DIABETES MELLITUS AND ORAL HEALTH: A COMPARISON BETWEEN DIABETIC AND NON-DIABETIC SUBJECTS

by

Nonhlanhla Radebe

A thesis submitted in partial fulfilment of the requirements for

the degree of MSc (Dent) in Dental Public Health,

University of the Western Cape

September 2009

Supervisor: Prof Sudeshni Naidoo

ABSTRACT

Diabetes is often associated with a number of medical complications as a result of the metabolic changes taking place systemically. There is considerable evidence it is associated with oral complications including among others, gingivitis, periodontal disease, xerostomia, oral candidiasis, dental caries, periapical abscesses, lichen planus, burning mouth syndrome and an altered taste sensation (Lamster et al. 2008; Skamagas et al. 2008; Vernillo, 2003). The aim of the present study was to compare the oral health status in diabetic and non-diabetic patients with regards to their oral health problems, oral pathology and self perceived quality of life.

A comparative cross-sectional study to determine the common oral complications prevalent in diabetics and non-diabetics was carried out in KwaZulu-Natal, at Prince Mshiyeni Memorial, EThekwini District, Umlazi. The study sample consisted of 150 diabetic patients and 150 non-diabetic patients attending the hospital. The oral health status was assessed clinically for each patient and recorded prior to the interview. The DMFT, plaque index and appearance of marginal gingiva were used to assess oral health status. An intra-oral examination was carried out to identify oral pathology lesions and other oral problems. Patients were then interviewed on the self perceived quality of life and the impact that diabetes has had on their lives.

Plaque Index and DMFT were significantly higher among the diabetic group as opposed to the non-diabetic group. Periodontal disease was observed in more than half of the diabetic group as opposed to only 14.7% of the non-diabetic group.

Except for tooth decay, the diabetic patients had more oral health problems compared to the non-diabetic group. More than half of the diabetic group reported having xerostomia compared to only 7.3% of the non-diabetic group. Altered taste sensation was also more prevalent in the diabetic group. In general, the diabetic group demonstrated a higher prevalence of oral pathology lesions and medical diabetes complications compared to the non-diabetic group.

The self perceived quality of life was said to have deteriorated in 92% of diabetic subjects due to concomitant diabetic complications and 75% of this group indicated that they were not satisfied with their current quality of life.

Diabetic patients were significantly less likely to perceive their quality of life as very satisfied after having adjusted for age and gender variables (OR=0.0188; CI: 0.0059-0.0594). Furthermore, diabetic patients were almost 6 times more likely to perceive themselves as "not satisfied" with their quality of life (QOL) as compared to non-diabetic patients.

Individuals with diabetes exhibited poorer oral health when compared to non-diabetics. They exhibited a higher DMFT and had a significantly higher average number of missing teeth compared to the non-diabetic group. Special care needs to be given to diabetic patients because of the associated complications to improve their quality of life. A more detailed and in-depth study that utilises a diabetes-specific quality of life instrument may provide a more accurate way of determining the quality of life as well as periodontal disease in patients.

DECLARATION

I, the undersigned, Nonhlanhla Matron Radebe hereby declare that the work contained in this dissertation is my original work and has not been previously in its entirety or in part been submitted at any university for a degree.

_____ _____

Dr NM Radebe Date

DEDICATION

This dissertation is dedicated my loving parents, my husband Manqoba and my son Yenzile Mkhize for all the support that they gave me whilst studying. It is also dedicated to the patients that willingly participated in this study with so much honesty and openness.

ACKNOWLEDGEMENTS

I would like to thank Professor Sudeshni Naidoo for her patience, unfailing support and expert guidance throughout this programme. A special “thank you” also goes to Ms NM Hlela, Ms Debra Mahlobo and the CEO of Prince Mshiyeni Hospital for all their support during my data collection period. I also wish to thank all the participants without whom this study would not have been possible.

TABLE OF CONTENTS

CHAPTER 3: AIMS AND OBJECTIVES

CHAPTER 4: RESEARCH METHODOLOGY

CHAPTER 5: RESULTS

CHAPTER 6: DISCUSSION AND CONCLUSION

LIST OF TABLES

LIST OF FIGURES

LIST OF APPENDICES

CHAPTER ONE: INTRODUCTION

Diabetes mellitus (DM) is described as one of the most common endocrine diseases in medicine (Clark, 2006; Bjelland et al. 2002; Zielinski et al. 2002) or the most common chronic disorder (Almas et al. 2003). It has reached epidemic proportions (Papadopoulos et al. 2007) despite the fact that not every diabetic case is reported and it is expected to rise as the ageing of the population will result in more people succumbing to it (Bjelland et al. 2002).

Diabetes Mellitus encompasses a heterogeneous group of disorders with the common characteristic of altered glucose tolerance and impaired lipid and carbohydrate metabolism (Karima et al. 2005). The disease is a growing public health problem (Mealey, 2008; Ogunbanjo, 2006; Tan et al. 2006; Murrah, 1985) because its associated complications give rise to socio-economic and disease burdens that put an enormous strain on the health care systems in many countries (Wilder et al. 2009; Papadopoulos et al. 2007 and Mbokazi, 2006), including South Africa. Diabetes exerts a heavy economic burden on society because of health system costs incurred in managing the disease, indirect costs resulting from productivity losses and premature mortality (Kirigia et al. 2009). Diabetes mellitus accounts for 5% of all deaths worldwide and it results in morbidity and mortality in both developing and developed countries (Skamagas et al. 2008). In the United States alone about 20.8 million people are estimated to have diabetes (Kapp et al. 2007) and it continues to rise due to the increasing number of overweight and obese individuals (King, 2008). There is evidence to suggest that women are at a higher risk of mortality and morbidity from diabetes (Gucciardi et al. 2008).

DM is a pernicious chronic disorder of the carbohydrate, fat and protein metabolism (Crawford, 1997; Vernillo, 2003) which is regarded as a true metabolic disorder as its effects are far-reaching, leading to pathological changes in every tissue of the body. It is characterized by a very distinct feature, the elevation in blood glucose levels that may occur as a result of a defect in insulin secretion from the pancreas, a change in insulin action or both (Almas et al. 2003; Mealey, 2003; Crawford, 1997).

The process leading to hyperglycaemia is fairly complex and it involves changes in chemical pathways and metabolic changes. An increase in cellular resistance to the action of insulin causes a series of metabolic irregularities involving carbohydrates, fats and proteins (Lamster et al. 2008), that result in micro- and macrovascular disease - the leading causes of death among diabetic patients.

Diabetes reduces the life expectancy of an individual diagnosed at 40 by about 12 years for men and 14 years for women (Skamagas et al. 2008). This is a cause for concern as a substantial increase in the number of diabetic people is anticipated worldwide. By the year 2025, the prevalence of diabetes is estimated at more than 300 million (Taylor and Borgnakke, 2008; Campus et al. 2005), a clear indication that this public health problem is on the increase.

It is critical therefore that oral health workers have a clear understanding of the disease in terms of its diagnosis, prevention and treatment so as to improve the quality of care offered to such patients (Rees, 2000).

Dentists can play a pivotal in identifying a diabetic patient, especially since diabetes is associated with oral complications. They can influence the diagnosis, treatment, management and prognosis of such patients by diagnosing their diabetes-associated oral complications and promptly referring the patient to a physician. There is no cure for diabetes and the dentist can play a role in glycaemic control of this disease by maintaining good oral hygiene on their diabetic patients and by motivating them to maintain good glycaemic control as this will prevent or slow down the progression of associated medical and oral diabetic complications. An elevation in blood glucose levels (hyperglycemia) affects almost all body tissues including the oral cavity although most literature focuses on the medical effects of diabetes rather than on oral health effects. The aim of the present study was to determine the prevalence of oral lesions amongst diabetic and non-diabetic patients, including periodontal deterioration; the self perceived quality of life and the prevalence of medical and oral complications closely related to diabetes.

CHAPTER TWO: LITERATURE REVIEW

This chapter provides an overview of diabetes in terms of its classification, epidemiology, associated risk factors, pathogenesis, diagnosis and management. The oral complications of diabetes are discussed as well as the role that dentists may play in management of the diabetic patient with special reference to diabetic emergencies.

Diabetes is a heterogenous group of disorders caused by a relative or absolute insulin deficiency which causes abnormalities of carbohydrate, protein and lipid metabolism (Ogunbanjo, 2006). It is pernicious in character and has strong associations with numerous co-morbid conditions. Its primary feature is hyperglycaemia that results from decreased insulin production, insulin dysfunction or lack of insulin receptor responsiveness at target organs (Almas et al. 2003; Mealey, 2003; Bjelland et al. 2002).

2.1. Classification of diabetes

The classification of Diabetes Mellitus is based on pathogenic processes that can lead to absolute or relative insulin deficiency, leading to hyperglycaemia, a cardinal feature of diabetes (Kidambi and Patel, 2008). It can be classified into three broad categories according to its signs and symptoms, namely Type 1, Type 2 and gestational diabetes that occur exclusively in pregnant women (Kim and Amar, 2006). Type 1 usually has its onset in childhood and adolescence (Bjelland et al. 2002) and is only prevalent in 5-10% of all diabetic patients (Kidambi and Patel, 2008). It is one of the most common chronic childhood illnesses affecting 18-20 per 100 000 children in the UK (Devendra et al. 2004). The term Type 1 diabetes has replaced what was previously known as insulin-dependent diabetes mellitus (IDDM), juvenile-onset diabetes mellitus (JODM) and early-onset diabetes (Clark, 2006). The previous names were used because Type 1 diabetics needed insulin for life and usually the disease occurred before 30 years of age. The new classification is an attempt to introduce appropriate, uniform terminology and to provide a functional working classification of diabetes that reflects the current knowledge about the disease instead of classification based on treatment methodology (Clark, 2006).

The American Diabetes Association recommended that Type 1 DM be further divided into Type 1A and Type 1B (Clark, 2006). Type 1A is caused by cell-mediated autoimmune destruction of the beta cells of the pancreas while Type 1B refers to non-immune mediated diabetes (NIMD) with severe insulin deficiency. Type 1B is mainly found in people of Asian or African descent. The characteristic features of this Type are very similar to those of Type 2 DM which are ketoacidosis and absence of autoimmune markers (Clark, 2006; Rees, 2000).

Type 1 diabetes is a slowly progressive T-cell-mediated autoimmune disease (Frier and Fisher, 2006; Rees, 2000) although its onset is often abrupt (Kidambi and Patel, 2008, Mealey, 2003; Moore et al. 1999; Crawford, 1997). This type of diabetes traditionally occurs in individuals younger than 30 years, mostly in adolescence or childhood, but according to recent epidemiological studies the incidence seems to be similar in both under 30s and adults (Bjelland et al. 2002). In Type 1 diabetes, the beta cells of the Islets of Langerhans in the pancreas are destroyed and are unable to produce insulin (Bjelland et al. 2002; Kim and Amar, 2006). This results in an inexorable cascade of metabolic reactions that eventually manifest as complications associated with diabetes. Hyperglycaemia, accompanied by the classical symptoms of diabetes, occurs only when 70-90% of the beta cells have been destroyed and this, together with familial studies has served as proof that this Type of diabetes has a very slow-onset (Frier and Fisher, 2006).

Insulin is vital for glucose metabolism and the body cannot function well in its absence, as it is unable to transport glucose to the cells where it is needed. It facilitates the entry of glucose which is already absorbed in the blood stream into the cells of the body by using glucose transporters (Mbokazi, 2006). This is the reason why exogenous insulin therapy is given to all Type 1 diabetic patients to prevent the build-up of glucose in the tissue fluids and blood stream (Bjelland et al. 2002).

Type 1 diabetes often predisposes one to a condition known as diabetic ketoacidosis (DKA). DKA occurs as a result of poor insulin control and it interferes with bone coupling and healing (Bender and Bender, 2003).

When glucose cannot enter the bloodstream, fat is metabolized through lipolysis and this is when glycerol and free fatty acids are released. The glycerol is converted to glucose and the fatty acids to ketones which then accumulate in the body fluids. Ketones can be detected in plasma using dipsticks. A slowly evolving variant of Type 1 diabetes is known as latent autoimmune diabetes in adults (LADA). This Type can be detected by specific auto antibodies called glutamic acid decarboxylase (GAD) the GAD65Ab being specifically associated with LADA in middle-aged people (Frier and Fisher, 2006). This subgroup is often masqueraded as Type 2 diabetes until evidence of autoimmune activity against pancreatic beta cells is detected.

Type 2 is the most common form of diabetes (King, 2008; Bjelland et al. 2002; Zielinski et al. 2002) and it refers to non-insulin –dependant diabetes mellitus. It occurs as a result of a reduced responsiveness to insulin or insulin resistance at target organs leading to a series of complications which include retinopathy, neuropathy, vascular degeneration and nephropathy. While there is still no scientific explanation why the insulin resistance occurs in Type 2 patients, it remains well accepted that excessive production of glucose in the liver accompanied by under-utilization of glucose in the skeletal muscle result from resistance to the actions of insulin (Frier and Fisher, 2006). This Type has its onset above 40 years of age (Kidambi and Patel, 2008).

In both Type1 and Type 2, the vascular system where the exchange of oxygen, nutrients and waste products occurs, is affected. The capillaries are affected in two ways: Firstly, they can be damaged due to atheromatous deposits accumulating in the lumen of the blood vessels and secondly, they may develop a thickening of the basement membrane which reduces the activity of leukocytes. The reduction in the activity of leukocytes leads to a reduction in polymorphoneucleocytes' (PMNs) killing ability making the diabetic patient more vulnerable to progress to a more severe infection than what is otherwise expected in a non-diabetic patient (Mbokazi, 2006; Bender and Bender, 2003).

The accompanying hyperglyceamia causes advanced glycation endproducts (AGEs) and the release of glycoheamoglobin ($HgbA_{1c}$) that also contributes to the thickening of the basement membrane of the blood vessels. Unlike hemoglobin, the glycoheamoglobin is less adept when it comes to transportation of oxygen. Other pathological mechanisms associated to elevated hyperglyceamia are activation of the sorbitol pathway, damaging effects of oxidative stress and altered lipid metabolism (Lamster et al. 2008).

Diabetes Mellitus affects the entire body through its medical complications. Tissues affected are those rich in blood vessels like the kidney, retina and nerves (Orbak et al. 2008). Complications such as renal disease, neuropathy, retinopathy, peripheral vascular disease and coronary heart disease may occur (Mbokazi, 2006). These serious chronic complications are associated with long-term biochemical and functional abnormalities that occur in poorly controlled diabetes and they often lead to premature and increased morbidity and mortality. The mortality and morbidity occur as a local response to the generalized vascular damage that occurs as a result of vascular permeability (Frier and Fisher, 2006). At present the annual mortality rate in adult diabetic patients is double that of non-diabetic adults as a result of these associated complications (Mbokazi, 2006).

This is largely due to large blood vessel disease which accounts for about 70% of all deaths which are mostly from myocardial infarction and stroke. Myocardial infarction is the most common cause of death in Type 2 diabetes mellitus (Zielinski et al. 2002). Scientific evidence shows that women have a significantly higher risk of morbidity and mortality as a result of diabetes associated complications (Gucciardi et al. 2008).

Gestational diabetes is defined as either onset or first recognition of glucose intolerance during pregnancy in a woman that has not had this condition before (Dasanayake et al. 2008; Kim and Amar, 2006). It occurs in pregnancy and it usually has its onset during the third trimester (Mealey, 2003). It is a pernicious condition that is responsible for perinatal morbidity and mortality as it is closely linked to pre-eclampsia, caesarian delivery, premature rupture of membranes and preterm delivery (Dasanayake et al. 2008). Its pathophysiology closely mimics that of Type 2 diabetes as it is associated with increased insulin resistance.

A diagnosis of this Type of diabetes during pregnancy usually leads to Type 2 diabetes within a decade post-delivery which is why gestational diabetes is now seen as a harbinger of DM in later life (Kidambi and Patel, 2008). Diagnosis of this condition is very significant in order to implement interventions against Type 2 DM and possible fetal abnormalities. Apart from the Types of diabetes already discussed, the American Diabetes Association has included other Types of diabetes classified according to specific etiology or pathophysiology (Kidambi and Patel, 2008; Frier and Fisher, 2006) and these are listed in Table 1.

Table 1: Other specific types of diabetes

1	Genetic defects of beta cell function: maturity-onset diabetes of the young (MODY) e.g. MODY 1, MODY 2.
2	Pancreatic disease or injuries (e.g. Pancreatitis, pancreactomy, neoplastic disease, etc).
3	Drug induced diabetes (e.g. corticosteroids, thiazide diuretics, phenytoin and nicotinic acid).
4	Endocrine Disorders: Hyperthyroidism, Cushing's syndrome, acromegaly, pheochromocytocoma, etc.
5	Other genetic syndromes (Down's syndrome, Klinefelter's Syndrome, Turner's Syndrome, etc
6	Viral infections (e.g. Congenital rubella, mumps, Coxsackie B virus and Cytomegalovirus).
7	Rare Immune Mediated Diabetes Mellitus: stiff man syndrome and anti-insulin receptor antibodies.

2.2 Risk Factors

Diabetes risk factors can be broadly classified as modifiable and non-modifiable. Those that are non-modifiable include factors such as genetic predisposition, increasing age and ethnicity (Skamagas et al. 2008). Diabetes Mellitus is associated with familial history (Bjelland, 2002), although the mode of inheritance of the susceptible genes is rather elusive (Skamagas et al. 2008). The fact that Type 1 diabetes occurs very frequently in people of Northern European descent and less commonly in Blacks, Native Americans and Asians (Clark, 2006) or that Indigenous Australians have more than twice the diabetes prevalence rates compared to the prevalence found in other Australians (Bjelland et al. 2002) may be an indication of how ethnicity affects diabetes prevalence.

Modifiable risk factors include obesity, sedentary lifestyles, hypertension, smoking and hyperlipidaemia, especially low-density lipoprotein hypercholesterolemia. Changing lifestyles characterized by very little exercise and a high fat diet lacking in fibre which significantly contributes to obesity (Bjelland et al. 2002; Skamagas et al. 2008). Obesity (with a body mass index >30) is a major risk factor for Type 2 diabetes (Bjelland et al. 2002) as it plays a very significant role in the patho-physiology of diabetes (Ogunbanjo, 2006). In fact, obesity is an integral part of metabolic syndrome X and it predisposes to Type 2 diabetes. According to one study done in South Australia 67.6% of the diabetic population were either overweight or obese (Bjelland et al. 2002). Knowledge of these risk factors is essential for non-drug management of diabetes. Diabetes often enhances the effects of other major cardiovascular risk factors like smoking, hypertension and dyslipidaemia (Frier and Fisher, 2006), which is why diabetic patients should receive some form of dietary and lifestyle counseling, and should be strongly discouraged from smoking and unhealthy eating habits.

Other modifiable risk factors distinctive of diabetes mellitus are impaired glucose tolerance (IGT) and impaired fasting glucose (IFG). Both these factors are referred to as pre-diabetes. These represent a metabolic state that occurs between diabetes and normoglyceamia (Bjelland et al. 2002).

People with this condition appear to have worse cardiovascular and mortality outcomes, especially those with IGT (Skamagas et al. 2008). To diagnose pre-diabetes, a glucose tolerance test has to be done. When carrying out the test, the blood glucose levels are measured after the patient has been fasting from the previous night. The patient is given an oral dose of glucose and the test is done minutes to hours thereafter (Skamagas et al. 2008). A fasting glucose value of 100-125mg/dl as well as a post-glucose challenge of 140-199mg/dl is used to define IFG and IGT respectively (Skamagas et al. 2008).

If a patient is non-diabetic the blood glucose level rises moderately and returns to normal ranges within an hour. In diabetic patients the blood glucose levels rise abnormally and remain elevated for hours after the oral dose of glucose. The reasons for the prolonged hyperglycaemia could either be attributed to a lack of insulin release by the pancreas or impaired target tissue response to insulin if not both (Crawford, 1997). Although these are not considered as clinical entities, they are important risk factors for developing diabetes in the future (Mealey, 2003; Rees, 2000).This is because this prediabetes phase represents an active and deleterious phase where progressive beta cell deterioration and insulin resistance occurs (Skamagas et al. 2008).

2.3 Epidemiology

Diabetes Mellitus is a common epidemic yet its prevalence is not easy to determine because many people remain undiagnosed and many are without any clinical signs and symptoms of diabetes (Kapellas and Slade, 2008; Kim and Amar, 2006; Bjelland et al. 2002). The prevalence of diabetes increases with age has a strong positive correlation to obese and overweight individuals. In the year 2000 diabetes was estimated to affect more than 171 million people worldwide (Brunton, 2008; Clark, 2006 Mealey, 2003; Crawford, 1997). World Health Organization currently estimates that more than 180 million people have diabetes worldwide (Kirigia et al. 2009). In the United States of America (USA), it currently affects more than 12 million people.

In the African Region, diabetes was estimated at 7.02 million people in the year 2000. and the estimates for 2030 suggest that the prevalence of the disease will increase to 366 million, most of which will be Type 2 (Brunton, 2008) since it is the most common form (Bjelland, 2002).With improvements in treatment, most people are likely to survive for longer periods after diagnosis, thereby, further increasing the prevalence (Ogunbodede, 2005).

In South Africa (SA), about 4 million people were reported to have diabetes in 2006 (Clark, 2006). The prevalence of Type 1 DM varies according to the different socio-economic groups and geographical location. There seems to be a vast international variation in the incidence of Type 1 diabetes. For example, a child in Finland is 40 times more likely to develop Type 1 diabetes than a child in Japan and almost 100 times more likely to get Type 1 diabetes than a child in the Zunyi Region of China (Devendra et al. 2004). There is also considerable evidence that a low income also contributes to a higher prevalence of diabetes and diabetes related complications (Rabi et al. 2006) and this is supported by Velupillai et al (2008), who reported that disadvantaged communities suffer higher levels of physical ill health than more advantaged communities. Not only is the prevalence and incidence of disease higher in areas of deprivation, but also the nature of the problem appears to be qualitatively different; and treatment is less successful (Velupillai et al. 2008). People from low-income groups are vulnerable because they have poor self-esteem, feelings of helplessness and physical and emotional poverty which all play a significant role in their state of health.

Type 1B is commonly found in patients of Asian or African descent (Crawford, 1997). Type 2 diabetes accounts for 80-90% of all forms of DM and its prevalence is increasing due to increasing obesity patterns (King, 2008). The Pima Indians in Arizona, USA have the highest prevalence and incidence rates in the world for Type 2 diabetes (Bjelland, 2002) followed by the Sardinian populations, with more than 90% of diabetes cases (Campus et al. 2005). The prevalence of Type 2 diabetes is higher in African Americans, Native Americans, Hispanics and Pacific Islanders (Mealey, 2003) and it has been reported to be between 3-7% in Western countries (Almas et al. 2003).

Among Aboriginal Canadians Type 2 diabetes has become an epidemic, with a prevalence of 3-5 times higher than non-Aboriginal Canadians (Ley et al. 2009). In South Africa, the Indian community has the highest prevalence (10%) followed by the African community at 5-6% (Ogunbanjo, 2006).

2.4 Pathophysiology

In order to understand the pathophysiology of diabetes, it is crucial to have a fair knowledge of the basic carbohydrate metabolism and insulin action (Mealey, 2003). After every meal, there's an increase in the levels of glucose and insulin is required to regulate these levels back to normal. The metabolic regulation of carbohydrates involves not only the beta cells, which secrete the insulin that reduces plasma glucose but also the alpha cells of the pancreas, the corticoadrenal hormones and the anterior pituitary hormones which increase plasma glucose (Zielinski et al. 2002). The hypothesized mechanism following ingestion of a high glycaemic carbohydrate is that blood glucose rises quickly, as does the secretion of insulin and secretion of insulin continues to bring the blood glucose level even lower than the initial glucose level leading to a hypoglycaemic state which then triggers the secretion of counterregulatory hormones like glucagon and epinephrine which will try to maintain euglyceamia (Janket et al. 2008).

Through this hypoglycaemic state, pathways like glycogenolysis and lipolysis cause an abundance release of free fatty acids which we already know to cause insulin resistance. Insulin is a hormone that is manufactured and stored in the Islets of Langerhans in the pancreas. This hormone plays a significant role in the patho-physiology of diabetes as it is the only hormone that lowers blood glucose levels (Mealey, 2003).When there is a rise in the blood glucose level the release of this hormone is triggered.

Insulin is one of the major anabolic hormones that are vital to the body for the following reasons (Crawford, 1997):

1. Transport of glucose and amino acids across membranes
2. Conversion of glucose into triglycerides
3. Nucleic acid synthesis
4. Protein synthesis
5. Glycogen formation in the liver and skeletal muscles

Its main function is to facilitate glucose transport into special cells of the body at a faster rate. These body cells include striated muscle cells, myocardial cells, fibroblasts and fat cells (Crawford, 1997). It is implicit therefore that should insulin production and secretion be impaired, all blood glucose dynamics will be altered (Mealey, 2003). Insulin secretion by beta cells requires glucose transport to enter the cells and that process is mediated by glucose transporter-2 (GLUT-2). If for whatever reason insulin production is decreased, hyperglycaemia will result, because glucose entry into the blood stream will be prevented or slowed-down. If insulin is secreted but not used by the target cells, hyperglycaemia will result.

If insulin secretion is increased, blood glucose levels may become too low as more glucose will enter the target tissues, leaving very little glucose in the blood stream. This will again cause a release of counterregulatory hormones due to prevailing hypoglycaemia and with enough repetition of the hyperglycaemia and hypoglycaemia cycles; beta cell failure may eventually result, causing Type 2 diabetes mellitus (Janket et al. 2008).

2.4.1 Type 1 Diabetes

Type 1 diabetes is both heterogenous and polygenic. In identical twins the relative risk for developing diabetes is 33% and the chance of a child developing Type 1 diabetes when another first degree relative has diabetes is only 5-10% (Clark, 2006). According to

2.4.2 Type 1A

Genetic factors

Genetic variants or alleles associated with Type 1 diabetes can either cause susceptibility to diabetes or can offer protection from diabetes (Devendra et al. 2004; Kidambi and Patel, 2008). A combination of genetic and environmental factors is believed to trigger a cell-mediated auto-immune attack on beta cells (Crawford, 1997). Genetic factors account for about one third of the susceptibility to Type 1 diabetes (Frier and Fisher, 2006). Diabetes is associated with certain human leukocyte antigens (HLA) which are found within the major histo-compatibility complex on the short arm of chromosome 6 (Frier and Fisher, 2006). The HLADRB1*09 and HLADQB*0302 are associated with Type 1 diabetes in black South Africans (Clark, 2006; Crawford, 1997). Type 1A diabetes is associated with human leukocyte antigens (HLA), HLA-DR and HLA-DQ and is inherited (Crawford, 1997). Most patients who develop Type 1 diabetes either have DR 3, DQ2 or DR4, DQ8 haplotypes (Devendra et al. 2004). There is a close correlation between Type 1 diabetes and other autoimmune conditions; especially thyroid disease (Devendra et al. 2004), coeliac disease, Addison's disease, pernicious anaemia and vitiligo (King, 2008; Frier and Fisher, 2006).

Environmental factors

If a susceptible individual is exposed to an environmental trigger, diabetes may occur. These triggers include viruses (enteroviruses, coxsackie and congenital rubella), toxins or foods (dietary exposure to non-human proteins in early infancy). The body then responds to these triggers through T-cell activation which leads to inflammation whereby mononuclear cell infiltration of pancreatic islands takes place. This infiltration is also known as insulitis. The beta cell response happens after the insulitis has produced auto antibodies to insulin. What results thereafter, is the loss of insulin secretory reserve and insulin deficiency (Crawford, 1997).

Environmental factors play a critical role in diabetes pathogenesis. It is believed that limited exposure to microorganisms during early childhood prevents maturation of the immune system and thus predisposes one to autoimmune disease (Frier and Fisher, 2006). In certain studies, viral particles known to cause autoimmune damage to beta cells have been isolated from pancreatic tissue of diabetic patients serving as evidence that viral infection could be an etiological factor for Type 1 diabetes. Viruses such as Cytomegalovirus, Epstein-Barr virus, Rubella, Mumps, Retroviruses and Coxsackie B4 have all been implicated in Type 1 DM.

Dietary factors such as cow's milk are presumed to trigger Type 1 diabetes (Frier and Fisher, 2006) although one study called the diabetes autoimmune study of the young (DAISY), where newborns were observed to date, has found no evidence that bovine milk ingestion does predispose to diabetes (Devendra et al. 2004). Nitrosamines are found in smoked and cured meats and which are found in coffee are also implicated in the aetiology of diabetes.

Stress may also trigger Type 1 diabetes by stimulating the secretion of counter-regulatory hormones and modulating immune activity (Frier and Fisher, 2006; Karima et al. 2005). During periods of stress, the endogenous production of epinephrine and cortisol causes blood glucose levels to be elevated and this can interfere with glyceamic control (Mealey, 2003).

2.4.3 Type 1B

This Type of diabetes is not as common as Type 1A and has no known aetiology. Patients with this form of diabetes initially present with ketoacidosis, absence of auto-immune markers and show physical characteristics which are more common in Type 2 diabetic patients.

2.4.4 Type 2 diabetes

The patho-physiology of Type 2 DM is closely associated with obesity, insulin resistance and environmental factors like inactivity and excessive eating (Figure 1). Type 2 diabetes is postulated to be a disruption in homeostasis of glucose metabolism (Janket et al. 2008).

This Type of diabetes proves to be more complex in nature since it is characterized by a combination of insulin resistance and impairment of ß cell function leading to relative insulin deficiency (Frier and Fisher, 2006). How these environmental and genetic factors interact is still unclear but it is widely accepted that genetic factors do contribute significantly to diabetes incidence as shown in Figure 1.

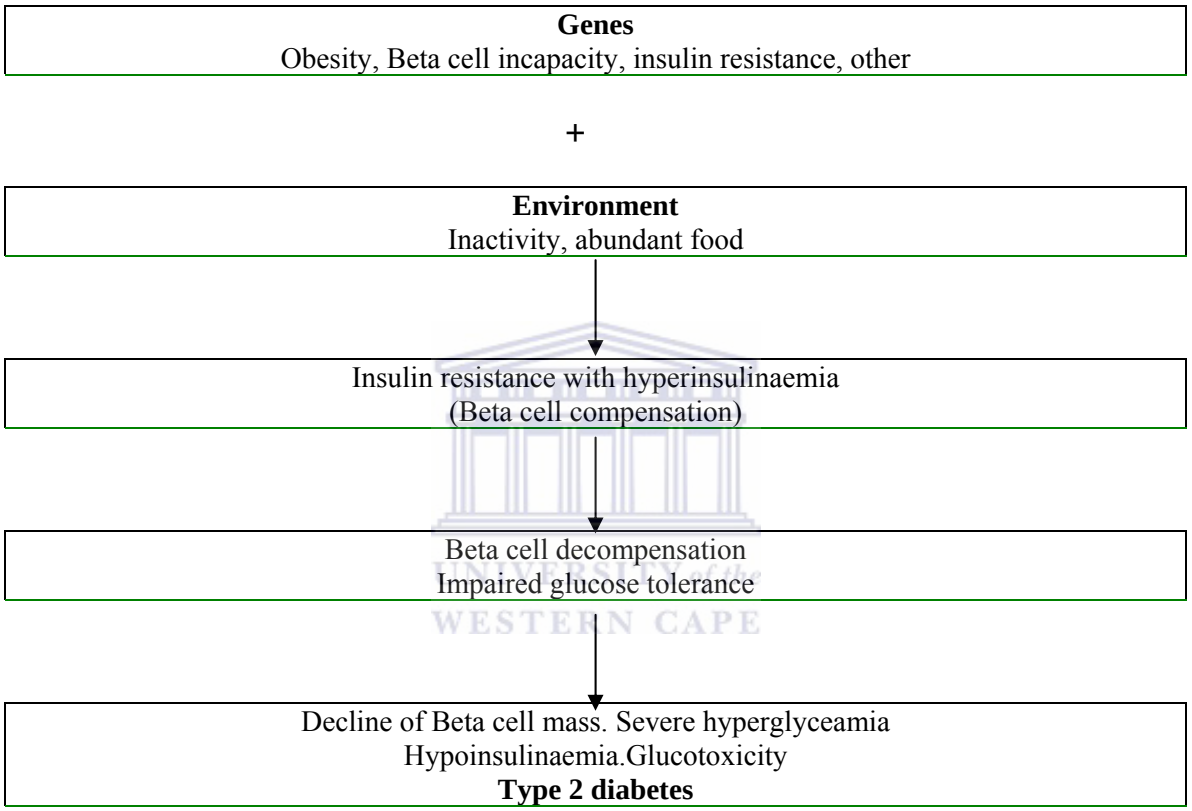

Fig. 1. Pathogenesis of Type 2 diabetes (Ogunbanjo, 2006).

Similar to Type 1 diabetes, environmental factors interact with genetic factors but the underlying genes precipitating environmental factors and pathophysiology differ from that of Type 1. Type 2 diabetics are not prone to diabetic ketoacidosis or other metabolic imbalances seen in Type 1 diabetics. However, under stressful situations and trauma, ketoacidosis may result from the influence of elevated levels of certain stress hormones.

Genetic Factors

Three gene polymorphisms have been identified to be closely associated with Type 2 diabetes. According to Frier and Fisher (2006) these polymorphisms can increase susceptibility by 20%. They are contained in the genes of the PPARγ, Kir6.2 subunit of the ß cell K_{ATP} channel (Frier and Fisher, 2006).

Environmental Factors

Obesity and lack of physical exercise are the major contributory factors for Type 2 diabetes. Individuals with a body mass index >30kg m^2 have 10 times more risk of developing Type 2 diabetes (Frier and Fisher, 2006). This is in contrast to Type 1 diabetes which is commonly known as the disease of the lean (Kidambi and Patel, 2008; Skamagas et al. 2008). Factors such as genetics, sedentary lifestyle, obesity, and chronic inflammation are known to enhance insulin resistance (King, 2008). Type 2 diabetics have to pay attention to their diet foods that contain highly refined carbohydrates may increase the demand for insulin secretion (Janket et al. 2008).

Apart from hyperglyceamia, Type 2 diabetes patients have a group of disorders which are called metabolic syndrome X. This syndrome is characterized by the following: abdominal obesity, atherogenic dyslipidaemia, raised blood pressure, Insulin resistance (with or without glucose intolerance), pro-inflammatory state and prothrombin state. All these conditions predispose to cardiovascular disease (Ley et al. 2009).

The incidence of Type 2 diabetes is increasing at the same time that the overweight and obesity profiles are increasing (Brunton, 2008). In Type 2 diabetic patients, visceral abdominal fat tissue is metabolically active, and this generates large quantities of free fatty acids (FFA). They induce insulin resistance because they compete with glucose as a fuel supply for oxidation in peripheral tissues such as muscle (Janket et al. 2008; Frier and Fisher, 2006). The cytokines generated by the visceral abdominal fat are called adipokines and are strongly associated with insulin resistance, diabetes, hypertension and dyslipidemia which all significantly increase the risk of microvascular and macrovascular complications (Skamagas et al. 2008).

2.5 Pathogenesis

Inflammation and oxidative stress are important factors in the pathogenesis of diabetes and they both contribute to the pathogenesis of diabetic complications (Ding et al. 2007). In addition, advanced glycation endproducts (AGEs) formation is considered to be major causal factors for the pathogenesis of diabetes complications (Taylor and Borgnakke, 2008). However, the pathogenesis is much more complex and involves a series of interrelated metabolic changes which gradually lead to the diagnosis of diabetes.

2.5.1 Type 1 diabetes

Insulitis (infiltration of the islets with mononuclear cells containing activated macrophages, helper cytotoxic and suppressor T lymphocyte) or the selective destruction of insulin-producing cells in the pancreas is the hallmark of Type 1 diabetes (Devendra et al. 2004). T-cell abnormalies are believed to be the major cause of autoimmune disease in Type 1 diabetes, leading to the destruction of pancreatic islets (King, 2008). This autoimmune destruction of pancreatic cells is the main reason behind the insulin deficiency that occurs in Type 1 diabetes but conditions such as pancreatitis and surgical removal or destruction through cystic fibrosis will lead to insulin deficiency too (Kidambi and Patel, 2008).

There is evidence to suggest no involvement of Type 1 diabetes to autoantibodies contrary to the belief that expression of diabetes-related autoantibodies in young children will serve as a major risk factor to the development of Type 1 diabetes (Devendra et al. 2004). Beta cell destruction is associated with the following cells: CD4, CD8, T lymphocytes and macrophages. These events eventually lead to loss of the first phase insulin response (Devendra et al. 2004). Once the beta cells are destroyed, the classic "3 P" symptoms known as polydipsia, polyuria and polyphagia appear (Bender and Bender, 2003).Type 1 diabetes has a strong genetic predisposition (Kidambi and Patel, 2008).

2.5.2 Type 2 diabetes

Type 2 diabetes is preceded by insulin resistance and the target tissues have a decreased response to the normal levels of circulating insulin (Kidambi and Patel, 2008). Patients with Type 2 diabetes have a slow-onset of relative insulin deficiency (Frier and Fisher, 2006). The results are that more and more insulin is required by the target tissues therefore hyperinsulinemia occurs.

The genetic predisposition for Type 2 diabetes is much stronger than for Type 1 diabetes (Kidambi and Patel, 2008). In a study conducted by Das and Rao (2007) it was observed that genes involved in carbohydrate, lipid and amino acid metabolism pathways, glycan of biosynthesis, metabolism of cofactors and vitamin pathways, ubiquitin mediated proteolysis, signal transduction pathways, neuroactive ligand-receptor interactions, nervous system pathways and neurodegenerative disorder pathways are upregulated in obesity compared to health subjects. This study further identified that genes involved in signal transduction, regulation of actin cytoskeleton, complement and coagulation cascades were upregulated in subjects with Type 2 diabetes.

These findings therefore suggest that genes involved in carbohydrate metabolism, lipid and amino acid metabolic pathways and inflammation have a significant role in the pathogenesis of Type 2 diabetes (Das and Rao, 2007). In these patients hyperglycaemia develops gradually over a long period and the renal threshold (capacity of renal tubules to reabsorb glucose from the glomerular filtrate) for glucose rises, so that osmotic symptoms (polyuria and polydipsia) are usually mild (Frier and Fisher, 2006). Type 2 DM is also associated with dyslipidaemia. Obesity and Type 2 diabetes are characterized by chronic oxidative stress and inflammatory stress (Brunton, 2008).

2.6 Diagnosis

Diagnosing Diabetes Mellitus is the realm of the physician (Mealey, 2003) as dentists are not qualified to make a diagnosis (Vernillo, 2003). However, it is still important for dentists to understand how the diagnosis is reached since they have a significant role to play in identifying those individuals at risk or who may have undiagnosed DM. The American Diabetes Association has endorsed the screening of all those who are at risk for diabetes and all those above 45 years of age (Skamagas et al. 2008).

Several tests are used in order to diagnose DM but the primary methods are fasting venous plasma glucose levels and oral glucose tolerance tests (Bjelland et al. 2002). Generally, a fasting glucose test is used as a way of screening patients but it remains limited since it cannot detect all forms of diabetes. A fasting glucose of 126mg dl^{-1} or more is used to distinguish a diabetic patient (Skamagas et al. 2008).

A post oral glucose challenge value of 200mg dl^{-1} or more is another way of distinguishing a diabetic patient. When the above tests have been done and a diabetic patient is distinguished, the next step is to do an oral glucose tolerance test (oGTT) for those who had impaired fasting glucose or in a high-risk individual with a normal fasting glucose. In the past, oral glucose tolerance test used to give false positive results as a result of stress-induced adrenaline release which impairs the response to glucose loading and this is why this test is not as popular anymore (Bjelland et al. 2002). For this test the readings are taken twice; one reading at baseline and another 2 hours after ingesting a 75g load of glucose. Should two abnormal readings be taken on different days then it's undeniably a case of diabetes (Skamagas et al. 2008).

2.7 Medical complications of Diabetes Mellitus

Diabetes complications have important effects on the patients' quality of life (Papadopoulos et al. 2007) and in order to improve the quality of life of diabetic patients it is important to understand these complications. The most common complications are retinopathy, neuropathy, cardiovascular disease, peripheral vascular disease and periodontal disease (King, 2008). Organs and tissues which have abundant capillary vessels are the ones which are severely affected by diabetes due to the changes that take place in the capillary basement membrane. In Type 1 diabetes the common complications are as a result of microvascular disease while in Type 2 diabetes both microvascular and macrovascular disease cause complications (Kidambi and Patel, 2008). Most complications associated with diabetes will occur secondary to the development of microangiopathy. The histopathological hallmark of diabetic microangiopathy is the thickening of the capillary basement membrane with associated increased vascular permeability throughout the body (Frier and Fisher, 2006).

Metabolic control is directly responsible for the extent or severity of diabetic complications that a diabetic individual will present with, but factors like the patients' socio-economic status and the duration of diabetes play an even bigger role. These complications present as diseases of small blood vessels (microangiopathy) or disease of large blood vessels (macrovascular). Microangiopathy causes morbidity and disability through conditions such as blindness due to diabetic retinopathy, difficulty in walking, chronic ulceration of the feet, and bowel and bladder dysfunction (Frier and Fisher, 2006). Macrovascular complications lead to morbidity through myocardial infarction, stroke, angina, cardiac failure and intermittent claudication (Frier and Fisher, 2006). Almost three quarters of the diabetic population develop cardiovascular disease and die from cardiovascular complications (Janket et al. 2008).Vascular problems increase anaerobic infection (Bender and Bender, 2003). Metabolic syndrome X and Type 2 diabetes resulting from sustained hyperglycaemia are also considered risk factors for cardiovascular disease although their mechanism in cardiopathogenesis has not been clearly outlined (Janket et al. 2008).

Another common complication is called atherosclerosis. Increased lipid deposition and atheroma formation is seen in the larger blood vessels coupled with increased thickness of arterial walls (Mealey, 2003). The pathological changes associated with atherosclerosis in diabetic patients are no different from those observed in non-diabetic patients, it is just that in diabetics they occur much earlier in life and are more severe (Frier and Fisher, 2006). They can be exacerbated by the presence of other risk factors such as smoking, hypertension and dyslipidemia (Kidambi and Patel, 2008). Due to the insidious nature of Type 2 diabetes patients are often diagnosed too late, when most of the damage to tissues and organs has been done. As a result a lot of pathological changes may be detected in more than one organ.

Manifestations of diabetes complications may be detected in a patient who is not known to have diabetes and this may well lead to the patient being diagnosed with diabetes following a comprehensive investigation testing blood glucose levels. On the contrary, Type 1 diabetes is characterized by abrupt onset of symptoms, decreased insulin in the serum, dependence on exogenous insulin and a tendency for ketosis (Murrah, 1985). Patients with Type 1 diabetes are especially susceptible to microvascular complications like neuropathy, retinopathy and nephropathy.

Diabetes Mellitus also affects joint function as well as bone density and dentists should be cognizant of this when treating diabetic patients. The increased stiffness and loss of flexibility detected in diabetic patients may affect the functioning temporomandibular joint (TMJ) (Kidambi and Patel, 2008). There are 2 possible mechanisms for the complications of diabetes - firstly the polyol pathway ensures that glucose is reduced to sorbitol (a tissue toxin) by the enzyme aldol reductase and secondly the production of advanced glycation endproducts (AGEs) through the non-enzymatic addition of hexoses to proteins. This leads to alterations of many body proteins like collagen, haemoglobin, lipoproteins and their functions (Soskolne, 1998).

2.8 Management of Diabetes Mellitus

The rationale behind the management of diabetes mellitus is to keep blood glucose levels as close to normal as possible so as to prevent microvascular and macrovascular complications of diabetes. Diabetes complications have significant effects on patients' quality of life which is why the guidelines for treatment of Type 2 DM emphasize that one of the primary objectives is to improve health related quality of life (HRQOL) (Papadopoulos et al. 2007).

There is considerable evidence to show that it is possible to reverse microvascular disease if early interventions are pursued and this is supported in part by Tan et al (2006), who reported that early diagnosis and aggressive treatment of the disease can delay or prevent progression of the major chronic complications. Strict glycaemic control has been shown to reduce microvascular complications in Type 1 diabetes (Skamagas et al. 2008).

Although no cure exists for diabetes, the disease can and its complications can be prevented, delayed and managed by identifying risk factors and detecting the condition at an early stage (Ley et al. 2009). The Diabetes Control and Complications Trial (DCCT) which lasted 9 years demonstrated strong evidence of a 60% overall reduction in the risk of developing diabetic complications in those patients on intensive therapy with strict glyceamic control (mean HbA_{1c} around 9%) (Skamagas et al. 2008; Frier and Fisher, 2006).

The United Kingdom Prospective Diabetes Study (UKPDS) also showed that diabetic complications and progression thereof is much slower with good glyceamic control and effective treatment of hypertension, irrespective of the Type of therapy used (Frier and Fisher, 2006). There are different methods of treatment that can be used to treat diabetes ranging from diet modification and lifestyle changes, oral antidiabetic drugs to insulin.

2.8.1. Non Drug Management

2.8.1.1 Diet and lifestyle changes

Diet control is the first vital step in individuals with Type 2 diabetes with the aim of reducing the intake of refined sugar and high lipid containing foods (Bjelland et al. 2002). The recommended diet should contain 50-65% carbohydrate, 30% total fat with saturated fat being less than 10% and 10-20% of protein (Ogunbanjo, 2006). Patients may be advised to lose weight and to engage in physical exercise to reduce their BMI. The recommended exercise regime is moderate intensity physical exercise of at least 150 min/week or intense aerobic activity for at least 90 min/week with not more than 2 consecutive days of not exercising (Clark, 2006). Patients should be discouraged from alcohol consumption as well as smoking since these are known risk factors for diabetes. If good glyceamic control is not achieved by dietary modification and lifestyle modification alone, oral anti-diabetic drugs may be used (Frier and Fisher, 2006).

2.8.2 Drug therapy

Oral hypoglyceamics

The use of oral hypoglycaemic drugs is very common in treatment of Type 2 diabetes. The aim of these drugs is to promote insulin release from the pancreas, encourage insulin uptake in the target organs and suppress appetite (Bjelland et al. 2002).

This mode of treatment was previously limited to sulfonylureas, pork and beef insulins and metformin but advances in medicine have led to new classes of oral medication, injectable medication recombinant human insulins and novel insulin delivery systems (Skamagas et al. 2008; Kidambi and Patel, 2008).

Metformin, a biguanide is the drug of choice for patients who are younger than 80 years and who have no end organ disease. Its mechanism of action is prevention of hepatic gluconeogenesis and improving insulin sensitivity in muscle and adipose tissue (Skamagas et al. 2008).

Oral biguanides are also used in overweight patients with normal renal function due to their anorexic effects. Sulfonylureas which are insulin secretagogues are prescribed for other Type 2 diabetes mellitus patients. They act by binding to sulfonylurea receptors on the beta cells triggering the release of insulin (Kidambi and Patel, 2008). They can lower the HbA_{1c} but often lead to hypoglyceamia and weight gain.

Thiazolidinediones (TZD) including pioglitazones and rosiglitazone act by increasing tissue sensitivity to insulin and by decreasing blood glucose levels (Mealey, 2003). They do not however cause hypoglyceamia. Their mechanism of action is through activation of peroximose proliferator-activated receptor γ to affect glucose and lipid metabolism (Kidambi and Patel, 2008). In recent years this drug was believed to have anti-inflammatory effects and to have the ability to preserve beta cells but the data generated in clinical trials only proved its enhanced insulin sensitivity effect (Skamagas et al. 2008). Its side effects include fluid retention and weight gain (Skamagas et al. 2008) and in susceptible people it may precipitate congestive heart failure and increase the risk of bone loss (Kidambi and Patel, 2008).

Arcabose can inhibit intestinal enzymes that metabolize carbohydrates into monosaccharides and this leads to a delay in carbohydrate absorption (Skamagas et al. 2008; Mealey, 2003). This agent inhibits α-glucosidase in the stomach which then prevents the breakdown of some complex carbohydrates into simple sugars making them inabsorbable (Kidambi and Patel, 2008).They often have gastrointestinal side-effects like flatulence and diarrhea.

Incretins are the newest group of oral agents for treatment of Type 2 diabetes and as such, they target the incretin pathway. They act by inhibiting the rapid breakdown of two intestinally secreted hormones (glucagon-like-peptide-1 and gastric inhibitory peptide) which are released following a meal (Kidambi and Patel, 2008).They increase insulin secretion, decrease glucagon secretion and delay gastric emptying (Kidambi and Patel, 2008; Frier and Fisher, 2006).

Insulin therapy

More often than not, good glyceamic goals are not met by most of these oral hypoglyceamic drugs and that leads to insulin therapy being introduced. Insulin therapy may be initiated if there is end organ disease or if the side effects that prevent the use of oral medication in severely uncontrolled Type 2 diabetes and Type 2 patients with reduced insulin production. In this case basal insulin or long-acting insulin is used to normalize fasting glucose in the morning (Skamagas et al. 2008). Glargine and Detemir are ideal since they are long acting.

For Type 1 diabetes insulin is the mainstay of treatment (Kidambi and Patel, 2008). Physiologic insulins are used and they can be administered by multiple injections with a basal-bolus regimen, or continuous subcutaneous insulin injection using an insulin pump (Kidambi and Patel, 2008; Skamagas et al. 2008). There are different Types of insulin preparations varying from short-acting, mediate-acting to long-acting. Modern insulin therapy combines the use of short-acting, rapid onset insulin with longer acting forms so that glucose levels are maintained with minimal peaks and troughs through the day (Bjelland et al. 2002). In South Africa all insulin varieties are biosynthetic (Clark, 2006). Insulin therapy should also mimic the physiological release of insulin, which is characterized by a continuous basal secretion, to prevent fasting hyperglyceamia, as well as prandial insulin release to prevent postprandial hyperglyceamia (Kidambi and Patel, 2008).

The success of this therapy bases itself on the ability of patients to monitor their own blood glucose levels with the use of glucometers. This will allow the patients to adjust their insulin dose, diet and exercise to produce normoglyceamia and prevent hypoglyceamia (Kidambi and Patel, 2008).

Combined oral anti-diabetic drug and insulin

In certain instances a diabetic patient may need increasing doses of a sulfonylurea or biguanide, either alone or in combination with each other or with a thiazolidinedione, the introduction of a single dose of intermediate- or long-acting insulin, administered at bedtime, may improve glyceamic control (Frier and Fisher, 2006).

2.8.3 Control of other risk factors

Certain randomized clinical trials have proved that aggressive management of lipids and blood pressure is beneficial in managing diabetes mellitus (Frier and Fisher, 2006). The use of ACE-inhibitors is recommended for its ability to improve outcomes in heart disease and in preventing diabetic nephropathy (Skamagas et al. 2008, Frier and Fisher, 2006). Also, smoking is a risk indicator for periodontal disease in diabetic patients (Tan et al. 2006). Smokers are therefore at a high risk for developing more periodontitis than non-smokers (Wilkins, 1999). Delayed wound healing after surgical and non surgical procedures may be a common finding in those patients who are smokers. Due to these reasons, an effort should be made to advise patients to stop smoking and to effectively treat hypertension and dyslipidemia. Alcohol, which is another risk factor for diabetes, can raise blood pressure and contribute to other health problems so its use by a diabetic patient should be discouraged (Wilkins, 1999).

2.8.4 Patient Education

Apart from the pharmaceutical preparations used to treat diabetes, patient education still plays a significant role in management of a diabetic patient. Diabetes can be a fairly complex disease to comprehend and as such, it is crucial that patients are made to comprehensively understand the very nature of diabetes so that they know how to cope with any given complications.

Those on insulin need to learn to measure their dose correctly and to administer the insulin to themselves on the basis of blood glucose values and other factors like exercise, illness and episodic hypoglyceamia (Frier and Fisher, 2006). Diabetic patients should be advised to carry a card with information on their name, address, their diabetes status, the nature and dosage of their drug treatment and the particulars of their physician in case there is an emergency where the patient is unconscious (Frier and Fisher, 2006).

2.8.5 Self-assessment of glyceamic control

Patients should be taught to perform capillary blood glucose measurements using blood glucose test strips. The recommended targets for adults are pre-prandial capillary plasma glucose of 5.0-7.2.mmol/l and peak postprandial capillary plasma of <10.0.mmol/l (Clark, 2006).This step has an added advantage that information on glucose levels is readily available and the patient can thus adjust treatment appropriately (Frier and Fisher, 2006).

2.9 Diabetes and the oral cavity

Similar changes to those that occur in the kidney, nerves and retina also take place in the oral mucosa. Oral health complications associated with diabetes include xerostomia, tooth loss, gingivitis, periodontitis, odontogenic abscesses, caries and soft tissue lesions of the tongue and mucosa (Vernillo, 2003; Moore et al.1999; Moore et al. 1998). Burning mouth syndrome, poor wound healing and an increased incidence of acute oral infection are all very common features in diabetic patients. Oral complications are more pronounced and occur more readily in Type 1 uncontrolled DM than in Type 2 subjects (Vernillo, 2003).

2.9.1 Xerostomia

Xerostomia is a commonly reported oral complication in the diabetic patient (Murrah, 1985) and is attributed to a decrease in salivary flow. Approximately one in five adults report having xerostomia (Thomson et al. 2006). It is associated with polyuria which is also commonly experienced by diabetic patients. It is not clear whether xerostomia occurs as a result of the diabetes or due to the medication used in treatment.

In some patients xerostomia may present with an enlargement of the parotid glands (Mealey, 2003). The changes in the salivary secretion are believed to occur as a result of the accompanying neuropathy of the autonomic nervous system and this alters the normal oral cavity environment thus predisposing a healthy mouth to dental caries (Vernillo, 2003). Dryness of the mouth may lead to oral tissues being more susceptible to trauma and opportunistic infections such as Candida but also an increased accumulation of bacterial plaque and food debris that contributes to dental caries and periodontal disease (Rees, 2000). Xerostomia may be associated with difficulties in chewing, swallowing, tasting and speaking which leads to poor dietary intake, malnutrition and poor socialization (Soell et al. 2007). It has recently been shown to affect the oral health-related quality of life (Thomson et al. 2006).

2.9.2 Candida

Oral candidiasis is commonly found in diabetics and it is believed to occur due to the dryness of the mucosa. This opportunistic fungal infection is caused by *Candida albicans* and is commonly associated with hyperglyceamia. It is often associated with marginally or uncontrolled diabetes and has a positive correlation to salivary gland dysfunction (Vernillo, 2003; Soell et al. 2007). Its occurrence in the diabetic patient may well be attributed to the fact that diabetics have impaired immune defense mechanisms and that makes them more susceptible to infection (Bjelland et al. 2002).

2.9.3 Burning mouth sensation

This condition is of unknown aetiology but is closely associated with salivary gland dysfunction, candidiasis and neurological abnormalities such as depression (Vernillo, 2003). Patients experiencing a burning mouth sensation do not present with any detectable lesion except the symptoms of pain and a burning sensation on the tongue. It is also suggested with no definite proof that this condition is common in severe diabetes mellitus where diabetic neuropathy exists as a complication (Rees, 2000).

By definition, gingivitis is a condition where inflamed gingival tissues are associated with a tooth, with no attachment loss or with previous attachment and bone loss, but is not currently losing attachment or bone (Tan et al. 2006). This is the most prevalent mild form of periodontal disease which is characterized by inflammation of the gums, erythema, swelling and frequent bleeding (Kim and Amar, 2006).

This inflammation of the gingiva is caused by bacterial plaque accumulation and is reversible if treatment is initiated early enough (Soell et al. 2007). Gingival bleeding is an indicator of inflammation and it is possible that the vascular changes that take place in the diabetic patient enhance this bleeding (Orbak et al. 2008). Apart from the microvascular changes leading to gingival bleeding, there's a correlation of gingiva with accumulation of plaque and calculus which act as local irritants to the gingival, leading to inflammation of the gums (Orbak et al. 2008). Increased salivary glucose may also contribute to the inflammatory process by way of increased bacterial substrate and plaque formation (Vernillo, 2003).

2.9.5 Periodontitis

Periodontitis is defined as an inflammatory disease of the supporting tissues of the teeth caused by specific microorganisms or groups of specific microorganisms resulting in progressive destruction of the periodontal ligament and alveolar bone with pocket formation, recession or both (Tan et al. 2006).

This condition is now classified as the 6th most common complication of diabetes (Kidambi and Patel, 2008; Almas et al. 2003; Vernillo, 2003; Zielinski et al. 2002) after retinopathy, nephropathy, neuropathy, peripheral vascular disease and cardiovascular disease. It is often referred to as the 6th "opathy" of diabetes (Mansour and Abd-Al-sada, 2005).

Periodontitis is a bacterial infection caused by Gram-negative anaerobes found in the subgingival plaque. The most commonly implicated bacteria causing periodontitis are *Porphyromonas gingivalis, Prevottella intermedia, Tannerella forsythensis and Treponema denticola* (Soell et al. 2007).

This is an infection characterized by oxidative stress, a factor which plays a major role in diabetic vascular complications (Karima et al. 2005). In a diabetic patient, periodontitis is presumed to occur due to an exaggerated inflammatory response to the periodontal micro flora (Lamster et al. 2008). This causes a chronic inflammatory condition characterized by loss of connective tissue attachment and alveolar bone height (Almas et al. 2003). The incidence and severity of periodontitis is influenced by the presence or absence of diabetes mellitus as well as the degree to which the disease is controlled by the patient (Tan et al. 2006; Mealey, 2003; Rees, 2000).

While periodontal infections have an impact on diabetic control, diabetes is a major risk factor for the development of periodontal disease (Wilder et al. 2009; Tan et al. 2006; Karima et al. 2005) and this is underpinned by epidemiological studies and case reports (Tan et al. 2006).This explains the reciprocal relationship that exists between diabetes and periodontitis. In fact, there's evidence to suggest that periodontal disease may increase the risk of experiencing poor metabolic control (Lamster et al. 2008) or that periodontal disease has the potential to have adverse impact on glycaemic control in patients with diabetes mellitus (Herring and Shah, 2006).

Periodontitis is a very common feature in diabetes and it becomes even more severe as the diabetic state progresses (Moore et al. 1999). In one meta-analysis of 18 comparative cross-sectional studies, subjects with diabetes had substantially more severe periodontal disease when compared to healthy subjects (Wilder et al. 2009). Unequivocally, diabetes impairs the immune system's defense mechanisms making diabetic patients not only more susceptible to infections but also the manifestations of those infections being more severe in comparison to non-diabetic patients (Bjelland et al. 2002). Periodontitis as a complication of diabetes is believed to occur following a series of exaggerated inflammatory response to the periodontal micro flora (Lamster et al. 2008). This belief is supported, in part, by the findings that there seems to be no difference in subgingival micro flora between periodontitis patients and their non-diabetic counterparts (Campus et al. 2005). Factors that increase the severity of inflammatory disease like neutrophil dysfunction have been associated with the pathogenesis of diabetes. Periodontal damage in diabetics is initiated by microbial plaque and the prevailing hyperglyceamia.

In a hyperglycaemic environment, proteins like collagen undergo a glycosylation process to form advanced glycation end products (AGEs), which play a significant role in periodontal tissue destruction (Tan et al. 2006). The deterioration in the patient's periodontal health which occurs as a result often presents as deep pockets and tooth mobility often resulting in teeth being extracted. After the extraction the quality of life of an individual is affected as patients cannot chew their foods resulting in unhealthy eating habits and consumption of less fibre-rich foods and more refined carbohydrates. This is supported by Moore et al (1999) who reports that periodontal disease and tooth loss may impact on overall health by compromising a patient's ability to maintain a healthy diet and proper glyceamic control.

Mechanism of action

Development of periodontitis occurs as a result of many factors including the microbial challenge, genetic risk factors, environmental factors and acquired risk factors (Tan et al. 2006). The thickening of the basement membrane which occurs as a result of hyperglycaemia in diabetes alters the oxygen diffusion, waste elimination, leukocyte migration and the diffusion of immune factors. These alterations may all contribute to the pathogenesis of periodontitis (Bjelland et al. 2002).

In a hyperglycaemic environment, proteins like collagen undergo a glycosylation process to form advanced glycation end-products (AGE) (Tan et al. 2006). Greater vascular permeability in AGE-enriched gingival tissue will lead to greater breakdown of collagen fibres and accelerated destruction of both connective tissue and bone (Mansour and Abd-Al-sada, 2005).

Infection in diabetics become more severe and last longer because of neutrophil microbicidal suppression and synergism of aerobic and anaerobic bacteria, as a result of the reduced oxygen diffusion across capillary walls causing anoxia (Bender and Bender, 2003). In a diabetic patient advanced glycation endproducts (AGEs) are the first byproduct to form once there's excess glucose which has come into contact with structural proteins. These AGEs then bind to cellular receptors, which are found on endothelial cells and monocytes, known as RAGE. Once the AGE-RAGE complex has formed, it triggers certain proinflammatory events which lead to accelerated destruction of connective tissue and bone in a diabetic patient (Lamster et al. 2008). AGE is also believed to affect wound healing by inducing apoptosis of extracellular matrix producing cells (Taylor and Borgnakke, 2008).

In addition to the proinflammatory responses, other pathophysiologic changes like reduction in leukocyte chemotaxis, phagocytosis, apoptosis, serum opsonic activity and reduced cellular immunity may predispose diabetic patients to periodontitis. The depressed immune response resulting from diabetes may explain why it may not be

possible to totally eradicate periodontal infection in a diabetic patient, even after conventional periodontal therapy (Tan et al. 2006). During the inflammatory process, neutrophils are stimulated to produce large quantities of superoxide and hydrogen peroxide which are both naturally occurring chemicals within the neutrophil. This process is catalyzed by an oxidative complex known as NADPH and this pathway results in increased oxidative stress which has been shown to significantly contribute to periodontitis (Karima et al. 2005).

The inflammed periodontal tissue also releases cytokines similar to those released by the visceral abdominal fat tissue and they are made up of tissue necrosis factor-alpha (TNF-α) and Interleukin-6 (IL-6) and they both may contribute to aggravate diabetes (Skamagas et al. 2008). It has been postulated that gingival crevicular fluid neutrophil elastase activity, age and smoking are all risk indicators for periodontal disease in diabetic patients (Tan et al. 2006). Enzyme ß-glucoronidase is a lysosomal enzyme which is also released during the inflammatory process when degranulation of PMNs takes place (Tan et al. 2006). It is also believed that the release of reactive oxygen species activates metalloproteinase, which degrade the collagen in the ligaments thus causing loss of attachment on the tooth (Herring and Shah, 2006).

2.9.6 Dental Caries

There seems to be no evidence linking diabetes mellitus to dental caries and information that has been published has often lacked consistency (Soell et al. 2007; Zielinski et al. 2002). Perhaps this inconsistency is as a result of different behavioural patterns among diabetic patients, where those that are compliant and motivated will engage in good oral hygiene practices and stick to regular dental appointments as opposed to those that are non-compliant. Since most diabetic patients limit their intake of fermentable carbohydrates, the less cariogenic diet may limit caries incidence (Mealey, 2003). There are reports of increased incidence of dental caries among uncontrolled or marginally controlled diabetes mellitus in both animals and human studies (Rees, 2000).

2.9.7 Salivary Dysfunction

This condition is difficult to diagnose since decreased salivary flow may occur as a result of factors like medication, age, neuropathy and thirst. However, this is a common finding in diabetic patients. It is associated with xerostomia and may lead to dryness of the mucosa and may contribute to dental caries formation. It is often accompanied by mucositis, ulcers and desquamation and often the tongue is inflamed and depapillated (Vernillo, 2003).

2.9.8 Altered taste sensation

The change in taste has been attributed to altered glucose receptors or early manifestation of diabetic neuropathy (Bender and Bender, 2003). Taste strongly influences food preference and intake and taste receptor variants have been associated with differences in taste perception (Dotson et al. 2008). It has also been attributed to xerostomia and candidiasis (Boyce and Shone, 2006); as a direct result of diabetic neuropathy or medication. Decreased taste sensation may be more pronounced for sucrose as opposed to other taste tests, and this indicates a direct effect of diabetes mellitus (Rees, 2000). The taste receptors involved are the TAS1R and TAS2R and they are responsible for sweet and bitter taste stimuli respectively (Dotson et al. 2008). There's a belief that altered taste sensation is an age-related phenomenon. One theory is that normal ageing produces taste loss because of changes in taste cell membranes involving altered function of ion channels and receptors (Boyle and Shone, 2006). This condition has been reported in diabetes patients who receive hemodialysis (Lamster et al. 2008).

2.9.9 Acute oral infections

This refers to recurrent episodes of herpes simplex virus, periodontal abscesses and palatal ulcers that occur in marginally controlled diabetics (Vernillo, 2003). These infections are believed to occur as a result of the same pathogenic mechanism which is associated with increased susceptibility to periodontal infections (Vernillo, 2003).

2.10 Management of oral complications

2.10.1 Management of xerostomia

Treatment of xerostomia is essential to encourage salivary stimulation to keep the mouth moist, prevent caries and Candida infection to provide palliative relief (Vernillo, 2003). The use of saliva substitutes and stimulants is recommended for this purpose. Artificial saliva containing methylcellulose or a mucin base can be used. Patients could also be encouraged to use sugarless chewing gum to stimulate salivary flow. Topical fluorides should be used so as to prevent caries.

2.10.2 Management of candidiasis

It is crucial for the dentist to remember the association of candidiasis with marginally or uncontrolled diabetes and the positive correlation to salivary gland dysfunction as this information will influence the drug of choice for a patient. As a precautionary measure a dentist should assess the sugar content of the intended medication (Vernillo, 2003) so as to not contribute to the hyperglycaemic state of the patient. Topical and systemic agents are available for treatment of candidiasis. Some of these preparations contain corticosteroids that provide both anti-inflammatory and anti-pruritic benefits, however, steroids can potentially cause hyperglyceamia due to their antagonistic effect on the action of insulin (Vernillo, 2003). The following treatment options are available for these patients (Vernillo, 2003):

0.5% gentian violet aqueous solution applied topically in the mouth 3 times a day.
Nystatin vaginal suppositories-although this preparation is not designed for oral use, clinicians have found it useful in treatment of oral candidiasis when the sugar content of other topical antifungal medications is a concern (Vernillo, 2003).

Clotrimazole troches given as 1-10mg troche 5 times/day for 2 weeks. These troches have a very high sugar content and should be used with caution so as not to affect glycaemic control.

Fluconazole 100mg/day for 2 weeks

Ketaconazole 200mg/day for 2 weeks

Itraconazole 200mg/day for 2 weeks. This drug is effective against resistant strains of *Candida albicans.*

2.10.3 Management of burning mouth syndrome

Due to its association with xerostomia and Candida, some relief may be achieved through proper management of these two factors. In uncontrolled diabetes it is recommended that the glyceamic control be improved so as to prevent complications associated with hyperglyceamia. Agents such as benzodiazepines, tricyclic anti –depressants and anti-convulsants given in low dosages can be useful in treating the burning mouth syndrome however; these should be used sparingly as they can be addictive (Vernillo, 2003).Symptoms of burning mouth have been found in undiagnosed cases of Type 2 diabetes, most of which have been resolved after medical diagnosis and subsequent treatment, directed at improving glycaemic control (Vernillo, 2003).

2.10.4 Management of gingivitis

Gingivitis may progress to periodontitis if it is not treated early enough. The rationale behind treatment of gingivitis is to eliminate the local irritants thereby reducing the inflammation of the gums. Plaque has to be removed through scaling and polishing because the components of microbial plaque have the potential to induce an initial infiltrate of inflammatory cells which may lead to connective tissue destruction (Vernillo, 2003). The use of a mouthwash is recommended as an adjunct to treatment (see 2.10.5). Although there are many mouthwashes to choose from, the efficacy of chlorhexidine has been shown to be very good. This drug has been shown to be the most effective antiplaque and antigingivitis chemotherapeutic agent available (Wilkins, 1999).

2.10.5 Management of periodontitis

Periodontal treatment is an essential requirement in a diabetic patient. In a meta-analysis of ten intervention studies, periodontal treatment resulted in a 0.66 percent reduction in absolute HbA_{1c} levels among patients with diabetes (Wilder et al. 2009).

The therapeutic goals in periodontal disease are to alter or eliminate the origin of the microbes and all contributing factors so as to prevent disease progression and to prevent recurrent periodontitis (Kim and Amar, 2006). Although periodontitis rarely causes death, it can lead to more serious oral infections such as fascial space infections and may be a major factor in bacteraemia (Bjelland et al. 2002).

A conventional approach to management of periodontitis is preferable. Patients with diabetes should receive regular scaling so as to remove plaque and calculus deposits. Local debridement with 1% povidone-iodine should be done. Sound oral hygiene practices like brushing 3 times/day and flossing should be reinforced by the dental team. The use of antibiotics is recommended and the following drugs are often used:

Amoxil 250mg three times/ day for 5 days for patients who are not allergic to penicillin
Erythromycin 250mg three times/ day for 5 days for patients who are allergic to penicillin.
Each of the above-mentioned drugs must be accompanied by any of the following:
Metronidazole 200mg three times/ day for 5 days or Clindamycin 300mg three times/day for 5 days or Clavulanic acid and Amoxil 375mg three times/ day for 5 days.
These drugs target a broad spectrum of bacteria which is why they are used for periodontitis management.

Periodontal surgical procedures can be performed on patients with advanced periodontitis but not without reservations as surgery may potentiate unwanted complications. Often there is a need to modify the patient's medication before and after surgery and there may be delayed wound healing since the patient is diabetic (Vernillo, 2003).

Surgical treatment of periodontitis involves removal of inflamed tissues to reduce the damage to the alveolar bone around the infected area and this has an added advantage since it enhances accessibility to areas where root planning and scaling could not be reached to remove plaque and calculus (Kim and Amar, 2006).

With the causative bacteria eliminated either through scaling and local debridement or through surgical methods, healing can be anticipated in the bone and soft tissues. Since the surgical treatment poses more risk, a non-surgical approach is recommended in the form of debridement and tetracycline antibiotic therapy. Tetracycline is preferable because of its known association with improvement of glycaemic control (Vernillo, 2003). This drug has also been reported as an inhibitor of human matrix metalloproteinases which are connective tissue degrading enzymes.

The use of 0.2% Chlorhexidine gluconate mouthwash has beneficial effects because it is bactericidal. This drug is rapidly absorbed to teeth and pellicle and is released slowly, thus prolonging the bactericidal effect (Wilkins, 1999). Phenol compounds like Listerine may also be used because they are known to be effective in preventing the development of supragingival plaque and gingivitis (Wilkins, 1999). Oxygen compounds like Triclosan are mainly directed against strict anaerobes which cannot survive in the presence of oxygen and may be beneficial in management of periodontal disease (Wilkins, 1999). This drug is both bacteriostatic and bacteriocidal. Patients with periodontal disease should be recalled regularly to monitor progress and to assist the patient with professional removal of plaque and calculus where indicated.

2.10.6 Management of dental caries

Dental caries should be managed according to the size and depth of the lesion otherwise the caries will progress and eventually lead to tooth loss. It is important to remember the association of caries with salivary dysfunction and xerostomia since they may well be the contributing factors to dental caries in a diabetic patient.

Apart from dental fillings, topical treatments such as fluoride-containing mouthrinses and salivary substitutes may help prevent caries. Fluoride compounds, gels, aqueous solutions and dentrifices are all possible options in treatment of dental caries. Topical fluoride can be subdivided into fluorides applied by professionals or those that can be applied at home. From the professionally-applied fluorides, stannous fluoride has been found to be 3 times more effective in inhibiting enamel dissolution by weak acids than sodium fluoride (Wilkins, 1999). Fluoride varnishes like Duraphat, Epoxylite and Elmex can be applied on patients by a professional. The use of fluoride-containing toothpaste for caries prevention at home should be encouraged. Sodium fluoride mouthrinses are also believed to be beneficial because of their fluid nature which allows the fluoride to reach areas which are inaccessible to the toothbrush (Wilkins, 1999). Oral health care workers should also reinforce sound oral hygiene practices and dietary counselling to the patient so that the patient can brush, floss and choose less cariogenic foods, all of which will enhance caries prevention.

2.10.7 Management of salivary dysfunction

This condition should be managed because of its potential complications. Difficulty in lubricating, masticating tasting and swallowing are some of the complications resulting from this condition and may all contribute to impaired nutritional intake of the diabetic patient (Vernillo, 2003). As in xerostomia, saliva substitutes and stimulants can be used to treat saliva hypofunction.

2.10.8 Management of altered taste sensation

Since this condition has a strong correlation for Candida and xerostomia, a positive outcome may be achieved by treating the accompanying fungal infection. Improvements in altered taste sensation may occur when diabetes mellitus metabolic control is established or when xerostomia and associated candidiasis are controlled (Rees, 2000).

2.10.9 Management of acute oral infections

These can be managed by achieving good glycaemic control in a diabetic patient as this will reduce the impact of acute oral infections. For those diabetic patients who might have recurrent Herpes Simplex Virus infection, agents such as oral acyclovir can be used for this infection both prophylactically and therapeutically (Vernillo, 2003). This drug is contraindicated for patients who are diabetic and have renal insufficiency as it can lead to nephrotoxicity (Vernillo, 2003) but it is safe to use on any other diabetic patient. Other drugs of choice are Valacyclovir 100mg three times / day or Famciclovir 250mg three times/ day for 7 days.

For acute oral infections like periodontal abscesses antibiotics similar to the ones used for periodontitis management are used. A mouthwash like chlohexidine gluconate may also be used as an adjunct.

2.11 Diabetic Emergencies in the dental surgery

Dentists should be alert to hypoglycaemia, a condition that is highly dangerous as it may lead to the patient getting a seizure or even loosing consciousness (Kidambi and Patel, 2008, Mealey, 2003). The classical signs and symptoms of this condition include sweating, tremors, confusion, agitation, anxiety, dizziness, tingling or numbness and tachycardia. If this condition is suspected it can be confirmed by taking a glucometer reading and the patient must be given 15g of glucose orally. In the event that a patient is unable to take this glucose orally, an intravenous line (IV) should be set up and 25-50ml of 50% dextrose solution should be administered (Kidambi and Patel, 2008; Mealey, 2003). A subcutaneous injection of 1mg of glucagon should be injected in case it is not possible to set up the IV line. After the treatment, the signs and symptoms of hypoglycaemia should resolve between 10-15 minutes. As a precaution, the patient must be observed for 30-60 minutes after recovery and the blood glucose levels can then be rechecked using the glucometer.

It is not uncommon for marked hyperglycaemia patients to present with the exact symptoms as described for hypoglycaemia. Again, the important step is to confirm the glucose level with a glucometer. However, should a glucometer not be available it is safer to treat this as hypoglycaemia because the extra dose of glucose given will not have a significant or detrimental effect on the hyperglycaemia, but if the patient was not treated as hypoglycaemia, he could suffer life threatening outcomes (Mealey, 2003).

CHAPTER THREE: AIMS AND OBJECTIVES

3.1 Aim: To compare the oral health problems in diabetic and non-diabetic patients attending at Prince Mshiyeni Memorial Hospital, Durban, KwaZulu-Natal

3.2 Objectives:

- To compare the oral health status, oral problems and dietary habits of diabetic and non-diabetic patients attending Prince Mshiyeni Hospital.
- To describe the oral health characteristics of the diabetic patients attending Prince Mshiyeni Hospital.
- To determine the effect of diabetes on the self perceived quality of life.

CHAPTER FOUR: RESEARCH METHODOLOGY

4.1 Introduction

This chapter discusses the research design and methodology. It discusses the study population, study site, study design, data collection, selection of study population, sample size estimation, data analysis and ethical considerations. It also describes the research instrument as well as the data collection methods.

4.2 Study design

The study design was an analytic cross-sectional survey that was carried out in October 2008 to December 2008 at Mshiyeni Memorial Hospital.

4.3 Study site

The study site was the Prince Mshiyeni Memorial Hospital (PMMH) in Umlazi, Durban. This is a 1200 bed facility regarded as a Regional Hospital, providing both district level services and specialist services for the most part of KwaZulu-Natal.

4.4 Study population

The study population was defined as all the diabetic and non-diabetic patients whose ages ranged between 25 and 75 years and who were attending PMMH from October 2008 to December 2008. The three months time frame for data collection was decided upon as this would have given the researcher more time to collect data, because the diabetic clinic opens once a week. The diabetic subjects recruited for this study were patients attending the diabetic clinic at Prince Mshiyeni Memorial Hospital. The non-diabetic subjects included those patients with a clear medical history of no diabetes and who attended the same hospital's out-patients department for other reasons. The age range of 25-75 years was decided upon because it was perceived as appropriate since it would include both Type 1 and Type 2 patients since the study was planned to focus on these two types of diabetes. The decision on this age range was based on information obtained from the literature (Sandberg et al (2000), Ogunbodede et al (2005), Campus et al (2005) and other studies discussed in Taylor and Borgnakke (2008).

Inclusion criteria

All patients who were dentate, with at least 6 teeth, presenting at the out patients department and the diabetic clinic, aged between 25 and 75 years of age

Exclusion criteria

All patients who were less than 25 and more than 75 years of age and patients who were edentulous.

4.5 Selection of study participants

A nonrandom convenience sampling was used in the selection of the study participation. Patients who fulfilled the inclusion criteria were recruited as they came for consultation at the hospital. The non-diabetic patients were recruited as they presented for medical consultation in the out-patients department during the study period. Patients who presented with their files to the diabetic clinic for collection of diabetes medication were included in the study as the diabetic patients.

4.6 Sample size estimation

The sample size calculations were based on the following assumptions:

Type I error of 5% (two tailed), Type II error of 10%.

Among the diabetic patients the prevalence of oral health problems was expected to be 45% and the prevalence of oral health problems among non-diabetic patients was expected to be 25%. Therefore the required number of patients in each group was 150 (two independent samples). The study consisted of 300 patients (150 diabetic and 150 non-diabetic) who all attended PMMH during October-December 2008.

4.7 Measurements and data collection instruments

A data capture sheet and administered questionnaires were used for data collection. The purpose of the questionnaire was to collect factual and/or attitudinal data for measurement. It was designed to obtain accurate and valid responses.

Design rules

The same rules of design apply to all types of questionnaire:

- It must suit the aim of the study
- It must suit the nature of the respondent
- It should be clear, simple, unambiguous
- The design should minimize potential errors from respondents and coders
- The subject of the questionnaire should interest the respondent, encourage their co-operation and elicit truthful answers
- Well worded questions are essential, and pitfalls must be avoided, for example, 'double-barreled questions' that is, when two questions are included in one- the questions will have to be separated so that the respondent and the researcher can distinguish between the two.
- The wording of the questions should not lead the respondent to feel obliged to answer in a particular way, which may not be truthful
- Questions must not alienate either the respondent or the researcher
- Efficient and meaningful analysis of the acquired data should be possible.

Instrument used

Questionnaires and data capture sheets (Appendix I) with open and closed ended questions were the instruments used to collect the data. They were designed to ensure that it suited the aim and objectives of the study and were simple, clearly understood and unambiguous.

The development of the study questionnaire

Planning of the questionnaire began in March 2008. It was developed following group discussions with patients with diabetes and health care professionals caring for them. After a thorough review of the literature, the questions were formulated. The data from the questionnaires was grouped into the following categories: Demographics, Oral health status, Oral problems and dietary habits, exercise habits and perceived quality of life. The diabetic cohort were asked about the duration of their disease, history of oral problems, medication taken and their perceived quality of life since being diagnosed with diabetes.

Piloting the questionnaire

In September 2008, the completed questionnaires were tested on six participants. The pilot study was carried out to:

- Test the suitability of the method of collecting the data
- Check the adequacy of the questionnaire
- Check that all questions were clear and unambiguous
- Remove any items that did not yield usable data.

Preparation for the final draft

After the pilot study, irrelevant and ambiguous questions for the interviews were identified and either reformulated or deleted. This resulted in a general improvement of the questionnaire and an increase in the efficiency of the enquiry. The final draft of the questionnaire (Appendix I) were then printed and copied for the larger study. The design and construction of the instrument took about 6 months to be completed. The questionnaire was piloted. Data was collected using a structured data capture sheet and questionnaire (Appendix I). The questionnaire was piloted (see *Piloting the questionnaire*, Section 4.7). The questionnaire was translated into IsiZulu. Section A was for oral examination and section B for the interview.

4.8 Data collection

Data was collected using a data capture sheet and a questionnaire by the principal investigator (qualified dentist) fluent in English and isiZulu. Firstly the purpose of the study was explained to the patients by the researcher. When the patients agreed to participate in the study they were asked to sign an informed consent form (Appendix II). The oral examination was performed by a qualified dentist (the researcher) to assess oral health status. The oral examination was performed with the use of a wooden spatula, dental mirror explorer probe and in daylight. The sequence of the examination was the teeth, gingiva, floor of the mouth, tongue, buccal mucosa and palate (see Section 4.9.3 for detailed description) and the information collected included DMFT, oral health status, gingival index and oral pathology lesions. Section A of the questionnaire was completed during the oral examination process. After the oral examination, the researcher interviewed the patients on their oral functions and section B of the questionnaire was completed.

The information collected during the interview included oral function, history of oral problems, dietary habits, physical activities and self-perceived quality of life. Any patient with oral health problems was referred to the oral health clinic at the same hospital for treatment and further management.

4.9 Oral examination

4.9.1 Plaque index (Pl I)

The plaque index, based on the debris index of Greene & Vermillion (1964) was used to assess the coronal extension of plaque for the purpose of his study. A total of 6 teeth per patient was assessed. It was perceived as a precise and reliable index to measure oral cleanliness in this study. None of the study participants had the entire teeth covered by plaque.

Codes and criteria

0 None (no debris or stain present)

1= soft debris covering not more than one third of the tooth surface being examined, or the presence of extrinsic stains without debris, regardless of surface area covered

2= soft debris covering more than one third but not more than two thirds of the exposed tooth surface.

3= Soft debris covering more than two thirds of the exposed tooth surface

9= Not recorded

4.9.2 Dentition status and treatment need – DMFT

The decayed, missing and filled teeth index was used to determine dental caries experience, past and present. Third molars were not counted. A systematic approach to the assessment of the dentition status and treatment was adopted using the WHO Guidelines to Oral Health Surveys (1997).

Codes and criteria

0 **Sound crown.** A crown was recorded as sound if it showed no evidence of treated or untreated clinical caries. In addition, a crown with the following defects was also coded as sound: white or chalky spots, discoloured or rough spots that were not soft to touch with the metal explorer probe, stained pits or fissures in the enamel that did not have visual signs of undermined enamel, or softening of the floor or walls detectable with an explorer probe, dark, shiny, hard, pitted areas of enamel in a tooth showing signs of moderate to severe fluorosis, lesions that, on the basis of distribution or history, appeared to be due to abrasion.

Sound root. A root was recorded as sound when it was exposed and showed no evidence of treated or untreated clinical caries. (Unexposed roots are coded as 8)

1 **Decayed crown.** Caries were recorded as present when a lesion in a pit or fissure, or on a smooth tooth surface, had an unmistakable cavity, undermined enamel, or a detectable softened floor or wall. A tooth with a temporary filling, or one which is

sealed (code 6) but also decayed was also included in this category. The explorer probe was used to confirm visual evidence of caries on the occlusal, buccal and lingual surfaces. Where any doubt existed, caries was not recorded as being present.

Decayed root. Caries was recorded as present when a lesion felt soft or leathery to probing with the explorer probe. If the root caries was discrete from the crown and required a separate treatment, it was recorded as root caries. For single carious lesions affecting both the crown and the root, the likely site of origin of the lesion was recorded as decayed. When it was not possible to judge the site of origin, both the crown and the root were recorded as decayed.

2 **Filled crown, with decay**. A crown was considered filled, with decay, if it had one or more permanent restorations and one or more areas that were decayed.

Filled root, with decay. A root was considered filled, with decay, if it had one or more permanent restorations and one or more areas that were decayed. For any restoration involving both the crown and the root with secondary caries, the most likely site of the primary lesion was recorded as filled, with decay. When it was not possible to judge the site of origin of the primary carious lesion, both the crown and the root were recorded as filled, with decay.

3 **Filled crown, no decay**. A crown was considered filled, without decay, when one or more permanent restorations were present and there was no caries anywhere on the crown. A tooth that had been crowned because of previous decay was recorded in this category. A tooth that had been crowned for other reasons (e.g. a bridge abutment), was coded as 7.

Filled root, no decay. A root was considered filled, without decay, when one or more permanent restorations were present and there was no caries anywhere on the root. For any restoration involving both crown and root, the most likely site of the primary lesion was recorded as filled.

4 **Missing tooth, as a result of caries**. This code was used for teeth that had been extracted because of caries and was recorded under coronal status. The root status of a tooth that had been scored as missing because of caries was coded as 7 or 9.

5 **Tooth missing, for any other reason**. This code was used for teeth judged to be absent congenitally, or extracted for orthodontic reasons, periodontal disease, trauma, etc.

6 **Fissure sealant**. This code was used for teeth in which a fissure sealant had been placed on the occlusal surface.

7 **Bridge abutment, crown, veneer**. This code was used under coronal status to indicate that a tooth formed part of a fixed bridge i.e. is a bridge abutment. It was also used for crowns placed for reasons other than for caries and for veneers or laminate covering the labial surface of a tooth on which there was no evidence of caries or a restoration. Missing teeth replaced by bridge pontics were coded 4 or 5 under coronal status, while root status was scored 9.

8 **Unerupted crown**. This code was used for a tooth space with an unerupted permanent tooth. Teeth scored as unerupted were excluded from all calculation concerning dental caries. This category does not include congenitally missing teeth, or teeth lost as a result of trauma, etc.

Trauma (fracture). A crown was scored as fractured when some of its surface was missing as a result of trauma and there was no evidence of caries.

9 **Not recorded**. This code was used for any tooth that could not be examined for any reason. It was used under root status to indicate either that the tooth had been extracted or that calculus was present to such an extent that a root examination was not possible.

4.9.3 Oral mucosa examination procedure

An examination of the oral mucosa and soft tissues in and around the mouth was made on every participant. Any abnormalities of the mucosa or of the gingiva were recorded on the chart on the data capture sheet (Roed-Petersen and Renstrup, 1969). In addition, a full description of the lesion's size, shape, type and anatomical site was documented. The oral examination was thorough and systematic and was performed in the following sequence:

(i) Labial mucosa and labial sulci (upper and lower)
(ii) Labial part of the commisures and buccal mucosa (right and left)
(iii) Tongue (dorsal and ventral surfaces, margins)
(iv) Alveolar ridges/gingiva (upper and lower)
(v) Floor of the mouth
(vi) Hard and soft palate

Two mouth mirrors or wooden spatulas were used to retract the tissues. The following procedure was used and the following codes were used to record the absence, presence or suspected presence of the condition: The lips were examined with the mouth closed and open. The colour, texture and any surface abnormalities of the vermillion border were noted. The mandible vestibule was examined visually with the mouth partially opened. The colour and any swelling of the vestibular mucosa were observed. The maxillary vestibule and fraenulum with the mouth partially opened was examined. Using the plane mouth mirrors as retractors and the mouth wide open, the entire buccal mucosa extending from the commisures and back to the anterior tonsillar pillar was examined. Any changes in pigmentation, colour, texture and mobility of the mucosa were noted. Alveolar ridges were examined from all sides (bucally, palatally, lingually).

With the tongue at rest and the mouth partially opened the dorsum of the tongue was inspected for any swelling, ulceration, coating or variation in colour or texture. The patient was then asked to protrude the tongue and the examiner noted any abnormality of mobility. The margins of the tongue were inspected with the aid of the mouth mirrors and then the ventral surface was observed.

While the tongue was still elevated, the floor of the mouth was inspected for swellings or any other abnormalities. With the mouth wide open and the subject's head tilted backwards, the base of the tongue was gently depressed. The hard palate was inspected first followed by the soft palate. Any mucosal or facial tissues that seemed to be abnormal, as well as the submandibular and cervical lymph nodes, were palpated.

4.9.4 Appearance of marginal gingiva (adapted from Löe & Silness, 1963) with an addition of criteria 3

The appearance of the marginal gingiva by Silness and Löe (1964) was adapted for use as the gingival index to assess the gingival area using the following code:

Code and criteria:

0 = normal gingiva
1 = mild marginal gingivitis, slight colour change & oedema
2 = moderate gingival inflammation, redness & glazing
3 = a distinct red band along the marginal gingiva from papilla to papilla
4 = severe inflammation, marked redness & oedema, *with* ulceration or spontaneous bleeding.

4.9.5 Criteria for interview questions and self-reported responses

The **periodontal status** was assessed by checking for obvious tooth mobility, loss of attachment and gingival recession and recording these during charting. The presence of any of these was regarded as evidence for periodontal disease and was documented on the data capture sheet. Pocket depths were not measured.

Sensitivity to the teeth was defined as any tooth that was sensitive to cold air or cold water at the time of the examination. This was verified by blowing some air from the air syringe and squirting some cold water and observing the response from the patient.

Gum problems were defined by any of the following:

1. Bleeding gums when eating or brushing
2. Swollen gums
3. Painful gums
4. Spontaneous bleeding of the gums

Poor wound healing was evaluated by asking the following questions:

1. How long does it take for your wound to heal if you have an injury?
2. Do you think that your wounds heal fast if you have been injured?
3. How long did it take before your socket healed when you last extracted a tooth?

A response of two weeks or more, coupled with the patients perception of poor wound healing was classified as poor wound healing.

Xerostomia was assessed by asking the subjects the following questions:

1. Does your mouth constantly feel dry?
2. Do you ever have difficulties swallowing foods if you don't drink water as you eat?
3. When eating your meal does your mouth often feel dry?

Subjects who responded positively for 2 or more of these questions coupled with a positive intraoral examination for dryness were classified as having xerostomia.

Halitosis was regarded a problem only if the patient thought they have a bad breath and if more than one person has complained about their bad breath. This was assessed by smelling the patient's exhaled air as well as the oral cavity for verification by the researcher.

Healthy Eating was assessed by asking the following questions:

1. How many meals do you eat in one day?
2. How often do you eat "take-aways" in one week?
3. When preparing your meal you

a. fry with cooking oil.
b. boil with little or no salt

c. grill with olive oil

d. Remove obvious fat tissue or chicken skin before boiling.

4. Which one of these food groups does your daily meal contain?

   a. protein

   b. fat

   c. carbohydrate

   d. vegetables

Subjects who consumed 3 meals or more and ate a balanced diet, with an effort to limit oil were regarded as healthy eaters.

To evaluate **how life changed after a diagnosis of diabetes** subjects were asked to speak about those symptoms which they experienced only after being diagnosed with diabetes. These symptoms were then grouped as neuropathy, nephropathy, cardiovascular, perioral, peripheral vascular disease or retinopathy according to signs and symptoms.

4.9.6 Treatment guidelines for patients

Treatment requirements will be assessed for the whole tooth, including both coronal and root caries. Immediately after the status of a tooth is recorded, and before proceeding to the next tooth, the type of treatment required, if any, will be recorded. If no treatment is required, code 0 will be assigned next to the tooth. The codes and criteria for treatment needs will be as follows:

0= *No treatment* .This code applies to a crown and a root which are both sound and therefore should not receive any treatment.

P= *Preventive, caries arresting care*

F= *Fissure sealant*

1= *One surface filling*

2= *Two or more surface filling*

Code P, F, 1 or 2 will be used to indicate treatment required to:

- Treat initial, primary or secondary caries
- Treat discolouration of a tooth, or a developmental effect

- Treat lesions due to trauma, abrasion, erosion or attrition
- Replace unsatisfactory fillings or sealants

A sealant will be considered unsatisfactory if partial loss has extended to exposure of a fissure, pit or junction or surface of the dentine which requires resealing.

A filling will be considered unsatisfactory of one or more of the following conditions exist:

- *a deficient margin* to an existing restoration that has leaked or is likely to permit leakage into the dentine
- *an overhanging margin* of an existing restoration that is causing obvious local irritation to the gingival and cannot be removed by recontouring of the restoration.
- *a fracture* of an existing restoration that either causes it to be loose or permits leakage into the dentine
- *discolouration*

3 *Crown for any reason*

4 *Veneer or laminate*

5 *Pulp care and restoration*. This code will be used to indicate that a tooth probably needs pulp care prior to restoration with a filling or crown because of deep extensive caries or because of trauma.

6 *Extraction.* A tooth will be recorded as indicated for extraction when caries has destroyed the tooth and it cannot be restored

Periodontal disease has progressed so far that the tooth is loose, painful or functionless, and cannot be restored to a functional state.

A tooth needs to be extracted for a prosthesis

Extraction is required for cosmetic reasons or because of impaction

7 *Fillings.* A tooth will be recorded as indicated for a filling when:

- Evidence of caries exists on the crown or root and no pulpal involvement is suspected.

8 Patients were referred for scaling and polishing where gross calculus deposits, tooth mobility and dental plaques have been identified

- Where there's evidence of inflammation of the gingival due to the presence of bacterial plaque or calculus.

9. Oral pathology. This code will be given to all patients presenting with oral pathology lesions and such patients will be referred for further investigations.

4.10 Intra-examiner calibration

For the oral examination there was only one examiner. Consistency was determined by examining a randomly selected group of 10 patients twice, on successive days. These patients possessed, collectively, the full range of conditions expected to be assessed in the study. By comparing the results of the two examinations, the examiner was able to obtain an estimate of the extent and nature of the diagnostic errors. If the level of agreement between the examinations did not meet the recommended minimum level, the examiner reviewed the interpretation criteria and additional calibration examinations were conducted until an acceptable consistency was achieved. Agreements were in the range of 85-90%.

4.11 Data analysis

The demographic and DMFT characteristics of the study participants were summarized for both groups. Some of the characteristics considered were age, gender, race, plaque index, number of teeth decayed, number of teeth missing and number of teeth filled. Continuous variables were presented using the following summary statistics, number of patients (n) mean, standard deviation (sd), minimum (min) median and maximum (max).

Categorical variables were presented using frequencies and percentages for each response category were given for diabetic and non-diabetic patients. Continuous variables were compared using t-tests, whereas categorical variables were compared using chi-squared test or Fisher's exact test.

The level of significance used for the comparisons in this study was 5%. Logistic regression techniques were used to examine the effect of diabetes on self perceived quality of life controlling for age, gender, healthy eating plan and exercising activities. The magnitude of the risk was assed using odds ratios (OR). The percentages of diabetic and non-diabetic were compared by calculating a point estimate (OR) and 95% confidence interval for the risk difference.

4.12 Validity and reliability

The principal investigator (fluent in English and isiZulu) was the only investigator involved in conducting interviews, keeping records, gathering and interpretation of data, thereby assuring confidentiality and the standardized recording of information.

4.13 Ethical considerations

The study was approved by the Ethics Committee at Prince Mshiyeni Memorial Hospital as well as the Provincial Health Research Committee at the Health Research and Knowledge Management sub-component for KwaZulu-Natal Department of Health and the Senate Research Committee, University of the Western Cape. The participants in the study were required to complete a consent form (Appendix II) if they were willing to participate in the study. Participation was voluntary and each individual had the right to refuse to be included in the study or to withdraw from the study at any time. Furthermore, it was re-iterated that their decision to participate or not, will not affect their management or care in any way whatsoever. The participants were assured of confidentiality regarding the information they provide should they decide to take part. The CPITN attachment loss and mobility indices were not performed in this study due to lack of resources. However, all the patients with a recession and tooth mobility were referred to the dental clinic at the same hospital.

CHAPTER FIVE: RESULTS

The study findings are discussed in this chapter. Demographic information is presented followed by a comparison of oral health status between diabetic and non-diabetic patients, comparison of oral problems and dietary habits between the disease status categories, a description of diabetic-specific oral health characteristics and finally the impact of diabetes on the self perceived quality of life.

5.1 Demographic information

Table 2 shows the demographic information of the study participants by diabetic diseases status. The study consisted of 300 patients (150 diabetic and 150 non-diabetic). Most study participants (90%) were black African and nearly 77% of the participants in each group were females. The gender distribution was similar for diabetic and non-diabetic groups (p=0.891). The age ranged from 32 to 74 years and 25 to 75 years for diabetic and non-diabetic patients respectively. The difference in average age was statistically significant (p<0.0001) with the average amongst non-diabetics being younger than the diabetic group.

Table 2
Demographic information by diabetic disease status

		Disease status				
		Diabetic (n=150)		Non-diabetic(n=150)		p-value
Variable	**Category**	**Freq**	**%**	**Freq**	**%**	
Gender						0.891
	Male	34	22.67	35	23.33	
	Female	116	77.33	115	76.67	
Race						0.562
	African	148	98.67	149	99.33	
	Indian	2	1.33	1	0.67	
Age						
	Mean± S.D		55.3±8.5		42.3±3.2	<0.0001
	Min		32		25	
	Q1		49		31	
	Q3		61		51	
	Median		55		41.5	
	Max		74		75	

Table 3 shows the plaque index between diabetic and non-diabetic patients. There was a statistically significant association between plaque index and diabetic status ($p<0.0001$). A higher percentage of diabetic patients had a plaque index of greater than 1/3 compared to non-diabetic patients (61% vs. 23%)

Table 3
Plaque index by diabetic disease status

		Disease status				
		Diabetic (n=150)		Non-diabetic(n=150)		p-value
Variable	**Category**	**Freq**	**%**	**Freq**	**%**	
Plaque Index						<0.0001
	None	7	4.67	44	29.33	
	<1/3	51	34.00	71	47.33	
	>1/3	92	61.33	35	23.33	
	Max		74		75	

The total number of decayed, missing or filled teeth among diabetic and non-diabetic patients is presented in Table 4. The mean DMFT was higher for diabetic patients compared to non-diabetic patients (7.2 vs 5.4) with a median of 6 for diabetic patients and 4.5 for non-diabetic patients. The difference in the DMFT was statistically significant (p= 0.0019). On average, the number of teeth missing was 5.5 for diabetic patients and 3.2 for non-diabetic patients and this was statistically significant.

Table 4
The number of teeth decayed, filled or missing by diabetic diseases status (DMFT)

Variable	Statistic	Diabetic (n=150)	Non-diabetic (n=150)	p-value
Total number of teeth with problems (DMFT)	**Mean± sd**	7.2± 5.5	5.4±4.2	0.0019
	Min	0.0	0.0	
	Q1	3.0	2.0	
	Q3	10.0	7.0	
	Median	6.0	4.5	
	Max	21.0	20.0	
Number of teeth decayed				
	Mean± sd	1.4± 1.7	1.4±1.6	0.9458
	Min	0.0	0.0	
	Q1	0.0	0.0	
	Q3	3.0	2.0	
	Median	1.0	1.0	
	Max	11.0	8.0	
Number of teeth missing				
	Mean± sd	5.5±5.1	3.2±3.2	<0.0001
	Min	0.0	0.0	
	Q1	1.0	1.0	
	Q3	8.0	5.0	
	Median	4.0	2.0	
	Max	20.0	16.0	
Number of teeth filled				
	Mean± sd	0.2±0.7	0.8±1.7	<0.0001
	Min	0.0	0.0	
	Q1	0.0	0.0	
	Q3	0.0	1.0	
	Median	0.0	0.0	
	Max	6.0	9.0	

Note: These figures were rounded off to one decimal place.

5.2 Oral Health Status

Figure 2 shows a comparison of the appearance of marginal gingiva between diabetic and non-diabetic patients. More than half (54%) of the diabetic patients had a moderate gingival index compared to 23% of the non-diabetic patients. The majority of non-diabetic patients were in the mild category (39.3%). The distinct red band and severe categories (both an indication of severe inflammation of the marginal gingiva for this study) were more prevalent in the diabetic patients compared to non-diabetic patients (19.3% vs. 6.7%) and (3.3% vs. 0.7%) respectively.

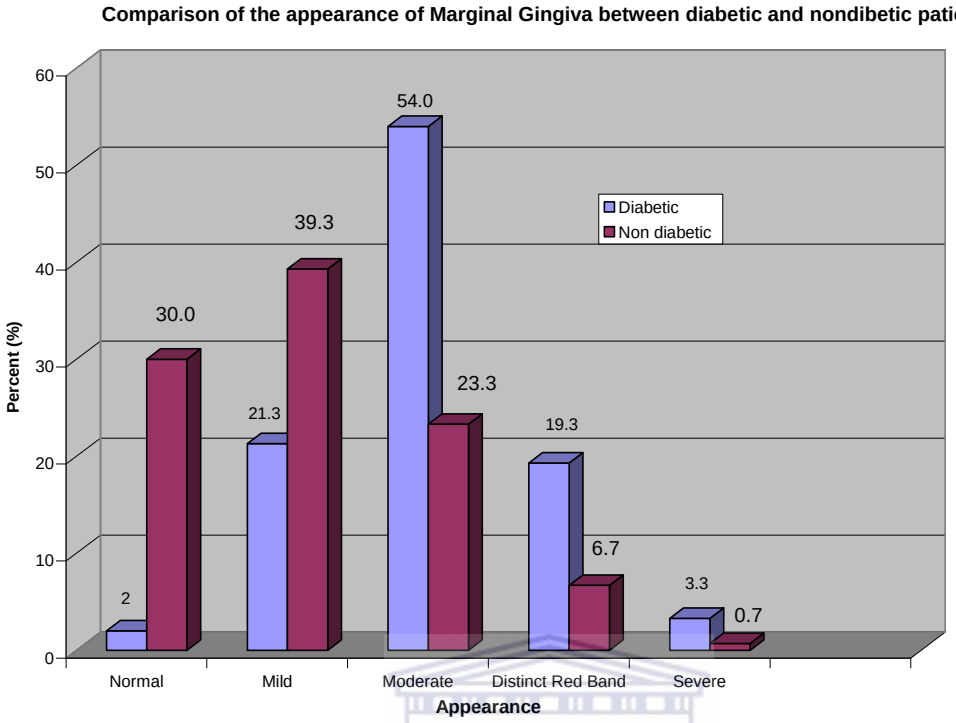

Figure 2: Comparison of the appearance of marginal gingiva between diabetic and non-diabetic patients

Figure 3 depicts a comparison of reported oral lesions between diabetic and non-diabetic patients. A total of 182 oral lesions were observed among diabetic patients and 151 among the non-diabetic patients (Appendix III). The number of oral pathology lesions was expressed as a percentage of 150 patients and these are compared in Figure 3. The oral lesions commonly observed among the diabetic patients compared to non-diabetic patients were candida (41.3% vs 8.0%), dental abscess (16.0% vs 2.7%), and ulcers (14.0% vs 2.0%). Nearly 90% of non-diabetic patients did not have any lesion compared to 43% of the diabetic patients. Some patients presented with more than one lesion.

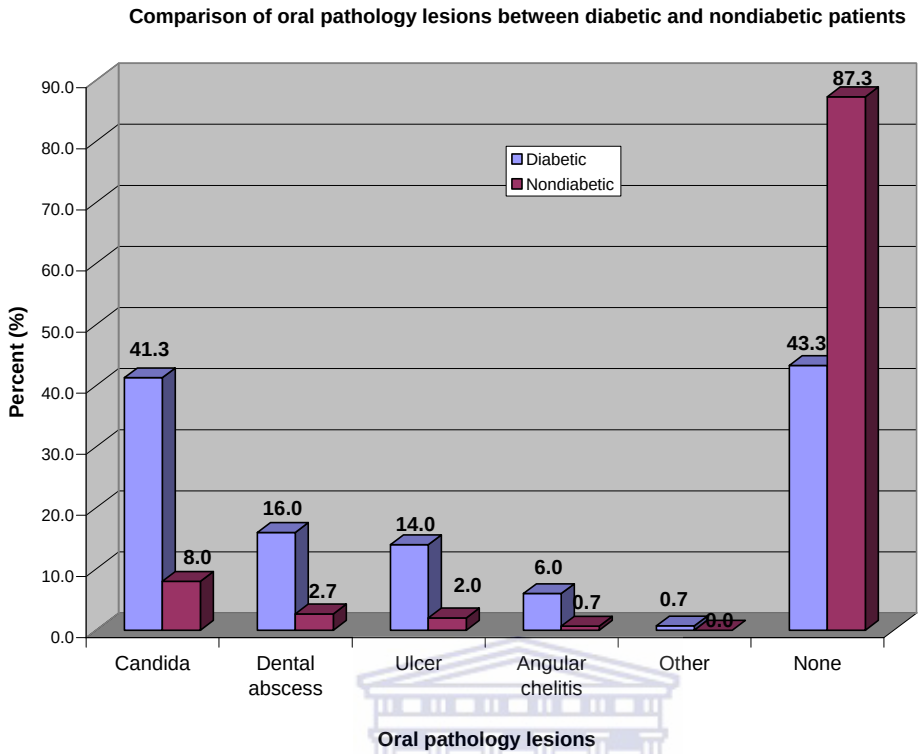

Figure 3: Comparison of oral lesions between diabetic and non-diabetic patients

A comparison of oral problems experienced between diabetic and non-diabetic patients is shown in Figure 4. A total of 328 oral lesions were observed among diabetic patients and 190 among the non-diabetic patients (Appendix III). The number of problems within the oral cavity was expressed as a percentage of 150 patients and these are compared in Figure 4. The most prevalent oral cavity problems were periodontal disease (measured by recession and mobility during oral examination/charting) and xerostomia among the diabetic patients. Tooth decay was more common among the non-diabetic patients. Periodontal problems as well as xerostomia were reported by more than half of the diabetic patients, as oral cavity problems, followed by tooth decay (49.3%). In 58% of the 150 non-diabetic patients tooth decay was the main oral cavity problem followed by periodontal disease (14.7%).

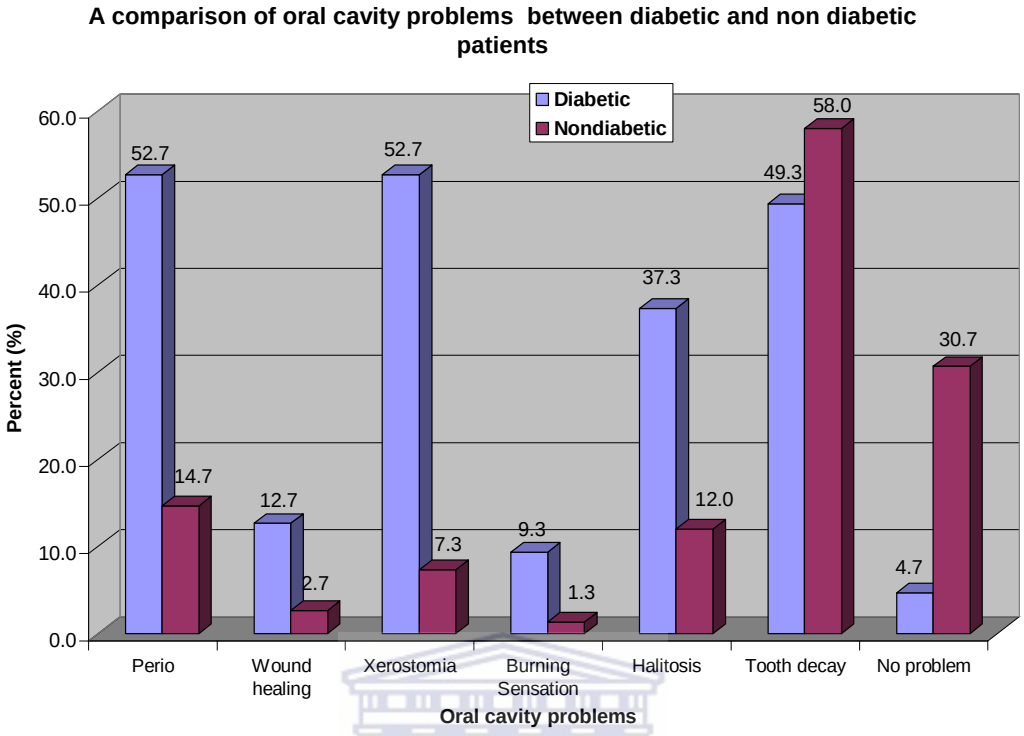

Figure 4: Comparison of oral cavity problems between diabetic and non-diabetic patients

Table 5 shows a comparison of the frequency of oral function problems between diabetic and non-diabetic patients. There was a statistically significant association between pain in mouth and diabetic status (p=0.004). A higher percentage of diabetic patients often reported having pain in the mouth "often" compared to non-diabetic patients (22.0% vs 11.3%) with one third having pain "sometimes" among diabetics compared to nearly one quarter of the non-diabetic patients.

Teeth problems were experienced "sometimes" by nearly 41% of the diabetic patients compared to nearly 29% of the non-diabetic patients. The purpose of this question was to assist the researcher in case of any teeth-related problems that may have been missed during the intra-oral examination. Teeth problems were defined as caries, mobility, sensitivity or toothache. Where possible the responses were verified by the researcher using a probe or air syringe.

The percentage that reported experiencing teeth problems "often" were almost similar between the two groups (19% vs 18%). The association between the frequency of experiencing teeth problems and diabetic status is not statistically significant (p=0.053). There was a statistically significant association (p<0.001) between the frequency of gum problems and the diabetes status. Problems of the gums were defined as gums that were swollen, bleeding when brushing, painful, spontaneous bleeding, hyperplastic or suppurated and the responses were checked against the clinical presentation. Gum problems were experienced more "often" by diabetic patients compared to non-diabetic (18.7% vs 4.7%). A quarter (34%) of diabetic patients "sometimes" experienced gum problems compared to nearly 21% of the non-diabetic patients.

Table 5
Comparison of oral function by diabetes status

Variable		Diabetic (n=150)		Non-diabetic (n=150)		
	Categories	**Freq**	**%**	**Freq**	**%**	**p-value**
Pain in mouth						0.004
	Never	67	44.67	94	62.67	
	Sometimes	50	33.33	39	26.00	
	Often	33	22.00	17	11.33	
Problems with teeth						0.053
	Never	58	38.93	80	53.33	
	Sometimes	61	40.94	43	28.67	
	Often	29	19.46	27	18.00	
Do you have problems with gums						<0.0001
	Never	71	47.33	112	74.67	
	Sometimes	51	34.00	31	20.67	
	Often have	28	18.67	7	4.67	

5.3 Oral problems and dietary habits

Figure 5 shows a comparison of the problems associated with the teeth between diabetic and non-diabetic patients. A total of 177 oral problems were reported (and verified by the researcher) among diabetic patients and 174 among the non-diabetic patients (Appendix III).

Problems of caries, sensitivity, toothache and mobility were verified by the researcher. The number of oral problem citations was expressed as a percentage of 150 patients and these are compared in Figure 5. The teeth problem reported most was caries where 34% was reported by diabetic patients and 28% by non-diabetic patients. Toothache was reported by 21.3% and 23.3% of diabetic and non-diabetic patients respectively. Sensitivity (whether due to caries, abrasion or recession) was reported by 12.0% of diabetic and 9.3% of non-diabetic patients. Mobile teeth (measured by applying two blunt ends of metal instruments for horizontal mobility and applying one blunt end on the occlusal or incisal edge for vertical mobility) were more common among diabetic patients compared to non-diabetic patients (11.3% vs 2.0%).

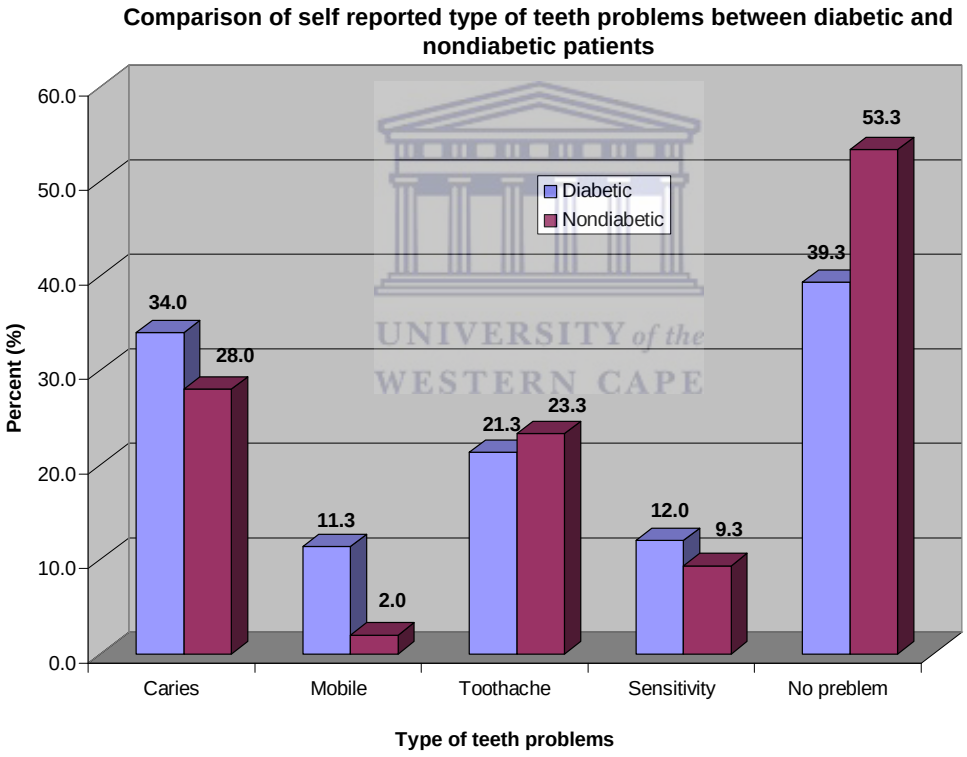

Figure 5: A comparison of the self reported type of teeth problems between Diabetic and

Non-diabetic patients.

A comparison of gum problems (defined as bleeding gums, swollen gums, painful gums without any inflammation or gums with spontaneous bleeding) between diabetic and non-diabetic patients is presented in Figure 6. A total of 161 gum problems were reported

among diabetic patients and 158 among the non-diabetic patients (Appendix III) since some patients reported having more than one problem. The reported number was expressed as a percentage of 150 patients and these are compared in Figure 6. The most common gum problem reported was bleeding with 38.7% by diabetic patients and 22.0% by non-diabetic patients, followed by swollen gums (8.7% and 2.0%). Painful gums were reported by 6.7% of diabetic and 5.3% of non-diabetic patients. Spontaneous bleeding was more common among diabetic patients compared to non-diabetic patients (6.0% vs 1.3%).

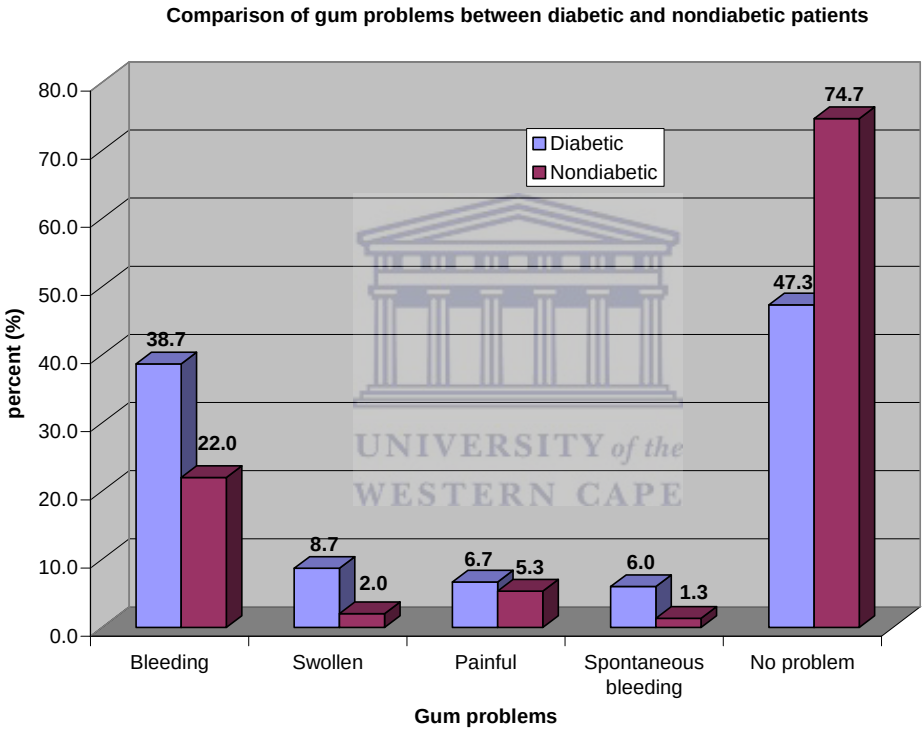

Figure 6: A comparison of self-reported gum problems between diabetic and non-diabetic patients.

Table 6 displays a comparison of dietary characteristics between diabetic and non-diabetic. Healthy eating was defined as the following: Subjects who consumed 3 meals or more per day and ate a balanced diet, with an effort to limit the amount of oil (See Section 4.8.5). There was a statistically significant association between a reported healthy eating plan and diabetes status ($p<0.0001$).

A higher proportion of diabetic patients followed a healthy eating plan compared to non-diabetic patients (76.7% vs 35.3%). Nearly one third of the diabetic patients began a healthy eating plan more than ten years ago compared to 6% of the non-diabetic patients. This difference was statistically significant (p<0.0001).

Table 6

A comparison of dietary characteristics between Diabetic and Non-diabetic

		Diabetic		Non-diabetic		
		Freq	**%**	**Freq**	**%**	**pvalue**
Do you follow a healthy eating plan?						<0.0001
	No	35	23.33	97	64.67	
	Yes	115	76.67	53	35.33	
When did you start following a healthy eating plan?						<0.0001
	Never	33	22.00	91	60.67	
	0 to 4 years	32	21.33	35	23.33	
	5 to 9 years	36	24.00	15	10.00	
	>10 years	49	32.67	9	6.00	
Is it beneficial to eat healthy when diabetic?						0.149
	No	2	1.33	0	0.00	
	Yes	147	98.00	146	97.33	
	Unsure	1	0.67	4	2.67	

5.4 Exercising characteristics and perceived quality of life

Exercising characteristics among diabetic and non-diabetic patients are shown in Table 7. There was a statistically significant association between following an exercising routine and diabetic status (p=0.015). A higher proportion of diabetic patients reported following an exercising routine compared to non-diabetic patients (55.3% vs 38.7%). The frequency of exercise during the previous month was not statistically significantly associated with diabetic status (p=0.086). A higher proportion of diabetic patients exercised more (for one day or more) during the previous month compared to non-diabetic patients (58.7% vs 45.7%). There was a statistically significant association between "reason for exercise" and diabetic status (p=0.021).

Almost two thirds of diabetic patients reported that they exercised because they wanted to be healthy and fit compared to 43% of non-diabetic patients. There was a statistically significant association ($p<0.0001$) between perceived quality of life and diabetic status. Only a quarter of diabetic patients perceived themselves as 'very satisfied' or "somewhat satisfied" with their quality of life compared to nearly 95% of non-diabetic patients. The majority of diabetic patients (86%) were "not satisfied" with their perceived quality of life. The reasons cited for this were those of medical complications associated with being diabetic.

Table 7
A comparison of exercising characteristics and perceived quality of life between Diabetic and Non-diabetic patients

		Diabetic		Non-diabetic		
		Freq	**%**	**Freq**	**%**	**p-value**
Do you follow any exercising routine?						0.015
	No	61	40.67	83	55.33	
	Yes	83	55.33	58	38.67	
	Sometimes	6	4.00	9	6.00	
How many times did you exercise last month?						0.086
	None	62	41.33	81	54.00	
	1 to 10 days	13	8.67	7	4.67	
	10 to 20 days	15	10.00	17	11.33	
	20 to 30 days	60	40.00	45	30.00	
Reason for exercising						0.021
	Don't exercise	59	39.33	82	54.67	
	Health and fitness	88	58.67	64	42.67	
	Weight loss	3	2.00	4	2.67	
Is it important to exercise when diabetic?						0.002
	No	4	2.67	0	0.00	
	Yes	146	97.33	142	94.67	
	Unsure	0	0.00	8	5.33	
Perceived quality of life						<0.0001
	Very satisfied	6	4.00	131	87.33	
	Somewhat satisfied	31	20.67	12	8.00	
	Not satisfied	113	75.33	7	4.67	
Reasons for perceived quality of life (QOL)						<0.0001
	Accepted my diabetes status	18	12.00	0	0.00	
	Diabetes complications	129	86.00	0	0.00	
	Other	3	2.00	150	100.00	

5.5 Description of oral health characteristics among diabetic patients

This section reported on the diabetic patient sample. Table 8 presents the results of their oral health characteristics. These patients experienced problems with their sense of taste with a quarter experiencing the problem "sometimes" and 3% experiencing the problem "often". Nearly 80.5% of the 41 patients who indicated having sense of taste problems experienced a bitter taste. Almost all (97%) of diabetic patients indicated that being diabetic had changed their life. A third (35%) indicated that their life changed because they had developed 3 or more diabetes complications while 44% indicated that their life had changed because they had developed 2 or more complications.

Table 8
Oral health among Diabetic patients only

		Frequency	Percent
Altered sense of taste			
	Never	109	72.67
	Sometimes	37	24.67
	Often	4	2.67
Type of sense of taste (n=41)			
	Bitter taste	33	80.49
	Certain foods funny	6	14.63
	Not applicable	2	4.88
History of oral problems			
	Before diabetes	16	10.67
	After diabetes	129	86.00
	Not applicable	5	3.33
Has being diabetic changed your life in anyway?			
	Yes	146	97.33
	No	4	2.67
How did your life change after becoming diabetic?			
	3 or more complications	53	35.33
	2 complications	66	44.00
	1 complication	26	17.33
	No complications	5	3.33

5.6 Length of time diagnosed with diabetes.

The length of time since the diagnosis of diabetes is shown in Figure 7. Most of the diabetic patients had been diagnosed with diabetes for more than 5 years while 22% had been diagnosed for a period of 3 to 5 years.

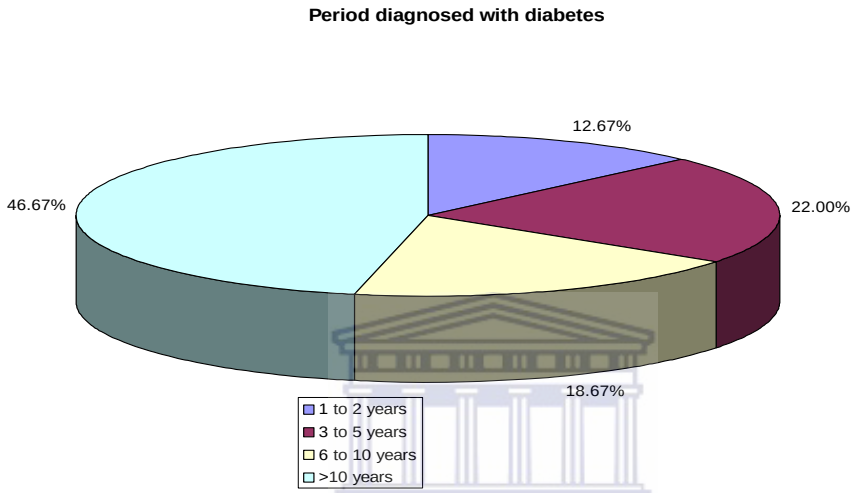

Figure 7: The length of time diagnosed with diabetes

The type of the diabetes medication taken by the participants is shown in Figure 8. Three quarter of the diabetic patients were taking oral hypoglycemic medication alone, 17% were taking a combination of insulin and oral hypoglycemics and only 8% were taking insulin alone.

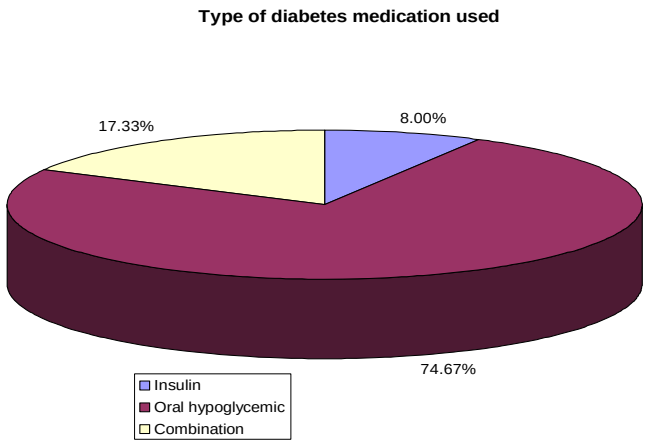

Figure 8: Type of diabetes medication taken by the diabetic participants

5.7 Self perceived quality of life of the entire study sample

This section reports on the factors that influence self perceived quality of life among all the study participants. Self perceived quality of life (PQOL) was measured using a question "How do your perceive your quality of life?" with responses "Very satisfied=3" "Somewhat satisfied=2" and "Not satisfied=1". To explore the effect of diabetes on the probability of being "Very satisfied" with QOL and "Not satisfied" with QOL adjusting for age and gender, a multinomial logistic regression model was used with the reference group being "Somewhat satisfied".

Table 9 shows the factors included in the model and the reference group, the odds ratio (OR) and their corresponding 95% confidence intervals (CI) and p-value. Diabetic patients were statistically significantly less likely to perceive their quality of life as very satisfied after adjusting for age and gender (OR=0.0188; CI: 0.0059-0.0594). Furthermore, diabetic patients were almost six times more likely to perceive their life as not satisfied compared to non-diabetic. This perception was statistically significant (2.0073- 17.8047).

Table 9
Multinomial logistic regression model of the impact of diabetic on PQOL adjusting for age and gender

	PQOL	OR	Wald statistic p-value	95% CI for OR
Ref: Somewhat satisfied	**Very satisfied**			
Ref: Non-diabetic	Diabetic	0.0188	0.000	0.0059- 0.0594
	Age	0.9867	0.488	0.9502- 1.0247
Ref: Male	Female	0.5892	0.355	0.1920- 1.8082
Ref: Somewhat satisfied	**Not satisfied**			
Ref: Non-diabetic	Diabetic	5.9782	0.001	2.0073-17.8047
	Age	1.0134	0.501	0.9748- 1.0536
Ref: Male	Female	2.0510	0.093	0.8864- 4.7458
Log likelihood value		-166.29095		

Table 10 shows the results of the multinomial model adjusting for other confounding factors: a healthy eating plan and exercising. It presents factors included in the model and the reference group, the odds ratio (OR) and their corresponding 95% confidence intervals (CI) and p-value. Diabetic patients were again statistically significantly less likely to perceive their quality of life as very satisfied (OR=0.0245; CI: 0.0071-0.0843).

The inclusion of healthy eating plan and exercising changed the magnitude of the effect of being diabetic: diabetic patients were now almost 10 times more likely to perceive their life as not satisfied compared to non-diabetic. This perception was statistically significant (2.7368- 33.3969). After adjusting for the other factors in the model, it was found that patients who do not exercise were significantly less likely to perceive their quality of life as being not satisfied compared to those patients who exercise.

Table 10
The result of a multinomial logistic regression model of the impact of diabetic on PQOL adjusting for age, gender, exercise and healthy eating plan

	PQOL (n=300)	Odds Ratio	Wald Statistic p-value	95% Cl for OR
Ref: Somewhat satisfied	**Very satisfied**			
Ref: Non-diabetic	Diabetic	0.0245	0.000	0.0071- 0.0843
Ref: No	Healthy eating plan-Yes	0.4303	0.122	0.1477- 1.2531
Ref: Male	Female	0.6183	0.409	0.1977- 1.9337
	Age	0.9874	0.522	0.9497- 1.0265
Ref: Yes exercise	No Exercise	0.8712	0.791	0.3146- 2.4129
	Somewhat exercise	0.3801	0.262	0.0702- 2.0587
Ref: Somewhat satisfied	**Not satisfied**			
Ref: Non-diabetic	Diabetic	9.5603	0.000	2.7368-33.3969
Ref: No	Healthy eating plan-Yes	0.2212	0.005	0.0764- 0.6401
Ref: Male	Female	2.3711	0.053	0.9884- 5.6881
	Age	1.0208	0.312	0.9808- 1.0624
Ref: Yes exercise	No Exercise	0.3627	0.017	0.1572- 0.8368
	Somewhat exercise	0.3474	0.200	0.0690- 1.7489
Log likelihood value		-159.27419		

A logistic regression model was then used to determine the impact of diabetes on PQOL after combining "very satisfied" and "somewhat satisfied" responses into one group called "satisfied". This resulted in a dichotomous outcome variable PQOL with only two responses, "satisfied" and "not satisfied".

Table 11 shows the factors included in the logit model and the reference group, the odds ratio (OR) and their corresponding 95% confidence intervals (CI) and p-value. The effect of diabetes on PQOL drastically increased compared to the effect of the multinomial logistic regression. Diabetic patients were now almost 91 times more likely to perceive their quality of life as being "not satisfied" compared to non-diabetic patients. However this estimate lacked significance due to the large confidence intervals.

Table 11

Logistic regression model of the impact of diabetic on PQOL adjusting for age, gender, exercise and healthy eating plan

Ref: Satisfied	Not satisfied	Odds Ratio	Wald Statistic P-value	95% CI for OR
Ref: Non-diabetic	Diabetic	90.5158	0.000	29.8594- 274.3896
Ref: No plan	Healthy eating plan	0.30338	0.015	0.11559 - 0.7963
Ref: Male	Female	2.65718	0.017	1.1898 - 5.9345
	Age	1.02551	0.170	0.9893 - 1.0631
Ref: Yes Exercise	No Exercise	0.38359	0.014	0.1790 - 0.8218
	Sometimes exercise	0.45849	0.355	0.08790 - 2.3916
Log likelihood		-104.38441		

5.8 Summary

The results of the present study indicated that there are statistically significant differences in oral health problems in diabetics. Diabetic patients displayed a higher prevalence of oral pathology lesions, poor oral hygiene status as well as a higher gingival index. There amount of decayed teeth in the diabetic patients were no different from the diabetic group. The average DMFT was significantly higher in the diabetic group compared to the non diabetic group. A higher proportion of diabetic patients indicated that they were following a healthy eating plan and had been diagnosed with diabetes for a period longer than five years. Nearly 75% of diabetic patients used oral hypoglycemic drugs to control the disease. Most diabetic patients were not satisfied with their quality of life when compared to non-diabetic patients.

CHAPTER 6: DISCUSSION AND CONCLUSION

The aim of the present study was to compare the oral health problems in a sample of 150 diabetic and 150 non-diabetic patients attending at Prince Mshiyeni Memorial Hospital, Durban, KwaZulu-Natal. In this study, diabetic patients exhibited poorer oral health when compared to their non-diabetic counterparts. Diabetic patients presented with more oral lesions, as is expected, due to the attenuated immunity caused by diabetes.

Almost all of the participants were black. This was because the study setting mainly offers services in a predominantly black community. The study indicated that oral health problems were more common among the middle-aged people. The study findings consistently revealed that oral health problems were more prevalent among diabetic patients compared to non-diabetic patients.

Nearly two thirds of diabetic patients had a Plaque Index greater than one third. These results are similar to Sandberg et al (2000) where 2 diabetics were compared to non-diabetics and the results showed that the diabetic patients exhibited more surfaces with bacterial plaque than non-diabetic patients, after using the Plaque Control Record method of measuring plaque (Sandberg et al. 2000).

The mean DMFT was statistically different between the two groups and this concurs with Zielinski et al (2002) where the mean vales of DMFT were higher in the diabetic group compared to the non-diabetic group. In the present study, diabetic patients had a significantly higher average number of missing teeth compared with non-diabetic ones, unlike Zielinski et al (2002) who reported that the mean number of missing teeth was slightly higher among the non-diabetic group, although not statistically significant (Zielinski et al. 2002).

There was a statistically significantly higher number of oral problems (candida, dental abscess and periodontitis) reported by the diabetic patients compared to non-diabetics. This difference may be due to the immune-suppression caused by diabetes.

Lamster et al (2008) indicated that the presence of candidiasis is a common manifestation of immuno-compromised state and these acute oral infections are frequent complications of marginally controlled or uncontrolled diabetes.

Periodontal disease and tooth decay were reported as the most common oral cavity problems among diabetic and non-diabetic patients respectively. Periodontal disease is a recognized and well-documented complication of diabetes (Lamster, et al. 2008). In a review done by Taylor and Borgnakke (2008) two out of three studies where patients were not classified as Type 1 or 2 diabetes reported a greater prevalence, extent and severity of periodontal disease for at least one measure or index of periodontal disease. Mealey (2003) and Vernillo (2003) found that increased gingival crevicular fluid glucose levels were much higher in the diabetic patients, and this may diminish the ability of periodontal fibroblasts to contribute to periodontal healing. This may indicate that the prevalence of moderate to severe gingival inflammation was generally high among diabetic patients compared to non-diabetic patients. Ogunbodede et al (2005) found a higher average gingival index and a higher plaque index among diabetic patients compared to non-diabetic patients.

In this thesis, problems with the teeth were either experienced “sometimes” or “often” more by the diabetic patients compared to just less than half of the non-diabetic patients. Pain in the mouth was the most reported problem among diabetics. These findings may be directly related to the fact that diabetic patients exhibited more caries, periodontal problems and oral pathology which are all associated with pain and discomfort.

The most common teeth problems reported by diabetic patients and non-diabetic patients were caries and tooth ache respectively. The literature shows a lack of consistency regarding the relationship between diabetes and dental caries. Some investigators (Lamster et al. 2008; Mealey, 2003 and Zielinski et al. 2002) observed higher levels of dental caries among the diabetic patients. In some studies, the increased rates of dental caries were associated with xerostomia or increased gingival crevicular fluid glucose levels (Mealey, 2003).

In others, the increased rate of caries was attributed partially to decreased salivary flow and increased levels of carbohydrates in the parotid saliva (Murrah, 1985).

The most commonly reported gum problem was bleeding, followed by painful gums. Because gingivitis is a reversible inflammatory condition, it is characterized by bleeding and this could be indicative of the fact that diabetic patients had more severe gingival inflammation. Tan et al (2006) found that diabetic patients had poorer oral hygiene as measured by plaque index, more severe gingival disease as measured by the gingival index and higher severity of periodontal disease based on probing depths and clinical attachment levels compared to non-diabetic patients.

Most diabetic patients indicated that they experienced problems with sense of taste. Taste disturbances have been reported in patients with DM, although not by all researchers. Its occurrence is thought to be due to reduced salivary flow and changes in food intake associated with diabetic disease management (Lamster et al. 2008).

Diet, exercise, weight control and medication are the mainstays of diabetic care (Mealey, 2003). A higher proportion of diabetic patients followed a healthy eating plan compared to non-diabetic patients. This may be due to the health education instructions on healthy eating and exercising, as part of the overall management of diabetic patients in order to keep diabetes under control. During patient education, health workers often emphasize the need to exercise and eat healthily and results of the present study perhaps indicated how well the health workers have managed to educate diabetic patients in this area of disease management. Diabetic patients reported exercising because they wanted to be healthy and fit. Diabetic patients may be exercising more than non-diabetic patients in order to comply with proper management of their diabetes status as a recommendation from their physicians.

Nearly all the diabetic patients perceived their quality of life as “not satisfactory” due to the complications of diabetes compared to less than five per cent of non-diabetic patients. There is much morbidity and mortality is associated with diabetes and as such diabetes complications have important effects on the patients’ quality of life. Papadopoulos et al (2007) found that nearly all their study sample suffered from co-morbidity. In the present study, almost all of diabetic patients indicated that being diabetic had changed their life for the worse. Perhaps the fact that diabetes affects multiple organ systems had an impact on how diabetic patients perceived their life once they had been diagnosed with diabetes.

Almost all the diabetic patients acknowledged that their life changed after being diagnosed with diabetes. The majority of diabetic patients started experiencing oral health problems after being diagnosed as a diabetic and reported having at least one diabetic complication. Three quarters of the diabetic patients were taking oral hypoglycemic drugs to control their condition. A higher proportion of diabetic patients were “not satisfied” with their quality of life, and this perception could be due to the medication, dietary restrictions and the actual symptoms of the disease, all of which lead to a deterioration in QOL (Papadopoulos et al. 2007). Logistic regression models indicated that diabetic patients were almost six times likely to perceive their life as “unsatisfactory” compared to non-diabetic after having adjusted for age and gender variables. This difference in perception was statistically significant. After adjusting for age, gender, healthy eating and exercising characteristics, diabetics were now almost ten times more likely to perceive themselves as “not satisfied” with their QOL. This indicates that not exercising has an influence on the perceived quality of life, even though healthy eating habits may be practiced. This perception was statistically significant although the confidence interval was wide.

6.2 Conclusions

It can be concluded from the present study that diabetic patients had more oral health problems compared to non-diabetic patients. This study found that diabetic subjects do exhibit oral complications and should therefore be encouraged to consult dentists on a regular basis to ensure that their oral cavity is as healthy as possible. This will allow them to be compliant with the nutritional recommendations of a diabetic patient. The dental team could have a significant, positive effect on the oral and general health of patients with diabetes mellitus (Lamster et al. 2008). This may indicate the need for targeted intervention programs aimed at educating the diabetic patient on oral health problems.

Intervention strategies for diabetics are needed to assist patients in maintaining normo-glycaemia as this has an impact on the progression of diabetic complications and thus on the quality of life in general. Furthermore, aggressive management of periodontal disease in a patient with diabetes may diminish the inflammatory milieu's detrimental effects on diabetes control and the cardiovascular health of the patient (Skamagas et al. 2008).

There is a need for an increased collaboration between physicians and the entire dental team to enable more interaction in the management of diabetic patients and improve the outcomes for diabetics. The dentist can play a role by assisting the patient to maintain glycaemic control; by treating oral infections and by instructing the patient with diabetes to maintain rigorous oral hygiene and a proper diet (Vernillo, 2003). Furthermore, dentists can play a very crucial role in identifying individuals at risk or with poorly controlled diabetes because of associated conditions like periodontitis and candida which are consistent with diabetes-related conditioning of periodontal responses to plaque (Bjelland et al. 2002). The oral cavity may exhibit the first signs or symptoms of an undiagnosed or poorly controlled diabetic condition, so dentist and dental hygienists must be aware of these signs and symptoms and know what to do if they are present (Mealey, 2008). Although not pathognomic for diabetes, conditions such as multiple periodontal abscesses, mobile teeth including bone loss, inflammation spreading through the attached gingiva etc may result from uncontrolled hyperglycaemia (Bjelland et al. 2002).

Detection of these conditions provides a means for early diagnosis of systemic disease like diabetes since the dentist has to refer suspicious cases to a physician for further investigations.

There is a great need for oral health promotion strategies targeted at the community at large so that they can understand the intimate and bidirectional relationship that exists between diabetes and oral health, and be alert to any mouth problems. This would enable individuals to cope better should they develop diabetes and it would empower others to take better care of those family members who are diabetic. There's a need for future studies among diabetic patients which will measure the mobility index, loss of attachment, CPITN index glyceamic control and compare how the findings relate to the Type of diabetes, medication taken and the period that a patient has been diabetic.

6.3 Study limitations

Variables related to smoking, stress and access to health care were not recorded. The oral examination did not include a mobility index, loss of attachment or periodontal index to measure pocket depths. However, a longitudinal study design would be best to elicit accurate information regarding the development and progression of oral diseases especially periodontal disease. There is a paucity of South African studies carried out on this topic and therefore most of the literature and statistics used were based on literature done from countries abroad.

REFERENCES

Almas K, Al-Lazzam S and Al-Quadairi A. The effect of oral hygiene instructions on diabetic Type 2 male patients with periodontal diseases. J Contemp Dent Pract. 2003; 4 (3): 040-051.

Bender IB and Bender AB. Diabetes and the dental pulp. J Endodontics. 2003; 29(6):383-389.

Bjelland S, Bray P, Gupta N and Hirsch R. Dentists, diabetes and periodontitis. Australian Dental Journal.2002; 47(3):202-207.

Boyce JM and Shone GR. Effects of ageing on smell and taste. Postgrad Med J. 2006; 82:239-241.

Brunton SA. The changing shape of Type 2 diabetes. Medscape J Med. 2008; 10 (6):143-154.

Campus G, Salem A, Uzzau S, Baldani E and Tonolo G. Diabetes and periodontal disease: a case control study. J Periodontol.2005; 76: 418-425.

Clark C. Type 1 Diabetes Mellitus. CME. 2006; 24(10)578-585.

Crawford JM. The pancreas. In: Basic Pathology 6th ed. Eds. Kumar V, Cotran RS and Robbins SL. WB Saunders.Tokyo.1997: 557-575.

Das UN and Rao AA. Gene expression profile in obesity and Type 2 diabetes mellitus. Lipids Health Dis. 2007; 6: 35.

Dasanayake AP, Chhun N, Tanner ACR, Craig R.G, Lee M.J, Moore A.F and Norman R.G. Periodontal pathogens and gestational diabetes mellitus. J Dent Res.2008; 87(4):328-333.

Devendra D, Lui E and Eisenbarth G.S. Type 1 diabetes: recent developments.BMJ.2004; 328 (7442):750-754.

Ding Y, Kantarci A, Hasturk H, Trackman P.C, Malabanan and VanDyke TE. Activation of RAGE induces elevated O_2^- generation by molecular phagocytes in diabetes. J Leukoc Biol. 2007; 81 (2): 520-527.

Dotson CD, Zhang L, Xu H, Shin Y, Vigues S, Ott SH, Elson AET, Choi HJ, Shaw H, Egan JM, Mitchell BD, Li X, Steinle NI and Munger SD. Bitter taste receptors influence glucose homeostasis. Plos ONE. 2008; 3 (12):e3974.

Frier BM, Fisher M. Diabetes mellitus In: Davidson's principles and practices of medicine 20th ed. Eds Boon NA, Colledge NR and Walker B. Churchill Livingston. Edinburgh, 2006.

Greene JC and Vermillion JR. The simplified oral hygiene Index. J Am Dent Assoc.1964; 68 (7).

Gucciardi E, Wang SC, DeMelo M, Amaral L and Stewart DE. Characteristics of men and women with diabetes. Can Fam Physician. 2008; 54: 219-27.

Herring ME and Shah SK. Periodontal disease and control of diabetes mellitus. JAOA.2006; 106(7): 416-421.

Janket S, Jones JA, Meurman JH, Baird AE and VanDyke TE. Oral infection, hyperglycaemia and endothelial dysfunction. Oral Surg Oral Med Oral Pathol Oral Radiol Endod.2008; 105(2): 173-179.

Kapellas K, Slade G. The relationship between diabetes and oral health among Australian adults. Australian Dental Journal.2008; 53:93-96.

Kapp JM, Boren S.A, Yun S and LeMaster J. Diabetes and tooth loss in a national sample of dentate adults reporting annual dental visits. Prev Chronic Dis.2007; 4(3):A59.

Karima M, Kantarci A, Ohira T, Hasturk H, Jones VL, Nam B-H, Malaban P. Trackman C, Nadwey J.A and VanDyke T.E. Enhanced superoxide release and elevated protein kinase C activity in neutrophils from diabetic patients: association with periodontitis. J Leukoc Biol.2005; 78 (4):862-870.

Kidambi S, Patel S.B. Diabetes mellitus: considerations for dentistry. J Am Dent Assoc. 2008; 139:8S-18S.

Kim J, Amar S. Periodontal disease and systemic conditions: a bidirectional relationship.Odontology.2006; 94(1):10-21.

King GL. The role of inflammatory cytokines in diabetes and its complications. Journal of Periodontology. 2008; 79(8s):1527-1534.

Kirigia JM, Sambo HB, Sambo LG and Barry SP. Economic burden of diabetes mellitus in the WHO African region. BMC International Health and Human Rights.2009; 9:6.

Lalla E, Park DB, Papapanou P.N and Lamster IB. Oral disease burden in Northern Manhattan patients with diabetes mellitus. Am J Public Health.2004; 94(5):755-758.

Lamster IB, Lalla E, Borgnakke WS and Taylor GW. The relationship between oral health and diabetes mellitus. J Am Dent Assoc.2008; 139:19S-24S.

Ley SH, Harris SB, Mamakeesick M, Noon T, Fiddler E, Gittelsohn J, Wolever TMS, Connelly PW, Hegele RA, Zinman B and Hanley AJG. Metabolic syndrome and its components as predictors of incident type 2 diabetes mellitus in Aboriginal community. CMAJ. 2009; 180(6): 617-24.

Mansour AA and Abd-Al-sada N. Periodontal disease among diabetics in Iraq. Med Gen Med.2005; 7(3):2-7.

Mbokazi AJ. Diabetes mellitus: chronic complications. CME. 2006; 24(10):558.

Mealey B. Diabetes Mellitus. In: Burket's Oral Medicine. Diagnosis and Treatment.10th ed. Eds. Greenberg M.S and Glick M. BC Decker Inc. Ontario, 2003.

Mealey BL. The interactions between physicians and dentists in managing the care of patients with diabetes mellitus. J Am Dent Assos 2008; 139:4S-7S.

Moore PA, Weyant RJ, Mongelluzzo MB, Myers DE, Rossie K, Guggenheimer J, Hubar H, Block HM and Orchard T. Type 1 diabetes mellitus and oral health: assessment of tooth loss and edentulism. J of Public Health Dentistry. 1998; 58(2):135-142.

Moore PA, Weyant RJ, Mongelluzzo MB, Myers DE, Rossie K, Guggenheimer J, Hubar H, Block HM and Orchard T. Type 1 diabetes mellitus and oral health. J Periodontol. 1999; 70(4):409-417.

Murrah V.A. Diabetes mellitus and associated manifestations: a review. J Oral Pathol.1985; 14:271-281.

Orbak R, Simsek S, Orbak Z, Kavrut F and Colak M. The influence of Type 1 diabetes mellitus on dentition and oral health in children and adolescents. Yonsei Med J.2008; 49(3):357-365.

Ogunbanjo GA. Type 2 diabetes: an evidence-based approach to its management by the family practitioner. CME. 2006; 24(10):568-572.

Ogunbodede EO, Fatusi OA, Akintomide A, Kalawole K, Ajayi A. Oral health status in a population of Nigerian diabetics. J Contemp Dent Pract.2005; 6(4): 075-084.

Papadopoulos AA, Kontodimopoulos N, Frydas A, Ikonomakis E and Niakas D. Predictors of health-related quality of life in Type 2 diabetic patients in Greece. BMC Public Health. 2007; 7:186.

Rabi DM, Edwards AL, Southern DA, Svenson LW, Sargious PM, Norton P, Larsen ET and Ghali WA. Association of socio-economic status with diabetes prevalence and utilization of diabetes care services. BMC Health Serv Res. 2006; 6: 124.

Rees D. Periodontal management of the patient with diabetes mellitus. Periodontology 2000. 2000; 23:63-72.

Roed-Petersen B, Renstrup G. A topographical classification of the oral mucosa suitable for electronic data processing. Its application to 560 leukoplakias. Acta Odontol Scand.1969; 27: 681-95.

Sandberg GE, Sundberg HE, Fjellstrom CA and Wikblad KF. Type 2 diabetes and oral health: a comparison between diabetic and non-diabetic subjects. Diabetes Res Clin Pract.2000; 50:27-34.

Silness J, Löe H .Periodontal Disease in Pregnancy. II. Correlation between oral hygiene and periodontal conditions. Acta Odontol Scand 1964; 22: 121.

Skamagas M, Breen TL and LeRoith D. Update on diabetes mellitus: prevention, treatment and association with oral disease. Oral Disease.2008; 14:105-114.

Soell M, Hassan M, Miliauskaite A, Haïkel Y and Selimovic D. The oral cavity of the elderly patient in diabetes. Diabetes and Metabolism.2007; 33:S10-S18.

Soskolne WA. Epidemiological and clinical aspects of periodontal disease in diabetics. Ann Periodontol.1998; 3(1):3-12.

Tan WC, Tay FBK and Lim LP. Diabetes as a risk factor for periodontal disease: current status and future considerations. Ann Acad Med Singapore 2006; 35:571-81.

Taylor GW and Borgnakke WS. Periodontal disease: associations with diabetes, glycaemic control and complications. Oral Diseases. 2008; 14: 191-203.

Thomson WM, Poulton R, Broadbent JM and Al-kubaisy. Xerostomia and medications among 32-year-olds.Acta Odontol Scand. 2006; 64(4):249-254.

Velupillai YN, Packard CJ, Batty GD, Bezlyak V, Burns H, Cavanagh J, Deans K, Ford I, McGiny A, Millar K, Sattar N, Shiels P and Tannahill C. Psychological, social and biological determinants of ill-health (pSoBid): study protocol of a population-based study.2008; 8:126.

Vernillo AT. Dental considerations for the treatment of patients with diabetes mellitus. JADA.2003; 134:24S-33S.

Wilder RS, Iacopino AM, Feldman CA, Guthmiller J, Linfante J, Lavigne S and Paquette D. Periodontal-systemic disease education in US and Canadian dental schools. Journal of Dental Education. 2009; 73(1):38-52.

Wilkins EM. Clinical practice of the dental hygienist. 8th edition. Lippincott Williams & Wilkins.Philadelphia.1999:293-313; 345-346.

World Health Organisation. Oral Health Surveys. Basic Methods. 4th edition. WHO, Geneva.1997.

Zielinski M, Fedele D, Forman LJ and Pomerantz SC. Oral health in the elderly with non-insulin-dependent diabetes mellitus. Spec Care Dentist.2002; 22(3):94-98.

Appendix I - Questionnaire

DATA CAPTURE SHEET FOR DIABETIC PATIENTS

1. **Date of examination**:……..
2. **Patient's name:**……..
3. **File no:**……

DEMOGRAPHIC INFORMATION

4. **Date of birth** (YY/M/D):……
5. **Gender:** ……..
6. **Ethnic group:**…….
7. **Do you have diabetes?:**…Y/N……..
8. **How long have you had diabetes?** ……..
9. **Type of medication taken** ……

SECTION A

10. Intra-oral Examination:

10.1. DMFT:

Tooth number	Status	Tooth number	Status
18		38	
17		37	
16		36	
15		35	
14		34	
13		33	
12		32	
11		31	
21		41	
22		42	
23		43	
24		44	
25		45	
26		46	
27		47	
28		48	

D	M	F	DMFT Total

11. Plaque Index based on Debris Index (Greene and Vermillion, 1964)

0 = none (no debris or stain)
1 = <1/3 of the tooth surface has debris
2 = >1/3 of the tooth surface but not more than 2/3 has debris
3 = more than 2/3 tooth surface has debris
9 = not recorded

12. Appearance of marginal gingiva adapted from (Löe & Silness, 1963) with addition of criteria 3

0 = normal gingiva
1 = mild marginal gingivitis, slight colour change & oedema
2 = moderate gingival inflammation, redness & glazing
3 = Severe inflammation, marked redness & oedema, *with* ulceration or spontaneous bleeding

13. Oral Pathology

	Lesion	**Yes**	**No**
13.1	**No abnormalities**		
13.2	**Candida**		
13.3	**Angular Cheilitis**		
13.4	**Dental Abscess**		
13.5	**Minor Aphthous Ulcers**		
13.6	**Major Aphthous Ulcers**		
13.7	**Other**		

14. Problems with the oral cavity:

	Type of problem	**Yes**	**No**
14.1	**Periodontitis**		
14.2	**Poor wound healing**		
14.3	**Xerostomia**		
14.4	**Burning Sensation**		
14.5	**Halitosis**		
14.6	**Tooth decay**		
14.7	**Other (Please specify)**		

SECTION B

15. Oral function:

15.1	Do you have pain in the mouth?	never	sometimes	often
15.2	Do you have problems with your teeth?	never	sometimes	often
	If yes, please explain			
15.3	Do you have problems with your sense of taste?	never	sometimes	often
15.4	Do you have problems with your gums?	never	sometimes	often

16. History of oral problems

When did you notice you had these problems?

Before I became diabetic	After I became diabetic

17. Lifestyle:

17.1 Do you follow any exercise routine? Yes No Sometimes ……..

17.2 If yes, why do you exercise?.....

17.3 Do you follow a healthy eating plan? Yes No ……

17.4 When did you start following a healthy eating plan?.....

17.5 How many times did you exercise last month?.....

17.6 Is it important to exercise when diabetic?.....

17.7 Is it beneficial to eat healthily when diabetic?.....

17.8 Has being diabetic changed your life in any way? Yes or No ……

17.9 If yes, how has your life changed since becoming diabetic?.....

18.1 Quality of life

Since becoming diabetic my quality of life has: Please tick appropriate answer.

improved	
deteriorated	
stayed the same	

18.2 How do you perceive your quality of life? Please tick appropriate answer.

I am very satisfied with it	
I am somewhat satisfied with it	
I am really not satisfied with it	

18.3 What is the reason for the above answer?.....

IPHEPHA LOKUGCWALISA IMININGWANE YALABO ABANOSHUKELA

1. **Usuku lokuhlolwa**:……..
2. **Igama:**……..
3. **Inombolo ye fayili:**……

IMININGWANE YESIGULI

4. Usuku lokuzalwa (YY/M/D):……

5. Ubulili: ……**……..**

6. Uhlanga:……..

7. Ngabe unaso isifo sikashukela? Yebo/Cha:……

8. Usunesikhathi esingakanani unoshukela……

9. Udla muthi muni?.....

ISIGABA SIKA "A"

10. Ukuhlolwa ngaphakathi komlomo:

10.1. DMFT :(Amazinyo awabolile, awangasekho nawagcwalisiwe)

Inombolo yezinyo	Isimo sezinyo	Inombolo yezinyo	Isimo sezinyo
18		38	
17		37	
16		36	
15		35	
14		34	
13		33	
12		32	
11		31	
21		41	
22		42	
23		43	
24		44	
25		45	
26		46	
27		47	
28		48	

D	M	F	Isamba seDMFT

11. I indeksi ye plakhi

0=ayikho
1=<1/3
2=>1/3
3= ingxenye enkulu yezinyo imbozwe iplakhi
9=ayibhaliwe

12. Isimo sezinsini isuselwe ku (Loe & Silness, 1963) kwase kongezwe inamba yesithathu

0 = izinsini azinalutho
1 = kune gingivitis encane, um'bala uhlukile kancane & zivuvukele
2 = kune gingivitis ebonakaloyo, umbala obomvana& nokukhazimula
3 = Kunebhande elibomvu elibonakala ngokugcwele kwi gingiva lusuka kwi phaphila liye kwenye
4 = Kuvuvukele kabi, umbala ubomvu kakhulu & kuvuvukele, kwaze ezilonda kanye nokopha okuzenzekelayo

13. Izifo zomlomo

	Isifo	**Yebo**	**Cha**
13.1	**Akukho zifo**		
13.2	**Inkwethu**		
13.3	**Amapapisi**		
13.4	**Ithumba lezinyo**		
13.5	**Isilonda esincane**		
13.6	**Isilonda esikhulu**		
13.7	**Okunye**		

14. Izinkinga ngomlomo wakho:

	Uhlobo lwenkinga	**Yebo**	**Cha**
14.1	**I periodontitis**		
14.2	**Izilonda azisheshi ukuphola**		
14.3	**Umlomo owomile**		
14.4	**Ukushoshozela**		
14.5	**Ukunukelwa kabi umlomo**		
14.6	**Ukubola kwamazinyo**		
14.7	**Okunye (Sicela ukuchaze)**		

ISIGABA SIKA "B"

15. Ukusebenza komlomo:

15.1	Unabo ubuhlungu emlonyeni?	Cha,angikaze	Ngezinye izikhathi	kujwayelekile
15.2	Unazo izinkinga ngamazinyo akho?	Cha,angikaze	Ngezinye izikhathi	kujwayelekile
	Uma ikhona chaza kabanzi			
15.3	Unayo inkinga ngokunambitha kwakho?	Cha,angikaze	Ngezinye izikhathi	kujwayelekile
	Uma ikhona chaza kabanzi			
15.4	Unayo inkinga ngezinsini zakho?	Cha,angikaze	Ngezinye izikhathi	kujwayelekile

16. Umlando wezinkinga zasemlonyeni

Ngabe waqala nini ukubona ukuthi unezinkinga zomlomo?

Ngaphambi kokuba noShukela	Ngemuva kokuba noShukela

17. Isitayela ophila ngaso

17.1 Ngabe uyawuvocavoca umzimba? Yebo Cha Ngesinye isikhathi

17.2 Uma ukwenza ukwenzelani ukuvocavoca mzimba?.....

17.3 Emasontweni amabili adlulile uye kangaki ejimini?.....

17.4 Ngabe indela odla ngayo inempilo noma umsoco?.....

17.5 Kulenyanga edlule uzikhiphe kangaki ukuyodla?.....

17.6 Ngabe kubalulekile yini ukujima uma unoShukela?.....

17.7 Ngabe kunayo yini inzuzo ukudla okunomsoco uma unoShukela? Yebo Cha.....

17.8 Ngabe ukuba noShukela kuyishintshile impilo yakho? Yebo Cha .....

17.9 Ishintshe kanjani impilo yakho njengoba unoShukela?.....

18. Izinga lempilo

18.1 Emveni kokuba noShukela izinga lempilo yami li: Sicela umake impendulo efanele.

Likhuphukile	
Lehlile	
Lisafana, akushintshanga lutho	

18.2 Ulibona linjani izinga lempilo yakho? Sicela umake impendulo efanele

Linganelisa kakhulu	
Nganelisekile nje	
Anganelisekile neze	

18.3 Ngabe yisiphi isizathu sempedulo engenhla?.....

Appendix II-Informed Consent Form

UNIVERSITY OF THE WESTERN CAPE

FACULTY OF DENTISTRY

INFORMED CONSENT FOR ORAL EXAMINATION

Dear .....…….....

I am Dr NM Radebe, a Masters student from the Department of Community Oral Health at the University of the Western Cape. Diabetes mellitus is described as one of the most common diseases in medicine and it is a growing public health problem. It is as a chronic disorder of the carbohydrate, fat and protein metabolism. It is characterized by a very distinct feature - the elevation in blood glucose levels. This may occur as a result of a defect in insulin secretion from the pancreas, a change in insulin action or both. An elevation in blood glucose levels is also known as hyperglycemia and tends to affect almost all body tissues including those of the mouth. We are interested in interviewing you on regarding any habits that you may have to see if there are ways in which we can prevent any mouth problems from developing or help with any mouth problems you may have.

The interview will take about 10-15 minutes. There are no risks in participating. All information gathered in the study will be treated as strictly confidential. No one will have access to this information except the researcher. Neither your name nor anything that identifies you will be used in any reports of this study. All information collected will be maintained and stored in such a way so as to keep it as confidential as possible. Your participation is voluntary and you may withdraw from the study at anytime without any penalties. If you would like to take part in the study, please sign the bottom of this letter. If you would like to know anything more about the study, please contact Dr N Radebe on telephone number at work 031-2624471 or at home on 031-2692891.

Thank you for your co-operation.
Yours sincerely

Dr NM Radebe

I understand what will be required of me to take part in the study. I agree to participate in the research being undertaken by Ms N Radebe. I understand that at any time I may withdraw from this study without giving a reason and without affecting my treatment in the future.

Name:……. **……..**
(print in block letters) **(Signature)**

(Witness) **Date: ……**

Appendix III- Tables of the Results presented in the Figures

Comparison of the appearance of marginal gingival between diabetic and non-diabetic patients

Figure 2	Diabetic (n=150)		Non-diabetic(n=150)	
	freq	%	Freq	%
Normal	2	2.0	45	30
Mild	32	21.33	59	39.33
Moderate	81	54	35	23.33
Distinct Red Band	29	19.33	10	6.67
Severe	5	3.33	1	0.67
Total	150	100.0	150	100.0

Comparison of the appearance of oral pathology lesions between diabetic and non-diabetic patients

Figure 3		Diabetic (n=150)		Non-diabetic (n=150)
	Freq	**%**	**freq**	**%**
Candida	62	41.3	12	8.0
Angular chelitis	9	6.0	1	0.7
Dental abscess	24	16.0	4	2.7
Ulcer	21	14.0	3	2.0
Other	1	0.7	0	0.0
None	65	43.3	131	87.3
Total	**182**		**151**	

Comparison of the appearance of oral cav ity problems between diabetic and non-diabetic patients

Figure 4	Diabetic (n=150)		Non-diabetic (n=150)	
	Freq	**%**	**freq**	**%**
Perio	79	52.7	22	14.7
Wound	19	12.7	4	2.7
Xerostomia	79	52.7	11	7.3
Burning Sensation	14	9.3	2	1.3
Halitosis	56	37.3	18	12.0
Tooth decay	74	49.3	87	58.0
No problem	7	4.7	46	30.7
Total	**328**		**190**	

Comparison of the appearance of the type of teeth problems between diabetic and non-diabetic patients

Figure 5	Diabetic (n=150)		Non-diabetic (n=150)	
	Freq	%	freq	%
Caries	51	34.0	42	28.0
Mobile	17	11.3	3	2.0
Toothache	32	21.3	35	23.3
Sensitivity	18	12.0	14	9.3
Not applicable	59	39.3	80	53.3
Total	**177**		**174**	

Comparison of the appearance of gum problems between diabetic and non-diabetic patients

Figure 6	Diabetic (n=150)		Non-diabetic (n=150)	
	Freq	%	freq	%
Bleeding	58	38.7	33	22.0
Swollen	13	8.7	3	2.0
Painful	10	6.7	8	5.3
Spontaneous bleeding	9	6.0	2	1.3
Not applicable	71	47.3	112	74.7
Total	**161**		**158**	

Length of time diagnosed with diabetes

Figure 7	Diabetic (n=150)	
	Freq	%
1 to 2 years	19	12.67
3 to 5 years	33	22.0
6 to 10 years	28	18.67
More than 10 years	70	46.67

Type of diabetes medication taken by the diabetic participants

Figure 8	Diabetic (n=150)	
	Freq	%
Insulin	12	8
Oral hypoglycemic	112	74.67
Combination	26	17.33