A Pilot Study to Measure the Relationship Between Myocardial Infarction and Periodontal Disease: Application of a Case-control Design

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Submitted to: Dr. W. Montelpare
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Abstract

Recent public health reports have identified Northwestern Ontario and Thunder Bay for increased risk of coronary heart disease among several cohorts.

The present study investigated the relationship between myocardial infarction and periodontal disease in individuals' aged 55 and over. As well, a biological mechanism, which may be an indicator to coronary heart disease and periodontal disease, was measured among the sample population.

Both myocardial infarction and periodontal disease can result from inflammation of the micro-vascular blood supply that provides oxygenated blood to the tissue of the heart and gums, respectively. Several research studies have reported a link between periodontal disease and coronary heart disease/myocardial infarction. Furthermore, the suggested association between myocardial infarction and periodontal disease is based on the simultaneous occurrence of the two events and therefore the potential co-incidence may have a measurable probability. In both coronary heart disease and periodontal disease clinicians may observe an increased concentration of c-reactive protein and/or fibrinogen. Not only are these proteins prevalent in individuals who are diagnosed with coronary heart disease, myocardial infarction and periodontal disease, but also the presence of these proteins may further exacerbate the inflammatory condition and thereby, degrade the integrity of the micro-vascular system. Thus suggesting that the underlying causal mechanism for both myocardial infarction and periodontal disease might be the presence of c-reactive protein and/or fibrinogen.

Participants were identified and recruited from a population of cardiac patients in the region of Thunder Bay. All participants have cardiac disease confirmed clinically and objectively by symptoms, stress test, cardiolite and/or angiogram. At least half of the selected participants had experienced a myocardial infarction within the past twelve months (February 2001 to February 2002) as verified by the cardiologist.

The periodontal evaluation included, a count of the number of teeth present, a measure of gingivitis, the average probing depth (six sites- medial and distal), and the individual's BANA hydrolase (will be referred to as BANA from this point) test score. The composite of the measures was thus considered representative of the individual's periodontal disease.

Forty-two people were involved in this study as research participants. The results indicated that negative relationship was apparent between the risk of having a myocardial infarction and the measure of periodontal disease. However, a relationship was found between the risk of having a myocardial infarction and the blood measures of c-reactive protein and fibrinogen. This research has provided a baseline for future research, and many suggestions and recommendations have come from the results.

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CHAPTER ONE: INTRODUCTION

Background and rationale for the proposed research study

Coronary heart disease is among the most prevalent diseases in the general population of North America, but more disturbing is the health report by the Institute for Clinical Evaluative Sciences (ICES, 1999) which identified Northwestern Ontario and especially Thunder Bay for higher risk of coronary heart disease among several cohorts (ICES, 1999). While this statistic is alarming, a cursory review of traditional risk factors at the community level, such as prevalence of smoking, ethnicity, predisposing diseases and/or such conditions as diabetes, hypertension and obesity, diet and socio-economic status combined with lifestyle, support the finding of a "higher risk" status.

Considering the plethora of previous research in cardiovascular and coronary heart disease (CHD) it is relatively easy to determine risk for coronary heart disease and coronary heart disease related events such as a myocardial infarction (MI). For example, smokers have an increased risk of coronary heart disease and/or myocardial infarction. Obese individuals are more likely to have a myocardial infarction than individuals with a higher lean body mass. Individuals, especially males or post menopausal women, who have a family history of coronary heart disease are more likely to have coronary heart disease or experience coronary heart disease related events than individuals who do not have such family histories.

Despite such overwhelming evidence, society is slow to change behaviours, which are known causes of coronary heart disease, and coronary heart disease related events. Among the many possible reasons for behaviour change is the recognition of direct risk associated with a known condition or event. That is, while smoking is a known cause of several diseases and conditions, individuals are less likely to seek

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smoking cessation programs until experiencing a coronary heart disease event or until they are diagnosed with a "direct" condition such as emphysema, reduced lung function or cancer.

Research in periodontal disease may provide yet another identifier of "direct" risk for coronary heart disease and coronary heart disease related events. Several research studies have reported an association between periodontal disease and coronary heart disease based on the simultaneous occurrence of the two events (Arbes, Lade, and Beck, 1999; Beck, 1998; Howell, Ridker, Ajani, Hennekens and Christen, 2000; Loesche et al. 1995, Loesche, Schork, Terpenning, Chen, Domingues, and Grossman, 1998; Mattila, Valtonen, Nieminen, and Huttunen, 1995; Seymour and Steel, 1998). Both coronary heart disease and periodontal disease result from inflammation of the micro-vascular blood supply that provides oxygenated blood to tissue. According to Beck et al. (1996) the underlying influences of the two diseases occurring simultaneously may be due to a genetic condition called hyperinflammatory monocyte phenotype trait.

In coronary heart disease and/or myocardial infarction and periodontal disease clinicians may observe an increased concentration of c-reactive protein and/or fibrinogen. Not only are these proteins prevalent in individuals who are diagnosed with coronary heart disease, but also the presence of these proteins may further exacerbate the inflammatory condition and thereby lead to a degradation of the integrity of the micro-vascular system as a precursor to periodontal disease. This would suggest that the underlying casual mechanism for both coronary heart disease and periodontal disease might be the presence of c-reactive protein and/or fibrinogen (Kornman, Pankow, Offenbacher, Beck, di Giovine and Duff, 1999). Most important, the observed co-

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incidence of myocardial infarction and periodontal disease, as a result of the presence of c-reactive protein and/or fibrinogen, may have a measurable probability.

Purpose and research hypothesis

The purpose of this research study was to determine the association between periodontal disease and myocardial infarction under a case-control design. The research hypothesis is that the odds of having a myocardial infarction are higher if participants had periodontal disease. The secondary hypothesizes are 1) C-reactive protein and fibrinogen will be elevated more often in participants with a myocardial infarction than in participants without a myocardial infarction; lifestyle scores will be lower and 2) c-reactive protein and fibrinogen will be elevated more often in participants with periodontal disease than in participants without periodontal disease; lifestyle scores will be lower.

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CHAPTER TWO: LITERATURE REVIEW

Introduction

Risk factors for coronary heart disease include high blood pressure, high blood cholesterol, smoking, obesity, diabetes, and physical inactivity (Grundy, 1998). In addition Arbes, et al. (1999) and Mattila, et al. (1995) suggested that periodontal disease might also be considered as a risk factor for coronary heart disease. Recognizing periodontal disease as a risk factor for coronary heart disease is particularly important to Northwestern Ontario, given the identification of Thunder Bay as a "hot spot" for increased coronary heart disease by the 1999 report of the Ontario Institute for Clinical Evaluative Sciences (ICES) (1999). Most important, changes in behavioural factors such as lifestyle can have both direct and indirect effects on coronary heart disease and periodontal disease (Burt, 1996; Loesche, et al. 1998).

Beck, Gracia, Heiss, Vokonas, and Offenbacher (1996) and Loesche (2000) suggested that there might be a common mechanism, which may relate coronary heart disease, including myocardial infarction, and periodontal disease. Kornman et al. (1999) suggested that the underlying causal mechanism for both diseases might be the presence of elevated levels of c-reactive protein and/or fibrinogen. Both c-reactive protein and fibrinogen are acute phase proteins which claim a relationship with inflammation. This association is based on the simultaneous occurrence of the two events, which may be due to a genetic condition called hyperinflammatory (monocyte phenotype) trait (Beck et al. 1996). The hyperinflammatory trait may induce concentration levels of c-reactive protein and/or fibrinogen.

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Coronary Heart Disease and Lifestyle

Both, coronary heart disease and periodontal disease can both be reduced by modifications in behaviour. Joshipura, Rimm, Douglass, Trichopoulos, Ascherio, and Willett (1996) stated that people who take care of their dentition may be at lower risk for coronary heart disease, simply because they practice healthy behaviours. Similarly, health status has been associated with periodontal disease (Jette, Feldman and Tennstedt, 1993) and coronary heart disease (Johansen, 1999). The results of a study by Johansen (1999) displayed that those reporting heart disease have several medical problems. More than a third (36%) of the participants reported having at least two other major illnesses, including, arthritis, stroke, and high blood pressure.

Modifications to traditional lifestyle and risk factors for coronary heart disease could help to decrease the likelihood of developing a myocardial infarction. Jousilahti, Vartiginen, Tuomilehto, Pekkanen, and Puska (1995) examined the effect of traditional risk factors on coronary heart disease in males and females. Nearly half of the decline of death due to a myocardial infarction was associated with the difference in risk factors, primarily total serum cholesterol and blood pressure. According to Stampher (2000) smoking reduction and an increase in postmenopausal hormones may influence a decline in the incidence of coronary heart disease.

Stampher (2000) and Jousilahti et al. (1995) emphasized the importance of risk factor modification by focussing on the contributions a negative lifestyle has on developing coronary heart disease. These contributing risk factors may also influence a person's oral health, thus increasing their risk of periodontal disease. If an individual's medical health is not a priority then their oral health may be affected negatively (Jette et al. 1993; Johansen, 1999; Syrjanen, 1990).

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Periodontal Disease and Coronary Heart Disease

Several research studies have been completed to determine if there is an association between coronary heart disease and /or myocardial infarction and periodontal disease. Mattila, et al.'s (1989) study reported periodontal disease and myocardial infarction to be associated in two separate case control studies. A total of 100 patients with myocardial infarction and 102 controls were selected from the community, at random, to analyze the role of chronic bacterial infections as a risk factor for myocardial infarction. Observations showed dental caries or periodontal disease or both were more common among patients with myocardial infarction than among controls. Mattilla, et al. (1989) suggested this could be attributed to two things: (1) dental caries and coronary heart disease share several common aetiological factors (smoking, diabetes, and resistance to insulin) and/or; (2) subjects who take care of their dentition may also be concerned about other areas of their health.

A separate study by Mattila, Nieminen, Vantonen, and Hietaniemi (1993) showed a positive association between periodontal disease scores and coronary heart disease in males. Mattila et al. (1993) used a pantomography x-ray to identify dental infections. The measures included vertical bone pockets and periapical lesions, tertiary caries, pericoronitis and furcation lesions. Males with more severe stenosis showed the highest dental disease scores (Mattila et al., 1993). However, due to the low number of females no conclusions were drawn about the females. Syrjanen (1990) and Beck, Gracia, Heiss, Vokonas and Offenbacher (1996) also reported an association between periodontal disease and cardiovascular diseases, including coronary heart disease.

A more recent study by Mattila, Asikainen, Wolf, Jousimies-Somer, Valtonen, and Nieminen (2000) used a matched case-control study to analyse the dental pathology

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found in coronary heart disease patients. Three categories were used to classify the 85 participants: coronary heart disease without a myocardial infarction, coronary heart disease with a myocardial infarction in the past, or a recent (< 2 weeks) myocardial infarction. The dental exam consisted of measuring the probing depth and the presence or absence of bleeding. A radiographic examination was also used to record the number of teeth, roots, carious teeth, impacted teeth, periapical lesions, pericoronitis, vertical bone pockets and furcation lesions. The results of the study indicated that all dental indices, for males and females, were higher in the coronary heart disease patients than in the controls (Mattila et al., 2000).

The coronary heart disease-periodontal disease association was also reported by Morrison, Ellison, and Taylor (1999) in a retrospective cohort study, in a prospective study by DeStefano, Anda, Kahn, Williamson and Russel (1993), and a study by Loesche et al. (1998), which utilized a convenience sample. DeStefano et al. (1993) indicated that participants with periodontal disease had more than a two fold increased risk of dying compared with men who had no periodontal disease at baseline. Morrison et al., (1999) reported coronary heart disease was more likely to be found in people with one to 14 teeth than in people with 15 to 28 teeth. Similar to Morrsion's et al.'s (1999) results, Loesche et al. (1998) reported that individuals with coronary heart disease were more likely to be wearing at least one full denture.

However, there is very little information, which describes the physiology of the association between coronary heart disease and periodontal disease. The link between coronary heart disease and periodontal disease is not clear from the literature available. The following section is going to provide hypotheses that may link coronary heart disease and periodontal disease together.

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C-reactive Protein, Fibrinogen and Coronary Heart Disease

An acute phase protein is a biological marker for underlying systemic inflammation (Ridker, Cushman, Stampfer, Tracy, and Hennekens, 1998). C-reactive protein and fibrinogen are two of the most common types of acute phase proteins and have been noted to have an association with coronary heart disease and periodontal disease. Ebersole, Machen, Steffen and Willmann (1996), Levenson, Giral, Razavian, Gariepy, and Simon (1995), Toss, Lindahl, Siegbahn, and Wallentin (1997), Ridker, Burning, Shih, Matias, and Hennekens (1998), Ridker et al.(1997) reported high levels of fibrinogen and c-reactive protein as a sign of inflammation in patients with coronary heart disease. C-reactive protein and/or fibrinogen can contribute with blood clotting and subsequently can lead to the development of coronary heart disease and cardiovascular disease (El-Sayed, 1996; Kannel et al., 1987; Toss et al, 1997).

Ridker et al. (1997) also reported an association between inflammation and myocardial infarction. There was a reported risk of myocardial infarction in individuals with elevated concentrations of c-reactive protein (Ridker et al., 1997). Ridker et al. (1997) reported that individuals who had higher levels of c-reactive protein were three times more likely to experience a myocardial infarction.

High concentrations of c-reactive protein and fibrinogen have been associated with an increased risk of atherosclerosis and coronary heart disease (Levenson and Tracy, 1995). In the Atherosclerosis Risk in Communities (ARIC) Study, the Scottish Heart Health Study, and in a study by Kannel, Wolf, Castelli and D'Agostino (1997) and a study by Eriksson, Egberg, Wamala, Orth-Gomer, Mittleman, and Schenck-Gustafsson (1999) plasma fibrinogen was reported as a risk factor for coronary heart disease in women.

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Lindahl, Toss, Siegbahn, Venge and Wallentin (2000) reported that concentrations of c-reactive protein in healthy men can predict the risk of first myocardial infarction and ischemic stroke. Moreover, it was suggested that c-reactive protein was not only a short term biological marker of risk, but was also a long-term marker, even in events occurring six or more years later.

Eriksson et al. (1999) have suggested that the inflammatory process involved in atherosclerotic diseases may explain elevated fibrinogen levels in patients with coronary heart disease and myocardial infarction. Eriksson et al. (1999) reported that fibrinogen remained statistically significant after controlling for other factors (c-reactive protein), thereby suggesting that fibrinogen is not only a marker of coronary heart disease, but is directly involved in the pathogenesis of coronary heart disease.

Ferreiros et al. (1999) analysed concentration levels of c-reactive protein in patients with coronary heart disease. Levels of c-reactive protein were measured at hospital admission and 90 days later or after the occurrence of a major cardiac event. Levels of c-reactive protein changed significantly between admission and discharge (Ferreriros et al., 1999).

Elevation of c-reactive protein in unstable angina is common and may indicate the presence of evolving inflammation (Ferreriros et al., 1999). By knowing that c-reactive protein is elevated in patients with unstable angina and disrupted plaque, medical professionals will be able to perform more aggressive therapy on people who may need it. High levels of c-reactive protein and fibrinogen may indicate persistent plaque and identify those at risk for a reoccurring cardiac event (Ferreiros et al., 1999).

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Periodontal Disease

Defining Periodontal Disease

Periodontal disease includes inflammation of the gingival tissues with some loss of both the attachment of the periodontal ligament and boney support (Scannapieco and Genco, 1999). Periodontal disease is generally identified by the number of infected sites with clinical attachment loss and the probing depth of each infected site (Scannapieco and Genco, 1999). Clinical attachment loss and probing depth can be used to measure the severity of periodontal disease (Scannapieco and Genco, 1999). As defined by Wilkins (1999) probing depth is the distance from the gingival margin to the location of the periodontal probe tip at the coronal border of attached periodontal tissues.

Approximately 90% of all adults, aged 55 to 64, are affected with periodontal disease with a probing depth of two millimetres (Burt, 1996). However, only 64% of those aged 55 to 64 have periodontal disease with four millimetres or more (Burt, 1996).

Periodontal Disease, Tooth Loss and Coronary Heart Disease

Burt (1996) examined the epidemiology of periodontal disease and reported that poor oral hygiene and periodontal disease were related to socio-economic status and education levels. More specifically, clinical attachment loss (of seven mm or more) was closely related to educational levels. Persons with periodontal disease at baseline had a 25% greater risk of subsequent coronary heart disease than individuals without periodontal disease and periodontitis. Again, illustrating the association between periodontal disease and coronary heart disease.

Joshipura et al. (1996) and Paunio, Impiuaara, Tiekso and Maki (1993) found that tooth loss and periodontal disease showed an association with increased risk of coronary heart disease and ischemic heart disease in two separate studies. Both studies included

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participants over the age of 40. The results showed that oral infection, expressed as missing teeth was an independent explanatory factor for coronary heart disease and ischemic heart disease.

Loesche et al. (1995) used a questionnaire, in conjunction with a dental exam, to measure number of teeth missing and gingivitis (assessed by a papillary bleeding score) to measure dental complications in seniors. Independent-living participants and dependent-living participants were compared. The four groups were independent-living group veterans affair (VA), independent-living group non- VA, dependent nursing home group and dependent hospitalized group. The study showed that the independent living groups had better dental health and less periodontal disease than the dependent living participants (Loesche et al., 1995). More specifically, Loesche et al. (1995) found that the independent groups had lower levels of edentia (toothless), fewer missing teeth and less periodontal disease (decayed/missing/filled teeth, gingivitis and saliva) than the dependent living group. The dependent nursing home group had 50% more edentia than the independent-living group veterans' affair.

Ninety percent of the dependent living hospitalized group were only removed from their independent status for one to three weeks prior to the study. Due to the short time span, Loesche et al. (1995) compared the dependent group to the independent living veterans affair group. The people who entered the dependent living group had high levels of dental morbidity (edentulous), which cannot be explained from dependent living. This raised the question that dental decay and periodontal disease are somehow related with the illness that brought them to the hospital.

All variables (age, gender, education, marital background, smoking, drinking habits and diabetes) were similar, except there were differences in the individual's

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periodontal disease status which included high level of edentia, tooth loss and decay, high prevalence of difficulty chewing certain foods, low salivary flow, high level of gingivitis and high plaque index (Loesche et al., 1995). The patients may have participated in activities that were conducive to their medical condition and their oral health, which may explain why some of the patients were hospitalized (Loesche et al., 1995). Loesche's et al. (1995) research study supported the hypothesis that dental health, particularly periodontal disease, and physical health may be related.

Periodontal disease and dental carries are the main causes for tooth loss in Canada and the United States (Ong, 1998; Paunio et al. 1993). Phipps and Stevens (1995) and Murray, Clarke, and Locker (1997) reported more tooth loss due to periodontal disease than to caries in Canada and the United States. Ong (1998) has also reported attachment loss to correlate with tooth mortality due to periodontal disease. Burt et al. (1990) showed that attachment loss (of four mm or more) is one of the greatest risk factors for tooth mortality and tooth loss. Greater periodontal attachment loss is associated with a higher rate of tooth loss (Ong, 1998). Moreover, if a patient suffers more severe periodontal disease, their chances of tooth loss are greater (Ong, 1998).

Periodontal disease may be induced by cigarette smoking, thus potentially leading to tooth loss (Jette et al., 1993, and McGuire and Nunn, 1996). A study by Hamel and Graig (1997) estimated that regular smokers were twice as likely to develop periodontal disease than non-smokers.

Generally there is more severe periodontal disease and tooth loss in smokers than in non-smokers (Hamel and Graig, 1997; Stoltenberg, Osborn, Phlstrom, Hersberg, Acppl and Fischer, 1993). More diseased teeth and sites, an increase in probing depths, alveolar bone loss, attachment loss and tooth loss is found more often in smokers (Hamel and

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Graig, 1997; Linden and Mullally, 1994; Grossi et al., 1995). Exposure to smoke and poor oral hygiene will increase the risk of developing periodontal disease and tooth loss (Holm, 1994) (cited in Hamel and Graig, 1997).

C-reactive Protein and Periodontal Disease

Increased levels of acute-phase proteins have been associated with gingival inflammation, including gingivitis and periodontitis (Ebersole, Machen, Steffen and Willmann, 1997). In a study by Ebersole et al. (1997) c-reactive protein was found to be elevated above a normal baseline in patients with clinically diagnosed periodontitis. In Ebersole's et al. (1997) study periodontitis was defined as bone loss with loss of attachment, bleeding on probing, and periodontal pockets greater or equal to five millimetres. Those participants with the most severe disease exhibited the greatest levels of c-reactive protein (Ebersole et al., 1997).

C-reactive Protein and Fibrinogen as an Underlying Mechanism to Coronary Heart Disease and Periodontal Disease

Kornman et al. (1999) suggested several hypothetical mechanisms, which may explain the epidemiological association between coronary heart disease and periodontal disease. There has been evidence that the plaque from oral diseases can enter the blood stream and be present as atheromatous plaques (the abnormal fatty deposit in an artery) (Loeche, 2000). This suggests, the direct involvement of periodontal bacteria in the atheroma processes. As well, the direct involvement of inflammatory mediators (c-reactive protein and/or fibrinogen) from periodontal disease on the atheroma processes has been hypothesized as a mechanism to associate the two diseases. There are also common predisposing risk factors that influence both diseases, which may link coronary heart disease, and periodontal disease. Kornman et al. (1999) suggested that

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combinations of the above interactions might be critical in determining the association between coronary heart disease and periodontal disease.

Beck et al. (2000; 1996) suggested that the potential biological mechanism between coronary heart disease and periodontal disease might be an underlying hyper inflammatory trait among certain individuals. This hyper inflammatory trait may serve as a common antecedent to both coronary heart disease and periodontal disease and may be induced by genetic, behavioural and environmental exposure (Beck et al., 2000). Certain factors, such as diet, may exacerbate the hyper inflammatory monocyte pheno type and may thereby contribute to atherosclerosis and periodontal disease. High concentrations of plasma c-reactive protein and/or fibrinogen will indicate inflammation and will be the result of the hyper inflammatory trait (Beck et al., 2000).

There is an association between increased fibrinogen and/or c-reactive protein levels and early signs of atherosclerosis (Toss et al., 1997). Fibrinogen and/or c-reactive protein interact with the particles involved with blood clotting and may contribute to the formation and progression of atherosclerotic plaques and may increase the likelihood of experiencing a myocardial infarction (Toss et al., 1997). Moreover, fibrinogen concentration levels increase after myocardial damage (Toss et al., 1997). Increased levels of these acute phase proteins may also increase the risk of a cardiac reoccurrence.

The inflammatory process involved in coronary heart disease may partially explain elevated plasma fibrinogen levels in a study by Eriksson et al. (1999). Fibrinogen remained statistically significant after controlling for c-reactive protein. This suggests that fibrinogen is not only a marker for coronary heart disease, but is involved in the pathogenesis of occlusive thrombus formation (Eriksson et al., 1999).

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Beck et al. (2000) suggested that a person with the hyperinflammatory trait who suffers from periodontal disease might increase their likelihood of atherosclerosis and coronary heart disease. Moreover, the bacteria from periodontal infection may directly contribute to the pathogenesis of atherosclerosis by providing inflammatory cytokines. Loesche (2000) stated that levels of acute phase proteins, specifically c-reactive protein and fibrinogen, can be elevated from the bacteria from periodontal disease and oral infections. The consequence of these bacteria could be plaque bacteria cells depositing on the lining of fatty streaks, which could collect and potentially generate inflammation (Loesche, 2000). Moreover, dental plaque has been shown to be present in atheromatous plaques (Loesche, 2000). If bacteria, related to periodontal disease gains access to the blood stream, the bacteria could cause particles in the blood to collect into a mass causing a blood clot (Loesche, 2000). This process is known as platelet aggregation (Loesche, 2000). The blood clot can potentially lead to a myocardial infarction by blocking the pathway(s) to the heart and restricting the flow of blood and oxygen. Common behavioural risk factors and/or genetics may be the underlying mechanism that relates the two diseases. However, more research needs to be done in order to address the biological relationship(s) between the coronary heart disease and periodontal disease.

Summary

The likelihood of developing coronary heart disease and /or myocardial infarction may be increased if other areas of an individual's health are not taken care of. It has been demonstrated that individuals with periodontal disease may increase their risk of developing coronary heart disease and thus potentially experiencing a myocardial infarction (Arbes et al., 1999; Beck et al., 1998; Howell et al., 2000; Loesche et al., 1995, 1998; Mattila et al., 1995; Scannapeico and Genco, 1999; Seymour and Steel, 1998).

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Research has suggested that the link between the two diseases may be explained by common behaviourial risk factors (smoking, aging, diabetes and education) (Mattila et al., 1989; Offenbacher, 1999; Paunio, 1993) and/or a biological association (Loesche et al, 1995, 1998; Kannel et al., 1987; Toss et al., 1997). However the link between myocardial infarction and periodontal disease is not clear from the available literature.

Consistency between different studies, have provided support to suggest there is a relationship, between myocardial infarction and periodontal disease in which researchers should be interested in (Arbes et al., 1999; Beck, 1998; Howell et al., 2000; Loesche et al., 1995, 1998; Mattila et al., 1995; Seymour and Steele, 1998). Research on coronary heart disease risk factor modification can potentially improve the health and well being of individuals at risk of developing coronary heart disease and /or myocardial infarction.

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CHAPTER THREE: METHODOLOGY

Purpose

The purpose of this research study was to determine the association between periodontal disease and myocardial infarction under a cross sectional case-control design using a 2x2 table. My primary objective was to determine if a relationship exists between myocardial infarction and periodontal disease. My primary hypothesis is, the odds of having a myocardial infarction is higher if a participant has periodontal disease. My secondary objective was to assess three potentially confounding variables. They were c-reactive protein, fibrinogen, and lifestyle. The first hypothesis is that c-reactive protein, and fibrinogen will be elevated more often in participants with a recent myocardial infarction than in participants without a myocardial infarction, and lifestyle scores will be lower. My second hypothesis is that c-reactive protein, and fibrinogen will be elevated more often in participants with periodontal disease than in participants without periodontal disease, and lifestyle scores will be lower.

Research Design

The computation of the "odds ratio" was used to test the research hypothesis under a case-control design. The two by two structure of the research design is illustrated in Figure 1, below. Periodontal scores for individuals who have experienced a myocardial infarction were compared to individuals who never experienced a myocardial infarction (all participants had coronary heart disease).

	CHD +	CHD -
Periodontal Disease +	A	B
Periodontal Disease -	C	D

A further explanation of the "odds ratio" is presented in Appendix F.

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Procedure for Data Collection

Participants

Inclusion Criteria

Participants were identified and recruited from a population of cardiac patients with confirmed myocardial infarction, or ischemic heart disease or coronary artery disease, in the Region of Thunder Bay. At least half of the selected participants experienced a myocardial infarction between February 2001 and February 2002 as verified by Dr. C. Lai. The participant group who experienced a recent myocardial infarction was identified through patient files from Dr. C. Lai's office and had been discharged for home convalescence. An individual was determined to be positive for myocardial infarction if they experienced a clinically diagnosed myocardial infarction between February 2001 and February 2002. Medical records verified positive myocardial infarction status.

Recruitment

Four approaches were used for patient recruitment. The researcher went through the cardiologist's files and selected patients based on the inclusion criteria. The potential participants were then contacted by telephone to determine if they were interested in participating in the study. Patients were also recruited from Cardiac Rehabilitation classes in at Thunder Bay Regional Hospital and The Canada Games Complex. The researcher then contacted these potential participants by telephone. The third approach was an advertisement in the local newspaper.

Preparation

The tools used to record the participant's information included:

- A consent form (see Appendix B).

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- An identification page (see Appendix C).
- A FANTASTIC Lifestyle Check-list (questionnaire) (see Appendix E).
- A periodontal disease record sheet (see Appendix D).
- The completed x-ray was added to the patient's file.

Individuals that agreed to participate in this study were asked to review and sign a letter of informed consent (Appendix A and B) outlining the purpose, risk, and potential benefits associated with this study. The study protocol and consent forms were approved by Lakehead University ethics committee and by the Thunder Bay Regional Hospital ethics committee. Individuals were informed that they were under no obligation to participate, that they may withdraw from the study at any time, and that all information regarding them will remain confidential. The letter described all aspects of the study including the requirements that the participant agreed. The requirements included, providing a blood sample, to participate in a test for periodontal disease and to complete a questionnaire that provided an assessment of coronary heart disease risk factors. The participants were encouraged to ask any questions they may have had regarding the research and assessment techniques used throughout the study. After all questions were answered, the participants were asked to sign a letter of informed consent allowing them to participate. Individuals were not allowed to participate in the study until they signed the letter of informed consent. Individuals were also required to complete a form with their name and address and the following question, "Have you ever had a heart attack? If so when?" The participant's address was required in order to mail out the results at the end of the study (Appendix C). Participants were scheduled 15 minutes apart and no more than three people were scheduled at the same time.

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The researcher also searched the patient files at Dr. Lai's office. Those individuals who met the inclusion criteria were called to participate in the study.

An advertisement in the local newspaper was also used to recruit volunteers for the study. However, no phone calls were generated through the advertisement.

Sample

The minimum sample size for this "pilot" study was n = 40. Under advice from the cardiologist the sample size was chosen. Participants were organized into four distinct groups:

i) Myocardial infarction (+) periodontal disease (+)

ii) Myocardial infarction (-) periodontal disease (+)

iii) Myocardial infarction (+) periodontal disease (-)

iv) Myocardial infarction (-) periodontal disease (-)

Each participant provided a blood sample, which was used to determine resting levels of c-reactive protein and fibrinogen. The procedures used to collect blood are given below. In addition, each individual participated in a periodontal assessment using a panoramic screening to count number of missing teeth, as well as an assessment of gingival condition and a measure of periodontal probing depth and the BANA test. The BANA test determined if P. gingivalis, B. forsythus or T. denticola were present. Finally lifestyle and behaviour were evaluated using the standard "FANTASTIC Lifestyle Questionnaire" (CSEP).

Blood Sample Assessments

Seventeen milliliters of blood was drawn from each participant using the vacutainer® technique under standard and accepted procedures of phlebotomy to measure c-reactive protein, and fibrinogen. A certified phlebotomist completed the blood

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sampling. C-reactive protein was determined by rate nephelometry and was measured in micrograms per milliliter. C-reactive protein was stratified as: less than or equal to four mg/L (recorded as four), or greater than four mg/L (recorded as a continuous variable).

Fibrinogen was measured according to the anti-cardiolipin antibody (ACL) method based on the total change in light scatter associated with the formation of a fibrin clot. Fibrinogen was stratified and measured as less than or equal to 2.8 g/L, 2.8 to 3.1 g/L, 3.1 to 3.6 g/L and greater than or equal to 3.6 g/L. Fibrinogen was stratified as less than or equal to 3.1 g/L, and greater than 3.1 g/LThe determination of c-reactive protein and fibrinogen strata are consistent with the suggested approaches of Eriksson et al. (1999), Kannel et al. (1987), and Toss et al. (1997).

Periodontal Assessment

There is no gold standard in defining a periodontal disease. For the purpose of this study the periodontal assessment were based on current literature and advice from the clinical advisors. The four indicators used to assess for periodontal disease are listed and described below.

Panoramic screening to count number of missing teeth

Panoramic screening uses an x-ray image of the face to make a complete half circle from ear to ear. The x-ray produced a complete dimensional representation of all the teeth. Prior to the test the researcher briefly explained the procedure. Following the x-ray, participants were given instructions for the next stage of the dental assessment (probing depth). Teeth were counted and stratified in to two groups. An individual who had 12 or more teeth scored negative for periodontal disease and an individual who had less than 12 teeth scored positive for periodontal disease.

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Probing Depth

Following the panoramic screening, the probing depth measures of the participant were taken. As defined by Wilkins (1999) probing depth is the distance from the gingival margin to the location of the periodontal probe tip at the coronal border of attached periodontal tissues. A calibrated periodontal probe was used to measure the probing depth of six sites, medial and distal points (tooth number, 16,21,24,36,41,and 44). Probing depth was measured in millimeters and was classified as none (less than two millimeters), mild (greater than two millimeters and less than five millimeters) or severe (greater than five millimeters). These measures are consistent with Beck, Garcia, Heiss, Vokonas and Offenbacher (1996).

A score in millimeters was recorded for each of the six sites, medial and distal; a total of 12 measurements. These measurements were then summed and divided by the number of sites measured (12) to calculate the mean. If a tooth was missing the tooth beside was utilized. If there was not a tooth to measure than the total number of sites was reduced.

Gingival Condition

Prior to the gingival assessment, the researcher briefly explained the procedure. The dental hygienist used a periodontal probe to assess for bleeding gums and to determine gingival condition. Gingival condition was classified as none, mild gingivitis or severe gingivitis, numbered one, two, or three respectively. For the purpose of this study, mild gingivitis was defined as obvious inflammation in the free gingiva of one or more tooth area where the inflammation did not completely surround the tooth. Severe gingivitis was defined as marked redness, swelling, tendency to bleed and ulceration

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which may or may not have extended around the tooth. These definitions are consistent with Morrison et al. (1999).

Each of the six sites (tooth number, 16,21,24,36,41, and 44) was given a score for gingival condition (one, two or three) for the; a total of 6 measurements. These measurements were then summed and divided by the number of sites (6), to find the mean. If a tooth was missing than the tooth beside was utilized. If there was no tooth to measure than the total number of sites was reduced and divided by the number sites used.

BANA Test

The BANA test was used to detect Bacteroides gingivalis, Bacteroides forsythus and Treponema denticola. A PERIOSAN Reagent strip was used to test for the bacteria in the mouth. Several individuals who participated in the study did not have teeth, thus the sample (biofilm plaque (tongue plaque)) was taken from the tongue. It is recommended that the sample be taken from the individual's teeth. A sample biofilm was taken from the tongue and was applied to the PERIOSCAN reagent strip. Next the upper test matrix was moistened with distilled water using a cotton ball, then the PERIOSCAN reagent strip was folded in half and heated for 15 minutes (at 55 degrees celsius). Small, faint traces of blue coloration on a pale red-brown background suggests a weak positive. Distinct blue patches of blue suggests a positive, while no blue coloration represent a negative.

Lifestyle and Behaviour Assessment

FANTASTIC Lifestyle Questionnaire

The FANTASTIC Lifestyle Questionnaire provided a score regarding the participants' lifestyle (see Appendix E) (CSEP). The nine areas measured were family and friends, activity, nutrition, tobacco, alcohol, sleep, type of behaviour, insight and

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career. Participants were given a statement and the reader responded by choosing almost never, seldom, some of the time, fairly often or almost always. At the end of the questionnaire the researcher counted the total number of each response. Depending on the response it was then multiplied by a number between zero and four. The scores were then added for a grand total. The maximum score is 100. A high score of 85 -100 is excellent (lifestyle and behaviour), 70-84 is very good, 55-69 is good, 35-54 is fair and 0-34 needs improvement. This data provided an estimate of "health risk" based on lifestyle for each participant.

For the purpose of this research study 100 - 70 were stratified as healthy and 69 and under were stratified as unhealthy.

Periodontal Score Calculation

If an individual scored positive on two or more of the periodontal disease indicators they were identified as positive for periodontal disease, in this study.

- Individuals with more than 12 teeth were provided a negative score in the count of teeth (negative score for periodontal disease) and individuals with less than 12 teeth were provided a positive score for periodontal disease in the *count of teeth.*
- A gingival score greater than or equal to two was positive for this indicator of periodontal disease and a score less than two were negative for this periodontal disease indicator when calculating the average score for *gingival condition.*
- A score less than five was considered negative for this periodontal disease indicator and a score greater than five was considered positive for this periodontal disease indicator when calculating the average score for *probing depth.*

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For both gingival and probing depth scores, six sites, medial and distal, (tooth number, 16,21,24,36,41,44) for each participant were measured. If a person was missing a tooth the tooth beside was measured. It is important to note that some people had zero teeth.

- *The BANA test* provided a positive or negative score.

Table 1, Sample Computation for the Composite Score Representing Periodontal Disease, provides information on the dental calculation for a hypothetical individual. The individual scored positive on three of the four tests used to determine an individual's periodontal disease status. They were, number of teeth present, degree of gingivitis and probing depth measurements. Thus, the individual was rated as positive for periodontal disease.

Table 1:
Sample Computation for the Composite Score Representing Oral Health*

DATA	SCORE	GUIDELINES
Number of teeth	7 teeth present = positive	Individuals with more than 12 teeth were provided with a negative score in the *count of teeth* and individuals with less than 12 teeth were provided with a positive score in the *count of teeth.*
Average gingival score	2.7 = positive	A gingival score greater than two was positive and a score less than two was negative when calculating the average score for *gingival condition.*
Average probing depth score	1.2 = negative	A score less than five was considered negative and a score greater than five was considered positive when calculating the average score for *probing depth.*
BANA score	Positive	*The BANA test* provided a positive or negative score.

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Coronary Heart Disease and Myocardial Infarction Measures

Coronary Heart Disease

Participants in this study were diagnosed with progressive coronary heart disease as determined by the cardiologists' files.

Myocardial Infarction

An individual was determined to be positive for myocardial infarction if they experienced a clinically diagnosed myocardial infarction in the past 12 months (February 2001 to February 2002). Medical records verified positive myocardial infarction status.

Confidentiality

All data collected from this study will be stored such that the participant's name will not be attached to the data. Once the results of the blood analyze, dental tests and questionnaire have been entered into the database there will be no way for anyone except the researcher and associates to discern one subject from another. All data will be stored for seven years under the direction of Dr. William Montelpare at the School of Kinesiology, Lakehead University.

Statistical Analysis

The statistical analysis will consist of descriptive statistics and computation of the "odds ratio" and confidence interval for each reproach hypotheses.

Potential Benefits and risks from the Proposed Study

The potential risks associated with participation in this study were minimal. There may have been some slight discomfort and bruising at the point of venipuncture and possibly minor bleeding and discomfort with intra-oral examination. As previously stated, the risk associated with participation in this study was explained to each participant prior to the data collection session. The risks assumed with participation in

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the dental evaluation for missing teeth and gingivitis are the same as those an individual would incur during any visit to the dentist's office.

The potential benefits associated with participation in this study will be derived from the findings. All participants received, without charge, feedback about c-reactive protein, and fibrinogen levels. Finally, all participants were given a summary of the findings of this study in the form of a general health profile at the completion of the analysis.

These results provide health researchers with the information necessary to evaluate the role of periodontal disease as a predictor of coronary heart disease and coronary heart disease related events. These findings are important to our understanding of risk factor evaluation in the diagnosis of coronary heart disease suspected patients.

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CHAPTER FOUR: RESULTS AND DISCUSSION

Background

The primary objective of the current study was to determine if periodontal disease was associated with individuals who had a recent myocardial infarction (between February 2001 and February 2002). That is, do individuals who have had a recent myocardial infarction have greater odds of also having periodontal disease?

The current study utilized four periodontal indicators to evaluate an individual's periodontal disease status. These measures included, a count of the number of teeth present, a measure of gingivitis based on Morrison et al. 's research study in 1999, the average probing depth (six sites- medial and distal), and the individual's BANA test score. These four measurements are consistent with previous literature that has evaluated periodontal disease and/or oral health (Beck, Garcia, Heiss, Vokonas and Offenbacher, 1996). The composite of the measures was thus considered representative of the individual's oral health, specifically periodontal disease.

Sample

The study was compromised of 29 males and 13 females. All individuals were over the age of fifty-five at the start of the study.

Displayed below, in Table 2, is a summary of results from the data abstraction. There is a discussion, which follows the summary table. The discussion highlights each individual variable studied.

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Table 2
Odds Ratio, and Confidence Intervals (95%)

MI and PD	No. of Participants	Effect (Odds Ratio& CI)
MI and PD	42	0.38 [0.11,1.31]
MI and Gignival Assessment	42	5.87 [0.60, 57.79]
MI and Probing Depth	42	2.59 [0.22, 30.99]
MI and No. Of teeth	42	0.85 [0.25, 2.90]
MI and BANA	42	0.07 [0.00, 1.46]
Explanatory Factors and MI	**No. of Participants**	**Effect (Odds Ratio& CI)**
CRP and MI	40	1.31 [0.37, 4.64]
Fibrinogen and MI	37	1.07 [0.23, 4.84]
Lifestyle and MI	42	0.59 [0.17, 2.06]
Explanatory Factors and PD	**No. of Participants**	**Effect (Odds Ratio& CI)**
CRP and PD	40	0.97 [0.28, 3.40]
Fibrinogen and PD	37	0.14 [0.02, 0.79]
Lifestyle and PD		2.75 [0.77, 9.86]
CRP and Gingival	40	1.12 [0.17, 7.60]
CRP and Probing Depth	40	1.52 [0.13, 18.33]
CRP and No. of teeth	40	0.60 [0.16, 2.16]
CRP and BANA	40	1.40 [0.18, 11.08]
Fibrinogen and Gingival	37	0.75 [0.07, 7.73]
Fibrinogen and Probing Depth	37	0.38 [0.02, 8.14]
Fibrinogen and No. of teeth	37	0.20 [0.04, 1.00]
Fibrinogen and BANA	37	0.27 [0.03, 2.27]

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Table 3
Frequencies

FREQUENCIES			
EVENTS	FREQUENCY		TOTAL
	YES	NO	
MI in past 12 months	19	33	42
Probing Depth Scores	3	39	42
Gingival Scores	5	37	42
Number of Teeth Present	23	19	42
BANA Test	38	4	42
FANTASTIC Lifestyle Scores	38	4	42
C-reactive Protein	23	17	40
Fibrinogen	9	26	37

Gingivitis

As part of the total periodontal disease score individuals were rated for degree of gingivitis. In the present study gingivitis was measured as none, mild, or severe. For the purpose of this study, mild gingivitis was defined as obvious inflammation in the free gingiva of one or more tooth area where the inflammation did not completely surround the tooth. Severe gingivitis was defined as marked redness, swelling, tendency to bleed and ulceration which may or may not have extended around the tooth. These definitions are consistent with Morrison et al. (1999). A mean score from the six sites determined the final score. If an individual's average score was less than or equal to two they were identified as positive for this indicator (gingival score) and if the average score was greater than two the individual was identified as negative for this indicator.

The current study identified an odds ratio of 5.87, for degree of gingivitis and myocardial infarction. The confidence interval ranged from 0.60, 57.29 at 95%. The odds ratio is not significant because the confidence interval included one. Although the odds ratio is not significant we can see that there is a positive trend between the gingival

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score and myocardial infarction. A larger sample size may increase power, and change the relationship.

Morrison et al. (1999) reported an association between periodontal disease and risk of fatal coronary heart disease. In Morrison et al.'s (1999) study a rate ratio of 2.15 was observed for severe gingivitis and coronary heart disease, suggesting a positive association with coronary heart disease. The current study reported similar results in that individuals with severe gingivitis will have an increased chance of experiencing a myocardial infarction. Both the current study and Morrison et al. (1999) agree that poor dental health, including periodontal disease, is associated with an increased risk of coronary heart disease and coronary heart disease risk factors.

Probing Depth

The average probing depth for six sites (if appropriate), medial and distal points, was calculated for each individual participating in the current study. Teeth numbered 16, 21, 24, 36, 41, and 44 were used in the calculation; two sites were measured for each tooth. Probing depth was measured in millimeters and was classified as none (less than two millimeters), mild (greater than two millimeters and less than five millimeters) or severe (greater than five millimeters). These measures/tools were consistent with Beck, Garcia, Heiss, Vokonas and Offenbacher (1996). The current study identified no relationship between probing depth and myocardial infarction. The odds ratio was reported at 2.59 and was not significant (the confidence interval included one). The confidence interval was reported at 0.22, to 30.99, at 95%. In the current study, 39 individuals (93%) were identified as negative when their average score for probing depth was calculated. These 39 participants had an average probing depth score less than four mm, thus suggesting good periodontal health (as opposed to poor). The relationship

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between myocardial infarction and average probing depth score is opposite from what was expected. It was expected that positive probing depth scores (average score greater than four millimeters) would have been observed between myocardial infarction and average probing depth scores.

Several individuals in the current study had zero teeth or a partial denture. Those participants with zero teeth and those with a partial denture may have skewed the results because it is not possible to measure an individual's probing depth if there are no teeth present.

In order to calculate a more accurate score, more specification could be used when calculating the average probing depth. For example, more sites (teeth) could be used and more sites per tooth. In this study six sites (teeth) were measured and two sites per tooth, (mesial and distal point were used). It is possible to probe the cheek side (buccale), the tongue side (lingual), and/or the mesial point of each tooth. Including more measures will increase the accuracy of the calculation and will consequently increase the time and cost of the tests.

Beck (1996) identified a relationship between clinical probing depth (greater than 3 mm) and coronary heart disease (OR=1.5) with confidence interval reported at 1.06; 2.15 at 95%. In Beck's (1996) study, a relationship was identified between total coronary heart disease and less than half of the mouth with teeth. The odds ratio was identified at 2.2 with confidence interval reported at 1.25; 4.01 at 95% (Beck, 1996). Perhaps by changing the scoring of probing depth in the current study significant results may have been observed.

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Count of the Number of Teeth

Each individual participating in the study consented to a panoramic x-ray. The panoramic x-ray allowed the dental hygienist to count the number of teeth present. The count of teeth was confirmed with the intra oral inspection. There was no relationship reported between the count of teeth and myocardial infarction in the current study. The odds ratio was identified at 0.85 with confidence interval reported at 0.25, 2.90 at 95%. The odds ratio was not significant as the confidence interval includes one.

According to Paunio (1993) individuals with fewer teeth were reported to have an increase in the prevalence of coronary heart disease. Individuals with at least half of their teeth missing doubled their prevalence of coronary heart disease (Paunio, 1993). The prevalence of heart disease doubled in individuals with at least half their teeth missing from 10% to 20% (Paunio, 1993).

Joshipura et al. (1996) also reported an association between tooth loss and coronary heart disease. The association is identified in men who reported a periodontal disease history. Men with zero to ten teeth reported a relative risk of 1.67, with confidence interval of 1.03; 2.71, at 95%. Joshipura et al. (1996) reported a higher incidence of coronary heart disease (in men) with less than ten teeth, compared to men with an intact dentition.

The current study is in agreement with Paunio (1993) and Joshipura et al. (1996) in that an association was reported between the number of teeth and myocardial infarction. The current study does not show a positive relationship, however there is a positive trend between the number of teeth and myocardial infarction. These studies are in agreement that number of teeth is positively associated with myocardial infarction profile.

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Both tooth loss and coronary heart disease risk can be reduced through lifestyle modifications. Thus, Joshipura et al. (1996) has suggested that people who take good care of their dentition may be at lower risk for coronary heart disease. Good oral health and a lower risk for coronary heart disease can be achieved through healthy behaviours (Joshipura et al., 1996).

As well, tooth loss may be a marker for more severe periodontal measures (Joshipura et al.,1996). In the current study data regarding dental caries was not collected. Therefore we cannot know whether teeth were extracted due to caries or external conditions ie. Accident. However, Paunio et al., (1993) stated that in older age groups the main cause for tooth extractions is periodontitis.

The BANA Test

The BANA test is used to detect Bacteroides gingivalis, Bacteroides forsythus and Treponema denticola. Several individuals who participated in the study did not have teeth, thus the bacteria sample was taken from the tongue. A biofilm sample is taken from the tongue and is then applied to the PERIOSCAN reagent strip. The PERIOSCAN reagent strip will then report if Bacteroides gingivalis, Bacteroides forsythus and Treponema denticola are present in the mouth. It is recommended that the sample be taken from each individual tooth. This would allow the researcher to identify which tooth/teeth are diseased.

In the current study the BANA test was used to determine if a relationship was present between myocardial infarction and the BANA scores. An odds ratio of 0.07 was reported in the current study. The odds ratio was not significant as the confidence interval included one. The confidence interval was reported at 0.00, 1.46 at 95%. In a

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future study it is recommended that the sample be taken from the teeth, rather than the tongue.

Total Periodontal Disease Score

The Total Dental Index (TDI) is a system in which various dental conditions are arbitrarily assigned scores, and the sum of the scores are used to establish the severity of the dental infection. Mattila et al. (2000) used the TDI to compare individuals with coronary heart disease (coronary heart disease without myocardial infarction, coronary heart disease with myocardial infarction, or recent myocardial infarction) and matched controls. In Mattila's et al. (2000) study the results were not statistically significant, however the dental indices were higher in each of the coronary heart disease categories. In 1989 and 1995 Mattila et al. used the TDI and reported periodontal disease to be more common among patients with myocardial infarction than the controls.

The total periodontal score in the current study resembled the TDI used by Mattila et al. (2000). The current study was not able to use the TDI because the tests were inappropriate ie. this study did not have access or funds for the tools and professionals that Mattila et al. (2000) used. The TDI is comprised of caries, periodontitis, periapical lesions and pericoronitis. As well, the TDI is not validated. The individuals are provided with a score of one to ten, increasing with the severity of the disease. The periodontal disease tests used in the current study were more feasible for this study than Mattila et al.'s (2000) index.

An odds ratio of 0.38 was reported and was not significant for the relationship between myocardial infarction and total periodontal score. The confidence intervals were reported at 0.11. 1.31 at 95% confidence. The confidence interval included one thus the odds ratio is not significant.

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In summary, the degree of gingivitis, probing depth, number of teeth and the BANA scores were not related to myocardial infarction in the current study, as all odds ratios were not significant.

FANTASTIC Lifestyle Questionnaire and the Coronary Heart Disease Profile

The FANTASTIC Lifestyle Questionnaire provided a score out of 100 in regards to the participant's lifestyle (healthy being 100). The nine areas measured from the questionnaire were family and friends, activity, nutrition, tobacco, alcohol, sleep, type of behaviour, insight and career. The FANTASTIC Lifestyle Questionnaire was used to determine a relationship between lifestyle and myocardial infarction.

The current study reported no relationship between the FANTASTIC Lifestyle Questionnaire and myocardial infarction. The odds ratio was reported at 0.59 and was not significant, as the confidence interval included one. The confidence interval was reported at 0.17, 2.06 at 95%.

Individuals are often informed by general practitioners to improve their physical health, i.e. to quit smoking, reduce alcohol and follow a healthy eating plan. Thus, it is expected to see a healthy (high score) FANTASTIC Lifestyle Questionnaire Score in those individuals who have experienced a myocardial infarction in the past year. These participants have recently visited the doctor and have potentially worked on modifying their lifestyle. The healthy (high) FANTASTIC Lifestyle Questionnaire scores may be a result of this behaviour, which is often encouraged by physicians.

FANTASTIC Lifestyle Questionnaire and Periodontal Disease

The current study reported a positive relationship between the FANTASTIC Lifestyle Questionnaire and periodontal disease. The odds ratio was reported at 2.8 and

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was significant, as the odds ratio does not include one. The confidence interval was reported at 1.46; 4.04 at 95%.

A positive relation between the questionnaire and periodontal disease was reported. Thus suggesting that those participants with a healthy lifestyle are likely to experience periodontal disease. The positive relationship between the questionnaire and periodontal disease speaks to the lack of specificity of these two tests being joined together. In other words it is not logical that those who score healthy would score high for periodontal disease. The problem may be that the tests are inappropriate. The questionnaire is broad and covers nine areas of health in a one-page questionnaire. It would be more appropriate to have a specific questionnaire, which would focus on specific risk factors, i.e. smoking or physical activity. It is suggested that a questionnaire be used which was created for cardiac purposes.

C-reactive Protein, Fibrinogen and Coronary Heart Disease

The odds ratio in the present study was reported at 1.07 and was not significant (confidence interval includes one) between fibrinogen and myocardial infarction. The confidence intervals were reported at 0.23, 4.84 at 95% confidence.

The current study set out to see an association similar to Toss, Lindahl, Siegbahn and Wallentin's (1997). Toss et al., (1997) studied the influences of increased fibrinogen and c-reactive protein levels and unstable coronary artery disease and myocardial infarction. Toss et al., (1997) investigated the prognostic influences of fibrinogen and c-reactive protein on levels of myocardial infarction and death in patients with coronary artery disease. The results of Toss et al.'s (1997) study reported that mean fibrinogen levels were significantly higher in participants who had died or had a new myocardial infarction during the follow-up period. The current study trends towards support

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however, we did not find a significant association. Both studies reported similar results in that fibrinogen levels were associated with an increased risk of experiencing a myocardial infarction.

Toss et al., (1997) also reported a significantly higher median score for c-reactive protein in patients who died during the follow-up period. The odds ratio for c-reactive protein and myocardial infarction was reported at 1.31 and was not significant. The confidence intervals were reported at 0.37, 4.64 at 95%. Thus, displaying a positive trend between c-reactive and myocardial infarction.

Ridker et al., (1997) measured c-reactive protein as a marker for systemic inflammation in participants (men) who had experienced a myocardial infarction, stroke, or venous thrombosis. Ridker et al., (1997) reported the levels of c-reactive protein were significantly higher among those who had experienced a myocardial infarction, stroke, or venous thrombosis. C-reactive protein was stratified into four quartiles. The four quartiles reported a relative risk of 1.0, 1.7, 2.6, and 2.9, respectively. Moreover, the current study is in agreement with Ridker et al., (1997) that c-reactive protein levels are increased in individuals who have experienced a myocardial infarction.

Woodward, Lowe, Tunstall-Pedoe (1998) measured fibrinogen to determine if there was a relationship between fatal and non-fatal coronary heart disease, with a follow-up of eight years. Coronary heart disease in Woodward et al.'s study included coronary death, non-fatal infarction and coronary artery surgery. In Woodward et al.'s study fibrinogen increased with severity of coronary heart disease. The current study is in unison with Woodward et al.'s in that those with a higher fibrinogen levels appear to have a greater chance of experiencing coronary heart disease.

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C-reactive Protein, Fibrinogen and Periodontal Disease

In the current study a no association was reported between c-reactive protein and periodontal disease as determined by the odds ratio (less than one). The odds ratio was reported at 0.97 and was not significant (confidence interval includes one). The confidence interval was reported at 0.28, 3.40 at 95%. However, there is a positive trend between c-reactive protein and periodontal disease. Perhaps with a larger sample size (more power) they would have been different results.

In the current study no association was reported between fibrinogen and periodontal disease (odds ratio less than one). The odds ratio was reported at 0.14 and was not significant. The confidence interval was reported at 0.21, 0.79 at 95%.

Noack, Genco, Trevisan, Grossi, Zambon, and De Nardin (2001) conducted a study to examine if there is a relation to severity of periodontal disease and c-reactive protein levels in 174 participants. Periodontal disease was measured similar to the current study (similar tools). Noack et al., (2001) used probing depth measurements, percentage of pocket loss greater than five, and clinical attachment loss. The presence of periodontal pathogens was also measured. Periodontal pathogens included were, Prevotella intermedia, Campylobacter recta, and Bacteroides forsythus. They were measured by immunofluorescence microscopy.

Noack et al., (2001) reported a statistically significant association between periodontal disease and increased levels of c-reactive protein. Participants with periodontal disease and higher levels of mean clinical attachment loss had significantly higher mean c-reactive protein levels than controls (Noack et al., 2001). The presence of periodontal pathogens in subgingival samples was positively related with elevated c-reactive protein levels. The current study is consistent with Noack et al., (2001) in that

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both the BANA score and c-reactive protein levels reported a positive relationship with periodontal disease. The current study is in agreement with Noack et al., (2001) in that the positive correlation between c-reactive protein levels and periodontal disease might be a possible underlying pathway in the association between periodontal disease and the observed higher risk for coronary heart disease.

Slade, Offenbacher, Beck, Heiss, and Pankow (2000) also examined c-reactive protein levels and periodontal disease. Slade et al. (2000) reported that individuals with periodontal disease (more than 10% of sites with periodontal pockets of four plus mm) had an increase of one-third in mean c-reactive protein. C-reactive protein levels were also higher in edentulous people (Slade et al., 2000). The current study is in agreement with Slade et al.'s (2000) study in that there was an increase in c-reactive protein levels with individuals who had positive periodontal disease.

A study by Abou-Raya, Naeem, Abou-El Kheir, Beltagy and Egypt (2002) evaluated the association between periodontal disease and coronary heart disease. Abou-Raya's et al. (2002) study was similar to the current study in that both studies used similar tests to measure periodontal disease. The difference was that the current study focused on coronary heart disease and Abou-Raya et al. (2002) targeted coronary artery disease. The sample size, and the age of the participants were similar in both studies. There were 50 participants (male and female) in Abou-Raya et al. (2002) study and the mean age was 63.78.

In Abou-Raya's et al. (2002) study periodontal disease was measured by gingival bleeding, periodontal pocket depths, loss of teeth, calculus, and attachments levels. The total score was summed and increased with the severity of disease similar to the current study. Abou-Raya et al. (2002) demonstrated an association between c-reactive protein

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and fibrinogen levels in individuals with periodontal disease and individuals with coronary artery disease.

In the current study c-reactive protein and fibrinogen were both not associated with periodontal disease total score. The odds ratio was reported at .97. There was a positive trend in the relationship between these two proteins and periodontal disease. As well, it is important to note that the periodontal disease indicators did show a positive trend with c-reactive protein. C-reactive protein and the gingival assessment displayed an odds ratio of 1.12 (not significant), with a confidence interval of 0.17, 7.60 at 95%. Probing depth measurements and c-reactive protein also displayed a positive relationship, with an odds ratio of 1.52. The confidence interval ranged form 0.13, 18.33 at 95% (not significant). The odds ratio for c-reactive protein and the BANA scores displayed an odds ratio of 1.4 (positive), with a confidence interval of 0.18, 11.08 (not significant). The relationship between c-reactive protein and count of teeth reported an odds ratio of 0.6, and a confidence interval ranging from 0.16, 2.16 (not significant). Although none of the periodontal disease indicators reported a positive, significant odds ratio there was an observed positive trend in the relationship between c-reactive protein and periodontal disease.

As well, fibrinogen did show a positive trend with the periodontal disease indicators, however none of the relationships were positive. Once again, the gingival indicator and fibrinogen indicator displayed a strong trend towards a positive association. The odds ratio for the gingival assessment and periodontal disease was reported at 0.75 with a confidence interval of ranging from 0.07, to 7.73 at 95%. The odds ratio for fibrinogen and probing depth was reported at 0.38 with the confidence interval spanning from 0.02, to 8.14 at 95%. The odds ratio was reported at 0.27 for the relationship of

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fibrinogen and the BANA scores, with a confidence interval of 0.03, 2.27 at 95%. The odds ratio was reported at 0.20 for the relationship of fibrinogen and number of teeth, with a confidence interval of 0.04, 1.00 at 95%.

Summary of Expectations

The biological link between periodontal disease and coronary heart disease is not yet clearly established, however there is a plausible mechanism which links the two disease. Individuals with periodontal disease have been observed with high fibrinogen levels and/or c-reactive protein levels (Abou-Raya et al., 2002). Such increases in fibrinogen and/or c-reactive protein may promote atherosclerosis and thrombosis, which can lead to coronary heart disease and/or a myocardial infarction (Abou-Raya et al., 2002). Specifically, this may lead to platelet aggregation (Abou-Raya et al., 2002).

As well, Loeche (2000) has suggested a biological link between periodontal disease and coronary heart disease. Loeche (2000) suggested that the plaque from oral disease may enter the blood stream and be present as atheromatous plaques. Thus suggesting the direct involvement of periodontal bacteria in the atheroma processes. Loeche (2000), is in agreement with Abou-Raya et al., (2002) stated that c-reactive protein and/or fibrinogen can be elevated from the bacteria from periodontal disease. Moreover, c-reactive protein and/or fibrinogen interact with the particles involved with blood clotting and may contribute to the formation and progression of atherosclerotics plaques and may increase the likelihood of experiencing a myocardial infarction (Abou-Raya et al., 2002; El-Sayed, 1996; Kannel et al., 1987; and Toss et al., 1997). The interactions of c-reactive protein and/or fibrinogen may be critical in determining the association between coronary heart disease and periodontal disease.

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As well, Offenbacher et al. (1999) has discussed the relationship between periodontal disease and coronary heart disease and suggested that both diseases share a common background. For example smoking, nutrition, and diabetes may increase the likelihood of developing both diseases. Additionally, both diseases are chronic and multifactorial and may potentially share common etiology pathways (Offendacher et al., 1999). Offenbacher et al. (1999) is also exploring the possibility that there are susceptible genes which may be inherited and place certain individuals at risk for both a myocardial infarction and periodontal disease.

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Recommendations for Future Research

One limitation of this research project was the difficulty of recruiting people who had a recent myocardial infarction. Originally, it was intended that recent myocardial infarction would be defined as an individual who experienced a myocardial infarction within six months (not twelve months) of data collection. It was difficult to recruit participants who had experienced a myocardial infarction in that time period and expect them to participate in a research study. Thus, defining a recent myocardial infarction to 12 months was necessary in order to recruit the required number of people. One method of correcting this problem, in the future, would be to have a cardiac nurse, from the corresponding hospital, on the research committee from the start of the project. The cardiac nurse(s) involvement could potentially assist in the recruitment of potential patients from the Hospital as soon as they experience a myocardial infarction. The cardiac nurse may help to increase participant compliance because the nurse may have a better rapport with the patients than a researcher and/or stranger.

Patient recruitment from the Hospital would be ideal to increase the sample size of the project, particularly individuals who have experienced a recent myocardial infarction. It is difficult to recruit individuals who have experienced a recent myocardial infraction. There are a limited number of individuals who experience a myocardial infarction in one year who reside in Thunder Bay. Thus, by including more hospitals or more doctors the sample size could increase and inclusion and exclusion criteria may become stricter. For example, it becomes more necessary to include more locations or more doctors into the study if the researcher were to focus on one gender. In future research it is being recommended to invite several hospitals from all over Ontario to be

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involved in the study. This will increase the likelihood of recruiting people who have had a recent myocardial infraction (past week). The definition of a recent myocardial infarction may be able to be identified as one week if a cardiac nurse(s) were involved in the study.

A tighter definition of a recent myocardial infarction may increase the accuracy of the results. It would not be advised that the periodontal tests are done immediately after a person has a myocardial infarction, however the sooner the periodontal tests are completed, in conjunction with the myocardial infarction, the more accurate the results will be.

Defining a recent myocardial infarction, for example, as one week rather than one year may also increase the levels of accuracy of c-reactive protein and/or fibrinogen. C-reactive protein and/or fibrinogen are at their highest immediately after a myocardial infarction and may not be accurate after one year. Therefore a recommendation for future research is to measure the levels of c-reactive protein and/or fibrinogen immediately after the myocardial infarction (with one week).

Another recommendation for future research is to re-consider two of the periodontal indicators; the count of teeth and the BANA test. The count of teeth may not be the best indicator because the researcher is uncertain how the individual lost their teeth (ie. accident, disease, or financial reasons). As well, if the BANA test is repeated the sample should be take from the teeth being studied rather than the tongue. Samples taken from the teeth will provide more accurate results. It is suggested that a sample should be taken from each site (tooth), as compared to one site, the tongue. If the sample is taken from the teeth the researcher can indicate if the bacteria is in one or all of the sites (teeth). Whereas, by taking the sample from the tongue is not as specific.

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As well, if this study was to be repeated with a larger sample size the researcher should reconsider which questionnaire to use. Perhaps a questionnaire, which is created to measure specific factors or a questionnaire created specifically for cardiac events, would be ideal. Lastly, a case match study would be ideal to ensure that all groups are equivalent.

Implications for Future Research

Based on the results of this study, the following recommendations are made for future research:

1. Change periodontal disease indicators; specifically count of teeth and the BANA test. The sample for the BANA test should be taken from the teeth rather than the tongue.
2. Tighten definition of myocardial infarction.
3. A different Lifestyle questionnaire to focus on specific factors would be ideal.
4. Consider a case match study to ensure all groups are equivalent.
5. Increase the sample size.

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Summary

The present pilot study was useful and was able to provide an excellent starting point for future research between myocardial infarction and periodontal disease. This was the first study to take place in Northern Ontario, specifically Thunder Bay, which focused on the association between myocardial infarction and periodontal disease. Suggestions have been provided in this document to assist any future work in this area. These suggestions are based on the learning experiences from the data abstraction processes.

Although there was no positive association(s) seen in this Northern sample between myocardial infarction and periodontal disease this study was a great contribution to community health and specifically, the community of Thunder Bay, as we did see a positive trend between the two diseases. Perhaps with a larger sample size the results would prove to be different. With an increase in participants we can assume there would be more power. The researchers however, were able to document a positive trend in the association between c-reactive protein and fibrinogen and 1) myocardial infarction and 2) periodontal disease. These positive trends are important to public health professionals. C-reactive protein and/or fibrinogen are currently noted as mechanisms, which may link myocardial infarction and periodontal disease together. Moreover, the current results have supported previous literature suggesting the link between myocardial infarction and periodontal disease may be the presence of c-reactive protein and/or fibrinogen.

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APPENDIX A (INFORMED CONSENT)

Dear Participant:

Student researchers, Cindy Morash, Ryan Rankka and Joe Ellard, under the direction of Dr. Bill Montelpare and Dr. Chris Lai, are conducting a study to determine the relationship between coronary heart disease and periodontal disease. Our research is investigating the possible inflammatory role of c-reactive protein, cholesterol and fibrinogen on this suggested relationship.

The purpose of this research is to measure the suggested relationship in a group of individuals who have had a heart attack and a group of individuals who have not.

If you agree to participate in this study, we will require you to complete a questionnaire concerning your lifestyle and specific health related behaviours (see attached). We expect that the completion of the questionnaire will take about 15 minutes. In addition, you will also be asked to provide a blood sample so that we can determine the concentration, c-reactive protein, cholesterol, and fibrinogen in your blood. The required blood sample will be drawn by an approved technician and the blood sample will be processed at Lakehead University. There may be some discomfort and/or bruising with the blood sampling.

The final step of this research study will be to participate in a dental assessment, which includes a dental x-ray, and an assessment of gingivitis and periodontal disease. The dental assessment will take about 30 minutes. You may experience some discomfort during the dental assessment.

There will be no direct benefit from this participating in this research study. However, your participation will benefit research in heart disease and periodontal disease in your community. Your participation is greatly appreciated.

All information you provide will remain confidential and securely stored at Lakehead University for the recommended time limit of seven years. The findings of this project will be made available to you, at your request, upon completion of the project.

If you have any concerns about your rights as a research participant you may contact
Mary Jane Kurm at (807) 346-3413

If you have any additional questions, comments, or concerns please contact
Dr. William J. Montelpare, Ph. D.
School of Kinesiology, Lakehead University
(807) 343-8481 (office)
(807) 346-7749 (fax)

Thank you for your assistance.

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APPENDIX B (INFORMED CONSENT)
INFORMED CONSENT: A PILOT STUDY TO MEASURE THE RELATIONSHIP BETWEEN CORONARY HEART DISEASE AND PERIODONTAL DISEASE

Please initial each item	
	I have read the letter of informed consent and I understand all of the tasks that I will be required to complete.
	I have had a chance to answer questions throughout the introduction and description of this study.
	I agree to participate in this study freely.
	I understand that I can withdraw from this study at any point even after the consent forms have been signed.
	I understand that there will be no direct financial benefit from participating in the research study.
	I understand that I will receive a copy of my results at the end of this study.

Signed: _____ Witness: _____

Date: _____

This research project has been approved by both the Ethics Committees of the Thunder Bay Regional Hospital and Lakehead University.

If you have any concerns about your rights as a research participant you may contact
Mary Jane Kurm at (807) 346-3413

If you have any additional questions, comments, or concerns please contact
Dr. William J. Montelpare, Ph. D,
School Of Kinesiology, Lakehead University
(807) 343-8481 (office), (807) 346-7749 (fax)

Thank you for your assistance, Cindy Morash

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APPENDIX C (ID PAGE)

NAME: _____

ADDRESS: _____

_____

_____

HAVE YOU EVER HAD A MI (HEART ATTACK)? YES ☐ NO ☐

IF YES, WHEN? _____

DAY MONTH YEAR

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APPENDIX D (PERIODONTAL RECORD)

NAME: _____

DATE: _____

PROBING DEPTH	BACK	FRONT
TOOTH # 16		
#21		
#24		
#36		
#41		
#44		

GINGIVAL SCORE (SCORE)_____

0 HEALTHY
1 MILD, NO BLEEDING ON PROBING
2 MODERATE INFLAMMATION, BLEEDING ON PROBING
3 SEVERE INFLAMATION

BANA SCAN

NEGATIVE POSITIVE

PASTE BANA SCAN HERE

MISSING TEETH:

_____
_____
_____
_____
_____
_____
_____

COMMENTS:

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FANTASTIC LIFESTYLE CHECKLIST

Instructions: Unless otherwise specified, place an 'X' beside the box that best describes your behavior or situation in the past month. Explanations of questions and scoring are provided on the next page.

Family Friends	I have someone to talk to about things that are important to me	almost never	seldom	some of the time	fairly often	almost always
	I give and receive affection	almost never	seldom	some of the time	fairly often	almost always
Activity	I am vigorously active for at least 30 min per day (e.g., running, cycling, etc.)	less than once a week	1-2 times/ week	3 times/week	4 times/ week	5 or more times/week
	I am moderately active (gardening, climbing stairs, walking, housework)	less than once a week	1-2 times/ week	3 times/week	4 times/ week	5 or more times/week
Nutrition	I eat a balanced diet (see explanation, page 43)	almost never	seldom	some of the time	fairly often	almost always
	I often eat excess: (1) sugar, or (2) salt, or (3) animal fats, or (4) junk foods	four of these	three of these	two of these	one of these	none of these
	I am within _____ kg of my healthy weight	not within 8 kg (20 lb)	8 kg (20 lb)	6 kg (15 lb)	4 kg (10 lb)	2 kg (5 lb)
Tobacco Toxics	I smoke tobacco	more than 10 times/week	1-10 times/ week	none in the past 6 months	none in the past year	none in the past 5 years
	I use drugs such as marijuana, cocaine	sometimes				never
	I overuse prescribed drugs or 'over the counter' drugs	almost daily	fairly often	only occasionally	almost never	never
	I drink caffeine-containing coffee, tea, or cola	more than 10 times/week	7-10/day	3-6/day	1-2/day	never
Alcohol	My average alcohol intake per week is _____ (see explanation, page 43)	more than 20 drinks	13-20 drinks	11-12 drinks	8-10 drinks	0-7 drinks
	I drink more than four drinks on an occasion	almost daily	fairly often	only occasionally	almost never	never
	I drive after drinking	sometimes				never
Sleep	I sleep well and feel rested	almost never	seldom	some of the time	fairly often	almost always
Seatbelts	I use seatbelts	never	seldom	seldom	most of the time	always
Stress	I am able to cope with the stresses in my life	almost never	seldom	seldom	fairly often	almost always
	I relax and enjoy leisure time	almost never	seldom	seldom	fairly often	almost always
Safe sex	I practice safe sex (see explanation, page 43)	almost never	seldom	seldom	fairly often	always
Type of behavior	I seem to be in a hurry	almost always	fairly often	fairly often	seldom	almost never
	I feel angry or hostile	almost always	fairly often	fairly often	seldom	almost never
Insight	I am a positive or optimistic thinker	almost never	seldom	seldom	fairly often	almost always
	I feel tense or uptight	almost always	fairly often	fairly often	seldom	almost never
	I feel sad or depressed	almost always	fairly often	fairly often	seldom	almost never
Career	I am satisfied with my job or role	almost never	seldom	seldom	fairly often	almost always

Step 1 Total the X's in each column	→	☐	☐	☐	☐	☐
Step 2 Multiply the totals by the numbers indicated (write answers in box below)	→	0	×1	×2	×3	×4
Step 3 Add your scores across bottom for your grand total	→		☐ +	☐ +	☐ +	☐ = ☐ Grand total (see explanation)

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▼ A balanced diet:

According to Canada's Food Guide to Healthy Eating (for people four years and over):

Different People Need Different Amounts of Food

The amount of food you need every day from the four food groups and other foods depends on your age, body size, activity level, whether you are male or female, and if you are pregnant or breast feeding. That's why the Food Guide gives a lower and higher number of servings for each food group. For example, young children can choose the lower number of servings, while male teenagers can select the higher number. Most other people can choose servings somewhere in between.

Grain products	Vegetables & fruit	Milk products	Meat & alternatives	Other foods
Choose whole grain and enriched products more often.	Choose dark green and orange vegetables more often.	Choose lower fat milk products more often.	Choose leaner meats, poultry and fish, as well as dried peas, beans, and lentils more often.	Taste and enjoyment can also come from other foods and beverages that are not part of the 4 food groups. Some of these are higher in fat or calories, so use these foods in moderation.
recommended number of servings per day:				
5-12	5-10	Children 4-9 yrs: 2-3 Youth 10-16 yrs: 3-4 Adults: 2-4 Pregnant and breast-feeding women: 3-4	2-3	

▼ Alcohol intake:

1 drink equals:		Canadian	Metric	U.S.
1 bottle of beer	5% alcohol	12 oz.	340.8 ml	10 oz.
1 glass wine	12% alcohol	5 oz.	142 ml	4.5 oz.
1 shot spirits	40% alcohol	1.5 oz.	42.6 ml	1.25 oz.

▼ Safe sex:

Refers to the use of methods of preventing infection or conception.

What does the score mean?

85-100	70-84	55-69	35-54	0-34
Excellent	Very good	Good	Fair	Needs improvement

Note: A low total score does not mean that you have failed. There is always the chance to change your lifestyle—starting now. Look at the areas where you scored a 0 or 1 and decide which areas you want to work on first.

Tips:

1. Don't try to change all the areas at once. This will be too overwhelming for you.
2. Writing down your proposed changes and your overall goal will help you to succeed.
3. Make changes in small steps towards the overall goal.
4. Enlist the help of a friend to make similar changes and/or to support you in your attempts.
5. Congratulate yourself for achieving each step. Give yourself appropriate rewards.
6. Ask your physical activity professional, family physician, nurse, or health department for more information on any of these areas.

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APPENDIX F (ODDS RATIO)

The two by two table can be used to determine the odds of a particular event, in a case-control design. The odds ratio will measure association between the exposed and non exposed; weather the exposure, myocardial infarction, is associated with the disease, periodontal disease. The proportion of the cases that were exposed and the proportion of the controls who were not exposed will be discussed. Odds of an event are calculated as the number of events divided by the number of non-events. Odds ratios are always used in case-control studies where disease prevalence is not known: the apparent prevalence depends solely on the ratio of sampling cases to controls, which is artificial.

The odds ratio is defined as the ratio of the number of ways the event can occur to the number of ways the event cannot occur. The odds ratio will report the chance of an individual who has periodontal disease and a myocardial infarction. In other words, the potential of someone with periodontal disease having a myocardial infarction.

An odds ratio is calculated by dividing the odds in the exposed group by the odds in the control groups.

Odds ratio = odds that case was exposed / odds that a control was exposed

In a two by two table that is ad/bc.

If the exposure is not related to the disease, the odds ratio will equal 1. If the exposure is positively related to the disease, the odds will be greater than1. If the exposure is negatively related to the disease, the odds will be less than 1. As well, relative risk can be estimated from the odds ratio in a case-control design.

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