

Association between Periodontal Disease and the Intake of Carbohydrate, Fat and Total Calories in Humans

Thesis submitted in partial fulfillment of the
requirement for the degree of Master of Science

Ahmed A Alhassani, BDS

Department of Periodontology
Tufts University School of Dental Medicine
Boston, MA
2013



Committee Members:

Rory O'Neill, DMD, BDS, MSc

Associate Professor, Department of Periodontics
Tufts University School of Dental Medicine, Boston, MA

Wanda Wright, DDS, MS, MSD

Assistant Professor, Department of Public Health & Community Service
Tufts University School of Dental Medicine, Boston, MA

Paul Stark, MS, ScD

Professor & Director, Division of Advanced & Graduate Education
Tufts University School of Dental Medicine, Boston, MA

Carole Palmer Ed.D, RD, LDN

Professor, Department of Public Health & Community Service
Tufts University School of Dental Medicine, Boston, MA

Elizabeth A. Krall Kaye, PhD, MPH

Professor, Department of Health Policy & Health Services Research
Boston University School of Dental Medicine, Boston, MA

Abstract:

Background: There is increasing evidence in the literature about the association between diet and periodontal disease. Due to the proposed effect of diet on oral and systemic environments, we hypothesized that there is an association between periodontal disease and carbohydrate, fat and total caloric intake.

Methods: Data from the National Health and Nutrition Examination Survey (NHANES) 2009-2010 was used. After the application of inclusion and exclusion criteria, 2636 individuals were included in the sample. The outcome variable was periodontitis, defined as the presence of at least one site with both attachment loss ≥ 3 mm and probing depth ≥ 4 mm. Exposure variables were: percentage of calories in diet from carbohydrate, percentage of calories in diet from fat, and total caloric intake. The 2010 Dietary Guidelines for Americans were used to categorize carbohydrate and fat into 3 categories each. Caloric requirement for each subject was estimated using Harris-Benedict equation and individual's reported level of physical activity. Actual caloric intake was compared with the estimated caloric requirement in order to classify participants into: reduced, average and excess caloric intake.

The following covariates were selected as potential confounders: smoking, diabetes, age, sex, race/ethnicity, education, socioeconomic level, waist circumference and whether a subject has had previous periodontal treatment or not. Descriptive statistics, bivariate analysis and logistic regression models were used for data analyses.

Results: The prevalence of periodontitis in the sample was 39.1%. A statistical significance was detected between the outcome variable and all the covariates. No statistical significance was found between any of the exposure variables and periodontitis.

Conclusions: the results of this study showed no statistically significant association between periodontal disease and carbohydrate, fat and total caloric intake. More studies are needed to further investigate such association.

Table of Contents:

Introduction	2
Aim and Hypothesis.....	10
Research Design and Methods.....	11
Data management	16
Results	26
Discussion	30
Conclusion	35
Tables	36
Figures	48
References	53

List of Tables:

Table 1: Characteristics of study population.

Table 2: Distribution of study population according to the exposure variables

Table 3: Sample distribution among the activity factor values

Table 4: Overall prevalence of periodontitis

Table 5: Prevalence of periodontitis among the study population categories

Table 6: Distribution of study population according to exposure variables and gender.

Table 7: Distribution of study population according to exposure variables and race/ethnicity.

Table 8: Distribution of study population according to exposure variables and age categories.

Table 9: Logistic regression model 1

Table 10: Logistic regression model 2

Table 11: Logistic regression model 3

Table 12: Logistic regression model 4

Table 13: Logistic regression model 5

List of Figures:

Figure 1: Distribution of carbohydrate intake by periodontitis

Figure 2 Distribution of fat intake by periodontitis

Figure 3: Distribution of total caloric intake by periodontitis

Figure 4: Distribution according to percentage of calories acquired from carbohydrate (uncategorized)

.

Figure 5: Distribution according to percentage of calories acquired from carbohydrate (uncategorized)

Figure 6: Distribution according to percentage of actual caloric intake/estimated caloric requirement

Association between Periodontal Disease and the Intake of Carbohydrate, Fat and Total Calories in Humans

Introduction:

Periodontitis is an inflammatory disease that leads to break down of the tooth-supporting apparatus through destruction of the periodontal ligament which is attached to the bundle bone and the cementum; alveolar bone resorption and apical migration of the junctional epithelium. Gingivitis is defined as inflammation of the superficial periodontal tissue that does not lead to periodontal attachment loss. The primary etiologic factor for both diseases is bacterial biofilm that involves colonization by periodontal pathogens.¹ While the gingivitis lesion is reversible and does not cause loss of periodontal support once the etiologic factor(s) is/are removed, the periodontitis lesion is irreversible. These periodontitis lesions progress with loss of attachment and supporting bone, gingival recession, increasing tooth mobility and eventual tooth loss.¹

Periodontitis is one of the most common chronic diseases in human adults. Eke *et al.* used the data from the National Health and Nutrition Examination Survey (NHANES) 2009-2010 data, to estimate the prevalence of periodontitis in the United States.² Data from that study estimated that 47.2% of the US population aged 30 years or older had some degree of periodontal disease, with 30% and 8.5% of all the subjects having moderate and severe periodontitis, respectively.² The remaining subjects (8.7%) would have slight periodontitis.² Special attention has to be given to the findings of this study, as the data set used by Eke *et al.* represents the first NHANES data in which a full-mouth periodontal examination was performed, versus random partial-mouth examination in the previous NHANES.³ Several reports in the past have concluded that partial mouth exam in NHANES would lead to underestimation of the prevalence of periodontal disease.^{3,4,5}

Several systemic, local and environmental factors have been associated with periodontitis.⁶ As with most chronic disorders, studying the association between periodontitis and risk factors/indicators, represents a huge challenge due to the overlap between different confounders and the time required to produce a measurable disease outcome. Ethical concerns add to the difficulty of studying such an association. The best study design to examine any association would be a randomized clinical trial (RCT) on human subjects, where an investigator has control over the study design. It would be unethical to leave some patients with periodontal disease untreated. In addition, once an exposure (e.g. treatment modality) is proved to be effective, a study has to be terminated, as it would be unacceptable to deprive the other groups of the benefit of treatment. Feasible study designs to investigate periodontal disease would be cross sectional studies, cohort studies and only limited RCTs.

Albinder has studied the risk factors of periodontal disease.⁶ Increasing age has been associated with increased periodontal break down.⁶ It is not clear, however, whether this is merely due to the 'passage of time' or due to other changes at the host level that increase the risk for periodontal disease. Studies have shown that men have a higher risk of periodontal disease than women.⁶ Individuals with high socioeconomic status tend to have a lower risk for periodontal disease when compared to those with a low socioeconomic status.⁶ Smoking, diabetes, poor oral hygiene, genetics, obesity, level of physical activity diet and many other factors have been shown in the literature to be associated with periodontitis.⁶⁻¹⁰ Strength of association varies. For instance

smoking and diabetes are proven to be strong risk factors for periodontal diseases while the relationship with diet is still controversial.⁶⁻¹⁰

Periodontal disease results from the interaction between the bacterial challenge and the host response. There is substantial evidence that the host immune response, through the release of pro-inflammatory mediators, such as: tumor necrosis factor- α (TNF- α), Interleukin-1 (IL-1), IL-6, IL-8 and Prostaglandin E₂ (PGE₂), causes most of the tissue destruction in periodontitis.¹¹ Nevertheless, the pathogenesis of periodontitis is not yet completely fully understood. Gingivitis is widely accepted as a prerequisite for periodontitis, only a few gingivitis sites develop into periodontitis.¹² Looking from this prospective, one can understand the importance of studying the association between periodontal disease and the potential risk factors.

Obesity and poor quality diet have been associated with periodontitis in cross sectional, and a few longitudinal, studies.^{9,10,13,14} Obesity has also been associated with elevated levels of inflammatory mediators that may play a role in the pathogenesis of periodontitis.⁸ In a recent prospective study, Gorman *et al.* concluded that there is a positive association between adiposity changes (gains in body weight, waist circumference, and arm fat) and periodontal disease when 893 non-diabetic male subjects were followed up to four decades.¹³ In addition, Jimenez *et al.* followed 36,910 healthy men, as a part of the Health Professionals Follow-Up Study and found a positive association between self-reported periodontal disease and adiposity measures which included body mass index (BMI), waist circumference (WC) and waist-to-hip ratio (WHR).¹⁴

When looking at the human diet, one can easily appreciate the huge variation of what people eat. Professional dietary recommendations are revised and changed over relatively short periods of time. The United States Department of Agriculture (USDA) released the “Food Wheel” in 1984 as a guide for adequate and moderate nutrition. Eight years later, the USDA developed the “Food Guide Pyramid”, which was later updated in 2005 when “MyPyramid Food Guidance system” was introduced. Recently, in 2011, the USDA announced the “MyPlate” guide (more information can be found on the website: www.choosemyplate.gov).¹⁵

Studying human nutrition is extremely complicated. There is a great variation among persons and among communities. This presents a huge challenge on how to record dietary intake. Furthermore, diet can be looked at from several perspectives (e.g. total intake, macronutrients, key elements, food groups, etc.). How dietary information is recorded is another challenge. Bias, recall problems, high cost and compliance issues are among these difficulties.^{16,17}

A number of methods are available for researchers who are interested in measuring dietary intake. The most common methods are: diet record (diary), 24-hour recall and food frequency questionnaires (FFQ). Diet record involves participants self-reporting their daily dietary intake in detail for a day or more (most commonly 3-7 days). In order to enhance accuracy, training and instructions are provided to the participants. The 24-hour recall method is conducted by trained interviewers to obtain detailed information about diet that was consumed in a 24-hour period. Although accurate information can be gained this way, it can be costly. In FFQs, questionnaires are sent to the participants to self-report their average, overall consumption of food over a

period of time, which is typically weeks or months. Less attention is given to details with this method versus the other two methods. FFQs are very useful in tracking long-term dietary patterns when conducting cohort studies.¹⁶⁻¹⁸

As mentioned earlier, the relationship between periodontal diseases and diet is controversial. Although many reports have succeeded in establishing an association between various elements of diet, oral health, and periodontal diseases, others have failed to confirm such an association.^{7-10,16} Several factors can contribute to such conflict in the literature. Different studies have targeted different dietary elements. When designing a study it is crucial to take into consideration that the relationship between most nutrients and diseases is not linear.^{16,17} In other words, a study design must ensure the presence of enough subjects who fit into either the 'deficient' or 'excess' groups vs. the 'optimal' group.¹⁷ Another important point is the difference in study designs among various studies. Confounding factors add to these difficulties.¹⁷

Most of the studies investigating the relationship between diet and the periodontal status in humans looked at Vitamin D and Calcium intake. Many found an improved periodontal health when these nutritional supplements are taken.¹⁹⁻²³ Only a few reports looked at other aspects of dietary intake. Al-Zahrani *et al.*²⁴ have found a positive association between poor dietary habits and the degree of calculus deposition on the teeth. In another study they reported that individuals with poor diets are three times more likely to develop periodontitis.⁸ It is worth mentioning, however, that Al-Zahrani *et al.* used the original healthy eating index (HEI), which is a measure of the overall dietary quality that the USDA developed the 1995.^{8,9}

Yoshihara *et al.* conducted a longitudinal study that followed 600 individuals aged 70 years for 6 years. They found a negative association between vegetable intake and periodontal disease.²⁵

As a part of the Health Professional Follow-up Study, Merchant *et al.* concluded that the risk of developing periodontitis was reduced with increasing whole-grain intake.²⁶ In another prospective study, Schwartz *et al.* followed 625 men for an average of 15 years.²⁷ This study was a part of the Veterans Affairs Dental Longitudinal Study.²⁷ The study concluded that higher dietary fiber content reduced progression of periodontal disease in men who were ≥ 65 years old ($n=204$).²⁷

Several mechanisms by which diet can affect periodontal status are mentioned in the literature. Earlier studies suggest a direct effect of the food content on the bacterial oral environment.²⁸ For example, simple sugars may provide nutrition to some of the biofilm organisms, while fibrous food content might have a cleansing effect.^{29,30}

Following consumption of meals, there is a transient state of systemic inflammation that is associated with oxidative stress and the release of reactive oxygen species; this is exaggerated with meals that are heavy in fat and/or refined carbohydrate.^{31,32} In addition, high caloric diets that are rich in carbohydrate and fat are associated with obesity and increased adipose tissue storage.^{31,32} Several *in vitro* and *in vivo* studies have shown the increased capability of adipose tissue to produce key pro inflammatory mediators such as TNF- α , IL-1 and IL-6.^{31,33} In addition, increased insulin production as a result of carbohydrate ingestion contributes to increased fat

tissue storage.³¹ There is increased evidence in the literature that links increased levels of pro-inflammatory mediators and insulin resistance.³⁴

A high fat diet has been associated with a change of intestinal flora into a more gram-negative bacterial flora.³⁴ An association between this shift in microbiota and the oral environment has been suggested in the literature.^{34,35} In fact, Blasco-Baque *et al.*³⁵ found a positive association between a fat enriched diet and the prevalence of periodontal pathogens such as *Fusobacterium nucleatum* and *Prevotella intermedia*. Their results also indicated increased gingival inflammation and alveolar bone loss.³⁵

Caloric restriction has been associated with a reduction in systemic chronic inflammation. Interestingly, Reynolds et al. studied the association between dietary caloric restriction and periodontal diseases in rhesus monkeys.³⁶ The experimental group received a 30% caloric reduction in diet, compared to the control group, for 13 to 17 years.³⁴ They concluded that caloric restriction reduced the risk for inflammatory periodontal disease only in male subjects.³⁶

Due to the strong evidence available that indicates vast involvement of the host immune system in the development and progression of periodontal disease,¹¹ it is possible that dietary elements essential for the immune system (such as protein intake) may have some effect on the risk of developing periodontal disease.³⁷

Based on our current knowledge, varying levels of evidence suggest an association between dietary intake and periodontal disease. Based on the limited data available, this association may be due to the direct and indirect effect of nutrients on the oral

flora, oral environment, immune status and systemic inflammation. Hence, we hypothesized that there is positive association between total caloric, carbohydrate and fat, and periodontal disease. To the authors' knowledge, no study has looked at the effect of carbohydrate, fat and total caloric intake on the periodontal status in human subjects. The aim of the present study was to assess if there was an association between periodontal disease and certain dietary aspects (total caloric, carbohydrate and fat intake) using a nationally representative US sample (NHANES data).

Aim and Hypothesis:

Aim: to determine if there was an association between specific aspects of diet (total caloric, carbohydrate and fat intake) and periodontal disease in humans.

Hypothesis: There is a positive association between:

- Periodontal disease and total caloric intake
- Periodontal disease and percentage of calories from carbohydrate
- Periodontal disease and percentage of calories from fat

Research Design and Methods:

This study used data from NHANES 2009-2010.³⁸ NHANES is a cross sectional survey that involves a sample representative of the United States population. In NHANES, calibrated examiners performed the periodontal examination. For participants who were ≥ 30 years old, probing depth (PD), recession, clinical attachment loss (CAL) and gingival bleeding were recorded on six sites per tooth (mid-facial, mid-lingual, mesio-facial, mesio-lingual, disto-facial and disto-lingual) for all the teeth excluding third molars. Individuals with contributory medical history findings were excluded from the examination. Dietary data for a 24-hour period was collected by personal interview. Three to 10 days later, a further 24-hour period data set was collected by a phone interview. Only subjects who completed both interviews were included in the study. Dietary data were averaged, using the two 24-hour surveys.

Independent variables in this study were:

- Total caloric intake
- Percentage of calories from carbohydrate
- Percentage of calories from fat

Periodontitis was defined based on the presence of at least one site with both a PD of ≥ 4 mm and CAL of ≥ 3 mm. This definition has been used in previous NHANES studies; in order to include only what most likely are true periodontitis lesions.^{7,8,10}

Data were adjusted for the following potential confounders: smoking, diabetes, age, sex, race/ethnicity, education, socioeconomic level, waist circumference (WC) and whether a subject has had periodontal treatment before or not.

Participants' smoking status was defined as either current, former or never smoker.

Education was classified as less than, equal to or more than 12 years of education.

Individuals in NHANES 2009-2010 were categorized according to their race as follows:

- Non-Hispanic White
- Non-Hispanic Black
- Mexican-American
- Other Race-including Multi-Racial.
- Other Hispanic

Participants with a history of diabetes were considered diabetics, with the exception of females with a history of gestational diabetes.

The 2010 Dietary Guidelines for Americans³⁹ were used in order to define categories for percentages of calories from both carbohydrate and fat, as follows:

- Percentage of calories acquired from carbohydrate:
 - Less than 45% is a low carbohydrate diet
 - Between 45-65% is an average carbohydrate diet
 - Greater than 65% is a high carbohydrate diet.

- Percentage of calories acquired from fat:
 - Less than 20% is a low fat diet,
 - Between 20-35% is an average fat diet
 - Greater than 35% is a high fat diet.

In order to categorize subjects according to their total caloric intake, the Harris-Benedict equation (reevaluated by Roza and Shizgal) ⁴⁰ which is a formula used to determine an individual's basal metabolic rate (BMR) using his/her weight, height, and age was used. The resulting BMR value is multiplied by a factor that is determined by the level of physical activity. The result is the recommended average daily caloric requirement in order to maintain the current body weight of the individual.

In our study, we used the Harris-Benedict equation to determine the average daily caloric requirement.⁴⁰ If a subject consumed 25% more calories than their average daily requirement, they were considered 'excess caloric intake'. If they consumed less than 75% of their average requirement, they were be considered 'reduced caloric intake'.

Periodontal examination was complete for 4086 individuals. After exclusion of edentulous participants, the sample size decreased to 3743. Having included only subjects who succeeded to complete both 24-hour dietary interviews, 506 individuals were excluded from the sample. Subjects without complete body measurements were also excluded, dropping the sample size to 2644. Physical activity was calculated in this study using information that was collected during the interviews about the

number of days and minutes of vigorous-intensity work, the number of days and minutes of vigorous recreational activities, the number of days and minutes of moderate-intensity work, the number of days and minutes of moderate recreational activities and the number of sedentary activity (details in the data management section). Only subjects with recordable physical activity using this method were included in the sample i.e. 2636 individual.

Power Calculation

Prior to starting the study, power calculation was performed using nQuery Advisor (Version 7.0, Statistical Solutions, Los Angeles, CA). A sample of at least 300 subjects with periodontal disease and 300 subjects without periodontal disease (which is the least anticipated sample size following application of inclusion and exclusion criteria) is adequate to obtain a type I error rate of 5% and a power of 80% assuming an effect size of $\Delta^2 = 0.02$ or greater.

Statistical Analysis

Bivariate analysis between the independent and dependent variables was conducted using Chi square test.

Five logistic regression models were created in order to further examine the relationship between the dependent and independent variables when other covariates were added. The models were as follows:

Model 1: the association between periodontal disease and the following factors: smoking, diabetes, age categories, gender, race/ethnicity, education, ratio of family income to poverty, WC and whether a subject has had periodontal treatment or not.

Model 2: the association between periodontal disease and the following factors: percentage of calories from carbohydrate, smoking, diabetes, age categories, gender, race/ethnicity, education, ratio of family income to poverty, WC and whether a subject has had periodontal treatment or not.

Model 3: the association between periodontal disease and the following factors: percentage of calories from fat, smoking, diabetes, age categories, gender, race/ethnicity, education, ratio of family income to poverty, WC and whether a subject has had periodontal treatment or not.

Model 4: the association between periodontal disease and the following factors: total caloric intake, smoking, diabetes, age categories, gender, race/ethnicity, education, ratio of family income to poverty, WC and whether a subject has had periodontal treatment before or not.

Model 5: the association between periodontal disease and the following factors: percentage of calories from carbohydrate, percentage of calories from fat, total caloric intake, smoking, diabetes, age categories, gender, race/ethnicity, education, ratio of family income to poverty, WC and whether a subject has had periodontal treatment or not.

Data Management:

Data management and analysis were conducted using SAS (Version 9.3, SAS Institute, Cary, NC).

The following files were downloaded from the NHANES website,

<http://www.cdc.gov/nchs/nhanes.htm> 2009-2010 data: ³⁸

DEMO_F.xpt (Demographic Variables and Sample Weights)

DR1TOT_F.xpt (Total Nutrient Intakes -- First Day)

DR2TOT_F.xpt (Total Nutrient Intakes – Second Day)

BMX_F.xpt (Body Measures)

OHXPER_F.xpt (Oral Health – Periodontal)

OHXDEN_F.xpt (Oral Health – Dentition)

OHQ_F.xpt (Oral Health)

DIQ_F.xpt (Diabetes)

PAQ_F.xpt (Physical Activity)

SMQ_F.xpt (Smoking - Cigarette Use)

Files were sorted, and then merged by the variable: **SEQN** (respondent sequence number).

Periodontitis was defined as the presence of at least one site with both a probing depth (PD) of ≥ 4 mm and clinical attachment loss (CAL) of ≥ 3 mm. Each tooth present in the mouth (excluding 3rd molars) had 6 variables that had the PD values. Those variables were extracted from the file **OHXPER_F.xpt**. Variables names are in appendix1.

Similar to PD values, each tooth present in the mouth (excluding 3rd molars) had 6 variables that had the CAL values. Those variables were extracted from the file **OHXPER_F.xpt**. Variables names are in appendix 2.

A new variable was created for each site using an "IF" statement i.e. if a site has a LA value ≥ 3 AND a PD value ≥ 4 , the value for the new variable would be 1; otherwise the value would 0. For each participant, the new variable values were summed up. If the sum value was ≥ 1 , the participant was categorized as having periodontitis. This was done by creating a variable that had a value of 1 if a subject had periodontitis and a value of 0 if the subject did not have periodontitis.

In order to get the number of permanent teeth present, excluding third molars, the following variables were extracted from the file **OHXDEN_F.xpt**:

**OHX02TC, OHX03TC, OHX04TC, OHX05TC, OHX06TC, OHX07TC,
OHX08TC, OHX09TC, OHX10TC, OHX11TC, OHX12TC, OHX13TC,
OHX14TC, OHX15TC, OHX18TC, OHX19TC, OHX20TC, OHX21TC,
OHX22TC, OHX23TC, OHX24TC, OHX25TC, OHX26TC, OHX27TC,
OHX28TC, OHX29TC, OHX30TC, OHX31TC.**

A value of 1 for any of the previous variables indicated the presence of a permanent tooth in that particular site. A new variable for each tooth site was created using an "IF" statement. If the value for any site =1, the new variable's value would be 1. Otherwise, the value of the new variable would be 0. The number of teeth for each individual was obtained by adding the values of the new variable for all the sites.

For the dietary data, each participant's total energy intake was present in the files **DR1TOT_F.xpt** and **DR2TOT_F.xpt**. The variables names were **DR1TKCAL** and **DR2TKCAL**. A new variable was created that contains the sum of the two previous variables divided by 2 i.e. the average total energy in k-calories.

Regarding the percentage of calories from carbohydrate, the following steps were done.

First, the values of the two variable **DR1TCARB** and **DR2TCARB** – (total carbohydrate in grams) were averaged. This value was then multiplied by 4 in order to get the amount of calories a participant gets from carbohydrate.

After that, another variable was created as follows:

- If the value from the first step is < 0.45 of the value of the average total energy ($\sum \text{DR1TKCAL DR2TKCAL}/2$), the new variable will be given the value of 1. (low carbohydrate diet)
- If the value from first step is between ≥ 0.45 and ≤ 0.65 of the value of the average total energy ($\sum \text{DR1TKCAL DR2TKCAL}/2$), the new variable will be given the value of 2 (average carbohydrate diet)
- If the value from first step is > 0.65 of the value of the average total energy ($\sum \text{DR1TKCAL DR2TKCAL}/2$), the new variable will be given the value of 3 (high carbohydrate diet)

Similar steps were done to determine the percentage of calories from fat; as follows: first, the values of the two variable **DR1TTFAT** and **DR2TTFAT** - (total fat in grams.) This value was then multiplied by 9 in order to get the amount of calories a participant gets from fat.

After that, another variable was created as follows:

- If the value from first step is < 0.20 of the value of the average total energy $(\sum \text{DR1TKCAL DR2TKCAL}/2)$, the new variable will be given the value of 1. (low fat diet)
- If the value from first step is ≥ 0.20 and ≤ 0.35 of the value of the average total energy $(\sum \text{DR1TKCAL DR2TKCAL}/2)$, the new variable will be given the value of 2 (average fat diet)
- If the value from first step is > 0.35 of the value of the average total energy $(\sum \text{DR1TKCAL DR2TKCAL}/2)$, the new variable will be given the value of 3 (high fat diet)

More complex steps had to be done in order to study the effect of total caloric intake.

First, a new variable called **BMR** (basal metabolic rate) was created and calculated using the formula:

$88.362 + (13.397 \times \text{weight in kg}) + (4.799 \times \text{height in cm}) - (5.677 \times \text{age in years})$ for male participants (value of 1 for in the variable **RIAGENDR** – Gender)

if a participant had a value of 2 for the variable **RIAGENDR** – Gender (female participant) the following formula $447.593 + (9.247 \times \text{weight in kg}) + (3.098 \times \text{height in cm}) - (4.330 \times \text{age in years})$ was used.

The following variables were used for this calculation:

BMXWT - Weight (kg)

BMXHT - Standing Height (cm)

RIDAGEYR - Age at Screening Adjudicated - Recode

After that, another variable was created that represented the average caloric requirement for each participant. This variable was calculated by multiplying the

BMR value by an activity factor that depends on the individual's level of physical activity (values of 1.2, 1.375, 1.55, 1.725 and 1.9).⁴¹⁻⁴³

In order to determine a participant's level of activity, the following variables were used:

PAQ610 - Days vigorous work

PAD615 - Minutes vigorous-intensity work

PAQ655 - Days vigorous recreational activities

PAD660 - Minutes vigorous recreational activities

PAQ625 - Number of days moderate work

PAD630 - Minutes moderate-intensity work

PAQ670 - Days moderate recreational activities

PAD675 - Minutes moderate recreational activities

PAD680 - Minutes sedentary activities

Those variables are in the file **PAQ_F.xpt** (Physical Activity).

For each subject,

- The value of the variable **PAQ610** was multiplied by the value of the variable **PAD615** to determine weekly minutes of vigorous work
- The value of the variable **PAQ655** was multiplied by the value of the variable **PAD660** to determine weekly minutes of vigorous recreational activities.
- The value of **PAQ625** was multiplied by the value of the variable **PAQ630** to determine the weekly minutes of moderate work.
- The value of the variable **PAQ670** was multiplied by the value of the variable **PAD675** to determine weekly minutes of moderate recreational activities.

For each subject, the values of the weekly minutes of moderate activity and the weekly minutes of moderate recreational activities were divided by 2 and added to the values of weekly minutes of vigorous work and weekly minutes of vigorous recreational activities.

This was done in order to adjust for the difference between the types of activities (moderate vs. vigorous) and to create a single variable that represented the adjusted physical activity for each individual.^{44,45}

Individuals who had any value > 0 for the variable **PAD680**-minutes sedentary activity, and had a zero adjust physical activity, were considered sedentary and their BMR value was multiplied by the activity factor of 1.2.

Participants with adjusted physical activity >0 were categorized into quartiles, as follows:

- Individuals with physical activity value that were in the lowest quartile, had their **BMR** value multiplied by the activity factor of 1.375.
- Individuals with physical activity value that were in the second quartile, had their **BMR** value multiplied by the activity factor of 1.55.
- Individuals with physical activity value that were in the third quartile, had their **BMR** value multiplied by the activity factor of 1.725
- Individuals with physical activity value that were in the highest quintile, had their **BMR** value multiplied by the activity factor of 1.9.

A variable with the result from the previous step determined the recommended average daily caloric requirement in order to maintain the current body weight of each individual. If the value of the average total energy ($\Sigma \text{DR1TKCAL DR2TKCAL}/2$) was:

- < 0.75 of the daily caloric requirement, the subject was categorized as 'reduced calorie intake'.
- ≥ 0.75 and ≤ 1.25 of the daily caloric requirement, the subject was categorized as 'average calorie intake'.
- > 1.25 of the daily caloric requirement, the subject was categorized as 'excess calorie intake'.

The following will explain how the potential confounders variables were categorized.

For Smoking, the following variables were used:

SMQ020 - Smoked at least 100 cigarettes in life

SMQ040 - Do you now smoke cigarettes

Subjects were categorized into:

- Current smoker: value of 1 for **SMQ020**, and value of 1 or 2 for **SMQ040**
- Former smoker: value of 1 for **SMQ020**, and value of 3 for **SMQ040**
- Non smoker: value of 2 for **SMQ020**.

For Diabetes, the following variable was used:

DIQ010 - Doctor told you have diabetes

Subjects were categorized into:

- Diabetics: value of 1 for **DIQ010**
- Non diabetics: value of 2 for **DIQ010**

For age, the following variable was used:

RIDAGEYR - Age at Screening Adjudicated - Recode

Subjects were categorized according to the value of the variable into:

- < 35
- 35>= and < 50
- 50 >= and < 65
- 65>=

For gender, the following variable was used:

RIAGENDR - Gender

Subjects were categorized into:

- Male: value of 1 for RIAGENDR
- Female: value of 2 for RIAGENDR

For race, the following variable were used:

RIDRETH1 - Race/Ethnicity - Recode

Subjects were categorized into:

- Mexican American: value of 1 for **RIDRETH1**
- Other Hispanic: value of 2 for **RIDRETH1**
- Non-Hispanic White: value of 3 for **RIDRETH1**
- Other Hispanic Black: value of 4 for **RIDRETH1**
- Other Race – Including Multi – Racial: value of 5 for **RIDRETH1**

For Education, the following variable was used:

DMDEDUC2 - Education Level - Adults 20+

Subjects will be categorized into:

- Less than 12 years of schooling: value of 1, 2 or 3 for **DMDEDUC2**
- Equal or more than 12 years of schooling: value of 4 or 5 for **DMDEDUC2**

For Socioeconomic level, the following variable was used:

INDFMPIR - Ratio of family income to poverty

Subjects were categorized according to the value of the variable into:

- < 1
- ≥ 1 and < 2
- ≥ 2 and < 3
- ≥ 3 and < 4
- ≥ 4 and

BMXWAIST- Waist Circumference (cm)

Subjects will be categorized into:

- Men:
 - ≤ 102
 - > 102
- Women:
 - ≤ 88
 - > 88

For whether a subject has had periodontal treatment before, the following variable was used:

OHQ850 - Ever had treatment for gum disease?

Subjects were categorized into:

- Yes: value of 1 for **OHQ850**
- No: value of 2 for **OHQ850**

The following weight, strata and cluster variables were used for data analysis.

WTMEC2YR - Full Sample 2 Year MEC Exam Weight

SDMVSTRA - Masked Variance Pseudo-Stratum

DMVPSU - Masked Variance Pseudo-PSU

Results

The number of individuals included in the analysis was 2636 subjects. Table 1 shows the characteristics of the study population. Based on the weighted sample, 52.45% of the study population were females. Seventy three percent of the subjects were categorized as non-Hispanic White. Most individuals belonged to 35-49 age category, followed by the 50-64 age set. Sixty two percent of the participants, completed 12 years or more of schooling. Current smokers constituted 16.8% of the sample while 26.7% of the individuals were former smokers. Individuals who answered 'yes' to the diabetes question were 7.4% of the study subjects. A hundred and fifteen subjects in our sample had missing values for some variables. Diabetes information was missing for 53 individuals, education data was missing for 8 subjects and previous gum treatment data for 56 participants.

Table 2 shows the sample distribution among exposure variables. Only 3.6% of individuals were categorized in the high carbohydrate intake, while 33.1% were in the low carbohydrate category. According to fat intake, 41.5% were in the high group versus 3.5% in the low fat group. Thirteen percent of the sample fell in the excess caloric intake class, while 35% were in the reduced caloric intake group. The mean total caloric intake averaged between the two 24-hours interviews was 2139.3 kcal. The average estimated caloric requirement was 2448.6.8kcal. The mean of the average share of calories from carbohydrate was 48.8%, the range was from 14.2% to 85%. The mean of the average portion of calories from fat was 33.5%, the range was from 6.9% to 68.4%. After adjusting for the difference between intensity of physical activity (1 moderate intensity activity minutes= 1/2 vigorous activity minute) the weekly physical activity minutes ranged from 5 to 6930 (mean=517.3). Table 3

show the distribution of the weighted sample according the values of activity the factor. In addition, it shows the range of adjusted weekly minutes on each quartile.

As a whole, 39.1% of the population were categorized as periodontitis patients (Tables 4-5). Periodontitis was statistically significantly higher in smokers ($p<0.0001$), high WC ($p<0.05$), diabetics ($p<0.05$), individuals with history of gum treatment ($p<0.05$) and male subjects ($p<0.0001$). According to the race/ethnicity, a statistical significance was detected between different groups ($p<0.0001$). Non-Hispanic white had the lowest prevalence of periodontitis while Mexican Americans had the highest prevalence of periodontitis. Individuals who had completed 12 years or more of education, and subjects with higher ratio of family income to poverty, had statistically significantly lower prevalence of periodontitis ($p<0.0001$).

Periodontitis was detected in 40.4% of the low carbohydrate group. That percentage was 38.4 and 39.5 in the average and high carbohydrate groups respectively. No statistical significance was detected in the relationship between carbohydrate intake and periodontal disease. Of the individuals belonging to the average fat intake, 39.6% were periodontitis patients. The periodontitis prevalence was slightly lower (38.6%) in the high fat group, and 36.7% in the low fat group. Statistical significance was not detected. Regarding total caloric intake, prevalence of periodontitis was 40.4%, 37.1% and 44% for the reduced, average and excess categories respectively, with no statistical significance.

Table 6 shows the distribution of the exposure variables in relation to gender. The majority of male and female subjects was in the average categories for all exposure

variables. Statistical significance was detected in the carbohydrate distribution ($p<0.0001$).

Table 7 shows the distribution of the exposure variables in relation to race/ethnicity. A statistical significance was detected for carbohydrate and fat intakes ($p<0.0001$). Table 8 shows the effect of age categories on the exposure variables. Only the fat groups had a statistical significance between them ($p<0.05$).

Tables 9 through 13 show the results of the 5 logistic regression models. In all models, statistical significance for the effect smoking, gender, race/ethnicity, history of periodontal treatment, age and the ratio of family income to poverty was detected. No statistical significance was shown for any of the exposure variables neither separate nor combined in one model. Education had a statistical significance in models 1, 2, 3 and 5 while WC was statistically significant in model 4.

Model 2 showed that individuals in the low carbohydrate group were 17% more likely to have periodontitis when compared to the high carbohydrate group (95% confidence interval(CI) 0.66 to 2.08), while the average carbohydrate group are 13% more likely to have periodontitis when compared to the high carbohydrate group (95% CI 0.68 to 1.88).

Model 3 displayed that individuals in the low fat group are 16% less likely to develop periodontitis when compared to the high fat group (95% CI 0.44 to 1.24), while the average fat group are 4% more likely to have periodontitis when compared to the high fat group (95% CI 0.84 to 1.29).

Model 4 showed that individuals in the reduced calorie group had 10% less the odds of having periodontal disease when compared to the excess calorie group (95% CI

0.68 to 1.19), while the average calorie group are 16% less likely to have periodontitis when compared to the excess calorie group (95% CI 0.61 to 1.15).

Model 5 presented that individuals in the low carbohydrate group had 7% more chance of having periodontal disease when compared to the high carbohydrate group (95% CI 0.56 to 2.01), while the average carbohydrate group are 1% more likely to have periodontitis when compared to the high carbohydrate group (95% CI 0.60 to 1.32). In addition, it showed that individuals in the low fat group had 24% less odds of having periodontal disease when compared to the high fat group (95% CI 0.44 to 1.32), while the average fat group are 7% more likely to have periodontitis when compared to the high fat group (95% CI 0.85 to 1.35). Finally, this model showed that individuals in the reduced calorie group have 10% less odds of having periodontal disease when compared to the excess calorie group (95% CI 0.68 to 1.19), while the average calorie group are 17% less likely to have periodontitis when compared to the high calorie group (95% CI 0.60 to 1.15).

Figures 1 to 3 show the distribution of the exposure variables by periodontitis

Figure 4 shows the sample distribution according to the percentage of calories acquired from carbohydrate (uncategorized). Figure 5 shows the distribution according to the percentage of calories acquired from carbohydrate (uncategorized). Figure 6 shows the distribution of the study population according to percentage of the actual caloric intake by the estimated caloric requirement.

Discussion

In the present study, the effect of carbohydrate, fat and total caloric intake on periodontal disease was studied using data from the NHANES 2009-2010 cycle.

NHANES is a complex multistage survey that includes both a physical examination and health and nutrition questionnaires. Data from the 2009-2010 cycle represent the first NHANES that included full-mouth periodontal examination i.e. 6 sites per tooth, excluding third molars. Eke *et al*² found an increased periodontal disease prevalence when NHANES 2009-2010 data were analyzed compared to previous NHANES.² Their finding came in agreement with publications that quibbled about the efficiency of partial mouth exam, used in the earlier NHANES in representing the true prevalence of periodontal disease status.³⁻⁵

The definition of periodontitis used in this study was the presence of at least one site with both PD \geq 4mm and CAL \geq 3mm. This delineation of periodontitis aims to include only what is considered a true periodontal lesion. In addition, this definition has been used in previous NHANES publications and it appeared to eliminate the confounding effect of age.^{8,46} The prevalence of periodontitis in the weighted sample (N=2636) was 39.1%. When the association between periodontal disease and the selected potential confounders was tested, a statistical significance was detected in all the bivariate analyses conducted between periodontitis and each of the covariates, making our results in agreement with the published literature vis-à-vis risk factors of periodontal disease.^{2,6-10,47}

Male gender is the most common risk factor for periodontal disease.⁴⁷ In the current study, 48.6% of men in the weighted sample had periodontal disease, in contrast to

29.8% of women ($p<0.0001$). This difference in gender has been found in multiple studies, and can be attributed to the social dissimilarities between the two genders as well as certain biological differences.^{4,47,48}

Diabetes is a global public health problem. Approximately, 8.3% of the US population are diabetics.⁵⁰ This percentage goes up to 26.9% among individuals who were 65 years or older.⁵⁰ The deleterious effect of diabetes on the periodontal condition has been shown on cross sectional and longitudinal studies.^{4,47} Periodontal disease on the other hand has been associated with poorer glycemic control in diabetic patients.⁴⁷ Two hundred forty eight individuals were categorized as diabetic in the current study sample, and 52.7% of them were periodontitis patients versus 38.1% among non-diabetic individuals ($p<0.0001$).

Most studies that examined the association between smoking and periodontal disease have found a positive association between them. Smokers are 2 to 7 times more likely to experience periodontal breakdown when compared to non-smokers.⁴ This relationship between smoking and periodontal disease has been presented in both cross-sectional and longitudinal studies.⁴⁷ In addition, there is strong evidence in the literature that smoking cessation slows the pathological periodontal breakdown process.⁴⁹ According to the present study, current smokers had the highest prevalence of periodontitis 59.5% followed by former smokers 41.3%.

When looking at the age categories in the current study sample, it is noticeable that the prevalence of periodontitis increases with age ($p<0.0001$). Individuals who were 65 years and older had a prevalence of 47.8% periodontal disease, while the youngest

age category had a prevalence of 29.9%. This finding is in agreement with the previously published data, and as mentioned earlier, it is not obvious whether this positive association between age and periodontal disease is due to changes occurring in the host or the passage of time.⁴

A negative association has been found between socioeconomic status and the risk of periodontal disease.^{4,51,52} In addition, low education attainment is associated with higher risk of periodontal disease.^{52,53} Our findings agree with these notions, as both socioeconomic status and education level are negatively associated with prevalence of periodontal disease ($p<0.0001$).

Individuals of Black and Mexican heritages have been associated with a higher risk of periodontal disease.^{4,54} In the weighted sample 64.6% of Mexican Americans had periodontal disease. Non-Hispanic Black had the second highest prevalence of periodontal disease, 54.1 %, while non-Hispanic white had the lowest prevalence ($p<0.0001$).

Although obesity was not among the main exposure variables in the current study, it was considered a potential confounder. Obesity was measured in our study using WC. Individuals were divided into either normal WC or high WC. Periodontitis was statistically significantly associated with high WC ($p<0.05$). Obesity has been associated with periodontal disease in both cross sectional (Al-Zahrani et al.) and longitudinal studies (Gorman et al. and Jimenez et al.).^{10,13,14} WC was used in the previous studies as one of the obesity/adiposity measures.

Nutritional epidemiology is a relatively new field that focuses on studying the association between nutritional factors and disease. Studying human nutrition is complex.¹⁷ The huge diversity of dietary intake, the involvement of many confounding factors, the difficulty in obtaining authentic data and the trouble faced with long term follow up, are some of the challenges associated with examining the human diet.^{17, 18} Strong associations in nutritional studies are not commonly found, which are an inherent limitation in this type of research e.g.: a relative risk of 0.7 to 1.5 is considered of great significance.¹⁷

Periodontitis, on the other hand, is a common chronic infectious disease, with several local, systemic and environmental factors contributing to its etiopathology, making it one of the most demanding diseases to examine.

Many reports have been recently published discussing the correlation between periodontal disease and nutritional elements. Most of the published studies looked at vitamins, minerals and/or few food groups.¹⁶ In the current study, the effect of total caloric intake and percentages of carbohydrate and fat in the human diet was examined. The 2010 Dietary guidelines for Americans³⁹ were used in order to categorize individuals' diet with respect to carbohydrate and fat intake. Total caloric intake classification was done relying on the Harris-Benedict equation and the reported physical activity level for each subject. No statistical significance was detected between any of the exposure variables (total caloric intake, percentage of calories from carbohydrate and percentage of calories from fat) and periodontal disease. Even after controlling the effect of covariates in the logistic regression

models, no statistical significance could be detected for the effect of the exposure variable on periodontal disease.

The authors are not familiar with any previous publication that looked at the association between periodontal disease and the above selected aspects of nutrition, therefore, comparison with other studies is not possible. The results of this study, however, must be interpreted with caution. Due to the inherent restrictions of the study design in that it being cross sectional in nature and a diet study, this association may have been weakened. The aspects of diet that we choose, required a 24-hour recall method of data collection. Although this method provides immense details about diet quality and quantity, conducting such surveys require a massive effort and funding. In order to more accurately report total energy intake and macronutrients 4 to 5 24-hour surveys would be preferred.¹⁷ In the current study, only individuals who completed the two 24-hour dietary questionnaire were included and their dietary values were averaged among the two data sets. This should have at least alleviated concerns about diet record accuracy.

Our research question obligated the need for both accurate periodontal and dietary data. The fact that the 2009-2010 NHANES is the only one to date with released data with a full periodontal mouth examination, has limited our ability to combine additional NHANES cycles to increase the sample size. Although, the final sample size of 2636 was relatively large, both periodontal disease and diet are influenced by an endless number of confounding factors. However, there is a major leverage in using NAHNES data set. The accurate data collection, the ability to merge and

analyze future data release and the national representation of this data, are among the strengths of this study.

A limitation of this study was the fact that it looked at both carbohydrate ,and fat intake as a whole. More information should be obtained by investigating the different types of carbohydrate and different types of fat.

As the relationship between diet and disease is almost always not linear, ^{16,17} there is a possibility that periodontal status is not sensitive to the categorization of carbohydrate, fat and total caloric intake in our study. A larger sample size would allow for a more restrictive sample grouping.

Periodontitis and other chronic life threatening conditions such as diabetes and heart diseases share several risk factors.^{55,56} Diet may play a more crucial role in periodontal disease than what our current understanding accepts. Therefore, more research is needed to add to the body of knowledge in the relationship between periodontal disease and dietary factors.

Conclusion

This study looked at the association between periodontal disease and carbohydrate, fat and total caloric intake using data from 2009-2010 NHANES. No statistically significant finding were detected. However, the results of this study cannot be generalized. More research needs to be done to investigate the effect of diet on periodontal status.

Table 1.

Characteristics of study population

	N	Weighted N	SD of Wgt N	Weighted %	SE of Wgt %
Gender					
Male	1316	49869652	3291982	49.55	0.72
Female	1320	50769386	3806652	50.45	0.72
Race/Ethnicity					
Mexican American	486	7862889	1635654	7.8130	2.0250
Other Hispanic	277	4703016	1197961	4.6732	1.2562
Non-Hispanic White	1369	73543217	8021325	73.0762	3.2882
Non-Hispanic Black	387	8603621	819654	8.5490	0.7999
Other incl Multiracial	117	5926293	891284	5.8887	0.9615
Age					
30-34	290	11733526	961260	11.66	0.64
35-49	939	38656953	2448800	38.41	1.63
50-64	792	32455551	2851202	32.25	1.30
65+	615	17793006	1920384	17.68	0.96
Hx of gum Tx					
No	1980	78616080	6409447	79.31	2.03
Yes	600	20506531	2239424	20.69	2.03
N of missing = 56				.	
Ratio of family Income to poverty					
<1	643	16044332	1022112	15.94	1.35
>=1 and <2	601	15706330	1276413	15.61	0.71
>=2 and <3	376	14370829	1502777	14.28	1.05
>=3 and <4	275	12721733	1902079	12.64	1.32
>=4	741	41795814	3395979	41.53	1.29
Smoking					
None	1437	56886459	3723478	56.53	1.79
Current	516	16932765	1295782	16.83	0.76
Former	683	26819814	3065323	26.65	1.78
Diabetes					
No	2335	91884905	6554192	93.0287	0.5327
Yes	248	6885545	521423	6.9713	0.5327
N of missing = 53					
WC					
Normal	1217	49624401	3557721	49.31	1.48
High	1419	51014636	4012129	50.69	1.48
Education					
<12 years	1257	37935002	3051255	37.78	1.62
>=12 years	1371	62472950	4675494	62.22	1.62
N of missing = 8					

Table 2.

Distribution of study population according to the exposure variables

	N	Weighted %	SE of Wgt %
Carbohydrate intake			
Low	775	33.12	1.57
Average	1739	63.30	1.43
High	122	3.58	0.42
Fat intake			
Low	117	3.51	0.41
Average	1529	55.04	1.73
High	990	41.45	1.78
Total caloric intake			
Reduced	967	34.98	1.16
Average	1325	51.89	0.88
Excess	344	13.13	1.11

*Based on the percentage of calories from carbohydrate: less than 45% is a low carbohydrate diet, between 45-65% is an average carbohydrate diet, and greater than 65% is a high carbohydrate diet.

** Based on the percentage of calories from fat: less than 20% is a low fat diet, between 20-35% is an average fat diet and greater than 35% is a high fat diet.

*** Based on the estimated caloric requirement versus actual total caloric intake.

Table 3.

Sample distribution among the activity factor values

Activity Factor	N	Weighted %	SE of Wgt %	Range of adjusted weekly minutes*
1.2	848	26.65	1.66	N/A
1.375	463	18.07	0.84	5 and 90
1.55	434	18.79	0.88	>90 and 250
1.725	444	19.38	1.13	>250 and 665
1.9	447	17.11	0.93	>665 and 6930

* 1 moderate intensity physical activity = ½ vigorous intensity physical activity

Table 4.

Overall prevalence of periodontitis

Periodontitis	N	Weighted N	SD of Wgt N	%	SE of %
No	1384	61275080	4895638	60.89	1.65
Yes	1252	39363957	2847612	39.11	1.65
Total	2636	100639037	6965824	100.000	

Table 5.

Prevalence of periodontitis among study population categories

	N	Weighted %	SE of weighted %	Prevalence of periodontal disease	SE of Prevalence of periodontal disease	P Value
Carbohydrate						0.6215
Low	775	33.12	1.57	40.42	1.56	
Average	1739	63.30	1.43	38.41	2.12	
High	122	3.58	0.42	39.47	5.10	
Fat						0.8066
Low	117	3.51	0.41	36.78	6.03	
Average	1529	55.04	1.73	39.62	1.79	
High	990	41.45	1.78	38.64	1.99	
Caloric Intake						0.2437
Reduced	967	34.98	1.16	40.35	2.83	
Average	1325	51.89	0.88	37.05	2.00	
Excess	344	13.13	1.11	43.98	3.96	
Smoking						<0.0001
None	1437	56.5252	1.7857	32.0369	2.2778	
Current	516	16.8252	0.7607	59.4512	2.7766	
Former	683	26.6495	1.7803	41.2851	2.4542	
Diabetes						0.0040
No	2335	93.03	0.53	38.07	1.81	
Yes	248	6.97	0.53	52.67	4.38	
WC						0.0342
Normal	1217	49.31	1.48	36.65	2.06	
High	697	21.04	1.13	41.51	1.95	
Education						<0.0001
<12 years	1	1257	37.7809	1.6179	50.80	2.4620
>= 12 years	1	525	19.8843	0.9646	31.96	1.5479
Gender						<0.0001
Male	1316	49.55	0.72	48.62	1.70	
Female	1320	50.45	0.72	29.78	2.20	

Cont. Table 5.

Prevalence of periodontitis among study population categories

	N	Weighted %	SE of weighted %	Prevalence of periodontal disease	SE of Prevalence of periodontal disease	P Value
Race/Ethnicity						<0.0001
Mexican American	486	7.8130	2.0250	64.5559	1.7370	
Other Hispanic	277	4.6732	1.2562	45.9151	2.6809	
Non-Hispanic White	1369	73.0762	3.2882	34.6353	1.8752	
Non-Hispanic Black	387	8.5490	0.7999	54.1438	3.4210	
Other incl Multiracial	117	5.8887	0.9615	33.7206	4.1681	
Age						<0.0001
30-34	290	11.66	0.64	29.94	3.38	
35-49	939	38.41	1.63	33.11	2.11	
50-64	792	32.25	1.30	44.82	3.07	
65+	615	17.68	0.96	47.80	3.28	
Hx of gum Tx						0.0097
No	1980	79.31	2.03	36.19	1.92	
Yes	600	20.69	2.03	50.05	4.70	
Ratio of family income to poverty						<0.0001
<1	643	15.94	1.35	52.9	1.72	
>=1 and <2	601	15.61	0.71	51.98	3.25	
>=2 and <3	376	14.28	1.05	42.66	2.76	
>=3 and <4	275	12.64	1.32	36.37	3.20	
>=4	741	41.53	1.29	28.59	2.41	

*Based on the percentage of calories from carbohydrate: less than 45% is a low carbohydrate diet, between 45-65% is an average carbohydrate diet, and greater than 65% is a high carbohydrate diet.

** Based on the percentage of calories from fat: less than 20% is a low fat diet, between 20-35% is an average fat diet and greater than 35% is a high fat diet.

*** Based on the estimated caloric requirement versus actual total caloric intake.

Table 6.

Distribution of study population according to exposure variables and gender

Gender	Carbohydrate Intake*	N	Wgt %	SE	Fat Intake**	N	Wgt %	SE	Tot caloric Intake***	N	Wgt %	SE
Male	Low	449	37.91	2.06	Low	60	3.12	0.37	Reduced	467	34.27	1.75
	Average	817	59.34	2.16	Average	748	54.15	2.67	Average	665	51.85	1.73
	High	50	2.75	0.49	High	508	42.74	2.61	Excess	184	13.88	1.43
Female	Low	326	28.41	1.55	Low	57	3.90	0.72	Reduced	500	35.68	1.26
	Average	922	67.19	1.36	Average	781	55.91	1.49	Average	660	51.93	1.14
	High	72	4.40	0.67	High	482	40.19	1.36	Excess	160	12.39	1.25
	<i>P</i> <0.0001				<i>P</i> =0.9963				<i>P</i> =0.5596			

*Based on the percentage of calories from carbohydrate: less than 45% is a low carbohydrate diet, between 45-65% is an average carbohydrate diet, and greater than 65% is a high carbohydrate diet.

** Based on the percentage of calories from fat: less than 20% is a low fat diet, between 20-35% is an average fat diet, and greater than 35% is a high fat diet.

*** Based on the estimated caloric requirement versus actual total caloric intake.

Table 7.

Distribution of study population according to exposure variables and race/ethnicity

Race/Ethnicity	Carbohydrate Intake*	N	Wgt %	SE	Fat Intake**	N	Wgt %	SE	Tot caloric Intake***	N	Wgt %	SE
Mexican American	Low	104	22.39	1.97	Low	28	5.07	0.93	Reduced	178	36.23	2.32
	Average	350	72.03	2.24	Average	327	68.90	1.76	Average	234	48.50	2.56
	High	32	5.58	1.13	High	131	26.03	1.55	Excess	74	15.27	1.57
Other Hispanic	Low	47	17.72	2.43	Low	18	7.16	2.06	Reduced	124	42.47	2.82
	Average	215	77.27	2.13	Average	188	66.17	3.14	Average	130	48.85	2.61
	High	15	5.00	1.27	High	71	26.67	2.57	Excess	23	8.68	2.47
Non Hispanic White	Low	476	36.08	2.02	Low	44	2.71	0.44	Reduced	480	34.52	1.36
	Average	850	61.18	1.87	Average	710	51.78	2.15	Average	708	52.32	1.18
	High	43	2.73	0.40	High	615	45.51	2.28	Excess	181	13.15	1.56
Non Hispanic Black	Low	121	30.72	2.64	Low	17	4.46	0.77	Reduced	150	39.85	2.37
	Average	245	63.67	3.12	Average	227	59.1	3.47	Average	184	46.51	2.07
	High	21	5.61	1.19	High	143	36.36	2.92	Excess	53	13.64	1.90
Other incl Multirac	Low	27	26.23	4.56	Low	10	7.12	3.14	Reduced	35	25.95	4.17
	Average	79	66.41	5.08	Average	77	62.22	6.61	Average	69	61.31	3.58
	High	11	7.36	1.92	High	30	30.66	5.49	Excess	13	12.74	4.59
	<i>P<0.0001</i>				<i>P<0.0001</i>				<i>P=0.0863</i>			

*Based on the percentage of calories from carbohydrate: less than 45% is a low carbohydrate diet, between 45-65% is an average carbohydrate diet, and greater than 65% is a high carbohydrate diet.

** Based on the percentage of calories from fat: less than 20% is a low fat diet, between 20-35% is an average fat diet, and greater than 35% is a high fat diet.

*** Based on the estimated caloric requirement versus actual total caloric intake.

Table 8.

Distribution of study population according to exposure variables and age categories

Age	Carbohydrate Intake	N	Wgt %	SE	Fat Intake	N	Wgt %	SE	Tot Caloric Intake	N	Wgt %	SE
30-34	Low	84	30.21	2.71	Low	19	5.56	1.86	Reduced	95	31.39	1.89
	Average	190	64.32	2.55	Average	180	61.08	3.05	Average	162	56.52	2.37
	High	16	5.47	1.38	High	91	33.36	2.95	Excess	33	12.09	1.68
35-49	Low	269	32.18	1.53	Low	41	3.68	0.53	Reduced	360	37.78	2.02
	Average	627	64.06	1.45	Average	565	57.15	2.95	Average	447	49.08	1.36
	High	43	3.76	0.65	High	333	39.16	2.87	Excess	132	13.14	1.98
50-64	Low	251	35.82	3.18	Low	37	3.39	0.70	Reduced	302	34.49	1.66
	Average	497	60.84	2.95	Average	443	51.37	2.09	Average	391	52.21	1.65
	High	44	3.34	0.72	High	312	45.24	2.00	Excess	99	13.29	1.57
65+	Low	171	32.14	1.84	Low	20	2.00	0.63	Reduced	210	32.14	2.13
	Average	425	65.48	1.82	Average	341	53.15	1.53	Average	325	54.37	1.63
	High	19	2.38	0.79	High	254	44.85	1.71	Excess	80	13.50	1.81
	<i>P=0.2357</i>					<i>P=0038</i>			<i>P=0.2891</i>			

Table 9. Logistic regression model 1.

Effect	OR	95% Wald Confidence Limits		P Value
Smoking (none vs former)	0.802	0.651	0.988	<.0001
Smoking (current vs former)	2.307	1.714	3.106	<.0001
diabetes (no vs yes)	0.888	0.617	1.278	0.5215
Education (<12 Y vs >=12)	1.436	1.224	1.685	<.0001
WC (normal vs high)	0.796	0.628	1.009	0.0596
Gender (male vs female)	2.689	2.141	3.377	<.0001
Race/ethnicity* (MA vs ORM)	2.816	2.043	3.882	<.0001
Race/ethnicity* (OH vs ORM)	1.434	1.012	2.033	0.8634
Race/Ethnicity* (NHW vs ORM)	0.901	0.618	1.315	<.0001
Race/Ethnicity* (NHB vs ORM)	1.830	1.282	2.612	0.0597
Had gum Tx before (No vs yes)	0.630	0.419	0.946	0.0259
Age (30-34 vs 65+)	0.290	0.185	0.455	<.0001
Age (35-49 vs 65+)	0.408	0.271	0.615	0.0004
Age (50 vs 65+)	0.854	0.550	1.325	0.0005
Ratio of f income to poverty (<1 vs >=4)	1.870	1.361	2.568	0.0306
Ratio of f income to poverty (>=1 to<2 vs >=4)	2.095	1.547	2.837	0.0040
Ratio of f income to poverty (>=2 to<3 vs >=4)	1.554	1.017	2.374	0.7302
Ratio of f income to poverty (>=3 to<4 vs >=4)	1.187	0.849	1.661	0.1057

Effect	DF	Wald Chi-Square	P Value
Smoking	2	32.3628	<.0001
Diabetes	1	0.4108	0.5215
Education	1	19.6780	<.0001
WC	1	3.5497	0.0596
Gender	1	72.4160	<.0001
Race/Ethnicity	4	109.7046	<.0001
Hx of gum Tx	1	4.9658	0.0259
Age	3	46.7408	<.0001
Ratio of f income to poverty	4	33.2194	<.0001

* MA=Mexican American, OH=Other Hispanic, NHW=Non-Hispanic White,
NHB=Other Hispanic Black , ORM=Other Race – including Multiracial

Table 10. Logistic regression model 2.

Effect	OR	95% Wald Confidence Limits		P Value
Carbohydrate* (low vs high)	1.168	0.655	2.084	0.6080
Carbohydrate* (average vs high)	1.129	0.677	1.883	0.7594
Smoking (none vs former)	0.806	0.654	0.995	<.0001
Smoking (current vs former)	2.314	1.719	3.117	<.0001
diabetes (no vs yes)	0.892	0.624	1.276	0.5326
Education (<12 Y vs ≥12)	1.436	1.223	1.686	<.0001
WC (normal vs high)	0.797	0.628	1.012	0.0628
Gender (male vs female)	2.677	2.143	3.345	<.0001
Race/ethnicity** (MA vs ORM)	2.807	2.029	3.884	<.0001
Race/ethnicity** (OH vs ORM)	1.432	1.003	2.043	0.8780
Race/Ethnicity** (NHW vs ORM)	0.894	0.604	1.323	<.0001
Race/Ethnicity** (NHB vs ORM)	1.820	1.265	2.618	0.0634
Had gum Tx before (No vs yes)	0.629	0.417	0.947	0.0265
Age (30-34 vs 65+)	0.291	0.185	0.457	<.0001
Age (35-49 vs 65+)	0.408	0.270	0.616	0.0004
Age (50 vs 65+)	0.853	0.548	1.329	0.0005
Ratio of f income to poverty (<1 vs ≥4)	1.880	1.355	2.608	0.0321
Ratio of f income to poverty (≥1 to <2 vs ≥4)	2.113	1.547	2.887	0.0036
Ratio of f income to poverty (≥2 to <3 vs ≥4)	1.561	1.025	2.377	0.7195
Ratio of f income to poverty (≥3 to <4 vs ≥4)	1.187	0.851	1.656	0.0952

Effect	DF	Wald Chi-Square	P Value
Carbohydrate*	2	0.2790	0.8698
Smoking	2	33.2221	<.0001
Diabetes	1	0.3895	0.5326
Education	1	19.5380	<.0001
WC	1	3.4618	0.0628
Gender	1	75.2219	<.0001
Race/Ethnicity	4	110.4463	<.0001
Hx of gum Tx	1	4.9248	0.0265
Age	3	46.4000	<.0001
Ratio of f income to poverty	4	30.2341	<.0001

*Based on the percentage of calories from carbohydrate: less than 45% is a low carbohydrate diet, between 45-65% is an average carbohydrate diet, and greater than 65% is a high carbohydrate diet.

** MA=Mexican American, OH=Other Hispanic, NHW=Non-Hispanic White,
NHB=Other Hispanic Black, ORM=Other Race – including Multiracial

Table 11. Logistic regression model 3

Effect	OR	95% Wald Confidence Limits		P Value
Fa*t (low vs high)	0.742	0.444	1.241	0.2588
Fat* (average vs high)	1.041	0.840	1.291	0.3239
Smoking (none vs former)	0.802	0.650	0.990	<.0001
Smoking (current vs former)	2.318	1.728	3.109	<.0001
diabetes (no vs yes)	0.889	0.619	1.276	0.5227
Education (<12 Y vs >=12)	1.441	1.224	1.696	<.0001
WC (normal vs high)	0.794	0.623	1.011	0.0616
Gender (male vs female)	2.690	2.148	3.368	<.0001
Race/ethnicity** (MA vs ORM)	2.771	2.004	3.831	<.0001
Race/ethnicity** (OH vs ORM)	1.421	0.989	2.042	0.8722
Race/Ethnicity** (NHW vs ORM)	0.889	0.603	1.310	<.0001
Race/Ethnicity** (NHB vs ORM)	1.804	1.257	2.589	0.0638
Had gum Tx before (No vs yes)	0.629	0.418	0.948	0.0266
Age (30-34 vs 65+)	0.292	0.186	0.458	<.0001
Age (35-49 vs 65+)	0.409	0.271	0.616	0.0003
Age (50 vs 65+)	0.856	0.550	1.331	0.0004
Ratio of f income to poverty (<1 vs >=4)	1.876	1.373	2.564	0.0282
Ratio of f income to poverty (>=1 to<2 vs >=4)	2.106	1.555	2.851	0.0036
Ratio of f income to poverty (>=2 to<3 vs >=4)	1.550	1.025	2.345	0.7467
Ratio of f income to poverty (>=3 to<4 vs >=4)	1.189	0.850	1.664	0.1059

Effect	DF	Wald Chi-Square	P Value
Fat	2	1.2955	0.5232
Smoking	2	33.1325	<.0001
Diabetes	1	0.4086	0.5227
Education	1	19.2289	<.0001
WC	1	3.4945	0.0616
Gender	1	74.2906	<.0001
Race/Ethnicity	4	112.2973	<.0001
Hx of gum Tx	1	4.9169	0.0266
Age	3	47.4554	<.0001
Ratio of f income to poverty	4	33.3053	<.0001

* Based on the percentage of calories from fat: less than 20% is a low fat diet, between 20-35% is an average fat diet and greater than 35% is a high fat diet.

** MA=Mexican American, OH=Other Hispanic, NHW=Non-Hispanic White, NHB=Other Hispanic Black, ORM=Other Race – including Multiracial

Table 12. Logistic regression model 4

Effect	OR	95% Wald Confidence Limits		P Value
Total Caloric intake* (reduced vs excess)	0.901	0.681	1.192	0.9084
Total Caloric intake* (average vs excess)	0.837	0.611	1.147	0.4083
Smoking (none vs former)	0.807	0.652	0.998	<.0001
Smoking (current vs former)	2.281	1.673	3.112	<.0001
diabetes (no vs yes)	0.879	0.616	1.255	0.4778
Education (<12 Y vs ≥12)	0.794	0.624	1.011	0.0609
WC (normal vs high)	1.441	1.223	1.699	<.0001
Gender (male vs female)	2.691	2.142	3.381	<.0001
Race/ethnicity** (MA vs ORM)	2.773	2.004	3.838	<.0001
Race/ethnicity** (OH vs ORM)	1.421	1.008	2.003	0.8668
Race/Ethnicity** (NHW vs ORM)	0.892	0.617	1.291	<.0001
Race/Ethnicity** (NHB vs ORM)	1.808	1.281	2.553	0.0618
Had gum Tx before (No vs yes)	0.629	0.420	0.944	0.0251
Age (30-34 vs 65+)	0.292	0.186	0.458	<.0001
Age (35-49 vs 65+)	0.408	0.272	0.612	0.0003
Age (50 vs 65+)	0.854	0.551	1.323	0.0005
Ratio of f income to poverty (<1 vs ≥4)	1.864	1.357	2.561	0.0300
Ratio of f income to poverty (≥1 to <2 vs ≥4)	2.082	1.547	2.803	0.0038
Ratio of f income to poverty (≥2 to <3 vs ≥4)	1.547	1.018	2.351	0.7360
Ratio of f income to poverty (≥3 to <4 vs ≥4)	1.184	0.839	1.670	0.1122

Effect	DF	Wald Chi-Square	P Value
Total caloric intake*	2	1.3589	0.5069
Smoking	2	28.8983	<.0001
Diabetes	1	0.5040	0.4778
Education	1	3.5124	0.0609
WC	1	18.9798	<.0001
Gender	1	72.2758	<.0001
Race/Ethnicity	4	99.6907	<.0001
Hx of gum Tx	1	5.0172	0.0251
Age	3	47.7945	<.0001
Ratio of f income to poverty	4	32.4814	<.0001

* Based on the estimated caloric requirement versus actual total caloric intake.

** MA=Mexican American, OH=Other Hispanic, NHW=Non-Hispanic

White, NHB=Other Hispanic Black, ORM=Other Race – including Multiracial

Table 13. Logistic regression model 5

Effect	OR	95% Wald Confidence Limits		P Value
Carbohydrate *(low vs high)	1.077	0.577	2.010	0.7376
Carbohydrate *(average vs high)	1.011	0.601	1.700	0.8497
Fat** (low vs high)	0.758	0.436	1.318	0.2988
Fat** (average vs high)	1.073	0.852	1.351	0.3019
Total Caloric intake *** (reduced vs excess)	0.902	0.682	1.194	0.9397
Total Caloric intake *** (average vs excess)	0.831	0.602	1.148	0.3812
Smoking (none vs former)	0.812	0.657	1.003	<.0001
Smoking (current vs former)	2.290	1.684	3.113	<.0001
diabetes (no vs yes)	0.882	0.620	1.255	0.4865
Education (<12 Y vs ≥12)	1.445	1.222	1.709	<.0001
WC (normal vs high)	0.794	0.622	1.013	0.0633
Gender (male vs female)	2.679	2.151	3.338	<.0001
Race/ethnicity† (MA vs ORM)	2.725	1.952	3.804	<.0001
Race/ethnicity† (OH vs ORM)	1.410	0.976	2.035	0.8914
Race/Ethnicity† (NHW vs ORM)	0.879	0.598	1.293	<.0001
Race/Ethnicity† (NHB vs ORM)	1.777	1.244	2.538	0.0689
Had gum Tx before (No vs yes)	0.628	0.417	0.945	0.0259
Age (30-34 vs 65+)	0.293	0.186	0.462	<.0001
Age (35-49 vs 65+)	0.407	0.271	0.611	0.0003
Age (50 vs 65+)	0.855	0.548	1.332	0.0005
Ratio of f income to poverty (<1 vs ≥4)	1.881	1.359	2.604	0.0288
Ratio of f income to poverty (≥1 to <2 vs ≥4)	2.105	1.550	2.859	0.0032
Ratio of f income to poverty (≥2 to <3 vs ≥4)	1.553	1.029	2.344	0.7297
Ratio of f income to poverty (≥3 to <4 vs ≥4)	1.182	0.841	1.660	0.1001

Effect	DF	Wald Chi-Square	P Value
Carbohydrate	2	0.2199	0.8959
Fat	2	1.1198	0.5713
Total caloric intake	2	1.3647	0.5054
Smoking	2	30.0897	<.0001
Diabetes	1	0.4842	0.4865
Education	1	18.4760	<.0001
WC	1	3.4486	0.0633
Gender	1	77.2660	<.0001
Race/Ethnicity	4	100.1813	<.0001
Hx of gum Tx	1	4.9655	0.0259
Age	3	48.5212	<.0001
Ratio of f income to poverty	4	30.4422	<.0001

*Based on the percentage of calories from carbohydrate: less than 45% is a low carbohydrate diet, between 45-65% is an average carbohydrate diet, and greater than 65% is a high carbohydrate diet.

** Based on the percentage of calories from fat: less than 20% is a low fat diet, between 20-35% is an average fat diet and greater than 35% is a high fat diet.

*** Based on the estimated caloric requirement versus actual total caloric intake.

† MA=Mexican American, OH=Other Hispanic, NHW=Non-Hispanic White, NHB=Other Hispanic Black, ORM=Other Race – including Multiracial

Figure 1.

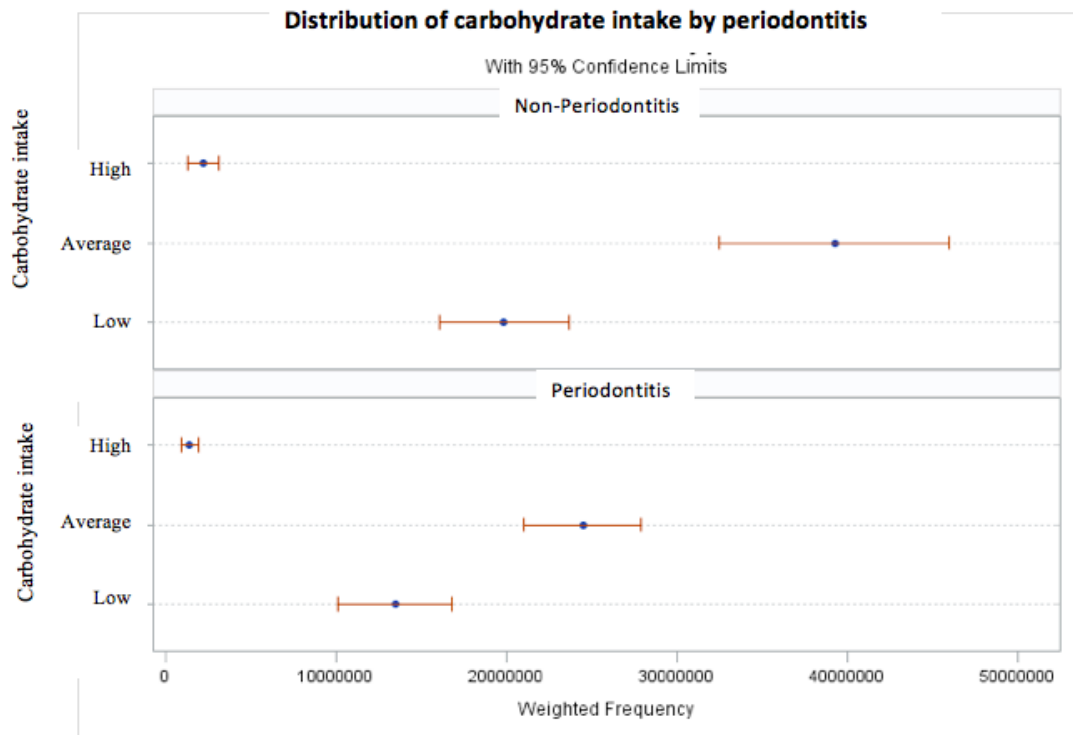


Figure 2.

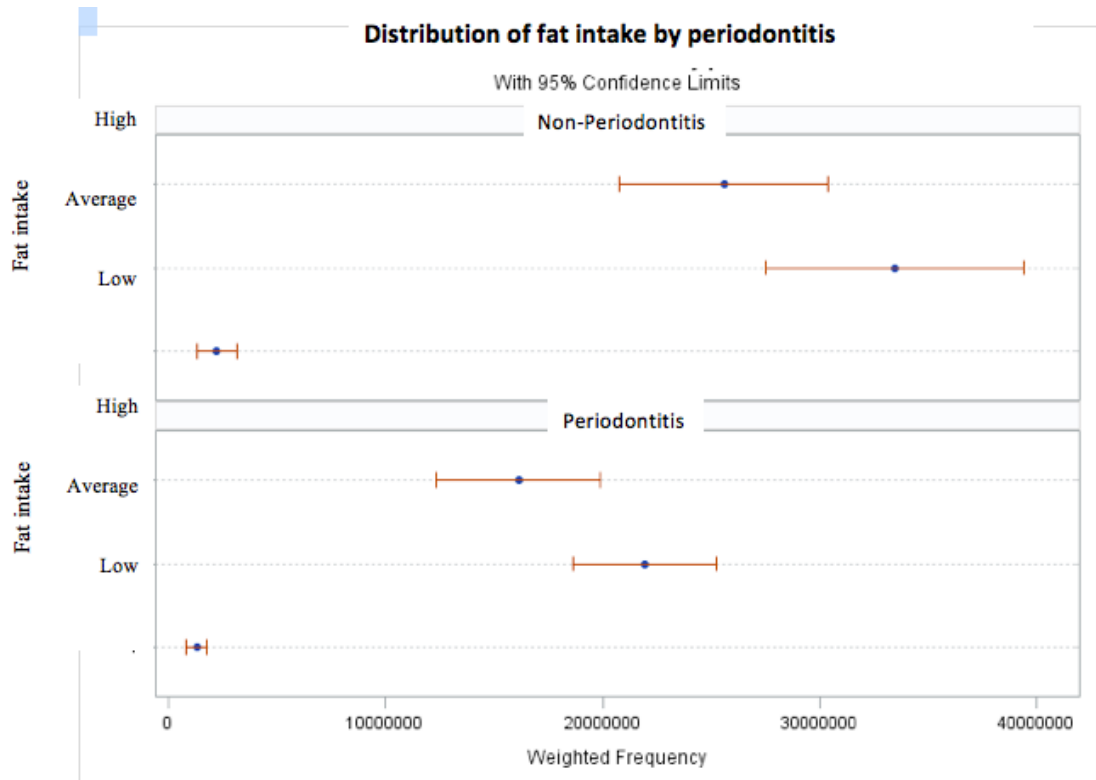


Figure 3.

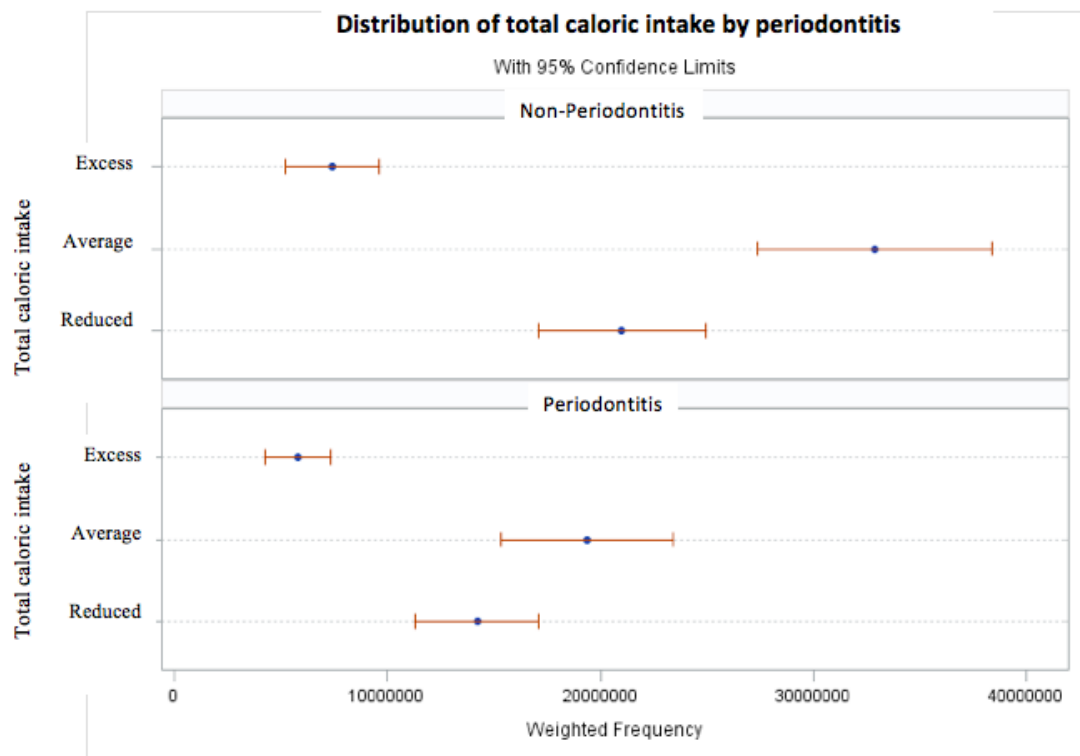


Figure 4.

Distribution according to % of calories acquired from carbohydrate (uncategorized)

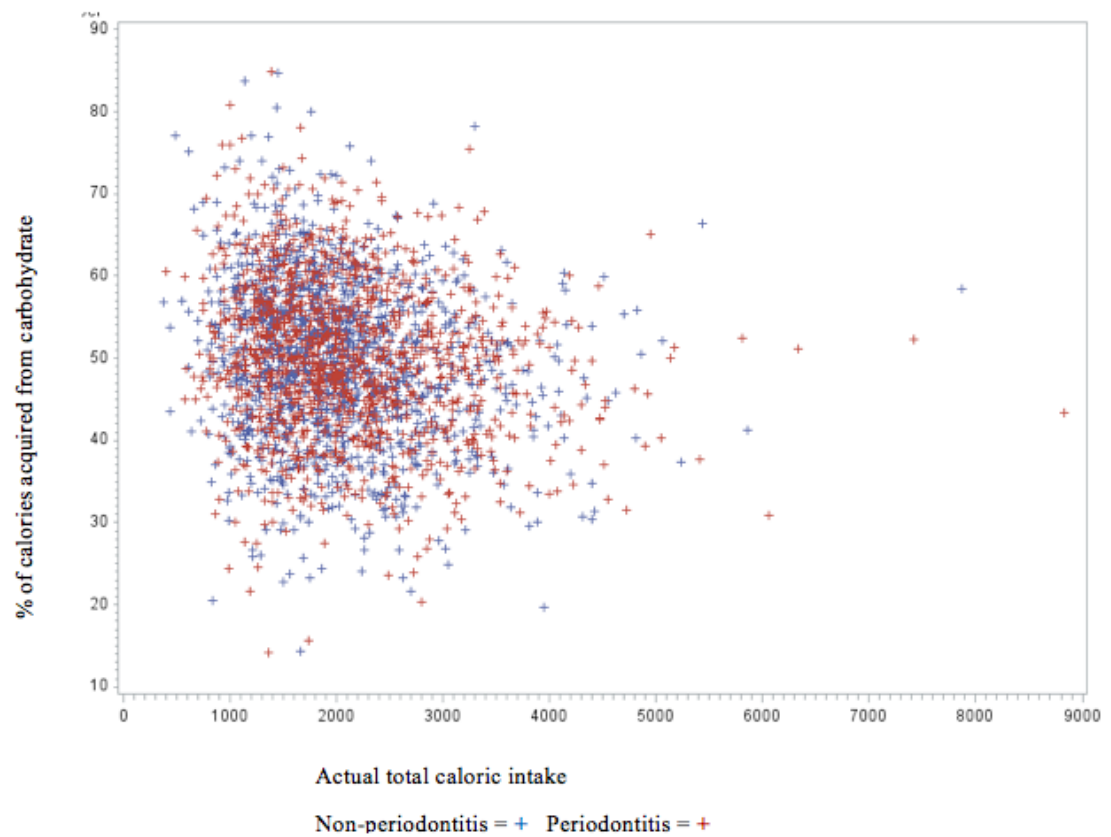


Figure 5.

Distribution according to % of calories acquired from fat (uncategorized)

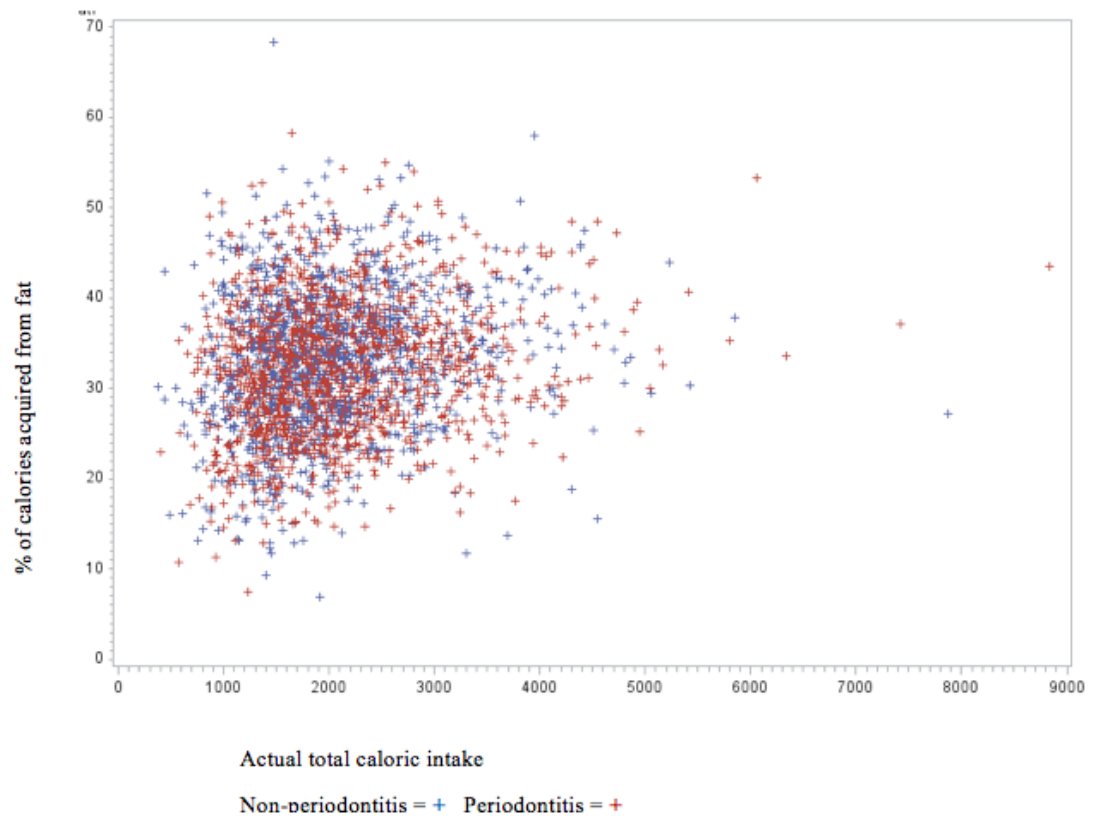
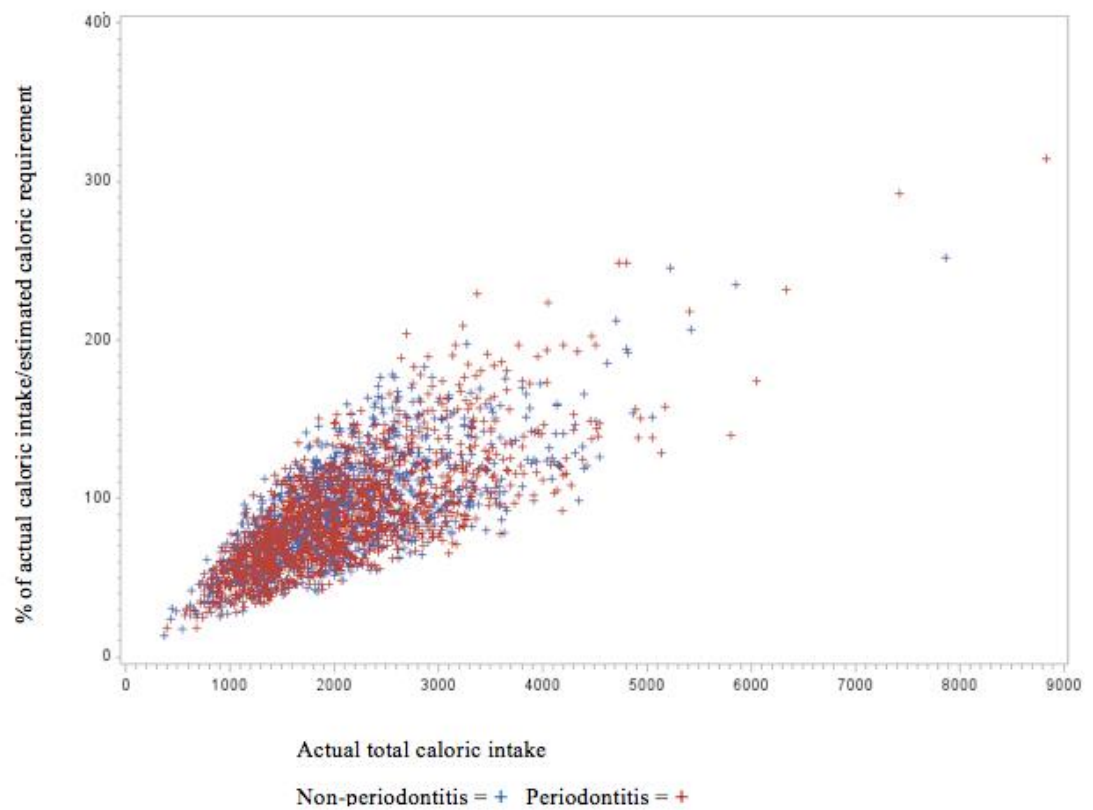


Figure 6.

Distribution according to % of actual caloric intake/estimated caloric requirement



References:

1. Newman MG, Takei HH, Klokkevold PR, Carranza FA. Carranza's Clinical Periodontology. 10th Edition 2006. Saunders.
2. Eke PI, Dye BA, Wei L, Thornton-Evans GO, Genco RJ. Prevalence of Periodontitis in Adults in the United States: 2009 and 2010. J Dent Res. 2012 Oct;91(10):914-920.
3. Papapanou PN. The prevalence of periodontitis in the US: forget what you were told. J Dent Res. 2012 Oct;91(10):907-8.
4. Albandar JM. Underestimation of periodontitis in NHANES surveys. J Periodontol. 2011 Mar;82(3):337-41.
5. Eke PI, Thornton-Evans GO, Wei L, Borgnakke WS, Dye BA. Accuracy of NHANES periodontal examination protocols. J Dent Res. 2010 Nov;89(11):1208-13.
6. Albandar JM. Epidemiology and risk factors of periodontal diseases. Dent Clin North Am. 2005 Jul;49(3):517-32, v-vi.
7. Al-Zahrani MS, Borawski EA, Bissada NF. Increased physical activity reduces prevalence of periodontitis. J Dent. 2005 Oct;33(9):703-10.
8. Al-Zahrani MS, Borawski EA, Bissada NF. Periodontitis and three health-enhancing behaviors: maintaining normal weight, engaging in recommended level of exercise, and consuming a high-quality diet. J Periodontol. 2005 Aug;76(8):1362-6.
9. Al-Zahrani MS, Bissada NF, Borawski EA. Diet and periodontitis. J Int Acad Periodontol. 2005 Jan;7(1):21-6.
10. Al-Zahrani MS, Bissada NF, Borawski EA. Obesity and periodontal disease in young, middle-aged, and older adults. J Periodontol. 2003 May;74(5):610-5.
11. Schenkein H, Cochran DL, Van Dyke TE, Blieden T, Cohen RE; Hallmon WW, Hinrichs JE, Mariotti A, Raulin LA, Somerman MJ; Genco RJ, Greenstein G, Iacono VJ. The pathogenesis of periodontal diseases. J Periodontol. 1999 Apr;70(4):457-70.
12. Burt B; Research, Science and Therapy Committee of the American Academy of Periodontology. Position paper: epidemiology of periodontal diseases. J Periodontol. 2005 Aug;76(8):1406-19.
13. Gorman A, Kaye EK, Nunn M, Garcia RI. Changes in body weight and adiposity predict periodontitis progression in men. J Dent Res. 2012 Oct;91(10):921-6.
14. Jimenez M, Hu FB, Marino M, Li Y, Joshupura KJ. Prospective associations between measures of adiposity and periodontal disease. Obesity (Silver Spring). 2012 Aug;20(8):1718-25.

15. The website for MyPlate, the United State Department of Agriculture.
<http://www.choosemyplate.gov/>
Last visited: October 2013
16. Kaye EK. Nutrition, dietary guidelines and optimal periodontal health. *Periodontol* 2000. 2012 Feb;58(1):93-111.
17. Willett W. Nutritional epidemiology, 3rd Edition 2012. Oxford.
18. Hu F. Obesity Epidemiology, 1st Edition 2008. Oxford.
19. Stein SH, Tipton DA. Vitamin D and its impact on oral health--an update. *J Tenn Dent Assoc*. 2011 Spring;91(2):30-3; quiz 34-5.
20. Bashutski JD, Eber RM, Kinney JS, Benavides E, Maitra S, Braun TM, Giannobile WV, McCauley LK. The impact of vitamin D status on periodontal surgery outcomes. *J Dent Res*. 2011 Aug;90(8):1007-12. Epub 2011 May 9.
21. Boggess KA, Espinola JA, Moss K, Beck J, Offenbacher S, Camargo CA Jr. Vitamin D status and periodontal disease among pregnant women. *J Periodontol*. 2011 Feb;82(2):195-200.
22. Garcia MN, Hildebolt CF, Miley DD, Dixon DA, Couture RA, Spearie CL, Langenwaller EM, Shannon WD, Deych E, Mueller C, Civitelli R. One-year effects of vitamin D and calcium supplementation on chronic periodontitis. *J Periodontol*. 2011 Jan;82(1):25-32.
23. Jabbar S, Drury J, Fordham J, Datta HK, Francis RM, Tuck SP. Plasma vitamin D and cytokines in periodontal disease and postmenopausal osteoporosis. *J Periodontal Res*. 2011 Feb;46(1):97-104.
24. Al-Zahrani MS, Borawski EA, Bissada NF. Poor overall diet quality as a possible contributor to calculus formation. *Oral Health Prev Dent*. 2004;2(4):345-9.
25. Yoshihara A, Watanabe R, Hanada N, Miyazaki H. A longitudinal study of the relationship between diet intake and dental caries and periodontal disease in elderly Japanese subjects. *Gerodontology*. 2009 Jun;26(2):130-6.
26. Merchant AT, Pitiphat W, Franz M, Joshupura KJ. Whole-grain and fiber intakes and periodontitis risk in men. *Am J Clin Nutr*. 2006 Jun;83(6):1395-400.
27. Schwartz N, Kaye EK, Nunn ME, Spiro A 3rd, Garcia RI. High-fiber foods reduce periodontal disease progression in men aged 65 and older: the Veterans Affairs normative aging study/Dental Longitudinal Study. *J Am Geriatr Soc*. 2012 Apr;60(4):676-83.

28. Bowden GH, Li YH. Nutritional influences on biofilm development. *Adv Dent Res.* 1997 Apr;11(1):81-99.
29. König KG, Navia JM. Nutritional role of sugars in oral health. *Am J Clin Nutr.* 1995 Jul;62(1 Suppl):275S-282S; discussion 282S-283S. Review. 7598084.
30. Morhart RE, Fitzgerald RJ. Nutritional determinants of the ecology of the oral flora. *Dent Clin North Am.* 1976 Jul;20(3):473-89.
31. Chapple IL. Potential mechanisms underpinning the nutritional modulation of periodontal inflammation. *J Am Dent Assoc.* 2009 Feb;140(2):178-84.
32. Calder PC, Ahluwalia N, Brouns F, Buetler T, Clement K, Cunningham K, Esposito K, Jönsson LS, Kolb H, Lansink M, Marcos A, Margioris A, Matusheski N, Nordmann H, O'Brien J, Pugliese G, Rizkalla S, Schalkwijk C, Tuomilehto J, Wärnberg J, Watzl B, Winklhofer-Roob BM. Dietary factors and low-grade inflammation in relation to overweight and obesity. *Br J Nutr.* 2011 Dec;106 Suppl 3:S5-78.
33. Rosen ED, Spiegelman BM. Adipocytes as regulators of energy balance and glucose homeostasis. *Nature.* 2006 Dec 14;444(7121):847-53.
34. Burcelin R, Serino M, Chabo C, Blasco-Baque V, Amar J. Gut microbiota and diabetes: from pathogenesis to therapeutic perspective. *Acta Diabetol.* 2011 Dec;48(4):257-73.
35. Blasco-Baque V, Serino M, Vergnes JN, Riant E, Loubieres P, Arnal JF, Gourdy P, Sixou M, Burcelin R, Kemoun P. High-fat diet induces periodontitis in mice through lipopolysaccharides (LPS) receptor signaling: protective action of estrogens. *PLoS One.* 2012;7(11):e48220.
36. Reynolds MA, Dawson DR, Novak KF, Ebersole JL, Gunsolley JC, Branch-Mays GL, Holt SC, Mattison JA, Ingram DK, Novak MJ. Effects of caloric restriction on inflammatory periodontal disease. *Nutrition.* 2009 Jan;25(1):88-97.
37. Schifferle RE. Nutrition and periodontal disease. *Dent Clin North Am.* 2005 Jul;49(3):595-610, vii.
38. The website for Centers for Diseases Control and Prevention; National Health and Nutrition Examination Survey
[Http://www.cdc.gov/nchs/nhanes.htm](http://www.cdc.gov/nchs/nhanes.htm)
Last Visited: January 2013
39. The website for The Center for Nutrition Policy and Promotion, an agency of the United State Department of Agriculture.
[Http://www.cnpp.usda.gov](http://www.cnpp.usda.gov)
Last visited: October 2012.

40. Roza AM, Shizgal HM. The Harris Benedict equation reevaluated: resting energy requirements and the body cell mass. *Am J Clin Nutr.* 1984 Jul;40(1):168-82.
41. Lin PH, Proschan MA, Bray GA, Fernandez CP, Hoben K, Most-Windhauser M, Karanja N, Obarzanek E; DASH Collaborative Research Group. Estimation of energy requirements in a controlled feeding trial. *Am J Clin Nutr.* 2003 Mar;77(3):639-45.
42. Douglas CC, Lawrence JC, Bush NC, Oster RA, Gower BA, Darnell BE. Ability of the Harris Benedict formula to predict energy requirements differs with weight history and ethnicity. *Nutr Res.* 2007 Apr;27(4):194-199
43. The Website: [bmi-calculator.net](http://www.bmi-calculator.net)
<http://www.bmi-calculator.net/bmr-calculator/harris-benedict-equation/>
Last visited: July 2013
44. The website for Centers for Diseases Control and Prevention; Physical Activity for a Healthy Weight.
[Http://www.cdc.gov/healthyweight/physical_activity/index.html](http://www.cdc.gov/healthyweight/physical_activity/index.html)
Last visited: January 2013
45. The website for the World Health organization; Physical Activity and Adults.
[Http://www.who.int/dietphysicalactivity/factsheet_adults/en/index.html](http://www.who.int/dietphysicalactivity/factsheet_adults/en/index.html)
Last visited: January 2013
46. Tomar SL, Asma S. Smoking-attributable periodontitis in the United States: findings from NHANES III. National Health and Nutrition Examination Survey. *J Periodontol.* 2000 May;71(5):743-51.
47. Genco RJ, Borgnakke WS. Risk factors for periodontal disease. *Periodontol* 2000. 2013 Jun;62(1):59-94.
48. Haas AN, Gaio EJ, Oppermann RV, Rösing CK, Albandar JM, Susin C. Pattern and rate of progression of periodontal attachment loss in an urban population of South Brazil: a 5-years population-based prospective study. *J Clin Periodontol.* 2012 Jan;39(1):1-9.
49. Johnson GK, Hill M. Cigarette smoking and the periodontal patient. *J Periodontol.* 2004 Feb;75(2):196-209.
50. The website for Centers for Diseases Control and Prevention; National Diabetes Fact sheet, 2011.
http://www.cdc.gov/diabetes/pubs/pdf/ndfs_2011.pdf
51. Thomson WM, Sheiham A, Spencer AJ. Sociobehavioral aspects of periodontal disease. *Periodontol* 2000. 2012 Oct;60(1):54-63.
52. Borrell LN, Crawford ND. Social disparities in periodontitis among United States adults 1999-2004. *Community Dent Oral Epidemiol.*

53. Boillot A, El Halabi B, Batty GD, Rangé H, Czernichow S, Bouchard P. Education as a predictor of chronic periodontitis: a systematic review with meta-analysis population-based studies. *PLoS One*.
54. Albandar JM, Brunelle JA, Kingman A. Destructive periodontal disease in adults 30 years of age and older in the United States, 1988-1994. *J Periodontol*. 1999 Jan;70(1):13-29. Erratum in: *J Periodontol* 1999 Mar;70(3):351.
55. Hung HC, Colditz G, Joshipura KJ. The association between tooth loss and the self-reported intake of selected CVD-related nutrients and foods among US women. *Community Dent Oral Epidemiol*. 2005 Jun;33(3):167-73.
56. Al-Zahrani MS, Kayal RA, Bissada NF. Periodontitis and cardiovascular disease: a review of shared risk factors and new findings supporting a causality hypothesis. *Quintessence Int*. 2006 Jan;37(1):11-8.

Appendix 1

Variables for probing depth.

**OHX02PCD, OHX02PCM, OHX02PCS, OHX02PCP, OHX02PCL,
OHX02PCA, OHX03PCD, OHX03PCM, OHX03PCS, OHX03PCP,
OHX03PCL, OHX03PCA, OHX04PCD, OHX04PCM, OHX04PCS,
OHX04PCP, OHX04PCL, OHX04PCA, OHX05PCD, OHX05PCM,
OHX05PCS, OHX05PCP, OHX05PCL, OHX05PCA, OHX06PCD,
OHX06PCM, OHX06PCS, OHX06PCP, OHX06PCL, OHX06PCA,
OHX07PCD, OHX07PCM, OHX07PCS, OHX07PCP, OHX07PCL,
OHX07PCA, OHX08PCD, OHX08PCM, OHX08PCS, OHX08PCP,
OHX08PCL, OHX08PCA, OHX09PCD, OHX09PCM, OHX09PCS,
OHX09PCP, OHX09PCL, OHX09PCA, OHX10PCD, OHX10PCM,
OHX10PCS, OHX10PCP, OHX10PCL, OHX10PCA, OHX11PCD,
OHX11PCM, OHX11PCS, OHX11PCP, OHX11PCL, OHX11PCA,
OHX12PCD, OHX12PCM, OHX12PCS, OHX12PCP, OHX12PCL,
OHX12PCA, OHX13PCD, OHX13PCM, OHX13PCS, OHX13PCP,
OHX13PCL, OHX13PCA, OHX14PCD, OHX14PCM, OHX14PCS,
OHX14PCP, OHX14PCL, OHX14PCA, OHX15PCD, OHX15PCM,
OHX15PCS, OHX15PCP, OHX15PCL, OHX15PCA, OHX18PCD,
OHX18PCM, OHX18PCS, OHX18PCP, OHX18PCL, OHX18PCA,
OHX19PCD, OHX19PCM, OHX19PCS, OHX19PCP, OHX19PCL,
OHX19PCA, OHX20PCD, OHX20PCM, OHX20PCS, OHX20PCP,
OHX20PCL, OHX20PCA, OHX21PCD, OHX21PCM, OHX21PCS,**

OHX21PCP, OHX21PCL, OHX21PCA, OHX22PCD, OHX22PCM,
OHX22PCS, OHX22PCP, OHX22PCL, OHX22PCA, OHX23PCD,
OHX23PCM, OHX23PCS, OHX23PCP, OHX23PCL, OHX23PCA,
OHX24PCD, OHX24PCM, OHX24PCS, OHX24PCP, OHX24PCL,
OHX24PCA, OHX25PCD, OHX25PCM, OHX25PCS, OHX25PCP,
OHX25PCL, OHX25PCA, OHX26PCD, OHX26PCM, OHX26PCS,
OHX26PCP, OHX26PCL, OHX26PCA, OHX27PCD, OHX27PCM,
OHX27PCS, OHX27PCP, OHX27PCL, OHX27PCA, OHX28PCD,
OHX28PCM, OHX28PCS, OHX28PCP, OHX28PCL, OHX28PCA,
OHX29PCD, OHX29PCM, OHX29PCS, OHX29PCP, OHX29PCL,
OHX29PCA, OHX30PCD, OHX30PCM, OHX30PCS, OHX30PCP,
OHX30PCL, OHX30PCA, OHX31PCD, OHX31PCM, OHX31PCS,
OHX31PCP, OHX31PCL, OHX31PCA

Appendix 2

OHX02LAD, OHX02LAM, OHX02LAP, OHX02LAS, OHX02LAL,
OHX02LAA, OHX03LAD, OHX03LAM, OHX03LAS, OHX03LAP,
OHX03LAL, OHX03LAA, OHX04LAD, OHX04LAM, OHX04LAS,
OHX04LAP, OHX04LAL, OHX04LAA, OHX05LAD, OHX05LAM,
OHX05LAS, OHX05LAP, OHX05LAL, OHX05LAA, OHX06LAD,
OHX06LAM, OHX06LAS, OHX06LAP, OHX06LAL, OHX06LAA,
OHX07LAD, OHX07LAM, OHX07LAS, OHX07LAP, OHX07LAL,
OHX07LAA, OHX08LAD, OHX08LAM, OHX08LAS, OHX08LAP,
OHX08LAL, OHX08LAA, OHX09LAD, OHX09LAM, OHX09LAS,
OHX09LAP, OHX09LAL, OHX09LAA, OHX10LAD, OHX10LAM,
OHX10LAS, OHX10LAP, OHX10LAL, OHX10LAA, OHX11LAD,

OHX11LAM, OHX11LAS, OHX11LAP, OHX11LAL, OHX11LAA,
OHX12LAD, OHX12LAM, OHX12LAS, OHX12LAP, OHX12LAL,
OHX12LAA, OHX13LAD, OHX13LAM, OHX13LAS, OHX13LAP,
OHX13LAL, OHX13LAA, OHX14LAD, OHX14LAM, OHX14LAS,
OHX14LAP, OHX14LAL, OHX14LAA, OHX15LAD, OHX15LAM,
OHX15LAS, OHX15LAP, OHX15LAL, OHX15LAA, OHX18LAD,
OHX18LAM, OHX18LAS, OHX18LAP, OHX18LAL, OHX18LAA,
OHX19LAD, OHX19LAM, OHX19LAS, OHX19LAP, OHX19LAL,
OHX19LAA, OHX20LAD, OHX20LAM, OHX20LAS, OHX20LAP,
OHX20LAL, OHX20LAA, OHX21LAD, OHX21LAM, OHX21LAS,
OHX21LAP, OHX21LAL, OHX21LAA, OHX22LAD, OHX22LAM,
OHX22LAS, OHX22LAP, OHX22LAL, OHX22LAA, OHX23LAD,
OHX23LAM, OHX23LAS, OHX23LAP, OHX23LAL, OHX23LAA,
OHX24LAD, OHX24LAM, OHX24LAS, OHX24LAP, OHX24LAL,
OHX24LAA, OHX25LAD, OHX25LAM, OHX25LAS, OHX25LAP,
OHX25LAL, OHX25LAA, OHX26LAD, OHX26LAM, OHX26LAS,
OHX26LAP, OHX26LAL, OHX26LAA, OHX27LAD, OHX27LAM,
OHX27LAS, OHX27LAP, OHX27LAL, OHX27LAA, OHX28LAD,
OHX28LAM, OHX28LAS, OHX28LAP, OHX28LAL, OHX28LAA,
OHX29LAD, OHX29LAM, OHX29LAS, OHX29LAP, OHX29LAL,
OHX29LAA, OHX30LAD, OHX30LAM, OHX30LAS, OHX30LAP,
OHX30LAL, OHX30LAA, OHX31LAD, OHX31LAM, OHX31LAS,
OHX31LAP, OHX31LAL, OHX31LAA