Alcohol intake and periodontal outcomes

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Thesis

ALCOHOL INTAKE AND PERIODONTAL OUTCOMES

by

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ALCOHOL INTAKE AND PERIODONTAL OUTCOMES

CHRISTINE CHIAO

ABSTRACT

Objective: Periodontal disease is a highly prevalent inflammatory disease with a wide range of causes and clinical manifestations. Excessive alcohol consumption is a significant public health problem, and is a risk factor for a variety of diseases; however, the relationship between alcohol intake and overall oral health remains unclear. This study seeks to identify the relationship between heavy alcohol consumption (consuming two or more drinks per day) and specific indicators of periodontal health.

Methods: Cross-sectional analysis utilized data from the Dental Longitudinal Study, a longitudinal study conducted at the Boston Veterans Affairs medical center, in which the initial cohort consisted of healthy male veterans residing in Greater Boston. Using demographic and behavioral information collected from surveys, and oral health data collected from clinical examinations, bivariate data analysis was conducted to compare periodontal health outcomes between those who drank less than two drinks per day (non-drinkers and moderate drinkers, n = 949) and those who drank two or more drinks per day (heavy drinkers, n = 237). Selected oral health outcomes for analysis were indicators of poor periodontal health and included: number of teeth, bleeding on probing, calculus and plaque levels, tooth mobility, alveolar bone loss, periodontal pocket depth measurements, and gingival recession.
**Results:** Results showed that the heavy alcohol consumption group was significantly associated with increased whole mouth mean alveolar bone loss and with periodontal pocket depths exceeding 4mm and 5mm, with a trend in heavy drinkers to have more teeth with increased levels of pocket depth.

**Discussion:** The detected association between heavy drinking and alveolar bone loss and pocket depth measurement is a significant clinical finding, and suggests that alcohol intake should be minimized in the interest of periodontal health. The results of this study point towards the need for future longitudinal studies to investigate the possible role of alcohol as a risk factor for periodontal outcomes.
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<tr>
<td>ABL</td>
<td>Alveolar bone loss</td>
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<td>ADA</td>
<td>American Dental Association</td>
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<tr>
<td>AIDS</td>
<td>Acquired immunodeficiency syndrome</td>
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<tr>
<td>BMI</td>
<td>Body mass index</td>
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<td>BOP</td>
<td>Bleeding on probing</td>
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<tr>
<td>BRFSS</td>
<td>Behavioral Risk Factor Surveillance System</td>
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<tr>
<td>CAL</td>
<td>Clinical attachment loss or clinical attachment level</td>
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<tr>
<td>CDC</td>
<td>Centers for Disease Control</td>
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<tr>
<td>CMI</td>
<td>Cornell Medical Index</td>
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<tr>
<td>DLS</td>
<td>Dental Longitudinal Study</td>
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<td>HIV</td>
<td>Human immunodeficiency virus</td>
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<td>NIAAA</td>
<td>National Institute on Alcohol Abuse and Alcoholism</td>
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<td>NIH</td>
<td>National Institute of Health</td>
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<td>PTSD</td>
<td>Post traumatic stress disorder</td>
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<tr>
<td>US</td>
<td>United States</td>
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<td>VA</td>
<td>Veterans Affairs</td>
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<td>WHO</td>
<td>World Health Organization</td>
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INTRODUCTION

Periodontal disease biology

*Periodontal disease*, also known as *gum disease*, is a common inflammatory
disease, caused by a combination of biological and environmental factors. It has been
found to be associated with diet, stress, and tobacco use (Pihlstrom, Michalowicz, & Johnson,
2005). Tobacco use has been found to be a major risk factor for periodontal disease,
possibly due to the effects tobacco on inflammatory mediators and vasculature
(Bergström, 2004; Johnson, 2001). While periodontal disease often develops on its own,
there are also many diseases with periodontal manifestations including diabetes,
leukemia, HIV/AIDS, psoriasis, herpes, lupus, Crohn’s disease, neutropenias, and others
(Pihlstrom et al., 2005). The term periodontal disease includes the milder form of the
disease, gingivitis, where there is inflammation of the gums, and periodontitis, where
inflammation has increased to affect the tissue surrounding the teeth itself (Pihlstrom et al.,
2005). There are many forms of periodontitis, with the most common being chronic
periodontitis, a slowly progressing condition in adults (Pihlstrom et al., 2005; Preshaw &
Taylor, 2012). Nonetheless, periodontal disease is a broad term and also includes the
following conditions: plaque-induced gingivitis, non-plaque induced gingivitis,
aggressive periodontitis, necrotizing or acute periodontal disease, periodontal abscesses,
and other periodontal deformities (Wiebe & Putnins, 2000).

In the oral cavity, microbial flora supports the build-up of plaque on teeth, which
can irritate the gums and cause inflammation. As the disease progresses, the gingiva, or
the gums, pull away from the teeth and the resulting space can become infected by
colonizing bacteria, and this infection can spread to surrounding tissue and bone which leads to increased loss of periodontal support. Over 300 different species of microbial pathogens have been found in periodontal pockets, and between 30 and 100 species may be found at a single site in one person (Haffajee & Socransky, 1994). Not only does there exist pathogens in both periodontal disease patients and disease-free individuals, but it is also difficult to discern the species that are found in different subtypes of periodontal disease (Haffajee & Socransky, 1994). This makes it difficult to identify specific microbial agents that cause periodontal disease. For this reason, periodontal disease is usually diagnosed by dentists based on clinical features instead of microbial presence (Loesche, 1996).

The histopathology of periodontal disease shows immune cell infiltration of connective tissue, in which the immune cells release inflammation inducing enzymes leading to destruction of collagen and proliferation of the local epithelial tissue (Preshaw & Taylor, 2012). As part of the inflammatory response, vasodilation occurs thus leading to the clinically apparent swelling of the gums (Preshaw & Taylor, 2012). Histologically, several stages of lesions occur. As the lesion progresses to an advanced lesion, the transition from gingivitis to periodontitis occurs with increased breakdown of connective tissue and inflammatory mediators activating pathways that lead to osteoclastic reabsorption of underlying bone (Preshaw & Taylor, 2012).

The disease itself manifests in symptoms of sensitive and swollen gums and a receding gum line, leading primarily to loosening of the teeth and eventual edentulism. Halitosis may also be present. Long-term consequences of edentulism include aesthetic
consequences and chewing difficulties, as well as the financial burden of dentures or implants. Additionally, the formation of infected pockets can foster microbial growth and thus promote the development of caries especially near the roots of teeth (Carranza & Camargo, 2012). There may be related systemic consequences as well, as recent studies have also found gum disease to be associated with a number of systemic health complications including adverse birth outcomes, cardiovascular outcomes, and diabetes (Humphrey, Fu, Buckley, Freeman, & Helfand, 2008; National Institute of Dental and Craniofacial Research [NIDCR], 2000; Taylor, 2001; Xiong, Buekens, Fraser, Beck, & Offenbacher, 2006).

While treatment varies with origin of periodontal deficiency, initial treatment of adult chronic periodontal disease generally involves non-surgical clinical techniques and patient education, with the goal to remove bacteria and calcified biofilms in order to restore periodontal health (Carranza & Takei, 2012; Claffey & Polyzois, 2008). The clinical therapy typically involves scaling and root planning to remove calculus, and if necessary it may also involve caries control, either local or systemic antimicrobial therapy, occlusal therapy (bite adjustment or use of night guards), correction of prosthetic irritational factors, or minor orthodontic movement (Carranza & Takei, 2012). Patient education aspects include diet control and oral hygiene instruction for home care to minimize disease progression. For many patients, individual behavioral change and maintenance of oral self-care procedures is a key component of successful periodontal disease treatment. In more severe cases of periodontal disease, surgical procedures such
as gingivectomy, flap procedures, tissue regeneration procedures, or osseous surgeries may be required (Wennström, Heijl, & Lindhe, 2008).

**Periodontal disease epidemiology**

Periodontal disease is a significant public health problem in the US, with over 47% of the adult population estimated to be affected by mild, moderate, or severe forms of the disease (Eke, Dye, Wei, Thornton-Evans, & Genco, 2012). This prevalence drastically increases to 64% in the elderly population (Eke et al., 2012). The WHO has identified older people as a target group due to their high oral health burden, with issues of periodontal health and edentulism with other comorbidities being of particular concern (World Health Organization [WHO], n.d.). Periodontal disease also disproportionately affects certain social groups, including males, non-Hispanic Blacks, Latinos, and those of lower socio-economic status and lower educational attainment (Eke et al., 2012). It is also highly prevalent on a global scale, with increased prevalence in developing nations, which often suffer from poorer oral health outcomes (Peterson & Ogawa, 2005).

However, estimates of periodontal disease vary widely across studies, in part due to the lack of a uniform methodology for measuring periodontal disease- such as varying methods to measure diagnostic criteria including inflammation, loss of periodontal support, alveolar bone loss, and other characteristics (Papapanou & Lindhe, 2008). This methodological issue not only contributes to inconsistency across existing epidemiological studies, but it also limits comparability between studies (Irfan, Dawson, & Bissada, 2001; Papapanou & Lindhe, 2008).
Clinical features of periodontal disease

Periodontal disease has a wide range of severity and manifests in a range of symptoms. *Plaque-induced gingivitis*, is a condition in which there is gum irritation and bleeding on probing, but no loss of periodontal support around the tooth or alveolar bone loss. Gingivitis can progress to the more severe forms of periodontitis if left untreated. In *chronic periodontitis*, there is not only bleeding upon probing but also increased pocket depths and loss of periodontal support and alveolar bone. Periodontal disease is most typically assessed using clinical attachment loss (CAL, also known as clinical attachment level), which is an estimate of the position of structures that stabilize and support the tooth (Nield-Gehrig, 2013). CAL is the most accurate estimation of the tooth’s periodontal support, and is measured by *probing pocket depth* and *gingival margin level* (Nield-Gehrig, 2013). Pocket depth is a measurement of the depth of pockets around the tooth due to loss of tissue between tooth and the gingiva (Loesche, 1996). Note that probing pocket depth alone does not suffice to diagnose periodontal disease because it fails to account for gingival changes due to swelling or recession, such as recession in the case of severe periodontitis (Nield-Gehrig, 2013).

There are several other pathological characteristics that may also be used to assess periodontal status and are important indicators of an individual’s gum health. Gingival recession may be an additional important factor for assessing periodontal disease severity, because with increased disease progression there is sometimes reduced probing pocket depth due to increased recession (Albandar & Rams, 2002). Gingival recession is an extremely common feature of periodontal disease and highly prevalent in the general
population. Gingival recession measurements are also taken into account when calculating clinical attachment loss. Epidemiological studies have estimated that over 50% of the adult population and 88% of the elderly population has gingival recession (Kassab & Cohen, 2003). There is a wide spectrum of causes of gingival recession, including aging, anatomical factors, physiological factors, microbial factors, trauma, and oral hygiene (Kassab & Cohen, 2003).

Alveolar crestal bone refers to the bone that holds the teeth in place (American Dental Association [ADA], 2014). It contains sockets that support the tooth roots and for periodontal fiber attachment, and thus is critical for maintaining periodontal stability. Alveolar bone loss (ABL) is a hallmark feature of chronic periodontitis (“Parameter on Chronic Periodontitis,” 2000), as its deterioration leads to loosening of the tooth from the periodontal socket and attachment loss (Nield-Gehrig, 2013). Although resorption of underlying bone occurs as a protective mechanism to prevent bacterial invasion of the bone, it has many negative consequences (Preshaw & Taylor, 2012). CAL, gingival recession, and ABL all also contribute to tooth mobility, which is indicative of loss of periodontal support surrounding the tooth.

Bleeding on probing (BOP) due to gingival inflammation is also commonly used as a predictor of periodontal health; it has been shown to have a strong negative predictive value, and when combined with other diagnostic techniques to have a moderate positive predictive value for periodontal disease (Claffey, Nylund, Kiger, Garrett, & Egelberg, 1990; Joss, Adler, & Lang, 1994; Lang, Adler, Joss, & Nyman, 1990).
Lastly, the buildup of calculus and plaque are important to take into account when looking at periodontal health. Calculus forms when plaque calcifies and hardens, creating hard deposits both sub-gingivally and supra-gingivally (White, 1997). Sub-gingival calculus co-occurs with periodontal disease, and in populations with poor oral hygiene, extreme calculus is associated with both gingival recession and attachment loss (White, 1997). Removal of sub-gingival calculus through scaling and root planning is used widely for periodontal therapy and has been shown to be successful in preventing periodontal disease progression (White, 1997). Together, these aforementioned features of periodontal disease serve as indicators for periodontal health and overall oral health, and can be used to diagnose and treat periodontal disease patients.

**Alcohol & oral health**

Excessive alcohol consumption is a risk factor for a wide array of medical conditions due to its harmful physiological and behavioral effects. These conditions range from acute to chronic conditions, including both mental health disorders and systemic illnesses. Alcohol consumption is among the leading causes of mortality in the US (Mokdad, Marks, Stroup, & Gerberding, 2004). Chronic alcoholism can have negative consequences on the central nervous system, liver, blood, heart, immune system, gastrointestinal tract, as well as developmental effects (Schreiber, 2001). Binge drinking episodes, defined as having 5 or more drinks on one occasion, is associated with a number of behavioral effects including increased injuries, violence, dangerous driving behaviors, and more (Wechsler, Davenport, Dowdall, Moeykens, and Castillo, 1994).
Nonetheless, in recent years there has been increasing scientific evidence discussing possible benefits of a moderate level of alcohol intake. Moderate drinkers have been found to have decreased all-cause mortality and lower risk for coronary heart disease compared to non-drinkers and heavy drinkers (Gaziano et al., 2000; Keil, Chambless, Döring, Filipiak, & Stieber, 1997; McElduff & Dobson, 1997; White, 1999). This may be explained by an anti-inflammatory effect of alcohol such as by reducing levels of C-reactive protein (Imhof et al., 2001).

In recent years, there has been increasing evidence supporting that oral health and systemic health are closely related. Naturally, it can thus be expected that alcohol be either directly or indirectly be related to a number oral health conditions. However, in general, there is a lack of studies on the mechanistic link between alcohol and gum health.

It has been reported that excessive alcohol consumption is among several behavioral risk factors for periodontal disease, along with tobacco use, unhealthy diet, and other lifestyle components (Peterson & Ogawa, 2005). There are several reasons for why excessive alcohol use may be correlated with oral health outcomes. Severe and chronic alcoholism sometimes manifests in poor self-care, thus poor periodontal outcomes in chronic alcoholics may be caused by poor oral hygiene habits (Novacek et al., 1995; Sakki, Knuuttila, Vipari, & Hartikainen, 1995; Schreiber, 2001). Indeed, studies have found alcoholics to exhibit increased dental caries, missing teeth, alveolar bone loss, periodontal disease, and tooth erosion (Enberg et al., 2001; Kranzler, Babor, Goldstein, & Gold, 1990).
The physiological effects of alcohol on bone may point towards the biological plausibility of a causal relationship between alcohol and periodontal health. Alveolar bone is the bone that holds the teeth by anchoring the periodontal fibers which teeth are attached to. Like any other bone in the body, alveolar bone is dynamic and undergoes constant remodeling and regulation by a complex interplay of nutritional, hormonal, vascular, mechanical, and genetic factors with the action of osteoblasts and osteoclasts (Clark, 2008; Kini & Nandeesh, 2012). One of the reasons why alveolar bone must undergo constant remodeling is to compensate for tooth movement and migration throughout life (Lindhe, Karring, and Araujo, 2008). In the human body, excess alcohol intake is believed to affect bones through its effects on several hormones that regulate bone, such as parathyroid hormone, calcitonin, vitamin D, estrogen, and other factors (Sampson, 1998). Previous research has shown that alcohol decreases osteoblast and osteoclast function, providing another possible mechanistic explanation for alcohol’s effect on decreasing bone formation (Sampson, 1998).

However, in general, studies investigating the association between drinking level and bone status have found mixed results (Sampson, 1998). Kanis et al. (2005) found heavy alcohol intake to be a risk factor for bone fracture when controlling for other related variables. Another study on elderly women found heavy alcohol intake to be related to increased bone loss (Hannan et al., 2000). On the other hand, moderate alcohol consumption was found protective for preserving spinal bone mineral density in women (Kröger, Tuppurainen, Honkanen, Alhava, & Saarikoski, 1994), and also associated with decreased fracture risk in elderly women (Felson, Zhang, Hannan, Kannel, & Kiel, 1995;
Hansen, Overgaard, Riis, & Christianson, 1991), and decreased verbal deformity in elderly women (Diaz, O’Neill, & Silman, 1997).

The possible effects of alcohol on alveolar bone can have important clinical implications because alveolar bone loss can contribute to attachment loss, leading to issues including edentulism and gingival recession. Alveolar bone fenestration or dehiscence can contribute to gingival recession (Kassab & Cohen, 2003), and with preexisting alveolar bone deficiencies, tooth brushing may mitigate recession by further inflammation of gingival tissue (Baker & Seymour, 1976; Wennström, Lindhe, Sinclair, & Thilander, 1987). Despite the biological plausibility of this relationship, there lacks a clear consensus on an association between alcohol intake and alveolar bone status, nor is there a known mechanistic pathway to explain this relationship.

A few epidemiological studies have found a relationship between alcohol and periodontal disease. Several cross-sectional studies have found that when controlling for oral hygiene status, alcohol consumption levels was associated with periodontal disease status (Amaral, Luiz, & Leão, 2008; Kim et al., 2014; Susin, Haas, & Albandar, 2014; Tezal, Grossi, Ho, & Genco, 2004; Tezal, Grossi, Ho, & Genco, 2001). There are even fewer longitudinal studies that investigate this relationship, and because most existing studies have been cross-sectional in design, the ability to determine causality in the association is limited (Kim et al., 2014; Tezal et al., 2004; Tezal et al., 2001). One Swedish longitudinal study found no conclusive relationship between alcohol consumption and periodontal disease (Jansson, 2008). However, a 2003 study using longitudinal data on male health professionals found a positive, dose-dependent
relationship between alcohol intake and periodontitis, in which men who drank over 1 glass a day had an 18-27% higher relative risk for developing periodontitis than non-drinkers (Pitiphat, Merchant, Rimm, & Joshipura, 2003).

**Aim and importance of present study**

The overall inconclusiveness of published literature on alcohol intake and oral health, and specifically periodontal disease, points towards the importance of increased studies to investigate this relationship. Americans consume large quantities of alcohol, with the CDC Behavioral Risk Factor Surveillance System (BRFSS) reporting over half of Americans drinking at all in the last month at the time of survey, 16.8% reporting binge drinking (five or more drinks per occasion for males, four or more drinks per occasion for females), and 6% being heavy drinkers as defined by the CDC BRFSS (average consumption of more than two drinks per day for men and more than one drink per day for women) (Center for Disease Control and Prevention [CDC], 2013). Yet the relationship between alcohol intake and oral health remains unclear. In recent years, with growing interest in the possible benefits of moderate alcohol consumption, it has become increasingly important to understand the specific risks and benefits associated with varying levels of drinking.

To better ascertain this relationship, we used data from the Dental Longitudinal Study (DLS) to investigate the relationship between alcohol intake and periodontal health. We specifically looked to assess the relationship between heavy drinking (2 or more drinks per day), and a variety of oral health outcomes relating to the gums: bleeding
on probing, calculus and plaque levels, mobility, alveolar bone loss, pocket depth, and gingival recession.
METHODS

Study population

The study population was from the Dental Longitudinal Study (DLS), a prospective longitudinal study initially composed of 1,231 males, which began as an offshoot of the Veterans Affairs (VA) Normative Aging Study. The initial cohort of the DLS began in 1968, and the study population was composed of healthy male residents of the Greater Boston area who were free from any chronic disease. The study design of the DLS has been detailed extensively in previous studies (Feldman, Douglass, Loftus, Kapur, & Chauncey, 1982; Glass, Loftus, Kapur, & Alman, 1973; and others). The majority of the participants are veterans, but most do not receive care from the VA healthcare system and receive physical health and dental care from private physicians. The demographics of this group of men was representative of the Greater Boston area at the time of the study origination, in that the majority of men were Caucasian. Ages ranged from 26 to 84, with the mean age being 48 with a standard deviation of 9 years.

Participants in the DLS receive comprehensive physical and dental examinations roughly every three years, during which they also complete thorough questionnaires on oral hygiene, dental care history, health care history, and behavioral questions. Over the years, the exam has evolved to encompass a more in-depth survey and different measurements of clinical parameters. The present study is a cross-sectional analysis using variables collected for the most part from first cycle of dental exams taking place between the years 1969 and 1973, with recession variables taken from the second cycle of dental exams (taking place between the years 1972 and 1976). This was selected
because the cohort size decreased in subsequent cycles due drop-out, thus the earliest cycles contained the largest sample size for data analysis.

**Oral health examination data**

Clinical parameters were measured through comprehensive oral examinations, which included both clinical and radiograph assessment. Trained dental examiners in this study have been found to have a high degree of inter-examiner agreement for assessing periodontal outcomes (Feldman et al., 1982). Clinical parameters utilized for this present bivariate analysis included: number of teeth, bleeding on probing, calculus, plaque, mobility, alveolar bone status, pocket depth, and gingival recession, as shown in table 2. Whole mouth scores were calculated by summing the scores for each variable, and dividing by the number of present teeth. Note that the sample size changes slightly for each periodontal outcome due to missing values, such as for individuals who have 0 teeth.

*Number of teeth* was counted, excluding third molars. Individuals who had 0 teeth were assigned *edentulous* status. *Gingival bleeding* was quantified by counting the number of teeth that bleed when probed with an instrument. *Calculus* was scored on a scale of 1 to 3 for each tooth. A given tooth was scored 1 if there were discontinuous flecks of calculus on the tooth. A score of 2 was given with a non-continuous band of calculus on the tooth, and a score of 3 was given with a continuous band of calculus. *Plaque* accumulation was assessed using a plaque disclosing solution, and scored on a scale of 1 to 3 for each tooth. A score of 1 was given if there was plaque accumulation on
interproximal surfaces only, a score of 2 given if there was plaque accumulation on the interproximal surfaces continuing to the buccal or lingual surfaces, and a score of 3 was given if there was plaque accumulation on more than two-thirds of the tooth. Mobility was measured by “manual palpitation” using instruments. Tooth mobility was scored on a scale of 1 to 3; 1 = slight mobility (<0.5 mm), 2 = moderate mobility (0.5 mm to 1 mm), and 3= substantial mobility (≥1mm). Alveolar bone loss was assessed by using radiographs, and quantified by percentage of loss out of maximum bone height and by measuring mm of loss. Above 40% ABL was selected as a level reflective of moderate to severe bone loss. ABL was also scored on a five point scale, modified from Schei, Waerhaug, Lovdal, & Arno (1959). Periodontal pocket depth was measured using a calibrated probe inserted into the periodontal pocket, with the maximal depth from the bottom of the pocket to the free gingival margin for a given tooth being recorded. Information on gingival recession was included as part of oral examinations starting from cycle two of exams. Recession was measured in millimeters using a calibrated probe by measuring the distance between the cement-enamel junction and the most apical part of the free gingiva.

Alcohol intake data

Average daily alcohol intake was ascertained as part of the Cornell Medical Index (CMI), using the survey question, “Do you usually take two or more alcoholic drinks a day?”, answered either “yes” or “no”. Definitions for moderate drinking and heavy drinking, low-risk and at-risk drinking, vary across studies, organizations, and time
periods. For the purposes of this study, we will refer men who indicated “no” to the CMI question as non-drinkers or moderate drinkers, and men who indicated “yes” to the CMI question as heavy drinkers. These definition are based on the definitions by the CDC and NIH National Institute on Alcohol Abuse and Alcoholism (NIAAA): moderate alcohol consumption is defined as an average daily consumption of up to 2 drinks per day for men and up to 1 drink per day for women (CDC, 2014; National Institute on Alcohol Abuse and Alcoholism [NIAAA], n.d.). Heavy drinking, as defined by the CDC, is excessive alcohol consumption in the form of average daily consumption of 15 or more drinks per week for men or 8 or more drinks per week for women, which is equivalent to more than two drinks per day for males (CDC, 2014). Thus our data analysis can be seen as comparing non-drinkers and moderate drinkers to heavy drinkers.

Data for other covariates

Caffeine consumption was also measured using the CMI by the question, “do you drink more than six cups of coffee or tea a day?”, answered “yes” or “no”. Body mass index (BMI) was calculated using height and weight information from physical examinations. Education attainment level was assessed by a survey question, and participants were assigned to one of three groups: high school graduate or less, some college, or college graduate. Education attainment level was dichotomized to non-college graduate and college graduate for analysis purposes. Participants were grouped by smoking status into three groups: non-smoker, cigarette smoker, and cigar smoker. Oral hygiene information was obtained by questionnaire during the oral examinations.
Brushing frequency was dichotomized to two or more times per day or less than two times per day. Flossing frequency was dichotomized to either flossing or no flossing. Prophylaxis, a procedure where a dental professional removes plaque and calculus from teeth ("Glossary of Dental Clinical and Administrative Terms"), within one year was answered yes or no.

**Statistical analysis**

Cross-sectional analysis was conducted using de-identified data from cycle one, in which a total of 1,231 participants were enrolled in the original cohort to complete oral examinations and surveys. SAS 9.3 was used for all data analysis. Based on their answer to the CMI survey question on alcohol intake, participants were grouped into one of two categories: under than 2 drinks per day, or 2 or more drinks per day. Data on alcohol intake was unavailable for 45 participants, and thus they were not included in the present analysis, therefore the current study utilizes a population of 1,186 individuals who underwent oral examination and had available data on alcohol intake. Independent samples student’s t-tests and chi-squared tests were used to detect any significant differences in baseline demographic or clinical parameters between heavy drinkers and moderate or non-drinkers. The bar graph for pocket depth trends was constructed using Microsoft Excel.

Using SAS 9.3, multiple linear regression models were used to control for the following independent variables: age, BMI, number of teeth, education, caffeine intake, smoking status, brushing frequency, floss use, and recent prophylaxis treatment.
Regression models were constructed for clinical outcomes that were found significantly different between the two groups by t-test; we constructed models for number of teeth, calculus levels, plaque levels, alveolar bone loss, and for pocket depth. Backward selection was used to remove any variables that were insignificantly related to the outcome of interest with p-values above 0.10. Standard regression coefficients were used to compare the relative effects of independent variables associated with the outcome of interest.
RESULTS

Of 1,231 participants from cycle 1, data on alcohol intake was missing for 45 patients. Table 1 shows that of the remaining n=1,186 participants, 80.02% (n=949) drank less than 2 drinks per day, while 19.98% (n=237) were heavy drinkers (consuming 2 or more drinks per day). Chi-squared tests showed that on average, those who drank 2 or more drinks per day were less likely to be a college graduate, more likely to drink more than 6 caffeinated drinks per day, and to be cigarette smokers (p = 0.0001, p = 0.036, p= 0.040, respectively). There was no significant difference between moderate/non-drinkers and heavy drinkers in regards to BMI, brushing and flossing habits, or receiving dental prophylaxis in the past year.

Table 1. Demographic characteristics.

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<thead>
<tr>
<th>Characteristic</th>
<th>&lt;2 alcoholic drinks per day</th>
<th>≥2 alcoholic drinks per day</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>n= 949 (80.02%)</td>
<td>n= 237 (19.98%)</td>
</tr>
<tr>
<td>Age</td>
<td>48.80 ± 9.17</td>
<td>48.75 ± 9.36</td>
</tr>
<tr>
<td>BMI</td>
<td>26.12 ± 2.90</td>
<td>26.03 ± 2.95</td>
</tr>
<tr>
<td>College graduate *</td>
<td>305 (32.14%)</td>
<td>45 (18.99%)</td>
</tr>
<tr>
<td>&gt;6 cups of coffee or tea per day *</td>
<td>129 (13.59%)</td>
<td>45 (18.99%)</td>
</tr>
<tr>
<td>Cigarette smoking *</td>
<td>238 (25.08%)</td>
<td>90 (37.97%)</td>
</tr>
<tr>
<td>Oral hygiene characteristics</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Brush twice daily</td>
<td>436 (46.28%)</td>
<td>115 (48.94%)</td>
</tr>
<tr>
<td>Floss use</td>
<td>315 (33.23%)</td>
<td>75 (31.65%)</td>
</tr>
<tr>
<td>Prophylaxis cleaning in past year</td>
<td>747 (78.80%)</td>
<td>175 (73.84%)</td>
</tr>
</tbody>
</table>

* p< 0.05 using chi-square test
Student’s t-test showed that of examined clinical parameters, those who drank 2 or more drinks per day had poorer oral health in the form of: fewer teeth, increased calculus, increased teeth with plaque score = 1, greater alveolar bone loss, and more teeth with increased pocket depth (see table 2). There was no significant difference between the two groups in number of teeth with bleeding on probing, whole mouth plaque score or number of teeth with plaque scores = 2 or 3, tooth mobility, or gingival recession measures (see table 2).

**Table 2. Clinical parameters.**

<table>
<thead>
<tr>
<th>Clinical parameter¹</th>
<th>&lt;2 alcoholic drinks per day n= 949 (80.02%)</th>
<th>≥2 alcoholic drinks per day n= 237 (19.98%)</th>
<th>P-value for difference</th>
</tr>
</thead>
<tbody>
<tr>
<td>Number of teeth (excluding third molars) ^</td>
<td>21.12 ± 2.47</td>
<td>20.04 ± 7.83</td>
<td>p = 0.049</td>
</tr>
<tr>
<td>Edentulous²</td>
<td>55 (5.80%)</td>
<td>15 (6.33%)</td>
<td>p = 0.75</td>
</tr>
<tr>
<td>Number of teeth with bleeding on probing</td>
<td>16.30 ± 6.51</td>
<td>16.16</td>
<td>p = 0.78</td>
</tr>
<tr>
<td>Calculus and plaque</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Whole mouth calculus score ^</td>
<td>1.34 ± 0.75</td>
<td>1.54 ± 0.77</td>
<td>p = 0.0005</td>
</tr>
<tr>
<td>Number of teeth with calculus score = 1</td>
<td>16.54 ± 7.23</td>
<td>17.13 ± 7.20</td>
<td>p = 0.28</td>
</tr>
<tr>
<td>Number of teeth with calculus score = 2 ^</td>
<td>9.13 ± 7.03</td>
<td>10.64 ± 7.60</td>
<td>p = 0.0049</td>
</tr>
<tr>
<td>Number of teeth with calculus score = 3 ^</td>
<td>3.33 ± 4.84</td>
<td>4.09 ± 5.10</td>
<td>p = 0.038</td>
</tr>
<tr>
<td>Whole mouth mean plaque score</td>
<td>1.54 ± 0.51</td>
<td>1.56 ± 0.53</td>
<td>p = 0.59</td>
</tr>
<tr>
<td>Number of teeth with plaque score = 1 ^</td>
<td>19.23 ± 5.94</td>
<td>18.33 ± 6.14</td>
<td>p = 0.045</td>
</tr>
</tbody>
</table>

¹ Increased calculus and plaque scores, tooth mobility, alveolar bone loss, pocket depth, and gingival recession are indicative of poorer periodontal health.

² Edentulous individuals lack teeth.
<table>
<thead>
<tr>
<th></th>
<th>Mean</th>
<th>SD</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td>Number of teeth with plaque score = 2</td>
<td>12.15 ± 6.23</td>
<td>11.59 ± 6.17</td>
<td>0.23</td>
</tr>
<tr>
<td>Number of teeth with plaque score = 3</td>
<td>2.51 ± 3.70</td>
<td>2.58 ± 3.42</td>
<td>0.80</td>
</tr>
<tr>
<td><strong>Tooth mobility</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Whole mouth mobility score</td>
<td>0.15 ± 0.28</td>
<td>0.21 ± 0.40</td>
<td>0.055</td>
</tr>
<tr>
<td>Number of teeth with mobility ≥1mm</td>
<td>0.070 ± 0.40</td>
<td>0.10 ± 0.51</td>
<td>0.40</td>
</tr>
<tr>
<td><strong>Alveolar bone status</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Whole mouth mean alveolar bone loss score ^</td>
<td>0.71 ± 0.63</td>
<td>0.89 ± 0.75</td>
<td>0.0014</td>
</tr>
<tr>
<td>Whole mouth mean alveolar bone loss in mm ^</td>
<td>3.22 ± 1.04</td>
<td>3.56 ± 1.18</td>
<td>0.0030</td>
</tr>
<tr>
<td>Number of teeth with alveolar bone loss ≥40% ^</td>
<td>0.70 ± 1.70</td>
<td>1.04 ± 2.28</td>
<td>0.042</td>
</tr>
<tr>
<td><strong>Pocket depth</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Number of teeth with pocket depth ≥3mm</td>
<td>8.56 ± 5.81</td>
<td>9.40 ± 6.18</td>
<td>0.059</td>
</tr>
<tr>
<td>Number of teeth with pocket depth ≥4mm ^</td>
<td>3.38 ± 3.99</td>
<td>4.18 ± 4.89</td>
<td>0.025</td>
</tr>
<tr>
<td>Number of teeth with pocket depth ≥5mm ^</td>
<td>1.09 ± 2.04</td>
<td>1.59 ± 3.05</td>
<td>0.022</td>
</tr>
<tr>
<td><strong>Gingival recession</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Whole mouth mean recession score (cycle 2)</td>
<td>1.17 ± 0.69</td>
<td>1.24 ± 0.72</td>
<td>0.20</td>
</tr>
<tr>
<td>Number of teeth with recession ≥3mm</td>
<td>14.05 ± 6.65</td>
<td>13.62 ± 6.77</td>
<td>0.43</td>
</tr>
<tr>
<td>Number of teeth with recession ≥4mm</td>
<td>8.75 ± 6.44</td>
<td>9.03 ± 6.41</td>
<td>0.61</td>
</tr>
<tr>
<td>Number of teeth with recession ≥5mm</td>
<td>1.89 ± 2.98</td>
<td>2.07 ± 2.69</td>
<td>0.46</td>
</tr>
</tbody>
</table>

^ p < 0.05 using student’s t-test

Multiple linear regression models controlling for associated independent variables showed that of the chosen clinical parameters, there existed a statistically significant relationship between heavy alcohol consumption (2 or more drinks per day) and two clinical features of periodontal disease: whole mouth mean alveolar bone loss score, and
number of teeth with pocket depths greater than 4mm and greater than 5mm (table 3 and table 4).

Alveolar bone loss

For the three measurements of alveolar bone loss (whole mouth mean alveolar bone loss score, mean millimeters of alveolar bone loss, and # teeth with alveolar bone loss over 40%), heavy alcohol intake was found to be significantly associated with whole mouth mean alveolar bone loss score (p = 0.024) when controlling for age, number of teeth, education attainment level, caffeine intake, smoking status, and floss use. The regression models showed weaker p-values for association with millimeters of ABL and number of teeth with over 40% ABL (p = 0.090 and p = 0.060 respectively), indicative of a trend for alcohol to be related to these outcomes.

Table 3. Multiple linear regression models for alveolar bone loss.

| Variable                              | Parameter Estimate | Standard Error | t Value | Pr >|t|   |
|---------------------------------------|--------------------|----------------|---------|------|----|
| Alcohol intake (<2 or ≥2 drinks per day) | 0.093              | 0.041          | 2.27    | 0.024|
| Age                                   | 0.017              | 0.0019         | 8.74    | <0.0001|
| Number of teeth (excluding third molars) | -0.049             | 0.0031         | -15.52  | <0.0001|

3 Alveolar bone loss refers to the deterioration of the bone holding the teeth in place; increased alveolar bone loss (ABL) indicates poorer periodontal health.
ABL exceeding 40% is reflective of moderate to severe bone loss.

| Variable                                                                 | Parameter Estimate | Standard Error | t Value | Pr > |t| |
|------------------------------------------------------------------------|--------------------|----------------|---------|------|---|
| Education attainment (high school, some college, or college graduate)  | -0.089             | 0.021          | -4.17   | <0.0001 |
| Caffeine intake (≤6 cups or >6 cups of coffee or tea)                   | 0.189              | 0.047          | 4.01    | <0.0001 |
| Smoking status (nonsmoker, cigarette smoker, or cigar smoker)          | 0.053              | 0.021          | 2.49    | 0.013  |
| Floss use (yes or no)                                                  | -0.061             | 0.035          | -1.73   | 0.084  |

Multiple linear regression of mean mm ABL with associated variables

| Variable                                                                 | Parameter Estimate | Standard Error | t Value | Pr > |t| |
|------------------------------------------------------------------------|--------------------|----------------|---------|------|---|
| Alcohol intake (<2 or ≥2 drinks per day)                               | 0.18               | 0.10           | 1.68    | 0.093 |
| Age                                                                    | 0.031              | 0.0051         | 5.91    | <0.0001 |
| Number of teeth (excluding third molars)                               | -0.059             | 0.0085         | -7.00   | <0.0001 |
| Education attainment (high school, some college, or college graduate)  | -0.12              | 0.051          | -2.28   | 0.023 |
| Caffeine intake (≤6 cups or >6 cups of coffee or tea)                   | 0.41               | 0.11           | 3.56    | 0.0004 |
| Smoking status (nonsmoker, cigarette smoker, or cigar smoker)          | 0.16               | 0.051          | 3.19    | 0.0015 |
| Flossing use (yes or no)                                               | -0.15              | 0.083          | -1.80   | 0.072 |

Multiple linear regression of # teeth with ABL ≥ 40% with associated variables

| Variable                                                                 | Parameter Estimate | Standard Error | t Value | Pr > |t| |
|------------------------------------------------------------------------|--------------------|----------------|---------|------|---|
| Alcohol intake (<2 or ≥2 drinks per day)                               | 0.25               | 0.13           | 1.89    | 0.0595 |
| Age                                                                    | 0.030              | 0.0063         | 4.75    | <0.0001 |
| Number of teeth (excluding third molars)                               | -0.032             | 0.010          | -3.15   | 0.0017 |
| Education attainment (high school, some college, or college graduate)  | -0.13              | 0.070          | -1.81   | 0.070 |

ABL exceeding 40% is reflective of moderate to severe bone loss.
On average, participants who drank 2 or more alcoholic drinks per day had more teeth with pocket depths exceeding 3mm, 4mm, and 5mm (see figure 1). For the three levels of pocket depth measurement (number of teeth with pocket depth over 3mm, over 4mm, and over 5mm), multiple linear regression models showed a significant association for number of teeth with pocket depth over 4mm and 5mm in the presence of other variables, with p = 0.049 and p= 0.017, respectively. The model exhibited a weak p-value of p= 0.069 for the association between alcohol and number of teeth with pocket depth over 3mm; the statistical trend is indicative of a pattern for heavy drinking to be related to increased pocket depth (see table 4).

<table>
<thead>
<tr>
<th>Smoking status (nonsmoker, cigarette smoker, or cigar smoker)</th>
<th>0.16</th>
<th>0.070</th>
<th>2.24</th>
<th>0.025</th>
</tr>
</thead>
<tbody>
<tr>
<td>Floss use (yes or no)</td>
<td>-0.24</td>
<td>0.12</td>
<td>-2.06</td>
<td>0.040</td>
</tr>
</tbody>
</table>

*Pocket Depth*

On average, participants who drank 2 or more alcoholic drinks per day had more teeth with pocket depths exceeding 3mm, 4mm, and 5mm (see figure 1). For the three levels of pocket depth measurement (number of teeth with pocket depth over 3mm, over 4mm, and over 5mm), multiple linear regression models showed a significant association for number of teeth with pocket depth over 4mm and 5mm in the presence of other variables, with p = 0.049 and p= 0.017, respectively. The model exhibited a weak p-value of p= 0.069 for the association between alcohol and number of teeth with pocket depth over 3mm; the statistical trend is indicative of a pattern for heavy drinking to be related to increased pocket depth (see table 4).
**Figure 1.** Pocket depth of moderate/non-drinkers vs. heavy drinkers.

![Bar chart showing number of teeth with pocket depths over 3mm, 4mm, and 5mm.](image)

* Chi-square test p< 0.05

**Table 4.** Multiple linear regression models for pocket depth.

| Variable                              | Parameter Estimate | Standard Error | t Value | Pr>|t| |
|---------------------------------------|--------------------|----------------|---------|-----|
| Alcohol intake (<2 or ≥2 drinks per day) | 0.78               | 0.43           | 1.82    | 0.069 |
| Body mass index (kg/m2)               | 0.18               | 0.059          | 2.98    | 0.0029 |

5 Pocket depth refers a measurement of the space between the tooth and the gums, in which the depth of from bottom of the periodontal pocket to the free gingival margin is measured. Increased pocket depth indicates poorer periodontal health. Normal healthy gums have pocket depth in health measurements between 1mm and 3mm (NIDCR, 2013).
### Multiple linear regression of number of teeth with pocket depth \( \geq 4\text{mm} \) with associated variables

| Variable                                                                 | Parameter Estimate | Standard Error | \( t \) Value | \( \text{Pr} >|t| \) |
|--------------------------------------------------------------------------|--------------------|----------------|---------------|-----------------|
| Alcohol intake (<2 or \( \geq 2 \) drinks per day)                       | 0.63               | 0.32           | 1.98          | 0.0485          |
| Body mass index (kg/m2)                                                  | 0.13               | 0.044          | 3.03          | 0.0025          |
| Number of teeth (excluding third molars)                                 | 0.10               | 0.024          | 4.36          | <0.0001         |
| Education attainment (high school, some college, or college graduate)    | -0.39              | 0.17           | -2.37         | 0.0178          |
| Caffeine intake (\( \leq 6 \) cups or >6 cups of coffee or tea)         | 1.01               | 0.36           | 2.77          | 0.0057          |
| Smoking status (nonsmoker, cigarette smoker, or cigar smoker)            | 0.54               | 0.17           | 3.21          | 0.0014          |
| Floss use (yes or no)                                                    | -0.90              | 0.28           | -3.27         | 0.0011          |

### Multiple linear regression of number of teeth with pocket depth \( \geq 5\text{mm} \) with associated variables

<p>| Variable                                                                 | Parameter Estimate | Standard Error | ( t ) Value | ( \text{Pr} &gt;|t| ) |
|--------------------------------------------------------------------------|--------------------|----------------|---------------|-----------------|
| Alcohol intake (&lt;2 or ( \geq 2 ) drinks per day)                       | 0.42               | 0.18           | 2.40          | 0.0168          |
| Body mass index (kg/m2)                                                  | 0.062              | 0.024          | 2.55          | 0.011           |</p>
<table>
<thead>
<tr>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>Education attainment (high school, some college, or college graduate)</td>
<td>-0.16</td>
<td>0.090</td>
<td>-1.79</td>
<td>0.073</td>
</tr>
<tr>
<td>Smoking status (nonsmoker, cigarette smoker, or cigar smoker)</td>
<td>0.20</td>
<td>0.093</td>
<td>2.14</td>
<td>0.033</td>
</tr>
<tr>
<td>Floss use (yes or no)</td>
<td>-0.34</td>
<td>0.15</td>
<td>-2.27</td>
<td>0.023</td>
</tr>
</tbody>
</table>
DISCUSSION

This study sought to compare oral health outcomes between men who drank less than two drinks a day (non-drinkers and moderate drinkers) and men who drank two or more drinks a day (heavy drinkers). After controlling for covariates, heavy drinkers had significantly increased whole mouth mean alveolar bone loss and number of teeth with pocket depths greater than 4mm and greater than 5mm. There was no association found between heavy alcohol consumption and number of teeth, bleeding on probing, calculus and plaque, tooth mobility, or gingival recession.

According to the CDC BRFSS, heavy drinking (defined by the BRFSS as over 2 drinks per day for males and over 1 drink per day for females), occurred in 6.2% of the US population during the year of 2013 (CDC, 2013). The higher prevalence of drinking 2 or more drinks per day found in our study sample (19.98%, see table 1), may be attributed to several factors. Military veterans are at increased risk for PTSD and alcohol dependence that is often associated with psychological trauma (Taft et al., 2007), and studies have found Vietnam veterans to have higher alcohol consumption than non-veterans (Boscarino, 1981). Additionally, an abundance of literature has found that males consume higher amounts of alcohol than females (Nolen-Hoeksema, 2004; Wilsnack, Vogeltanz, Wilsnack, & Harris, 2000). It has been reported that men have two to three times the odds of having alcohol use disorder, alcohol abuse, or alcohol dependence (Hasin, Stinson, Ogburn, & Grant, 2007), and that men have substantially increased episodes of binge drinking among both moderate and heavy drinkers (Naimi et al., 2003). Furthermore, it is possible that during the time of data collection, the late 1960’s to early
1970’s, differing social norms for drinking made for different alcohol consumption rates compared to present day. In this sample, there was also a tendency for drinkers to be cigarette smokers, which is concordant with existing literature that finds that smoking and alcohol dependence often goes hand-in-hand (Room, 2003). A number of studies have found smoking to be a risk factor for periodontal disease and to affect alveolar bone status, thus it is important to have controlled for tobacco use in our data analysis (Bergström, 2004; Bolin, Eklund, Frithiof, & Lavstedt, 1992).

Our data showed no significant differences between non-drinkers/moderate drinkers and heavy drinkers for brushing and flossing habits and recent prophylactic dental care (using chi-square tests, see table 1). This indicates that heavy drinkers do not appear to have poorer oral hygiene habits in comparison to non-drinkers, which is in contrast to existing literature suggesting that poor periodontal health in drinkers are in part due to poor hygiene habits. This may be reflected in our study, in that there was no significant difference in filled or decayed tooth surfaces between the two groups (data not shown). It may be the case that the threshold of two drinks per day is too low to detect differences in oral hygiene habits between groups. Moreover, brushing and recent prophylactic care did not appear to be significantly related to the outcomes in most of the regression models.

Consuming two or more drinks per day appeared to be primarily associated with two unfavorable clinical parameters: alveolar bone loss and pocket depth. The finding that more than moderate drinking is associated with whole mouth mean alveolar bone loss suggests a possible mechanism for alcohol to affect periodontal disease by its action
on bone. Additionally, despite non-significant p-values for the association between mean mm ABL and number of teeth with ABL over 40%, statistical analysis points towards a trend for heavy alcohol consumption to be related to ABL (see table 3). These results on alveolar bone loss are an important clinical finding, because loss of alveolar bone directly leads to loss of tooth attachment. ABL also makes it more difficult to receive procedures such as dental implants, because the success of an implant is contingent on the presence and stability of the alveolar bone process (Hammerle & Jung, 2008). Significant loss of alveolar bone in more severe cases of periodontal disease can thus lead to more complicated treatment plans, such as requiring bone grafts to stimulate bone regeneration and restore the alveolar ridge (Hammerle & Jung, 2008).

These results are possibly explained by the effects of alcohol bone to affect bone remodeling by inducing bone resorption (Sampson, 1998), although this supposition should be interpreted with caution as we are unable to confirm a causal relationship between heavy alcohol exposure and periodontal outcomes. Our results are reflective of past animal studies, where studies have found a dose-dependent relationship between alcohol consumption and increased alveolar bone loss in rats (Souza, Ricardo, Kantoski, & Rocha, 2009). However, there exist few epidemiological studies on humans looking specifically at the effect of alcohol consumption on alveolar bone loss. One epidemiological study found no association between alveolar bone loss and alcohol intake: Tezal et al. (2001) found that individuals consuming 10 or more drinks a week was not related to alveolar bone status although it was related to bleeding and CAL.
The finding that heavy alcohol consumption is associated with pocket depth greater than 4mm and 5mm is of important clinical significance. Healthy gums have average pocket depths of 1mm to 3mm, and pocket depth measurements under 3mm generally indicate to dentists that the gums are healthy and no clinical action is needed (Cutress, Ainamo, & Sardo-Infirri, 1987; NIDCR, 2013). Pocket depths over 3mm suggest moderate or severe periodontal disease, and points towards the need for dentists to provide either oral hygiene education and/or deep ultrasonic cleaning to remove plaque and tartar and to prevent periodontal disease progression (Cutress et al., 1987; Zimmerman et al., 2015). With severe periodontal disease and extremely deep pockets, treatment plans can again become increasingly complex and involve surgical procedures such as flap surgery to decrease pocket depth (Karring & Lindhe, 2008). It is possible that the increased pocket depths among heavy drinkers is related to loss of alveolar bone, because deep pockets can foster growth of microbiota which promote bone loss (Tanner, Socransky, & Goodson, 1984).

Our result that heavy drinking, or consuming 2 or more drinks per day, is associated with increased pocket depth in a greater number of teeth is in agreement with past studies which have found an association between alcohol consumption and pocket depth and/or clinical attachment level. Similar to our results, Amaral et al. (2008) found that alcoholism was associated with more teeth having increased CAL and increased pocket depths, while there was no association between alcoholism and plaque or bleeding on probing. A number of studies have found alcohol intake to be related to CAL (Kim et al., 2014; Susin et al., 2014; Tezal et al, 2004; Tezal et al., 2001), which we can interpret
as being along the same lines as our findings due to pocket depth measurements being integral to the calculation of CAL.

**Strengths and limitations**

One major limitation of this study is that we are unable to confirm any causal mechanisms between alcohol intake and clinical outcomes due to the cross-sectional design of this study. This is because data from a single time point was used, thus we are unable to determine whether drinking status preceded occurrence of clinical outcomes. Nonetheless, the cross-sectional study design is advantageous because it allows us to minimize possible effects of loss to follow-up in an aging study sample. Results of this study provide preliminary evidence for longitudinal studies to investigate a causal relationship between alcohol intake and indicators of periodontal disease. Furthermore, this study is unique in that it investigates a wide range of periodontal clinical parameters. This allows us to pinpoint the specific ways that our exposure, heavy alcohol consumption, affects periodontal health. As mentioned previously, epidemiological studies on periodontal disease often deal with methodological inconsistencies between studies due to varying criteria for assessing periodontal disease status (Irfan et al., 2001; Papapanou & Lindhe, 2008). Assessing a wide range of periodontal health characteristics in this present study allows us to somewhat overcome this obstacle, because we are not utilizing a set criteria for periodontal disease status and instead have analyzed a spectrum of periodontal health indicators, independent of periodontal disease diagnosis.
The observational nature of this study also presents with possible confounding factors, which may lead to certain individuals appearing to have poorer or better health outcomes due to factors that were not accounted for as part of our analysis. Furthermore, misclassification bias may occur such as if those who did not drink previously recently became categorized as heavy drinkers, or if previously heavy drinkers recently became abstainers. The latter phenomenon is known as the “sick quitter” hypothesis, a common concern for bias in alcohol epidemiology research. This describes the potential misclassification of former drinkers to the non-drinker group due to recent behavioral change to stop drinking, often because of health problems associated with problem drinking. (Marmot & Brunner, 1991; Shaper, Wannamethee, & Walker, 1988; Shaper, 2011). This information bias may have affected our study results by causing health outcomes between heavy drinkers and non-drinkers/ moderate drinkers to be less pronounced. Additionally, because the collection of many of the variables was through self-reported data, this leads to the possibility of response bias for some variables, including for alcohol intake level. Although our question on alcohol intake (“Do you usually take two or more alcoholic drinks a day?”) was intended to ascertain average daily alcohol intake, participants may have incorrectly interpreted it as intake level on days that alcohol was consumed, leading to incorrect assignment for alcohol intake status. A last form of bias which may have occurred is selection bias, due to the enrollment of voluntarily participating study subjects; certain individuals may be less inclined or able to be study candidates. For example, it may be that severe alcoholics were less motivated to volunteer to be a part of the study.
Another limitation to this study is that it does not differentiate between patterns of alcohol intake, because only it only utilizes information on average daily consumption. The present study groups “moderate drinkers” with non-drinkers, thus we are unable to differentiate any possible differences that may exist between moderate drinkers and those who abstain from alcohol entirely. Distinguishing between moderate drinkers and non-drinkers may be important, as there is much debate on the possible benefit or detriment of moderate alcohol consumption. The “heavy” alcohol consumption group (consuming two or more drinks daily) may also include a wide range of alcohol consumption levels, but we are unable to further stratify levels of alcohol consumption due to the nature of our survey question. Furthermore, we are unable to ascertain the effect of other drinking patterns such as binge drinking frequency or alcohol dependence, making it difficult for us to generalize our study results to the oral health of all types of problem drinkers. The comparability of this study is further limited due to the fact that our study sample only included adult males and the majority of the participants were Caucasian, thus limiting the generalizability of our study results to a more ethnically diverse population, to the adolescent population, or to the female population.

A final limitation is that our definition of heavy alcoholic intake, which we define as consuming two or more drinks per day for men, varies slightly from the CDC and NIAAA definition that heavy drinking is consuming more than two drinks per day for men. This is due to the nature of the survey question used during the time of study. Because our study included a daily consumption level of two drinks in the heavy drinking
category, comparison of prevalence information from our study on “heavy drinking” to other studies should be approached with caution.

**Implications for future research**

In conclusion, our results support that heavy drinking is related to certain poor periodontal health outcomes; alveolar bone loss and increased periodontal pocket depth; both of which indicate loss of periodontal support and are clinically important hallmarks of periodontal disease. This study provides evidence that when developing treatment plans for periodontal disease patients who are consume excessive alcohol, dentists should consider advising patients to decrease alcohol consumption as a possible way to minimize periodontal disease progression.

The results of this study point towards the importance of further research on the effects of alcohol and oral health. Both pocket depth and alveolar bone loss are common clinical manifestations and diagnostic criteria of periodontal disease, thus the possible effects of alcohol consumption on these parameters is an important finding. Further studies should aim to clarify this relationship on both an epidemiological and physiological level. This includes studies to investigate the molecular mechanisms on how alcohol affects oral health, and in particular, how it affects alveolar bone status. This should also include studies comparing between more levels of alcohol consumption, different patterns of drinking, and also between types of alcoholic beverage consumed. The general lack of consensus on this relationship also points towards the need for review studies or meta-analyses on the subject. Large-scale prospective longitudinal studies
would be instrumental in establishing the possible role of alcohol as a risk factor for subsequent periodontal disease and the clinical manifestations that come with it.
REFERENCES


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CHRISTINE CHIAO

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Birth year: 1991

EDUCATION

BOSTON UNIVERSITY
School of Graduate Medical Sciences
Candidate for Master of Science in Medical Sciences, May 2015

School of Public Health
Candidate for Master of Public Health in Social and Behavioral Sciences, May 2015

College of Arts and Sciences
B.A. in Biology, May 2013

PROFESSIONAL EXPERIENCE

Boston University School of Dental Medicine, Boston MA
Health Policy and Health Services Research Department
Research Assistant
July 2014 - April 2014

• Helped to input examination data from the VA hospital Dental Longitudinal Study into Excel. Performed statistical analysis of data to assess the relationship between alcohol intake and periodontal status, culminating to a thesis paper for completion of a Masters degree in Medical Sciences at Boston University.

Boston University School of Public Health, Boston MA
Core Course Tutor
February 2015 - May 2015

• Tutored students in core courses for environmental health and epidemiology at the BU School of Public Health. Met with students on a weekly basis to review lectures topics, practice problems, and study skills.

Boston University School of Social Work, Boston MA
Asian Women’s Health Initiative Project
Research Assistant
May 2012 – November 2013

• Assisted in writing a grant proposal for an HIV-risk intervention for Asian-American women. Specifically, assisted in session development and created in-depth questionnaires to assess eligibility criteria and outcomes. Assisted in a qualitative research project to develop a framework for understanding Asian-American women’s suicide and self-harm behaviors. Performed data analysis and helped to write a peer-reviewed journal article.
**Boston University School of Medicine**, Boston MA  
Cancer Center  
*RResearch Assistant*  
April 2011 - August 2013

- Worked with lab members to investigate the role of the SIRT1 protein relating to epigenetics involved in cancer progression. Performed laboratory assays including PCR, western blotting, microscopy, cell culture procedures. Performed data organization and analysis, also aided in paper and grant writing.

**SKILLS**

*Computer*: SPSS (beginner), SAS (beginner), Atlas-ti, Zotero, EndNote, REDCap, Adobe Photoshop, Microsoft Office

*Laboratory*: cell culture, cell staining and counting, viral infection and transfection, blotting techniques, PCR, pipetting, dilutions and preparation of solutions, mouse care and tail-vein injections

*Other*: IRB protocol, NIH grant writing, Protecting Human Subjects Research Participants NIH and CITI program certified

**PUBLICATIONS AND PRESENTATIONS**


