	357.40	389.00	378.50	265.00	287.00	277.00
Glycated haemoglobin (mmol/l)	9.70	11.13	10.61	10.83	11.73	11.43	9.63	11.16	10.52
Blood glucose (mmol/l)	11.73	11.83	11.79	9.99	10.84	10.56	7.34	9.04	8.31
Protein (g/l)	74.86	74.50	74.63	74.80	74.50	74.60			75.51
Albumin (g/l)	44.86	41.92	43.00	46.40	43.95	44.77			41.18
Haemoglobin (g/dl)	14.30	12.76	13.33	14.00	13.63	13.76	9.76	9.16	9.41
Haematocrit (%)	43.10	39.41	40.77	43.58	41.01	41.93	46.11	43.82	44.75
Total cholesterol (mmol/l)	5.16	5.35	5.28	5.63	5.53	5.56	4.74	5.61	5.24
HDL-cholesterol (mmol/l)	0.87	1.05	0.98	0.98	0.83	0.98	1.02	1.02	1.02
%HDL/Total cholesterol	16.86	19.63	18.56	14.74	19.17	17.63	22.39	19.70	20.85
LDL-cholesterol (mmol/l)	3.80	4.00	3.90	4.30	4.10	4.18	3.40	4.30	3.92
Triglycerides (mmol/l)	2.46	1.70	1.98	2.48	1.73	1.98	1.57	1.49	1.52
Fibrinogen (g/l)	5.56	6.59	6.21	5.34	5.76	5.62	3.00	3.49	3.28
Sodium (mmol/l)	137.29	139.50	138.69	142.40	138.25	139.63			137.25
Potassium (mmol/l)	4.34	4.38	4.36	4.49	4.23	4.31			4.12
Chloride (mmol/l)	102.86	104.42	103.84	110.60	103.55	105.90			102.48
Carbon dioxide (mmol/l)	21.43	22.25	21.95	18.90	21.45	20.60			23.31
Phosphorus (mmol/l)	1.13	1.03	1.07	1.06	1.06	1.06			1.12
Calcium (mmol/l)	2.39	2.34	2.36	2.44	2.39	2.41			2.36
Osmolality (mosmol/l)	282.71	285.33	284.40	290.50	281.70	284.60			290.03

HDL = high-density-lipoprotein cholesterol

TC = total cholesterol

LDL = low-density-lipoprotein cholesterol

Twelve patients (40 %) of the test group and five (26 %) of the control group were very badly controlled. These percentages correlate excellently with those of Silvis who found that 34 % of the patients in her study were very badly controlled and correlates well with those of O'Connor et al. who found that 36 % of the patients had poor control. However, four patients (13 %) of the test group were in excellent control and only one of the control group (ten percent of all patients) in contrast with the 24 % of the patients in the study of Silvis who were controlled excellently.

There was a substantial difference between the mean HbA_{1c} values of patients treated with insulin and oral agents. Control worsened more in the oral agent users than in the insulin-treated patients, possibly because of a more important influence of diet on the control in the patients who use oral hypoglycaemic agents. The blood glucose values showed a small but not significant improvement from baseline to five months in both groups. Only one blood glucose value was determined each month and it can therefore not be seen as the mean value for the whole month.

There was an excellent positive correlation between the fructosamine values and the glucose and HbA_{1c} values, especially in the test group (see Tables 6.11 and 6.12). The glycated haemoglobin values did not correlate as strongly with the glucose values. These trends could be expected, because fructosamine values are indicators of glycaemic control over two weeks, which are nearer to day to day glucose control than the 120 days of HbA_{1c}. The fructosamine and HbA_{1c} levels did not correlate well with any of the other variables.

Haematocrit values were on the lower end of the normal range for all the patients and lower than the values reported by Silvis (1989) (see Table 6.10). Small but statistically significant changes in mean haematocrit values occurred between baseline and five months in the women of the control group, and in the total test group.

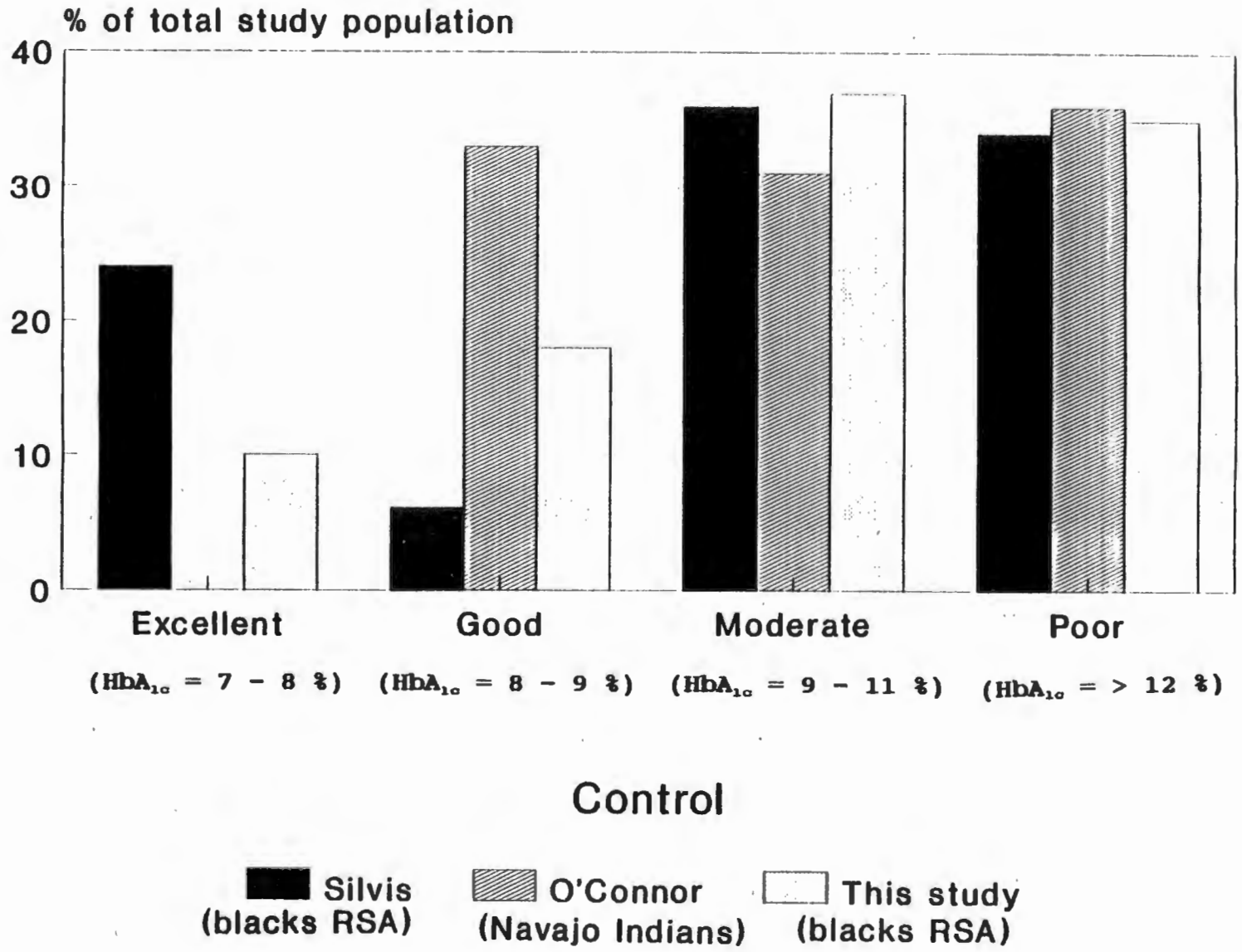


Figure 6.9 Distribution of glycaemic control according to HbA_{1c} values in different studies

Table 6.11 Correlations between various variables in the test group

	Glucose	HbA_{1c}	Fibrin	BP sis	BPdias	Choles	Triglc	HDL	LDL	Apo A	Apo B	WHR
Apolipoprot.A												
Baseline												
Spearman	0.27	0.49 bc	−0.04	−0.17	−0.13	−0.11	−0.33	0.82 abcx	−0.23		−0.26	−0.26
p-value	0.15	0.01	0.82	0.38	0.48	0.57	0.08	0.001	0.23		0.16	0.17
One month												
Spearman	0.03	0.38 bd	−0.13	−0.01	−0.06	−0.09	−0.36	0.76 dbx	−0.12		−0.36	
p-value	0.89	0.04	0.50	0.97	0.77	0.64	0.05	0.001	0.52		0.05	
Five months												
Spearman	0.38	0.40 dc	0.29	−0.17	−0.15	−0.05	−0.25	0.86 dcx	−0.20		−0.33	−0.52 a
p-value	0.04	0.03	0.13	0.39	0.43	0.80	0.20	0.001	0.31		0.08	0.01
Apolipoprot.B												
Baseline												
Spearman	0.05	−0.11	−0.20	0.24	−0.001	0.73 abcx	0.42 abc	−0.28	0.73 abcx	−0.26		0.17
p-value	0.78	0.55	0.29	0.19	1.00	0.001	0.02	0.13	0.001	0.16		0.36
One month												
Spearman	0.04	0.04	−0.11	0.31	0.44	0.86 abdx	0.43 db	−0.29	0.85 dbx	−0.36		
p-value	0.82	0.83	0.58	0.10	0.01	0.001	0.02	0.12	0.001	0.05		
Five months												
Spearman	0.24	0.003	−0.04	0.12	0.12	0.80 adcx	0.69 dcx	−0.44	0.83 adcx	−0.33		0.09
p-value	0.22	0.99	0.84	0.52	0.52	0.001	0.001	0.02	0.001	0.08		0.66
Body mass index												
Baseline												
Spearman	−0.07	−0.03	0.10	0.11	0.31	−0.33	−0.18	0.05	−0.30	−0.03	−0.17	−0.37
p-value	0.71	0.88	0.59	0.58	0.09	0.07	0.33	0.80	0.10	0.87	0.37	0.04
One month												
Spearman	−0.05	−0.15	−0.08	−0.05	0.05	−0.20	0.06	0.22	−0.23	0.29	−0.34	
p-value	0.78	0.44	0.68	0.79	0.81	0.30	0.74	0.24	0.21	0.12	0.06	
Five months												
Spearman	−0.19	−0.08	0.48	0.10	−0.05	−0.33	−0.14	0.47	−0.41	0.42	−0.41	−0.32
p-value	0.32	0.69	0.01	0.60	0.79	0.08	0.48	0.01	0.03	0.02	0.03	0.10
Fructosamine												
Baseline												
Spearman	0.72 abcx	0.76 abcx	−0.32	0.06	0.12	0.06	0.01	0.26	0.02	0.35	0.11	−0.21
p-value	0.001	0.001	0.09	0.74	0.52	0.74	0.97	0.17	0.93	0.06	0.57	0.26
One month												
Spearman	0.61 dbx	0.82 abdx	−0.002	0.05	−0.03	0.36	−0.08	0.38 d	0.32	0.32	0.15	
p-value	0.00	0.001	0.99	0.81	0.88	0.05	0.66	0.04	0.08	0.09	0.42	
Five months												
Spearman	0.75 dcx	0.69 dcx	−0.04	−0.13	0.02	0.22	−0.26	0.38 d	0.16	0.34	0.08	−0.27
p-value	0.001	0.001	0.84	0.50	0.92	0.25	0.18	0.04	0.42	0.07	0.68	0.17
HbA_{1c}												
Baseline												
Spearman	0.54 bc		−0.10	−0.08	−0.004	−0.08	−0.06	0.42 b	−0.13	0.49 b	−0.11	−0.10
p-value	0.001		0.62	0.67	0.98	0.69	0.75	0.02	0.48	0.01	0.55	0.60
One month												
Spearman	0.55 bd		−0.13	−0.04	−0.04	0.28	−0.25	0.43 b	0.28	0.38 bd	0.04	
p-value	0.001		0.49	0.85	0.84	0.13	0.18	0.20	0.13	0.04	0.83	
Five months												
Spearman	0.64 dcx		0.02	0.09	0.17	0.16	−0.07	0.35	0.09	0.40 d	0.003	−0.29
p-value	0.001		0.93	0.65	0.38	0.41	0.73	0.07	0.64	0.03	0.99	0.13

Triglyc = triglycerides
Apo A = Apolipoprotein A
Apo B = Apolipoprotein B
Fibrin = fibrinogen
BP dias = diastolic blood pressure
BP sis = systolic blood pressure

a = good correlation between both test and control groups
b = good correlations during baseline and after one month
c = good correlations during baseline and after five months

Table 6.12 Correlations between various variables in the control group

	Glucose	HbA_K	Fibrin	BP sis	BPdias	Choles	Triglc	HDL	LDL	Apo A	Apo B	WHR
Apolipoprot.A												
Baseline												
Spearman	0.02	0.11	−0.25	0.19	0.20	0.17	−0.20	0.58 ac	0.12		−0.11	−0.34
p-value	0.92	0.66	0.31	0.43	0.41	0.48	0.41	0.01	0.61		0.67	0.16
One month												
Spearman	0.05	−0.10	0.23	−0.24	−0.53	0.56	−0.07	0.38	0.52		0.47	
p-value	0.84	0.69	0.38	0.36	0.03	0.02	0.80	0.13	0.03		0.06	
Five months												
Spearman	−0.04	−0.21	0.07	−0.03	0.07	0.31	−0.61 x	0.80 cx	0.24		0.23	−0.55 a
p-value	0.89	0.43	0.79	0.93	0.79	0.24	0.01	0.001	0.37		0.40	0.04
Apolipoprot.B												
Baseline												
Spearman	0.10	0.23	0.13	0.12	0.09	0.78 abcx	0.80 ax	−0.36	0.67 abcx	−0.11		0.06
p-value	0.68	0.34	0.59	0.62	0.71	0.001	0.001	0.14	0.001	0.67		0.80
One month												
Spearman	−0.05	−0.20	0.19	0.22	0.12	0.55 abd	0.29	−0.16	0.56 bd	0.47		
p-value	0.86	0.44	0.46	0.40	0.66	0.02	0.26	0.54	0.02	0.06		
Five months												
Spearman	0.57	0.13	0.22	0.20	0.13	−0.74 adcx	0.38	−0.19	0.67 adcx	0.23		0.15
p-value	0.02	0.62	0.42	0.46	0.62	0.001	0.14	0.47	0.001	0.40		0.61
Body mass index												
Baseline												
Spearman	−0.07	0.33	0.36	−0.02	−0.22	0.02	0.18	−0.13	0.003	−0.21	0.15	−0.35
p-value	0.76	0.17	0.13	0.93	0.36	0.94	0.47	0.60	0.99	0.40	0.53	0.14
One month												
Spearman	−0.17	0.12	0.11	0.34	0.23	0.27	−0.40	0.07	0.30	−0.11	−0.01	
p-value	0.50	0.66	0.68	0.19	0.38	0.29	0.11	0.78	0.24	0.67	0.98	
Five months												
Spearman	−0.47	0.15	0.05	0.35	0.13	−0.07	−0.08	0.16	−0.01	−0.16	−0.21	−0.23
p-value	0.07	0.57	0.07	0.19	0.62	0.80	0.78	0.56	0.96	0.55	0.44	0.43
Fructosamine												
Baseline												
Spearman	0.19 ax	0.55 ab	−0.08	−0.32	−0.41	0.18	0.05	−0.03	0.17	−0.22	0.15	−0.04
p-value	0.001	0.01	0.75	0.18	0.08	0.46	0.82	0.92	0.47	0.37	0.55	0.86
One month												
Spearman	0.46	0.90 abx	−0.04	0.16	0.29	−0.37	−0.20	−0.03	−0.30	0.01	−0.21	
p-value	0.06	0.001	0.88	0.54	0.25	0.15	0.44	0.90	0.25	0.96	0.42	
Five months												
Spearman	0.42	0.36	−0.29	−0.07	0.12	0.13	0.09	−0.004	0.24	0.20	0.40	−0.30
p-value	0.11	0.17	0.27	0.80	0.65	0.62	0.73	0.99	0.37	0.47	0.12	0.30
HbA_{1c}												
Baseline												
Spearman	0.53		−0.02	−0.38	−0.41	0.10	0.34	−0.07	0.03	0.11	0.23	−0.36
p-value	0.02		0.93	0.11	0.08	0.67	0.15	0.76	0.90	0.66	0.34	0.13
One month												
Spearman	0.34		0.07	0.27	0.43	−0.26	−0.13	−0.05	−0.21	−0.10	−0.20	
p-value	0.18		0.78	0.29	0.08	0.31	0.63	0.84	0.42	0.69	0.44	
Five months												
Spearman	0.16		−0.10	0.14	−0.04	0.04	0.02	−0.16	−0.004	−0.21	0.13	−0.39
p-value	0.56		0.71	0.60	0.87	0.88	0.94	0.56	0.99	0.43	0.62	0.17

HbA_{1c} = glycated haemoglobin
Fibrin = fibrinogen
BP dias = diastolic blood pressure
BP sis = systolic blood pressure
Triglyc = triglycerides
Apo A = Apolipoprotein A
Apo B = Apolipoprotein B
WHR = waist-to-hip circumference ratio

b = good correlations during baseline and after one month
c = good correlations during baseline and after five months
d = good correlations after one and five months
x = correlation ≥ 0.6, p ≤ 0.5

Mean total haemoglobin values were within the normal range for men and women in both groups.

6.4.4 Lipid profiles

The serum lipid values measured before and during the study are given in Table 6.13.

The mean cholesterol values for both groups were within the normal ranges for this age group but slightly higher for men and lower for women than the values reported by Silvis (1989) (see Table 6.10). Values for black diabetics reported by other researchers varied from 4.06 to 5.77 mmol/l (Silvis, 1989:123). Values for healthy black rural males are, however, 4.1 (Vorster et al., 1987a) and 4.0 mmol/l (Walker et al., 1978), which are lower than the values found for the diabetic men. It seems that these black diabetics did not have markedly increased cholesterol values and that they had lower values than reported for white diabetics of the same sex and age groups (Billingham et al., 1989; Ohlson et al., 1988; Vorster et al., 1988b) (see Figure 6.10).

Cholesterol values decreased slightly from baseline to one and five months for men and women in both groups. The decrease was not clinically significant and only statistically significant for the men in the test group and for the total test group, from baseline to one month. If the food intake as reported by the subjects can be taken as valid, it seems that, although the reported fat and cholesterol intakes of the test group were lower and the plant protein intake higher than these of the control group, the diet did not have a significant influence on total serum cholesterol values of the patients.

The baseline HDL-cholesterol values for both groups, although within normal ranges, were slightly lower than those found by Silvis (1989) (see Table 6.10). The values were below the 1.37 mmol/l, shown to be the cut-off point for high and low risk for

Table 6.13 Serum lipids and plasma fibrinogen at baseline, after one month and at the end of the study (after five months)

	CONTROL GROUP						TEST GROUP						ORAL AGENT		INSULIN		TOTAL		Normal Ranges
	Men		Women		Total group		Men		Women		Total group								
	Mean	SD	Mean	SD	Mean	SD	Mean	SD	Mean	SD	Mean	SD	Mean	SD	Mean	SD	Mean	SD	
Tot.Cholesterol mmol/l																			3.90 6.50
Baseline n = 49	5.16	1.20	5.35	1.30	5.28	1.2	5.63 a	1.0	5.53	1.5	5.56 a	1.3	5.44	1.3	5.55	0.4	5.45	1.3	
One month n = 47	4.14	0.30	5.40	1.10	5.03	1.1	5.20 a	0.9	5.36	1.5	5.31 a	1.3	5.12	1.3	5.79	1.0	5.21	1.3	
Five months n = 45	4.96	0.70	4.78	0.90	4.85	0.8	5.37	1.1	5.29	1.2	5.30	1.2	5.14	1.1	5.26	1.0	5.20	1.1	
HDL-cholesterol mmol/l																			0.65 1.72
Baseline	0.87	0.10	1.05	0.20	0.98	0.2	0.83	0.2	1.06	0.3	0.98	0.3	0.96	0.2	1.15	0.3	0.98	0.2	
One month	0.84	0.10	1.11	0.20	1.03	0.2	0.76	0.2	1.05	0.2	0.98	0.3	0.95	0.2	1.21	0.3	0.98	0.2	
Five months	0.78	0.20	1.19	0.30	1.03	0.3	0.77	0.2	1.08	0.2	0.97	0.3	0.96	0.2	1.25	0.4	1.00	0.3	
%HDL:TC																			>18
Baseline	16.86		19.63		18.56		14.74		19.17		17.63		17.65		20.72		17.98		
One month	20.29		20.56		20.48		14.62		19.59		18.46		18.55		20.90		18.81		
Five months	15.73		24.90		21.24		14.34		20.42		18.30		18.68		23.76		19.23		
LDL-cholesterol mmol/l																			M 1.55 5.57 F 1.55 5.70
Baseline	3.80 b	1.20	4.00 b	1.10	3.90 ab	1.10	4.30 ab	1.00	4.10 ab	1.50	4.18 ab	1.30							
One month	2.87 c	0.30	3.95 c	1.10	3.63 ac	1.10	4.05 a c	0.90	3.96 a c	1.50	3.99 a c	1.30							
Five months	3.48 bc	0.80	3.32 bc	0.90	3.38 cb	0.80	4.23 bc	1.10	3.88 bc	1.20	4.00 bc	1.10							
Triglycerides mmol/l																			0.34 1.70
Baseline	2.46	1.50	1.70	1.00	1.98	1.2	2.48	1.0	1.73	0.7	1.98	0.9	2.00	1.1	1.86	0.6	1.98	1.0	
One month	2.15	0.90	1.75	1.10	1.87	1.1	1.98	1.0	1.79	0.7	1.85	0.8	1.94	0.9	1.26	0.5	1.86	0.9	
Five months x	3.55	3.20	1.39	0.70	2.20	2.2	1.89	1.1	1.63	0.6	1.70	0.8	1.97	1.5	1.41	0.5	1.89	1.5	
Apolipoprot A g/l																			1.00 2.00
Baseline x	1.25	0.10	1.37	0.10	1.33	0.1	1.24	0.3	1.40	0.3	1.35	0.3	1.32	0.2	1.50	0.3	1.34	0.2	
One month	1.33	0.30	1.47	0.20	1.43	0.2	1.16	0.3	1.50	0.3	1.39 c	0.3	1.39	0.3	1.51	0.3	1.40	0.3	
Five months	1.25	0.20	1.43	0.20	1.36	0.2	1.11	0.2	1.40	0.2	1.30 c	0.2	1.29	0.2	1.54	0.2	1.32	0.2	
Apolipoprot B g/l																			0.70 1.50
Baseline	1.30	0.30	1.23	0.30	1.26	0.3	1.50	0.3	1.59	1.3	1.56	1.1	1.46	0.9	1.31	0.2	1.44	0.9	
One month	1.21	0.20	1.19	0.30	1.20	0.2	1.37	0.3	1.29	0.4	1.32	0.4	1.28	0.3	1.27	0.2	1.27	0.3	
Five months	1.37	0.50	1.15	0.20	1.23	0.4	1.31	0.3	1.27	0.4	1.28	0.3	1.27	0.4	1.25	0.2	1.27	0.3	
Fibrinogen g/l																			2.00 4.00
Baseline	5.56	1.60	6.59 ab	1.20	6.21 a	1.4	5.34	1.7	5.76	1.6	5.62 b	1.6	5.80	1.4	6.24	2.8	5.85	1.6	
One month	3.90	2.40	5.98 a	0.90	5.37 a	1.7	5.60	1.8	5.70	1.0	5.67	1.2	5.44	1.4	6.37	0.9	5.56	0.4	
Five months	5.23	1.90	4.90 b	1.70	5.02	1.7	4.36	1.4	5.08	1.4	4.83 b	1.5	4.80	1.4	5.51	2.2	4.90	1.5	

Tot. cholesterol = total cholesterol

% HDL:TC = percentage of HDL-cholesterol/total cholesterol

a = significant differences ($p \leq 0.05$) between visit 1 and 2

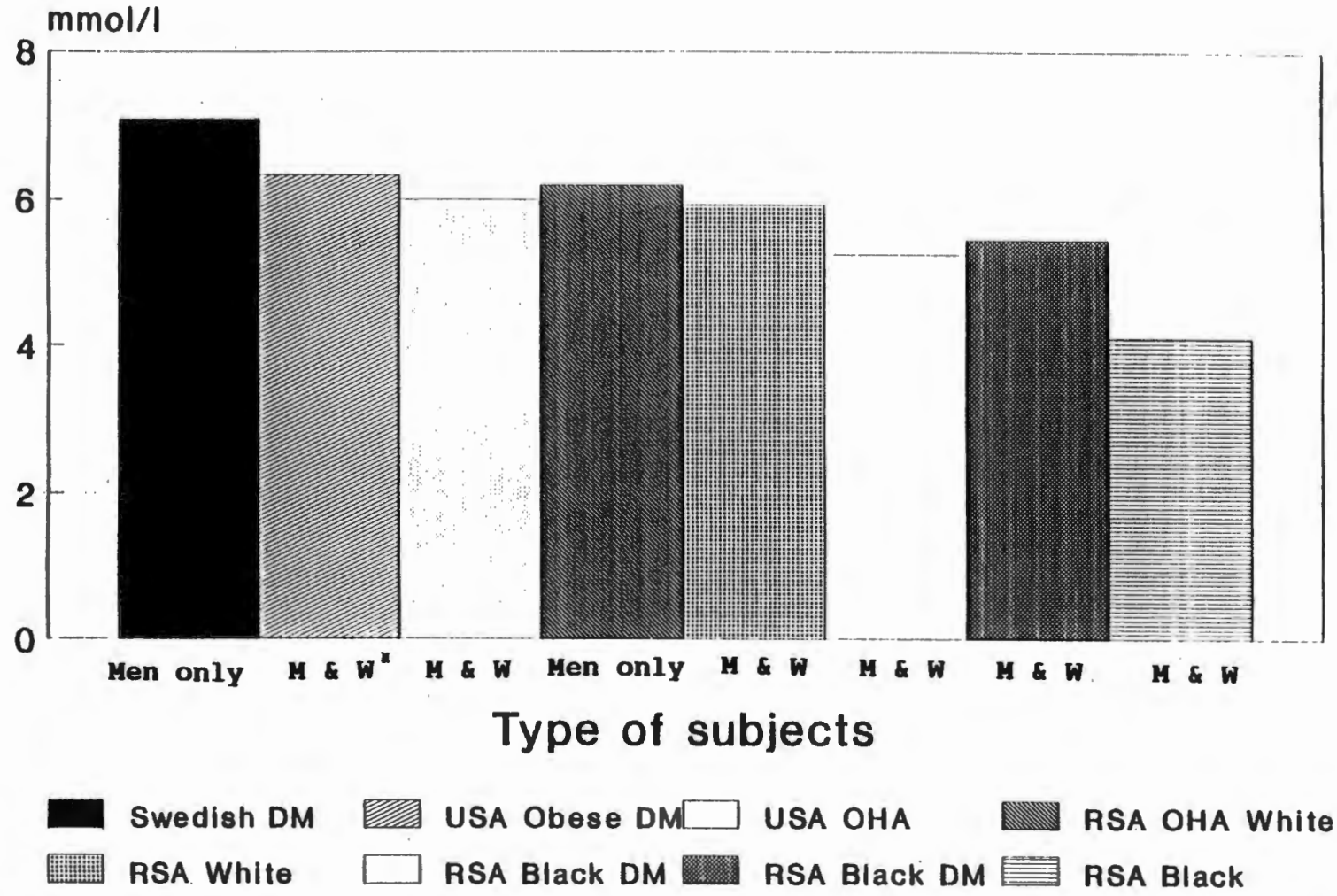
b = significant differences ($p \leq 0.05$) between visit 1 and 5

Apolipoprot. A = Apolipoprotein A

Apolipoprot. B = Apolipoprotein B

c = significant differences ($p \leq 0.05$) between visit 2 and 5

x = significant differences ($p \leq 0.05$) between control and test group



Swedish DM = Ohlson et al., 1988 (mean age 54)
USA Obese DM = Uusitupa et al., 1990 (mean age 51)
USA OHA = Billingham et al., 1989 (mean age 58)
RSA OHA White = Vorster et al., 1988 (mean age 57)
RSA White = Vermaak et al., 1988 (adults, all ages)
RSA Black DM = Silvis, 1989 (mean age 53)
RSA Black DM = This study (mean age 54)
RSA Black = Vorster et al., 1987 (mean age 28)
M & W* = men and women

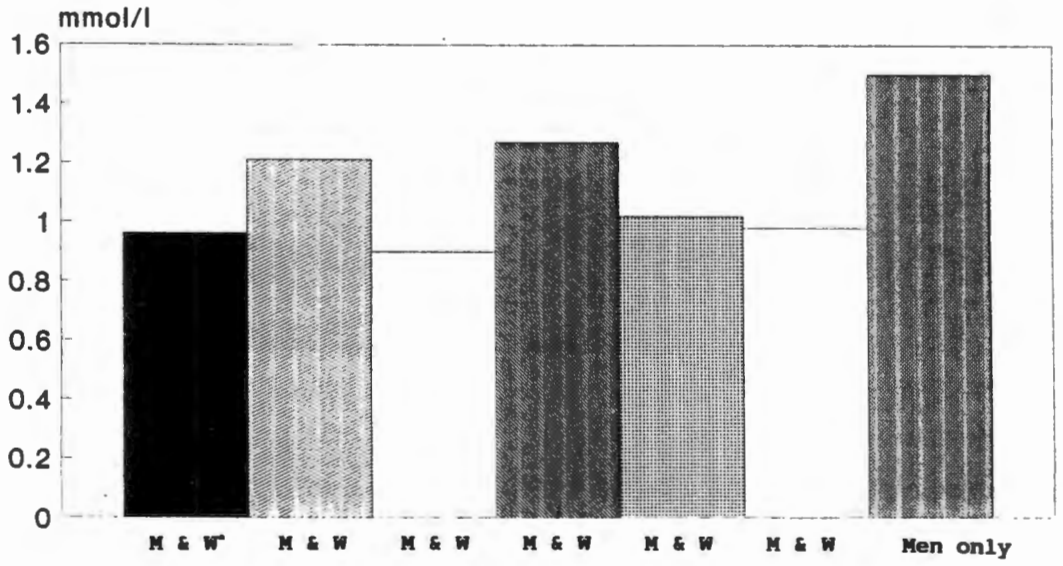
Figure 6.10 Mean total serum cholesterol values from different studies

coronary heart disease (Anon, 1990). Mean values for women of both groups were higher than those of the men. In both this study and the study of Silvis, the HDL-cholesterol values of the diabetic patients were lower than those of healthy blacks (Vorster et al., 1987a). This trend can also be seen in healthy and diabetic whites (Vermaak et al., 1988; Vorster et al., 1988b) (see Figure 6.11).

The percentage HDL:total cholesterol was lower in this study than in the study of Silvis (1989). The mean percentages in the control and test group and for both the men and women were lower than 20 %, which is regarded as the cut-off point of the ratio for protection against cardiovascular complications (Miller, 1980). The mean percentage HDL:total cholesterol in healthy rural black men was found to be 37 % in the study of Vorster et al. (1987a) and that for white diabetics on oral hypoglycaemic agents 15 % (Vorster et al., 1988b), compared to the 21 % of healthy whites (Vermaak et al., 1988) of the same age. The same trend of low ratios and increased risk for cardiovascular complications are therefore seen in blacks as in whites.

The HDL-cholesterol values of the insulin-treated subjects were much higher than those of the patients on oral hypoglycaemic agents. This was also found by Vorster et al. (1988b) in white diabetics in South Africa and Laakso and Pyörälä (1988) in Finnish diabetics (see Figure 6.12). It seems that the NIDDM patients on oral medication have a more unfavourable lipid pattern with respect to risk of coronary heart disease.

There were no significant differences between the HDL-cholesterol values at baseline, neither after one month and at the end of the study for any of the groups, nor between groups. The same applies to the percentage HDL:total cholesterol. As with total cholesterol, the diet did not have any significant effect on these lipid values. According to Reaven (1987) diabetic women usually have a greater lowering of HDL-cholesterol levels than diabetic men. This may explain the increase in coronary heart

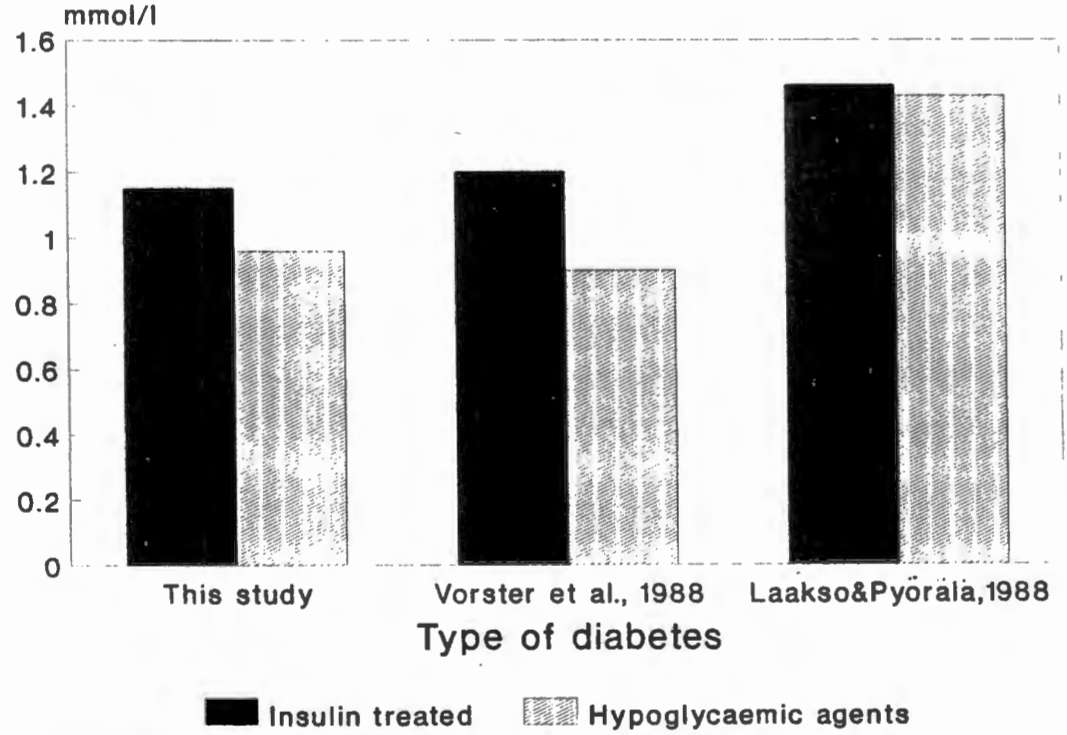


USA ObeseDM USA OHA RSA OHAWhite RSA White

RSA Black DM RSA Black DM RSA Black *M & W = Men and women

USA ObeseDM = Uusitupa et al., 1990 (mean age 51)
USA OHA = Billingham et al., 1989 (mean age 58)
RSA OHA White = Vorster et al., 1988 (mean age 57)
RSA White = Vermaak et al., 1988 (adults, all ages)
RSA Black DM = Silvis, 1989 (mean age 53)
RSA Black DM = This study (mean age 54)
RSA Black = Vorster et al., 1987 (mean age 28)

Figure 6.11 Mean HDL-cholesterol values from different studies



Insulin treated Hypoglycaemic agents

This study = Men and women, mean age 54 years, black subjects
Vorster et al., 1988 = Men and women, mean age 57 years, white subjects
Laakso & Pyörälä, 1988 = Men and women, mean age 56 years, white subjects

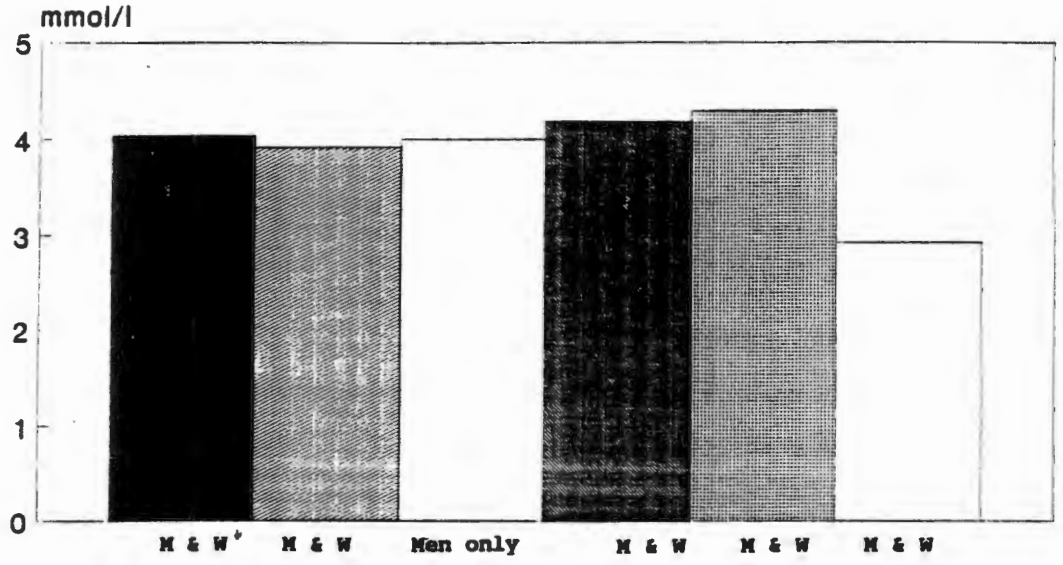
Figure 6.12 Differences in HDL-cholesterol values of patients on insulin and oral hypoglycaemic agents

disease in diabetic women compared to men. However, in this study the women in both groups had higher HDL-cholesterol values than the men and their values increased in contrast with those of the men during the study. Walker et al. (1979) suggests that there is a genetic difference in HDL-cholesterol of blacks compared to whites. This difference may possibly explain the higher HDL-values in the black women.

Values for LDL-cholesterol in this study and the study of Silvis (1989) compare well (see Table 6.10) and are within the normal ranges. The same values in subjects of the same age were also found for diabetics on oral hypoglycaemic agents by Vorster (1988b) in white South Africans and slightly higher values were found by Laakso and Pyörälä (1988) in Finnish and Billingham et al. (1989) in American subjects. Iwai et al. (1990) found lower values in Japanese subjects (see Figure 6.13). Vorster et al. (1987a) found lower values (mean value of 2.1 mmol/l) in healthy black men in a rural area.

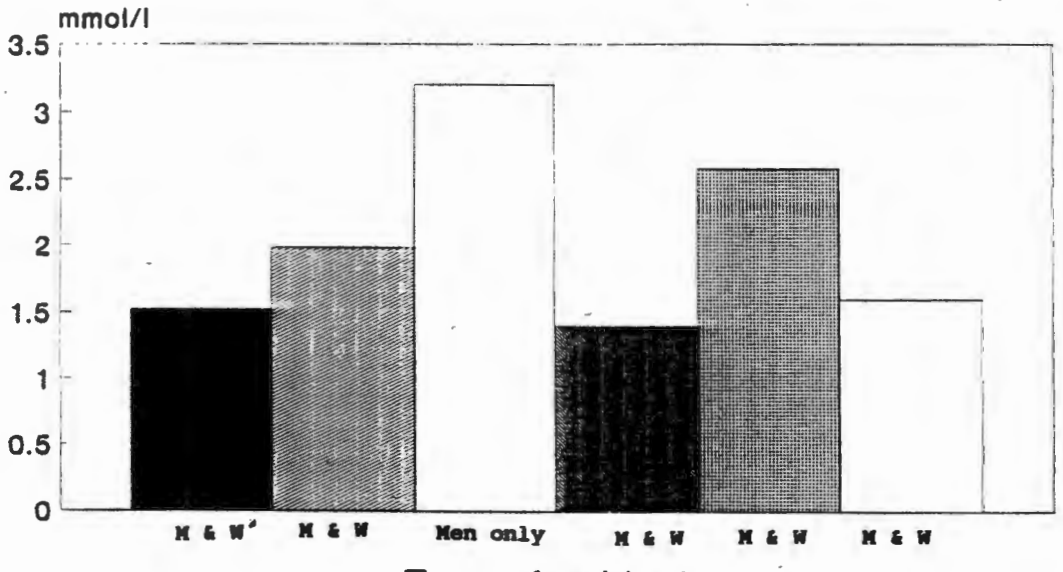
There was a statistically significant decrease in LDL-levels in both the men and women in the control as well as the test group from baseline to one month and a slight increase from one to five months. Values at five months were, however, significantly lower than values at baseline. The decrease was, however, not clinically significant and could not have been caused by the test diet because the decrease was also significant in the control group. The attention paid to the diet and the motivation of the patients might have played a role in the reduction of fat intake as could be seen from Table 6.7 (diet at baseline) and Table 6.8 (diet during the study). The increase in dietary fibre intake which may lead to lower LDL-cholesterol levels (Anderson & Geil, 1988; Hagander et al., 1989), could also be responsible.

The triglyceride values in the patients in this study are higher than those reported by Silvis (1989) and of American subjects on oral hypoglycaemic agents reported by Billingham et al. (1989) (Figure 6.14). However, Vorster et al. (1988b) and Laakso and



RSA Black DM = This study (mean age 54)
RSA Black DM = Silvis, 1989 (mean age 53)
RSA Black = Vorster et al., 1987 (mean age 28)
Finnish DM = Laakso & Pyörälä, 1988 (mean age 57)
USA DM = Billingham et al., 1989 (mean age 58)
Japanese DM = Iwai et al., 1990 (mean age 58)

Figure 6.13 LDL-cholesterol levels found in various studies



RSA Black DM = Silvis, 1989 (mean age 53)
RSA Black DM = This study (mean age 54)
RSA Black = Vorster et al., 1988 (mean age 28)
RSA White DM = Vermaak et al., 1988 (all ages)
Finnish DM = Laakso & Pyörälä, 1988 (mean age 56)
USA DM = Billingham et al., 1989 (mean age 58)

Pyörälä (1988) found higher values in white South African diabetics on oral hypoglycaemic agents and Finnish NIDDM subjects respectively. In healthy black South African men from a rural area the triglyceride levels were lower (Vorster et al., 1987a). The mean triglyceride values in this study are, therefore high in comparison with those of other South African blacks, but low in comparison with levels in white diabetic subjects.

Elevated triglyceride levels are often the result of poorly controlled blood glucose values and obesity (Hagan & Wylie-Rosett, 1989). It was seen in these patients that glycaemic control was poor and all of them could be classified as overweight or obese. These factors could, therefore be the main reasons for the observed high triglyceride levels. The subjects who took part in the study of Silvis (1989) are mainly from the Orange Free State and therefore from the Northern-Sotho tribe, while most of the subjects in this study are Tswanas from the Pretoria area. A genetic difference in these two tribes is not impossible. The nutrient compositions of the baseline diets in the two studies did not differ much. However, the differences in mean HbA_{1c} levels, suggest difference in "success" of treatment between the two clinics.

There were no statistically or clinically significant differences in the triglyceride values in the test or control group from baseline to five months. However, the mean triglyceride values in the control group increased while the mean triglyceride values in the test group decreased with the result that there was a statistically significant difference between the control and the test group after five months. Glycaemic control was not significantly different in the control and test group. There was a slight but not significant decrease in weight in the test group (but not in the control group). There should, therefore, be another reason for the significant difference in triglyceride levels. The higher intake of legumes in the test group and resultant lower GI of the diet, could have been responsible (Erdman & Fordyce, 1989) as well as the lower animal protein

intake (Simpson et al., 1981).

No data on the apolipoprotein A-status of black diabetics in South Africa could be found. However, the apolipoprotein A values of patients in this study (1.24 g/l for men and 1.39 g/l for women) compared well with the data of Billingham et al. (1989) (1.26 g/l for NIDDM men and 1.5 g/l for women). Iwai et al. (1990) found a mean apolipoprotein A-value of 1.65 g/l in Japanese NIDDM subjects. Concentrations of apolipoprotein A of these black patients were, therefore, higher in females but within the normal range for both sexes.

At the baseline of the study, the apolipoprotein A levels differed significantly between the control and test groups. The test group had the highest value, but the difference was not clinically significant. No other statistically significant differences were found during the study, except for the mean value of the test group that decreased after one month to the last visit (after five months). Apolipoprotein A is very sensitive to dietary changes (Weinberg, Dantzker & Patton, 1990) and the lower fat content of the test diet could explain the lower apolipoprotein levels at the end of the study.

The apolipoprotein A levels in the insulin-treated subjects were higher than the levels in the other patients. It increased during the five months of the study, while the levels in the oral hypoglycaemic agent users decreased.

No data on the apolipoprotein B-status of black diabetics in South Africa could be found. The apolipoprotein B values in this study (1.26 g/l in control group, 1.56 g/l in test group) compare well with the 1.51 g/dl found by Kubisz, Parizek and Cronberg (1986) in IDDM patients, but are higher than the levels of 0.94 g/l in oral hypoglycaemic users and 0.91 g/l in insulin-treated patients in Japan (Iwai et al., 1990). Increased apolipoprotein B levels are associated with decreased HDL-cholesterol and increased risk for atherosclerosis (Stein, 1986). The high mean

and individual values in the test group are cause for concern.

No statistically significant changes in apolipoprotein B occurred during the study. The levels in the men of the control group increased from baseline to five months, but the levels in the women decreased. Apolipoprotein B values in both men and women of the test group decreased so that the mean value of 1.28 g/l after five months of dietary treatment was within the normal range. This change seems clinically significant. The lower fat content of the test diet and relatively good compliance could have caused the decrease (Weinberg, Dantzker & Patton, 1990). There is also a strong association between HDL-cholesterol levels and apolipoprotein B. The mean total HDL-level in the test group also showed a small decrease.

There was an excellent correlation between the cholesterol levels and the LDL-cholesterol and apolipoprotein B levels during all the visits in both the control and test groups (see Tables 6.14 and 6.15). All three measurements are indications of risk of macrovascular disease, as discussed already. The fact that all three these values were within the normal ranges, but at the higher end of the range, could indicate some risk of coronary heart disease. However, the results suggest that serum lipid values do not change as drastically in the black population with poor diabetic control in comparison with white patients. It seems that black NIDDM patients might have some genetic protection against these changes.

There were consistent, high and significant negative correlations between triglycerides and HDL-cholesterol levels during all visits in both groups. The good positive correlation between the triglycerides and apolipoprotein B in the test group supports the arguments on the reasons for reduction discussed above. The strong correlation between the HDL levels and apolipoprotein A, especially in the test group, together with the above-mentioned correlation of the triglycerides and apolipoprotein B in the test group, indicates that the test diet had a positive influence on

Table 6.14 Correlations between various variables in the test group

	Glucose	HbA_c	Fibrin	BP sis	BPdias	Choles	Triglc	HDL	LDL	Apo A	Apo B	WHR
Cholesterol												
Baseline												
Spearman	0.05	- 0.08	- 0.09	0.23	- 0.12		0.16	0.01	0.97 abcx	- 0.11	0.73 abcx	0.06
p-value	0.80	0.69	0.65	0.22	0.53		0.41	0.94	0.001	0.57	0.001	0.76
One month												
Spearman	0.14	0.28	- 0.19	0.26	0.38		0.11	-0.001	0.98 abdx	- 0.09 aa	0.86 abdx	
p-value	0.46	0.13	0.30	0.17	0.04		0.56	1.00	0.001	0.64	0.001	
Five months												
Spearman	0.35	0.16	0.29	0.01	0.04		0.55	0.17	0.97 adcx	- 0.05	0.80 adcx	-0.001
p-value	0.07	0.41	0.13	0.96	0.83		0.001	0.39	0.001	0.80	0.001	0.99
Triglycerides												
Baseline												
Spearman	- 0.04	- 0.06	- 0.25	- 0.02	- 0.10	0.16		-0.53 abc	0.10	- 0.33	0.42 abc	0.17
p-value	0.82	0.75	0.18	0.91	0.61	0.41		0.001	0.58	0.08	0.02	0.37
One month												
Spearman	0.05	-0.25	0.21	- 0.14	0.06	0.11		- 0.37 abd	0.08	- 0.36	0.43 db	
p-value	0.81	0.18	0.27	0.46	0.76	0.56		0.04	0.68	0.02	0.02	
Five months												
Spearman	0.01	- 0.07	0.12	0.29	0.18	0.55		- 0.43 adc	0.55	- 0.25	0.69 dcx	0.27
p-value	0.95	0.73	0.53	0.13	0.35	0.001		0.02	0.001	0.20	0.001	0.16
HDL-cholesterol												
Baseline												
Spearman	0.18	0.42	0.11	- 0.02	- 0.27	0.01	-0.53 abc		- 0.09	0.82 abcx	- 0.28	-0.42 ac
p-value	0.33	0.02	0.56	0.34	0.15	0.94	0.001		0.62	0.001	0.13	0.02
One month												
Spearman	0.08	0.43	- 0.01	0.07	0.002	-0.001	- 0.37 abd		- 0.10	0.76 dbx	- 0.29	
p-value	0.67	0.02	0.97	0.70	0.99	1.00	0.04		0.60	0.001	0.12	
Five months												
Spearman	0.36	0.35	0.23	-0.09	-0.09	-0.17	-0.43 adc		- 0.33	0.86 dcx	- 0.44	-0.54 ac
p-value	0.05	0.07	0.22	0.64	0.64	0.39	0.02		0.08	0.001	0.02	0.001
Fibrinogen												
Baseline												
Spearman	- 0.15	- 0.10		0.16	0.77	-0.09	- 0.25	0.11	- 0.09	- 0.04	- 0.20	
p-value	0.43	0.62		0.40	0.68	0.65	0.18	0.56	0.62	0.82	0.29	
One month												
Spearman	0.28	- 0.13		- 0.05	- 0.24	- 0.19	0.21	- 0.01	- 0.25	- 0.13	- 0.11	
p-value	0.14	0.49		0.80	0.20	0.30	0.27	0.97	0.18	0.50	0.58	
Five months												
Spearman	0.05	0.02		0.12	0.06	0.29	0.12	0.23	0.22	0.29	- 0.04	
p-value	0.81	0.93		0.52	0.76	0.13	0.53	0.22	0.24	0.13	0.84	

HbA_c = glycated haemoglobin
Fibrin = fibrinogen
BP dias = diastolic blood pressure
BP sis = systolic blood pressure
WHR = waist-to-hip circumference ratio
Triglyc = triglycerides
Apo A = Apolipoprotein A
Apo B = Apolipoprotein B

a = good correlation between both test and control groups
b = good correlations during baseline and after one month
c = good correlations during baseline and after five months
d = good correlations after one and five months
x = correlation ≥ 0.6, $p \leq 0.5$

Table 6.15 Correlations between various variables in the control group

	Glucose	HbA_{1c}	Fibrin	BP sis	BPdias	Choles	Triglc	HDL	LDL	Apo A	Apo B	WHR
Cholesterol												
Baseline												
Spearman	0.14	0.10	0.21	0.02	−0.05		0.42	−0.05	0.97 abcx	0.17	0.78 abcx	−0.08
p-value	0.58	0.67	0.39	0.93	0.84		0.08	0.84	0.001	0.48	0.001	0.73
One month												
Spearman	−0.23	−0.26	0.66	0.07	−0.25		−0.18	0.36	0.97 abdx	0.56 a	0.55 b	
p-value	0.38	0.31	0.001	0.78	0.33		0.49	0.15	0.001	0.02	0.02	
Five months												
Spearman	0.25	0.04	0.44	0.21	0.31		0.13	0.08	0.90 adcx	0.31	0.74 acx	0.10
p-value	0.35	0.88	0.09	0.44	0.24		0.64	0.77	0.001	0.24	0.001	0.73
Triglycerides												
Baseline												
Spearman	−0.07	0.34	0.04	0.04	0.11	0.42		−0.59 abc	0.27	−0.20	0.80 ax	0.24
p-value	0.78	0.15	0.87	0.86	0.64	0.08		0.01	0.27	0.41	0.001	0.33
One month												
Spearman	−0.15	−0.13	−0.27	−0.19	0.08	−0.18		−0.56 abd	−0.20	−0.07	0.29	
p-value	0.56	0.63	0.30	0.46	0.77	0.49		0.02	0.43	0.80	0.26	
Five months												
Spearman	0.50	0.02	0.03	0.23	0.05	0.13		−0.90 adcx	0.14	−0.61 x	0.38	0.64 x
p-value	0.05	0.94	0.91	0.40	0.85	0.64		0.001	0.61	0.01	0.14	0.01
HDL-cholesterol												
Baseline												
Spearman	0.19	−0.07 b	−0.24	0.08	0.12	−0.05	−0.59 abc		−0.05	0.58 ac	−0.36	−0.46 ac
p-value	0.42	0.76	0.33	0.74	0.63	0.84	0.01		0.83	0.01	0.13	0.05
One month												
Spearman	0.05	−0.05 b	0.45	−0.24	−0.49	0.36	−0.56 abd		0.18	0.38	0.16	
p-value	0.85	0.84	0.07	0.34	0.05	0.15	0.02		0.49	0.13	0.54	
Five months												
Spearman	−0.48	−0.16	0.12	−0.12	0.05	0.08	−0.90 adcx		0.07	0.80 cx	−0.19	−0.64 acx
p-value	0.06	0.56	0.66	0.67	0.86	0.77	0.001		0.79	0.001	0.47	0.01
Fibrinogen												
Baseline												
Spearman	−0.03	−0.02		−0.08	−0.21	0.21	0.04	−0.24	0.28	−0.25	0.13	
p-value	0.91	0.91		0.74	0.39	0.39	0.87	0.33	0.24	0.31	0.59	
One month												
Spearman	0.001	0.07		−0.23	−0.34	0.66 x	−0.27	0.45	0.61	0.23	0.19	
p-value	1.00	0.78		0.38	0.19	0.001	0.30	0.07	0.01	0.38	0.46	
Five months												
Spearman	−0.29	−0.10		0.02	0.06	0.44	0.03	0.12	0.48	0.07	0.22	
p-value	0.27	0.71		0.93	0.84	0.09	0.91	0.66	0.06	0.79	0.42	

HbA_{1c} = glycated haemoglobin
Fibrin = fibrinogen
BP dias = diastolic blood pressure
BP sis = systolic blood pressure
Triglyc = triglycerides
Apo A = Apolipoprotein A
Apo B = Apolipoprotein B

WHR = waist-to-hip circumference ratio
a = good correlation between both test and control groups
b = good correlations during baseline and after one month
c = good correlations during baseline and after five months
d = good correlations after one and five months
x = correlation ≥ 0.6, p ≤ 0.5

the lipid values in these patients. This diet therefore seems to be of importance. Although lipid values of black NIDDM patients are not of such major concern as in white diabetics, the westernisation of the black population leads to a higher risk of coronary heart disease (Silvis, 1989). These results indicate that a relatively small dietary intervention could influence serum lipid levels of black NIDDM subjects positively.

Numerous studies have shown an increase in plasma fibrinogen levels in diabetic subjects (Osterman & Van der Loo, 1986). The fibrinogen values in this study (Table 6.13) were also much higher than the normal values of 2 - 4 g/l used at the laboratory for healthy black subjects. The values were also much higher than those found by Silvis (1989) (see Table 6.10) for black diabetics and those found by Vorster et al. (1987a) (2.79 g/l) for healthy black men. In a study in the Swedish population, Wilhelmsen et al. (1984) found that fibrinogen values higher than 3.56 g/l were associated with macrovascular disease. Hyperfibrinogenaemia and fibrinogen survival are most probably associated with the level of glycaemia and can be reversed with better glycaemic control (Ulutin, 1986). The high fibrinogen levels in this study population are, therefore, of concern. It should be noted, however, that the fibrinogen in this study was measured by a different method (using EDTA-plasma) than that used by Silvis (1989) and Vorster et al. (1987a) (using citrate plasma).

There was a slight decrease in fibrinogen in the men in the control group during the study, and a significant decrease in the women from baseline to one and five months. In both the men and women of the test group there were slight decreases in fibrinogen. The decrease for the whole group from baseline to five months was statistically significant, although possibly not clinically significant. Glycaemic control did not improve according to the same pattern as fibrinogen and was probably not responsible for the improvement in fibrinogen values. The reduction in weight could probably have had an influence. Other

haemostatic variables were not measured and, therefore, definite conclusions on reasons for the improvement in fibrinogen cannot be drawn.

6.4.5 Electrolytes and other minerals

The change in blood electrolytes and other minerals can be seen in Table 6.16.

The mean sodium levels of both groups were within the normal limits at baseline and during the study and compared well with values found by Silvis (1989) (see Figure 6.15). Sodium intake was also within normal ranges (see Tables 6.7 and 6.8) (American Diabetes Association, 1990a). However, the restriction of sodium could get attention due to the high prevalence of high blood pressure (Anderson & Geil, 1988), even in patients on anti-hypertensive medication.

The serum sodium values in the control group increased slightly in both men and women during the study, although the increase was not statistically significant. The same applied to the women and total population of the test group. There was a slight, non-significant decrease in the sodium levels in the men of the test group.

Potassium levels were also within the normal ranges and compared well with the values found by Silvis (1989) (see Figure 6.15). A slight decrease in potassium levels was found for both groups, but only the men of the control group showed a statistically but not clinically significant decrease.

The men in the test group were the only subjects with a higher than normal mean chloride level, which was slightly higher than the values found by Silvis (1989) (see Figure 6.15). Hyperchloraemia could occur in diabetics with renal failure (Tietz, Pruden & Siggaard-Andersen, 1986), which was not present in any of the patients (according to their medical records).

Table 6.16 Serum minerals and electrolytes at baseline, after one month and at the end of the study (after five months)

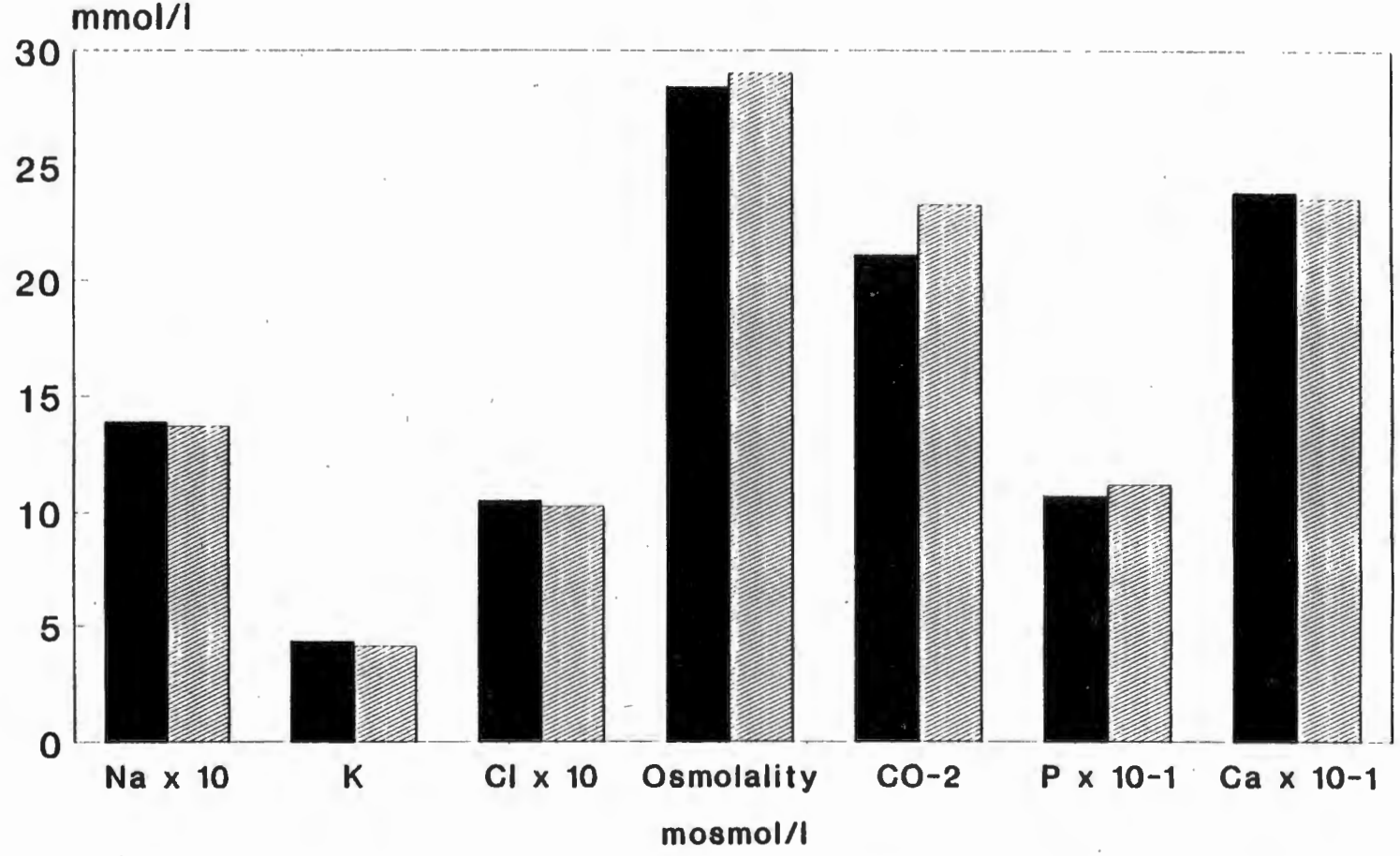
	CONTROL GROUP						TEST GROUP						ORAL AGENT		INSULIN		TOTAL		Normal
	Men		Women		Total	group	Men		Women		Total	group							
	Mean	SD	Mean	SD	Mean	SD	Mean	SD	Mean	SD	Mean	SD	Mean	SD	Mean	SD	Mean	SD	Ranges
Sodium																			
mmol/l																			137.00
Baseline	137.29	10.1	139.50	5.50	138.69	7.3	142.40	2.5	138.25	6.4	139.63	5.7	139.07	6.6	140.67	3.3	139.27	6.3	144.00
n = 49																			
One month	140.60	1.90	137.25	10.6	138.24	9.0	142.30	3.2	139.50	5.1	140.43	4.7	139.32	6.9	141.83	1.9	139.64	6.5	
n = 47																			
Five months	139.00	3.60	141.40	2.90	140.50	3.3	141.60	3.2	140.37	2.5	140.79	2.7	140.64	3.1	141.00	1.7	140.69	2.9	
n = 45																			
Potassium																			
mmol/l																			3.60
Baseline	4.34	0.50	4.38	0.40	4.36	0.4	4.49	0.4	4.23	0.5	4.31	0.5	4.34	0.4	4.27	0.5	4.33	0.5	4.70
One month	4.30 c	0.60	4.28	0.50	4.29	0.5	4.53	0.4	4.29	0.7	4.37	0.6	4.34	0.6	4.30	0.5	4.34	0.6	
Five months	3.90 c	0.30	4.22	0.30	4.10	0.4	4.34	0.4	4.21	0.6	4.26	0.5	4.24	0.5	3.95	0.2	4.20	0.5	
Chloride																			
mmol/l																			98.00
Baseline	102.86	7.60	104.42	5.30	103.84	6.1	110.60	4.8	103.55	6.0	105.90	6.5	105.09	6.5	105.17	5.9	105.10	6.4	108.00
One month	107.20	1.60	105.17	7.90	105.77	6.7	108.70 c	6.0	103.90	5.7	105.50	6.2	105.37	6.7	107.17	1.0	105.60	6.3	
Five months	105.83	4.40	108.20	3.20	107.31	3.7	110.90 c	4.1	105.37	3.9	107.28	4.7	107.41	4.6	106.50	2.9	107.29	4.4	
Phosphorus																			
mmol/l																			0.60
Baseline	1.13	0.20	1.03	0.10	1.07	0.2	1.06	0.2	1.06	0.1	1.06	0.1	1.07	0.1	1.03	0.2	1.07	0.2	1.40
One month	1.12	0.10	1.09	0.10	1.10	0.1	1.08	0.2	1.09	0.2	1.08	0.2	1.09	0.1	1.06	0.2	1.09	0.1	
Five months	1.10	0.10	1.05	0.10	1.07	0.1	1.07	0.1	1.11	0.1	1.10	0.1	1.10	0.1	1.04	0.1	1.09	0.1	
Calcium																			
mmol/l																			2.20
Baseline	2.39	0.20	2.34	0.10	2.36	0.2	2.44 b	0.1	2.39	0.2	2.41	0.1	2.39	0.1	2.39	0.1	2.39	0.1	2.60
One month	2.44	0.10	2.32	0.30	2.35	0.2	2.39	0.1	2.40	0.1	2.40	0.1	2.37	0.2	2.44	0.1	2.38	0.2	
Five months	2.37	0.10	2.36	0.10	2.36	0.1	2.35 b	0.1	2.40	0.1	2.38	0.1	2.37	0.1	2.42	0.1	2.38	0.1	
Osmolality																			
mosmol/l																			275.00
Baseline	282.71	22.9	285.33	11.4	284.40	16.0	290.50	6.3	281.70 b	12.0	284.60	11.1	283.90	13.7	288.80	6.1	284.50	13.1	295.00
One month	288.20	3.30	279.67	21.5	282.18	18.4	288.80	6.7	284.05	11.2	285.63	10.0	283.90	14.4	287.80	3.9	284.40	13.6	
Five months	287.83	8.10	287.60	6.60	287.70	6.9	286.70	7.3	287.37 b	6.3	287.10	6.6	287.70	6.7	284.70	5.9	287.30	6.6	

a = significant differences ($p \leq 0.05$) between visit 1 and 2

b = significant differences ($p \leq 0.05$) between visit 1 and 5

c = significant differences ($p \leq 0.05$) between visit 2 and 5

x = significant differences ($p \leq 0.05$) between control and test groups



Na = sodium, K = potassium, Cl = chloride,
CO-2 = Carbon dioxide, P = Phosphorus, Ca = calcium

Figure 6.15 Comparison of electrolyte and mineral values of two studies on black diabetes mellitus patients

However, there might have been some renal damage because of poor diabetic control of these patients (Brownlee, 1985). Metabolic acidosis, which could be present to some extent (because the urinary excretion of ketones were high in these patients) could also have contributed to the elevated chloride levels.

Chloride is the major extracellular anion and is involved in the maintenance of osmotic pressure. The men in the test group had a slightly, but not statistically significant higher mean blood osmolality than the other patients, which could be explained by the higher chloride levels. The osmolality was within the normal ranges.

The men and women in both groups showed an increase in blood chloride levels during the study. Only the increase from one to five months in the men of the test group was statistically significant. Slight increases in the mean osmolality of both groups were also found. The increase was significant from baseline to five months in the women of the test group. The men in the test group experienced a mean decrease in osmolality, in contrast with the increase in their mean chloride levels.

The mean phosphorus levels of both groups were within the normal ranges and did not show any statistically significant changes. The same applied to the mean calcium levels, except in the case of the men in the test group where a statistically significant decrease in the mean calcium level was found. The calcium intake of the men in the test group was much higher than the intake of the other patients at the baseline of the study and they also had a slightly higher mean calcium blood level. During the study, their intake was lower and more in line with the intake of the other patients. That could have caused the statistically significant decrease in serum calcium levels.

There was no difference between the electrolyte and other mineral levels of the insulin-treated and the patients treated with oral hypoglycaemic agents.

6.4.6 Metabolites and excretion products

The mean values of metabolites and excretion products measured in serum are given in Table 6.17.

Total blood ureum nitrogen levels were within normal ranges. As already mentioned, the patients were not malnourished. The baseline value for the men of the control group were higher than those of the other patients. These patients had a much higher mean intake of animal protein, which could explain their mean higher total blood ureum nitrogen level. A slight decrease, not statistically significant, was observed for all the patients except the women in the test group. The decrease in protein intake during the study could have been responsible for this. The protein intakes and mean nitrogen levels of the two groups are compared in Figure 6.16.

The uric acid values were also within the normal ranges and very little change took place during the study. There was a statistically significant difference between the test and control group at the baseline of the study, but not during the study. The control group showed a statistically significant decrease in mean uric acid level, but the decrease was not clinically significant.

The mean creatinine level for the men in the control group was higher than the normal range and that for the women in both groups lower than normal. The mean creatinine level of all the patients (87.5 µmol/l) was, however, within the normal range and higher than the mean of 79.9 µmol/l found by Silvis (1989) in black NIDDM subjects. The increase in creatinine in the control group was statistically significant from baseline to five months. The same applies to the increase from baseline to five months and from one to five months for the women of the test group. They still had a mean creatinine level after five months that was lower than the normal range. No clinically significant change took place during the study.

Table 6.17 Serum excretion products at baseline, after one month and at the end of the study (after five months)

	CONTROL GROUP						TEST GROUP						ORAL AGENT		INSULIN		TOTAL		Normal
	Men		Women		Total in group		Men		Women		Total group								
	Mean	SD	Mean	SD	Mean	SD	Mean	SD	Mean	SD	Mean	SD	Mean	SD	Mean	SD	Mean	SD	Ranges
Total BUN mmol/l																			3.60 7.80
Baseline n = 49	7.44	4.7	5.67	2.3	6.32	3.4	6.75	3.5	5.26	1.9	5.76	2.6	5.91	2.9	6.48	2.8	5.98	2.9	
One month n = 47	5.46	2.5	5.23	1.9	5.29	2.0	6.16	3.3	5.08	1.9	5.44	2.4	5.32	2.3	5.82	2.5	5.39	2.3	
Five months n = 45	7.38	4.3	5.50	1.9	6.21	3.0	6.34	2.9	5.33	1.9	5.68	2.3	5.94	2.6	5.38	2.2	5.86	2.6	
Uric acid mmol/l																			0.21 0.47
Baseline x	0.39	0.1	0.31	0.1	0.34 b	0.1	0.35	0.1	0.31	0.1	0.33	0.1	0.33	0.1	0.33	0.1	0.33	0.1	
One month	0.35	0.1	0.30	0.1	0.31	0.1	0.35	0.0	0.30	0.1	0.32	0.1	0.32	0.1	0.31	0.0	0.32	0.1	
Five months	0.40	0.1	0.29	0.0	0.33 b	0.1	0.36	0.1	0.30	0.1	0.32	0.1	0.32	0.1	0.33	0.0	0.33	0.1	
Creatinine umol/l																			81.00 114.00
Baseline	119.84	65.3	78.15	29.6	93.51 b	48.8	111.13	51.4	69.98 b	14.8	83.69	36.8	87.73	42.9	85.90	34.2	87.50	41.7	
One month	102.02	28.9	73.50	26.7	81.90	29.6	109.09	49.4	70.77 c	14.8	83.54 c	35.2	83.03	33.7	82.37	30.4	82.95	33.0	
Five months	129.05	84.7	77.62	27.1	96.91 b	59.1	111.64	51.5	75.47 bc	16.7	87.94 c	36.6	91.80	47.6	86.78	29.4	91.13	45.4	
TB umol/l																			4.00 30.00
Baseline	4.74	2.2	2.88	1.2	3.56	1.8	3.42 b	1.9	4.16	3.6	3.91	3.1	3.71	2.6	4.25	3.8	3.78	2.7	
One month	5.64	2.5	4.00	1.3	4.48	1.8	3.63	1.2	4.00	2.0	3.88	1.8	4.00	1.9	4.72	1.1	4.09	1.8	
Five months	4.82	3.7	3.81	1.2	4.19	2.4	4.66 b	2.0	4.75	1.9	4.72	2.4	4.54	2.2	4.47	1.3	4.53	2.1	
DB umol/l																			0.10 4.00
Baseline	0.20	0.4	0.10 a	0.3	0.14 b	0.3	0.07	0.2	0.45	0.6	0.32	0.5	0.26	0.5	0.20	0.4	0.25	0.5	
One month x	0.34	0.5	0.35 a	0.4	0.35	0.4	0.16	0.1	0.16	0.2	0.16 c	0.2	0.19	0.3	0.48	0.4	0.23	0.3	
Five months	0.28	0.5	0.26	0.3	0.27 b	0.4	0.38	0.5	0.22	0.1	0.27 c	0.3	0.28	0.4	0.22	0.2	0.27	0.3	
Carbon dioxide mmol/l																			23.00 29.00
Baseline	21.43	2.10	22.25 a	2.10	21.95	2.1	18.90	3.1	21.45 b	2.4	20.60 b	2.8	21.21	2.7	20.50	2.1	21.12	2.7	
One month	20.60	2.30	20.08 ac	2.70	20.24 c	2.5	20.10 c	2.8	21.44 c	2.9	21.00 c	2.9	20.68	2.9	21.00	1.7	20.72	2.8	
Five months	22.50	2.00	22.44 c	2.10	22.46 c	2.0	21.40 c	2.7	23.74 b c	2.8	22.93 b c	2.9	22.80	2.7	22.57	1.7	22.76	2.6	

Total BUN = Total blood ureum nitrogen
TB = Total bilirubin
DB = Direct bilirubin

a = significant differences ($p \leq 0.05$) between visit 1 and 2
b = significant differences ($p \leq 0.05$) between visit 1 and 5
c = significant differences ($p \leq 0.05$) between visit 2 and 5
x = significant differences ($p \leq 0.05$) between control and test groups

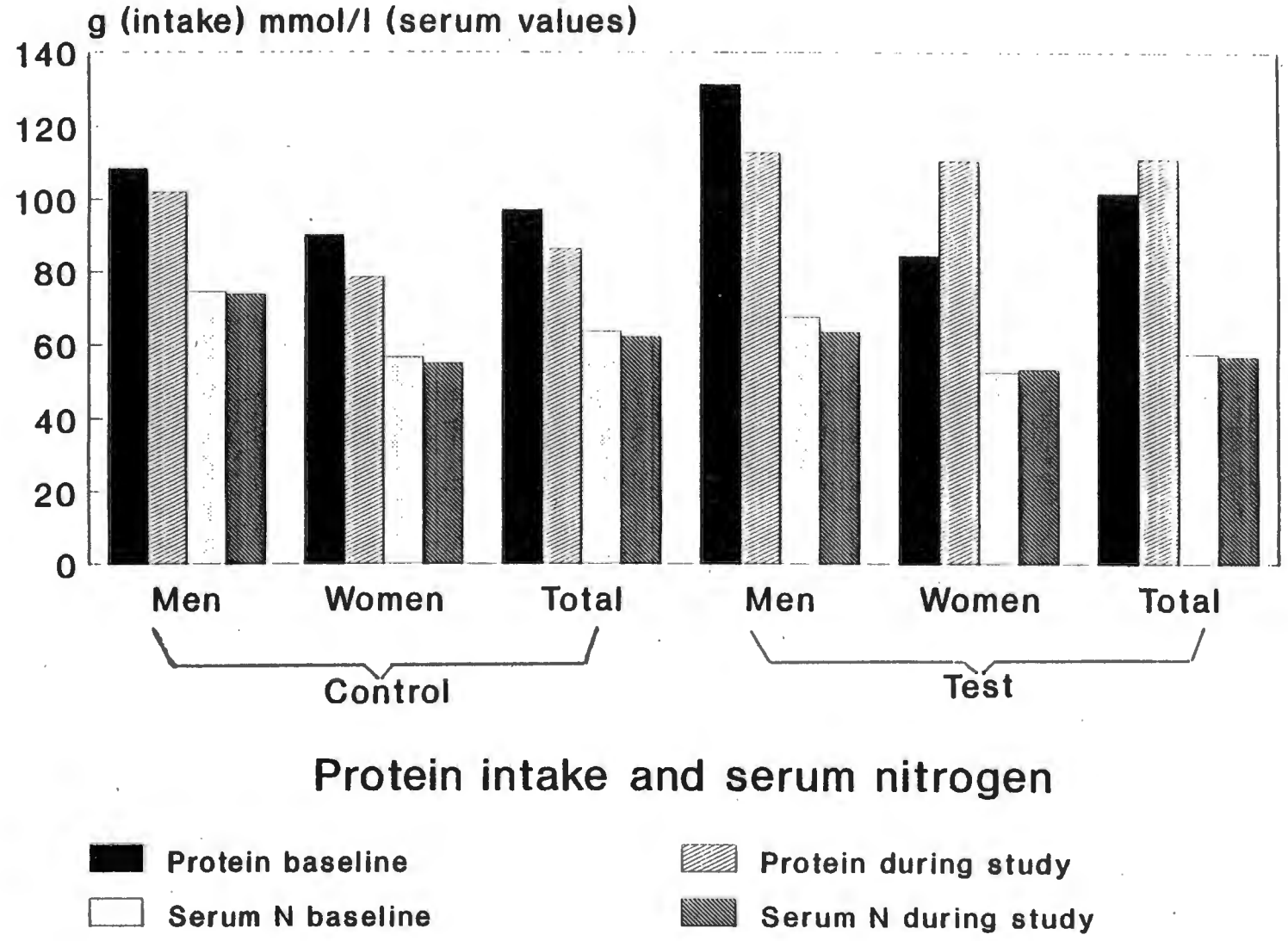


Figure 6.16 Comparison of protein intake and total plasma nitrogen at baseline and during the study

The total and direct bilirubin levels in the women of the control group and men of the test group were lower than the normal range. The men of the control group and women of the test group had mean values near to the lower limit of the normal range. All values were much lower than the 6.96 µmol/l for total bilirubin and 0.67 µmol/l for direct bilirubin found by Silvis (1989) for black NIDDM subjects. However, low values of bilirubin are not clinically important (Balistreri & Shaw, 1986). The total bilirubin levels in both groups increased during the study. The increase was only statistically significant in the case of the men of the test group - the mean level increased to 4.66 µmol/l, which is within the normal range. The direct bilirubin levels in both groups also increased, except in the women of the test group which showed a statistically significant decrease from one to five months. The increase was statistically significant for the women of the control group from baseline to one month and for the total control group from baseline to five months. These increases resulted in normal values in both cases. There was a statistically significant difference between the values of the control and test groups after one month, with the value of the test group below the normal range.

The mean carbon dioxide levels of both groups were below the normal ranges and also lower than the values found by Silvis (1989) (see Figure 6.15) at the baseline of the study. However, the blood samples were stored -20 °C for five months before analysis was done and that could have caused lower values (Tietz, Pruden & Siggaard-Andersen, 1986). The carbon dioxide levels were higher and very close to the normal ranges in the samples that were analysed soon after they were taken at the end of the study.

6.4.7 Enzymes and proteins

Values obtained from serum analysis of enzymes and proteins are given in Table 6.18.

Table 6.18 Serum enzymes and proteins at baseline, after one month and at the end of the study (after five months)

	CONTROL GROUP						TEST GROUP						ORAL AGENT		INSULIN		TOTAL		Normal
	Men		Women		Total in group		Men		Women		Total group								
	Mean	SD	Mean	SD	Mean	SD	Mean	SD	Mean	SD	Mean	SD	Mean	SD	Mean	SD	Mean	SD	Ranges
AST																			11.00
IE/l																			32.00
Baseline	14.00	4.5	18.75 a	14.7	17.00 b	12.0	14.80	6.0	13.50 b	8.5	13.93	7.7	13.93	9.1	23.67	9.0	15.12	9.6	
One month	14.80	4.3	15.10 a	11.9	15.00 c	10.1	14.70	4.9	12.80	4.4	13.43	4.6	13.37	7.1	18.33	5.5	14.00	7.0	
Five months	16.33	3.7	19.90	13.7	18.56 bc	11.0	16.70	7.2	14.00 b	5.2	14.93	6.0	15.44	8.3	21.33	6.0	16.22	8.2	
LDH																			60.00
IE/l																			200.00
Baseline	143.14	43.2	160.92 b	49.7	154.37 b	47.0	137.00 b	46.6	157.25 b	33.9	150.50	39.0	144.65	38.3	204.67	26.4	152.00	41.8	
One month	146.60	34.8	160.75	69.5	156.59	60.6	164.30	44.2	167.40 c	42.4	166.37 c	42.3	153.29	38.8	228.00	66.5	162.83	49.2	
Five months	167.50	34.9	202.10 b	40.2	189.13 b	40.9	176.40 b	34.2	188.63 bc	42.1	184.41 c	39.4	176.74	29.5	246.83	45.2	186.09	39.5	
ALT																			8.00
IE/l																			30.00
Baseline	8.14 b	2.1	9.67	8.8	9.11 b	7.1	12.40	9.9	10.60 b	10.2	11.20	10.0	10.21	9.1	11.67	7.9	10.39	8.9	
One month	8.60	4.2	7.90 c	7.1	8.12 c	6.3	13.10	11.0	9.00 c	5.6	10.37 c	7.9	9.63	7.8	9.00	3.5	9.55	7.3	
Five months	16.67 b	2.4	15.30 c	13.9	15.81 bc	10.9	13.40	6.5	14.20 bc	6.9	13.93 c	6.6	15.05	8.7	11.67	4.5	14.60	8.3	
GGT																			12.00
IE/l																			43.00
Baseline	36.43	23.0	27.50	14.3	30.79	17.9	36.60 b	12.6	26.55	14.4	29.90	14.5	29.74	15.7	33.83	16.7	30.25	15.7	
One month	25.00	6.0	26.58	15.4	26.12 c	13.1	31.40	8.3	26.80	11.3	28.33	10.5	27.37	12.0	28.67	6.8	27.53	11.4	
Five months x	39.00	15.3	28.50	22.6	32.44 c	20.3	29.90 b	8.9	27.50	12.8	28.31	11.5	30.00	16.1	28.30	6.4	29.78	15.1	
AP																			40.00
IE/l																			104.00
Baseline	95.29	21.0	105.08	39.3	101.47 b	33.4	82.20	23.3	94.05 b	29.5	90.10	27.8	90.79	26.2	121.17	45.1	94.51	30.3	
One month	95.40	18.6	101.50	29.8	99.71	26.6	79.30	17.9	98.35	33.6	92.00	30.4	92.17	28.6	112.67	27.6	94.79	29.0	
Five months	103.50	18.7	115.50	35.2	111.00 b	29.9	81.00	22.4	103.05 b	28.6	95.45	28.3	99.56	28.2	110.17	38.9	100.98	29.5	
Total protein																			66.00
g/l																			79.00
Baseline	74.86	8.3	74.50	4.2	74.63	5.8	74.80	4.8	74.50	4.4	74.60	4.5	74.26	4.9	77.17	4.9	74.61	5.0	
One month	75.80	3.6	72.50	6.8	73.47	6.1	73.80	4.7	74.40	4.0	74.20	4.1	73.44	4.8	77.33	4.2	73.94	4.9	
Five months	75.33	3.2	73.80	4.0	74.38	3.7	72.70	4.4	74.58	4.6	73.93	4.5	73.77	4.4	76.17	2.1	74.09	4.2	
Albumin																			39.00
g/l																			50.00
Baseline	44.86	3.9	41.92	2.4	43.00	3.3	46.40	2.7	43.95	3.8	44.77	3.6	44.19	3.7	43.33	2.9	44.08	3.6	
One month	45.00	1.6	40.83	4.5	42.06	4.3	45.60	2.4	43.90	3.3	44.43	3.1	43.49	3.8	44.17	3.4	43.57	3.7	
Five months	44.83	2.7	42.10	2.3	43.13	2.7	45.20	3.1	44.21	3.7	44.55	3.5	44.03	3.3	44.17	3.0	44.04	3.3	
PA ratio																			
Baseline	1.67	0.1	1.78	0.1	1.74	0.1	1.62	0.1	1.70	0.1	1.67	0.1	1.69	0.1	1.79	0.1	1.70	0.1	
One month	1.69	0.1	1.78 c	0.1	1.75	0.1	1.62	0.1	1.70	0.1	1.68	0.1	1.70	0.1	1.76	0.1	1.70	0.1	
Five months	1.69	0.1	1.76 c	0.1	1.73	0.1	1.61	0.1	1.69	0.1	1.67	0.1	1.68	0.1	1.73	0.1	1.69	0.1	

AST = serum aspartate aminotransferase
LDH = serum lactate dehidrogenase
ALT = serum alanine aminotransferase
GGT = serum gamma-glutamyltransferase
AP = serum alkaline phosphatase
PA ratio = protein to albumin ratio

a = significant differences ($p \leq 0.05$) between visit 1 and 2
b = significant differences ($p \leq 0.05$) between visit 1 and 5
c = significant differences ($p \leq 0.05$) between visit 2 and 5
x = significant differences ($p \leq 0.05$) between control and test groups

The mean serum aspartate aminotransferase (AST) values were within the normal range but nearer to the lower limit and much lower than the values found by Silvis (1989) (see Figure 6.17). During the study there was an increase in the AST levels. After an initial significant decrease in the mean values of the women of the control group, they then had an increase in values. There was a statistically significant increase in both the total control group and the women of the test group from baseline to five months and in the control group from one to five months. The insulin-treated subjects had a much higher mean AST value than the patients on oral hypoglycaemic agents. None of the changes in AST levels were of clinical significance.

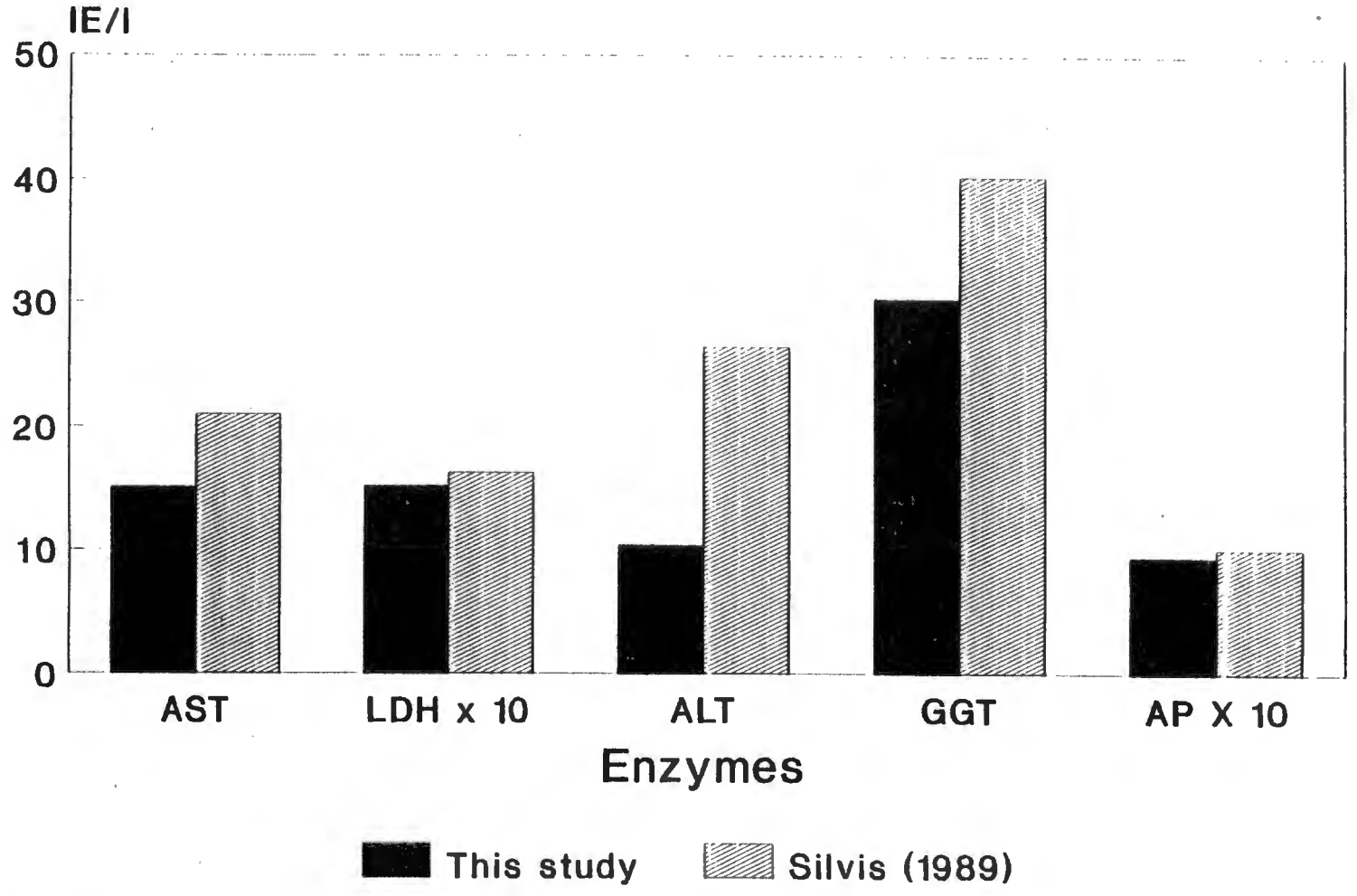
The serum lactate dehydrogenase (LDH) levels of both groups were within the normal range but nearer to the upper limit of the range and compare well with the values of Silvis (1989) (see Figure 6.17). The values for both groups increased during the study. A statistically significant increase for the women of both groups, the men of the test group and the total control group from baseline to five months and for the women in the test group and the total test group from one to five months were observed. The mean value for the women in the control group at the end of the study exceeded the upper limit of the normal range. The high positive correlation between systolic blood pressure and LDH levels in both groups during the baseline and one month later was interesting (see Tables 6.19 and 6.20). LDH activity is often used to determine the possibility of myocardial infarction in which case blood pressure is also high (Moss, Henderson & Kachmar, 1986). The insulin-treated subjects had much higher values than the other patients. A moderately high LDH level, as found in this study in the insulin-treated subjects, may be a sign of liver damage due to liver anoxia secondary to decreased oxygen perfusion. In some patients it may also be a sign of renal damage (Moss, Henderson & Kachmar, 1986).

The alanine aminotransferase (ALT) levels were all within the normal range but nearer to the lower limit of the range and much

lower than the values found by Silvis (1989) (see Figure 6.17). A large increase in ALT levels was noted in the control group (both men and women) and the increase was statistically signifi= cant in the men and total group from baseline to five months and in the women and the total group from one to five months. A statistically significant increase was found in the women of the test group, but not in the men. The ALT levels of the insulin-treated subjects did not differ from those of the other patients.

The gamma-glutamyltransferase (GGT) levels were within the normal range but nearer to the upper limit. The levels were also lower than those measured by Silvis (1989) (see Figure 6.17). The men in both groups had higher GGT levels than the women and there was in increase in the levels for all except the men of the test group who had a statistically significant decrease from baseline to five months. This caused a statistically significant difference between the two groups in the GGT levels after five months.

The values for alkaline phosphatase (AP) were near the upper limit of the normal range and, in the women of the control group, even higher than the normal range. A high AP level is often caused by biliary obstruction (Moss, Henderson & Kachmar, 1986). If the high BMI of these patients is taken into consideration, then the possibility of biliary obstruction cannot be excluded (Krause & Mahan, 1984). As with the other enzymes the levels for both groups, except for the men in the test group, increased during the study and the increase was statistically significant from baseline to five months in the case of the total control group and the women of the test group. There was a poor correlation between the AP values of the test group and their cholesterol levels during baseline and after five months (see Tables 6.19 and 6.20).



AST = serum aspartate aminotransferase
LDH = lactate dehidrogenase
ALT = serum alanine aminotransferase

GGT = serum gamma-glutamyltransferase
AP = serum alkaline phosphatase

Figure 6.17 Comparison of enzyme levels in black NIDDM patients in this study and the study of Silvis (1989)

Table 6.19 Correlations between various variables in the test group

	TEST Glucose	HDA_{1C}	Fibrin	BP sis	BPdias	Choles	Triglc	HDL	LDL	Apo A	Apo B
AST											
Baseline											
Spearman	0.03	− 0.10	− 0.09	0.09	0.06	0.12	0.31	− 0.30	0.09	0.05	0.15
p-value	0.89	0.59	0.64	0.62	0.74	0.52	0.10	0.11	0.65	0.80	0.42
One month											
Spearman	− 0.25	− 0.22	0.13	0.35	0.13	0.01	0.03	− 0.16	− 0.01	− 0.11	0.16
p-value	0.19	0.24	0.48	0.06	0.50	0.97	0.88	0.39	0.95	0.57	0.39
Five months											
Spearman	− 0.22	− 0.09	− 0.19	0.04	− 0.07	0.22	0.34	− 0.10	0.19	0.04	0.21
p-value	0.24	0.65	0.31	0.84	0.70	0.26	0.70	0.62	0.33	0.82	0.28
LDH											
Baseline											
Spearman	0.03	0.19	0.06	0.41 ab	0.55 b	−0.16	−0.12	0.14	− 0.16	0.27	−0.09
p-value	0.87	0.31	0.77	0.03	0.001	0.40	0.54	0.45	0.39	0.14	0.64
One month											
Spearman	− 0.08	0.25	0.08	0.42 b	0.37 b	0.09	− 0.14	0.15	0.07	− 0.12	0.08
p-value	0.68	0.18	0.69	0.02	0.05	0.62	0.47	0.94	0.73	0.53	0.69
Five months											
Spearman	− 0.30	−0.005	0.02	− 0.09	− 0.23	0.08	0.001	0.14	0.01	0.33	− 0.02
p-value	0.11	0.98	0.92	0.66	0.24	0.67	1.00	0.46	0.97	0.08	0.91
ALT											
Baseline											
Spearman	0,25	0.03	0.02	−0.28	− 0.12	−0.07	0.39	− 0.32	−0.08	0.001	0.11
p-value	0.19	0.89	0.93	0.13	0.54	0.72	0.03	0.09	0.66	1.00	0.55
One month											
Spearman	− 0.01	− 0.30	0.17	0.10	0.26	0.01	0.32	−0.28	0.01	−0.31	0.26
p-value	0.94	0.10	0.37	0.62	0.17	0.94	0.09	0.13	0.95	0.09	0.17
Five months											
Spearman	0.07	0.12	−0.21	0.01	−0.03	0.16	0.30	0.12	0.10	0.22	0.14
p-value	0.72	0.55	0.27	0.95	0.87	0.42	0.11	0.55	0.60	0.25	0.48
GGT											
Baseline											
Spearman	−0.02	− 0.07	− 0.12	− 0.18	− 0.01	−0.002	0.23	− 0.45 b	0.03	− 0.23	0.07
p-value	0.91	0.73	0.52	0.33	0.95	0.99	0.23	0.01	0.89	0.22	0.70
One month											
Spearman	−0.15	− 0.21	−0.09	0.19	0.23	−0.05	0.17	− 0.43 b	−0.001	− 0.22	0.16
p-value	0.44	0.27	0.63	0.31	0.21	0.77	0.37	0.02	1.00	0.23	0.41
Five months											
Spearman	−0.19	− 0.05	0.03	0.05	−0.11	−0.03	0.06	− 0.02	0.02	0.12	− 0.05
p-value	0.32	0.79	0.90	0.81	0.58	0.88	0.76	0.90	0.90	0.55	0.78
AP											
Baseline											
Spearman	0.19	0.15	0.001	− 0.17	− 0.35	0.42 c	0.29	−0.001	0.01	0.36	0.35
p-value	0.32	0.43	1.00	0.37	0.06	0.02	0.001	1.00	0.97	0.05	0.05
One month											
Spearman	0.16	0.11	0.11	0.09	0.14	0.19	0.29	0.004	0.16	0.03	0.28
p-value	0.41	0.56	0.55	0.63	0.46	0.31	0.12	0.99	0.39	0.86	0.14
Five months											
Spearman	0.60	0.43	− 0.22	− 0.20	−0.09	0.39 c	0.25	−0.01	0.36	0.17	0.43
p-value	0.001	0.02	0.24	0.29	0.64	0.04	0.20	0.97	0.06	0.39	0.02

Triglyc = triglycerides
Fibrin = fibrinogen
BP dias = diastolic blood pressure
BP sis = systolic blood pressure
WHR = waist-to-hip circumference ratio
Apo A = Apolipoprotein A
Apo B = Apolipoprotein B
x = correlation ≥ 0.6, p ≤ 0.5

a = good correlation between both test and control groups
b = good correlations during baseline and after one month
c = good correlations during baseline and after five months
d = good correlations after one and five months

Table 6.20 Correlations between various variables in the control group

	Glucose	HbA	Fibrin	BP sis	BPdias	Choles	Triglc	HDL	LDL	Apo A	Apo B
AST											
Baseline											
Spearman	0.06	0.14	0.06	0.25	0.23	−0.27	−0.09	0.21	−0.29	0.16	0.27
p-value	0.82	0.57	0.81	0.30	0.34	0.27	0.71	0.38	0.23	0.51	0.27
One month											
Spearman	−0.25	−0.10	−0.24	0.37	0.10	−0.15	−0.43	0.25	−0.16	−0.08	−0.21
p-value	0.33	0.71	0.35	0.14	0.70	0.57	0.08	0.33	0.53	0.77	0.41
Five months											
Spearman	−0.16	−0.03	−0.32	0.06	−0.19	−0.39	−0.39	0.38	−0.44	0.31	−0.12
p-value	0.54	0.91	0.22	0.83	0.49	0.13	0.13	0.15	0.09	0.25	0.65
LDH											
Baseline											
Spearman	−0.09	−0.20	0.37	0.46 ab	0.29	−0.22	−0.22	0.12	−0.23	−0.02	−0.24
p-value	0.71	0.41	0.12	0.05	0.23	0.37	0.37	0.63	0.35	0.95	0.32
One month											
Spearman	0.10	0.22	−0.12	0.58 b	0.44	−0.07	−0.31	0.25	−0.12	−0.25	−0.09
p-value	0.69	0.39	0.65	0.01	0.07	0.79	0.23	0.33	0.66	0.33	0.74
Five months											
Spearman	−0.05	0.33	0.24	0.29	0.34	0.26	−0.17	0.31	0.47	0.20	0.32
p-value	0.87	0.21	0.38	0.28	0.20	0.32	0.53	0.24	0.07	0.46	0.23
ALT											
Baseline											
Spearman	0.14	−0.01	0.08	0.36	0.42	0.22	0.22	0.14	0.16	−0.08	0.19
p-value	0.56	0.98	0.75	0.14	0.08	0.36	0.35	0.57	0.51	0.74	0.43
One month											
Spearman	0.49	0.12	−0.35	0.45	0.48	−0.26	−0.19	0.01	−0.23	−0.09	0.23
p-value	0.05	0.66	0.17	0.07	0.05	0.32	0.47	0.96	0.37	0.72	0.37
Five months											
Spearman	0.16	0.14	0.01	0.26	0.38	0.16	0.13	−0.24	−0.11	0.34	0.23
p-value	0.55	0.61	0.97	0.33	0.15	0.57	0.62	0.37	0.69	0.19	0.39
GGT											
Baseline											
Spearman	0.002	0.08	−0.004	0.05	0.10	0.37	0.30	0.15	0.31	−0.06	0.37
p-value	0.99	0.73	0.99	0.84	0.68	0.12	0.21	0.95	0.20	0.82	0.12
One month											
Spearman	0.30	0.003	0.07	0.25	0.15	0.13	−0.31	0.09	0.17	0.32	0.43
p-value	0.24	0.99	0.80	0.33	0.56	0.62	0.23	0.72	0.51	0.21	0.08
Five months											
Spearman	−0.21	0.22	0.06	0.34	0.08	0.06	0.10	−0.17	−0.10	−0.34	0.08
p-value	0.43	0.41	0.81	0.20	0.77	0.82	0.71	0.54	0.71	0.20	0.76
AP											
Baseline											
Spearman	0.19	0.05	0.59	0.03	−0.24	−0.08	−0.15	−0.04	−0.01	−0.24	−0.13
p-value	0.44	0.85	0.01	0.92	0.33	0.75	0.54	0.88	0.98	0.32	0.59
One month											
Spearman	0.06	0.36	0.28	0.26	0.22	−0.02	−0.54	0.19	0.01	−0.12	−0.15
p-value	0.83	0.16	0.28	0.32	0.39	0.94	0.03	0.46	0.97	0.65	0.55
Five months											
Spearman	0.12	0.28	−0.07	0.17	0.03	−0.13	0.004	0.06	−0.03	0.05	0.15
p-value	0.65	0.30	0.81	0.54	0.92	0.63	0.99	0.82	0.93	0.86	0.59

HbA$_{1c}$ = glycated haemoglobin
Fibrin = fibrinogen
BP dias = diastolic blood pressure
BP sis = systolic blood pressure
Triglyc = triglycerides
Apo A = Apolipoprotein A
Apo B = Apolipoprotein B
WHR = waist-to-hip circumference ratio

a = good correlation between both test and control groups
b = good correlations during baseline and after one month
c = good correlations during baseline and after five months
d = good correlations after one and five months

The serum protein values were within the normal range and very much the same for both groups. The same applies to the albumin values. No statistically significant changes in the plasma protein and albumin levels took place during the study. The protein/albumin ratio was discussed in section 6.3.1

6.4.8 Full blood count

Values measured with the full blood count can be seen in Table 6.21.

All values for leucocytes and erythrocytes were within normal ranges. The leucocyte counts decreased in the control group during the study and increased in the test group. No statistically significant differences could be found. Although the mean leucocyte count was normal, there may be functional abnormalities (Bagdade, Stewart & Walters, 1978) which could not have been detected by the leucocyte count. The mean erythrocyte count was on the lower end of the normal range and decreased during the study. This was not necessarily caused by the diet (the iron intake of the test group nearly doubled). However, no statistically or clinically significant changes took place. There could have been functional abnormalities in the erythrocytes that would not have been detected with the erythrocyte count (Jones & Peterson, 1981). Because of the low values, erythrocyte counts should be repeated on these patients from time to time.

The mean corpuscular haemoglobin was normal. A significant difference was found at the baseline between the test and control groups for both the mean corpuscular haemoglobin and mean corpuscular haemoglobin concentration. A significant decrease in the mean corpuscular haemoglobin took place from one to five months in the women of the control group as well as in the women of the test group from baseline to one month. However, no change was clinically significant. The mean corpuscular haemoglobin concentration was on the lower end of the normal range for all

Table 6.21 Full blood counts at baseline, after one month and at the end of the study (after five months)

	CONTROL GROUP						TEST GROUP						ORAL AGENT		INSULIN		TOTAL		Normal
	Men		Women		Total in group		Men		Women		Total in group								
	Mean	SD	Mean	SD	Mean	SD	Mean	SD	Mean	SD	Mean	SD	Mean	SD	Mean	SD	Mean	SD	Ranges
ukocyte count																			4.80
0																			10.80
seline	5.94	2.10	6.05	1.50	6.01	1.7	5,66	1.3	4.95	1.4	5.20	1.4	5.47	1.2	5.83	3.2	5.52	1.6	
= 49																			
e month	5.80	2.10	5.85	1.50	5.84	1.6	5.88	1.1	5.52	1.4	5.64	1.3	5.53	1.2	6.96	2.0	5.71	1.4	
= 47																			
ve months	5.29	1.50	5.58	1.00	5.47	1.1	5.75	1.2	5.96	1.5	5.89	1.4	5.61	1.3	6.58	1.0	5.74	1.3	
= 45																			
ythrocyte cnt																			M 4.7
0^2 /µl																			6.10
seline	4.91	0.70	4.55	0.40	4.68	0.6	4.83	0.5	4.69	0.3	4.74	0.4	4.71	0.5	4.83	0.3	4.72	0.5	F 4.2
e month	4.90	0.70	4.48	0.50	4.60	0.6	4.73	0.4	4.74	0.4	4.73	0.4	4.67	0.5	4.78	0.4	4.69	0.5	5.40
ve months	4.81	0.60	4.48	0.40	4.60	0.5	4.69	0.5	4.69	0.4	4.69	0.4	4.65	0.4	4.70	0.3	4.66	0.40	
H																			27.00
																			31.00
seline x	29.17	2.40	28.19	2.40	28.55	2.4	28.98	1.6	29.16 a	0.9	29.10	1.2	28.93	1.8	28.40	1.7	28.88	1.80	
e month	29.56	2.20	27.58 c	2.40	28.17	2.4	29.45	2.6	28.36 a	1.2	28.73	1.8	28.55	2.1	28.38	2.2	28.52	2.10	
ve months	29.03	2.50	28.49 c	2.70	28.69	2.6	29.35	2.3	29.10	1.5	29.18	1.8	29.08	2.1	28.57	2.1	29.01	2.10	
HC																			33.00
dl																			37.00
seline x	33.17	2.20	32.38	1.40	32.67 b	1.7	32.15	0.7	33.26	0.6	32.86	0.8	32.84	1.3	32.32	0.7	32.79	1.30	
e month	33.64	2.50	31.99	1.20	32.48 c	1.8	33.18	1.9	32.92	1.2	33.01 c	1.5	32.78	1.6	33.03	1.8	32.81	1.60	
ve months	33.88	1.80	33.25	1.90	33.49 bc	1.9	32.86	1.1	33.57	1.2	33.33 c	1.2	33.48	1.4	32.75	1.7	33.38	1.50	
atelet count																			130.00
0^2 /µl																			400.00
seline	274.29	56	287.83	84.3	282.84	73.7	277.50	69.3	288.17	46.9	284.36	54.9	276.81	61.0	342.00	43.9	283.74	62.4	
e month	303.60	51	285.17	80.0	290.59	71.5	262.90	47.3	287.79	58.8	279.21	55.6	279.97	63.8	306.33	38.0	283.41	61.4	
ve months x	254.17	53	275.30	98.1	267.38	82.5	274.80	60.7	295.47	37.9	288.34	47.0	274.25	60.7	324.00	56.1	280.89	61.9	
V																			7.20
																			11.10
seline	9.13	1.00	9.14	0.90	9.14 a	0.9	9.31	0.9	9.24 b	0.6	9.26 b	0.7	9.26	0.8	8.82	0.4	9.21	0.80	
e month	8.48	1.10	8.83 c	0.70	8.73 ac	0.8	9.07	0.5	9.11 c	0.9	9.09 c	0.8	8.92	0.8	9.20	0.5	8.96	0.80	
ve months	9.08	1.00	9.51 c	0.90	9.35 c	0.9	9.38	0.7	9.70 bc	0.8	9.59 bc	0.7	9.53	0.9	9.32	0.2	9.50	0.80	
V																			M 80
																			94.00
seline	88.04	6.80	86.98	5.70	87.37 b	6.0	90.16 a	5.1	87.51 a	3.4	88.45 ab	4.2	88.03	5.0	87.94	5.0	88.02	5.00	F 81
e month	88.02	7.20	86.14	5.90	86.69 c	6.1	88.71 a	4.3	86.24 a	3.1	87.09 a	3.7	87.11	4.8	85.88	3.8	86.95	4.70	99.00
ve months	85.65	5.90	85.62	5.70	85.63 bc	5.6	89.27	5.2	86.63	3.3	87.54 b	4.2	86.80	4.7	87.23	5.4	86.86	4.70	

MCH = mean corpuscular haemoglobin

MCHC = mean corpuscular haemoglobin concentration

MPV = mean plasma volume

MCV = mean corpuscular volume

a = significant differences ($p \leq 0.05$) between visit 1 and 2

b = significant differences ($p \leq 0.05$) between visit 1 and 5

c = significant differences ($p \leq 0.05$) between visit 2 and 5

x = significant differences ($p \leq 0.05$) between control and test groups

patients and slightly lower than the normal range in the women of the control group and the men of the test group. There was an improvement in concentration for both groups during the study, which was statistically significant in the total control group and from one to five months in the total test group.

The platelet count was normal for both groups and approximately in the middle of the normal range. The platelet count decreased in the control group and increased in the test group to cause a statistically significant difference at the end of the study. The difference had no clinical significance. There was a large difference between the platelet count of the insulin-treated subjects and the other patients.

The mean plasma volume and mean corpuscular volume were normal in both groups and increased statistically but not clinically significantly in both groups during the study.

The full blood counts of both groups were, therefore normal.

Table 6.22 Statistical probabilities of differences in variables between visits (Wilcoxin ranked test)

	CONTROL GROUP									TEST GROUP								
	Men			Women			Total	in	group	Men			Women			Total	in	group
	V1+V2	V1+V5	V2+V5	V1+V2	V1+V5	V2+V5	V1+V2	V1+V5	V2+V5	V1+V2	V1+V5	V2+V5	V1+V2	V1+V5	V2+V5	V1+V2	V1+V5	V2+V5
Age	na	na	na	na	na	na	na	na	na	na	na	na	na	na	na	na	na	na
Height	na	na	na	na	na	na	na	na	na	na	na	na	na	na	na	na	na	na
Weight	>0.1	>0.1	>0.1	>0.1	>0.1	>0.1	>0.1	>0.1	>0.1	0.035	0.035	>0.1	>0.1	>0.1	>0.1	0.075	<0.01	0.015
Body mass index	>0.1	>0.1	>0.1	>0.1	>0.1	>0.1	>0.1	>0.1	>0.1	0.075	0.035	0.075	>0.1	>0.1	>0.1	>0.1	0.007	<0.01
Blood pressure	>0.1	>0.1	>0.1	>0.1	>0.1	>0.1	>0.1	>0.1	>0.1	>0.1	>0.1	>0.1	>0.1	>0.1	>0.1	>0.1	>0.1	>0.1
WHR	na	>0.1	na	na	>0.1	na	na	>0.1	na	na	>0.	na	na	0.035	na	na	0.075	na
Cholesterol	>0.1	>0.1	>0.1	>0.1	>0.1	>0.1	>0.1	>0.1	>0.1	0.035	>0.1	>0.1	>0.1	>0.1	>0.1	0.046	0.200	0.703
HDL-cholesterol	>0.1	>0.1	>0.1	>0.1	0.075	>0.1	>0.1	>0.1	>0.1	>0.1	>0.1	>0.1	>0.1	>0.1	>0.1	0.218	0.485	0.949
LDL-cholesterol	>0.1	0.035	0.035	>0.1	<0.01	<0.01	<0.01	<0.01	<0.01	<0.01	<0.01	<0.01	<0.01	<0.01	<0.01	<0.01	<0.01	<0.01
Triglisérides	>0.1	>0.1	>0.1	>0.1	>0.1	>0.1	>0.1	>0.1	>0.1	>0.1	0.075	>0.1	>0.1	>0.1	>0.1	0.800	0.416	>0.1
Apolipoprot A	>0.1	>0.1	>0.1	>0.1	>0.1	>0.1	>0.1	>0.1	>0.1	>0.1	>0.1	>0.1	>0.1	>0.1	0.075	0.388	>0.1	0.035
Apolipoprot B	>0.1	>0.1	>0.1	>0.1	>0.1	>0.1	>0.1	>0.1	>0.1	0.075	0.075	>0.1	>0.1	>0.1	>0.1	0.280	>0.1	0.233
Fibrinogen	>0.1	>0.1	>0.1	0.035	0.015	0.075	0.035	>0.1	>0.1	>0.1	>0.1	>0.1	>0.1	0.075	0.075	0.501	0.007	0.120
Fructosamine	>0.1	>0.1	>0.1	>0.1	>0.1	>0.1	0.075	>0.1	>0.1	>0.1	>0.1	>0.1	>0.1	>0.1	>0.1	0.407	0.603	0.367
HbA_{1c}	>0.1	>0.1	>0.1	>0.1	>0.1	>0.1	0.075	>0.1	>0.1	>0.1	>0.1	>0.1	>0.1	>0.1	>0.1	>0.1	0.208	0.040
Haemoglobin	>0.1	>0.1	>0.1	0.015	>0.1	0.075	0.075	>0.1	>0.1	>0.1	>0.1	>0.1	>0.1	>0.1	>0.1	>0.1	>0.1	>0.1
Hematocrit	>0.1	>0.1	>0.1	0.075	0.035	>0.1	>0.1	>0.1	>0.1	>0.1	0.075	>0.1	>0.1	>0.1	>0.1	>0.1	0.040	>0.1
Blood glucose	>0.1	>0.1	>0.1	>0.1	>0.1	>0.1	>0.1	>0.1	>0.1	>0.1	>0.1	>0.1	>0.1	>0.1	>0.1	0.494	0.713	0.761
Sodium	>0.1	>0.1	0.075	>0.1	>0.1	>0.1	>0.1	>0.1	>0.1	>0.1	>0.1	>0.1	>0.1	>0.1	>0.1	>0.1	>0.1	>0.1
Potassium	>0.1	>0.1	0.020	>0.1	>0.1	>0.1	>0.1	>0.1	>0.1	>0.1	>0.1	>0.1	>0.1	>0.1	>0.1	>0.1	>0.1	>0.1
Chloride	0.075	>0.1	>0.1	>0.1	0.075	>0.1	>0.1	>0.1	>0.1	>0.1	>0.1	0.015	>0.1	>0.1	>0.1	>0.1	>0.1	0.075
Carbon dioxide	>0.1	>0.1	>0.1	0.015	>0.1	0.035	>0.1	>0.1	0.035	>0.1	0.075	0.035	>0.1	<0.01	<0.01	>0.1	<0.01	<0.01
Phosphorus	>0.1	>0.1	>0.1	>0.1	>0.1	>0.1	>0.1	>0.1	>0.1	>0.1	>0.1	>0.1	>0.1	>0.1	>0.1	0.269	>0.1	>0.1
Calcium	>0.1	>0.1	>0.1	>0.1	>0.1	>0.1	>0.1	>0.1	>0.1	>0.1	0.015	>0.1	>0.1	>0.1	>0.1	0.542	0.071	>0.1
Osmolality	>0.1	>0.1	>0.1	>0.1	>0.1	>0.1	>0.1	>0.1	>0.1	>0.1	>0.1	>0.1	>0.1	0.035	>0.1	0.939	>0.1	>0.1
BUN	>0.1	>0.1	>0.1	>0.1	>0.1	>0.1	>0.1	>0.1	>0.1	>0.1	>0.1	>0.1	>0.1	>0.1	>0.1	0.254	>0.1	0.269
Uric acid	>0.1	0.075	>0.1	>0.1	>0.1	>0.1	>0.1	0.035	>0.1	>0.1	>0.1	>0.1	>0.1	>0.1	>0.1	0.355	0.424	0.657
Creatinine	>0.1	>0.1	>0.1	>0.1	>0.1	>0.1	>0.1	0.015	>0.1	>0.1	>0.1	>0.1	>0.1	0.015	>0.1	0.075	0.008	
Tot. Bilirubin	>0.1	>0.1	>0.1	0.075	0.075	>0.1	>0.1	>0.1	>0.1	>0.1	0.035	0.075	>0.1	0.075	>0.1	0.674	0.01	0.075
Dir. Bilirubin	>0.1	>0.1	>0.1	0.015	>0.1	>0.1	>0.1	0.035	>0.1	>0.1	0.075	>0.1	>0.1	>0.1	>0.1	>0.1	>0.1	0.035
AST	>0.1	0.075	>0.1	0.035	>0.1	0.075	>0.1	<0.01	0.035	>0.1	>0.1	0.075	>0.1	0.035	>0.1	>0.1	0.035	>0.1
LDH	>0.1	>0.1	>0.1	<0.01	>0.1	>0.1	>0.1	0.035	0.075	>0.1	<0.01	>0.1	>0.1	<0.01	0.015	0.077	0.001	0.029
ALT	>0.1	0.035	>0.1	>0.1	>0.1	<0.01	>0.1	<0.01	0.015	>0.1	>0.1	>0.1	>0.1	<0.01	<0.01	>0.1	0.015	<0.01
GGT	>0.1	>0.1	0.075	>0.1	>0.1	>0.1	>0.1	>0.1	0.035	0.075	0.035	>0.1	>0.1	>0.1	>0.1	>0.1	>0.1	>0.1
AP	>0.1	>0.1	>0.1	>0.1	>0.1	>0.1	>0.1	0.035	>0.1	>0.1	>0.1	>0.1	>0.1	0.075	<0.01	>0.1	0.186	<0.01	0.075
Total Protein	>0.1	>0.1	>0.1	>0.1	>0.1	>0.1	>0.1	>0.1	>0.1	>0.1	>0.1	>0.1	>0.1	>0.1	>0.1	>0.1	>0.1	>0.1
Albumin	>0.1	>0.1	>0.1	>0.1	>0.1	>0.1	>0.1	>0.1	>0.1	>0.1	>0.1	>0.1	>0.1	>0.1	>0.1	>0.1	>0.1	>0.1
PA ratio	>0.1	>0.1	>0.1	>0.1	>0.1	0.015	>0.1	>0.1	>0.1	>0.1	>0.1	>0.1	>0.1	>0.1	>0.1	>0.1	>0.1	>0.1
Leucocyte count	>0.1	>0.1	>0.1	>0.1	>0.1	>0.1	>0.1	>0.1	>0.1	>0.1	>0.1	>0.1	>0.1	>0.1	>0.1	>0.1	>0.1	>0.1
Erythrocyte cnt	>0.1	>0.1	>0.1	>0.1	>0.1	>0.1	>0.1	>0.1	>0.1	>0.1	>0.1	>0.1	>0.1	>0.1	>0.1	>0.1	>0.1	>0.1
MCH	>0.1	>0.1	>0.1	>0.1	>0.1	0.035	>0.1	>0.1	>0.1	>0.1	>0.1	>0.1	<0.01	>0.1	0.075	>0.1	>0.1	0.075
MCHC	>0.1	>0.1	>0.1	>0.1	0.075	<0.01	>0.1	0.015	<0.01	>0.1	0.075	>0.1	>0.1	>0.1	0.075	>0.1	0.015	0.035
Platelet count	>0.1	>0.1	>0.1	>0.1	>0.1	>0.1	>0.1	>0.1	>0.1	>0.1	>0.1	>0.1	>0.1	>0.1	>0.1	>0.1	>0.1	>0.1
MPV	>0.1	>0.1	>0.1	>0.1	>0.1	0.035	0.035	>0.1	<0.01	>0.1	>0.1	>0.1	>0.1	0.035	<0.01	>0.1	0.075	<0.01
MCV	>0.1	>0.1	0.075	>0.1	>0.1	>0.1	>0.1	0.035	0.035	0.035	>0.1	>0.1	0.035	0.075	>0.1	<0.01	0.015	>0.1

WHR = middle-to-hip circumference ratio
Apolipoprot A = apolipoprotein A
Apolipoprot B = apolipoprotein B
HbA_{1c} = glycated haemoglobin
BUN = total blood ureum nitrogen
Tot. Bilirubin = total bilirubin
Dir. Bilirubin = direct bilirubin
AST = serum aspartate aminotransferase
LDH = serum lactate dehidrogenase
ALT = serum alanine aminotransferase
GGT = serum gamma-glutamyltransferase
AP = serum alkaline phosphatase

Table 6.23 Statistical probabilities of differences in variable between the test and control group (Mann-Whitney test)

	Baseline	One month	Five months
Age	0.38	0.58	0.24
Height	0.11	0.04	0.15
Weight	0.31	0.18	0.24
BMI	0.46	0.34	0.44
Blood pressure			
Sistolic	0.47	0.85	0.02
Diastolic	0.21	0.98	0.11
WHR	0.01	1.00	0.08
PA ratio	0.95	0.81	0.85
Cholesterol	0.98	0.69	0.31
HDL-cholesterol	0.09	0.78	0.28
Triglycerides	0.33	0.28	0.01
Apolipoprot. A	0.01	0.13	0.45
Apolipoprot. B	0.28	0.14	0.72
Fibrinogen	0.95	0.21	0.62
Fructosamine	0.64	0.44	0.04
Protein	0.37	0.44	0.36
HbA_{1c}	0.88	0.19	0.05
Haemoglobin	0.14	0.83	0.40
Hematocrit	0.77	0.64	0.83
Glucose	0.33	0.45	0.02
Sodium	0.35	0.35	0.49
Potassium	0.95	0.34	0.31
Chloride	0.90	0.72	0.42
Phosphorus	0.67	0.55	0.49
Calcium	0.42	0.11	0.99
Osmolality	0.30	0.37	0.44
BUN	0.48	0.48	0.61
Uric acid	0.02	0.72	0.42
Creatinine	0.25	0.93	0.34
Tot. Bilirubin	0.20	0.88	0.85
Dir. Bilirubin	0.11	0.00	0.28
AST	0.15	0.07	0.65
LDH	0.40	0.23	1.00
ALT	0.21	0.68	0.46
GGT	0.74	0.59	0.03
AP	0.57	0.74	0.58
Protein	0.37	0.44	0.36
Albumin	0.82	0.44	0.64
PA ratio	0.95	0.81	0.85
Carbon dioxide	0.15	0.37	0.17
Leucocyte cnt	0.30	0.59	0.49
Erythrocyte cnt	0.39	0.40	0.26
MCH	0.01	0.14	0.15
MCHC	0.00	0.45	0.07
Platelet cnt	0.06	0.36	0.04
MPV	0.38	0.88	0.39
MCV	0.25	0.07	0.27

WHR = middle-to-hip circumference ratio

Apolipoprot A = apolipoprotein A

Apolipoprot B = apolipoprotein B

HbA_{1c} = glycated haemoglobin

BUN = total blood ureum nitrogen

Tot. Bil = total bil

Dir. Bil = direct bil

6.5 DISCUSSION AND CONCLUSIONS

This part of the study was designed to examine the hypothesis that the traditional African diet has a positive effect on the long-term metabolic control and risk markers of cardiovascular complications in black NIDDM subjects. The prescribed control and test diets were not principally designed for weight loss. The total energy content of the test diet was, however, slightly lower than that of the reference diet (14 600 and 10 700 kJ for men, 11 500 and 8 500 kJ for women of the control and test groups respectively). The lower GI of the test diet could also lead to weight reduction (Wolever, 1990). Small reductions in weight of subjects in the test group could have been expected, and were in fact observed.

However, the energy intake and changes in weight of the subjects did not correlate well (see Table 6.24). This discrepancy could have occurred either because of higher energy intakes than reported by the subjects (something that happens commonly in obese subjects on a diet), or, the theory that the metabolism of the black patient and especially the NIDDM patient, has adapted to famine situations and cannot cope with the abundance of food available in the westernised urban environment (Zimmet, 1982), could also be true in these patients. This needs further investigation. The food intake data of the patients were evaluated carefully and they were questioned repeatedly by experienced interviewers (see section 6.3.4.2). Higher than the reported energy intakes could, therefore be likely in some cases but not in all - it would have been detected. A lower metabolism might therefore also play a role. Both reasons mentioned above could therefore be responsible for the discrepancy.

Therefore, the traditional African diet alone, as prescribed for these patients, did not result in significant weight loss and a reducing diet with a low energy content should therefore be prescribed for the overweight black NIDDM patient.

The high prevalence of hypertension, even in those patients on anti-hypertensive medication, is cause for concern.

Table 6.24 Comparison between energy intakes and weight changes of subjects.

Variable	Control group		Test group	
	Men (n = 8)	Women (n = 13)	Men (n = 11)	Women (n = 19)
Energy intakes (kJ)				
Prescribed intake	14574	10738	11497	8496
Intake at baseline	11257	8911	11037	8538
Intake during study	9156	7681	9076	7801
Mean weight (kg)				
Baseline	79.6	83.0	84.4	81.1
±SD	14.4	16.8	9.9	16.1
One month	76.3	83.5	83.6	80.9
±SD	17.1	17.2	9.7	15.7
Five months	80.2	83.5	82.8	80.0
±SD	16.8	17.9	9.4	16.4
Mean weight changes (kg)				
Baseline to one month	- 3.29	+ 0.55	- 0.81	- 0.26
Baseline to five months	+ 0.63	+ 0.53	- 1.58	- 1.11
Expected mean weight changes (kg)				
Actual to prescribed intake (5 months)	+13.09	+ 7.21	+ 1.82	- 0.17
Actual to actual intake (5 months)	- 8.29	- 4.86	- 7.70	- 2.88

Except for the women in the control group, the mean WHR of all groups indicated android or upper body obesity if 0.85 is taken as cut-off point, as advised by Kaplan (1989). Of the men all but one had a WHR higher than 0.85 and of the women 50 % (twelve from the test group and four from the control group) had a WHR

higher than 0.85. Obesity is common among young and middle-aged black women in the RSA (Seftel, 1977). It was recently shown in a study on rural black women that gynoid or as it was termed "healthy" obesity, was more prevalent than android obesity (Walker et al., 1989). It is interesting to note that 50 % of the black women in this study with DM had gynoid obesity. This underlines the importance of gynoid obesity as a risk factor for DM and coronary heart disease (Freedman et al., 1990; Ostlund et al., 1990; Van Gaal et al., 1988) and raises questions as to whether the connotation of "healthy" obesity is valid.

The hypothesis that the black NIDDM patient can follow the traditional African diet was successfully proved with a dietary compliance of 71 %. The study further indicated that, with proper motivation and attention, the patients of the test group succeeded in reducing their fat intake from a mean of 33 % of total energy to 23 % of energy. Even if the actual energy intakes were higher than the reported intakes, as discussed previously, the energy distribution was probably correct, as the method of data collection was valid (see section 6.4.2) and therefore the reported lower fat intake would be correct.

The small but significant weight loss in the test group was accompanied by small but significant decreases in triglycerides, apolipoprotein B, fibrinogen and total cholesterol (in the men). The question arises whether the small but significant improvements seen in the lipid profiles were a result of the reduced fat intake, the low GI of the diet, or of the observed weight loss (Anderson & Geil, 1988; Streja, Boyko & Rabkin, 1980). It is possible that all these factors contributed. The lower GI of the test diet (because of the low GI of two meals in the diet - see Chapter 5) probably also contributed to the improved serum lipids (Jenkins et al., 1985; Jenkins et al., 1987b).

The absence of an effect of the low GI test diet on glycaemic control was disappointing. The question could be asked whether

it would be possible to improve glycaemic control of obese NIDDM patients without substantial weight loss and/or medication adjustments. This study supports the notion that although adjustments in energy distribution of the diet may beneficially influence lipid profiles, it will not influence glycaemic control without substantial weight loss, as was also concluded by Silvis (1989). It seems, therefore, that the initial or first dietary adjustment for obese NIDDM patients should be an energy reduction which could lead to substantial weight loss. The problem would be compliance to such low-energy diets.

The lipid profile of the NIDDM patients in this study was worse than in the healthy black population but better than in the South African white population (Vermaak et al., 1988). Dietary intake data showed that at least this section of the black population is in the process of westernisation and that they are prone to the diseases caused by the new life-style as was also found by Silvis (1989). The good reported compliance and reduction in weight and serum lipids on the test diet also show that, if the patients could be motivated not to change their traditional life-style drastically, it could only be to their advantage. Motivation strategies should therefore be investigated urgently.

The relatively low fibrinogen levels in the study of Silvis (1989) (Table 6.10) led to the conclusion that these levels could probably be one of the reasons why the black NIDDM population do not get coronary heart disease as easily as the white South African NIDDM population. A direct comparison between the higher results of this study with the results of Silvis (1989), could not be made, because a different method was used for the determination of fibrinogen. The glycaemic control of the patients in the study of Silvis (1989) was also better. The fibrinogen levels and other reasons for the protection against macro-vascular disease should be investigated further. It is possible that the relatively normal lipid profiles (if compared with white diabetics) protect black NIDDM subjects to some extent against the macrovascular complications of the disease. Poor

glycaemic control is associated with the microvascular complications of DM (Brownlee, 1985) and one could therefore expect micro rather than macrovascular complications in the black diabetic patient. This possibility should be investigated further.

The electrolyte and other mineral profiles of the patients in this study were normal, except for relatively high chloride levels (Table 6.16). Low carbon dioxide levels may by due to the storage of the blood before analysis. The chloride levels could indicate some renal and macro-vascular damage and should get attention. The values for the excretion products and full blood count profiles were within the normal ranges (Table 6.17 and Table 6.21). The enzyme and protein levels of the patients (Table 6.18) were normal to normal-high.

CHAPTER 7

COMBINED DISCUSSION, CONCLUSIONS AND RECOMMENDATIONS FOR FURTHER RESEARCH

It was clear from the literature survey that more research needs to be done on black NIDDM patients in South Arica. This study examined:

- the eating habits and nutrient intake of black NIDDM patients in the Ga-Rankuwa area;
- the GI of a traditional African meal and the acute effects of this meal on short term glycaemic control in black NIDDM patients;
- the long-term effect on glycaemic control and influence on risk markers of macro-vascular disease of the traditional African diet.

The black NIDDM patients studied are clearly in the process of westernisation. The macro-nutrient distribution of their diet lies between that of the prudent diet as recommended for diabetic subjects, and the western diet. This was reflected by their increased intakes of fat, animal protein products and refined foods and a decrease in dietary fibre intake. The current concern of South African health personnel, that urbanisation and westernisation of the black population may lead to an increasing prevalence of typical western degenerative diseases, is supported by the observations in this study.

The study has further shown that obese black NIDDM patients can successfully comply with a prescribed diet, low in fat and animal protein foods. Therefore, if effective nutrition education can be implemented early enough, before they become used to the western diet and a high fat and animal protein intake, nutritional prevention of western diseases, and especially diabetes and the complications thereof, should be successful. The fact that their eating habits are relatively constant, especially during the week, simplifies nutrition education and

may contribute to successful motivation to follow the prudent diet for diabetics in the form of an adapted African diet.

Obesity is one of the major environmental factors in determining the prevalence of NIDDM in a population (Krolewski & Warram, 1985). Energy intake should therefore be restricted to a very low energy level in most of the black NIDDM patients because of their high mean body mass index. It is also possible that their energy needs are much lower than the RDA and that metabolic adaptation took place during years of famine, so that they cannot metabolise high energy loads effectively.

The type of foods included in the black diabetic diet should get attention, because the mean micro-nutrient intakes of these patients are low. The use of legumes as source of plant proteins, vegetables and fruit should be advocated.

More research is, therefore, needed for:
- effective weight loss programmes in the black NIDDM patients;
- successful motivation strategies to follow prescribed diets;
- effective nutrition education programmes.

The GI of the traditional African meal, as adapted for this study, was relatively low (71 % if incremental area with t_o as baseline is used) compared to white bread. It should therefore be much lower if compared to glucose. The dietary fibre content of the diet was, however, low (5.8 g) due to the removal of the leguminous fibre during processing in the soya mince product. This could have influenced the GI values obtained.

The predicted GI, based on the glycaemic indices of the individual food items, correlated well with the GI of the traditional African meal in the women but not in the men. The reasons why the men and the women reacted differently regarding the GI of the test meal are not clear.

The glycaemic response to a second meal, given three hours after

the low GI meal, caused a Staub-Traugott effect in both the men and the women, and a SMR in the women, supporting the hypothesis of Wolever (1990) and Jenkins et al. (1982b), that a low GI meal will lead to an improved glucose disposal of a subsequent meal.

More research is needed on:
- the effect an African meal with unrefined leguminous fibre on the glycaemic response of the black NIDDM patient;
- reasons why the glycaemic response differed in the women and the men; and
- possible mechanisms through which the second meal effect is mediated.

If answers for these problems could be found, the GI and SMR might be used successfully in designing diabetic diets and improving glycaemic control through diet.

A high prevalence of hypertension was observed among the black NIDDM patients. This was also reported by Silvis (1989) for black NIDDM subjects. Research on the mechanisms involved and strategies to improve blood pressure profiles in these patients are urgently needed.

The poor medical care and unavailability of dietitians for nutritional counselling due to shortage of funds are areas of concern in these black diabetic patients. Even the excellent work that is done by the nursing staff of the outpatient clinics cannot compensate for these deficiencies.

On the long-term (period of five months), the low GI, traditional African diet did not markedly influence glycaemic control. It did, however, induce small improvements on some of the risk markers of macro-vascular disease, namely weight, blood lipid profiles and fibrinogen levels, but most changes were not statistically or clinically significant. It was shown that the GI of the African meal was lower in women than in men (Chapter

5); if the low GI of the diet was solely responsible for the effects mentioned above, larger effects in the women than in the men could have been expected, which was not always observed. Therefore, although the low GI diet possibly contributed, it could not have been the only reason.

Some of the improvements were also found in the patients on the reference diet, but to a smaller extent. Reasons for this relatively low effectivity of the test diet are not clear; if the reported intakes were the actual intakes of these patietnts, then compliance was relatively good (71 %), possibly because most of the food of the prescribed diet was provided.

Differences between the composition of the reference and the test diet (low GI African diet) were not large and could explain the observed results. The energy distribution of the reference diet was, for example, 18 % from protein, 30 % from fat and 49 % from carbohydrates with the plant protein intake nearly as high as the animal protein intake, compared to the 23 % from protein, 23 % from fat and 53 % from carbohydrates of the test diet, but with a plant protein intake of nearly four times that of the animal protein intake. Both diets were mainly composed of maize meal porridge which has a low GI and the reference diet therefore also had a relatively low GI. (The blacks still eat large amounts of maize meal porridge as staple.) The only large difference between the reference and test diet was, therefore, the large plant protein component of the test diet. In an analysis of the minced soya product used in the test diet, it was found that most of the leguminous dietary fibre had been removed, probably during processing. It therefore seems that the soya product, without the leguminous fibre, had little effect on long-term metabolic control.

A low GI leguminous diet with a high fibre content (unprocessed legumes) will probably effect a substantial improvement in metabolic control and risk markers of macro-vascular complications (Potter et al., 1981; Simpson et al., 1981; Erdman

& Fordyce, 1989). The effect of such a diet in black NIDDM patients should be investigated.

The diabetic patients who participated in the study, were already part of the busy western life style. Most of them were skilled workers or professional people. The long preparation time of unprocessed legumes is, therefore, a major problem and was the main reason why the processed soya mince was chosen for this study. Solutions for this problem, such as the use of canned legume products with a low GI (Jenkins et al., 1987c) should be investigated.

Finally, there is no question that NIDDM, which already affects at least 400 000 urban blacks and an unknown number of rural blacks, is increasing in South Africa. It should get serious attention and the occurrence of the disease should be prevented by all means. Research to develop an effective, cheap screening procedure, successful preventive strategies, and useful education programmes for prevention of NIDDM and if already present, prevention of complications, are of utmost importance. The high monetary toll of an increasing number of NIDDM patients cannot be afforded by this country. Prevention thereof should be one of the primary goals of all health authorities, personnel and researchers.

From the results of this study it is clear that the dietitian should be an integral part of the medical team. He/she could play an important role in the prevention and treatment of NIDDM. Patients can comply with a prudent diabetic diet, if they get the necessary nutrition education, motivation and attention. Such a diet can improve glycaemic control and risk markers of long-term complications of DM. The insight of the dietitian in the nutritional problems of the DM patient should enable him/her to provide this education, motivation and attention, from diagnosis of diabetes, throughout the course of the disease. He/she could therefore play an important role in improving and maintaining quality of life for the NIDDM patient.

APPENDIX A

1983 METROPOLITAN HEIGHT AND WEIGHT TABLES

MEN					WOMEN				
Height		*Frame size*			*Height*		*Frame size*		
Feet	*Inches*	*Small*	*Medium*	*Large*	*Feet*	*Inches*	*Small*	*Medium*	*Large*
5	2	128-134	131-141	138-150	4	10	102-111	109-121	118-131
5	3	130-136	133-143	140-153	4	11	103-113	111-123	120-134
5	4	132-138	135-145	142-156	5	0	104-115	113-126	122-137
5	5	134-140	137-148	144-160	5	1	106-118	115-129	125-140
5	6	136-142	139-151	146-164	5	2	108-121	118-132	128-143
5	7	138-145	142-154	149-168	5	3	111-124	121-135	131-147
5	8	140-148	145-157	152-172	5	4	114-127	124-138	134-151
5	9	142-151	148-160	155-176	5	5	117-130	127-141	137-155
5	10	144-154	151-163	158-180	5	6	120-133	130-144	140-159
5	11	146-157	154-166	161-184	5	7	123-136	133-147	143-163
6	0	149-160	157-170	164-188	5	8	126-139	136-150	146-167
6	1	152-164	160-174	168-192	5	9	129-142	139-153	149-170
6	2	155-168	164-178	172-197	5	10	132-145	142-156	152-173
6	3	158-172	167-182	176-202	5	11	135-148	145-159	155-176
6	4	162-176	171-187	181-207	6	0	138-151	148-162	158-179

(Goodhart & Shils, 1980:7,8)

APPENDIX B

QUESTIONNAIRE

HABITUAL DIETARY INTAKE

Name.....
Surname.....
Age.....

WHAT DO YOU USUALLY EAT?

Before breakfast

For breakfast

Tea-time

Lunch

Coffee time

When you get home

Supper

At bedtime

During the night

FOOD FREQUENCY LIST - MEDUNSA DIABETIC NUTRITION PROGRAMME

Ethnic group .....
Age .....
Sex .....
Name.....
Surname .....
Date .....

ANYTHING USED LESS THAN ONCE IN TWO WEEKS SHOULD BE IGNORED

State which of the following was eaten and how much.

A. MILK AND DAIRY PRODUCTS

When	Type	Quantity	Times/day	Times/week
As such or as a milk drink Cup = 180 ml Mug = 225 ml Glass = 200 ml				
In tea and coffee In cup = 25 ml In mug = 35 ml				
Over porridge 60 - 100 ml/ 250ml				
Other uses (custard, sauce)				
Type = full cream fresh, full cream powder, skimmed, skim milk powder, blend, low fat milk, condensed, evaporated, buttermilk, yoghurt, thick milk, goats milk, whey, fermented milk, macheu				
Cheese				
Cream				
Ice-cream				
Non-dairy creamer				

B. MEAT AND PROTEIN DISHES

Type	Description	g/day	g/week	Preparation	Bone
Beef, lean					
, with fat					
, other					
Lamb + Mutton					
, lean					
, with fat					
, other					
Pork, lean					
, with fat					
, bacon					
, other					
Goat, lean					
, with fat					
, other					
Other=brains, heart, kidney, liver, tongue, tripe, feet, biltong					
Mince, lean					
, with fat					
Boerewors					
Sausages					
Venison					
Chicken					
, with skin					
, without skin					
, giblets etc.					
Fish, white					
, oily					
, canned					
, crumbed					
, batter					
, shellfish					
Processed meat					
Meat pies, etc.					
Eggs					
Legumes, soup					
, stew					
, other					
Baked beans					
TVP (Toppers)					
Nuts					
Peanut butter					

C. CEREALS AND CEREAL PRODUCTS

Type	g(vol)/day	g(vol)/week	Comments
Bread, white			
, brown			
, whole wheat			
Provita, etc.			
Porridge, maize refined			
, maize unrefined			
, maize fermented			
, oats			
, maltabella			
, other			
Breakfast cereals			
Cake			
Rusks			
Cookies/ Biscuits			
Pies			
Vetkoek			
Scones			
Samoosas			
Pastas, refined			
, unrefined			
Rice, white			
, brown			
Mealie rice			
Samp			
Pearled wheat			
Barley			
Pudding			
Type.....			
STARCHY VEGETABLES			
Potatoes			
, cooked			
, mashed			
, chips, hot			
, chips, dry			
, salad			
, roasted			
, baked			
, in stews etc			
Sweet potatoes			
Mealies			
Sweet corn			

D. VEGETABLES

Type	g/day	g/week	Preparation
Salad vegetables			
tomatoes, raw			
, cooked			
lettuce			
celery			
cucumber, raw			
, cooked			
chilis			
green peppers			
Green vegetables			
spinach			
imifino/ marogo			
Brussels sprouts			
broccoli			
green beans			
green peas			
Yellow vegetables			
carrots			
pumpkin			
Other			
beetroot			
asparagus			
onions, alone			
, in stews			
cabbage			
cauliflower			
radishes			
mixed			
mushrooms			
brinjals			
other			
Tomato and onion sauce			
Fat added			
Sugar added			
Sauces added			

E. FRUIT

Type	g/day	g/week	Comments
Apples			
Pears			
Peaches			
Plums			
Apricots			
Quinces			
Prickly pear			
Strawberries			
Other berries			
Cherries			
Guavas			
Bananas			
Grapes			
Oranges			
Lemons			
Limes			
Naartjies			
Grapefruit			
Sweet melons/cantelopes			
Watermelons			
Paw-paws			
Pineapples			
Mangoes			
Litchi's			
Maroelas			
Figs			
Rhubarb			
Dates			
Granadilla			
Avocados			
Olives			
Stewed, fresh			
, dried			
Dried			
Fruit rolls			
Canned with syrup			
Canned with water/juice			
Fruit juice			
Fruit nectar			
Fruit salad			
Other			

F. FATS AND OILS

Type	When	g/day	g/week	How
Butter				
Margarine, hard				
, soft				
,light				
Beef fat				
Sheep fat				
Drippings				
Oil, cooking				
Mayonnaise				
Salad dressings				

G. SUGAR AND SWEETS

Type	Quantity	Comments
Sugar, white		
, brown		
, other		
Artificial Sweeteners		
Jam		
Honey		
Syrup		
Sweets		
Chewing gum		
Peppermints		
Chocolates		
Other		
Cold drinks		
Syrups, conc.		
Carbonated		

H. MISCELLANEOUS

Type	Quantity	Comments
Atchar		
Chutney		
Tomato sauce		
Other		

I. BEVERAGES

Type	Quantity	Comments
Coffee		
Tea		
Milo		
Cacao		
Ovaltine		
Fermented beer		
, alcoholic		
, non-alcoholic		
Beer		
Wine		
Brandy		
Other alcoholic drinks		

APPENDIX C

CONSENT FORM

I, ..., hereby give consent for the proposed procedure to be performed on me as part of the research project: "The effects of a traditional African diet on the metabolic control of black patients with Type II diabetes mellitus". Mrs. A. Gresse has given me a full explanation of the probable advantages and possible dangers inherent in the procedure of the study.

She has also explained to me in what way the proposed precedure differs from the usual procedure. The procedure consists of test meals that will be eaten and blood samples that will be drawn. Anthropometric measurements will also be taken. The study will be executed by Mrs. A. Gresse and her staff.

My consent is freely given on the understanding that it may be withdrawn at any time.

Name of subject:.....

Signature:.....

Signature of witness (1):.....

Signature of witness (2):.....

Project leader, Mrs. A. Gresse:.....
MEDUNSA advisor/guardian: Prof. I.I. Glatthaar
Department of Human Nutrition

Date: 1990-03-29

APPENDIX D

DIABETIC PROJECT DIET SHEET SOYA GROUP

NAME.....

		Men	Women
Breakfast:	Porridge, soft, maize meal	3 cups	2 cups
	Apple/Orange	1	1
	Coffee/Tea (skim milk)	1 cup	1 cup
10:00	Bread, brown	3 slices	2 slices
	Coffee/Tea (skim milk)	1 cup	1 cup
Lunch:	Porridge, stiff, maize meal	2 cups	1 cup
	Soya mince	1 portion = ½ cup	
	Vegetables (spinach, cabbage)	2 portions of ½ cup each	
15:00:	Bread, brown	3 slices	2 slices
	Coffee/Tea (skim milk)	1 cup	1 cup
Supper:	Porridge, stiff, maize meal	3 cups	2 cups
	Soya mince	1 portion = ½ cup	
	Vegetables	2 portions of ½ cup each	
Bedtime:	Bread, brown	3 slices	2 slices
	Skim milk	1 glass	

APPENDIX E

DIABETIC PROJECT DIET SHEET MEAT GROUP

NAME.....

		Men	Women
Breakfast:	Porridge, soft, maize meal	3 cups	2 cups
	Milk	½ cup	½ cup
	Apple/Orange	1	1
	Coffee/Tea (skim milk)	1 cup	1 cup
10:00	Bread, brown	3 slices	2 slices
	Coffee/Tea (skim milk)	1 cup	1 cup
Lunch:	Starch	2 cups	1 cup
	Meat/egg/cheese	1 portion = ½ cup	
	Vegetables (spinach, cabbage)		
15:00:	Bread, brown	3 slices	2 slices
	Coffee/Tea (skim milk)	1 cup	1 cup
Supper:	Starch	3 cups	2 cups
	Meat/etc.	1 portion = ½ cup	
	Vegetables		
Bedtime:	Bread, brown	3 slices	2 slices
	Skim milk	1 glass	

BIBLIOGRAPHY

ABBOT, W.G.H., Boyle, V.L., Grundy, S.M. & Howard, B.V. 1989. Effects of replacing saturated fat with complex carbohydrate in diets of subjects with NIDDM. *Diabetes Care*, 12(2):102-107.

ABRAHAMSON, M.J. 1988. Diabetes mellitus, hypertension and insulin. *South African Medical Journal*, 74(4):148-149.

ABRAMS, J.J., Ginsberg, H. & Grundy, S.M. 1982. Metabolism of cholesterol and plasma triglycerides in nonketotic diabetes mellitus. *Diabetes*, 31(10):903-910.

ALBERTI, K.G.M.M. & Hockaday, T.D.R. 1987. Diabetes mellitus (In Wheatherall, D.J., Ledingham, J.G.H. & Warrell, D.A. (Eds.) Oxford textbook of medicine. 2nd Ed. Oxford: Oxford University Press.) p. 9.51-9.101.

ALMéR, L.-O. & Pandolfi, M. 1976. Fibrinolysis and diabetic retinopathy. *Diabetes*, 25(no. 9, suppl 2):807-810.

AMERICAN DIABETES ASSOCIATION. 1986. Glycemic effects of carbohydrates. *Diabetes Care*, 7(6):607-608.

AMERICAN DIABETES ASSOCIATION. 1987. Task Force on Nutrition and Exchange Lists: Nutritional recommendations and principles for individuals with diabetes mellitus. *Diabetes Care*, 10(1):126-132.

AMERICAN DIABETES ASSOCIATION. 1990a. Nutritional recommendations and principles for individuals with diabetes mellitus. *Diabetes Care*, 13(suppl 1):18-25.

AMERICAN DIABETES ASSOCIATION. 1990b. Diabetes mellitus and exercise. *Diabetes Care*, 13(7):785-789,804-805.

AMERICAN DIABETES ASSOCIATION. 1990c. Office guide to diagnosis and classification of diabetes mellitus and other categories of glucose intolerance. *Diabetes Care*, 13(suppl 1):3-4.

AMERICAN DIETETIC ASSOCIATION. 1987. Position of the American Dietetic Association: Appropriate use of nutritive and non-nutritive sweeteners. *Journal of the American Dietetic Association*, 87(12):1689-1694.

AMERICAN DIETETIC ASSOCIATION. 1988. Position of the American Dietetic Association: Health implications of dietary fiber. *Journal of the American Dietetic Association*, 88(2):216-221.

AMERICAN HEART ASSOCIATION. 1990. The cholesterol facts. *Circulation*, 81(5):1721-1733.

AMERICAN MEDICAL ASSOCIATION. 1983. Council on scientific affairs: Dietary and pharmacologic therapy for the lipid risk factors. *Journal of the American Medical Association*, 250(14):1873-1879.

ANASTASI, A. 1983. Psychological Testing. New York: MacMillan.

ANDERSON, J.W. 1980. High fiber diets in diabetes and hypertriglyceridemia. *Journal of the Canadian Medical Association*, 123:975-979.

ANDERSON, J.W. 1982. Dietary fibre and diabetes. (In Vahouny, G.V. & Kritchevsky, D. Eds. Dietary fibre in health and disease. New York: Plenum Press. pp. 151-168.)

ANDERSON, J.W. 1983. Plant fibre and blood pressure. *Annals of Internal Medicine*, 98(2):842-846.

ANDERSON, J.W. 1986. Dietary fibre in nutrition management of diabetes. (In Vahouny, G.V. & Kritchevsky, D. Eds. Dietary Fibre: Basic and Clinical Aspects. New York: Plenum Press. pp. 343-359).

ANDERSON, J.W. & Geil, P.B. 1988. New perspectives in nutrition management of diabetes mellitus. *American Journal of Medicine*, 85(suppl 5A):159-165.

ANDERSON, J.W., Gustafson, N.J., Bryant, C.A. & Tietyen-Clark, J.T. 1987. Dietary fiber and diabetes: A comprehensive review and practical application. *Journal of the American Dietetic Association,* 87(9):1189-1197.

ANDERSON, J.W. & Sieling, B. 1981. High-fiber diets for diabetics: Unconventional but effective. *Geriatrics*, 36(5):64-72.

ANON. 1963. Diabetes in non-white people in Southern Africa. *South African Medical Journal*, 37(48):1193-1194.

ANON. 1990. HDL - most important measure of coronary risk? *Hospital Medicine*, 12(10):20-21.

AOKI, T.T. 1985. Hormone-fuel interrelationships in normal, fasting, and diabetic man. (In Marble, A., Krall, L.P., Bradley, R.F., Christlieb, A.R. & Soeldner, J.S. Eds. Joslin's diabetes mellitus. 12th Ed. Philadelphia: Lea & Febiger. p. 138-157).

ASHBY, J.P. & Frier, B.M. 1988. Is serum fructosamine a clinically useful test? *Diabetic Medicine*, 5:118-121.

ASMAL, A.C., Dayal, B., Jialal, I., Leary, W.P., Omar, M. A. K., Pillay, N.L. & Thandroyen, F.T. 1981. Non-insulin-dependent diabetes mellitus with early onset in Blacks and Indians. *South African Medical Journal*, 60(2):93-96.

ASMAL, A.C. & Leary, W.P. 1975. Carbohydrate tolerance, plasma insulin, growth hormone and lipid levels in Indian and Black diabetics. *South African Medical Journal*, 49(20):810-812.

AVOGARO, P., Bon, G.B., Cuzzolato, G. & Quinci, G.B. 1979. Are apolipoproteins better discriminators than lipids for atherosclerosis? *Lancet*, 1(8122):901-904.

AXELSON, J.M. & Csernus, M.M. 1983. Reliability and validity of a food frequency checklist. *Journal of the American Dietetic Association*, 83(2):152-155.

BAGDADE, J.D., Stewart, M. & Walters, E. 1978. Impaired granulocyte adherence. A reversible defect in host defence in patients with poorly controlled diabetes. *Diabetes*, 27(6):677-681.

BAILEY, K.D. 1982. Methods of social research. 2nd Ed. London: Collier MacMillan.

BAILEY, C.J. & Day, C. 1989. Traditional plant medicines as treatments for diabetes. *Diabetes Care*, 12(8):553-564.

BALISTRERI, W.F. & Shaw, L.M. 1986. Liver function. (In Tietz, N.W. Ed. Textbook of clinical chemistry. London: W.B. Saunders Co. pp.1373-1434).

BANGA, J.D. & Sixma, J.J. 1986. Diabetes mellitus, vascular disease and thrombosis. *Clinics in Haematology*, 15(2):456-492.

BARNARD, R.J., Lattimore, L., Holly, R.G., Cherny, S. & Pritikin, N. 1982. Response of non-insulin-dependent diabetic patients: an intensive program of diet and exercise. *Diabetes Care*, 5(4):370-374.

BEEBE, C.A., Pastors, J.C., Powers, M.A. & Wylie-Rosett, J. 1991. Nutrition manangement of individuals with noninsulin-dependent diabetes mellitus in the 1990s: a review by the diabetes care and education dietetic practice group. *Journal of the American Dietetic Association*, 91(2):196-207.

BEHALL, K.M., Scholfield, D.J. & Canary, J. 1988. Effect of starch structure on glucose and insulin responses in adults. *American Journal of Clinical Nutrition*, 47(3):428-432.

BEMBRIDGE, T.J. 1987. Some aspects of household diet and family income problems in Transkei. *South African Medical Journal*, 72(6):425-428.

BENNET, P.H. 1983. Diabetes in developing countries and unusual populations. (In Mann, J.I., Pyörälä, K. & Teuscher, A. (Eds). Diabetes in epidemiological perspective. New York: Churchill Livingstone. p. 43-57).

BERGER, M. & Berchtold, P. 1985. Insulin transport and action at target cells. (In Marble, A., Krall, L.P., Bradley, R.F., Christlieb, A.R. & Soeldner, J.S. Eds. Joslin's diabetes mellitus. 12th Ed. Philadelphia: Lea & Febiger. p. 82 -109).

BERN, M.M. & Busick, E.J. 1985. Disorders of blood and diabetes. (In Marble, A., Krall, L.P., Bradley, R.F., Christlieb, A.R. & Soeldner, J.S. Eds. Joslin's diabetes mellitus. 12th Ed. Philadelphia: Lea & Febiger. p. 748-752).

BEYERS, M.J.C., Hammer, M.L. & Groenewald, G. 1979. Foods commonly used by the Xhosa-speaking people of Transkei and Ciskei: Weights for various household measures. *Journal of Dietetics and Home Economics*, 7(3):96-100.

BILLINGHAM, M.S., Milles, J.J., Bailey, C.J. & Hall, R.A. 1989. Lipoprotein subfraction composition in NIDD treated by diet, sulphonylurea, and insulin. *Metabolism*, 38(9):850-857.

BJÖRNTORP, P & Sjöström, L. 1978. Carbohydrate storage in man: speculations and some quantitative considerations. *Metabolism*, 27(12, suppl 2):1853-1865.

BOEIJEN, W.G.M., Brummer, H.J.M. & Cramwinckel A.B. 1985. Het gebruik van foto's bij het voedingsondersoek. *Voeding*, 46(2):63-65.

BOLLAND, J.E., Yuhas, J.A. & Bolland, T.W. 1988. Estimation of food portion sizes: Effectiveness of training. *Journal of the American Dietetic Association*, 88(7):817-821.

BORNET, F.R.J., Costagliola, D., Rizkalla, S.W., Blayo, A., Fontvieille, A.-M., Haardt, M.-J., Letanoux, M., Tchobroutsky, G., Slama, G. 1987. Insulinemic and glycemic indexes of six starch-rich foods taken alone and in a mixed meal by type 2 diabetics. *American Journal of Clinical Nutrition*, 45(3):588-595.

BORRELLI, R., Cole, T.J., Di Biase, G. & Contaldo, F. 1989. Some statistical considerations on dietary assessment methods. *European Journal of Clinical Nutrition*, 43(7):453-464.

BRAND, J.C., Nicholson, P.L., Thorburn, A.W. & Truswell, A.S. 1985. Food processing and the glycemic index. *American Journal of Clinical Nutrition*, 42(6):1192-1196.

BRIGGS, B.R., Jackson, W.P.U., Du Toit, E. & Botha, M.C. 1980. The histocompatibility (HLA) antigen distribution in diabetes in southern African Blacks (Xhosa). *Diabetes*, 29(1):68-70.

BROWNLEE, M. 1985. Microvascular disease and related abnormalities; Their relation to control of diabetes. (In Marble, A., Krall, L.P., Bradley, R.F., Christlieb, A.R. & Soeldner, J.S. Eds. Joslin's diabetes mellitus. 12th Ed. Philadelphia: Lea & Febiger. p. 185-216).

BROWNLEE, M., Vlassara, H. & Cerami, A. 1983. Nonenzymatic glycosylation reduces the susceptibility of fibrin to degration by plasmin. *Diabetes*, 32(7):680-684.

CAHILL, G.F. 1985. Current concepts of diabetes (In Marble, A., Krall, L.P., Bradley, R.F., Christlieb, A.R. & Soeldner, J.S. Eds. Joslin's diabetes mellitus. 12th Ed. Philadelphia: Lea & Febiger. p. 1-11).

CALLE-PASCUAL, A.L., Bordiu, E., Romeo, S., Romero, C., Martin-Alvarez, P.J. & Maranes, J.P. 1986. Food glycaemic index or meal response? *Human Nutrition: Applied Nutrition*, 40A(4):282-286.

CANADIAN DIABETES ASSOCIATION. 1981. Special report committee of the Canadian Diabetes Association. 1980 guidelines for the nutritional management of diabetes mellitus: A special report from the Canadian Diabetes Association. *Journal of the Canadian Dietetic Association*, 42:110-118.

CARON, D., Poussier, P., Marliss, E.B. & Zinman, B. 1982. The effect of postprandial exercise on meal-related glucose intolerance in insulin-dependent diabetic individuals. *Diabetes Care*, 5(4):364-369.

CEFALU, W.T., Parker, T.B. & Johnson, C.R. 1988. Validity of serum fructosamine as index of short-term glycemic control in diabetic outpatients. *Diabetes Care*, 11(8):662-664.

CHEW, I., Brand, J.C., Thorburn, A.W. & Truswell, A.S. 1988. Application of glycemic index to mixed meals. *American Journal of Clinical Nutrition*, 47(1):53-56.

CHRISTAU, B.L., Kromann, H., Andersen, O.O., Christy, M., Buschard, K., Arnung, K., Kristensen, I.H., Peitersen, B., Steinrud, J. & Nerup, J. 1977. Incidence, seasonal and geographical patterns of juvenile-onset insulin-dependent diabetes mellitus in Denmark. *Diabetologia*, 13(5):281-284.

CLARKE, W.L., Cox, D., Gonder-Frederick, L.A., Carter, W. & Pohl, S.L. 1987. Evaluating clinical accuracy of systems for self-monitoring of blood glucose. *Diabetes Care*, 10(5):622-628.

CLEMENTS, R.S. & Reynertson, R. 1977. Myoinositol metabolism in diabetes mellitus. Effect of insulin treatment. *Diabetes*, 26(3):215-221.

COHN, P.F., Gabbay, S.I. & Weglicki, W.B. 1976. Serum lipid levels in angiographically defined coronary artery disease. *Annals of Internal Medicine*, 84(3):241-245.

COLAGIURI, S., Miller, J.J. & Edwards, R.A. 1989. Metabolic effects of adding sucrose and aspartame to the diet of subjects with noninsulin-dependent diabetes mellitus. *American Journal of Clinical Nutrition*, 50(3):474-478.

COLLER, B.S., Frank, R.N., Milton, R.C. & Gralnick, H.R. 1978. Plasma cofactors of platelet function: correlation with diabetic retinopathy and hemoglobins A_{1a-c}. *Annals of Internal Medicine*, 88(3):311-314.

COLLIER, G.R. & O'Dea, K. 1983. The effect of co-ingestion of fat on the glucose, insulin, and gastric inhibitory polypeptide responses to carbohydrate and protein. *American Journal of Clinical Nutrition*, 37(6):941-944.

COLLIER, G.R., Wolever, T.M.S., Wong, G.S. & Josse, R.G. 1986. Prediction of glycemic response to mixed meals in non-insulin-dependent diabetic subjects. *American Journal of Clinical Nutrition*, 44(3):349-352.

COLLINGS, P., Williams, C. & Macdonald, I. 1981. Effects of cooking on serum glucose and insulin responses to starch. *British Medical Journal*, 282(6269):1032.

COOPPAN, R. 1987. Determining the most appropriate treatment for patients with non-insulin-dependent diabetes mellitus. *Metabolism*, 36(2)(suppl 1):17-21.

COULSTON, A.M., Hollenbeck, C.B., Donner, C.C., Williams, R., Chiou, Y.A.M. & Reaven, G.M. 1985. Metabolic effects of added dietary sucrose in individuals with noninsulin-dependent diabetes mellitus (NIDDM). *Metabolism*, 34(10):962-966.

COULSTON, A.M., Hollenbeck, C.B., Liu, G.C., Williams, R.A., Starich, G.H., Mazzaferri, E.L. & Reaven, G.M. 1984. Effect of source of dietary carbohydrate on plasma glucose, insulin and gastric inibitory polypeptide responses to test meals in subjects with noninsulin-dependent diabetes mellitus. *American Journal of Clinical Nutrition*, 40(5):965-970.

COULSTON, A.M., Hollenbeck, C.B., Swislocki, A.L.M. & Reaven G.M. 1987. Effect of source of dietary carbohydrate on plasma glucose and insulin responses to mixed meals in subjects with NIDDM. *Diabetes Care*, 10(4):395-400.

COULSTON, A.M., Hollenbeck, C.B., Swislocki, A.L.M. & Reaven G.M. 1989. Persistence of hypertriglyceridemic effect of low-fat high-carbohydrate diets in NIDDM patients. *Diabetes Care*, 12(2):94-101.

CRAPO, P.A., Reaven G.M. & Olefsky, J. 1976. Plasma glucose and insulin responses to orally administered simple and complex carbohydrates. *Diabetes*, 25(4):741-747.

CRAPO, P. & Vinik, A.I. 1987. Nutrition controversies in diabetes management. *Journal of the American Dietetic Association*, 87(1):25-26.

CROUS, J.M. & Borchardt, S. 1982. Eetgewoontes van die Pedi-huisgesinne in Atteridgeville. *Journal of Dietetics and Home Economics*, 10(1):29-31.

CROUS, J.M. & Borchardt, S. 1984. Dieetpatrone van Venda-gesinne in Atteridgeville. *Journal of Dietetics and Home Economics*, 12(2):41-44.

CROUS, J.M. & Borchardt, S. 1986. Dieetpatrone van Venda-gesinne in Tshikunda Malema. *Journal of Dietetics and Home Economics*, 14(2):44-47.

D'ANTONIO, J.A., Ellis, D., Doft, B.H., Becker, D.J., Drash, A.L., Kuller, L.H., Orchard, T.J. 1989. Diabetes complications and glycemic control. *Diabetes Care*, 12(10):694-700.

DEAN, A.G. 1986. EPI INFO. 3rd Ed. Stone Mountain: USD Inc.

DEFRONZO, R.A. 1981. The effect of insulin on renal sodium metabolism: a review with clinical implications. *Diabetologia*, 21(3):165-171.

DEFRONZO, R.A., Ferrannini, E. & Koivisto, V. 1983. New concenpts in the pathogenesis and treatment of noninsulin dependent diabetes mellitus. *American Journal of Medicine*, 74:52.

DEL TOMA, E., Clementi, A., Marcelli, M., Cappelloni, M. & Lintus, C. 1988. Food fiber choices for diabetic diets. *American Journal of Clinical Nutrition*, 47(2):243-246.

DESPRES, J.-P., Moorjani, S., Lupien, P.J., Tremblay, A., Nadeau, A. & Bouchard, C. 1990. Regional distribution of body fat, plasma lipoproteins, and cardiovascular disease. *Arteriosclerosis*, 10:497-511.

DIAZ, E.O., Villar, J., Immink, M. & Gonzales, T. 1989. Bioimpedance or anthropometry? *European Journal of Clinical Nutrition*, 43(2):129-137.

DISTILLER, L.A. 1989. Insulin therapy in non-insulin dependent diabetes mellitus. *Hospital Medicine*, May/June:8-9.

DOMINICZAK, M.H., Smith, L.A., McNaught, J. & Paterson, K.R. 1988. Assessment of past glycemic control. *Diabetes Care* 11(4):358-359.

DU PLESSIS, J.N. 1963. Die invloed van stamgewoontes op die voedingspatroon van die Noord-Sotho. (Lecture to students in Anthropology, University of Pretoria.) Pretoria. 6 p. (Unpublished).

DWYER, J.T., Krall, E.A. & Coleman, K.A. 1987. The problem of memory in nutritional epidemiology research. *Journal of the American Dietetic Association*, 87(11):1509-1512.

EDINGTON, J.D., Geekie, M., Carter, R., Benfield, L., Ball, M. & Mann, J. 1989. Serum lipid response to dietary cholesterol in subjects fed a low-fat, high-fiber diet. *American Journal of Clinical Nutrition*, 50(1):58-62.

EDITORIAL. 1963. Diabetes in non-white people in Southern Africa. *South African Medical Journal*, 37(48):1193-1194.

EGEBERG, O. 1963. The blood coagulability in retinopathy. *Scandinavian Journal of Clinical Laboratory Investigation*, 15:522-535.

ENGLYST, H.N., Bingham, S.A., Runswick, E., Collinson, E. & Cummings, J.H. 1988. Dietary fibre (non-starch polysaccharides) in fruit, vegetables and nuts. *Journal of Human Nutrition and Dietetics*, 1:247-286.

ENGLYST, H.N., Bingham, S.A., Runswick, E., Collinson, E. & Cummings, J.H. 1989. Dietary fibre (non-starch polysaccharides) in cereal products. *Journal of Human Nutrition and Dietetics*, 2:253-271.

ENGLYST, H.N. & Cummings, J.H. 1985. Digestion of the polysaccharides of some cereal foods in the human small intestine. *American Journal of Clinical Nutrition*, 42(5):778-787.

ENGLYST, H.N., Trowell, H., Southgate, D.A.T. & Cummings, J.H. 1987. Dietary fiber and resistant starch. *American Journal of Clinical Nutrition*, 46(4):873-874.

ERDMAN, J.W. & Fordyce, E.J. 1989. Soy products and the human diet. *American Journal of Clinical Nutrition*, 49(5):725-737.

ENTMACHER, P.S., Krall, L.P. & Kranczer, S.N. 1985. Diabetes mortality from vital statistics. (In Marble, A., Krall, L.P., Bradley, R.F., Christlieb, A.R. & Soeldner, J.S. Eds. Joslin's diabetes mellitus. 12th Ed. Philadelphia: Lea & Febiger. p. 278-297).

ERKELENS, D.W., Stofkooper, A., Van den Bogaard, E. & Van der Snoek, B.E. 1985. Glycaemic effect of mono-, di- and polysaccharides in a mixed meal in diabetic patients. *Netherlands Journal of Medicine*, 28(2):157-163.

EVERS, S.E., McCracken, E. & Deagle, G. 1989. Body fat distribution and NIDDM in North American Indians. *Nutrition Research*, 9(9):977-988.

FABER, O.K., Beck-Nielsen, H., Binder, C., Butzer, P., Damsgaard, E.M., Froland, F., Hjollund, E., Lindskov, H.O., Melander, A., Pedersen, O., Petersen, P., Sorensen, N.S. & Wahlin-Boll, E. 1990. Acute actions of sulfonylurea drugs during long-term treatment of NIDDM. *Diabetes Care*, 13(3, suppl):26-31.

FEHILY, A.M., Milbank, J.E., Yarnell, J.W.G., Hayes, T.M., Kubiki, A.J. & Eastham, R.D. 1982. Dietary determinants of lipoproteins, total cholesterol, viscosity, fibrinogen and blood pressure. *American Journal of Clinical Nutrition*, 36(4, suppl):890-896.

FLOOD, T.M., Halford, B.M., Cooppan, R. & Marble, A. 1985. Dietary management of diabetes. (In Marble, A., Krall, L.P., Bradley, R.F., Christlieb, A.R. & Soeldner, J.S. Eds. Joslin's diabetes mellitus. 12th Ed. Philadelphia: Lea & Febiger. p. 357-372).

FLüCKIGER, R., Woodtli, T. & Berger, W. 1987. Evaluation of the fructosamine test for the measurement of plasma protein glycation. *Diabetologia*, 30(8):648-652.

FOOD AND NUTRITION BOARD. 1989. Recommended dietary allowances (RDA's). Revised 1989. Washington DC: National Academy of Sciences - National Research Council.

FRANK, G.C., Hollatz, A.T., Webber, L.S. & Berenson, G.S. 1984. Effect of interviewer recording practices on nutrient intake - Bogalusa Heart Study. *Journal of the American Dietetic Association*, 84(12):1432-1439.

FRANZ, M.J. 1987. Exercise and the management of diabetes mellitus. *Journal of the American Dietetic Association*, 87(7):872-880.

FREEDMAN, D.S., Jacobsen, S.J., Barboriak, J.J., Sobocinski, K.A., Anderson, A.J., Kissebah, A.H., Sasse, E.A. & Gruchow, H.W. 1990. Body fat distribution and male/female differences in lipids and lipoproteins. *Circulation*, 81(5):1498-1506.

FULLER, J.H., Keen, H., Jarret, R.J., Omer, T., Meade, T.W., Chakrabarti, R., North, W.R.S. & Stirling, Y. 1979. Haemostatic variables associated with diabetes and its complications. *British Medical Journal*, 20(Oct):964-966.

GANDA, O.P. 1980. Pathogenesis of macro-vascular disease in the human diabetic. *Diabetes*, 29(11):931-942.

GANDA, O.P. 1985a. Hormones and related factors affecting the secretion and actions of insulin. (In Marble, A. Krall, L.P., Bradley, R.F., Christlieb, A.R. & Soeldner, J.S. Eds. Joslin's diabetes mellitus. 12th Ed. Philadelphia: Lea & Febiger. p. 158-186).

GANDA, O.P. 1985b. Pathogenesis of macrovascular disease including the influence of lipids. (In Marble, A. Krall, L.P., Bradley, R.F., Christlieb, A.R. & Soeldner, J.S. Eds. Joslin's diabetes mellitus. 12th Ed. Philadelphia: Lea & Febiger. p. 217-250).

GANDOLFO, G.M., De Angelis, A. & Torresi, M.V. 1980. Determination of antithrombin III activities by different methods in diabetic patients. *Haemostasis*, 9:15-17.

GANNON, M.C. & Nuttall, F.Q. 1987. Factors affecting interpretation of postprandial glucose and insulin areas. *Diabetes Care*, 10(6):759-763.

GARN, S.M., Leonard, W.R. & Hawthorne, V.M. 1986. Three limitations of the body mass index. *American Journal of Clinical Nutrition*, 44(6):997-997.

GARN, S.M., Leonard, W.R. & Rosenberg, K. 1986. Body build dependence, stature dependence and influence of lean tissue on the body mass index. *Ecology of Food and Nutrition*, 19(2):163-165.

GELFAND, M. 1973. The dietary habits of the African and European, with special reference to the Shona-speaking peoples. *South African Medical Journal*, 47(33):1501-1503.

GERICKE, G.J. & Muller, Y. 1987. Glukemiese indeks van voedsel: 'n oorsig. *Tydskrif vir Dieetkunde en Huishoudkunde*, 15(3):89-92.

GOLAY, A., Coulston, A.M., Hollenbeck, C.B., Kaiser, L.L., Wursch, P. & Reaven, G.M. 1986. Comparison of metabolic effects of white beans processed into two different physical forms. *Diabetes Care*, 9(3):260-266.

GOODHART, R.S. & Shils, M.E. 1980. Modern nutrition in health and disease. Philadelphia. Lea & Febiger.

GOODNIGHT, S.H., Harris, W.S., Connor, W.E. & Illingworth, D.R. 1982. Polyunsaturated fatty acids, hyperlipidemia and thrombosis. *Arteriosclerosis*, 2:87-113.

GOUWS, E. & Langenhoven, M.L. 1986. RIND Food Composition Tables. 2nd Ed. Parow : South African Medical Research Council.

GRESSE, A. 1987. Die koolhidraatsamestelling van sekere groentesoorte wat aan verskillende prosesseringstegnieke onderwerp is. Bloemfontein. 190 p. (Dissertation (M Sc Home Economics - University of the Orange Free State).

GRUNDY, S.M., Barret-Connor, E., Rudel, L.L. Miettinen, T & Spector A.A. 1988. Workshop on the impact of dietary cholesterol on plasma lipoproteins and atherogenesis. *Arteriosclerosis,* 8:95-101.

GUARE, J.C., Wing, R.R, Marcus, M.D., Epstein, L.H., Burton, L.R. & Gooding, W.E. 1989. Analysis of changes in eating behaviour and weight loss in Type II diabetic patients. Which behaviours to change. *Diabetes Care,* 12(7):500-503.

GUILFORD, J.P. & Fruchter, B. 1978. Fundamental statistics in psychology and education. 6th Ed. Tokyo: McGraw Hill Kogakusha.

GUTHRIE, H.A. 1989. Interpretation of data on dietary intake. *Nutrition Reviews,* 47(2):33-38.

HAGAN, J. & Wylie-Rosett, J. 1989. Lipids: Impact on dietary prescription in diabetes. *Journal of the American Dietetic Association,* 89(8):1104-1111.

HAGANDER, B., Asp, N.-G., Efendic, S., Nilsson-Ehle, P. & Schersten, B. 1988. Dietary fiber decreases fasting blood glucose levels and plasma LDL concentration in noninsulin-dependent diabetes mellitus. *American Journal of Clinical Nutrition,* 47(5):852-858.

HAGANDER, B., Asp, N.-G., Ekman, R., Nilsson-Ehle, P. & Schersten, B. 1989. Dietary fibre enrichment, blood pressure, lipoprotein profile and gut hormones in NIDDM patients. *European Journal of Clinical Nutrition*, 43(1):35-44.

HALTER, J.B. & Morrow, L.A. 1990. Use of sulfonylurea drugs in elderly patients. *Diabetes Care*, 13(suppl 2):86-92.

HAMMAN, R.F. 1983. Diabetes in affluent societies. (In Mann, J.I., Pyörälä, K. & Teuscher, A. (Eds). Diabetes in epidemiological perspective. New York: Churchill Livingstone. p. 7-42).

HANKIN, J.H. 1986. 23rd Lenna Frances Cooper Memorial Lecture. A diet history method for research, clinical and community use. *Journal of the American Dietetic Association*, 86(7):868-875.

HARRIS, M.I. 1989. Impaired glucose tolerance in the US population. *Diabetes Care*, 12(7):464-474.

HARRISON, H.E., Reece, A.H. & Johnson, M. 1980. Effect of insulin treatment on prostacyclin in experimental diabetes. *Diabetologia*, 18:65-68.

HEATON, K.W., Marcus, S.N., Emmet, P.M. & Bolton, C.H. 1988. Particle size of wheat, maize and oat test meals - effect on plasma glucose and insulin responses and on the rate of starch digestion *in vitro*. *American Journal of Clinical Nutrition*, 47(4):675-682.

HEINS, J.M., Wylie-Rosett, J. & Green-Davis, S. 1987. The new look in diabetic diets. *American Journal of Nursing*, Feb:196-198.

HENRY, D., Bell, G.D. & Glithero, P. 1979. Plasma high-density lipoproteins. *New England Journal of Medicine*, 300(14):797-798.

HENRY, H.J., McCarron, D.A., Morris, C.D. & Parrott-Garcia, M. 1985. Increasing calcium intake lowers blood pressure: the literature reviewed. *Journal of the American Dietetic Association*, 85(2):182-185.

HENRY, R.R., Wiest-Kent, T.A., Scheaffer, L. 1986. Metabolic consequences of very-low-calorie diet therapy in non-insulin-dependent diabetic and non-diabetic subjects. *Diabetes*, 35(2):155-164.

HERMANN, L.S. 1990. Biguanides and sulfonylureas as combination therapy in NIDDM. *Diabetes Care*, 13(3, suppl):37-41.

HILL, M., ed. 1987. BMPD user's digest. 4th Ed. Los Angelis, Calif: BMPD Statistical Software, Inc.

HJOLLUND, E., Pedersen, O., Richelsen, B., Beck-Nielsen, H. & Sorensen, N. 1983. Increased insulin binding to adipocytes and monocytes and increased insulin sensitivity of glucose transport and metabolism in adipocytes from non-insulin-dependent diabetes after a low-fat, high-starch/fibre diet. *Metabolism*, 32(11):1067-1075.

HOLLENBECK, C.B., Coulston, A.M. & Reaven, G.M. 1989. Effects of sucrose on carbohydrate and lipid metabolism in NIDDM patients. *Diabetes Care*, 12(1, suppl 1):62-66.

HOOVER-PLOW, J., Savesky, J. & Dailey, G. 1987. The glycemic response to meals with six different fruits in insulin-dependent diabetics using a home blood-glucose monitoring system. *American Journal of Clinical Nutrition*, 45(1):92-97.

HOTHER-NIELSEN, O., Faber, O., Sorensen, N.S.T. & Beck-Nielsen, H. 1988. Classification of newly diagnosed diabetic patients as insulin-requiring or non-insulin-requiring based on clinical and biochemical variables. *Diabetes Care*, 11(7):531-537.

HUDDLE, K.R.L. 1989. Insulin dependent diabetes mellitus. *Hospital Medicine*, May/June:11-13.

HUGHES, T.A., Atchison, J., Hazelrig, J.B. & Boshell, B.R. 1989. Glycemic responses in insulin-dependent diabetic patients: effect of food composition. *American Journal of Clinical Nutrition*, 49(4):658-666.

ISAACSON, C., Milne, F.J. & Van Niekerk, I. 1989. Hypertension in black South Africans - new perspectives on old material. *South African Medical Journal*, 76(7):323-324.

IWAI, M., Yoshino, G., Matsushita, M., Morita, M., Matsuba, K., Kazumi, T. & Baba, S. 1990. Abnormal lipoprotein composition in normolipidemic diabetic patients. *Diabetes Care*, 13(7):792-796.

JACKSON, W.P.U. 1978a. Epidemiology of diabetes in South Africa. (In Miller, M. & Bennet, P.H. (Eds) Advances in metabolic disorders. Vol. 9. New York: Academic Press) p. 111-146.

JACKSON, W.P.U. 1978b. The genetics of diabetes mellitus. *South African Medical Journal*, 53(13):481-490.

JACKSON, W.P.U. & Huskisson J.M. 1965. Diabetics: Inter-racial comparisons. *South African Medical Journal*, 39(23):526-531.

JACKSON, C.E. & Meier, D.W. 1968. Routine serum magnesium analysis. *Annals of Internal Medicine*, 69(4):743-748.

JAKOBSEN, J. 1978. Peripheral nerves in early experimental diabetes. Expansion of the endoneurial space as a cause of increased water content. *Diabetologia*, 14(2):113.

JENKINS, D.J.A., Ghafari, H., Wolever, T.M.S., Taylor, R.H., Jenkins, A.L., Barker, H.M., Fielden, H. & Bowling, A.C. 1982a. Relationship between rate of digestion of foods and post-prandial glycaemia. *Diabetologia*, 22(6):450-455.

JENKINS, D.J.A., Thomas, D.M., Wolever, M.S., Collier, G.R., Ocana, A., Rao, A.V., Buckley, G., Lam Y., Mayer, A. & Thompson, L.U. 1987a. Metabolic effects of a low-glycemic-index diet. *American Journal of Clinical Nutrition*, 46(6):968-975.

JENKINS, D.J.A., Thomas, D.M., Wolever, M.S., Kalmusky, J., Guidici, S., Giordano, C., Patten, R., Wong, G.S., Bird, J.N., Hall, M., Buckley, G., Csima, A. & Little, J.A. 1987b. Low-glycemic-index diet in hyperlipidemia: use of traditional starchy foods. *American Journal of Clinical Nutrition*, 46(1):66-71.

JENKINS, D.J.A., Thomas, D.M., Wolever, M.S., Kalmusky, J., Guidici, S., Giordano, C., Wong, G.S., Bird, J.N., Patten R., Hall, M., Buckley, G. & Little, J.A. 1985. Low glycemic index carbohydrate foods in the management of hyperlipidemia. *American Journal of Clinical Nutrition*, 42(4):604-617.

JENKINS, D.J.A., Thomas, D.M., Wolever, M.S., Taylor, R.H., Barker, H., Fielden, H., Baldwin, J.M., Bowling, A.C., Newman, H.C., Jenkins, A.L. & Goff, D.V. 1981. Glycaemic index of foods: a physiological basis for carbohydrate exchange. *American Journal of Clinical Nutrition*, 34(2):362-366.

JENKINS, D.J.A., Thomas, D.M., Wolever, M.S., Thorne, M.J., Jenkins, A.L., Wong, G.S., Josse, R.G. & Csima, A. 1984b. The relationship between glycemic response, digestibility, and factors influencing the dietary habits of diabetics. *American Journal of Clinical Nutrition*, 40(6):1175-1191.

JENKINS, D.J.A., Thomas, D.M., Wolever, M.S., Wong, G.S., Kenshole, A., Josse, R.G., Thompson, L.U. & Lam, K.Y. 1984c. Glycemic responses to foods: possible differences between insulin-dependent and noninsulin-dependent diabetics. *American Journal of Clinical Nutrition*, 40(5):971-981.

JENKINS, D.J.A., Wolever, T.M.S., Bacon, S., Nineham, R., Lees, R., Rowden, R., Lover, M., Hockaday, T.D. 1980a. Diabetic diets: high carbohydrate combined with high fiber. *American Journal of Clinical Nutrition*, 33:1729-1733.

JENKINS, D.J.A., Wolever, T.M.S., Buckley, G., Lam, K.Y., Giudici, S., Kalmusky, J., Jenkins, A.L., Patten, R.L., Bird, J., Wong, G.S., Josse, R.G. 1988. Low-glycaemic-index starchy foods in the diabetic diet. *American Journal of Clinical Nutirtion*, 48(2):248-254.

JENKINS, D.J.A., Wolever, T.M.S., Collier, G.R., Ocana, A., Rao, A.V., Buckley, G., Lam, Y., Mayer, A. & Thompson, L.U. 1987c. Metabolic effects of a low-glycemic-index diet. *American Journal of Clinical Nutrition*, 46(6):968-975.

JENKINS, D.J.A., Wolever, T.M.S., Jenkins, A.L., Josse, R.G. & Wong, G.S. 1984a. The glycaemic response to carbohydrate foods. *The Lancet*, 2(8399):388-391.

JENKINS, D.J.A., Wolever, T.M.S., Jenkins, A.L., Thorne, M.J., Kalusky, R.L.J., Reichert, R. & Wong, G.S. 1983a. The glycaemic index of foods tested in diabetic patients: A new basis for carbohydrate exchange favouring the use of legumes. *Diabetologia*, 24(4):257-264.

JENKINS, D.J.A, Wolever, T.M.S., Leeds, A.R., Gassull, M.A., Haisman, P., Dilawari, J., Goff, D.V., Metz, G.L. & Alberti, K.G.M.M. 1978. Dietary fibres, fibre analogues, and glucose tolerance: importance of viscosity. *British Medical Journal*, 1(6124):1392-1394.

JENKINS, D.J.A., Wolever, T.M.S & Nineham, R. 1980b. Improved glucose tolerance four hours after taking guar with glucose. *Diabetologia*, 19:21-24.

JENKINS, D.J.A., Wolever, T.M.S., Taylor, R.H., Griffiths, C., Krzeminska, K., Lawrie, J.A., Bennet, C.M., Goff, D.V., Sarson, D.L., & Bloom, S.R. 1982b. Slow release carbohydrate improves second meal tolerance. *American Journal of Clinical Nutrition*, 35(6):1339-1346.

JENKINS, D.J.A., Wong, G.S., Patten, R., Bird, J., Hall, M. Buckley, G.C., McGuire, V., Reichert, R. & Little, J.A. 1983b. Leguminous seeds in the dietary management of hyperlipidemia. *American Journal of Clinical Nutrition*, 38(4):567-573.

JOFFRES, M.R., Reed, D.M. & Yano, K. 1987. Relationship of magnesium intake and other dietary factors to blood pressure: the Honolulu heart study. *American Journal of Clinical Nutrition*, 45(2):469-475.

JONES, R.L. & Peterson, C.M. 1979. Reduced fibrinogen survival in diabetes mellitus. *Journal of Clinical Investigations*, 63(3):485-493.

JONES, R.L. & Peterson, C.M. 1981. Hematologic alterations in diabetes mellitus. *The American Journal of Medicine*, 70(1):399-352.

KAHN, C.R. 1983. The insulin receptor and insulin; The lock and key to diabetes. *Clinical Research*, 31(3):326-335.

KAHN, C.R. 1985. Pathophysiology of diabetes mellitus; An Overview. (In Marble, A., Krall, L.P., Bradley, R.F., Christlieb, A.R. & Soeldner, J.S. Eds. Joslin's diabetes mellitus. 12th Ed. Philadelphia: Lea & Febiger. p. 43 - 50).

KALDANY, A., Busick, E.J. & Eisenbarth, G.S. 1985. Diabetes mellitus and the immune system. (In Marble, A., Krall, L.P., Bradley, R.F., Christlieb, A.R. & Soeldner, J.S. Eds. Joslin's diabetes mellitus. 12th Ed. Philadelphia: Lea & Febiger. p. 51 - 64).

KANNEL, W.B., Wolf, P.A., Castelli, W.P. & D'Agostino, R.B. 1987. Fibrinogen and risk of cardiovascular disease. The Framingham Study. *Journal of American Medical Association*, 258(9):1183-1186.

KAPLAN, N.M. 1989. The deadly quartet. Upper-body obesity, glucose intolerance, hypertriglyceridemia, and hypertension. *Archives of Internal Medicine*, 149:1514-1520.

KARKECK, J.M. 1987. Improving the use of dietary survey methodology. *Journal of the American Dietetic Association*, 87(7):869-871.

KARVETTI, R-L. & Knuts, L-R. 1985. Validity of the 24-hour dietary recall. *Journal of the American Dietetic Association*, 85(11):1437-1442.

KATAN, M.B., Beynen, A.C., De Vries, J.H. & Nobels, A. 1986. Existence of consistent hypo- and hyperresponders to dietary cholesterol in man. *American Journal of Epidemiology*, 123(3):221-234.

KEEN, H., Jarret, R.J., Fuller, J.H. & McCarthy, P. 1981. Hyperglycemia and arterial disease. *Diabetes*, 30(suppl 2):49-53.

KERLINGER, F.N. 1986. Foundations of behavioural research. 3rd Ed. New York: Association Press.

KHIEM, T.G., Anderson, J.W. & Ward, K. 1976. Beneficial effects of a high carbohydrate, high fiber diet on hyperglycemic diabetic men. *American Journal of Clinical Nutrition*, 29(5):895-899.

KING, H., Zimmet, P., Raper, L.R. & Balkau, B. 1984. Risk factors for diabetes in three pacific populations. *American Journal of Epidemiology*, 119(3):396-409.

KINSELLA, J.E. 1986a. Food components with potential therapeutic benefits: the n-3 polyunsaturated fatty acids of fish oils. *Food Technology*, Feb.:89-97.

KINSELLA, J.E. 1986b. Dietary fish oils. Possible effects of n-3 polyunsaturated fatty acids in reduction of thrombosis and heart disease. *Nutrition Today*, Nov/Dec:7-14.

KIRSTEN, C.M. 1975. Die graandieetpatroon in die distrik Mount Ayliff. *Journal of Dietetics and Home Economics*, 3(1):17-19.

KIRSTEN, C.M. 1977. The complement of the cereal diet of Transkei people in the Mount Ayliff district. *Journal of Dietetics and Home Economics*, 5(1):19-22.

KLEIN, R., Klein, B.E.K., Moss, S.E., Davis, M.D. & DeMets, D.L. 1988. Glycosylated hemoglobin predicts the incidence and progression of diabetic retinopathy. *Journal of American Medical Association*, 260(19):2864-2871.

KOHNER, E.M. 1976. The problems of retinal blood flow in diabetes. *Diabetes*, 25(suppl 2):839.

KOLTERMAN, O.G. & Olefsky, J.M. 1984. The impact of sulfonylurea treatment upon the mechanisms responsible for the insulin resistance in Type II diabetes. *Diabetes Care*, 7(suppl.1):81-88.

KOSTNER, G.M. & Karadi, I. 1988. Lipoprotein alterations in diabetes mellitus. *Diabetologia*, 31(10):717-722.

KRALL, L.P. 1985a. Oral hypoglycemic agents. (In Marble, A., Krall, L.P., Bradley, R.F., Christlieb, A.R. & Soeldner, J.S. Eds. Joslin's diabetes mellitus. 12th Ed. Philadelphia: Lea & Febiger. p.412-452).

KRALL, L.P. 1985b. Education: a treatment for diabetes. (In Marble, A., Krall, L.P., Bradley, R.F., Christlieb, A.R. & Soeldner, J.S. Eds. Joslin's diabetes mellitus. 12th Ed. Philadelphia: Lea & Febiger. p.465-484).

KRALL, E.A. & Dwyer, J.T. 1987. Validity of a food frequency questionnaire and a food diary in a short-term recall situation. *Journal of the American Dietetic Association*, 87(10):1374-1377.

KRAUSE, M.V. & Mahan, L.K. 1984. Food, nutrition and diet therapy. 7th Ed. Philadelphia: WB Saunders.

KRóLEWSKI, A.S. & Warram, J.H. 1985. Epidemiology of diabetes mellitus. (In Marble, A., Krall, L.P., Bradley, R.F., Christlieb, A.R. & Soeldner, J.S. Eds. Joslin's diabetes mellitus. 12th Ed. Philadelphia: Lea & Febiger. p. 12-22).

KRòLEWSKI, A.S., Warram, J.H. & Christlieb, A.R. 1985. Onset, course, complications and prognosis of diabetes mellitus. (In Marble, A., Krall, L.P., Bradley, R.F., Christlieb, A.R. & Soeldner, J.S. Eds. Joslin's diabetes mellitus. 12th Ed. Philadelphia: Lea & Febiger. p. 251-277).

KRUGER, M.M. 1987. Die voorkoms van koronêre hartvatsiekte risikofaktore by 'n groep swartwerkers van die PU vir CHO. Potchefstroom. 45 p. (Projects (B. Sc Hons Physiology) - PU vir CHO.)

KUBISZ, P., Parizek, M. & Cronberg, S. 1986. Apolipoprotein B and platelet function in diabetes mellitus. *Haemostasis*, 16:424-427.

KUZWAYO, P. 1990. Personal communication. MEDUNSA : Pretoria.

LAAKSO, M. & Pyörälä, K. 1988. Lipid and lipoprotein abnormalities in diabetic patients with perpheral vascular disease. *Atherosclerosis*, 74:55-63.

LAAKSO, M., Pyörälä, K., Voutilainen, E. & Marniemi, J. 1987. Plasma insulin and serum lipids and lipoproteins in middle-aged non-insulin-dependent diabetic and non-diabetic subjects. *American Journal of Epidemiology*, 125(4):611-621.

LAINE, D.C., Thomas, W., Levitt, M.D. & Bantle, J.P. 1987. Comparison of predictive capabilities of diabetic exchange lists and glycemic index of foods. *Diabetes Care*, 10(4):387-394.

LANGENHOVEN, M.L., Conradie, P.J., Gouws, E., Wolmarans, P. & Van Eck, M. 1986. NRIND Food Quantities Manual. Parow:SAMRC.

LARKIN, F.A., Metzner, H.L., Thompson, F.E., Flegal, K.M. & Guire, K.E. 1989. Comparison of estimated nutrient intakes by food frequency and dietary records in adults. *Journal of the American Dietetic Association*, 89(2):215-223.

LEEDS, A.R. & Judd, P.A. 1986. Dietary fiber and weight management (In Vahouny, G.V. & Kritchevsky, D. Eds. Dietary fiber. New York: Plenum Publishing Corp.)

LEFLOCH, J.-P., Escuyer, P., Baudin, E., Baudin, D. & Perlemuter, L. 1990. Blood glucose area under the curve. Methodological aspects. *Diabetes Care*, 13(2):172-175.

LEGG, M.A. & Harawi, S.J. 1985. The pathology of diabetes mellitus. (In Marble, A., Krall, L.P., Bradley, R.F., Christlieb, A.R. & Soeldner, J.S. Eds. Joslin's diabetes mellitus. 12th Ed. Philadelphia: Lea & Febiger. p. 298-331).

LETCHER, R.L., Chien, S., Pickering, T.G., Sealey, J.E. & Laragh, J.H. 1981. Direct relationship between blood pressure and blood viscosity in normal and hypertensive subjects. Role of fibrinogen and concentration. *The American Journal of Medicine*, 70(6):1195-1202.

LEV-RAN, A. & Hill, L.R. 1987. Different body-fat distributions in IDDM and NIDDM. *Diabetes Care*, 10(4):491-494.

LOUW, D.A. 1982. Inleiding tot die psigologie. Johannesburg: McGraw Hill.

LUBBE, A.M. 1971. Dietary evaluation. *South African Medical Journal*, 45(45):1289-1297

LUFKIN, E.G., Fass, D.N., O'fallon, W.M. & Bowrie, E.J.W. 1979. Increased von Willebrand factor in diabetes mellitus. *Metabolism*, 28(1):63-69.

LUNDGREN, H., Bengtsson, C., Blohme, G., Lapidus, L. & Sjöström, L. 1989. Adiposity and adipose tissue distribution in relation to incidence of diabetes in women: results from a prospective population study in Gothenberg, Sweden. *Internation Journal of Obesity*, 13:413-423.

MADSBAD, S. 1990. Classification of diabetes in older adults. *Diabetes Care*, 12(suppl 2):93-96.

MAHLEY, R.W. 1981. Cellular and molecular biology of lipoprotein metabolism in atherosclerosis. *Diabetes*, 30(suppl 2):60-65.

MANN, J.I. 1984. Lines to legumes: changing concepts of diabetic diets. *Diabetic Medicine*, 1(1):191-198.

MANN, J.I. 1989. Diabetic dietary prescriptions. The cost of oral hypoglycaemic agents could be cut by sensible dietary advice. *British Medical Journal*, 298(6687):1535-1536.

MARBLE, A. 1976. Late complications of diabetes. A continuing challence. *Diabetologia*, 12:193.

MARBLE, A. 1985. Insulin in the treatment of diabetes. (In Marble, A., Krall, L.P., Bradley, R.F., Christlieb, A.R. & Soeldner, J.S. Eds. Joslin's diabetes mellitus. 12th Ed. Philadelphia: Lea & Febiger. p. 380.405).

MARBLE, A. & Ferguson, B.D. 1985. Diagnosis and classification of diabetes mellitus and nondiabetic meliturias. (In Marble, A., Krall, L.P., Bradley, R.F., Christlieb, A.R. & Soeldner, J.S. Eds. Joslin's diabetes mellitus. 12th Ed. Philadelphia: Lea & Febiger. p. 332-352).

MARINE, N., Vinik, A.I., Edelstein, I. & Jackson, W.P.U. 1969. Diabetes, hyperglycemia and glucosuria among Indians, Malays and Africans (Bantu) in Cape Town, South Africa. *Diabetes*, 18(12):840-857.

McCANCE, D.R., Coulter, D., Smye, M. & Kennedy, L. 1987. Effect of fluctuations in albumin on serum fructosamine assay. *Diabetic Medicine*, 4:434-436.

McCARTY, M.F. & Rubin, E.J. 1984. Rationales for micronutrient supplementation in diabetes. *Medical Hypotheses*, 13:139-151.

MEADE, T.W., Brozovic, M., Chakarabarti, R.R., Haines, A.P., Imeson, J.D., Mellows, S., Miller, G.J., North, W.R.S., Stirling, Y. & Thompson, S.G. 1986. Haemostatic function and ischaemic heart disease: principal results of the Northwick Park Heart Study. *Lancet*, Sept., 2(8506):533-537.

MEDLIN, C. & Skinner, J.D. 1988. Individual dietary intake methodology: A 50-year review of progress. *Journal of the American Dietetic Association,* 88(10):1250-1257.

MELANDER, A, Lebovitz, H.E. & Faber, O.K. 1990. Sulfonylureas. Why, which and how? *Diabetes Care*, 13(3, suppl):18-25.

METZ, R. & Friedenberg, R. 1970. Effects of repetitive glucose loads on plasma concentrations of glucose, insulin and free fatty acids: paradoxical insulin responses in subjects with mild glucose intolerance. *Journal of Clinical Endocrinology*, 30:602-608.

MEYER, T.W., Anderson, S. & Brenner, B.M. 1983. Dietary protein intake and progressive glomerular sclerosis: role of capillary hypertension and hyperfusion in the progression of renal disease. *Annals of Internal Medicine*, 98(5 part 2):832-838.

MICOZZI, M.S., Albanes, D., Jones, D.Y. & Chumlea, W.C. 1986. Correlations of body mass indices with weight, stature, and body composition in men and women in NHANES I and II. *American Journal of Clinical Nutrition*, 44(6):725-731.

MILLER, G.J. 1980. High density lipoproteins and atherosclerosis. *Annual Review of Medicine,* 31:97-108.

MILLER, G.J., Martin, J.C., Webster, J., Wilkes, H., Miller, N.E., Wilkinson, W.H. & Meade, T.W. 1986. Association between dietary fat intake and plasma Factor VII coagulant activity - a predictor of cardiovascular mortality. *Atherosclerosis*, 60: 269 -277.

MOGENSON, C.E., Osterby, R. & Gundersen, H.J.G. 1979. Early functional and morphologic vascular renal consequences of the diabetic state. *Diabetologia*, 17:71-75.

MOLLENTZE, W.F., Panzegrouw, D.F. & Steyn, A.F. 1990. Diabetes mellitus, pulmonary tuberculosis and chronic calcific pancreatitis revisited. *South African Medical Journal*, 78(4):235-236.

MOLLER-JENSEN, O., Wahrendorf, J., Rosenqvist, A. & Geser, A. 1984. Ther reliability of questionnaire-derived historical dietary information and temporal stability of food habits in individuals. *American Journal of Epidemiology*, 120(2):281-289.

MÖNNIG, H.O. 1978. The Pedi. Pretoria : J.L. van Schaik Limited. 235 p.

MOORADIAN, A.D. & Morley, J.E. 1987. Micronutrient status in diabetes mellitus. *American Journal of Clinical Nutrition*, 45(5):877-895.

MORGAN, K.J., Johnson, S.R., Rizek, R.L., Reese, R. & Stampley, G.L. 1987. Collection of food intake data. An evaluation of methods. *Journal of the American Dietetic Association*, 87(7):888-898.

MORLEY, J.E., Lowenthal, M.N., Asvat, M.S., Kopelowitz, W., Klein, C. & Kokoris, N. 1977. Problems experienced in a diabetic clinic for blacks. *South African Medical Journal*, 52(6):215-218.

MOSS, D.W., Henderson, A.R. & Kachmar, J.F. 1986. Enzymes. (In Tietz, N.W. Ed. Textbook of clinical chemistry. London: W.B. Saunders Co. pp.619-762).

MOUROT, J., Thouvenot, P., Couet, C., Antoine, J.M., Krobicka, A. & Debry, G. 1988. Relationship between the rate of gastric emptying and glucose and insulin responses to starchy foods in young healthy adults. *American Journal of Clinical Nutrition*, 48(4):1035-1040.

MOUTON, D.P. & Gill, A.J. 1988. Prevalence of diabetic retinopathy and evaluation of risk factors. *South African Medical Journal*, 74(8):399-402.

MULLEN, B.J., Krantzler, N.J., Grivetti, L.E., Schutz, H.G. & Meiselman, H.L. 1984. Validity of a food frequency questionnaire for the determination of individual food intake. *American Journal of Clinical Nutrition*, 39(1):136-143.

MUSGRAVE, K.O., Giambalvo, L., Leclerc, H.L., Cook, R.A. & Rosen, C.J. 1989. Validation of a quantitative food frequency questionnaire for rapid assessment of dietary calcium intake. *Journal of the American Dietetic Association*, 89(10):1484-1488.

NATIONAL DIABETES DATA GROUP. 1979. Classification and diagnosis of diabetes mellitus and other categories of glucose intolerance. *Diabetes*, 28(10):1039-1057.

NATIONAL INSTITUTES OF HEALTH. 1987. Consensus development conference on diet and exercise in non-insulin-dependent diabetes mellitus. *Diabetes Care*, 10(5):639-643.

NIELSEN, H. & Nielsen, G.C. 1989. Preprandial blood glucose values: influence on glycemic response studies. *American Journal of Clinical Nutrition*, 49(6):1243-1246.

NIEWOEHNER, C.B., Allen, J.I., Boosalis, M., Levine, A.S. & Morley, J.E. 1986. The role of zinc supplementation in type II diabetes mellitus. *American Journal of Medicine*, 81(1):63-68.

NORUM, K.R. & Drevon, C.A. 1986. Dietary n-3 fatty acids and cardiovascular diseases. *Arteriosclerosis*, 6:352-355.

NOSADINI, R., Avogaro, A., Trevisan, R., Valerio, A., Tessari, P., Dumer, E., Tiengo, A., Velussi, M., Del Prato, S., De Kreutzenberg, S., Muggeo, M. & Crepaldi, G. 1987. Effect of metformin on insulin-stimulated glucose turnover and insulin binding to receptors in Type II diabetes. *Diabetes Care*, 10(1):62-67.

NUTTALL, F.Q. 1979. Dietary recommendations for individuals with diabetes mellitus: Summary of report from the Food and Nutrition Committee of the American Diabetes Association. *American Journal of Clinical Nutrition*, 33(6):1311-1312.

NUTTALL, F.Q. 1988. The high-carbohydrate diet in diabetes management. *Advances in Internal Medicine*, 33:165-184.

O'CONNOR, P.J., Fragneto, R., Coulehan, J. & Crabtree, B.F. Metabolic control in non-insulin-dependent diabetes mellitus. Factors associated with patient outcomes. *Diabetes Care*, 10(6):697-701.

O'DEA, K. 1984. Marked improvement in carbohydrate and lipid metabolism in diabetic Australian Aborigines after temporary reversion to traditional lifestyle. *Diabetes*, 33(6):596-603.

O'DEA, K., Nestel, P.J. & Antoff, L. 1980. Physical factors influencing postprandial glucose and insulin responses to starch. *American Journal of Clinical Nutrition*, 33(5):760-765.

O'DEA, K., Traianedes, T., Hopper, J.L., Larkins, R.G. 1988. Impaired glucose tolerance, hyperinsulinemia and hypertriglyceridemia in Australian Aborigines from the desert. *Diabetes Care*, 11(1):23-29.

O'DEA, K., Traianedes, K., Ireland, P., Naill, M., Sadler, J., Hopper, J. & De Luise, M. 1989. The effects of diet differing in fat, carbohydrate, and fiber on carbohydrate and lipid metabolism in type II diabetes. *Journal of the American Dietetic Association*, 89(8):1076-1086.

OHLSON, L.-O., Larsson, B., Björntorp, P., Eriksson, H., Svärdsudd, K., Welin, L., Tibblin, G. & Wilhelmsen, L. 1988. Risk factors for type 2 (NID) diabetes mellitus. Thirteen and one-half years of follow-up of the participants in a study of Swedish men born in 1913. *Diabetologia*, 31(11):798-805.

OLEFSKY, J.M. & Crapo, P.A. 1980. Fructose, xylitol, sorbitol. *Diabetes Care*, 3(5):390-393.

O'RAHILLY, S.O., Wainscoat, J.S. & Turner, R.C. 1988. Type 2 (non-insulin-dependent) diabetes mellitus: New genetics for old nightmares. *Diabetologia*, 31(7):407-414.

OSTERMANN, H. & Van de Loo, J. 1986. Factors of the hemostatic system in diabetic patients. *Haemostasis*, 16:386-418.

OSTLUND, R.E., Stalen, M., Kohrt, W.M., Schultz, J. & Malley, M. 1990. The ratio of waist-to-hip circumference, plasma insulin level and glucose intolerance as independent predictors of the HDL-cholesterol level in older adults. *New England Journal of Medicine*, 322(4):229-234.

OUDKERK, A.C.F. 1965. Eating habits of the urban Bantu, with special reference to the school-going child. *South African Journal of Nutrition*, supplement to *South African Medical Journal*, 39(45):1148-1150.

PANZRAM, G. 1987. Mortality and survival in Type 2 (non-insulin-dependent) diabetes mellitus. *Diabetologia*, 30(3):123-131.

PAOLISSO, G., Sheen, A., Onofrio, F.D. & Lefèbre, P. 1990. Magnesium and glucose homeostasis. *Diabetologia*, 33(6):511-514.

PARILLO, M., Giacco, R., Riccardi, G., Pacioni, D. & Rivellese, A. 1985. Different glycaemic responses to pasta, bread, and potatoes in diabetic patients. *Diabetic Medicine*, 2(2):374-377.

PEEPLES, L.H., Carpenter, J.W., Israel, R.G. & Barakat, H.A. 1989. Alterations in low-density lipoproteins in subjects with abdominal adiposity. *Metabolism*, 38(10):1029-1036.

POTTER, J.G., Coffman, K.P., Reid, R.L., Krall, J.M. & Albrink, M.J. 1981. Effect of test meals of varying dietary fiber content on plasma insulin and glucose response. *American Journal of Clinical Nutrition*, 34(2):328-334.

QUIN, P.J. 1964. Foods and feeding habits of the Pedi. *South African Medical Journal*, 38(42):969-971.

RABINOWITZ, M.B., Gonick, H.C., Levin, S.R. & Davidson, M.B. 1983. Effects of chromium and yeast supplements on carbohydrate and lipid metabolism in diabetic men. *Diabetes Care*, 6(4):319-327.

RAY, T.K., Mansell, K.M., Knight, L.C., Malmud, L.S., Owen O.E. & Boden, G. 1983. Long-term effects of dietary fiber on glucose tolerance and gastric emptying in noninsulin-dependent diabetic patients. *American Journal of Clinical Nutrition*, 37(3):376-381.

REAVEN, G.M. 1987. Abnormal lipoprotein metabolism in non-insulin-dependent diabetes mellitus. *The American Journal of Medicine*, 83(suppl 3A):31-40.

REAVEN, G.M., Lardinois, C.K., Greenfield, M.S., Schwartz, H.C. & Vreman, H.J. 1990. Effect of acarbose on carbohydrate and lipid metabolism in NIDDM patients poorly controlled by sulfonylureas. *Diabetes Care*, 13(3, suppl):32-36.

RESHEF, A. & Epstein, L.M. 1972. Reliability of a dietary questionnaire. *American Journal of Clinical Nutrition*, 25(1):91-95.

RICCARDI, G., Rivellese, A., Pacioni, D., Genovese, S., Mastranzo, P & Mancini, M. 1984. Separate influence of dietary carbohydrate and fibre on the metabolic control in diabetes. *Diabetologia*, 26(2):116-121.

RIVELLESE, A.A., Giacco, R., Genovese, S., Patti, L., Marotta, G., Pacioni, D., Annuzzi, G. & Riccardi, G. 1990. Effects of changing amounts of carbohydrate in diet on plasma lipoproteins in type II diabetes patients. *Diabetes Care*, 13(4):446-448.

RIVELLESE, A., Riccardi, G., Giacco, A., Pacioni, D., Genovese, S., Maltioli, P.L. & Manoni, M. 1980. Effect of dietary fibre on glucose control and serum lipoproteins in diabetic patients. *Lancet*, 30 Aug:447-450.

ROSENDORFF, C. 1989. Blood pressure and blood glucose - is there a link? *South African Medical Journal*, 76(12):640-642.

ROSS, S.W., Brand, J.C., Thorburn, A.W. & Truswell, A.S. 1987. Glycemic index of processed wheat products. *American Journal of Clinical Nutrition*, 46(4):631-635.

RUSSEL, D.S., Gherzi, R., Johnson, E.J., Chou, E.K. & Rosen, O.M. 1987. The protein-tyrosine kinase activity of the insulin receptor is necessary for insulin mediated receptor down-regulation. *Journal of Biological Chemistry*, 262(24):11833-11840.

SAMANTA, A., Burden, A.C. & Jones G.R. 1985. Plasma glucose responses to glucose, sucrose, and honey in patients with diabetes mellitus: an analysis of glycaemic and peak incremental indices. *Diabetic Medicine*, 2(2):371-373.

SAMET, J.M., Humble, C.G. & Skipper, B.E. 1984. Alternatives in the collection and analysis of food frequency interview data. *American Journal of Epidemiology*, 120(4):572-581.

SARLUND, H., Laakso, M., Pyörälä, K. & Penttila, I. 1987. Association of serum lipids and lipoproteins with plasma C-peptide concentration in non-insulin-dependent diabetic and non-diabetic subjects. *Metabolism*, 36(9):840-845.

SAS® User's Guide. 1985. Basics, version 5 edition; statistics, version 5 edition. Cary, North Carolina: SAS Institute Inc.

SCHARF, O. 1990. Dietary Manager. 6th Ed. Program Management: Johannesburg.

SCHARF, O. 1989. Dietary Manager. 2 nd Ed. Program Management. Johannesburg.

SCHMID-SCHÖNBEIN, H. & Volger, E. 1976. Red-cell aggregation and red-cell deformability in diabetes. *Diabetes*, 25(10, suppl 1):897-902.

SEEDAT, M.A. 1989a. Non-insulin-dependent diabetes mellitus in Southern Africa. *Hospital Medicine*, May/June:18.

SEEDAT, Y.K. 1989b. Nutritional aspects of hypertension. *South African Medical Journal*, 75(4):175-177.

SEEDAT, Y.K., Seedat, M.A. & Reddy, K. 1978. The prevalence of hypertension in the Indian population of Durban. *South African Medical Journal*, 54(1):10-15.

SEFTEL, H.C. 1977. Diseases in urban and rural populations. *South African Medical Journal*, 51(3):121-123.

SEFTEL, H.C., Keely, K.J. & Walker, A.R.P. 1963. Studies in glucosuria and diabetes in non-white populations of the Transvaal. Part I: Africans. *South African Medical Journal*, 37(48):1213-1216.

SGAMMATO, J. 1987. Fibrinogen: A major new risk factor for CHD. *Patient Care*, 21(1):19-20.

SHAHEEN, S.M. & Fleming, S.E. 1987. High-fiber foods at breakfast: Influence on plasma glucose and insulin responses to lunch. *American Journal of Clinical Nutrition*, 46(5):804-811.

SHEKELLE, R.B., Shyrock, A.M., Paul, O., Lepper, M., Stamler, J., Liu, S & Raynor, W.J. 1981. Diet, serum cholesterol and death from coronary heart disease. The Western Electric Study. *New England Journal of Medicine*, 304(2):65-70.

SHEPERD, J. 1990. A focus on Acipimox. *Clinician*, 8(1):35-41.

SILVERMAN, L.M., Christenson, R.H. & Grant, G.H. 1986. Amino acids and proteins. (In Tietz, N.W. Ed. Textbook of clinical chemistry. London: W.B. Saunders Co. pp.519-618).

SILVIS, N. 1988. Fish oil, prostaglandin metabolism and cardiovascular disease. *Journal of Dietetics and Home Economics*, 16(3):95-100.

SILVIS, N. 1989. The influence of two dietary supplements, a dietary fibre concentrate and eicosapentaenoic acid on metabolic control, serum lipids and haemostasis of black type 2 diabetes mellitus subjects. Potchefstroom. 279 p. (Thesis (D. Sc Dietetics) - PU for CHE.)

SILVIS, N., Vorster, H.H., Mollentze, W.F., De Jager, J. & Huisman, H.W. 1990. Metabolic and haemostatic consequences of dietary fibre and n-3 fatty acids in black type 2 (NIDDM) diabetic subjects: a placebo controlled study. *International Clinical Nutrition Review*, 10(3):362-380.

SIMOPOULOS, A. 1985. Dietary control of hypertension and obesity and body weight standards. *Journal of the American Dietetic Association*, 85(4):419-421.

SIMPSON, H.C.R., Mann, J.I., Chakrabarti, R., Imeson, J.D., Stirling, Y., Tozer, M., Woolf, L. & Meade, T.W. 1982. Effect of high-fibre diet on haemostatic variables in diabetes. *British Medical Journal*, 284:1608.

SIMPSON, H.C.R., Simpson, R.W., Lousley, S., Carter, R.D., Geekie, M., Hockaday, T.D.R. & Mann, J.I. 1981. A high-carbohydrate leguminous fibre diet improves all aspects of diabetic control. *Lancet*, 1(8210):1-5.

SLAMA, G., Haardt, M.J., Jean-Joseph, P., Costagliola, D., Goicolea, I., Bornet, F., Elgrably, F. & Tchobroutsky, G. 1984. Sucrose taken during mixed meal has no additional hyperglycaemic action over isocaloric amounts of starch in well-controlled diabetics. *Lancet*, 2(8395):122-124.

SMART, L.M., Howie, A.F., Young, R.J., Walker, S.W., Clarke, B.F. & Smith, A.F. 1988. Comparison of fructosamine with glycosylated hemoglobin and plasma proteins as measures of glycemic control. *Diabetes Care*, 11(5):433-436.

SMIT, G.J. 1983. Psigometrika. 2de Ed. Pretoria: HAUM.

SMITH, E.B. 1986. Fibrinogen, fibrin and fibrin degradation products in relation to atherosclerosis. *Clinics in Haematology*, 15(2):355-370.

SNEDECOR, G.W. & Cochran, W.G. 1976. Statistical methods. 6th Ed. Ames. Iowa State University Press, 273-275.

STANLEY, J.C. & Hopkins, K. 1972. Educational and psychological measurement and evaluation. 4th Ed. Englewood Cliffs: Prentice-Hall.

STEIN, E.A. 1986. Lipids, lipoproteins, and apolipoproteins. (In Tietz, N.W. Ed. Textbook of clinical chemistry. London: W.B. Saunders Co. pp.829-900).

STEINER, G. 1981. Diabetes and atherosclerosis. *Diabetes*, 30(suppl 2):1-7.

STONE, M.C. & Thorpe, J.M. 1985. Plasma fibrinogen - a major coronary risk factor. *Journal of the Royal College of General Practitioners*, 35:565-569.

STORY, L., Anderson, J.W., Chem, W.J.L., Karounos, D. & Jefferson, B. 1985. Adherence to high-carbohydrate, high-fibre diets: long-term studies of non-obese diabetic men. *Journal of the American Dietetic Association*, 85(9):1105-1110.

STREJA, D.A., Boyko, E. & Rabkin, S.W. 1980. Changes in plasma high-density lipoprotein cholesterol concentration after weight reduction in grossly obese subjects. *British Medical Journal*, 281(6243):770-772.

STUFF, J.E., Garza, C., Smith, E. O'B., Nichols, B.L. & Montandon, C.M. 1983. A comparison of dietary methods in nutritional studies. *American Journal of Clinical Nutrition*, 37(2):300-306.

STUNKARD, A.J. & Waxman, M. 1981. Accuracy of self-reports of food intake. *Journal of the American Dietetic Association*, 79(5):547-551.

TALMAGE, H. & Rasher, S.P. 1981. Validity and reliability issues in measurement instrumentation. *Journal of Nutrition Education*, 3(3):83-90.

TALMAGE, H. & Rasher, S.P. 1982. Design issues in developing and selecting measurment instruments. *Journal of Nutrition Education*, 14(2):54-59.

THOMPSON, L.U. 1988. Antinutrients and blood glucose. *Food Technology*, April:123-132.

THOMPSON, D.G., Wingate, D.L., Thomas, M. & Harrison, D. 1982. Gastric emptying as a determinant of the oral glucose tolerance test. *Gastroentorology*, 82(1):51-55.

THORBURN, A.W., Brand, J.C. & Truswell, A.S. 1986a. The glycaemic index of foods. *The Medical Journal of Australia*, 144:580-582.

THORBURN, A.W., Brand, J.C. & Truswell, A.S. 1986b. Salt and the glycaemic response. *British Medical Journal*, 292(6537):1697-1699.

THORBURN, A.W., Brand, J.C. & Truswell, A.S. 1987. Slowly digested and absorbed carbohydrate in traditional bushfoods: a protective factor against diabetes? *American Journal of Clinical Nutrition*, 45(1):98-106.

TIETZ, N.W., Pruden, E.L. & Siggaard-Andersen, O. 1986. Electrolytes, blood gases, and acid-base balance. Section One. Electrolytes. (In Tietz, N.W. Ed. Textbook of clinical chemistry. London: W.B. Saunders Co. pp.1172-1190).

TIMPERLY, W.R., Williams, E., Ward, J.D. & Preston F.E. 1977. Abnormalities of small blood vessels in diabetic neuropathy. *Diabetologia*, 13:437-439.

TODD, K.S., Hudes, M. & Calloway, D.H. 1983. Food intake measurement: problems and approaches. *American Journal of Clinical Nutrition*, 37(1):139-146.

TOPPING, D.L. 1977. Metabolic effects of carbon monoxide in relation to atherogenesis. *Atherosclerosis*, 26(1):129.

TUCK, M. 1988. Treatment of hypertensive diabetic patients. *Diabetes Care*, 11(10):828-831.

TUOMILEHTO, J. & Wolf, E. 1987. Primary prevention of diabetes mellitus. *Diabetes Care*, 10(2):238-248.

TURNER, R.C. & Holman, R.R. 1990. Insulin use in NIDDM. Rationale based on pathophysiology of the disease. *Diabetes Care*, 13(9):1011-1020.

ULUTIN, O.N. 1986. Atherosclerosis and hemostasis. *Seminars in Thrombosis and Hemostasis*, 12(2):156-162.

URBERG, M., Parent, M., Hill, D & Zemel, M. 1986. Evidence for synergism between chromium and nicotinic acid in normalizing glucose tolerance. *Diabetes*, 35(suppl 1):147A(abs).

UUSITUPA, M.I.J., Laakso, M., Sarlund, H., Majander, H., Takala, J. & Penttilä, I. 1990. Effects of a very-low-calorie diet on metabolic control and cardiovascular risk factors in the treatment of obese non-insulin-dependent diabetics. *American Journal of Clinical Nutrition*, 51(5):768-773.

VAN GAAL, L.F., Rillaerts, E., Creten, W. & De Leeuw, I.H. 1988. Relationship of body fat distribution pattern to atherogenic risk factors in NIDDM. *Diabetes Care*, 11(2):103-106.

VAN GAAL, L.F., VanSant, G.A. & De Leeuw, I.H. 1989. Upper body adiposity and the risk for atherosclerosis. *Journal of the American College of Nutrition*, 8(6):504-514.

VATASSERY, G.T., Morley, J.E., Kuskowski, M.A. 1983. Vitamin E in plasma and platelets of human diabetic patients and control subjects. *American Journal of Clinical Nutrition*, 37(4):641-644.

VENTER, C.S., Vorster, H.H., Van Rooyen, A., Kruger-Locke, M.M. & Silvis, N. 1990. Comparison of the effects of maize porridge consumed at different temperatures on blood glucose, insulin and acetate levels in healthy volunteers. *The South African Journal of Food Science and Nutrition*, 2(1):2-5.

VERMAAK, W.J.H., Steyn, K., Rossouw, J.E., Barnard, H.C., Esterhuysen, A.J., Potgieter, G.M. & Van Jaarsveld, H. 1988. Towards standardization of pre- and post-analytical variables in the assessment of lipid risk factors for ischaemic heart disease. *South African Medical Journal*, 74(1):485-490.

VIGNATI, L. & Cunningham, L.N. 1985. Exercise and diabetes. (In Marble, A., Krall, L.P., Bradley, R.F., Christlieb, A.R. & Soeldner, J.S. Eds. Joslin's diabetes mellitus. 12th Ed. Philadelphia: Lea & Febiger. p. 453-464).

VIGNERI, R. & Goldfine, I.D. 1987. Role of metformin in treatment of diabetes mellitus. *Diabetes Care*, 10(1):118-122.

VINIK, A.I. & Jenkins, D.J.A. 1988. Dietary fiber in management of diabetes. *Diabetes Care*, 11(2):160-172.

VORSTER, H.H. 1986. Die moderne dieetbehandeling van diabetes mellitus. *Journal of Dietetics and Home Economics*, 14(2):59-64.

VORSTER, H.H. 1987. Sommige fisiologiese effekte van dieetvesel met besondere vewysing na die plasmastollingsfaktore. Potchefstroom. 345 p. (Thesis (D. Sc - PU vir CHO)).

VORSTER, H.H, Silvis, N., Venter, C.S., Van Ryssen, J.J., Huisman, H., Van Eeden, T.S. & Walker, A.R.P. 1987a. Serum cholesterol, lipoproteins, and plasma coagulation factors in South African blacks on a high-egg but low-fat intake. *American Journal of Clinical Nutrition*, 45(1):52-57.

VORSTER, H.H., Van Tonder, E., Kotzé, J.P. & Walker, A.R.P. 1987b. Effects of graded sucrose additions on taste preference, acceptablility, glycemic index and insulin response to butter beans. *American Journal of Clinical Nutrition*, 45(3):575-579.

VORSTER, H.H., Venter, C.S. & Silvis, N. 1990. The glycaemic index of foods: a critical evaluation. *The South African Journal of Food Science and Nutrition*, 2(1):13-17.

VORSTER, H.H., Venter, C.S., Menssink, E., Van Staden, D.A., Labadarios, D., Strydom, A.J.C., Silvis, N., Gericke, G.J. & Walker, A.R.P. 1990. Nutrient intakes and vitamin status of adult Vendas in an isolated rural village. *Journal of American Dietetic Association*, In Press.

VORSTER, H.H., Venter, C.S., Silvis, N., Van Eeden, T.S., Huisman, H.W., Walker, A.R.P. 1988a. Dietary influences on haemostasis may affect risk for coronary heart disease. *South African Journal for Science*, 84(5):289-293.

VORSTER, H.H., Venter, C.S., Van Eeden, T.S., Silvis, N., Huisman, H.W., Van Ryssen, J.C.J. & Walker, A.R.P. 1988b. Benefits from supplementation of the current recommended diabetic diet with gel fibre. *International Clinical Nutrition Review*, 8(3):140-146.

WALKER, A.R.P., Bhamjee, D., Walker, B.F. & Martin, A.P. 1979. Serum high-density lipoprotein cholesterol, glucose tolerance and other variables in obese black adolescent girls. *South African Medical Journal*, 56(6):221-224.

WALKER, A.R.P. & Walker, B.F. 1984 Glycaemic index of South African foods determined in rural blacks - a population at low risk of diabetes. *Human Nutrition: Clinical Nutrition*, 38C:215-222.

WALKER, A.R.P., Walker, B.F., Walker, A.J. & Vorster, H.H. 1989. Low frequency of adverse sequelae of obestiy in South African rural black women. *International Journal of Vitamin and Nutrition Research*, 59:224-228.

WARREN, S., LeCompte, P.M. & Legg, M.A. 1966. The pathology of diabetes mellitus. 4th Ed. Philadelphia: Lea & Febiger.

WEINBERG, R.B., Dantzker, C. & Patton, C.S. 1990. Sensitivity of serum apolipoprotein A-IV levels to changes in dietary fat content. *Gastroenterology*, 98(1):17-24.

WEISS, E.H., Kien, C.L. & Clark, G. 1988. Validation of a photographic method for recording the selection of foods by individuals. *Journal of the American Dietetic Association*, 88(5):599-600.

WEYMAN-DAUM, M., Fort, P., Recker, B., Lanes, R. & Lifshitz, F. 1987. Glycemic response in children with insulin-dependent diabetes mellitus after high- or low-glycemic-index breakfast. *American Journal of Clinical Nutrition*, 46(5):798-803.

WHEELER, M.L., Delahanty, L. & Wylie-Rosett, J. 1987. Diet and exercise in noninsulin-dependent diabetes mellitus: Implications for dietitians from the NIH Consensus Development Conference. *Journal of the American Dietetic Association*, 87(4):480-485.

WIGAND, J.P., Anderson, J.H., Jennings, S.S. & Blackard, W.G. 1979. Effect of dietary composition on insulin receptors in normal subjects. *American Journal of Clinical Nutrition*, 32(1):6-9.

WILHELMSEN, L., Svärdsudd, K., Korsan-Bengsten, K., Larsson, B., Welin, L. & Tibblin, G. 1984. Fibrinogen as a risk factor for stroke and myocardial infarction. *New England Journal of Medicine*, 311(8):501-505.

WILLET, W.C., Reynolds, R.D., Cottrell-Hoehner, S., Sampson, L. & Browne, M.L. 1987. Validation of a semi-quantitative food frequency questionnaire: comparison with a 1-year diet record. *Journal of the American Dietetic Association*, 87(1):43-47.

WILLET, W.C., Sampson, L., Stampfer, M.J., Rosner, B., Bain, C., Witschi, J., Hennekens, C.H. & Speizer, F.E. 1985. Reproducability and validity of a semi-quanititative food frequency questionnaire. *American Journal of Epidemiology*, 122(1):51-65.

WILLIAMS, P.T., Krauss, R.M., Vranizan, K.M. & Wood, P.D.S. 1990. Changes in lipoprotein subfractions during diet-induced and exercise-induced weight loss in moderately overweight men. *Circulation*, 81(4):1293-1304.

WING, R.R., Epstein, L.H., Paternostro-Bayles, M., Kriska, A., Nowalk, M.P. & Gooding, W. 1988. Exercise in a behavioural weight control programme for obese patients with type 2 (non-insulin-dependent) diabetes. *Diabetologia*, 31(12):902-909.

WINGARD, D.L., Barrett-Connor, E., Criqui, H. & Suarez, L. 1983. Clustering of heart disease risk factors in diabetic compared to non-diabetic adults. *American Journal of Epidemiology*, 117(1):19-26.

WOLEVER, T.M.S. 1990. The glycaemic index (In Bourne, G.H. (Ed.) Aspects of some vitamins, minerals and enzymes in health and disease. World Review of Nutrition and Dietetics, vol. 62. Basil, Karger. pp. 120-185).

WOLEVER, T.M.S., Csima, A., Jenkins, D.J.A., Wong, G.S. & Josse, R.G. 1989. The glycemic index: Variation between subjects and predictive difference. *Journal of the American College of Nutition*, 8(3):235-247.

WOLEVER, T.M.S., Jenkins, D.J.A., Josse, R.G., Wong, G.S. & Lee, R. 1987b. The glycemic index: Similarity of values derived in insulin-dependent and non-insulin-dependent diabetic patients. *Journal of the American College of Nutrition*, 6(4):295-305.

WOLEVER, T.M.S., Jenkins, D.J.A., Ocana, A.M., Rao, V.A. & Collier, G.R. 1988. Second meal effect of low-glycemic-index foods eaten at dinner improve subsequent breakfast glycemic response. *American Journal of Clinical Nutrition*, 48(4):1041-1047.

WOLEVER, T.M.S., Jenkins, D.J.A., Thompson, L.U., Wong, G.S. & Josse, R.G. 1987a. Effect of canning on the blood glucose response to beans in patients with type 2 diabetes. *Human Nutrition: Clinical Nutrition*, 41C(1):135-140.

WOLEVER, T.M.S, Jenkins, D.J.A, Vuksan, V., Josse, R.G., Wong, G.S. & Jenkins, A.L. 1990. Glycemic index of foods in individual subjects. *Diabetes Care*, 13(2):126-132.

WOLEVER, T.M.S., Nuttall, F.Q., Lee, R., Wong, G.S., Josse, R.G., Csima, A. & Jenkins, D.J.A. 1985. Prediction of the relative blood glucose response of mixed meals using the white bread glycemic index. *Diabetes Care*, 8(5):418-428.

WOODS, H.F. & Bax, N.D.S. 1982. Sweetness in the diabetic diet. *Diabetologia*, 23(3):213-215.

WORLD HEALTH ORGANISATION STUDY GROUP. 1985. Diabetes mellitus. World Health Organisation technical report. Series no. 727. Geneva: World Health Organisation.

WYLIE-ROSETT, J. 1988. Evaluation of protein in dietary management of diabetes mellitus. *Diabetes Care*, 11(2):143-147.

WU, M.S., Johnston, P., Sheu, W. H.-H., Hollenbeck, C.B., Jeng, C.-Y., Goldfine, I.D., Chen, Y.D.I. & Reaven, G.M. 1990. Effect of metformin on carbohydrate and lipoprotein metabolism in NIDDM patients. *Diabetes Care*, 13(1):1-8.

WU, M.L., Whittemore, A.S. & Jung, D.L. 1986. Errors in reported dietary intakes. *American Journal of Epidemiology*, 124(5):826-835

ZILLIKENS, M.C. & Conway, J.M. 1990. Anthropometry in blacks: applicability of generalized skinfold equations and differences in fat patterning between blacks and whites. *American Journal of Clinical Nutrition*, 52(1):45-51.

ZIMMET, P. 1982. Type 2 (non-insulin-dependent) diabetes - an epidemiological overview. *Diabetologia*, 22(6):399-411.