Published in final edited form as:
Am J Cardiol. 2008 December 1; 102(11): 1509–1513. doi:10.1016/j.amjcard.2008.07.047.

Usefulness of Self-Reported Periodontal Disease to Identify Individuals with Elevated Inflammatory Markers at Risk of Cardiovascular Disease

Heidi Mochari, MPH, RD,
Columbia University Medical Center/New York Presbyterian Hospital

John T. Grbic, DMD, M Med Sc, and
Columbia University, College of Dental Medicine

Lori Mosca, MD, MPH, PhD
Columbia University College of Physicians and Surgeons/New York-Presbyterian Hospital

Abstract

Periodontal disease has been associated with cardiovascular disease (CVD) and inflammation may represent a common pathophysiology. Oral health screening in the context of CVD risk assessment represents a potential opportunity to identify persons at risk for CVD. The purpose of this study was to determine if self-reported oral health status is independently associated with inflammatory markers and if oral health assessment as part of CVD risk screening can identify at-risk persons without traditional CVD risk factors. A baseline analysis was conducted among participants in the NHLBI **F**amily **I**ntervention **T**rial for **Heart** Health (**F.I.T. Heart**) (n=421; mean age 48±13.5y; 36% non-white) without CVD or diabetes who underwent standardized assessment of oral health, lifestyle, CVD risk factors and inflammatory markers high sensitivity c-reactive protein (hsCRP) and lipoprotein-associated phospholipase A_2 (Lp-PLA_2). Statistical associations between oral health, risk factors and inflammatory markers were assessed and logistic regression was used to adjust for effects of lifestyle and potential confounders. Periodontal disease was independently associated with being in the top Lp-PLA_2 quartile versus the lower three (OR=1.9; 95%CI=1.1–3.2) after adjustment for lifestyle and risk factors. History of periodontal disease was reported by 24% of non-overweight, non-hypertensive, non-hypercholesterolemic participants and among these participants, 37% had elevated hsCRP (≥ 3mg/L) or Lp-PLA_2 (≥ 215ng/mL). In conclusion, self-reported periodontal disease is independently associated with inflammation and common in persons without traditional CVD risk-factors.

Keywords

Oral Health; Cardiovascular Disease; Inflammation; Lipoprotein-Associated Phospholipase A2

Corresponding Author: Lori Mosca, MD, MPH, PhD, 601 West 168th Street, Suite 43, New York, NY 10032, Tel: (212) 305-4866; FAX: (212) 342-5238, Email: lmr2@columbia.edu, ljm10@columbia.edu.

INTRODUCTION

Periodontal disease is highly prevalent, affecting approximately 34% of adults over the age of 30 years (~36 million persons) (1) and has been correlated with the presence of cardiovascular disease (CVD) (2). Whether this association is causal is not yet determined, however research suggests that there may be common underlying pathophysiology. Chronic infection has been linked to vascular inflammation, and both infection and inflammation have been associated with periodontal disease and incident CVD events (2,3). But, not all prior research has controlled for diet and/or lifestyle factors that may be associated with CVD and periodontal disease (3). The purpose of this study was 1) to examine whether self-reported oral health status is independently associated with novel inflammatory markers for CVD risk including high sensitivity c-reactive protein (hsCRP) and lipoprotein-associated phospholipase A_2 (Lp-PLA_2) and 2) to assess whether self-reported oral health status identifies persons with a history of periodontal disease who do not have traditional CVD risk factors in a diverse population of participants eligible for the primary prevention of CVD.

METHODS

This was a cross sectional baseline sub-study of consecutively enrolled participants in the National Heart Lung and Blood Institute (NHLBI) **F**amily **I**ntervention **T**rial for **Heart** Health (F.I.T. Heart) (n=421; mean age 48 ± 13.5 years; 36% racial/ethnic minority) designed to test the effectiveness of a screening and educational intervention for family members of patients hospitalized with CVD. Participants were eligible if they were family members or cohabitants of patients hospitalized with CVD, were 20–79 years of age, did not have CVD or diabetes, and spoke either English or Spanish. Demographic information was obtained by standardized questionnaire. All participants signed written informed consent to be a part of the study. The study was approved by the Columbia University Medical Center Internal Review Board.

Oral health status data were collected using the following standardized questions: 1) “Have you ever been informed that you have periodontal (gum) disease?”, 2) “Have you ever received treatment for periodontal disease?”, 3) “Do you have removable partial or complete dentures?”, and 4) “When was the last time you had your teeth cleaned?” Self reported measures of periodontal disease such as these have been shown to be predictive of clinical attachment loss and periodontitis (4,5).

Dietary assessments were completed using the full length Block 98 Food Frequency Questionnaire (6–8). High saturated fat intake was defined as ≥10% of calories from saturated fat per day. High dietary cholesterol intake was defined as ≥ 300 mg/day. Suboptimal fruit and vegetable intake was defined as fewer than 5 combined servings of fruits and vegetables per day. Higher alcohol intake was defined as being at or above the 75^{th} percentile for daily percent of calories from alcohol.

Physical activity level was assessed using standardized questions adapted from the Centers for Disease Control Behavioral Risk Factor Surveillance System Questionnaire (9). Suboptimal exercise level was defined as exercise less than three days per week for 30 minutes per session. Current smoking status was defined by self report as smoker vs. non smoker and confirmed using carbon monoxide breath testing.

Waist circumference and body mass index (BMI) were assessed by trained examiners using NHLBI Clinical Guidelines (10). Above optimal waist circumference was defined as > 102 centimeters [> 40 inches] in men and > 88 centimeters [> 35 inches] in women. Overweight or obese status was defined as BMI ≥ 25.0 kg/M^2.

Systolic and diastolic blood pressure was assessed by an automated blood pressure monitor in the Columbia University Clinical and Translational Science Award (CTSA) Center using standard protocol (11). Hypertension was defined as a systolic blood pressure ≥ 140 mmHg or a diastolic blood pressure ≥ 90 mmHg (12).

All participants underwent fasting blood draw at their baseline study visits. Serum and plasma aliquots were prepared from fasting blood samples immediately after blood draw. Determination lipids were performed on blood analyzed (Roche Diagnostics) in the Columbia University CTSA. Low density lipoprotein (LDL) cholesterol was calculated using the Friedewald equation. High sensitivity c-reactive protein (hsCRP) and lipoprotein-associated phospholipase A_2 (Lp-PLA_2), both inflammatory markers that are indicators of CVD risk, were systematically measured in all participants using blood collected at baseline visit. HsCRP was assessed using Kamiya hsCRP kit on serum samples. High hsCRP levels were defined as ≥ 3 mg/L vs. < 3 mg/L. HsCRP values ≥ 10 mg/L were excluded from analysis based on previous work suggesting they reflect acute/non-vascular inflammation (13). Lp-PLA_2 mass was measured using the PLAC test (diaDexus Inc. South San Francisco, CA, USA) on plasma samples. Elevated Lp-PLA_2 levels were defined as being at or above the 75th percentile.

Metabolic syndrome was evaluated using National the Cholesterol Education Program Adult Treatment Panel III definition of the presence of 3 or more of the risk factors 1) abdominal obesity, 2) triglycerides ≥ 150 mg/dL, 3) HDL-cholesterol level < 40 mg/dL (men); < 50 mg/dL(women), 4) blood pressure ≥ 130/85 mmHg and 5) fasting glucose ≥ 110 mg/dL (14).

All data were collected on standardized forms, double entered into a Microsoft Access database and exported to SAS version 9.1, SAS Institute Inc., Cary, North Carolina, USA for statistical analysis. Continuous and categorical variables were characterized using means and frequency statistics respectively. Spearman coefficients were used to assess the correlation between hsCRP and Lp-PLA_2 and between each of these inflammatory markers and age. Associations between oral health and inflammatory markers, oral health and CVD lifestyle and risk factors, and between CVD lifestyle and risk factors and inflammatory markers were assessed using chi-square statistics. Multiple logistic regression models were used to assess the association between oral health exposure variables and inflammation markers, controlling for age, sex, race/ethnicity, smoking, and potential confounders.

RESULTS

The baseline characteristics of 421 consecutively enrolled F.I.T. Heart study participants in this analysis are listed in Table 1. Mean age was 48.2 ± 13.4 years. The median hsCRP level was 1.05 mg/L (hsCRP ≥ 10.0 mg/L excluded (n=1)). The median Lp-PLA_2 level among all participants was 189.3 ng/mL with 25% of participants having Lp-PLA_2 levels ≥ 215 ng/mL. Age and hsCRP were not correlated (r=−.01; p=.85), nor were age and Lp-PLA_2 (r=.05; p=.28) and Lp-PLA_2 and hsCRP were not correlated with each other (r=.04; p=.39).

Approximately one in four (23%) of participants had a history of diagnosed periodontal disease. Partial or complete removable dentures were utilized by 10% of participants (n=44) and of them 38% reported a history of diagnosed periodontal disease. A duration of greater than 12 months since their last dental cleaning was reported by 23% of participants (n=97). Among those with greater than 12 months since last cleaning, 19% reported diagnosed periodontal disease.

Participants with a history of diagnosed periodontal disease were almost two times more likely to have Lp-PLA_2 levels in the highest quartile versus the lower three (OR=1.8; 95%CI=1.1–3.0) than those who did not have a history of diagnosed periodontal disease. Those reporting no dental cleaning in greater than a year were more likely to have hsCRP ≥ 3 mg/L vs. < 3 mg/

L (OR=1.7; 95%CI=1.0–2.9) compared to those who reported a cleaning within the past year. Neither a history of treatment for periodontal disease, or having partial or complete removable dentures was significantly associated with elevated levels of hsCRP or Lp-PLA_2 (Table 2).

Males were less likely than females to have elevated hsCRP (≥ 3 mg/L vs. < 3 mg/L) and more likely than females to have Lp-PLA_2 in the highest quartile. Non-white participants were more likely to have elevated hsCRP compared to whites and less likely to have Lp-PLA_2 in the highest quartile. Age, education level and having health insurance were not associated with hsCRP or Lp-PLA_2 (Table 2).

In univariate assessment of the association between CVD lifestyle and traditional CVD risk factors, high waist circumference was positively associated with hsCRP levels as was overweight or obese status. Neither was significantly associated with Lp-PLA_2 levels. Saturated fat intake was significantly associated with higher Lp-PLA_2 and not with hsCRP. Likewise, higher LDL cholesterol levels were significantly associated with higher Lp-PLA_2 levels. Dietary cholesterol, alcohol, fruit and vegetable intake, exercise level, smoking and blood pressure were not significantly associated with increased inflammatory marker levels in this study (Table 2).

The observed association between self-reported periodontal disease and increased Lp-PLA_2 levels remained statistically significant when controlling for age, sex, race, smoking, LDL-cholesterol and dietary saturated fat in a multiple logistic model (OR=1.9; 95%CI=1.1–3.2). The association between last dental cleaning > 12 months ago and hsCRP level did not retain statistical significance after adjustment for age, sex, race, smoking, waist circumference and BMI.

We examined the prevalence of self-reported history of periodontal disease among participants without traditional cardiovascular risk factors and found that diagnosed periodontal disease was present 23–29% of the time in the absence of individual and multiple CVD lifestyle and risk factors traditionally measured during CVD risk assessment (Table 3). Over one in five (24%) of non-hypertensive participants without elevated LDL-cholesterol or overweight/obesity reported a history of periodontal disease diagnosis. Among those with periodontal disease and without these three traditional CVD risk factors 37% had elevated hsCRP or Lp-PLA_2.

DISCUSSION

In a diverse population of individuals eligible for the primary prevention of CVD, a history of periodontal disease was associated with significantly higher levels of Lp-PLA_2 compared with those without periodontal disease. When adjusted for potential confounders and lifestyle factors, this association remained statistically significant. To our knowledge this is the first report of an association between oral health and Lp-PLA_2. These data support a possible independent association between oral health and inflammation, suggesting that inflammation may be a factor in the relationship between oral health and CVD. We also showed that diagnosed periodontal disease is present in patients without traditional CVD risk factors and that many of these individuals have increased inflammatory markers (hsCRP or Lp-PLA_2).

In contrast to some studies, we did not find an association between oral health and hsCRP after adjustment. This may be related to our primary prevention population with lower mean hsCRP levels compared to means reported in patients with established CVD (15–17) where both hsCRP and Lp-PLA_2 have been independently correlated with CVD risk (18,19). Alternatively, because we controlled for BMI, smoking and other lifestyle factors, our results may discordant due to control for confounding.

We did not show a correlation of hsCRP and Lp-PLA_2 with each other, which may reflect disparate inflammatory pathways (20) and correlation with varying CVD risk factors. For example, central adiposity and obesity were strongly associated with increased hsCRP levels in our study, and in others (19), but were not found to be associated with higher levels of Lp-PLA_2. This is consistent with past research in both men and women which also failed to correlate Lp-PLA_2 levels with body mass index (21–23) and supports specificity of Lp-PLA_2 to vascular rather than systemic inflammatory processes (24). Higher levels of Lp-PLA_2 have been identified on inflamed, rupture prone plaques, and the active Lp-PLA_2 product lysophosphatidylcholine has been correlated with human endothelial dysfunction in persons with early stages of atherosclerosis (24,25).

Periodontal disease diagnosis was reported by 23% of our screening population which is lower than nationally estimated prevalence among adults aged ≥ 30 years (1). This could be attributed to 10% of study participants being under the age of 30 years with only 4 participants in this age range reporting a diagnosis of periodontal disease.

Strengths of this study include our population with well characterized CVD risk, lifestyle, and other risk factors allowing us to examine interrelation among these, oral health, and inflammation. Participants were ethnically diverse, eligible for the primary prevention of CVD, and actively seeking CVD risk screening suggesting these findings may be generalizable and applicable to other primary prevention populations.

A limitation of this study is that we used self-reported data instead of clinical examination to assess oral health status. Nonetheless past research has shown good correlation between self report and clinical evaluation (26) and non-differential misclassification of disease status would generally bias towards the null making a spurious association unlikely. We are limited in drawing conclusions about directionality of relations between oral health and inflammatory markers due to the cross sectional design; specifically it is not possible to conclude from these data that oral health is a risk marker for CVD or CVD outcomes, or that any therapy based on oral health status will be effective. Our data suggest that “at risk” persons who may otherwise not be identified by traditional CVD risk screening might be identified through oral health screening. Further research prospectively examining the relationship between oral health status, CVD biomarkers and incident CVD is needed to determine if there is a causal link between oral health and CVD.

Acknowledgements

This study was funded by a National Institute of Health (NIH) Research Project Award to Dr. Mosca (R01 HL 075101-01A1) and supported in part by the Clinical and Translational Science Award (CTSA) Center at Columbia University Medical Center, and an NIH Research Career Award to Dr. Mosca (K24 HL07634601A1).

References

1. Eke PI, Genco RJ. CDC periodontal disease surveillance project: background, objectives, and progress report. J Periodontol 2007;78:1366–1371. [PubMed: 17610396]
2. Genco R, Offenbacher S, Beck J. Periodontal disease and cardiovascular disease epidemiology and possible mechanisms. J Am Dent Assoc 2002;133:14S–22S. [PubMed: 12085720]
3. Ridker PM. Inflammation, infection and cardiovascular risk. How good is the clinical evidence? Circulation 1998;97:1671–1674. [PubMed: 9591759]
4. Slade GD. Interim analysis of validity of periodontitis screening questions in the Australian population. J Periodontol 2007;78:1463–1470. [PubMed: 17608615]
5. Genco RJ, Falkner KL, Grossi S, Dunford R, Trevisan M. Validity of self-reported measures for surveillance of periodontal disease in two Western New York population-based studies. J Periodonal 2007;78:1439–1454.

6. Boucher B, Cotterchio M, Kreiger N, Nadalin V, Block T, Block G. Validity and reliability of the Block98 food-frequency questionnaire in a sample of Canadian women. Public Health Nutr 2006;9:84–93. [PubMed: 16480538]
7. Nutritionquest. Questionnaires & Screeners. 2008 Feb 06 [Accessed: 2008-02-06]. URL:http://www.nutritionquest.com/products/questionnaires_screeners.htm (Archived by WebCite® at http://www.webcitation.org/5VPUsTdh6).
8. USDA. USDA National Nutrient Database for Standard Reference. 2008 Feb 06 [Accessed: 2008-02-06]. URL:http://www.ars.usda.gov/Services/docs.htm?docid=8964 (Archived by WebCite® at http://www.webcitation.org/5VPV4Nrei).
9. Behavioral Risk Factor Surveillance System Questionnaires. 2008 Feb 06 [Accessed: 2008-02-06]. URL:http://www.cdc.gov/brfss/questionnaires/english.htm (Archived by WebCite® at http://www.webcitation.org/5VPVDeN4r).
10. Clinical guidelines on the identification, evaluation, and treatment of overweight and obesity in adults: executive summary. Expert Panel on the Identification, Evaluation, and Treatment of Overweight in Adults. Am J Clin Nutr 1998;68:899–917. [PubMed: 9771869]
11. Pickering TG, Hall JE, Appel LJ, Falkner BE, Graves J, Hill MN, Jones DW, Kurtz T, Sheps SG, Roccella EJ. Recommendations for blood pressure measurement in humans and experimental animals. Part 1: blood pressure measurement in humans: a statement for professionals from the Subcommittee of Professional and Public Education of the American Heart Association Council on High Blood Pressure Research. Hypertension 2005;45:142–161. [PubMed: 15611362]
12. Chobanian AV, Bakris GL, Black HR, Cushman WC, Green LA, Izzo JL Jr, Jones DW, Materson BJ, Oparil S, Wright JY Jr, Roccella EJ. Joint National Committee on Prevention, Detection, Evaluation, and Treatment of High Blood Pressure. National Heart, Lung, and Blood Institute; National High Blood Pressure Education Program Coordinating Committee. Seventh report of the Joint National Committee on Prevention, Detection, Evaluation, and Treatment of High Blood Pressure. Hypertension 2003;42:1206–1252. [PubMed: 14656957]
13. Pearson TA, Mensah GA, Alexander RW, Anderson JL, Cannon RO 3rd, Criqui M, Fadi YY, Fortmann SP, Hong Y, Myers GL, Rifai N, Smith SC Jr, Taubert K, Tracey RP, Vinicor F. Markers of inflammation and cardiovascular disease: application to clinical and public health practice: A statement for healthcare professionals from the Centers for Disease Control and Prevention and the American Heart Association. Circulation 2003;107:499–511. [PubMed: 12551878]
14. Third Report of the National Cholesterol Education Program (NCEP) Expert Panel on Detection, Evaluation, and Treatment of High Blood Cholesterol in Adults (Adult Treatment Panel III) final report. Circulation 2002;106:3143–3421. [PubMed: 12485966]
15. Koenig W, Twardella D, Brenner H, Rothenbacher D. Lipoprotein-associated phospholipase A_2 predicts future cardiovascular events in patients with coronary heart disease Independently of traditional risk factors, markers of inflammation, renal function, and hemodynamic stress. Arterioscler Thromb Vasc Biol 2006;26:1586–1593. [PubMed: 16627803]
16. Brilakis ES, McConnell JP, Lennon RJ, Elesber AA, Meyer JG, Berger PB. Association of lipoprotein-associated phospholipase A2 levels with coronary artery disease risk factors, angiographic coronary artery disease, and major adverse events at follow-up. Eur Heart J 2005;26:137–144. [PubMed: 15618069]
17. Corsetti JP, Rainwater DL, Moss AJ, Zareba W, Sparks CE. High lipoprotein-associated phospholipase A_2 is a risk factor for recurrent coronary events in postinfarction patients. Clin Chem 2006;52:1331–1338. [PubMed: 16709622]
18. Tselepis AD, John Chapman M. Inflammation, bioactive lipids and atherosclerosis: potential roles of a lipoprotein-associated phospholipase A_2, platelet activating factor-acetylhydrolase. Atheroscler Suppl 2002;3:57–68. [PubMed: 12573364]
19. Ridker PM. C-reactive protein and the prediction of cardiovascular events among those at intermediate risk: moving an inflammatory hypothesis toward consensus. J Am Coll Cardiol 2007;49:2129–2138. [PubMed: 17531663]
20. Zalewski A, Macphee C. Role of lipoprotein-associated phosholipase A2 in atherosclerosis: biology, epidemiology, and possible therapeutic target. Arterioscler Thromb Vasc Biol 2005;25:923–931. [PubMed: 15731492]

21. Packard CJ, O'Reilly DS, Caslake MJ, McMahon AD, Ford I, Cooney J, Macphee CH, Suckling KE, Krishna M, Wilkinson FE, Rumley A, Lowe GD. Lipoprotein-associated phospholipase A2 as an independent predictor of coronary heart disease. West of Scotland Coronary Prevention Study Group. N Engl J Med 2000;343:1148–1155. [PubMed: 11036120]

22. Koenig W, Khuseyinova N, Lowel H, Trischler G, Meisinger C. Lipoprotein-associated phospholipase A_2 adds to risk prediction of incident coronary artery events by c-reactive protein in apparently healthy middle aged men from the general population. Circulation 2004;110:1903–1908. [PubMed: 15451783]

23. Persson M, Nilsson JA, Nelson JJ, Hedblad B, Berglund G. The epidemiology of Lp-PLA_2: distribution and correlation with cardiovascular risk factors in a population-based cohort. Atherosclerosis 2007;190:388–396. [PubMed: 16530769]

24. Kolodgie FD, Burke AP, Skorija KS, Ladich E, Kutys R, Makuria AT, Virmani R. Lipoprotein-associated phospholipase A2 protein expression in the natural progression of human coronary atherosclerosis. Arterioscler Thromb Vasc Biol 2006;26:2523–2529. [PubMed: 16960105]

25. Lavi S, McConnell JP, Rihal CS, Prasad A, Mathew V, Lerman LO, Lerman A. Local production of lipoprotein-associated phospholipase A2 and lysophosphatidylcholine in the coronary circulation. Association with early coronary atherosclerosis and endothelial dysfunction in humans. Circulation 2007;115:2715–2721. [PubMed: 17502572]

26. Miller K, Eke PI, Schoua-Glusberg A. Cognitive evaluation of self-report questions for surveillance of periodontitis. J Periodontol 2007;78:1455–1462. [PubMed: 17610399]

Table 1

Characteristics of Cardiovascular Disease Risk Screening Participants (n=421)

	n (%)
Male	147 (35%)
Age ≥ 65 years	49 (12%)
Non-white race	151 (36%)
≤ High school education	92 (22%)
No health insurance	62 (15%)
Low density lipoprotein cholesterol ≥ 3.37 mmol/L [≥ 130 mg/dL]	159 (38%)
Blood pressure ≥ 140/90 mmHg	99 (24%)
Waist circumference > 102 cm (M); > 88 cm (F)	161 (38%)
Body mass index ≥ 25 kg/M^2	274 (66%)
Metabolic syndrome	75 (18%)
Saturated fat ≥ 10% of calories/day	240 (57%)
Dietary cholesterol ≥ 300mg/day	107 (26%)
Fruit + vegetables < 5 servings/day	254 (60%)
Dietary alcohol ≥ 75th percentile (≥ 5.2% kcal/day)	108 (25%)
Exercise < 3 days per week for 30 minutes	290 (69%)
Current smoker	41 (10%)
Framingham risk ≥ 10%	35 (8%)
Ever diagnosed with periodontal disease	95 (24%)
Ever treated for periodontal disease	82 (20%)
Wears partial/complete removable dentures	44 (10%)
Last dental cleaning >12 months ago	97 (23%)

Table 2

Associations between Oral Health, Demographic or Lifestyle Factors and Inflammatory Markers

Variable	hsCRP (≥3mg/L vs. <3mg/L) OR (95% CI)	Lp-PLA$_2$ (≥215ng/mL vs. <215ng/mL) OR (95% CI)
Ever diagnosed with periodontal disease	1.2 (.68–2.0)	**1.8 (1.1–3.0)**
Ever treated for periodontal disease	1.2 (.65–2.1)	1.5 (.89–2.6)
Wears removable dentures	.98 (.45–2.1)	1.1 (.54–2.2)
Last dental cleaning >12 months ago	**1.7 (1.0–2.9)**	1.5 (.88–2.4)
Male	**.58 (.34–.98)**	**1.6 (1.0–2.5)**
≥ 65 years old	1.2 (.60–2.4)	1.1 (.56–2.2)
Non-white	**2.2 (1.4–3.5)**	**.36 (.21–.61)**
≤ High school education	1.2 (.70–2.2)	1.0 (.60–1.7)
No health Insurance	1.4 (.73–2.6)	1.4 (.77–2.5)
Low density lipoprotein cholesterol ≥3.37 mmol/L [≥ 130 mg/dL]	.87 (.53–1.4)	**2.0 (1.3–3.2)**
Blood pressure ≥ 140/90 mmHg	1.2 (.69–2.1)	1.3 (.76–2.1)
Waist circumference > 102cm (Male); > 88cm (Female)	**3.7 (2.2–6.0)**	1.2 (74–1.8)
Body mass index ≥ 25 kg/M^2	**3.1 (1.7–5.5)**	1.4 (.87–2.3)
Metabolic syndrome	**2.1 (1.2–3.7)**	1.1 (.64–2.0)
Saturated fat ≥ 10% of calories/day	1.3 (.82–2.2)	**2.0 (1.2–3.2)**
Dietary cholesterol ≥ 300 mg/day	1.1 (.62–1.8)	1.3 (.82–2.2)
Fruit + vegetables < 5 servings/day	1.4 (.88–2.4)	.72 (.46–1.1)
Dietary alcohol ≥ 75th percentile	.66 (.37–1.2)	1.1 (.66–1.8)
Exercise < 3 days per week 30 minutes	1.2 (.74–2.1)	.80 (.50–1.3)
Current smoker	.60 (.24–1.5)	.84 (.39–1.8)
Framingham risk ≥ 10%	.62 (.23–1.6)	1.1 (.48–2.3)

Table 3

Prevalence of Periodontal Disease and Inflammatory Markers by Risk Factor Status

Variable	At Risk Factor Goal	Percent At Risk Factor Goal with Periodontal Disease	Percent At Risk Factor Goal with Periodontal Disease *and* Elevated Lp-PLA_2 or hsCRP
Low density lipoprotein cholesterol < 3.37 mmol/L [< 130 mg/dL]	259 (62%)	24%	11%
Blood pressure < 140/90 mmHg	322 (76%)	24%	11%
Waist circumference ≤ 102 cm (M); ≤ 88 cm (F)	260 (62%)	23%	9%
Body mass index < 25 kg/M^2	144 (34%)	24%	10%
Saturated fat < 10% of calories/day	179 (43%)	28%	11%
Dietary cholesterol < 300mg/day	312 (74%)	24%	13%
Fruit and vegetable intake ≥ 5 servings/day	167 (40%)	29%	15%
Exercise ≥ 3 days/week for 30 minutes	130 (31%)	23%	13%
Non- Smoker	378 (90%)	24%	12%
Low density lipoprotein cholesterol < 3.37 mmol/L [<130mg/dL] *and* blood pressure <140/90mmHg *and* body mass index <25 kg/M^2	87 (21%)	24%	9%