THE PREVALENCE AND SEVERITY OF PERIODONTAL DISEASE IN TYPE II DIABETICS

A mini-thesis submitted in partial fulfilment of the requirements for the degree of Master of Science in Dental Sciences in Periodontics at the Faculty of Dentistry
University of the Western Cape

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THE PREVALENCE AND SEVERITY OF PERIODONTAL DISEASE AND TYPE II DIABETES

Keywords

Periodontitis

Type II Diabetes

Inflammation

Glycaemic control

Coloured South Africans

HbA1c

Oral hygiene

SUMMARY

Introduction: The relationship between Periodontal Disease and Type II diabetes has been reported in recent literature. More recent studies suggest that further research is required into the relationship of glycaemic control on Periodontitis.

The main aetiological factor in Periodontal Disease is plaque, however other secondary factors such as Diabetes Mellitus, neutrophil abnormalities, smoking, socio-economic status, age, stress, Human Immunodeficiency Virus (HIV) infections, pregnancy, sex hormones, osteoporosis and several other conditions play an important causative role (Genco, 1993).

Aim: The purpose of this study is to test the hypothesis that the prevalence and severity of periodontal disease is greater for patients with poorly controlled Type II Diabetes Mellitus (DM-2) compared to those with better controlled Type II Diabetes Mellitus.

Methods: **'**Coloured female patients', who were diagnosed with Type II diabetes were included in the study. Demographic information, medical history and HbA1c levels were recorded by the attending physician in the diabetes unit. Periodontal examination was carried out by a single examiner. This included a plaque index (PI), a gingival index (GI), bleeding on probing (BoP), probing depths (PD) and clinical attachment loss (CAL). These measurements were recorded on Ramfjord teeth. The presence of any one sextant showing PD of ≥ 4 mm or clinical loss of attachment of ≥ 3 mm was diagnosed as periodontitis.

Results: Poor glycaemic control was associated with more severe periodontitis.

Conclusion: The results of the study showed that it could be justified that the regression approach (correlation) be applied to the complete sample of 63 individuals. Most of these correlation coefficients were positive and significantly different from zero, indicating that “HbA1c” had a detrimental influence on the periodontal measurements; within the limitations of the study. This link could be indirect in that some other properties of diabetes, and not necessarily “HbA1c”, affected the dental health of diabetics adversely.

DECLARATION

I hereby declare that, “the prevalence and severity of periodontal disease in Type II diabetes“, is my own work, that it has not been submitted before for any degree or examination at any other university or institution, and that all the sources I have used or quoted have been indicated and acknowledged by complete references.

James R. Hyslop

October 2011

Signed:……

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GLOSSARY, DEFINITIONS and ABBREVIATIONS

Risk is the probability that an individual will get a specific disease in a given period. The risk of developing the disease will vary from individual to individual.

Risk factors may be environmental, behavioural, or biological factors that, when present, increase the likelihood that an individual will get the disease. Risk factors are identified through longitudinal studies of patients with the disease of interest.

HbA1c- Haemoglobin A1c (HbA1c) is a minor component of haemoglobin to which glucose is bound. HbA1c also is referred to as glycosylated or glucosylated haemoglobin.

Glycaeted haemoglobin assay, measures the amount of glucose irreversibly bound to the haemoglobin molecule.

Casual plasma glucose concentration- 'Casual' is defined as any time of the day without regard to the time since the last meal.

Fasting plasma glucose- Fasting is defined as no caloric intake for at least 8 hours.

CI — Calculus Index

DM — Diabetes Mellitus

GI — Gingival Index

PD — Periodontal Disease

PI — Periodontal Index

DEDICATION

To my supervisor whose guidance, encouragement, help and support made this project possible.

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CHAPTER 1

INTRODUCTION

The motivation for this study is outlined in this chapter. The relationship between Periodontal Disease and Type II Diabetes has been researched, however it has been suggested in previous studies that the relationship of glycaemic control and periodontitis needs further elucidation (Navarro-Sanchez *et al.*, 2007; Promsudthi *et al.*, 2005; Kiran *et al.,* 2005; Tsai *et al.*, 2002).

Various factors predispose individuals to periodontal disease. These include Diabetes Mellitus, neutrophil abnormalities, smoking, socio-economic status, age, stress, Human Immunodeficiency Virus (HIV) infections, pregnancy, osteoporosis as well as race (Van Dyke & Dave, 2005; Genco, 1993). The association between periodontal disease and systemic conditions such as diabetes and cardiovascular disease has been explored in several studies, (Faria-Almeida *et al.*, 2006; Southerland *et al.*, 2006; Genco *et al.*, 2005; Geerts *et al.*, 2004; Teng *et al.*, 2002). It has been established that periodontal disease is more prevalent and more severe in persons with diabetes than in non-diabetic persons. Indeed, periodontal disease has been recognized for some time as the "sixth complication" of diabetes (Loe, 1993).

The current classification of Diabetes Mellitus is based upon the pathophysiology of each form of the disease (Mealey & Oates, 2006; American Diabetes Association, 2005).

Type I diabetes results from cellular mediated auto-immune destruction of pancreatic ß-cells, usually leading to a total cessation of insulin secretion. Markers of autoimmune destruction have been identified and can be used for diagnosis or risk assessment (American Diabetes Association, 2005). Type I diabetes usually presents in children and adolescents, although some studies

demonstrated 15% to 30% of all cases being diagnosed after 30 years of age, (Laakso, 1985). In older Type I patients, the ß-cell destruction occurs more slowly than in children, with a less abrupt onset of symptoms. This indicates that the rate and the extent of cellular destruction may vary between patients. The lack of insulin production in patients with Type I diabetes makes the use of exogenous insulin necessary to sustain life, hence the former name "insulin-dependent diabetes," (American Diabetes Association, 2005). In the absence of insulin these patients develop ketoacidosis, a life-threatening complication.

Type II diabetes, previously called "non-insulin-dependent diabetes", results from insulin resistance, which alters the use of endogenously produced insulin at the target cells (American Diabetes Association, 2005). People affected by Type II diabetes have altered insulin production without destruction of ß-cells, and they retain the capacity for some insulin production. The presence of insulin (however minimal) in the latter group of individuals results in a lower occurrence of ketoacidosis compared to the Type I group. Type II patients can remain undiagnosed for many years because the hyperglycaemia appears gradually and often without symptoms, (DeFronzo & Ferrannini, 1991). Often, especially early in the disease process, pancreatic insulin production increases to compensate for insulin resistance. As the condition progresses, pancreatic insulin production diminishes due to the prolonged increase in secretory demand (Mealey & Oates, 2006). Insulin secretion becomes insufficient to compensate for insulin resistance. Although Type II diabetics do not always necessarily need insulin to survive, insulin may be used as part of the medical management of Type II diabetes. About 90% of affected persons are affected by Type II diabetes.

The impact of Diabetes Mellitus on the oral cavity has been well researched, and there is strong evidence demonstrating that diabetes is a risk factor for gingivitis and periodontitis (Mealey & Ocampo, 2007; Southerland *et al.*, 2006). The degree of glycaemic control is an important variable in the bi-directional relationship between diabetes and periodontal diseases. A higher prevalence

and severity of periodontal disease is seen in individuals with poor glycaemic control. Significant improvements in both glycaemic control and periodontal condition may be achieved by the implementation of periodontal therapy (Garcia, 2007; Mealey & Ocampo, 2007; Navarro-Sanchez *et al.*, 2007; Promsudthi *et al.*, 2005; Kiran *et al.*, 2005). Large epidemiological studies have shown that diabetes increases the risk of alveolar bone loss and attachment loss approximately three-fold when compared to non-diabetic individuals (Mealey & Ocampo, 2007). Tsai, Hayes & Taylor (2002) found the degree of glycaemic control to be the major factor affecting the risk of periodontal disease. In a large epidemiological study, the authors reported that adults with poorly controlled Diabetes Mellitus had a 2.9-fold increased risk of having periodontitis compared to non-diabetic patients; conversely, patients with well-controlled Diabetes Mellitus had no significant increase in the risk for periodontitis.

Various parameters over the years have been used to investigate the level of glycaemic control in Diabetes Mellitus. These include haemoglobin A1 (HbA1), (Taylor *et al.*, 1998), haemoglobin A1c (HbA1c), (Tsai *et al.*, 2002) and random blood sugar (RBS), (Almas *et al.*, 2001). According to the American Diabetes Association (2005) glycaeted haemoglobin assay measures the amount of glucose irreversibly bound to the haemoglobin molecule. This bound glucose value is proportional to blood glucose levels. Glycaeted haemoglobin (HbA1c) records the serum glucose level over 2-3 months (American Diabetes Association, 2005).

In 1998, the WHO revised the diagnostic criteria for diabetes, and introduced the concept of impaired fasting glycaemia (IFG). More recently, the American Diabetes Association (ADA), 2005, proposed the upper limit of normal for fasting plasma glucose be lowered from 6.1 to 5.6mmol/l.

The aim of this study is to investigate the prevalence and severity of periodontal disease in Type II diabetics, compared to a control group of non-diabetics. This literature review will pay special attention to HbA1c levels and its relationship with the prevalence and severity of periodontal disease. The purpose of this study is to test the hypothesis that, 'the prevalence and severity of periodontal disease is greater, for patients with poorly controlled Type II Diabetes Mellitus compared to those who are better controlled. Diabetic control will be monitored by using HbA1c readings which are recorded.

CHAPTER 2

LITERATURE REVIEW

2.1 Definition of Diabetes Mellitus

Diabetes Mellitus is one of the most common endocrine disorders resulting from relative or absolute deficiency of insulin and is characterized by persistently raised blood glucose levels (hyperglycaemia). Diabetes, coronary heart disease and adult periodontitis are common chronic diseases observed in a significant proportion of the adult South African population. Diabetes is a metabolic disease that, due to disturbances in insulin production, leads to abnormal fat, sugar, and protein metabolism and resultant hyperglycaemia that can ultimately induce diverse multiple system pathologies (Winer and Sowers, 2004). The classic symptoms of diabetes include polyuria, polydipsia, and unexplained weight loss.

Literature that elucidates the relationship between periodontal disease and Type II diabetes, in particular, studies in which glycaemic control was monitored, by measuring HbA1c are reviewed in this chapter.

2.2 Risk Factors for Type II Diabetes

1. Genetic predisposition to diabetes.
2. Obesity.
3. Lack of exercise.
4. Gestational diabetes.
5. Pre-diabetes.
6. Ageing.

2.3 Epidemiology

Diabetes Mellitus (DM) is a chronic metabolic disorder that was reported to affect more than 100 million people worldwide in 1998. This number is expected to treble by year 2025 (Alberti & Zimmet, 1998). Hough, (2007) reported that, the prevalence of Type II Diabetes Mellitus is more than 200 million, currently. Moreover, the incidence of Type II diabetes is predicted to increase by 30-70% in Europe and the USA in the next 15-20 years, (Hough, 2007).

The prevalence of Diabetes Mellitus in South Africans was reported using epidemiological data from KwaZulu-Natal (Motala, Omar *et al.*, 2003) Western Cape (Levitt *et al.*, 2006) and Free State (Mollentze *et al.*, 2006). The disease is common in all population groups. Figures quoted from the studies were: Indian 13%, Coloured 11%, Black 5-8% and White 4-5%, respectively. An increasing westernised life-style, urbanisation and ageing may result in a 160-200% increase in the total prevalence of this disease by 2030, (Hough, 2007). Earlier predictions by Levitt and Bradshaw (2006) that the impact of the HIV/AIDS epidemic may decrease the prevalence of Type II diabetes in South Africa can possibly be explained by the early deaths of HIV positive cases

Studies by Erasmus *et al.*, (1999) assessed glycaemic control in stable Type II black South African diabetics attending a peri-urban clinic. The study showed that glycaemic control was poor in the majority of patients, irrespective of sex, treatment or duration of diabetes. The findings of this study agreed with the results of Weatherspoon (1994). The results were also supported by some race-based studies that found glycaemic control to be poorer in the black population. Eberhardt (1990) revealed that black women had high HbA1c levels, indicating that gender may play a significant role in poor glycaemic control. The poor control of blood glucose levels both at the beginning of the study and after attendance at the diabetic clinic may reflect the following: 1) insufficient

motivation, 2) lack of knowledge, 3) incorrect beliefs about diabetes, and 4) particular cultural values. These factors are all recognized as potential barriers to improving diabetic care for blacks, (Erasmus *et al.*, 1999).

2.4 Diagnostic Parameters of Diabetes Mellitus

In 1998, the World Health Organization adopted the diagnostic parameters for diabetes established by the American Diabetes Association, (American Diabetes association, 1997; Alberti & Zimmet, 1998; American Diabetes Association, 2005). Currently there are 3 methods of diagnosing Diabetes Mellitus (American Diabetes Association, 2005):

1) Symptoms of diabetes plus casual plasma glucose concentration ≥200mg/dl (≥11.1 mmol/l). 'Casual' is defined as any time of the day without regard to time since the last meal.
2) Fasting plasma glucose ≥126 mg/dl (≥7.0mmol/l). Fasting is defined as no caloric intake for at least 8h;
3) 2-h post-load glucose ≥ 200mg/dl (≥11.1 mmol/l) during an oral glucose tolerance test. The test should be performed as described by the WHO, using a glucose load containing the equivalent of 75g anhydrous glucose dissolved in water.

Any one of the methods listed above is necessary to accompany a single random laboratory test and is required to be performed on a different day. Impaired glucose tolerance can be diagnosed by an oral glucose tolerance test; and occurs when the 2-h post-load plasma glucose concentration is ≥140 mg/dl but ≤199 mg/dl (between 7.8 and 11.1 mmol/l). Impaired fasting glucose is diagnosed after a fasting plasma glucose test and is defined by plasma glucose levels of ≥100mg/dl but ≤125 mg/dl (between 5.6 and 6.9 mmol/l).

The ***haemoglobin A1c test*** is used to monitor the overall glycaemic control in people known to have diabetes. It is not recommended for diagnosis because there is not a 'gold standard' assay for haemoglobin A1c and because many countries do not have ready access to the test, (Mealey & Ocampo, 2007).

2.5 Glycated Haemoglobin (haemoglobin A1c)

Numerous proteins in the body are capable of being glycated. Glycohaemoglobin is formed continuously in erythrocytes as a product of the non-enzymatic reaction between the haemoglobin protein, the function of which is to carry oxygen molecules, and glucose. The binding of glucose to haemoglobin is stable and haemoglobin remains glycated for the life span of the erythrocyte, (Virtue *et al*, 2004; Mealey & Ocampo, 2007). Glycohaemoglobin levels estimate the average blood glucose level. Over time, higher haemoglobin A1c levels reflect higher average blood glucose levels, (Rohlfing, 2002). Measurement of haemoglobin A1c is of major clinical value and accurately reflects the mean blood glucose concentration over the preceding 1-3 months. Haemoglobin A1c levels correlate well with the development of diabetic complications, and may become established as a test for the diagnosis and progression of diabetes, (Davidson *et al.*, 2000).

It is generally recommended by the American Diabetes Association, (American Diabetes association, 1997; Alberti & Zimmet, 1998; American Diabetes Association, 2005), that the haemoglobin A1c test is performed at least twice a year in affected people meeting treatment goals. Affected individuals who have not met their treatment goals or had changes to their therapy would benefit from a test every three months. The recommended haemoglobin A1c target value for people with diabetes is <7% (non-diabetic persons is <6%), (American Diabetes association, 1997; Alberti & Zimmet, 1998; American Diabetes Association, 2005). It would appear that achieving this goal is difficult. A recent population

study showed that only 36% of people with Type II diabetes achieved a target haemoglobin A1c of <7%, (Koro *et al.*, 2004). Estimates of HbA1c may be reduced in individuals suffering from anaemia or during pregnancy (Manfredi *et al.*, 2004).

2.6 Aetiology of Periodontal Disease

Periodontal disease is a microbe-induced chronic inflammatory condition that leads to gingival inflammation, periodontal tissue destruction, and alveolar bone loss, (Nassar, 2007). The condition is characterized by destruction of the periodontal tissues and results in the loss of connective tissue attachment, loss of alveolar bone, and the formation of pathological pockets around the diseased teeth. There is no particular age at which it occurs (Flemming, 1999). Chronic periodontitis can be considered a disease of the middle age, the majority of patients first presenting between the ages of 40 to 50. Periodontal disease has been found world-wide and affects all population groups. It is responsible for a substantial proportion of the tooth loss in adulthood (Loe, 1967; Ramfjord, 1967 Machtei, 1992 and Milward & Chapple, 2003). Periodontal diseases cannot be classified according to their aetiology because they are complex diseases that are polymicrobial and polyimmuno-inflammatory in nature. Thus, periodontal disease is the outcome of complex, unpredictable interactions between microbial complexes and the host's inflammatory/immune response. Periodontal disease is primarily caused by the accumulation of plaque, i.e. microorganisms, with the establishment of and persistent role of other factors.

2.7 Risk Factors of Periodontal Disease

There are several risk factors predisposed to periodontal disease. The clinical assessment of patients with periodontal disease must include an evaluation of risk and susceptibility. While removal of bacterial plaque and plaque retentive factors remains the focus of treatment, the successful, long-term management of periodontal diseases needs to include the removal and/or control of known risk factors (Irwin *et al.*, 2007).

Risk factors may be ***modifiable or non-modifiable. Modifiable risk factors*** are usually environmental or behavioural in nature, whereas ***non-modifiable risk factors*** are usually intrinsic to the individual and therefore not easily changed. These risk factors have been identified by longitudinal cohort studies or controlled clinical trials which identify a causal relationship and can provide evidence of the benefit of eliminating the risk factor, respectively, (Van Dyke & Dave, 2005).

2.7.1 Modifiable Risk Factors for Periodontal Disease

1. Tobacco Smoking is a well-established risk factor for periodontitis. A direct relationship between smoking and the prevalence of periodontal disease has been demonstrated in numerous studies, (Kibayashi & Tanaka, 2007; Irwin *et al.*, 2007). This association is independent of other factors such as oral hygiene or age, (Ismail, Burt & Eklund, 1983). Cross sectional and longitudinal data show that the risk of developing periodontal disease increases with increased smoking, (Van Dyke & Dave, 2005). Cross sectional and longitudinal studies have shown that former smokers (clinically defined as two or more years since quitting smoking) experience less attachment loss than current smokers but more than never-smokers. Also, the likelihood of developing increasing periodontal disease is dose dependent, (Tomar & Asma, 2000).

A number of studies have established that smoking is associated with reduced gingival bleeding (Bergstrom & Bostrom, 2001; Muller *et al.*, 2001). This occurrence may reflect an alteration of the calibre of blood vessels perfusing the gingival tissues (Mirbod *et al.*, 2001). Reduced bleeding may be the result of a disruption of the immune response which in turn may account for the increased loss of clinical attachment and alveolar bone. The use of gingival bleeding as an indicator of gingival inflammation therefore is not a reliable indicator of a smoker's periodontal health.

2. Diabetes is a modifiable risk factor as there is no cure. However, it can be controlled. Epidemiological studies have demonstrated on numerous occasions that the prevalence and severity of periodontitis is significantly higher in patients with Type I and Type II diabetes than in those without diabetes and that the level of diabetic control is important, (Mealey & Oates, 2006; Van Dyke & Dave, 2005; Saremi *et al.*, 2005; Taylor *et al.*, 1996; Emrich, Shlossmann & Genco, 1991).

3. Microorganisms and periodontal disease. Bacterial plaque at the gingival margin results in the development of gingivitis, a condition that is reversible with the implementation of oral hygiene measures. Of all the various microorganisms that colonize the mouth, there are three, known as '**Red Complex':**

(i) Porphyromonas gingivalis,

(ii) Treponema denticola, and

(iii)Tannerella forsythia (formerly Bacteroides forsythia)

that have been implicated as aetiological agents in periodontitis, relating strikingly to clinical measures of periodontal disease, particularly pocket depth and bleeding on probing (Socransky *et al.*, 1998).

Bacterial species exist in complexes in subgingival plaque. The purpose of an investigation by Socransky *et al.*, (1998) was to define the bacterial complexes using data from large numbers of plaque samples and different clustering and

ordination techniques. Subgingival plaque samples were taken from the mesial aspect of each tooth in 185 subjects; 160 with periodontitis and 25 without periodontitis. 5 major complexes were consistently observed using any of the analytical methods.

(1) Red Complex consisting of the tightly related group:
(See list above)

(2) Orange Complex consisting of a tightly related core group,

(i) F. nucleatum

(ii) Prevotella intermedia

(iii) Prevotella nigrescens, and

(iv) Peptostretococcus micros.

Species associated with this group included: *Eubacterium nodatum, Campylobacter rectus, Campylobacter showae, Streptococcus constellatus, and Campylobacter gracilis.*

(3) Green Complex consisting of the 3 *Capnocytophaga* species:

(i) Campylobacter concisus

(ii) *Eikenella corrodens, and*

(iii)*Actinobacillus Aggregatibacter actinomycetemcomitans.*

(4) Yellow Complex consist of a group of streptococci:

(i) Streptococcus mitis,

(ii) Streptococcus sanguis, and

(ii) Streptococcus oralis, were closely related in this group.

(5) Purple Complex

(i) Actinomyces odontolyticus, and

(ii) *Veillonella parvula.*

Socransky *et al.*, (1998) investigated the relationship of the species in the different complexes to pocket depth and bleeding on probing. All species in the 'Red and Orange complexes' showed a strong relationship with pocket depth, showing an increase in prevalence and numbers with increasing pocket depth. The relationship was found to more significant with the 'Red Complex'. Also the red complex and the individual species in that group were also strongly associated with bleeding on probing.

The presence of periodontal pathogens is not sufficient to cause disease. The chance of developing periodontal disease in an individual who harbours one of the putative periodontal pathogens is not high enough to consider them a risk factor (Ezzo and Cutler, 2003). Therefore the presence of these microorganisms may be risk indicators of periodontal disease, but other factors need to be present; anatomic factors such as furcations, root concavities, developmental grooves, restorative margin overhangs etc. which all harbour bacterial plaque and make these areas difficult for the clinician to instrument, (Van Dyke and Dave, 2005).

2.7.2 Non-modifiable Risk Factors for Periodontal Disease

1. Genetic Factors

Although bacterial infection is the etiologic agent in periodontal disease, studies of identical twins suggest 50% of susceptibility to periodontal disease is due to host factors, (Michalowicz *et al.*, 2000). Similarly, indigenous and relatively isolated populations have been shown to develop distinct periodontal disease presentations that differ from group to group, (Ronderos *et al.*, 2001).

2. Host response

There is evidence to suggest that tissue destruction occurring in periodontal disease results from an improper immune response to bacteria rather than directly to the destructive effects of the bacterial pathogens, (Van Dyke and Serhan, 2003). In localized aggressive periodontitis, overly active or "primed" neutrophils may be responsible for mediating tissue destruction, (Van Dyke and Serhan, 2003). Interleukin-1 (IL-1) gene polymorphisms have been linked to periodontal disease by Guzman *et al*., (2003). The authors have demonstrated a possible relationship between IL-1 genotype and periodontal status in diabetics. To date no definitive IL-1 genotype that places individuals in any given population at risk for periodontal disease. Furthermore, the evidence suggesting possible interactions between IL-1, smoking and diabetes suggest that there is interplay between genetic an environmental factors that results in periodontal disease, (Van Dyke and Dave, 2005).

3. Osteoporosis

Osteoporosis has been suggested as another risk factor for periodontitis. Several studies have shown that alveolar bone density is altered in osteoporitic individuals. Lerner, (2006), stated that in females with postmenopausal osteoporosis, the possibility exists that a lack of oestrogen influences the activity of bone cells and immune cells by enhancing the progression of alveolar bone loss. Fewer studies have demonstrated a relationship with clinical attachment

levels. However, these results have been contraindicated by several other studies. Phipps *et al.*, (2007), evaluated the association between periodontal disease and bone mineral density (BMD) in a cohort of 1347 older men, who were recruited from the 'Osteoporotic Fractures in Men Study.' The authors found no association between periodontitis and skeletal bone mineral density among older men.

4. Other Systemic Diseases

Several deficiencies of neutrophil function have been related to periodontal disease. These include Chediak-Higashi syndrome, cyclic neutropenias, lazy leukocyte syndrome, agranulocytosis, leukocyte adhesion deficiency, Downs syndrome and Papillon Lefevre syndrome. Except for Downs syndrome, these diseases are very rare, so probable though not definitive relationships to periodontal disease have not been established (Deas *et al.*, 2003).

5. HIV

It has been hypothesized that the immune dysfunction associated with human immunodeficiency virus (HIV) infection and acquired immunodeficiency syndrome (AIDS) increases susceptibility to periodontal disease; characterised by necrotizing ulcerative periodontitis. Aas *et al.*, (2007) compared the predominant bacterial and fungal species associated with gingivitis, periodontitis, and linear gingival erythema, in HIV positive subjects with different immune status. The classical putative periodontal pathogens of the 'Red Complex', were not detected in these subjects while other species, such as *Gemella, Streptococcus* and *Candida albicans* were evident. These findings suggest that alternative bacterial and fungal species may be involved in the periodontal diseases associated with HIV. These are indicative of opportunistic infections in a highly susceptible immunocompromised host.

2.8 Periodontal Infections and Diabetes Mellitus

The association between periodontal disease and diabetes has been explored in several studies. Periodontal disease is more prevalent and more severe in persons with diabetes than non-diabetic persons.

Diabetes mellitus type II is a complex metabolic disease characterized by chronic hyperglycaemia. The peripheral resistance to insulin action, impaired insulin secretion and increased glucose production in the liver, means there is an increase in glucose in the gingival fluid and blood, with similar plaque and gingival scores. This could change the environment of the micro-flora, inducing qualitative changes in bacteria that could account for the severity of periodontal disease in poorly controlled diabetics. These patients tend to suffer from a greater loss of attachment, increased bleeding on probing, and increased tooth mobility, with a retarded healing capacity. Chronic hyperglycaemia adversely affects the synthesis, maturation, and maintenance of collagen and extracellular matrix. Advanced glycation end products (AGEs) are formed in the hyperglycaemic state, by numerous proteins and matrix molecules undergoing a nonenzymatic glycosylation. These AGEs crosslink with collagen, making it less soluble and less likely to be normally repaired or replaced, therefore rendering the periodontal tissues more prone to destruction. This altered collagen metabolism, means the healing potential of diabetics is reduced. This coupled with the fact that they have an altered polymorphonuclear Leukocyte function, therefore leading to further susceptibility to infection, means they are more prone to periodontal breakdown, as the diabetic control decreases.

Studies that have examined the relationship between diabetes and periodontitis are heterogeneous in design and aim. Thus, both positive and negative conclusions have been drawn with respect to the relationship between the two diseases. There is no difference in impact between Types I and II Diabetes Mellitus, (Van Dyke & Dave, 2005). Diabetic parameters examined

include glycaemic control, duration of the disease, presence of other diabetes associated complications and population studied. Periodontal parameters examined have included gingivitis, clinical attachment loss, and alveolar bone loss (Tomar and Asma, 2000). A review of the literature by Mealey and Oates, (2006) stated that, 'Diabetes increases the risk of periodontal diseases, and biologically plausible mechanisms have been demonstrated in abundance'. The impact of periodontal diseases on glycaemic control of diabetes and the mechanisms through which this occurs is less clear. Inflammatory periodontal diseases may increase insulin resistance in a similar way to obesity, aggravating glycaemic control. Further research is needed to clarify this aspect of the relationship between periodontal diseases and diabetes, (Mealey & Oates, 2006; Kinane & Chestnutt, 1997). Studies have shown a relationship between poor glycaemic control and periodontal disease parameters (Navarro-Sanchez *et.al*., 2007, Guzman *et.al*., 2003; Tsai *et at.*, 2002; Cutler *et al.*, 1999). Navarro-Sanchez *et al.* (2007) have suggested a complex two-way relationship between Diabetes Mellitus and periodontitis; each disease having a potential impact on the other. The study revealed that clinical and immunological improvements were obtained by significantly decreased HbA1c values in Type II diabetic subjects. The authors suggested that larger studies are needed to confirm the finding and establish whether periodontal therapy had a significant effect on glycaemic control. Cross- sectional studies on Pima Indians, a group displaying the highest prevalence of Type II diabetes in the world, show an odds ratio of 2.8 to 3.4 for developing periodontal disease in Type II diabetics compared to non-diabetics (Emrich *et al.*, 1991). In a prospective longitudinal study of 628 subjects, (age ≥35 years), Saremi *et al.*, (2005) examined the effect of periodontal disease on overall and cardiovascular disease; mortality in Pima Indians with Type II diabetes. Periodontal abnormality was classified as no or mild, moderate and severe, based on panoramic radiographs and clinical dental examinations. Based on the results of this study the authors concluded that, 'periodontal disease is a strong predictor of mortality from ischaemic heart disease and diabetic nephropathy in Pima Indians with Type II diabetes. The

effect of periodontal disease is additional to the effects of traditional risk factors for these diseases. Similarly, longitudinal studies have shown increased risk of on-going periodontal destruction in diabetics as compared to non-diabetics with an odds ratio of 4.2. Finally, studies have been done which suggest that poorly controlled diabetics respond less successfully to periodontal therapy compared to well-controlled and non-diabetics (Navarro- Sanchez *et al.*, 2007; Promsudthi *et al.*, 2005; Kiran *et al*., 2005; Tsai, Hayes and Taylor, 2002).

Non-surgical periodontal treatment is associated with improved glycaemic control in Type II diabetic patients and can be undertaken along with the standard measures for the diabetic patient care. Non-surgical periodontal treatment resulted in a significant decrease in HbA1c levels (Garcia, 2007).

Most of the studies done over the years have been on the relationship between periodontal disease and IDDM (Insulin Dependent Diabetes Mellitus). There seemed to be only limited information available on the relationship between periodontal disease and NIDDM (Non-Insulin Dependent Diabetes Mellitus).

The most extensive studies on the relationship between periodontal status and Diabetes Mellitus have been done amongst the Pima Indians from the Gila River Indian community in Arizona. This tribe has the highest prevalence of Type II diabetes in the world. Data from 2,483 non-diabetic and 736 diabetic subjects was assessed. Panoramic radiographs were used to assess interproximal bone loss while probing depth and clinical attachment loss were taken with a graduated probe (Shlossman *et al.*, 1990).

In one particular review, of the 'Bidirectional interrelationships between diabetes and periodontal diseases', 8 reports were identified limited to subjects with Type II diabetes. Three of these reports included only adults. The remaining 5 reports are from an epidemiological study in the Pima Indians of the

Gila River Indian community, Arizona and include subjects ages 5 and older or 15 and older. These 8 studies all reported significantly poorer periodontal health in subjects with diabetes. Emrich *et al.*, (1991) and Taylor (2001) reported that the odds were approximately 3 times greater for people with diabetes to have destructive periodontal disease after controlling other important factors. Nelson *et al.*, (1990) found a 2.6-fold greater risk of advanced periodontal disease incidence, and Taylor *et al.* (1998) reported that subjects with Type II diabetes had a 4-fold greater risk for more severe alveolar bone loss progression. Some studies have also reported no association (Ervasti, *et al.*, 1985). Therefore there is still a need to generate more data on diabetes and periodontal disease so that the issue of the association of the two diseases becomes clearer. Some of the studies which took the level of glycaemic control into consideration demonstrate that poor glycaemic control contributes to a higher prevalence or more severe form of the periodontal disease (Almas *et al.*, 2001; Tsai, Hayes & Tailor, 2002). A study with insulin dependent diabetics found a correlation between clinical attachment loss and duration of diabetes (Firalti, 1997) and another did not find a significant relationship between duration of diabetes and periodontal status (Bridges, 1996). Therefore, the need to carry out more studies assessing the severity and prevalence of periodontal disease in Type II diabetics, compared to poorly controlled Diabetes Mellitus on the basis of glycaemic control is evident.

One South African study showing the relationship between Type II Diabetics and the severity of periodontal disease (Matu, 2009) is reported. Statistics show that 7-8% of the South African population suffers from Diabetes, thus, there is a definite need for further research in this area.

CHAPTER 3

AIMS AND OBJECTIVES:
Research Design and Methodology

3.1 Introduction

In this chapter the research design and method used to obtain data are presented. The purpose of this study was to test the hypothesis that the prevalence and severity of periodontal disease is greater, for individuals with poorly controlled Type II Diabetes Mellitus compared to those with better controlled Type II Diabetes Mellitus.

3.2 Research questions

This study will address the following question:

1. What is the prevalence and severity of periodontal disease amongst poorly controlled and controlled Type II diabetics, attending Tygerberg Hospital, Cape Town?

Aim of the study

To determine the prevalence and severity of periodontal disease in poorly controlled and better controlled Type II diabetics compared to a control group of non-diabetics, at Tygerberg hospital.

Objectives

1. To determine the prevalence and severity of periodontal disease in Type II diabetics and its association with glycaemic control.

3.3 Null Hypothesis

There is no difference in the prevalence and severity of periodontal disease in poorly controlled and better controlled non insulin dependant diabetics Type II.

3.4 Study design

The study will be an observer blind, descriptive cross sectional study. A regression analysis will be undertaken of existing records (18 months).

3.5 Study area

The study was carried out at Tygerberg Hospital, located in the Tygerberg area, Cape Town, South Africa. The hospital is an outpatient facility under The Department of Health in the Western Cape.

3.6 Study population

The study population consisted of Coloured female patients aged between 30-60 years, with a medical history of Type II diabetes who attend the Tygerberg Hospital.

1. Those who had an appointment to see the doctor for review purposes. The review appointments were scheduled according to the level of control.
2. Those that belong to a "members club". These had received diet education as a way of managing their diabetic status.
3. Those who came to collect medicine from the hospital pharmacy.

Groups one and two attended the hospital on Mondays and Tuesdays.

3.7 Exclusion criteria

Diabetic patients were excluded from the study if:

1. Three Ramfjord teeth selected for recording or their substitutes were absent.
2. They were on prophylactic antibiotics or on antibiotic treatment one month prior to the examination.
3. They were pregnant.
4. They had only been diagnosed and treated as Type II diabetic < 18 months.
5. Calculus obstructed the cement-enamel junction or sulcus made it difficult to record the pocket depth measurement.
6. Edentulous patients.
7. They did not belong to the group consisting of coloured female patients aged between 30 to 60 years.
8. Patients who had had NSAIDS for the last 5 days prior to examination.
9. They suffered from any bleeding disorders or were on anticoagulant therapy, e.g. warfarin.
10. If the assessment of glycaemic control during the past 18 months was not possible; if greater than or equal to the last 3 HbA1c values were not available, to obtain an average value.

11. They were taking anti-epileptic drugs, as these may have caused gingival overgrowth.

3.8 Sample Size

In order to calculate the size of the study a retrospective analysis of patient records was carried out. One hundred randomly selected records were assessed so as to ascertain the prevalence of poorly controlled versus controlled Type II diabetics i.e. one hundred HbA1c records were categorized according to their assay. Recording of HbA1c of >7% was considered to be readings of poorly controlled diabetes. HbA1c recordings of <7% are considered to be better controlled diabetics.

3.9 Sampling Procedure

All Type II diabetic patients that were available during each day of examination, that consent in writing to the study, met the inclusion criteria and had valid HbA1c readings taken in the recent 3 months.

The clinical examination was done 'Observer Blind', to avoid bias, i.e. the clinical researcher didn't know the diabetic status prior to examination. The individual's HbA1c calibration needed to have been recorded within the last 3 months. The information was verified by the dental assistant but the control factor was not revealed to the clinical researcher. The method used to determine HbA1c at Tygerberg Hospital was an automated one employing the Advia-1800 auto analyzer; reported CV= 5-10%.

3.10 Calibration

Prior to carrying out the study, the investigator carried out an exercise to become familiar with the various indices to be used. He was calibrated against an experienced clinician. Fourteen patients were examined for plaque, gingival health and probing depth. The teeth of each patient was divided so that each examiner had an equal number of teeth to evaluate in a random manner.

The average score for each examiner was calculated per patient. All the above variables were assessed. The differences in the scores of the two examiners were calculated by a statistician using Wilcoxon Signed Rank Sum test.

3.11 Data Collection

The faculty manager was requested to inform the staff of Tygerberg medical hospital about the study. These in turn informed the patients. A trained dental assistant recruited patients to the dental examination area. The dental assistant performed a preliminary interview to ascertain whether the individuals met the criteria for the study. When in doubt, an opinion of a medical practitioner was sought to confirm the patient's diagnosis by consulting the patient's medical records.

Patients were provided with a consent form (appendix I). They were allowed to ask questions and any information they needed to know was clarified verbally. Only those who signed the document in writing were recruited into the study.

3.11.1 Dental examination

In order to remove operator variability in the recording of data, a single examiner did examinations, The patient was seated on a dental chair with standard overhead light in a well-ventilated room with good natural light. A questionnaire-interview (appendix II) was used to obtain demographic data, dental history, medical history and social history. In order to avoid bias, the medical history was not revealed to the examiner until completion of scoring for periodontal disease. The examiner then counterchecked the information recorded in the questionnaire-interview to confirm that it was correct.

3.11.1.1 Scoring for periodontal disease

Data was captured in a record sheet (Appendix III). Scoring for periodontal disease was done on six "index teeth" as described by Ramfjord, (1967). These are:

i) Maxillary right first molar (16)
ii) Maxillary left central incisor (21)
iii) Maxillary left first bicuspid (24)
iv) Mandibular left first molar (36)
v) Mandibular right central incisor (41)
vi) Mandibular right first bicuspid (44)

If the "index tooth" was missing, the tooth immediately distal to it was used, and the deepest probing depth was recorded. If this tooth was also missing, the tooth immediately mesial to the “index tooth” was used. If no acceptable substitute was found, no information was recorded for that area of the mouth.

The following was scored for:

3.11.1.2 Bacterial plaque

Plaque index (P1I) as described by Loe (1967) (appendix IV) was used to score for bacterial plaque. Scoring was done on mesial, buccal, distal and palatal (or lingual) surfaces of "each index" tooth. A periodontal probe was used for checking presence of plaque. The average score of all the sites was calculated and taken as the plaque score for the patient. Percentage of sites with each of the four scores (0-3) were calculated.

3.11.1.3 Gingival health

Scoring for gingival health was done using gingival index (GI) as described by Loe (1967) (appendix V). Gingival index was scored on mesial, buccal, distal and palatal along the coronal side of the pocket. The average score of all the sites was calculated and taken as the gingival score for the patient. Percentage of sites with each of the four scores (0-3) was calculated.

3.11.1.4 Calculus

Absence or presence of calculus was recorded for each "index tooth". This was examined using a dental explorer probe. The percentage of teeth with calculus was calculated for each patient.

3.11.1.5 Probing depth

This was measured with a graduated periodontal probe from the gingival margin to the bottom of the gingival pocket on six points of each "index tooth". These are midbuccal, midlingual, mesiobuccal, mesiolingual (or mesiopalatal), distobuccal and distolingual (or distopalatal). The average pocket depth of each individual was calculated. The percentage of sites with pocket depths of ≥ 4mm, 6mm and 8mm was calculated.

3.11.1.6 Clinical recession

This was measured with a graduated periodontal probe from the cementoenamel junction to the bottom of the gingival pocket on six points of each "index tooth". These are midbuccal, midlingual, mesiobuccal, mesiolingual (or mesiopalatal), distobuccal and distolingual (or distopalatal). The average clinical attachment loss of each individual was calculated. The percentage of sites with clinical attachment loss of ≥ 4mm and above was calculated.

3.11.1.7 Number of teeth

The number of clinically visible teeth in the mouth was recorded in both the poorly controlled diabetics and controlled Type II diabetic control group.

3.11.1.8 Measuring glycaemic control

To be included in the study the patient was to have had ≥3 HbA1c calibrations recorded in the previous 18 months, and the last recording must have been in the last three months.The results were recorded in their medical notes. These details were confirmed by the dental assistant prior to the clinical examination. On the basis of this information the individuals were categorized into poorly controlled, (with a HbA1c value >7%) or better controlled, (<7%) group.

3.12 Data capture and statistical analysis

Data analysis was done in consultation with a statistician. Data was captured by means of MS Excel, as well as the basic transformations also in Excel©. Statistical analysis was performed using SPSS (Statistical package for social sciences version 14.0) and NCSS (Number cruncher statistical system). Basic descriptive statistics were determined for all the measurements within the two groups (poorly controlled and better controlled). Applicable statistical tests were performed to compare the two groups. Statistical tests, significant at the 5% level ($p \leq 0.05$) were recorded.

CHAPTER 4

Ethics

4.1 Ethics Statement:

Patients who participated in this study had an oral examination. They were asked questions about their dental and medical health and also some aspects of their social habits. The oral examination was a simple, minimally invasive procedure with minimal discomfort.

Patients who were diagnosed as being affected by periodontal disease were offered a referral to the department of Periodontology at Tygerberg hospital or Mitchell's Plain oral health centre. These individuals were placed on a waiting list for a periodontal assessment in the undergraduate clinic, or were referred to the postgraduate clinic depending on the severity of their disease. Alternatively, individuals were advised of their periodontal status and prompted to seek advice and treatment from their local General Dental Practitioner.

Patient participation in the study was voluntary. Participation had no harmful effects and individuals could withdraw from the study any time they wished.

The study was approved by the Ethics Committee of the University of the Western Cape. Informed written consent was obtained from all patients and controls.

All patient information obtained from this study remained confidential, as all medical in confidence protocols were observed.

CHAPTER 5

RESULTS

5.1 Introduction

The influence of the level of HbA1c on the dental health of diabetic patients (Type II) was studied and it was decided to concentrate on female patients so as to exclude variables between the genders. A feature of Type II Diabetics is that there is a HbA1c of more than six per cent. The experimental layout of such a study could be planned in two ways; firstly a group of Type II Diabetics could be compared to a Control Group (without diabetes). Secondly, a "Regression Approach" could be followed, in which Type II Diabetics and the Controls were grouped together. In the "Regression Approach" the influence of the information contained in HbA1c values are better utilised.

The sample size of the total group to investigate the influence of the level of HbA1c on the dental health of diabetic patients by means of the "Regression Approach" was decided to be sixty.

A quarter of the cohort was used as Controls (with HbA1c less than six, as these are classified as Non-diabetic controls). The sample resulted in 47 Type II Diabetics and 16 Control female subjects.

Discussion of Results in Chapter 6

Table 1
Descriptive Statistics of the Ages of individuals in the sample as well as a comparison of the Median Ages

	Diabetic	Non-diabetic	Total
Sample Sizes (The ages of all subjects were available)	47	16	63
Average Age	45.0	39.7	43.6
Median	43.0	36.0	42.0
Standard Deviation	9.50	8.58	9.49
Minimum	30	32	30
Maximum	63	58	63

cx

Wilcoxon Rank Sum Test for the Median Ages
Large Sample Approximation
Test Statistic Z = 2.1396
P-Value = 0.0324

Table 1 illustrated that the difference between the two Medians was seven years. The average age of the Non-diabetic Control Group was less. The minimum and maximum of the two groups were comparable. The age difference would therefore not influence the results of the study. However, the Median ages were significantly different only on the 5% level.

Table 2
Descriptive Statistics of the Mean HbA1c Values of individuals in the sample as well as a comparison of the Median of HbA1c. (The HbA1c was not measured for the Non-diabetics and was assumed to be less than five)

	Diabetic	Non-diabetic	Total
Sample Sizes (The HbA1c values of all subjects were available)	47	16	63
Average of Mean HbA1c Values	9.63	<5.0	8.45
Median	9.60	<5.0	8.47
Standard Deviation	1.96	—	2.64
Minimum	6.1	—	5
Maximum	14.07	—	14.07

Wilcoxon Rank Sum Test
Large Sample Approximation
Test Statistic Z = 5.9372
P-Value = 0

Sixteen healthy female volunteers, with at least nine teeth each, were selected for the Non-diabetic Control Group. Due to the sample structure and the selection of the participants the two groups differed statistically ($p<0.01$ the assumptions of the Wilcoxon Rank Sum Test is not satisfied in this case, mainly because HbA1c was not determined for the Control Group but assumed to be less than six). Professor Hough advised that, to avoid personal complications, it must be assumed that the control group's HbA1c levels were less than six.

Table 3
Descriptive Statistics of the Number of Teeth of individuals in the sample as well as a comparison of the Median of the Number of Teeth

	Diabetic	Non-diabetic	Total
Sample Sizes (The number of teeth of all subjects were available)	47	16	63
Average of number of Teeth	19.98	19.13	19.76
Median	22.00	21.50	22.00
Standard Deviation	6.83	5.99	6.59
Minimum	7	10	7
Maximum	31	26	31

Wilcoxon Rank Sum Test
Large Sample Approximation
Test Statistic Z = 0.5211
P-Value = 0.6023

From the Averages and Medians of the two sub-groups it could be deduced that it was the same for the two sub groups.

Table 4
Descriptive Statistics of the Observed Gingivitis Index of individuals in the sample as well as a comparison of the Median of Observed Gingivitis Index

	Diabetic	Non-diabetic	Total
Sample Sizes (The Observed Gingivitis indexes of all subjects were available)	47	16	63
Average of Gingivitis Index	1.56	1.06	1.43
Median	1.63	1.13	1.54
Standard Deviation	0.43	0.53	0.50
Minimum	0.38	0.08	0.08
Maximum	2.6	1.83	2.6

Wilcoxon Rank Sum Test
Large Sample Approximation
Test Statistic Z = 3.087
P-Value = 0.002

The Medians of the Gingivitis Index of the sub-groups were different ($p<0.01$) and it indicated that the Non-diabetic Control Group had less gingivitis than the Diabetic Group.

Table 5
Descriptive Statistics of Observed Plaque Index of individuals in the sample as well as a comparison of the Median of Observed Plaque Index

	Diabetic	Non-diabetic	Total
Sample Sizes (The Observed Plaque Indexes of all subjects were available)	47	16	63
Average of Plaque Index	1.45	0.97	1.33
Median	1.42	0.94	1.25
Standard Deviation	0.56	0.50	0.58
Minimum	0.45	0.08	0.08
Maximum	3	1.67	3

Wilcoxon Rank Sum Test
Large Sample Approximation
Test Statistic Z = 2.6844
P-Value = 0.0073

The Medians of the Plaque Index of the sub-groups were different ($p<0.01$) and it indicated that the Non-diabetic Control Group had less plaque than the Diabetic Group.

Table 6
Descriptive Statistics of Observed Calculus Index of individuals in the sample and a comparison of the Median of Observed Calculus Index

	Diabetic	Non-diabetic	Total
Sample Sizes (The Observed Calculus Indexes of all subjects were available)	47	16	63
Average of Calculus Index	0.46	0.27	0.41
Median	0.33	0.33	0.33
Standard Deviation	0.30	0.21	0.29
Minimum	0	0	0
Maximum	1	0.67	1

Wilcoxon Rank Sum Test
Large Sample Approximation
Test Statistic Z = 1.9422
P-Value = 0.0521

The Medians of the Calculus Index of the two sub-groups were less different than that of the Gingivitis Index and the Plaque Index but only on the 10% significance level. For the averages the differences were considerable but for the medians the estimate was the same.

Table 7
Descriptive Statistics of Observed Mean Pocket Depths of the Maxilla of individuals in the sample as well as a comparison of the Median of Mean Pocket Depths

	Diabetic	Non-diabetic	Total
Sample Sizes	47	16	63
Count of Observed Maxilla Pocket Depths	39*	11*	50
Average of Maxilla Pocket Depths	3.41	1.91	3.08
Median	3.11	1.83	2.72
Standard Deviation	1.27	0.34	1.30
Minimum	1.9	1.5	1.5
Maximum	7	2.6	7

*8 Missing *5 Missing

Wilcoxon Rank Sum Test
Large Sample Approximation
Test Statistic Z = 4.3092
P-Value < 0.0001

There were a considerable number of missing values due to the absence of teeth in the maxilla's of the participants. Eight subjects had missing values for Maxilla Pocket Depths (17% missing) and five subjects had missing values for Maxilla Pocket Depths (31% missing) in the Diabetes Mellitus and Non- Diabetes Mellitus respectively. The Medians of the remaining observations were significantly different ($p<0.0001$).

Table 8
Descriptive Statistics of Observed Mean Pocket Depths of Mandibles of individuals in the sample as well as a comparison of the Median of Mean Pocket Depths

	Diabetic	Non-diabetic	Total
Sample Sizes (The Mandible Pocket depths of all subjects were available)	47	16	63
Average of Mandible Pocket Depths	2.91	1.93	2.66
Median	2.78	1.75	2.56
Standard Deviation	0.71	0.47	0.79
Minimum	1.50	1.11	1.11
Maximum	4.33	2.89	4.33

Wilcoxon Rank Sum Test
Large Sample Approximation
Test Statistic Z = 4.4766
P-Value < 0.0001

There were no missing values due to the sufficient presence of teeth in the mandibles. The Medians of the observations were highly significantly different ($p<0.0001$).

Table 9
Descriptive Statistics of Observed Mean Recession Values of the Maxilla of individuals in the sample as well as a comparison of the Median of Mean Recession Values

	Diabetic	Non-diabetic	Total
Sample Sizes	47	16	63
Count of Maxilla Recession Values	39*	11*	50
Average of Maxilla Recession Values	1.18	0.69	1.07
Median	1.11	0.67	1.03
Standard Deviation	0.58	0.40	0.58
Minimum	0.06	0.28	0.06
Maximum	2.75	1.56	2.75

*8 Missing *5 Missing
(Not measured)

Wilcoxon Rank Sum Test
Large Sample Approximation
Test Statistic Z = 2.5996
P-Value = 0.0093

Because many participants did not have maxillary teeth Due to the absence of maxillary teeth of the participants there were a considerable number of missing values. Eight subjects had missing values for Recession of the Maxilla (17% missing) and five subjects had missing values for Receding Values (31% missing). The Medians of the remaining observations were significantly different ($p<0.01$).

Table 10
Descriptive Statistics of Observed Mean Recession Values of the Mandibles of individuals in the sample as well as a comparison of the Median of Mean Receding Values

	Diabetic	Non-diabetic	Total
Sample Sizes (The Mandible Recession Values of all subjects were available)	47	16	63
Average of Mandible Recession Values	1.37	0.88	1.24
Median	1.28	0.92	1.17
Standard Deviation	0.68	0.31	0.64
Minimum	0.39	0.33	0.33
Maximum	3.44	1.33	3.44

Wilcoxon Rank Sum Test
Large Sample Approximation
Test Statistic Z = 2.8344
P-Value = 0.0046

There were no missing values due to the sufficient presence of teeth in the mandibles. The Medians of the observations were highly significantly different ($p<0.01$).

5.2 Regression and Correlation Relationships present in the sample

The statistical methods, Regression and correlation analysis, are the two sides of the same coin. The Pearson Correlation Coefficient varies between –1 and +1 and regression has high predictability when the correlation is near to –1 and +1. When the correlation is positive, the two measurements increase together. A negative correlation indicates that as the one measurement increases, the other decreases, an inverse relationship.

Instead of the Pearson Correlations, Spearman Rank Correlation Coefficients, stored in matrices, were used to summarise the relationships between the measured variables Age, Mean HbA1c, Number of teeth, Gingivitis Index, Plaque Index and Calculus Index. Spearman Rank Correlations were used because it is more dependable in the presence of possible outliers.

Table 11(a)

Table of Spearman Rank Correlations; each cell contains the correlation, its significance value, p value, (whether correlation coefficient was different from zero) and the number of data pairs. Measurement headings are indicated.

Only Diabetics

	Age	Mean HbA1c	Number of Teeth	Gingivitis Index	Plaque Index	Calculus Index
Age	1 0 47	-0.234 0.1138 47	-0.421 0.0032 47	-0.006[1] 0.9701 47	0.202 0.1731 47	0.306 0.0367 47
Mean HbA1c	-0.234[5] 0.1138 47	1 0 47	0.073 0.6249 47	0.169 0.2548 47	0.199 0.1788 47	0.025 0.8669 47
Number of Teeth	-0.421 0.0032 47	0.073 0.6249 47	1 0 47	-0.406 0.0046 47	-0.552 0.0001 47	-0.442 0.0019 47
Gingivitis Index	-0.006 0.9701 47	0.169 0.2548 47	-0.406[2] 0.0046 47	1 0 47	0.817[4] 0.0000 47	0.389[3] 0.0068 47

Remarks on the matrix in Table 11(a)

1. No relationship between increasing age and gingivitis index, Spearman Rank Correlation (r_s = -0.006).

2. The gingivitis index shows a significant negative correlation with the number of teeth, i.e. as the number of teeth increases the gingivitis index decreases.

3. The gingivitis index shows a significant positive correlation with the calculus index, i.e. as the gingivitis index increases the calculus index increases.

4. Very strong positive relationship between increasing gingivitis and plaque index, (r_s = 0.817).

5. The mean HbA1c increases as the age decreases, i.e. a negative correlation. However the Spearman Rank Correlation, r_s = -0.234.is not significant at the 10% (0.1) significance level.

The Spearman Rank Correlations of, “Number of teeth”, “Gingivitis Index”, “Plaque Index”, “Calculus Index”, were small and positive and not significantly related to HbA1c.

Table 11(b)
Correlation Matrix of Both Groups- Diabetics and Non-diabetics

	Age	Mean HbA1c	Number of Teeth	Gingivitis Index	Plaque Index	Calculus Index
Age	1 0 63	0.076 0.5542 63	-0.470 0.0001 63	0.134 0.2959 63	0.283 0.0245 63	0.284 0.0241 63
Mean HbA1c	0.076[1] 0.5542 63	1 0 63	0.094[2] 0.4616 63	0.390[3] 0.0016 63	0.365[4] 0.0033 63	0.206[5] 0.1045 63
Number of Teeth	-0.470 0.0001 63	0.094 0.4616 63	1 0 63	-0.326 0.0090 63	-0.453 0.0002 63	-0.334 0.0075 63
Gingivitis	0.134 0.2959 63	0.390 0.0016 63	-0.326 0.0090 63	1 0 63	0.856 0.0000 63	0.483 0.0001 63

Remarks on the matrix in Table 11(b)

1. No significance- there is no relationship between increasing age and HbA1c. But due to the combination of the two samples the negative of Table 11(a) disappeared.

2. No significance- there was no relationship between HbA1c and number of teeth.

3. There was a positive relationship between mean HbA1c and Gingivitis.

4. There was a positive relationship between mean HbA1c and Plaque Index.

5. There was a positive relationship between Calculus Index and mean HbA1c, but it was not significant.

The start of the correlation matrix (four rows and six columns) was presented above. The correlations with "Age" within the complete group (Sample size equal to 63) will be discussed initially because "Age" can be seen as a background variable. There was a strong correlation between **"Age"** and "Number of Teeth" and relationships of lesser importance with "Plaque Index" and "Calculus Index". When the correlations in the first row (with "Age") were contrasted to the corresponding Spearman Correlations, the reduction in the absolute value of the correlation indicated that the relationship weakened in those cases. (See 1)

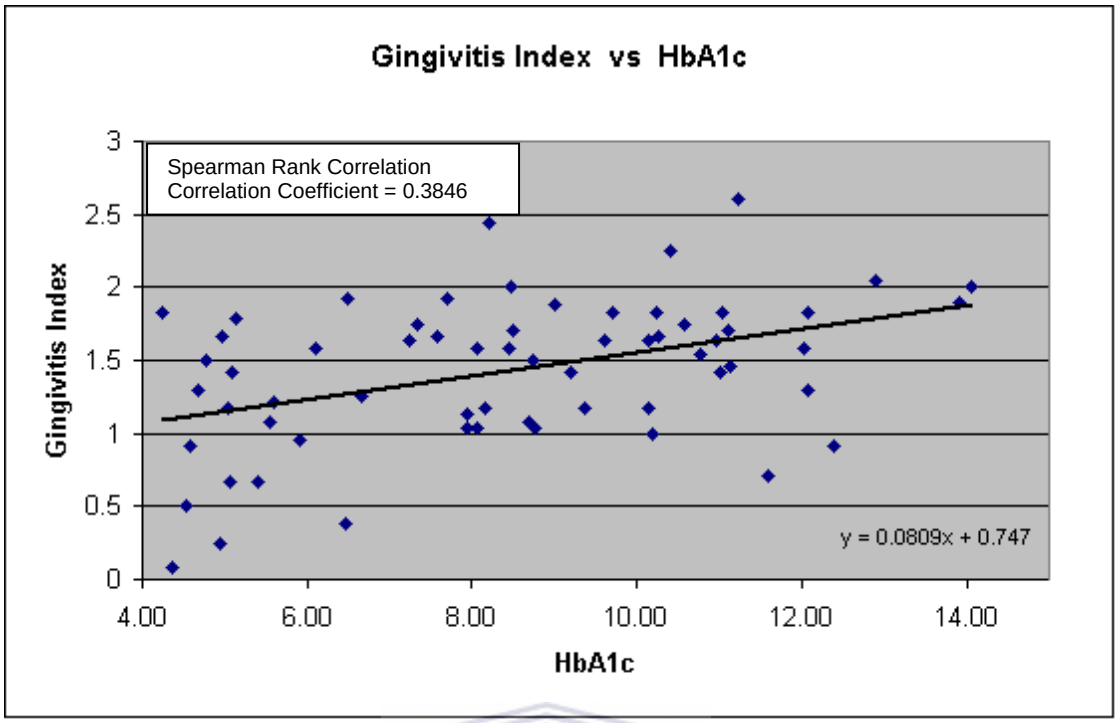

Figure 1
The relationship between HbA1c and Gingivitis Index with the Spearman Rank Correlation indicated in the upper left hand corner and the equation of the fitted least squares line indicated in the bottom right hand corner

A reasonably strong relationship existed between HbA1c and the Gingivitis Index. The linear regression equation fitted, was 'Gingivitis Index' = 0.0809 x 'HbA1c' + 0.747; the slope of this line was 0.0809, implying that for each unit increase in the value of HbA1c the Gingivitis Index increased with 0.0809.

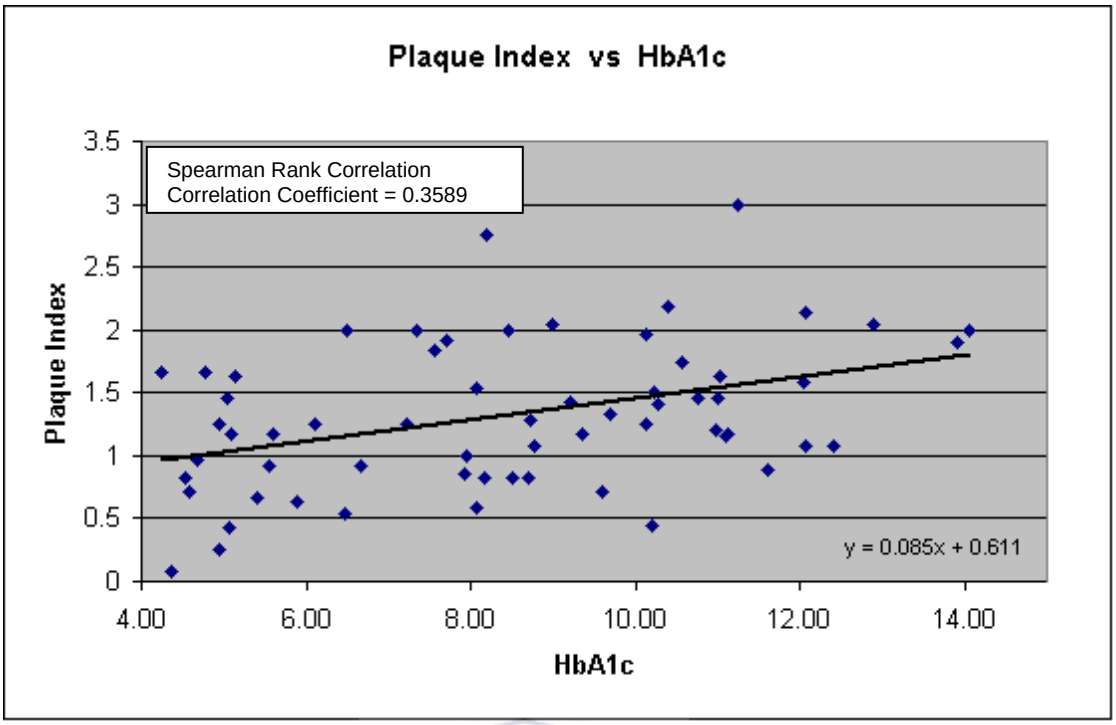

Figure 2
The relationship between HbA1c and Plaque Index with the Spearman Rank Correlation indicated in the upper left hand corner and the equation of the fitted least squares line indicated in the bottom right hand corner

A positive association existed between HbA1c and the Plaque Index and similar deductions could be made as in the case of the Gingivitis Index and HbA1c.

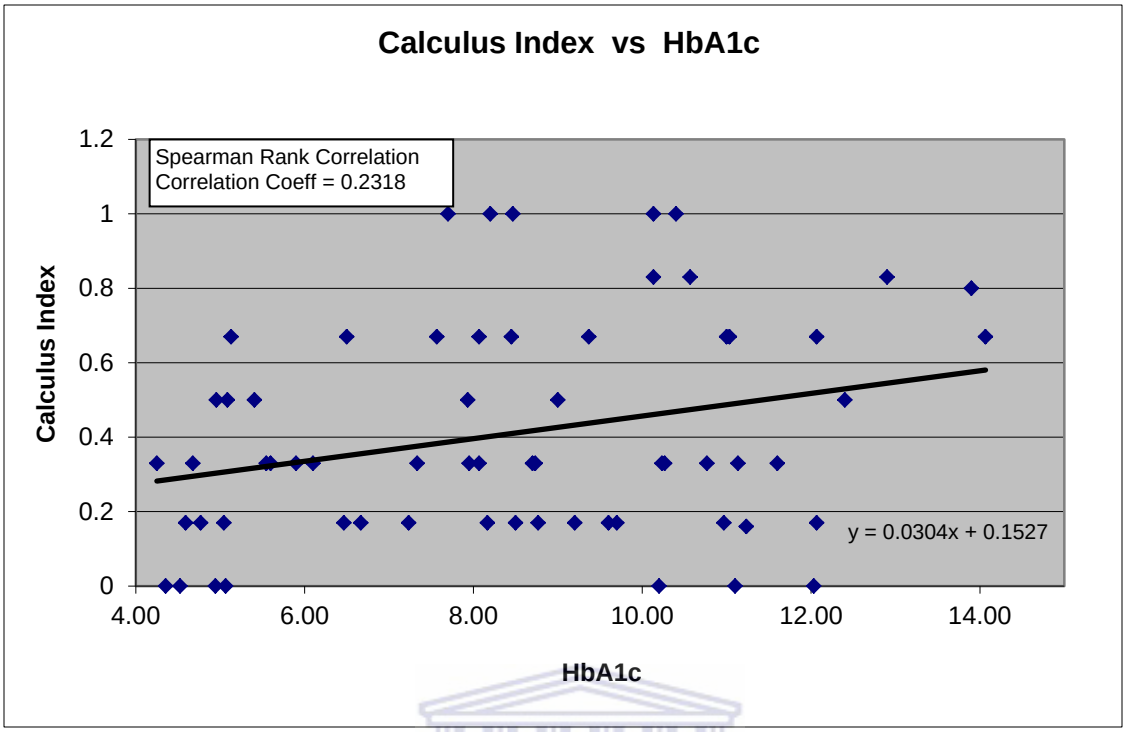

Figure 3
The relationship between HbA1c and the Calculus Index with the Spearman Rank Correlation indicated in the upper left hand corner and the equation of the fitted least squares line indicated in the bottom right hand corner

A weak positive relationship was observed between HbA1c and the Calculus Index.

Table 12
Table of Spearman Rank Correlations; each cell contains the correlation, its significance value, p value, (whether correlation coefficient was different from zero) and the number of data pairs. Measurement headings are indicated.

Correlation Matrix for Both Groups

	Maxilla Pocket Depths					
	Average Pocket Depth Distal Buccal	Average Pocket Depth Buccal	Average Pocket Depth Mesial Buccal	Average Pocket Depth Distal Palatal	Average Pocket Depth Palatal	Average Pocket Depth Mesial Palatal
Age	0.295 0.0377 50	0.439 0.0014 50	0.311 0.0280 50	0.342 0.0152 50	0.347 0.0135 50	0.301 0.0337 50
Mean HbA1c	0.521 0.0001 50	0.464 0.0007 50	0.469 0.0006 50	0.633 0.0000 50	0.503 0.0002 50	0.545 0.0000 50
Number of Teeth	-0.201 0.1616 50	-0.292 0.0394 50	-0.219 0.1269 50	-0.174 0.2270 50	-0.165 0.2533 50	-0.234 0.1019 50
Gingivitis Index	0.736 0.0000 50	0.602 0.0000 50	0.657 0.0000 50	0.665 0.0000 50	0.496 0.0002 50	0.653 0.0000 50

The correlations with “Age” within the incomplete group (50 data pairs remaining, 13 contained missing values), will be discussed at the outset because “Age” can be seen as a background variable. There was a strong correlation between **“Age”** and “Average Pocket Depth Top Buccal” and relationships of lesser extent with **{**“Average Pocket Depth Top D Buccal”; “Average Pocket Depth Top M Buccal”; “Average Pocket Depth Top D Palatal”; “Average Pocket Depth Top Palatal”; “Average Pocket Depth Top M Palatal”**}**.

The aim of this study was to investigate the possible relationship between “HbA1c” and periodontal parameters. The measurement “HbA1c” was strongly related with **{**“Average Pocket Depth Top D Buccal”; “Average Pocket Depth Top Buccal”; “Average Pocket Depth Top M Buccal”; “Average Pocket Depth Top D

Palatal"; "Average Pocket Depth Top Palatal"; "Average Pocket Depth Top M Palatal"**}**. The scatter plot of "Average Pocket Depth Top D Palatal" and "HbA1c" is given below (the Spearman Rank Correlation of this pair of measurements was the maximum in the list of six correlations).

Ignoring the Control Group caused a drop in the significance of the correlations, because the number of data pairs was reduced from 50 to 39. The slopes of the regressions of "HbA1c", however, stayed positive.

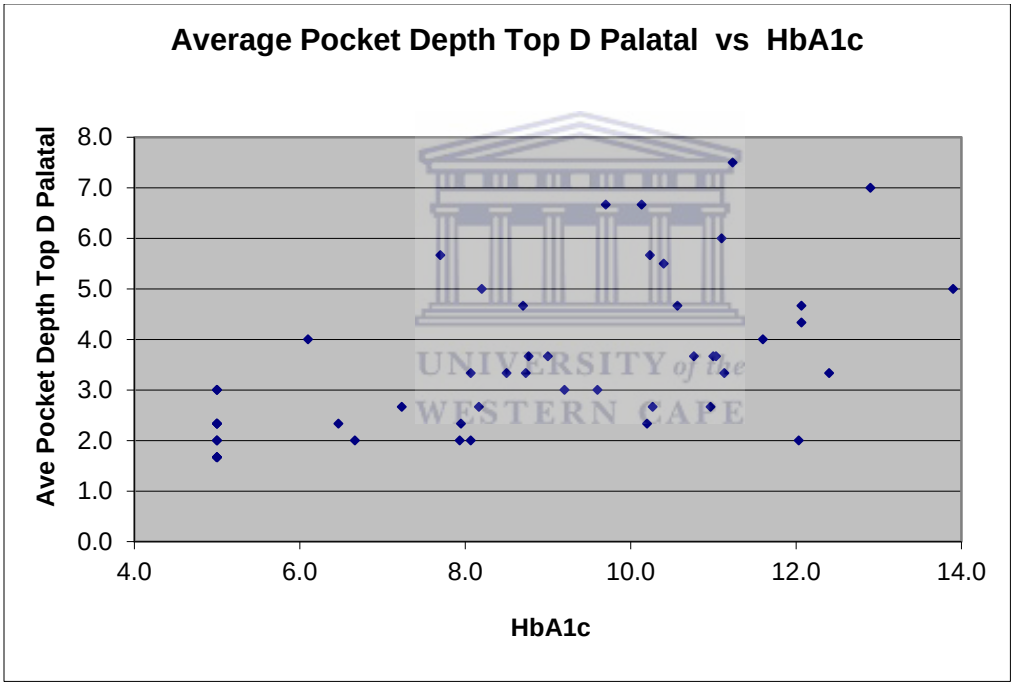

Figure 4
Scatter Diagram of "Average Pocket Depth Top D Palatal" and "HbA1c"

The presence of heteroscedasticity in the relationship between "Average Pocket Depth Top D Palatal" and "HbA1c" could clearly be observed in that the dispersion of "Average Pocket Depth Top D Palatal" for the lower values of "HbA1c" is less than the dispersion of it at the higher values of "HbA1c" e g 12.

Table 13
Table of Spearman Rank Correlations. Each cell contains the correlation, its significance value (whether correlation coefficient was different from zero) and the number of data pairs. Measurement headings are indicated

Correlation for Both Groups

	Mandible Pocket Depths					
	Average Pocket Distal Buccal	Average Pocket Depth Buccal	Average Pocket Depth Mesial Buccal	Average Pocket Depth Distal Lingual	Average Pocket Depth Lingual	Average Pocket Depth Mesial Lingual
Age	0.279 0.0266 63	0.169 0.1858 63	0.379 0.0022 63	0.380 0.0021 63	0.258 0.0415 63	0.355 0.0043 63
Mean HbA1c	0.503 0.0000 63	0.367 0.0031 63	0.572 0.0000 63	0.510 0.0000 63	0.372 0.0027 63	0.505 0.0000 63
Number of Teeth	-0.166 0.1948 63	-0.131 0.3043 63	-0.131 0.3069 63	-0.113 0.3799 63	-0.177 0.1660 63	-0.198 0.1204 63
Gingivitis Index	0.634 0.0000 63	0.515 0.0000 63	0.618 0.0000 63	0.568 0.0000 63	0.485 0.0001 63	0.591 0.0000 63

A similar pattern existed for the correlation coefficients for the "Bottom(Mandibular) Pocket Depths" compared to the "Top (Maxilla) Pocket Depths" except that the "Maxilla Pocket Depths" were calculated on fewer observations due to the higher number of missing values. The gingivitis index had a strong positive correlation to the "Pocket Depth measurements". Interestingly, there was evidence of weak negative correlations between the number of teeth and the "Pocket Depth measurements" implying that, the smaller the number of teeth, the greater the "Pocket Depth measurements".

Table 14
Table of Spearman Rank Correlations. Each cell contains the correlation, its significance value (whether correlation coefficient was different from zero) and the number of data pairs. Measurement headings are indicated

Correlation for Both Groups

	Maxilla Recession Distance					
	Average Receding Distal Buccal	Average Receding Buccal	Average Receding Mesial Buccal	Average Receding Distal Palatal	Average Receding Palatal	Average Receding Mesial Palatal
Age	0.437 0.0015 50	0.321 0.0228 50	0.468 0.0006 50	0.502 0.0002 50	0.393 0.0047 50	0.523 0.0001 50
Mean HbA1c	0.347 0.0135 50	0.162 0.2621 50	0.326 0.0208 50	0.424 0.0022 50	0.310 0.0284 50	0.411 0.0030 50
Number of Teeth	-0.497 0.0002 50	-0.488 0.0003 50	-0.492 0.0003 50	-0.454 0.0009 50	-0.349 0.0129 50	-0.380 0.0065 50
Gingivitis Index	0.308 0.0297 50	0.369 0.0083 50	0.328 0.0199 50	0.451 0.0010 50	0.407 0.0034 50	0.405 0.0035 50

The number of observed pairs for the Maxillae (Table 14) was less than that of the Mandibles (Table 13) and the number of missing values corresponded exactly with that of the "Pocket Depths" of the Maxillae (Table 12). The "HbA1c" correlated reasonably well with **{**"Average Receding Top D Buccal"; "Average Receding Top Buccal"; "Average Receding Top M Buccal"; "Average Receding Top D Palatal"; "Average Receding Top Palatal"; "Average Receding Top M Palatal"**}**. Some of the correlations between "Gingivitis Index" and "Recession Distances" in Table 14 were significantly different from zero. There were much stronger negative correlations between the number of teeth and the "Recession Distances" implying that, the smaller the "Number of teeth", the greater the "Recession Distances".

Table 15

Table of Spearman Rank Correlations and each cell contains the correlation, its significance value (whether correlation coefficient was different from zero) and the number of data pairs. Measurement headings are indicated

Correlation matrix for Both Groups

	Mandible Receding Distance					
	Average Receding Distal Buccal	Average Receding Buccal	Average Receding Mesial Buccal	Average Receding Distal Lingual	Average Receding Lingual	Average Receding Mesial Lingual
Age	0.514 0.0000 63	0.351 0.0048 63	0.512 0.0000 63	0.569 0.0000 63	0.407 0.0009 63	0.514 0.0000 63
Mean HbA1c	0.243 0.0547 63	0.192 0.1325 63	0.256 0.0425 63	0.233 0.0657 63	0.300 0.0169 63	0.242 0.0556 63
Number of Teeth	-0.511 0.0000 63	-0.560 0.0000 63	-0.489 0.0000 63	-0.553 0.0000 63	-0.482 0.0001 63	-0.538 0.0000 63
Gingivitis Index	0.321 0.0104 63	0.412 0.0008 63	0.362 0.0036 63	0.408 0.0009 63	0.506 0.0000 63	0.403 0.0010 63

A similar correlation pattern existed for the “Average Receding Bottom: Buccal & Lingual” compared to the “Average Receding Top: Buccal & Lingual” with respect to “HbA1c”, but the correlations were even weaker than that of the Maxillae.

It could be justified that the regression approach (correlation) be applied to the complete sample of 63 individuals. Most of these correlation coefficients were positive and significantly different from zero indicating that “HbA1c” had a detrimental influence on the dental measurements. This link could be indirect in that some other properties of diabetes, and not necessarily “HbA1c”, affected the dental health of diabetics adversely.

CHAPTER 6

DISCUSSION

6.1 Analysis and interpretation of results

In this study, Type II diabetics had significantly higher gingival index (GI), plaque index (PI), calculus index (CI) and probing depth (PD).

There were obvious difficulties in obtaining the patient numbers due to the high proportion of edentulous individuals attending the diabetes clinic. A regressional approach was therefore used to test the Null hypothesis. The results were conclusive in that the percentage of sites with high GI, PI, CI and PD was higher in diabetics than non-diabetics. The regression analysis showed that as the HbA1c increased so did the GI, PI, CI and PD.

Therefore, the Null Hypothesis was disproved as this means that in this study diabetics suffered from more severe and had a higher prevalence of periodontal disease than non-diabetics.

Many studies have reported a positive correlation between the prevalence and severity of periodontal disease and Diabetes Mellitus . This study confirms the results from previous cross-sectional studies, that Type II diabetics have a higher percentage of gingival sites with deeper probing depths than non-diabetics (Bridges, *et al.*, 1996 and Persson, *et al.*, 2003). In contrast, Tervonen and Knuuttila, (1986) found no difference in percentage of gingival sites involved between diabetics and non-diabetics.

This study showed that there was a difference in GI, PI, CI and PD between Type II diabetics and non-diabetics. In addition, it illustrated a

correlation between HbA1c, PI, CI and PD. These results concur with studies by Taylor, *et al.*, (1996); Tervonen and Knuuttila, (1986); Taylor, (1998); Taylor, Burt, Becker, *et al.* (1998); Tsai, Hayes and Taylor, (2002). Whilst in contrast Bridges *et al.* (1996) reported no difference. These differences might be due to real differences or reasons coming from statistical summary measures for example sample size or outliers.

The use of a control group in this study served as a standard by which the severity and prevalence of periodontal disease in the diabetic group could be measured and compared. The nature of the study was cross-sectional, and does not enable one to conclude a causal relationship between diabetes mellitus and periodontal disease. HbA1c provided a reliable way of assessing glycaemic control in the diabetic group, as described by Kilpatrick *et al.* (1994). Other studies have used other variables such as fasting blood plasma glucose, (Tsai, Hayes and Taylor, 2002; and random blood sugar, Almas *et al.*, 2001).

The critical level for categorising people into well controlled or poorly controlled has been arbitrarily set at different levels in different studies, for example Taylor, *et al.* (1998), used HbA1c <9% for better controlled and ≥ 9% for poorly controlled. In order to avoid misclassifying individuals, it was decided to use the regression approach instead, where the influence of the HbA1c values on the periodontal measurements are better utilised. For applying the regression approach the data on HbA1c had enough dispersion to make valid inferences.

6.2 LIMITATIONS

There are some important limitations to the present study:

Partial mouth recordings protocol was used for the estimation of periodontal diseases which may be accurate and efficient in estimating the mean periodontal measures but could severely under- and/or over-estimate the prevalence of periodontal disease. This effect would not be significant in this study because it was applied to both controls and the diabetic group. So, if underestimation occurred in one group, it was compensated for in the other group.

The sample size was not large enough to represent the Type II diabetes population.

Other risk factors including smoking, lipid abnormalities, renal impairment, and even osteoporosis which not infrequently attend the diabetic state may account for periodontal disease independent of Type II Diabetes Mellitus; but these risk factors were not considered when doing the statistical analysis.

6.3 RECOMMENDATIONS

It is recommended that an association between Diabetes Mellitus and Periodontal Disease must be investigated through large, prospective randomized clinical studies, as well as interventional studies. The preliminary findings deduced form this study must be interpreted with caution due to study limitations.

Multi-centre studies with larger groups of Type II Diabetic populations could contribute favourable to more conclusive results.

Ramfjord (1967) index teeth or their substitutes were used for scoring the various parameters of periodontal disease. This method of recording is very

useful in large epidemiological studies, but may have underestimated disease prevalence in some individuals in this small study. To compensate for this possible bias, these teeth were used in both the diabetic and control group. Ideally, periodontal examination should be performed using a full mouth, recording protocol to establish the effect of glycaemic control on periodontitis more accurately.

As part of their regular medical regimen it would be advisable to educate diabetic sufferers that they are more likely to be affected by severe Periodontal Disease. The importance of controlling their glycaemic levels need to be strongly emphasized.

Both diabetics and non-diabetics in this population should be educated and encouraged to attend more regular dental health checks and have their dental health managed by alternative treatment methods other than just extractions.

Health educators should be aware and advise diabetics that periodontal disease is a complication of their diabetic state and they should be encouraged to maintain their dental health in the optimum condition.

CHAPTER 7

CONCLUSIONS

This study was conducted to investigate whether the prevalence and severity of periodontitis is more prevalent in poorly controlled Type II diabetic patients, and increases on a regressive basis.

The results of the study suggested that there is a higher prevalence of periodontal disease in poorly controlled Type II diabetic patients.
It could be justified that the regression approach (correlation) be applied to the complete sample of 63 individuals. Most of these correlation coefficients were positive and significantly different from zero indicating that “HbA1c” had a detrimental influence on the periodontal measurements. This link could be indirect in that some other properties of diabetes, and not necessarily “HbA1c”, affected the periodontal health of diabetics adversely.

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Appendix I

CONSENT FORM FOR PARTICIPATION IN RESEARCH PROJECT

Title of the Project: The prevalence and severity of periodontal disease in Type II diabetics.

Names of Researchers: Dr James Hyslop (Principal investigator), Prof L Stephen (Clinical co-ordinator).

If you would like to participate in this study please tick the relevant boxes:

1. Have you read and understood the attached information sheet?	*YES*	*NO*
2. Has the purpose of the research project been explained to you?	*YES*	*NO*
3. Do you understand the method of sample collection and the risks involved?	*YES*	*NO*
4. Do you grant permission for information from your medical records to be disclosed to the research team as and when necessary?	*YES*	*NO*
5. Do you agree that data collected for research can be stored for possible use in future research projects conducted by the above named researchers and /or other research collaborators?	*YES*	*NO*

I declare that my participation in this research project is voluntary and that I am free to withdraw from the study at any stage or my approval for use of the sample(s) at any time without giving a reason and without my medical treatment or legal rights being affected. I understand that any information contained in my file will remain confidential and that I (or my doctor) will be informed if any of the results of the medical tests done (as part of the research) have implications for my health. I know how to contact members of the research team should I change my mind about participating in this study.

…….	…….	……
Name of patient (BLOCK CAPITALS)	**Date**	**Signature**
…….	……	……
Name of person taking consent	**Date**	**Signature**
……	……	……
Name of researcher	**Date**	**Signature**

THANK YOU FOR AGREEING TO PARTICIPATE IN THIS RESEARCH – IT IS MUCH APPRECIATED

ADDENDUM A

INFORMATION SHEET

Prospective participants are requested to read this information sheet carefully and to ask questions if anything is not clear, before signing the attached consent form. This sheet must be detached and retained by the participant and the consent form filed in the record.

The purpose of the research project is to compare the periodontal disease present among poorly controlled and controlled Type II diabetic patients attending the diabetes clinic in the Tygerberg hospital. The literature suggests that Type II Diabetes may predispose to periodontal (gum) disease.

The dentist will examine your mouth. Patients will be asked questions about their dental and medical health and also some aspects of their lifestyle history. All diabetic patients will have blood taken for purposes of laboratory investigations of their blood sugar levels; HbA1c recording in order to categorize them accordingly.

The examination of the mouth will use oral clinical indices in making a clinical diagnosis of periodontal disease; it will be carried out by a dentist. The clinical measurements are painless, non-invasive and safe and will be carried out with the utmost care to ensure the comfort of the patient.

It must be brought to the attention of the individual that periodontal disease may present with bleeding gums and that any bleeding during the procedure may therefore be due to this condition (as happens during tooth brushing) and not due to injury inflicted by the dental examiner. The bleeding is usually of short duration and will pose no major threat or discomfort to the individual. If the gums are healthy, no bleeding will occur.

Patients are required to sign the attached form to be able to participate in the study, thereby granting consent for the above-mentioned procedures to be carried out, for the subsequent use of the clinical parameters recorded. The patient will also be required to grant permission for his/her other medical history to be disclosed. Participants will be coded by a number and not be recorded by name, and information will also be coded to protect the identity of the individual. However, the coding will be used by the clinic to trace the individual if relevant medical information (as a result of the study) needs to be passed to him/her or his/her doctor. Where necessary, participants with severe periodontal disease will be referred for treatment.

Participation in this study is voluntary and refusal to participate will not prejudice the treatment of the patient in any way. Consent to participate will be recorded by completing the attached form. Should individuals agree to participate and later change their minds, they may withdraw from the study at any time by calling: Dr James Hyslop, University of the Western Cape,

Department of Oral Medicine and Periodontology, Tel: 021 9373000, or email: jimhyslop@hotmail.com

Appendiks 1
Instemmingsvorm tot deelname aan navorsingsprojek

Titel van die Projek: Die algemeenheid en felheid van die periodentale siekte in tipe II diabeties.

Name van Navorsers: Dr. James Hyslop (Hoof navorser), Prof. L Stephen (Kliniese koördineerder)

As u wil deelneem aan die studie, tik asseblief die relevante blok:

1.	Het u die aangehegde informasie gelees en verstaan?	*JA*	*NEE*
2.	Is die rede vir die navorsingsprojek aan u verduidelik?	*JA*	*NEE*
3.	Verstaan u die metode waardeur die monsters bymekaargemaak gaan word en die risiko's daaraan verbonde?	*JA*	*NEE*
4.	Gee u toestemming dat inligting vanaf u mediese verslae aan die navorsingspan bekend gemaak mag word soos en wanneer dit nodig is?	*JA*	*NEE*
5.	Stem u in dat data wat tydens die navorsing ingesamel word gestoor mag word vir moontlike toekomstige navorsingsprojekte wat deur die bovermelde navorsers en/of ander navorsers-medewerkers gedoen mag word.	*JA*	*NEE*

Ek verklaar dat my deelname aan hierdie navorsingsprojek vrywillig is en dat ek die vryheid het om op enige stadium van die studie te onttrek of my instemming om monsters te neem enige tyd mag staak sonder om 'n rede te verskaf en sonder dat my mediese regte en dienste beïnvloed sal word. Ek verstaan dat enige informasie soos in my mediese lêer vervat, vertroulik sal bly en dat ek (of my Dokter) in kennis gestel sal word indien die mediese toetse enige implikasies op my gesondheid het. Ek weet hoe om die lede van die navorsingspan te kontak indien ek van besluit verander oor my deelname aan die navorsing.

…….	…….	……
Naam van pasiënt (HOOF BLOKLETTERS)	**Datum**	**Handtekening**
…….	……	……
Naam van persoon wie toestemming gee	***Datum***	***Handtekening***
……	……	……
Naam van Navorsers	***Datum***	***Handtekening***

DANKIE VIR U INSTEMMING TOT DEELNAME AAN HIERDIE NAVORSING – DIT WORD BAIE WAARDEER.

BYLAAG A
INLIGTINGSBLAD

Voornemende deelnemers word versoek om die inligtingsblad deeglik deur te lees en om vrae te vra indien daar enigiets is wat onduidelik is, voordat die aangehegde instemmingsvorm onderteken word. Hierdie blad moet losgemaak en gehou word deur die deelnemer en die instemmingsvorm word in die verslag geliasseer.

Die doel van die navorsingsprojek is om die periodentale siekte wat voorkom tydens swak beheerde diabete te vergelyk met die van tipe II diabeties by pasiënte wat die diabetiese kliniek by Tygerberg hospitaal besoek. Die literatuur stel voor dat tipe II diabeties pasiënte moontlik meer vatbaar maak vir periodentale (tandvleis) siekte.

Die tandarts sal u mond ondersoek. Daar sal vrae aan pasiënte gevra word oor hulle mond- en mediese gesondheid en ook seker aspekte van hulle lewenstyl gesiedenis. Daar sal bloed getrek word van alle diabetiese pasiënte, vir laboratoriumondersoeke na hulle bloedsuikervlakke; HbA1c opname ten einde die bloed te kategoriseer.

Die ondersoek van die mond sal mond-kliniese aanwysings gebruik vir die maak van ’n kliniese diagnose van die periodentale siekte; dit sal deur ’n tandarts uitgevoer word. Die kliniese meetings is pynloos, ingreepvry en veilig en sal met die grootste sorg uitgevoer word om die pasiënt se gemak te verseker.

Dit moet onder die aandag van die individu gebring word dat periodentale siekte mag lei tot bloeiende tandvleis en dat die bloeding tydens die prosedure moontlik as gevolg van die toestand is (soos wat gebeur tydens tandeborsel) en nie as gevolg van die besering wat toegedien word deur die tandondersoeker nie. Die bloeding is gewoonlik van korte duur en sal geen groot gevaar of ongemak vir die individu inhou nie. As die tandvleis gesond is, sal geen bloeding voorkom nie.

Ten einde deel te neem aan die studie word daar van pasiënte verwag om die aangehegde vorm te onderteken. Daardeur word ingestem dat die bovermelde prosedure uitgevoer mag word, en dat die gevolglike kliniese parameters gedokumenteer mag word. Daar sal ook van die pasiënt verwag word om toestemming te verleen dat sy/haar ander mediese geskiedenis bekendgemaak mag word. Deelnemers sal gekodeer word deur middel van ’n nommer en nie deur middel van hulle naam nie, en inligting sal ook so gekodeer word dat die identiteit van die individu beskermd bly. Die kodering sal egter deur die kliniek gebruik word indien ’n individu opgespoor moet word ten einde relevante mediese inligting (as ’n resultaat van die studie) aan hom/haar of sy/haar dokter deur te gee. Indien nodig sal deelnemers met ernstige periodentale siekte verwys word vir behandeling.

Deelname aan hierdie studie is vrywillig en weiering om deel te neem sal geen nadelige invloed op die behandeling van die pasiënt hê nie. Instemming tot deelname word bevestig deur die voltooiing van die aangehegde vorm. Sou individue instem om deel te neem en op ’n later stadium van plan verander, mag hulle op enige tydstip van die studie onttrek deur Dr. James Hyslop van die Universiteit van die Wes-Kaap, Departement van Orale Medisyne en Periodentologie te kontak. Die telefoon nommer is 021 9373000 en e-pos: jimhyslop@hotmail.com

Appendix II

QUESTIONNAIRE – INTERVIEW

Date……..... ... Hospital No……

Study No…….. .....

Name……. Race: 1) White ( )

2) Black ( )

Age (years)…… 3) Coloured ( )

4) Indian ( )

Sex: male ( ) female ( ) 5) Other ( )

Occupation……

Residence…….. Duration (years)…….

Income per month (In rands):

1. 0-499 ( ) 2. 500-2399( ) 3. 2400-5000 ( ) 4. >5000 ( )

DENTAL HISTORY

1. What do you think about the condition of your gums?
   a. Excellent ( ) b. Very good ( ) c. good ( ) d. poor ( )

2. Have you visited a dentist in the last: a. 6 months ( ) b. 1 year ( ) c. 2 years ( )
   d. never ( )
   If yes to the above, what treatment was given?
   Oral hygiene advice including tooth brushing instruction ( )
   Scaling and prophylaxis ( )
   Other (specify)……..

3. Do you brush your teeth? Yes ( ) No ( )

   If yes:

   How often do you brush your teeth every day?

   Once ( ) Twice ( )

   Other (please specify)……

4. Do you employ any other method of cleaning your teeth?

   If yes, which one.....

MEDICAL HISTORY

1. Besides diabetes do you suffer from any chronic illness?

   Yes ( ) No ( )

   If yes, specify the condition……

2. Last menstrual period where applicable…….....

   Pregnant: Yes ( ) No ( )

3. Are you on any medication? Yes ( ) No ( )

   If on medication (specify) a) Type……

   b) Duration……

SOCIAL HISTORY

1. Do you use tobacco? Yes ( ) No ( )

2. Do you drink alcohol? Yes ( ) No ( )

   If yes please specify:

   a. Type/form……...

   b. Amount in a 1) Day……

   2) Week…….

   3) Month……

3. Please indicate level of education:

a. Never attended school ( )

b. Primary school education ( )

c. High school ( )

d. College ( )

e. University ( )

TYPE II DIABETES MELLITUS HISTORY (To be completed by a Diabetologist)

Control (past 18months), HbA1C (last 3 readings) -

-

- Mean of last 3 readings

Appendix IV

Plaque index (P11) by Loe, 1967

0 = No plaque in the gingival area

1= A film of plaque adhering to the free gingival margin and the adjacent area of the tooth. The plaque may only be recognized by running a probe across the tooth surface.

2 = Moderate accumulation of soft deposits within the gingival pocket, on the gingival margin and/or adjacent tooth surface, which can be seen by the naked eye.

3 = Abundance of soft matter within the gingival pocket and/or on the gingival margin and adjacent tooth surface.

Appendix V

Gingival index (G1) as illustrated in Loe, 1967

0 = Normal Gingival

1 = Mild inflammation – slight change in colour, slight oedema.*No bleeding on probing.*

2 = Moderate inflammation – redness oedema and glazing.*Bleeding on probing.*

3 = Severe inflammation – marked redness and oedema.Ulceration. *Tendency to spontaneous bleeding.*

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