TOBACCO USE AS A RISK INDICATOR FOR PERIODONTAL DISEASE IN A SAMPLE OF NORTHWESTERN ONTARIO RESIDENTS

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ABSTRACT

Introduction

According to Pairo and Neill (2002), tobacco use has been identified as a significant risk factor in the development and progression of periodontal disease. However, throughout the literature the effects of smoking on individual variables such as bleeding on probing, amounts of plaque and calculus, probing depth, attachment loss and recession are fraught with controversy and ambiguity. The purpose of the present study was to determine the influence of tobacco use on the periodontal health status of Northwestern Ontario residents. Specifically, clinical features of periodontal disease in adults in relation to smoking behaviours were measured. The study will contribute new information about the risk of tobacco use and oral hygiene practices. It is anticipated that this study may be useful to dental professionals, researchers and local health promoters who are challenged with the responsibility of educating people about the negative consequences of tobacco use and the importance of maintaining appropriate oral hygiene practices.

Statement of Problem

- To determine a measure of prevalence of periodontal disease in an urban community in Northwestern Ontario
- To determine the likelihood of periodontal disease between tobacco users and non-users
- To determine the oral hygiene practices between tobacco users and non-users
- To determine the level of periodontal disease in tobacco users versus non-tobacco users

Method

One-hundred adult patients were recruited over a six-month period from a dental clinic in Thunder Bay, Ontario. Participants completed a survey while waiting for their routine dental appointment. The questionnaire was designed to determine tobacco use: frequency, type, duration and quantity. Moreover, the questionnaire was used to collect information about oral hygiene practices, age and gender, and general health. All patients had undergone a clinical and radiographic assessment by the dentist and hygienist. The periodontal status was based on the following clinical measures: gingival bleeding tendency, level of gingival inflammation, levels of bacterial plaque and calculus, periodontal pocket depth, clinical attachment loss, and number of teeth retained.

Results and Conclusion

Tobacco use is a causal mechanism of periodontal disease. The results obtained in this study support previous research, which demonstrated that smokers are at greater risk of developing severe periodontal disease than non-smokers. Odds ratios reported in the present study indicated that tobacco users are twice as likely to show severe periodontal disease. Furthermore, the present study also demonstrated that tobacco users were more than twice as likely to demonstrate higher levels of plaque and both supragingival and subgingival calculus, which are noted precursors of gingival inflammation leading to periodontal disease. While these findings are important alone, the study also showed that self-reported oral hygiene behaviours were not significantly different between smokers and non-smokers, suggesting that despite the best intentions of smokers to self maintain good oral health through subscribing to regular dental visits and practices, periodontal disease and its related sequelae continue to develop.

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1. INTRODUCTION

1.1 Overview and Problem Statement

The impact of smoking on the health of Canadians has been noted extensively in the literature. As demonstrated in the most recent Canadian Tobacco Use Monitoring Survey (CTUMS-2005), 21% of Canadians (approximately 5 million people), aged 15 and over reported that they are current smokers. Further, 16% of the smokers indicated that they smoked daily. A further categorization of smokers by gender indicates that 22% of all males ages 15 and older were current smokers. Comparatively, 17% of women over the age of 15 are smokers (Health Canada, 2005). According to the CTUM Survey, individuals between 15 and 24 years of age are most at risk to initiate smoking. Trends documented in 2003, showed that the proportion of smokers among young adults ages 20 to 24 remained the highest across all age groups (30%), with minimal difference in male and female rates (Health Canada, 2003). The proportion of smokers over the age of 24 tends to decrease with age. Since 1985, fewer Canadians are smoking and those that do smoke are smoking fewer cigarettes (Health Canada, 2003). Although smoking rates and consumption (volume smoked) have been declining since 1985, tobacco use continues to account for more than 21% of Canada's mortality rate in any given year (Health Canada, 2003). In addition, the use of tobacco accounts for a large proportion of the morbidity rates across Canada. Examples of diseases where cigarettes or tobacco products are considered substantial contributing risks are lung cancer and cardiovascular disease; two of Canada's greatest diseases.

In addition to the more commonly known effects of smoking, several studies have identified tobacco use as a risk indicator for periodontal disease (Albandar, 2002a;

Bergstrom & Preber, 1994; Ogawa, Yoshihara, Hirotomi, Ando, & Miyazaki, 2002; Rivera-Hidalgo, 2003). For example, it has been suggested that the smoking attributable risk indicates that smokers were between 2 and 14 times more likely to develop periodontal disease than non-smokers (Grossi, Genco, & Machtei, 1995 cited in Calsina, Ramon, Echeverria, 2002; Linden & Mullally, 1994; Papapanou, 1996 cited in Tonetti, 1998). National clinical epidemiological studies from developed countries have repeatedly estimated that over 90% of the general population has some form of periodontal disease (Borrell, Burt, Gillespie, Lynch & Neighbors, 2002; Morris, Steele, & White, 2002). In addition, it has been reported that between 10 and 20 percent of the population in most countries have severe forms of periodontal disease (Albandar, Brunelle & Kingman, 1999; Hugoson & Laurell, 2000). Although there have been many improvements in other oral health behaviours and treatments, such as a reduction in dental caries, periodontal disease has remained prevalent and shows little sign of improvement in severity (Downer, 1998). Throughout the literature, the effects of smoking studied on individual variables such as, bleeding upon probing, amounts of plaque and calculus, probing depth, attachment loss, and gingival recession are fraught with controversy and ambiguity. However, the literature supports that an association between tobacco use and overall health exists (Figure 1.1).

Figure 1.1 A concept map demonstrating logic flow for the collection of data

Tobacco Use → Periodontal Disease → Systemic Physiology → Overall Health

The use of tobacco is linked with periodontal disease and periodontal disease is linked with systemic physiology, which is subsequently associated with overall health.

Oral health is an important component of the overall well-being of an individual. Research has shown a connection between poor oral health and systemic diseases, such as heart disease, diabetes, respiratory diseases, and premature and low birth weight babies (Health Canada, 2004). Although researchers are still exploring the link between oral disease and general health, gingivitis and periodontal disease lead to oral pain, tooth loss, and poor functionality.

Research Questions

The present study was conducted in order to explore the relationship between oral health, oral hygiene practices and tobacco use in a sample of residents of Northwestern Ontario. The following four specific research objectives were investigated:

- To determine a measure of prevalence of periodontal disease in the region of Thunder Bay, Ontario, Canada. In determining prevalence, the sample was subdivided by age and gender; similarly, within the subdivisions, the data were classified according to existing medical conditions and medication use.
- To determine the likelihood of periodontal disease between tobacco users versus non-users. Particularly, cigarette consumption (volume smoked) and frequency of tobacco use have been investigated.
- To determine the oral hygiene practices between tobacco users and non-users. Variables regarding oral hygiene, specifically, visits to the dentist, frequency of dental cleanings, and frequency of brushing and flossing were compared with frequency of tobacco use and cigarette consumption.

- To determine the level of periodontal disease in tobacco users versus non-tobacco users. Associations between plaque, calculus, bleeding on probing values, clinical attachment level, and probing depths were evaluated across smoking status.

1.2 Rationale

From a Canadian perspective, very little has been published on the relationship between periodontal disease and tobacco use. As a next step in measuring the relationship between periodontal disease and tobacco use in a Canadian population, the present study selected a sample of individuals from a dental clinic.

The intent of this study was to initiate the gathering of data necessary to determine the oral health status, oral hygiene practices, and tobacco use of this particular area. Included in this investigation was information regarding participant knowledge on the effects of tobacco on the mouth, cessation assistance for smokers, and second-hand smoke in the household of both tobacco users and non-users. Additional information regarding alcohol consumption, medication use and existing medical conditions/diseases was obtained.

Although national surveys provide important information on trends in tobacco use and periodontal disease for large populations, studying local information is also required for regions of Canada. This is required to provide an understanding of the oral health status and the oral effects of tobacco use, which will assist in developing and evaluating interventions. An important objective of this research study was specifically intended to determine the relationship between tobacco use and periodontal disease in a sample of individuals from Northwestern Ontario.

If a relationship exists between periodontal disease and tobacco use and the estimated risk is found to be high, additional oral health and anti-tobacco use initiatives can be implemented to target those individuals at greatest risk. The amount and frequency of tobacco use are essential information when evaluating the risk factors associated with smoking (American Academy of Periodontology, 1999). The study will provide useful information for dental and health professionals to further educate patients. The research will provide direction on where the direct focus should be implemented for health education, specifically with regard to tobacco use and oral hygiene practices. The following postulates were made prior to the analysis of the study:

- An association will exist between tobacco use and periodontal disease. It is anticipated that current cigarette smokers will exhibit a more severe form of periodontal disease than non-smokers, whereas former smokers will exhibit some level of periodontal disease between those of the current and non-smokers.
- Oral hygiene practices will differ across the sample.
- The risk of tobacco on oral health status in relation to the prevalence of periodontal disease in the measured sample will be estimable.

1.3 Summary

The present research study will validate the suggested relationship between periodontal disease and tobacco use. As well, the independent examination of oral health variables, such as calculus, plaque and bleeding on probing, in response to tobacco use will contribute to existing literature on the relationship between smoking and periodontal disease. The findings of the research aim to provide a foundation for the link between periodontal disease and tobacco use in Northwestern Ontario and

subsequently inspire further research in this area. Ultimately, more advanced research on this topic could address the oral health of citizens and the needs for implementation of tobacco cessation and oral health promotion experts.

2. LITERATURE REVIEW

2.1 Literature Search Strategy and Search Terms

For purposes of this research, the literature used to formulate the study was drawn from the following databases: Web of Science, Proquest, PubMed, E-Journals @ Scholars Portal, Cochrane Databases of Systematic Reviews. All databases searched were in the English language, with peer-reviewed articles retrieved. Date of publication was not a factor when searching for documents. Information from clinical textbooks was also used. Most articles are recent, although a few are published prior to 1990. In addition to article retrieval, important facts and statistics were obtained from the World Health Organization, Health Canada, and Statistics Canada websites.

The search terms tobacco and periodontal disease were used to search a number of journals including Periodontology 2000, Journal of Periodontology, The Journal of the American Dental Association, and Journal of Clinical Periodontology. In addition, relevant articles were obtained from reference lists using a "snowball approach". The abstracts of the articles were read prior to retrieval for relevance and use in this review. The search strategy used to identify relevant articles involved using combinations of the following categories:

1) Risk factors for periodontal disease: tobacco, smoking, cigarette smoking, oral hygiene, dose-relationship
2) Risk: periodontal disease, risk, odds ratio

3) Variables that define periodontal disease: tooth mobility, bleeding, clinical attachment level, probing depth, bone loss, calculus, plaque

4) Prevalence

2.2 What is Periodontal Disease?

Periodontal disease is an oral disease that includes a variety of conditions characterized by inflammation and degeneration of the gingivae (gums), alveolar bone, periodontal ligament, and cementum (Tortora, 2002).

The alveolar bone comprises the maxilla and mandible, providing a socket where the tooth sits, and is covered superficially by the gingivae. Periodontal ligaments of fibrous connective tissue line the sockets and act to anchor the tooth in the socket. Teeth are comprised mainly of dentin, a calcified connective tissue. Each tooth is made of three distinct parts: the crown, neck, and root. In healthy individuals, the crown of the tooth is the part which is exposed; the root is the portion that is embedded in the socket of the alveolar bone; and the neck forms the junction between the two (Tortora, 2002).

Periodontal diseases are chronic infectious disorders caused predominantly by bacteria (Albandar, 2002a). Generally, they are processes that affect dental support tissues. Although there is no universally accepted consensus regarding the classification of periodontal disease, it can be divided into two major categories: gingivitis which is related to coronal plaque or the smooth supragingival surfaces in the gingival area of the tooth and, periodontitis which is known to be associated with subgingival plaque (Liebana, Castillo & Alvarez, 2004).

Gingivitis:

Gingivitis is a non-destructive form of periodontal disease (Liebana et al., 2004). The accumulation of dental plaque biofilms on clean tooth surfaces results in the development of an inflammatory process encompassing local gingival and periodontal tissues around teeth (Oh, Eber & Wang, 2002). Gingivitis is mediated by host/microorganism interactions, which only involves the gingival tissues and not the underlying periodontal ligament, cementum or alveolar and supporting bone (American Academy of Periodontology, 1992). These clinical syndromes are related to host response and involve microorganisms that create biofilms by colonizing the tooth surfaces of the gingival margin and gingival sulcus (Oh et al., 2002). The term biofilm denotes microbial communities that associate with any surface that does not shed; in the mouth they are known as plaque. The initial clinical findings in gingivitis include redness and swelling of marginal gingival, as well as bleeding upon probing (Liebana et al., 2004). The individual exhibits little or no discomfort at this stage. There is no detectable loss of bone or connective tissue attachment with gingivitis (Oh et al., 2002). As the condition persists the tissue may be fibrotic. Gingival margins that are normally knife-edged in contour may become rolled and interdental papillae may become bulbous and enlarged (Oh et al., 2002). It has also been shown through research that the local inflammation will persist as long as the bacterial biofilm is present, adjacent to the gingival tissues, and that the inflammation may resolve when the microbial biofilm is removed (Oh et al., 2002). Thus, gingivitis is reversible with professional treatment and good oral hygiene.

Bacterial Origin:

Microbiological studies have shown that the oral cavity has a complex and diverse microbial flora comprising of more than 500 different bacterial types. More than 40 different bacterial species have been isolated from gingival conditions with elevated plaque levels. The flora comprise, in most cases, of 50% anaerobic bacteria and 45% strict anaerobic germs and up to 5% treponemas. Anaerobic bacteria are bacteria that do not require oxygen for growth and strict (obligate) anaerobic germs are organisms that die when exposed to atmospheric levels of oxygen. The anaerobic bacteria comprise of oral streptococci and *Actinomyces species*. *Veillonella species* comprise the strict anaerobic germs. Oral streptococci predominate and are typically found at the gingival level of the tooth. The microorganisms survive in areas of lower oxygen reduction potential and between the supra- and sub-gingival areas (Liebana et al., 2004). Equilibrium between the organisms and gum tissues exists. However, a plaque-related gingival disease occurs when there is an imbalance between the two. The presence of non-specific plaque at the gingival portion of the tooth is common to periodontal diseases. Plaque and calculus (tartar) trigger the inflammatory process.

Periodontitis:

Gingivitis is the precursor to periodontitis. The destruction of the dental support system defines the inflammatory disease periodontitis (Liebana et al., 2004). It may follow a course of progressive stages, starting with an initial stage and moving on to an advanced stage; it may be aggressive or chronic; and, can be localized or generalized. Subgingival plaque is located in the gingival sulcus and in healthy periodontia it presents minimal colonization. The presence of periodontitis exists as the amount and diversity of

the microorganisms increases, developing a biofilm and turning the space into a true pocket, which in turn, leads to the destruction of alveolar bone (Liebana et al., 2004). Thus, periodontitis is characterized by gingival pocket formation, loss of the soft tissue attachment of the teeth, alveolar bone loss, mobility, and abscesses, all of which may progress to tooth loss (Page, 1998).

Aggressive periodontitis usually begins during childhood or in early adulthood. There are two types of aggressive periodontitis: prepubertal periodontitis and juvenile periodontitis, both which progress rapidly. Pathogenic bacteria that are associated with the prepubertal type include *Actinobacillus actinomycetemcomitans*, *Capnocytophaga species*, *Porphyromonas gingivalis*, and *Eikenella corrodens*. *Actinobacillus actinomycetemcomitans* is mainly associated with juvenile periodontitis as well as other gram-negative anaerobic rods (Oh et al., 2002). Chronic periodontitis is clinically relevant starting at the age of 35 years, though it may be age independent. It evolves slowly and involves a wide range of microorganisms, such as *Prevotella gingivalis, Prevotella intermedia, Prevotella nigrescens* and others (Liebana et al., 2004).

Other forms of periodontal diseases include infection of non-bacterial origin and non-infectious periodontal diseases. Necrotizing periodontal diseases are infections that are characterized by necrosis of gingival tissues, periodontal ligament and alveolar bone. These lesions are most commonly observed in individuals with systemic conditions, such as herpetic gingivostomatitis, HIV infection, malnutrition, immunosuppression, and others (Albandar, 2002a). Non-infectious periodontal diseases include gingival inflammation caused by mechanical, thermal, and chemical factors. The pathogenesis of gingival inflammation caused by viral infections is not fully understood. The tissue

response to these infections is difficult to assess. Local inflammation will result if the gingival tissues are exposed to a traumatic agent (Albandar, 2002a).

Recently, it has been suggested that tissue destruction in periodontal disease is due to the nature of infiltrating lymphocytes, and is mainly mediated by interleukin-1 (Gemmell, Yamazaki & Seymour, 2002). Population studies have firmly established diabetes mellitus, smoking and poor oral hygiene as risk factors in the development of periodontitis (Albandar, 2002a). Additionally, gender, socioeconomic status, age, race, ethnicity and psychological and genetic factors all impact the incidence of the disease (Albandar, 2002a). Specifically, elderly African-American males of low socioeconomic status are at greater risk for developing periodontal disease. Weinberg, Westphal, Palat, and Froum (2001) stated that the progression of periodontal disease may be modified by stress in addition to these factors. Periodontitis may be related to the development and manifestation of certain systemic diseases, such as cardiovascular disease, cerebral infarction, diabetes and rheumatoid arthritis (Lagervall, Jansson & Bergstrom, 2003).

The various forms of periodontitis differ in etiology; however, they share common pathways of tissue destruction (Page, 1998). Connective tissue degradation and alveolar bone resorption are mediated by matrix metalloproteins and prostaglandins (mainly PGE_2), which are secreted by host cells in response to bacterial toxins (Page, 1998). In recent studies, it has been documented that in most patients periodontal deterioration is not continuous, but progresses in periods of exacerbation and remission (Page, 1998; Socransky & Haffajee, 1997).

2.3 Classification of Periodontal Disease

In 1999, the Workshop on Classification of Periodontal diseases and Conditions proposed a new comprehensive classification system for periodontal disease (Armitage, 1999). The classification is not dependent on age or progression rate. The degree of periodontal disease is classified as localized when less than 30% of the sites are involved and classified as generalized when more than 30% of the sites are involved. Severity of the disease is based on the amount of clinical attachment loss (CAL), with "slight" defined as 1 to 2 mm of CAL, "moderate" as 3 to 4 mm of CAL, and "severe" as 5 mm or more (Armitage,1999). Probing depth alone does not indicate the amount of periodontal destruction. Attachment loss occurs when the epithelium migrates apically from the cemento-enamal junction (CEJ) due to destruction of the connective tissue and bone destruction. Clinical attachment level (CAL) is calculated by addition of the probe depth and recession measurement. Refractory periodontal disease describes disease that is unresponsive to periodontal therapy (Daniel & Harfarst, 2002).

Some researchers question whether gingivitis should be considered a periodontal disease, since it does not cause bone loss or significant loss of periodontal support. However, most researchers have concluded otherwise (Ranney, 1993).

Chronic periodontitis is the new classification of the previous adult periodontitits and does not rely on the age of the patient to make a diagnosis. It is the most common form of periodontal disease affecting both adults and adolescents and has a slow rate of progression (Weinberg et al., 2001). It is directly related to the presence of plaque. Other risk factors in addition to the ones previously mentioned include calculus,

overhanging restorations, and hormonal factors. Tooth mobility may not necessarily be evident in chronic periodontitis.

Aggressive periodontitis is the new classification from early onset periodontitis. Aggressive periodontitis is less dependent on the age of the individual and is divided into localized and generalized. Individuals with aggressive periodontitis have distinct clinical findings from those with chronic periodontitis. Those with aggressive periodontitis are generally healthy except for the presence of periodontitis. They present with rapid attachment loss and bone destruction, which occurs around the time of puberty. Localized aggressive periodontitis affects those under the age of thirty and is localized at the first molar/incisor. Generalized aggressive periodontitis can affect older individuals and is defined by interproximal attachment loss and bone destruction affecting at least three permanent teeth other than first molars and incisors (Weinberg et al., 2001)

2.4 The Definition of Periodontal Disease in Research Designs

Numerous studies use a wide variety of case definitions for disease and different study designs. This variation may affect the results and the evaluation of risk associated with periodontal disease, as well as the magnitude of the risk estimates. The definition of the disease is an important factor that can be expected to contribute to the variation in magnitude of the risk estimate and other comparisons between tobacco use and periodontal disease associations (Bergstrom, 2003). Although general acceptance of a unanimous definition of periodontal disease does not exist, oral health is still studied by using a variety of demarcation points to differentiate disease from non-disease. This wide definition of disease among studies accounts for the wide variation in risk estimates when evaluating the association between cigarette smoking and periodontal diseases. A

narrow definition results in a low prevalence and a high risk, whereas a broader definition will result in a higher prevalence and a lower risk (Bergstrom, 2003).

There is a wide variation among studies regarding diagnostic criteria for the determination and classification of disease. There is extensive variation in regards to the choice of boundary or cut-off borderline for the discrimination of disease from non-disease. Such discrepancies include, choosing one pocket of 5 mm probing depth, one tooth with an attachment loss of 7 mm, or a majority of teeth exhibiting alveolar bone loss exceeding one-third of the root-length. Some other reasons for the large variation may be due to the type of study, and the character and size of the population studied.

Studies in this field indicate various differences in design, conduct and type of measurement of periodontal health. Oral health has been evaluated using various clinical and radiographic variables, as well as different indices (Lagervall et al., 2003). The prevalence and severity of periodontal disease can been based on alveolar bone height, number of missing teeth, oral hygiene, clinical attachment level, probing pocket depth, gingival recession, tooth mobility, bleeding on probing, plaque index, and the amount and location of calculus (Calsina et al., 2002; Lagervall et al., 2003).

2.5 Cigarette Smoking and Periodontal Disease Indicators

It is well established that using tobacco reduces life span and causes an increased incidence of cancer, ischemic heart disease, strokes, myocardial infarctions and chronic lung diseases (Das, 2003). In addition to being detrimental to systemic health, tobacco use also affects oral health. Smoking has been associated with certain oral conditions (acute necrotizing ulcerative gingivitis, candidiasis), cancers, and periodontal disease. The main periodontal disease indicators that have been reviewed for the purpose of this

study are probing depth, clinical attachment level, and radiographic alveolar bone levels, gingival bleeding on probing, and the amount of plaque and calculus levels. Throughout the literature reviewed, former smokers, non-smokers and current smokers are defined as follows, unless otherwise stated. Former smokers are individuals who were not smoking at the time of the study, non-smokers are individuals who have never smoked and current smokers are those who smoked at the time of the study.

Probing Depth and Bone Height

Bergstrom, Eliasson & Dock (2000a) studied the exposure of tobacco smoking to periodontal health in a cohort of professional musician's, ages 20 to 69 years old with a high standard of awareness. In this cross-sectional study of a representative sample of the Swedish population, the mean frequency of diseased sites (probing depth of 4 mm or more) was 16.8, 6.6 and 5.2 among current smokers, former and non-smokers, respectively. This trend was also evident among severely diseased sites (mean probing depth of 6 mm or more), (Bergstrom et al., 2000). This demonstrates a greater frequency of diseased sites among current smokers compared to that of former smokers and non-smokers. The greatest differences existed in the age group of 40 to 69 years, whereas the smoking duration was approximately 30 years compared to 10 years among the younger age groups. This age group presented with a mean frequency of diseased sites of 27.0, 8.5 and 7.8, for current, former and non-smokers, respectively. Whereas, the age group of 20 to 39 years presented differences that were small and insignificant, as the mean scores of frequency of diseased sites were 3.8, 3.1 and 3.2 for current, former and non-smokers, respectively. With increased duration of smoking and age, a greater amount of disease sites are present. Differences among these age groups were similar for severely diseased

sites. This illustrates that symptoms of disease become clinically or radiographically apparent to such an extent that the effects of smoking can be detected after a long incubation period. In addition to higher probing depths in current smokers, a significantly greater bone height reduction was indicated in both current and former smokers compared to non-smokers. Furthermore, a dose-response association between smoking and pocketing was revealed. Those who consumed greater than 10 cigarettes per day exhibited an increased frequency of diseased sites, (= 4 mm), and more reduced bone height compared to those who consumed less than 10 cigarettes per day (Bergstrom et al., 2000; Martinez-Canut, Lorca, & Magan, 1995). In addition to quantity of consumption, duration of smoking habit also affected the probing depth. Long-term smokers of 15 years or more had statistically greater frequency of diseased sites and reduced bone height than short-term smokers (less than 15 years). Smokers with a life-time exposure of 200 cigarette-years or more exhibited a greater frequency of diseased sites than smokers with less than 200 cigarette-years. Current smokers with heavy life-time exposure had a significantly greater frequency of diseased sites than former smokers with heavy life-time exposure ($p < 0.05$) significant in the age group of 40 to 69 years. Therefore, this study found that the effects of smoking appeared in middle age, suggesting a rather long incubation time until symptoms of disease become clinically or radiographically apparent. The periodontal health condition of former smokers in terms of periodontal pocketing resembled that of non-smokers, although the bone height of former smokers was still reduced when compared to non-smokers. This suggests that smoking cessation may be accompanied by a resolution of periodontal pockets and a normalization towards the levels of non-smokers (Bergstrom et al., 2000a).

A cross-sectional study by Schuller and Hoist (2001) found that smoking is positively related to alveolar bone loss. This study generated a hypothesis that the "relationship between smoking duration and alveolar bone loss might be S-shaped" when plotted graphically. The researchers of this study state that this hypothesis should be studied using a prospective design, which may affect the results of this study and the hypothesis generated. This study also confirms that former smokers have an intermediate position for alveolar bone loss between the alveolar bone loss of current smokers and never smokers. This implies that a slow progression of periodontal disease could explain why the initial years of smoking seemed to have no effects on alveolar bone loss. Similarly, a study conducted on 60 to 75 year olds, concluded that it took approximately 30 or more years of smoking to have a significant impact on alveolar bone loss (Persson, Kiyak, Wyatt, MacEntee & Persson, 2005). Clinical measures of probing depth and clinical attachment level reflect the presence of periodontitis, and radiographic evidence of alveolar bone loss reflects the long-term effects caused by all factors on alveolar bone. However, analyzing the risk of bone loss as an effect of smoking failed to identify smoking as a clinically significant risk in this study because they found that it may take a minimum of 30 years of smoking before smoking is a clinical risk factor for periodontitis. Also, tooth loss in this older cohort of adults could have occurred from numerous effects: caries, periodontitis, prosthetic or other reasons which are impossible to evaluate. Since there was only a small difference in the number of remaining teeth between the smoking and non-smoking groups, this study does not provide an indication that smoking can be attributed to tooth loss (Persson et al., 2005). It has been suggested that the past effects of smoking on the periodontium, specifically bone height, cannot be reversed, but that

smoking cessation is beneficial to periodontal health (American Academy of Periodontology, 1999). Bergstrom's study (2004) investigated the magnitude of the long-term influence of chronic smoking on periodontal bone height and found that a reduction of periodontal bone height occurs irrespective of smoking, but the reduction is 2.7 times greater among smokers than non-smokers. This reduction suggests almost a 3-fold elevated bone height reduction rate under the influence of smoking (Bergstrom, 2004). The reduction in bone height was consistent in all quadrants of the dentition and the results are consistent with another study conducted previously (Bergstrom, Eliasson & Dock 2000b).

It is apparent from previous research that smoking has a dose relationship with probing depth. An increase in probing depth further progresses periodontal destruction.

Clinical Attachment Level and Recession

The increase in mean level of attachment loss in smokers compared to non-smokers can be quite remarkable. Schenkein, Gunsolley, Koertge, Schenkein, and Tew (1995) found mean attachment level differences in smokers compared with non-smokers. The differences for 211 adult periodontitis subjects, 112 generalized early onset periodontitis and 141 generalized early onset probands was 0.32, 0.64, and 0.70 mm, respectively. Axelsson, Paulander and Lindhe (1998) in a study of 1093 randomly selected subjects found that mean attachment levels differed significantly between smokers and non-smokers. Mean attachment level differences were 0.37, 0.88, 0.85, and 1.33 mm in the 35-, 50-, 65-, and 75 year olds, respectively. Thus, this study showed that smoking increases attachment loss of smokers compared to those who do not smoke. Haffajee and Socransky (2001) determined patterns of attachment loss by conducting a

study of 289 adult periodontitis subjects. At all levels of mean attachment loss, smokers exhibited more disease than non-smokers. Specifically, individuals at the severe end of the distribution of adult periodontitis who smoked were likely to have about 0.6 to 0.8 mm more mean attachment level loss than subjects with less severe disease who never smoked. There was a significantly greater attachment loss observed at maxillary lingual sites and lower anterior teeth (Haffajee et al., 2001; Haber et al., 1993). This distribution also occurs with pocket depth in that smokers had a greater proportion of sites with a probing pocket depth of greater than or equal to 5 mm in the anterior, premolar and molar regions (Van der Weijden, De Slegte, Timmerman, & Van der Velden, 2001). The distribution of attachment loss and periodontal pockets in smokers versus non-smokers has suggested a local effect of cigarette smoking on the periodontium (Haffajee & Socransky, 2001). In addition, Haffajee and Socransky (2001) found that both smoking and age contributed in a synergistic manner to increased mean attachment level, meaning that the younger subjects who smoked, approximately lost the same attachment that took 27 years to achieve in non-smokers. Martinez-Canut et al. (1995) cited that each extra year of life increases the average clinical attachment loss in each tooth by 0.05 mm (0.7%), which they found to be consistent with other studies. They found that each extra cigarette produces an increase in attachment loss of 0.5%. This 0.5% increase for each cigarette seems only significantly evident from10 cigarettes per day (Martinez-Canut et al., 1995). Age was statistically significant in this study, increasing the severity of periodontitis, but differences were not found between subjects of the 41 to 50 year olds and those over the age of 51, which may due to the older age group having fewer teeth with greater attachment loss. In addition, the researchers found a greater severity of

clinical attachment loss in men than women and noted that this finding is consistent with other studies they have looked at extensively.

Although attachment loss may evolve with age, it increases dramatically as a result of smoking. The greater loss of attachment seen in smokers was clinically observed to have a local effect; affecting only the periodontal support system. Furthermore, smoking displays a dose relationship in regards to clinical attachment level with 10 cigarettes consumed per day being the amount where the effects become most evident. Since attachment loss is one of the main variables that determine periodontal disease, it can be concluded from the evidence provided, that smoking causes periodontal disease by the effects it presents on attachment level.

In regards to gingival recession, Muller, Stadermann and Heinecke (2002) found in a study on young adults ranging from 19 to 30 years old, that there was no significant difference in the prevalence of gingival recession between smokers and non-smokers. This study does not support the hypothesis that smokers are at an increased risk for the development of gingival recession. However, the relative youth may account for the negative findings. Conversely, it was suggested that smoking is a risk factor for gingival recession in adults with minimal periodontal destruction (Gunsolley et al., 1998). This is also consistent with the studies that measured clinical attachment level. Since recession development has been associated with brushing techniques and frequency, (Vehkalait, 1989; Khocht, Simon, Person, & Denepitiya, 1993; Checchi, Daprile, Gatto & Pellicioni, 1999, cited in Muller et al., 2002), it may be considered that excessive usage of a harder toothbrush and abrasive dentifrice might partially explain the development of more recession in smokers than non-smokers. Other studies have also suggested that gingival

recession in older people may be common and not necessarily the effect of periodontitis (Persson et al., 2005). Therefore, some ambiguity exists if recession alone results from tobacco smoking and if it is increased by the habit. The studies conducted regarding clinical attachment level calculate this by adding the value of recession to the value of probing depth. Additional studies must be conducted to determine whether the increase in clinical attachment level is a result of smoking mainly due to the probing depth value and not the recession value. From the studies retrieved, a definite conclusion on the effects of tobacco on recession cannot be made.

Bleeding on Probing

Bleeding on probing is generally apparent 10 seconds after probing. The force used while probing is ideally 0.24 N, which clinically represents a "light probing force" (Lang, Joss, & Tonetti, 1996). Bleeding on probing indicates inflammation within the connective tissue. Disease stability can be monitored during periodontal maintenance on the basis of bleeding on probing. Bleeding sites in deep pockets seem to have an increased risk for progression of periodontitis. The absence of bleeding on probing is a better indicator of gingival health than periodontal disease (Lang et al., 1996). All loss of attachment begins as gingival inflammation. The amount of bleeding may be indicative of the level of inflammation but not necessarily level of attachment loss. It has been stated that it is not unusual, particularly in smokers, to have minimal bleeding on probing even in the presence of severe attachment loss (Daniel & Harfst, 2002). The gingival tissue in smokers is often thin and fibrotic, with a noticeable decrease in marginal inflammation. Clinically, the gingival tissues may appear to look healthy. Bleeding detection is important in ongoing evaluation. Increased attachment loss was evident

when more than 30% of the sites bled when probed at four consecutive recall appointments (Lang et al. 1996).

The effect of tobacco smoking on gingival bleeding

Many local factors may influence the probability of a site to bleed on probing or modify the effect of smoking. The mechanism by which smoking suppresses gingival bleeding is unclear. In their article, "The effect of cigarette smoking on gingival bleeding", Dietrich, Bernimoulin, and Glynn (2004) state that previous studies have confirmed that nicotine causes acute vasoconstriction in skin. However, gingival blood flow is found to increase upon smoking.

In Dietrich's article (2004), it is stated that numerous observational and experimental studies have demonstrated that current cigarette smoking suppresses the gingival inflammatory response to a given amount of plaque. This was measured by bleeding on probing. Dietrich et al. (2004) provides evidence to support the magnitude of the suppressive effect smoking presents on gingival bleeding at the site level. This study investigated a dose-dependent effect of cigarette smoking on bleeding on probing. A dose-dependent association was presented with the least amount of bleeding in heavy smokers, (> 10 cigarettes per day), and greatest amount in former smokers, (= 100 lifetime cigarettes). A lowered bleeding response in former smokers would indicate a more "chronic" effect of smoking. In addition, few studies have examined gingival bleeding of former smokers and the findings were inconsistent (Bergstrom et al., 2000b; Bergstrom & Bostrom, 2001). There was a threshold at which bleeding did not vary among heavy smokers, that is, more than 10 cigarettes per day did not change or decrease the amount of bleeding any further. Thus, the smoking effect reached a plateau at 10 to

20 cigarettes per day. This dose dependent suppressive effect is also in agreement with another study, (Albandar, Brunelle & Kingman, 1999).

Calculus and increased probing depths were associated with increased risk of gingival bleeding; the effect of smoking was more pronounced if either or both of these were present. In healthy sites, heavy smoking reduced the likelihood of bleeding on probing by almost 50%; this is most distinct in sites with calculus and/or increased probing depths (= 4 mm). Thus, in never-smokers bleeding on probing occurs more frequently at sites with calculus and/or increased probing depths. In Dietrich's study (2004), the examiner did not probe to the bottom of the pocket, only 2 mm into the gingival sulcus. When used as a clinical diagnostic parameter, bleeding is assessed after probing to the bottom of the pocket, possibly yielding higher average bleeding scores. Thus, the results cannot be generalized to probing to the bottom of the pocket. Nevertheless, a suppressive effect was demonstrated when not probing to the base of the pocket. This study inferred that the presence of plaque was not a confounding effect, although it was not proven, as the amount of plaque was not taken into account.

Along with many other epidemiological and experimental studies, this study evaluated the association between gingival bleeding and smoking. However, the findings of such research studies are somewhat conflicting. For instance, some researchers have observed less gingival bleeding in smokers than non-smokers (Bergstrom, 1990; Bergstrom & Preber, 1994; Bergstrom & Bostrom, 2001; Calsina et al., 2002, Rivera-Hildalgo, 2003), while others have found an elevation of gingival inflammatory response in smokers compared to non-smokers (Amarasena, Ekanayaka, Herath, & Miyazaki, 2003; Linden & Mullally, 1994). In a cross-sectional study, Muller et al. (2000) showed

that bleeding on probing was less prevalent in non-smokers than smokers with minimal periodontal destruction, as the participants were classified as having mild-plaque induced gingivitis. Al-Wahadni and Linden (2003) found in a case-control study of young adults ages 20 to 35 years that smokers had more sites which bled in response to probing. Still others have shown no significant difference in gingival status, specifically bleeding sites between smokers and non-smokers (Liede et al., 1999, Bergstrom et al., 2000b, Van der Weijden et al., 2001). In addition, only few studies found on smokeless tobacco have failed to demonstrate a significant relationship among the smokeless tobacco users and non-users (Ernster et al., 1990; Robertson et al., 1990). In a cross-sectional study of Sri Lanka residents, betel chewers appeared to demonstrate a greater number of bleeding sites, thus higher levels of gingivitis (Amarasena et al., 2003). Calsina et al. (2002) reported that former smokers showed more bleeding on probing than smokers and non-smokers. Haffajee and Socransky (2001) also concluded that current smokers had less bleeding than non-smokers and past smokers, with past smokers having the highest percentage of bleeding on probing sites. Since the findings are inconsistent, the relationship between tobacco users and gingival bleeding requires further investigation.

Among the studies listed and the findings briefly explained, an inconsistent outcome in results is evident. Moreover, among the inconsistencies, a majority of the studies observed less gingival bleeding in smokers, indicating a suppressive effect of tobacco on the gingival tissue.

Oral Hygiene and Plaque

Oral hygiene is mostly reported by use of questionnaires and plaque is commonly measured by using the Silness & Loe 1964 Plaque Index, which provides a numerical

value that quantifies the amount of plaque present. Some epidemiologic and longitudinal studies use the Simplified Oral Hygiene Index (OHI-S) developed by Green and Vermillion (1964). Methods of measuring plaque are important epidemiologically and are useful to practitioners treating the disease. Oral hygiene self-care is a preventative measure to maintaining a healthy periodontium. The most preventative measure in periodontics is to remove supra- and subgingival dental plaque. Also, brushing should be carried out on a regular basis. The number of times a day an individual must brush is not concrete, since the frequency of brushing is modified to meet individual needs. Factors that affect the recommendation for frequency of brushing include: rate of dental plaque formation, plaque visibility, susceptibility of oral disease, and diet and contributing factors. Low-risk patients defined by the American Dental Association (ADA) are those who have no new or incipient carious lesion and high-risk patients are defined by having a history of numerous caries, frequent sugar exposure, decreased salivary flow, irregular dental visits, inadequate fluoride exposure or compromised oral hygiene. Low-risk patients should brush at least twice a day, whereas high-risk patients should brush more frequently. To support this, the ADA recommends brushing at least 2 times per day and flossing at least once a day because of their high risk for poor oral health. The recommended time for brushing is 3 minutes, although people think they actually brush longer than they do (Weinberg et al., 2001).

Compliance of oral hygiene regimens and differences in plaque scores between smokers and non-smokers:

Most studies reported similar plaque levels for smokers and non-smokers (Bergstrom J, 1990; Calsina G et al., 2002; Haffajee & Socransky, 2001; Axelsson et al.,

1998; Linden & Mullally, 1994). In addition, compliance with oral hygiene instructions was shown to be similar between smokers and non-smokers (Bergstrom, 1990). Dietrich et al. (2004) states that in experimental gingivitis studies, no difference between smokers and non-smokers with regard to plaque accumulation was observed. Conversely, higher plaque levels in smokers were found by some researchers (Bergstrom et al., 2000a; Muller et al., 2002). In a prospective study with a sample of 19 to 30 year old, Muller et al. (2000) adults found that the mean brushing frequencies were comparable in smokers and non-smokers (1.9 ± 0.4 versus 1.8 ± 0.5 times per day). As well, 39% of the smokers and 31% of the non-smokers indicated regular use of dental floss, although these differences were not significant. The smokers had higher mean plaque index scores and a greater proportion of sites covered with supragingival plaque although the tooth brushing frequencies were comparable in this study.

Likewise, Al-Wahadni & Linden (2003), in a case-control study of young adults between the ages of 20 and 35 years, found that smokers brushed their teeth less frequently and were less likely to use accessory methods of interdental cleaning (almost a 50% increase among non-smokers); consequently, smokers had higher plaque scores than non-smokers. It was also reported by Mendoza, Newcomb and Nixon (1991) that smokers were less compliant patients than non-users. Torrungruang et al. (2005) and Rivera-Hildalgo (2003) also reported plaque scores to be highest among current smokers compared to former and non-smokers.

There have been only a few studies that examine oral hygiene regimens and the use of smokeless tobacco. Andrews, Severson, Lichenstein and Gordon (1998) assessed both smokeless tobacco and cigarette smoking with self-reported frequency of brushing

and flossing. Andrews et al. (1998) reports that age and education are positively related to oral hygiene and tobacco use; younger, less-educated, single men reported brushing and flossing less frequently than women, older, more-educated, married patients. Male cigarette users flossed more frequently than male smokeless tobacco users. In contrast, other studies found that most tobacco users met the American Dental Association guidelines for brushing, but not flossing. In addition, non-users brushed and flossed more frequently than both cigarette smokers and smokeless tobacco users (Andrews et al., 1998).

In earlier studies, the adverse effect of smoking on periodontal tissues was masked by the comparable findings of smoking and poor oral hygiene (Arno, Schei, Lovdal, Waerhaug, 1959., Ainamo, 1971, cited in Torrungruang et al., 2005). Presently, it is established that poorer oral hygiene among smokers cannot solely explain their poorer periodontal health (Muller et al., 2000). This concept has been confirmed in a longitudinal study conducted in a population of professional musicians with high standard of oral hygiene. The overall plaque score of these subjects was 0.8 using the Silness and Loe index. Although these subjects exhibited good plaque control, smokers showed increased frequency of diseased sites and more loss of alveolar bone height compared to non-smokers whose periodontal conditions remained stable throughout the 10-year study period (Bergstrom et al., 2000b). Another existing study agrees with the notion that tobacco has a direct periodontal effect, and does not exist solely as a result of poor oral hygiene (Haber et al., 1993).

Therefore, among the literature studied for this section, there are major differences in the results obtained. The main difference being that some authors report

no difference in plaque amounts between smokers and non-smokers, whereas others report higher plaque amounts in smokers compared to former and non-smokers. In the majority of these articles, it has been suggested that tobacco use is a risk factor for low compliance with oral hygiene regimens. Since oral hygiene practices and plaque accumulation could influence the periodontal condition, these factors are likely to be interrelated. On the other hand, some authors found that smokers had higher plaque scores even when they brushed and flossed their teeth just as frequently as non-smokers. Tobacco may have a direct effect on periodontal health irrespective of plaque amounts. Thus, the inconsistency among these research study outcomes directs a need for further research that examines the relationship between smoking and oral hygiene.

Calculus

Calculus is considered the most important local contributing factor for periodontal disease. It is essentially calcified dental plaque, but may even form in the absence of bacteria. Mineralization of bacterial plaque results in the formation of supragingival calculus. The rate at which calculus forms varies between individuals. Subgingival calculus forms more slowly in a thinner layer and is firmly attached to the root. Calculus on the root surface is usually more difficult to remove. Calculus is always covered by plaque and retains toxic bacterial products. Subgingival calculus is commonly deposited in rings or ledges on root surfaces and is associated with progressive periodontal disease. Hence, the removal of calculus will prevent further loss of attachment and promote healing in periodontal patients (Weinberg et al., 2001). The population is more aware of plaque and its relationship to gingivitis than it is of subgingival plaque and its relationship to periodontitis. Awareness is lacking in the need to remove plaque to

prevent periodontal disease and how to remove supragingival and subgingival plaque. Calculus has a porous surface and contributes to the accumulation of plaque. It is unknown if calculus covered with bacterial plaque is more damaging to tissues than plaque alone (Weinberg et al., 2001). The mineralization process occurs separately for supragingival and subgingival plaque; therefore, different amounts can arise in both areas. Supragingival calculus can occur on any clinical crown, exposed root surface or restoration. It is associated most frequently with sites that are adjacent to a salivary source, such as parotid gland and salivary caruncle. The mineral components of calculus are derived from saliva. Subgingival calculus is derived from gingival crevicular fluid and any inflammatory exudates. Subgingival calculus can attach to root surfaces by locking into irregularities in the cementum, or into areas of cementum resorption by an organic pellicle or by penetrating into bacteria (not accepted by all).

Supragingival Calculus

Supragingival calculus deposits are predominant on the lingual surfaces of mandibular incisors and vestibular surfaces of maxillary molars (Bergstrom, 1999). The effects of tobacco smoking on the severity of supragingival calculus have received little attention in terms of research and are ambiguous among the literature. In older studies, it has been claimed that higher calcium levels in plaque and saliva in individuals with periodontitis may reflect an influence of smoking (Sewon, Soderling & Karjalainen, 1990). Bergstrom (1999) reports a significantly ($p = 0.016$) greater amount of supragingival calculus in the mandibular and maxillary quadrants in smokers compared to former smokers and non-smokers. Former and non-smokers had almost similar amounts, with former smokers having slightly more calculus. He also reports that with

increasing life-time exposure to tobacco, the mean score of supragingival calculus also increases, thus heavier smokers had a greater amount of supragingival calculus compared to light smokers. Current smokers had a 3-fold increased risk of exhibiting supragingival calculus compared to non-smokers. Bergstrom (1999) also found that the mandibular regions contained more supragingival calculus compared to the maxillary regions across all three groups (current, former and non-smokers); almost 3 times as much in the mandibular regions compared to the maxillary regions. As well, the influence of age on the occurrence and severity of supragingival calculus was demonstrated, although he reports that the reasons for this are not completely understood.

Subgingival Calculus

Bergstrom (1999) conducted a study on the effect of subgingival calculus and periodontal disease. He reported the prevalence of subgingival calculus (calculus below the gingival margin) among current smokers to be 71% and 53% for former smokers and 28% among non-smokers. The specific differences between current smokers and non-smokers, and former smokers and non-smokers were statistically significant. Radiographic assessment alone was used and thus an underestimation may have resulted because only proximal aspects of the root surfaces were viewed. Bergstrom also found that prevalence was significantly higher in heavy exposure smokers than light exposure smokers in terms of duration and lifetime exposure. One such study conducted by Martinez-Canut, Benlloch and Izquierdo (1999) reported that subgingival calculus was found to be lower in smokers.

In either case, the plaque on the surfaces of the calculus contains living bacteria and is detrimental to the tissue. In this sense, calculus is a contributing factor to

periodontal diseases. Specifically, the role of supragingival calculus is a major contributor to periodontal disease among smokers. Since tobacco may increase calculus levels, which in turn increase the amount of periodontal destruction, there is a direct association between periodontal disease and smoking by association of supragingival calculus. However, there seems to be some controversy in regards to the role of smoking in subgingival calculus deposition. Subgingival calculus and smoking in Bergstrom's study may simply reflect a confounding influence of periodontal disease, as periodontal disease is more prevalent and severe in smokers.

2.6 Attributed Risk of Periodontal Disease: Smokers versus Non-smokers

In a review by Albandar (2002a), the author indicated that cross-sectional studies have consistently shown a higher prevalence, extent and severity of various periodontal disease outcomes in smokers than non-smokers. This review also identifies that smoking is associated with between a 2 and 7 fold increase in risk for having periodontitis and/ or having periodontal tissue loss compared to non-smokers. A study assessed the occurrence of severe loss of periodontal attachment and deep probing depth in cigarette smokers and non-smokers who were regular dental attendees and found a significant increase in risk (OR = 14) in young adult smokers aged 20-33 years of age compared to non-smokers of the same age (Linden & Mullally, 1994 cited in Albandar 2002a). Increased risk from smoking has also been found in older age cohorts (Jette, Feldman & Tennstedt, 1993 cited in Albandar 2002a).

In a review of recent publications (1999-2002), Rivera-Hidalgo (2003) reported the relative risk for smokers was 3.97 and the risk for former smokers was 1.68. These values were retrieved from the data of the United States Third National Health and

Nutrition Examination Survey. The odds of periodontitis have also increased with smoking exposure. The odds of acquiring periodontal disease increased from 2.79 if nine or fewer cigarettes were smoked per day to 5.88 for 31 or more cigarettes per day. The odds of acquiring periodontal disease decreased to 3.22 after quitting for the first two years and decreased to 1.15 after 11 or more years. In another such study, (Hashim, Thomson & Pack, 2001, cited in Rivera-Hidalgo, 2003), reported that if smoking occurs from mid-adolescence and into adulthood, the likelihood of periodontitis would double by the mid-twenties.

A cross-sectional study conducted in a remote Canadian community, found that the strongest association observed was with smoking, which had an odds ratio of 6.3 compared to missing teeth, dental visiting, flossing frequency, and age (Sbaraglia, Turnbull & Locker, 2002). Each of these variables showed independent effects. This study had a high odds ratio, which may be attributed to the younger age population (mean age 38.1 years, SD = 11.5), although the age studied was from 21 to 82 years old. A possible limitation to this study was in the methodology, as only 2 sites on each tooth measured for probing depth. Therefore, the results may have been different if all six sites on each tooth were measured for probing depth/ clinical attachment loss.

In summary, the majority of the evidence suggests that smoking is a significant risk factor for the development of periodontal disease. Among these studies the majority of them have been conducted in U.S. Risk estimates may differ according to demographics between and among U.S. and Canadian citizens.

2.7 Prevalence of Periodontal Disease

A review of the epidemiology of periodontal diseases in the North American populations has been conducted and is based on a systematic review of relevant studies published in peer-reviewed journals. There are a greater number of surveys that have been conducted in the United States, which assess the epidemiology of periodontal diseases. However, there are only a few studies found from Canada.

The prevalence of periodontal problems among adults in industrialized countries has been the subject of several studies. National clinical oral epidemiological studies from developed countries have repeatedly estimated that over 90% of the general population has some form of periodontal disease (Morris, Steele & White, 2002, Borrell, Burt, Gillespie, Lynch & Neighbors, 2002). In addition, it was reported that between 10% and 20% of the population in most countries have severe forms of periodontal disease (Albandar et al., 1999, Hugoson & Laurell, 2000). However, despite the dramatic improvements in other oral health states in recent decades, such as dental caries, periodontal disease has remained prevalent with little signs of improvement in the severity of the disease (Downer, 1998).

The World Health Organization's (WHO) compilation of more than 100 studies measuring the Community Periodontal Index of Treatment Needs (CPITN) indicates that most adults present with calculus or gingival bleeding or both, and depending on the country, 5% to 20% of people 40 years of age suffer from severe periodontal diseases (Miyazaki, Pilot, Leclercq & Barmes, 1991). Many studies of periodontal disease experience have used the Community Periodontal Index of Treatment Needs (CPITN), the limitations of this measure for characterizing the periodontal disease experience of

populations has led to its replacement by more valid indicators, such as mean periodontal attachment loss.

Periodontal problems continue to affect millions of Americans. In the United States, Albandar (2002b) reviewed the prevalence of periodontal disease in the population based on the results of 3 national studies: National Health and Nutrition Examination Survey (NHANES I (1971-74), III (1988 to 1994) and National Institute of Dental Research Survey (NIDR) of 1985-86. Douglass et al. (1993) also reviewed NHANES I, NIDR, National Health Examination Survey (NHES of 1960-62) and the Research Triangle Institute from 1981. Douglass et al. (1993) found that the proportion of adults with periodontal diseases decreased from 1962 to 1986. However, because these studies used different sampling and measurement methods, it was not possible to know whether the decline was valid or simply a result of these differences (Douglass et al., 1993). Nonetheless, all five studies showed that the proportion of persons with periodontal diseases increased with age and was higher among black men than in white men.

NHANES I conducted during 1971-1974 included a sample of approximately 28,000 subjects. It utilized a periodontal index (PI) designed by Russell Al (1956) to assess the periodontal condition. Russell's index merely relies on visual inspection using a dental mirror to estimate the severity of disease and does not include probing or clinical attachment measurement of teeth. Thus, Russells's PI has major limitations in its validity to assess periodontal disease and gingivitis, due to its subjective nature of measurement. The use of this survey in the NHANES I survey is a drawback, thus the findings regarding the prevalence and severity of periodontal disease in the U.S. population may

hold little value. However, some inferences could be made. A greater number of males versus females had periodontal disease. As well, periodontal disease was higher among blacks than whites and increased with age (Albandar, 2002b).

NHANES III was conducted during the period of 1988-1994 and included 30, 818 people, 2 months and older. It looked at such key indicators of periodontal disease as attachment loss and gingival bleeding. The study found that 31% of U.S. adults aged 30 years and older had advanced periodontitis, 9.5% had moderate, 21.8% had mild periodontitis and 65.5% had no periodontitis. However, moderate and advanced periodontitis increased in prevalence between 30 and 70 years of age, leveled off and slightly declined thereafter. Moderate attachment loss of 3-4 mm was found in 30% of 25 to 34 year olds, in 63% of 45 to 54 year olds and in 80% of 65 year old individuals and older. Overall, women had better periodontal health than did men, and whites had fewer periodontal problems than did blacks or Mexican Americans. The survey found that the prevalence and extent of periodontal attachment loss increased with age (Albandar, 2002b).

It is important to note that in the NDIR and NHANES III studies, the proportion of periodontal pockets was underestimated, as measurements were taken on only 2 sites (mesiovestibular and vestibular sites) rather than all around the tooth.

Borrell et al., (2002) compared the results of the NHANES III and NHANES 1988-2000. The study analyzed non-Hispanic black, non-Hispanic white, and Mexican-American adults aged 18+ years in the NHANES III (n = 12,088) or the NHANES 1999–2000 (n = 3214). The prevalence of periodontitis for the NHANES III and the NHANES 1999–2000 were 7.3% and 4.2%, respectively. In multivariable analyses, blacks were

1.88 times (95%CI: 1.42, 2.50) more likely to have periodontitis than whites surveyed in the NHANES III. However, the odds of periodontitis for blacks and Mexican-Americans did not differ from the whites surveyed in the NHANES 1999–2000. Their findings indicated that the prevalence of periodontitis has decreased between the NHANES III and the NHANES 1999–2000 for all racial/ethnic groups in the U.S.

Hujoel, Bergstrom, del Aguila and De Rouen (2003) reported on the hidden periodontitis epidemic during the 20th century in the U.S. predominately due to the causal link between smoking and periodontitis. In this study, it was estimated that the incidence of advanced periodontitis decreased by 31% between 1955 and 2000. In his report he identifies that the changes in smoking habits, and consequently the changes in the incidence of periodontitis, depended strongly on education and gender. Between 1966 and 1998, an estimation of 43% decreased periodontitis incidence among college-educated individuals versus only 8% decrease among individuals with less than a high school education occurred. Between 1955 and 1999, this study calculated a 41% decrease among males versus a 14% decrease among females. He also predicts that the incidence may decrease to 43% by 2020 from its level in 1955. Smoking was not identified for most of the 20th century as one of the main risk factors for chronic periodontitis, thus a hidden periodontitis epidemic fueled by smoking may have occurred during this time. At the beginning of the 20th century smoking was most prevalent among males and among higher socioeconomic classes in the developed world. It is now becoming increasingly prevalent among lower socioeconomic classes and women, and in certain developing countries. These shifts in socio-demographic patterns of smoking are already affecting the periodontal profession. The harm associated with cigarettes manufactured in the

middle of the 20th century may have been underestimated due to the lack of filters and relatively higher nicotine and tar delivery. A limitation to the study conducted by Hujoel et al., (2003) was the inability to verify the extent at which the periodontal disease incidence changed during the 20th century, such as the use of diagnostic codes. The only data available to track changes in periodontitis for epidemiological studies are substituted periodontitis markers collected during national surveys. Due to this inability to confirm the extent of periodontal disease Hujoel et al. (2003) cited that "the incidence of periodontal disease is unknown" and that no reliable trend data of the epidemiology of periodontitits is available.

In Canada, there are very few studies on the prevalence of periodontal disease indicators among adults. Hoover and Tynan (1986) conducted a study consisting of 260 adults aged 19 years and over living in Saskatoon, Saskatchewan. A periodontal examination was performed on 4 sites per tooth on a total of 6 teeth. The authors reported that among subjects ages 30 to 44 years old, 34% had 4 to 5 mm periodontal pockets and 15% had periodontal pockets of = 6 mm. The Nutrition Canada National Survey was conducted in 1971-72 and showed that 26% of Canadians aged 19 years and older suffered from serious gingivitis and 15% had periodontal pockets. A study conducted in Ontario, Canada examined periodontal disease in 624 adults ages 50 years and older (Locker & Leake, 1993). They reported 76.6% to have an attachment loss of 2 mm. Only 12.8% had fewer than half their sites showing evidence of previous disease. Although this study utilized the CPITN as their form of measurement, the study has contributed valuable information on periodontal attachment loss and risk indicators and markers for periodontal disease in Canadian adults. The extent and severity of disease in

this population was greater than in some recent U.S. reports. They reported that the most consistent independent effects as assessed via multivariate analyses appeared to be age, education, current smoking status and number of teeth.

A cross-sectional study completed in 1994-1995 in Quebec, assessed the periodontal health of the Quebec population aged 35 to 44 years (Brodeur et al., 2001). This study reported that there was a high prevalence of calculus and gingival bleeding (50%) for both on at least 5 teeth. This study also used the CPITN, which evaluated periodontal treatment needs. Although it holds limitations in validity, the study indicated that 67.8% of individuals were classified as presenting with calculus or a periodontal pocket of 4 to 5 mm, or both, on at least one tooth. As well, 21.4% of the subjects had at least one tooth with a periodontal pocket = 6 mm. According to the CPITN, only 5.2% of this group did not require any treatment needs. This study only examined a specified age group and did not assess the smoking habits of this population. The prevalence rates of various periodontal parameters were significantly higher in this study compared to the U.S. national survey. Differences in study design and examination methodologies should not be disregarded. For example, the examination of the this cohort (Canadian) used the WHO's CPITN periodontal probe and a significantly longer examination time than in the NHANES III survey. Additionally, the CPITN records the worst condition for each tooth in the dentition and only uses a fixed subset of teeth which is predetermined (Albandar & Kingman, 1999). A recent recommendation of the WHO is to evaluate attachment loss of the 10 index teeth. The CPITN does not evaluate attachment loss of the 10 index teeth, which leads to a lower estimation of periodontal disease (Albandar & Kingman, 1999). In addition, the study of the Quebec population examined the worst site around the tooth

on all teeth, versus only two sites in two quadrants in the NHANES III survey; a more comprehensive examination in the Canadian study may have influenced the outcome. Individuals with low socio-economic status may contribute to a higher level of disease (Brodeur et al., 2001), as well as other individuals such as minorities of certain ethnic groups with other risk factors and health concerns.

A more recent study evaluated periodontal attachment loss, number of teeth, oral hygiene habits and smoking in a remote Canadian community located in Kirkland Lake, Ontario (Sbaraglia et al., 2002). This study collected data from 187 adult patients. The smoking status was reported by use of a questionnaire; 39.6% reported to be current smokers, 14.4% were former smokers, and 46% claimed they had never smoked. Among this group of individuals, 48.1% of subjects had a mean loss of 4 mm or more, while 9.6% had a mean loss of 6 mm or more. The mean proportion of sites per subject with loss of 2 mm or more was 0.89 and 0.35 for a loss of 5 mm or more. The overall mean loss was quite higher: 3.89 mm compared to 2.95 mm reported in Locker and Leake's (1993) study, indicating that the periodontal health of the former to be poorer. This higher value may be attributed to the sample of people not attending a dentist regularly and also possessing high rates of smoking with relatively poor oral hygiene practices. The age variation among the Canadian studies could also account for the differences in results, although this is not verified.

There is a lack of information on the periodontal health status of Canadians living in smaller cities and even rural communities. Thus, further research will contribute to the knowledge regarding the prevalence of periodontal disease in smaller Canadian cities.

2.8 Prevalence of Tobacco Smoking in Canada

The latest results from the Canadian Tobacco Use Monitoring Survey (CTUMS) confirm that the prevalence of smoking continues to decline in Canada. According to CTUMS, in the first half of 2002 an estimated 5.4 million people representing roughly 21% of the population aged 15 and older, were current (daily or occasional) smokers. Approximately 23% of men aged 15 and older were current smokers, slightly higher than the proportion of women, which is 20% (Health Canada, 2005).

In 2005, Canadian per capita (age 15+) cigarette consumption (including roll-your-own) fell for the ninth consecutive year: 1997-2005 inclusive. Based on annual Statistics Canada sales data released, per capita sales volumes including roll-your-own fell by 7.3% in 2005. For the nine years, the cumulative decline in per capita consumption is 39.1% as compared to 1996. In Ontario, according to Statistics Canada data, 25% of the smokers in 1994-1995 quit by the year 2000-2001. British Columbia again reported the lowest prevalence of smoking among Canadians aged 15 years and older (17%), closely followed by Ontario (19%). The highest rates were in Quebec (27%), and Newfoundland and Labrador (25%). Quebec also reported the highest average number of cigarettes consumed per day by daily smokers (17.4), closely followed by Prince Edward Island at 17.3 and Saskatchewan at 17.2, while Manitoba reported the lowest average (14.7). Not only are fewer Canadians smoking, but they are also smoking less. In 1985, daily smokers consumed an average of 20.6 cigarettes per day. Since then, the number of cigarettes smoked has been gradually but steadily declining to the current level of 16.4 cigarettes per day, reported for the first half of 2002. Men continue to

smoke more than women: 17.7 cigarettes per day for males as compared to 14.9 for females (Health Canada, 2005).

Pairo and Neill (2002) acknowledge that the percentage of the adult population that smoke cigarettes has been declining since the 1970s, but this decline is less among women and certain minorities. In addition, they cite that tobacco use has grown more popular among youth (Garfinkel, L 1997, cited in Pairo and Neill, 2002).

2.9 Summary

Throughout the literature, a consensus does not exist in regards to oral health indicators and smoking. The oral health indicators that predominately identify periodontal disease are bleeding on probing, clinical attachment level, probing depth, recession and plaque and calculus scores. In addition, there is minimal research on these factors and the impact of smoking on Canadian populations. This study specifically targets residents of Northwestern Ontario and aims at identifying the prevalence of periodontal disease, the likelihood of periodontal disease between tobacco users and non-users, the standard of dental care and finally the oral hygiene practices of tobacco users and non-users. The literature review illustrates a need for further research conducted in Canada, specifically at the local level. As well, it demonstrates the need for further confirmation of the association between periodontal disease and tobacco use.

3. RESEARCH METHODOLOGY

3.1 Participant Selection and Sampling Recruitment

University Ethics Review

The study was approved by the Research Ethics Board of Lakehead University. A researcher's agreement form, proposal and a copy of the research instruments utilized including the cover letter and informed consent were provided and reviewed. Upon ethics approval from Lakehead University, potential dental facilities were contacted to explain the research and request their participation in the study. Participants were only included in the study after they signed the letter of informed consent form.

Sampling and the Characteristics of Subjects

The sample size was determined from a Statistics Canada reference population of approximately 82,000 adults older than 20 years of age. This age range accounted for approximately 75% of the population of Thunder Bay. Of the number of adults, 34.5% were expected to have periodontal disease, where periodontal disease is defined as the presence of attachment loss of 3 mm or more together with a presence of a probing depth of = 3 mm at the same sites (Albandar, 2002a). The sample size estimated for this study was 100 adults age 20 years and older from a dental clinic in Thunder Bay, based on an a level of $p < 0.05$ with 10% accuracy of estimation (Appendix B1). The researcher is confident that the sample size allowed for generalizations of the adult population in Thunder Bay and other Northwestern Ontario residents.

The final sample consisted of individuals who were at least 19 years of age. The sample was designed to include persons who receive regular dental care, as well as those who visit the dentist infrequently or on an emergency basis only. The sample was

restricted to an adult cohort because of the legal restriction in the province of Ontario concerning tobacco use. It was assumed that periodontal disease and gingival status might be associated with oral hygiene and tobacco use behaviours.

3.2 Instrument Development

The study methodology combined a self-reported questionnaire and specific clinical variables to identify oral health status (Appendix A1-A3). The questionnaire collected the following information: i) demographics—age, and gender; ii) health — under the care of a physician, the presence of a condition or illness, the use of prescribed medications; iii) previous periodontal surgery or special treatment/cleanings involving the gum tissue (Appendix B2). Similarly, oral hygiene practices were determined by regularity of dental visits, frequency of professional dental cleanings, frequency of brushing, frequency of flossing, and the use of additional dental aids. Questions regarding patient knowledge on the effects of tobacco on the mouth and second hand smoke exposure and cessation were also included. Tobacco use was determined from questions about the use of cigarettes, cigars, chewing tobacco and pipes. Current tobacco users were defined as those who currently used tobacco products at the time of completing the survey. Former tobacco users were those who had quit at the time of completing the survey. Non-tobacco users were individuals who indicated that they had never used tobacco products. Type, quantity, frequency and duration of tobacco use were also recorded. The questionnaire used a combination of multiple-choice, yes/no, and fill in the blank styles. Questions were selected from previously published Centre for Disease Control Oral Health, Smoking and Tobacco Use Questionnaire and the Canadian Tobacco Use Monitoring Survey.

After collecting the questionnaires, the researcher reviewed the medical history of the participants to ensure that they were reported to be in good general health. Individuals that had a medical condition(s) were included in the study but categorized according to type and number of conditions.

3.3 Method of Data Collection

The data set for the present study consisted of responses on the questionnaire and results from oral/periodontal examination records.

Recruitment Procedures for Dental Clinics

The main source of dental record information used in this study was the Confederation College Dental Clinic. This site was selected because it is centrally located in Thunder Bay, there are comprehensive dental records available for each patient, and all participants selected from this clinic will have had a standard dental examination using all clinical variables as well as a full mouth probing depth assessment using six sites for each tooth. In addition, the dental charts at the selected clinic were easily accessible to the researcher. The letter to request their participation and the consent form are included in Appendix A4 and A5.

Recruitment Procedures for Dental Patients

As patients arrived at the clinic for their regular appointment, they were greeted individually by the researcher. The patient was then asked if they would be willing to participate in the study (Appendix A6). If they agreed, the purpose and expectations of the research was explained to them. Participants completed the questionnaire booklet while they were waiting for their appointment. The questionnaire booklet also included a cover letter, and the letter of informed consent. Patients agreeing to participate in this

study gave signed permission to the researcher to access information from their dental records. On average the researcher visited the dental clinic three times per week to request patient participation. Some individuals chose not to participate once the purpose of the study was described to them. Many dental patients stated that they did not wish to disclose their smoking status or have their dental information recorded.

Clinical Assessment

Clinical assessments of the patients were conducted by a group of fifteen dental hygiene students. To ensure accuracy, four different trained and experienced dental hygiene clinical instructors (two dentists and two hygienists) re-measured probing depths and re-evaluated the classification of disease for each patient. A mouth mirror and standard probe was used for all examinations. During the oral examination, gingival status, oral health status, classification of gingival status and classification of periodontal status was identified. The researcher developed a gingival/periodontal assessment form based on questions from the Standard Dentrix Computer Software. The assessment form and probing depth chart are included in Appendix A7 and A8.

The periodontal examination involved the measurement of pocket depths, recorded as the distance from the free gingival margin to the base of the pocket, measured at six sites on all remaining teeth. These are the mesial, mid and distal of the buccal and lingual aspects. All teeth in each of the four quadrants were examined. Gingival recession was measured on two sites of each tooth the midbuccal and midlingual sites. Recession was measured as the distance from the cementoenamel junction (CEJ) to the height of the free gingival margin. Clinical attachment level was calculated as the sum of the probing depth and gingival recession and represents the distance from the CEJ to the

base of the pocket. The mean was computed for clinical attachment level, probing depth and recession. The surfaces (lingual/ buccal) of recession were summed for each participant. Third molars were not included in the statistical analysis involving these measurements.

The gingival status section included the visible characteristics of the gingivae such as colour, consistency, texture, marginal contour and papillary contour. Gingival status was classified as being either acute or chronic, and either generalized or localized. Further, the gingival status was classified as either mild, moderate, or severe and papillary, marginal or diffuse.

The periodontal examination included an oral health status section which consisted of a measure of the level of plaque, the level of supra-gingival calculus and sub-gingival calculus as well as a review of the general location, case type, number of teeth present, number of teeth that present with mobility and number of teeth that present with furcation involvement. Plaque was scored using the Silness and Löe Plaque Index (PII) for all teeth. Patients were classified as demonstrating light (< 10% with plaque), moderate (10-30% covered with plaque) or heavy (> 30% of the surfaces covered) plaque scores.

Mobility and furcation involvement were not given a grade; instead, each variable was calculated as a dichotomous variable indicating how many teeth were mobile and how many teeth presented with furcation involvement. Bleeding upon probing was measured as a dichotomous variable, categorized as present or not present for each of the six sites probed on each tooth. The number of bleeding sites was recorded and tabulated as a percentage ([number of bleeding sites ÷ number of sites probed] x 100). Levels of

calculus either subgingival or supragingival were determined visually. The number of sites with the presence of exudates/pus was also recorded.

Periodontal disease was classified as localized when less than 30% of the existing sites were involved or generalized when more than 30% of those sites were involved. The Case Type indicated by the hygienist was based on the standard criteria of the Workshop of Classification of Periodontal Diseases and Conditions (1999) listed as follows.

- Case Type I Gingivitis is identified by having no recession and shallow probing depths and no bone loss. There is inflammation of the gingival and bleeding upon probing.
- Case Type II Slight Periodontitis defined as having slight bone loss and 1-2 mm of clinical attachment loss (CAL).
- Case Type III Moderate Periodontitis is defined as more bone and soft tissue destruction than in Type II, 3-4 mm of CAL. There may be bone loss in the furcation area of multi-rooted teeth. Tooth mobility may be a feature.
- Case Type IV Advanced Periodontitis is a more advanced stage of Type III and having a CAL of 5 mm or more. Furcation involvement of multi-rooted teeth and tooth mobility are likely.
- Case Type V Refractory Periodontitis occurs when patients continue to show periodontal breakdown despite appropriate periodontal treatment and optimal self-care.

The hygienists also used radiographs, specifically bitewings to assess the level of bone loss and thereby to classify the periodontal status of each patient.

The questionnaire took each participant approximately 15 minutes to complete. The dental exam was a component already included in their regular appointment, and therefore not considered as part of the time commitment for this study. Each patient's questionnaire was matched with their dental record, the data was compiled and entered into an electronic database using a number system to ensure confidentially and anonymity. Questionnaire responses as well as information from the periodontal probing chart and gingival assessment form were stored in a secure electronic database at Lakehead University under the direction of Dr. William Montelpare, School of Kinesiology. Data collection occurred from October 2005 to March 2006.

3.4 Data Preparation

The three different data sources (questionnaire, gingival form and periodontal probing form) were organized and entered into an electronic database using a customized data entry program. SAS (the statistical analysis system) programs were developed for each data set.

4. DATA ANALYSIS AND RESULTS

The results of this study were based on information collected from three specific surveillance tools: i) the survey of smoking behaviour and oral hygiene; ii) the gingival and periodontal clinical assessment index; and iii) the periodontal probing depth measurement form. These data provide both discrete and continuous measures to explain the relationship between smoking behaviour and oral hygiene with periodontal disease. The results are presented in the following four sections. First, the sample of participants in the study were described. Second, responses on the survey of smoking behaviour and oral hygiene are tabulated and compared between smokers and non-smokers, across age groups and levels of periodontal disease. Third, periodontal health status was determined from data collected on the gingival and periodontal clinical assessment index and the periodontal probing depth measurement form. Finally, the relationship between periodontal health, smoking and hygiene is presented.

The first step in analyzing these data was to prepare and condition the responses collected on the questionnaires, the gingival assessment forms, and the periodontal probing chart. Once the data were organized and compiled in specific datasets, SAS programs were run to determine descriptive statistics for each data set, and measures of association between selected variables.

4.1 Subjects

Subjects were a convenience sample of 100 individuals ($n_{males} = 48$, $n_{females} = 52$) that visited the Community College Dental Clinic. An individual was only included in the study if they agreed to participate in all parts of the study. The average age of the

entire group was 40 ± 17. Females were slightly older on average than males (Average Age_{males} = 39 ± 16, Average $Age_{females}$ = 41 ± 18).

Table 1: Age Distribution by Gender

Age Group (years)	Males	Females
= 20	2%	12%
21 - 40	58%	48%
41 - 60	23%	21%
60 +	17%	19%
	N = 48	N = 52
	x = 39.3 ± 16.3	x = 40.7 ± 18.2

The minimum and maximum ages in the total sample were 19 and 80 years old, respectively. The maximum age of the male cohort was 74 and the minimum age was 19. In comparison, the female cohort had a maximum age of 80 and minimum age of 19 years.

Table 2: Confidence Interval for Age Using 95% C.I.

Cohort	Lower limit	Mean	Upper limit
entire sample	36.6	40	43
males	34.4	39	43.9
females	35.7	40.7	45.6

According to the data collected, we are 95% confident that μ is between 36.6 years of age to 43 years of age. Smokers (n = 52) were younger than non-smokers (n = 48), (Average $Age_{smokers}$ = 36.6 ± 15.1, Average $Age_{non\text{-}smokers}$ = 43.7 ± 18.77).

Table 3: Confidence Interval for Tobacco Users and Non-users by Age (95% C.I).

Cohort	Lower limit	Mean	Upper limit
Non-smokers	38.4	43.7	48.9
smokers	32.5	36.6	40.68

Overall Health Status Indicators:

The variables in the survey that were associated with the participant's overall health status score included the number of existing medical conditions, and the use of medication, daily. Ten males (4.8%) and 13 females (25%) indicated that they were currently under the care of a physician for a medical condition. On average, the participants reported 1.2 ± 0.43 medical conditions. Fourteen males (29.2%) and 29 females (55.7%) indicated that they were currently taking a drug or medication. On average, the participants reported using 2.7 ± 1.73 medications.

4.2 Oral Hygiene Measures

Listed below are the four variables used to produce a score for oral hygiene and oral health.

i) regularity of dental visits,

ii) frequency of professional dental cleaning,

iii) frequency of brushing and flossing,

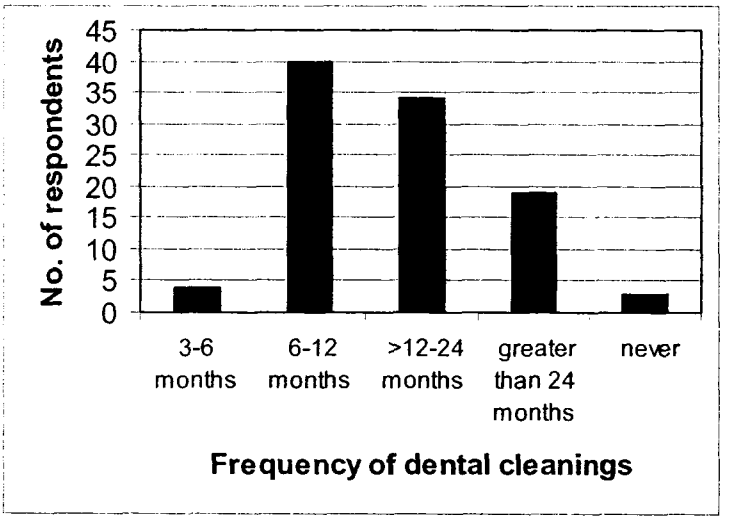
iv) and if the gingiva bleed when flossing or brushing.

The frequency distribution for dental cleanings, brushing and flossing indicate the level of oral hygiene of the sample. The question "do you visit the dentist regularly," was dichotomous, with yes and no responses. Of the 100 participants, half of the sample indicated that they regularly visited a dentist.

Figure 2 presents the frequency of dental cleanings among the participant group. A majority of the participants visited the dentist for a cleaning by a dental professional every 6-12 months (40%), some 34% of the participants visited the dental office for a

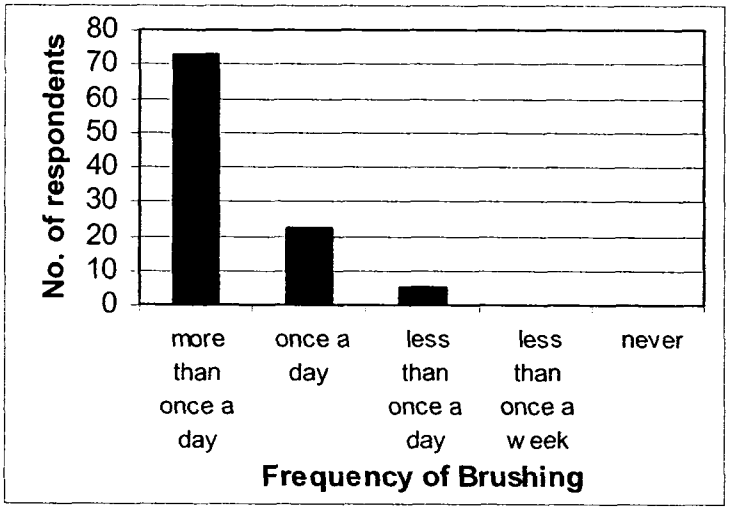
cleaning every one (greater than 12 months) to two years, and a small number of participants visited the dentist every 3-6 months (4%).

Figure 2: Frequency of Dental Cleanings



The majority of the participants (73%) indicated that they brushed their teeth more than once a day, while only a few participants (5%) indicated that they brushed their teeth less than once a day. No respondents indicated that they brushed "less than once a week" or "never". Figure 3 depicts the reported brushing frequency among participants.

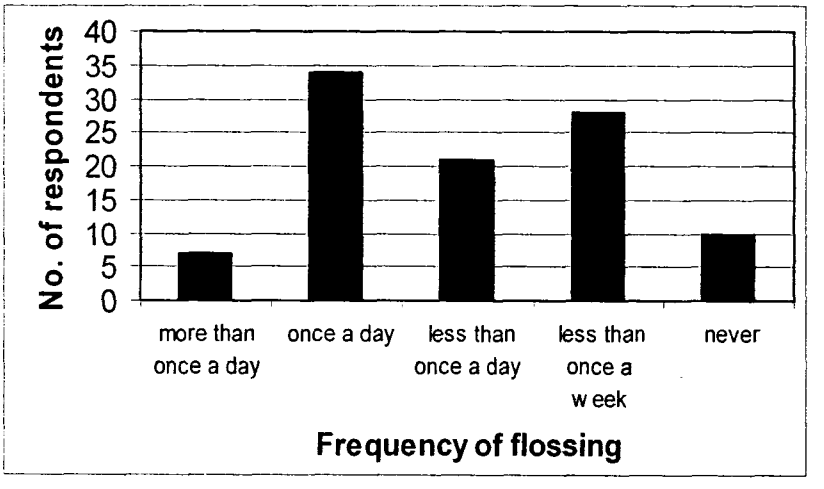
Figure 3: Frequency of Brushing



A good indicator of regular brushing and flossing is determined by whether or not an individual reports bleeding gums when they brush or floss. In this sample, 59% indicated that they had no bleeding whereas 41% indicated that they had bleeding. These self-perception scores were later validated by the actual bleeding estimates of bleeding on probing.

As displayed below (Figure 4), the majority of respondents flossed at least once a day (34%), while 21% reported flossing less than once per day, and 27% reported flossing less than once per week. It is interesting to note that as many as 10% of the individuals reported never flossing.

Figure 4: Frequency of Flossing



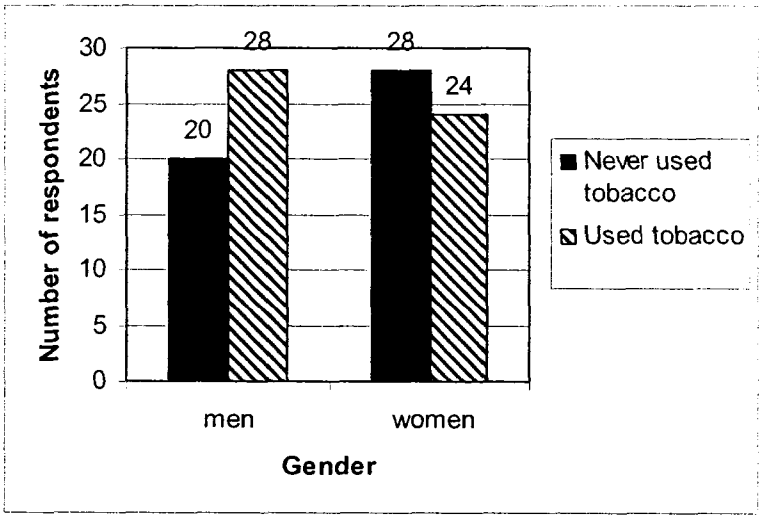
4.3 Tobacco Use

Questions on tobacco comprised a large part of the survey. Questions included use of tobacco, either currently or previous to the time the survey was administered. Questions about frequency, duration and quantity smoked were asked for cigarette smoking, pipe smoking, cigar smoking and use of chewing tobacco. Finally, with respect

to cigarette smoking, questions included a summary of type and frequency most recently smoked in the last thirty days.

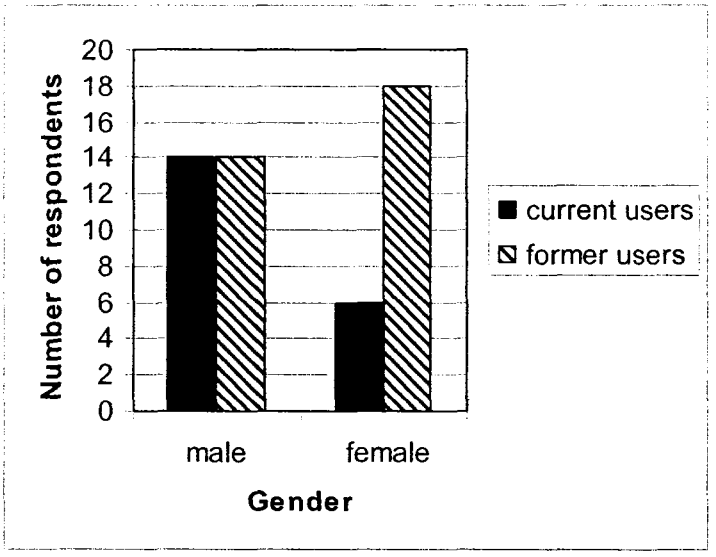
Of the total respondents, 52% indicated that they either currently or previously used tobacco products. More males than females reported using tobacco products (Figure 5).

Figure 5: Status of Tobacco Use Separated by Gender



Most respondents indicated that currently they were not smokers (only 6 females in the entire sample were current smokers). Of those who ever used tobacco (52 individuals), 20 (38%) were current tobacco users and 32 (61%) were former tobacco users (not presently using tobacco products). More former tobacco users were females (56%). There was an equal proportion of current and former tobacco users among males (Figure 6).

Figure 6: Current and Former Tobacco Users Separated by Gender



Former Tobacco Use:

The former tobacco users were asked specific questions regarding form of tobacco used, time since cessation, duration of tobacco use, and quantity used per day. The different forms of tobacco containing products were cigarettes, cigar, chewing tobacco and pipe. When grouped by gender, among the former male tobacco users, 13 indicated that they used cigarettes, 1 used chewing tobacco, 1 used a cigar and no individuals responded to using a pipe. Eighteen female former tobacco users responded to using cigarettes, with no respondents using other forms.

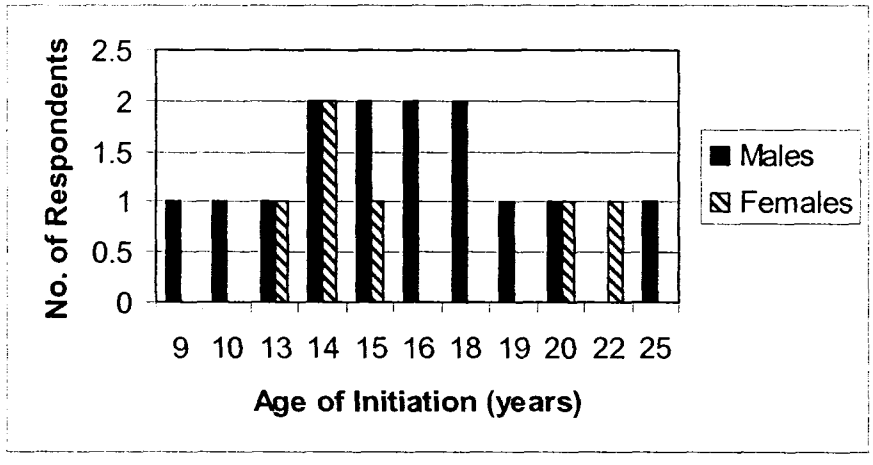
Of the former male tobacco users (N = 14), the average number of years since they quit is 12.5 ± 14.1, compared to the females (N = 18) who on average quit 10.3 ± 9.95 years ago. Former male tobacco users were users for a longer duration of time, mean of 9.14 ± 7.4 years versus female (Average = 5.8 ± 5.5). Consistent with the average values, males had a greater value for maximum duration of tobacco use; 24 years versus 20 years. The number of cigarettes, wads, pipefuls, or cigars consumed was

greater among the male cohort (Average per day = 13.8 ± 11.0) compared to females (Average per day = 9.7 ± 8.3).

Current Tobacco Use:

Of the current tobacco users, males were younger when they initiated tobacco use (Average $Years_{males}$ = 15.9 ± 4.1), compared to females (Average $Years_{females}$ = 16.3 ± 3.7) (Figure 7). The earliest age of initiation reported by males was 9 years old and 13 years old by females.

Figure 7: Age of Initiation of Tobacco Products



Cigarette Use:

Of the current smokers, males smoked a greater number of cigarettes per day and for a longer duration (in years) compared to females (Table 4 & 5).

Table 4: Number of Cigarettes Smoked Per Day

Cohort	Minimum	Mean	Maximum
males (N = 11)	1	12 ± 8.2	25
females (N = 6)	5	9.3 ± 3.2	13

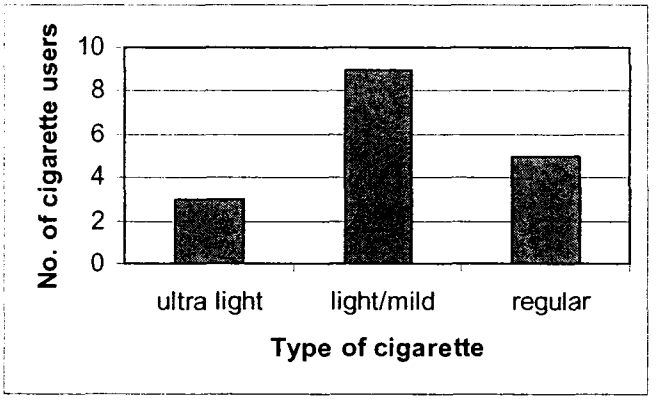
Table 5: Smoking Duration (in years)

Cohort	Minimum	Mean	Maximum
males (N = 11)	1	14.9 ± 12.3	43
females (N = 6)	2	11 ± 2.4	31

When asked the question "on how many of the past 30 days did you smoke a cigarette", there was no significant difference in the average values between genders ($Average_{males} = 27.5 \pm 7.5$; $Average_{females} = 28.5 \pm 3.2$).

The quantity smoked in the past 30 days was also questioned. Males smoked slightly more per day than females ($Average_{males} = 11.8 \pm 6$; $Average_{females} = 9.3 \pm 3.2$). This is consistent with the data obtained previously regarding lifetime tobacco use. Of the current cigarette users, 53% of the cigarette users use light/mild cigarettes, 29% use regular and 18% use ultra light (Figure 8).

Figure 8: Cigarette Type



Cigar Use:

The number of male current tobacco users who indicated that they smoke cigars was 7. The average number of cigars smoked per day was $1.3 \pm .75$ with a maximum value of 3 and minimum value of 1. Of these respondents, the mean number of years cigars were smoked was 5.7 ± 4.2 years. Compared to the responses of the males who smoked cigarettes, the quantity and duration of cigar use was significantly lower.

Chewing Tobacco Use:

Only one participant indicated that they currently used chewing tobacco and consumed 4 portions ("plugs/wads") per day for 10 years.

Pipe Use:

One individual indicated at the time of the study to occasionally smoke a pipe. On average this participant indicated that they smoke a pipe once a day and have done so for the last 10 years.

4.4 Gingival and Periodontal Assessment Outcomes

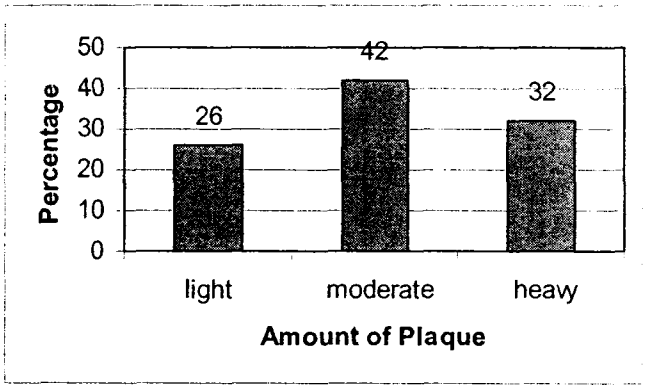
Descriptive statistics were computed for each of the measures of dental health and periodontal disease. Specifically, frequency distributions were used to compute amount of plaque, case-type, and gingival characteristic variables. The univariate procedure was used to compute mean, standard deviation and confidence intervals for continuous variables (number of teeth, bleeding, clinical attachment level and probing depths).

Oral Health Status Visual Indicators

The average number of teeth for the sample was 26.5 ± 3.5. The median number of teeth was 28, with the greatest number of 32 and lowest number of 9. The number of bleeding on probing sites is a good indicator of gingivitis and periodontal disease. The mean number of bleeding on probing was 16.6 ± 17.6 sites.

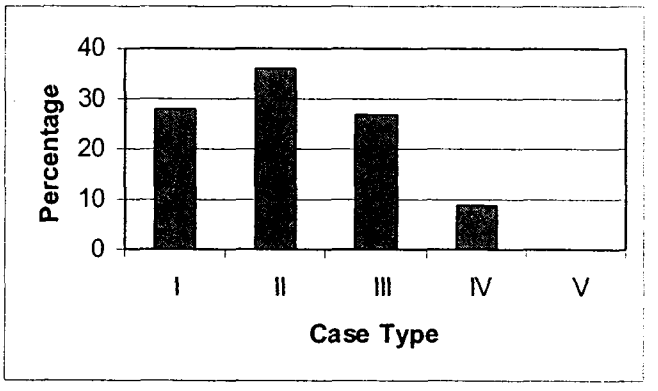
The amount of plaque for each individual was classified as light, moderate, or heavy. The majority (42%) of the participants had moderate amounts of plaque, with a small proportion of the sample (26%) having a light amount of plaque (Figure 9).

Figure 9: Plaque Scores



Each participant was categorized according to their gingival/periodontal status into one of the five different categories. The frequency distribution across the first three categories was fairly even, with 28% having gingivitis (Case I), 36% having slight periodontitis (Case II), and 27% having moderate periodontitis (Case III). A small percentage (9%) presented with advanced periodontitis, with no participants classified in the Refractory stage (Figure 10).

Figure 10: Stages of Disease



The participants were classified based on gingival status. Almost all except one presented with chronic disease versus acute. The majority (95%) of participants had a generalized form of gingival inflammation. The status of the gingival tissue was also categorized as being mild, moderate or severe. A frequency distribution of this

classification indicated that approximately fifty percent had a mild (47%) form and (50%) had a moderate form of gingival destruction.

The average periodontal probing depth of the sample was 2.42 ± 0.42 mm with a maximum value of 3.68 mm and minimum of 1.57 mm. The average clinical attachment for the sample was 4.367 ± 0.8511 mm ($CAL_{maximum}$ = 6.50 mm, $CAL_{minimum}$ = 2.55 mm). For the entire sample, age did not predict mean probing depth or CAL ($p > 0.05$).

Participants that reported they were currently taking medications were compared with CAL using the chi square goodness of fit. It showed no significant difference between the two variables ($p > 0.05$).

4.5 Relationship Between Smoking, Oral Hygiene and Oral Health Status

Measures of periodontal probing depths and clinical attachment levels were computed from the separate data collection charts and later merged with data from the smoking and oral hygiene questionnaire. For many of the subsequent analyses both current and former smokers were grouped together to allow for a sufficient number of subjects within each subgroup. The group of current and former smokers combined was given the title ever-smokers.

Prevalence

Periodontal probing depth measures were highest among current smokers, followed by former smokers and non-smokers. With regard to clinical attachment level (CAL), the non-smokers had the lowest scores, yet former smokers had a higher mean CAL than current smokers. This trend compared to the trend of mean probing depth may suggest that recession values are irreversible, even after the individual has quit smoking (Table 6).

Table 6: Probing Depth and Clinical Attachment Level according to Smoking Status

Smoking Status		Mean Probing Depth	Mean Clinical Attachment Levels (CAL)
Non-user	N = 48	2.41 ± 0.4 S.E. = 0.064 95% CI = 2.391 - 2.428	4.28 ± 0.91 S.E. = 0.131 95% CI = 4.243 - 4.317
Current user	N = 20	2.44 ± 0.45 S.E. = 0.100 95% CI = 2.396 - 2.484	4.38 ± 0.818 S.E. = 0.18 95% CI = 4.301 - 4.459
Former user	N = 32	2.43 ± 0.37 S.E. = 0.065 95% CI = 2.386 - 2.452	4.48 ± 0.78 S.E. = 0.138 95% CI = 4.432 - 4.527

S.E= Standard Error, CI= confidence interval

The results presented in Table 6 indicate that while the average CAL and probing depth values differ across groups (smokers versus non-smokers), a further analysis of the means using a one-way analysis of variance found that there was no significant difference ($p > 0.05$).

An important observation in this study was that all participants scored poorly on the CAL index, 100% > 2.5 mm (demonstrating moderate periodontal disease). A further analysis of the data, separating ever-smokers from non-smokers by clinical attachment score is presented in the Table 7.

Table 7: Smoking and Clinical Attachment Level

C.A.L (mm)	Non-smokers	Smokers	Row sum
= 3.5	N = 9 Column% = 19	N = 8 Column% = 15	17
> 3.5 = 4.5	N = 25 Column% = 52	N = 20 Column% = 38	45
> 4.5 = 5.5	N = 9 Column% = 19	N = 22 Column% = 42	31
> 5.5	N = 5 Column% = 10	N = 2 Column% = 4	7
Column Sum	48	52	

The results of the goodness of fit test applied to these data produced a chi-square observed of 7.2032 ($df = 3$; $p < 0.066$). Although this result is not significant, the difference should be further investigated as a prevalence of higher periodontal disease is seen among the ever-smokers compared to the non-smokers (Figure 10).

Figure 10: Clinical Attachment Level and Smoking Status

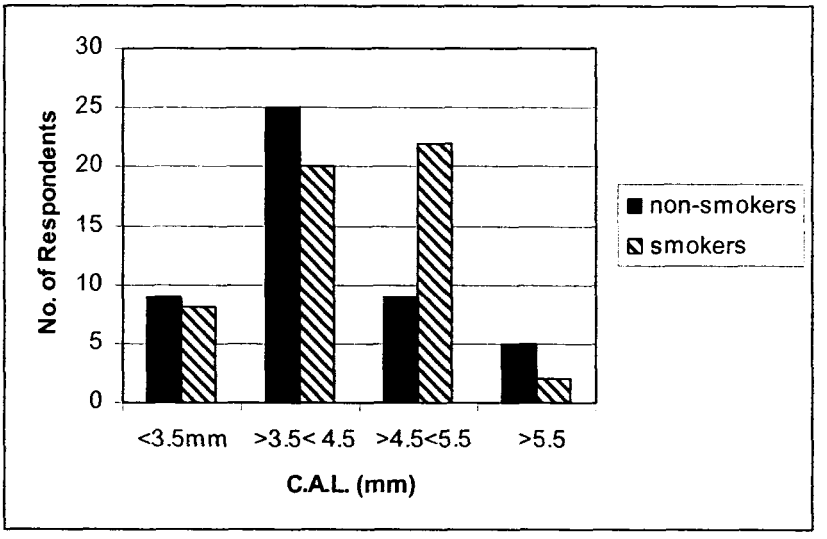


Table 8: Classification of Periodontal Disease According to Smoking

	Severe periodontal disease	Less severe periodontal disease
Smoked	24	28
Never smoked	14	34
Odds ratio = 2.08		

The results of this study provide a comprehensive overview of the relationship between smoking and periodontal disease. The results indicated that all participants reported a level of periodontal disease above normal (> 2.0 and < 6.5). However, when separating the group into severe periodontal disease and less severe periodontal disease, the computation of the odds ratio showed that the odds of developing severe periodontal disease was twice as likely for individuals who had ever smoked versus those who did not.

Ever-smokers and non-smokers were compared with case-type. When performing a goodness of fit test, the chi-square observed was 2.97 ($df = 2$; $p > 0.05$), indicating that there was no significant difference between smoking status and case-type (X^2_{obs} 2.9 < X^2_{crit} 5.99). Although the result was not significant, a higher percentage of ever-smokers had either moderate or severe periodontitis compared to non-smokers (Table 9).

Table 9: Case-Type according to Smoking status

Case-Type	Smokers	Non- Smokers
Gingivitis (Case 1)	15 (28%)	13 (27%)
Slight Periodontitis (Case 2)	16 (30.7%)	20 (42%)
Moderate-Severe Periodontitis (Case 3 and 4)	21 (42%)	15 (31%)

Quantity and Duration

Very few participants in this study identified themselves as current/former smokers. Seventeen participants reported the amount and duration they smoked. When computing a chi square goodness of fit analysis with clinical attachment level, no significant difference was observed. The number of pack years smoked for the sample was calculated by using the following formula:

Pack years = No. of cigarettes / 20 cigarettes in a pack · 365 days in a year · years smoked

For current smokers, the number of pack years does not predict mean probing depth or CAL ($p > 0.05$).

4.6 Indicators of Disease

Number of Teeth

When computing the number of teeth between ever-smokers and non-smokers, there was no significant difference (Table 10).

Table 10: Smoking Status and Number of Teeth

Smoking Status		Number of teeth
Never smoked	N = 48	26.35 ± 4.15 S.E. = 0.6 95% CI = 26.18 - 26.52
Smoked	N = 52	26.71 ± 2.8 S.E. = 0.39 95% CI = 26.60 - 26.82

Bleeding on Probing

When computing goodness of fit, there was no significant difference between bleeding on probing and smoking status (Table 11).

Table 11: Smoking Status and Bleeding on Probing

Smoking Status		Bleeding on Probing (%)
Never Smoked	N = 48	9.318 ± 12.0 S.E. = 1.7 95% CI = 8.84 - 9.79
Smoked	N = 52	11.06 ± 9.5 S.E. = 1.31 95% CI = 10.88 - 11.24

Calculus and Plaque Levels

Table 12: Amount of Supra-Gingival Calculus According to Smoking Status

Smoking Status		None to light (amounts)	Moderate to heavy (amounts)
Never Smoked	N = 48	37	11
Smoked	N = 52	31	21

As expected, a comparison of ever-smokers versus non-smokers for the supra-gingival calculus measure demonstrated an important difference ($p = 0.06$). A greater proportion

of the smokers presented with moderate to heavy amounts of supra-gingival calculus. A greater proportion of smokers were also identified as having moderate to heavy levels of sub-gingival calculus compared to non-smokers. When computing an odds ratio using a 2 x 2 design, the results showed that ever-smokers were 2.38 times more likely to demonstrate moderate to heavy amounts of calculus for both sub- and supra- types compared to those who do not smoke.

Table 13: Amount of Calculus (sub- and supra-) According to Smoking Status

	Calculus (none –light)	Calculus (Moderate - Heavy)
Smoked	25	15
Never smoked	31	8
Odds ratio = 2.38		

The amount of plaque recorded by the dental hygienist is a good indicator of oral hygiene practices. Tobacco users had a higher amount of plaque (moderate to heavy) than non-tobacco users (Table 14). The goodness of fit test showed that the chi-square observed value of 4.25 was greater than the chi-square critical value of 3.84 ($p < 0.05$; $df = 1$). Thus, the null hypothesis is rejected and the two distributions are not equal, indicating the association between tobacco use and plaque scores to be significant. The odds ratio computation indicated that ever-smokers are 2.62 times more likely to have heavier plaque indices than non-smokers.

Table 14: Amount of Plaque According to Smoking Status

	Plaque (light)	Plaque (Moderate - Heavy)
Smoked	9	43
Never smoked	17	31
Odds ratio = 2.62		

Oral Hygiene Behaviours

Visits to Dentist:

Smokers in this study were less likely to visit the dentist on a regular basis. Six non-smokers compared to 16 smokers waited 24 months or longer before visiting the dentist for a routine dental cleaning.

Brushing and Flossing:

There was no significant difference between brushing and/or flossing behaviours among the smoking and non-smoking groups.

4.7 Validity of Clinical Measurements

Clinical attachment level (CAL) and "case-type" showed a significant association based on simple linear regression as presented in Table 15 below ($p < 0.0001$). This test of validity is consistent with the expectation that the clinical team that evaluated each patient was accurate and reliable in their CAL assessment and categorized each patient by the appropriate case-type.

Table 15: Simple Linear Regression Model of Probing Depth and Case-Type

Analysis of Variance					
Source	**DF**	**Sum of Squares**	**Mean Square**	**F Value**	**Significance**
Model	1	5.61822	5.61822	46.71	<.0001
Error	98	11.78715	0.12028		
Corrected Total	99	17.40537			
Model Summary					
Root MSE	**Dependent Mean**	**Coefficient of Variance**	**R- Square**	**Adjusted R-Square**	
0.34681	2.42331	14.31140	0.3228	0.3159	
Parameter Estimates					
Variable	**DF**	**Parameter Estimate**	**Standard Error**	**t-value**	**Significance**
Intercept	1	1.87536	0.08735	21.47	<.0001
Casetype	1	0.25251	0.03695	6.83	<.0001

5. DISCUSSION AND CONCLUSIONS

Tobacco use is a causal mechanism of periodontal disease. The results of this study support previous research by Axelsson et al., 1998, Bergstrom & Preber., 1994, Bergstrom et al., 2000a, & Bergstrom, 2003 which demonstrated that smokers are at greater risk of developing severe periodontal disease than non-smokers. Odds ratios reported in the present study indicated that not only were tobacco users twice as likely to show severe periodontal disease but the present study also demonstrated that tobacco users were more than twice as likely to demonstrate higher levels of plaque and both supra- and sub-gingival calculus, which are noted precursors of gingival inflammation leading to periodontal disease. While this finding is important alone, the study also showed that self-reported oral hygiene behaviours were not significantly different between ever-smokers and non-smokers, suggesting that despite the best intentions of smokers to self maintain good oral health through subscribing to regular dental visits and practices, periodontal disease and its related sequelae continue to develop.

5.1 Oral Hygiene Behaviours and Tobacco Use Questionnaire

Reported oral hygiene behaviours, specifically, the frequency of visiting a dental professional for cleaning was greater than that which would be expected from a random sample of individuals drawn from a community. Similarly, the frequency of brushing more than once a day was reported to be higher than expected. Conversely, the frequency of flossing was inconsistent across the sample, with the flossing options being equally distributed across respondents. Based on the self-reported responses, one might be led to believe that this cohort had good oral hygiene behaviours, and were therefore minimally at risk with regard to their access or awareness of dental care. However, as

with most dental patients, it should be noted that the technique of brushing and flossing might be incorrect and more important, that the frequency of preventive dental behaviours was over reported among participants in this sample. Certainly, the results of probing depth and clinical attachment measurements support the notion that while participants believed that their behaviours were adequate and appropriate, the specific measures demonstrate that these individuals were all at moderate risk for dental disease.

One challenge that emerged in this study was that although half of the participants reported ever using a tobacco product, even fewer indicated that they were current smokers. Further, although the study demonstrated the residual effects of smoking on periodontal disease, the relatively small number of current smokers may have had a direct influence on several comparisons. These findings suggest that either individuals who visited the clinic selected for this study were not reporting previous smoking history accurately, or that participants who volunteered from the selected clinic were more likely not to smoke.

Within the sample of smokers, proportional differences among the former and current smoking groups with respect to gender were noted. However, regardless of gender, the majority of smokers reported using cigarettes as a form of tobacco versus other products such as cigars, chew or pipe. Gender differences observed in the present study, comparing the number of males versus females that continue to smoke and the duration of tobacco use and consumption volume, each showed that males were higher in both categories. These findings are consistent with the research on gender differences in smoking. However, trends in smoking among women are increasing with a decline in smoking prevalence among men.

5.2 Clinical Oral Health Measures

As noted previously, although the sample reported high frequency of brushing, these individuals showed higher amounts of plaque than should be expected given their self-reports. Likewise, although few participants showed advanced periodontitis, there was higher than expected distribution of gingivitis, slight periodontitis, and moderate periodontitis in this group.

Clinical attachment, which is a measure of the combination of probing depth and gingival recession, is a standard measure of progressive periodontitis. In the present sample, the average clinical attachment level of slightly more than 4 mm suggests a higher than expected observation, especially in a random sample of individuals visiting an urban community dental clinic. Moreover, the reported clinical attachment score in the present study suggests that more individuals are experiencing severe periodontal disease than that reported in other studies. For example, two relevant Canadian research studies by Locker and Leake (1993) and Sbaraglia et al. (2002), reported 2.95 mm and 3.89 mm clinical attachment levels, respectively, when considering the entire sample.

In the present study, clinical attachment loss was not separated by age, however, other researchers reported that older individuals present with a higher mean attachment loss (Sbaraglia et al., 2002), even though Beck and coworkers did not find a significantly higher attachment loss when studying an isolated group of seniors (Beck, Kock, Roxier & Tudor, 1990).

The average attachment loss observed for participants in the present study was greater than that reported for similar communities studied. The subjects in the present study ranged from 19 to 80 years old with an average age of 40. When comparing the

participants across age strata to participants in other studies, the difference in mean clinical attachment levels is notable. Another study of similar age distribution reported lower average CAL's (Bagramian et al., 1994). In the present study, all participants scored poorly on the CAL index – showing an average CAL of greater than 2.5 mm. The important note of these findings is that the periodontal health of participants in the present study was poorer than other populations. It is not known as to why this sample showed a relatively poor level of oral health. One assumption of why the sample showed a relatively poor level of oral health may be a result of the type of clinic used to retrieve data. The fee for dental work at the community college dental clinic is lower than that of a private practice dental office which may have targeted lower S.E.S individuals. However, this is only a speculation as the S.E.S of the sample was not identified.

In an attempt to ensure the most comprehensive estimates of the clinical variables, the present study used techniques of internal validation for the main outcome measures of periodontal pocketing, clinical attachment level and diagnosis. Full mouth examinations were used to obtain data for each variable, thereby obtaining a large number of measurements for each participant. In order to compute periodontal pocketing and clinical attachment level, six sites on each tooth were examined. The selected approach increased the number of items used to compute the entire estimate of probing depth and clinical attachment level. In turn, the sensitivity of the measure was increased. In conjunction with evaluating the number of retained teeth, additional clinical and radiographic measures were used to classify the periodontal condition. In addition, the study demonstrated that by relating two variables, clinical attachment level and case-type, one could show the consistency between observations to evaluate the categorization of

variables by the clinical evaluation team. This significant association thus validates the assessment and categorical precision of the clinicians.

5.3 Relationship: Oral Hygiene, Smoking Behaviour and Periodontal Disease

In bivariate analyses, many variables were associated with periodontal attachment loss. However, some variables were not significant under the ANOVA and/or Chi-Square tests. A dose-response relationship was not found when "pack-years" and mean probing depths were compared. The interpretation of this finding along with others needs to be treated with caution as the absence of the significant association may reflect the fact that the number of individuals who reported to be current smokers was too small for the relationship to be assessed.

Consistent with Van der Weijden et al. study (2001), no differences in bleeding tendency were detected between ever-smokers and non-smokers in the present study. In regards to gingivitis and bleeding on probing values measured in previous studies, most report smokers having less bleeding on probing than non-smokers. Dietrich (2004) found that smokers had less gingival bleeding than non-smokers. Decreased gingival bleeding in smokers has been explained by the vasoconstrictive effects of nicotine on peripheral blood vessels. Conversely, one study reported smokers to have an increased bleeding tendency (Haber et al., 1993).

Socio-economic status has also been reported to be related to smoking habits (Bergstrom, 2000a). The socio-economic status of the sample was not identified, thus, a homogenous population may have not been obtained. Since socio-economic status was not controlled for in this study, other socio-economically related factors may have exerted a confounding influence on the outcome variables and thus, influenced the

results. A lower socio-economic cohort of the population with perhaps a greater amount of smokers and inferior standards of dental care and oral hygiene could have presented a greater degree of periodontal disease than that obtained in this study. Obtaining participants from a dental setting may have been a limitation since these individuals are dentally aware patients.

There was no significant difference in the clinical attachment levels of those taking medications or those not taking medications. Certain medications may have had an indirect effect on oral health. However, no effects were observed for the present study.

Even though there is some controversy over the role of bacteria and tobacco as a regulator of periodontal disease, there is also evidence to suggest that the use of oral hygiene regimens to control plaque can prevent or slow down the process of periodontal disease. Since periodontal disease is associated with tobacco use, cigarette smokers are more likely to develop periodontal disease as a result of their poor technique or lack of oral hygiene habits and their tobacco use. Although not significantly different, ever-smokers were less likely to visit the dentist as frequently as non-smokers. The reported frequencies of brushing and flossing were similar between ever-smokers and non-smokers. However, ever-smokers had higher amounts of plaque than non-smokers. Ever-smokers were more than twice as likely to have heavier plaque scores than non-smokers. As well, ever-smokers had odd ratios of more than twice that of non-smokers for subgingival and supragingival calculus compared to non-smokers. This is reflective of their oral hygiene practices being incorrect or an indication that the smokers may have over-reported their frequency of brushing and flossing. In comparison, Andrews et al. (1998) reported that non-tobacco users practiced better self-reported oral hygiene.

Overall, severe periodontal disease, as defined in this study as having an average clinical attachment level of 4.5 mm or greater, was higher in current smokers versus former and non-smokers. The non-smokers had the lowest CAL score; whereas former smokers had a higher mean CAL than current smokers suggesting that smoking may be so destructive that its effects are irreversible. However, the trend in periodontal probing depths was that former smokers were higher than non-smokers but lower than current smokers. From this, the benefits of smoking cessation may be identified. This study did not focus on how long after cessation benefits can be observed, however, the benefits of cessation outweigh the harmful effects of smoking. Longitudinal studies would be beneficial in determining periodontal changes over time following smoking cessation.

Ever-smokers had a higher prevalence of severe periodontal disease versus non-smokers. The odds ratio of 2.08 for developing severe periodontal disease (CAL of > 4.5 mm), is consistent with Papapanou (1996) who found an odds ratio of 2.82 (95% CI, 2.36-3.39).

Generally, assessment of risk in other studies showed that smoking is associated with between a 2 and 7 fold increase and even up to 11.8 fold increase in risk for having periodontal tissue loss compared to non-smokers (Axelsson et al., 1998, Bergstrom & Preber., 1994, Bergstrom et al., 2000a, Bergstrom., 2003, Haber., 1993 & Torrungruang et al., 2005). The broad range risk estimate may be due to the type of study and most importantly, the different case definitions of periodontal disease. A narrower definition could result in higher risk estimates and vice versa (Bergstrom, 2003). From this study, one can conclude that individuals are twice as likely to get severe periodontal disease if

they smoke versus those who do not. Therefore, consistent with other studies, cigarette smoking should be considered a major risk factor for severe periodontal disease.

5.4 Study Limitations and Strengths

The specific limitations of this study were related to issues of design, sample size, and reliability of self-reported data. The cross-sectional design may be considered a limitation because it is collecting data at a single episode in time, but by using clinical attachment level as a main outcome variable, the researcher was able to demonstrate cumulative periodontal destruction over time (Loe, Anerud, Boysen & Smith, 1978, cited in Torrungruang et al., 2005). Clinical attachment is a good indicator of periodontal disease, as it demonstrates the progressive pathology related to the reduction of bone loss and increase in gingival recession.

The reliability of self-reported data is also a limitation of the study, as an external validator was not used in the study to confirm the actual exposures to nicotine or tobacco related products. Thus, the reports of nicotine exposure may be suspect for some individuals who may have been less accurate in their estimates of tobacco product exposure. The term consumption describes the amount of use of tobacco products at the time of the study and does not take into account changes over time or possible intervals when the individual was not smoking.

The sample size of 100 respondents was a reasonable size, although the number of participants that reported tobacco use was fewer than expected from a random sample of community participants. This smaller sample of smokers and the wide range of variability among these individuals limited a more comprehensive analysis of the effects of smoking on the periodontium, such as the researcher's ability to estimate a tobacco

dose-response relationship. The small sample of current smokers as well as the reported high frequency of dental cleanings by the entire sample could have been due to the location of participant recruitment. Participants recruited from a non-dental facility may have a lower standard of dental awareness.

An additional limitation is inter-rater reliability for the collection of the clinical measurements; more than one individual was taking measurements. Probing force ranges between clinical examiners (Daniel & Harfst, 2002).

Other possible limitations are related to the development of the questionnaire and include the number of items used and the types of questions. The tobacco use questions as well as the oral hygiene behaviour questions were taken from published surveys but the items from the various instruments were not previously combined. The questionnaire was not pretested which could have resulted in participant uncertainty. The validity and reliability of the questionnaire should be investigated further.

The research was intended to be both an epidemiological survey of dental health as well as an exploration of the association between lifestyle behaviours, most notably smoking and dental health outcomes in a sample of individuals from Northwestern Ontario. To date, few Canadian studies have been published on this topic, and no studies have been reported at the local level pertaining to the effects of tobacco on the periodontium. The current study is useful in that the findings reported here support the suggestion that a relationship exists between smoking and poor dental health. Yet most important, these findings suggest that the health consequences demonstrated by smokers cannot be masked or reversed simply by dental hygiene practices.

The questionnaire contained predominately valid and reliable items from previous published instruments. The quantitative and qualitative clinical measurements complemented the questionnaire to form a good basis for analysis. An oral health profile of adults in Northwestern Ontario, Canada that did not previously exist was created and the study used the appropriate clinical measurements to evaluate periodontal status of each individual.

5.5 Conclusions

The main objectives of the present study were to explore the relationship between, oral health, oral hygiene practices and tobacco use in a sample of residents of Northwestern Ontario. Information about the periodontal status, oral hygiene behaviours and tobacco use of adults recruited from a centrally located dental clinic was obtained. Clinical attachment level was the main outcome variable used for the classification of periodontal status.

The study sample was equally distributed across varying age groups and gender. In addition, the study had a low proportion of individuals who reported to be currently using tobacco. The sample reported having a high standard of dental care with some form of dental disease, ranging from gingivitis to severe periodontitits. This sample of Northwestern Ontario residents are an important group to study as information from this geographic area is lacking in the literature. Nevertheless, the association between tobacco users in this area and their overall dental health has been identified, even among a small sample of self-identified smokers.

The strength of the association of the precursors and risk factors of periodontal disease is an important finding. Since both the ever-smoking and non-smoking groups

had similar reported oral hygiene habits, with the exception of dental visits, an important concept is established. Although smokers may have reported similar oral hygiene practices, this group was more than twice as likely to have higher plaque scores, subgingival and supragingival calculus levels and most importantly twice as likely to develop more severe forms of periodontal disease versus non-smokers. The sequence of events that cause periodontal destruction is initiated by plaque which mineralizes into calculus and primarily causes gingival inflammation. This study confirmed that smoking is a risk factor for severe periodontal disease.

The study's instruments can now be used to initiate ongoing data collection of adults within the area. It is anticipated that this study may be of assistance to dental professionals, researchers and epidemiologists in learning more about the tobacco-periodontal association as well as contributing to the current surveillance of the oral health profile of individuals in the area. The relevance of the findings of this study for the effects of tobacco on the oral health of Canadians and the high prevalence of periodontal disease in the sample are important for tobacco cessation and prevention and proper oral hygiene behaviour. The implications of the association between periodontal disease and smoking are that smoking cessation efforts should be considered when treating periodontitis. Furthermore, the intervention of counselling to prevent periodontal diseases should be integrated into community education.

5.6 Recommendations

1. Further confirmation of the relationship between periodontal disease and tobacco use is required using different design strategies. These may include longitudinal and prospective designs.
2. Recruit participants from a non-biased sample from the general public rather than a dental setting. Those from the general public may be more likely to be non-subscribers to dental health and could exhibit a greater degree of periodontal disease and contain a larger sample of tobacco users.
3. Validate tobacco levels to eliminate the unreliability of self-reported smoking habits.

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APPENDIX A

RESEARCH INSTRUMENTS AND CONSENT FORMS

1. Cover Letter
2. Participant Consent Form
3. Participant Questionnaire
4. Letter to Dental Clinic
5. Coordinator/Dentist Consent Form
6. Script for Researcher
7. Gingival/Periodontal Disease Assessment Tool
8. Probing Depth Computerized Assessment Form

A2. PARTICIPANT CONSENT FORM

My signature on this form indicates that I, _____, consent to participate in a study by Ann Marie Chlebovec entitled "Tobacco use as a risk indicator for periodontal disease in a sample of Northwestern Ontario residents."

I have read and understand the cover letter, which explains the nature of the study and my role as a volunteer in the study.
I understand the following:

1. My participation in the study is purely voluntary and I can withdraw from the study at any time, with no obligations.
2. I will be expected to complete a questionnaire.
3. I am allowing the researcher to access dental information provided by the dentist (ie. Periodontal measurements).
4. The data I will provide will remain confidential; any information that is gathered about me during this study will not to be disclosed, and if the results are published, I will not be identified in any way
5. I have been provided with a contact number and an e-mail address should I have any questions or concerns regarding the study.
6. I will receive a summary of the study, upon request, following the completion of the project.

Signature of Participant Date

Initial of Witness Date

A3. PARTICIPANT QUESTIONNAIRE

SECTION A: Participant Information:

- Do you have natural teeth? YES or NO (please circle)
    - If you **do not** have any natural teeth your participation is not required. Thank You.

- Your Name (please print):_____
- Date of Birth YY/MM/DD_____/_____/_____
- Sex ____Male ____Female
- Are you under the care of a physician for a medical condition? YES or NO.
  If yes, explain_____
  _____
- Are you currently taking any drugs or medications? YES or NO. If yes, please list_____
  _____
  _____
  _____

- Have you ever undergone periodontal surgery or extensive surgery involving your gum tissue? _____YES _____NO

- Do you see a periodontist to receive special cleanings/ treatment for your gums? ____YES _____NO

- Is your alcohol intake none_____, light____, moderate_____, heavy_____.

Please make a check mark on the line that best describes your practices.

SECTION B: The following questions are about oral hygiene practices

#	QUESTIONS	RESPONSES
1	Do you visit the dentist regularly (within a 12 month period)?	____ Yes ____ No
2	How often do you have your teeth cleaned by a dental professional?	____ every 3 to 6 months ____ every 6 to 12 months ____12-24 months ____ greater than 24 months, specify: _____years ago ____I have never had my teeth cleaned by a dental professional
3	Do you use anything other than a toothbrush and dental floss to care for your teeth and gums?	____Yes ____No
4	How often do you brush your teeth?	____more than once a day ____once a day ____less than once a day ____less than once a week ____never
5	Do your gums bleed when you brush or floss?	____Yes ____No
6	How often do you floss your teeth?	____more than once a day ____once a day ____less than once a day ____less than once a week ____never

SECTION C: The following questions are going to ask you about tobacco use

#	QUESTIONS	RESPONSES
7	Are you aware of the effects of tobacco on your mouth?	____Yes ____No
8	Have you been exposed to second-hand smoke in your household as you were growing up?	____Yes ____No
9	Are you or have you ever used tobacco products: cigarettes, cigar, chewing tobacco, etc?	____Yes ____No (go to end of questionnaire)
10	Have you ever been counseled about tobacco cessation?	____Yes ____No
11	Are you a current or former tobacco user? (any tobacco product: cigarettes, cigars, pipes, chewing tobacco, etc)	___ CURRENT tobacco user (go to question 16) ___ FORMER tobacco user (go to the next question)
12	As a former tobacco user, how long ago did you quit?	___ ___years OR ___ ___ months ago
13	How long were you a tobacco user for?	____ ___years OR ____ ____months
14	What form of tobacco did you use?	_____cigarettes _____cigar _____chewing tobacco _____pipe
15	How much did you use this form of tobacco?	_____ Number of cigarettes /wads /pipefuls /cigars per day?

FORMER TOBACCO USERS GO TO END OF QUESTIONNAIRE

16	At what age did you begin to smoke or use smokeless tobacco products?	____ ____years old
17	Do you currently smoke **cigarettes?**	____Everyday ____Occasionally ____I use other types of tobacco (go to question 24)
18	On average, how many cigarettes do you now smoke per day? 1 pack equals 25 cigarettes If less than 1 per day, enter 1 If 95 or more per day, enter 95	__ __ __Number of cigarettes (per day)
19	For about how many years have you smoked this amount? If less than 1 year, enter 1	__ __ enter number of years
20	Is the cigarette product filtered or non-filtered?	___filtered ___non-filtered
21	What type of cigarette do you use?	____ultra light ____light/ mild ____regular
22	On how many of the past 30 days did you smoke a cigarette?	__ __ enter number of days
23	During the past 30 day, on the days that you did smoke, about how many did you smoke per day?	__ __ __ enter number of cigarettes (per day)
24	Do you smoke **cigars**?	____everyday ____occasionally ____ never, I use other forms of tobacco (go to question 27)
25	How many cigars do you smoke per day? If less than 1 per day, enter 1	__ __ ___ enter number of cigars

26	For about how many years have you smoked this amount? If less than 1 year, enter 1	__ __ enter number of years
27	Do you use **chewing tobacco (snuff)?**	____everyday ____occasionally ____I am a pipe user (go to question 30) ____I do not use any other forms of tobacco (go to end of questionnaire)
28	How many "plugs", "wads", or "chaws" of chewing tobacco do you use per day? If less than 1 per day, enter 1	__ ___ enter number of plugs, wads or chaws
29	For how many years have you used this amount? If less than 1 year, enter 1	___ ___ years
30	How often do you smoke a **pipe**?	____everyday ____occasionally ____ never, I do not use a pipe (go to end of questionnaire)
31	How many pipefuls of tobacco do you smoke per day? If less than 1 enter 1.	___ ___ number of pipefuls
32	For how many years have you smoked that amount?	___ ___ years

END of QUESTIONNAIRE
THANK YOU FOR YOUR PARTICIPATION

A5. CLINIC COORDINATOR/DENTIST CONSENT FORM

I agree to participate in a study conducted by Ann Marie Chlebovec, a graduate student of Lakehead University.

I have read and understand the cover letter, which explains the purpose of the study and my role in the study.

I understand the following:

1. My participation in the study is purely voluntary and I can withdraw from the study at any time, with no obligations.
2. The researcher will ask adult patients to complete a questionnaire as well as collect them upon completion.
3. I am allowing the researcher to access dental information from the dental chart for each patient participant on a weekly basis. This includes entering dental information into a computer program.
4. The data that the patient participants will provide will remain confidential; any information that is gathered during this study will not to be disclosed, and if the results are published, the patient participants will not be identified in any way.
5. I have been provided with a contact number and an e-mail address should I have any questions or concerns regarding the study.
6. I will receive a summary of the study, upon request, following the completion of the project.

Signature of Dental Hygiene Coordinator Date

Signature of Dentist(s) Date

Signature of Witness Date

A6. SCRIPT FOR RESEARCHER

Hello, we are involved in a research study about dental health. Would you be willing to complete a short questionnaire while you are waiting for your appointment?

A7. GINGIVAL/ PERIODONTAL DISEASE ASSESSMENT TOOL

Patient Name: _____

Gingival Status				
Colour: ? Pink ? Red ? Reddish Blue ? Blue	**Consistency:** ? Spongy ? Edematous ? Retractable ? Hyperplastic	**Texture:** ? Heavy stippling ? Smooth & Shiny ? Sloughing	**Contour Marginal:** ? Rolled ? Rounded ? Recessed ? Cleft	**Contour Papillary:** ? Bulbous ? Blunted ? Cratered
Oral Health Status				
Plaque: ? Light ? Moderate ? Heavy	**Supra Calculus:** ? None ? Light ? Moderate ? Heavy ? Local ? General	**Sub Calculus:** ? None ? Light ? Moderate ? Heavy ? Local ? General	**Case Type:** ? I Gingivitis ? II Slight Perio ? III Moderate Perio ? IV Advanced Perio ? Refractory	**Number of Teeth present** _____
Tooth Mobility: Number of teeth mobile: _____	**Furcation Involvement:** Number of teeth affected: _____			
Classification of Gingival Status				
? Acute ? Chronic ? Generalized ? Localized	? Mild ? Moderate ? Severe ? Papillary ? Marginal ? Diffuse	**Gingival recession:** Number of teeth involved:_____ # of teeth in each range: ___4 mm ___5-7 mm ___8+ mm	**Bleeding on Probing:** Number of bleeding on probing sites: _____	**Suppuration (pus):** ? Yes ____sites ? No
Classification of Periodontal Status				
? Generalized ? Localized	? Slight ? Moderate ? Advanced ? Refractory	**Attachment Loss** ? Local ? General		

A8. PROBING DEPTH COMPUTERIZED ASSESSMENT FORM

Probing Depths (mm)

Px_name: id_Num:

Pocket Chart 1

11_F:		**R_F =**		11_L:		**R_L =**	
12_F:		**R_F =**		12_L:		**R_L =**	
13_F:		**R_F =**		13_L:		**R_L =**	
14_F:		**R_F =**		14_L:		**R_L =**	
15_F:		**R_F =**		15_L:		**R_L =**	
16_F:		**R_F =**		16_L:		**R_L =**	
17_F:		**R_F =**		17_L:		**R_L =**	
18_F:		**R_F =**		18_L:		**R_L =**	

Pocket Chart 2

21_F:		**R_F =**		21_L:		**R_L =**	
22_F:		**R_F =**		22_L:		**R_L =**	
23_F:		**R_F =**		23_L:		**R_L =**	
24_F:		**R_F =**		24_L:		**R_L =**	
25_F:		**R_F =**		25_L:		**R_L =**	
26_F:		**R_F =**		26_L:		**R_L =**	
27_F:		**R_F =**		27_L:		**R_L =**	
28_F:		**R_F =**		28_L:		**R_L =**	

Pocket Chart 3

31_F:		R_F =		31_L:		R_L =	
32_F:		R_F =		32_L:		R_L =	
33_F:		R_F =		33_L:		R_L =	
34_F:		R_F =		34_L:		R_L =	
35_F:		R_F =		35_L:		R_L =	
36_F:		R_F =		36_L:		R_L =	
37_F:		R_F =		37_L:		R_L =	
38_F:		R_F =		38_L:		R_L =	

Pocket Chart 4

41_F:		R_F =		41_L:		R_L =	
42_F:		R_F =		42_L:		R_L =	
43_F:		R_F =		43_L:		R_L =	
44_F:		R_F =		44_L:		R_L =	
45_F:		R_F =		45_L:		R_L =	
46_F:		R_F =		46_L:		R_L =	
47_F:		R_F =		47_L:		R_L =	
48_F:		R_F =		48_L:		R_L =	

Enter source of data (college, dental office)

Click here to write the data from this form to the drive Click ME to submit data to file

... or click here to clear the form Clear

APPENDIX B

SAMPLE INFORMATION

1. Sample Size Calculations
2. Medications Reported and Their Oral Effects

B1. SAMPLE SIZE CALCULATIONS

Sample size calculations for the adult population (over age 20) in Thunder Bay, Ontario. According to Statistics Canada, Census of Canada 2001, the population in Thunder Bay was 109,016 as of 2001. The population 20 years of age and older is 82,110 as of 2001.

Percent Error	Confidence Interval		
	90% (Z_a=1.64)	95% (Z_a=1.96)	99% (Z_a=2.58)
2%	1492	2114	3596
5%	242	346	597
10%	61	87	150

The number of males 39280 (~48%) and females 42835 (~52.3%) in the 20 and over range (Statistics Canada, 2002). The mean age of males is 38.2 and females is 40.1 for all age ranges. The expected case rate of periodontal disease (defined as the presence of attachment loss of 3mm or more together with a presence of a probing depth of = 3mm at the same sites) is 34.5% (used for this calculation). The expected case rate of periodontal attachment loss of 3mm or more is 53%.

* The following figures were computed with the use of a webulator designed by Dr. Montelpare found at http://giant.lakeheadu.ca/~wmontelp/index.html.

B2. MEDICATIONS REPORTED AND THEIR ORAL EFFECTS

Generic name	Brand name(s)	Oral side effects	No. of subjects using
Warfarin sodium	Coumadin	Gingival bleeding, stomatitis, salivary gland pain/swelling	2
Estrogens and Progestins combination	Oral Contraceptives	Gingival bleeding, dry socket	9
Telmisartan	Micardis	None	1
Norfloxacin	Noroxin	Dry mouth, stomatitis	1
Irbessartan	Avapro	None	2
Atenolol	Tenormin	Dry mouth	3
Hydrochlorothiazide (HTCZ)	Apo-hydro, Diuchlor H, Neo-Codema, Novo-Hydrazide, Urozide	Dry mouth, increased thirst, lichenoid reaction	4
atorvastatin calcium	Lipitor	Angioneurotic edema, lichenoid reaction	3
paroxetine HCl	Paxil, Paxil CR	Dry mouth, glossistis, aphthous stomatitis	1
omeprazole	Losec	Dry mouth, mucosal atrophy of tongue, taste perversion, candidiasis	2
Vitamin D	Vitamin D	Metallic taste, dry mouth can be early sign of toxicity	1
lansoprazole	Prevacid	Candidiasis, stomatitis, halitosis, dry mouth, taste alteration	1
desipramine HCl	Norpramin	Dry mouth, unpleasant taste, bleeding, stomatitis	1
rampiril	Altace	Angioedema (lips, tongue, mucous membranes), dry mouth	8
metaformin HCl	Novo-Metaformin, Apo-Metformin, Gen-Metformin, Glycon, Nu-Metformin	Unpleasant taste, metallic taste	1
Ranitidine	Novo-Ranitidine	None	1
Tetracycline/tetracycline HCL	Apo-Tetra, Novotetra, Nu-Tetra	Tooth discoloration in children < 8yr, candidiasis, tongue discoloration and hypertrophy of papilla, enamel hypoplasia, bleeding (long-term use), stomatitis, erythema, multiforme	1

Cefixime	suprax	Candidiasis, glossitis	1
Lisinopril	Zestril, Prinivil	Drug mouth, angioedema	1
Atorvastatin calcium	Lipitor	Angioneurotic edema, lichenoid reaction	3
Aspirin (acetylsalicylic acid)	Entrophen, Novasen, ASA, Arthrisin	Increased bleeding (chronic, high doses)	4
Albuterol, albuterol sulfate	Ventolin, Gen-Sal butamol, Proventil	Dry mouth, teeth discoloration, taste changes	2
Diltiazem	Apo-Diltaz, Diacor XR, Cardizem	None	
Simvastatin	Zocor	None	3
glyburide	DiaBeta, Apo-Glyburide, Euglucon, Gen-Glybe	Pts. on chronic therapy may have symptoms of bleeding and poor healing.	2
Venlafaxine HCL	Effexor, Effexor XR	Dry mouth, glossitis (rare), cheilitis, gingivitis, candidiasis	2
Bupropion hydrochloride	Wellbutrin, Wellbutrin SR, Zyban	Dry mouth, taste alteration	1
Rosiglitazone maleate	Avandia	None	1
sotalol	Sotacor, Betapace	None	1
Sumatriptan succinate	Imitrex	Discomfort in jaw/ mouth/tongue	1
Levothyroxine sodium	Eltroxin, Levothroid	None	1
primidone	APO-Primidone, Mysoline	None	1
valsartan	Diovan	Taste alterations	1
indapamide	Lozol, Apo-indapadmide	Dry mouth	1
Allopurinol/allopurinol sodium	Zyloprim, Purinol, Apo-Allopurinol	Metallic taste, stomatitis, lichenoid drug reaction, salivary gland swelling	1
Amlodipine besylate	Norvasc	Dry mouth, altered taste, gingival overgrowth has been reported with other channel blockers	1
Metformin HCL	Glucophage, Novo-Metformin, Apo-Metformin, Glycon, Gen-Metformin	Unpleasant taste, metallic taste	1
Metoprolol tartrate	Lopressor, Apo-Metoprolol, Loresor SR	Dry mouth	3

Meloxicam	Mobic, Mobicox	Facial edema, dry mouth, ulcerative stomatitis, taste perversion	1
Etanercept	Enbrel	None	1
Estrogen A, conjugated synthetic; *estropipate*	Cenestin; *Ogen, Ortho-Est*	None; *Exacerbates gingivitis, bleeding*	2
Pantoprazole sodium	Pantaloc, Protonix	Aphthous stomatitis, candidiasis, dry mouth, dysphagia (all <1%)	1
Cyclobenzaprine HCL	Flexeril	None	1
Thyroid USP	Cholaxin	None	1
Diazide	Triamterene	Dry mouth	1
Advair	Fluticasone	Oral candidiasis	2
Advil	Ibuprofen	Dry mouth, bleeding, stomatitis, lichenoid reactions	1
Arimidex	Anastrozole	None	1
Didrocal	Calcium Carbonate & Etidronate Disodium	Altered taste, glossitis	1
Eletriptan	Relpax	Dry mouth, facial edema	1

(Gage & Pickett, 2005)