# NUTRITIONAL STATUS, GLYCEMIC CONTROL AND BARRIERS TO TREATMENT COMPLIANCE AMONG PATIENTS WITH TYPE 2 DIABETES ATTENDING DIABETES CLINICS IN MASERU, LESOTHO

**Mohlakotsana Mokhehle**

**Dissertation submitted in accordance**

**with the academic requirements for the degree**

**M.Sc. Dietetics**

**in the**

**Faculty of Health Sciences**

**Department of Nutrition and Dietetics**

**University of the Free State**

**Bloemfontein**

**South Africa**

**July 2014**

**Supervisor: Dr VL van den Berg**

**Co-Supervisor: L Janse van Rensburg**

## DECLARATION

I declare that the dissertation hereby submitted by me for M.Sc Dietetics at the University of the Free State is my own independent work and has not previously been submitted by me to another university/faculty. I further cede copyright of this research report in favour of the University of the Free State.

_____

Mohlakotsana Mokhehle

July 2014

## ACKNOWLEDGEMENTS

I would like to thank Dr van den Berg  my supervisor and Liska Janse van Rensburg my co-supervisor for their guidance and supervision throughout the years of my study.

I would like to pass my sincere thanks to Dr Jacques Raubenheimer, a staff member at the Department of Biostatistics for analysing the data.

I would also like to convey my special thanks to the following
Serialong, my daughter for patience and understanding during all the years of my study;
My family for their support and encouragement;
Lisemelo Seheri for her mutual support and assistance;
The staff of Domiciliary and Lesotho Defense Force clinics for their assistance throughout the collection of data;
The staff of Pathcare laboratory in Lesotho for handling and sending blood samples for HbA1c analysis;
Not forgetting the participants of the study.

## DEDICATION

I dedicate the work of this research to my daughter Serialong Mosa Mokhehle.

## TABLE OF CONTENTS

**CHAPTER 2: LITERATURE REVIEW**

**LIST OF TABLES** **PAGES**

**LIST OF FIGURES** **PAGES**

## LIST OF APPENDICES

## LIST OF ABBREVIATIONS

| | |
|---|---|
| **ACCORD** | Action to Control Cardiovascular Risk in Diabetes |
| **ACE** | American College of Endocrinology |
| **ACSM** | American College of Sports Medicine |
| **ADA** | American Diabetes Association |
| **ADA** | American Dietetic Association |
| **AHEAD** | Action for Health in Diabetes |
| **AIDS** | Acquired Immunodeficiency Syndrome |
| **ALA** | Alpha-Linolenic Acid |
| **AMDR** | Acceptable Macronutrient Distribution Range |
| **ART** | Anti-Retroviral Therapy |
| **BAI** | Body Adiposity Index |
| **BMI** | Body Mass Index |
| **CDC** | Centre for Disease Control and prevention |
| **CHASE** | The Child Heart And Health Study in England |
| **CIA** | Central Intelligence Agency |
| **CVD** | Cardiovascular Disease |
| **DARTS** | Diabetes Audit and Research in Tayside, Scotland |
| **DAWN** | Diabetes, Attitudes, Wishes and Needs |
| **DASH** | Dietary Approach to Stop Hypertension |
| **DCCT** | Diabetes Control and Complications Trial |
| **DECODE** | Diabetes Epidemiology Collaborative Analysis of Criteria in Europe |
| **DHMT** | District Health Management Team |
| **DHA** | Docosahexaenoic Acid |
| **DiOGenes** | The Diet Obesity and Genes trial |
| **DPP** | Diabetes Prevention Program |
| **DRIs** | Dietary Reference Intakes |
| **DSME** | Diabetes Self-Management Education |
| **eAG** | estimated Average Glucose |
| **EAL** | Evidence Analysis Library |
| **EASD** | European Association for the Study of Diabetes |
| **EDTA** | Ethylene diamine tetraacetic Acid |
| **EPA** | Eicosapentaenoic Acid |
| **FFQ** | Food Frequency Questionnaire |
| **FPG** | Fasting Plasma Glucose |
| **GDM** | Gestational Diabetes Mellitus |
| **GDP** | Gross Domestic Product |
| **GI** | Glycemic Index |
| **Hb** | Hemoglobin |
| **HbA1c** | Glycated Hemoglobin |
| **HDL** | High Density Lipoprotein |
| **HFCS** | High Fructose Corn Syrup |
| **HIV** | Human Immuno Virus |
| **IDF** | International Diabetes Federation |
| **IPAQ** | International Physical Activity Questionnaire |
| **IPAQ-L** | International Physical Activity Questionnaire – Long form |
| **KAP** | Knowledge, Attitudes and Perception |
| **LDF** | Lesotho Defense Force |
| **LDHS** | Lesotho Demographic Health Survey |

| | |
|---|---|
| **LDL** | Low-Density Lipoprotein |
| **LRFS** | Lesotho Risk Factor Survey |
| **LNNS** | Lesotho National Nutrition Survey |
| **LNNP** | Lesotho National Nutrition Policy |
| **MDGs** | Millennium Development Goals |
| **METs** | Metabolic equivalents |
| **MOH** | Ministry of Health |
| **MNT** | Medical Nutrition Therapy |
| **MUFAs** | Monounsaturated Fatty Acids |
| **NCDs** | Non-Communicable Diseases |
| **NGSP** | National Glycohemoglobin Standardization Program |
| **NHANES** | National Health and Nutrition Examination Survey |
| **OGTT** | Oral Glucose Tolerance Test |
| **IOM** | Institute of Medicine |
| **PPG** | Post-Prandial Glucose |
| **PPP** | Private Public Partnership |
| **PUFAs** | Polyunsaturated Fatty Acids |
| **QE II** | Queen Elizabeth II Hospital |
| **QMMH** | Queen Mamohato Memorial Hospital |
| **RCT** | Randomized Controlled Trial |
| **RD** | Registered Dietician |
| **RDAs** | Recommended Dietary Allowances |
| **RMR** | Resting Metabolic Rate |
| **SCFA** | Short-chain Fatty Acids |
| **SADHS** | South Africa Demographic and Health Survey |
| **SAFBDG** | South African Food Based Dietary Guidelines |
| **SEMDSA** | Society for Endocrinology, Metabolism and Diabetes of South Africa |
| **SMBG** | Self Monitoring Blood Glucose |
| **SSBs** | Sugar Sweetened Beverages |
| **T1DM** | Type 1 Diabetes Mellitus |
| **T2DM** | Type 2 Diabetes Mellitus |
| **TB** | Tuberculosis |
| **UFS** | University of Free State |
| **UK** | United Kingdom |
| **UKZN** | University of Kwazulu Natal |
| **US** | United States |
| **USD** | United State Dollar |
| **USDA** | United States Diabetes Association |
| **VLDL** | Very-Low Density Lipoprotein |
| **WC** | Waist Circumference |
| **WHO** | World Health Organization |
| **WHtR** | Waist-to-Height Ratio |
| **WHR** | Waist-Hip Ratio |

# CHAPTER 1: INTRODUCTION AND MOTIVATION FOR THE STUDY

## 1.1 Introduction

The International Diabetes Federation (IDF) and the World Health Organization (WHO) (2010) describes diabetes mellitus as a "metabolic disorder" characterized by a chronic high level of blood glucose (hyperglycemia), with disturbances to carbohydrate, fat and protein metabolism resulting from insulin deficiency, insulin resistance or both. Common symptoms of diabetes associated with hyperglycemia include excessive thirst, fatigue, frequent urination, hunger, and weight loss (Amod *et al.*, 2012:S5; Franz, 2012:677).

Diabetes is one of the most common non-communicable diseases globally: according to the IDF, 382 million people suffered from diabetes in 2013 (IDF, 2013b:12) and this number is estimated to rise to 592 million by 2035, which is an increase of 55% (IDF, 2013b:12). While it is the fourth leading cause of death in most high-income countries, 80% of current cases occur in low-and-middle income countries (IDF, 2013b:13, 31). The IDF (2013b:14) further estimated that in 2013, three quarters of deaths from diabetes among people younger than 60 years of age occurred in Africa.

Diabetes is among the top ten causes of disability, resulting in life-threatening complications such as heart disease, stroke, renal failure, lower limb amputations and blindness (IDF, 2013b:24). According to the IDF (2013b:14), it was projected that by the end of 2013 diabetes will have caused 5.1 million deaths globally (a person dies from diabetes every six seconds), and at least US$548 billion in healthcare expenditures, and this amount is projected to exceed US$627 billion by 2035. Furthermore, an estimated 175 million (46%) people with diabetes worldwide remain undiagnosed and unaware that they have the disease (IDF, 2013b:11, 30). In Africa, it is estimated that as many as 81% of people with diabetes are undiagnosed (IDF, 2012: Online; Amod *et al.*, 2012:S4).

The impact of diabetes was recognized as a development issue by the United Nations Resolution 61/225 of 2006, which stated that "diabetes is a chronic, debilitating and costly disease associated with severe complications, which poses a serious challenge to families of people with diabetes, to governments, as well as to the achievement of internationally agreed

development goals, including the Millennium Development Goals (MDGs) 1 (to eradicate extreme poverty and hunger), 5 (to improve maternal health), and 6 (to combat Human Immunodeficiency Virus (HIV) and Acquired Immunodeficiency Syndrome (AIDS), malaria, and other diseases)" (IDF, 2011:Online; Carter *et al.*, 2010:4229). Therefore, this resolution called upon all countries to develop national policies, protocols and guidelines for the prevention, care, and treatment of diabetes mellitus (IDF, 2011: Online; Unwin *et al.*, 2010:2).

## 1.2 Type 2 Diabetes Mellitus (T2DM)

Four types of diabetes are defined based on etiology. In type 1 diabetes mellitus (T1DM), the primary defect is pancreatic ß-cell destruction which usually leads to absolute insulin deficiency (IDF, 2013b:22; ADA, 2013a:S11; Amod *et al.*, 2012:S6; Franz, 2012:676). The second type is Type 2 diabetes mellitus (T2DM), which is characterized by a combination of insulin resistance and ß-cell failure (IDF, 2013b:23; ADA, 2013a:S11; Amod *et al.*, 2012:S6; Franz, 2012:678). Thirdly, gestational diabetes mellitus (GDM) is defined as any degree of glucose intolerance with onset or first recognition during pregnancy (IDF, 2013b:23; ADA, 2013a:S11; Amod *et al.*, 2012:S5; Franz, 2012:679). The fourth type of diabetes occurs secondary to specific genetic syndromes (for example Down syndrome), diseases of the exocrine pancreas (for example cystic fibrosis), drugs or chemicals (for example drugs used in the treatment of HIV and AIDS), surgery (for example, organ transplantation), infections (for example *Congenital rubella*), and other diseases (for example hypothyroidism) (ADA, 2013a:S11; Amod *et al.*, 2012:S5; Franz, 2012:681).

In T2DM, hyperglycemia is caused by four basic defects, namely: insulin-resistance, decreased insulin secretion, increased hepatic glucose production and reduced glucagon-like peptide-1 levels (ADA, 2013a:S67; Franz, 2012:679). T2DM is common in both males and females, with the poor being as vulnerable as the rich (IDF, 2013b:16), and contributes about 90 to 95% of all cases of diagnosed diabetes (IDF, 2012: Online; Amod *et al.*, 2012:S4; Franz, 2012:678). Risk factors for T2DM include a family history of diabetes, older age (being over 40 years old), obesity (especially intra-abdominal obesity), a history of gestational diabetes, impaired glucose tolerance, hypertension, dyslipidemia, high energy

intake, physical inactivity, smoking and harmful use of alcohol (IDF 2013b: 23; Amod *et al.*, 2012:S9; Franz, 2012:678).

The diagnosis of T2DM usually occurs around the age of 40 years, but recently there have been increasing reports of children (18 years and younger) also developing T2DM due to an epidemic of obesity and physical inactivity among children and adolescents (IDF, 2013b:23; ADA, 2013a:S14; Copeland *et al.*, 2013:366). In the United States of America (USA), T2DM currently account for one out of three new cases of diabetes diagnosed in youths (Copeland *et al*., 2013:366). T2DM can remain asymptomatic for years and the diagnosis is often made incidentally through abnormal blood test results or from diabetes-associated long-term complications (IDF, 2011: Online).

The current diagnostic criteria for T2DM in a patient with classic symptoms of hyperglycemia (polyuria, polydipsia, and weight loss) or with a hyperglycemic crisis (diabetic ketoacidosis, or hyperosmolar nonketotic hyperglycemia), include: glycated hemoglobin ($HbA_{1c}$) of greater or equal to 6.5% ($HbA_{1c}$ is a test that reflects the average blood glucose concentration over the life span of red blood cells, which is 120 days, and is expressed as the percentage of total hemoglobin with glucose attached) (ADA, 2013a:S12; Amod *et al.*, 2012:S7; Franz, 2012:682; Reddigan, 2010:106), or fasting plasma glucose (FPG) level of ≥ 7.0 mmol/L (fasting is defined as no energy intake for at least eight hours), or 2-hours plasma glucose level (during an oral glucose tolerance test [OGTT], as described by the WHO, using a glucose load containing the equivalent of 75g anhydrous glucose dissolved in 250 ml of water) of ≥11.1 mmol/L, or a random plasma glucose level of ≥11.1 mmol/L (ADA, 2013a:S12; Amod *et al.*, 2012:S7; Franz, 2012:682).

Studies suggest that at the time of diagnosis, most patients with T2DM normally would have had diabetes for at least four to seven years (Khardori & Griffing, 2012:1). The longer people remain undiagnosed, the more likely it is that their glucose will be poorly controlled and therefore the higher their risk of developing diabetes-associated complications (IDF, 2013b:23). Amod *et al.* (2012:S4) demonstrated that 20% of patients with diabetes have complications at the time of diagnosis. Among patients with T2DM, 25% are believed to have retinopathy, 9% neuropathy, and 8% nephropathy at the time of diagnosis (Khardori & Griffing, 2012:1).

According to Hammond (2012:165) nutritional status is defined as a "measurement of the extent to which an individual's physiologic need for nutrients is being met". A good nutritional status contributes to the wellbeing of a patient with diabetes by improving the glycemic control, as well as cardiovascular risk factors (hypertension and dyslipidemia) (Amod *et al.*, 2012:S7; Hammond, 2012:165).

The risk for complications are linked to poor nutritional status (at-risk anthropometric measurements, high energy intake, lack of physical activity, harmful use of alcohol and smoking), poor glycemic control (high FPG and $HbA_{1c}$), high diastolic blood pressure, infections, dyslipidaemia, longer duration of diabetes and poor self-care; all of which can be prevented and managed (ADA, 2013a:S28; Amod *et al.*, 2012:S54; Franz, 2012:704).

Patients with diabetes could achieve a good nutritional status by maintaining a body mass index (BMI) between 18.5 to 25 kg/m², waist circumference (WC) of ≤ 80 cm in females and ≤ 94 cm in males (cut-offs for metabolic complications and insulin resistance recommended by the IDF for sub-Saharan populations) (Amod *et al.*, 2012:S58), a waist-to-height ratio (WHtR) of 0.5 in both females and males at all ages (Browning *et al.*, 2010:248), and a healthy body adiposity index (BAI) of 20%-38% in females and 8%-25% in males (Bergman *et al.*, 2011:1084).

Good nutritional status should be achieved through acceptable biochemical values ($HbA_{1c}$ of < 7%, fasting blood glucose between 4.0 to 7.0 mmol/L, total serum triglycerides of < 1.7 mmol/L, total serum cholesterol of < 4.5 mmol/L, blood pressure of 140/80 mmHg or below), through a prudent dietary pattern, which include adequate energy intake and appropriately proportioned intakes of macronutrients: carbohydrates (45-60% of total energy), proteins (15-20% of total energy), and fats (<35% of total energy) (Amod *et al.*, 2012:S16; Franz, 2012:684), and a healthy lifestyle (including physical activity, safe use of alcohol and non-smoking) all contribute to a good nutritional status (ADA, 2013a:S32; Amod *et al.*, 2012:S57).

Glycemic control is defined as the achievement of normal blood glucose levels (FPG between 4.0 to 7.0mmol/l and $HbA_{1c}$ of <7.0%), which lowers the risk for diabetes-associated long-term complications (both microvascular and macrovascular), improves quality of life, and

reduces hospital admissions and mortality (Klein *et al.*, 2013: Online; Gavin *et al.*, 2010:5). Furthermore, good glycemic control is shown to increase employment retention and workplace productivity and reduces medical costs and utilization of health care resources (Gavin *et al*., 2010:5). According to Amod *et al.* (2012:Online), T2DM is generally not well managed even in developed countries, as less than 50% of patients with diabetes are able to meet their glycemic targets, while less than 10% of these patients manage to achieve lipid and blood pressure targets.

The two main techniques that are used to determine glycemic control are self-monitoring of blood glucose (SMBG) and $HbA_{1c}$ (ADA, 2013a:S17; Amod *et al.*, 2012:S20). Daily self-monitoring and recording of blood glucose levels are strongly advised for all T2DM patients, but requires expensive glucometers and glucose test strips (ADA, 2013a:S17; Franz, 2012:693). The biochemical test of choice for evaluating long term blood glucose control is $HbA_{1c}$ (ADA, 2013a:S21; Amod *et al.*, 2012:S18; Gavin *et al.*, 2010:5).

As a long term monitoring tool of glycemic control, $HbA_{1c}$ reflects adherence to both medication and diet, verifies the accuracy of the patient's glucometer, as well as the adequacy of the SMBG testing schedule (ADA, 2013a:S18; Gavin *et al.*, 2010:5). $HbA_{1c}$ also provides a guide for treatment, reinforces overall glycemic control, and provides patients with information regarding the success of their efforts (Gavin *et al.*, 2010:5). $HbA_{1c}$ should be done at least twice a year if blood glucose control is stable, and every three months if treatment has been changed or if blood glucose control is not stable (ADA, 2013a:S18; Amod *et al.*, 2012:S21).

The optimal $HbA_{1c}$ level in patients with diabetes according to Amod *et al.*, (2012:S20) and the ADA (2013a:S21), is <7.0%, which corresponds to an average whole blood glucose concentration over the last three to four months, of less than 9.57mmol/L (Average Plasma Blood Glucose (mmol/L)=($HbA_{1c}$ x 1.98)-4.290; or an average plasma glucose concentration over the last three to four months, of less than 8.55 mmol/L (Average whole blood glucose = Plasma Blood Glucose/1.12) (Rohlfing *et al.*, 2002:275).

Good glycemic control is however unlikely to be achieved with insulin or oral therapy when diet and exercise are neglected, especially when the patient is also overweight (Mann &

Morenga, 2013:453; Maghsoudi & Azadbakht, 2012: Online; Gavin, *et al.*, 2010:5). As indicated by Gavin *et al.* (2010:5) "every drug that has ever been approved for the treatment of T2DM is predicated on being adjunct to lifestyle modification".

Therefore, good glycemic control requires first and foremost adherence to a prudent diet and lifestyle which will eventually result in weight loss (Mann & Morenga, 2013:453; Maghsoudi & Azadbakht, 2012: Online). Most patients with T2DM are overweight and obese. Weight loss of greater or equal to five percent (≥5%) of current body weight in overweight and obese patients with diabetes has been shown to yield good outcomes with regard to improved insulin sensitivity and blood glucose control (Mann & Morenga, 2013:453; Maghsoudi & Azadbakht, 2012:Online; Pells *et al.*, 2012:Online).

A prudent dietary pattern is based on whole grains, fish, legumes, nuts, vegetable oils, poultry, vegetables and fruits (Quirk *et al.*, 2013:175; Maghsoudi & Azadbakht, 2012: Online). The total daily carbohydrate intake should contribute 45% to 60% of total energy (Amod *et al.*, 2012:16). The dietary reference intake (DRI) for carbohydrate is 130g per day based on the amount of glucose required to maintain the needs of the central nervous system (Institute of Medicine, 2002). Monitoring of carbohydrate intake could be done by carbohydrate counting, use of a food exchange system, or through experience-based estimation (ADA, 2013a:S22; Amod *et al.*, 2012:16). Emphasis on carbohydrates with low glycemic index, and low glycemic loads may also be beneficial for glycemic control (Ajala *et al.*, 2013:506; Amod *et al.*, 2012:16; Franz, 2012:684).

A prudent diet should also include a daily proteins intake of between 15-20% of total energy for T2DM patients without renal problems (ADA, 2013a:S22; Amod *et al.*, 2012:S16, Franz, 2012:686). Protein intake should be limited to 0.8-1.0 g per body weight per day when there are signs of renal damage (signaled by micro-albuminuria), and to 0.8g per kg body weight per day in later stages of renal damage (Beasley & Wylie-Rosett, 2013: Online). The recommended daily intake of total fat (not specific to patients with diabetes) is 20-35% of total energy per day (Evert, 2013:10).

Currently there are no recommendations for micronutrients supplementation (particularly of beta-carotene, vitamin E and C and other antioxidant) in patients with T2DM who show no

underlying deficiencies, as there is no evidence regarding their use and long-term safety (ADA, 2013a:S23; Amod *et al.*, 2012:S16; Franz, 2012:687). Nonetheless, a multivitamin supplement may be recommended to the elderly, or to pregnant or lactating women, strict vegetarians, or those on energy-restricted diets (Franz, 2012:687).

A prudent lifestyle includes the safe use of alcohol, tobacco avoidance and being physically active (Maghsoudi & Azadbakht, 2012: Online). In a prudent lifestyle alcohol intake should be limited to a moderate amount, which translates to one drink or 15g alcohol or less for adult females, and 2 drinks or 30g alcohol per day or less for adult males, and at least one alcohol free day per week (ADA, 2013a:S23; Amod *et al.*, 2012:S16; Franz, 2012:687). One drink is equivalent to 330ml beer (3-4% alcohol by volume); 150ml of wine; 25ml of distilled spirits (40% alcohol by volume); or 50ml of fortified wine such as sherry or port (20% alcohol by volume). Patients on insulin need to be cautioned against alcohol-induced hypoglycemia, and hence be advised to take alcohol with food (Amod *et al.*, 2012:16). When excessive amounts of alcohol (>3 drinks per day) are consumed on a consistent basis may contribute to hyperglycemia (Evert *et al.*, 2013: Online), and development of cardio metabolic factors (dyslipidaemia and hypertension) (Heianza *et al.*, 2013: Online).

Cigarette smoking is linked to increased insulin resistance and to the risk for developing T2DM (CDC, 2011: Online; Nyamdorj, 2010:21). Smoking is specifically discouraged for patients with diabetes, therefore patients with diabetes are recommended not to use any form of tobacco (Glass *et al*., 2009:40). Furthermore, several studies demonstrated that smoking increases the risk of cardiovascular complications in patients with diabetes and hypertension (Raz, 2009:S149).

Physical activity is defined by Franz, (2012:688), as a "planned, structured and repetitive bodily movement performed to improve or maintain one or more components of physical fitness" (De Feo & Schwarz, 2013: Online; Franz, 2012:688; Hovanec *et al.*, 2012: Online. Physical activity improves glycemic control in patients with T2DM by increasing insulin sensitivity, which eventually results in increased peripheral use of glucose during and after exercise (ADA, 2013a:S25; Franz, 2012:688). Physical activity is shown to reduce cardiovascular risk factors (dyslipidemia, hypertension) and depression; to decrease the dosages of chronic medication required to control blood glucose levels; and to improve

weight management, physical movements, cognitive function, and quality of life (ADA, 2013a:S25; Laverty, *et al.*, 2013:282; Plotnikoff *et al.*, 2013:3; Amod *et al.*, 2012:18; Roden, 2012: Online; Conn *et al.*, 2011: Online).

The recommendations for physical activity include that patients with T2DM are to perform at least 150 minutes per week of moderate-intensity aerobic exercises (such as brisk walking, bicycling, continuous swimming, dancing and gardening) at 50% to 70% of maximum heart rate, or at least 90 minutes per week of vigorous aerobic exercise (such as brisk walking up a slope, jogging, aerobics, hockey, basketball) at more than 70% of maximum heart rate (ADA, 2013a:S24; Amod *et al.*, 2012:18). Another recommendation is to distribute physical activity over at least three days per week, with no more than two consecutive exercise-free days (ADA, 2013a:S24; Amod *et al.*, 2012:18; Franz, 2012:689).

Patients without contraindications should be encouraged to perform resistance exercise (such as weight lifting, using resistance providing machines) three times a week, targeting all major muscle groups (ADA, 2013a:S24; Amod *et al.*, 2012:18; Franz, 2012:689; Hovanec *et al.*, 2012:284635). Franz (2012:689) further indicated that the patient's well-being needs to be assessed, and high-risk patients should be advised to start with short periods of low-intensity exercise and increase the intensity and duration slowly. Therefore, physical activity should vary depending on the interest, age, general health, and level of physical fitness of the patient (Franz, 2012:688).

Lastly, good glycemic control is also dependent on adherence to the prescribed medical treatment (oral glucose lowering drugs and where necessary, insulin), compliance to treatment, and proper self-management (ADA, 2013a:S17; Klein *et al.*, 2013: Online; Mbaezue *et al.*, 2010: Online). Diabetes self-management entails performance of SMBG, adherence and compliance to multiple medications, maintenance of foot hygiene, adherence to a healthy diet and meal plans, and engagement in an exercise program (Mbaezue *et al.*, 2010:Online). SMBG is one of the tools in diabetes care that have been shown to improve glycemic control, and is recommended at least once a day for patients with T2DM (ADA, 2013a:S17; Mbaezue *et al.*, 2010: Online).

Gavin *et al*. (2010:8) indicated that adherence to medication among patients with diabetes ranges from 36% to 87% with oral agents, and from 54% to 81% with insulin-only regimens. Factors which have been shown in studies to challenge the medication adherence in patients with diabetes are: lack of knowledge about the disease, side effects of medication such as gastrointestinal disturbances and weight loss, regimen complexity, dosing frequency (more than twice per day), cost, lack of confidence in the medication's benefits, lack of education about the use of the medication, depression, fear of hypoglycemia, an awareness of the complexity of diabetes management (for instance, what to eat, when to eat, how much to eat, and taking of medications) which may be overwhelming for some patients, provision of below standards services by some clinics, and failure to encourage patients with diabetes to do regular blood glucose monitoring (Gavin *et al*. 2010:11).

Good glycemic control also depends on screening and monitoring for the development of long-term complications (ADA, 2013a:S17). According to ADA and SEMDSA guidelines, blood pressure should be measured at every routine visit to the diabetes clinic or primary health care provider; WC and weight should be measured at each visit, BMI should be evaluated annually; a comprehensive foot examination should be done annually or more often in patients with high risk of foot conditions; micro-albuminuria and serum creatinine should be evaluated annually; eye examinations to screen for retinopathy should be performed annually or more frequently if significant retinopathy is present; and neuropathy should be screened for in all patients at least annually (ADA, 2013a:S29; Amod *et al.*, 2012:S12).

### 1.3 The diabetes situation in Sub-Saharan Africa

The literature indicated that until the mid-1980s, Africa was considered safe from many of the so called "disease of the affluence", including diabetes, which were already common in developed countries (Amod *et al.*, 2012:S12). However, due to the effects of urbanization, rapid cultural changes, an aging population, and the high prevalence of obesity and unhealthy lifestyles, the prevalence of diabetes in Africa has rapidly escalated (IDF, 2013b:23). The prevalence of diabetes in the Africa region is currently estimated at 19.8 million (4.9%) (IDF, 2013b:56). The Africa region have also been shown to have the highest proportion of undiagnosed diabetes (at least 63%) (IDF, 2013b:56).

In the Africa region the prevalence of diabetes is the highest in the Islands of Reunion (15.4%), followed by Seychelles (12.1%), Gabon (10.7%), and Zimbabwe (9.7%) (IDF, 2013b:56). Some of Africa's most populated countries also have the highest numbers of people with diabetes, and include: Nigeria (3.9 million), South Africa (2.6 million), Ethiopia (1.9 million), and the United Republic of Tanzania (1.7 million) (IDF, 2013b:56). Furthermore, 8.6% of all deaths in the Africa region are attributed to diabetes, while in 2013, 522 600 (76.4%) of those deaths occurred in people under the age of 60 years (IDF, 2013b:57). The health expenditure on diabetes was estimated at USD 4 billion in 2013, and this is projected to increase to 58% by 2035 (IDF, 2013b:57).

### 1.4 The diabetes situation in Lesotho

Lesotho is a small mountainous kingdom (Figure 1.1) completely surrounded by the Republic of South Africa. The country is divided into ten administrative districts with a total area of about 30,355 square kilometers, and less than 10.1% of the land is arable (CIA, 2012:Online; LDHS, 2009:1). Lesotho is primarily a country of subsistence farming. The major agricultural products are wheat, corn, sorghum, barley, pulses and livestock (CIA, 2012: Online). The gross domestic product (GDP) is M9.013 million, with an annual growth rate of 4.4% (LDHS, 2009:1). The inflation rate is estimated at 6.1%, and unemployment rate is 34.4% (CIA, 2012: Online). Agriculture contributes about 6.7% of the GDP, while manufacturing accounts for 34.6% of the GDP (CIA, 2012: Online; LDHS, 2009:1). Approximately, 56.6 % of the population lives below the poverty line (<US$ 1 per day) and such inequalities have adverse health consequences on Basotho nation (LNNS, 2007:49).

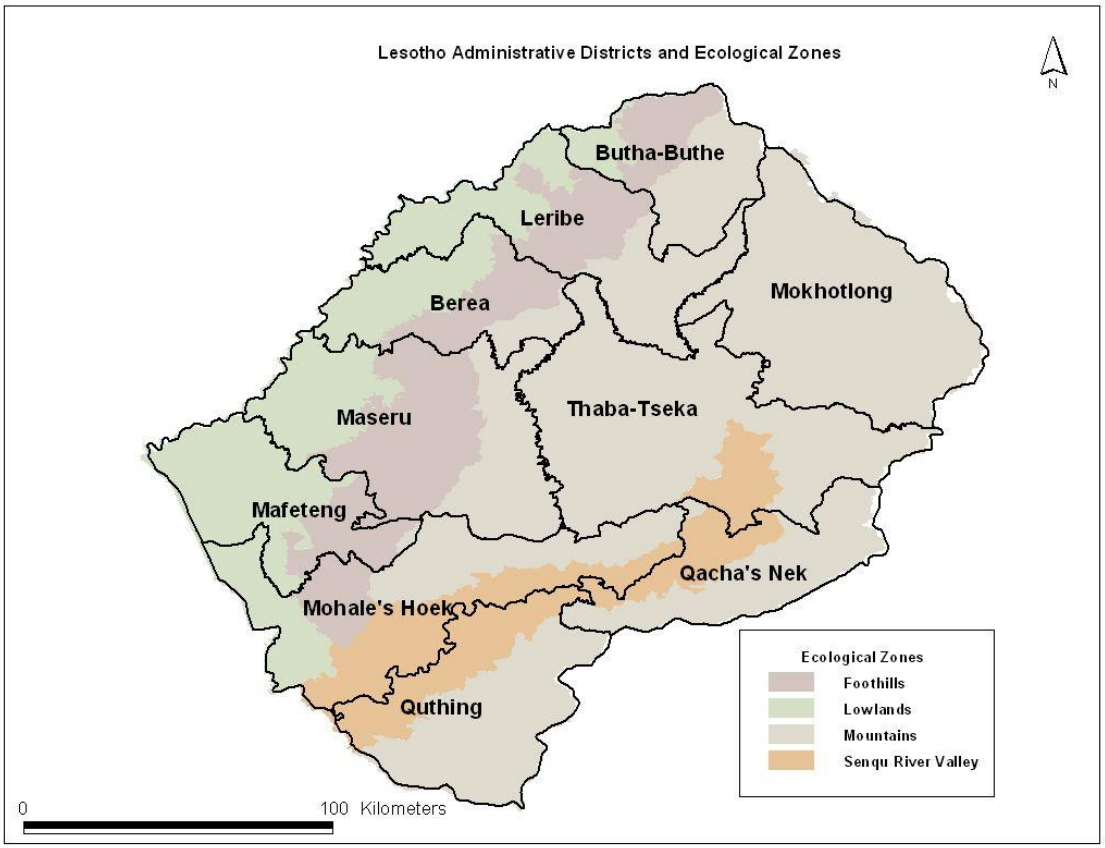


**Figure 1.1:** Administrative districts and ecological zones of Lesotho (LNNP, 2011:5, Figure 1).

The CIA World Fact Book (2012: Online) estimated the 2013 Lesotho population at 1,936,181, consisting of 99.7% Basotho. The birth rate was estimated for 2013 at 26.31 births per 1000 population and the death rate at 15.02 deaths per 1000 (CIA, 2012: Online). Maseru is the capital city of Lesotho with a population of 220 000 (CIA, 2012: Online).

The situation in Lesotho is no different from the rest of Africa, or from that of neighboring South Africa with respect to rapid cultural changes, an aging population, increasing urbanization, unhealthy lifestyles, high prevalence of obesity, and unhealthy behavioral patterns without prevention and control preparedness. Lesotho, like other low-middle income countries is undergoing social and economic changes, which are resulting in increased urbanization with a potentially negative impact on health related behavior (Echouffo-Tcheugui & Kengne, 2011:9). Data from population censuses show that while the population of Lesotho is still predominantly rural, there is an observed population increase in the urban areas (LDHS, 2009:1). In 2010, 27% of the population lived in urban areas and it was estimated that the urban population will increase annually by 3.4% from 2010 to 2015 (CIA,

2012: Online). This increasing urbanization is exposing Basotho to a more sedentary lifestyle and highly processed and unhealthy foods, which are high in refined carbohydrates, saturated and trans-fats, salt and sugar. Lesotho, like the rest of Africa, is also experiencing the double burden of communicable (tuberculosis (TB), HIV and AIDS), and non-communicable diseases (obesity, heart diseases, hypertension, T2DM, and cancer), which if nothing is done, will overwhelm the healthcare systems and resources (Echouffo-Tcheugui & Kengne, 2011:9).

Diabetes is among diseases which appear in the higher ranks in the morbidity data sheet in Lesotho, and the cost incurred on the health care of patients with diabetes in primary, secondary and tertiary institutions is greatly subsidized by the government through the Ministry of Health (MOH) (LRFS, 2001:11). The Lesotho Risk Factor Survey (LRFS) done in 2001, identified diabetes as the second most treated disease in Lesotho hospitals, and reported that 1.5% of the population had diabetes (1.1% had diabetes and 0.4% had impaired glucose tolerance). The prevalence rate of diabetes in the capital city, Maseru, was at 1.9% (LRFS, 2001:11). However by 2012, the IDF Diabetes Atlas reports the prevalence of T2DM in Lesotho at 3.46%, with the highest rate occurring in the rural areas and women being affected most (IDF, 2012: Online).

In Maseru, health care services are rendered to people with diabetes by public clinics and hospitals, and by private clinics and hospitals, as well as general practitioners. Some patients with T2DM seek treatment from private health care facilities, especially over weekends, on public holidays, in emergency situations, and/or as a matter of preference. Health care services received from private health care facilities are usually financed through medical aids and/or self financed. Some patients also seek health care services from non-conventional medicine due to a combination of factors such as loss of trust in conventional medicine, lack of knowledge and individual preference (LRFS, 2001:11).

Queen Elizabeth II Hospital (QE II) was a referral hospital offering a range of specialist services in the country. As Lesotho's major hospital, QE II also acted as a primary care facility for the urban population of Maseru where diabetes clinics were held every Wednesday. As from 1st October 2011, QE II was closed down and the patients of the diabetes clinic were redirected to the new Queen Mamohato Memorial Hospital (QMMH)

(Gateway clinic) nearby, where diabetes clinics are continued to be conducted, as well as to three other Private-Public-Partnership (PPP) clinics (Ts'epong clinics) and two District Health Management Team (DHMT) clinics around Maseru.

The LRFS (2001) reported that admission rates for patients with diabetes, who present with either acute or chronic complications were escalating in Lesotho, and were possibly related to lack of compliance, lack of knowledge about the disease, and use of traditional and herbal medicines instead of allopathic medicines prescribed by clinicians (LRFS, 2001:11).

While working as a dietician at the QE II diabetes clinic prior to its closure in 2011, the researcher observed several challenges which may have negatively impacted on the level of diabetes control of patients attending the clinic, and which may remain possible problems in the new settings. Firstly, patients were requested to attend the clinic once every third month, while during the two months in-between, they only collected their medication from the pharmacy. Thus patients only had their blood glucose levels checked at the clinic every third month (without $HbA_{1c}$ test).

Secondly, some patients from outside Maseru also preferred to attend the clinic at QE II hospital. Previously patients had to arrive early and wait for long periods of time to be attended to at the diabetes clinic at QE II. Hence attendance seemed to depend on the motivation, resources and mobility of the patients which may have contributed to poor compliance and adherence to prescribed medications, as some patients with diabetes missed their clinic dates and defaulted on their treatment. The researcher is of the opinion that the diabetes population attending the clinic at QE II was too large for provision of good services, but this is expected to improve at the new facility.

Thirdly, the researcher observed that few patients with diabetes were referred for nutritional counseling, implicating that the importance of lifestyle and dietary modification in the management of diabetes were not well recognized at the QE II diabetes clinic.

Fourthly, a high turnover of professionals working with patients with diabetes, especially doctors and nurses was noticed, which may have impacted negatively on the treatment of the

patients with diabetes who need to be closely monitored and followed up to ensure that they achieve and maintain their treatment goals.

Fifthly, the researcher observed that some patients with diabetes opt for use of traditional and herbal medicines instead of hypoglycemic medications prescribed by doctors, because they lack information, have low socio-economic status, and/or are also bound by cultural beliefs to do so.

Lastly, the researcher observed that most patients with diabetes previously admitted at QE II hospital with complications, seemed to lack knowledge about the disease, lifestyle modification, medical management, and self-care.

These factors, combined with challenges related to socio-economic status, level of education, social environment, lack of psychosocial and emotional support, lack of good communicative relationship between the patient and health care providers, and patients' knowledge, attitudes, beliefs and perceptions regarding dietary, lifestyle changes and medical therapies, may add up to significant barriers that may prevent compliance with treatment needed for optimal glycemic control.

## 1.5 Problem Statement

The LRFS, 2001 reported escalating admission rates for patients with diabetes, with acute and/or chronic complications. Lack of compliance, lack of knowledge about the disease, and use of traditional and herbal medicines instead of allopathic medicines prescribed by clinicians, were identified as contributing factors (LRFS, 2001:11). The researcher's observations while working as a dietician at the diabetes clinic of the former QE II Hospital until its closure in October, 2011, corroborate these findings.

There is however no literature assessing the nutritional status, including dietary compliance and related risk factors such as obesity, physical activity, smoking, alcohol usage; and glycemic control; nor the perceived barriers which may influence the compliance to treatment among patients with diabetes attending diabetes clinics in Lesotho.

In order to bridge this gap and also to expand on the LRFS, the current study was undertaken to describe the nutritional status and the glycemic control of patients with diabetes attending clinics in Maseru, Lesotho, as well as to describe the barriers that these patients perceive and encounter, which may negatively impact on their compliance to medical and lifestyle treatment.

The findings of this study may provide crucial baseline information that can be used in the planning and implementation of prevention, control and treatment strategies for T2DM in Lesotho.

## 1.6 Aim and Objectives

### 1.6.1 Aim

The aim of the study was to determine the nutritional status, glycemic control, and barriers that impact on treatment compliance among patients with T2DM who attend diabetes clinics in Maseru, Lesotho.

### 1.6.2 Objectives

In order to achieve the aim, the following objectives needed to be determined:

**1.6.2.1** Socio-demographic factors (age, gender, residential area, marital status, level of education, employment, income level, number of dependents);

**1.6.2.2** Nutritional status, through:

i) Anthropometric measurements (BMI, WC, WHtR, BAI);

ii) Usual dietary intake (total energy, and macronutrients); and

iii) Lifestyles factors (alcohol intake, tobacco use, physical activity);

**1.6.2.3** Medical history (year of diagnosis, biochemical results, glucose lowering medication, comorbid conditions, complications of diabetes mellitus);

**1.6.2.4** Glycemic control ($HbA_{1c}$); and

**1.6.2.5** Barriers that may impact on treatment compliance (factors related to provision of health care services at the clinic, socio-economic status, social environment, and patients' knowledge, attitudes, beliefs and perceptions regarding dietary and lifestyle changes, and medical therapies).

## 1.7 Layout of the dissertation

**Chapter 1** – Introduction and motivation of the study are discussed, including the background, problem statement, aim, objectives and the layout of the dissertation.

**Chapter 2** – The literature review include the definition of diabetes, types of diabetes, etiology and risks factors, and management of diabetes.

**Chapter 3** – Methods used to conduct the study are described in this chapter. The ethical approval and permission, the study design, study population and sampling, measurements – variables and operational definitions; techniques, study procedure; selection and standardization of techniques to ensure validity and reliability are described. Ethical considerations, the pilot study and the statistical analysis are included. The limitations of the study, and steps taken to overcome these, are discussed.

**Chapter 4** – Results of the study are discussed and summarized, and the problems encountered during the study are outlined.

**Chapter 5** – The results of the study are interpreted and discussed in the context of the relative literature. The current results are compared with previous studies in similar and different settings.

**Chapter 6** – Conclusions are drawn from the results of this study and recommendations for improvements and further research, as well as recommendations for the planning and implementation of prevention, control and treatment strategies for T2DM in Lesotho, based on the findings of the current study, are made.

## CHAPTER 2: LITERATURE REVIEW

In this chapter diabetes mellitus is reviewed with regard to definition and classifications, prevalence, etiology and risk factors, diagnosis, management, complications, glycemic control and barriers to treatment compliance.

### 2.1 Introduction

Diabetes Mellitus is emerging as a major public health concern across the world and is increasingly being diagnosed in the low-and-middle income countries, including Lesotho. Diabetes (especially T2DM) is one of the main threats to human health and the achievement of the MDGs particularly MDG 1, 5, and 6 (IDF, 2011: Online; Stuckler *et al.*, 2010: Online). For instance, in order to achieve MDG 1 (to eradicate extreme poverty and hunger), the prevention and management of diabetes need to be strengthened, because those individuals who are at low-socio-economic status are also shown to be at an increased risk of diabetes (IDF, 2011:Online; Stuckler *et al.*, 2010:Online).

Research further showed that 80% of diabetes cases occur in low-and-middle income countries due to rapid growth in economy (IDF, 2013b:16), unequal distribution of resources and an imbalance between economic development and education (IDF, 2011: Online). This confirms that diabetes is a disease associated with poverty, as it seems to affect the lower socio-economic and disadvantaged groups as well (IDF, 2013b:16; Stuckler *et al.*, 2010: Online). WHO reported that there are approximately 80% of diabetes deaths that occur in low-and-middle income countries, and if no urgent action is taken, diabetes deaths are projected to increase by more than 50% in the next ten years (WHO, 2010:Online).

There is a great challenge in achieving MDG 5 - to improve maternal health, as uncontrolled and undiagnosed diabetes during pregnancy is associated with delivery of macrosomic babies, which can result in life threatening and costly complications for the mother, as well as the new born child (IDF, 2011:Online; Stuckler *et al.*, 2010:Online). Inadequate intrauterine nutrition was proven to result in low-birth weight and poor development of the central nervous system in infants (Zimmet *et al.*, 2014: Online; Wang *et al.*, 2014: Online; Hales & Barker, 2013: Online; Malik *et al.*, 2011: Online), and low-birth weight seems to be early

markers of subclinical risk of future development of T2DM (James-Todd *et al.*, 2013: Online). Wang *et al.* (2014: Online) also stated that preterm birth may be a risk factor for the future development of insulin resistance and T2DM. Furthermore, IDF (2013b:13) indicated that more than 21 million live births were affected by diabetes during gestation period in the year 2013.

In low-and-middle income countries, Lesotho not an exception, there is an interrelation and interdependence of infectious and chronic diseases, MDG 6 (to combat HIV and AIDS, malaria, and other diseases) (IDF, 2011: Online). People with diabetes are three times more likely to develop TB (diabetes is one of the factors that impair the host's defense against TB infections and diseases) (Han *et al.*, 2012:2088; IDF, 2011:Online), and the life-long anti-retroviral treatment (especially protease inhibitors) can triple the risk of diabetes (due to insulin resistance) in people with HIV and AIDS (Han *et al.*, 2012: 2088; IDF, 2011:Online; Levitt *et al.*, 2011:Online; Maher *et al.*, 2010:2; Stuckler *et al.*, 2010:Online).

### 2.2. Definition and classifications of diabetes mellitus

Diabetes mellitus is described as a "metabolic disorder" characterized by chronic high level of blood glucose (hyperglycemia), with disturbances to carbohydrate, fat and protein metabolism resulting from insulin deficiency, insulin resistance or both (ADA, 2013b:S67; Franz, 2012:676; IDF and WHO, 2010:Online). Insulin is a hormone produced by the beta-cells of the pancreas and released into the body to transport glucose from the blood into the cells so that it can be used as an energy source (IDF, 2013b:27; Franz, 2012:676). Insulin also contributes to protein synthesis and stimulates the storage of free fatty acids in the adipose tissue (IDF, 2013b:27). However, the main role of insulin is to control the storage of glucose in the liver as glycogen and release of glucose, as a result maintaining blood glucose within the normal range (IDF, 2013b:27; ADA, 2013b:S67; Franz, 2012:676 ).

The disease is defined by $HbA_{1c}$ of 6.5% or above, fasting plasma glucose (FPG) of 7.0mmol/l or above, or random plasma glucose of 11.1mmol/l or above, or an abnormal oral glucose tolerance test of 11.1mmol/l or above (ADA, 2013a:S13; Amod *et al.*, 2012:S7; Franz, 2012:682). Common symptoms of diabetes associated with hyperglycemia include

excessive thirst, frequent urination, polyphagia, blurred vision and weight loss (Amod *et al.*, 2012:S5; Franz, 2012:677; IDF, 2011: Online).

Diabetes is classified according to the causes and symptoms as follows:

### 2.2.1 Type 1 diabetes mellitus (T1DM)

T1DM used to be called insulin-dependent, immune-mediated or idiopathic in origin diabetes, and is characterized by an absolute insulin deficiency, and accounts for 5% to 10 % of diabetes cases (Amod *et al.*, 2012:S6; Franz, 2012:676). It is caused by the destruction of the insulin-producing cells (beta-cells) of the pancreas (mainly as a result of an auto-immune reaction) (IDF, 2013b:22; ADA, 2013a:S11; Amod *et al.*, 2012:S6; Franz, 2012:676). T1DM can affect people of any age, but usually found in children (below 18 years of age) or young adults (below 30 years of age), hence is one of the most common endocrine and metabolic conditions in childhood (Amod *et al.*,2012:S6; Franz, 2012:676). The incidences of T1DM are increasing probably due to changes in environmental risk factors, increased height and overweight and obesity, increased maternal age at delivery, some aspects of diet (decreased intake of some micronutrients) and exposure to some viral infections, which may initiate autoimmunity (IDF, 2013b:22; Franz, 2012:676).

### 2.2.2 Type 2 diabetes mellitus (T2DM)

T2DM accounts for 90% to 95% of all diabetes cases, and is characterized by insulin resistance and relative insulin deficiency, leading to chronic hyperglycemia (IDF, 2013b:23; ADA, 2013a:S11; Amod *et al.*, 2012:S6; Franz, 2012:678). T2DM is caused by four basic defects, namely insulin-resistance, decreased insulin secretion, increased hepatic glucose production and reduced glucagon-like peptide-1 levels, which may already be present during diagnosis (Franz, 2012:679). T2DM is common in both males and females, especially in population with high diabetes prevalence (Asians) with the poor being as vulnerable as the rich (IDF, 2011: Online). Diagnosis of T2DM usually occurs after the age of 40 years but could occur earlier, and there are increasing reports of children (younger than 18 years) who also develop T2DM (ADA, 2013a:S14; Copeland *et al.*, 2013:366; IDF, 2013b:23).

T2DM can remain asymptomatic for many years and diagnosis is often made from associated complications (such as blindness and incurable wounds), or incidentally through an abnormal

blood glucose test results (IDF, 2011: Online). It is often, but not always associated with obesity, which itself can cause insulin resistance and lead to elevated blood glucose levels (Franz, 2012:678; IDF and WHO, 2010: Online). Some independent risk factors of T2DM include a family history of diabetes, increasing age (being over 40 years old), obesity (especially abdominal obesity), gestational diabetes, impaired glucose tolerance, hypertension, dyslipidemia, high energy diet, less than optimum intrauterine environment, ethnicity, physical inactivity, smoking and harmful use of alcohol (IDF, 2013b:23; Amod *et al.*, 2012:S4; Franz, 2012:678).

Unlike T1DM, people with T2DM are not dependent on exogenous insulin and are not ketosis-prone, but may eventually require insulin for control of hyperglycemia if not achieved with diet alone or with oral hypoglycemic agents (tablets) (IDF, 2013b:23; Franz, 2012:679). The number of people with T2DM is increasing in every country, and is common between 40 to 59 years of age (IDF, 2013b:30, 34). The rising prevalence is associated with cultural and social changes, ageing populations, increasing urbanizations, dietary changes, reduced physical activity and other unhealthy lifestyle and behavioral patterns (IDF, 2013b:23).

### 2.2.3 Gestational diabetes mellitus (GDM)

GDM is a glucose intolerance of varying degrees of severity which starts during pregnancy (IDF, 2013b:23; ADA, 2013a:S11; Amod *et al.*, 2012:S6; Franz, 2012:679). The prevalence of GDM among pregnant women is approximately 7%, and 5% to 10% of women with GDM will have an increased risk of developing T2DM in later years (Franz, 2012:679). Maintenance of normal blood glucose levels during pregnancy reduces the risk to the baby as an increased maternal glucose level could result in complications in the baby including large size at birth (macrosomic), birth trauma, hypoglycemia and jaundice (IDF, 2013b:23; Amod *et al.*, 2012:S76). Babies born to mothers with GDM have an increased risk of obesity and abnormal glucose metabolism during childhood and developing T2DM during adulthood (IDF, 2013b:23).

### 2.2.4 Other specific types of diabetes

Other specific types of diabetes include those secondary to other causes such as genetic defects of beta cell function, defects of insulin action, pancreatic disease (e.g. pancreatitis, neoplastic disease, pancreatectomy, Cystic fibrosis etc.), excess endogenous production of

hormonal antagonists to insulin (e.g. growth hormone, glucagon, glucocorticoids, catecholamines, and thyroid hormones), viral infections (e.g. congenital rubella, mumps, Coxackie virus B, Cytomegalovirus), diseases of the exocrine pancreas, endocrinopathies (e.g. acromegaly, Cushing's syndrome, hyperthyroidism) and drugs-induced (e.g. corticosteroid, thiazide, diuretics, beta-blockers and phenytoin) (ADA, 2013a:S11; Franz, 2012:681; Amod *et al.*, 2012:S6). Other genetic syndromes sometimes associated with diabetes are Down's syndrome, Klinelfelter's syndrome, Turner's syndrome, although these types of diabetes are relatively uncommon (ADA, 2013a:S11; Franz, 2012:681; Amod *et al.*, 2012:S6).

### 2.3. Global prevalence of diabetes mellitus

T2DM is one of the most common non-communicable disease (NCDs) globally. According to IDF (2013b:12), 382 million people suffered from diabetes in 2013, and this number is estimated to rise to 592 million by 2035, a 55% increase, and the majority of people have T2DM. While it is the fourth leading cause of death in most high-income countries among people under the age of 60, (approximately 5.1 million patients die in a year from the disease) (IDF, 2013b:14; IDF, 2012:Online), 80% of cases occur in low-and-middle income countries (IDF, 2013b:16, 31). The greatest number of people with diabetes is currently between 40 and 59 years of age, and by the year 2035, this age group is still expected to comprise the largest number of people with diabetes (IDF, 2013b:30, 34; Carter *et al.*, 2010:4229).

Diabetes is among the top ten causes of disability, resulting in life-threatening complications such as heart disease, stroke, renal failure, lower limb amputations and blindness (IDF, 2012: Online). The annual healthcare expenditure was calculated at US$548 billion in 2013, and this number is projected to increase to US$627 billion by 2035 (IDF, 2013b:48). An estimated 175 million (46%) people with diabetes are undiagnosed and unaware that they have the disease (IDF, 2013b:38; Amod *et al.*, 2012:S4). This shows that the global burden of diabetes is far larger than previously estimated, as indicated in Table 2.1.

According to the data obtained from 2005-2008, 25.6 million (11.3%) of all people above 20 years of age in USA, have diagnosed (1.9 million are newly diagnosed) or undiagnosed diabetes (Nakamura & Omaye, 2012: Online; CDC, 2011: Online). Hence, IDF (2012:

Online) estimated the number of people with diabetes (between 20 to 79 years) in USA to be 24.1 million. While the data collected by the Centers for Disease Control and Prevention in 2010 showed similar results with an estimated total of 25.8 million (8.3%) people having diabetes (Molitch, 2013:Online).

**Table 2.1: Global and Regional Prevalence of Diabetes (IDF, 2012: Online)**

| Region | Diabetes Population (Million) | Prevalence (%) | Undiagnosed (%) |
|---|---|---|---|
| **World** | 371 | 8.3 | 50 |
| **Africa** | 15 | 4.3 | 81.2 |
| **Middle East and North Africa** | 34 | 10.9 | 52.9 |
| **South- East Asia** | 70 | 8.7 | 51.1 |
| **North America and Caribbean** | 38 | 10.5 | 29.2 |
| **South and Central America** | 26 | 9.2 | 45.5 |
| **Western Pacific** | 132 | 8.0 | 57.9 |
| **Europe** | 55 | 6.7 | 38.6 |

The increase in T2DM in the USA is associated with changes in diet, particularly an increase in intake of refined carbohydrates, including sugary beverages (Nakamura & Omaye, 2012: Online). Diabetes is one of the most common chronic childhood diseases in the US; more than 13,000 youths are diagnosed with diabetes every year, and T2DM accounts for 8% to 45% of new childhood diabetes due to sedentary lifestyles (CDC, 2011:Online). Hence, the prevalence of T2DM in childhood has increased by 33% in the past 15 years, as a result of increasing rates of overweight and insulin resistant (CDC, 2011:Online).

According to IDF (2012:Online), the prevalence of diabetes in other developed countries was as follows: 5.59% in England, 4.2% in Sweden, 5.89% Netherlands, 5.79% Finland, 7.15% Spain and 5.57% Italy, 5.52% in Germany, and 9.31% in Cyprus. The top five Asian countries with the highest prevalence of diabetes are: India (63.0 million), China (92.3 million), Pakistan (6.6 million), Japan (7.1 million) and Indonesia (7.6 million) (IDF, 2012: Online; Herman & Zimmet, 2012: Online).

However, the latest national study conducted in China revealed that, China has overtaken India and has become "the global epicenter of the diabetes epidemic" with 98.4 million adults with diabetes (IDF, 2013:Online; Tuomi *et al.*, 2013:Online). The prevalence of diabetes in

China showed the following characteristics: it increased with age, males have higher prevalence than females, it is common among adults aged 20 to 60 years, and there is a positive relationship between central obesity, education and incidence of diabetes (IDF, 2013b:34; Tuomi *et al.*, 2013:Online; IDF, 2011:Online).

### 2.3.1 Prevalence of diabetes mellitus in Sub-Saharan Africa

The literature indicated that until the mid-1980s, Africa was considered safe from many of so called 'disease of the affluence' such as diabetes, which was already common in the developed countries (Amod *et al.*, 2012:S12). However, due to the effects of urbanization, rapid cultural changes, an aging population, high prevalence of obesity and unhealthy lifestyles, the prevalence of diabetes in Africa has escalated rapidly (IDF, 2013b:23), and is estimated at 19.8 million (4.9%) (IDF, 2013b:56).

The Africa Region is also shown to have the highest proportion of undiagnosed diabetes (IDF, 2013b:56). What is even more worrying is that diabetes epidemic is becoming more prevalent among people in their reproductive years (below 60 years) and those contributing to economic development (IDF, 2011:Online). Maher *et al.* (2010:Online) indicated that in Africa, the NCDs burden is likely to increase as more HIV-infected people receive anti-retroviral treatment (ART), as ART result in metabolic side effects more especially T2DM. Levitt *et al.* (2011:Online) reported that with the epidemic of HIV and AIDS, the prevalence of diabetes has risen tremendously in Sub-Saharan Africa, and the introduction of ART has resulted in increased weight gain, change in the distribution of fat deposition, and this seem to result in a high prevalence of dysglycemia in HIV-infected patients on ART.

It is estimated that 81% of people with diabetes are undiagnosed and progressing towards complications unaware (IDF, 2012: Online; Amod *et al.*, 2012:S4). In the Africa Region the highest prevalence of diabetes is as follows: Islands of Reunion (15.4%), followed by Seychelles (12.1%), Gabon (10.7%), and Zimbabwe (9.7%) (IDF, 2013b:56). Some of Africa's most populated countries also have the highest numbers of people with diabetes, and include: Nigeria (3.9 million), South Africa (2.6 million), Ethiopia (1.9 million), and the United Republic of Tanzania (1.7 million) (IDF, 2013b:56). Furthermore, 8.6% of all deaths in Africa Region are attributed to diabetes, about 522, 600 (76.4%) of those deaths occurred in people under the age of 60 years (IDF, 2013b:57). The health expenditure on diabetes was

estimated at US$4 billion in 2013, and this is projected to increase by 58% in the year 2035 (IDF, 2013b:57).

According to Whiting *et al.* (2011) it is estimated that the prevalence of diabetes in Sub-Saharan Africa will increase by 90% in the year 2030. The IDF Diabetes Atlas 2012 estimates of the current prevalence of diabetes in Sub-Saharan Africa are summarized in Table 2.2.

In 1994 the prevalence of diabetes in Cameroon was 0.8% in the rural, and 1.6% in the urban adult population (Echouffo-Tcheugui & Kengne, 2011:44). By the year 2003 there was a 10-fold increase in diabetes prevalence in Cameroonian adults (Echouffo-Tcheugui & Kengne, 2011:44). IDF estimated the national prevalence of diabetes among adults aged 20 to 79 years at 6.15% (IDF, 2012: Online). Hence 80% of the adult population in Cameroon has undiagnosed diabetes, and glycemic control in known diabetes patients is often very poor (only a quarter of diabetes population achieve optimal fasting blood glucose levels) (Echouffo-Tcheugui & Kengne, 2011:44).

In Zimbabwe, diabetes was reported as the fifth among the top ten most common diseases (Hjelm & Mufunda, 2010:7). The prevalence of diabetes increased from 150 to 550 per 100,000 people between the years 1990 to 1997, implying that the overall prevalence of diabetes in Zimbabwe has increased threefold (Hjelm & Mufunda, 2010:7). The Zimbabwe National Profiles of 1996 to 1998 demonstrated that the newly diagnosed diabetes from ages of 15 years and above had increased from 2734 cases in 1996 to 5114 cases in 1998, which is an increase of 87% of recorded cases (Hjelm & Mufunda, 2010:7). The IDF (2012: Online) estimated the comparative prevalence of diabetes in Zimbabwe to be 9.75%, while the number of people with undiagnosed diabetes are 469.79 in 1000s.

Furthermore, Whiting *et al.* (2011: Online) reported that Sub-Saharan Africa would have the greatest proportional increase in diabetes by the year 2030, compared to all other IDF regions. In Tanzania diabetes was estimated to increase by 48,000, Malawi would increase by 21,000, and the Democratic Republic of Congo would increase by 36,000, and suggested that the age-adjusted prevalence of T2DM in Sub-Saharan Africa was 5% and would increase to 5.9% by the year 2030 (Whiting *et al.*, 2011:Online).

**Table 2.2: The Sub-Saharan Africa prevalence estimates of Diabetes mellitus (IDF, 2012: Online)**

| Country | Diabetes cases (20-79) in 1000s | Diabetes comparative prevalence (%)WHO standard | Diabetes related deaths (20-79) | Mean DM related expenditure/ person with diabetes (USD) | Number of people with undiagnosed diabetes (20-79) in 1000s. |
|---|---|---|---|---|---|
| **Angola** | 192.61 | 2.91 | 4,154 | 276.70 | 154.09 |
| **Botswana** | 96.42 | 10.80 | 2,962 | 814.16 | 77.13 |
| **Burkina Faso** | 182.44 | 2.95 | 4.826 | 68.64 | 150.71 |
| **Cameroon** | 517.86 | 6.15 | 14.588 | 109.04 | 414.29 |
| **Cote d'Ivoire** | 421.03 | 4.93 | 10.263 | _ | 336.82 |
| **D. R. of Congo** | 737.09 | 3.03 | 16.355 | 25.03 | 608.91 |
| **Ethiopia** | 1,386.64 | 3.32 | 23.869 | 24.91 | 1,145.50 |
| **Ghana** | 354.02 | 3.16 | 6,973 | 114.76 | 292.45 |
| **Kenya** | 720.73 | 4.66 | 17,733 | 57.58 | 595.40 |
| **Lesotho** | 29.96 | 3.46 | 2,133 | 101.09 | 23.97 |
| **Madagascar** | 477.47 | 5.09 | 6,973 | 114.76 | 292.45 |
| **Malawi** | 363.94 | 5.63 | 12,776 | 31.27 | 300.65 |
| **Mozambique** | 305.05 | 3.14 | 11,325 | 36.82 | 252 |
| **Namibia** | 75.73 | 7.68 | 1,727 | 469.48 | 60.58 |
| **Niger** | 293.93 | 4.15 | 5,333 | 38.54 | 242.82 |
| **Nigeria** | 3,165.31 | 4.83 | 88.681 | 129.17 | 2,532.25 |
| **Senegal** | 160.11 | 3.26 | 2,430 | 109.52 | 132.27 |
| **South Africa** | 1,978.25 | 7.04 | 63, 061 | 695.06 | 1, 582.60 |
| **Swaziland** | 14.20 | 3.07 | 856 | 246.31 | 11.36 |
| **Tanzania** | 492.95 | 2.81 | 15,156 | 40.26 | 407.22 |
| **Uganda** | 319.73 | 2.85 | 11.296 | 83.61 | 150.71 |
| **Zambia** | 268 | 5.13 | 10, 535 | 124.96 | 221.39 |
| **Zimbabwe** | 568.68 | 9.75 | 29,987 | 55.58 | 469.79 |

IDF (2012: Online) estimated the prevalence of diabetes for South Africans aged between 20 to 79 years to be 7.04%. While the South African Demographic and Health Survey (SADHS) of 2003 confirmed that 6.5% of people older than 15 years have diabetes (Amod *et al.*, 2012: Online). SADHS also revealed that 50% to 85% of diabetes sufferers (especially in rural areas) remain undiagnosed (Amod *et al.*, 2012: Online). The high prevalence of diabetes in South Africa is linked to rapid cultural and social changes, aging populations, increasing urbanization, unhealthy eating and reduced physical activity (Amod *et al.*, 2012:Online).

### 2.3.2 Prevalence of Diabetes Mellitus in Lesotho

Lesotho's situation is no different from the rest of Africa, or from that of neighboring South Africa with respect to rapid cultural and social changes, an aging population, increasing urbanization, unhealthy lifestyles, high prevalence of obesity, and unhealthy behavioral patterns without prevention and control preparedness. According to the LRFS, 1.5 % of the population had diabetes (1.1% had diabetes and 0.4% had impaired glucose tolerance), while the prevalence rate in the capital city Maseru was at 1.9% (LRFS, 2001). The IDF Diabetes Atlas reports the prevalence of T2DM in Lesotho at 3.46%, with the highest rate occurring in the rural areas and females being affected most (IDF, 2012: Online).

Though Diabetes Mellitus has been generally discussed in the previous sections, the study is intended to focus mainly on T2DM; hence the following sections will only refer to T2DM.

## 2.4 Etiology and risks factors of T2DM

The literature has generally demonstrated the cause of chronic diseases including diabetes, to be the result of the underlying socio-economic status, cultural factors, political and environmental determinants such as globalization, urbanization and aging population (IDF, 2013b:23). T2DM is caused by the four basic defects, namely insulin-resistance, decreased insulin secretion, increased hepatic glucose production and reduced glucagon-like peptide-1 levels (Franz, 2012:679; ADA, 2013b:S67). Nonetheless, insulin resistance (which is caused by an insulin signaling defect, glucose transporter defect or lipotoxicity) and ß-cell dysfunction, believed to be caused by amyloid (a glycoprotein) deposition in the islets, oxidative stress, excess fatty acid or lack of incretin effect, were shown to be the major defects that drive T2DM (Taylor, 2013:Online). Figure 2.1 in the next page depicts the hypothesis of the etiology of T2DM in detail.

Taylor (2013: Online) define the etiology of diabetes (Figure 2.1) as follows: *"During long-term intake of more calories than are expended each day, any excess carbohydrate must undergo de novo lipogenesis, which particularly promotes fat accumulation in the liver. Because insulin stimulates de novo lipogenesis, individuals with a degree of insulin resistance (determined by family or lifestyle factors) will accumulate liver fat more readily than others because of higher plasma insulin levels. In turn, the increased liver fat will cause relative*

*resistance to insulin suppression of hepatic glucose production. Over many years, a modest increase in fasting plasma glucose level will stimulate increased basal insulin secretion rates to maintain euglycemia. The consequent hyperinsulinaemia will further increase the conversion of excess calories to liver fat. A cycle of hyperinsulinaemia and blunted suppression of hepatic glucose production becomes established. Fatty liver leads to increased export of VLDL triacylglycerol, which will increase fat delivery to all tissues, including the islets. This process is further stimulated by elevated plasma glucose levels. Excess fatty acid availability in the pancreatic islet would be expected to impair the acute insulin secretion in response to ingested food, and at a certain level of fatty acid exposure, postprandial hyperglycemia will supervene. The hyperglycemia will further increase insulin secretion rates, with consequent enhancement of hepatic lipogenesis, spinning the liver cycle faster and driving the pancreas cycle. Eventually, the fatty acid and glucose inhibitory effects on the islets reach a trigger level that leads to a relatively sudden onset of clinical diabetes"* (Taylor, 2013:Online).

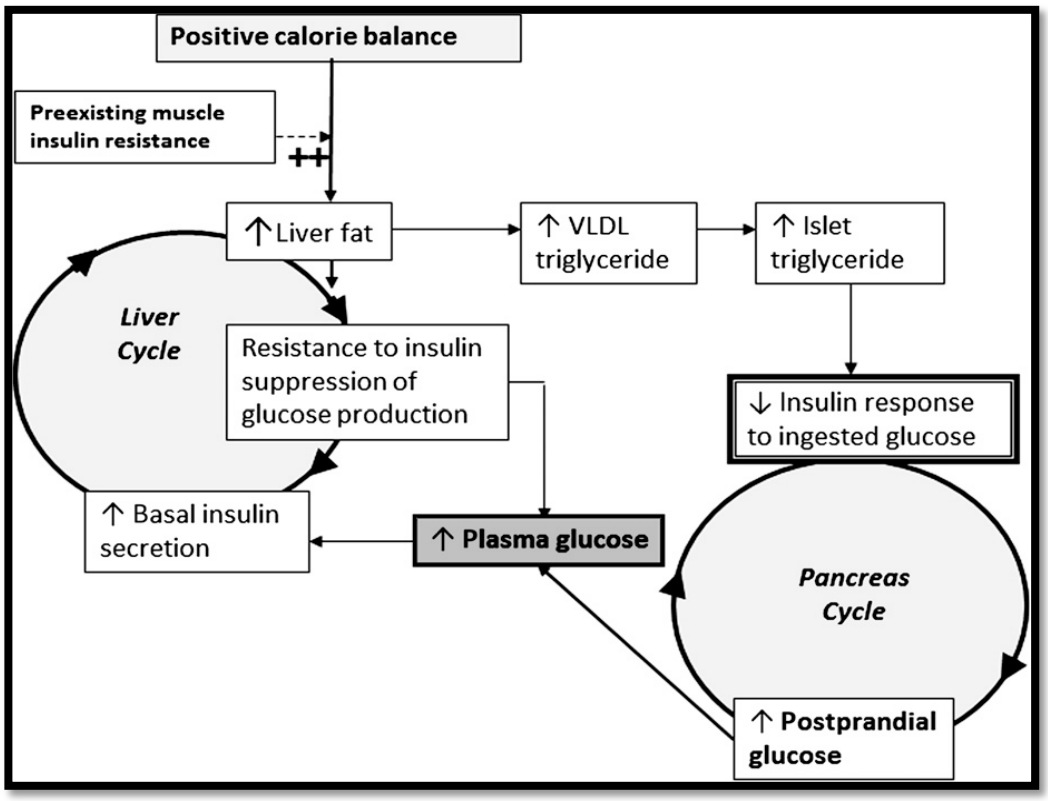


**Figure 2.1:** The twin cycle hypothesis of the etiology of type 2 diabetes (Taylor, 2013: Online).

Therefore, T2DM is shown to result from a collision between genes and environment, but genetic predisposition only establishes susceptibility, while rapid changes in the environment (for an example unhealthy diet and physical inactivity) are shown to be the main contributors for the increase in the incidence of diabetes (Tuomi *et al.*, 2013:Online). T2DM is common in both males and females, especially in populations (Asians) with the high diabetes prevalence, with the poor being as vulnerable as the rich (IDF, 2013b:16).

The risk factors of T2DM are divided into preventable and modifiable risk factors (for example, rapid cultural changes, increasing urbanization, Westernized diet, high energy intake, physical inactivity, unsafe alcohol intake, and tobacco use) (IDF, 2013b:16), and non-preventable and non-modifiable risk factors (for example, an aging population, ethnicity and genetic influence) (IDF, 2013b:22). However, in low-middle income countries the risk factors of T2DM are mainly associated with a family history, ethnicity, older age, gestational diabetes and lifestyle changes, which include obesity (especially intra-abdominal obesity), a poor or unhealthy diet, physical inactivity (IDF, 2013b:23; Franz, 2012:681; Miller, 2012:1838).

### 2.4.1 Family history of diabetes

T2DM is shown to be strongly inherited, but the susceptibility genes have not yet been identified (ADA, 2013b:S69; Qi *et al.*, 2009: Online), as many genes are shown to be implicated in the pathogenesis of T2DM (McCathy *et al.*, 2010: Online). The loci associated with obesity, pancreatic ß-cell dysfunction, decrease ß-cell mass, environmental mutations are also shown to be associated with the risk of developing T2DM (Tuomi *et al.*, 2013: Online; McCathy *et al.*, 2010: Online; Stitzel *et al.*, 2010: Online). McCathy *et al.*, (2010: Online) further indicated that the genes associated with T2DM in Europe might be different from the genes associated with T2DM in Africa. Though, it is believed that the genetic predisposition merely establishes susceptibility to T2DM (Tuomi *et al.*, 2013: Online).

The major genetic defects discovered in diabetes are of the ß-cell and the insulin action (ADA, 2013b:S69). There are three forms of diabetes identified under genetic defects of the ß-cell: the first form of diabetes is associated with the ß-cell function (characterized by impaired insulin secretion with minimal or no defects in insulin action) (ADA, 2013b:S69). The second form is associated with mutations in the glucokinase gene (glucokinase is an

enzyme responsible to convert glucose to glucose-6-phosphate, which eventually stimulates insulin secretion by the ß-cell), that is glucokinase serves as the glucose sensor for the ß-cell (ADA, 2013b:S69). The third form is due to the inability to convert proinsulin to insulin, though the resultant glucose intolerance is mild (ADA, 2013b:S69).

Kahn *et al.* (2013: Online) explained that the development of T2DM would occur only when genes for abnormal ß-cell function and body adiposity interact with environmental factors (high energy intake, nutrient composition, physical inactivity). Figure 2.2 illustrate the role of genes and the environment in the development of obesity and T2DM.

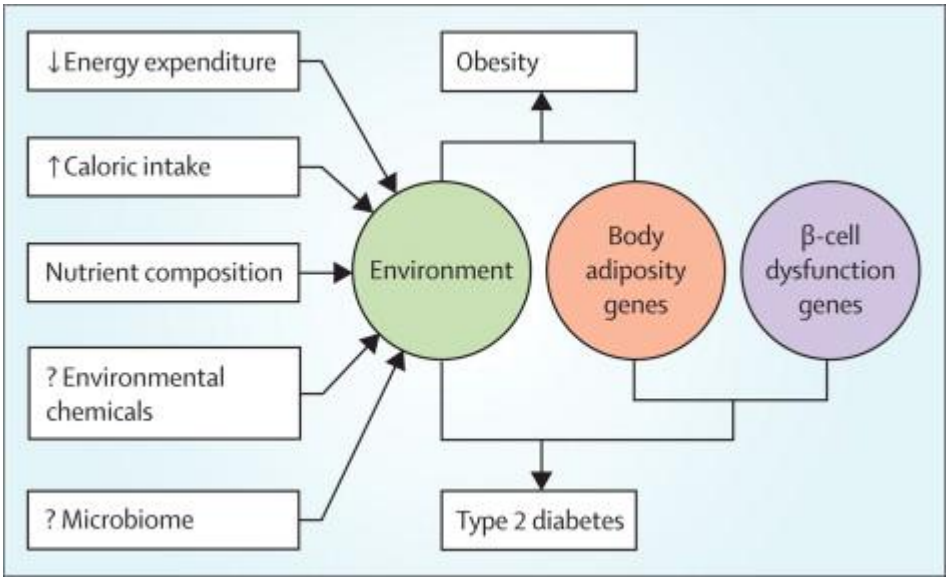


**Figure 2.2:** Role of genes and the environment in the development of obesity and Type 2 Diabetes Mellitus (Kahn *et al.*, 2013:Online, Figure 2).

The genetic defects in insulin action, is associated with mutations of the insulin receptor and may range from hyperinsulinaemia and modest hyperglycemia to severe diabetes, some individuals with these mutations may have acanthosis nigricans, females may present with enlarged cystic ovaries (ADA, 2013b:S69).

In the longitudinal cohort study performed by Li *et al.* (2011:777), it was suggested that Westernized dietary pattern may increase the diabetes risk, particularly in the genetically high-risk population groups (Li *et al.*, 2011:777; Qi *et al.*, 2009: Online). The Western dietary pattern, which is characterized by a high intake of red meat, processed meat products, high-fat dairy products, eggs, butter, and refined grains has been shown to cause insulin

resistance and risk of T2DM (Qi *et al.*, 2009:Online). In addition, cooking, preserving and processing of these food items result in the production of other chemicals that might have a toxic effect on ß-cells or induce insulin resistance (Qi *et al.*, 2009:Online).

Qi *et al.* (2009:Online) further demonstrated that the heme-iron found in red meat is the main nutrient that is found to interact with genetic variation, and result in the high iron stores in the body, which eventually may impair insulin sensitivity and glucose homeostasis. The interaction between genetic contribution with the environment as important risk factors for T2DM has long been recognized (Zimmet *et al.*, 2014: Online). There is evidence that "maternal and childhood epigenetic exposure and gene-expression changes" may also increase the risk of T2DM in later life (Kahn *et al.*, 2013: Online; Whiting *et al.*, 2011: Online).

### 2.4.2 Ethnicity

T2DM is more common in individuals with a family history of the disease and in members of certain racial/ethnic groups (Asians and African origins) (Whincup *et al.*, 2010:e1000263; Kahn *et al.*, 2013:62155). The Child Heart and Health Study in England (CHASE) study revealed that Asians and Africans have an increased risk of T2DM compared with white Europeans (Whincup *et al.*, 2010:e1000263). In population-based studies, Asians appeared to have raised plasma glucose, $HbA_{1c}$, and fasting insulin, lower HDL-cholesterol concentrations and low C-reactive protein levels, which is strongly associated with insulin resistance and T2DM (Whincup *et al.*, 2010:e1000263).

While in South Africa the Asian/Indian descent have a higher risk of T2DM, followed by the black people dwelling in urban areas, then the whites and coloured people (Amod *et al.*, 2012:83). Amod *et al.* (2012:83) further demonstrated that the Asian/Indian group become at risk even from as young as 10 years of age.

Furthermore, one study conducted at the University of the Witwatersrand, comparing groups of 8 to 15 black and white females, it was demonstrated that black females had significantly less visceral adipose tissue as compared to white females (Goedecke *et al.*, 2006:Online). However, despite the black females having less visceral fat, they presented with more insulin resistance (a cause of T2DM) than the white females (Goedecke *et al.*, 2006: Online).

### 2.4.3 Advancing age

The well-established public health services and an increased life expectancy in some countries have resulted in a great number of older people (60 years and above) (IDF, 2013b:102). This increase in life expectancy has contributed to an increase in the number of older people with diabetes (IDF, 2013b:102). Aging is associated with T2DM because it results in a decline in oxidative capacity and mitochondrial function (Taylor, 2013: Online), this may be caused by both genetic and environmental factors (Tuomi *et al.*, 2013: Online).

In addition, during aging there is a decrease in glucokinase (the main enzyme in the utilization of glucose by the liver) activity which is a common problem in patients with diabetes; this lead to insulin resistance in the liver (Ling & Groop, 2009:2720). Older patients with diabetes are at increased risk of having diabetes-related complications, and also often present with comorbid conditions (such as hypertension, physical disability, cognitive dysfunction, falls and fractures, depression, pressure sores, impaired vision and hearing problems) (IDF, 2013b:102).

According to IDF Atlas 6$^{th}$ edition, the estimated global prevalence of diabetes among people aged between 60 and 79 years is 18.6%, that is more than 134.6 million people, and this number is projected to increase to beyond 252.8 million by 2035 (IDF,2013b:102). IDF (2013b:58) reported that in Europe Region 37% of the population are over 50 years of age, and this is expected to increase to 44% by 2035. European adult population with diabetes is estimated to be 56.3 million (8.5%), and countries with the highest number of people with diabetes are in the Western part of Europe (Germany, Spain, Italy, France, and United Kingdom) (IDF, 2013b:58). The "South Africa burden of disease study" has estimated the prevalence of T2DM to be about 5.5% for people older than 30 years of age, and indicated that colored people above 60 years of age seem to be at increased risk (Amod *et al.*, 2012:83).

### 2.4.4 Obesity

Obesity is described as an imbalance between energy intake and energy output, such that the excess energy is stored as adipose tissue in the fat cells (Goedecke *et al.*, 2006:Online). Obesity is defined by the BMI of >30kg/m² according to the WHO criteria. Obesity has become more widespread, and is increasingly recognized as one of the global health problems, and the WHO estimated the global prevalence of obesity at 9.8% in 2005 (Ejima *et*

*al.*, 2013:17). Makinga & Beke (2013:194) indicated that the high prevalence of obesity might be associated with the following factors: genetic makeup, intrauterine and early life influences, parity, physical inactivity, poor dietary habits, low level of education, socio-cultural factors and stress.

Obesity serves as one of the most important risk factors of chronic diseases, including T2DM (Ejima *et al.*, 2013:17), and overweight and obesity are shown to contribute approximately 70% of diabetes cases (Krishnan *et al.*, 2012:466; WHO, 2011: Online). According to Goedecke *et al.* (2006:Online) obesity and T2DM are closely related in both females and males of all ethnic groups, and the risk of T2DM increases with the extent and duration of overweight and the degree of central adiposity (Goedecke *et al.*, 2006:Online).

In the Nurses' Health Study, which included 114 281 female nurses, the risk of diabetes was found to increase 40-fold when BMI increased from 22 to 35kg/m² (Goedecke *et al.*, 2006: Online). While the Health Professional Follow-Up Study that included 51,529 males, found that the relative risk of developing diabetes was 42-fold in males with BMI of >35kg/m² compared to BMI of < 23kg/m² (Goedecke *et al.*, 2006:Online). In addition, the government reviews in the UK, indicated that BMI >30kg/m² is associated with a relative risk of 12.7 for T2DM in females and 5.2 in males (Goedecke *et al.*, 2006: Online).

The Diabetes Prevention Program (DPP), which concentrated on reducing intake of fats and energy, resulted in weight loss averaging 7% at 6 months and maintenance of 5% weight loss at 3 years, and these was associated with a 58% reduction in incidence of T2DM (ADA, 2013a:23). While a Randomized Controlled Trial (RCT) indicated that the Mediterranean dietary pattern reduced the incidence of diabetes in the absence of weight loss by 52% compared to the low-fat control group (ADA, 2013a:23).

Though obesity is a well-recognized risk factor for T2DM, but a number of individuals who develop T2DM are not always obese, suggesting that in the absence of obesity, there are other independent risk factors (Krishnan *et al.*, 2012:466; WHO, 2011:Online; Bergman *et al.*, 2011:1085; Ling & Groop, 2009:2720). For instance Asian populations are generally less in overweight and obese than those in Western countries, but the Asian populations are still at a

higher risk of diabetes (Tuomi *et al.*, 2013:Online; Krishnan *et al.*, 2012:466; WHO, 2011:Online; Bergman *et al.*, 2011:1085).

Accumulation of fat in the abdominal area, particularly in the visceral compartment is associated with increased risk of insulin resistance, diabetes, hypertension, dyslipidaemia and atherosclerosis (Goedecke *et al.*, 2006: Online). Obesity-related fat accumulation in the liver plays a major role in triggering insulin resistance, and is also associated with prediabetes, implying that it is more harmful than the one accumulated in other parts of the body (Tuomi *et al.*, 2013:Online). However, the fat that is distributed to other parts of the body is shown to impair insulin secretion, hence it is the one that forms a link between obesity and T2DM (Tuomi *et al.*, 2013: Online).

The upper body obesity ("male" pattern or "android" pattern) is associated with increased insulin resistance and T2DM (Goedecke *et al.*, 2006: Online). In females, increasing WC is associated with greater metabolic risk, while other fat depot (such as in hips, thighs, upper arms, and breasts) appears to be relatively unimportant to metabolic disease risk (CDC, 2010: Online). Hence, the estimation of T2DM requires measurement of both the BMI and the WC (Feller *et al.*, 2010: 476).

### 2.4.5 Pregnancy-specific factors

Evidence from different studies has implicated a role of early-life exposures, maternal diet during pregnancy, postnatal growth, and childhood diet in chronic disease etiology (Malik *et al.*, 2011: Online). Thus Zimmet *et al.* (2014:Online) concluded that the first nine months shape the rest of one's life and early life programmes diseases such as diabetes and obesity. The "Dutch Hunger Winter Famine" birth cohort study, which took place in the late stages of the second World War serves as a good example on the cause of T2DM and other chronic disorders (Zimmet *et al.*, 2014:Online; Wang *et al.*, 2014:Online; Hales & Barker, 2013:Online). Hence, GDM is shown to have important implications for the prevention of T2DM and control of the diabetes epidemic (Zimmet *et al.*, 2014: Online).

#### 2.4.5.1 Foetal under-nutrition

The "Dutch Hunger Winter famine" birth cohort study of 1944-45 (at the end of the second World War in Netherlands), has shown an association between foetal malnutrition and

increased risk of diabetes in adult life, hence inadequate intrauterine nutrition (allocated rations were 1680 to 3360 kilojoules per day) was proven to result in low-birth weight (small-for-gestational age) and poor development of the central nervous system in infants (Zimmet *et al.*, 2014: Online).

This effects were found to be prevalent among individuals who were exposed to famine while in utero during the first and second trimesters, and not to those exposed during the third trimester (Hales and Barker, 2013:Online; Goedecke *et al.*, 2006:Online). James-Todd *et al.* (2013: Online) stated that low-birth weight seems to be early markers of subclinical risk of future development of T2DM, and may be a useful screening tool to identify high-risk females.

Hales & Barker (2013: Online) in their review indicated that there is a deficiency of beta cells (ß-cells) in T2DM, which result from abnormal supply of insulin during foetal growth. Evidence from other studies suggested that amino acids are major nutrients controlling ß-cell growth and development, as well as insulin secretion until late foetal life (Hales & Barker, 2013:Online). Insulin seems to determine foetal growth, while glucose has very little effect until late gestation (Hales & Barker, 2013: Online).

The risk of T2DM is shown to result from inadequate foetal nutrition and to be more prevalent among people exposed to overnutrition and rapid weight gain during the early life of adulthood (Zimmet *et al.*, 2014: Online). Hence, it is essential to focus on the importance of foetal nutrition during pregnancy, and after delivery, that is during lactation and the first few years of life (first 1000 days of the life) (Zimmet *et al.*, 2014: Online; Hales & Barker, 2013: Online). The "Dutch Hunger Winter famine" scenario demonstrated a potential effect that famines, natural disasters, and malnutrition can have on trends in T2DM many decades after the events (Zimmet *et al.*, 2014: Online). The current famine conditions (such as those in Africa due to wars and droughts), could result in similar scenarios in 30 to 40 years to come (Zimmet *et al.*, 2014: Online).

As long as an individual persists in the underfed state, there is no need to produce much insulin, but a sudden transition to good or over-nutrition seemed to expose individuals to the reduced state of ß-cell function and hence diabetes results (Wang *et al.*, 2014:Online; Hales &

Barker, 2013:Online). The similar situation was demonstrated by the Ethiopian Jews after moving to Israel among whom a high prevalence of diabetes was observed (Wang *et al.*, 2014: Online; Hales & Barker, 2013: Online). Another experience was also observed among the Nauruan Islanders, who suffered severe nutritional deficiency before and during the World War II, and after the war they became more affluent from phosphate mining, and diabetes became epidemic (Zimmet *et al.*, 2014:Online; Wang *et al.*, 2014:Online; Hales & Barker, 2013:Online).

However, amino acids deficiency does not only result in reduced ß-cell mass but also lead to abnormal islet structure and vascularisation, which would not be corrected by refeeding normally (the damage is irreversible) (Hales & Barker, 2013:Online). Thus, proteins and amino acids play a key role in maternal nutrition, but not excluding other nutritional defects (Hales & Barker, 2013: Online). In the study conducted in India, which investigated folate and vitamin $B_{12}$ (one of the feeders into methylation) levels during pregnancy and followed the newborn infants till the age of six years for indicators of insulin resistance, it showed that vitamin B12 deficiency could have a direct effect on gene function and towards development of diabetes.

In addition, the "Carnivore Connection Hypothesis" mean that during the human evolution there was a scarcity of dietary carbohydrates, which together with the high consumption of animal proteins resulted in insulin resistance (Wickert, 2012:Online). This hypothesis provided a survival and reproductive advantage as it redirected glucose from maternal use to foetal metabolism, as a result increasing the birthweight and survival of the infant (Wickert, 2012:Online). However, this was thought to have detrimental effect (high prevalence of insulin resistance and T2DM) particularly to those infants who are exposed to a high carbohydrate environment (Wickert, 2012: Online).

#### 2.4.5.2 Pre-term birth

Premature is defined as any birth occurring before 37 weeks of gestation (Wang *et al.*, 2014: Online). Preterm babies are shown to be at a high risk of infant mortality, childhood morbidity and have problems with neurodevelopment, such as hearing problems, and higher risk of other conditions (Wang *et al.*, 2014:Online). However, the link between being born preterm and metabolic disorders is still under recognized (Wang *et al.*, 2014: Online).

Infant born prematurely have higher insulin levels at birth and in early childhood, and this serves as an additional evidence that preterm birth may be a risk factor for the future development of insulin resistance and T2DM (Wang *et al.*, 2014:Online). Though premature births have been linked to T2DM in later life (during childhood, adolescence, and adulthood), it is suggested that insulin resistance exhibited by adolescents and adults born preterm may originate in utero and the developmental programming that occurs in low-birth weight births may be similar to the one for preterm births (Wang *et al.*, 2014:Online).

James-Todd *et al.* (2013:Online) explained that preterm births also result in mothers (9%) been at increased risk of developing T2DM, and their risk being evident at around 11 to 15 years after the first pregnancy. It is further suggested that preterm births seem to be early markers of subclinical risk of future development of T2DM, and may be a useful screening tool to identify high-risk women (James-Todd *et al.*, 2013:Online).

#### 2.4.5.3 Gestational diabetes

Gestational diabetes is a glucose intolerance of varying degrees of severity which starts during pregnancy (IDF, 2013b:23; ADA, 2013a:S11; Franz, 2012:679; Amod *et al.*, 2012:S6). Evidence from several studies has indicated that early life effects, especially during pregnancy could strongly affect the risk of T2DM in adult life (Zimmet *et al.*, 2014: Online), hence GDM is a well-established risk factor for T2DM in females, and it is believed that 70% of mothers who develop GDM will eventually have T2DM within 5 to 20 years after pregnancy (James-Todd *et al.*, 2013: Online; Osgood *et al.*, 2011: Online).

Intrauterine hyperglycemia is demonstrated to result in foetal hyperinsulinaemia, which eventually result in excessive weight gain (macrosomia), thus infants exposed to high levels of glucose, and high blood pressure during pregnancy are at increased risk of developing maternal T2DM in their adult life even in the absence of GDM (Zimmet *et al.*, 2014: Online; James-Todd *et al.*, 2013: Online; Osgood *et al.*, 2011: Online).

The abnormal glucose tolerance which occur during pregnancy eventually affect both the mother and their infants, and both will have an increased chance of developing obesity, metabolic syndrome and then T2DM in later life (Zimmet *et al.*, 2014: Online; Osgood *et al.*, 2011: Online). Nonetheless, breastfeeding seems to offer more protective benefits, as

lactation is shown to lower the risk of developing T2DM in both the mother and the child (Osgood *et al.*, 2011:Online). Lactation also increases metabolism, facilitates postpartum weight loss, and enhances glucose tolerance though it is on a short-term basis (Osgood *et al.*, 2011: Online).

### 2.4.6 Unhealthy dietary/eating pattern

Dietary/eating pattern is a term used to describe combinations of different foods or food groups that characterize relationship between nutrition and health promotion, and disease prevention (Evert *et al.*, 2013: Online). There are factors that impact eating patterns, and include, but are not limited to: food access or availability of healthful foods, tradition, cultural food systems, health beliefs, knowledge of foods that promote health and prevent disease, and economics/resources to buy health promoting foods (Evert *et al.*, 2013:Online).

Dietary intake is an important modifiable environmental risk factor in the development and prevention of T2DM (Naja *et al.*, 2012: Online). It is evident that dietary patterns may exert greater effects on health than individual foods, nutrients, or food groups (Naja *et al.*, 2012: Online). According to Samieri *et al.* (2013: Online), if one improves the quality of the diet at midlife, that would result in good health and well-being during elderly.

#### 2.4.6.1 Westernized diet

The nutrition transition of the rural population has contributed to increased adoption of a more westernized diet, which is higher in fat and carbohydrates (Goedecke *et al.*, 2005: Online). The Western dietary pattern, which is characterized by a high intake of red meat, processed meat products, high-fat dairy products, eggs, butter, and refined grains has been shown to cause insulin resistance and T2DM (Maghsoudi & Azadbakht, 2012:Online; Naja *et al.*, 2012:Online; Qi *et al.*, 2009:Online).

The excess consumption of high energy nutrient-poor foods, high-GI foods, and sugar sweetened beverages are associated with an increased risk of T2DM (ADA, 2013a:S23; Naja *et al.*, 2012: Online; Krishnan *et al.*, 2010:467). High energy intake as well as an increased consumption of sugar-sweetened sodas and fruit drinks, a low intake of fiber, and a high intake of foods with a high glycemic load (refers to the amount of carbohydrate in addition to

its glycemic index) are preventable risk factors that increase the risk of diabetes (ADA, 2013a:S23; Naja *et al.*, 2012: Online).

According to Weickert (2012:Online), it is believed that dietary fiber intake and reduction in diabetes risk are due to the gel-forming properties of soluble fiber from fruits and vegetables, and the beneficial metabolic effects of short-chain fatty acids (SCFA) derived from the colonic fermentation of non-digested fiber by the gut microorganisms. Even the meta-analysis of large US prospective cohort studies showed a reduced diabetes risk with high cereal-fibre intake, but not with fruits or vegetables intake (Weickert, 2012: Online). The improved insulin resistance that result from the intake of high cereal-fibre, is postulated by the fact that cereal fibre hinder the digestion and absorption of dietary protein in the upper gut, thus preventing amino acid-induced insulin resistance (Weickert, 2012:Online).

In Western dietary pattern there is an increased intake of red meat and processed meats, which are major sources of proteins and fats, hence associated with cardiovascular diseases and diabetes (Pan *et al.*, 2011:Online). There are several mechanisms that explain the effects of red meat on T2DM, the first one is the heme-iron (Pan *et al.*, 2011: Online). Iron (from red meat) is a strong pro-oxidant that catalyses several cellular reactions in the production of reactive oxygen species, thus increases the level of oxidative stress (Ajala *et al.*, 2013: Online; Pan *et al.*, 2011: Online). Production of oxidants can cause damage to tissues, particularly the pancreatic beta cells, hence high concentrations of iron in the body has been associated with an elevated risk of T2DM (Ajala *et al.*, 2013:Online; Pan *et al.*, 2011:Online).

The second association may be through the presence of sodium, nitrates and nitrites, which are found in red meat and processed meats (Ajala *et al.*, 2013: Online; Pan *et al.*, 2011: Online). These compounds are converted to nitrosamines in the stomach or within the food product, and nitrosamines are extremely toxic to the pancreatic beta cells resulting in endothelial dysfunction and impaired insulin response (Ajala *et al.*, 2013: Online; Pan *et al.*, 2011: Online).

Another contributing factor may be due to advanced glycation end-products or increased concentrations of inflammatory mediators and gamma-glutamyltransferase with high intake of red meat (Pan *et al.*, 2011: Online). Lastly, the association may be due to weight gain and

obesity, as red meat intake is linked to weight gain, for example, a red meat intake of 250g per day would result in 2kg weight gain after a period of five years (Pan *et al.*, 2011: Online).

The cohort study conducted in Lebanon revealed that adherence to a Western dietary pattern during adolescence was associated with 29% increase in the risk of T2DM in middle aged women (Naja *et al.*, 2012: Online). In addition, the National Nutrition Survey conducted in Lebanon in 2009, further indicated that Western dietary pattern, metabolic syndrome, and obesity are associated with increased risk of T2DM (Naja *et al.*, 2012: Online).

#### 2.4.6.2 Fast-foods

Fast-foods which are characterized by high intakes of chips and fries, sandwiches, full cream milk and milk products, and added fats were correlated mainly with high energy and fat intake, therefore may increase the risk of T2DM (Naja *et al.*, 2012: Online). Foods eaten from the restaurants tend to have a higher energy density than foods eaten at home; portion sizes are larger and result in increased intake of energy, saturated and trans-fats, sodium, and carbonated beverages and lower intakes of fruits and vegetables, and milk (examples are foods such as burgers, fried chicken, and chips) (Krishnan *et al.*,2010:467).

Fast-foods contain hydrogenated oils, which may result in insulin resistance, rapid weight gain and obesity (Krishnan *et al.*, 2010:467). Fast-foods patterns possess a high-GI as well as a high glycemic load, and such foods have been shown to have a direct association with T2DM (ADA, 2013a:S23; Naja *et al.*, 2012: Online). Foods with a high glycemic load are shown to trigger higher postprandial insulin levels, which may result in age related decline in insulin secretion and eventually lead to the onset of T2DM (ADA, 2013a:S23; Naja *et al.*, 2012:Online).

The association between glycemic load and T2DM risk is moderate to strong, depending on the prevailing circumstance (Livesey *et al.*, 2013: Online, Rossi *et al.*, 2000: Online). People who consume diets of more than 100g of glycemic load per 2000kcal (8400kJ), appear to be at greater risk of T2DM with greater glycemic load intake (Livesey *et al.*, 2013: Online). Therefore, the meta-analysis supported that glycemic load is an important and underestimated dietary characteristic that contributes to the incidence of T2DM (Livesey *et al.*, 2013: Online).

### 2.4.6.3 High sugar intake

The consumption of sugar (in the form of soft drinks and fruit drinks) has risen tremendously, and is believed to be more than 50-fold higher than in 1800s (Malik *et al.*, 2010: Online; Bray, 2010: Online). The introduction of high fructose corn syrup (HFCS), which is made by converting glucose into fructose using grown bacteria and then diluting the fructose to provide the commercially available HFCS solutions that have 55% fructose (Bray, 2010:Online). Most soft drinks and many other foods are sweetened with this product because it is very cheap and has useful manufacturing properties (Malik *et al.*, 2010: Online; Rizkallan, 2010:82). Steyn & Temple (2012:502) revealed that one can (330ml) of carbonated sugar sweetened beverages (SSB) (soft drink) provides 40-50g of sugar and 630kJ, in the form of high fructose corn syrup.

This high content of rapidly absorbable carbohydrates such as sucrose (50% glucose and 50% fructose) and HFCS (most often 45% glucose and 55% fructose), with the large volumes of beverages (>1 can per day) consumed may increase the risk of metabolic syndrome and T2DM, not only through obesity but also by increasing dietary glycemic load, leading to insulin resistance, cell dysfunction, and inflammation (Naja *et al.*, 2012: Online; Malik *et al.*, 2010: Online). Feinman and Fine (2013:45) showed that high fructose intake is associated to insulin resistance and metabolic syndrome.

In a meta-analysis of eight prospective cohort studies, a diet high in consumption of SSBs was associated with the development of T2DM, and individuals in the highest quintile of SSBs intake had a 26% greater risk of developing diabetes (ADA, 2013a:S23). Mann and Morenga (2013:Online) demonstrated that high-GI foods and dietary glycemic load without proper advice on food choices could result in substantial intake of foods that are high in sugars (including high-fructose corn syrup) and/or fats, and that are high in energy. Table 2.3 lists ingredients that consumers can use to identify sugars.

**Table 2.3: Ingredients on food labels consumers can use to identify sugars (Academy of Nutrition and Dietetics, 2012)**

| | |
|---|---|
| Anhydrous dextrose | Malt syrup |
| Confectioner's powdered corn syrup | Maltose |
| Corn syrup | Maple syrup |
| Corn syrup solids | Molasses |
| Dextrose | Nectors (e.g. peach nector, pear nector) |
| Fructose | Pancake syrup |
| High-fructose corn syrup | Raw syrup |
| Honey | Sucrose |
| Invert sugar | White granulated sugar |
| Lactose | |

The Nurses Health Study found that females who had one or more SSB drinks per day over a period of four years gained more weight than those who decreased their intake of SSBs (Naja *et al.*, 2012:Online). Females who had one or more SSBs had a relative risk of T2DM of 1.83 compared with those who consumed less than one drink a day, while sweetened fruit juices had a relative risk of two (Naja *et al.*, 2012:Online).

The American Heart Association's Diet and Lifestyle Recommendations stated that females should eat or drink no more than 420kJ per day (25g or 6tsp) from added sugars, and males should eat or drink no more than 630kJ per day (38g or 10tsp) from added sugars. While the WHO Global Strategy on Diet, Physical Activity and Health, endorsed at the 57$^{th}$ World Health Assembly recommended that 10% of energy could be provided by added sugars. The current sugar guideline for South Africa is to use foods and drinks that contain sugar sparingly and not between meals (Steyn & Temple, 2012:502).

### 2.4.7 Physical inactivity

Physical activity is the "expenditure of energy above that of resting by the contraction of skeletal muscle to produce bodily movement", while exercise is a "a type of physical activity that involves planned, structured and repetitive bodily movement performed for the purpose of improving physical fitness" (De Feo & Schwarz, 2013: Online; Franz, 2012:688; Hovanec *et al.*, 2012: Online). Physical activity has declined steadily since 1960s, reflecting declining work related activity, increasing sedentary activity (for an example more time is spent using computers or watching television), increased vehicle travelling from home to work, and increases in vehicle use versus walking or public transit (Laverty *et al.*, 2013: Online).

Poor physical fitness and low $VO_2$max (the maximum or optimum rate at which the heart, lungs, and muscles can effectively use oxygen during exercise, used as a way of measuring a person's individual aerobic capacity) predict risk of developing T2DM (Roden, 2012: Online). Mitochodrial dysfunction, changes in muscle fibre-type composition, and insulin resistance are thought to be potential mechanisms linking poor physical fitness with an increased risk for T2DM (Roden, 2012: Online). Physical activity is also postulated to induce the expression of a number of genes that regulate glucose uptake in skeletal muscle (Roden, 2012: Online).

Adequate physical activity is associated with important health outcomes, including reductions in cardiovascular disease, T2DM, weight management, improvements in physical function, and improved quality of life (Conn *et al.*, 2011:Online; CDC, 2011:Online; Wilde, 2009:368), and a reduction of 27% in the risk of diabetes (Lachat *et al.*, 2013:Online). The American College of Sports Medicine (ACSM) and the ADA recommended that 150 minutes per week of moderate/vigorous physical activity should be undertaken to prevent or delay the development of T2DM (Roden, 2012:Online).

Lachat *et al.* (2013:Online) from their investigations concluded that increased rates of walking and cycling were associated with a higher number of adults who achieved the recommended levels of physical activity, a lower percentage of adults with obesity, and a lower percentage of adults with diabetes. The research suggested that even 30 minutes per day of moderate-intensity physical activity, if performed regularly contribute to significant health benefits (Lachat *et al.*, 2013:Online). While Laine *et al.*( 2013:Online), hypothesized that vigorous physical activity during young adulthood protects from disturbances in glucose regulation in later life, and the study further proved that a career as an elite athlete protects one against T2DM in later life.

The "Understanding Society", a national representative survey of United Kingdom residents of 2009/2011, which investigated the differences among socio-demographic groups in their use of active travel, and whether active travel to work was associated with overweight/obese, diabetes or hypertension (Laverty *et al.*, 2013: Online). The study showed an association between active travel to work and a reduced likelihood of being overweight, having diabetes, and having hypertension (Laverty *et al.*, 2013: Online). Hence, the study advocated for active travel, and that it should be included and prioritized within national and local prevention strategies for obesity, diabetes , and cardiovascular disease (Laverty *et al.*, 2013: Online).

The 2008 Physical Activity Guidelines for Americans indicated that moderate levels of activity, however achieved, are associated with a 30-40% decreased risk of developing T2DM and metabolic syndrome (Roden, 2012: Online).

### 2.4.8 Other risk factors

Other risk factors include metabolic syndrome, HIV/AIDS, use of tobacco and alcohol, lack of sleep, psychosocial and work stress, climate changes and certain environmental exposures.

#### 2.4.8.1 Metabolic Syndrome

Metabolic syndrome is a group of interrelated risk factors for cardiovascular, which include hypertension, dyslipidaemia (high levels of triglycerides, low levels of high-density lipoprotein cholesterol, high levels of low-density lipoprotein cholesterol), obesity (especially abdominal) and the presence of insulin resistance as a linking factor (Tuomi *et al.*, 2013: Online; Amod *et al.*, 2012:S57; Alberti *et al.*, 2009: Online). The features and targets of metabolic syndrome are shown in Table 2.4.

**Table 2.4: The features and target for metabolic syndromes (Amod *et al.*, 2012:S57, Table I)**

| Features | Targets |
|---|---|
| Total cholesterol | < 4.5 mmol/l |
| LDL cholesterol<br>HDL cholesterol | < 1.8 mmol/l<br>> 1.0 mmol/l (Males)<br>> 1.2 mmol/l (Females) |
| Triglycerides | < 1.7 mmol/l |
| Waist Circumference | < 94 cm (Males)<br>< 80 cm (Females) |
| Body Mass Index | < 25kg/$m^2$ |
| Blood pressure: Systolic<br>Diastolic | < 140 mmHg<br>< 80 mmHg |
| Fasting blood glucose | ≥ 5.6 mmol/l |

Other contributing features may include the polycystic ovarian syndrome, non-alcoholic fatty liver disease and sleep apnea (Amod *et al.*, 2012:S57). According to the WHO, the criteria for diagnosing the metabolic syndrome could be based on markers of insulin resistance including the two additional risk factors, either obesity, hypertension, high triglycerides levels, reduced high-density lipoprotein cholesterol level, or microalbuminuria (Alberti *et al.*, 2009:Online).

Insulin is produced in the pancreas by the islets of langerhans of the beta cells (IDF, 2013b:27). Insulin transports glucose into the cells and directs the storage of excess nutrients in the form of glycogen, triglycerides, and proteins (IDF, 2013b:27). The major tissue targets of insulin are muscle, liver, and adipose tissue. Insulin is secreted in response to a number of factors (increased blood glucose, amino acids, fatty acids, gastrointestinal hormones, and

other hormones), but most importantly increased blood glucose, and it promotes the uptake of glucose from the blood into the cells thereby decreasing blood glucose (IDF, 2013b:27).
Insulin resistance means that the body is able to produce insulin, but either this is not sufficient or the body is unable to respond to its effects, resulting in a build-up of glucose in the blood (IDF, 2013b:27). Insulin resistance occurs when there is an abnormal signaling defect of the insulin and glucose transporter defects or lipotoxicity (defect in mitochondrial function is associated with extremes of insulin resistance in skeletal muscle) (Taylor, 2013: Online; Tuomi *et al.*, 2013: Online), and high fat content of the viscera and the liver (Tuomi *et al.*, 2013: Online). Hence, fat accumulation in the liver serves as an important trigger of insulin resistance (Tuomi *et al.*, 2013: Online).

Metabolic syndrome is best managed by lifestyle change (diet adjustment, weight loss and regular exercise) as well as insulin sensitizers', such as metformin (Amod *et al.*, 2012:S57). Lifestyle change can delay, or even prevent the onset of T2DM in patients with the metabolic syndrome (Amod *et al.*, 2012:S57).

#### 2.4.8.2 HIV and AIDS

Patients with human immunodeficiency virus (HIV) infection who are on antiretroviral therapy (ART) experience chronic metabolic complications (insulin resistance, uncontrolled blood glucose, dyslipidaemia, lipodystrophy, and accelerated atherosclerosis) due to HIV itself, but mainly as a result of iatrogenic factors (for example use of ARTs particularly protease inhibitors, and drugs used to treat opportunistic diseases) (Parikh *et al.*, 2013: Online; Amod *et al.*, 2012:S85; Levitt *et al.*, 2011: Online). Therefore, the risk factors for development of diabetes in HIV include: HIV virus itself (viral load, CD4 count, and duration of disease), rapid weight gain after the catabolic phase, co-infection with hepatits C, dyslipidemia with lipotoxicity, lipodystrophy and iatrogenic factors (Amod *et al.*, 2012:S85; Levitt *et al.*, 2011: Online).

The mechanism on how HIV, fluctuating viral load, and ARTs could result in diabetes is as follows: it is postulated that HIV induces inflammatory state, resulting in an increase in cytokines and a decrease in adiponectin levels, which may eventually induce insulin resistance (Amod *et al.*, 2012:S85; Levitt *et al.*, 2011: Online). While nucleoside reverse transcriptase inhibitors cause mitochondrial toxicity, lipodystrophy and pancreatitis, which

result in insulin resistance, and protease inhibitors induce insulin resistance by lipodystrophy, impaired glucose transporter type 4 translocation, reduced adipocyte differentiation, reduced insulin secretion and dyslipidaemia with lipotoxicity (Amod *et al.*, 2012:S85).

The mechanism of how protease inhibitors induce lipodystrophy is not yet known, but some studies have reported that up to 83% of patients on protease inhibitors develop lipodystrophy, and 35% of those eventually develop diabetes (Amod *et al.*, 2012:S85).

#### 2.4.8.3 Harmful use of alcohol

There is a growing consensus that alcohol consumption is not one of the influencing factors of diabetes (Baliunas *et al.*, 2009: Online). There is not enough evidence as to how alcohol is not entirely a risk factor of diabetes, but there are several factors that may explain the relationship, which include increases in insulin sensitivity after moderate alcohol consumption, changes in levels of alcohol metabolites, and increases in HDL cholesterol concentrations, or via the anti-inflammatory effect of alcohol (Baliunas *et al.*, 2009: Online). The U-shaped relationship is observed between average amount of alcohol consumed per day and risk of incident T2DM among females and males (Heianza *et al.*, 2013: Online; Baliunas *et al.*, 2009: Online).

A moderate alcohol intake has been linked to a lower risk of developing T2DM, while heavy alcohol consumption has been associated with a risk of developing diabetes (Heianza *et al.*, 2013:Online). The NHANES cross-sectional study demonstrated that males who used to consume more or equal to three (≥ 3) drinks at one occasion developed cardiometabolic factors, regardless of drinking frequency over a period of one week (Heianza *et al.*, 2013:Online). However, studies that investigated the effect of binge-drinking habits (≥3 drinks over a short period) on the risk of developing diabetes showed inconsistent results (Heianza *et al.*, 2013: Online).

Short-term randomized clinical trials done on postmenopausal females reported that moderate alcohol consumption improved insulin sensitivity and lowered triglyceride concentration (Heianza *et al.*, 2013: Online). While, the study done on Japanese males reported that those with light-to-moderate alcohol consumption (4-7 drinks/week) had a lower risk of diabetes than did the nondrinking males (Heianza *et al.*, 2013: Online). These results suggested that

high alcohol consumption on one drinking occasion significantly increased the risk of diabetes regardless of frequency of drinking (Heianza *et al.*, 2013: Online).

A more protective effect of moderate consumption was found in females, and it is recommended that alcohol intake be limited to moderate amounts and heavy consumption should be discouraged (Baliunas *et al.*, 2009: Online). According to Crandall *et al.*(2009: Online) the moderate (one to three drinks per day) consumption of alcohol is associated with reduction by 33-56% in diabetes in both females and males, and the risk with high and chronic consumption is not clearly explained (Heianza *et al.*, 2013: Online; Crandall *et al.*, 2009: Online).

#### 2.4.8.4 Smoking

Smoking may have a role in the development of T2DM (CDC, 2011: Online). Cigarette smoking is linked to increased insulin resistance and to the risk for developing T2DM by 44%, though in some studies the results were inconsistent (Nyamdorj, 2010:21). Furthermore, smoking increase the risk of cardiovascular complications in patients with diabetes and hypertension (Raz, 2009: Online).

Smoking, particularly during adolescence is associated with overweight and abdominal obesity during young adulthood, of which are risk factors for T2DM and cardiovascular diseases (Saami *et al.*, 2009: Online). This association of overweight and abdominal obesity with smoking may come about as a result of changes in glucocorticoids metabolism, and psychosocial stress (Saami *et al.*, 2009: Online).

The Surgeon General's report of 2014, indicated that smoking can cause T2DM , and the risk of developing diabetes is as high as 30-40%, and is higher for active smokers than non-smokers (CDC, 2014: Online). Furthermore, the risk of developing diabetes increases as the number of cigarettes smoked increased (CDC, 2014: Online). In addition the use of smokeless tobacco (snuff, taken in an unburnt form through chewing or sniffing) contains several carcinogens, and has been associated with oral cancer, hypertension, heart disease, T2DM, and other diseases (Lee & Hamling, 2009: Online).

#### 2.4.8.5 Lack of adequate sleep

The daily experience of sleep is critical to "setting" the clock, hence inadequate sleep (≤5 hours per night) time is of public health concern (Boyko *et al.*, 2013: Online). Sleep deprivation (troubled sleep, short sleep, sleep apnea) are known to have behavioural consequences such as decreased alertness, accidents, emotional disturbances (Boyko *et al.*, 2013: Online). Short sleep duration is associated with overweight and obesity and with the risk of developing diabetes (Boyko *et al.*, 2013: Online). Recent research in humans indicates that sleep disturbances also play a causal role in generating or exacerbating problems of energy balance and insulin function (Boyko *et al.*, 2013: Online).

#### 2.4.8.6 Psychosocial and work stress

"Burnout", which is defined as an experience of physical, emotional, and mental exhaustion caused by long-term involvement in situations that are emotionally demanding, may have some effects on physical health, including diseases like T2DM and cardiovascular diseases (Eriksson *et al.*, 2013:Online). In addition years of rotating night-shift work has been associated with a modestly increased risk of T2DM (Eriksson *et al.*, 2013: Online).

Chronic and acute psychosocial stress is shown to be associated with obesity and morbidity of various chronic diseases (T2DM) (Lee, 2010: Online). During the 17$^{th}$ century, physicians used to associate diabetes to prolonged sorrow, which is called psychosocial stress in today's language (Heraclides *et al.*, 2009: Online). First, psychosocial stress has been linked to increased glucose levels and glucose intolerance among diabetes patients. Secondly, psychosocial stress has been linked to obesity and metabolic syndrome, which are well known risks factors of T2DM (Heraclides *et al.*, 2009: Online).

Heraclides *et al.* (2009: Online) also indicated that females exposed to high job demands and low job control (job strain) had a higher risk of T2DM compared with those not exposed to the combination of work stressors, and females exposed to a low work social support (iso-strain) had a two-fold risk of developing T2DM (Heraclides *et al.*, 2009: Online). While other studies (for example the Nurses' Health Study II) revealed that job strain was not associated with incident T2DM, but working overtime was associated with increased risk for T2DM, though the mechanisms underlying this relationship were not clearly defined (Eriksson *et al.*, 2013: Online; Heraclides *et al.*, 2009: Online).

It is postulated that shift work result in disturbed sleep, which is believed to increase the risk of T2DM, as it interferes with the normal synchrony between the light-dark cycle, sleeping, and eating, which may eventually alter the functioning of the endocrine system (Eriksson *et al.*, 2013: Online; Heraclides *et al.*, 2009: Online) This relationship between shift work and sleep disturbances, insulin resistance, and T2DM may be mediated through adiposity or weight gain influenced by the alteration of the hormone systems or changed dietary and exercise patterns (Eriksson *et al.*, 2013: Online), thus sleep deprivation may be indirectly linked to chronic diseases (Lee, 2010: Online). Hence, work stress is associated with the development of T2DM, particularly in females, but it is unclear whether this is due to the work situation or an accumulation effect from work stress and stress in the home situation (Eriksson *et al.*, 2013: Online).

#### 2.4.8.7 Climate changes

According to IDF (2013: Online), 25% of the global burden of diseases have close ties to environmental factors. Clean water and sanitation are two environmental factors that have an association with mortality and morbidity rates, and without access to safe water and minimum sanitation, the health of the people would be at risk (IDF, 2013: Online). Climate change (cold weather, heat waves, and increased seasonal temperature variations) affects blood vessel-related heart disease, dyslipidaemia and hypertension which are the comorbid conditions found in T2DM patients (McMichael, 2013: Online; Millett *et al.*, 2013: Online; Liao *et al.* 2010:4252)**.**

In general, a healthy individual will be able to cope with thermal stress through the efficient regulation of heat (Liao *et al.* 2010:4252). The changes in blood pressure, blood viscosity, and heart rate associated with physiological adjustments to cold and warmth, may explain the increase in mortality due to diseases of the cardiovascular system (a major cause of death in people with diabetes) (McMichael, 2013: Online; Liao *et al.* 2010:4252). Studies have shown that human health is significantly affected by the level of and variation in climate-related variables, since the risk of death will be increased if the changes of temperature exceed the limits that the cardiovascular system can withstand (Liao *et al.* 2010:4252).

#### 2.4.8.8 Environmental exposures

Not enough research has been done in regard to the relationship between other environmental exposures (arsenic, maternal smoking during pregnancy, organic tin compounds, bisphenol A, pesticides, vehicle fumes, smoke from burning wood, coal or tyres) and T2DM (Krishnan *et al.*, 2012:120).

## 2.5 Diagnosis of T2DM

In 2009, an international Expert Committee that included representatives of ADA, the IDF, and the European Association for the Study of Diabetes (EASD) recommended the use of the HbA1c test to diagnose diabetes, with the threshold of ≥ 6.5%, the new criteria was adopted by ADA in 2010 (ADA, 2013a:S13). It is recommended that the diagnosis of diabetes if done for clinical purposes be confirmed with the repeated test (preferably the same test) on another day, except in the presence of hyperglycemia with acute metabolic decompensation or obvious classic symptoms of hyperglycemia (such as polyuria, polydipsia and weight loss) (Amod *et al.*, 2012:7). Severe hyperglycemia detected under conditions of acute infective, traumatic, cardiovascular or other stress, may be transitory and should not be regarded as diagnostic of diabetes until confirmed subsequently (Amod *et al.*, 2012:8).

The diagnosis of diabetes should be based on the formal laboratory testing, not on point-of-care or bedside instruments (for an example glucose reflectance meters or single-use HbA1c kits), and venous plasma glucose reading is preferred to capillary blood glucose (Amod *et al.*, 2012:7). When using the capillary blood glucose measurements, the plasma glucose value will need to be derived with the following conversion factor: Plasma glucose (mmol/l) = 0.102 + 1.066 × capillary blood glucose (Amod *et al.*, 2012:7).

**The WHO criteria for the diagnosis of diabetes are as follows:**

HbA1c of ≥ 6.5% (the test should be performed in a laboratory using a method that is National Glycohemoglobin Standardization Program (NGSP) certified and standardized to the Diabetes Control and Complications Trial (DCCT) assay (ADA, 2013a:S12, S13; ADA, 2013b:S72 Amod *et al.*, 2012:7), and there should be no conditions present which preclude its accurate measurements (Amod *et al.*, 2012:7)

**Or**

Fasting plasma glucose (FPG) ≥ 7.0 mmol/l (fasting is defined as no energy intake for at least 8 hours) (IDF, 2013b:109; ADA, 2013a:S13, Amod *et al.*, and 2012:7)

**Or**

2-hours plasma glucose ≥ 11.1 mmol/l during an OGTT (the test should be performed as described by the WHO, using a glucose load containing the equivalent of 75g anhydrous glucose dissolved in 250 ml water ingested over five minutes (IDF, 2013b:109; ADA, 2013a:S13, Amod *et al.*, 2012:7)

**Or**

Random plasma glucose of ≥11.1 mmol/l (in patient with classic symptoms of hyperglycemia or hyperglycemic crisis) (IDF, 2013b:109; ADA, 2013a:S13, Amod *et al.*, and 2012:7). Random means casual, and refers to any time of the day, without regard to time of last meal. The classic symptoms of hyperglycemia include polyuria, polydypsia, and weight loss, while hyperglycemic crisis refers to diabetic ketoacidosis, hyperosmolar nonketotic hyperglycemia (Amod *et al.*, 2012:7).

Advantages of $HbA1_c$ include: it has a greater convenience (since fasting is not required) to the FPG and OGTT, has less day-to-day perturbations during periods of stress and illness, and has a greater preanalytical stability (ADA, 2013a:S19; Franz, 2012:681).

Disadvantages of $HbA_{1c}$ are: it is expensive, there is limited availability of $HbA_{1c}$ testing in certain regions of the developing world, there is incomplete correlation between $HbA_{1c}$ and average glucose in certain individuals, $HbA_{1c}$ levels may vary with patients' race/ethnicity (ADA, 2013a:S19).

The $HbA_{1c}$ is subject to certain limitations: for conditions that affect erythrocytes turnover (for an example anaemia from haemolysis, iron deficiency or blood loss, and pregnancy) (ADA, 2013a:S18; Franz, 2012:681), haemoglobin variants must be considered, and $HbA_{1c}$ does not provide a measure of glycemic variability or hypoglycemia (ADA, 2013a:S18).

The WHO consultation on the use of $HbA_{1c}$ in the diagnosis of diabetes mellitus, however, has conditions that must be met before $HbA_{1c}$ can be used to diagnose diabetes mellitus and include: the test method used must meet stringent quality assurance criteria, the assay must be standardized to criteria aligned with international reference values (NGSP certified), the assay

must be standardized to the DCCT assay, and there must be no conditions that preclude the accurate measurement of $HbA_{1c}$ (IDF, 2012:9; Amod *et al.*, 2012:7).

## 2.6 Management of T2DM

According to Evert *et al.* (2013: Online), the main components of diabetes management involve a Diabetes Self-Management Education (DSME), Self-Monitoring Blood Glucose (SMBG), healthful eating pattern, regular physical activity, and often medication. The goals of diabetes management are to: relief symptoms, achieve healthy eating habit and normal physical activity, as well as achievement and/or maintenance of normal body weight (BMI of 20-25 kg/m²), fasting plasma glucose levels between 4.0mmol/l-7.0mmol/l, 2-hr postprandial plasma glucose ≤ 11.1mmol/l, and $HbA_{1c}$ levels ≤ 7% (Amod *et al.*, 2012: S7). The overall aim of diabetes management is to achieve and maintain as near normal metabolic control (glycemic control, lipids, and blood pressure) as is practicable through lifestyle modifications (ADA, 2013a: S16; Evert *et al.* 2013: Online).

In order to manage diabetes patients more effectively and efficiently, Amod *et al.*, (2012:10) recommended inclusion of the following, (as summarized in Table 2.5 below): well trained and dedicated personnel, calibrated and functioning equipment, management and referral protocols, continuous supply of medication, a register of all patients to facilitate recall for non attendance, and for specific aspects of regular care, legible patient records (flow charts and annual review cards) are useful for following of clinical and biochemical measures.

**Table 2.5**: **Requirements for a diabetes clinic (Amod *et al.*, 2012:S10, Table 1)**

| | |
|---|---|
| Dedicated and appropriately trained staff | |
| Adequate space | For individual consultation<br>For group education |
| Protocols covering | Screening<br>Regular care, including referrals |
| Equipment | Tape measure<br>Scale<br>Height measure<br>Accurate sphygmomanometers, with two cuff sizes<br>Monofilament or tuning fork<br>Glucometers in good working order<br>HbA1c testing equipment, to enable testing on the site<br>Educational material |
| Regular supply of medication | |
| Register with recall system for non-attendees | |
| Annual audits | Numbers of patients reaching targets for glycemic control, blood pressure and lipids<br>Numbers of patients receiving designated processes of care |

### 2.6.1 Diabetes self-management education (DSME)

DSME is essential for achievement of successful health-related outcomes, as well as learning of coping mechanisms (Shrivastava *et al.*, 2013: Online; Amod *et al.*, 2012:13). The seven important self care behaviours in people with diabetes which predict good outcomes are healthy eating, being physically active, monitoring of blood sugar, compliant with medications, good problem-solving skills, healthy coping skills and risk-reduction behaviours (Shrivastava *et al.*, 2013: Online). Proper education is shown to result in a better compliance and adherence to treatment (Amod *et al.*, 2012:13).

The main aim of diabetes education is to promote patient self-management and improve the quality of life (ADA, 2013a:S24; Amod *et al.*, 2012:13), to provide patients with the knowledge, skills, and motivation (Franz, 2012:693). Effective DSME education in conjunction with effective medical management has been shown to improve glycemic control, which in turn reduces hospital admissions and a range of other adverse diabetes outcomes (Shrivastava *et al.*, 2013: Online; ADA, 2013a:S24). Self-management is the cornerstone of modern diabetes care and providing patients with the information, skills, and support they need to manage the disease is a critical issue for health care providers and systems (ADA, 2013a:S24).

In an ideal situation, patients with diabetes should receive DSME at the time of diagnosis (ADA, 2013a:S24; IDF, 2012:21; Amod *et al.*, 2012:13). DSME should be evidence-based, patient-centered, with a well-structured education programmes, and to be on an ongoing basis, based on routine assessment of needs of the patients (ADA, 2013a:S24; IDF, 2012:21; Amod *et al.*, 2012:13). The education need to be given by trained multi-disciplinary team (physicians, dietitians, nutritionists, nurses, pharmacists, and mental health professionals with expertise in diabetes) to group of people with diabetes or individually (ADA, 2013a:S16; IDF, 2012:21; Amod *et al.*, 2012:13; Franz, 2012:693).

Where desired a family member or a friend might be included (IDF, 2012:21; Amod *et al.*, 2012:13). DSME should be adapted for the elderly, the handicapped and people who live alone (ADA, 2013a:S24; IDF, 2013b:23; Amod *et al.*, 2012:13). Specialized pre-conception education should be offered to improve pregnancy outcomes (ADA, 2013a:S24; IDF, 2013b:23; Amod *et al.*, 2012:13). Small group education is the most cost-effective option,

and it is shown to reduce $HbA_{1c}$ by 1% per 23.6 hours of education provided (Amod *et al.*, 2012:13). The literature suggests that a strong core group of topics are beneficial in the design of the curriculum for teaching self-management, and need to be made available to all people with diabetes, irrespective of culture, race, language, level of education or socio-economic status (ADA, 2013a:S16; IDF, 2012:21; Amod *et al.*, 2012:13; Franz, 2012:693).

Amod *et al.*, (2012:13) recommended topics to be covered under DSME to include: the basic knowledge of diabetes, importance of good comprehensive control (nutrition counseling, instructions in managing insulin, other medications, and in the use of blood glucose meters) and methods to achieve, insulin injection techniques and sites of injection, self-monitoring of blood glucose, recognition and management of acute and chronic complications, foot care, smoking cessation and responsible alcohol use, pre-conception care, pregnancy (preparing, managing diabetes during pregnancy), and appropriate postnatal care, psychosocial issues, stress management and coping skills, training of caregivers and family of people with diabetes, managing diabetes emergencies (importance of an identification disc or bracelet) (Amod *et al.*, 2012:13).

Children with T2DM should be referred for specialist assessment and diabetes education (Amod *et al.*, 2012:13), while elderly patients need to be assessed for knowledge and understanding of diabetes, evaluated for ability to learn and apply new self-care skills, assessed for nutrition and physical activity, assessed for cognitive dysfunction, depression and physical disability, and to address poly-pharmacy and co-morbidities, quality of life versus life expectancy (IDF, 2013b: 23; Amod *et al.*, 2012:13).

ADA (2013a:S24) indicated that DSME is associated with increased use of primary and preventive services and lower use of acute, inpatient hospital services. Patients who participated in diabetes education were more likely to follow best practice treatment recommendations, and had lower medical costs (ADA, 2013a:S24). However, most patients with diabetes do not receive any structured diabetes education and/or nutrition therapy (Evert *et al.*, 2013: Online).

The National data in USA, indicated that almost half of diabetes patients reported receiving some type of diabetes education, and fewer were ever referred to a dietitian (Evert *et al.*,

2013: Online). Self-management education can be provided to patients with diabetes in a variety of settings, using any of a variety of models and methods, and may be provided in a one-on-one format or in group settings (Amod *et al.*, 2012:S13).

According to ADA (2013a:S24), several studies demonstrated that DSME is associated with improved diabetes knowledge and self-care behavior, improved clinical outcomes such as lower self-reported weight, improved quality of life, healthy coping, and lower medical costs. However, better outcomes were reported for DSME interventions that were longer and included follow-up support, that were culturally and age appropriate and were tailored to individual needs and preferences, and that addressed psychosocial issues and incorporated behavioral strategies (Evert *et al.* (2013: Online).

Klein *et al.*, (2013: Online) in their study, which addressed whether DSME interventions helped patients with T2DM to sustain glycemic control, revealed that DSME does assist patients with T2DM to achieve their control, but some patients had difficulty in altering their long-term behavioral patterns, while others were unwilling even to try. Nevertheless, Klein *et al.*, (2013: Online) suggested that innovative DSME programs that build mental models to help people realize anomalies, identify possible causes, and generate corrective actions will result in more patients eventually achieving their $HbA_{1c}$ targets.

### 2.6.2. Medical nutrition therapy

The goals of nutrition therapy (MNT) that apply to adults with T2DM include: promotion and support of healthful eating patterns, emphasizing a variety of nutrient dense foods in appropriate portion sizes in order to improve overall health and to achieve the metabolic control (attain $HbA_{1c}$ < 7%, blood pressure of 140/80mmHg, and lipids targets-LDL cholesterol of < 1.8 mmol/l, HDL cholesterol of > 1.3 mmol/l for females and > 1.0 mmol/l for males, triglycerides of < 1.7 mmol/l, achieve and maintain body weight goals (5-10% weight loss), and delay or prevent complications of diabetes (Evert *et al.*, 2013: Online; Amod *et al.*, 2012: S57).

MNT is important for prevention, treatment and self-management of diabetes, as well as in delaying the onset of diabetes-related complications (ADA, 2013a:S22; IDF, 2013b:19; Amod *et al.*, 2012:S15). It is shown that MNT can reduce $HbA_{1c}$ levels by 1-2%, depending

on the duration of diabetes (Amod *et al.*, 2012:S15; Franz, 2012: 685; IDF, 2012: Online), while the secondary benefits include weight loss, reduction in risk factors for common co-morbidities of T2DM such as hypertension (5mmHg), dyslipidaemia (9-12%), and cardiovascular diseases (Franz, 2012:685). Apart from preventing and controlling diabetes, MNT has been recognized as an essential component of the healthy lifestyle (ADA, 2013a:S13; Amod *et al.*, 2012:S15).

In order for the MNT to be successful, it requires a coordinated team effort including registered dietitians and/or nutritionists, preferably dietitians who are knowledgeable and skilled in implementing current nutrition therapy recommendations for diabetes (ADA, 2013a:S13; IDF, 2012: Online). MNT requires an individualized approach and effective nutrition self-management education and counseling (IDF, 2013b:19; ADA, 2013a:S22). Randomized controlled trials and systemic reviews demonstrated that nutrition therapy is very effective for improving glycemic control, various markers of cardiovascular and hypertension risk (Evert *et al.*, 2013: Online).

Some studies had shown that MNT provided by a registered dietitian (RD) to participants with an abnormal lipid profile reduced daily fat intake by 5-8%, saturated fat by 2-4%, and energy intake by (974kJ/day-2982kJ/day), and lowered triglycerides by 11-31%, LDL cholesterol by 7-22%, and total cholesterol by 7-21% levels (Evert *et al.*, 2013:Online; IDF, 2012: Online). However, the management of T2DM by nutrition therapy and physical activity alone (without medications) does not seem to effectively maintain $HbA_{1c}$ after some time in most patients with T2DM (Evert *et al.*, 2013: Online; IDF, 2012: Online).

MNT should follow a patient-centered approach, include the patient's nutritional status assessment and diabetes self-management knowledge and skills, identify and negotiate individualized nutrition goals, tailor the nutritional intervention to allow the match between a meal-planning approach and educational materials with the patient needs, and to allow for flexibility (IDF, 2013b:19; Amod *et al.*, 2012:S15). Lastly, the MNT should ensure that there is ongoing monitoring, evaluation, support and assessment (Amod *et al.*, 2012:S15).

In one study done among 18 404 patients with diabetes, 9.1% reported that they had at least one nutrition visit within a 9-year period (Evert *et al.*, 2013: Online). In fact many patients

with diabetes, including their health care providers are not aware that there are services such as nutrition therapy available to them (Evert *et al.*, 2013: Online).

#### 2.6.2.1 Nutrition care plan

Nutrition care plan encompasses different steps, which include nutrition assessment, nutrition diagnosis, nutrition intervention (for example meal planning, education and counseling), nutrition monitoring and evaluation with ongoing follow-up to support long term lifestyle changes in order to deliver MNT (Franz, 2012:694). Some individuals with diabetes may receive MNT individually, while others may receive it in group sessions (Evert *et al.*, 2013: Online; Franz, 2012:694).

ADA recommend that for newly diagnosed patients with diabetes or during the first referral, MNT be provided by an RD in an initial series of three to four meetings each lasting 45 to 90 minutes (Evert *et al.*, 2013:Online; Franz, 2012:694). At least one follow-up meeting is recommended annually to reinforce lifestyle changes and to evaluate and monitor outcomes that may affect the need for changes in MNT or medication (Evert *et al.*, 2013: Online; Franz, 2012:694).

**i) Nutrition assessment**

Nutrition assessment involves obtaining information before and during the meeting to identify nutrition-related problems (Franz, 2012:694). Nutrition assessment data can be obtained from the referral source or the patient's medical record and from the patient (Franz, 2012:694). Nutrition assessment should be an ongoing process that will involve the initial data collection, the reassessment and analysis of patient data and needs (Franz, 2012:694).

The three specific assessments that are recommended by the ADA (2013a:S13) include: to assess food intake (focusing on carbohydrates), medication, metabolic control (glycemic control, lipids, and blood pressure), anthropometric measurements, and physical activity as the basis for the implementation of the nutrition prescription, goals and interventions (Franz, 2012:694). Second, to assess glycemic control and focus MNT to achieve and maintain blood glucose levels in the target range, and the need for cardio-protective nutrition interventions (Franz, 2012:694). Thirdly, to assess the relative importance of weight management for persons with diabetes who are overweight and obese (Franz, 2012:694).

**a) Anthropometric measurements**

Anthropometric measurements are the most basic methods of assessing body composition. Anthropometric measurements describe body mass, size, shape, and level of fatness (Gibson, 2005:44). Since the body size changes with weight gain, anthropometry gives the researcher an adequate assessment of the overall adiposity of an individual (Gibson, 2005:44).

**BMI**

One way to overcome the lack of specificity in body weight is to use the BMI. BMI is a descriptive index of body habitus that encompasses both the lean and the obese (Nyamdorj, 2009:70), and is expressed as the current measured body weight in kilograms (kg) divided by measured height in meter squared ($m^2$), which provide a gross evaluation of total body fat (Knowles *et al.*, 2011:Online; Browning *et al.*, 2010:247, Nyamdorj, 2009:70). BMI has been the most common measure that was used, and its concept dates back to 1869 as Quetelet's index, which was shown as a fairly good indicator of general fatness (Bergman *et al.*, 2011:1084; Nyamdorj, 2010:70).

BMI is particularly useful in monitoring the treatment of obesity, with a weight change of 3.5kg needed to produce a unit change in BMI (Nyamdorj, 2009:70). BMI levels above 25kg/$m^2$ are associated with increased risk of morbidity and mortality, with BMI levels of 30kg/$m^2$ and greater indicating obesity (Nyamdorj, 2009:70). According to WHO (2000), BMI is considered a valid tool to determine overweight and obesity in adults, as classified in chapter 3 (Table 3.2).

Klisiewicz and Raal (2009:15) stated that BMI provide a guide for appropriate weight for height, but does not take into account the effects of distribution of body fat. BMI is found to predict diabetes very well in all ethnic groups, and is associated with T2DM (Nyamdorj, 2009:70). BMI is particularly useful in monitoring the treatment of obesity (Nyamdorj, 2009:70). There is also a less measurement of error with BMI than with WC, and the measure for BMI is easy to standardize, as results are easy to compare between different populations throughout the world (Nyamdorj, 2009:70). There is no difference between BMI and WC in their predictive ability (Leitzmann *et al.*, 2011: Online), and BMI alone cannot independently be associated to the risk of incident diabetes (Leitzmann *et al.*, 2011: Online; Bororgmanesh *et al.*, 2011: Online).

The adiposity varies by age, sex, and ethnicity for a given BMI (Nyamdorj, 2009:70). BMI is widely used to estimate body fat, not only in epidemiological studies, but also in clinical practice despite warnings that it is not a very accurate measure of adiposity in individual patients (Bergman *et al.*, 2011:1084). BMI is particularly inaccurate in subjects with elevated lean body mass, such as athletes, and cannot be generalized among different ethnic groups (Bergman *et al.*, 2011:1084).

A defining criteria of "healthy" weight for the elderly poses a challenge as there are physiological changes related to aging (BMI lacks the ability to distinguish between fat-mass and fat-free mass), especially in body composition and height (Leitzmann *et al.*, 2011: Online). Thus, the accuracy of BMI as an indicator of adiposity decreases with increasing age (fat-free mass decreases with aging, without a change in overall weight) (Leitzmann *et al.*, 2011: Online).

**WC**

WC is described as a measure of the horizontal plane midway between the superior iliac crest and the lower margin of the last rib or border of the iliac crest, thus, provides a simple and reliable assessment of obesity and central adiposity (Nyamdorj, 2009:70). WC measurement is considered a good indicator of intra-abdominal (visceral) fat in adults (Ashwell & Browning, 2011:70; Browning *et al.*, 2010:248; Feller *et al.*, 2010: Online; Nyamdorj, 2009:70).

WC is an independent risk factor for cardiovascular disease, diabetes and other endocrine abnormalities, and assessment of central obesity holds greater prognostic value than BMI alone (Leitzmann *et al.*, 2011: Online; Browning *et al.*, 2010:248; Feller *et al.*, 2010: Online). Ethnic-specific cut-off points recommended by the IDF for Sub-Saharan Africa include, central obesity defined as WC of ≥80 cm in females and ≥94 cm in males (Amod *et al.*, 2012:S58).

WC is found to predict T2DM very well in all ethnic groups, and is recommended due to its close association with unfavorable health consequences, not because its measurement error is less (Leitzmann *et al.*, 2011: Online; Nyamdorj, 2009:70). Visceral or abdominal fat is shown to be more metabolically active than subcutaneous fat (thought to produce substances

that may have a favorable effect to glucose metabolism) and secretes more hormones and cytokines hence may be more deleterious to health (Feller *et al.*, 2010: Online), as individuals in the upper percentiles for abdominal circumference are considered obese and at increased risk for morbidity, specifically T2DM (Feller *et al.*, 2010: Online; Amato *et al.*, 2010: Online).

WC was shown to be strongly correlated with BMI, to be more reliable predictor of cardiovascular disease, diabetes and related risk factors (Leitzmann *et al.*, 2011: Online; Browning *et al.*, 2010:266), and a better correlate of visceral fat deposits than waist-hip-ratio (WHR) (Amato *et al.*, 2010: Online). However, WC is found to over-and-under evaluate risk for tall and short individuals with similar WC (Browning *et al.*, 2010:248), while WC alone does not assist in distinguishing between subcutaneous and visceral fat mass (Amato *et al.*, 2010: Online), and cannot independently be associated with the risk of diabetes (Bororgmanesh *et al.*, 2011: Online).

Feller *et al.*, (2010: Online), further explained that individuals with low muscle mass may have a greater risk of diabetes than those with larger muscle mass, at a given fat mass. The important result from Feller's study revealed that the risk of diabetes was high for individuals with low or normal weight, and with above average waist circumference (females: > 80 cm; males: > 94cm) (Feller *et al.*, 2010: Online). Furthermore, the Epic-Potsdam study done in Germany, confirmed that individuals of normal weight with large waist circumference showed an increased risk of mortality (Feller *et al.*, 2010: Online). The findings of the study showed that the precise estimation of the T2DM risk requires measurement of both the BMI and the waist circumference, hence the study advocated for the inclusion of the group with < 25kg/m² and a large waist circumference in the guidelines on diabetes prevention (Feller *et al.*, 2010: Online).

**WHtR**

WHtR is the current waist circumference in centimeters (cm) divided by height in meter (m), and is considered to be the best tool to determine abdominal fatness, thus predicting risk for diseases, especially cardiovascular diseases and diabetes (Kruger *et al.*, 2013: Online; Ashwell *et al.*, 2012: Online; Knowles *et al.*, 2011: Online; Ashwell, 2011: Online; Browning

*et al.*, 2010:248). The use of WHtR for detecting obesity, and health risks associated with it, was first proposed in the mid-1990s (Ashwell *et al.*, 2012: Online).

WHtR has demonstrated to be very useful among different ethnic, age and sex groups (Ashwell, 2011: Online; Browning *et al.*, 2010:248). The WHtR of 0.5 is correlated with total body fat, in both females and males (Browning *et al.*, 2010:248). The advantages of using WHtR over other anthropometric indices (BMI and WC), though not yet universally adopted include: WHtR is more sensitive that BMI as an early warning of health risks, the cut-off value of 0.5 demonstrate increased risk in both females and males, and of different ethnic groups, WHtR is cheap and easy to measure (as measuring weight require accurate scales and some degree of subject undressing), and calculate than BMI, and allows the same boundary values for children and adults, and boundary values can be easily converted into a consumer-friendly chart (Browning *et al.*, 2010:265).

Cross-sectional studies in both adults and children on diabetes outcomes revealed that WHtR was significantly associated with diabetes outcomes in six of the seven studies (Browning *et al.*, 2010:252). Ashwell (2011: Online), further indicated in a "National Diet and Nutrition Survey" that screening for cardiovascular health risk by BMI alone would miss 14% of females and 35% of males who would be within the normal range, but have central fat distribution, defined by a cut-off value of WHtR greater than 0.5.

In a meta-analysis of abdominal obesity indices comparing BMI, WC, WHR, and WHtR, researchers concluded that WHtR was the best predictor for both hypertension and dyslipidaemia (comorbid conditions of T2DM) for both females and males followed by WC, and BMI (Ashwell *et al.*, 2012: Online; Knowles *et al.*, 2011: Online; Sluik *et al.*, 2011: Online). According to Ashwell *et al.*, (2012: Online), evidence from the studies involving 300 000 adults in several ethnic groups showed the superiority of WHtR over WC and BMI for detecting cardiometabolic risk factors in both females and males.

**BAI**

BAI is the current hip circumference in centimeters (cm) divided by height in meters (m) to the power of 1.5, subtract 18 (cm/ $(m)^{1.5}$-18) (Bergman *et al.*, 2011:1084). BAI is a newly developed method to estimate adiposity of individuals, and offers a direct estimate of

percentage body fat (% body fat). Unlike BMI, BAI provides % body fat in both females and males of differing ethnicities without numerical correction (Bergman *et al.*, 2011: 1084). BAI can be measured without weighing, which may be very useful in settings where measuring accurate body weight is a great problem (Bergman *et al.*, 2011:1085).

However, BAI proved not to be as good as BMI and WC in predicting diabetes (Schutze *et al.*, 2012: Online). According to Bergman *et al.*, (2011:1084) a healthy BAI is 20% - 38% in females and 8% - 25% in males.

**b) Dietary intake**

Dietary assessment is classified as the medical and nutritional assessment methods used to gather information on the habitual food consumption of the individual (Lee & Nieman, 2010:83). The measurement of dietary intake is essential for investigating the causes of malnutrition, interventions to combat malnutrition, the diet-health relationships (for example the relationship between diet and diabetes) (Lee & Nieman, 2010:83). Dietary assessment can also be used in the formulation of policies and guidelines to improve health and nutritional status, in predicting the adequacy of the food supply, and to monitor trends in food use, exposure to additives, contaminants, and compliance with dietary guidelines (Lee & Nieman, 2010:83). The widely used methods in research include the 24-hour dietary recall method, dietary history method and food frequency questionnaire (Lee & Nieman, 2010:83).

**24-hour dietary recall**

In 24-hour recall the subject is interviewed to find out the actual food intake during the immediate past, usually the past 24 or 48 hours or during the preceding day (Gibson, 2005:42). The method is quick and relatively cheap, and it can be used equally well with both literate and illiterate subjects (Gibson, 2005:42). However, a single 24-hour recall is not sufficient to describe an individual's usual intake of food and nutrients, and three to seven 24-hour recalls are shown to provide a better estimation (Lee & Nieman, 2010:83). Single 24-hour recall can be used to assess actual intakes of food and nutrients, sometimes required for metabolic studies, or for counseling purposes (Gibson, 2005:42).

The advantages of 24-hour recall include, it places little burden on the subjects, it is unlikely to alter eating behavior, memory problems are usually minimal, participants are generally

willing to respond to the interviewer, and thus refusals are less likely to occur (Gibson, 2005:42). The interviewer also assists the respondent to estimate portion sizes (Lee & Nieman, 2010:77).

The disadvantages include, the respondents tend to withhold or alter information about what they have eaten because of poor memory, embarrassment or intent to please or impress the interviewer (Lee & Nieman, 2010:83). Respondents tend to also underreport binge eating, consumption of alcoholic beverages and foods perceived as unhealthy (Lee & Nieman, 2010:83). Another disadvantage is inaccuracy in recalling the kinds of food and amount of food consumed, and tendency for persons to over report low intakes and under report high intakes of food (Hammond, 2008: 398; Lee & Nieman, 2010:83).

**Food Frequency Questionnaire**

A Food Frequency Questionnaire estimates how often foods are eaten by an individual, and usually qualitative (does not include usual portion sizes), expressed per day, per week, per month or seasonally (Lee & Nieman, 2010:86). However, FFQ may also include quantitative assessment of usual portion size (Hammond, 2008:397). FFQs are mainly used in studies that are designed to determine associations between food intake and disease, that is, it proved to be good when used in large epidemiologic studies on diet and chronic diseases (Lee & Nieman, 2010:86). When the 24-hour recall is used concurrently with a FFQ, the accuracy of intake estimates is improved (Lee & Nieman, 2010:86).

The advantages of FFQ include, it impose less burden on respondent than most of the other dietary assessment tools, the results are easy to collect and process and are generally taken to represent usual intakes over an extended period of time, it is relatively inexpensive for large sample sizes (Lee & Nieman, 2010:86), it provides an overall picture of the dietary intake, and easy to standardize (Hammond, 2008:397).

The disadvantages of FFQ include, the information obtained is only on the frequency of consumption of a particular food at a given period of time, rather than reflecting the context in which the food was eaten (Lee & Nieman, 2010:86). FFQ does not provide the data of the meal pattern, require knowledge of portion sizes, and literacy skills if self administered (Hammond, 2008:422).

**ii) Nutrition intervention**

Most diabetes patients find it very difficult to determine what to eat, when and how much, there is no "one-size-fits-all" in eating pattern for individuals with diabetes (Evert *et al.* 2013: Online). IDF (2013a:19) recommended that the nutrition care plan should be individualized and need to consider the person's food preferences, eating routines, religion and culture, and physical and cognitive health status (particularly for elders) (IDF, 2013a:19), as well as health literacy and numeracy, access to healthful food choices, willingness and ability to make behavioral changes, and barriers to change (Evert *et al.* 2013: Online).

The meal plan should include a variety of foods to ensure essential vitamins, minerals, proteins, and fibre are consumed in adequate amounts, and the times for medicine administration must coincide with meal times (IDF, 2013a:19). Weickert (2012: Online), recommended the use of a Mediterranean-like dietary pattern, to avoid excess intake of dietary fat, substitute SFA and TFA by MUFA and omega-6 PUFA, and emphasizing the cereal fibre content in the diet and keeping exercise levels high.

**a). The Diabetes Food Pyramid**

The Diabetes Food Guide Pyramid was designed by the American Diabetes Association (ADA) and the American Dietetic Association (ADA) (ADA and ADA, 2005: Online). The Diabetes Food Guide Pyramid (Figure 2.3) represents a method of meal planning that is used to visually represent the recommended servings from each food group per day (ADA and ADA, 2005:Online).

Similar to the USDA Food Guide Pyramid, the Diabetes Food Guide Pyramid is used to guide patients with group of foods based on their carbohydrate and protein content, because these foods affect blood glucose levels, which is the primary concern to people with diabetes (ADA and ADA, 2005:Online). There are slight differences between the two systems, for example on the Diabetes Food Guide Pyramid potatoes and other starchy vegetables are grouped into the grains, beans and starchy vegetables group instead of the vegetables group, and cheese is grouped into the meat group instead of the milk group (ADA and ADA, 2005:Online). A serving of pasta or rice is ⅓ cup in the Diabetes Food Pyramid, and ½ cup in the USDA pyramid (ADA and ADA, 2005: Online). Fruit juice is ½ cup in the Diabetes Food Pyramid and ¾ cup in the USDA pyramid (ADA and ADA, 2005: Online). This difference is to make

the amount of carbohydrates about the same in all the servings listed (ADA and ADA, 2005: Online).

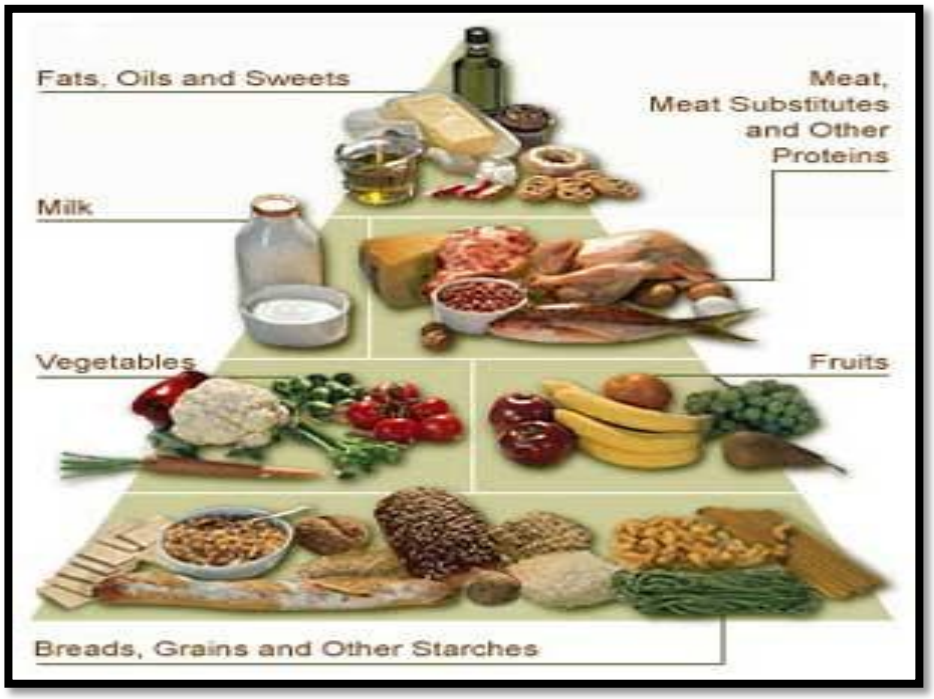


**Figure 2.3:** Diabetes Food Guide Pyramid (ADA and ADA, 2005: Online)

**Breads, grains and starches**

Grains and starches are situated at the base of the pyramid; these are foods containing most carbohydrates (ADA and ADA, 2005: Online). Bread, cereal, rice, and pasta are food in this group mostly made of grains, such as wheat, rye, and oats (ADA and ADA, 2005: Online). Starchy vegetables like potatoes, corn, and peas also belong to this group, along with dry beans such as pinto beans and black eyed peas. Starchy vegetables and beans are in this group because they have about as much carbohydrate in one serving as a slice of bread (ADA and ADA, 2005: Online). As for beans and starches, they are in a group together because they affect blood glucose in the same way (ADA and ADA, 2005: Online).

Daily intakes of six to eleven (6-11) servings are recommended (ADA and ADA, 2005: Online).

**Fruits**

Fruits are important because they provide important vitamins, minerals, and fiber. Fruits also contain carbohydrates (ADA and ADA, 2005: Online). It is advisable to consume whole fruits rather than juices because of the fiber contained; and to avoid fruits and fruit juices that contain sweeteners or syrups added. This latter group includes blackberries, grapefruit and tangerines, strawberries, oranges, apples, bananas, peaches, pears, and apricots (ADA and ADA, 2005: Online).

Daily intakes of two to four (2-4) servings are recommended (ADA and ADA, 2005: Online).

**Vegetables**

Vegetables have plenty of vitamins, minerals, fiber, and naturally all of them are low in fat (ADA and ADA, 2005). Vegetables that should be at the top of the list are dark green and deep yellow vegetables, such as spinach, broccoli, lettuce, carrots, cucumbers, chilies and peppers. Try to get fresh or frozen vegetables rather than canned vegetables because they have less sauces, fats and salt added (ADA and ADA, 2005: Online).

Daily intakes of three to five (3-5) servings are recommended (ADA and ADA, 2005: Online).

**Milk and milk products**

Milk products contain a lot of protein and calcium as well as many other vitamins. Try to choose low-fat or non-fat milk products for the great taste and nutrition without the saturated fat (ADA and ADA, 2005: Online).

Daily intakes of two to three (2-3) servings are recommended (ADA and ADA, 2005: Online).

**Meat and meat substitutes**

Meat and meat substitutes includes beef, chicken, turkey, fish, eggs, tofu, dried beans, cheese, cottage cheese and peanut butter (ADA and ADA, 2005: Online). Meat and meat substitutes are great sources of protein and many vitamins and minerals. Fish and poultry are recommended over red meat, because they are less fatty. The portion sizes should be kept

small and trim away all the visible fat off meat before cooking. Baking, roasting or grilling is preferable to frying (ADA and ADA, 2005: Online).

Daily intakes of two to three (2-3) servings are recommended (ADA and ADA, 2000: Online).

**Sweets, fats and alcohol**

Sweets, fats and alcohol should be taken in small amounts, the body needs fats for other functions, but avoids eating too much of it (ADA and ADA, 2005: Online). Although sugary foods like candy and cookies are simple carbohydrates that can give quick energy, they are usually loaded with calories and do not offer much nutrients. In the right amounts, though, fats, alcohol and sweets can spike up the flavor in meals and snacks (ADA and ADA, 2005: Online). The recommendation stated to use these foods sparingly; to eat only a little bit at a time, and not very often (ADA and ADA, 2005: Online).

**b). The Idaho plate model**

Idaho plate model as demonstrated in Figure 24, is another method that could be used for meal planning, it is a good starting point for healthy meal planning that can be used until referral to a dietitian for MNT (Amod *et al.,* 2012:S17). The plate model proved to be effective for both managing diabetes and losing weight (Amod *et al.,* 2012:S17). It allows patients to choose the foods that they enjoy, but within the recommended portion sizes. It focuses on increasing the portion sizes of non-starchy vegetables, and decreasing the portion sizes of starches (Amod *et al.*, 2012:S17). A plate with illustrated portion sizes encourages patients to consume carbohydrates throughout the day, which will assist with blood glucose control (Amod *et al.*, 2012:S17).

The adequate proportions of the macronutrients (carbohydrates, proteins and fats) distributions are not well clarified (Ajala *et al.*, 2013: Online). When guidance is required in meal planning, the dietary reference intakes (DRIs) for healthy eating, which recommend that adults should consume 45% to 60% of total energy from carbohydrates, 20% to 35% from fat, and 15% to 20% from proteins can be very useful (Table 3.7, chapter 3) (Franz, 2012:684). Alternatively, when implementing the nutrition therapy, the initial areas that need much attention should include replacing red meat intake with legumes and fish, increasing the

amount of fruits and vegetables consumed per day, and replacing saturated fats with monounsaturated fats (Benson *et al.*, 2011:Online).

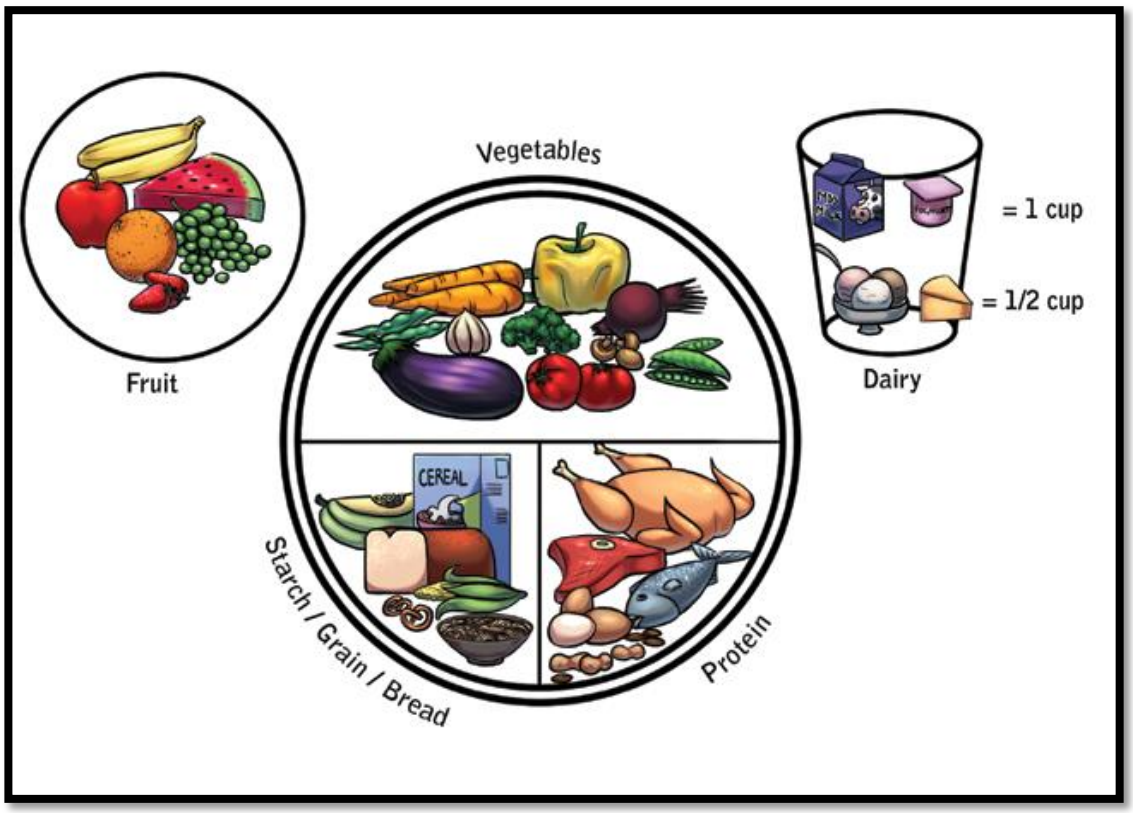


**Figure 2.4:** The Idaho plate model (Amod *et al.*, 2012:S17)

**How to use a plate model**

A 22 cm plate is marked with a line down the centre, one half of the plate is divided into two equal sections (Amod *et al.*, 2012:S17). One half of the plate is filled with non-starchy vegetables (such as spinach, carrots, cabbage, green beans, broccoli, cauliflower, tomatoes, cucumber, lettuce, beetroot, mushrooms, peppers and other greens (Amod *et al.*, 2012:S17). One quarter section is filled with starchy foods (such as whole-grain breads, whole-grain high fibre cereal, cooked oatmeal, brown or long-grain rice, pasta, baby potatoes, green peas, sweet potatoes, whole-grain crackers and fat-free popcorn (Amod *et al.*, 2012:S17). The last quarter of the plate is filled with meat and meat substitutes, such as skinless chicken, turkey portions, fish and other seafood, lean cuts of beef, pork, tofu, soya, eggs and low-fat cheese (Amod *et al.*, 2012:S17).

Avoid processed meats (for example salami, Vienna sausages, Russians and polony), which are high in fat and salt (Amod *et al.*, 2012:S17). Eat or drink a glass of non-fat or low-fat

milk or 180ml of low-fat yoghurt at least two servings a day (Amod *et al.*, 2012:S17). Add a medium portion of fruit (e.g. oranges, apples, pears or small bananas), or two small fruits (e.g. plums or peaches), or three quarters of a cup of fresh fruit salad (Amod *et al.*, 2012:S17). Instead of eating fruit with meals, it can be used as a snack between meals, and eat only two or three servings per day (Amod *et al.*, 2012:S17).

**iii) Nutrition Prescription**

When developing the nutrition prescription it is essential to learn about the patient's lifestyle and eating habits (Franz, 2012:699). Food and diet or eating histories can be done several ways, with the objective being to determine a schedule and pattern of eating that will not disrupt the lifestyle of the individual with diabetes and at the same time, will facilitate improved glycemic control (Franz, 2012:699). Nutrition prescription need to be done bearing in mind that there is no standard meal plan or eating pattern that work best for all patients with T2DM, and for it to be effective it should be individualized (Evert *et al.*, 2013:Online).

Therefore, asking the individual either to record or report what, how much, and when he or she typically eats during a 24-hour period may be the most useful way of obtaining a diet history (Franz, 2012:699). Alternatively, the patient may be asked to bring a 3-day or 1 week food intake record (Franz, 2012:699). It is also important to learn about the patient's daily routine and schedule, may include the time of walking, usual meal and eating times, work schedule or school hours, type, amount, and timing of exercise, and usual sleep habits (Franz, 2012:699).

Using the assessment data, and food and diet history information collected, a food and meal plan can be designed with the use of exchange list as illustrated in Table 2.5 below, and if the patient desires, sample menus may be provided (Franz, 2012:699). To determine the appropriateness of the meal plan for diabetes management, distribution of the meals or snacks must be assessed along with the types of medications prescribed and treatment goals (Franz, 2012:699).

**Table 2.6: The Exchange list (Franz, 2012:700, Table 31.9).**

| Exchange list | Energy (KJ) | Protein (g) | Fat (g) | Carbohydrates (g) |
|---|---|---|---|---|
| **Milk:** Skim | 375 | 8 | Trace | 12 |
| Low –fat | 525 | 8 | 5 | 12 |
| Whole | 640 | 8 | 8 | 12 |
| **Starch/bread** | 285 | 3 | Trace | 15 |
| **Fruit** | 250 | - | - | 15 |
| **Vegetables** | 105 | 2 | - | 5 |
| **Meat:** Very lean | 155 | 7 | 1 | - |
| Lean | 230 | 7 | 3 | - |
| Medium fat | 315 | 7 | 5 | - |
| High fat | 420 | 7 | 8 | - |
| **Fat:** | 190 | - | 5 | - |
| **Sugar:** | 85 | - | - | 5 |
| **Alcohol:** | 45ml distilled beverage | 2 fats | | |
| | 120ml red/rose/dry wine | 2 fats | | |
| | 120ml sweet wine/sherry | ⅓bread and 2 fats | | |
| | 340ml beer | 1 bread and 2 fats | | |

**Note:** 1g of carbohydrates and 1 g of protein yields 17kJ; 1g of fat yields 38 kJ, and 1g of alcohol yields 29 kJ.

In addition, the SAFBDG could be used if adapted to reflect the diabetes-specific recommendations (Steyn & Temple, 2012:502) and include:

- Enjoy a variety of foods (eat regular small meals)
- Be active
- Make starchy foods the basis of most meals, (including those that are slowly digested, high in fibre)
- Eat plenty of fruit and vegetables everyday (minimum of 5 servings per day)
- Eat cooked dried beans, peas, lentils and soya regularly
- Low fat meat, fish, chicken, milk, yoghurt, cheese or eggs can be eaten every day (but a diabetes patient is advised to limit eggs intake to 3 eggs per week)
- Eat fats sparingly, (especially saturated fats, and try to avoid trans-fatty acids)
- Use salt sparingly
- Use food and drinks containing sugar sparingly and not between meals
- Drink lots of clean safe water
- If you drink alcohol, drink sensibly (have an alcohol free day per week)

There are eating patterns studied among T2DM patients to evaluate their impact on diabetes nutrition goals and include: Mediterranean, vegetarian, low-fat, low-carbohydrate, and DASH diets (Evert *et al.*, 2013: Online). The Mediterranean-style eating pattern, though studies

were mostly done in the Mediterranean region, has been shown to improve cardiovascular risk factors (for example the lipids, blood pressure, triglycerides), more especially when supplemented with mixed nuts or olive oil (Evert *et al.*, 2013: Online; Estruch *et al.*, 2013: Online). For those individuals following an energy-restricted Mediterranean-style eating pattern improvements in glycemic control was also achieved (Evert *et al.*, 2013: Online; Benson *et al.*, 2011: Online).

The reviewed studies on vegetarian and low-fat eating patterns in individuals with T2DM, all the six studies neither improved glycemic control nor cardiovascular risk factors, except when energy intake was restricted and weight was lost. However, both diets resulted in a significant weight loss (Evert *et al.*, 2013: Online).

The DASH eating pattern is frequently recommended as a healthful eating pattern for the general population (Evert *et al.*, 2013: Online). However, there is a limited evidence on the effects of the DASH eating plan on health outcomes particularly in individuals with diabetes, but would expect similar results as for people without diabetes, whom DASH eating pattern was shown to help control blood pressure and lower risk for cardiovascular disease (Evert *et al.*, 2013:Online).

Evidence from different studies suggests that different macronutrient distributions may lead to improvements in glycemic control and/or cardiovascular disease risk factors (Evert *et al.*, 2013: Online). There is no ideal eating pattern (even percentage of energy from carbohydrates, proteins and fats) that is expected to benefit all individuals with diabetes (Evert *et al.*, 2013: Online). However, Benson *et al.*(2011: Online) recommended that the Mediterranean diet (a traditional eating pattern) may be a simpler strategy to help patients with diabetes achieve more optimal glycemic control and reduce the risk of complications. Thus, it is essential that the total energy intake (portion sizes) is considered for each person no matter which eating pattern is chosen, hence a variety of eating patterns are acceptable for the management of T2DM (Evert *et al.*, 2013: Online).

**a) Carbohydrates intake**

ADA (2013a:S23) recommendation of 130g per day of carbohydrates intake is based on providing adequate glucose that is required for the central nervous system without reliance on

glucose production from proteins or fats. Conventional high-carbohydrates diets are shown to increase postprandial glucose and insulin concentrations, and may compromise fat oxidation, and metabolic flexibility (Weickert, 2012: Online).

According to Wheeler *et al.* (2010: Online), the definition of low- or- high carbohydrate diets are not well explained in the literature, but the definitions currently used are: very low-carbohydrate diet (21-70g/day of carbohydrate), moderately low-carbohydrate diet (30% to 40% of total energy as carbohydrate), moderate-carbohydrate diet (40% to 65% of total energy as carbohydrates), high-carbohydrate diet (>65% of total energy as carbohydrate). It has been shown that patients with diabetes follow an eating pattern that is about 45% to 65% of energy from carbohydrates (Amod *et al.*, 2012:16; Wheeler *et al.* 2010: Online).

In a review by Ajala *et al.*, (2013: Online), a low-carbohydrates diet (20-60g per day, and <45% of daily energy intake) resulted in a reduction of weight, improved glycemic control and reduced lipid profile (increased HDL levels by 10%, reduced triglycerides by 9% and reduced LDL levels by 1%). However, it is indicated that long term metabolic effects of carbohydrates lower than 130g per day are not clearly defined, but low-carbohydrates diets may eliminate most foods that are important sources of energy, dietary fiber, vitamins and minerals (ADA, 2013a:S23; Ajala *et al.*, 2013:Online; Franz, 2012:685), and low carbohydrates foods are not palatable (ADA, 2013a:S23).

Consumption of carbohydrates is shown to have a direct effect on postprandial glucose levels in patients with diabetes, and is the most important macronutrient in glycemic management (Evert *et al.*, 2013: Online). For maintenance of good health, carbohydrate intake from vegetables, fruits, whole grains, legumes, and dairy products should be advised over intake from other carbohydrates sources, especially those that contain added fats, sugars, or sodium (Evert *et al.*, 2013: Online).

Kim and Park (2012: Online), showed that sorghum could lower blood glucose concentration, without changing the insulin levels, and these beneficial effects of sorghum indicate that it may have bioactive components that could exert blood lipids profiles in humans. It is also important to consider the amount of carbohydrates consumed and available insulin, as are important factors influencing glycemic response post-prandially (Evert *et al.*, 2013: Online).

There is insufficient evidence to support one specific amount of carbohydrate intake for people with diabetes (Evert *et al.*, 2013: Online). Some published studies comparing a low-carbohydrate intake (<45 % of total energy) to high-carbohydrate intake (>65% of total energy), indicated that there were improved markers of glycemic control and insulin sensitivity with lower carbohydrate intakes (Evert *et al.*, 2013: Online). However, four randomized clinical trials indicated no significant difference in glycemic markers with a low-carbohydrate eating pattern compared with high-carbohydrate intakes, though the studies were small, of short duration, and/or had low retention rates (Evert *et al.*, 2013: Online; Wheeler *et al.*, 2010: Online).

Despite the inconclusive results of the studies evaluating the effect of differing percentages of carbohydrates in people with diabetes, monitoring carbohydrate amounts (by carbohydrate counting, exchanges or experienced-based estimation) is a useful strategy for improving postprandial glucose control (Evert *et al.*, 2013: Online, Amod *et al.*, 2012:S16). There is evidence to confirm that the total amount and type of carbohydrate in the food would influence blood glucose levels and glycemic response (Evert *et al.*, 2013: Online). Hence, Amod *et al.*, (2012:S16) recommend that sucrose intake be limited to 10% of total energy per day, and to limit intake of sugar alcohols (isomalt, lactitol, mannitol, maltitol, sorbitol, xylitol) to <10g per day.

**Glycemic index and glycemic load**

The GI of a carbohydrate is a measure of how much that food raises blood glucose compared with a standard carbohydrate (usually glucose or white bread), while glycemic load takes into account the amount of carbohydrate in addition to its glycemic index (Evert *et al.*, 2013: Online; Solomon *et al.*, 2009:1225). Substitution of a low-glycemic load for high-glycemic load foods is thought to improve glycemic control (Evert *et al.*, 2013: Online). The use of GI and glycemic load may provide an additional benefit compared to considering total carbohydrate content (Amod *et al.*, 2012:S16).

The use of low GI and glycemic load carbohydrates may also be beneficial for glycemic control (Rossi *et al.*, 2013: Online; Ajala *et al.*, 2013: Online). Rossi *et a.l*, (2013: Online) demonstrated that a high glycemic load diet increases blood glucose and insulin levels. If insulin levels rise for a considerable long time, this may result in pancreatic beta cell failure,

which will eventually lead to impaired glucose tolerance and increase insulin resistance. A high glycemic load is also implicated in individuals with diabetes who do not control their blood sugar (Rossi *et al.,* 2013: Online). A diet low in glycemic load minimizes the spiking of postprandial glucose, and these benefits are thought to improve insulin demand and ß cell function (de Koning *et al.*, 2011: Online). Milk proteins such as whey, which are relatively low in glycemic load, may have insulinotropic properties (de Koning *et al.*, 2011: Online).

Low-GI carbohydrate-based foods slow down digestion and regulate uptake of nutrients, and are therefore capable of prolonging satiety (Franz, 2012:685). While high-GI foods are digested more quickly and can induce very short-term satiety in response to plasma glucose levels (Wilde, 2009:368; Franz, 2012:685). A low-GI diet in addition to exercise training may lead to a decrease in whole-body fat mass and thus greater improvements in insulin sensitivity (Solomon *et al.*, 2009:1225).

Ajala *et al.,* (2013: Online) indicated that low-GI diets are shown to result in lower $HbA_{1c}$ (reduced by 0.14%), and increased HDL, but seemed not to affect weight loss. A low-GI diet may suppress the mean arterial pressure in overweight/obese young adults, due to reductions of insulinemia (Solomon *et al.,* 2009:1225). Insulin is known to have a direct effects on sodium-potassium transport mechanisms, thus the lower concentrations induced by a low-GI diet may have a positive effect on blood pressure (Solomon *et al.,* 2009:1225). These findings highlight the potential therapeutic effect of low-GI foods on elevated arterial pressure (Solomon *et al.*, 2009:1225).

Prospective cohort studies revealed that a high-GI diet is associated with elevated risk of T2DM (Sun *et al.*, 2013). In a randomized clinical trial, which replaced white rice with whole grains, the postprandial glucose and insulin levels decrease significantly (Sun *et al.*, 2013:Online). White rice has a high-GI, thus the removal of the bran and some of the germ results in loss of important nutrients (fiber, vitamins, magnesium and other minerals, lignans, phytoestrogens, and phytic acid), which may be protective towards T2DM (Sun *et al.*, 2013: Online), while brown rice may play a preventive role towards T2DM risk, because of its essential nutrients such as fiber, vitamins, and minerals (Sun *et al.*, 2013: Online).

Observational studies have linked the intake of high-GI foods to higher prevalence of insulin resistance, whereas low-GI diets improved the whole-body insulin resistance in patients with T2DM, though not all studies showed protective effects of low-GI diets on insulin resistance and diabetes risk (Weickert, 2012: Online).

It has also been postulated that a diet high in legumes (beans, lentils, peanuts, peas, and soybeans) may be beneficial for the prevention of T2DM (Evert *et al.*, 2013: Online; Solomon *et al.,* 2009:1225). Legumes are good sources of fiber and have a low-GI (Evert *et al.*, 2013: Online; Solomon *et al.,* 2009:1225). The protective effect of legumes on T2DM may involve multiple biological pathways, including increased fiber content in the diet, a reduction in the GI of mixed meals, or both (Evert *et al.*, 2013: Online). Legumes contain polyphenols, such as isoflavones and lignans, which have an antioxidant effect and may be responsible for the protective role of legumes against the development of T2DM (Bahadoran *et al.,* 2013: Online).

**Dietary fiber and whole grains**

Whole grains are referred to foods containing the entire seed, bran, germ and endosperm (Evert *et al.,* 2013: Online). The beneficial effects of fibre intake that is observed among patients with T2DM, include the viscous and/or gel-forming properties of soluble fibre from fruit and vegetables that influence the GI and blood lipids, as well as the metabolic effects of short-chain fatty acids derived from colonic fermentation of non-digested fibre by the gut microorganisms (Weickert, 2012: Online). However, the main source of fibre is obtained from wheat bran that is insoluble in water, non-viscous and moderately fermentable (Weickert, 2012: Online). In addition, cereal fibre may hinder the digestion and/or absorption of dietary protein in the upper gut, as a result preventing amino acid-induced insulin resistance (Weickert, 2012: Online).

Patients with T2DM should consume the amount of fiber and whole grains recommended for the general populations, which is to increase the intake of soluble and insoluble fibre to 25-50g per day (Amod *et al.*, 2012:S16). The recommendations for the patients with T2DM are to increase fibre intake to about 25g/day for adult females and 38g/day for adult males (Evert *et al.*, 2013: Online).

Some studies have shown modest lowering of pre-prandial glucose and $HbA_{1c}$ with intakes of 50g of fiber per day. Studies have mixed findings regarding the effect of fiber on cardiovascular risk factors, however, total fiber intake from natural food sources seems to have a beneficial effect on serum cholesterol levels and other cardiovascular risk factors such as blood pressure (Evert *et al.*, 2013: Online). The Nurses' Health Study looking at the effects of whole grains and their components in relation to all-cause and mortality due to cardiovascular diseases among T2DM patients, indicated the benefit of whole-grains intake in reducing mortality and cardiovascular disease (Evert *et al.*, 2013: Online).

Other forms of fiber include: resistant starch, which is defined as the starch physically enclosed within intact cell structures as in some legumes, in raw potatoes, and retrograde amylase from plants modified by plant breeding to increase amylose content, fructans are an indigestible type of fiber that has been hypothesized to have a glucose-lowering effect, and inulin is a fructan commonly added to many processed food products in the form of chicory root (Evert *et al.*, 2013: Online).

It has been proposed that foods containing resistant starch or high amylose foods (such as specially formulated cornstarch) may modify postprandial glycemic response, prevent hypoglycemia, and reduce hyperglycemia (Evert *et al.*, 2013: Online). Though there are no published long-term studies in subjects with T2DM to confirm the benefit from the use of resistant starch (Evert *et al.*, 2013: Online). There are no published long-term studies in subjects with T2DM to prove benefit from the use of fructans (Evert *et al.*, 2013: Online).

**Fructose**

Fructose is a monosaccharide found naturally in fruits (free fructose), and it is also a component of added sugars found in sweetened beverages and processed snacks (Evert *et al.*, 2013: Online). Free fructose may result in better glycemic control compared with intake of sucrose or starch, as well as triglycerides provided the intake is not excessive (12% of total energy), however, patients with T2DM are advised to limit or avoid intake of sugar-sweetened beverages (SSBs) in order to reduce risk of weight gain and worsening of cardio-metabolic risk profile (Evert *et al.*, 2013: Online). It is recommended that fructose be limited to 60g per day (Amod *et al.*, 2012:S16).

Evidence from different studies indicated that consumption of high levels of fructose-containing beverages may have an adverse effects on selective deposition of ectopic and visceral fat, lipid metabolism, blood pressure, insulin sensitivity, and de novo lipogenesis, compared with glucose-sweetened beverages (Evert *et al.*, 2013:Online). There is a concern that fructose may also increase serum triglycerides (Evert *et al.*, 2013: Online). Fructose is also not a good option to treat hypoglycemia, as it was found to be least effective in correcting blood glucose as compared with sucrose or glucose (Evert *et al.*, 2013:Online).

**Non-nutritive sweeteners**

Non-nutritive sweeteners reduce energy and carbohydrates intake, and are found not to increase blood glucose, although they might affect blood glucose due to other ingredients in the product (Evert *et al.*, 2013: Online). American Heart Association and ADA scientific statement on non-nutritive sweeteners intake have indicated that there is not enough evidence to conclude that non-nutritive sweeteners' use leads to reduction in body weight or reduction in cardio-metabolic risk factors (Evert *et al.*, 2013: Online). However, the use of artificial sweeteners, which include acesulfame-K, aspartame, saccharine and sucralose are safe when consumed within the daily limit recommended by the Food and Drug Association (FDA) (Amod *et al.*, 2012:S16).

**b) Protein intake**

There is inconclusive evidence regarding an ideal amount of protein for optimizing glycemic control or improving cardiovascular risk factors to patients with T2DM without kidney disease, and it is recommended that the goals should be individualized (Evert *et al.*, 2013: Online; Amod *et al.*, 2012:S16). But for T2DM patients with kidney disease (micro- or macro-albuminuria), to reduce the amount of protein intake below the usual intake (< 0.8-1.0g/kg body weight) is not recommended as it does not alter glycemic control, cardiovascular risk factors or the course of glomerular filtration rate decline (Evert *et al.*, 2013: Online).

Hence, the recommendation for protein intake is 15-20% of the total energy intake per day for patients with T2DM without renal problems (ADA, 2013a:S22; Amod *et al.*, 2012:S16; Franz, 2012:686), and has been shown to improve glycemic response, reduce lipids levels and hormones, to have no long term effect on insulin requirements (Franz, 2012:686). While in

patients with signs of renal damage (micro-albuminuria), protein intake should be limited to 0.8-1.0g per body weight per day (ADA, 2013a:S35; Beasley & Wylie-Rosett, 2013: Online), and to 0.8g per body weight per day in the later stage of renal damage (ADA, 2013a:S35; Beasley & Wylie-Rosett, 2013: Online).

Furthermore, among patients with T2DM, protein intake seems to increase insulin response without increasing plasma glucose concentrations (Evert *et al.*, 2013: Online; Amod *et al.*, 2012:S16). Thus carbohydrate sources high in protein should not be used to treat or prevent hypoglycemia (Evert *et al.*, 2013: Online; Amod *et al.*, 2012:S16). Several randomized clinical trials comparing protein levels in patients with diabetic kidney disease, four of the studies reported no difference in glomerular filtration rate and/or albumin excretion rate, while only one study found some potentially beneficial renal effects with a low-protein diet (Evert *et al.*, 2013: Online).

High protein diets play an important role in lowering blood lipids, body composition and weight loss, but only for a short period of time (Weickert, 2012: Online). Due to the satiating effect of dietary protein, a higher protein intake may help to reduce energy intake (Weickert, 2012: Online). The Diet, Obesity and Genes (DiOGenes) trial, a European multicentre study indicated that high protein diets (and high-GI carbohydrates) may increase inflammation, and eventually worsen insulin resistance in the whole body (Weickert, 2012: Online).

**Red meat intake**

The adverse effects of red meat on T2DM patients can be best explained by the heme-iron, which is found in red meat (Ajala *et al.*, 2013: Online; de Koning *et al.*, 2011: Online). Iron is a strong pro-oxidant that catalyses several cellular reactions in the production of reactive oxygen species, thus increases the level of oxidative stress (Ajala *et al.*, 2013: Online). Production of oxidants can cause damage to tissues, particularly the pancreatic beta cells, hence high concentrations of iron in the body has been associated with an elevated risk of T2DM (Ajala *et al.*, 2013: Online; de Koning *et al.*, 2011: Online). Excessive intake of red meat may increase concentrations of inflammatory mediators and gamma-glutamyltransferase (Ajala *et al.*, 2013: Online).

There was a positive association seen between red meat and/or processed meat consumption and risk of stroke, and the risk increased by 15% for red and processed meat intake (Chen *et al.*, 2013: Online). Heme iron has also been suggested to increase risks of atherosclerosis, T2DM and coronary heart disease, all of which contribute to increase the risk of stroke (Chen *et al.*, 2013: Online). Red meat was found to be positively associated with risk of blood pressure and to increase the incidence of hypertension, the comorbid of T2DM (Chen *et al.*, 2013: Online).

Processed meats contain saturated fat, sodium and nitrites (chemicals used in preservation of processed meats), which together with nitrates are converted to nitrosamines (Ajala *et al.*, 2013: Online; de Koning *et al.*, 2011: Online). Furthermore, nitrites and nitrates are converted to nitrosamines (through interaction with amino compounds either in the stomach and intestines or within the food product), and nitrosamines have been shown to be toxic to pancreatic beta cells (Ajala *et al.*, 2013: Online; de Koning *et al.*, 2011: Online). High concentrations of the nitrites in the blood of adults have been shown to be related to endothelial dysfunction and impaired insulin response (Ajala *et al.*, 2013: Online). A prospective study in Finland demonstrated that the association of processed meat and incidence of diabetes was mainly due to the presence of high sodium concentration (Ajala *et al.*, 2013: Online). While advanced glycation end products formed during cooking of meat at high temperatures may also induce insulin resistance (de Koning *et al.*, 2011: Online).

**Dairy intake**

The milk and dairy products intake are associated with an increase in HDL cholesterol and reduction in blood pressure, as well as many other disease processes including diabetes (Elwood *et al.*, 2010: Online). Several studies in adults have shown that dairy product intake is inversely associated with the metabolic syndrome and T2DM (O'Connor *et al.*, 2013: Online; Malik *et al.*, 2011: Online). The intake of dairy products from adolescent until adulthood is demonstrated to be significantly associated with lower incidences of T2DM (Malik *et al.*, 2011: Online).

It is possible that high dairy consumption is associated with an overall healthy diet and lifestyle, which may track throughout the lifecycle and ultimately lowers the risk of T2DM.

However, consumption of dairy products has been displaced by sugar-sweetened beverages, which are associated with obesity and T2DM (Malik *et al.*, 2011: Online).

The components found in dairy products (high-quality proteins, vitamin A, vitamin D, vitamin $B_{12}$, menaquinones-vitamin $K_2$, riboflavin, calcium, magnesium, potassium and lactose) may be responsible for the beneficial effects (O'Connor *et al.*, 2013:Online; Malik *et al.*, 2011: Online). For instance, milk proteins such as whey may have insulinotropic (de Koning *et al.*, 2011: Online) properties with a relatively low glycemic load, which may improve glucose tolerance (Malik *et al.*, 2011: Online). Menaquinones are found in fermented foods, and probiotic bacteria have been shown to improve the lipid profile and antioxidant status in patients with T2DM (O'Connor *et al.*, 2013: Online). While other dairy product components, including medium-chain fatty acids, calcium, and magnesium may reduce insulin resistance or inflammation and lowers oxidative stress (Malik *et al.*, 2011: Online; de Koning *et al.*, 2011: Online).

**Probiotics**

The human gastrointestinal tract contains bacteria, which affects the expression of various host genes that regulate nutrient uptake, metabolism, mucosal barrier function, development of the enteric nervous system and maturation of mucosal immunity (Alokail *et al.*, 2013: Online). A diet that normalizes the gut microflora and metabolic functions is beneficial in the management of most chronic diseases, particularly T2DM (Alokail *et al.*, 2013: Online).

Therefore, addition of probiotics in the diet of patients with T2DM will influence the gut microflora, hence affect the systemic effect of endotoxin (are derived from the Gram negative bacteria in the gut, and act as important mediator of inflammation in liver disease), and low levels of endotoxin lead to reduction in inflammatory cytokines and inflammatory response) (Alokail *et al.*, 2013: Online).

**c) Fat intake**

There is inconclusive evidence in regard to an ideal amount of total fat intake for people with T2DM. It is therefore suggested that goals should be individualized as the fat quality is more important than quantity (Evert *et al.*, 2013: Online). There is no adequate intake or recommended daily allowance for total fat intake for T2DM, but IOM define an acceptable

macronutrient distribution range (AMDR) for total fat of 20-35% of total energy with no tolerable upper intake level defined (Evert *et al.*, 2013: Online).

Dietary fat refer to a mixture of saturated (SFA), trans-saturated (TFA), mononunsaturated (MUFA) and polyunsaturated fatty acids (PUFA), PUFA are further classified as omega-3 and omega-6 polyunsaturated fatty acids (Evert *et al.*, 2013: Online; Wickert, 2012: Online). The type of fatty acid consumed is more important in controlling metabolic goals and influencing the risk of cardiovascular disease than total fat in the diet (Evert *et al.*, 2013: Online). Thus, that high intakes of total fat (>37% of total energy) worsen insulin resistance due to the interference with the binding of insulin to its receptors and accumulation of triglycerides in the skeletal muscle. But with the intake of < 30% the opposite effects occur (Wickert, 2012: Online).

Therefore, the type of fat should be prioritized when individualizing goals and all patients with diabetes should be encouraged to take fat moderately, in consistent with their goals to lose or maintain weight (Evert *et al.*, 2013: Online). The recommendations include: the total fat intake should be restricted to < 35% of total energy per day, reducing SFAs to < 7% of total energy per day, MUFAs to < 10% of total energy per day, while PUFAs intake should be < 10% of the total energy intake per day, and TFAs to < 1% of total energy, and aiming for < 300mg of total dietary cholesterol per day (Evert, 2013:11; Amod *et al.*, 2012:S16).

**Saturated fatty acids, dietary cholesterol, and trans-fats**

Intake of saturated fatty acids causes an interference with cell membrane function, inflammatory pathways, endoplasmatic reticulum stress and toxic effects on pancreatic beta-cells, which may eventually result in insulin resistance (Weickert, 2012: Online). Few research studies have explored the relationship between the amount of saturated fatty acids in the diet and glycemic control, and cardiovascular risk in patients with diabetes (Evert *et al.*, 2013: Online). In one systemic review that compared a low-saturated diet (8% of total energy) with a high-saturated diet (17% of total energy), there was no significant difference in glycemic control and cardiovascular risk factors (Evert *et al.*, 2013: Online).

The adverse effects of total fatty acids on cardiovascular disease are well known, but their role in the development of insulin resistance and T2DM is not well established (Evert *et al.*,

2013: Online). The Nurses, Health Study showed a dose-dependent association between total fatty acid intake and risk of T2DM (Evert *et al.*, 2013: Online). For instance a high intake of milk and dairy products, which contain about 70% of saturated fatty acid, has been associated with reduced diabetes risk (Weickert, 2012:Online). However, studies on low-fat and fat-free dairy products suggested that fat from the dairy products cannot be the driving factor for reduced diabetes risk (Weickert, 2012: Online).

One large prospective cohort study also found a 37% increase in cardiovascular risk for every 200mg cholesterol/1000kcal (Evert *et al.*, 2013: Online). Therefore, it is recommended that patients with T2DM should consume 10% of calories from saturated fatty acids, aiming for 300mg dietary cholesterol/day, and limiting intake of trans-fats in order to reduce cardiovascular risk (Evert *et al.*, 2013: Online). This could be achieved by replacing foods high in saturated fatty acids (full cream milk and full cream dairy products, butter, bacon, coconut or palm oils) with foods rich in MUFAs and PUFAs (vegetable and nut oils, including canola, corn, soy, and sunflower, vegetable oil spreads, and avocados) (Evert *et al.*, 2013: Online).

**Monounsaturated fatty acids/polyunsaturated fatty acids**

T2DM patients may benefit with a Mediterranean-style and monounsaturated fatty acids-rich eating pattern, as they are shown to improve glycemic control and reduce cardiovascular risk factors (Evert *et al.*, 2013: Online). Evidence from prospective cohort studies, clinical trials, and systematic review of randomized controlled trials indicated that MUFAs diets are linked to improved glycemic control and cardiovascular risk factors (Evert *et al.*, 2013: Online).

The replacement of saturated fatty acids by MUFAs is shown to reduce insulin resistance by 10% (Weickert, 2012: Online). MUFAs improvement of insulin resistance may be the effects of cell membrane fatty acid composition, with functional effects on membrane fluidity, ion permeability, insulin receptor binding/affinity and up regulation of glucose transporters (Weickert, 2012: Online). However, MUFA are not associated with reduced risk of T2DM in prospective cohort studies (Weickert, 2012: Online).

In 2011, the Evidence Analysis Library (EAL) of the academy of Nutrition and Dietetics demonstrated that replacing saturated fatty acids with monounsaturated fatty acids (MUFAs)

by 5% energy improves insulin sensitivity and insulin-resistant in T2DM subjects (Evert *et al.*, 2013:Online). However, there is limited information on the effects of omega-6 polyunsaturated fatty acids (PUFAs) in patients with T2DM (Evert *et al.*, 2013: Online). De Koning *et al.* (2011: Online) indicated that PUFAs from vegetable oils and nuts may reduce postprandial triglycerides and increase skeletal muscle cell membrane fluidity and glucose uptake.

There is also a controversy about the best ratio of omega-6 to omega-3 fatty acids; but PUFAs and MUFAs are recommended fatty acids rather than saturated or trans-fats (Evert *et al.*, 2013: Online; Amod *et al.*, 2012:S16). In humans studies, intake of omega-6 PUFA showed that it might improve insulin resistance and probably also diabetes risk, while results for omega-3 PUFAs are inconsistent (Weickert, 2012: Online).

**Omega-3 fatty acids**

It is recommended that patients with T2DM should increase their intake of foods containing long-chain omega-3 fatty acids from fatty fish (EPA and DHA) and omega-3 linolenic acid (ALA), as they are shown to have beneficial effects on lipoproteins, prevent heart diseases, and are associated with positive health outcomes (Evert *et al.*, 2013: Online). Patients with T2DM are advised to eat fish at least two times per week (Evert *et al.*, 2013: Online) in order to obtain the recommended amounts of omega-3 PUFAs (Amod *et al.*, 2012:S16).

In one of the longest trials, in which T2DM patients were supplemented with 1g/day of omega-3 fatty acids compared with placebo, there was no reduction in the rate of cardiovascular events or death from any cause (Evert *et al.*, 2013: Online). While in randomized controlled trials, omega-3 fatty acids supplementation did not improve glycemic control, and cardiovascular risk factors were not altered, however higher doses) of omega-3 fatty acid decreased triglycerides levels in the blood (Evert *et al.*, 2013: Online).

Studies using omega-3 fatty acids had shown mixed effects on fasting blood sugar and HbA1c levels (Evert *et al.*, 2013: Online). However, one study reported that omega-3 rich diets and Omega-6 diets had no detrimental effects on glucose measures, and both diets improved insulin sensitivity and lipoprotein profiles (Evert *et al.*, 2013: Online). Furthermore, Carter *et al*. (2010: Online) indicated that the fatty acid profile of the diet is

essential in determining the fatty acid composition of the phospholipid bilayer, which is related to insulin sensitivity within skeletal muscle (Carter *et al.*, 2010: Online).

**Plant stanols and sterols**

Plant stanols and sterols are the type of fats that are shown to benefit patients with T2DM and dyslipidemia, as consumption of 1.6-3.0g/day of plant stanols or sterols could reduce total and LDL cholesterol (Evert *et al.*, 2013: Online). Plant sterol and stanol esters block the intestinal absorption of dietary and biliary cholesterol (Evert *et al.*, 2013: Online). The EAL from the Academy of Nutrition and Dietetics recommends that individuals with dyslipidaemia should include 2-3g of plant sterol and stanol esters per day (from plant sterol and stanol ester enriched foods including many spreads, dairy products, grains and bread products, and yoghurt) as part of the cardio-protective diet (Evert *et al.*, 2013: Online).

**d) Fruits and vegetables intake**

Fruits and vegetables are rich in polyphenols, which are natural phytochemical compounds in plant-based foods (Bahadoran *et al.* (2013: Online). Polyphenolic compounds (for example phenolic acids, flavonoids, stilbenes, lignans, and polymeric lignans) have been identified in whole plant foods, and are secondary metabolites of plants that act as a defense against ultraviolet radiation, oxidants and pathogens (Bahadoran *et al.* (2013: Online).

Polyphenols act as antioxidants, anti-allergic, anti-inflammatory, anti-viral and anti-microbial, anti-proliferative, anti-mutagenic, anti-carcinogenic, free radical scavenging, regulation of cell cycle arrest, apoptosis, and induction of antioxidant enzymes (Bahadoran *et al.*, 2013: Online). Fruits and vegetables high phenolic acid content include berry fruits, kiwi, cherry, apple, and pear, black grapes, strawberries, blueberries, red cabbage, onions, curly kale, leeks, broccoli, soybeans and soy products (Bahadoran *et al.*, 2013: Online).

Polyphenols has hypoglycemic effects, which involve reduction of intestinal absorption of dietary carbohydrate, modulation of the enzymes involved in glucose metabolism, improvement of ß-cell function and insulin action, stimulation of insulin secretion, and the antioxidative and anti-inflammatory properties of these components (Bahadoran *et al.*, 2013: Online). Polyphenols (particularly flavonoids, phenolic acids and tannins), work on carbohydrates metabolism by inhibiting α-glucosidase and α-amylase, the key enzymes

responsible for digestion of dietary carbohydrates to glucose (Bahadoran *et al.*, 2013: Online). Some polyphenols are able to regulate the key pathways of carbohydrate metabolism and hepatic glucose homeostasis including glycolysis, glycogenesis and gluconeogenesis, which are usually impaired in diabetes (Bahadoran *et al.*, 2013: Online).

"Oxidative stress is defined as imbalance between the generation of free oxygen radicals and the antioxidant defence system and results from increased production of reactive oxygen species known to trigger cytotoxic reactions that are damaging to membrane lipids, proteins, nucleic acids and carbohydrates" (Gariballa *et al.*, 2013: Online). Several studies have linked oxidative stress with obesity, diabetes, and other related complications, and green leafy vegetables intake has been reported to reduce the risk of T2DM. In addition dietary intake of antioxidants (through a high intake of fruits and vegetables) may lower development of metabolic syndrome features, which include adiposity, hypertension, hyperglycaemia, dyslipidaemia, and some inflammatory biomarkers in healthy people (Gariballa *et al.*, 2013: Online).

Carter *et al.* (2010: Online) demonstrated that an increased amount of green leafy vegetables in an individual's diet could help in reduction of T2DM. Thus, several studies examining dietary patterns and incidence of T2DM have consistently shown that fruits and vegetables are important components of the dietary patterns associated with a decreased risk of T2DM. Carter *et al.*, 2010: Online, also stated that 1.15 servings of green vegetables (such as spinach, cabbage) per day were associated with 14% decrease in incidence of T2DM.

This benefit is thought to be contributed to the presence of antioxidants (ß carotene and vitamin C, magnesium and polyphenols), which play a major role in reduction of systemic oxidative stress (Bahadoran *et al.*, 2013: Online). Green leafy vegetables also contain α-linolenic acid (which is an omega-3 PUFAs), which are essential in determining the fatty acid composition of the phospholipid bilayer, and is related to insulin sensitivity within skeletal muscle (Carter *et al.*, 2010: Online). Furthermore, Carter *et al.* (2010: Online) indicated that "foods" rather than isolated components such as antioxidants are beneficial for health.

**e) Micronutrients and herbal supplements intake**

Even though uncontrolled diabetes is often associated with micronutrient deficiencies, patients with diabetes should be made aware of the importance of acquiring daily vitamin and mineral requirements from natural food sources and a balanced diet (Evert *et al.*, 2013: Online). It is recommended that meal plans should contain optimal nutrients to meet the recommended dietary allowance (RDAs) or dietary reference intake for all micronutrients (DRIs) (Evert *et al.*, 2013: Online).

There is no evidence to support routine supplementation with vitamins and minerals, as well as use of herbal products in patients with T2DM who do not have the underlying deficiencies (Evert *et al.*, 2013: Online). However, the elderly, pregnant or lactating women, vegetarians, and those on energy-restricted diets, a multivitamin supplement may be necessary (Evert *et al.*, 2013: Online; Amod *et al.*, 2012:S16; Franz, 2012:687).

Green tea is a polyphenol and could interact with absorption of glucose from the intestines via inhibition of $Na^+$ dependent glucose transporters, and is also able to regulate postprandial glycemia and inhibit the development of glucose intolerance by a facilitated insulin response and secretion of glucose dependent insulinotropic polypeptide, and glucagon like polypeptide-1 (GLP-1) (Bahadoran *et al.*, 2013: Online). However, herbal products are not standardized, hence vary in the content of active ingredients and may interact with other medications (Evert *et al.*, 2013: Online).

**f) Sodium intake**

The main source of sodium in the diet is the salt added in food during cooking or at the table, and also contained in packaged and processed foods (Amod *et al.*, 2012:S16). There are limited studies published on the benefits of sodium reduction in patients with diabetes, and randomized controlled trials indicated that decreasing sodium intake reduces blood pressure in patients with diabetes (Evert *et al.*, 2013: Online). Following the DASH diet and reducing sodium intake to about 2300mg led to improvements in blood pressure and other measures on cardiovascular risk factors (Evert *et al.*, 2013: Online).

There is no scientific evidence proving the benefit of low sodium diet in patients with both diabetes and hypertension, but intake lower that 2300mg/day should be considered on an

individual basis, considering the palatability, availability, and additional cost of specialty low sodium products (Evert *et al.*, 2013: Online; Amod *et al.*, 2012:S16). Meta-analysis, clinical trials and expert committees support the role of reduced sodium intake, modest weight loss (4-5kg), increased physical activity, a low-fat diet that includes fruits, vegetables and low-fat dairy products, and moderate alcohol intake, in reducing blood pressure, which is a comorbid condition commonly found in T2DM (Evert *et al.*, 2013).

Therefore patients with T2DM are advised to reduce the intake of salt, however, the association between dietary salt intake and mortality outcomes has not been well established (Ekinci *et al.*, 2011: Online). High salt intake was to be independently associated with all-cause and cardiovascular mortality in patients with T2DM such that the highest mortality risks were observed in individual with the lowest sodium intake (Ekinci *et al.*, 2011: Online).

**g) Alcohol intake**

Epidemiological data showed some improved glycemic control, cardiovascular risk and mortality with moderate alcohol intake in patients with T2DM (Evert *et al.*, 2013: Online). However, alcohol may place patients who use insulin and insulin secretagogue at increased risk for delayed hypoglycemia (consuming alcohol with food can minimize the risk of nocturnal hypoglycemia) (Evert *et al.*, 2013: Online, Amod *et al.*, 2012:S16).

The recommendation for alcohol consumption for people with diabetes include: adults with T2DM who choose to take alcohol should limit their intake to one serving or less per day for females and two servings or less per day for males (Evert *et al.*, 2013:Online; Amod *et al.*, 2012:S16). Amod *et al.*, (2012:S16) showed that drinking alcohol in moderation will not result in an increase in blood sugar or hypoglycemia.

Excessive amounts of alcohol (>3 drinks per day) consumed on a consistent basis may contribute to hyperglycemia (Evert *et al.*, 2013: Online). One alcohol-containing beverage is defined as 330ml of beer, 150ml wine, 25ml spirit, and 50ml fortified wine, sherry or port, each containing approximately 15g of alcohol (ADA, 2013a:S23; Evert *et al.*, 2013:Online; Franz, 2012:687). However, patients with T2DM should be advised to abstain from alcohol, particularly those with a history of alcohol abuse or dependence, women during pregnancy,

and people with medical conditions such as liver disease, pancreatitis, advanced neuropathy, or severe hypertriglyceridimia (Evert *et al.*, 2013: Online).

#### 2.6.2.2 Weight loss

Three out of four adults with diabetes are overweight, while half of patients with diabetes are obese (Evert *et al.*, 2013: Online). There is a relationship between body adiposity and insulin resistance, therefore weight loss, and maintenance of weight are the major recommendations in the management of T2DM (Evert *et al.*, 2013: Online). But use of some medications (for example insulin, insulin secretagogues, and thiazolidinediones) has made it difficult for patients with diabetes to reduce weight (Evert *et al.*, 2013: Online).

However, apart from medications, most patients with T2DM do regain their lost weight, and other factors that are thought to contribute to the inability of maintaining the lost weight are a reduction in glucosuria (resulting in retention of extra energy which might be lost as an effect of therapeutic intervention), changes in food intake or energy expenditure, socioeconomic status, an unsupportive environment, and also due to physiological changes (for an example compensatory changes in circulating hormones that encourage weight gain) (Evert *et al.*, 2013:Online).

Weight loss is an important therapeutic intervention on all overweight and obese individuals who have T2DM (ADA, 2013a:S13; Mann & Morenga, 2013: Online). In order to lose weight, T2DM should follow a healthy lifestyle change, including reduction in energy intake (regular distribution of meals with equal carbohydrate intake), consumption of less saturated fats, trans-fats, cholesterol and sodium, and increasing physical activity (Evert *et al.*, 2013:Online; ADA, 2013a:S13). Patients with T2DM should follow intensive lifestyle interventions (nutrition therapy, physical activity, and behavior change) with ongoing support, particularly to patients who are early in the disease process (Evert *et al.*, 2013: Online).

Short-term studies have demonstrated that moderate weight loss (5-10% of body weight) in people with T2DM is associated with decreased insulin resistance, improved glycemic control and dyslipidemia, and reduced blood pressure (ADA, 2013a:S23; Evert *et al.*, 2013: Online; Mann & Morenga, 2013: Online; Bergman *et al.*, 2011:1084). The optimal rate of weight

loss should be between one to two kilograms per month (Amod *et al.*, 2012:S55). To reduce weight the energy restriction is more important than the quality adjustments (Amod *et al.*, 2012:S55). But very low-energy diets (3360kJ per day) should be prescribed at centers with experience (preferably as part of bariatric surgery work-up) (ADA, 2013a:S23).

Patients with T2DM could achieve a good nutritional status and glycemic control by maintaining a normal BMI of ≤ 25kg/m², waist circumference (WC) of ≤ 94 cm in males and ≤ 80 cm in females (cutoffs for metabolic complications and insulin resistance recommended by the IDF for sub-Saharan populations), and a healthy body adiposity index (BAI), which is 20%-38% in females and 8%-25% in males (Bergman *et al.*, 2011:1084; Alberti, 2009:1643).

The above interventions are part of an effort to improve glycemic control, dyslipidaemia and blood pressure (ADA, 2013a:S13; Amod *et al.*, 2012:S20). Evidence suggests that commercial weight-loss programs (for example low-carbohydrate, low-fat calorie-restricted or Mediterranean diets) that follow healthy-eating principles can be an effective strategy for weight-loss (ADA, 2013a:S13). However, restrictive and fad diets (carb-free, high protein, fat-free etc) should be avoided as they offer no long term benefit over conventional healthy eating plans (Amod *et al.*, 2012:S15).

The patients with T2DM who maintain ideal body weights, might achieve normal blood glucose levels, improved metabolic profile, and lower incidences of complications (Evert *et al.*, 2013: Online; Norris *et al.*, 2009:470). In general, greater weight loss has been associated with a larger magnitude of improvement in $HbA_{1c}$ levels and fasting blood glucose concentration (Evert *et al.*, 2013: Online; Norris *et al.*, 2009:470). These findings suggest that weight loss may enhance the efficacy of medications (hypoglycemic agent) to induce clinically relevant improvements in T2DM management (Norris *et al.*, 2009:470).

The Look Action for Health in Diabetes (AHEAD), which is a large clinical trial designed to determine whether long-term weight loss will improve glycemic control and prevent cardiovascular events in subjects with T2DM (Evert *et al.*, 2013:Online; ADA, 2013a:S23). After one year, the results of the intensive lifestyle intervention showed an average of 8.6% weight loss, significant reduction on $HbA_{1c}$, and reduction in several cardiovascular risk

factors (increase in HDL cholesterol, a decrease in triglycerides and blood pressure), and those benefits were sustained at four years (Evert *et al.*, 2013:Online; ADA, 2013a:S23).

After 11 years, there was no difference in the primary cardiovascular outcome between weight loss and standard care group, and cardiovascular risk factors were improved with weight loss, and participants were on fewer medications to achieve these improvements (ADA, 2013a:S23), and experienced several additional health benefits (for example reduced sleep apnea, depression, urinary incontinence and generally improved health-related quality of life) (Evert *et al.*, 2013: Online).

The Look AHEAD study demonstrated that weight loss strategies associated with lower BMI in overweight or obese patients with T2DM should include a weekly self weighing, regular consumption of breakfast, and reduced intake of fast foods (Evert *et al.*, 2013: Online). Other strategies included are increasing physical activity, reducing portion sizes, using meal replacements, and encouraging patients with T2DM to eat those foods with the greatest consensus for improving health (Evert *et al.*, 2013: Online).

The literature does not support any particular nutrition approach to reduce weight, and the macronutrient intake to support reduction in excess weight has not been established (Evert *et al.*, 2013: Online). A weight loss of 6kg (approximately 7% to 8.5% loss of initial body weight), regular physical activity, and frequent contact with RDs proved to be the beneficial recommendations of weight loss interventions (Evert *et al.*, 2013: Online).

According to Canadian Diabetes Association and ADA guidelines obesity in T2DM can be managed through lifestyle measures, pharmacotherapy and surgical procedures (bariatric surgery) (ADA, 2013a:S27). Bariatric surgery is recommended for morbidly obese T2DM patients (BMI >35kg/m$^2$) or those with BMI between 30kg/m$^2$ and 35kg/m$^2$, who cannot adequately control their blood glucose by medical regimen, and in the presence of other major cardiovascular risk factors (ADA, 2013a:S27).

### 2.6.3 Physical activity

The WHO, 2004 recommendations (based on physical activity recommendations by the US Department of Health and Human Services and American College of Sports Medicine-

ACSM) for physical activity levels for promoting and maintaining health among adults should be a structured or unstructured character at moderate-intensity (>50-70% of maximum heart rate, examples include cycling, brisk walking, dancing, swimming, gardening/raking leaves) for 150 minutes per week or 75 minutes of vigorous-intensity (> 70% of maximum heart rate), examples are brisk walking up an incline, jogging, aerobics, hockey, basketball, fast swimming, fast dancing) activity throughout the week as a means of health enhancement (ADA, 2013a:S24; Khalaf *et al.*, 2013: Online; Amod *et al.*, 2012:S18).

While ADA, (2013a:S24) recommends that adults with T2DM should be advised to perform at least 150 minutes per week of moderate-intensity aerobic physical activity (50%-70% of maximum heart rate), for at least three days per week with no more than two consecutive days without exercise. In the absence of contraindications, adults with T2DM should be encouraged to perform resistance training at least two times per week and it is important to guide the patients on adjusting medications (particularly insulin) and/or inclusion of snacks (carbohydrates) during the physical activity (ADA, 2013a:S24; IDF, 2012: Online).

Exercise forms part of the diabetes management plan, and regular exercise has been shown to improve blood glucose control, reduce cardiovascular risk factors, contribute to weight loss, and improve the overall well-being of patients with T2DM (ADA, 2013a:S25; Amod *et al.*, 2012:S18). Randomized controlled trials have indicated that patients with T2DM benefit more from physical activity as it improves glycemic control and reduces cardiovascular risk factors, and may lower medications dosages (Amod *et al.*, 2012:S18). Regular physical activities have been shown to improve symptoms of depression and improve the overall well-being of T2DM patients (Amod *et al.*, 2012:S18). Large cohort studies have demonstrated that regular physical activity and moderate to high levels of cardio-respiratory fitness are associated with reductions in cardiovascular and overall mortality of 39-70% over a period of 15 to 20 years (Amod *et al.*, 2012:S18).

Eventually patients with T2DM are to have the following benefits from physical activity: increased cardio-respiratory fitness, improved glycemic control, decreased insulin resistance, improved blood pressure, maintenance of weight loss, reduced abdominal and overall fat percentage, improved well-being and decreased stress and anxiety, and reduced medication dosages (Amod *et al.*, 2012:S18).

High intensity exercises are shown to improve $HbA_{1c}$ and fitness of the patients with T2DM, and structured exercise interventions of at least eight weeks duration have been shown to lower $HbA_{1c}$ by an average of 0.66% in patients with T2DM, even if there was no change in BMI (ADA, 2013a:S25). Aerobics training (such as brisk walking, cycling, dancing, swimming and running) activates large muscle groups to perform activities, as a result strengthening the heart, lungs, and muscle mitochondria to meet the maximum oxygen demands, eventually improving the cardio-respiratory fitness (Amod *et al.*, 2012:S18; Hovanec *et al.*, 2012: Online).

Resistance training activates the muscular system to generate force against a resistive load, using exercise machines, lifting free-weights (for example dumbbells), or doing calisthenics such as situps, pushups, crunches, and lunges (Hovanec *et al.*, 2012: Online). Resistance training often results in increased muscle mass and improvements in muscular fitness if performed regularly (Hovanec *et al.*, 2012: Online). Resistance training may be more beneficial in patients with T2DM who are overweight and sedentary, older adults, obese, and/or frail as they tend to lose muscle strength (sarcopenia) (Hovanec *et al.*, 2012: Online).

While patients with T2DM, in particular have worse muscle quality, reduced upper and lower body strength, greater visceral adipose content, and higher risk for functional decline and disability (Hovanec *et al.*, 2012: Online). Hence, resistance training is shown to benefit older adults living with T2DM through muscle hypertrophy, enhanced muscle quality, strength gains for greater power development with more effective mobility function, and glycemic profile improvements (Hovanec *et al.*, 2012: Online).

The guidelines suggest that adults should also do resistance training (muscle-strengthening activities that involve all major muscle groups) for two days per week (ADA, 2013a:S24; Amod *et al.*, 2012:S18). The guideline further suggested that adults over the age of 65 years, or those with disabilities, to follow the adult guidelines if possible or (if not possible) be as physically as they are able (ADA, 2013a:S24; Amod *et al.*, 2012:S 18). Clinical trials have demonstrated a strong evidence for the lowering of HbA1c from resistance training in older adults with T2DM (Hovanec *et al.*, 2012: Online). Resistance exercise is shown to improve insulin sensitivity in older males with T2DM to the same or even a greater extent as aerobic exercise (Hovanec *et al.*, 2012: Online).

The comparison of all 50 USA countries, indicated that high rates of walking and cycling to work were associated with, an increased percentage of adults who achieved recommended levels of physical activity, a decreased percentage of adults with obesity, and a lower percentage of adults with diabetes (Lachat *et al.*, 2013: Online). Promotion of walking and cycling instead of the use of motor vehicles will reduce vehicular emissions, which are risk factors for cardiovascular disease and diabetes (Bhalla, 2013: Online).

Active travelling (walking, cycling and use of public transport) has been linked to reduced risk of overweight, T2DM, and hypertension (Laverty *et al.*, 2013: Online). Meta-analysis of studies done before 2007 demonstrated that active travel to work is associated with an 11% reduction in cardiovascular risk (Laverty *et al.*, 2013: Online). A study done in USA indicated that people who used public transport to commute, walk an average of 19 minutes as part of their journey to work (Laverty *et al.*, 2013: Online). The findings of the study indicated that increasing active travel should be prioritized within national and local prevention strategies for obesity, diabetes, and cardiovascular disease (Laverty *et al.*, 2013: Online).

The Look AHEAD Study by the National Institutes of Health demonstrated that an intensive lifestyle intervention that produces weight loss and improves fitness could slow the loss of mobility in such patients (De Feo & Schwarz, 2013: Online). In 10 000 years ago, people used to walk for > 20 000 steps/day for daily survival. Of late, people walk < 5000 steps/day, burn less energy, gain visceral adiposity, and accumulate chronic diseases such as diabetes (De Feo and Schwarz, 2013: Online). A T2DM patients who walks 1000 steps per day more than their average per day could achieve a reduction of postprandial blood glucose by 1.6mmol/l over a period of 2 years, and help reduce the daily medication (hypoglycemic agents), and other medications such as antihypertensive or lipid-lowering drugs (De Feo & Schwarz, 2013: Online).

It is important that patients with T2DM (particularly those with cardiovascular risk factors for coronary artery disease) are evaluated before commencing any physical activity, and the following conditions are contraindicated to certain types of exercises or can predispose to injury: uncontrolled hypertension, severe autonomic neuropathy, severe peripheral neuropathy or history of foot ulcers, unstable proliferative retinopathy, and orthopaedic

injuries (ADA, 2013a:S25; Amod *et al.*, 2012:S19). High risk patients should be encouraged to start with short periods of low-intensity exercise and increase the intensity and duration slowly (ADA, 2013a:S25). The patient's age and previous physical activity level should be considered (ADA, 2013a:S25; Amod *et al.*, 2012:S19).

However, physical activity can result in hypoglycaemia if the medication dose or carbohydrate intake is not altered, especially in T2DM patients on insulin and/or insulin secretagogues (ADA, 2013a:S25). An intake of additional carbohydrate will be required if preexercise glucose reading is < 5.6mmol/l (ADA, 2013a:S25). Hence, blood glucose measurements before, during and after exercise will assist in the management of snack size and dosage adjustments (Amod *et al.*, 2012:S19). Excess carbohydrate intake during physical activity for T2DM patients taking metformin or alpha-glucosidase inhibitors alone is not necessary (Amod *et al.*, 2012:S19).

### 2.6.4 Medication

During the time of diagnosis management of hyperglycemia in T2DM include lifestyle (MNT and physical activity) and metformin (Franz, 2012:689). Amod *et al.*, (2012:S36) suggested that use of metformin should be given the first choice in the management of T2DM, particularly in overweight patients. It is further suggested that because of its tolerability, metformin should be maintained, even when other classes of drugs are added, and in cases where there are no contraindications (Amod *et al.*, 2012:S36). In situations whereby $HbA_{1c}$ is 7% or more, the next medication to be added can be either sulfonylurea or basal insulin (Franz, 2012:689). Table 2.7 and Table 2.8 depict the treatment algorithm and antihyperglycemic agents used in T2DM.

**Table 2.7:** Treatment algorithm for type 2 diabetes (Amod *et al.*, 2012:S38, Table 1).

| | **Preferred therapies** | **Alternative therapies for special circumstances** |
|---|---|---|
| Lifestyle measures **plus** | | |
| **Step I:** Initiate at least one oral drug at diagnosis | Metformin | Sulphonylureas **or** DPP-4i **or** Acarbase |
| **Step 2:** Combine any two drugs | Metformin + sulphonylureas | Incretin **or** Acarbase **or** Basal Insulin |
| **Step 3:** Combine three drugs | Metformin + sulphonylureas + basal insulin (or metformin + pre-mix insulin) | Metformin + Sulphonylureas + Incretin **or** Metformin + Sulphonylureas + Acarbase |
| **Step 4:** More advanced therapies | Refer to specialist for basal + mealtime insulin ± metformin ± acarbase ± incretin | Metformin + Pre-mix insulin (if not used yet) |

Note: DPP-4i (Dipeptidyl peptidase-4 inhihitors).

**Table 2.8**: Antihyperglycemic agents used in type 2 diabetes (Amod *et al.*, 2012:S39, Table: II).

| **Class** | **Drug (brand name)** | **Effect on $HbA_{1c}$** | **Therapeutic considerations** | **Disadvantages** |
|---|---|---|---|---|
| **Alpha glucosidase inhibitors** | Acarbose (Glucobay) | ↓ | Negligible hypoglycemia risk as monotherapy<br>Non-Systemic effect<br>Weight neutral<br>Targets post-prandial hyperglycemia | Gastrointestinal effects (flatulence, diarrhea)<br>Frequent dosing (meal times) |
| **Biguaride** | Metformin (Glucophage) | ↓↓ | Negligible hypoglycemia risk, as monotherapy<br>Weight neutral as monotherapy, promotes less weight gain when combined with other antihyperglycemic agents, including insulin.<br>Proven reduction in cardiovascular events and mortality in obese subjects.<br>Metformin extended-release formation has better gastrointestinal tolerability and, in the event of metformin intolerance, is preferred to switching to another class of drug. | Frequent gastrointestinal side-effects (diarrhea, abdominal cramping), 5-10% discontinuation<br>Lactic acidosis (rare)<br>Vitamin $B_{12}$ deficiency appears to be more common than initially appreciated<br>Renal impairment: reduce dose to 1000mg/day if estimated glomerular filtration rate (eGFR) <30ml/minute/1.73m² |
| **Incretins** | DPP-4i<br><br><br><br><br><br>GLP-1 agonists | ↓<br><br><br><br><br><br>↓↓ | Negligible hypoglycemia risk, as monotherapy<br>Weight neutral<br>Improves postprandial control<br>Drug-specific recommendations for hepatic and renal disease<br>Negligible hypoglycemia risk, as monotherapy<br>Enhances satiety and causes weight loss<br>Possible potential for improved beta-cell mass and function<br>Avoid initiating therapy in individuals in whom the potential for dehydration poses a considerable risk e.g. trial elderly, multiple co-morbid conditions. | Occasional reports of urticaria and angioedema<br>Cases of pancreatitis observed<br>Newer agents with unknown long-term safety<br>Injectable<br>Initial gastrointestinal/side-effects (nausea, vomiting, diarrhea)<br>Cases of acute pancreatitis observed<br>Liraglutide causes C-cell hyperplasia and medulary thyroid tumours in animals<br>Newer agents with unknown long-term safety. |
| **Insulin Secretagogues** | Sulphonylureas<br>Glibenclamide (Daonil) | ↓↓ | Generally well tolerated<br>Proven reduction in microvascular | Hypoglycemia relatively common, but variable |

| | | | | |
|---|---|---|---|---|
| | Meglitinides | ↓ | end-points (UKPDS and ADVANCE studies), reduction in cardiovascular events and mortality in the long term (UKPDS post-trial monitoring).<br>Relatively rapid glucose-lowering response, useful in the patient at high risk of hypoglycemia e.g. the elderly, renal and hepatic failure. If a sulphonulurea must be used in such individuals, gliclazide modified-release is associated with the lowest incidence of hypoglycemia.<br>Glimepride and gliclazide are associated with less hypoglycemia than glibenclamide.<br>Nateglinide is the least effective secretagogue<br>Targets postprandial glycemia, use if fasting glucose is at target, but $HbA_{1c}$ remains high. Associated with less hypoglycemia than the sulphonulurea in the context of missed meals, useful for patients with unpredictable mealtimes. | Can cause severe hypoglycemia, including episodes, necessitating hospital admission and causing death (particularly glibenclamide, and particularly when renal function is impaired).<br>Causes weight gain (2.5kg), worst with glibenclamide<br>May blunt myocardial ischaemic preconditioning (particularly glibenclamide)<br>Renal impairment: glibenclamide<br>Contraindicated if eGFR <60ml/minute/1.73m², glimeptide and glipizide dose may need to be reduced.<br>Causes hypoglycemia<br>Causes weight gain<br>May blunt myocardial ischaemic preconditioning<br>Frequent dosing (at mealtimes) |
| **Insulin** | Rapid-acting analogues (e.g. Aspart, Lispro)<br>Short-acting regular (Actrapid, Humulin-R)<br>Intermediate-acting (e.g. NPH, Protophane)<br>Long-acting basal analogues e.g. Glargine<br>Pre-mixed human e.g.Actraphane, Humulin 30/70 | Depends on regimen and dosing but up to ↓↓↓ | Potentially greatest $HbA_{1c}$ and no maximal dose.<br>Numerous formulations and delivery systems allow for regimen flexibility.<br>When initiating insulin, consider adding bedtime intermediate-acting insulin or long-acting insulin analogue to daytime oral antihyperglycemic agents (although other regimens can be used) to attain glycemic targets.<br>Premix (biphasic) insulins are somewhat less effective but have wider patient acceptability and appeal. | Significant risk of hypoglycemia<br>Hypoglycemic risk highest with regular and NPH insulin, use analogue insulin in this circumstances.<br>Increased risk of weight gain relative to sulphonylureas and metformin.<br>Injectable.<br>Oedema is usually transit, but can be severe.<br>Initial reports of malignancies with glargine are unverified. |

Note: DPP-4i (Dipeptidyl peptidase-4 inhibitors), GLP-1(Glucagon-Like Peptide-1)

### 2.6.5 Glycemic control and self-monitoring of blood glucose

The main aim of managing blood glucose in T2DM is to prevent or delay the onset of microvascular and macrovascular complications, and this could be achieved through evaluation of $HbA_{1c}$ and blood-glucose levels (ADA, 2013a:S18; Amod *et al.*, 2012:S20). Good glycemic control is defined as achievement of normal blood glucose levels (Amod *et al.*, 2012:S20). Good glycemic control has been shown to reduce diabetes-related complications, hospitalization; mortality, and hence improve patient quality of life; increase employment retention and workplace productivity; and to reduce medical costs and utilization of health care resources (Gavin *et al.*, 2010:5).

This, however requires patients suffering from T2DM to understand the risks associated with the disease as well as the potential benefits of glycemic control and other measures such as maintaining a healthy weight, eating a proper diet, smoking cessation and taking care of the feet (ADA, 2013a:S18). Glycemic control include first and foremost adherence to a prudent diet and lifestyle (adequate levels of physical exercise, prudent use of alcohol, non-smoking) all of which refers to a patient's nutritional status (Gavin *et al.*, 2010:5). Glycemic control is also dependent on adherence to the prescribed medical treatment (oral glucose lowering drugs and where necessary insulin) (Gavin *et al.*, 2010:5; Khattab *et al.*, 2010: Online). The two primary techniques that are used by the health care providers and patients to assess the effectiveness of the management plan on glycemic control include: patient self-monitoring of blood glucose (SMBG) and $HbA_{1c}$ (ADA, 2013a:S17).

The biochemical test of choice for evaluating long term blood glucose control is $HbA_{1c}$ (ADA, 2013a:S17; Amod *et al.*, 2012:S21; Franz, 2012:684; Gavin *et al.*, 2010:5). ADA (2013a:S18) recommended that $HbA_{1c}$ should be tested at least two times per year in patients who are meeting the treatment goals (and who have stable glycemic control), in patients whose therapy has changed or who are not meeting the glycemic goals, $HbA_{1c}$ should be tested every quarter, $HbA_{1c}$ testing should be performed routinely in all newly diagnosed patients with diabetes, at initial assessment and then as part of continuing care.

Thus, $HbA_{1c}$ measurements every three months will determine whether patient's glycemic targets have been met and maintained (ADA, 2013a:S18; Amod *et al.*, 2012:S20). $HbA_{1c}$ is also shown to have a strong predictive value for diabetes complications, and may serve as a check on the accuracy of the patient's glucometer (ADA, 2013a:S18). As a long term monitoring tool of glycemic control, $HbA_{1c}$ reflects adherence to both medication and diet (Amod *et al.*, 2012:S21). $HbA_{1c}$ also helps to provide a guide to treatment; reinforces overall glycemic control and provides patients with information regarding the success of their efforts (Gavin *et al.*, 2010:5). Optimal $HbA_{1c}$ levels in patients with diabetes according to Amod *et al.*, (2012:S21) and the ADA (2013a:S19) should be < 7.0%.

It is shown that reducing $HbA_{1c}$ to less than 7.0% has result in less microvascular complications and if done from the time of diagnosis can result in reduction of macrovascular diseases as well (ADA, 2013a:S19). $HbA_{1c}$ of 6.5% is recommended for selected individual

patients (for example newly diagnosed diabetes patients, patients with long life expectancy, and those without cardiovascular disease), while $HbA_{1c}$ of 8% is recommended for patients with a history of severe hypoglycemia, limited life expectancy, advanced microvascular or macrovascular complications, extensive comorbid conditions, and those with long-standing uncontrolled diabetes despite DSME, appropriate glucose monitoring, and effective doses of oral hypoglycemic agents and insulin.

According to Khattab *et al.* 2010: Online), poor compliance to treatment is often the cause of poor glycemic control. Gavin *et al*. (2010:8) indicated that adherence to medication ranges from 36% to 87% with oral agents, and from 54% to 81% with insulin-only regimens. Good glycemic control is however unlikely to be achieved with insulin or oral therapy when diet is neglected, especially when the patient is also overweight (Mann & Morenga, 2013:453; Maghsoudi & Azadbakht, 2012: Online; Gavin, *et al.*, 2010:5). As indicated by Gavin *et al.* (2010:5) "every drug that has ever been approved for the treatment of T2DM is predicated on being adjunct to lifestyle modification".

Epidemiological evidence has shown that $HbA_{1c}$ level of > 7.5% has a 2.5 to 5-fold greater risk of causing microvascular complications, and a fivefold risk of developing artery disease. Hence, an $HbA_{1c}$ target of 7.0% is the recommended level (Amod *et al.*, 2012:S20). But the IDF and the American College of Endocrinology (ACE) have recommended a target $HbA_{1c}$ level of < 6.5%, and further showed that a low target in patients with long-duration T2DM, will improve the cardiovascular outcomes (Amod *et al.*, 2012:S20).

Therefore it is important to individualize $HbA_{1c}$ targets (Table 15), and for newly diagnosed patients and T2DM patients with cardiovascular disease, an $HbA_{1c}$ target of < 6.5% seems a reasonable target (Amod *et al.*, 2012:S20). While for the elderly, the infirm, those without cardiovascular disease or those with hypoglycemic unawareness, a target of 7.5% (or even up to 8.0%) may be acceptable, and in the majority of patients, an $HbA_{1c}$ target of < 7.0% is reasonable (Amod *et al.*, 2012:S20).

The correlation between $HbA_{1c}$ and average glucose (Table 2.8 and Table 2.9 below) is shown to be strong enough to justify reporting both the $HbA_{1c}$ result and an estimated average

glucose (eAG) result when a clinician orders the $HbA_{1c}$ test, and it is recommended that laboratories should report both values (Amod *et al.*, 2012:S21).

**Table 2.9: Targets for $HbA_{1c}$, fasting plasma glucose and postprandial glucose in different patients (Amod *et al.*, 2012:S21, Table 1).**

| Patient type | Target HbA1c | Target FPG | Target PPG |
|---|---|---|---|
| Young | < 6.5% | 4.0-7.0mmol/l | 4.4-7.8mmo/l |
| Low risk | | | |
| Newly diagnosed | | | |
| No cardiovascular disease | | | |
| Majority of patients | < 7.0% | 4.0-7.0mmol/l | 5.0-10.0mmol/l |
| Elderly | < 7.5% | 4.0-7.0mmol/l | < 12.0mmol/l |
| High risk | | | |
| Hypoglycemic unaware | | | |
| Poor short-term prognosis | | | |

**Table 2.10: Translating $HbA_{1c}$ into average glucose (Amod *et al.*, 2012:S22, Table II)**

| $HbA_{1c}$ (%) | eAG (mmol/l) |
|---|---|
| 6 | 7.0 |
| 7 | 8.6 |
| 8 | 10.2 |
| 9 | 11.8 |
| 10 | 13.4 |
| 11 | 14.9 |
| 12 | 16.5 |

Daily self-monitoring and recording of blood glucose levels are strongly advised for all T2DM patients, but requires expensive glucometers and strips. The frequency will depend on the insulin regimen used, for patients injecting insulin two to four times per day, testing should be undertaken at least three times per day (Amod *et al.*, 2012:S21). While in those patients on once-daily insulin, with or without oral hypoglycemic agents, testing should be performed at variable times (Amod *et al.*, 2012:S21). ADA (2013a:S17) also advised that patients on multiple-dose insulin should do SMBG at least before meals and snacks, occasionally postprandially, at bedtime, before exercises, when patient suspect low blood glucose, after treating low blood glucose until they are normoglycemic, and before any involvement in critical task such as driving.

SMBG results may be helpful to guide treatment decisions and/or patient self-management for patients using less frequent insulin injections or oral hypoglycemic agents. Major clinical trials have suggested that SMBG is an effective component of management, and it allows patients to evaluate their individual response to medication and diet therapy (ADA, 2013a:S17). Patients using oral hypoglycemic agents SMBG is essential, especially in association with $HbA_{1c}$ testing as it provides information on, and help avoid hypoglycemia, it assesses changes in blood glucose control due to medications and lifestyle changes, it monitors the effects of foods on postprandial glycemia, as well as changes in blood glucose levels during intercurrent illness (ADA, 2013a:S17).

However, there is a conflicting evidence in regard to SMBG in patients using oral hypoglycemic agents, but in newly diagnosed patients with T2DM, SMBG together with structured testing and education has been shown to be beneficial (Amod *et al.*, 2012:S21). Thus, IDF advised that SMBG should only be undertaken by patients who have been taught how to incorporate the testing into their diabetes care plan, and those with skills and are willing to use the information (IDF, 2012: Online; Amod *et al.*, 2012:S21).

The recommendation for patients on oral agents include: SMBG should be considered in patients on oral hypoglycemic agents and who have been given an education that accompanies initiation of testing, and three to five tests per week should be sufficient, testing must be structured and have a meaning for the patient (Amod *et al.*, 2012:S21). In addition, patients with T2DM must understand their glycemic targets, and know what to do if the targets are not being achieved (Amod *et al.*, 2012:S21).

The following require more frequent SMBG: acute illness, periods of poor glycemic control, frequent hypoglycemic episodes, pregnancy, and adjustments to therapy (Amod *et al.*, 2012:S21). Patients with hypoglycemia unawareness, those with frequent hypoglycemic episodes, and with discrepant $HbA_{1c}$ and SMBG results, and for additional monitoring during pregnancy (Amod *et al.*, 2012:S21).

## 2.7 Prevention and management of diabetes chronic complications

The mechanism by which diabetes leads to chronic complications is complex, and not yet fully understood, but it is thought to be a result of the toxic effect of high glucose levels, the impact of elevated blood pressure, abnormal lipid levels and both "functional and structural abnormalities" of small and large blood vessels (Robbins *et al.*, 2008:658). Patients with T2DM who attend clinics often present with complications caused by uncontrolled blood glucose which over time damages nerves and blood vessels, which typically causes microvascular diseases (diseases of the small blood vessels e.g. nephropathy, neuropathy, retinopathy) and macrovascular diseases (diseases of the large blood vessel e.g. hypertension and dyslipidemia) (Franz, 2012:704; IDF, 2010:Online; WHO, 2010: Online).

The risk for complications is linked to poor nutritional status (longer duration of diabetes, increased BMI, being sedentary and smoking), poor glycemic control, high diastolic blood pressure, infections, dyslipidemia and poor self-care all of which are modifiable and manageable (ADA, 2013a:S18).

The major chronic complications of diabetes are: cardiovascular disease (CVD) (major cause of death in diabetes and include angina, myocardial infarction, stroke, peripheral artery disease, congestive heart failure), nephropathy (common cause of renal failure in diabetes and of end stage renal disease), neuropathy (uncontrolled blood glucose and blood pressure can harm the nerves resulting in problems with digestion, urination, impotence, loss of feeling in the feet and toes, and legs), retinopathy (diabetes can harm sight and cause blindness, and increases the risk of cataracts and glaucoma) and foot ulceration (result in amputations) (Solomon *et al.*, 2009:1225).

### 2.7.1 Macrovascular diseases

Macrovascular disease affects the large blood vessels, and includes coronary heart disease, peripheral vascular disease, and cerebrovascular disease. Patients with T2DM have a two to three fold for males and three to five fold chance in females of having cardiovascular disease (Amod *et al.*, 2012:S57). The risk factors for cardiovascular diseases are hypertension and dyslipidaemia, which are common conditions that coexist with T2DM (ADA, 2013a:S18).

Dyslipidaemia and hypertension are also shown to be the main cause of macrovascular disease or atherosclerosis (ADA, 2013a:S18).

IDF (2012: Online) indicated that all patients with T2DM aged 60 years and above are at increased risk of cardiovascular diseases and should always be considered for cardiovascular disease prevention measures. Though the benefits of the cardiovascular interventions may take a number of years to show the results (IDF, 2012: Online). Cardiovascular risk factors (for example dyslipidaemia, hypertension, smoking, a positive family history of premature coronary disease, and the presence of micro-or macro-albuminuria) as summarized in Table 2.10, should be assessed at least annually in all patients with diabetes (Amod *et al.*, 2012:S57).

#### 2.7.1.1 Hypertension

Hypertension is a common comorbidity of T2DM, and the prevalence depends on type of diabetes, age, obesity, and ethnicity (ADA, 2013a:S18; Franz, 2012:704). Therefore, it is recommended for every patient, that the blood pressure be measured at every routine visit (ADA, 2013a:S18; Franz, 2012:704). Patients with T2DM and hypertension should be treated to a systolic blood pressure of 140mmHg and diastolic blood pressure of 80mmHg (ADA, 2013a:S18).

Treatment of patients with blood pressure of 120/80mmHg should be advised on lifestyle changes (weight loss, if overweight, DASH-style dietary pattern, sodium reduction, increased potassium intake, moderate alcohol consumption, and increased physical activity) to reduce blood pressure, while those with a confirmed blood pressure above 140/80mmHg should be treated with both lifestyle and pharmacological therapy (ACE inhibitors or an angiotensin receptor blocker) in order to achieve blood pressure goals (ADA, 2013a:S18).

Therefore measurement of blood pressure should be done by trained staff, who follows the guidelines established (measurement in the seated position, with feet on the floor and arm supported at heart level, after five minutes of rest, and cuff size should be appropriate for the upper arm circumference (ADA, 2013a:S18). It is recommended that the blood pressure should be measured in both arms at the first consultation, and thereafter in the arm with the higher blood pressure (Amod *et al.*, 2012:S61).

The ACCORD trial demonstrated that lowering blood pressure to systolic blood pressure of 120mmHg, reduced the stroke incidences, and albuminuria, but there were no differences in renal function and other microvascular complications (ADA, 2013a:S18).

Though there are no well-controlled studies of diet and exercise in the treatment of elevated blood pressure in patients with T2DM, the DASH study in people without diabetes has shown antihypertensive effects similar to pharmacological monotherapy (ADA, 2013a:S18). The lifestyle therapy consists of reducing sodium intake to below 1,500mg/day, and excess body weight, increasing consumption of fruits, vegetables (8-10 servings per day), and low-fat dairy products (2-3 servings per day), limiting alcohol intake (no more than one serving per day for females and no more than two servings per day for males), and increasing activity levels. Thus, the glycemic and lipid control may be positively affected when using this strategy, but their effects on cardiovascular events have not yet been established (ADA, 2013a:S18).

#### 2.7.1.2 Dyslipidemia

Dyslipidemia is a common comorbidity of T2DM (ADA, 2013a:S18), and it is suggested that a full fasting lipid profile (total cholesterol, triglycerides, HDL cholesterol and LDL cholesterol) should be tested during the first encounter with the T2DM patient (Amod *et al.*, 2012:S57). If the results of the fasting lipid profile are satisfactory, the next lipid profile should be done once every year (ADA, 2013a:S18; Amod *et al.*, 2012:S57). If the results are unsatisfactory, the test should be repeated in three months, after the patient has been on a lipid lowering diet, weight reduction programme, an established glucose control, and lipid lowering drugs have been instituted (Amod *et al.*, 2012:S57).

Lifestyle intervention for dyslipidaemia should focus on reducing saturated fat, trans-unsaturated fat and cholesterol intake, increase of omega-3 fatty acids, viscous/soluble fiber (for an example oats, legumes, citrus), and plant stanols/sterols, smoking cessation, weight loss (if indicated), and increased physical activity (ADA, 2013a:S18). Nutrition intervention should consider the patient's age, pharmacological treatment, lipid levels, and other medical conditions (ADA, 2013a:S18). It is recommended that for patients with T2DM, statin therapy need to be added to lifestyle therapy, regardless of baseline lipid levels, and in those patients

with clinical cardiovascular disease or who are above the age of 40 years with other cardiovascular disease risk factors (ADA, 2013a:S18).

**Table 2.11: Cardiovascular risk factors and targets for type 2 diabetes (Amod *et al.*, 2012:S57, Table 1)**

| Traditional Cardiovascular risk factors | Targets |
|---|---|
| Cigarette smoking | Cessation |
| **Dyslipidaemia** | |
| Total cholesterol | < 4.5 mmol/l |
| LDL cholesterol | < 1.8 mmol/l |
| HDL cholesterol | < 1.2 mmol/l (females); < 1.0 mmol/l (males); |
| Triglycerides | < 1.7 mmol/l |
| **Obesity** | |
| Waist circumference | < 80 cm (females); < 94 cm (males); |
| Body Mass Index | < 25 kg/m² |
| **Hypertension** | |
| Systolic blood pressure | < 140mmHg |
| Diastolic blood pressure | < 80 mmHg |

Diet is the cornerstone of cardiovascular disease therapy, and all patients with T2DM should be encouraged to receive standard advice on healthy eating habits and recommended food choices from a dietitian/nutritionist with particular emphasis on energy intake, fats, fibre, and alcohol intake (Amod *et al.*, 2012:S57). Table 2.10 summarizes the traditional modifiable risk factors for CVD and the recommended target values in diabetes.

### 2.7.2 Microvascular disease

Microvascular diseases are associated with diabetes and involve the small blood vessels, and include nephropathy, neuropathy and retinopathy (Franz, 2012:704).

#### 2.7.2.1 Nephropathy

Diabetic nephropathy is prevalent in 20-40% of patients with diabetes, and it is the main cause of end-stage renal disease (ADA, 2013a:S35; Franz, 2012:705). Nephropathy is a preventable long-term complication of diabetes mellitus, which result in morbidity and mortality among T2DM patients (Amod *et al.*, 2012:S63). Nephropathy can be delayed or even prevented through regular follow-ups of all patients with T2DM and adhering to a strict treatment protocol (Amod *et al.*, 2012:S63).

Diabetic nephropathy is shown to develop from a subclinical disease to the earliest clinically detectable stage, which is characterized by the presence of proteins in urine (Amod *et al.*, 2012:S63). Recurrent albuminuria (microalbuminuria) between 30-299mg/24hr has been shown to be a marker for development of nephropathy in T2DM, and also a marker of increased cardiovascular risk (ADA, 2013a:S35; Franz, 2012:705). Patients with T2DM who progress to macroalbuminuria are more likely to progress to end-stage-renal disease (ADA, 2013a:S35). Prospective randomized studies have shown that intensive blood glucose control could delay the onset and progression of increased urinary albumin excretion in patients with T2DM (ADA, 2013a:S35).

Patients with T2DM should be screened for renal disease at diagnosis by a random urine sample (for determination of the albumin-to-creatinine ratio) (Amod *et al.*, 2012:S63), and a serum creatinine concentration, for conversion into the estimated glomerular filtration rate (ADA, 2013a:S35; Amod *et al.*, 2012:S63). It is recommended that screening be done annually if the albumin-to-creatinine ratio falls within the normal range, and the estimated glomerular filtration rate is > 60ml per minute (Amod *et al.*, 2012:S63).

To prevent or delay chronic kidney disease, the T2DM patients should control their blood glucose and blood pressure, including other cardiovascular risk factors (for example smoking, dyslipidaemia) (Amod *et al.*, 2012:S63). In order to monitor disease progression and response to treatment, a random urine albumin-to-creatinine and serum creatinine converted into estimated glomerular filtration rate must be performed at least every six months in patients with T2DM and chronic kidney disease (Amod *et al.*, 2012:S63). A potassium restricted diet should be considered in case of a mild to moderate increase in serum potassium concentration (Amod *et al.*, 2012:S63).

ADA, (2013a:S35) suggested that all adults with T2DM patients regardless of the degree of urine albumin excretion, should be screened for serum creatinine at least every year. Thus, the serum creatinine should be used to estimate glomerular filtration rate and measure the level of chronic kidney disease, if present. Nutritional recommendation include: reducing protein intake to 0.8-1.0g/kg body weight per day in individuals with diabetes and in the early stages of chronic kidney disease, and to 0.8g/kg body weight per day in the late stages of

chronic kidney disease, may improve renal function (urine excretion rate, glomerular filtration rate) (ADA, 2013a:S35).

Studies in patients with nephropathy have shown that protein restriction of dietary protein helps slow the progression of albuminuria, glomerular filtration rate decline, and occurrence of end-stage-renal disease, but recently done studies have provided conflicting results (ADA, 2013a:S35; Franz, 2012:705). However, studies suggest that dietary protein restriction might be considered in patients whose nephropathy seems to be progressing despite optimal glucose and blood pressure control and use of antihypertensive drugs (ADA, 2013a:S35).

#### 2.7.2.2 Neuropathy

High levels of blood glucose over a long time results in nerve damage and 60% to 70% of patients with diabetes will have mild to severe forms of nervous system damage (Franz, 2012:706). ADA (2013a:S37) recommended that all T2DM patients should be screened for distal symmetric polyneuropathy, and signs of cardiovascular autonomic neuropathy at diagnosis. Currently there is no specific treatment for underlying nerve damage other than improved glycemic control, which may only delay the progression of neuropathy, but not reverse neuronal loss (Franz, 2012:706).

Peripheral neuropathy usually affects the nerves that control sensation of the feet and hands, while autonomic neuropathy affects nerve function controlling various organ systems (Franz, 2012:706). Peripheral neuropathy cause damage to the nerves innervating the gastrointestinal tract and in the oesophagus causing nausea and oesophagitis, in the stomach as unpredictable emptying, in the small bowel as loss of nutrients, and in the large bowel as diarrhea or constipation (Franz, 2012:706).

The treatment of neuropathy include: maintaining optimal glycemic control, and MNT, this will assist to minimize abdominal stress (Franz, 2012:706). Therefore, small frequent meals may be tolerated well than three meals a day, and the meals should be low in fiber and fat (Franz, 2012:706). Liquid meals may be provided in cases where solid meals are not well tolerated, and for patients using insulin, insulin administration should be matched with the delayed nutrient absorption (Franz, 2012:706).

The signs and symptoms of diabetic autonomic neuropathy include resting tachycardia, exercise intolerance, orthostatic hypotension, constipation, gastroparesis, erectile dysfunction, sudo-motor dysfunction, impaired neurovascular function, and failure in response to hypoglycemia (ADA, 2013a:S38). Therefore, ADA (2013a:S38) recommended that patients with diabetes should be screened annually for distal symmetric polyneuropathy using pinprick sensation, vibration (using a 128-Hz tuning fork), 10-g monofilament pressure sensation at the distal plantar aspect of both great toes and metatarsal joints, and assessment of ankle reflexes.

**i) Foot care**

ADA (2013a:S38), recommended that comprehensive foot examination should be performed for all patients with diabetes, to determine the risk factors of ulcers and amputations. The foot examination should include inspection, assessment of foot pulses, and testing for loss of protective sensation (10-g monofilament plus testing any one of the following: vibration using 128-Hz tuning fork, pinprick sensation, ankle reflexes, or vibration perception threshold). Screening for peripheral arterial disease should include a history for claudication and an assessment of pedal pulses.

It is imperative that all patients with T2DM and high-risk foot conditions are provided with general foot self-care education (regarding their risk factors and appropriate management) (Amod *et al.*, 2012:S63). Patients at risk should also be educated about the implications of the loss of protective sensation, the importance of foot monitoring on a daily basis, the proper care of foot, as well as nail and skin care, and the selection of appropriate footwear (Amod *et al.*, 2012:S63). Patients with visual difficulties, physical constraints or cognitive impairment would need assistance from other members of the family. Patients with neuropathy or evidence of increased plantar pressure (for example callus, erythema) may be managed with well-fitted walking shoes or athletic shoes that cushion the feet and redistribute pressure (Amod *et al.*, 2012:S63).

According to Amod *et al.*, (2012:S69) recommended that general prevention strategies for foot ulceration and amputations should include: optimal glycemic, blood pressure and cholesterol control, and smoking cessation, regular inspection and examination of the shoes (to wear shoes that fit well, and check that the shoes are correct length and width, allow

enough room for toes, have a smooth lining without seams, have a flexible sole that can bend easily, have a heel no higher than 4cm, slip-on shoes and slippers are not recommended) and annual examination of feet (assessment of the skin, bones, nerves and vasculature of the feet), and education about foot care to the patient, family and healthcare providers (Amod *et al.*, 2012:S69).

#### 2.7.2.3 Retinopathy

Diabetes management aiming to achieve normal glycemic control, and lowering of blood pressure has been shown to prevent and/or delay the onset and progression of diabetic retinopathy (ADA, 2013a:S36). Diabetic retinopathy is a highly specific vascular complication of T2DM, with prevalence strongly related to the duration of diabetes, chronic hyperglycemia, nephropathy, and hypertension (ADA, 2013a:S36). Diabetic retinopathy is the most frequent cause of new cases of blindness among adults aged between 20 to 74 years (glaucoma, cataracts, and other disorders of the eye) occur earlier and more frequently in people with diabetes (ADA, 2013a:S36).

Patients with T2DM should have an initial dilated and comprehensive eye examination by an ophthalmologist or optometrist immediately after the diagnosis of diabetes (ADA, 2013a:S18). Retinopathy is the major cause of visual impairment in T2DM, about 40% of patients with diabetes mellitus have retinopathy, and is comprised of cataract and glaucoma formation (ADA, 2013a:S36). Smoking cessation and optimal glycemic and blood pressure control can reduce the incidence of retinopathy in T2DM patients (ADA, 2013a:S36).

Therefore, regularly examining the eyes through dilated pupils and testing of visual acuity could identify patients at risk of sight threatening complications in time (ADA, 2013a:S36). It is recommended that all patients with T2DM should be screened (by indirect opthalmoscopy, direct ophthalmoscopy or fundus photographic methods) for retinopathy at diagnosis (ADA, 2013a:S36). Subsequent examinations should be performed at least annually by an ophthalmologist or optometrist (ADA, 2013a:S36).

Studies show that diabetes complications can be prevented through early effective control of hyperglycemia by proper medication and lifestyles modifications, including treatment of cormorbid conditions (obesity, hypertension and dyslipidemia) (ADA, 2013a:S28). The

severity of diabetes complications is usually influenced by many factors such as few visits for diabetes patient care (ADA, 2013a:S28). Some researchers indicated that some patients with diabetes do source for care from the complementary and alternative practitioners such as traditional healers that rarely refer patients to conventional practitioners, except when complications have worsened (Azevedo & Alla, 2008:104).

### 2.8 Barriers to treatment compliance

Proper management of diabetes would allow the patient to live a completely normal life, and to remain symptom free with good health. Successful management of diabetes requires proper evaluation and understanding of the patients' lifestyles (including perceived barriers), perceptions, beliefs, family and social networks (Gning *et al*, 2007: Online).

Barriers to diabetes care or treatment compliance may include, socio-demographic and cultural barriers such as poor access to drugs, high cost, patient satisfaction with their medical care, patient relationship with the health-workers, degree of symptoms, unequal distribution of health workers between urban and rural areas (Shrivastava *et al.*, 2013: Online). Factors that are found to contribute to patients non-compliance include: lack of adherence to medications, attitudes, beliefs, knowledge about diabetes, culture and language capabilities, health literacy, financial resources, co-morbidities and social support (Shrivastava *et al.*, 2013: Online).

According to Funnell (2006: Online) some patients reported that their treatment was too complicated, while others mentioned that they were tired of taking their medications. The Diabetes Audit and Research in Tayside, Scotland (DARTS) study on the impact of regimen complexity with antidiabetic therapy, confirmed that the more complex the regimen is, the less the adherence to therapy, and advocated for the use of novel formulations, which include a single-tablet combinations (Reasner & Goke, 2002: Online).

Depression and other mental health disorders can prevent patients' ability to comply with treatment, and hence manage their diabetes (Gonzalez *et al.*, 2013: Online; Kruger & Spollett, 2012: Online). Clinical inertia can also be expressed by patients with T2DM, by resisting

treatment intensification (for example resist addition of insulin therapy) (Kruger & Spollett, 2012: Online).

'Clinical inertia', which is described as the failure of health care providers to initiate or intensify treatment when indicated (for example the health care provider may recognize a problem but fail to act) (Kruger & Spollett, 2012: Online). While other factors related to health professionals include attitude, belief and knowledge about diabetes, and effective communication (Shrivastava *et al.*, 2013: Online). In Diabetes, Attitudes, Wishes and Needs (DAWN) study, 43.4% of health care providers indicated that they preferred to delay initiation of medications until it is really necessary (Kruger & Spollett, 2012: Online).

Patients with T2DM are expected to follow a set of behavioural actions to care, and these actions involve engaging in positive lifestyle behaviour, which include following a prescribed meal plan, engaging in appropriate physical activity, taking medications (oral hypoglycaemic agents and insulin) as prescribed, monitoring blood glucose levels, responding to and self-treating diabetes-related symptoms, following foot-care guidelines, and seeking appropriate medical care for diabetes and other health-related problems (Shrivastava *et al.*, 2013: Online; Kruger & Spollett, 2012: Online).

However, it becomes a challenge to encooperate all of these behavioural tasks into a patient's daily routine (Shrivastava *et al.*, 2013: Online). Compliance and adherence to these behaviours have been found to reduce the chances of developing long-term complications, but has been found to be very low among patients with diabetes (Shrivastava *et al.*, 2013: Online). Gavin *et al*. (2010:8) indicated that adherence to medication ranges from 36% to 87% with oral agents, and from 54% to 81% with insulin-only regimens.

One study conducted among people with diabetes, demonstrated that only 30% of the participants were found to be compliant with drug regimens, and the lack of compliance was higher in those patients with low-socioeconomic status (Shrivastava *et al.*, 2013: Online). Shrivastava *et al., (*2013: Online) further indicated that good metabolic control among patients with T2DM would not be achieved through compliance to self-care behaviours alone.

**Summary**

The prevalence of T2DM has risen in Sub-Saharan Africa (including Lesotho) mainly due to the effects of underlying socio-economic status, urbanization, rapid cultural changes, an aging population, high prevalence of obesity and unhealthy lifestyles (poor diet and physical inactivity), and with the epidemic of HIV and AIDS (Pakish *et al.*, 2013: Online; Amod *et al.*, 2012:S85; Levitt *et al.*, 2011: Online). Unfortunately diabetes seems to be very prevalent among the age group of 40-60 years, the age in which people are more productive and thus contribute to the economy of the country.

Though diabetes is an ancient disease, its management is still suboptimal in most Sub-Saharan countries mainly due to limited resources, which are divided between fighting poverty, implementing education strategies, providing housing and appropriate sanitation, and dealing with the socio-economic and health burden of fighting the increasing incidence and prevalence of HIV and AIDS (IDF, 2012: Online; Levitt *et al.*, 2011: Online).

Another important factor impacting on diabetic course in Sub-Saharan Africa is poor access to health care that is, differential access to health care due to various reasons such as transportation difficulties, lack of trained health care providers, limited resources, inadequate health facilities and chronic shortages of drugs (including insulin) and their high cost, some patients with diabetes do source for care from the complementary and alternative practitioners such as traditional healers (Azevedo & Alla, 2008:105).

It thus appears that therapeutic lifestyle modification recommendations together with oral hypoglycemic agents, and insulin, are the basis to improve and maintain good glycemic control in order to minimize diabetic complications (ADA, 2013a:S22). Several studies have indicated that medical nutrition therapy is essential for prevention, treatment and self-management of diabetes, as well as in delaying the onset of diabetes-related complications (ADA, 2013a:S22; IDF, 2013:19; Amod *et al.*, 2012:S22).

There is an urgent need to intensify efforts to ensure follow-up of patients, whenever treatment has commenced in order to reduce and/or prevent the high morbidity and mortality rates from T2DM (Azevedo & Alla, 2008:105). T2DM care requires continuity of care by a

specific group of health care providers and continuity of information by means of hand-held records or electronic records if available, to ensure that even in the event of up-and-down-referral, there will not be a breakdown in the continuity of information, and a register would facilitate patient recall in instances of non-attendance (Levitt *et al.*, 2011: Online).

Consistent drug supplies are central to good chronic care, as well as the ability to prescribe medication for a reasonable duration to avoid repeat visits of the patients to clinics merely to collect medication (Levitt *et al.*, 2011: Online). Management of diabetes require regular monitoring and evaluation, which should include regular auditing of the quality of care provided, with feedback to direct interventions that lead to improvements of care provided, and support for patients with diabetes (Levitt *et al.*, 2011: Online).

# CHAPTER 3: METHODOLOGY

The aim of the study was to determine the nutritional status, glycemic control, and barriers that impact on treatment compliance among patients with T2DM who attend diabetes clinics in Maseru, Lesotho. In this chapter the methodology that was followed to meet the aim and objectives (as stipulated in Chapter 1), is discussed with regard to ethical approval, study design, study population, sample size and selection criteria, measurements, pilot study, procedures, statistical and nutritional analysis, ethical considerations, and challenges/limitations encountered during the study which may impact on the results. All data was collected by the researcher, a registered dietician who was trained at the University of Kwazulu Natal (UKZN) and has been working as a dietician in Maseru, particularly with patients with diabetes, for six years.

## 3.1 Ethical approval and permission to conduct the study

Approval to conduct the research was obtained from the Ethics Committee of the Faculty of Health Sciences of the University of the Free State (UFS) (ETOVS NR 76/2012) and the Ethics Committee of the Ministry of Health (MOH) (ID 45/2012). Permission to perform the study at the DHMT clinics, were obtained from the District Focal Officer for Maseru (Appendix G: A letter requesting for an approval from the District Medical Officer - Maseru district).

## 3.2 Study design

A quantitative descriptive study was conducted.

## 3.3 Study population

Approximately 100 to 150 patients with T2DM used to attend the clinic at QE II Hospital, in Maseru every Wednesday. Given that patients with T2DM attended the clinic once every three months, and that a typical three month period would include thirteen Wednesdays, the total study population was estimated at between 1300 and 2050 patients. After the closure of QE II hospital in October 2011, patients of the diabetes clinic were redirected to the new

QMM hospital clinic, as well as to three other PPP clinics (Ts'epong clinics) and two DHMT clinics around Maseru. Permission could only be obtained however, to conduct the study at the two clinics managed by the DHMT.

## 3.4 Sampling

The following procedures were followed to select the participants for the study.

### 3.4.1 Sample size

A sample size of 120 patients with T2DM was estimated to represent 10% of the total population of T2DM patients attending the diabetes clinics in Maseru. Given the time frame of this study, the researcher was not able to interview more patients, and considering the possibility of non-participation and other factors responsible for attrition, it was also estimated that only about 120 of the patients who used to attend the QE II clinic, would be available for the study.

### 3.4.2 Inclusion and exclusion criteria

Males and females attending the two clinics managed by the DHMT in Maseru, who had been formally diagnosed with T2DM by a health care professional; were between 30 to 69 years old; and received dietary and lifestyle treatment, hypoglycemic tablets and/or insulin; and who gave written informed consent (Appendix I); were included in the study. The age group 30 to 69 years old was included, as this age group is accommodated in the International Physical Activity Questionnaire (IPAQ), which was used to assess activity levels. Though T2DM does occur in patients younger than 30, it was the researcher's previous experience that all younger patients attending the clinics in Maseru has been patients diagnosed with T1DM and that the youngest patient she ever counseled with T2DM at these clinics, was 30 years old. This concurs with the LRFS (2001), which indicated that T2DM was more prevalent from the age of 30 years and above in Lesotho, as shown in Table 3.1.

Patients with T1DM were excluded because the focus of this study was T2DM only. Males and females with T2DM who attended the diabetes clinics in Maseru, but were not on any treatment; either dietary, hypoglycemic tablets and/or insulin; were self-diagnosed; or did not

give written informed consent; and/or were not between 30 to 69 years old; were excluded from the study.

**Table 3.1: Prevalence of T2DM by age in Lesotho (LRFS, 2001).**

| *Age group* | *Total number tested for diabetes* | *Number of people with T2DM* | *%* |
|---|---|---|---|
| 15-19 years | 302 | 0 | 0 |
| 20-24 years | 371 | 0 | 0 |
| 25-29 years | 308 | 0 | 0 |
| 30-34 years | 255 | 2 | 0.8 |
| 35-39 years | 288 | 2 | 0.7 |
| 40-44 years | 292 | 5 | 1.7 |
| 45-49 years | 284 | 4 | 1.4 |
| 50-54 years | 273 | 6 | 4.4 |
| >55 years | 1233 | 21 | 2.3 |

Patients were recruited according to these inclusion and exclusion criteria, from the two DHMT clinics situated in Maseru, namely: Domiciliary Clinic (on Tuesdays and Wednesdays) and Lesotho Defense Force (LDF) clinic (on Wednesdays and Thursdays). These clinics are centrally located, easily accessible, and provide health care delivery to the people of Maseru and its surrounding communities. The practice profile includes promotive, preventative, curative, and rehabilitative health care services. The majority of the Lesotho patients, including those with T2DM, usually seek health care services from clinics, due to free health care delivery system. The researcher randomly selected patients with T2DM, either during their visits to a clinic doctor, or when they came to collect their medicines, and continued until 127 patients were interviewed (October 2012 to March 2013).

### 3.5. Measurements

Measurements refer to the variables and operational definitions, and the techniques applied in this study.

#### 3.5.1. Variables and operational definitions

The variables measured in this study, included socio-demographic factors, nutritional status, glycemic control, and barriers that might impact on treatment compliance.

#### 3.5.1.1. Socio-demographic factors

Socio-demographic factors referred to age in years, gender, and marital status, level of education, employment status, income level, and number of dependents.

#### 3.5.1.2. Nutritional Status

Nutritional status referred to anthropometric measurements, usual dietary intake, and lifestyle factors.

**i) Anthropometric measurements**

Anthropometric measurements referred to BMI, WC, WHtR and BAI, as these predict risks of diabetes and other chronic diseases of lifestyle in all ethnic groups (Kruger, 2013:503; Amod *et al.*, 2012:S58; Bergman *et al*., 2011:1085; Browning *et al*, 2010: 252,265).

**a) BMI**

BMI refers to the current weight in kilograms (kg) divided by height in meter squared ($m^2$) and provides a gross evaluation of total body fat (Knowles *et al.*, 2011: Online; Browning *et al.*, 2010: 247; Nyamdorj, 2010:70). According to WHO (2000), BMI is considered a valid tool to determine overweight and obesity in adults, and is defined in Table 3.2.

**Table 3.2: BMI classification (WHO Consultation, 2000)**

| BMI categories ($kg/m^2$) | Classification |
|---|---|
| < 18.5 $kg/m^2$ | Underweight |
| 18.5 – 24.9 $kg/m^2$ | Normal weight |
| 25 – 29.9 $kg/m^2$ | Overweight |
| ≥ 30 $kg/m^2$ | Obese class I |
| ≥ 35 $kg/m^2$ | Obese class II |
| ≥ 40 $kg/m^2$ | Obese class III |

**b) WC**

WC refers to a measure of the horizontal plane midway between the superior iliac crest and the lower margin of the last rib, or on the upper border of the iliac crest (Nyamdorj, 2010:70). Assessment of central obesity holds greater prognostic value than BMI alone (Browning *et al.*, 2010:248; Feller *et al.*, 2010: Online). WC measurement is considered a good indicator of intra-abdominal fat in adults (Ashwell and Browning, 2011:70; Browning *et al.*, 2010:248;

Nyamdorj, 2010:70) and is an independent risk factor for cardiovascular disease, diabetes and other endocrine abnormalities. Ethnic-specific cut-off points recommended by the IDF for Sub-Saharan Africa were used, and central obesity was defined as WC of ≥ 94 cm in males and ≥ 80 cm in females (Amod *et al.*, 2012:S58). Participants were categorized based on WC, as having no risk, increased risk or substantially increased risk for chronic diseases of lifestyle

**c) WHtR**

WHtR refers to the current waist circumference divided by height and the suggested boundary line is 0.5 (Browning *et al.*, 2010:248). WHtR is considered to be the best tool to determine abdominal fatness, and thus predicting risk factors for cardiovascular diseases and diabetes, and has demonstrated to be very useful among different ethnic, age and gender groups (Kruger *et al.*, 2013: Online; Ashwell *et al.*, 2012: 281; Knowles *et al.*, 2011: Online; Ashwell, 2011:Online; Browning *et al.*, 2010:248). Participants were categorized based on WHtR, as having no risk or increased risk for chronic diseases of lifestyle.

**d) BAI**

BAI is a newly developed parameter to estimate adiposity in individuals, and offers a direct estimate of percentage body fat (% body fat). Unlike BMI, BAI provides % body fat in both males and females of differing ethnicities without numerical correction (Bergman *et al.*, 2011:1084). BAI is calculated as follows:

BAI (% body fat) = (Hip circumference (cm)) / (Height (m) $^{1.5}$) -18 (Bergman *et al.*, 2011:1084).

$BAI = Hip/Height^{1.5} - 18$

According to Bergman *et al.*, (2011:1084) a healthy BAI is summarized as 20% - 38% in females and 8% - 25% in males.

**ii) Usual dietary intake**

Usual diet intake in this study referred to (a) the usual daily food intakes with relation to number of servings from each food group; energy and macronutrients intakes; as well as (b) the frequency (daily, weekly, monthly) with which specific types of foods from the different food groups were consumed.

**a) Usual daily food intakes (energy, macronutrients)**

The usual daily food intake was recorded with a daily food intake questionnaire (see techniques) and interpreted according to the serving recommendations of the Diabetes Food Guide Pyramid (ADA & ADA, 2005: Online), which classifies food groups according to their carbohydrate, protein and fat content (ADA & ADA, 2005: Online). Usual food intake below the recommendations of the Diabetes Food Guide Pyramid was considered insufficient; an intake equal to the recommendations was considered adequate; and an intake above the recommendations was considered as high (Table 3.3) (ADA & ADA, 2005:Online) as indicated in Table 3.3.

**Table 3.3: Interpretation of dietary intake based on the serving recommendations of the Diabetes Food Guide Pyramid (ADA & ADA, 2005)**

| **Food Groups** | **Number of recommended servings per day** | | |
|---|---|---|---|
| | **Insufficient** | **Adequate** | **High** |
| Bread, grains and other starches | < 6 | 6– 11 | > 11 |
| Fruit | < 2 | 2 – 4 | > 4 |
| Vegetables | < 3 | 3 – 5 | * |
| Milk and milk products | < 2 | 2– 3 | > 3 |
| Meat and meat substitutes | < 2 | 2– 3 | > 3 |
| Fats/oils and sweets | Use sparingly | | |

***High intake of vegetables is considered beneficial even in people with diabetes as the carbohydrate content is very low to negligible.**

Total energy intake referred to the usual intake in kilojoules per day, and macronutrients intake referred to intakes of carbohydrates, proteins and fats in grams per day. The energy and macronutrients were quantified to kilojoules and grams respectively from the data collected from the usual daily food intake questionnaire, by grouping foods according to their food groups and calculating the energy and macronutrient content based on the standard serving sizes from an exchange list developed according to the guidelines of the ADA (Franz, 2012:701; Wheeler, 2008: 883) as shown in Table 3.4. (See Appendix A for food exchange list, alcohol equivalents, and the standard serving sizes) Carbohydrate intakes (g) were interpreted against the minimum recommendation of 130g/day (Rohlfing *et al.*, 2002:275).

**Table 3.4: Food exchange list Analysis (Franz, 2012:701; Wheeler, 2008:883).**

| Exchange list | Carbohydrates (g) | Protein (g) | Fat (g) | Energy (kJ) |
|---|---|---|---|---|
| **Milk:** Skim | 12 | 8 | Trace | 375 |
| Low fat (2%) | 12 | 8 | 5 | 525 |
| Whole | 12 | 8 | 8 | 640 |
| **Starch:** bread, cereals, grains, starchy vegetables, legumes | 15 | 3 | Trace | 285 |
| **Fruit** | 15 | - | - | 250 |
| **Vegetables with negligible carbohydrate content** | - | - | - | - |
| **Vegetables with higher carbohydrate content** | 5 | 2 | - | 105 |
| **Meat:** Very lean | - | 7 | 1 | 155 |
| Lean | - | 7 | 3 | 230 |
| Medium fat | - | 7 | 5 | 315 |
| High fat | - | 7 | 8 | 420 |
| Plant-based protein | - | 7 | Varies | Varies |
| **Fat** | - | - | 5 | 190 |
| **Sugar** | 5 | - | - | 85 |
| **Alcohol** | Varies | - | - | 420 |

**Note:** 1g of carbohydrates and 1 g of protein yields 17kJ; 1g of fat yields 38 kJ, and 1g of alcohol yields 29 kJ.

Distribution of macronutrient intake was expressed proportionately as percentages of total energy and interpreted according to the recommendations for adults with diabetes based on SEMDSA guidelines of 2012, and developed according to the guidelines of ADA. Macronutrients intakes less than the recommended percentage proportion were considered insufficient, intakes within the recommended percentage proportions were considered adequate, and macronutrients intakes above the recommended percentage proportion was considered as high, as indicated in Table 3.5.

**Table 3.5: Recommended macronutrient distribution for adults with diabetes (Amod *et al.*, 2012: S16, Table 1).**

| Macronutrients | Percentage Total Energy (% TE) | | |
|---|---|---|---|
| | Low (%) | Adequate (%) | High (%) |
| **Proteins** | < 15% | 15 – 20% | > 20% |
| **Carbohydrates** | < 45% | 45 – 60% | > 60% |
| **Fats** | < 20% | 20 – 35% | > 35% |

Protein intakes were further quantified as g/kg/day and compared the DRI of 0.8g/kg/day (ADA, 2013a:S35; Beasley & Wylie-Rosett, 2013: Online).

**b) Frequency with which specific foods are consumed (daily/weekly/monthly)**

A non-quantified FFQ was used to assess the frequency with which specific food and beverage items on the list were consumed, without asking about the preparation methods and portion sizes (Gibson, 2005:44; Johnson & Hankin, 2003:227). The FFQ also served as a checklist to ensure that data provided for the usual dietary intake was complete and valid.

**iii) Lifestyle factors**

Lifestyle factors referred to physical activity levels, alcohol intake, and use of tobacco products.

**a) Physical Activity**

Physical activity for this study referred to all movements that the patients incur in their everyday lives, which included work, recreation, exercise and sporting activities. The International Physical Activity Questionnaire-Long form (IPAQ-L) was adapted to reflect the types of activities of the study population and was used to categorize activity levels. The IPAQ was developed in Geneva in 1998 to obtain internationally comparable data on health-related physical activity. Between 1997 and 1998, an International Consensus Group developed four long and four short forms of IPAQ tools which may be administered by telephone interview or self-administration, with two alternate reference periods, either the "last 7 days" or a "usual week" to recall physical activity (Craig *et al*, 2003:1396).

Overall, the IPAQ was found to produce repeatable data, with comparable data from short and long forms. The short IPAQ form is recommended for national or large scale monitoring and the long form for research requiring more detailed assessment (Craig *et al*, 2003:1396). The IPAQ showed acceptable measurements properties for physical activity among 18 to 69 years old adults in diverse settings (Graff-Iversen *et al*., 2007:13).

Activity levels obtained with the IPAQ-L were categorized according to three levels, namely low, moderate and high (McGrady *et al*., 2007:93).

**Category 1: Low**

This category represented the lowest level of physical activity. Individuals who did not meet criteria for category 2 **or** 3 were considered as accumulating a "low" level of activity.

**Category 2: Moderate**

This category represented a medium level of activity that referred to three or more days of vigorous-intensity activity of at least 20 minutes per day;

**Or**

Five or more days of moderate-intensity activity and/or walking of at least 30 minutes per day;

**Or**

Five or more days of any combination of walking, moderate-intensity or vigorous-intensity activities, achieving a minimum total activity of at least 600 metabolic equivalents (METs) minutes/weeks.

Individuals meeting **at least one** of the above criteria were defined as accumulating a "moderate" level of activity.

**Category 3: High**

This category represents a higher level of participation in physical activity that referred to:
Vigorous-intensity activity on at least three days achieving a minimum total physical activity of at least 1500 MET-minutes/week.

**Or**

Seven or more days of any combination of walking, moderate-intensity or vigorous-intensity activities achieving a minimum total physical activity of at least 3000 MET-minutes/week.

Individuals meeting **at least one** of the above criteria would be defined as accumulating a "high" level of activity.

METs are units of measure that correspond to a person's metabolic rate during selected activities and are expressed as multiplies of resting metabolic rate (RMR). A MET value of 1

is the oxygen metabolized at rest, which is 3.5 ml of oxygen per kilogram of body weight per minute in adults (1 MET = ~ 3.5 ml of O2/kg/min), and can be expressed as 1 kcal per kilogram of body weight per hour (1MET = 1 kcal/kg/hour) (Frary & Johnson, 2008:34).

For the purpose of the IPAQ-L, a median and interquartile range is computed for walking (W), moderate-intensity activities (M), vigorous-intensity activities (V) and a combined total physical activity score. All continuous scores are expressed in MET- minutes/week and used to quantify levels of activity as follows:

**Walking** METs-minutes/week = 3.3×walking minutes per week;

**Moderate** METs-minutes/week = 4.0×moderate-intensity minutes per week; and

**Vigorous** METs-minutes/week = 8.0 × vigorous-intensity activity minutes per week.

**Total physical activity** MET-minutes/week = sum of Walking + Moderate + Vigorous MET minutes/week scores (Frary & Johnson, 2008:34; McGrady *et al*., 2007:93).

**b) Alcohol consumption**

Alcohol consumption for this study referred to the intake of any beverage (homebrew, beer, cider, wine, and spirits) that contains alcohol. In T2DM alcohol intake should be limited to a light to moderate amount, which represents ≤ 1 drink/units of alcohol (15g alcohol) per day for women and ≤ 2 drinks/units of alcohol (30g alcohol) per day for men (ADA, 2013a:S23; Amod *et al.*, 2012:S16; Franz, 2012:687).

**1 unit of alcohol:** 330ml of ordinary beer, lager or cider (3-4% alcohol by volume); or
150 ml wine; or
A small pub measure (25ml) of spirits (40% alcohol by volume); or
A standard pub measure (50ml) of fortified wine such as sherry or port (20% alcohol by volume) (ADA, 2013a:S23; Amod *et al.*, 2012:S16; Franz, 2012:687).

Based on these recommendations, the following categories were distinguished:

**Non-drinker:** referring to a participant who does not use any alcohol;

**Prudent drinker:** referring to a female participant who takes ≤ 1 unit/day, or a male participant who takes ≤ 2 units/day; and

**At risk drinker:** referring to a female participant who takes >1unit/day, or a male participant who takes > 2 units/day.

**c) Tobacco use**

Cigarette smoking is linked to increased insulin resistance and to the risk for developing T2DM (CDC, 2011: Online; Nyamdorj, 2010:21). Smoking and any form of tobacco use is specifically discouraged for patients with diabetes (Glass *et al*., 2009:40). Tobacco use for the purpose of this study referred to smoking of cigarettes (commercial or home-made) and/or a pipe, or use of and/or the use of snuff.

The following categories were distinguished:

**Non-tobacco user:** referring to a participant who never smoked cigarettes/pipe or used snuff;

**Ex-tobacco user:** referring to a participant who smoked cigarette/pipe or used snuff over a period of time in his/her life; but stopped;

**Current tobacco user:** referring a participant who smoked cigarettes/pipe or used snuff at the time of data collection. The type(s) of tobacco used, as well as the frequency of use.

**3.5.1.3 Medical history**

Medical history as obtained from the participants' medical booklets (bukanas), or self-reported during one-on-one interviews with the patients. Bukana is a small booklet in which all the information regarding the condition of the patient is recoded e.g. diseases, laboratory tests requested and results, blood pressure readings, medications prescribed and other procedures that might be performed. The bukana is kept by the patient, and has to be presented every time the patient visits the health care facility. All health professionals have access to the patients' bukana.

The bukanas would also have information regarding the year of diagnosis of T2DM, comorbid conditions (hypertension only), medications prescribed, and complications of diabetes (when tested, though they are not routinely done at the public clinics), and those reported in the study were self-reported by the patients. However at QE II hospital patients with eye problems used to be referred to eye specialists (because there was an ophthalmologic

department), while with other complications such as nephropathy and neuropathy were treated as the particular patient present any of those and get admitted in the hospital. The last recorded results/readings in the bukanas were used for this study.

The information that was obtained from the patient's bukanas included FPG readings, blood pressure readings, (and total cholesterol, triglycerides and $HbA_{1c}$ results if available, but are not routinely done in public clinics), and would be available to those patients whom would have consulted private doctors at some stage. Acceptable biochemical values for patients with T2DM are $HbA_{1c}$ of < 7%, fasting blood glucose between 4.0 to 7.0 mmol/L, total serum triglycerides of < 1.7 mmol/L, total serum cholesterol of < 4.5 mmol/L, blood pressure of 140/80 mmHg or below (Amod *et al.*, 2012:S57).

#### 3.5.1.4 Glycemic control

Glycemic control referred to an achievement of normal blood glucose levels, and was determined using fasting blood glucose levels (4.0 to 7.0 mmol/L) and $HbA_{1c}$ levels. Both current and retrospective (where available) $HbA_{1c}$ results were recorded and interpreted according to the following with cut-off points as recommended by (Amod *et al*., 2012: S) and (ADA, 2013a: S19):

**HbA1c < 7%** was considered as optimal;

**HbA1c = 7- 8%** was considered as acceptable; and

**HbA1c > 8%** was considered as sub-optimal and indicative that additional action must be taken (Amod *et al*., 2012: S20).

#### 3.5.1.5 Barriers that may impact on treatment compliance

Barriers for the purpose of this study referred to the challenges that the participants experience which might negatively impact on their compliance to diabetes treatment. Barriers related to the provision of health care services at the clinic or to personal factors, including socio-economic factors, level of education, social environment, and patients' knowledge, attitudes, beliefs and perceptions regarding dietary and lifestyle changes, and medical therapies, were assessed.

### 3.5.2 Measuring Techniques

Data regarding socio-demographic factors, dietary intakes and barriers that may impact on treatment compliance were obtained from the participants' in structured one-on-one interviews, using questionnaires. Anthropometrical data was obtained by direct measurements of the participants. Data on medical history and biochemical parameters to assess glycemic control and risk for diabetes-associated long-term complications were obtained from the participants medical booklets (bukanas), self-reported or by biochemical measurements (FPG and $HbA_{1c}$ results).

#### 3.5.2.1 Anthropometric measurements

To ensure reliability, anthropometrical values were measured by the single researcher who is a registered dietitian, using standardized techniques, and were recorded on the questionnaire (Appendix B).

**i) Weight**

Weight was measured with a calibrated digital "Seca" scale, placed on a flat, hard surface. Participants were measured in light clothes, without shoes. The measurements were taken after emptying the bladder and before a meal. The subjects were asked to stand in the middle of the platform and to look ahead, unassisted and relaxed. The readings obtained were recorded to the nearest 0.1 kg, and used to calculate the BMI and BAI (Gibson, 2005:252).

To ensure reliability the scale was calibrated before use and after every $10^{th}$ patient was weighed. The zero reading was checked periodically and each time after the scale was moved (Katzenellenbogen *et al*., 1997:176). Two readings were taken to the nearest 0.1 kg, and the averages of the readings were used if the readings were more than 1kg apart (Gibson, 2005: 281). The measurement obtained was used to calculate the BMI.

**ii) Height**

Height was measured to the nearest 0.1 cm using a portable wall-mounted stadiometer. Participants were requested to stand upright on a flat surface without shoes, with the back of the heels on the stadiometer (Gibson, 2005:252). Participants were asked to take a deep breath, relax and stand tall to aid the straightening of the spine. The headboard was lowered

until it reached of the crown the head and height was taken at maximum inspiration, with the researcher's eyes level with the headboard to avoid parallax errors (Gibson, 2005:247). To ensure reliability two readings were taken to the nearest mm, and the average of the readings was used if the readings were greater than 1mm apart (Gibson, 2005:281). The measurement obtained was used to calculate the BMI, WHtR and BAI.

**iii) Waist Circumference**

WC was measured with an unstretchable, flexible anthropometric tape measure. The participants were asked to stand with the feet apart, arms at each side of the body at a 30° angle. WC was measured over loose thin clothing (for cultural reasons) in a horizontal plane; midway between the inferior margin of the ribs and the superior border of the iliac crest. The measurement was taken at the end of a normal expiration, ensuring that the participant did not contract the abdominal muscles.

To ensure reliability two readings were taken to the nearest millimeter (mm) and the average of the readings were used if the readings were greater than 1mm apart (Gibson, 2005:281). The measurement obtained was interpreted and also used to calculate the WHtR.

**iv) Hip circumference**

Hip circumference was measured with a non-stretchable, flexible anthropometric tape measure. The participants were asked to stand with the feet apart, arms at each side of the body at a 30° angle. Hip circumference was measured at the level of the maximum extension of the buttocks posterior over loose thin clothing (for cultural reasons) in a horizontal plane. To ensure reliability two readings were taken to the nearest millimeter, and the average of the readings were used if the readings were greater than 1mm apart (Gibson, 2005:281). The measurement obtained was used to calculate the BAI.

### 3.5.2.2 $HbA_{1c}$ measurements

Blood samples were drawn with the assistance of a registered nurse, using an ethylene diamine tetraacetic acid (EDTA) (purple stopper) tube. The blood samples were placed in a cooler box containing ice-packs immediately after withdrawal, and transported immediately after the clinic session by car to the laboratory where $HbA_{1c}$ was measured according to

standardized techniques. $HbA_{1c}$ measurements do not require fasting blood samples and has a higher repeatability than measurement of fasting blood glucose levels (Nyamdorj, 2010:70).

#### 3.5.2.3 Questionnaires

Questionnaires were developed to record the socio-demographic data, usual dietary intake, lifestyle factors, and barriers that may impact on treatment compliance. The single researcher, who is a registered dietitian, completed the questionnaires during a one-on-one structured interview with a single participant at a time, conducted in a private room at the clinic. Interviews were conducted in Sesotho and/or English according to the preference of the participant. The questions in the different languages were translated as not to differ in meaning in any way, and asked in the same order. No leading questions or double-barreled questions were asked. The researcher provided appropriate verbal and non-verbal cues, avoided putting her own perspective into questioning, and practiced using the questionnaires until familiar with them. The following questionnaires were included:

**i) Socio-demographic factors**

Socio-demographic data was obtained by the researcher from the participants' medical booklets (bukanas) and also through one-on-one interviewing, and the information was recorded on the questionnaire (Appendix B).

**ii) Usual dietary intake**

Usual dietary intake was assessed using an (a) usual daily dietary intake questionnaire and (b) a non-quantified FFQ, during the interview with each participant (Appendix C).

**a) Usual dietary intake questionnaire**

Participants were asked to recall all foods/beverages eaten/drunk on a regular day including the portion sizes, and the preparation methods (Gibson, 2005:44; Johnson & Hankin, 2003:227). Participants were guided to estimate portion sizes using household measuring utensils, and food models. The researcher used probing questions to elicit specific details of each food item (Gibson, 2005:41-42).

**b) Non-quantified food frequency questionnaire (non-quantified FFQ)**

A non-quantified FFQ which is designed to provide additional information on the quality and diversity of the diet (Gibson, 2005:44), was used to assess the frequency with which specific food and beverages from a list were consumed (daily, weekly or monthly), without asking about the preparation methods and portion sizes (Gibson, 2005:44; Johnson & Hankin, 2003:227). The FFQ was also used as a checklist to ensure that data provided for the usual daily dietary intake was complete and valid. For instance, if a food item was not mentioned in the usual daily dietary intake questionnaire, but shown to be consumed daily in the non-quantified FFQ, the participant was asked about the particular food in order to incorporate it in the usual daily dietary intake record to provide an accurate picture of the individual's usual food intake. The FFQ was developed by the Department of Nutrition and Dietetics, UFS, was adapted by the researcher, who is a native of Lesotho, to include the traditional Basotho foods and foods typically consumed in Lesotho, to increase the content validity.

**iii) Lifestyle factors**

Information on physical activity, alcohol intake and smoking was also obtained during the structured one-on-one interviews. The IPAQ-L was adapted by the researcher as recommended by Craig *et al*, (2003:1396), to include the activities that were most relevant to the study population (Appendix D). The IPAQ questionnaire measured activities by asking the respondent to recall usual participation in activities or in sedentary behaviors over a set period of time. The activities range from household duties, gardening, taking the stairs, and walking briskly, to jogging, exercising and participating in sports. The level of physical activity needed to obtain a health benefit does not have to be strenuous (Graff-Iversen *et al*., 2007:13). The IPAQ was validated in Geneva in 2000, and extensive reliability and validity testing was done in 12 countries. This tool has since been used by the WHO in many countries (Craig *et al*, 2003:1396).

To ensure reliability, participants were assured on the confidentiality of the information they provided. The researcher used probing questions to elicit details and avoided putting her own perspective into questioning.

**iv) Medical history**

The information about the year of diagnosis, biochemical values (total cholesterol and triglycerides levels), comorbid conditions (only hypertension), and glucose lowering medications, was obtained from the participants' medical booklets (bukanas) or during the structured interview with the researcher. The information regarding chronic complications of diabetes (retinopathy, neuropathy and nephropathy) was self-reported by the patients (not obtained from the bukana), because the patients were not routinely screened for chronic complications in their respective clinics. Blood pressure and fasting blood glucose levels were measured prior to the interview by the nurse in charge. The information was recorded in the form (Appendix B).

**v) Barriers that may impact on treatment compliance**

The questionnaire was adapted from published 'Knowledge, Attitudes and Perception (KAP)' surveys (Dunstan *et al.*, 2002: Online) to include questions on the barriers that might negatively impact on treatment compliance in this population. Questions were included on provision of health care services at the clinic; personal factors; socio-economic factors; social environment; and the participants' knowledge, attitudes, beliefs and perceptions regarding dietary and lifestyle changes and medical therapies. Questions regarding participants' knowledge of the dietary recommendations for T2DM were structured around the SAFBDG Steyn and Temple, (2012:502), which is a tool developed and validated for advocating and teaching prudent dietary practices to the various population groups in South Africa. The people of Lesotho have more similar dietary habits to those of South Africans, rather than other population groups, like Americans and British for whom published dietary guidelines are available.

The SAFBDG are as follows and were adapted in the questionnaire to reflect diabetes-specific recommendations (Steyn and Temple, 2012:502):

- Enjoy a variety of foods (eat regular small meals);
- Be active;
- Make starchy foods the basis of most meals, (including those that are slowly digested, high in fibre);
- Eat plenty of fruit and vegetables everyday (minimum of 5 servings per day);
- Eat cooked dried beans, peas, lentils and soya regularly;

- Low fat meat, fish, chicken, milk, yoghurt, cheese or eggs can be eaten every day (but a diabetes patient is advised to limit eggs intake to 3 eggs per week);
- Eat fats sparingly (especially saturated fats, and try to avoid trans-fatty acids);
- Use salt sparingly;
- Use food and drinks containing sugar sparingly and not between meals
- Drink lots of clean safe water; and
- If you drink alcohol, drink sensibly (have an alcohol free day per week).

### 3.6 Study procedures

The study was conducted according to the following steps:

**Step 1:**

i) Approval to conduct the study was obtained from the Ethics Committee of the Faculty of Health Sciences, UFS (ETOVS NR 76/2012).

ii) Approval to conduct the study was obtained from the Ethics Committee of the Ministry of Health of Lesotho (Appendix F).

iii) Permission was obtained from the District Medical Officer Maseru District (Appendix G).

**Step 2:**

i) The researcher met with the clinic staff to explain the study and ask their assistance to access each participant's medical booklets (bukanas).

ii) The pilot study was conducted on five patients with T2DM meeting the inclusion criteria and were included in the major study.

**Step 3:**

i) Interviews were conducted at the diabetes clinic on Tuesdays, Wednesdays and Thursdays.

ii) Data was collected for a period of six months (from October, 2012 to March, 2013), and there was a six weeks break (two weeks of November and four weeks of December) due to work assignments which were carried out outside Maseru.

iii) Participants attending the clinic who fitted the inclusion criteria were approached and invited to participate in the study. Signed informed consent was obtained from the participants.

iv) Participants were then interviewed by the researcher after the nurse in charge had

checked fasting blood glucose levels (using a glucometer) and blood pressure. One-on-one structured interviews to determine the socio-demographic factors, dietary intake, lifestyle factors and barriers that might impact on treatment compliance were then conducted either before the participants consulted the doctor; while waiting for their medications; or after collection of their medications. An average of five patients were interviewed on Wednesdays and three on Tuesdays and Thursdays, and the interviews took approximately 45 minutes.

v) Anthropometric measurements and biochemical values were measured with relevant instruments.

vi) Blood was drawn with the assistance of a registered nurse.

vii) Relevant information was obtained from the patient medical booklets (bukanas).

**Step 4:**

i) The data was coded from 001 to 127), then the data was captured on excel by the Department of Biostatistics at UFS.

ii) The statistical analysis was performed by the Department of Biostatistics at UFS and was generated by SAS® software, and categorical data was presented as frequencies and percentages; and continuous data as medians and percentiles.

### 3.7 Reliability and validity

Validity refers to the adequacy with which any measurement, index, or indicator reflects what it was intended to measure (Gibson, 2005:11). Reliability (precision of measurement) refers to the degree to which repeated measurements of the same variable gives the same value and reflects the ability of a procedure to produce the same results repeatedly (Gibson, 2005:11).

To ensure reliability and validity of the anthropometric measurements all measurements were taken by the single researcher, who has been trained to accurately collect the required information, using standardized techniques and calibrated equipment (Gibson, 2005:281).

In the structured one-on-one interviews, reliability was improved by explaining the questionnaires to the participants in the language of their choice. The researcher used probing questions to help participants recall food items that they usually ate. Household measuring utensils and food models were used to help participants estimate portions sizes. The same

questionnaires used in the pilot study were included in the main study. Participants were assured of the confidentiality of the information they provided.

The biochemical measurements were performed using the NGSP, which was developed to standardize $HbA_{1c}$ to clinical data from the DCCT. The NGSP has a certification program for manufacturers and laboratories, as well as ongoing monitoring of the quality of $HbA_{1c}$ measurements. This program has been successful in reducing variability among laboratories (ADA, 2013a: S 23; ADA, 2013b: S67; IDF, 2013a:S13).

### 3.8 Pilot study

The purpose of a pilot study is to ensure that the participants understand and correctly interpret the questions in the relevant language, and to ensure that all the relevant information is included in the questionnaire. The pilot study was conducted, at Domiciliary Clinic on five patients with T2DM who met the inclusion criteria of the study and attended the diabetes clinic. Corrections were made to some questions from the "Barriers that may impact on treatment compliance questionnaire" (Appendix E). The five questionnaires from the pilot study were included in the major study.

### 3.9 Data analysis

Statistical analysis was performed by the Department of Biostatistics at UFS and was generated by SAS® software (copyright, SAS Institute – SAS and all other SAS Institute Inc. products or service names are registered trademarks or trademarks of SAS Institute Inc., NC, and USA). Categorical data was presented as frequencies and percentages; and continuous data as medians and percentiles.

### 3.10 Ethical aspects

The purpose of the study, the description of the information (Appendix H) to be collected from the participants and the procedures and processes involved, were communicated orally and in writing, in the language of the participants' choice. Participants were asked to voluntarily complete the consent forms provided to them (Appendix I) in the language of

their choice. Participation was voluntary and respondents were free to withdraw from the study at any time.

Confidentiality was retained during all stages of the research by ensuring that no names were disclosed or written on questionnaires. Codes were used in all data analysis and results. Results obtained were used for the purpose of the study only, and were not part of the patients' routine records.

Patients who require further management or those seriously ill, were referred to clinicians, while for those needing dietetic counseling, appointments were made with the researcher for provision of such services. The participants were provided with a snack (yoghurt and an apple), in order to curb their hunger after the interviews. There was no remuneration and no cost to the participants, and participants were informed that results obtained may be published.

No risks were encountered by the participants, since only 5 milliliters of blood were drawn. The procedure was performed by a registered nurse at the clinic. The participants experienced a very mild pain when the needle was inserted into the vein, which disappeared within a short period of time. To avoid infections, a surgical spirit was used as a disinfectant before insertion of the needle. Blood samples were used to determine $HbA_{1c}$, and for no other tests.

The benefits of the study, which would include contributions to improved knowledge, services and management of patients with diabetes in Maseru, would justify the effort taken by the participants. Feed-back and medical results were made available to participants on request, and a final report of findings will be made available to the MOH and the two DHMT clinics.

### 3.11 Challenges experienced during the study and possible limitations

The researcher originally planned to include all five clinics in Maseru in the study, but permission could only be obtained to conduct the study at the two clinics managed by the

Government (DHMT). The problems encountered during the research process included: time restraints, financial restraints, and accessing the participants.

### 3.11.1 Time restraints

As the protocol was originally approved to include the five Maseru clinics, but permission could only be obtained to conduct the study at the two clinics managed by DHMT, an addendum to the protocol (to exclude the three clinics) had to be presented again to the Ethics Committee UFS after the first approval. This delayed the commencement of the data collection. Furthermore, the data collection had to be stopped for six weeks for the researcher to perform assignments outside of Maseru for the Ministry of Health. The six weeks also included the Christmas holidays. Furthermore, the researcher had to conduct education sessions in between the interviews, on healthy eating and use of medications, especially to the participants who were on insulin. This was time consuming and also delayed the data collection process on the given day.

To make up some lost time, the researcher started to collect data for two days per week instead of only on Wednesdays (when diabetes sessions are routinely held at the clinics). At Domiciliary Clinic and at LDF Clinic, sessions are routinely held for patients with hypertension on Tuesdays and Thursdays, respectively. As some patients with diabetes are hypertensive as well, some attend these sessions and the researcher recruited them.

If the researcher had more time, she would have increased the sample size to be more representative, and to make the findings of this study more generalizable for the whole population of patients with T2DM in and around Maseru.

### 3.11.2 Financial restraints

The researcher had no sponsorship for the study, and had requested the laboratory in the Ministry of Health to procure $HbA_{1c}$ reagents and use of the analyzing machine (as originally planned in the protocol). Unfortunately due to budget cut downs in the laboratory department, reagents were never procured and the machine was out of order due to lack of the service.

To overcome the problem, the researcher had to take the blood samples to a private laboratory (*Pathcare*) for $HbA_{1c}$ analysis, which required more funds than budgeted for. Patients with diabetes are not routinely tested for $HbA_{1c}$ in public facilities (including clinics), except for few cases. While those patients who visited private facilities during emergency or as preferred would be tested for $HbA_{1c}$. The researcher's intention was to analyze blood samples of all participants interviewed, but due to budget constraints only 45 blood samples were analyzed for $HbA_{1c}$ and could be paid for. While the rest of $HbA_{1c}$ results were old (a period of one to two years) results obtained from the participants medical booklets/ bukanas. If the researcher had enough funds, she would have analyzed all blood samples of the participants, thus improving the findings of the study in regard to patients' glycemic control.

### 3.11.3 Difficulties in recruiting participants

Most patients with diabetes approached for the study, declined to participate due to fear of the needle used to get the blood samples. Some participants demanded to get the feedback of their blood results. Some patients felt that the researcher would be wasting their time, as they had to return to work after the clinic visit.

In an attempt to overcome these limitations, the researcher explained the benefits of knowing HbA1c results to the patients approached, and promised to send results with explanations to them via cell phone text messages. Provision of the snack became useful in some cases.

# CHAPTER 4: RESULTS

This chapter summarizes the results with regard to the socio-demographic information, nutritional status, medical history, glycemic control, and barriers that may impact on treatment compliance.

## 4.1 Introduction

A total of 127 patients participated in the study. Three of these participants were subsequently excluded from the study because most of their information was missing. The final sample size thus comprised of 124 participants.

## 4.2 Socio-demographic information

The socio-demographic results for the total sample, as well as according to gender, are presented in Table 4.1 and include age, residential area, marital status, level of education, employment status, income level, and the number of dependents.

Most participants were females (79.8%). The mean age of the total group was 53.9 (± 9.4) years (54.6 ± 9.2 years for the females and 51.0 ± 9.9 years for the males). The total group comprised of 90.0% married participants (69.7% of the females and 84.0% of the males); and 44.4% of the participants (42.4% of the females and 52.0% of the males) had three to four dependents. About half (49.2%) of the participants had a high school education (49.5% of the females and the 48.0% males), and less than half (42.7%) were employed (42.4% of the females and 44.0% of the males). Most females (62.6%) had a maximum income level at the lower end of the income scale (between M300 and M1 500), with only about 10% earning an income at the higher end of the scale (between M4 100 and M5 300). Contrarily, among the males, 37.5% had a maximum income at the higher end of the scale, with only 12% at the lower end of the scale. Overall about half of the participants (52.4%) had an income at the lower end of the income scale.

## Table 4.1: Socio-demographics of the participants (N=124)

| VARIABLES | FREQUENCY/PERCENTAGES | | | | | |
|---|---|---|---|---|---|---|
| | Total group (n=124) | | Females (n=99) | | Males (n=25) | |
| **Age** | **n** | **%** | **n** | **%** | **n** | **%** |
| 30 – 40 years | 13 | 10.5 | 9 | 9.1 | 4 | 16.0 |
| 41 – 50 years | 27 | 21.8 | 19 | 19.2 | 8 | 32.0 |
| 51 – 60 years | 46 | 37.1 | 39 | 39.4 | 7 | 28.0 |
| 61 – 70 years | 38 | 30.6 | 32 | 32.3 | 6 | 24.0 |
| **Residential area** | | | | | | |
| Maseru Urban | 113 | 91.1 | 88 | 88.9 | 25 | 100.0 |
| Maseru Rural | 11 | 8.9 | 11 | 11.1 | 0 | 0.0 |
| **Marital status** | | | | | | |
| Married | 90 | 72.6 | 69 | 69.7 | 21 | 84.0 |
| Single | 16 | 12.9 | 14 | 14.1 | 2 | 8.0 |
| Divorced | 2 | 1.6 | 1 | 1.0 | 1 | 4.0 |
| Separated | 16 | 12.9 | 15 | 15.2 | 1 | 4.0 |
| Widowed | 0 | 0.0 | 0 | 0.0 | 0 | 0.0 |
| **Number of dependents** | | | | | | |
| 1 – 2 | 24 | 19.4 | 16 | 16.2 | 8 | 32.0 |
| 3 – 4 | 55 | 44.4 | 42 | 42.4 | 13 | 52.0 |
| 5 – 6 | 36 | 29.0 | 32 | 32.3 | 4 | 16.0 |
| 7 – 8 | 6 | 4.8 | 6 | 6.1 | 0 | 0.0 |
| 9 – 10 | 3 | 2.4 | 3 | 3.0 | 0 | 0.0 |
| **Employment status** | | | | | | |
| Employed | 53 | 42.7 | 42 | 42.4 | 11 | 44.0 |
| Unemployed | 18 | 14.5 | 14 | 14.1 | 4 | 16.0 |
| Pensioner | 8 | 6.5 | 8 | 8.1 | 0 | 0.0 |
| Homemaker | 12 | 9.7 | 12 | 12.1 | 0 | 0.0 |
| Other (Self-employed) | 33 | 26.6 | 23 | 23.2 | 10 | 40.0 |
| **Level of Education** | | | | | | |
| Tertiary | 10 | 8.1 | 5 | 5.1 | 5 | 20.0 |
| College (including vocational schools) | 15 | 12.1 | 12 | 12.1 | 3 | 12.0 |
| High school | 61 | 49.2 | 49 | 49.5 | 12 | 48.0 |
| Primary school | 37 | 29.8 | 33 | 33.3 | 4 | 16.0 |
| None | 1 | 0.8 | 0 | 0.0 | 1 | 4.0 |
| **Income level (N=123)** | **n=123** | | **n=99** | | **n=24** | |
| M0.300 – M1,500 | 65 | 52.4 | 62 | 62.6 | 3 | 12.0 |
| M1,500 – M2,700 | 13 | 10.5 | 7 | 7.1 | 6 | 25.0 |
| M2,700 – M3,900 | 16 | 12.9 | 10 | 10.1 | 6 | 25.0 |
| M3,900 – M4,100 | 10 | 8.0 | 10 | 10.1 | 0 | 0.0 |
| M4,100 – M5,300 | 19 | 15.3 | 10 | 10.1 | 9 | 37.5 |

## 4.3 Nutritional status

Nutritional status for the purpose of this study included anthropometric measurements, dietary intake and lifestyle factors.

### 4.3.1 Anthropometric measurements

Anthropometric measurements included BMI, WC, WHtR and BAI as these predict risks of diabetes in all ethnic groups. Anthropometric values as measured by the researcher, using standardized techniques, are presented in Table 4.2 and Figures 4.1 and 4.2.

Among the participants, more than half (57.3%) of all participants, and more of the females (61.6%) than of the males (40%), were obese; with a further 31.3% (31% of the females and 32% of the males) being overweight (Table 4.2). There were no underweight participants and only females (8%) fell into the obesity class III category.

Based on WC (Table 4.2), almost all participants were at an increased risk for cardiovascular diseases, insulin resistance, and other endocrine abnormalities (Leitzmann *et al.*, 2011: Online; Browning *et al.*, 2010:266), with 91.1% (90.9% of the females and 92.0% of the males) having a substantially increased risk. This was supported by the WHtR which placed 96.8% of participants (98.0% of the females and 92.0% of the males) in the category of increased risk of cardiovascular diseases (Kruger *et al.*, 2013: Online; Ashwell *et al.*, 2012: Online; Knowles *et al.*, 2011: Online; Ashwell, 2011: Online; Browning *et al.*, 2010:248). Based on BAI (as a measure of fat percentage) more than half (65%) of all participants, and more of the females (78%) than of the males (12%), fell into the obese category. Another 60.8% of females and 84.0% of males were obese, based on BAI.

Figure 4.1 illustrated the percentages of participants who were classified as obese, based on BMI (88.5%), WC (98.3%), WHtR (96.8%), and BAI (60.8%). The percentages of obese participants stratified according to gender are further represented in Figure 4.2. BMI and WHtR indicated that more females were obese, while WC and BAI showed that more males were obese as compared to females.

**Table 4.2: Anthropometric measurements of the participants**

| VARIABLES | FREQUENCY/PERCENTAGES | | | | | |
|---|---|---|---|---|---|---|
| | Total group (n=124) | | Females (n=99) | | Males (n=25) | |
| **Body Mass Index (BMI) (N=124)** | **n** | **%** | **n** | **%** | **n** | **%** |
| Underweight | 0 | 0.0 | 0 | 0.0 | 0 | 0.0 |
| Normal | 14 | 11.5 | 7 | 7.1 | 7 | 28.0 |
| Overweight | 39 | 31.9 | 31 | 31.3 | 8 | 32.0 |
| Obese class I | 40 | 32.3 | 32 | 32.3 | 8 | 32.0 |
| Obese class II | 23 | 18.5 | 21 | 21.2 | 2 | 8.0 |
| Obese class III | 8 | 6.5 | 8 | 8.1 | 0 | 0.0 |
| **Waist Circumference (WC) (N=124)** | | | | | | |
| No risk | 2 | 1.6 | 2 | 2.0 | 0 | 0.0 |
| Increased risk | 9 | 7.3 | 7 | 7.1 | 2 | 8.0 |
| Substantially increased risk | 113 | 91.0 | 90 | 90.9 | 23 | 92.0 |
| **Waist-to-Height Ratio WHtR (N=124)** | | | | | | |
| No risk (≤ 0.5) | 4 | 3.2 | 2 | 2.0 | 2 | 8.0 |
| Increased risk (> 0.5) | 120 | 96.8 | 97 | 98.0 | 23 | 92.0 |

| **Body Adiposity Index (BAI) (n=122)** | **Females (n = 97)** | | | **Males (n = 25)** | | |
|---|---|---|---|---|---|---|
| | **Cut-off** | **N** | **%** | **Cut-off** | **n** | **%** |
| Underweight | < 20% | 0 | 0.0 | > 8% | 0 | 0.0 |
| Healthy | 20 – 38% | 38 | 39.2 | 8 - 25% | 4 | 16.0 |
| Obesity | > 38% | 59 | 60.8 | > 25% | 21 | 84.0 |

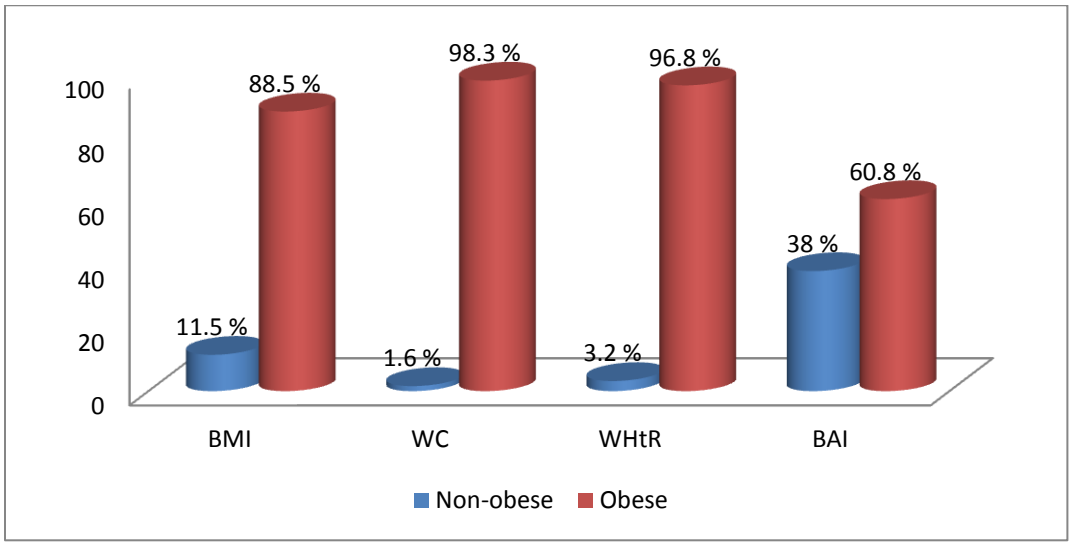


**Figure 4.1: Percentage distribution of non-obese and obese participants**

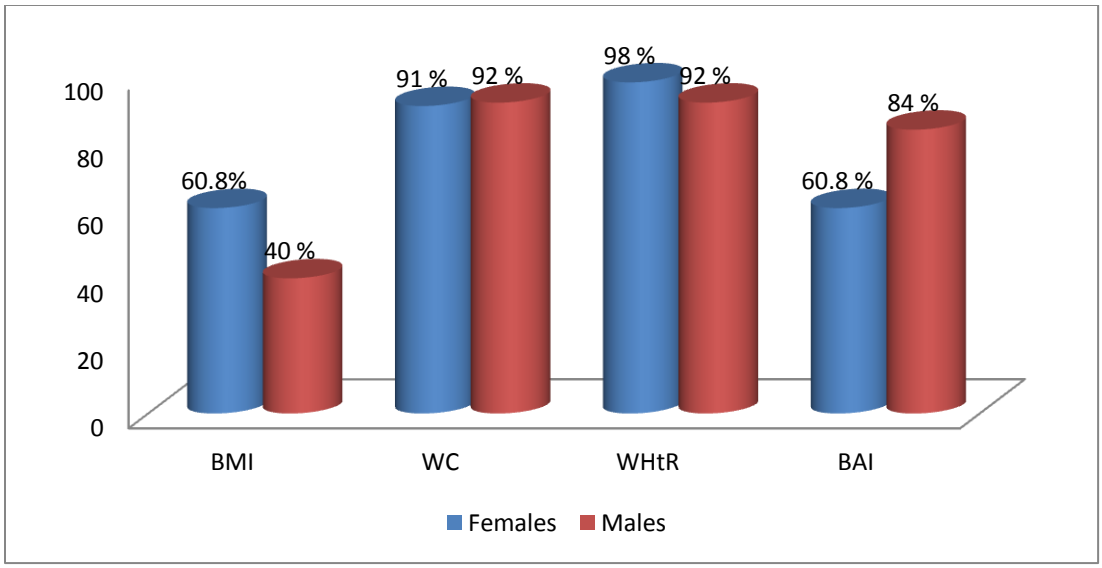


**Figure 4.2: Percentage distribution of obese females and males.**

### 4.3.2 Dietary intake

The dietary intake data was collected as usual dietary intake (to assess total energy and macronutrient intakes) and as frequency with which specific food types are consumed (on a daily/weekly/monthly basis).

#### 4.3.2.1 Usual dietary intake

The usual daily food intake was assessed according to the serving recommendations of the Diabetes Food Guide Pyramid (ADA & ADA, 2005: Online), which categorizes food into six food groups according to carbohydrate, protein and fat content (ADA & ADA, 2005: Online). Usual food intake below the recommendations of the Diabetes Food Guide Pyramid was considered insufficient; intake within the recommendations was considered adequate, and intake above the recommendations was considered as high (Table 4.3) (ADA & ADA, 2005: Online).

**Table 4.3: Usual daily dietary intake of the participants (N=124)**

| FOOD GROUPS | RECOMMENDATIONS | FREQUENCY/PERCENTAGES | | | | | |
|---|---|---|---|---|---|---|---|
| **Bread, grains & cereals** | | **Total group (n=124)** | | **Females (n=98)** | | **Males (n=25)** | |
| | | **n** | **%** | **n** | **%** | **n** | **%** |
| Below | < 6 servings per day | 0 | 0.0 | 0 | 0.0 | 0 | 0.0 |
| Within | 6 – 11 servings per day | 74 | 59.7 | 65 | 66.3 | 9 | 36.0 |
| Above | > 11 servings per day | 49 | 39.5 | 33 | 33.7 | 16 | 64.0 |
| **Fruits** | | | | | | | |
| Below | < 2 servings per day | 81 | 65.3 | 63 | 64.3 | 18 | 72.0 |
| Within | 2 – 4 servings per day | 39 | 31.5 | 34 | 34.7 | 5 | 20.0 |
| Above | > 4 servings per day | 3 | 2.4 | 1 | 1.0 | 2 | 8.0 |
| **Vegetables** | | | | | | | |
| Below | < 3 servings per day | 97 | 78.2 | 79 | 80.6 | 18 | 72.0 |
| Within | 3 - 5 servings per day | 25 | 20.2 | 18 | 18.4 | 7 | 28.0 |
| Above | > 5 servings per day | 1 | 0.8 | 1 | 1.0 | 0 | 0.0 |
| **Milk and milk products** | | | | | | | |
| Below | < 2 servings per day | 115 | 92.7 | 94 | 95.9 | 21 | 84.0 |
| Within | 2 – 3 servings per day | 8 | 6.5 | 4 | 4.1 | 4 | 16.0 |
| Above | > 3 servings per day | 0 | 0.0 | 0 | 0.0 | 0 | 0.0 |
| **Meat and meat substitutes** | | | | | | | |
| Below | < 2 servings per day | 24 | 19.3 | 21 | 21.4 | 3 | 12.0 |
| Within | 2 – 3 servings per day | 83 | 66.9 | 71 | 72.4 | 12 | 48.0 |
| Above | > 3 servings per day | 16 | 12.9 | 6 | 6.1 | 10 | 40.0 |
| **Fats/oils and sweets** | | | | | | | |
| Use sparingly | | | | | | | |

Table 4.3 indicates that no participants consumed less than the recommended number of servings from the bread, grains and cereal group per day, while almost 40% of all participants, and more of the males (64%) than of the females (33.7%), consumed more than the recommended number of daily servings from this food group.

Both females and males consumed fewer than the recommended number of servings from the fruit group (64.3% of the females and 72.0% of the males), the vegetable group (80.6% of the

females and 72.0% of the males), and the milk and dairy product group (95.9% of the females and 84.0% of the males).

Regarding meat and meat substitutes, almost a fifth (19.3%) of all participants (21.4% of females and 12 % of males) consumed fewer than the recommended 2 to 3 servings per day. On the other hand, 40% of the males, but only 6.1% of the females, consumed more than 3 servings of meat and meat substitutes per day.

The FGP recommends fats and oils are consumed sparingly, but do not quantify the amounts. While most participants (57.7%) consumed up to three servings of added fat and oils per day, 38.2% (33.7% of the females and 56.0% of the males) consumed more than three servings from this food group per day, and 4.1% of the participants consumed less than three servings per day.

Table 4.4 summarizes the usual dietary intakes of energy, carbohydrates, proteins and fats by the participants, and Table 4.5 summarizes the protein intakes of the participants expressed as g/kg/day.

As indicated in Table 4.4, the mean energy intakes, derived from the self-reported usual daily dietary intake reports, for females and males were 5360.2 kJ and 6893.0 kJ, respectively. Carbohydrates contributed about two thirds of energy intakes (65.7% for females and 63.4% for males). Proteins contributed less than 20% of energy intakes (18.6% for females and 19.3% for males), with almost 60% of females consuming less than 0.8 g per body weight per day (Table 4.5). Fats contributed less than 20% of energy (16% for females and 17.3% for males).

**Table 4.4: Energy and macronutrients intakes (means and standard deviations) of participants**

| Macronutrients | Females (n=97) | Males (n=25) |
|---|---|---|
| **Energy (kJ)** | 5360.2 ± 1235.8 | 6893.0 ± 1541.7 |
| **Carbohydrates (g)** | 206.8 ± 52.5<br>(65.7% of TE) | 257.1 ± 66.9<br>(63.4% of TE) |
| **Proteins (g)** | 58.5 ± 15.6<br>(18.6% of TE) | 78.1 ± 17.4<br>(19.3% of TE) |
| **Fats (g)** | 22.6 ± 7.9<br>(16.0% of TE) | 31.3 ± 10.7<br>(17.3% of TE) |

**Table 4.5: The daily protein intakes of participants per kg body weight**

| Proteins in g/kg/day | Females (n=97) | | Males (n=25) | |
|---|---|---|---|---|
| | N | % | n | % |
| < 0.8g/kg/day | 58 | 59.8 | 5 | 20.0 |
| 0.8 – 1.0g/kg/day | 21 | 21.6 | 11 | 44.0 |
| > 1.0g/kg/day | 18 | 18.6 | 9 | 36.0 |

### 4.3.2.2 Frequency of consumption of specific foods in the diet

The frequency with which specific foods were consumed, are summarized in Table 4.6 to 4.12 and generally supports the results from the usual daily dietary intake reports summarized above. As indicated in Table 4.6, the only foods from the bread, grains and cereals group (Table 4.6) that were consumed on a daily basis, were stiff porridge (papa) (91.9%) and brown bread (71.0%).

**Table 4.6: Frequency of bread, grains and cereals consumed by the participants (N=124)**

| Food and food groups | Daily | | Weekly | | Monthly | |
|---|---|---|---|---|---|---|
| | n | % | n | % | n | % |
| **Bread, grains and cereals (N= 124)** | | | | | | |
| Papa (mealie meal stiff porridge) | 114 | 91.9 | 5 | 4.0 | 2 | 1.6 |
| Mabele porridge | 9 | 7.3 | 101 | 81.5 | 3 | 2.4 |
| Oats porridge | 0 | 0.0 | 52 | 41.9 | 10 | 8.1 |
| Motoho (sour porridge) | 0 | 0.0 | 13 | 10.5 | 46 | 37.1 |
| Weetbix | 0 | 0.0 | 33 | 26.6 | 1 | 0.8 |
| Corn flakes | 0 | 0.0 | 3 | 2.4 | 0 | 0.0 |
| All bran | 0 | 0.0 | 3 | 2.4 | 0 | 0.0 |
| Muesli | 0 | 0.0 | 0 | 0.0 | 0 | 0.0 |
| Pronutro | 0 | 0.0 | 0 | 0.0 | 0 | 0.0 |
| Morvite | 0 | 0.0 | 2 | 1.6 | 0 | 0.0 |
| White bread | 1 | 0.8 | 2 | 1.6 | 0 | 0.0 |
| Brown bread | 88 | 71.0 | 29 | 23.4 | 1 | 0.8 |
| Provita | 0 | 0.0 | 0 | 0.0 | 0 | 0.0 |
| Pasta | 0 | 0.0 | 10 | 8.1 | 10 | 8.1 |
| Potatoes | 0 | 0.0 | 12 | 9.7 | 26 | 21.0 |
| Rice /Mealie rice | 0 | 0.0 | 111 | 89.5 | 8 | 6.5 |
| Samp | 0 | 0.0 | 26 | 21.0 | 65 | 52.4 |
| Dried beans | 2 | 1.6 | 114 | 91.9 | 4 | 3.2 |
| Baked beans | 0 | 0.0 | 0 | 0.0 | 10 | 8.1 |
| Corn on the cob | 0 | 0.0 | 34 | 27.4 | 15 | 12.1 |
| Popcorn | 0 | 0.0 | 2 | 1.6 | 0 | 0.0 |

While cooked breakfast cereals (mabele porridge [81.5%] and oats porridge [41.9%]) were consumed on a weekly basis, ready-to-eat cereals were seldom consumed, except for Weetbix (consumed by 26.6% of participants on a weekly basis). Sour porridge (motoho) was not very popular (consumed by 37.1%, but only on a monthly basis). Rice/mealie rice (89.5%) and corn on the cob (27.4%) were mostly consumed on a weekly basis, but most participants only consumed samp (52.4%), and potatoes (21%) on a monthly basis. Dried beans was consumed by 91.9% on a weekly basis, but very few of the participants reported consuming baked beans (available in tinned form) and then only on a monthly basis. Few participants consumed pasta or popcorn, while none of participants consumed crisp bread (Provita).

According to Table 4.7 which summarises the frequency of fruit and vegetable intakes of the participants, 62.1% of the participants consumed fruits daily, although according to Table 4.3 they did not meet the daily recommendations of 2 to 4 servings. Fruits were mostly consumed fresh, while only a small percentage of participant consumed fruit juice and mostly only on a monthly basis. Consumption of canned fruit was negligible and wild fruits and dried fruits were not consumed at all.

Very few participants consumed vegetables daily; rather vegetables were mostly consumed on a weekly basis, with spinach (92.7%), sepaile (91.9%), cabbage (91.1%), tomatoes (96.8%), green pepper (80.6%) and onions (87.1%), being the vegetables that were mostly used from the low carbohydrate group. Among the vegetables with higher carbohydrate content, carrots were the only vegetables consumed on a weekly basis (96.0%), while pumpkin (in the form of butternut) were mostly consumed on a monthly basis (64.5%). About a third of the participants (29.8%) consumedly green peas on a weekly, and another 38.7% on a monthly basis.

**Table 4.7: Frequency of fruit and vegetable consumed by the participants (N=124)**

| Food and food groups | Daily | | Weekly | | Monthly | |
|---|---|---|---|---|---|---|
| | n | % | n | % | n | % |
| **Fruits** | | | | | | |
| Fresh fruits | 77 | 62.1 | 39 | 31.5 | 3 | 2.4 |
| Dried fruits | 0 | 0.0 | 0 | 0.0 | 0 | 0.0 |
| Fruit juice | 2 | 1.6 | 10 | 8.1 | 17 | 13.7 |
| Canned fruit | 0 | 0.0 | 0 | 0.0 | 2 | 1.6 |
| Wild fruits | 0 | 0.0 | 0 | 0.0 | 0 | 0.0 |
| **Vegetables:**<br>**Vegetables with almost negligible carbohydrate content** | | | | | | |
| Spinach | 9 | 7.3 | 115 | 92.7 | 0 | 0.0 |
| Lepu (pumpkin leaves) | 2 | 1.6 | 13 | 10.5 | 56 | 45.2 |
| Sepaile | 5 | 4.0 | 114 | 91.9 | 2 | 1.6 |
| Radish | 0 | 0.0 | 17 | 13.7 | 11 | 8.9 |
| Wild vegetables | 0 | 0.0 | 10 | 8.1 | 2 | 1.6 |
| Cabbage | 1 | 0.8 | 113 | 91.1 | 3 | 2.4 |
| Green beans | 0 | 0.0 | 2 | 1.6 | 88 | 71.0 |
| Cauliflower | 0 | 0.0 | 4 | 3.2 | 4 | 3.2 |
| Broccoli | 0 | 0.0 | 1 | 0.8 | 3 | 2.4 |
| Mushrooms | 0 | 0.0 | 4 | 3.2 | 3 | 2.4 |
| Onions | 13 | 10.5 | 108 | 87.1 | 0 | 0.0 |
| Lettuce | 0 | 0.0 | 17 | 13.7 | 21 | 16.9 |
| Cucumber | 0 | 0.0 | 17 | 13.7 | 19 | 15.3 |
| Frozen vegetables | 0 | 0.0 | 5 | 4.0 | 9 | 7.3 |
| Mixed vegetables | 0 | 0.0 | 8 | 6.5 | 8 | 6.5 |
| Green pepper | 0 | 0.0 | 100 | 80.6 | 2 | 1.6 |
| Tomato | 0 | 0.0 | 120 | 96.8 | 2 | 1.6 |
| **Vegetable with higher carbohydrate content (on average 7g per 125ml of cooked vegetables)** | | | | | | |
| Butternut | 0 | 0.0 | 37 | 29.8 | 80 | 64.5 |
| Carrots | 2 | 1.6 | 119 | 96.0 | 0 | 0.0 |
| Green peas | 0 | 0.0 | 37 | 29.8 | 48 | 38.7 |

**Table 4.8: Frequency of milk and dairy products consumed by the participants (N=124)**

| Food and food groups | Daily | | Weekly | | Monthly | |
|---|---|---|---|---|---|---|
| | n | % | n | % | n | % |
| **Milk and milk products** | | | | | | |
| Full cream milk | 23 | 18.9 | 43 | 34.7 | 6 | 4.8 |
| Low-fat milk | 11 | 8.9 | 20 | 16.1 | 0 | 0.0 |
| Skimmed milk | 1 | 0.8 | 4 | 3.2 | 0 | 0.0 |
| Flavoured yoghurt | 0 | 0.0 | 1 | 0.8 | 2 | 1.6 |
| Plain yoghurt | 0 | 0.0 | 0 | 0.0 | 0 | 0.0 |
| Maas/sour milk | 1 | 0.8 | 66 | 53.2 | 2 | 1.6 |

In support of the usual dietary intake data, the non-quantified FFQ (summarized in Table 4.8) indicated that most participants did not consume dairy products on a daily basis. Of the participants, 18.9% consumed full cream milk, 8.9% low-fat milk, and 0.8% skimmed milk, daily; which counts up to about 30% at best, if it is assumed that all participants only used one of the milk types). While fresh milk, mostly as full cream milk, was the only dairy product consumed on a daily basis, sour milk seemed to be the dairy product which was most commonly used, albeit only on a weekly basis (by 53.2%). Yoghurt consumption was very uncommon among these participants.

According to Table 4.9, food from the meat and meat substitutes group were not consumed on a daily basis, with the exception of eggs (used by 13.7% daily). The meats that were mostly consumed by participants were chicken, tinned fish and pork, followed by beef. These meats were only consumed on a weekly basis (chicken by 95.2%; tinned fish, particularly pilchard, by 91.9%, and pork by 23.4%). Beef was consumed by only a small percentage weekly (14.5%) or monthly (15.3%). Lamb consumption was almost negligible, and very few participants consumed white fish (hake fillets). A small percentage of participants (20.2%) consumed organ meats (offals) on a weekly basis, and more than half of participants (57.3%) used processed meats (Russians, polony and viennas) on a weekly basis. While the majority of participants used eggs at least every week, cheese consumption was almost negligible.

With regard to legumes, nine out of ten (91.9%) participants used dried beans (Tables 4.9), and 38.7% used peanut butter weekly, but very few used texturized proteins and soya milk.

**Table 4.9: Frequency of meat and meat substitutes consumed by the participants**

| Food and food groups | Daily | | Weekly | | Monthly | |
|---|---|---|---|---|---|---|
| | n | % | n | % | n | % |
| **Meat and meat substitutes (N=124)** | | | | | | |
| Beef | 0 | 0.0 | 18 | 14.5 | 19 | 15.3 |
| Lamb | 0 | 0.0 | 1 | 0.8 | 13 | 10.5 |
| Pork | 0 | 0.0 | 29 | 23.4 | 15 | 12.1 |
| Chicken | 3 | 2.4 | 118 | 95.2 | 2 | 1.6 |
| White fish (Hake) | 0 | 0.0 | 9 | 7.3 | 14 | 11.3 |
| Tinned fish (pilchards, tuna) | 0 | 0.0 | 114 | 91.9 | 3 | 2.4 |
| Russians/polony/viennas | 0 | 0.0 | 71 | 57.3 | 7 | 5.6 |
| Offals | 0 | 0.0 | 25 | 20.2 | 6 | 4.8 |
| Soy milk | 1 | 0.8 | 0 | 0.0 | 0 | 0.0 |
| Texturized protein (Imana) | 0 | 0.0 | 4 | 3.2 | 0 | 0.0 |
| Eggs | 17 | 13.7 | 84 | 67.7 | 10 | 8.1 |
| Cheese | 1 | 0.8 | 7 | 5.6 | 5 | 4.0 |
| Peanut butter | 1 | 0.8 | 48 | 38.7 | 6 | 4.8 |

According to Table 4.10 the only type of food from the fats and oil group that was used with any regularity was sunflower oil, which was used by nine out of ten participants (90.3%) every day. A very small percentage of participants used canola or olive oils daily for food preparation. Less than 20% of participants used any margarine or mayonnaise in food on a weekly basis, and very few participants used butter or lard. Avocado, cream, bacon and non-dairy coffee creamer were not used at all. A very small percentage of participants consumed nuts, but only monthly, and seeds were not consumed at all.

**Table 4.10: Frequency of fats and oils consumed by the participants**

| Food and food groups | Daily | | Weekly | | Monthly | |
|---|---|---|---|---|---|---|
| | n | % | n | % | n | % |
| **Fats/oils (N=124)** | | | | | | |
| Canola oil | 6 | 4.8 | 0 | 0.0 | 0 | 0.0 |
| Olive oil | 5 | 4.0 | 1 | 0.8 | 0 | 0.0 |
| Sunflower oil | 112 | 90.3 | 0 | 0.0 | 0 | 0.0 |
| Margarine | 5 | 4.0 | 13 | 10.5 | 1 | 0.8 |
| Butter | 0 | 0.0 | 2 | 1.6 | 1 | 0.8 |
| Mayonnaise | 0 | 0.0 | 21 | 16.9 | 5 | 4.0 |
| Nuts | 0 | 0.0 | 2 | 1.6 | 16 | 12.9 |
| Seeds | 0 | 0.0 | 0 | 0.0 | 0 | 0.0 |
| Avocado | 0 | 0.0 | 0 | 0.0 | 0 | 0.0 |
| Cream | 0 | 0.0 | 0 | 0.0 | 0 | 0.0 |
| Bacon | 0 | 0.0 | 0 | 0.0 | 0 | 0.0 |
| Non-dairy coffee creamer | 0 | 0.0 | 0 | 0.0 | 0 | 0.0 |
| Lard | 0 | 0.0 | 1 | 0.8 | 0 | 0.0 |

Though the majority of participants reported using peanut butter on their bread, a few of them were using margarine ("rama"). As indicated in Table 4.11, almost half of participants (54.8%) used sugar daily to sweeten tea (the majority of participants used an average of two teaspoons of sugar in a cup of tea). One participant reported using either honey or sugar in tea. Honey, jam, as well as sweets, chocolates, desserts and biscuits were rarely consumed.

**Table 4.11: Frequency of sugar and sweets consumed by the participants**

| Food and food groups | Daily | | Weekly | | Monthly | |
|---|---|---|---|---|---|---|
| | n | % | n | % | n | % |
| **Sugar/sweets (N=124)** | | | | | | |
| Sugar | 68 | 54.8 | 3 | 2.4 | 2 | 1.6 |
| Syrup | 0 | 0.0 | 0 | 0.0 | 0 | 0.0 |
| Honey | 1 | 0.8 | 0 | 0.0 | 0 | 0.0 |
| Jam | 0 | 0.0 | 3 | 2.4 | 1 | 0.8 |
| Sweets | 2 | 1.6 | 1 | 0.8 | 1 | 0.8 |
| Chocolate | 0 | 0.0 | 0 | 0.0 | 0 | 0.0 |
| Desserts | 0 | 0.0 | 1 | 0.8 | 0 | 0.0 |
| Biscuits | 0 | 0.0 | 0 | 0.0 | 0 | 0.0 |

**Table 4.12: Frequency of miscellaneous foods /beverage consumed by the participants**

| Food and food groups | Daily | | Weekly | | Monthly | |
|---|---|---|---|---|---|---|
| | n | % | n | % | n | % |
| **Miscellaneous (N=124)** | | | | | | |
| Diabetic products | 1 | 0.8 | 0 | 0.0 | 0 | 0.0 |
| Spreads (cheese, fish) | 0 | 0.0 | 0 | 0.0 | 0 | 0.0 |
| Vinegar | 0 | 0.0 | 0 | 0.0 | 0 | 0.0 |
| Salt | 124 | 100.0 | 0 | 0.0 | 0 | 0.0 |
| Aromat | 8 | 6.5 | 45 | 36.3 | 1 | 0.8 |
| Spices | 15 | 12.1 | 64 | 51.6 | 0 | 0.0 |
| Herbs | 1 | 0.8 | 8 | 6.5 | 1 | 0.8 |
| Pepper | 0 | 0.0 | 8 | 6.5 | 0 | 0.0 |
| Stock cubes | 16 | 12.9 | 58 | 46.8 | 7 | 5.6 |
| Packet soups | 0 | 0.0 | 3 | 2.4 | 6 | 4.8 |
| Simbas | 0 | 0.0 | 2 | 1.6 | 14 | 11.3 |
| Fast foods | 0 | 0.0 | 2 | 1.6 | 7 | 5.6 |
| Sweeteners | 6 | 4.8 | 0 | 0.0 | 0 | 0.0 |
| Supplements | 0 | 0.0 | 0 | 0.0 | 0 | 0.0 |
| Bran | 0 | 0.0 | 0 | 0.0 | 0 | 0.0 |
| **Beverages (N=124)** | | | | | | |
| Soft drinks | 8 | 6.5 | 34 | 27.4 | 12 | 9.7 |
| Cordials | 3 | 2.4 | 11 | 8.9 | 1 | 0.8 |
| Fruit juice | 2 | 1.6 | 16 | 12.9 | 28 | 22.6 |
| Tea | 69 | 55.6 | 26 | 21.0 | 3 | 2.4 |
| Coffee | 0 | 0.0 | 1 | 0.8 | 1 | 0.8 |
| Hot chocolate | 0 | 0.0 | 0 | 0.0 | 0 | 0.0 |
| Milo | 0 | 0.0 | 0 | 0.0 | 0 | 0.0 |
| Alcohol | 2 | 1.6 | 2 | 1.6 | 9 | 7.3 |

As indicated in Table 4.12, less than 5% of participants used sweeteners (and those that did, did so daily). All participants added salt on a daily basis, and to flavour food. Other flavourants were used more on a weekly basis, including spices (51.6%), aromat (36.3%) and stock cubes (46.8%) were rather used on a weekly basis as food flavourants. Pepper, herbs and packet soups were not popular. Fast foods and hard chips (‘*Simbas’*) were only consumed by a few participants and mostly only on a monthly basis. Special diabetic foods were very rarely

consumed. None of the participants added bran to their foods or used sandwich spreads (like fish paste or cheese spreads), or any food supplements. Fast foods were also only consumed by a very small percentage of participants.

Tea was the most commonly consumed beverage (consumed by 55.6% on a daily basis). Coffee intake was negligible, and Milo and hot chocolate were not consumed at all. Soft drinks and cordials were consumed mostly on a weekly basis by 27.4% and 8.9% of the participants, respectively. About a third of participants consumed fruit juice (12.9% on a weekly basis and 22.6% on a monthly basis). Very low alcohol consumption was also reported in the FFQ.

### 4.3.3 Lifestyle factors

The information of the physical activity, alcohol intake and smoking habits were also obtained during the structured one-on-one interviews, and are summarized in Tables 4.13 to 4.15.

**Table 4.13: Physical activity levels of the participants (N=124)**

| VARIABLE | FREQUENCY/PERCENTAGES | | | | | |
|---|---|---|---|---|---|---|
| Self-reported physical activity level (assessed by IPAQ) | Total group (n=124) | | Females (n=99) | | Males (n=25) | |
| | N | % | n | % | n | % |
| Low | 2 | 1.6 | 1 | 1.0 | 1 | 4.0 |
| Moderate | 101 | 81.5 | 89 | 89.9 | 12 | 48.0 |
| High | 21 | 16.9 | 9 | 9.1 | 12 | 48.0 |

The self-reported physical activity is summarized in Table 4.13. According to the IPAQ, most participants (82%) were classified as moderately active. Almost half of the males (48%) were however classified as highly active, compared to only 9.1% of females who fell into this category. The majority (98%) of participants were moderately active for at least seven days of the week, hence meeting the recommendations.

While the majority of all participants (78.2%) reported not using any alcohol, half of the males (52%) in the study were risk drinkers. The most frequently consumed type of alcohol, particularly among the males, was commercial beer. Wine and spirits were not commonly

consumed, and home-brewed beer and ciders were not used at all. Most males reported taking between 4 and 6 units of alcohol per session.

**Table 4.14: Alcohol consumption habits of the participants (N=124)**

| VARIABLE | FREQUENCY/PERCENTAGES | | | | | |
|---|---|---|---|---|---|---|
| **Classification according to self-reported alcohol intake** | **Total group (n=117)** | | **Females (n=92)** | | **Males (n=25)** | |
| | **n** | **%** | **n** | **%** | **n** | **%** |
| Non-drinker | 97 | 78.2 | 86 | 86.9 | 11 | 44.0 |
| Prudent drinker | 2 | 1.6 | 1 | 1.0 | 1 | 4.0 |
| At-risk drinker | 18 | 14.5 | 5 | 5.1 | 13 | 52.0 |
| **The type of alcohol used regularly** | | | | | | |
| Spirits (rum, whisky, gin, vodka) | 2 | 1.6 | 0 | 0.0 | 2 | 8.0 |
| Wine | 4 | 3.2 | 3 | 3.0 | 1 | 4.0 |
| Cider | 1 | 0.8 | 1 | 1.0 | 0 | 0.0 |
| Commercial Beer | 13 | 10.5 | 2 | 2.0 | 11 | 44.0 |
| Homebrew | 0 | 0.0 | 0 | 0.0 | 0 | 0.0 |
| None | 104 | 83.9 | 93 | 93.9 | 11 | 44.0 |
| **The frequency of alcohol use** | | | | | | |
| Daily | 0 | 0.0 | 0 | 0.0 | 0 | 0.0 |
| Weekly | 5 | 4.0 | 2 | 2.0 | 3 | 12.0 |
| Monthly | 4 | 3.2 | 0 | 0.0 | 4 | 16.0 |
| Occasionally | 11 | 8.9 | 4 | 4.0 | 7 | 28.0 |
| Never | 104 | 83.9 | 93 | 93.9 | 11 | 44.0 |
| **Number of units used per session** | | | | | | |
| 0 units | 104 | 83.9 | 93 | 93.9 | 11 | 44.0 |
| 1 unit | 1 | 0.8 | 1 | 1.0 | 0 | 0.0 |
| 2 units | 1 | 0.8 | 0 | 0.0 | 1 | 4.0 |
| 3 units | 1 | 0.8 | 1 | 1.0 | 0 | 0.0 |
| 4 units | 9 | 7.3 | 2 | 2.0 | 7 | 28.0 |
| 5 units | 2 | 1.6 | 1 | 1.0 | 1 | 4.0 |
| 6 units | 5 | 4.0 | 1 | 1.0 | 4 | 16.0 |
| 7 units | 0 | 0.0 | 0 | 0.0 | 0 | 0.0 |
| 8 units | 1 | 0.8 | 0 | 0.0 | 1 | 4.0 |

As summarized in Table 4.15, the majority of participants (86.3%) reported that they have never used any form of tobacco, and 3.2% that they were ex-users. Just above 10% participants were current users with cigarette smoking being more common among the males, and snuff among the females. Most users reported using tobacco three times a day.

**Table 4.15: Tobacco habits of the participants (N=124)**

| VARIABLE | FREQUENCY/PERCENTAGES | | | | | |
|---|---|---|---|---|---|---|
| **Classification according to self-reported alcohol intake** | **Total group (n=124)** | | **Females (n=92)** | | **Males (n=25)** | |
| | **n** | **%** | **n** | **%** | **n** | **%** |
| Non-tobacco user | 107 | 86.3 | 87 | 87.9 | 20 | 80.0 |
| Current tobacco user | 13 | 10.5 | 10 | 10.1 | 3 | 12.0 |
| Ex-tobacco user | 4 | 3.2 | 2 | 2.0 | 2 | 8.0 |
| **The type of tobacco used** | | | | | | |
| Cigarettes | 3 | 2.4 | 0 | 0.0 | 3 | 12.0 |
| Snuff | 10 | 8.1 | 10 | 10.1 | 0 | 0.0 |
| Pipe | 0 | 0.0 | 0 | 0.0 | 0 | 0.0 |
| Don't use any tobacco | 111 | 89.5 | 89 | 89.9 | 22 | 88.0 |
| **The frequency of tobacco use / day** | | | | | | |
| 0 times | 112 | 90.3 | 89 | 89.9 | 23 | 92.0 |
| 1 time | 2 | 1.6 | 2 | 2.0 | 0 | 0.0 |
| 2 times | 1 | 0.8 | 1 | 1.0 | 0 | 0.0 |
| 3 times | 6 | 4.8 | 4 | 4.0 | 2 | 8.0 |
| 4 times | 0 | 0.0 | 0 | 0.0 | 0 | 0.0 |
| 5 times | 1 | 0.8 | 1 | 1.0 | 0 | 0.0 |
| 6 times | 1 | 0.8 | 1 | 1.0 | 0 | 0.0 |
| 7 times | 1 | 0.8 | 1 | 1.0 | 0 | 0.0 |

## 4.4 Medical history

Medical history for the purpose of this study included the year of diagnosis, biochemical results, and glucose lowering medication, comorbid conditions, and complications of diabetes mellitus. These results are summarised in Tables 4.16 to Table 4.19.

Table 4.16 illustrated that about half (53.2%) of all the participants (49.5% of the females and 68.0% of the males) had been diagnosed with T2DM for five years or less. Only 5 participants had been living with T2DM for more than 15 years. Most (67.7%) of the participants (68.7% of the females and 64.0% of the males) were on oral medication, 19.4% were using insulin, and very few (4.0%) were on diet and lifestyle therapy only.

Hypertension was the most common comorbid condition reported in the patient files for 92.9% of the females and all the males.

**Table 4.16: Medical history of the participants captured from their medical booklets (N=124)**

| VARIABLES | FREQUENCY/PERCENTAGES | | | | | |
|---|---|---|---|---|---|---|
| **Duration of diabetes** | **Total group (n=124)** | | **Females (n=99)** | | **Males (n=25)** | |
| | **n** | **%** | **n** | **%** | **n** | **%** |
| 0 – 5 years | 66 | 53.2 | 49 | 49.5 | 17 | 68.0 |
| 6 – 10 years | 33 | 26.6 | 29 | 29.3 | 4 | 16.0 |
| 11 – 15 years | 20 | 16.1 | 17 | 17.2 | 3 | 12.0 |
| 16 – 20 years | 2 | 1.6 | 2 | 2.0 | 0 | 0.0 |
| 21 – 25 years | 2 | 1.6 | 1 | 1.0 | 1 | 1.0 |
| 26 – 30 years | 1 | 0.8 | 1 | 1.0 | 0 | 0.0 |
| **Treatment/medication** | | | | | | |
| Diet only | 5 | 4.0 | 4 | 4.0 | 1 | 4.0 |
| Oral agents | 84 | 67.7 | 68 | 68.7 | 16 | 64.0 |
| Insulin | 24 | 19.4 | 17 | 17.2 | 7 | 28.0 |
| Combination* | 11 | 8.9 | 10 | 10.1 | 1 | 4.0 |
| **Comorbid conditions** | | | | | | |
| Hypertension (as measured & reported) | 117 | 94.4 | 92 | 92.9 | 25 | 100.0 |

*Combination of oral medication with insulin.

Table 4.17 summarizes the self-reported microvascular complications. The information regarding chronic complications of diabetes (retinopathy, neuropathy and nephropathy) was self-reported by the patients, because there was no routine screening for chronic complications in their respective clinics. Of the participants, 7.3% reported that they had retinopathy, and 4.0% that they had neuropathy. None of the participants however reported that they had nephropathy.

**Table 4.17: Self-reported chronic microvascular complications of diabetes (N=124)**

| VARIABLES | FREQUENCY/PERCENTAGES | | | | | |
|---|---|---|---|---|---|---|
| **Micro vascular complications** | **Total group (n=124)** | | **Females (n=99)** | | **Males (n=25)** | |
| | **n** | **%** | **n** | **%** | **n** | **%** |
| Retinopathy | 9 | 7.3 | 9 | 9.1 | 0 | 0.0 |
| Neuropathy | 5 | 4.0 | 4 | 4.0 | 1 | 4.0 |
| Nephropathy | 0 | 0.0 | 0 | 0.0 | 0 | 0.0 |

The results for the blood lipids (as captured from the patient files) and the blood pressure (as measured by the nurse at the clinic before the interview) are captured in Table 4.18. Blood lipids (triglycerides and total cholesterol levels) were not available in the files, except for one participant (with total cholesterol of 6.9 mmol/L). The majority of participants (94.4%) had uncontrolled blood pressure.

**Table 4.18: Blood lipids and blood pressure measurements of the participants**

| VARIABLES | FREQUENCY/PERCENTAGES | | | | | |
|---|---|---|---|---|---|---|
| **Total cholesterol (N=2)** (captured from patient booklets) | **n** | **%** | **n** | **%** | **n** | **%** |
| | **(n=2)** | | **(n=2)** | | **(n=0)** | |
| < 4.5 mmol/L | 0 | 0.0 | 0 | 0.0 | 0 | 0.0 |
| > 4.5 mmol/L | 2 | 100.0 | 2 | 100.0 | 0 | 0.0 |
| **Triglycerides (N=0)** (captured from patient booklets) | **(n=0)** | | **(n=0)** | | **(n=0)** | |
| < 1.7 mmol/L | 0 | 0.0 | 0 | 0.0 | 0 | 0.0 |
| > 1.7 mmol/L | 0 | 0.0 | 0 | 0.0 | 0 | 0.0 |
| **Blood pressure (N=124)** (measured by the nurse before the interview) | **(n=124)** | | **(n=99)** | | **(n=25)** | |
| < 140/80 mmHg | 7 | 5.6 | 7 | 7.1 | 0 | 0.0 |
| > 140/80 mmHg | 117 | 94.4 | 92 | 92.9 | 25 | 100.0 |

## 4.5 Glycemic control

Results for fasting blood glucose levels (measured at the clinic on the day of the interview) and the $HbA_{1c}$ levels (current and retrospective (where available), are summarized in Table 4.19.

**Table 4.19: Glycemic control of participants (N=124)**

| VARIABLES | | FREQUENCY/PERCENTAGES | | | | | |
|---|---|---|---|---|---|---|---|
| **Fasting blood glucose levels (N=124)** | | **Total group (n=124)** | | **Females (n=97)** | | **Males (n=25)** | |
| | | **n** | **%** | **n** | **%** | **n** | **%** |
| < 4 mmol/L | | 5 | 4.0 | 3 | 3.1 | 2 | 8.0 |
| 4 – 7 mmol/L | | 40 | 32.3 | 35 | 36.1 | 5 | 20.0 |
| > 7 mmol/L | | 77 | 62.1 | 59 | 60.9 | 18 | 72.0 |
| **$HbA_{1c}$ levels* (N=94)** | | **(n=94)** | | **(n=74)** | | **(n=20)** | |
| < 7% | Optimal | 38 | 30.6 | 33 | 44.6 | 5 | 25.0 |
| 7-8% | Acceptable | 11 | 8.9 | 9 | 12.2 | 2 | 10.0 |
| > 8% | Suboptimal | 45 | 36.3 | 32 | 43.2 | 13 | 65.0 |

***HbA1c levels (45 were current results and 49 retrospective)

Results demonstrated that three quarter of the participants (62.1%) had fasting blood glucose levels above 7.0mmol/L; more than a third (32.3%) of participants had acceptable fasting blood glucose levels (4-7mmol/L), while very few had fasting blood glucose of less than 4.0mmol/L. Assessment of blood glucose control over the previous three to four months by means of $HbA_{1c}$ measurements confirmed that most participants (36.3%) had suboptimal glucose control ($HbA_{1c}$ > 8%). The results for $HbA_{1c}$ further demonstrated a better blood glucose control among the females participants (44.6% with optimal control at $HbA_{1c}$ < 7%), than among the males (65.0% with suboptimal blood glucose control ($HbA_{1c}$ > 8%).

### 4.6 Barriers that may impact on treatment compliance

Barriers that may impact on treatment compliance, in this study referred to factors related to the logistics of attending the clinic, participants experience of the services at the clinics, and participants' knowledge, attitudes, perceptions and practices regarding diet and lifestyle, physical activity, alcohol and tobacco use, self-care and medical therapies, health beliefs, cultural and traditional attitudes towards diabetes care; information on which was gathered by the researcher in structured one-on-one interviews with the participants using a questionnaire. The results are summarized in Table 4.20 to 4.27.

**Table 4.20: Factors regarding the logistics of attending the diabetes clinic (N = 124)**

| QUESTIONS | OPTIONS (choose one) | FREQUENCY/PERCENTAGES | |
|---|---|---|---|
| | | n | % |
| 1. Did you used to attend the QE II diabetes clinic? | Yes | 78 | 62.9 |
| | No | 46 | 37.1 |
| 2. Which clinic do you attend now (since Oct 2011)? | Domiciliary | 54 | 43.5 |
| | LDF | 74 | 59.7 |
| | Other | 9 | 7.3 |
| 3. Why do you come to the city clinics? | Like clinics in Maseru | 0 | 0.0 |
| | Child lives in Maseru | 3 | 2.4 |
| | No DM clinic at home | 1 | 0.8 |
| | I live in Maseru | 120 | 96.8 |
| 4. How often do you go to a diabetes clinic? | Once per month | 0 | 0.0 |
| | Every two months | 0 | 0.0 |
| | Every third month | 124 | 100 |
| 5. Do you have to sleep over in Maseru when you attend the clinic? | Yes | 3 | 2.4 |
| | No | 121 | 97.6 |
| 6. a) If you sleep over do you have to pay for accommodation? | Yes | 1 | 0.8 |
| | No | 123 | 99.2 |
| b) If yes, how much? | M100-M150 | 2 | 1.6 |
| 7. How do you reach the clinic? | Drive self to the clinic | 0 | 0.0 |
| | Driven to the clinic | 9 | 7.3 |
| | Take a taxi | 94 | 75.8 |
| | Walk | 21 | 16.9 |
| 8. a) Do you pay for transport? | Yes | 91 | 74.0 |
| | No | 31 | 25.0 |
| b) If yes, how much? | Nothing | 31 | 25.0 |
| | M5.50 | 5 | 4.0 |
| | M10.00 | 0 | 0.0 |
| | M11.00 | 75 | 60.5 |
| | >M25.00 | 13 | 10.5 |
| 9. As a result of your diabetes, are you retired or currently not working? | Yes | 2 | 1.6 |
| | No | 121 | 97.6 |
| 10. Do you sometimes miss to attend clinic because of lack of money? | Yes | 48 | 38.7 |
| | No | 76 | 61.3 |

### 4.6.1 Factors regarding the logistics of attending the diabetes clinic

Table 4.20 summarizes the participants' responses to questions regarding the logistic of attending the clinic in Maseru. All but four of the participants lived in Maseru, and therefore attended the clinics there; 62.9% used to be regular attendees of the diabetes clinic at QE II hospital. Of the four from outside Maseru, three attended the Maseru clinics because their children lived in the city and one because there were no diabetes clinics near their home. As the group from outside Maseru was so small, information regarding the cost incurred by having to sleep over in the city when attending the clinics, could not be meaningfully evaluated.

All of the participants were scheduled to attend the clinic every third month only. More than three quarters (76%) of participants travelled to the clinic by taxi, while 17% walked there. Transport fees for most participants were M11.00 or more, with 10.5% paying more than M25.00. More than a third (38.7%) of participants reported that they sometimes missed to attend the clinic due to lack of money.

### 4.6.2 The participants experience of the services at the clinics

Table 4.21 summarizes the participants' responses to questions (questions 1 to 8) regarding their experience of the services they receive at the clinics. About a third reported that they are usually serviced at the clinic within an hour of arrival, but most (57.3%) report that they wait between one and two hours. The services offered entails having blood pressure and blood glucose measured by a nurse, then consulting a doctor, and then collecting their medicines from the pharmacy. Between each step the waiting period was reported to be for most participants between one and two hours. Thus the total time spent at the clinic tallies up to between three to six hours. Most (75.8%) of the participants indicated that they felt that the services at the clinics takes too long. Most participants (85.5%) reported that they arrive at the clinics between 6:00 and 8:00 in the morning (the rest arrive even earlier between 4:00 and 6:00), and arrive at home or back at work, between 12:00 and 13h00.

Nonetheless, participants (98.4%) acknowledged that the clinics provided good and up to standard services, and that the communication relationships between the participants and health care providers were good (66.1%). Most participants reported that they are not always serviced by the same nurse (86.3%) or doctor (64.5%), but most indicated that they did not have any problem whether or not they were serviced by the same nurse (93.0%) or doctor (61.0%). Most (96%) were satisfied that they spend adequate time with the doctor. 17.7% reported that they did not always receive all their medications from the pharmacy though. Almost two thirds of participants (61.3%) received the services for free and the rest mostly paid M15.00 per clinic visit. About a third of participants felt that they were not receiving psychosocial and emotional support from the clinic, family members and friends. Most of them (77.4%) did not know about the Lesotho Diabetes Association, hence only 1.6% were members.

**Table 4.21: The participants experience of the services at the clinics (N = 124)**

| QUESTIONS | OPTIONS (choose one) | FREQUENCY/PERCENTAGES | |
|---|---|---|---|
| | | N | % |
| 1. At what time do you arrive at the clinic? | 4:00-5:00 am | 2 | 1.6 |
| | 5:00-6:00 am | 15 | 12.1 |
| | 6:00-7:00 am | 56 | 45.2 |
| | 7:00-8:00 am | 50 | 40.3 |
| 2. How long do you wait before your blood pressure and blood sugar checked at the clinic? | 0:00-30mins | 3 | 2.4 |
| | 30mins-1hr | 34 | 27.4 |
| | 1hr-2 hrs | 71 | 57.3 |
| | 2 hrs-3hrs | 16 | 12.9 |
| 3. How long do you wait before you could be seen by the doctor? | 0:00-30mins | 6 | 4.8 |
| | 30mins-1 hr | 43 | 34.7 |
| | 1hr-2hrs | 68 | 54.8 |
| | 2 hrs-3hrs | 7 | 5.7 |
| 4. Do you feel you have enough time with your doctor? | Yes | 119 | 96.0 |
| | No | 5 | 4.0 |
| 5. What type of medication do you use to control your blood sugar? | Tablets | 84 | 67.7 |
| | Insulin injection | 24 | 19.4 |
| | Diet only | 5 | 4.0 |
| | Tablets and insulin | 11 | 8.9 |
| | Nothing | 0 | 0.0 |
| 6. Do you always receive all your medicines from the pharmacy? | Yes | 102 | 82.3 |
| | No | 22 | 17.7 |
| 7. How long do you wait before you can get your medicines? | 0:00-30mins | 0 | 0.0 |
| | 30mins-1hr | 16 | 12.9 |
| | 1hr-2hrs | 94 | 75.8 |
| | 2 hrs-3hrs | 14 | 11.3 |
| 8. At what time do you normally arrive back home, from the clinic? | 11:00-12:00 | 28 | 22.6 |
| | 12:00-13:00 | 58 | 46.8 |
| | 13:00-14:00 | 25 | 20.2 |
| | 14:00-15:00 | 10 | 8.1 |
| | After 15:00 | 3 | 2.4 |
| 9. Do you think the services at the clinics take too long? | Yes | 94 | 75.8 |
| | No | 14 | 24.2 |
| 10. How much do you pay to attend the clinic? | M15.00 | 45 | 36.3 |
| | M5.50 | 3 | 2.4 |
| | M5.00 | 0 | 0.0 |
| | M0.00 | 76 | 61.3 |
| 11. Do you think the clinic provides good or up to standard services? | Yes | 122 | 98.4 |
| | No | 2 | 1.6 |
| 12. Do you think there is a good communicative relationship between the patient and health care providers at the clinic? | Yes | 82 | 66.1 |
| | No | 42 | 33.9 |
| 13. a) Are your blood pressure and blood sugar checked by the same nurse each time you attend the clinic? | Yes | 17 | 13.7 |
| | No | 107 | 86.3 |
| b) If no. How do you feel about it? | Don't like it | 10 | 8.1 |
| | Not happy | 2 | 1.6 |
| | No problem | 93 | 75.0 |
| | Okay with it | 19 | 15.3 |
| 14. a). Are you consulted by the same doctor each time you attend the clinic? | Yes | 44 | 35.5 |
| | No | 80 | 64.5 |
| b) If no. How do you feel about it? | Don't like it | 20 | 16.1 |

| | | | |
|---|---|---|---|
| | Not happy<br>No problem<br>Okay with it | 27<br>61<br>16 | 21.8<br>49.2<br>12.9 |
| 15. Do you receive psychosocial and emotional support from the clinic, family members and friends? | Yes<br>No | 83<br>41 | 66.9<br>33.1 |
| 16. a) Do you know about the Lesotho Diabetes Association? | Yes<br>No | 28<br>96 | 22.6<br>77.4 |
| b) If yes. Are you a member? | Yes<br>No | 2<br>121 | 1.6<br>98.4 |

### 4.6.3 Knowledge, attitudes, perceptions and practices regarding diet and lifestyle

Tables 4.22 to 4.23 summarize the responses of the participants to questions which probed their knowledge, attitudes, perceptions and practices regarding diet and lifestyle.

According to Table 4.22, most of the participants (52.4%) indicated that they received some form of education from health care providers about healthy eating habits, but 74.2% indicated that they had never received any written instructions or education from a dietitian and/or nutritionist. Although most of the participants (>99%) could identify healthy dietary recommendations, and (82.3%) reported that they adhere to healthy eating habits, some reported that they failed to adhere to healthy eating guidelines because of the financial constraints (65.3%) and poor self-control (90.3%).

**Table 4.22: Knowledge, attitudes, perceptions and practices of participants regarding diet (N= 124)**

| QUESTIONS | OPTIONS (choose one) | FREQUENCY/PERCENTAGES | |
|---|---|---|---|
| | | n | % |
| 1. a) Did you receive any education from a health care provider about healthy eating habits? | Yes<br>No | 65<br>59 | 52.4<br>47.6 |
| b) If yes. Are these healthy dietary recommendations?: | | | |
| i) Increase fibre intake. | Yes<br>No | 123<br>1 | 99.2<br>0.8 |
| ii) Avoid animal fats. | Yes<br>No | 122<br>2 | 98.4<br>1.6 |
| iii) Avoid sugar. | Yes<br>No | 124<br>0 | 100<br>0.0 |
| iv) Eat sweets. | Yes<br>No | 2<br>122 | 1.6<br>98.4 |
| v) Use salt/oil liberally. | Yes<br>No | 4<br>120 | 3.2<br>96.8 |
| 2. Have you ever received a detailed written instructions regarding dietary intake by a health worker (dietitian/nutritionist)? | Yes<br>No | 32<br>92 | 25.8<br>74.2 |

| | | | |
|---|---|---|---|
| 3. a) Do you adhere to any form of healthy dietary guidelines? | Yes<br>No | 115<br>22 | 82.3<br>17.7 |
| b) If Yes. Which healthy dietary guidelines are you adhering to?: | | | |
| i) Eat a variety of foods. | Yes<br>No | 102<br>22 | 82.3<br>17.7 |
| ii) Make starchy foods the basis of most meals. | Yes<br>No | 115<br>9 | 92.7<br>7.3 |
| iii) Eat dry beans, peas, lentils and soya regularly. | Yes<br>No | 119<br>5 | 96.0<br>4.0 |
| iv) Eat chicken, fish, milk, meat or eggs daily. | Yes<br>No | 98<br>26 | 79.0<br>21.0 |
| v) Drink lots of clean, safe water. | Yes<br>No | 106<br>18 | 85.5<br>14.5 |
| vi) Eat plenty of vegetables and fruits every day. | Yes<br>No | 73<br>51 | 58.9<br>41.1 |
| vii) Eat fats sparingly, avoid saturated fats & trans-fats. | Yes<br>No | 111<br>13 | 89.5<br>10.5 |
| viii) Use salt sparingly. | Yes<br>No | 111<br>13 | 89.5<br>10.5 |
| ix) Use food and drinks containing sugar sparingly and not between meals. | Yes<br>No | 90<br>34 | 72.6<br>27.4 |
| x) Drinks alcohol sensibly. | Yes<br>No | 103<br>21 | 83.1<br>16.9 |
| xi) None of the above. | Yes<br>No | 0<br>124 | 0.0<br>100 |
| 4. What is your reason (s) for not adhering to the dietary recommendations? | | | |
| i) Often eating out. | Yes<br>No | 13<br>111 | 10.5<br>89.5 |
| ii) Financial constraints. | Yes<br>No | 81<br>43 | 65.3<br>34.7 |
| iii) Poor self-control. | Yes<br>No | 112<br>12 | 90.3<br>9.7 |
| iv) Difficulty of following a different diet from the rest of the family. | Yes<br>No | 41<br>83 | 33.0<br>66.9 |
| v) Lots of travelling. | Yes<br>No | 4<br>120 | 3.2<br>96.8 |
| vi) High frequency of social gatherings. | Yes<br>No | 1<br>123 | 0.8<br>99.2 |
| vii) Other (specify) No reasons. | Yes<br>No | 2<br>121 | 1.6<br>98.4 |
| 5. Do you feel that healthy dietary habits have a role to play in the management of T2DM? | Yes<br>No | 123<br>1 | 99.2<br>0.8 |
| 6. Do you believe that good dietary habits could help control and maintain your blood sugar? | Yes<br>No | 124<br>0 | 100<br>0.0 |

According to Table 4.23, almost all of participants believed that moderate physical activity has a role in the management of T2DM (99.2%) and in controlling blood glucose levels (99.2%), but none of the participants had ever received any written instructions from a healthcare worker

regarding an exercise program. Most considered gardening (88.7%) and housework (85.5%) as forms of exercise. Almost all participants (98.4%) reported that they felt they were doing exercises, with most (52.0%) selecting gardening as what they perceived to be their main exercise, followed by walking (selected by 24.4%) and housework (selected by 15.5%). Only 8.1% selected going to the gym or taking part in aerobic exercise or jogging. Almost half (49.2%) felt that they are physically active five times per week, and 68.6% reported that they were active for one or more hours at a time. On the other hand most participants also agreed that they do not exercise due to lack of time (87.9%) and/or workloads (69.4%).

**Table 4.23: Knowledge, attitudes, perceptions and practices of participants regarding physical activity, alcohol and tobacco use (N= 124)**

| QUESTIONS | OPTIONS (choose one) | FREQUENCY/PERCENTAGES | |
|---|---|---|---|
| | | n | % |
| 7. What do you understand by the word 'physical activity (exercise)'? | | | |
| i) Brisk walking | Yes | 124 | 100 |
| | No | 0 | 0.0 |
| ii) Housework | Yes | 106 | 85.5 |
| | No | 18 | 14.5 |
| iii) Aerobic exercises. | Yes | 123 | 99.2 |
| | No | 1 | 0.8 |
| **iv)** Gardening | Yes | 110 | 88.7 |
| | No | 14 | 11.3 |
| **v)** Sitting down. | Yes | 1 | 0.8 |
| | No | 123 | 99.2 |
| 8. Do you do any form of moderate physical activity (exercises)? | Yes | 122 | 98.4 |
| | No | 2 | 1.6 |
| 9. What form of physical activities (exercises) are you adhering to? | Walking | 30 | 24.4 |
| | Housework | 19 | 15.5 |
| | Gym/aerobic/jogging | 10 | 8.1 |
| | Gardening | 64 | 52.0 |
| 10. How often do you do your physical activities (exercises)? | 5/week | 61 | 49.2 |
| | 3/week | 35 | 28.2 |
| | 2/week | 17 | 13.7 |
| | 0/week | 11 | 8.9 |
| 11. What duration do you normally take to do your physical activities (exercises)? | ≥1hours | 85 | 68.6 |
| | 45 minutes | 18 | 14.5 |
| | 3 minutes | 18 | 14.5 |
| | 15 minutes | 3 | 2.4 |
| 12. Do you believe that moderate physical activity (exercise) has a role in the management of type 2 diabetes? | Yes | 123 | 99.2 |
| | No | 1 | 0.8 |
| 13. Do you believe that moderate physical activity (exercise) helps to control and maintain blood glucose levels? | Yes | 123 | 99.2 |
| | No | 1 | 0.8 |
| 14. Do you think that moderate physical activities may exacerbate other illnesses? | Yes | 2 | 1.6 |
| | No | 122 | 98.4 |
| 15. If you do not do any form of physical activity (exercise), what are your reasons? | | | |
| i) Unwillingness | Yes | 1 | 0.8 |

| | | | |
|---|---|---|---|
| | No | 121 | 99.2 |
| ii) Lack of time to exercise | Yes | 109 | 87.9 |
| | No | 15 | 12.1 |
| iii) Workloads | Yes | 86 | 69.4 |
| | No | 38 | 30.7 |
| iv) Lack of advice given by health care provider | Yes | 2 | 1.6 |
| | No | 122 | 98.4 |
| v) Coexisting diseases such as osteo-arthritis | Yes | 25 | 20.2 |
| | No | 99 | 79.8 |
| vi) Stressful environment | Yes | 2 | 1.6 |
| | No | 122 | 98.4 |
| 16. Have you ever received detailed written instructions regarding exercise programs from health care provider? | Yes | 0 | 0.0 |
| | No | 124 | 100 |
| 17. a) Do you use any alcoholic beverages? | Yes | 23 | 18.6 |
| | No | 101 | 81.5 |
| b) What is the recommended intake for you? | 1 portion | 7 | 5.7 |
| | 2 portions | 8 | 6.5 |
| | 3 portions | 4 | 3.2 |
| | No idea | 105 | 84.7 |
| 18. What type of alcohol do you think is good for you? | Beer | 5 | 4.0 |
| | Red wine | 56 | 45.2 |
| | Semi-sweet wine | 6 | 4.8 |
| | Whisky | 3 | 2.4 |
| | None | 54 | 43.6 |
| 19. Are you allowed to use any form of tobacco (cigarette or snuff)? | Yes | 0 | 0.0 |
| | No | 124 | 100 |

According to Table 4.23, less than a fifth (18.6%) of participants reported on this questionnaire that they use any form alcohol, but 84.7% of the 124 participants reported having no idea what the recommended limits for alcohol intake were, while 3.2% thought it was 3 drinks per day. Asked what type of alcohol they perceived to be "good for you", 43.6% chose the "no alcohol" option, while 45.2% chose the "red wine" option (although only 3.2% reported using wine [Table 4.14]). Although beer was the most consumed form of alcohol (by 10.5% of participants [Table 4.14]), only 4% thought it was "good for you". Although there were 10.5% current tobacco user among the participants (Table 4.15), all of the participants thought that using tobacco is forbidden in patients with diabetes.

### 4.6.4 Knowledge, attitudes, perceptions and practices regarding self-care

Table 4.24 summarizes the responses of the participants to questions which probed their knowledge, attitudes, perceptions and practices regarding self-care, which for the purpose of this study included the symptoms of elevated blood glucose levels, self-monitoring of blood glucose, complications of diabetes, compliance to medication, and foot care.

Most (98.4%) of the participants reported that they adhere to their prescribed medications, and that they (99.2%) do not just take the medications when symptoms occur, but regularly as prescribed (only 2.4% admitted that they sometimes forget to take their medications). Only one patient reported not having confidence in the benefits of the medications that they use. Most (91.9%) reported that they have received education about the use of the medicines, but 8.1% reported that they did not.

Most to all participants (82.3% to 100%) knew what the symptoms of high blood sugar were. Although 99% of participants reported that they think it is important to "regularly test your blood sugar", 94.4% only have their blood glucose levels tested every third month during their follow-up appointments to the clinics (which 96% reported that they attend as scheduled). Furthermore most participants did not know the normal reference range for fasting blood sugar, with 37.9% admitting that they have no idea what it should be.

**Table 4.24: Knowledge, attitudes, perceptions and practices of participants regarding self-care (N= 124)**

| VARIABLES | | FREQUENCY/PERCENTAGES | |
|---|---|---|---|
| | **OPTIONS (choose one)** | **n** | **%** |
| 1. Please tick the symptoms of high blood sugar? | | | |
| i) Drinking a lot of water | Yes | 124 | 100 |
| | No | 0 | 0.0 |
| ii) Passing a lot of urine | Yes | 124 | 100 |
| | No | 0 | 0.0 |
| iii) Feeling weak & tired | Yes | 123 | 99.2 |
| | No | 1 | 0.8 |
| iv) Swollen body | Yes | 0 | 0.0 |
| | No | 124 | 100 |
| v) Dry mouth | Yes | 102 | 82.3 |
| | No | 22 | 17.7 |
| 2. Please tick the consequences of high blood sugar, over a long period of time. | | | |
| i) Blindness | Yes | 124 | 100 |
| | No | 0 | 0.0 |
| ii) Foot ulcers | Yes | 113 | 91.1 |
| | No | 11 | 8.9 |
| iii) Asthma | Yes | 1 | 0.8 |
| | No | 123 | 99.2 |
| iv) Kidney problem | Yes | 108 | 87.1 |
| | No | 16 | 12.9 |
| v) Arthritis | Yes | 2 | 1.6 |
| | No | 122 | 98.4 |
| 3. What is the reference range of normal fasting blood sugar? | 4.0-7.0mmol/l | 12 | 9.7 |
| | 7.0-8.0mmo/l | 13 | 10.5 |
| | 8.0-10.0mmol/l | 27 | 21.8 |
| | 10.0 -12.0mmol/l | 25 | 20.2 |
| | No idea | 47 | 37.9 |
| 4.Do you think diabetes complications can be prevented? | Yes | 122 | 98.4 |
| | No | 2 | 1.6 |
| 5. Do you take your medicines as prescribed? | Yes | 122 | 98.4 |
| | No | 2 | 1.6 |
| 6.a) Do you take your medicines only when symptoms occur? | Yes | 1 | 0.8 |
| | No | 123 | 99.2 |
| b) If yes. What is/are your reason (s)? | I forget | 3 | 2.4 |
| | I don't know if I take them daily | 0 | 0.0 |
| | I think I am cured | 0 | 0.0 |
| | I don't | 121 | 97.6 |
| 7. Did you receive any education about the use of your medicines? | Yes | 114 | 91.9 |
| | No | 10 | 8.1 |
| 8. Do you have confidence in your medicines' benefits? | Yes | 123 | 99.2 |
| | No | 1 | 0.8 |
| 9. a) Do you think it is important to regularly test your blood sugar? | Yes | 123 | 99 |
| | No | 1 | 0.8 |
| b) How often do you test? | Once/month | 7 | 5.7 |

| | | | |
|---|---|---|---|
| | Weekly | 0 | 0.0 |
| | Every third month | 117 | 94.4 |
| 10. How often are the following checked? | | | |
| i) Eye sight | Three monthly | 0 | 0.0 |
| | Never | 124 | 100 |
| ii) Kidney function | Three monthly | 0 | 0.0 |
| | Never | 124 | 100 |
| iii) Blood pressure | Three monthly | 124 | 100 |
| | Never | 0 | 0.0 |
| iv) Feet | Three monthly | 0 | 0.0 |
| | Never | 124 | 100 |
| 11. Please tick daily personal foot care recommendations. | | | |
| i) Wash feet daily with soap & lukewarm water. | Yes | 124 | 100 |
| | No | 0 | 0.0 |
| ii) Dry feet thoroughly even in-between the toes | Yes | 124 | 100 |
| | No | 0 | 0.0 |
| iii) Carefully cut your nails. | Yes | 122 | 98.4 |
| | No | 2 | 1.6 |
| iv) Apply feet with lotions, except in-between toes | Yes | 123 | 99.2 |
| | No | 1 | 0.8 |
| v) Wear well fitting socks | Yes | 118 | 95.9 |
| | No | 5 | 4.1 |
| vi) Try to wear leather shoes | Yes | 114 | 91.9 |
| | No | 10 | 8.1 |
| vii) Avoid walking bare-footed in & out-doors. | Yes | 102 | 82.3 |
| | No | 22 | 17.7 |
| viii) Wear tight shoes. | Yes | 3 | 2.4 |
| | No | 121 | 97.6 |
| ix) Wash your feet with hot water. | Yes | 0 | 0.0 |
| | No | 124 | 100 |
| x) Keep your feet moist. | Yes | 0 | 0.0 |
| | No | 124 | 100 |
| 12. Do you attend your checkups as scheduled? | Yes | 119 | 96.0 |
| | No | 5 | 4.0 |

Participants could identify the micro vascular complications associated with uncontrolled blood sugar: all participants identified blindness; 91.1% identified foot ulcers and 87.1% identified kidney problems as long-term consequences; and they (98.4%) believed that the complications of diabetes are preventable. Yet, all of the participants reported that they are never screened for any of these complications (which concur with the lack of any formation on these screening tests in the patient files). Participants (90 to 100%) were however well informed about daily personal foot care recommendations (except for about a fifth not being sure about the recommendation to not walk barefoot outside).

### 4.6.5 Health beliefs and cultural and traditional attitudes towards diabetes care

Table 4.25 summarizes the responses of the participants to questions which probed their health beliefs about diabetes, and cultural/traditional attitudes towards diabetes care.

No participants believed that T2DM was caused by witchcraft or that it was punishment from God; while most believed that diabetes was inherited (family history) (99.2%), and/or caused by overweight (82.9%) and "wrong" diet (83.7%). Most of the participants (89.4%) consulted the doctor at the clinic when they first experienced the symptoms of diabetes mellitus; none reported consulting a traditional healer. However, when asked if they knew that diabetes is a life-long disease, almost a fifth (18.7%) answered "No".

Small percentages of participants reported that they use traditional medicines, with "Haelale" being the most common. Very few reported using popular herbal "home remedies", including cinnamon, garlic, ginger and green tea, as well as a small percentage of the participants also used nutritional supplements (12.2% used omega-3 and 5.7% used antioxidants). About 85.4% of the participants reported that, even if they took traditional medicines, herbs, and nutritional supplements, they continued with their prescribed medications (concurring with the confidence they expressed in their medicines in Table 4.24); 14.4% however indicated that when taking these substances they stopped taking their medicines.

## Table 4.25: Health beliefs and cultural and traditional attitudes of participants towards diabetes care (N= 124)

| VARIABLES | FREQUENCY/PERCENTAGES | | |
|---|---|---|---|
| | OPTIONS (choose one) | n | % |
| 14. What are the causes of diabetes mellitus? | | | |
| i) Heredity | Yes | 122 | 99.2 |
| | No | 1 | 0.8 |
| ii) Overweight | Yes | 102 | 82.9 |
| | No | 21 | 17.1 |
| iii) Wrong diet | Yes | 103 | 83.7 |
| | No | 20 | 16.3 |
| iv) Punishment from God | Yes | 0 | 0.0 |
| | No | 123 | 100 |
| v) Witchcraft | Yes | 0 | 0.0 |
| | No | 123 | 100 |
| 2. Whom did you consult when you first experience symptoms of diabetes mellitus? | Doctor at the clinic | 110 | 89.4 |
| | Doctor's surgery | 13 | 10.6 |
| | Popular sector | 0 | 0.0 |
| | Traditional Healer | 0 | 0.0 |
| 3. Do you know that diabetes mellitus is a life-long disease? | Yes | 100 | 81.3 |
| | No | 23 | 18.7 |
| 4. Do you think diabetes mellitus is the result of supernatural forces? | Yes | 3 | 2.4 |
| | No | 120 | 97.6 |
| 5. a) Do you use any traditional medicines? | Yes | 39 | 31.7 |
| | No | 84 | 68.3 |
| b) If yes, which ones? | | | |
| i) Lekhala (Aloe)** | Yes | 16 | 13.0 |
| | No | 107 | 87.0 |
| ii) Hloenya** | Yes | 12 | 9.8 |
| | No | 111 | 90.2 |
| iii) Khomo ea balisa** | Yes | 1 | 0.8 |
| | No | 122 | 99.2 |
| iv) Sehalahala sa matlaka** | Yes | 8 | 6.5 |
| | No | 115 | 93.5 |
| v) Cheche** | Yes | 0 | 0.0 |
| | No | 123 | 100 |
| vi) Other (specify): Haelale** | Yes | 18 | 14.6 |
| | No | 105 | 85.4 |
| 6. a) Do you use any herbal home remedies? | Yes | 14 | 11.4 |
| | No | 109 | 88.6 |
| b) If yes. Please indicate which ones. | | | |
| i) Cinnamon | Yes | 1 | 0.8 |
| | No | 122 | 99.2 |
| ii) Garlic | Yes | 5 | 4.1 |
| | No | 118 | 95.9 |
| iii) Ginger | Yes | 3 | 2.4 |
| | No | 120 | 97.6 |
| iv) Green tea | Yes | 7 | 5.7 |
| | No | 116 | 94.3 |

| | | | |
|---|---|---|---|
| v) Other (specify) Parsley and aloe vera. | Yes | 4 | 3.3 |
| | No | 119 | 96.8 |
| 7. a) Do you use any nutritional supplements? | Yes | 28 | 22.8 |
| | No | 95 | 77.2 |
| b) If yes. Please indicate which ones. | | | |
| i) Multivitamins | Yes | 1 | 0.8 |
| | No | 122 | 99.2 |
| ii) Omega 3's | Yes | 15 | 12.2 |
| | No | 108 | 87.8 |
| iii) Antioxidants | Yes | 7 | 5.7 |
| | No | 116 | 94.3 |
| iv) B-complex | Yes | 2 | 1.6 |
| | No | 121 | 98.4 |
| v) Vitamin C | Yes | 0 | 0.0 |
| | No | 123 | 100 |
| vi) Calcium / magnesium | Yes | 4 | 3.3 |
| | No | 119 | 96.8 |
| vii) Shake | Yes | 1 | 0.8 |
| | No | 122 | 99.2 |
| viii) Other (specify): Tre-en-en (A product sold by Golden) | Yes | 4 | 3.3 |
| | No | 119 | 96.8 |
| 8. When using complementary alternative medicines, do you still continue taking your medicines? | Yes | 105 | 85.4 |
| | No | 18 | 14.6 |

******Indigenous plants that grow in Lesotho.

## Summary

The results of the study indicated that the majority of these Basotho patients with T2DM who participated in the study, were between the ages of 51 and 60 years; married; and mainly resided in urban-Maseru. Although most were employed, most had a low level of income, particularly the females. Most participants had a low education level and had only obtained a high school certificate.

The majority of the participants had been diagnosed with diabetes for a period of five or less years, and was on oral medications. Almost all of the participants had been diagnosed with hypertension and were on hypertensive medications. However, there were no test results screening for dyslipidaemia (triglycerides and total cholesterol) and chronic complications (retinopathy, neuropathy and nephropathy) in the patient's medical booklets.

More than three quarter of the participants had fasting blood glucose above 7.0mml/L on the day of the interview. No SMBG results were done at home before clinic appointments, and very few

had been tested for $HbA_{1c}$. $HbA_{1c}$ levels were measured in the study for 45 of the participants, and the majority had levels above 8%, indicating a suboptimal blood glucose control.

Most participants were classified as overweight or obese, based on BMI, WC, WHtR and BAI measurements; which implies high risk of complications, particularly cardiovascular diseases. WC and WHtR identified more at-risk participants, than BMI, while BAI only identified about two thirds of the number of at-risk participants that WHtR did.

In addition, the participants did not follow prudent dietary recommendations. Their diets were inadequate in milk and dairy products, and fruits and vegetables. Based on the FFQ their diets were low in sources of omega 3 fatty acids.

Despite the poor dietary quality revealed by the analysis of daily intakes and the patterns revealed by the non-quantified FFQ, most participants believed that they were following the SA FBDG (which reflects prudent dietary advice). Nonetheless, some admitted that they sometimes fail to abide by the dietary and lifestyle recommendation, due to financial constraints and poor self-control. However, most patients acknowledged being educated on diet and medication usage, though most of them did not know the normal reference range for fasting blood glucose.

According to their own perceptions most participants reported that they comply with taking their medications as prescribed, although the fasting blood glucose levels and blood pressure readings were high.

According to the IPAQ most of them were moderately active (81.5%), while only 1.0% were inactive females as compared to 4.0% males. The participants however, thought that walking and gardening were the best form of physical activities, as 52.0% of participants reported that they adhered to gardening, and 24.4% to walking. Nonetheless, some admitted that they sometimes fail to abide by the lifestyle recommendation, due to lack of time and/or workload.

While most did not use either alcohol or tobacco, but 14.5% of participants were at-risk drinkers (used four to six units of alcohol per session), and often used commercial beer on a weekly basis.

One tenth of participants also reported that they use tobacco mainly cigarette (by 12.0% of males) and snuff (10.1% of females) three times a day.

Generally most participants were happy with the services offered at their respective clinics, despite the long waiting periods (between three to six hours) when coming for their appointments, not receiving all their medications, lack of screening for chronic complications, and lack of referral to other health professionals.

# CHAPTER 5: DISCUSSION OF RESULTS

## 5.1 Introduction

In this chapter, the results of the study with regard to socio-demographic factors, nutritional status, medical history, glycemic control and barriers that may impact on treatment compliance is discussed, and interpreted in context of the current literature. Limitations encountered in the study and how these may have influenced the findings, are also discussed.

## 5.2 Socio-demographic factors

The socio-demographic factors for this study included age, gender, residential area, marital status, level of education, employment status, income level and the number of dependents that the participants have.

### 5.2.1 Age

According to Ling and Groop (2009:2720) during aging there is a decrease in glucokinase (the main enzyme in the utilization of glucose by the liver) activity leading to increased insulin resistance in the liver; a common problem in patients with diabetes. The IDF (2013b:13,30,34) states that T2DM is common between the ages of 40 to 59 years worldwide and predicted that the same age group will still comprise the largest number of people with diabetes, by the year 2035. In the US, the highest percentage of people (25.9%) with diabetes is in the age group 65 years and above (National Diabetes Statistics Report, 2014). In South Africa, the SADHS of 2003 (SAHDS; 2007: 200), however found the highest self-reported diabetes prevalence in the pre-retirement age groups (55-64 years). Similarly in Lesotho, the LRFS (2001:28) indicated that the prevalence of T2DM in Lesotho was high in the age group 50 to 54 years.

For patients living with diabetes in Lesotho, Adebayo (2010:Online) found that in 50 randomly selected patients with T2DM seen at the outpatient department of Scott Hospital, Morija, 60 km south of Maseru, in 2008, 72% were above 50 years of age. Makinga & Beke (2013:193) found that the mean age was 54.7 years among 192 randomly selected out-patients at one public hospital and two state-aided hospitals in Lesotho between November 2004 and July 2005. In the

current study the mean age of the total group of participants was 52.6 (± 8.3) years; while 67.7% (Table 4.1) of the participants were above 50 years of age.

Interestingly though, almost half (48%) of the males in the current study were younger than 50 years compared to only 28.3% of the females (Table 4.1); the reason remains unclear. Furthermore almost half of the participants had had diabetes for more than five years at the time of the study (Table 4.16). Taken together with the facts that: i) an estimated 81% of people with diabetes in Africa are undiagnosed (IDF, 2012: Online; Amod *et al*., 2012:S4); and ii) that older patients with diabetes are at increased risk of having diabetes-related complications, and to present with comorbid conditions such as hypertension (IDF, 2013b:102), thus making it more probable that it is the older rather than the younger patients with T2DM that would seek medical attention from the clinics, there is a need for screening for, and raising awareness about diabetes among the Basotho in Lesotho from a young age.

### 5.2.2 Gender distribution

Although the participants were randomly selected, as indicated in chapter 3, the final study group comprised of 79.8% females and 20.2% males (representing a female to male ratio of about 3:1). This concurs with the findings of Makinga & Beke (2013:193), who also found a ratio of 3:1 (75.5% females: 24.5% males) in Maseru; as well as the LRFS (2001:28) which reported that 1.7% of females and 1.1% of males are diagnosed with T2DM in Lesotho (a female to male ratio of 3:2). Adebayo (2010) found an even higher prevalence among Basotho females attending outpatient departments in Morija, with a female to male ratio of 6:1. The IDF (2012: Online) concluded that though T2DM is common among both females and males, females are more affected by the disease in Lesotho than males.

In South Africa the overall self-reported diabetes prevalence in the SAHDS 2003 (SAHDS, 2007:200) was 4 % for females and 3 % for males (a female to male ratio of 3:2). The Turkish Epidemiology Survey of Diabetes, Hypertension, Obesity and Endocrine Disease (TURDEP-II) conducted in 2010, reported similar findings, as Satman *et al.* (2013:Online) found that females had a higher diabetes prevalence than males. Among Korean adults Lee (2010: Online) found a

female to male ratio of 2:1 for chronic diseases, hence the effects of education, income, and marital status on morbidity were stronger in females than in males.

In contrast in developed countries such as China, more males were found to have diabetes than females (Tuomi *et al.*, 2013: Online). Other developed countries where the prevalence of T2DM have been reported to be higher among males than females include the USA (11.2% females and 13.6% males), which would corresponds with a female to male ratio of about 0.5); Switzerland (3.8% females and 9.1% males; with a female to male ratio of about 3:7), Japan (7.3% females and 15.3% males; with a female to male ratio of about 1:2), and Finland (7.4% females and 10.2% males; with a female to male ratio of about 2:3) (Tuomi *et al.* (2013:Online).

Although it is possible in the current study that the higher number of females with T2DM that visited the clinics, may have been be due to bias, for instance that females may be somehow more compliant to adhere to their appointments than males; the fact that the results are supported by many studies in developing countries, including Lesotho and South Africa, suggest increased susceptibility among females in developing countries. In the Turkey study Satman *et al.* (2013: Online) found a higher prevalence of obesity among middle-aged, and older females, than among males and suggested that females are more sedentary as a result of traditional and cultural attitudes. In the current study, the prevalence of overweight and obesity was indeed higher among the females, while males were more active than females (as will be discussed later).

### 5.2.3 Area of residence

Although urbanization is a recognized risk factor for T2DM (IDF, 2013b:23; Tcheugui & Kengne 2011: Online), previous studies in Lesotho found that the highest prevalence of T2DM was in the rural areas (IDF; 2012: Online; LRFS; 2001:28). In the current study however, most participants (91.1%) who attended Domiciliary and LDF clinics, resided in the urban Maseru area. This may have simply reflected that those living in the rural areas chose to visit other clinics around Maseru- for example the clinic at Scott Hospital, Morija, 60 km south of Maseru, studied by Adebayo (2010: Online) in 2008.

### 5.2.4 Marital status

Most participants (90.0%) in the current study were married (69.7% of the females and 84.0% of the males). Similar findings were reported by Makinga & Beke (2013:193) in 2004/2005 among patients with T2DM in Maseru (68.2% of the participants in their study were married). According to Lee (2010: Online) marital status is not necessarily a risk factor for chronic diseases, but marital functions (for example stress and parity) have been suggested to be significant factors that influence mental and physical health of married females. Hence, marital status has been shown to influence the risk for comorbid conditions associated with T2DM.

### 5.2.5 Education level

The LRFS (2001:27) found that diabetes prevalence was high among professionals; and the IDF (2013b:68) also reports that there is a positive relationship between education and the incidence of diabetes in low-and-middle income countries.

In the current study the level of education was classified into tertiary, college (include vocational schools), high school, primary school and none (no education). Only 0.8% of the participants in the current study had no education, compared to 4% in the study by Adebayo (2010: Online). About a third (29.8%) of the participants in the current study had only a primary school education, compared 62% in the study by Adebayo (2010:Online); and in the current study most of those with only a primary school education were females (33.3% of the females compared to 16% of the males). Makinga & Beke (2013:193) reported that 50% of the Basotho participants with T2DM from this area in 2005/2006 had not completed high school. In the current study half (49.2%) of participants (49.5% of the females and 48.0% of the males) had attended high school, although some may not have completed high school, though this was not assessed in the questionnaire.

These findings were supported by the SADHS (2003:200), which found that the prevalence of chronic diseases were highest among the general population of South Africans with a low socio-economic status (low education and low income). Among Korean adults, Lee (2010: Online) found no risk of comorbid conditions among participants at the highest education level, even if they had high BMIs. Similarly Aliaezadeh *et al.* (2014: Online) reported an association between

lower educational level and the incidence of diabetes in the Women's Health Study in Canada. The International Depression Studies done on wealthier and more educated groups reported increased risks of diabetes among women and populations of low socio-economic status or low education level (Hosseinpoor *et al.*, 2012:Online).

### 5.2.6 Employment status, income level and number of dependents

Both Hosseinpoor *et al*. (2012: Online) and Aliaezadeh *et al.* (2014: Online) found that that low-socioeconomic status (unemployment, low family income and large families) was associated with a higher risk of diabetes, especially in females, while Lee (2010: Online) demonstrated a lack of association between comorbid conditions and high income level, even in the presence of high BMIs, in Korean adults.

Employment status in the current study distinguished between participants with T2DM who were employed, unemployed, pensioners, homemakers or 'other' (all participants that chose this option indicated that they were self-employed). Of the participants in the current study, which was conducted in 2013, only 14.5% were unemployed; in contrast to the study by Makinga & Beke (2013:193) conducted in the area in 2004/2005 which found that 51.6% of participants with T2DM were unemployed (the majority of participants from 2004/2005 study were residing in rural areas compared to the majority in the current study residing in urban areas).

In the current study females (62.6%) had a maximum income level at the lower end of the income scale (between M300 and M1, 500), as opposed to males among whom 37.5% had a maximum income at the higher end of the income scale (between M4, 100 and M5, 300). Females in Lesotho do not necessarily earn less than males, but it depends on the type of work one does. In this case most females were cleaning in Government departments, while some were factory workers and street vendors (the lowest paying jobs). Most males were either self-employed (some were taxi owners) or employed in private organizations, hence they had a higher income. Most of the participants also had at least three dependents.

**Summary**

International literature identifies advancing age, marital status, low education level and low socio-economic status (poverty) as risk factors of T2DM. In the current study that enrolled 124 Basotho patients with T2DM, the participants were mostly married, with low-socio-economic status and only a primary or a high school education, but most participants were in the pre-retirement age group, with almost 50% of the men being younger than 50 years. Females seemed to be at the most disadvantage with the lowest income levels and the lowest education levels. In contrast to findings in developed countries, three times as many of these Basotho who attended two clinics in Maseru during the time frame of the study were female; supporting the findings from other studies in Lesotho, as well as studies from other developing countries including surrounding South Africa. Based on the socio-demography, there seems to be a need for screening for diabetes, and raising awareness about diabetes, among the Basotho in Lesotho from a young age.

## 5.3 Nutritional status

Nutritional status for the purpose of this study included anthropometric measurements, dietary intake and lifestyle factors.

### 5.3.1 Anthropometric measurements

Anthropometric measurements included BMI, WC, WHtR and BAI as these predict risks of cardiovascular disease, insulin resistance, and other lifestyle diseases in all ethnic groups. Individuals with diabetes have a 2-6 fold higher incidence of cardiovascular disease than individuals without diabetes (Haffner *et al*, 1998:229). Patients with diabetes also more often die of cardiovascular disease rather than short-term complications of diabetes, like hypoglycemia (Morris *et al*, 2001:S14). In addition, patients with diabetes who develop cardiovascular disease, have a worse prognosis for survival and worse quality of life compared to than those without diabetes who develops cardiovascular disease (Grundy *et al*, 1999: 1134). Timely identification of patients with T2DM who are at risk for cardiovascular disease could therefore help to prevent or delay cardiovascular incidents like myocardial infarction and stroke.

#### 5.3.1.1 BMI

BMI is a descriptive index of body size that encompasses both the lean and the obese (Nyamdorj, 2009:70). BMI levels > 25 kg/m² are associated with increased risk of morbidity and mortality; while BMI levels ≥30 kg/m², indicating obesity, is associated with progressively higher risk (Nyamdorj, 2009:70). Increased BMI among patients with diabetes is associated with increased insulin resistance, hypertension, dyslipidaemia and atherosclerosis (Goedecke *et al.*, 2005: Online), as well as cardiovascular disease (Leitzmann *et al*., 2011: Online; Browning *et al*., 2010:266).

In the current study, based on BMI, more than half (57.3%) of the participants were obese, with a further 31.5% being overweight; thus 89.2% of participants had an above normal BMI, putting them at risk of cardiovascular complications of diabetes, as well as other chronic diseases. This was more evident among the females: 61.6% of the females were classified as obese and 31.3% as overweight; thus 92.9% of the females had a BMI above the normal cut-off value. Among the males, 40% of males were classified as obese and 32% as overweight; thus 72% of the males had a BMI above the normal cut-off value. These findings are similar to those of two previous studies among patients with T2DM in Lesotho. In 2005/2006, Makinga & Beke (2013:194) found that the overall prevalence of obesity among the participants with T2DM in Maseru was 67.7%, and also confirmed that obesity was more prevalent among the females (73.1%) than the males (51.1%).

Similarly, Adebayo (2010: Online) found that among patients with T2DM at Scott hospital outside Maseru in 2008, 78% of the participants were obese; with the prevalence among females (81%) again higher than among males (57%); also the 14% of the participants who were morbidly obese (BMI ≥40kg/$m^2$) were all females. The Prevalence of obesity in the current study as well as in the other two Maseru studies, are markedly higher than that reported in the LRFS (2001:35), which indicated that 47.3% of females and 18.8% of males with diabetes were obese. This may indicate that the obesity situation is escalating in Lesotho, similar to other countries in the world where it has been called a growing epidemic (James, 2008:S120).

The gender distribution of obesity in this and other Lesotho studies are similar to that found in South Africa, as well as other developing countries. National statistics from South African DHS (SADHS, 2003:281) indicated that among the general population of SA, more females (56.6%) than males (29.2%) were overweight and obese. Similar findings were reported in many SA studies among the black population (van den Berg *et al*., 2008: Online; Goedecke, 2005: Online). Goedecke (2005:Online) also reported that among South African females, black females (58.5%) were found to have the highest prevalence of either overweight and obesity, followed by females of mixed ancestry (52%), then white females (49.2%), and lastly Indian females (48.9%). Similarly, in Cameroon, situated in SSA, 50% of females and 25% of males living in urban areas were either overweight or obese.

#### 5.3.1.2 WC

WC measurement is considered a good indicator in adults of intra-abdominal (central) fat, which accumulates around the organs in the peritoneal cavity (Ashwell and Browning, 2011:70; Browning *et al.,* 2010:248; Nyamdorj, 2010:70). In China, for example, a positive relationship between central obesity and incidence of diabetes was shown (IDF, 2013b:68). The Epic-Potsdam study done in Germany by Feller *et al.* (2010: Online), found a high prevalence of obesity with increased WC (39.5%) of >88cm in females and >102cm in males (Feller *et al.*, 2010: Online). The IDF however recommends the use of ethnic-specific cut-off points for central obesity, which in Sub-Sahara Africa is WC of ≥ 94 cm in males and ≥ 80 cm in females (Amod *et al.*, 2012:S58).

As a measure of intra-abdominal obesity, WC is an independent risk factor for cardiovascular disease, insulin resistance and other endocrine abnormalities, and assessment of WC as a measure of central more prognostic for cardiovascular disease, diabetes and other endocrine abnormalities, than BMI alone (Ashwell & Browning, 2011:70; Browning *et al.,* 2010:248; Feller *et al*., 2010:Online).

Almost all participants in the current study 91.1%; (90.9% females and 92.0% males) had WC above the cut-offs, which indicated that almost all of these Basotho patients with T2DM had

substantially increased risk for insulin resistance, hypertension, dyslipidaemia, atherosclerosis and other cardiovascular diseases.

#### 5.3.1.3 WHtR

WHtR has been shown to be very useful among different ethnic, age and sex groups as an index of central obesity and predictor of risk for chronic diseases (Ashwell, 2011: Online; Browning *et al.*, 2010:248). WHtR has been recommended as more sensitive than BMI as an early warning sign of metabolic health risks (Ashwell & Hsieh; 2005:303). In Mangaung, South Africa, not far from Maseru, Lategan *et al.* (2014: Online) found that WHtR was a better predictor of hypertension among a black urban population with a high prevalence of hypertension and T2DM. An advantage of using WHtR is that even in different ethnic groups, the same cut-off point of 0.5 can be used for males and females (Ashwell & Hsieh, 2005:303).

In the current study 96.8% of participants (98.0% females and 92.0% males) had increased WHtR and thus increased risk for insulin resistance, hypertension, dyslipidaemia, atherosclerosis and other cardiovascular diseases. These findings are also consistent with that of the 2005/2006 study in Maseru by Makinga & Beke (2013:194), which showed that 95.3% of participants had increased WHtR.

#### 5.3.1.4 BAI

BAI is a newly developed method to estimate adiposity of individuals, and offers a direct estimate of percentage body fat (% body fat) (Bergman *et al.*, 2011:1084). In the current study based on BAI, the majority of participants were obese (>38% of body fat in females, and >25% in males), and males (84%) had the highest prevalence of obesity than females (60.8%). These findings correspond with that of Lategan *et al.* (2014:Online) among a black urban population with high prevalence of hypertension and T2DM, Mangaung, South Africa which found that 76.3% of participants were classified as either overweight or obese.

**Summary**

The findings indicated that majority of patients with T2DM in this study are at increased risk of developing complications, particularly cardiovascular diseases and associated risk factors such as hypertension, dyslipidaemia, and atherosclerosis as a result of a high prevalence of obesity.

Comparing the different anthropometry indices, (Figure 4.1), more participants were at risk for complications were indicated by WC and WHtR than BMI and BAI. These results indicated that WC and WHtR, instead of BMI and BAI, could be useful tools to screen for risk of cardiovascular complications among this study population. This also concur with the findings by Ashwell (2011: Online), which indicated that measures of abdominal obesity are better predictors of cardiovascular disease and T2DM risks than BMI.

### 5.3.2 Usual dietary intake

Usual dietary intake in this study referred to (a) the usual daily food intakes with relation to number of servings from each food group; energy and macronutrients intakes; as well as (b) the frequency (daily, weekly, monthly) with which specific types of foods from the different food groups were consumed.

#### 5.3.2.1 Energy intake

Most patients with diabetes find it very difficult to determine what to eat, when and how much, and there is no "one-size-fits-all" eating pattern for individuals with diabetes (Evert et al., 2013: Online). The optimal proportions of the macronutrients (carbohydrates, proteins and fats) in the diet are not well clarified (Ajala *et al.*, 2013: Online). When guidance is required in meal planning, the dietary reference intakes (DRIs) for healthy eating, which recommend that adults should consume 45% to 60% of total energy from carbohydrates, 20% to 35% from fat, and 15% to 20% from proteins can be very is useful (Table 3.7, chapter 3).

In the current study the mean total energy intake for both females (5360.2 ± 1235.8kJ (SD)) and males (6893.0± 1541.7kJ (SD)) were quite low in the light of the levels of overweight and obesity in this group). This may indicate underreporting which is one of the disadvantages of dietary recall. According to Lee and Nieman (2010:83) respondents tend to alter information

about what they ate due to poor memory, embarrassment, and/or intent to please or impress the interviewer. Respondents also tend to underreport binge eating, consumption of alcoholic beverages, and other foods perceived as unhealthy (Lee & Nieman, 2010:83). Similar relatively low energy intakes in the presence of high levels of overweight and obesity was also reported by van den Berg, *et al* (2012:Online) among black nursing students in the Eastern Cape, South Africa.

#### 5.3.2.2 Intakes of carbohydrate and food from the bread, grain and cereal group

The ADA (2013a:S23) recommendation of at least 130g per day of carbohydrates intake is based on providing adequate glucose that is required for the central nervous system without reliance on glucose production from proteins or fats. The ADA (2013a:S23) and Ajala *et al*. (2013:S23) indicated that long term metabolic effects of carbohydrates intake lower than 130g per day are not clearly defined, but may eliminate most foods in the diet that are important sources of dietary fiber, vitamins and minerals, and are not palatable. Evert *et al.* (2013: Online) stated that there is insufficient evidence to support one specific amount of carbohydrates intake for people with diabetes, hence a total energy of 45 to 60% as carbohydrates is recommended.

In the current study the percentage total energy intake from carbohydrates of the females was 63.4%, which is within the DRIs recommendation, while for the males was 65.7% and slightly above the DRIs recommendations. Despite the mean intake of carbohydrates being 206.8g ± 52.5 (SD) for females and 257.1g ± 66.9 (SD) for males, the minimum carbohydrates intake was 125g for females and 127g for males, indicating that some participants may be risk for developing hypoglycemia due to inadequate glucose intake to sustain the central nervous system without reliance on glucose production from proteins or fats. On the other hand, a maximum carbohydrates intake of 430g was reported among the males. These findings indicate lack of knowledge and understanding among the participants regarding food intakes or rather healthy eating in diabetes.

Solomon *et al.* (2009:1225) indicated that use of low-GI and glycemic load carbohydrates may be to be beneficial in regulating uptake of nutrients; and capable of prolonging satiety, therefore leading to a decrease in whole-body fat mass. Refined carbohydrates are deficient in fiber,

vitamins, magnesium and other minerals, lignans, phytoestrogens, and phytic acid (Sun *et al.*, 2010: Online). According to Sun *et al.* (2010:Online) prospective cohort studies in the US among females and males showed that consumption of white rice (high GI) was associated with higher risk of T2DM, compared to intakes of whole grains. The type of carbohydrates that the participants in the current study consumed daily (stiff porridge, brown bread, mabele porridge and rice) were not low-GI, which may contribute to poor glycemic control among participants (discussed later).

A diet high in legumes (beans, lentils, peanuts, peas and soybeans) is postulated to have a beneficial effect for the prevention of T2DM and dyslipidemia, as legumes are good sources of soluble fiber and have a low-GI (Maghsoudi & Azadbakht, 2012). Legumes also contain polyphenols such as isoflavones and lignans, which have an antioxidant, effect (Maghsoudi & Azadbakht, 2012). Nine out of ten (91.9%) participants in the current study used dried beans every week (although only 1.6% reported consuming dried beans daily), and very few participants used texturized proteins.

Patients with T2DM should consume the amount of fiber and whole grains recommended for the general populations, which is to increase the intake of soluble and insoluble fiber to 25-50g per day (Amod *et al.*, 2012:S16); about 25g/day for adult females and 38g/day for adult males (Evert *et al.*, 2013:Online). The benefits of fiber intake reported among patients with T2DM include the viscous and/or gel-forming properties of soluble fibre from fruit and vegetables that influence the GI and blood lipids, as well as the metabolic effects of short-chain fatty acids derived from colonic fermentation of nondigested fibre by the gut microorganisms (Weickert, 2012:Online). As the diet consumed by the participants in the current study was low in sources of fiber, including high fiber breakfast cereals, high grain bread and legumes, it could indicate less than optimum fiber intakes, particularly in light of the low intakes of fruit and vegetables (discussed later).

#### 5.3.2.3 Intake of protein and food from the meat and meat substitute group

According to Evert *et al.* (2013: Online), there is inconclusive evidence regarding an ideal amount of protein for optimizing glycemic control or improving cardiovascular risk factors to

patients with T2DM without kidney disease, and it is recommended that the goals be individualized. For patients with signs of renal damage (micro-albuminuria), protein intake should be limited to 0.8-1.0g per body weight per day (ADA, 2013a:S35; Beasley & Wylie-Rosett, 2013: Online), and to 0.8g per body weight per day in the later stage of renal damage (ADA, 2013a:S35; Beasley & Wylie-Rosett, 2013: Online). It is not recommended however to reduce the amount of protein intake to below 0.8g/kg body weight per day, as it does not alter glycemic control, cardiovascular risk factors or the course of glomerular filtration rate decline (Evert *et al.*, 2013: Online). Hence, the recommendation for protein contribution to total energy intakes is 15-20% for patients with T2DM without renal problems (ADA, 2013a:S22; Amod *et al.*, 2012:S16; Franz, 2012:686), and this has been shown to improve glycemic response, reduce lipids levels and hormones, to have no long term effect on insulin requirements (Franz, 2012:686).

The findings of the current study demonstrated that the overall intake of protein was 18.6% of total energy for females, and 19.3% of total energy for males, which falls within the recommendations if it is assumed that none of these patients suffer from renal damage.

The mean intake of proteins was 58.5g ± 15.6 and 78.1g ± 17.4 for females and males, respectively. The protein intake expressed in grams per kg body weight ranged from 0.29g/kg body weight to 1.79g/kg body weight for females, and 0.50g/kg body weight to 1.66g/kg body weight for males. Therefore some of the participants (particularly females) consumed proteins far below the recommended usual daily intake of 0.8-1.0g/kg body weight. Maximum intakes ranged from 78.1g in males to 99g in females. High protein diets (and high-GI carbohydrates) may increase inflammation, and eventually worsen the whole body insulin resistance in patients with diabetes (Weickert, 2012: Online).

The high biological value protein sources that was mostly consumed by participants was chicken (95.2%), followed by pork (23.4%), while less than 20% of participants consumed beef and lamb. In view of the high prices of beef and lamb in Lesotho, this probably reflects the participants' low socio-economic status and not necessarily health concerns. According to the findings by Ajala *et al.* (2013: Online), excessive intake of red meat may increase concentrations

of inflammatory mediators and gamma-glutamyl transferase. Red meat contains iron, which is a strong prooxidant that catalyzes several cellular reactions in the production of reactive oxygen species, thus producing oxidants that can cause damage to tissues, particularly also to the pancreatic beta cells (Ajala *et al.*, 2013: Online). Therefore excessive intake of red meat is discouraged in patients with T2DM.

Small percentage of participants (20.2%) consumed organ meats (offals) every week, and more than half of participants (57.3%) used processed meats (Russians, polony and viennas). Ajala *et al.* (2013: Online) indicated that frequent use of organ meats is discouraged in patients with T2DM as they contain high levels of saturated fat. Processed meats also contain saturated fats, sodium and nitrites (chemicals used in meat preservation). Nitrites and nitrates are converted to nitrosamines, which are shown to be toxic to pancreatic beta cells, and high concentrations of nitrites in the blood of adults have been linked with endothelial dysfunction and impaired insulin response (Ajala *et al.*, 2013:Online).

Most participants (91.9%) reported that they consume tinned fish (pilchards) on a weekly basis, particularly pilchard, while very few used white fish (hake fillets). Patients with T2DM are advised to eat fish at least twice per week (Evert *et al.*, 2013: Online; Steyn & Temple, 2012:502) in order to obtain the recommended amounts of omega-3 PUFAs (Amos *et al.*, 2012:S16). Omega-3 fatty acids from fatty fish (salmon, pilchards, tuna and sardines) (EPA and DHA) and linolenic acid (ALA), are shown to have a beneficial effects on lipoproteins, to prevent heart diseases, and are associated with positive health outcomes (Evert *et al.*, 2013: Online).

The majority of participants (67.7%) used eggs every week, while 13.7% used eggs on a daily basis, probably because eggs are a relatively cheap source of proteins the participants could afford to buy. The ADA recommends that cholesterol should be restricted to < 300mg of per day to protect against cardiovascular disease. Patients with diabetes are therefore advised to limit eggs which are containing high levels of cholesterol in the yolks, to 3 eggs per week (Steyn & Temple, 2012:502),

#### 5.3.2.4 Intake of fat and fat sources

Dietary fat refer to a mixture of saturated (SFA), trans-saturated (TFA), mononunsaturated (MUFA) and polyunsaturated fatty acids (PUFA), PUFA are further classified as omega-3 and omega-6 polyunsaturated fatty acids (Evert *et al.*, 2013: Online; Wickert, 2012: Online). Recent evidence show that the type of fatty acid consumed is more important in controlling metabolic goals and influencing the risk of cardiovascular disease than total fat in the diet (Evert *et al.*, 2013: Online). Therefore, the type of fat should be prioritized when individualizing goals, and all patients with diabetes should be encouraged to take fat moderately, in consistent with their goals to lose or maintain weight (Evert *et al.*, 2013: Online).

There is no defined adequate intake or recommended daily allowance for total fat intake for T2DM, but the IOM define an acceptable macronutrient distribution range (AMDR) for total fat of 20-35% of total energy with no tolerable upper intake level defined (Evert *et al.*, 2013:Online). The recommendations include: the total fat intake should be restricted to < 35% of total energy per day, while polyunsaturated fatty acids intake should contribute < 10% of the total energy intake per day, saturated fat < 7% of the total energy intake per day, and trans-fats < 1% of total energy intake per day (ADA,2013a:S22; Amod *et al.*, 2012:S16).

In the current study the percentage of total energy intake from fats for females and males were 16.0% and 17.3% respectively, which are below recommendations. The mean intake of fats/oils was 22.6g ± 7.9 and 31.3g ± 10.7 for females and males, respectively. The minimum intake was as low as 5g in females and 10.0g in males; while the maximum intake was 43g in females and 50.0g in males. These intakes are much lower than the mean intakes reported in the US (ADA, 2014: 136 and in light of the high prevalence of obesity in the study group, raise suspicions of under-reporting. However, many South African studies have demonstrated average intakes of fats in the country as 20-30% of total energy, with some pockets of the population consuming less than 20% of total energy as fats (Smuts & Wolmarans, 2013:Online). Interestingly the SADHS (2003:Online) showed the mean fat intakes are the lowest in the Northern Cape, Western Cape and Free State (situated closer to Lesotho, and hence share several characteristics).

With regard to the types of fatty acids in the diet, the majority of participants (90.3%) reported daily use of sunflower (a source of pro-inflammatory omega-6 PUFAs). A small percentage of the participants consumed organ meats (offals) on a weekly basis, and more than half of the participants (57.3%) used processed meats (Russians, polony and viennas) on a weekly basis, which are source of saturated fatty acids, nitrites and nitrates. The intake of fast foods was minimal, and reported buying chips and fat cakes, which are high in trans-fats.

While the majority of participants used eggs least at every week, cheese consumption was almost negligible. With regard to legumes, nine out of ten (91.9%) participants used dried beans (Tables 4.9), and 38.7% used peanut butter weekly, but very few used texturized proteins and soya milk.

**5.3.2.5 Intake of milk and dairy products**

High intake of milk and dairy products is associated with an overall healthy diet and lifestyle, which may track throughout the lifecycle and ultimately lowers the risk of T2DM. However, consumption of dairy products has been displaced by sugar-sweetened beverages, which are associated with obesity and T2DM (Malik *et al.*, 2011: Online).

The components found in dairy products (high-quality proteins, vitamin A, vitamin D, vitamin $B_{12}$, menaquinones-vitamin $K_2$, riboflavin, calcium, magnesium, potassium and lactose) may be responsible for the beneficial effects (O'Connor *et al.*, 2013: Online; Malik *et al.*, 2011: Online). For instance, milk proteins such as whey may have insulinotropic (de Koning *et al.*, 2011: Online) properties with a relatively low glycemic load, which may improve glucose tolerance (Malik *et al.*, 2011: Online). While other dairy product components, including medium-chain fatty acids, calcium, and magnesium, may reduce insulin resistance or inflammation and lowers oxidative stress (Malik *et al.*, 2011: Online; de Koning *et al.*, 2011: Online).

Only 6.5% of the participants in the current study met the recommendation of 2-3 servings of dairy per day; and the low daily intakes were also confirmed by the FFQ. These findings concur with two other studies in Lesotho. Seheri (2012:72) similarly found that 90% of the 16 year-old Basotho adolescent in Maseru, did not consume the required two to three servings of dairy per

day; while Lehlasoa (2011:75) found that none of older Basotho women (aged 28 to 86 years) studied, met this requirement. In South Africa van den Berg *et al* (2012: Online) similarly found that 92.6% of predominantly black nursing students in the Eastern Cape did not meet this requirement.

A reason for low dairy intake in this population may be due to lactose intolerance which is common among Black South Africans. The ability to digest lactose depends on the presence of the enzyme lactase-phlorizin hydrolase which is reduced by up to 90-95% in individuals with "lactase non-persistence" (the declining of lactase production after infancy) (Byers & Savaian, 2005:569). Research has however shown that individuals who are lactose intolerant, including African-Americans, can consume at least one cup (240ml) of milk without experiencing symptoms, and that tolerance can be improved by gradually introducing milk into the diet, by consuming the milk with a meal, choosing fermented sources of milk like yoghurt, sour milk or hard cheeses (US National Medical Association, 2009:3).

The intake of fermented dairy products also adds probiotics to the diet which positively influences the gut microflora. Studies show that this may alter the systemic effect of endotoxin which is derived from the Gram negative bacteria in the gut and act as an important mediator of inflammation. Lower levels of endotoxin lead to reduction in inflammatory cytokines and inflammatory response. A diet that normalizes the gut microflora and metabolic functions is therefore beneficial in the management of most chronic diseases, particularly T2DM (Alokail *et al.*, 2013: Online).

In the current study sour milk was the form of milk which was most consumed, indicating a preference for the fermented form of milk. However, consumption was still only on a weekly basis, indicating that other factors like cost, or lack of cold storage facilities (not assessed in this study) could also be other reasons for the low dairy intakes.

#### 5.3.2.6 Fruits and vegetable intake

Fruits and vegetables are sources of vitamins, minerals, fibre and many different phytochemicals which protects against cardiovascular disease, T2DM and cancer. Various protective mechanisms have been identified, including: acting as anti-oxidants; enhancing the performance of the liver detoxification enzyme systems, being anti-microbial, anti-proliferative, anti-mutagenic, and anti-carcinogenic and regulating apoptosis (Bahadoran *et al.* (2013: Online).

Polyphenols has hypoglycemic effects, which involve reduction of intestinal absorption of dietary carbohydrate, modulation of the enzymes involved in glucose metabolism, improvement of ß-cell function and insulin action, stimulation of insulin secretion, and the antioxidative and anti-inflammatory properties of these components (Bahadoran *et al.*, 2013:Online; Van Dokkum *et al*, 2008: 133).

Green leafy vegetables also contain α-linolenic acid (which is an omega-3 PUFAs), which are essential in determining the fatty acid composition the fatty acid composition of the phospholipid bilayer, and is related to insulin sensitivity within skeletal muscle (Carter *et al.*, 2010:Online).

Based on these protective effects patients with diabetes are advised to consume at least 2-4 fruits and 3-5 vegetable servings per day (ADA and ADA, 2005: Online). Most participants in the current study did not however meet these recommendations for fruits (65.3%) or vegetables (78.2%). According to the non-quantified FFQ, 62.1% of participants did report that they consume fruits daily, though they did not meet the daily recommendations of 2-4 servings per day (Table 4.7). The study was done during the season (October to March) when plenty of fruits (peaches, apricots, apples, pears, and grapes) are freely available in most households and their cost is minimal. These low intakes of fruits and vegetables may increase risk the participants for complications of T2DM.

#### 5.3.2.7 Sugar intake

The SEMDSA guidelines recommend that sucrose intake be limited to 10% of total energy per day, and to limit intake of sugar alcohols (isomalt, lactitol, mannitol, maltitol, sorbitol, xylitol) to <10g per day (Amod *et al.*, (2012:S16). Almost half of the participants used sugar daily to

sweeten tea (the majority of the participants reported using two teaspoons of sugar in a cup of tea), and preferred brown sugar to white sugar. Honey, syrup, jam, sweets, chocolates, desserts and biscuits were rarely used.

#### 5.3.2.8 Sodium Intake

The main source of sodium in the diet is the salt added in food during cooking or at the table, and also contained in packaged and processed foods (Amod *et al.*, 2012:S16). There are limited studies published on the benefits of sodium reduction in patients with diabetes, and randomized controlled trials indicated that decreasing sodium intake reduces blood pressure in patients with diabetes (Evert *et al.*, 2013:Online). Following the DASH diet principles and reducing sodium intake to about 2300mg led to improvements in blood pressure and other measures on cardiovascular risk factors (Evert *et al.*, 2013:Online). In the current study all participants' added salt to flavor food on a daily basis. Other salty flavourants that were used more on a weekly basis include spices, aromat, and stock cubes.

**Summary**

The majority of participants in the current study were either overweight or obese. The high prevalence of obesity among Basotho may be associated with the following factors: genetic makeup, intrauterine and early life influences, parity, physical inactivity, poor dietary habits, low level of education, socio-cultural factors and stress (Makinga & Beke (2013:104). Furthermore their dietary intake did not meet the recommendations for milk and dairy products and fruits and vegetables, and were low in sources of key nutrients. These findings indicate that the majority of the patients with T2DM in this study may be at increased risk of developing complications such as cardiovascular diseases, and associated risk factors such as hypertension (most of them were hypertensive already and not controlled as discussed later), dyslipidaemia, and atherosclerosis. Therefore there is a need for an individualized nutritional management among patients with T2DM, done well trained dieticians and/or nutritionists.

The study shows that WHtR may be a useful tool to screen for risk of complications in this population. A very practical and easy to understand way to raise patients awareness of their own risk, may be to cut them each a piece of rope equal to the heights, and educating them that this

rope should be able to fit around their waists at least twice to protect against cardiovascular complications of diabetes.

The value of protective food choices already portrayed by these Basotho patients with T2DM should be discussed with them and encouraged to increase the daily intakes, including consumption of chicken and tinned pilchards rather than beef and lamb, and the inclusion of sour milk, fresh fruits and green leafy vegetables and legumes. As their staple (for cultural and financial reasons) is cooked maize porridge with a high GI and low fiber content, they can be educated about cooling the porridge down and reheating it before consumption to decrease the GI; and choosing whole grain bread. Most of all a dietician/nutritionist could assist them individually and in groups, to plan more adequate, yet affordable diets using their limited resources.

### 5.3.3 Lifestyle factors

The information of the physical activity, alcohol intake and smoking was also obtained during the structured one-on-one interviews. Physical activities ranged from household duties, gardening, taking the stairs, and walking briskly, to jogging, exercising and participating in sports.

#### 5.3.3.1 Physical activity

Physical activity referred to all movements that the patients incur in their everyday lives, which included work, recreation, exercise and sporting activities. The CDC (2011: Online) indicated that physical activity is associated with important health outcomes, including reducing the risk for cardiovascular disease and T2DM.

Sedentary behavior is increasing worldwide risk factor and is a well-recognized risk factor for chronic diseases including cardiovascular (WHO, 2004:Online). In South Africa, the SADHS showed that the prevalence of inactivity among the general adult population in urban areas was 66% in females and 49% in males; and the prevalence of inactivity in adults females and males in rural areas, was 59% and 46% respectively. The SADHS further demonstrated that only 14% of females and 24% of males were sufficiently active, and levels of activity seemed to decline

with increasing age. In Lesotho the LRFS (2001:40), reported different findings, since it showed that among participants who performed exercises, 50.7% reported doing heavy activities, 33.4% did moderate activities, 13.6% did light activities.

For patients with T2DM a physical activity may contribute to increased cardio-respiratory fitness, improved glycemic control, decreased insulin resistance, improved blood pressure, maintenance of weight loss, reduced abdominal and overall fat percentage, improved well-being and decreased stress and anxiety (ADA, 2013a:S24). ADA, (2013a:S24) recommends that adults with T2DM should be advised to perform at least 150 minutes per week of moderate-intensity aerobic physical activity (50-70% of maximum heart rate), for at least three days per week with no more than two consecutive days without exercise (ADA, 2013a:S24). In the absence of contraindications, adults with T2DM should be encouraged to perform resistance training at least twice per week (ADA, 2013a:S24).

Makinga and Beke (2013:193) reported that 58.3% of patients with T2DM in Maseru reported that they practiced physical activities recommended for patients with diabetes, such as brisk walking. In the current study the IPAQ indicated that 82% of participants were moderately active; mostly through household tasks like gardening. More of the men than of the women were also classified as highly active. These results raise the possibility to encourage patients, particularly the females, to keep vegetable gardens which they should tend themselves. While this may help to raise physical activity level, it may also increase their vegetable intakes. A dietician/nutritionist may assist patients in planning and starting vegetables gardens.

#### 5.3.3.2 Alcohol intake

Alcohol consumption referred to the intake of any beverage (homebrew, beer, cider, wine, and spirits) that contains alcohol. In South Africa, the SADHS (2003:267) reported that higher rates of drinking in the urban areas as compared to the rural areas, and 10% of females and 30% of males reported that they had drunk alcohol at some point in time. Similar trends are reported for other developing countries. Echouffo-Tcheugui & Kengne (2011: Online) also reported that alcohol consumption is high in Cameroon, with the percentage of life-time abstainers estimated at only 18% in females and 11% in males, in 2008. In Lesotho, according to LRFS (2001:39),

the prevalence of alcohol intake among the general population was 38.8% in rural areas, and 33.7% in urban areas.

Adults with T2DM who choose to take alcohol are advised to limit their intake to one serving or less per day for females and two servings or less per day for males (Evert et al., 2013:Online; Amod et al., 2012:S16). At this moderate level of intake alcohol have been shown to somewhat improve glycemic control, cardiovascular risk and mortality in patients with T2DM (Evert *et al.*, 2013: Online). Moderate alcohol consumption with food also does not contribute to acute hyperglycemia or hypoglycemia (Amod *et al.*, 2012:S16), while excessive amounts of alcohol (>3 drinks per day) consumed on a consistent basis may contribute to hyperglycemia (Evert *et al.*, 2013: Online).

The LRFS (2001:23) found that 56% of patients with diabetes in Lesotho did not drink alcohol. Among patients with T2DM in Lesotho, Makinga and Beke (2013:193) reported that about 90% of patients with diabetes in a Maseru survey did not drink alcohol. Similarly, in the current study almost 80% of participants reported being non-drinkers. However, 52% of the males in the current study reported consumed alcohol above the recommended limit, mostly as commercial beer. Most males reported taking between 4 and 6 units of alcohol per session. Awareness of the risks associated with excessive alcohol intake with regard to blood glucose control and weight management need to be raised among these Basotho patients, particularly among the men.

Furthermore, patients with T2DM should be advised to abstain from alcohol if there is evidence of advanced neuropathy, or severe hypertriglyceridimia (Evert et al., 2013: Online). Therefore regular screening for these complications in these patients is important.

#### 5.3.3.3 Smoking

Smoking refers to use of tobacco products including cigarettes (commercial or home-made) and/or pipe, and/or snuff. Cigarette smoking is linked to increased insulin resistance. The Surgeon General's report of 2014 indicated that smoking can cause T2DM, and the risk of developing diabetes is 30-40% higher among active smokers than non-smokers (CDC, 2014:

Online). In addition the use of smokeless tobacco (snuff, taken in an unburnt form through chewing or sniffing) contains several carcinogens, and has been associated with oral cancer, hypertension, heart disease, T2DM, and other diseases (Lee & Hamling, 2009:Online).

The LRFS (2001:38) showed that the prevalence of smoking in the general population, in rural areas was higher (41.5%) than in the urban areas (32.7%), and was higher among males (47.9%) than females (34.2%). In South Africa, the SADHS reported that 10% of females and 35% of males smoked cigarettes either daily or occasionally, and also found a higher prevalence in urban areas (where 13% of females and 39% of males smoked), compared to rural areas where 6% of females and 28% of males smoked. In Cameroon, another developing country, Echouffo-Tcheugui & Kengne (2011: Online) quoted unpublished data showed a smoking prevalence of 6.4% (1.0% females and 8.2% males).

Smoking is specifically discouraged for patients with diabetes (Glass *et al*., 2009:40) as several studies demonstrated that smoking increase the risk of cardiovascular complications including hypertension in patients with diabetes (Raz, 2009:S149). In a study among patients with T2DM in Lesotho in 2005/2006, Makinga & Beke (2013:193) found that 14.6% of patients were using tobacco. In the current study 10% of participants were current tobacco users, and the most commonly used form of tobacco was smokeless tobacco in the form of snuff (which used by 10.0% of females). Interestingly the SADHS (2003:263) found that snuff was mostly used by black females living in the Free State (which mostly constitutes the South African Basotho [Sesotho] people), who are older and have limited education, which correspond to the profile of snuff users of the current study. Men in the current study, who used tobacco, mostly smoked cigarettes.

**Summary**

The findings of the study demonstrated that most of these Basotho patients with T2DM were trying to follow the recommended lifestyle habits, as the prevalence of smoking and alcohol use was lower among them than what was reported for the general population of Lesotho and surrounding South Africa. According to the IPAQ very few of the participants were also inactive. This is beneficial as smoking cessation lowers the risk of cardiovascular diseases,

while moderate activity as wells as prudent alcohol intake improves glycemic control and insulin sensitivity. These practices need to be discussed and encouraged among the patients. Special attention should also be given to the sub-groups of patients who were inactive and those who used alcohol excessively.

### 5.4 Medical history

The information about the year of diagnosis, biochemical values (fasting blood sugar, blood pressure, total cholesterol and triglycerides), comorbid conditions (obesity, hypertension and dyslipidemia), and glucose lowering medications was obtained from the participants' medical booklets (bukanas) or during the structured interview with the researcher.

#### 5.4.1 Duration of T2DM

Table 4.16 illustrated that majority of participants (females 49.5% and males 68.0%) had been diagnosed with T2DM for a period of five or less years. Controlling blood glucose levels and other metabolic risk factors from early on in the disease can significantly prevent or delay the onset of complications (ADA, 2013a:S18).

#### 5.4.2 Medications

The LRFS (2001:29) established that among patients with diabetes in Lesotho (diagnosed by a clinician), only 57% were on medications, while 43% were not on any medications. Moreover, 81.8% were on oral agents, 13.5% on insulin, 4.5% on traditional medicines, and nobody was on lifestyle and diet only. In the current study (67.7%) of participants (68.7% females and 64.0% males) were on oral agents only, 19.4% were on insulin, while very few (4.0% for both females and males) were on lifestyle and diet treatment only. Gavin *et al*. (2010:8) indicated that adherence to medication among patients with diabetes ranges from 36% to 87% with oral agents, and from 54% to 81% with insulin-only regimens.

#### 5.4.3 Comorbid conditions

The LRFS (2001:29) indicated that 70% of patients with diabetes in Lesotho had hypertension (76.7% females and 50% males), and 11.1% had hypercholesterolemia.

In the current study 94.4% (92.9% of females and all males) were hypertensive. Hypertension is a common comorbid of T2DM, and the prevalence depends on type of diabetes, age, obesity, and ethnicity (ADA, 2013a:S18; Franz, 2012:704).

Patients with T2DM and hypertension should be treated to achieve a systolic blood pressure of less than 140mmHg and diastolic blood pressure of less than 80mmHg (ADA, 2013a:S18). To this end it is recommended that patients should be advised on lifestyle changes (weight loss, if overweight, DASH-style dietary pattern, sodium reduction to below 1500mg/day, increased potassium intake, moderate alcohol consumption, increased physical activity, increasing consumption of fruits and vegetables and use low-fat dairy products) to reduce blood pressure. Those with a confirmed blood pressure above 140/80mmHg should be treated with both lifestyle and pharmacological therapy (ACE inhibitors or an angiotensin receptor blocker) in order to achieve blood pressure goals (ADA, 2013a:S18). Glycemic and lipid control may be positively affected when using this strategy, although the effects on cardiovascular events have not yet been established (ADA, 2013a:S18).

Although the participants in the current study had their blood pressure be measured at every routine visit to the health care facility, as is recommended (ADA, 2013a:S18; Franz, 2012:704), and despite receiving anti-hypertension drugs from the clinics, most participants' blood pressures remained uncontrolled.

Dyslipidemia is a common comorbid of T2DM (ADA, 2013a:S18), and it is suggested that the full fasting lipid profile (total cholesterol, triglycerides, HDL cholesterol and LDL cholesterol) should be tested during the first encounter with the patient with T2DM (Amod *et al.*, 2012:S57). If the results of the fasting lipid profile are satisfactory, a lipid profile should be done once every year (ADA, 2013a:S18; Amod *et al.*, 2012:S57). If the results are unsatisfactory, the test should be repeated in three months, during which the patient has been on a lipid lowering diet, weight reduction programme. If still unsatisfactory lipid lowering drugs are instituted (Amod *et al.*, 2012:S57). In the current study however, no evidence of results of screening for dyslipidaemia (triglycerides and total cholesterol) were available in the patient files (except for one patient with a total cholesterol level of 6.9 mmol/l).

Lifestyle intervention for dyslipidaemia should focus on reducing saturated fat, trans-unsaturated fat and cholesterol intake, increase of omega-3 fatty acids, soluble fiber (for an example oats, legumes, citrus), and plant stanols/sterols, smoking cessation, weight loss (if indicated), and increased physical activity (ADA, 2013a:S18). Nutrition intervention should consider the patient's age, pharmacological treatment, lipid levels, and other medical conditions (ADA, 2013a:S18). It is recommended that for patients with T2DM, statin therapy need to be added to lifestyle therapy, regardless of baseline lipid levels, and in those patients with clinical cardiovascular disease or who are above the age of 40 years with other cardiovascular disease risk factors (ADA, 2013a:S18).

Diet is the cornerstone of cardiovascular disease therapy, and all patients with T2DM should be receive standard advice on healthy eating habits and recommended food choices from a dietitian and/or nutritionist with particular emphasis on energy intake, fats, fibre, and alcohol intake (Amod *et al.*, 2012:S57).

### 5.4.4 Chronic complications

The mechanism by which diabetes leads to chronic complications is thought to be a result of the toxic effect of high glucose levels, the impact of elevated blood pressure, abnormal lipid levels and both "functional and structural abnormalities" of small and large blood vessels (Robbins *et al.*, 2008:658). The risk for complications is linked to poor nutritional status (longer duration of diabetes, increased body mass index (BMI), being sedentary and smoking), poor glycemic control, high diastolic blood pressure, infections, dyslipidemia and poor self-care all of which are modifiable and manageable (ADA, 2013a:S18).

The major chronic complications of diabetes are: cardiovascular disease (which is the major cause of death in diabetes and include angina, myocardial infarction, stroke, peripheral artery disease, congestive heart failure), nephropathy (common cause of renal failure in diabetes and of end stage renal disease), neuropathy (uncontrolled blood glucose and blood pressure can harm the nerves result in problems with digestion, urination, impotence, loss of feeling in the feet and toes, and legs), retinopathy (diabetes can harm sight and cause blindness, and increases the risk of cataracts and glaucoma) and foot ulceration.

Echouffo-Tcheugui & Kengne (2011: Online) reported that in Cameroon the prevalence of complications related to chronic diseases (particularly diabetes) were: retinopathy 42%; macular oedema 10.6%, and microalbumiuria 53.1%. A cross-sectional study done in Cameroon found the prevalence of diabetes-foot related lesions, neuropathy, ischemia, and foot deformity were 13.0%, 27.3%, 21.3% and 17.3% respectively.

In the current study, there was no evidence in the patient files of any screening for these complications and participants, when asked were not aware whether or not they were suffering of any of these complications. There were only a few who reported self-diagnosed retinopathy (7.3%) and neuropathy (4.0%). This is in contradiction to international guidelines for diabetes care which requires that screening tests for these complications should be performed annually (ADA, 2013a:S35).

### 5.4.5 Glycemic control

Glycemic control is defined as the achievement of normal blood glucose levels (FPG between 4.0 to 7.0 mmol/l and $HbA_{1c}$ of < 7.0%), which lowers the risk for diabetes-associated long-term complications (both microvascular and macrovascular), improves quality of life, and reduces hospital admissions and mortality (Klein *et al.*, 2013: Online; Gavin *et al.*, 2010:5).

Acceptable biochemical values ($HbA_{1c}$ of <7%, fasting blood glucose between 4.0 to 7.0 mmol/L, total serum triglycerides of <1.7 mmol/L, total serum cholesterol of <4.5 mmol/L, blood pressure of 140/80 mmHg or below), all contribute to a good nutritional status, and hence improved glycemic control and viewer complications (ADA, 2013a:S32; Amod *et al.*, 2012:S20).

According to Amod *et al.* (2012: Online), patients with T2DM do not generally control their blood glucose even in developed countries, as less than 50% of patients with diabetes are able to meet their glycemic targets, while less than 10% of these patients manage to achieve lipid and blood pressure targets. In the study by Khabbat *et al.* (2012: Online), poor glycemic control was observed in 65.1% of patients, and majority of these patients were on combination drugs and insulin.

Results of the current study demonstrated that the majority of participants (34.7%) had fasting blood glucose of greater than 10.0 mmol/L on the day of the interview, while 36.3% of the participants had suboptimal long-term glucose control as indicated by $HbA_{1c}$ level of >8% measured by the researcher. These participants were not achieving glycemic control, which puts them at increased risk of diabetes-related complications (retinopathy, neuropathy and nephropathy and macrovascular complications) (ADA, 2013a:S32; Amod *et al.*, 2012:S20). Furthermore, very few had evidence of recent $HbA_{1c}$ measurements recorded in their files.

**Summary**

In the current study, most participants (53.2%) had been diagnosed with T2DM for ≤5 years, and were on oral medication. $HbA_{1c}$ measurements (done by the researcher) indicated suboptimal glucose control in 36.3% of the total group and in 65% of the males. Most participants (94.4%) suffered from hypertension, which remained uncontrolled despite receiving anti-hypertension drugs from the clinics. Participants were not screened for retinopathy, neuropathy, nephropathy or dyslipidemia at the clinics, and few had $HbA_{1c}$ measurements recorded in their files. Even though chronic complications were not screened among participants, 7.3% of participants reported that they had symptoms of retinopathy, while 4.0% reported that they had symptoms of neuropathy. These findings indicate that these Basotho patients with T2DM are not receiving optimal health care as recommended by international standards, from the clinics.

### 5.5 Barriers that impact on treatment compliance

The barriers that might negatively impact on treatment compliance included provision of health care services at the clinic or to personal factors, socio-economical factors, social environment, and participants' knowledge, attitudes, beliefs and perceptions regarding dietary and lifestyle changes and medical therapies.

#### 5.5.1 Logistics regarding attendance of the clinic

All the participants in the current study visit the health care facilities only every third month. In 2005/2006 Makinga and Beke (2013:194) reported that almost half of the participants with T2DM in their study in Maseru consulted a physician every month, and the other half consulted a physician every two and/or three months. This they attributed to the private-public sector divide

in the country, finding that those who attended the public facilities (included in the current study) did so only every three months.

All but four of the participants in the current study lived in Maseru, and therefore attended the clinics there; 62.9% used to be regular attendees of the diabetes clinic at QE II hospital. Of the four from outside Maseru, three attended the Maseru clinics because their children lived in the city and one because there were no diabetes clinics near their home. As the group from outside Maseru was so small, information regarding the cost incurred by having to sleep over in the city when attending the clinics, could not be meaningfully evaluated.

More than three quarters (76%) of participants travelled to the clinic by taxi, while 17% walked there. Transport fees for most participants were M11.00 or more, with 10.5% paying more than M25.00. Although all of the participants were scheduled to attend the clinic only every third month, more than a third (38.7%) of participants reported that they sometimes fail to attend the clinic due to lack of money. This concurs with the low socio-economic status of the participants.

Most participants (85.5%) reported that they arrive at the clinics between 6:00 and 8:00 in the morning (the rest arrive even earlier between 4:00 and 6:00), and arrive at home or back at work, between 12:00 and 13h00. This entails many work hours lost for those who are employed.

Most (75.8%) of the participants indicated that they felt that the services at the clinics takes too long. About a third reported that they are usually serviced at the clinic within an hour of arrival, but most (57.3%) report that they wait between one and two hours. The services offered entails having blood pressure and blood glucose measured by a nurse, then consulting a doctor, and then collecting their medicines from the pharmacy. Between each step the waiting period was reported to be for most participants between one and two hours. Thus the total time spent at the clinic was 3-6 hours.

Nonetheless, participants (98.4%) indicated that the clinics provided good and up to standard services, and that they (66.1%) were satisfied with the communication with the health care providers. These findings contrast with by those of a recent study done in Zimbabwe, which

showed that most participants expressed that they were not satisfied with the services provided, citing delays by doctors, limited time for consultations and ability to ask questions and receive information as reasons for their dissatisfaction (Hjelm & Mufunda 2010:7).

Most participants in the current reported that they are not always serviced by the same nurse (86.3%) or doctor (64.5%), but most indicated that they did not have any problem whether or not they were serviced by the same nurse (75.0%) or doctor (49.2%). Most (96%) were satisfied that they spend adequate time with the doctor.

Almost a firth (17.7%) reported that they did not always receive all their medications from the pharmacy and then had to buy from another chemist. Almost two thirds of participants (61.3%) received the services for free and the rest indicated that they mostly paid a small fee of M15.00 per clinic visit.

About a third of participants felt that they were not receiving psychosocial and emotional support from the clinic, family members and friends. The majority (77.4%) of the participants was not aware of the Lesotho Diabetes Association, and only 1.6% was members.

### 5.5.2 Diet and lifestyle KAP

Analysis of the KAP of participants in the current study regarding diet and lifestyle factors related to the management of diabetes, showed that majority of the participants (52.4%) indicated that they had received education from health care providers about healthy eating habits, and knew what food to avoid.

Similar findings were supported by Makinga and Beke (2013:194), who used the Health Education Quantitative Index (HEQI), to assess the quantity, not the quality, of health education provided by the clinics for patients with T2DM in Maseru in 2005/2006. The HEQI revealed that out of 186 participants who were expected to have received education on all seven topics listed, only 11 had been educated on all seven topics, nine on six topics, 25 on five topics, 52 on four topics, 48 on three, 21 on two, and 10 on one topic only, hence the HEQI score of 49.9% was very low (the acceptable range is 80-100%) (Makinga & Beke, 2013:192). The majority of

participants (92.7%), however acknowledged that they received health education on healthy eating, physical activity, weight loss, use of alcohol and smoking, and proper foot care (Makinga & Beke, 2013:192). The common discussed topics were adherence to a prudent diet and physical activity (> 80%), while foot care (33%) seemed to be neglected in most cases (Makinga & Beke, 2013:192).

National data in US, indicated that almost 50% of patients with diabetes reported having received some type of health education on the management of diabetes, but less than 50% were ever referred to a dietitian (Evert *et al.*, 2013:Online).

In the current study, only about half (52.4%) reported that they received education about healthy eating habits from a health care provider, but 74.2% indicated that they had never received any written instructions or education from a dietitian and/or nutritionist. Most of the participants (>99%) could identify healthy dietary recommendations. Klein *et al.*, (2013:Online) showed that patients even after receiving some form of education on diabetes self-management topics, still had difficulty in altering their long-term behavioral patterns, while others were unwilling even to try.

Despite the poor dietary quality revealed by the analysis of daily intakes and the patterns revealed by the non-quantified FFQ, most participants (82.3%) believed that they were following the SAFBDG (which reflects prudent dietary advice). Nonetheless, some admitted that they sometimes fail to abide by the dietary and lifestyle recommendation due to financial constraints, (65.3%, which is in-line with their low socio-economic status as discussed before); as well as poor self-control (90.3%).

Therefore, involvement of an on-going dietetic care through individualized counseling would benefit the patients with T2DM.

Regarding the lifestyle habits, most of the participants reported on the KAP questionnaire, that they were doing some form of physical activity, and gardening seemed to be the most common. However none of them had ever received detailed written instructions for an exercise program

from a health care provider. The LRFS (2001:23) indicated that very few patients with diabetes used alcohol and were smoking as discussed before. Quite a significant number of participants (81.5%) in the current study reported on the KAP questionnaire that they did not use any form of alcohol, and knew that tobacco is forbidden in patients with diabetes (100%). These findings concur with the responses to the lifestyle questionnaires on the same topics. As discussed before, there were however about 50% of males in the study who were heavy drinkers, as well as about 12.0% of the participants who were using tobacco, concurring with the findings of Klein *et al.,* (2013:Online) that education on diabetes self-management topics, do not necessarily lead to altered behavior.

### 5.5.3 Knowledge and practices regarding self-care

Hjelm & Mufunda (2010:7) reported that there limited knowledge regarding self-care measures and most of the participants in the study were unable to identify the cause of their health problems and were not able to identify the disease at the onset, suspecting other diseases such as HIV and AIDS. There was also a limited knowledge regarding self-care measures and most of them were unable to identify the cause of their health problems.

All participants (100%) in the current study however knew about some of the symptoms of high blood sugar, as well as the consequences of uncontrolled blood sugar. Yet, 90% of the participants did not know about the normal reference range of fasting blood sugar and none of performed self-monitoring of blood glucose.

Participants could also identify the micro vascular complications associated with uncontrolled blood sugar: all participants identified blindness; 91.1% identified foot ulcers and 87.1% identified kidney problems as long-term consequences; and 98.4% believed that the complications of diabetes are preventable. Yet, none of participants reported that they are screened for any of these complications (which concur with the lack of any formation on these screening tests in the patient files). In contrast to the findings of Makinga & Beke among patients with T2DM in Maseru in 2005/2006 (2013:192) that foot care (33%) seemed to be neglected topic that participants received education on, in the current study the participants (90 to 100%) were very well informed about daily personal foot care recommendations.

In the current study, 91.9% reported that they received education about the use of medicines. According to their own perceptions most (98.4%) participants reported that they comply with taking their medications as prescribed, although the fasting blood glucose levels and blood pressure readings were high. Most (99.2%) of the participants reported that they do not just take the medications when symptoms occur, but regularly as prescribed (only 2.4% admitted that they sometimes forget to take their medications). All but one participant also reported having confidence in the benefits of the medications that they use.

Such confidence in the medications is a positive factor as it encourages compliance to the medical treatment. Reasner & Goke, (2012: Online) in the DARTS study revealed that 30% of patients with T2DM met the adherence goal while on monotherapy (either sulphonylurea or metformin), but when a combination of the two drugs was introduced, adherence goal dropped to 13%, indicating that polypharmacy could be one of the barrier towards medication adherence among the participants.

However over-reliance in the benefits of the medications to control blood glucose and prevent complications, could lead to patients to neglect their diets and lifestyle, which is still the cornerstone of diabetes management.

### 5.5.4 Alternative causes and treatments for diabetes

Hjelm & Mufunda (2010:7) found that in Zimbabwe most participants were not certain about the cause of diabetes: some said diabetes was caused by antihypertensive drugs or pancreatic disease, obesity and unhealthy diets, supernatural causes such as fate, punishment from God or spirits lying outside the person's own control. Participants also consulted traditional healers and and used traditional medicines, as well as complementary alternative medicines, including household remedies, herbs and prayers or holy water when they experienced symptoms of diabetes (Hjelm & Mufunda 2010:7). In the 2001 LRFS (2001: Online) some patients with diabetes were reported to visit traditional doctors and use traditional medicines in Lesotho.

On contrary, in the current study most of the participants could identify the signs and symptoms of diabetes, hence most of them consulted a doctor at the clinic, and none reported consulting a

traditional healer. None of the participants believed that diabetes was due to witchcraft or punishment from God. A small percentage of participants reported that they use traditional medicines, with "Haelale" being the most common. Only a few participants reported using "home remedies" including cinnamon, garlic, ginger and green tea. A small percentage of the participants also used nutritional supplements. Nonetheless, the majority of the participants reported that even if they use traditional medicines, herbs, and nutritional supplements, they still continued with their prescribed medications.

**Summary**

The majority of participants used to attend diabetes clinic at QE II hospital, and reported that they were currently visiting the clinic every third month, to have their blood glucose and blood pressures checked and to see the doctor. Participants reported that they were satisfied with the services offered at the clinics, though they felt that the services were taking too long. Participants acknowledged that they received education on dietary intake and medication usage, cessation of alcohol and smoking, but not on physical activity, though few participants still use alcohol, cigarette and smokeless tobacco (snuff). Only a small number of participants reported that they sometimes use traditional medicines, and nutritional supplements, but never stop using diabetes medications.

Overall the participants expressed confidence in the medical treatment and health care facilities, and showed good knowledge of the causes, consequences and associated risks of this disease. Yet their blood glucose levels and blood pressures were uncontrolled and they were never screen for complications. Their diets were inadequate in the sources of key nutrients and some participated in risky behavior regarding alcohol consumption and the use of tobacco. These Basotho patients with T2DM may benefit from referral to a dietician/nutritionist for individualized dietary and lifestyle programmes developed to take into account their socio-economic and cultural situation.

# CHAPTER 6: CONCLUSION AND RECOMMENDATIONS

In this chapter conclusions and recommendations will be drawn from the findings of the study regarding socio-demographic status, nutritional status, lifestyle habits, medical history, glycemic control, and barriers to treatment compliance.

## 6.1 Conclusion

According to the IDF, (2013b:24), diabetes is the fourth leading cause of mortality, and 80% of deaths attributed to diabetes occur in low-and-middle income countries; while three quarters of deaths from diabetes among people younger than 60 years of age, occur in Africa. Diabetes is also among the top ten causes of disability, resulting in life-threatening complications such as heart disease, stroke, renal failure, lower limb amputations and blindness (IDF, 2013b:24). It is estimated that in Africa, as many as 81% of people with diabetes are undiagnosed (IDF, 2012: Online; Amod *et al.*, 2012:S4). Hence diabetes was identified by the United Nations Resolution 61/225 of 2006 as a challenge in the achievement of agreed development goals, including the Millennium Development Goals (MDGs) 1 (to eradicate extreme poverty and hunger), 5 (to improve maternal health), and 6 (to combat Human Immunodeficiency Virus (HIV) and Acquired Immunodeficiency Syndrome (AIDS), malaria, and other diseases) (IDF, 2011: Online; Carter *et al.*, 2010:4229).

The current study found that patients with T2DM attending diabetes clinics in Maseru, Lesotho had low-socio-economic status, poor nutritional status, suboptimal glycemic control, and were at increased risks of cardiovascular diseases and other diabetes-related complications. The findings also supported previous studies that T2DM was associated with aging (30 years and above), female gender, rural-urbanization, low level of education, low income level, obesity, inadequate nutrients intakes (particularly micronutrients and essential fatty acids), and physical inactivity.

While some patients were able to achieve acceptable glycemic control, the prevalence of obesity (abdominal obesity) as measured by BMI, WC, WHtR, and BAI still placed them at increased risk of cardiovascular diseases and other diabetes-related chronic complications.

The patients with T2DM demonstrated positive attitudes towards services provided at the clinics despite the long waiting hours, and trusted and relied on the management services offered at the facilities, except for the few who opted to use traditional medicines and nutritional supplements together with their prescribed medicines.

The findings also revealed that patients received education on dietary intake, physical activity and use of medications by nurses and pharmacy technicians, though a number of them still did not adhere to lifestyle modifications (particularly with dietary intake). Therefore, provision of regular individualized nutritional counseling by dieticians and/or nutritionists and health education still remain a great challenge

The study found that the patients with T2DM were not monitored by either HbA1c measurements or self-monitoring of blood glucose (which recognizes the patterns of fasting blood glucose on a daily basis); and that the participants were not knowledgeable about the normal reference values for blood glucose. Furthermore, there were no screening done for chronic complications (retinopathy, neuropathy and nephropathy) at the clinics, nor were the patients referred for such screening. All these findings implied that the services offered to patients with diabetes were substandard (the patients are not being served adequately), and not in-line with the WHO recommendations. In some studies provision of below standards services by clinics has been shown to challenge the medication adherence in patients with diabetes. Hence, there is a need for a change towards improving the services offered to patients with diabetes, as poorly managed diabetes leads to serious complications, which increases the health expenditure.

### 6.2 Recommendations

The following recommendations are based on the above findings:

i) There is a need for the development of national policies, protocols and guidelines for prevention, care, and treatment of diabetes mellitus, which should include algorithm of referrals.

ii) A multidisciplinary team needs to be supported in the management of diabetes in all health care facilities, and should include clinicians, nurses, diabetes educators, dietitians and/or nutritionists, psychologists, pharmacists. Individualize nutritional counseling should be provided by dieticians and/or nutritionists as much as possible.

iii) Outreach programs should be improved and strengthened, in which health care workers will move out into the community to benefit the rural population (for example screening for diabetes among people in their mid-40 years). This may offer long-term solutions to the problems of management and prevention of diabetes. Females should be mostly targeted as there is a higher prevalence of diabetes among females than males in Lesotho as confirmed by many studies, including the current study. These Basotho females have a high prevalence of obesity, and their socio-economic status is very low, since most of them are not employed, and had only obtained a primary school education.

iv) Challenges such as irregular supply of medicines, inadequate health-care infrastructure, lack of adequate training and retraining of healthcare providers, and lack of education to patients living with diabetes and their families or care-givers, should be dealt with.

v) The use of confusing and complicated messages communicated to patients with diabetes should be avoided, for example the use of terms such as BMI. Other anthropometric measurements, which are easy to interpret and could, benefit the population as they predict diabetes and cardiovascular diseases very well, such as WC and WHtR could be explored. In the current study WHtR (waist circumference (cm) divided by height (cm)) above 0.5 identified the highest percentage of patients with T2DM at risk for macro-vascular complications. This offers a simple yet very practical solution to self-monitoring of body weight among these patients: by cutting them a piece of rope equal to their height and educating them that the string needs to be able to go around the waist at least twice for body weight to be acceptable.

vi) The Ministry of Health of Lesotho should strengthen maternal services in order to prevent pre-term births and intra-uterine malnutrition, one of the hypothesized risks factors of T2DM and other chronic diseases. Evidence from different studies has implicated a role of early-life exposures, maternal diet during pregnancy, postnatal growth, and childhood diet in chronic disease etiology. Thus, the first nine months shape the rest of one's life and early life programmes diseases such as diabetes and obesity (Malik *et al.*, 2011:Online).

vii) The Government of Lesotho should invest in early age interventions through legislations that promote physical activity and healthy eating in schools. As recently there have been increasing reports of children and adolescents (18 years and younger) also developing T2DM due to epidemic of obesity and physical inactivity.

viii) The National Diabetes Association should be strengthened, and its role of educating, screening and support among patients with diabetes be explained to all patients with diabetes and they should be motivated to become members.

With the concerted efforts to try and prevent diabetes and its chronic complications, the country could achieve the Millennium Development Goals 1, 5 and 6, hence cut-down the expenses of managing T2DM and the associated chronic complications. The WHO recommendations include: blood pressure should be measured at every routine visit to the diabetes clinic or primary health care provider; WC and weight should be measured at each visit, BMI should be evaluated annually; a comprehensive foot examination should be done annually or more often in patients with high risk of foot conditions; micro-albuminuria and serum creatinine should be evaluated annually; eye examinations to screen for retinopathy should be performed annually or more frequently if significant retinopathy is present; and neuropathy should be screened for in all patients at least annually (ADA, 2013a:S21; Amod *et al.*, 2012:S12).

**SUMMARY**

In 2013, 382 million people suffered from diabetes, with 80% of cases occurring in low-and-middle income countries. Diabetes and diabetes-related complications is the fourth most treated disease in Lesotho hospitals, incurring high medical costs which are largely subsidized by the Government. This study aimed to evaluate the socio-economic factors, nutritional status, medical history, glycemic control, and barriers to treatment compliance among Basotho out-patients with T2DM.

A quantitative descriptive study was conducted among 124 consenting patients with T2DM who attended Domiciliary and LDF Clinics in urban Maseru between October 2012 and March 2013. Information were recorded via questionnaires administered by structured one-on-one interviews with the participants; information from patient files and by anthropometrical measurements. Activity was assessed with the validated International Physical Activity Questionnaire.

Most participants were married (72.6%); employed (42.7%) or self-employed (26.6%); but 52.4% earned only M0.300 – M1500 per month; and most had only a primary (29.8%) or a high school (49.2%) education. Most were 40 - 60 years old, with almost 50% of the males being ≤50 years. Most participants were female, and females also had the lowest income and education levels.

Most (53.2%) had been diagnosed with T2DM for ≤5 years, and 67.7% were on oral glucose-lowering therapy, which 99.2% believed to be effective, and 98.4% reported strictly adhering to. All visit the clinics only every third month (61.3% for free and the rest for ≤ M15.00 per visit), while 40% reported that they sometimes failed to attend due to lack of money (75.8% travelled to the clinic by taxi).

Most (82.9%) believed that overweight causes diabetes, yet based on BMI, 89.2% were overweight/obese; and based on WC, WHtR, and BAI, 98.3%, 96.8% and 64.5% were at risk for diabetes-related complications. Almost all were knowledgeable about prudent dietary and lifestyle guidelines and the importance thereof for the management of T2DM, and reported that

they had received information on these subjects at the clinics, albeit for 92% never as written, individualized instructions from a dietician/nutritionist.

Most participants also did not meet the daily recommendations for intakes of milk and dairy products (92.7%), fruits (65.3%) and vegetables (78.2%); and mostly relied on maize porridge (91.9%) and brown bread (71%) as daily staples. Most cited financial constraints (65.3%) and poor self-control (90.3%) as reasons for non-compliance to dietary guidelines. Most (98%) were moderately active; 78.2% reported abstaining from alcohol, but 52% of the males reported taking 4-6 units of alcohol, mostly commercial beer, per session; and approximately 10% were current tobacco users.

None of the participants performed self-monitoring of blood glucose and 90.3% were ignorant about normal reference values. $HbA_{1c}$ measurements done by the researcher indicated suboptimal glucose control in 36.3%. Most (94.4%) suffered from hypertension, which remained uncontrolled despite receiving anti-hypertension drugs from the clinics. Participants were not screened for retinopathy, neuropathy, nephropathy or dyslipidemia at the clinics, and few had $HbA_{1c}$ measurements recorded in their files.

Despite the services falling short of international recommendations for the management of patients with T2DM, 98.4% of participants were satisfied with the services rendered and 82.0% were happy with the communication between patient and health care professional at the clinics. Most (75.8%) however felt that the services took too long, and 17.7% reported that the clinic did not always have all their medications available.

None of the participants believed that diabetes was due to witchcraft or punishment from God, or reported preferring to consult traditional healers. Few used traditional medicines (mostly "Haelale"); "home remedies" including cinnamon, garlic, ginger and green tea; or nutritional supplements; mostly in addition to (85.7%) rather than instead of, their prescribed medications.

In summary, this study identified shortcomings in the health services rendered to patients with T2DM at government-supported clinics in Maseru, as well as various barriers to compliance to

dietary and lifestyle recommendations, which need to be addressed: in recognition of patients' right to quality treatment, and to prevent diabetes-related complications which imparts a heavy financial burden on the health care system of a developing country like Lesotho.

**OPSOMMING**

In 2013, het 382 miljoen mense wêreldwyd aan diabetes gely, en 80% van gevalle het in lae- en middelinkomste-lande voorgekom. Diabetes is die vierde mees behandelde siekte in Lesotho-hospitale (twa verwante komplikasies), en lei tot hoë mediese kostes wat grootliks deur die staat gesubsidieer word. Hierdie studie het gepoog om die sosio-ekonomiese faktore, voedingstatus, mediese geskiedenis, glukemiese beheer en hindernisse tot die nakoming van behandeling onder Basotho buite-pasiënte met T2DM, te evalueer.

'n Kwantitatiewe beskrywende studie is uitgevoer onder 124 pasiënte met T2DM wat Domiciliary en LDF Klinieke in stedelike Maseru tussen Oktober 2012 en Maart 2013 besoek het, en ingeligte toestemming gegee het. Inligting is ingesamel mbv vraelyste tydens gestruktureerde een-tot-een onderhoude met die deelnemers; uit pasiëntlêers, en deur antropometriese metings. Aktiwiteit is beoordeel met die geldige "International Physical Activity Questionnaire".

Die meeste deelnemers was getroud (72.6%); indiensgeneem (42.7%) of self-indiensgeneem (26.6%); maar 52.4% het net M0.300 - M1500 per maand verdien; en die meeste het net primêre- (29.8%), of hoërskool (49.2%) opleiding gehad. Die meeste was 40 - 60 jaar ouds; met byna 50% van die mans ≤50 jaar. Die meeste deelnemers was vroulik, en vrouens het ook die laagste inkomste- en opvoedingsvlakke gehad.

Die meeste (53.2%) was ≤5 jaar met T2DM gediagnoseer, en 67.7% was op orale glukose-verlagende terapie; wat 99.2% geglo het doeltreffend was; en 98.4% aangedui het dat hulle streng volg. Almal het die klinieke slegs elke derde maand besoek (61.3% gratis, en die res teen ≤M15.00 per besoek), terwyl 40% gerapporteer het dat hulle soms besoeke oorslaan twa 'n gebrek aan geld (75.8% het per taxi na die kliniek gereis).

Hoewel die meeste (82.9%) geglo het dat diabetes deur oormassa veroorsaak kan word was 89.2% oormassa/vetsugtig volgens liggaamsmassa-indeks (LMI). Gebaseer op middelomtrek, middelomtrek: lengte-verhouding (WHtR), en liggaamsvetindeks (BAI), het 98.3%, 96.8% en

64.5% verhoodge risiko's vir diabeteskomplikasies gehad. Byna almal was goed ingelig oor aanbevole dieet- en lewenstylriglyne, en die belangrikheid daarvan vir die beheer van T2DM; en het gerapporteer dat hulle inligting oor hierdie onderwerpe by die klinieke ontvang het; hoewel 92% van deelnemers aangedui het dat hulle nooit 'n geskrewe, geïndividualiseerde program van 'n dieetkundige/voedingskundige ontvang het nie.

Die meeste deelnemers het nie aan die daaglikse aanbevelings vir inname van melk en suiwelprodukte (92.7%), vrugte (65.3%) en groente (78.2%) voldoen nie; en het meestal staatgemaak op mieliepap (91.9%) en bruinbrood (71%) as stapelvoedsels. Die meeste het finansiële beperkinge (65.3%) en swak selfbeheersing (90.3%) as redes aangevoer vir die nie-nakoming van die dieetriglyne. Die meeste (98%) was matig aktief; en 78.2% het geen alkoholinname gerapporteer nie, maar 52% van die mans het aangedui dat hulle 4-6 eenhede alkohol, meestal kommersiële bier, per sessie inneem. Ongeveer 10% was huidige tabakgebruikers.

Geen deelnemers het aangedui dat hulle self-monitering van bloedglukose doen nie en 90.3% was onbewus van normale verwysingwaardes. $HbA_{1c}$-metings deur die navorser, het suboptimale glukosebeheer in 36.3% aangedui. Die meeste (94.4%) het aan hipertensie gely, wat ongekontroleerd gebly het tsv van hipertensiemedikasie wat deur die klinieke aan hulle verskaf is. Geen deelnemers is vir retinopatie, neuropatie, nefropatie of dislipidemie gesif nie, en vir slegs 'n paar was $HbA_{1c}$ metings in hul lêers aangeteken.

Ten spyte daarvan dat die dienste nie aan internasionale aanbevelings vir die hantering van pasiënte met T2DM voldoen het nie, was 98.4% van die deelnemers daarmee tevrede, en 82.0% was tevrede met die kommunikasie tussen die pasiënt en die gesondheidsorgspan by die klinieke. Die meeste (75.8%) was egter van mening dat die dienste te lank neem, en 17.7% het gerapporteer dat die kliniek nie altyd al hul medisyne beskikbaar het nie.

Geen deelnemers het geglo dat diabetes aan heksery of straf van God toegeskryf moet word nie, of aangedui dat hulle 'n tradisionele geneser verkies nie. Min het tradisionele medisyne (meestal "Haelale"); "tuispreparate", insluitend kaneel, knoffel, gemmer en groen tee; of

voedingsaanvullings gebruik; meestal saam met (85.7%), eerder as in plaas van, hul voorgeskrewe medikasies.

Ter opsomming, het hierdie studie tekortkominge in die gesondheidsdienste wat aan pasiënte met T2DM by die regeringsondersteunde klinieke in Maseru gelewer word, asook verskeie hindernisse tot nakoming van dieet- en lewenstylaanbevelings, geïdentifiseer, wat aangespreek moet word: ter erkenning van die pasiënte se reg tot gehalte-behandeling, en om diabetes-verwante komplikasies te voorkom wat 'n swaar finansiële las op die gesondheidsorgstelsel van 'n ontwikkelende land soos Lesotho plaas.

**REFERENCES:**


Adebayo FO. 2010. *Dietary habits and prevalence of obesity among type 2 Diabetes patients seen at Scott Hospital, Morija, Lesotho*. Msc Dissertation. University of Limpopo (Medunsa Campus), South Africa.

American Diabetes Association. 2014. Position of the Academy of Nutrition and Dietetics: Dietary Fatty Acids for Healthy Adults. *J Acad Nutr Diet*, 114:136-153.

American Diabetes Association and American Dietetic Association. 2005. Diabetic Food Guide Pyramid. *Journal of the American Dietetic Association,* 95(11):1241-1360.

American Diabetes Association. 2013a. Standards of medical care in diabetes. *Diabetes care* [Online], 36 (1):S11-S66.

American Diabetes Association. 2013b. Diagnosis and Classification of Diabetes Mellitus. *Diabetes Care*, 36(Suppl 1): S67-S74.

American Diabetes Association. 2013c. Third-Party Reimbursement for Diabetes Care, Self-Management Education, and Supplies. *Diabetes Care* [Online], 36(Suppl 1):S98-S99.

Ajala O, English P & Pinkney J. 2013. Systematic review and meta-analysis of different dietary approaches to the management of type 2 diabetes. *Am J Clin Nutri*, 97(3):505-516. [Accessed January 30, 2013].

Alberti KG. 2005. Tight glycemic control: the key to improving the management of type 2 diabetes. *SA Journal of Diabetes and Vascular Disease*, 2 (3): 103-104.

Alberti KGMM, Eckel RH, Grundy SM, Zimmet PJ, Cleeman JI, Karen A, Donato SM. 2009. Harmonizing the Metabolic Syndrome: A Joint Interim Statement of the International Diabetes Federation Task Force on Epidemiology and Prevention: National Heart, Lung, and Blood

Institute; American Heart Association; World Heart Federation; International Atherosclerosis Society; and International Association for the Study of Obesity. *Circulation*, (120): 1640-1645.

Aliaezadeh B, Greiver M, Moineddin R, Meaney C, White D, Moazzam A, Moore KM & Belanger P. 2014. Association Between Socio-economic Status and Hemoglobin A1c Levels in a Canadian Primary Care Adult Population Without Diabetes. *BMC Fam Pract.*;15(7).

Alokail MS, Sabico S, Al-Saleh Y, Al-Daghril NM, Alkharfy KM, Vanhoutte PM & McTernan PG. 2013. Effects of probiotics in patients with diabetes mellitus type 2: study protocol for a randomized, double-blind, placebo-controlled trial. *Trial*, 14: 195. [Accessed July 4, 2013].

Amod A, Ascott-Evans BH, Berg GI, Blom DJ, Brown SL, Carrihil MM, Dave JA, Distiller LA, Ganie YN, Globler N, Heilbrunn AG, Huddle KRL, Janse van Rensburg G, Jivan D, Joshi P, Khutsoane DT, Levitt NS, May WM, Mollentze WF, Motala AA, Paruk IM, Pirie FJ, Raal FJ, Rauff S, Raubenheimer PJ, Randree HAR, Rheeder P, Tudhope L, Van Zyl DJ & Young M. 2012. The 2012 SEMDSA Guideline for the management of Type 2 Diabetes (Revised). *JEMDSA* [Online], 17(2) (suppl 1):S1-S95.

Amato MC, Giordano C, Galia M, Criscimanna A, Vitabile S, Midiri M & Galluzzo A. 2010. Visceral Adiposity Index. A reliable indicator of visceral fat function associated with Cardiometabolic risk. *Diabetes Care*, 33(4):920-922. [Accessed 12 January, 2010].

Ashwell M, Gunn P & Gibson S. 2012. Obesity Comorbidity Diagnostic. Waist-to-height ratio is a better screening tool than waist circumference and BMI for adult cardiometabolic risk factors: Systematic review and meta-analysis. *Obesity reviews*, 13:275-286. [Accessed 17 October, 2011].

Ashwell M. 2011. The Waist-to-Height Ratio is a Good, Simple Screening tool for Cardiometabolic Risk. *Nutrition Today*, 46(2):85-89.

Ashwell M & Browning LM. 2011. The increasing Importance of Waist-to-Height Ratio to Assess Cardiometabolic Risk: A Plea for Consistent Terminology. *The Open Obesity Journal*, 3: 70-77.

Ashwell M & Hsieh SD. 2005. Six reasons why the waist-to-height ratio is a rapid and effective global indicator for health risks of obesity and how its use could simplify the international public health message on obesity. *Journal of Food Sciences and Nutrition*, 56(5):303-307.

Azevedo M & Alla S. 2008. Diabetes in Sub-Saharan Africa: Kenya, Mali, Mozambique, Nigeria, South Africa and Zambia. *Int J Diabetes Dev Ctries*, 28 (4): 101-108.

Bahadoran Z, Mirmiran P & Azizi F. 2013. Dietary polyphenols as potential nutraceuticals in management of diabetes: a review. *Journal of Diabetes and Metabolic Disorders*, 12:43. [Accessed 13 August, 2013].

Baliunas DO, Taylor BJ, Irving H, Roerecke M, Patra J, Mohapatra S & Rehm J. 2009. Alcohol as a Risk Factor for Type 2 Diabetes. A systematic Review and meta-analysis. *Diabetes Care*, 32(11):2123-2132. [Accessed July 22, 2009].

Beasley JM & Wylie-Rosett J. 2013. The role of dietary proteins among persons with diabetes. *Curr Atheroscler Rep* [Online], 15(9):348. [Accessed January 30th, 2013].

Benson G, Pereira RF & Boucher JL. 2011. Rationale for the use of a Mediterranean Diet in Diabetes Management. *Diabetes Spectrum*; 24(1): 36-40.

Bergman RN, Stefanovski D, Buchanan TA, Sumner AE, Reynolds JC, Sebring NG, Xiang AH & Watanabe RM. 2011. A better Index of Body Adiposity. *Obesity (Silver Spring)* [Online], 19(5):1083-1089. [Accessed March 3rd, 2011].

Bhalla K. 2013. The Health Effects of Motorization. *PLoS Med*, 9(6): e1001458. [Accessed June 11, 2013].

Bororgmanesh M, Hadaegh F & Azizi F. 2011. Predictive Performance of the visceral adiposity index for a visceral adiposity-related risk: Type 2 diabetes. *Lipids Health Dis.*, 10:88.

Boyko EJ, Seelig AD, Jacobson IG, Hooper TI, Smith BS, Smith TC & Crum-Cianflone NF. 2013. Sleep characteristics, Mental Health, and Diabetes Risk. A prospective study of U.S. military service members in the Millennium Cohort Study. *Diabetes Care*; 36(10): 3154-61. [Accessed July 8th, 2013].

Bray GA. 2010. Fructose: Pure, White, and Deadly? Fructose, by any other name, is a Health Hazard. *J Diabetes Sci Technol*; 4(4): 1003-1007. [Accessed July 1st, 2010].

Browning LM, Hsieh SD & Ashwell M. 2010. Asystematic review of waist-to-height ratio as a screening tool for the prediction of cardiovascular disease and diabetes: 0.5 could be a suitable global boundary value. *Nutrition Research Reviews*, 23: 247-269.

Byers KG & Savaiano DA. 2005. The myth of increased lactose intolerance in African-Americans. *Am Coll Nutr*, 24(suppl 6):569S-573S.

Carter P, Gray LJ, Troughton J, Khunti K & Daviec MJ. 2010. Fruits and vegetables intake and incidence of Type 2 diabetes mellitus: Systematic review and meta-analysis. *BMJ* [Online], 341:C4229. [Accessed August 20th, 2010].

Central Intelligence Agency (CIA). 2012. The World Fact book [Online]. www.cia.gov/publications.

Centers for Disease Control and Prevention (CDC). 2011. National diabetes fact sheet: national estimates and general information on diabetes and prediabetes in the United States. www.cdc.gov/diabetes/pubs//.

Chen GC, Lv DB, Pang Z & Liu QF. 2013. Red and processed meat consumption and risk of stroke: a meta-analysis of prospective cohort studies. *European Journal of Clinical Nutrition*; 67:91-95. [Accessed November 21st, 2012].

Conn VS, Hafdahl AR & Mehr DR. 2011. Interventions to increase Physical Activity among Healthy Adults: Meta-analysis of outcomes. *Am J Public Health* [Online], 101 (4):751-758. [Accessed February 17th, 2011].

Copeland KC, Silvertein J, Moore KR, Prazer GE, Raymer T, Shiffman RN, Springer SC, Thaker VV, Anderson M, Spann SJ & Flinn SK. 2013. Management of Newly Diagnosed Type 2 Diabetes Mellitus in Children and Adolescents. *Pediatrics* [Online], 131 (2):364-382. [Accessed January 28th, 2013].

Crandall JP, Polsky S, Howard AA, Perreault L, Bray GA, Barrett-Connor E, Brown-Friday J, Whittington T, Foo S, Ma Y & Edelstein SL. 2009. Alcohol consumption and diabetes risk in the diabetes prevention program. *Am J Clin Nutr.*; 90(3): 595-601. [Accessed July 29th, 2009].

Craig CL, Marshall AL, Sjostrom M, Bauman AE, Booth ML, Ainsworth BE, Pratt M, Ekelund U, Ynqve A, Sallis JF & Oja P. 2003. International Physical Activity Questionnaire: 12-Country reliability and validity. *Med Sci Sports Exerc*, 35(8): 1381-1396.

De Feo P. & Schwarz P. 2013. Is Physical Exercise a Core Therapeutic Element for Most Patients with Type 2 Diabetes? *Diabetes Care*; 36 (Suppl2): S149- S154.

De Koning L, Chiuve SE, Fung TT, Willett WC, Rimm EB, & Hu FB. 2011. Diet quality scores and the risk of type 2 diabetes in men. *Diabetes Care*, 34:1150-1156. [Accessed March 1st 2011].

Department of Health, Republic of South Africa 2003. Health and Demographic Survey (Full report pp (276-277). Accessed on: 28th April, 2011. Available from: http://www.mrc.ac.za /bod/sadhs.htm.

Dunstan DW, Zimmet PZ, Welborn TA, Cameron AJ, Shaw J, de Courten M, Jolley D & Mc Carty DJ. 2002. Australian Diabetes, Obesity and Lifestyle Study (AusDiab). *Diabetes Res Clin Pract*; 57(2):119-129.

Echouffo-Tcheugui JB & Kengne AP. 2011. Chronic non-communicable diseases in Cameroon-burden determinants and current policies. *Global Health* [Online], 7: 44. [Accessed November 23rd, 2011].

Ejima K, Aihara K & Nishiura H. 2013. Modeling the obesity epidemic: Social contagion and implications for control. *Theoretical Biology and Medical Modelling*; 10:17.

Ekinci EI, Clarke S, Thomas MC, Moran JL, Cheong K, MacIsaac RJ & Jerums G. 2011. Dietary Salt Intake and Mortality in Patients With Type 2 Diabetes. *Diabetes Care*; 34:703-709.

Elwood PC, Pickering JE, Givens I & Gallacher JE. 2010. The consumption of Milk and Dairy Foods and the Incidence of Vascular Disease and Diabetes: An Overview of the Evidence. *Lipids*; 45(10):925-939. [Accessed April 16th, 2010].

Estruch R, Ros E, Salas-Salvado J, Covas M, Corella D, Aros F, Gomez-Gracia E, Ruiz-Gutierrez V, Lapetra J, Lamuela-Raventos RM, Serra-Majem L, Pinto X, Basora J, Munoz MA, Sorli JV, Martinez JM, & Martinez-Gonzalez MA. 2013. Primary Prevention of Cardiovascular Disease with a Mediterranean Diet. *N Engl J Med*; 368:1279-90. [Accessed February 25th, 2013].

Eriksson A, van den Donk M, Hilding A & Ostenson C. 2013. Work Stress, Sense of Coherence, and Risk of Type 2 Diabetes in a Prospective Study of Middle-aged Swedish Men and Women. *Diabetes Care, dc12-1738.* [Accessed May 1st, 2013].

Evert AB, Boucher JL, Cypress M, Dunbar SA, Franz MJ, Mayer-Davis EJ, Neumiller JJ, Nwankwo R, Verdi CL, Urbanski P & Yancy WS. 2013. Nutrition Therapy Recommendations

for the Management of Adults with Diabetes. *Diabetes Care* 36(11):3821-3842. [Accessed October 9th, 2013].

Feinman RD & Fine EJ. 2013. Fructose in perspective. *Nutrition & Metabolism,* 10:45. [Accessed July 1st, 2013].

Feller S, Boeing H & Pischon T. 2010. Body Mass Index, Waist Circumference, and the risk of Type 2 Diabetes Mellitus. Implications for Routine Clinical Practice. *Dtsch Arztebl Int*., 107(26):470-476. [Accessed July 2nd, 2010].

Food and Nutrition Coordinating Office, 2007. Lesotho National Nutrition Survey Report.

Funnell MM. 2006. The Diabetes Attitudes, Wishes, and Needs (DAWN) Study. *Clinical Diabetes*, 24(4):154-155.

Franz MJ. 2012. Medical Nutrition Therapy for Diabetes Mellitus and Hypoglycemia of nondiabetic origin. *Krause's Food and the Nutrition Care Process.* Ed. by Mahan KL, Escott-Stump S & Raymond JL. 13th ed. Philadelphia: W.B. Saunders Company: 675-709.

Gallagher D, Heymsfield SB, Heo M, Jebb SA, Murgatroyd PR & Sakamoto Y. 2000. Healthy percentage body fat ranges: an approach for developing guidelines based on body mass index. *Am J ClinNutr.*, 72(3): 694-701.

Ganiyu AB. 2009. *Non-adherence to lifestyle modification recommendations (diet and exercise) amongst Type 2 Diabetes Mellitus patients attending Extension II clinic in Gaborone, Botswana*. University of Limpopo (Medunsa campus): 83.

Gariballa S, Afandi B, AbuHattem M, Yassin J, Habib H & Ibrahim W. 2013. Oxidative damage and inflammation in obese diabetic Emirati subjects supplemented with antioxidants and B-vitamins: a randomized placebo-controlled trial. *Nutrition & Metabolism*, 10:21. [Accessed February 4th, 2013].

Gavin JR, Stolar MW, Freeman JS & Spellman CW. 2010. Improving outcomes in patients with Type 2 diabetes mellitus: Practical solutions for clinical challenges. *J Am Osteopath Assoc.* [Online], 110:2-14. [Accessed November 23rd, 2011].

Gibson RS. 2005. Anthropometric assessment. *Principles of Nutritional Assessment*. 2nd ed. USA: Oxford University Press: 6-283.

Glass JM, Buu A, Adam KM, Nigg JT, Puttler LI, Jester JM & Zucker RA. 2009. Effects of Alcoholism severity and Smoking on executive function Neurocognitive Function. *Addiction* [Online], 104(1):38-48. [Accessed January 30th, 2010].

Glazier RH, Creatore MI, Weyman JT, Fazli G, Matheson FI, Gozdyra P, Moineddin R, Shriqui VK & Booth GL. 2014. Density, Destinations or Both? Comparison of Measures of Walkability in Relation to Transportation Behaviours, Obesity and Diabetes in Toronto, Canada.

Goedecke JH, Jennings CL & Lambert EV. 2006. Obesity in South Africa. *In Chronic diseases of lifestyle in South Africa*: 1995-2005. pp. 65-79. Cape Town: MRC; available at http://www.mrc.ac.za/chronic/cdl/1995-2005.pdf [Accessed on November 11th, 2010].

Gonzalez JS, Schneider HE, Wexler DJ, Psaros C, Delahanty LM, Cagliero E & Safren SA. 2013. Validity of medication Adherence Self-Reports in Adults With Type 2 Diabetes. *Diabetes Care*, 36:831-837.

Graff-Iversen S, Anderssen SA, Holme IM, Jenum AK & Raastad T. 2007. An adapted version of the Long International Physical Activity Questionnaire (IPAQ-L): Construct validity in a low-income, multiethnic population study from Oslo, Norway. *Int J Behav Nutr Phys Act*, 4:13.

Grundy SM, Benjamin IJ, Burke GL. 1999. AHA Scientific statement. Diabetes and Cardiovascular disease. A statement for healthcare professionals from the American Heart Association. *Circulation*, 100(10): 1134-1146.

Haffner SM, Lehto S, Ronneman TR, Pyorala K & Laakso M. 1998. "Mortality from coronary heart disease in subjects with type 2 diabetes and non-diabetic subjects with and without prior myocardial infarction. *The New England Journal of Medicine*, 339(4): 229-234.

Hales CN & Barker DJP. 2013. Type 2 (Non-Insulin Dependent) Diabetes Mellitus. The Thrifty Hypothesis. *Int J Epidemiol*, 42(5): 1215-1222.

Hallal PC, Andersen LB, Bull FC, Guthold R, Haskell W & Ekelund U. 2012. Global physical activity levels: surveillance progress, pitfalls, and prospects. *Lancet*, 80(9838): 247-257.

Hammond K, 2008. Assessment: Dietary and Clinical Data. *In Krause Food and Nutrition Therapy*. Ed by LK Mahan & Escott-Stump S. 12th ed. Philadelphia: W.B. Saunders Company: 398-402.

Hammond KA. 2012. Assessment: Dietary and Clinical Data. *Krause's Food and the Nutrition Care Process.* Ed. by Mahan KL, Escott- Stump S & Raymond JL. 13th ed. Philadelphia: W.B. Saunders Company: 163-171.

Heianza Y, Arase Y, Saito K, Tsuji H, Fujihara K, Hsieh SD, Kodama S, Shimano H, Yamada N, Hara S & Sone H. 2013. Role of alcohol drinking pattern in Type 2 diabetes in Japanese men: Toranomon Hospital Health Management Center Study 11 (TOPICS 11). *Am J Clin Nutr*, 97(3): 561-568. [Accessed January 23rd, 2003].

Heraclides A, Chandola T, Witte DR & Brunner EJ. 2009. Psychosocial Stress at Work Doubles the Risk of Type 2 Diabetes in Middle-Aged Women: Evidence from the Whitehall II Study. *Diabetes Care*, 32(12): 2230 - 2235. [Accessed August 31st, 2009].

Herman WH & Zimmet P. 2012. Type 2 diabetes: An epidemic requiring global attention and urgent action. *Diabetes Care*, 35(5):943-944.

Hjelm K & Mufunda E. 2010. Zimbabwean diabetes' beliefs about health and illness: an interview study. *BMC International Health and Human Rights*; (10):7.

Hosseinpoor AR, Bergen N, Mendis S, Harper S, Verdes E, Kunst A & Chatterji. 2012. Socioeconomic inequality in the prevalence of non-communicable diseases in low-and middle-income countries: Results from the World Health Survey. *BMC Public Health*; 12:474.

Hovanec N, Sawant A, Overend RJ & Vandervoort AA. 2012. Resistance Training and Older Adults with Diabetes Mellitus: Strength of the Evidence. *J Aging Res* [Online], 2012:284635. [Accessed September 4th, 2012].

Howard ME & White ND. 2013. Potential benefits of cinnamon in type 2 Diabetes. *Am J Lifestyle Med*. 2013, 7(1):23-26.

Institute of Medicine. 2002. *Dietary reference intakes for energy, carbohydrate, fiber, fat, fatty acid, cholesterol, protein and amino acids*. Washington DC: The National Academies Press.

International Diabetes Federation. Definition and diagnosis of diabetes mellitus and intermediate hyperglycemia. 2010. Report of a WHO/IDF Consultation. www.who.int/diabetes/publications/diabetes/

International Diabetes Federation. 2011. IDF Diabetes Atlas, 5th edition. Brussels, Belgium: World Diabetes Day, 2011. November 14, 2011. 2011 Available from: http://www.idf.org/diabetesatlas.

International Diabetes Federation. 2012. Global Guideline for Type 2 Diabetes. IDF Diabetes Atlas, 5th edition. Available from: http://www.idf.org/diabetesatlas/5e/update.

International Diabetes Federation. 2013a. Managing Older People with Type 2 Diabetes: Global guideline.

International Diabetes Federation. 2013b. IDF Diabetes Atlas, 6th Edition. Available from: http://www.idf.org/diabetesatlas/data-visualisations.

James WPT. 2008. WHO recognition of the global obesity epidemic. *International Journal of Obesity*, 32:S120-S126.

James-Todd TM, Karumanchi A, Hibert EL, Mason SM, Vadrais MA, Hu FB & Rich-Edwards JW. 2013. Gestational Age, infant birth weight, and Subsequent risk of type 2 Diabetes in Mothers. *Prev Chronic Dis*, 10: 120336.

Jennings A, Welch AA, Spector T, Macgregor A & Cassidy A. 2014. Intakes of Anthocyanins and Flavones are Associated with Biomarkers of Insulin Resistance and Inflammation in Women. *J. Nutr.* 144(2):202-208. [Accessed December 11th, 2013].

Johnson RK & Hankin JH. 2003. Dietary Assessment and Validation. *In Nutritional*: *Assessment* by Lee RD and Nieman DC. 4th Edition. New York: McGraw Hill: 227-231.

Kahn SE, Cooper ME & Prato SD. 2013 Pathophysiology and treatment of type 2 diabetes Perspectives on the past, present, and future. *The Lancet*, 13:62154-6.

Katzenellenbogen JM, Jourbert G & Abdool Karim SS. 1997. *Epidemiology a Manual for South Africa.* Oxford University Press. Southern Africa.

Khalaf A, Ekblom O, Kowalski J, Berggren V, Westergren A & Al-Hazzaa H. 2013. Female University Students' Physical Activity Levels and Associated Factors-A Cross-Sectional Study in SouthWestern Saudi Arabia. *Int. J Environ Res Public Health*; 10(8):3502-3517. [Accessed August 9th, 2013].

Khardori R & Griffing GT. 2012. Type 2 Diabetes mellitus clinical presentation. http://www.medscape.com/article/117853-clinical.

Khattab M, Khader YS, Al-Khawaldeh A & Ajlouni K. 2010. Factors associated with poor glycemic control among patients with Type 2 Diabetes. *Journal of Diabetes and Its Complications*, 24:84-89.

Kim J & Park Y. 2012. Anti-diabetic effect of sorghum extract on hepatic gluconeogenesis of streptozotocin-induced diabetic rats. *Nutrition & Metabolism*, 9:106. [Accessed November 27th, 2012].

Klein HA, Jackson SM, Street K, Whitacre JC & Klein G. 2013. Diabetes Self-Management Education: Miles to Go. *Nurs Res Pract* [Online], 2013:581012. [Accessed March 20th, 2013].

Klisiewics AM and Raal F. 2009. Sub-optimal management of type 2 diabetes mellitus – a local audit. *JEMDSA*, 14(1):13-16.

Knowles KM, Paiva LL, Sanchez SE, Revillal L, Lopez T, Yasuda MB, Yanez ND, Gelaye B & Williams MA. 2011. Waist circumference. Body Mass Index, and other Measures of Adiposity in Predicting Cardiovascular Risk factors among Peruvian Adults. *Int J Hypertens*, 2011: 931402. [Accessed January 24th, 2011].

Krishnan M, Coogan PF, Boggs DA, Rosenberg L & Palmer JR. 2010. Consumption of restaurant foods and incidence of type 2 diabetes in African American women. *Am J Clin Nutr*, 91(2):465-471. (Accessed 16 December, 2009).

Kruger HS, Faber M, Schutte A & Ellis SM. 2013. A proposed cut-off point of WHtR for metabolic risk in African township adolescents. *Nutrition*, 29 (3): 502-507. [Accessed August 5th, 2012].

Kruger DF & Spollett GR. 2010. Addressing Barriers to Timely Intensification of Diabetes Care: The Relationship Between Clinical Inertia and Patient Behaviour. *Consultant* [Online], www.Consultantline.com.

Lachat C, Otchere S, Roberfroid D, Abdulai A, Seret FM, Milesevic J, Xuereb G, Candeias V & Kolsteren P. 2013. Diet and Physical Activity for the Prevention of Non-communicable Disease in Low-and Middle-Income Countries: A Systematic Policy Review. *PLoS Med.*, 10(6): e1001465. [Accessed June 11th, 2013].

Laine MK, Eriksson JG, Kujala UM, Wasenius NS, Kaprio J, Backmand HM, Peltonen M, Mertsalmi TH & Sarna S. 2013. A former career as a male elite athlete – does it protect against type 2 diabetes in later life? *Diabetologia*, 57(2): 270-4.

Lategan R, van den Berg VL, Walsh CM. 2014. Body adiposity indices are associated with hypertension in black, urban Free State Community. *Afr J Prm Health Care Fam Med*, 6(1): A vailable at: http://dx.doi.org/10.4102/phcfm.v6c1.581

Laverty AA, Mindel JS, Webb EA & Millett C. 2013. Active Travel to work and Cardiovascular Risk Factors in the United Kingdom. *Am J Prev Med* [Online], 45(3): 282-288. [Accessed March 20th, 2013].

Lee K. 2010. Sociodemographic Status and Self-Reported BMI-related Morbidity in Koreans. *Yonsei Med J.*, 51(2):171-177. [Accessed February 12th, 2010].

Lee PN & Hamling J. 2009. Systematic review of the relation between smokeless tobacco and cancer in Europe and North America. *BMC Med.* , 7:36. [Accessed July 29th, 2009].

Lee RD & Nieman DC. 2010. *Nutritional Assessment*. 3rd ed. New York: Mc Graw-Hill Companies: xv, 73-99.

Lehlasoa MR. 2011. *Prevalence of the known risk factors in women diagnosed with breast cancer at Queen II Hospital, Maseru.* Msc dissertation. University of the Free State, [Online]. Available at: http://www.bnl.gov/isd/documents/30546.pdf [Accessed:26 March 2014].

Leitzmann MF, Moore SC, Koster A, Harris TB, Park Y, Hollenbeck A & Schatzkin A. 2011. Waist Circumference as Compared with Body-Mass Index in Predicting Mortality from Specific Causes. *PLoS One*; 6(4): e18582. [Accessed April 26th, 2011].

Lemacks J, Wells BA, Ilich JZ & Ralston PA. 2013. Interventions for Improving Nutrition and Physical Activity Behaviors in Adults African American Populations. A Systematic Review, January 2000 Through December 2011. *Prev Chronic Dis*., 10: 120256.

Lesotho Demographic and Health Survey (LDHS). 2009. Ministry of Health and Social Welfare Maseru, Lesotho, 261-268.

Lesotho National Nutrition Survey Report. 2007: 49.

Lesotho Risk Factor Survey. 2001. Survey Report on Prevalence of Diabetes and Hypertension in Lesotho. Disease Control Division, Ministry of Health and Social Welfare.

Levitt NS, Steyn K, Dave J & Bradshaw D. 2011. Chronic noncommunicable diseases and HIV-AIDS on a collision course: relevance for health care delivery, particularly in low-resource settings-insights from South Africa. *Am J Clin Nutr*, 94(6):1690s-1696s. [Accessed November 9, 2011].

Li S, Zhao JH, Luan J, Langenburg C, Luben RN, Khaw KJ, Wareham NJ & Loos RJF. 2011. Genetic predisposition to obesity leads to an increased risk of T2DM in our large population-based, longitudinal cohort with an average follow-up period of 12.9 years. *Diabetologia*, 54(4):776-782 [Accessed January 26, 2011].

Liao S, Tseng W, Chen C, & Wu W. 2012. Estimating the Economic Impact of Climate Change on Cardiovascular Diseases-Evidence from Taiwan. *Int J Environ Res Public Health*, 7(12):4250-4266.

Ling C & Groop L. 2009. Epigenetics: A Molecular Link Between Environmental Factors and Type 2 Diabetes. *Diabetes*, 58(12):2718-2725.

Livesey G, Taylor R, Livesey H & Liu S. 2013. Is there a dose-response relation of dietary glycemic load to risk of type 2 diabetes? Meta-analysis of prospective cohort studies. *Am J Clin Nutr*, 97(3):584-596. [Accessed January 30th, 2013].

Maghsoudi Z & Azadbakht L. 2012. How dietary patterns could have a role in prevention, progression, or management of diabetes mellitus? Review on the current evidence. *J Res Med Sci.* [Online], 17(7):694-709. [Accessed March 20th, 2013].

Maher D, Waswa L, Baisley K, Karabarinde A, Unwin N & Gross-Kurth H. 2010. Distribution of hyperglycemia and related Cardiovascular disease risk factors in low-income countries: a cross-sectional population-based survey in rural Uganda. *Int. J Epidemiol*., 40(1):160-171. [Accessed October 5th, 2010].

Makinga PN & Beke A. 2013. A cross-sectional survey on the lifestyle and health-seeking behaviour of Basotho patients with diabetes. *S Afr Fam Pract*, 55 (2): 190-195.

Malik VS & Hu FB. 2011. Sugar-sweetened beverages and health: where does the evidence stand? *Am J Clin Nutr,* 94(5): 1161-1162. [ Accessed October 12th, 2011].

Malik VS, Popkin BM, Bray GA, Despr'es JP, Willett WC & Hu FB. 2010. Sugar-sweetened Beverages and Risk of Metabolic Syndrome and Type 2 Diabetes. A meta-analysis. *Diabetes Care*, 33(11): 2477-2483. [Accessed August 6th, 2010].

Malik VS, Sun Q, van Dam RM, Rimm EB, Willett WC, Rosner B & Hu FB. 2012. Adolescent dairy product consumption and risk of type 2 diabetes in middle-aged women. *Am J Clin Nutr*. [Accessed July 13th, 2011].

Mann JI & Morenga LT. 2013. Diet and diabetes revisited, yet again. *Am J Nutr*. [Online], 2013, 97 (3): 453-454. [Accessed January 30th, 2013].

Mbaezue N, Mayberry R, Gazmararian J, Quarshie A, Ivonye C & Heisler M. 2010. The impact of Health Literacy on self-monitoring of blood Glucose in patients with Diabetes Receiving care in an Inner-City Hospital. *J Natl Med Assoc*. [Online], 102(1):5-9. [Accessed September 14th, 2010].

McCathy MP, Best MJ & Betts RA. 2010. Climate change in cities due to global warming and urban effects. Geophysical Research letters, 37(9): 1-5. [Accesed 8 May, 2010].

McMichael AJ. 2013. Globalization, climate change, and Human Health. *N Engl J Med*; 368: 1335-1343.

Miller CK, Kristeller JL, Heading SA, Nagaraja H & Miser WF. 2012. Comparative effectiveness of a mindful eating intervention to a diabetes self-management intervention among adults with type 2 diabetes: A pilot study. *Journal of the Academy of Nutrition and Dietetics*, 112(11): 1835-1842.

Millett C, Agrawals S, Sullivan R, Vaz M & Kurpad A. 2013. Association between Active Travel to work and Overweight, Hypertension, and Diabetes in India: A cross-sectional study. *PLoS Med*, 10(6): e10014459.

Molitch ME. 2013. Current state of Type 2 Diabetes Management. *Am J Manag Care*, 19: S136-S142.

Morrish NJ, Wang SL & Stevens LK. 2001. Mortality and cause of death in the WHO multinational study of vascular disease in diabetes. *Diabetologia*, 44(2):S14-S21.

Naja F, Hwalla N, Itani L, Azar ST, Zeidan MN & Nasreddine L. 2012. Dietary Patterns and Odds of Type 2 Diabetes in Beirut, Lebanon. A Case-Control Study. *Nutr. Metab.*, 9:111.

Nakamura YK & Omaye ST. 2012. Metabolic diseases and pro- and prebiotics: Mechanistic insights. *Nutrition & Metabolism*, 9:60. [Accessed June 19th, 2012].

Nilsson PM. 2009. Hypertension and Diabetes: Should we treat early surrogates? What are the cons? *Diabetes care*, 32(Suppl 2): S290-S293.

Norris LE, Collene AL, Asp ML, Hsu JC, Liu LF, Richardson JR, Li D, Bell D, Osei K, Jackson RD & Belurly M. 2009. *Am J Clin Nutr*, 90(3):468-476.

Nyamdorj R. 2010. Anthropometric measures of obesity-their association with type 2 diabetes and hypertension across ethnic groups. *Public Health* [Online], M207:70. [Accessed January 30th, 2013].

O'Connor LM, Lentjes MAH, Luben RN, Khaw K, Wareham NJ & Forouhi NG. 2014. Dietary dairy product intake and incident type 2 diabetes: a prospective study using dietary data from a 7-day food diary. *Diabetologia* [Online], 98:1066-1083.

Osgood ND, Dyck RF & Grassmann WK. 2011. The Inter-and Intergenerational impact of Gestational Diabetes on the Epidemic of Type 2 Diabetes. *Am J Public Health*, 101(1):173-179.

Pan A, Sun Q, Bernstein AM, Manson JE, Willett WC & Hu FB. 2011. Red meat consumption and risk of type 2 diabetes: 3 cohorts of US adults and an updated meta-analysis. *Am J Clin Nutr*, 94(4):1088-1096. [Accessed August 10th, 2011].

Parikh SM, Obuku EA, Walker SA, Semeere AS, Auerbach BJ, Hakim JG, Mayanja-Kizza H, Mugyenyi PN, Salata RA & Kityo CM. 2013. Clinical Differences between Younger and Older Adults with HIV/AIDS Starting Antiretroviral Therapy in Uganda and Zimbabwe: A Secondary Analysis of the DART Trial. *PLoS One,* 8(10):e76158. [Accessed October 2nd, 2013].

Pells JJ, Stout AL, Rodriguez MM, Quigley CA & Musante GJ. 2012. Brief Intensive Lifestyle Intervention Leads to Rapid Diabetes Improvements. *Am J Lifestyle Med.* [Online], 6 (2): 178-185. [Accessed January 30th, 2013].

Plotnikoff RC, Costigan SA, Karunamuni ND & Lubans DR. 2013. Community-based Physical Activity Interventions for Treatment of Type 2 Diabetes: Asystematic Review with Meta-Analysis. *Front Endocrinol* [Online], 4:3. [Accessed January 29th, 2013].

Position of the Academy of Nutrition and Dietetics: Use of Nutritive and Non-nutritive Sweeteners. 2012. *J Acad Nutr Diet*; 112:739-758.

Qi L, Cornelis MC. Zhang C, van Dam RM and Hu FB. 2009. Genetic predisposition, Western dietary pattern, and the risk of type 2 diabetes in Men. *Am J Clin Nutr*, 89(5):1453-1458.

Quirk SE, Williams LJ, O'Neil A, Pasco JA, Jacka FN, Housden S, Berk M & Brennan SL. 2013. The Association between Diet Quality, Dietary Patterns and Depression in Adults. *Psychiatry* [Online], 13:175. [Accessed June 27th, 2013].

Raz I. 2009. Introduction to the Second World Congress, on Controversies to Consensus in Diabetes, Obesity and Hypertension (CODHy). *Diabetes Care* [Online], 32(Suppl 2): S149-S150. [Accessed June 27th, 2013].

Reasner CA & Goke B. 2002. Overcoming the barriers to effective glycemic control for type 2 diabetes. *British Journal of Diabetes & Vascular Disease*, 2:290.

Reddigan JI, Arden CI, Riddell MC & Kuk JL. 2010. Differences in the Association between Clinically Relevant Classifications of Glycemia Measures and All-cause and Cardiovascular Disease Mortality Risk. *J Diabetes Metab* [Online], 1:106. [Accessed November 01st, 2010].

Rizkallan S. 2010. Health implications of fructose consumption: A review of recent data. *Nutr. Metab (Lond)*, 7:82. [Accessed November 4th, 2010].

Robbins JM, Thatcher GE, Wabb DA & Valdmanis VG. 2008. *Diabetes care*, 31(4):655-660.

Roden M. 2012. Exercise in Type 2 diabetes: resist or to endure? *Diabetologia Clinical and Experimental Diabetes and Metabolism [Online]*, 55(5): 1273-82. [Accessed March 6th, 2012].

Rohlfing CL, Wiedmeer HM, Little RR, England JD, Tennil A, Goldstein DE. 2002. Defining the relationship between plasma glucose and HbA1c: analysis of glucose profiles and HbA1c in the Diabetes Control and Complications Trial. *Diabetes Care*, 25(2): 275-278.

Rossi M, Turati F, Lagiou P, Trichopoulos D, Augustin LS, Vecchia CL and Trichopoulos A. 2000. Mediterranean diet and glycaemic load in relation to incidence of type 2 diabetes: results from the Greek cohort of the population-based European Prospective Investigation into Cancer and Nutrition (EPIC). *Diabetologia*, 56(11):2405-2411. [Accessed July 9th, 2013].

Saami SE, Pietilainene K, Kantonen S, Rissanen A & Kaprio J. 2009. Association of smoking in Adolescence with Abdominal Obesity in Adulthood: A Follow-up Study of 5 Birth Cohorts of Finnish Twins. *Am J Public Health*, 99 (2): 348-354. [Accessed February, 2009].

Samieri C, Sun Q, Townsend MK, Chiuve SE, Okerere OI, Willett WC, Stampfer M & Grodstein F. 2013. The association between dietary patterns at midlife and health in aging: an observational study. *Ann Intern Med*, 159 (9): 584-591.

Satman I, Omer B, TutuncuY, Kalaca S, Gedik S, Dinccag N, Karsidag K, Genc S, Telci A, Canbaz B, Yilmaz T, Cakir B & Tuomilehto J. 2013. Twelve-year trends in the prevalence and risk factors of diabetes and prediabetes in Turkish adults. *Eur J Epidemiol.*, 28(2):169-180. [Accessed February 14th, 2013].

Segal I, Gagjee PP, Essop AR, Noormohamed AM. 1983. Lactase deficiency in the South African black population. *Am J Clin Nutr*, 38(6):901-905.

Seheri L. 2013. *The prevalence f overweight and obesity among adolescents in urban Maseru.* Msc dissertation. University of the Free State, [Online]. Available at: http://www.bnl.gov/isd/documents/30546.pdf [Accessed:26 March 2014].

Shrivastava SR, Shrivastava PS & Ramasamy J. 2013. Role of self-care in management of diabetes mellitus. *Journal of Diabetes & Metabolic Disorders*, 12:14. [Accessed March 5th, 2013].

Solomon TPJ, Haus JM, Kelly KR, Cook MD, Riccardi M, Rocco M, Kashyap SR, Barkoukis & Kirwan JP. 2009. Randomized trial on the effects of a 7-d low-glycemic diet and exercise intervention on insulin resistance in older obese humans. *Am J Clin Nutr*., 90(5):1222-1229. [Accessed September, 2009].

Sluik D, Boeing H, Motonen J, Pischon T, Kaaks R, Teucher B, Tjonneland A, Halkjaer J, Berentzen TL, Overvad K, Arriola L, Ardanaz E, Bendinelli B, Grioni S, Tumino R, Sacerdite C, Mattielo A, Spijkerman AMW, van der A DL, Beulens JW, van der Schouw YT, Nilsson PM, Hedblad B, Ronaldsson O, Franks PW & Nothlings U. 2011 Associations between general and abdominal adiposity and mortality in individuals with Diabetes Mellitus. *Am J Epidemiol*, 174 (1): 22-34.

Smuts CM & Wolmarans P. 2013. The Importance of the Quality or Type of fat in the diet: a food-based dietary guideline for South Africa. *S Afr J Nutr*, 26(3)(Supplement):S87-S99.

Steyn NP & Temple NJ. 2012. Evidence to support a food-based dietary guideline on sugar consumption in South Africa. *BMC Public Health*, 12:502. [Accessed October 4th, 2012].

Stiglitz J. 2010. Type 2 diabetes epidemic: a global education. *The Lancet*, 374:1654-1656.

Stuckler D, Basu S & Mckee M. 2010. Drivers of inequality in Millenium Development Goal Progress: A Statistical Analysis. *PLoS Med*, 7(3): e1000241.

Sun Q, Spiegelman D, van Dam RM, Holmes MD, Malik VS, Willett WC & Hu FB. 2010. White Rice, Brown Rice, and Risk of Type 2 Diabetes in US Men and Women. *Arch Intern Med*, 170(11): 961-969. [Accessed June 14th, 2010].

Taylor R. 2013. Type 2 Diabetes Etiology and reversibility. *Diabetes care*, 36:1047-1055.

Tuomi T, Santoro N, Caprio S, Cai M, Weng J & Groop L. 2013. The many faces of diabetes: a disease with increasing heterogeneity. *The Lancet*,. [Accessed December 3rd, 2013].

Unwin N, Gan D & Whiting D. 2010. IDF Diabetes Atlas: Providing evidence, raising awareness and promoting action. *Diabetes Res Clin Pract* [Online], 87 (1): 2-3. [Accessed March 6th, 2012].

US National Medical Association. 2009. Lactose intolerance and African-Americans: Implications for the consumption of appropriate intake levels of key nutrients. *J Natl Med Assoc*, 101: 1S-24S.

Van den Berg V, Okeyo AP, Dannhauser A & Nel M. 2012. Body weight, eating practices and nutritional knowledge amongst university nursing students, Eastern Cape, South Africa. *Afr J Prm Health Care Fam Med*, 4(1):1-9.

Van Dokkum W, Frolich W, Saltarch M & Gee J. 2008. The health effects of bioactive plant components in food: results and opinions of the EU COST 926 action. *Nutr Bull*, 33:133-139.

Wang G, Divall S, Radovick S, Paige D, Ning Y, Chen Z, Ji Y, Hong X, Walker SO, Caruso D, Pearson C, Wang MC, Zuckerman B, Cheng TL & Wang X. 2014. Preterm Birth and Random Plasma Insulin Levels at Birth and in Early Childhood. *JAMA*, 311(6): 587-596.

Wheeler ML, Daly A & Evert A. 2008. Choose Your Foods: Exchange Lists for Diabetes, 6th Edition. 2008: Description and Guidelines for Use. *J Am Diet Assoc*, 108:883-888.

Wheeler ML, Dunbar SA, Jaacks LM, Karmally W, Mayer-Davis E, Wylie-Rosett J & Yancy WS. 2012. Macronutrients, Food Groups, and Eating Patterns in the Management of Diabetes. A systematic review of the literature, 2010. *Diabetes Care*, 35(2):434-445.

Weickert MO. 2012. What Dietary Modification Best Improves Insulin Sensitivity and Why? *Clin Endocrinol*, 77(4):508-512.

Whincup PH, Nightingale CM, Owen CG, Rudnicka AR, Gibb I, Mckay CM, Donin AS, Salter N, George K & Cook DG. 2010. Early Emergence of Ethnic Differences in Type 2 Diabetes Precursors in the UK: The Child Heart and Health Study in England (CHASE Study). *PLoS Med*, 7(4):e1000263. [Accessed April 20th, 2010].

Whiting DR, Guariguata L, Weil C & Shaw J. 2011. IDF Diabetes Atlas: Global estimates of the prevalence of diabetes for 2011 and 2030. *Diabetes Research and Clinical Practice*, 94:311-321.

Wilde PJ. 2009. Eating for life: Designing Foods for Appetite Control. *J Diabetes Sci Technol*, 3(2): 366-370.

World Health Organization (WHO) Consultation. 2000. Obesity: Preventing and managing the global epidemic. Report of a WHO Consultation. WHO Technical Report series 894, Geneva. http://www.who.int/diabetes/publications/diagnosis-diabetes

World Health Organization. 2011. Global database on body mass index. [Online]. Available from: http://apps.who.int/bmi/index.jsp? Intropage =intro-3.html [Accessed 21st, 2011].

World Health Organization. 2004. Global strategy on diet, physical activity and health: www.who.int.hpr

Zimmet PZ, Magliaro DJ, Herman WH & Shaw JE. 2014. Diabetes a 21st challenge. *The Lancet Diabetes & Endocrinology*, 2(1): 56-64.

## Appendix A: Food Exchange lists, Portion sizes and Alcohol Equivalents.

| FOODS | 1 PORTION | FOODS | 1 PORTION |
|---|---|---|---|
| **STARCH/BREAD :** | | **MEAT:** | |
| Mabele/oats porridge: | ½ cup – 125g | **Lean meat & substitutes:** | |
| Papa | 1 heaped Tbsp | Lean beef (fat trimmed) | 30g |
| Pasta | ½ cup – 70g | Pork (lean ham) | 30g |
| Ready-to-eat cereals | ½ cup – 20g | Veal (chops & roasts) | 30g |
| Rice, white or brown | ½ cup – 70g | Poultry (chicken & turkey without skin) | 30g |
| Samp | ½ cup – 60g | Mutton (leg-fat trimmed) | 30g |
| Weetbix | 1 – 25g | Fish-hake, haddock, snoek | 30g |
| Provita | 3 – 20g | Tuna (canned in water) | ¼ cup – 35g |
| Popcorn | 1 ½ cups – 20g | Pilchards (canned in tomato sauce) | ¼ cup – 40g |
| | | Cheese (cottage, fat free and low-fat | ¼ cup – 50g |
| Beans, peas (dried, cooked) | ⅓ cup – 70g | parmesan) | 3 whites |
| Lentils (cooked) | ⅓ cup – 70g | **Other:** egg whites | |
| Baked beans | ¼ cup – 70g | | |
| | | **Medium fat meat & substitutes:** | |
| **Starchy vegetables:** | | Beef (mince, roasts, corned, steak) | 30g |
| Mealie, on cob | 1 cob – 80g | Pork (chops, roast, cutlets, ham) | 30g |
| Kernels (canned or frozen) | ½ cup – 95g | Veal (cutlet) | 30g |
| Peas, green (cooked) | ½ cup – 90g | Poultry (chicken & turkey with skin) | 30g |
| Mixed vegetables | ½ cup – 90g | Mutton-chops, fat trimmed lean stewing | 30g |
| Potato | 1 small – 90g | Fish – high fat fish (herring, butterfish, | 30g |
| Pumpkin, butternut, squash | ¾ cup – 170g | mackerel, eel) | |
| Sweet potato | ¼ cup – 80g | Tuna (canned in oil & drained) | ¼ cup – 35g |
| | | Cheese (low-fat hard cheeses) | 30g |
| **Bread:** | | **Other:** egg | 1 egg |
| Bread (white, brown) | 1 slice – 30g | Liver, heart, kidney | 30g |
| Bread rolls: - hamburger | ½ - 30g | | |
| - Hot dog | ½ - 30g | **High fat meat & substitutes:** | |
| Pita | ½ - 30g | Beef (fatty beef and sausages) | 30g |
| Raisin bread | 1 slice – 30g | Pork (spare ribs, sausages) | 30g |
| Rye, pumpernickel | 1 slice – 30g | Mutton (fatty mutton) | 30g |
| Scone | 1 small – 25g | Fish (any fried fish) | 30g |
| | | Cheese (cheddar, gouda, creamed cheese | ¼ cup – 50g |
| | | **Others:** luncheon meat, polony, vienna, | 30g |
| **MILK .** | | boerewors, frankfurter, Russians, salami | |
| **Skim milk:** | | | |
| Skim milk (fresh) | 1 cup/250ml | **Note: 30g = 1 matchbox size**. | |
| Skim milk powder | ¼ cup/25g | | |
| Plain skim milk yoghurt | 1 cup/250ml | **FAT :** | |
| Evaporated skim milk | ½ cup/125ml | **Unsaturated fats:** | |
| | | Avocado (mono) | ¼ /30g |
| **Low-fat milk:** | | Margarine (tub varieties) | 1 tsp/ 5g |
| Low fat milk 2% (fresh) | 1 cup/250ml | Margarine, lite | 1 ½ tsps/7g |
| Plain low fat yoghurt | 1 cup/250ml | Mayonnaise | 1 tsp/9g |
| Low fat yoghurt | 1 cup/250ml | Mayonnaise, reduced fat (trim) | 2 Tbsp/25g |
| Low fat maas | 1 cup/250ml | Nuts & seeds: - Almonds, dry roasted | 6 whole |
| | | -Cashews, dry roasted | 1 Tbsp |
| **Whole milk:** | | - Peanuts | 20 small/10 large |
| Full cream milk (fresh) | 1 cup/250ml | - Seeds | 1 Tbsp |
| Full cream milk powder | ¼ cup/30g | Oil | 1 tsp/5g |
| Evaporated whole milk | ½ cup/125ml | Olives | 10 small/5 large |
| Sour milk/maas | 1 cup/250ml | Salad cream | 1 tsp/5g |

| FOODS | 1 PORTION |
|---|---|
| **Saturated fats:** | |
| Butter | 1 tsp/5g |
| Bacon (crisp) | 1 rasher/8g |
| Coconut, desiccate | 1 Tbsp/8g |
| Cream, coffee | 2 Tbsp/30g |
| Light cream | 1 ½ Tbsp/20g |
| Heavy cream | 1 Tbsp/12g |
| Cream cheese | 1 Tbsp/15g |
| French dressing | 2 tsp/10g |
| Margarine (brick varieties) | 1 tsp/5g |
| **FRESH, FROZEN & UNSWEETENED CANNED FRUIT:** | |
| Apple (raw, small) | 1 apple/125g |
| Apple (canned or sauce) | ½ cup/125g |
| Apricots (raw) | 3-4 apricots/100g |
| Apricots (canned) | ½ cup/8 halves/125g |
| Banana (small) | 1 banana/65g |
| Berries (raw) | ¾ cup/115g |
| Berries (canned) | ⅓ cup/70g |
| Cherries (raw) | 12 cherries/85g |
| Cherries (canned) | ½ cup/85g |
| Figs (raw) | 2 figs/80g |
| Fruit cocktail (canned) | ½ cup/130g |
| Grapefruit (raw) | ½ grapefruit/160g |
| Grapefruit (canned) | ¾ cup/160g |
| Grapes (small) | 15 grapes/85g |
| Guava (raw) | 2 guavas/120g |
| Kiwi fruit | 1 kiwifruit/90g |
| Mango (raw, small) | ½ mango/95g |
| Melon (raw, cubed) | 1 cup/170g |
| Naartjies (raw) | 2 naartjies/135g |
| Nectarine (raw) | 1 nectarine/100g |
| Orange (raw) | 1 orange/125g |
| Paw paw (raw) | 1 cup/150g |
| Peach (raw) | 1 peach/140g |
| Peach (canned) | ½ cup/2 halves/140g |
| Pear (raw) | 1 pear/100g |
| Pear (canned) | ½ cup/2 halves/120g |
| Pineapple (raw, cubed) | ½ cup/100g |
| Pineapple (canned) | ¾ cup/120g |
| Plums (raw) | 2 plums/110g |
| Strawberries (raw, whole) | 1 ¼ cups/200g |
| Watermelon (raw, cubed) | 1 ¼ cups/200g |
| **Dried fruit** | |
| Apples | 5 rings/25g |
| Apricots | 3 halves/25g |
| Dates | 4 dates/22g |
| Figs | 1 fig (large)/24g |
| Peach | 2 halves/25g |
| Pears | 1 half (large)/23g |
| Prunes | 2-3 prunes/25g |
| Raisins | 1 heapedTbsp/20g |
| **FRUIT JUICE -UNSWEETENED:** | |
| Apple juice | ½ cup/125 ml |
| Apricot juice | ½ cup/125 ml |
| Grape juice | 1/3 cup/85 ml |
| Grapefruit juice | ½ cup/125 ml |
| Guava juice | ½ cup/125 ml |
| Orange juice | ½ cup/125 ml |
| Pear juice | 1/3 cup/85 ml |
| Peach juice | 1/3 cup/85ml |

**VEGETABLE :**

**One portion is:**

- ½ cup (125ml) cooked vegetables.
- ½ cup (125ml) vegetable juice.
- 1cup (250ml) raw vegetables.

Asparagus
Beans (green)
Bean sprouts
Beetroot
Brussels sprouts
Cabbage, cooked
Carrots, cooked
Carrots, raw
Cauliflower
Turnips
Tomato (1 large)
Vegetable juice
Baby marrow, cooked
Eggplant/brinjal
Gem squash
Green pepper
Greens /moroho, cooked
Leeks
Mushrooms, cooked
Onions
Peapods
Spinach, cooked

**NOTE:** starchy vegetables such as corn/maize, pumpkin, butternut, peas and potatoes are found on the starch/bread list.

**FREE FOOD :**

A free food is any food or drink that contains less than 84 kJ (20 cal) per serving. Those items that have no serving size specified may be eaten freely. .

**Drinks:**

Bouillon or broth, without fat
Carbonated drinks, sugar free
Cocoa powder, unsweetened (1 Tbsp) coffee/tea
Drink mixes/squashes, sugar free
Soda water

**Vegetables (raw, 1 cup):**

Cabbage
Mushrooms
Cucumber
Baby marrow
Celery
Radishes

**Salad greens:**

Endive
Lettuce
Spinach

**Sweet substitutes:**

Candy, hard, sugar free
Gum, sugar free
Gelatin, sugar free
Jelly, sugar free
Non- nutritive sugar substitutes

**Alcohol Equivalents:**

| | |
|---|---|
| 45ml distilled beverage | 2 fats |
| 120ml red/rose/dry wine | 2 fats |
| 120ml sweet wine/sherry | ⅓ bread and 2 fats |
| 360ml beer | 1 bread and 2 fats |

**Appendix B: Patients' Socio-demographic Questionnaire.**

**Instructions:**

**Write an appropriate number in the corresponding block(s) or on the space provided.**

| | | |
|---|---|---|
| **Interview date** | ☐☐☐☐☐☐ | 01-06 |
| **Patient No.** | ☐☐☐ | 07-09 |
| **Date of birth** | ☐☐☐☐☐☐ | 10-15 |
| **Gender**<br>1. Male<br>2. Female | ☐ | 16 |
| **Residential address**_____ | ☐ | 17 |
| **Marital Status**<br>1. Married<br>2. Single<br>3. Divorced<br>4. Separated<br>5. Widowed | ☐ | 18 |
| **Current working conditions**<br>1. Employed<br>2. Unemployed<br>3. Pensioner<br>4. Housewife<br>5. Other (specify)_____ | ☐ | 19 |
| **Level of education**<br>1. Tertiary<br>2. College<br>3. High school<br>4. Primary school<br>5. None | ☐ | 20 |
| **Net income**<br>1. M0.300 - M1,500<br>2. M1,500 - M2,700<br>3. M2,700 - M3,900<br>4. M3,900 - M4,100 | ☐ | 21 |

5. M4,100 - M5,300

**Dependents** [ ] 22
1. 01 -0 2
2. 03 – 04
3. 05 – 06
4. 07 – 08
5. 09 – 10

**Anthropometrics:**

| | | |
|---|---|---|
| Weight (kg) | [ ][ ][ ].[ ] | 23-27 |
| Height (m) | [ ].[ ][ ] | 28-31 |
| Waist circumference (cm) | [ ][ ][ ].[ ] | 32-36 |
| Hip circumference | [ ][ ][ ].[ ] | 37-41 |

**Biochemical indices:**

| | | |
|---|---|---|
| HbA1c: | [ ][ ].[ ] | 42-45 |
| Fasting blood glucose | [ ][ ].[ ] | 46-49 |
| Total serum cholesterol | [ ][ ].[ ] | 50-53 |
| Triglycerides | [ ][ ].[ ] | 54-57 |

**Cormobid conditions, 1 = Yes or 2 = No**

| | | |
|---|---|---|
| 1. Obesity | [ ] | 58 |
| 2. Hypertension | [ ] | 59 |
| 3. Dyslipidemia | [ ] | 60 |
| 4. Retinopathy | [ ] | 61 |
| 5. Neuropathy | [ ] | 62 |
| 6. Nephropathy | [ ] | 63 |

**Duration of DM _____** [ ][ ] 64-65

**Treatment/medication** [ ] 66
1. Dietary
2. Oral agents
3. Insulin
4. Combination with insulin
5. None of the above

**Thank you for your participation.**

**Khomeletso B: Lipotso tse amanang le bophelo ka kakaretso.**

**Litaelo:** Ngola nomoro e nepahetseng ka mabokoseng kapa sebakeng se siiloeng manapana le potso.

| | | |
|---|---|---|
| **Letsatsi la lipotso** | ☐☐☐☐☐☐ | 01-06 |
| **Nomoro ea mokuli** | ☐☐☐ | 07-09 |
| **Letsatsi la tsoalo** | ☐☐☐☐☐☐ | 10-15 |
| **Botona kapa bots'ehali**<br>1. Motona<br>2. Mots'ehali | ☐ | 16 |
| **Bolulo _____** | ☐ | 17 |
| **Boemo ba lenyalo**<br>1. Ke na le molekane<br>2. Ha ke na molekane<br>3. Karohano ea lenyalo.<br>4. Karohano e seng ea molao.<br>5. Mohlolohali | ☐ | 18 |
| **Boemo ba ts'ebetso**<br>1. Ke hiriloe<br>2. Ha ke sebetse<br>3. Ke phomolong<br>4. Ke etsa mosebetsi oa lelapa.<br>5. Mesebetsi e meng (E bolele)_____ | ☐ | 19 |
| **Boemo ba thuto**<br>1. Unifesiti<br>2. Kh'oleje<br>3. Thuto e phahameng<br>4. Thuto ea mathomo<br>5. Ha ke ea kena sekolo | ☐ | 20 |
| **Chelete e kenang ka lapeng.**<br>1. M0.300 - M1,500<br>2. M1,500 - M2,700<br>3. M2,700 - M3,900<br>4. M3,900 - M4,100<br>5. M4,100 - M5,300 | ☐ | 21 |

**Baphelisuoa** | 22

1. 01 -0 2
2. 03 – 04
3. 05 – 06
4. 07 – 08
5. 09 – 10

**Boima ba 'mele**

| | | |
|---|---|---|
| 1. Boima (kg) | ☐☐☐.☐ | 23-27 |
| 2. Botelele (m) | ☐.☐☐☐ | 28-31 |
| 3. Bophara be letheka (cm) | ☐☐☐.☐ | 32-36 |
| 4. Bophara ba liqholo (cm) | ☐☐☐.☐ | 37-41 |

**Liphetho tsa litlhatlhobo tsa mali**

| | | |
|---|---|---|
| 1. HbA1c | ☐☐.☐ | 42-45 |
| 2. Boemo be tsoekere pele ho lijo | ☐☐.☐ | 46-49 |
| 3. Mafura a bitsoang cholesterol | ☐☐.☐ | 50-53 |
| 4. Mafura a bitsoang triglycerides | ☐☐.☐ | 54-57 |

**Mafu a tsamaellanang le lefu la tsoekere a teng.**

1 = Ee 2 = Chee

| | | |
|---|---|---|
| 1. 'Mele o moholo | ☐ | 58 |
| 2. Phallo e phahameng ea mali | ☐ | 59 |
| 3. Mafura a mangata maling | ☐ | 60 |
| 4. Bofofu kapa bothata ba pono | ☐ | 61 |
| 5. Methapo kutlo e sa sebetseng hantle | ☐ | 62 |
| 6. Liphieo tse sa sebetseng hantle | ☐ | 63 |

**Ke nako e kae u ena le lefu la tsoekere _____** | ☐☐ | 64-65

**Mofuta oa phekolo kapa litlhare tseo u Lisebelisang** | ☐ | 66

1. Lijo feela
2. Lipilisi
3. Ente
4. Lipilisi le ente
5. ha hona letho

**Kea leboha.**

**Appendix C: Adapted 24-Hour Recall and Food Frequency Questionnaires.**

**Please indicate the meals, food type, preparation methods, quantity and time eaten.**

| Meals, preparation methods, time eaten. | Quantity/ Portions | Bread & Grains | Fruit | Vegs. | Milk & Prod. | Meat & Alter. | Fats /oil & Sweets |
|---|---|---|---|---|---|---|---|
| **Breakfast:** | | | | | | | |
| …… | …… | …… | …… | …… | ……. | …….. | ……... |
| …… | …… | …… | …… | …… | ……. | …….. | ……... |
| …… | …… | …… | …… | …… | ……. | …….. | ……... |
| …… | …… | …… | …… | …… | ……. | …….. | ……... |
| …… | …… | …… | …… | …… | ……. | …….. | ……... |
| …… | …… | …… | …… | …… | ……. | …….. | ……... |
| **Mid-morning snack:** | | | | | | | |
| …… | ……..... | …… | …… | …… | ……. | ……... | ……... |
| …… | …… | …… | …… | …… | ……. | ……... | ……... |
| …… | …… | …… | …… | …… | ……. | ……... | ……... |
| …… | …… | …… | …… | …… | ……. | ……... | ……... |
| …… | …… | …… | …… | …… | ……. | ……... | ……... |
| **Lunch:** | | | | | | | |
| …… | …… | …… | …… | …… | …… | ……... | ……... |
| …… | …… | …… | …… | …… | ……. | ……... | ……... |
| …… | …… | …… | …… | …… | ……. | ……... | ……... |
| …… | …… | …… | …… | …… | ……. | ……... | ……... |
| …… | …… | …… | …… | …… | ……. | ……... | ……... |
| …… | …… | …… | …… | …… | ……. | ……... | ……... |
| **Mid-afternoon snack:** | | | | | | | |
| …… | …… | …… | …… | …… | ……. | ……... | ……... |
| …… | …… | …… | …… | …… | ……. | ……... | ……... |
| …… | …… | …… | …… | …… | ……. | ……... | ……... |
| …… | …… | …… | …… | …… | ……. | ……... | ……... |
| …… | …… | …… | …… | …… | …….. | ……... | ……... |
| **Supper:** | | | | | | | |
| …… | …… | …… | …… | …… | ……. | ……... | ……... |
| …… | …… | …… | …… | …… | ……. | ……... | ……... |
| …… | …… | …… | …… | …… | ……. | ……... | ……... |
| …… | …… | …… | …… | …… | ……. | ……... | ……... |
| …… | …… | …… | …… | …… | ……. | ……... | ……... |
| …… | …… | …… | …… | …… | …….. | ……... | ……... |
| **Mid-night snack:** | | | | | | | |
| …… | …… | …… | …… | …… | ……. | ……... | ……... |
| …… | …… | …… | …… | …… | ……. | ……... | ……... |
| …… | …… | …… | …… | …… | ……. | ……... | ……... |
| …… | …… | …… | …… | …… | ……. | ……... | ……... |
| …… | …… | …… | ……... | …… | ……. | ……... | ……... |
| **Total** | | | | | | | |

**Instruction:**

**Indicate the frequency that the food is eaten.**

| **Food Type** | /day | | /Week | | /Month | | |
|---|---|---|---|---|---|---|---|
| **Bread and cereals** | | | | | | | |
| Mabele porridge | | | | | | | 01-06 |
| Oats porridge | | | | | | | 07-12 |
| Sour porridge/Motoho | | | | | | | 13-18 |
| Weetbix | | | | | | | 19-24 |
| Cornflakes | | | | | | | 25-30 |
| All bran | | | | | | | 31-36 |
| Muesli | | | | | | | 37-42 |
| Pronutro | | | | | | | 43-48 |
| Morvite | | | | | | | 49-54 |
| White bread | | | | | | | 55-60 |
| Brown bread | | | | | | | 61-66 |
| Provita | | | | | | | 67-72 |
| Pasta (macaroni, spaghetti) | | | | | | | 73-78 |
| Maize-meal Pap | | | | | | | 01-06 |
| Potatoes | | | | | | | 07-12 |
| Rice /mealie rice | | | | | | | 13-18 |
| Samp | | | | | | | 19-24 |
| Dried beans/Lentils/Peas | | | | | | | 25-30 |
| Baked beans | | | | | | | 31-36 |
| Corn on cob | | | | | | | 37-42 |
| Mixed vegetables | | | | | | | 43-48 |
| Green peas | | | | | | | 49-54 |
| Popcorn | | | | | | | 55-60 |
| Butternut/squash/pumpkin | | | | | | | 61-66 |
| **Milk:** | | | | | | | |
| Full cream milk | | | | | | | 67-72 |
| Skimmed milk | | | | | | | 73-78 |
| Low-fat milk | | | | | | | 01-06 |
| Plain low-fat yoghurt | | | | | | | 07-12 |
| Flavoured yoghurt | | | | | | | 13-18 |
| Sour milk/maas | | | | | | | 19-24 |
| Goat's milk | | | | | | | 25-30 |

| | /day | | /week | | /month | | |
|---|---|---|---|---|---|---|---|
| **Fruit:** | | | | | | | |
| Fresh fruits | | | | | | | 31-36 |
| Dried fruits | | | | | | | 37-42 |
| Fruit juice | | | | | | | 43-48 |
| Canned fruits | | | | | | | 49-54 |
| Wild fruits | | | | | | | 55-60 |
| **Vegetables :** | | | | | | | |
| Spinach | | | | | | | 61-66 |
| Lepu | | | | | | | 67-72 |
| Sepaile | | | | | | | 73-78 |
| Radish/Rapa | | | | | | | 01-06 |
| Wild vegetables | | | | | | | 07-12 |
| Cabbage | | | | | | | 13-18 |
| Green beans | | | | | | | 19-24 |
| Cauliflower | | | | | | | 25-30 |
| Broccoli | | | | | | | 31-36 |
| Mushrooms | | | | | | | 37-42 |
| Carrots | | | | | | | 43-48 |
| Lettuce | | | | | | | 49-54 |
| Cucumber | | | | | | | 55-60 |
| Frozen vegetables | | | | | | | 61-66 |
| Green pepper | | | | | | | 67-72 |
| Green onions/onions | | | | | | | 73-78 |
| Tomato | | | | | | | 01-06 |
| **Fat:** | | | | | | | |
| Canola oil | | | | | | | 07-12 |
| Olive oil | | | | | | | 13-18 |
| Sunflower oil | | | | | | | 19-24 |
| Margarine | | | | | | | 25-30 |
| Butter | | | | | | | 31-36 |
| Peanut butter | | | | | | | 37-42 |
| Mayonnaise/salad dressing | | | | | | | 42-48 |
| Nuts (almonds, peanuts) | | | | | | | 49-54 |
| Seeds | | | | | | | 55-60 |

| | | | | | | | |
|---|---|---|---|---|---|---|---|
| Avocado | | | | | | | 61-66 |
| Cream | | | | | | | 67-72 |
| Bacon | | | | | | | 73-78 |
| Creamer | | | | | | | 01-06 |
| Shortening/Lard (Holsum) | | | | | | | 07-12 |
| **Meat and meat alternatives:** | | | | | | | |
| Beef | | | | | | | 13-18 |
| Lamb | | | | | | | 19-24 |
| Pork | | | | | | | 25-30 |
| Chicken | | | | | | | 31-36 |
| White fish (Hake) | | | | | | | 37-42 |
| Tinned fish (pilchards, tuna) | | | | | | | 43-48 |
| Cheese | | | | | | | 49-54 |
| Eggs | | | | | | | 55-60 |
| Russian / polony/Vienna/ham | | | | | | | 61-66 |
| Peanut butter | | | | | | | 67-72 |
| Dried beans/peas/lentils | | | | | | | 73-78 |
| Texturerized protein (imana) | | | | | | | 01-06 |
| Offals – kidney, liver, heart | | | | | | | 07-12 |
| Soy milk | | | | | | | 13-18 |
| **Sweet/sugar :** | | | | | | | |
| Sugar | | | | | | | 19-24 |
| Syrup | | | | | | | 25-30 |
| Honey | | | | | | | 31-36 |
| Jam | | | | | | | 37-42 |
| Sweets | | | | | | | 43-48 |
| Chocolate | | | | | | | 49-54 |
| Desserts | | | | | | | 55-60 |
| Cakes | | | | | | | 61-66 |
| Biscuits | | | | | | | 67-72 |
| Synthetic sweeteners | | | | | | | 73-78 |
| **Drinks :** | | | | | | | |
| Soft drink | | | | | | | 01-06 |
| Cordials | | | | | | | 07-12 |
| Fruit juice | | | | | | | 13-18 |

| | | | | | | | |
|---|---|---|---|---|---|---|---|
| Hot chocolate | | | | | | | 19-24 |
| Coffee | | | | | | | 25-30 |
| Tea | | | | | | | 31-36 |
| Milo | | | | | | | 37-42 |
| Green tea | | | | | | | 43-48 |
| Alcohol | | | | | | | 49-54 |

**Miscellaneous:**

| | | | | | | | |
|---|---|---|---|---|---|---|---|
| Diabetic products | | | | | | | 55-60 |
| Fast foods | | | | | | | 61-66 |
| Vinegar | | | | | | | 67-72 |
| Spreads (cheese, fish) | | | | | | | 73-78 |
| Salt | | | | | | | 01-06 |
| Aromat | | | | | | | 07-12 |
| Pepper | | | | | | | 13-18 |
| Spices (chicken, barbecue) | | | | | | | 19-24 |
| Herbs (garlic, ginger) | | | | | | | 25-30 |
| Stock cubes (beef stock) | | | | | | | 31-36 |
| Packet soups | | | | | | | 37-42 |
| Supplements | | | | | | | 43-48 |
| Bran | | | | | | | 49-54 |
| Simbas | | | | | | | 55-60 |

**Thank you for your participation!!**

**Appendix D: Adapted International Physical Activity (Long form)Questionnaire.**

**Instructions: Please fill in the correct number in the block (s) or space provided.**

**Part 1: Job-related physical activity.**

**1. Do you currently have a job or do any unpaid work outside your home?** ☐ 01

1. Yes
2. No

**2. During the last 7 days, on how many days did you do vigorous physical activities, like heavy lifting, digging, heavy construction, or climbing up stairs as part of your work? Think about only those physical activities that you did at least 10 minutes at a time.** ☐ 02

1. _____days per week
2. _____no vigorous job-related physical activity

**3. How much times did you usually spend on one of those days doing vigorous physical activities as part of your work?** ☐ 03

1. _____hours per day
2. _____minutes per day

**4. Again, think about only those physical activities that you did at least 10 minutes at a time. During the last 7 days on how many days did you do moderate physical activities like carrying light loads as part of your work? Please do not include walking.** ☐ 04

1. _____days per week
2. _____no moderate job-related physical activity

**5. How much time did you usually spend on one of those days doing moderate physical activities as part of your work?** ☐ 05

1. _____hours per day
2. _____minutes per day

**6. During the last 7 days, on how many days did you walk for at least 10 minutes at a time as part of your work? Please do not count any walking you did to travel to or from work.** ☐ 06

1._____days per week
2. _____no job-related work

**7. How much time did you usually spend on one of those days walking as part of your work?**

1._____hours per day

2._____minutes per day

☐ 07

**Part 2: Transportation physical activity**

**1. During the last 7 days, on how many days did you travel in a motor vehicle like a taxi or car?**

1._____days per week

2. _____no travelling in a motor vehicle

☐ 08

**2. How much time did you usually spend on one of those days travelling in a taxi, bus, car or other kind of motor vehicle?**

1. _____hours per day

2. _____minutes per day

☐ 09

**3. During the last 7 days, on how many days did you bicycle for at least 10 minutes at a time to go from place to place.**

1. _____days per week

2. _____no bicycling from place to place

☐ 10

**4. How much time did you usually spend on one of those days to to bicycle from place to place?**

1. _____hours per day

2._____minutes per day

☐ 11

**5. During the last 7 days, on how many days did you walk for at least 10 minutes at a time go from place to place?**

1. _____hours per day

2. _____minutes per day

☐ 12

**6. How much time did you usually spend on one of those days walking from place to place?**

1. _____hours per day

2._____minutes per day

☐ 13

**Part 3: Housework, house maintenance, and caring for family.**

**1. Think about only those physical activities that you did for at least 10 minutes at a time. During the last 7 days, did you do 7 days, did vigorous physical activities like heavy lifting, chopping wood or digging in the garden or yard?** ☐ 14

1._____days per week
2. _____no vigorous activity in garden or yard.

**2. How much time did you usually spend on one of those days doing vigorous physical activities in the garden or doing yard?** ☐ 15

1. _____hours per day
2. _____Minutes per day

**3. Again, think about only those physical activities that you did for at least 10 minutes at a time. During the last 7 days on how days did you do moderate physical activities like carrying light loads sweeping, washing windows, raking in the garden or yard?** ☐ 16

1. _____days per week
2._____no moderate activity in garden or yard

**4. How much time did you usually spend on one of those days doing moderate physical activities in the garden or yard?** ☐ 17

1._____hours per day
2._____minutes per day

**5. Once again, think about only those physical activities that you did for at least 10 minutes at a time. During the last 7 days, how many days did you do moderate activities like carrying light loads, washing windows, scrubbing floors and sweeping in your home?** ☐ 18

1._____days per week
2._____no moderate activity inside home

**6. How much time did you usually spend on one of those doing moderate physical activities inside your home?** ☐ 19

1._____hours per day
2._____minutes per day

**Part 4: Recreation, sport, and leisure time physical activity.**

**1. Not counting any walking you have already mentioned, during the last 7 days, on how many days did you walk for at least 10 minutes at a time in your leisure time?**

1. _____days per week
2. _____no walking in leisure time

☐ 20

**2. How much time did you usually spend on one of those days walking in your leisure time?**

1. _____hours per day
2. _____minutes per day

☐ 21

**3. Think about only those physical activities that you did for at least 10 minutes at a time. During the last 7 days, on how many days did you do vigorous physical activities like aerobics, running,**
**fast bicycling, or fast swimming in your leisure time?**

1._____days per week
2. _____no vigorous activities in leisure time

☐ 22

**4. How much time did you usually spend on one of those days doing vigorous physical activities in your leisure time?**

1. _____hours per day
2._____minutes per day

☐ 23

**5. Again, think about only those physical activities that you did for at least 10 minutes at a time. During the last 7 days, on how many days did you do moderate physical activities like bicycling at a regular pace, swimming at a regular pace and doubles in your leisure time.**

1. _____days per week
2._____no moderate activity in leisure time

☐ 24

**6. How much time did you usually spend on one of those days doing moderate physical activities in your leisure time?**

1. _____hours per day
2._____minutes per day

☐ 25

**Part 5: Time spend sitting.**

**1. During the last 7 days, how much time did you usually spend sitting on a weekday?** ☐ 26

1. _____hours per day
2._____minutes per day

**2. During the last 7 days, how much time did you usually spend sitting on a weekend day?** ☐ 27

1. _____hours per day
2. _____minutes per day

**Part 6: Alcohol intake**

**1. Do you currently use alcohol?** ☐ 28

1.No
2.Yes

**2. What form of alcohol do you use regularly use?** ☐ 29

1. Spirits (rum, whisky, gin, vodka)
2.Wine
3. Cider
4. Beer
5. Homebrew

**3. How often do you use alcohol?** ☐ 30

1. Daily
2. Weekly
3. Monthly
4.Occasional

**4. How many drinks do you normally take per session?** ☐☐ 31-32

_____

**Part 7: Tobacco use**

**1. Do you currently use any tobacco?** ☐ 33

1. No
2. Yes

**2. What form of tobacco do you use?** ☐ 34

1. Cigarette
2. Snuff
3. Pipe

**3. How many times per day do you snuff?** ☐☐ 35-36

_____

**4. How many cigarettes do you use per day?** ☐☐ 37-38

_____

**5. How many pipes do you use per day?** ☐☐ 39-40

_____

**6. When did you stop smoking?** ☐ 41

1. 1 month
2. 3 months
3. 6 months
4. 1 year
5. Other (specify) _____

**Thank you for your participation!!!**

**Khomeletso D: Foromo (e telele) ea lipotso tsa boikoetliso le mesebetsi ea lapeng.**

**Litaelo: Ngola nomoro e nepahetseng ka mabokoseng kapa sebakeng se siiloeng manapana le potso.**

**Karolo ea pele: Boikoetliso bo amanang le ho sebetsa**

**1.Na u na le mesebetsi kapa o etsa mosebetsi oa lapeng o sa pataleng?** ☐ 01
1 = Ee
2 = Chee

**2. Matsatsing a supileng a fetileng, ke matsatsi a makae ao o ileng oa ikoetlisa/sebetsa ka matla joaloka ho phahamisa lintho tse boima, ho cheka, ho haha, ho hloa litepisi e le karolo ea mosebetsi oa hau. Nahana ka boikoetliso boo nkileng metsotso e leshome feela ka nako?** ☐ 02
1._____ matsatsi ka beke.
2. _____ ha hona mesebetsi e matla oe ke e entseng.

**3. Ke nako e kae eo u e nkileng matsatsing ao u etsa mesebetsi ka matla?** ☐ 03
1._____ Lihora ka letsatsi
2. _____ metsotso ka letsatsi

**4. Hape hopola mesebetsi eo nkileng metsotso e leshome ka nako. Matsatsing a supileng a fetileng, ke matsatsi a makae oa u ikoetlisitseng ka tekanyo, joaloka ho nka lintho tse bobebe e le mosebetsi oa hau? Se ke oa kopanya le ho tsamaea.** ☐ 04
1._____ matsatsi ka beke.
2._____ ha hona mesebetsi/boikoetliso bo entsoeng.

**5. U nka nako e kae ho ikoetlisa ka tekanyo, e le karolo ea mosebetsi oa hau?** ☐ 05
1._____ Lihora ka letsatsi.
2._____ metsotso ka letsatsi.

**6. Matsatsing a supileng a fetileng, ke matsatsi a makae ao u ileng oa tsamaea bonytane metsotso e leshome ka nako e le karolo ea mosebetsi?. Se ke oa bala ho tsamaea u e ea ebile u** ☐ 06

**khutla mosebetsing.**
1 _____ matsatsi ka beke.
2._____ ha hona mesebetsi o entsoeng.

**7. Ke nako e kae eo u e nkileng matsatsing ao o tsamaea e le karolo ea mosebetsi oa hau?** 07
1._____ lihora ka letsatsi
2._____ metsotso ka letsatsi.

**<u>Karolo ea bobeli: Boikoetliso ka mokhoa oa lipalamo.</u>**

**1. Matsatsing a supileng a fetileng, ke a makae ao u palameng koloi joaloka taxi?** 08
1 _____ matsatsi ka beke
2._____ ha ke es'o tsamae ka koloi.

**2. Ke nako e kae matsatsing ao u tsamaea ka taxi, bese, kapa mofuta o mong oa koloi?** 09
1. _____lihora ka letsatsi
2._____ metsotso ka letsatsi

**3. Matsatsing a supileng a fetileng, ke a makae ao o ileng oa palama bicycle metsotso e kabang leshome ka nako ho tloha sebakeng se itseng ho ea ho se seng?** 10
1._____ matsatsi ka beke
2._____ bicycle ha es'o palamoe ho tloha sebakeng ho ea ho se seng.

**4. Ke nako e kae eo u e nkileng u tsamaea ka bicycle ho tloha sebakeng se seng ho ea ho se seng?** 11
1. _____ lihora ka letsatsi
2. _____ metsotso ka letsatsi

**5. Matsatsing a supileng a fetileng, ke matsatsi a makae ao u ileng oa tsamaea metsotso e leshome ka nako ho tloha sebakeng se seng ho ea ho se seng?** 12
1._____ lihora ka letsatsi
2._____ metsotso ka letsatsi

**6. Ke nako e kae eo u e nkileng matsatsing ao u tsamaea ho** 13

**tloha sebakeng se itseng ho ea ho se seng?**
1._____lihora ka letsatsi
2._____metsotso ka letsatsi

**<u>Karolo ea boraro: Mesebetsi ea lapeng, le ho hlokomela lelapa.</u>**

**1. Nahana ka boikoetliso boo u bo entseng bonyane metsotso e leshome ka nako. Matsatsing a supileng a fetileng, na o kile oa etsa mesebetsi e matla joaloka ho nka lintho tse boima, ho ratha patsi kapa ho lema jareteng?** ☐ 14
1. _____ matsatsi ka beke
2. _____ha hona mosebetso o ke oentseng

**2. Ke nako e kae eo u e nkang matsatsing ao u etsa mesebetsi e matla jareteng?** ☐ 15
1._____ lihora ka letsatsi
2. _____ metsotso ka letsatsi

**3. Hopola hape mesebetsi kapa boikoetliso boo u bo nkileng metsotso e leshome ka nako. Matsatsing a supileng a fetileng ke a makae ao u entseng mesebetsi kapa boikoetliso bo bohareng joaloka ho nka lintho tse boima, ho fiela, ho hlatsoa lifensetere, ho haraka jareteng?** ☐ 16
1. _____ Matsatsi ka beke
2. _____ ha hona mesebetsi e bohareng e entsoeng.

**4. Ke nako e kae eo u e nkileng matsatsing ao u etsa mesebetsi kapa boikoetliso ka jareteng?** ☐ 17
1. _____ lihora ka letsatsi
2. _____ metsotso ka letsatsi

**5. Hopola hape mesebetsi kapa boikoetliso boo u bo entseng bonyane metsotso e leshome ka nako. Nakong ea matsatsi a supileng, ke matsatsi a makae ao u entseng mesebetsi kapa boikoetliso bo mahareng joaloka ho hlatsoa lifensetere, ho koropa fats'e le ho fiela lapeng?** ☐ 18
1. _____ Matsatsi ka beke
2. _____ ha hona mesebetsi e bohareng e entsoeng.

**6. Ke nako e kae eo u tloaetseng ho e nka ho etsa mesebetsi kapa boikoetliso bo bohareng?** 19

1. _____ Lihora ka beke
2. _____ metsotso ka letsatsi

**Karolo ea Bone: Boikoetliso ba ho ithapolla, boithabiso le lipapali.**

**1. U sa bale ho tsamaea ho seng u ho boletse, matsatsing a supileng a fetileng, ke a makae ao u tsamaileng bonyane metsotso e leshome ka nako ea hau ea phomolo?** 20

1. _____ matsatsi ka beke
2. _____ ha hona ho tsamaea ka nako ea phomolo

**2. Ke nako e kae eo u e nkang matsatsing ao u tsamaea ka nako ea hau ea phomolo?** 21

1. _____ lihora ka nako
2. _____ metsotso

**3. Hopola feela mesebetsi kapa boikoetliso boo u bo entseng bonyane metsotso e leshome ka nako. Matsatsing a supileng a fetileng, ke matsatsi a makae ao u entseng mesebetsi kapa boikoetliso bo matla joaloka li-aerobics, ho matha, ho palama bicycle ka pele, kapa ho sesa nakong ea hao ea phomolo?** 22

1. _____matsatsi ka beke
2. _____ ha hona mesebetsi kapa boithapollo bo matla ka nako ea phomolo?

**4. Ke nako e kae eo u tloaetseng ho e nka matsatsing ao u etsa mesebetsi ka matla ka nako ea phomolo?** 23

1._____ lihora ka letsatsi
2. _____ metsotso ka letsatsi

**5. Hape hopola ka mesebetsi kapa boikoetliso boo u bo entseng bonyane metsotso e leshome ka nako. Matsatsing a supileng a fetileng, ke a makae ao u entseng mesebetsi kapa boikoetliso ka ho palama bicycle ka lebelo le bohareng, ho sesa ka sekhahla se bohareng, ho bapala tenese ka nako ea hau ea ho phomolo?** 24

1. _____matsatsi ka beke

2. _____ ha hona mesebetsi kapa boikoetliso bo entsoeng ka nako ea phomolo?

**6. Ke nako e kae eo u e nkileng matsatsing ao u etsa mesebetsi kapa boikoetliso bo bohareng ka nako ea ho phomolo?** 25
1. _____ lihora ka letsatsi
2. _____ metsotso ka letsatsi

**Karolo ea Bohlano: Nako eo u entseng u lutse.**

**1. Matsatsing a supileng a fetileng, ke nako e kae eo u tloaetseng ho e nka u lutse feela mafelong a beke?** 26
1. _____ lihora ka letsatsi
2._____ metsotso ka letsatsi

**2. Matsatsing a supileng a fetileng, ke nako e kae eo u tloaetseng ho e nka u lutse feela mafelong a beke?** 27
1. _____lihora ka beke
2 _____ metsotso ka letsatsi

**Karolo ea Bots'elela: Ho sebelisa joala.**

**1. Na u sebelisa joala?** 28
1. Ee
2. Chee

**2. Ke mofuta o feng oa joala oo u tlaoetseng ho o sebelisa?** 29
1. Sepiriti (rum, whisky, gin, vodka)
2. Veine
3. Cider
4. Biri
5. Joala ba Sesotho

**3. U sebelisa joala joang?** 30
1. Letsatsi le leng le leng
2. Mafelo a beke feela
3. Mafelo a khoeli feela
4. Hoane le hoane (ka mekete).

**4. U sebelisa joala bo bokae ka nako? _____** | | | 31-32

**Karolo ea Bosupa: Ho sebelisa koae.**

**1. Na u sebelisa koae?** | | 33
1. Ee
2. Chee
3. Ke tlohetse

**2. U sebelisa koae e feng?** | | 34
1. Cigarette
2. Senifi
3. Peipi

**3. U sebelisa senifi ha kae ka letsatsi?_____** | | | 35-36

**4. U sebelisa cigarette tse kae ka letsatsi?_____** | | | 37-38

**5. U sebelisa peipi ha kae ka letsatsi?_____** | | | 39-40

**6. U tlohetse ho tsuba neng?** | | 41
1. Khoeli e fetileng
2. Khoeli tse tharo tse fetileng
3. Khoeli tse tseletseng tse fetileng
4. Selemo se fetileng
5. Tse ling (hlalosa)_____

**Ke a leboha!!!**

**Appendix E: Barriers that may impact on treatment compliance Questionnaire.**

**Instruction: Fill in appropriate number in the block (s) or space provided.**

**Part 1: Logistics regarding attending the clinic**

**1. Did you used to attend the QE II diabetes clinic?** ☐ 01
1. Yes
2. No

**2. Which clinic do you attend now (since Oct 2011)?**
1. Gateway clinic ☐ 02
2. Domicilliary clinic ☐ 03
3. Lesotho Defence Force (LDF) clinic ☐ 04
4. Qoaling clinic ☐ 05
5. Mabote clinic ☐ 06
6. Likotsi clinic ☐ 07
7. Other (specify)_____ ☐ 08

**3. If you stay outside Maseru, why do you come to the city clinics?** ☐ 09
_____

**4. How often do you go to a diabetes clinic?** ☐ 10
_____

**5. Do you have to sleep over in Maseru when you attend the clinic?** ☐ 11
1. Yes
2. No

**6. a). If you sleep over do you have to pay for accommodation?** ☐ 12
1. Yes
2. No

**b). If yes, how much?_____** ☐ 13

**7. How do you reach the clinic?** ☐ 14
1. Drive to the clinic
2. Driven by members of the family/friends/acquaintance
3. Take a taxi
4. Walk

**8. a). Do you pay for transport?** 15

1. Yes
2. No

**b). If yes, how much_____?** 16

**9. At what time do you arrive at the clinic?** 17

_____

**10. How long do you wait before checking your blood pressure and blood sugar, at the clinic?** 18

_____

**11. How long do you wait before you could be seen by the doctor?** 19

_____

**12. Do you feel you have enough time with your doctor?** 20

1. Yes
2. No

**13. What type of medication do you use to control your blood sugar?** 21

1. Tablets
2. Insulin injection
3. Diet only
4. Tablets and insulin
5. Nothing

**14. Do you always receive all your medicines from the pharmacy?** 22

1. Yes
2. No

**15. How long do you wait before you could get your medicines?** 23

_____

**16. What time do you normally arrive at home, from the clinic?** 24

_____

**17. Do you think the services at the clinics take too long?** 25

1. Yes
2. No

**18. How much do you pay to attend the clinic?** ☐ 26

_____

**19. Do you think the clinic provides good or up to standards services?** ☐ 27

_____

**20. Do you think there is a good communicative relationship between the patient and health care providers?** ☐ 28

_____

**21. a). Are your blood pressure and blood sugar checked by the s same nurse each time you attend the clinic?** ☐ 29

1. Yes
2. No

**b). If no. How do you feel about it?** ☐ 30

_____

**22. a). Are you consulted by the same doctor each time you attend the clinic?** ☐ 31

**1. Yes**
**2. No**

**b). If no. How do you feel about it?** ☐ 32

_____

**23. As a result of your diabetes, are you retired or currently not working?** ☐ 33

1. Yes
2. No

**24. Do you sometimes miss to attend clinic because of lack of money?** ☐ 34

1. Yes
2. No

**25. Do you receive psychosocial and emotional support from the clinic, family members and friends?** ☐ 35

_____

**26. a). Do you know about the Lesotho Diabetes Association?** ☐ 36

1. Yes
2. No

**b). If yes. Are you a member?_____** ☐ 37

**Part 2: Diet and lifestyle KAP.**

**1. a). Did you receive any education from a health care provider about healthy eating habits?** ☐ 38

1. Yes
2. No

**b). If yes. What are the healthy dietary recommendations?**

1. Increase fibre intake ☐ 39
2. Avoid animal fats ☐ 40
3. Avoid sugar ☐ 41
4. Eat sweets ☐ 42
5. Use salt and oil liberally ☐ 43

**2. Have you ever received a detailed written instructions regarding dietary intake (meal planning) from a health care provider (Dietitian)?** ☐ 44

1. Yes.
2. No

**3.a). Do you adhere to any form of healthy dietary guidelines?** ☐ 45

1. Yes.
2. No

**b). If Yes. Which healthy dietary guidelines are you adhering to?**

1. Eat variety of foods and regular small meals. ☐ 46
2. Include foods containing carbohydrates that are slowly digested. ☐ 47
3. Eat cooked beans, peas, lentils and soya regularly. ☐ 48
4. Eat low fat chicken, fish, milk, yoghurt, cheese, meat or eggs daily. ☐ 49
5. Drink 6 – 8 glasses of clean, safe water. ☐ 50
6. Eat plenty of vegetables and fruits everyday (at least 5 servings a day). ☐ 51
7. Eat fats sparingly, avoid saturated (animal) fats and trans fatty acids. ☐ 52
8. Use salt sparingly. ☐ 53
9. Use food and drinks containing sugar sparingly, not between meals. ☐ 54
10. Drinks alcohol sensibly. ☐ 55

11. None of the above ☐ 56

**4. What are your reason (s) for not adhering to the dietary recommendations?**

1. Often eating out. ☐ 57
2. Financial constraints. ☐ 58
3. Poor self control ☐ 59
4. Difficulty of following a different diet from the rest of the family. ☐ 60
5. Lots of travelling ☐ 61
6. High frequency of social gatherings. ☐ 62
7. Other (specify)_____ ☐ 63

**5. Do you feel that healthy dietary habits have a role to play in the management of type 2 diabetes mellitus?** ☐ 64

1. Yes
2. No

**6. Do you believe that good dietary habits could help control and maintain your blood sugar?** ☐ 65

1. Yes
2. No

**7. What do you understand by the word physical activity (exercise)?**

1. Brisk walking. ☐ 66
2. Housework ☐ 67
3. Aerobic exercises. ☐ 68
4. Gardening. ☐ 69
5. Sitting down. ☐ 70

**8. Do you do any form of moderate physical activity (exercises)?** ☐ 71

1. Yes
2. No

**9. What form of physical activities (exercises) are you adhering to?** ☐ 72

_____

**10. How often do you do your physical activities (exercises)?** ☐ 73

_____

**11. What duration do you normally take to do your physical activities** ☐ 74

**(exercises)?**

_____

**12. Do you believe that moderate physical activity (exercise) has a role in the management of type 2 diabetes?** ☐ 75

1. Yes
2. No

**13. Do you believe that moderate physical activity (exercise) helps to control and maintain blood glucose (sugar) levels?** ☐ 76

1. Yes
2. No

**14. Do you think that moderate physical activities may exacerbate other illnesses?** ☐ 77

1. Yes
2. No

**15. If you do not do any form of physical activity (exercise), what are your reasons?**

1. Unwillingness ☐ 78
2. Lack of time to exercise ☐ 79
3. Workloads ☐ 01
4. Lack of advice given by health care providers ☐ 02
5. Coexisting diseases such as osteo-arthritis ☐ 03
6. Stressful environment ☐ 04

**16. Have you ever received a detailed written instructions regarding exercise programs from a health care provider?** ☐ 05

1. Yes
2. No

**17. a). Do you use any alcoholic beverages?** ☐ 06

1. Yes
2. No

**b). If yes. What is the recommended intake for you?** ☐ 07

_____

**18. What type of alcohol do you think is good for you?** ☐ 08

_____

**19. Are you allowed to use any form of tobacco (cigarette or snuff)?** ☐ 09

1. Yes
2. No

**Part 3: Knowledge and practices regarding self-care.**

**1. Please tick the symptoms of high blood sugar?**

1. Drinking a lot of water. ☐ 10
2. Passing a lot of urine. ☐ 11
3. Feeling weak and tired. ☐ 12
4. Swollen body. ☐ 13
5. Dry mouth. ☐ 14

**2. Please tick the consequences of high blood sugar, over a long period of time.**

1. Blindness ☐ 15
2. Foot ulcers ☐ 16
3. Asthma ☐ 17
4. Kidney problem ☐ 18
5. Arthritis ☐ 19

**3. What is the reference range of normal fasting blood sugar?** ☐ 20

_____

**4. Do you think diabetes complications can be prevented?** ☐ 21

1. Yes
2. No

**5. Do you take your medicines as prescribed?** ☐ 22

_____

**6. a). Do you take your medicines only when symptoms occur?** ☐ 23

1. Yes
2. No

**b). If yes. What is/are your reason (s)?** ☐ 24

_____

**7. Did you receive any education about the use of your medicines?** ☐ 25

_____

**8. Do you have confidence in your medicines' benefits?** ☐ 26

_____

**9. a). Do you think it is important to regularly test your blood sugar?** ☐ 27

1. Yes
2. No

**b). If yes. How often do you test? _____** ☐ 28

**10. How often are the following checked?**

1. Eye sight _____ ☐ 29
2. Kidney function_____ ☐ 30
3. Blood pressure_____ ☐ 31
4. Feet_____ ☐ 32

**11. Please tick daily personal foot care recommendations.**

1. Wash your feet daily with soap and lukewarm water. ☐ 33
2. Dry your feet thoroughly even in-between the toes. ☐ 34
3. Carefully cut your nails. ☐ 35
4. Apply your feet with lotions, except in-between the toes. ☐ 36
5. Wear well fitting socks. ☐ 37
6. Try to wear leather shoes. ☐ 38
7. Avoid walking bare-footed in and out-doors. ☐ 39
8. Wear tight shoes. ☐ 40
9. Wash your feet with hot water. ☐ 41
10. Keep your feet moist. ☐ 42

**12. Do you attend your checkups as scheduled?** ☐ 43

_____

**<u>Part 4: Health, cultural beliefs and attitudes.</u>**

**1. What are the causes of diabetes mellitus?**

1. Heredity ☐ 44
2. Overweight ☐ 45
3. Wrong diet ☐ 46
4. Punishment from God. ☐ 47
5. Witchcraft ☐ 48

**2. Whom did you consult when you first experience symptoms of diabetes mellitus?** ☐ 49

1. Doctor at the clinic.
2. Doctor's surgery
3. Popular sector (friends, spouse)
4. Traditional Healers

**3. Do you know that diabetes mellitus is a life-long disease?** ☐ 50

1. Yes
2. No

**4. Do you think diabetes mellitus is the result of supernatural forces?** ☐ 51

1. Yes
2. No

**5.a). Do you use any traditional medicines?** ☐ 52

1. Yes
2. No

**b). If yes, which ones? Please tick the correct box.**

1. Aloe (lekhala) ☐ 53
2. Hloenya ☐ 54
3. Khomo ea balisa ☐ 55
4. Sehalahala sa matlaka ☐ 56
5. Cheche. ☐ 57
6. Other (specify)_____ ☐ 58

**6. a). Do you use any herbal home remedies?** ☐ 59

1. Yes
2. No

**b). If yes. Please indicate which ones.**

1. Cinnamon ☐ 60
2. Garlic ☐ 61
3. Ginger ☐ 62
4. Green tea. ☐ 63
5. Other (specify)_____ ☐ 64

**7.a) Do you use any nutritional supplements?** ☐ 65

1. Yes

2. No

**b). If yes. Please indicate which ones.**

| | |
|---|---|
| 1. Multivitamins | 66 |
| 2. Omega 3's. | 67 |
| 3. Antioxidants | 68 |
| 4. B-complex | 69 |
| 5. Vitamin C | 70 |
| 6. Calcium and magnesium | 71 |
| 7. Shake | 72 |
| 8. Other (specify)_____ | 73 |

**8. When using complementary alternative medicines, do you still continue taking your medicines?** 74

1. Yes
2. No

**Thank you for your participation!!!**

**Khomeletso E: Mabaka a ka sitisang bakuli ba lefu la tsoekere ho ts'epahalla litlhare le mekhoa e meng ea phekolo.**

**Litaelo: Ngola nomoro e nepahetseng ka mabokoseng kapa sebakeng se siiloeng manapana le potso.**

**Karolo ea pele: Logistics.**

**1. Na pele u n'o tsamaea kliniki ea QE II?** ☐ 01
1. Ee
2. Chee

**2. U s'o tsamaea kliniki e feng haa joale?**
1. Gateway kliniki ☐ 02
2. Lesotho Defence Force (LDF) kliniki ☐ 03
3. Domicilliary kliniki ☐ 04
4. Qoaling kliniki ☐ 05
5. Mabote kliniki ☐ 06
6. Likotsi kliniki ☐ 07
7. Tse ling (Hlalosa) _____ ☐ 08

**3. Haeba u sa lule Maseru, hobaneng u tsamaea kliniking ee?** ☐ 09
_____

**4. U tla kliniking hakae?** ☐ 10
_____

**5. Na u tlameha ho roballetsa ha o e tla kliniking?** ☐ 11
1. Ee
2. Chee

**6.a). Haeba u roballetsa, na u patalla boroko?** ☐ 12
1. Ee
2. Chee

**b). Haeba u patalla boroko. Ke bokae?** ☐ 13
_____

**7. U fihla joang kliniking?** ☐ 14
1. Ke ea ikhannela

2. Ke tlisoa ke ba lelapa/metsoalle/bahaisani ka koloi tsa bona
3. Ke sebelisa taxi
4. Ke tsamaea ka maoto

**8. a). U patala koloi chelete e kae ho tla kliniking?** ☐ 15
1. Ee
2. Chee

**b). Haeba ho le joalo. U patala bokae ka kakaretso?** ☐ 16
_____

**9. U fihla ka nako mang kliniking?** ☐ 17
_____

**10. U ema nako e kae pele u ka hlahlojoa phallo ea mali le tsoekere maling?** ☐ 18
_____

**11. U ema nako e kae pele u ka bonoa ke ngaka?** ☐ 19
_____

**12. Na u khotsofalla nako eo u bang le eona le ngaka?** ☐ 20
_____

**13. U sebelisa mokhoa o feng oa ho laola lefu la tsoekere?** ☐ 21
1. Lipilisi
2. Ente
3. Mokhoa oa ho ja
4. Lipilisi le ente 'moho
5. Ha hona letho leo ke le sebelisang.

**14. Na u fumana litlhare tsa hau kamehla ha u tlile kliniking?** ☐ 22
_____

**15. U ema nako e kae pele u fumana litlhare tsa hau?** ☐ 23
_____

**16. U fihla ka nako e feng hae hangata ha u tsoa kliniking?** ☐ 24
_____

**17. Na u nahana hore lits'ebeletso tsa kliniking li nka nako e** ☐ 25

**telele?**
1. Ee
2. Chee

**18. Na u nahana hore kliniki ee e fana ka lits'ebeletso tse tsoetseng pele?** 26

_____

**19. Na u nahana hore likamano tsa bakuli le basebelletsi ba bophelo lintle?** 27

_____

**20. U patala bokae ka kakaretso ha u tla kliniking?** 28

_____

**21. a). Na phallo ea mali le tsoekere li hlahlojoa ke mooki a le mong hangata ha u le kliniking?** 29
1. Ee
2. Chee

**b). Ha ho se joalo. U e bona joang taba eo?** 30

_____

**22.a). Na u bonoa ke ngaka e le 'ngoe hangata ha u etla kliniking?** 31
1. Ee
2. Chee

**b). Ha ho se joalo. U e bona joang taba eo?** 32

_____

**23. Na u phomotse ts'ebetsong ka lebaka la lefu la tsoekere?** 33
1. Ee
2. Chee

**24. Na u fumana ts'ehetso ea maikutlo kliniking, lapeng kapa ho metsoalle?** 34

_____

**25. Na ka nako e 'ngoe ha u ee kliniking hobane u sena chelete?** 35

1. Ee
2. Chee

**26. a). Na u tseba ka mokhatlo oa Lesotho oa batho ba nang le lefu**
**la tsoekere?** ☐ 36

1. Ee
2. Chee

**b). Ha ho le joalo. Na u setho sa mokhatlo?** ☐ 37

_____

**<u>Karolo ea bobeli: Tsebo, boits'oaro le litloaelo ka tsa ho ja le le tsela ea ho phela.</u>**

**1. a). Na u ile oa fumants'oa thuto ka lefu la tsoekere le hoja ho nepahetseng ke basebeletsi ba tsa bophelo?** ☐ 38

**1. Ee**
**2. Chee**

**b). Ha ho le joalo. Likhothalletso tsa ho ja hantle ke life?**

1. Ho ja lijo tse litlheferetsi ☐ 39
2. Ho qoba mafura a liphoofolo ☐ 40
3. Ho qoba tsoekere ☐ 41
4. Ho ja lipompong ☐ 42
5. Ho sebelisa letsoai le mafura haholo. ☐ 43

**2. Na u kile oa fumants'oa ka mokhoa oa mongolo ke e mong oa bahlanka ba tsa bophelo le phepo (dietitian).** ☐ 44

1. Ee
2. Chee

**3. a). Na u latela likhothalletso tsa ho ja ho nepahetseng?** ☐ 45

1. Ee
2. Chee

**b). Ha ho le joalo. Ke likhothalletso life tseo u li latelang?**

1. Ho ja mefuta e fapaneng ea lijo. ☐ 46
2. Ho etsa limatlafatsi karolo ea bohlokoa ea lijo tsa hau. ☐ 47
3. Ho ja linaoa, lierekisi, lensisi le soya khafetsa. ☐ 48

4. Ho ja nama ea khoho, tlhapi, lebese, nama e khubelu kapa mahe kamehla. ☐ 49
5. Ho noa metsi a hloekileng, a bolokehileng. ☐ 50
6. Ho ja meroho le litholoana kamehla. ☐ 51
7. Ho ja mafura ha nyenyane, haholo a tsoang liphoofolong. ☐ 52
8. Ho sebelisa letsoai ha nyenyane. ☐ 53
9. Ho sebelisa lijo le linomapholi tse tsoekere ha nyenyane. ☐ 54
10. Ho noa joala ka hloko. ☐ 55
11. Ha hona tseo ke li latelang. ☐ 56

**4. Ke mabaka a feng a u sitisang ho latela likhothalletso tsa ho ja ka**
**mokhoa u nepahetseng?**
1. Ho ja litsing tsa lijo. ☐ 57
2. Ho hloka chelete ☐ 58
3. Ho sitoa ho its'oara ☐ 59
4. Ho ba le bothata ba hoja lijo tse fapaneng le ba bang ba lelapa. ☐ 60
5. Ho nka maeto haholo ☐ 61
6. Ho ea meketeng haholo. ☐ 62
7. Le leng (Hlalosa) ☐ 63

**5. Na u lumela hore ho ja ho nepahetseng ho na le kabelo ho laoleng lefu la tsoekere?** ☐ 64
1. Ee
2. Chee

**6. Na u lumela hore ho ja ho nepahetseng ho ka laola le ho theola tsoekere maling?** ☐ 65
1. Ee
2. Chee

**7. U utloisisa eng ka ho thapolla 'mele?**
1. Ho tsamaea ka matla ☐ 66
2. Ho etsa mesebetsi ea lapeng ☐ 67
3. Ho ikoetlisa ☐ 68
4. Ho sebetsa jareteng ☐ 69
5. Ho lula feela ☐ 70

**8. Na hona le boithapollo boo u bo etsang?** ☐ 71
1. Ee
2. Chee

**9. U latela mekhoa e feng ea boithapollo?** ☐ 72

_____

**10. U ithapolla hakae bekeng?** ☐ 73

_____

**11. U ithapolla nako e kae?** ☐ 74

_____

**12. Na u lumela hore ho ithapolla ho na le seabo ho laoleng lefu la tsoekere?** ☐ 75

1. Ee
2. Chee

**13. Na u lumela hore ho ithapolla ho na le seabo ho laoleng le ho theoleng tsoekere maling?** ☐ 76

1. Ee
2. Chee

**14. Na u lumela hore ho ithapolla ho ka tsosa mafu a mang?** ☐ 77

1. Ee
2. Chee

**15. Haeba ha u etse boithapollo bofe kapa bofe. Mabaka a hau ke afe?**

1. Ha kena thahasello ☐ 78
2. Ha kena nako ☐ 79
3. Ke phathahannyoa ke mesebetsi. ☐ 01
4. Ha ke es'o fumane boeletsi bo joalo ☐ 02
5. Ke na le bokuli bo nts'itisang (j.j. arthritis) ☐ 03
6. Ke sitisoa ke tikoloho ☐ 04

**16. Na u kile oa fumana melaetsa e ngotsoeng ea tsela ea ho ithapolla, ho tsoa ho mohlanka oa tsa bophelo?** ☐ 05

1. Ee
2. Chee

**17. a). Na u sebelisa joala?** ☐ 06

1. Ee
2. Chee

**b). Ha ho le joalo. Likhothalletso li re u sebelise bo bokae?** ☐ 07

_____

**18. Ke mofuta o feng oa joala oo o loketseng batho ba nang le lefu la tsoekere?** ☐ 08

_____

**19. Na u lumelletsoe ho sebelisa mofuta o mong le o mong oa koae?** ☐ 09

1. Ee
2. Chee

**<u>Karolo ea Boraro: Tsebo le litloaelo ka ho itlhokomela.</u>**

**1. Khetha matsoao a ha tsoekere e phahame maling.**

1. Ho noa metsi a mangata ☐ 10
2. Ho nts'a metsi khafetsa ☐ 11
3. Ho ba le mokhathala le ho hloka matla. ☐ 12
4. Ho ruruha ha 'mele ☐ 13
5. Ho omella ka hanong. ☐ 14

**2. Khetha litlamorao ha tsoekere e lula a phahame maling nako e telele?**

1. Bofofu ☐ 15
2. Maqeba a sa foleng haholo maotong ☐ 16
3. Lets'oeea ☐ 17
4. Liphieo tse sa sebetseng hantle ☐ 18
5. Manolenyetso a bohloko ☐ 19

**3. Tsoekere maling e tlameha ho bala bokae pele u eja?** ☐ 20

_____

**4. Na u lumela hore litlamorao tsa tsoekere li ka thibeloa?** ☐ 21

1. Ee
2. Chee

**5. Na u noa litlhare tsa hao joalo ka ha u bolelletsoe.** ☐ 22

1. Ee
2. Chee

**6. a). Na u noa litlhare ha feela u utloa mats'oao a ha tsoekere e phahameng?** ☐ 23

1. Ee
2. Chee

**b). Ha ho le joalo. Mabaka a hau ke afe?** ☐ 24

_____

**7. Na u kile oa fumana thuto ka ts'ebeliso ea litlhare tsa hau?** ☐ 25

_____

**8. Na u na le tumelo ea hore litlhare tsa hau li ea sebetsa?** ☐ 26

_____

**9. a). Na u nahana hore ho bohlokoa ho hlahloba tsoekere maling khafetsa?** ☐ 27

1. Ee
2. Chee

**b). Ha ho le joalo. U hlahloba hakae?** ☐ 28

_____

**10. U hlahlojoa litho tse latelang hakae?**

1. Pono _____ ☐ 29
2. Liphieo _____ ☐ 30
3. Phallo e phahameng ea mali _____ ☐ 31
4. Maoto _____ ☐ 32

**11. Khetha likhothalletso tse nepahetseng tsa ho hlokomela maoto letsatsi ka letsatsi.**

1. Hlatsoa maoto a hau kamehla ka metsi a foofo le sesepa. ☐ 33
2. Omisa maoto a hau hantle le lipakeng tsa menoana. ☐ 34
3. Kuta manala a hau ka hloko. ☐ 35
4. Tlotsa maoto a hau hantle, feela qoba ho tlotsa pakeng tsa menoana. ☐ 36
5. Roala likausi tse lekanang hantle. ☐ 37
6. Leka ho roala lietta tsa letlalo. ☐ 38
7. Qoba ho tsamaea ka maoto fats'e, ekaba ka tlung kapa kantle. ☐ 39
8. Roala lieta tse u tiisang. ☐ 40
9. Hlatsoa maoto a hau ka metsi a chesang. ☐ 41
10. Boloka maoto a hau a le mongobo haholo. ☐ 42

**12. Na u ea kliniking ka nako e beiloeng?** ☐ 43

_____

**Karolo ea bone: Litumelo le likatamelo tsa bophelo le setso.**

**1. Lefu la tsoekere le bakoa ke eng?**

1. Lefutso ☐ 44
2. 'Mele o moholo ☐ 45
3. Lijo tse sa nepahalang ☐ 46
4. Kotlo e tsoang ho Molimo ☐ 47
5. Boloi ☐ 48

**2. U ile oa bona mang ha u qala ho ba le mats'oao a lefu la tsoekere?** ☐ 49

1. Ngaka kliniking
2. Ngaka e its'ebetsang
3. Batho ba bang joaloka metsoalle kapa bahaisane.
4. Ngaka ea setso

**3. Na u nahana hore lefu la tsoekere ha le phekolehe?** ☐ 50

1. Ee
2. Chee

**4. Na u nahana hore lefu la tsoekere ke litlamorao tsa meea e mebe?** ☐ 51

1. Ee
2. Chee

**5. a). Na u sebelisa litlhare tsa setso ho laola tsoekere?** ☐ 52

1. Ee
2. Chee

**b). Ha ho le joalo. Khetha tseo u li sebelisang.**

1. Lakhala (aloe) ☐ 53
2. Hloenya ☐ 54
3. Khomo ea balisa ☐ 55
4. Sehala-hala sa matlaka ☐ 56
5. Cheche ☐ 57
6. Tse ling (Hlalosa)_____ ☐ 58

**6.a). Na u sebelisa linoko (herbs) bakeng sa ho laola tsoekere?** ☐ 59

1. Yes
2. No

**b). Ha ho le joalo. Bonts'a hore na le life?**

1. Cinnamon ☐ 60
2. Konofono (garlic) ☐ 61
3. Ginger ☐ 62
4. Green tea ☐ 63

Tse ling (Hlalosa)_____ ☐ 64

**7. a). Na u sebelisa lijo tlatsetso (supplements) ho laola tsoekere ea hau?** ☐ 65

1. Ee
2. Chee

**b) Haeba ho le joalo. Bonts'a hore ke life?**

1. Multivitamin ☐ 66
2. Omega ☐ 67
3. Antioxidants ☐ 68
4. B-complex ☐ 69
5. Vitamin C ☐ 70
6. Calcium and magnesium ☐ 71
7. Shake ☐ 72

8. Tse ling (Hlalosa)_____ ☐ 73

**8. Ha u sebelisa lijo tlatsetso le meriana ea setso, na u tlohela meriana ea hau ea kliniking?** ☐ 74

1. Ee
2. Chee

**Kea Leboha!!!**

**Appendix F: A letter requesting an approval from the Ethics Committee of the Ministry of Health and Social Welfare.**

Ethics Committee
Ministry of Health and Social Welfare
P.O Box 514
Maseru, 0100

Dear Sir/Madam,

**Re: Permission to perform a research study at diabetes clinics in Maseru.**

I am currently a student registered for Masters Degree in Dietetics in the Department of Nutrition and Dietetics at the University of the Free State. As part of this degree, I am undertaking a research project titled **"Nutritional status, glycemic control, and perceived barriers that may impact on treatment compliance among patients with Type 2 diabetes mellitus attending diabetes clinics in Maseru, Lesotho"**. May I submit this letter to apply for a permission to undertake the study at diabetes clinics in Maseru**.**

The aim of the study is to determine the nutritional status, glycemic control, and perceived barriers to treatment compliance among patients with T2DM who attend the clinics in Maseru, Lesotho. The patients', health care professionals and decision makers in the Ministry of Health and Social Welfare will benefit from the findings generated from this study.

The study will entail the following standard procedures:

1. Anthropometric measurements, which will include weight and height to determine Body Mass Index (BMI), Waist Circumference (WC) and Body Adiposity Index (BAI).
2. Face-to-face structured interviews on socio-demographic factors, usual dietary intake, lifestyle factors, medical history and barriers that may impact on treatment compliance.
3. Glycated hemoglobin (HbA1c) measurements or analysis of blood sample, drawn from the participants.

The study procedures involve no foreseeable risks or harm to participants. Participation is voluntary and there will be no compensation for participation. Participants have the right to withdraw from the study at any time and without any penalties. There will be provision of snacks (yoghurt and a fruit) to all participants.

The study will be submitted for approval to an Evaluation Committee of the school of Allied professionals and the Ethics Committee of the Faculty of Health Sciences at the University of the Free State (UFS).

All information will be kept strictly confidential, and will not be used for purposes other than the research project. The results may be published but the participant will remain anonymous.

Questions regarding the study may be directed to the researcher at +266 58900297 and the Secretariat of the Ethics Committee of the Faculty of Health Sciences, UFS, Ms Strauss at +2751 4052812.

Sincerely,

_____

Mohlakotsana Mokhehle (2008094787)

**Appendix G: A letter requesting an approval from the District Medical Officer- Maseru District.**

District Medical Officer
DHMT
Maseru
100

Dear Sir/Madam,

**Re: Permission to perform a research study at diabetes clinics in Maseru.**

I am currently a student who has registered for Masters Degree in Dietetics in the Department of Nutrition and Dietetics at the University of the Free State. As part of this degree, I am undertaking a research project titled **"Nutritional status, glycemic control, and perceived barriers that may impact on treatment compliance among patients with Type 2 diabetes mellitus attending diabetes clinics in Maseru, Lesotho"**. May I submit this letter to apply for a permission to undertake the study at Domiciliary and RLDF clinics in Maseru**.**

The aim of the study is to determine the nutritional status, glycemic control, and perceived barriers to treatment compliance among patients with T2DM who attend the diabetes clinics in Maseru, Lesotho. The patients', health care professionals and decision makers in the Ministry of Health and Social Welfare will benefit from the findings generated from this study.

The study will entail the following standard procedures:

1. Anthropometric measurements, which will include weight and height to determine Body Mass Index (BMI), Waist Circumference (WC), and Body Adiposity Index (BAI).
2. Face-to-face structured interviews on socio-demographic factors, dietary intake, lifestyle factors, medical history and barriers that may impact on treatment compliance.
3. Glycated hemoglobin (HbA1c) measurements or analysis of blood sample, drawn from the participants.

The study procedures involve no foreseeable risks or harm to participants. Participation is voluntary and there will be no compensation for participation. Participants have the right to withdraw from the study at any time and without any penalties. There will be provision of snacks (yoghurt and a fruit) to all participants.

The study will be submitted for approval to an Evaluation Committee of the school of Allied professionals and the Ethics Committee of the Faculty of Health Sciences at the University of the Free State (UFS).

All information will be kept strictly confidential, and will not be used for purposes other than the research project. The results may be published but the participant will remain anonymous.

Questions regarding the study may be directed to the researcher at +266 58900297 and the Secretariat of the Ethics Committee of the Faculty of Health Sciences, UFS, Ms Strauss at +2751 4052812.

Sincerely,

_____

Mohlakotsana Mokhehle (2008094787)

**Appendix H: An information document for participants.**

**STUDY TITLE: Nutritional status, glycemic control and barriers that may impact on treatment compliance among Type 2 diabetes patients attending diabetes clinics in Maseru, Lesotho.**

Dear Participant,

I, Mohlakotsana Mokhehle a student at the University of the Free State, is pursuing a Masters Degree in Dietetics in the department of Nutrition and Dietetics. I am doing research on the study title stated above, and research is just the process to learn the answer to a question. In this study I want to determine the nutritional status, glycemic control, and barriers that may impact on treatment compliance among Type 2 diabetes patients attending diabetes clinics in Maseru, Lesotho.

This study only involve research and not routine care, hence the duplication of some procedures. The findings of this study may be crucial in the planning and implementation of the prevention, control and treatment strategies for Type 2 diabetes patients, which will eventually benefit all diabetic patients in Lesotho.

A quantitative descriptive cross-sectional study will be conducted from June to October, 2012. There will be 120 participants, selected from the diabetes clinics in Maseru, Lesotho. Both Males and females between the ages of 30 and 69 years old, who attend the clinics and have been diagnosed with T2DM, and are on prescribed dietary, hypoglycemic tablets or insulin treatment by a clinician, and who have signed a consent form, will be included in the study. All the procedures are expected to take approximately an hour.

The study will entail the following standard procedures:

1. Anthropometric measurements, which will include weight and height to determine Body Mass Index (BMI), Waist Circumference (WC) and Body Adiposity Index (BAI).
2. Face-to-face structured interviews on socio-demographic factors, dietary intake, lifestyle factors, and barriers that may impact on treatment compliance.

3. Glycated hemoglobin (HbA1c) measurements or analysis of blood sample, drawn from the participants.

The interviews will be conducted during the clinic and after the diabetic patients have received all the services, that is after checking the blood sugar and blood pressure levels, consultation with doctors, and receiving of medication. The researcher will schedule times for interviews according to the participants' preference.

The study procedures involve no foreseeable risks or harm to participants, and a 5ml blood sample will be taken by the Nurse on duty. The biochemical measurement to be determined will be HbA1c only. Participation is voluntary and refusal to participate will involve no penalty. Participants have the right to withdraw from the study at any time and without any penalties. No compensation will be given to participants, but there will be a provision of snack (yoghurt and a fruit) for all participants.

As a participant you will be given relevant information on the study while involved in the project and after the results are available. There will be no personal results issued since efforts will be made to keep personal information confidential.

Efforts will be made to keep personal information confidential. Absolute confidentiality cannot be guaranteed. Personal information may be disclosed if required by law.

The results may be published but the participant will remain anonymous.

Questions regarding the study may be directed to the researcher at any time at the following numbers +266 58900297 and/or the Secretariat of the Ethics Committee of the Faculty of Health Sciences, UFS, Ms Strauss +2751 4052812.

**Khoromeletso H: Tokomane e hlalosang phuputso.**

**SEHLOHO: Boemo be phepo, taolo ea tsoekere le mabaka a ka sitisang bakuli ba lefu la tsoekere ba tsamaeang likliniking tsa Maseru, Lesotho ho ts'epahalla kalafo.**

Monkarolo,

'Na, Mohlakotsana Mokhehle moithuti unifesithing ea Foreistata , ke ithutela Masters Degree ea phepo e nepahetseng fapheng la Phepo e Nepahetseng le Melaoana ea Lijo. Karolo ea thuto ena ke ho etsa phuputso ka sehlooho se boletsoeng ka holimo ho taba tsena.

Phuputso ena e qala ka Hlakubele ho isa Phupu selemong sa 2012. Phuputso ena e tlo hlahloba boemo ba 'mele, mokhoa oa ho ja, ho laola tsoekere le mabaka a etsang hore bakuli ba lefu la tsoekere, ba tsamaeang likliniki Maseru, ba sitoe ho noa litlhare tsa bona hantle kapa ho sebelisa likeletso tseo ba lifuoang kokelong. Phuputso e tlo etsuoa ho bakuli ba lefu la tsoekere ba 120. Bakuli batla kena liphuputsong batla ba lipakeng tsa lilemo tse mashome a mararo ho isa ho mashome a ts'eletseng a metso e robong.

Bakuli batla botsoa lipotso ke mofuputsi lekunutung, 'me ho tla botsoa mokuli ka mong. Mofuputsi o tla boela a nke boima le bolelele ba moithuti ka mong. Mokuli o tla kena phuputsong ena ho fihlela khoeling ea Phupu selemong sa 2012.

Ka hoo, ke memela mokuli ho tla nka karolo phuputsong ena. Lintlha tse latelang li tla etsoa e le ho phethahatsa liphuputso:

1. Boemo ba 'mele bo tla fumanoa ka ho nka boima, bolelele, bophara ba letheka le mafura a 'meleng a mokuli.
2. Lipuisano li tla etsoa pakeng tsa mokuli le mofuputsi ka boemo ba mokuli, mokhoa oa ho ja, litloaelo tsa mokuli, le mabaka a sitisang hore mokuli a latele litaelo tsa basebetsi ba bophelo, le ho noa litlhare tsa hae ka ts'oanelo.

3. Mali a tla nkuoa ho tla hlahloba taolo ea tsoekere khoeling tse tharo tse fetileng (HbA1c).

Phuputso le mokuli ka mong e nka bonyane hora (1 hour). Phuputso ena e etsoa kamorao hore mokuli a hlahlojoe tsoekere le phallo e phahameng ea mali, a bonoe ke ngaka le hore a fumane litlhare tsa hae. Nako ea phuputso e tla lokisoa ke mofuputsi le mokuli. Litaba tsa phuputso ena litla bolokoa ka mokhoa o bolokehileng ke mofuputsi oa litaba, ‘me ha li na ho aroleloana le mang kapa mang, e tla ba lekunutu la mofuputsi.

Ha tla nkuoa mali a etsang limeli-metara tse hlano (kapa khaba e noang meriana) ke Nurse e teng ts’ebetsong, ‘me ha hona kotsi e ka etsahalang. Tlhahlobo e tla etsoa ke e bitsoang HbA1c feela. Mokuli o bolokolohing ba ho lumela kapa ho hana ho nka karolo ea bakuli batla fuputsoa, ‘me a ka itokolla nako eohle ho se kotlo ea letho. Phuputso ena ha e behe bophelo ba mokuli ofe kapa ofe kotsing. Ha ho letho leo bakuli ba lebelletseng ho le pataloa ha ba nka karolo phuputsong ena. Feela ho tla ba le lijo tsa lipakeng (snack); yoghurt le tholoana.

Mokuli ea ikhethetseng ho nka karolo, o tla fuoa tokomane e hlalosang phuputso ena. Ha ho na letho le amanang le mokuli ka mongoe leo a tla le fuoa mabapi le litaba tsa phuputso. Litaba tsa phuputso ena li tla ba lekunutu la mofuputsi.

Litaba tsena li ka ‘na tsa phatlalatsoa empa mabitso a baithaopi ba nkileng karolo a ke ke a phatlalatsoa.

Lipotso mabapi le phuputso ena li etsoe ho mofuputsi oa litaba nomorong tse latelang: +266 58900297 kapa ho Mofumahali Strauss ea fumanehang ofising ea melao le melaoana ea batho lefapheng la Bophelo le Mahlale, Unifesithing ea Foreistata nomorong e latelang +27 51 405 2812.

**Appendix I: Consent form to participate in a Research.**

You have been asked to participate in a research study.

You have been informed about the study by Mohlakotsana Mokhehle.

You may contact Mohlakotsana Mokhehle at 58900297 any time if you have questions about the research or if you are injured as a result of the research.

You may contact the Secretariat of the Ethics Committee of the Faculty of Health Sciences, UFS at telephone number (051) 4052812 if you have questions about your rights as a research subject.

Your participation in this research is voluntary, and you will not be penalized or lose benefits if you refuse to participate or decide to terminate participation. There will be no cost and remunerations to participants, and results of this study may be published.

If you agree to participate, you will be given a signed copy of this document as well as the participant information sheet, which is a written summary of the research.

The research study, including the above information has been verbally described to me. I understand what my involvement in the study means and I voluntarily agree to participate.

_____ _____

Signature of participant Date

_____ _____

Signature of Witness Date

_____ _____

Signature of Translator Date

**Khorometso I: Boitlamo ba ho nka karolo liphuputsong.**

U kopuoa ho nka karolo liphuputsong.

U hlaloselitsoe ka liphuputso tse tlo etsoa ke Mohlakotsana Mokhehle.

Haeba u ena le lipotso kapa u hlaheloa ke kotsi liphuputsong tse tlo etsoa, tsebisa Mohlakotsana Mokhehle linomorong tse latelang; 58900297.

Haeba u ena le lipotso mabapi le litokelo tsa hau liphuputsong tsena, buisana le komiti ea boits'oaro, lekaleng la lithuto tsa mahlale, univesitine ea Frei Setata; linomorong tsa mohala tse latelang (051) 4052812.

Ha u tlamelloe ho nka karolo liphuputsong tsena, 'me u ke se haneloe ka lits'ebeletso hobane u sa nke karolo kapa u ka itokolla hara nako. Ha ho na patala e lebelletsoeng ho tsoa ho uena, kapa eona tefo e tla etsoa. Lipheto tsa liphuputso tsena li ka 'na tsa phatlallatsoa.

Haeba u nka karolo liphuputsong tsena, u tla fumants'oa tokomane le pampiri e hlalosang liphuputso ka bokhuts'oanyane.

Liphuputso tse tl'o etsoa le lihlakiso li buisanoe le 'na, 'me ke utl'oisisitse ka botlalo hore na ho lebelletsoe eng ho 'na liphuputsong tsena, ke lumela ho nka karolo.

_____ _____

Boitekeno ba hau. Letsatsi

_____ _____

Boitekeno ba molebelli Letsatsi

_____ _____

Boitekeno ba toloko Letsatsi