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Metabolic Syndrome and Periodontitis in Gullah African Americans With Type 2 Diabetes Mellitus

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Abstract

Aim—To assess associations of metabolic syndrome, and its individual components, with extent of severe periodontitis among patients with type 2 diabetes mellitus (T2DM).

Materials and Methods—We performed a secondary data analysis (N=283) using a cross-sectional study population of Gullah African Americans with T2DM. Extent of severe periodontitis was assessed as total diseased tooth-sites/person (evaluated as separate outcomes: 6+mm clinical attachment level [CAL], 5+mm periodontal probing depth [PPD]) using negative binomial regression techniques. Primary independent variables assessed in separate models included metabolic syndrome (yes/no), each metabolic syndrome component (low HDL, hypertension, high triglycerides, large waist circumference) and glycemic control (poor/good).

Results—Multivariable CAL-model results showed a significant association for metabolic syndrome status with extent of severe periodontitis (RR=2.77, p=0.03). The separate multivariable CAL-model including each metabolic syndrome component showed marginally increased rates among those with large waist circumference (RR=2.33, p=0.09) and those with HbA1c ≥ 7% (RR=1.73, p=0.06). Multivariable PPD-models showed marginally increased rates among those with metabolic syndrome (RR=2.18, p=0.06).

Conclusion—Metabolic syndrome is associated with the extent of severe periodontitis in this Gullah population with T2DM.

Keywords

Metabolic syndrome; diabetes mellitus; obesity; periodontitis; Gullah population

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Introduction

In chronic periodontitis, the interplay between periodontal pathogens and the host inflammatory immune system is responsible for destruction of connective tissue, loss of periodontal attachment and resorption of alveolar bone. Despite the fact that environmental factors seem to provide sufficient disease-provoking factors, not everyone seems to be equally susceptible to periodontal disease (Kornman, 2001). The response to inflammatory mediators is also influenced by the host's genetic composition, suggesting racial differences in susceptibility to periodontal diseases, as well as by acquired risk factors such as diabetes mellitus, obesity and hypertension.

Gullah African Americans (Gullah) are a specific ethnic population who are direct descendants of native Africans, primarily from West African coastal regions (Parra et al., 2001). The South Carolina Gullah, due to geographic and cultural factors, have remained isolated and have considerably less European genetic admixture (3.5%) compared to other African American population (17.7%) (McLean et al., 2005). This population has a high prevalence of chronic diseases, including type 2 diabetes mellitus (T2DM), hypertension, obesity and periodontitis. The relative risk of T2DM to siblings among the Gullah is 3.3, a risk that exceeds that in many other communities (Garvey et al., 2003). Given their genetic and socio-cultural homogeneity, studies involving the Gullah may offer unique insight into the significance of host and environmental factors in development of these diseases.

Metabolic syndrome (MetS) is defined by the National Cholesterol Education Program Adult Treatment Panel III (NCEP/ATP III) as the concomitant presence in an individual of three or more of the following five cardiovascular risk factors: abdominal obesity, hypertension, reduced HDL cholesterol, elevated triglycerides, and elevated fasting glucose (impaired fasting glucose or T2DM).

The relationship between MetS, as a clinical entity, and periodontitis has been examined using the Third National Health and Nutrition Examination Survey (NHANES III) data (D'Aiuto et al., 2008) as well as in several other studies (Shimazaki et al., 2007, Khader et al., 2008, Kushiyama et al., 2009, Morita et al., 2009, Li et al., 2009, Han et al., 2010). These studies produced conflicting results; thus, further investigation is required.

The cardiovascular risk factors, which comprise the diagnosis of MetS, have also been independently evaluated for their associations with periodontitis. Several publications have shown an increased prevalence of periodontitis among those with diabetes, including more severe disease for those with poor glycemic control (Han et al., 2010, Bandyopadhyay et al., 2010, Fernandes et al., 2009). Obesity has also emerged as a risk indicator for periodontal disease (Chaffee and Weston, 2010, Ritchie, 2007), and the presence of periodontitis has been positively associated with abnormal lipid metabolism in several cohort studies (Nibali et al., 2007, Losche et al., 2000). A Swedish study reported an association between diastolic blood pressure and the prevalence of deep periodontal pockets (Engstrom et al., 2007).

The aim of our study was to analyze the significance of the additional risk conferred by the presence of MetS, as well as its individual components, on the extent of severe periodontitis in a Gullah study population with T2DM.

Materials and Methods

Study population

We extracted data from a previous cross-sectional study limited to epidemiological data collection (Fernandes et al., 2009) regarding periodontal disease prevalence among adult

Gullah with T2DM. These subjects were recruited based on self-reported history of T2DM. This information was verified by hemoglobin A1c (HbA1c) testing and/or subjects' use of diabetes medication(s). The research protocol was clearly explained to potential subjects, and Institutional Review Board (IRB) approved consent and HIPAA forms were required for study inclusion. Two calibrated oral examiners (Hill et al., 2006) performed radiographic and soft tissue exams and evaluated six sites per tooth (excluding third molars) for bacterial plaque presence, periodontal probing depths (PPD), clinical attachment levels (CAL), and bleeding on probing (BOP). CAL and PPD exam measurements were rounded down to the next whole decimal number. Oral health behaviors (frequency of brushing, flossing, and dental visits) as well as social and medical history data were collected through a questionnaire administered individually by study personnel. Blood and urine samples were collected to assess renal function, lipid profiles, and HbA1c at the time of enrollment.

Data Analyses

All statistical analyses were performed using SAS software, version 9.2 for XP-Pro, Cary, NC, USA. Our investigation comprised a secondary analyses of data extracted from the previously described epidemiologic study (N=313) (Fernandes et al., 2009). Our study population was limited to those subjects with non-missing data for MetS status, glycemic control, smoking history, gender, age, serum albumin level, high-sensitivity C-reactive protein (CRP), total teeth, and periodontal measures of interest, CAL and PPD, (N=283). We assessed extent of severe periodontitis, defined as total tooth-sites per person measuring 6+mm for CAL and 5+mm for PPD, evaluated separately. These thresholds for diseased tooth-sites are clinically meaningful according to conventional definitions for established periodontitis (Machtei et al., 1992). After determining whether or not each tooth-site had exceeded this threshold, results for each separate measure (CAL and PPD) were summarized as total counts of diseased sites among all available tooth-sites for that individual.

The NCEP/ATP III definition for MetS is the presence in an individual of three or more of the following five components: abdominal obesity (waist circumference >102cm [40 inches] in men and >88cm [35 inches] in women); high blood pressure (BP) (systolic BP ≥130mmHg and /or diastolic BP ≥85mmHg or on therapy for hypertension); hypertriglyceridemia (serum triglyceride level ≥150mg/dl [1.7mmol/l]); low high-density lipoprotein (HDL) cholesterol (serum HDL cholesterol <40mg /dl [1mmol/l] in men and <50mg/dl [1.3mmol/l] in women); high fasting glucose (glucose level ≥ 100mg/dl [5.6mmol/l] or on drug therapy for elevated blood glucose) (Expert Panel on Detection, Evaluation and Treatment of High Blood Cholesterol on Adults, 2001). Since all subjects had an underlying diagnosis of T2DM, they required the presence of two additional risk factors, out of the remaining four from the above definition, in order to be classified as having MetS.

Primary independent variables assessed in the analyses included MetS (yes/no) and each MetS component (low HDL, hypertension, high triglycerides, large waist circumference) exhibited in addition to T2DM. Evaluated covariates included glycemic control (poor: $HbA1_c \geq 7\%$, good: $HbA1_{1c} < 7\%$) (AmericanDiabetesAssociation, 2006), total teeth, age (years), gender (male / female), smoking status (never / current / past), body mass index (BMI, with BMI<25kg/m^2 = normal / 25-30kg/m^2 = overweight / >30kg/m^2 = obese / data-missing), high sensitivity CRP (mg/ml, $\log_e$ transformed) and serum albumin (g/dl, $\log_e$ transformed).

Extent of severe periodontitis (total tooth-sites per person with CAL- and PPD-outcomes), glycemic control, and all other covariates were first summarized by mean and standard deviation (SD) (if continuous) or frequencies (if categorical), and results were reported by their overall and MetS specific distributions [Table 1]. To assess associations between MetS

and the rates of severe periodontitis per person, we used regression techniques appropriate for varying count event data – those that correct for different totals of tooth-sites available per subject. Results from univariable Poisson regression models showed over-dispersion; hence, we evaluated negative binomial (NB) models (Hilbe, 2007) with maximum likelihood parameter estimation (PROC GENMOD in SAS). NB models incorporate estimation of a dispersion parameter, which adequately corrects for over-dispersion (Hilbe, 2007). Our NB models showed values of Pearson chi-square and deviance per degrees of freedom close to 1 [Table 2-5], indicating adequate model fit.

Two sets (each containing two models) of multivariable NB models were fitted separately to each of the outcome measures (CAL and PPD) for a total of four models. In each set, we had a model for (i) MetS status (yes/no); and (ii) individual MetS components (low HDL, hypertension, hypertriglyceridemia, abdominal obesity) as the primary independent predictor(s). In addition to the previously described model covariates, we also tested for significant effect modification amongst factors of clinical importance. The presence of MetS models (i) were initially assessed for interaction effects between MetS status and glycemic control. Also, for the MetS components models (ii), numerous interaction effects were tested among the components factors and glycemic control, provided that our study population provided sufficient samples to do so (i.e., cell sizes of $n \geq 10$). These included low HDL by hypertension, low HDL by abdominal obesity, low HDL by hypertriglyceridemia, and hypertension by abdominal obesity as well as glycemic control by each of these individual components. However, none of the evaluated interaction terms were significant; hence these were dropped in further modeling.

Predictors other than the MetS variables, glycemic control, and any determined confounders were then successively removed through a process of backward elimination based on *p-values* of estimated regression coefficients (removing those with $p>0.05$). The final four models included MetS variables, glycemic control, determined confounders, and all other predictors that showed significant associations with the respective outcome measures. Estimated regression coefficients from these final models were used to calculate covariate adjusted rate ratios (RR) and associated 95% confidence intervals (CI) for the extent of severe periodontitis outcomes (by CAL and PPD) per person (Hilbe, 2007).

Results

Descriptive statistics of our study population (N=283) are listed in Table 1. Prevalence of MetS and HbA1c ≥7% were each 85.87% and 60.07%, respectively. Prevalence for MetS components was 49.47% for low HDL, 85.16% for hypertension, 14.84% for high triglycerides, and 84.45% for large waist circumference. CAL- (5.22±11.60%) and PPD-outcomes (5.24±10.94%) each ranged 0-79.17% tooth-sites/person, and total teeth (19.71±6.07) ranged 3-28 per person. The study population age ranged from 26 to 87 years (55.27±10.65), and 75.79% of participants were female, consistent with higher rates of enrollment among females than males for studies involving the Gullah (Johnson-Spruill et al., 2009). Most participants were never smokers (69.96%), while 14.84% were current smokers and 15.19% were past smokers. Prevalence of obesity and being overweight were 66.08% and 19.79%, respectively.

The covariate effects were interpreted separately for the PPD and CAL models

CAL Models—Final multivariable model results for MetS (yes/no) showed a significant association with extent of severe periodontitis (RR= 2.77; 95% CI=1.11-6.93; $p=0.03$) [Table 2]. Significantly decreased rates were observed for every additional tooth present per-person (RR=0.90; 95% CI=0.86-0.94; $p<0.0001$) as well as among females (RR=0.43, 95% CI=0.22-0.83, $p=0.02$). There were also marginally increased rates among current smokers

(RR=2.15; 95% CI=0.97-4.78; p=0.06) as well as with CRP elevations (RR=1.30; 95% CI=0.99-1.71; p=0.06). The separate multivariable CAL model for the components of MetS showed marginally increased rates among those with large waist circumference (RR=2.33; 95% CI=0.88-6.23; p=0.09) [Table 3]. However, results for low HDL, hypertension, and high triglycerides showed no associations. This model also showed significantly increased rates with CRP elevations (RR=1.37; 95% CI=1.05-1.79; p=0.02), while significantly decreased rates were observed for every additional tooth (RR=0.89; 95% CI=0.85-0.94; p<0.0001) as well as among females (RR=0.39; 95% CI=0.19-0.80; p=0.01). There were also marginally increased rates among those with HbA1c ≥7% (RR=1.73; 95% CI=0.97-3.11; p=0.06) as well as for current smokers (RR=2.16; 95% CI=0.88-5.29; p=0.09).

We also performed post-hoc analyses of these CAL-models to assess HbA1c as a continuous covariate (%). The final multivariable model for MetS (yes/no) showed a significant association with extent of severe periodontitis (RR=3.11, 95% CI=1.28-7.52, p=0.01); significantly increased rates were also observed with HbA1c elevations (RR=1.21, 95% CI=1.05-1.39, p<0.01). Results for low HDL, hypertension, high triglycerides, and high waist circumference showed no associations; however, significantly increased rates were observed with HbA1c elevations (RR=1.21, 95% CI=1.04-1.41, p=0.02).

PPD Models—Final multivariable PPD model results for MetS (yes/no) showed marginally increased rates for extent of severe periodontitis (RR=2.18; 95% CI=0.98-4.87; p=0.06) [Table 4]. Significantly decreased rates were observed among females (RR=0.52; 95% CI=0.30-0.92; p=0.02), while significantly increased rates were observed with CRP elevations (RR=1.37; 95% CI=1.09-1.71; p<0.01). The separate multivariable model for the components of MetS showed no associations for large waist circumference, hypertension, high triglycerides, or low HDL [Table 5]. Significantly increased rates were observed with CRP elevations (RR=1.37; 95% CI=1.10-1.70; p<0.01), while significantly decreased rates were observed among females (RR=0.50; 95% CI=0.28-0.91; p=0.02).

Post-hoc analyses were similarly performed for these PPD-models using HbA1c as a continuous covariate (%). Final multivariable model showed marginally increased rates among those with MetS (RR=1.97, 95% CI=0.91-4.28, p=0.09); marginally increased rates were also observed with HbA1c elevations (RR=1.13, 95% CI=0.99-1.29, p=0.07). Results for low HDL, hypertension, high triglycerides, and high waist circumference showed no significant associations; HbA1c was also not significant in this model.

Discussion

The World Health Organization initially defined MetS in 1998 as the association between markers of insulin resistance and two additional risk factors, including obesity, hypertension, high triglycerides level, reduced HDL cholesterol level, or microalbuminuria. This definition has been refined over the last decade by other organizations. We used the definition published by the NCEP/ATP III, which bases the diagnosis of MetS on the presence of three of the following five factors: reduced HDL cholesterol, elevated triglycerides, elevated blood pressure, abdominal obesity and elevated fasting glucose (impaired fasting glucose or T2DM). An important feature of MetS, although not part of the syndrome itself, is the association with raised concentrations of CRP and other markers of inflammation

Insulin resistance is an important etiologic component of the cardiovascular risk factors representative of MetS, and T2DM is present in a majority of MetS cases. However, according to the authors of the original World Health Organization and International

Diabetes Federation definitions of MetS, it is important to recognize obesity as the causal element within MetS. Current MetS definitions are more focused on CVD risk, as opposed to diabetes risk, and, used in conjunction with other shorter-term risk prediction algorithms and sound clinical judgment, should be considered a useful tool for the prevention of the serious consequences of diabetes and CVD (Cameron, 2010).

The impact of MetS on cardiovascular disease in patients with established T2DM has been addressed in several studies (de Simone et al., 2007, Bonora et al., 2004). While there is data supporting the increased risk of prevalent and incident cardiovascular disease in patients with T2DM and comorbid MetS, the added benefit of diagnosing MetS in patients with T2DM is difficult to quantify and apply in practice, considering the high baseline cardiovascular risk associated with T2DM.

Analysis of NHANES 1992-2002 data found a 34.5% prevalence of MetS in the general population, with 64.8% in African Americans with diabetes, ages 40 and older (Lin and Pi-Sunyer, 2007). Strikingly, in our study population of Gullah with T2DM and an average age of 55.27 years, the prevalence of MetS was 85.87% (with 85.50% among those ages 40 and older).

Several U.S. and international studies have found an association between MetS and periodontitis. Analysis of data from 13,677 participants in the cross-sectional NHANES III showed an association between periodontitis and the presence of MetS. Severe periodontitis was also associated with insulin resistance (OR=1.71; CI 1.16-2.54; $p<0.01$), as well as with MetS in subjects aged older than 45 years (OR 1.74; CI 1.13-2.76; $p<0.05$), in a later report (D'Aiuto et al., 2008). In another cross-sectional study comparing periodontal status among subjects >25 years old with and without MetS living in northern Jordan, the MetS group displayed more extensive periodontal disease (higher percentages of sites with CAL ≥ 3 mm and PD ≥ 3mm), compared to the group without MetS (Khader et al., 2008). The results of our study among Gullah with T2DM showed increased rates for extent of severe periodontitis among those with MetS. This association was statistically significant in CAL-models ($p=0.03$) and marginally significant in PPD-models ($p=0.06$), independent of glycemic control (evaluated as both HbA1c ≥7% versus HbA1c <7%, and additionally as continuous HbA1c in post-hoc analyses).

The individual components of MetS have been studied, especially during the last decade, as independent risk factors for the development of periodontitis. A review regarding the association between diabetes and periodontitis supported the evidence of a bi-directional relationship between these factors (Taylor, 2001). Diabetes had an adverse effect on periodontal status, and periodontitis had an adverse effect on glycemic control. The previously published and primary analysis of this cross-sectional study population of Gullah with T2DM (from which the subject data of this report was extracted) showed that the prevalence of periodontitis was significantly higher in Gullah with diabetes mellitus compared to other African Americans with diabetes mellitus (70.6% vs. 31.3%, $p<0.001$); however, the presence of periodontal disease was not associated with uncontrolled diabetes mellitus, defined as HbA1c>7% (Fernandes et al., 2009). In contrast, our analyses showed marginally increased rates ($p=0.06$) for the extent of severe periodontitis among those with HbA1c>7% in our final CAL-model for MetS components. Additionally, in post-hoc analyses with HbA1c as a continuous covariate, results showed significant associations ($p<0.05$) in both CAL-models and marginally increased rates ($p=0.07$) in our PPD-model with MetS (yes/no).

A previous study of NHANES III data showed that BMI ≥ 27kg/m^2 was positively correlated with more periodontal disease ($p<0.05$) and greater CAL ($p<0.01$). However,

overweight individuals with insulin resistance in the highest quartile exhibited an odds ratio (OR) of 1.48 (95% CI 1.13-1.93) for severe CAL, whereas this association was not significant for subjects with high BMI and low insulin resistance (Genco et al., 2005). Our results showed marginally increased rates among those with large waist circumference in the CAL-model (p=0.09), while there were no significant interactions or associations in PPD-model.

NHANES III has also been analyzed for the relationship between markers of periodontal inflammation and disease according to arterial blood pressure (Tsakos et al., 2010). This study showed higher odds for hypertension (OR 1.0-1.1; p<0.05) in individuals with gingival bleeding as a marker of current periodontal inflammation. However, there was no association between hypertension andmoderate or severe periodontitis after adjusting for all covariates. In our study, there was no significant association between hypertension and extent of severe periodontitis in either MetS components model (CAL and PPD).

The association between periodontitis and plasma lipid profile was evaluated in a German case-control study with 79 subjects. Their results showed significantly higher absolute levels of total cholesterol (p=0.03), LDL cholesterol (p=0.01) and triglycerides (p=0.02) as well as significantly higher frequency of above normal levels of total cholesterol (p=0.03), LDL cholesterol (p=0.003) and triglycerides (p=0.001) in subjects with periodontitis; however, no association was shown among those with low HDL levels (Losche et al., 2000). A Japanese cross-sectional study that enrolled women aged 40-79 years found that those with low HDL cholesterol level had higher OR for PD ≥ 2mm (OR 2.2; CI 1.4-3.6) and CAL ≥ 3mm (OR 2.8; CI 1.4-5.6) (Shimazaki et al., 2007). We found no significant association between high triglycerides levels or low HDL levels and the extent of severe periodontitis in either MetS components model (CAL or PPD).

Our study also evaluated associations for extent of severe periodontitis with other covariates (included as potential confounders with MetS status). Both final CAL-models showed significantly decreased rates for individuals with more total teeth and among females, while there were significantly increased rates with CRP elevations in the MetS components model . There were marginally increased rates among current smokers in both final CAL-models; while the MetS status CALmodel showed marginally increased rates with CRP elevations, and the MetS components CALmodel showed marginally increased rates among current smokers. Both final PPD-models also showed significantly decreased rates among females and significantly increased rates with CRP elevations.

Our study population was limited to Gullah-speaking African Americans; therefore, our results may not apply to other populations with a different genetic and socio-cultural background. Also, all subjects in our study had T2DM, so our findings may not apply to those with MetS without a diagnosis of T2DM. The relationships among the clinical entities of periodontitis, T2DM, and MetS are very complex, and further study regarding the periodontal status of individuals who have MetS but not T2DM is especially warranted. Analyses to address these questions are beyond the scope of the present study population under investigation, who all have T2DM. The subjects were also predominantly of lower socioeconomic status with limited access to dental care; poor oral health care has traditionally been linked to lower socioeconomic status and may have influenced our results. Lastly, this study is cross-sectional, thus, we cannot comment on a causal relationship between MetS and extent of severe periodontitis.

Our results suggest that MetS significantly influences the extent of severe periodontitis in a population with T2DM, even after adjusting for several key potentially confounding factors (including age, total teeth, smoking status, HbA1c and gender). Notably, our population was

at high risk of developing periodontitis due to underlying T2DM. The presence of only one additional cardiovascular risk factor (linked to insulin resistance), did not significantly increase the extent of severe periodontitis, while the presence of two such cardiovascular risk factors (which fulfilled the criteria for diagnosing MetS) did significantly increase the extent of severe periodontitis. The extent of severe periodontitis in individuals with T2DM may exhibit a threshold phenomenon, manifested by more advanced disease in association with involvement of multiple organs and systems by the insulin resistance phenomenon (the presence of MetS). Our findings provide further support regarding the clinical importance of evaluating patients with T2DM and, additionally, MetS for periodontitis.

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Clinical Relevance

Scientific rationale for study

Associations of metabolic syndrome (MetS) with periodontitis could be due to the confounding effect of type 2 diabetes mellitus (T2DM). We evaluated the association of MetS and its individual components with the extent of severe periodontitis in Gullah African Americans with T2DM.

Principal findings

MetS is associated with the extent of severe periodontitis in patients with T2DM, while individual components of MetS are not.

Practical implications

The findings support clinical evaluation for periodontitis among patients with T2DM and, additionally, MetS.

Table 1

Study population characteristics

	All (N=283)		With Metabolic Syndrome (N=243, 85.87%)		Without Metabolic Syndrome (N=40, 14.13%)	
Variable	**Mean / N**	**SD (Range) / %**	**Mean**	**SD (Range) / %**	**Mean**	**SD (Range) / %**
Percent of sites PPD≥5mm	5.24%	10.94% (0-79.17%)	5.61%	11.59% (0-79.17%)	3.02%	5.05% (0-22.81%)
Percent of sites CAL≥6mm	5.22%	11.60% (0-79.17%)	5.59%	12.22% (0-79.17%)	2.94%	6.32% (0-33.33%)
Total teeth	19.71	6.07 (3-28)	19.54	6.04 (3-28)	20.78	6.27 (3-28)
Age (years)	55.27	10.65 (26-87)	55.47	10.52 (26-87)	54.08	11.25 (33-86)
High sensitivity CRP (mg/ml)	0.96	5.22 (0.02-87.00)	1.05	5.63 (0.02-87)	0.40	0.50 (0.02-2.28)
Serum albumin (g/dl)	3.77	0.33 (2.40-4.50)	3.76	0.33 (2.40-4.50)	3.85	0.32 (2.90-4.50)
Hemoglobin A1c (%)	7.93	2.05 (5-16.40)	7.92	1.94 (5-15.10)	8.00	2.66 (5.4-16.40)
Hemoglobin A1c ≥7%	170	60.07%	150	61.73%	20	50%
Hemoglobin A1c <7%	113	39.93%	93	38.27%	20	50%
Low serum HDL cholesterol[1] positive	140	49.47%	135	55.56%	5	12.50%
Low serum HDL cholesterol[1] negative	142	50.53%	108	44.44%	35	87.50%
High blood pressure[2] positive	241	85.16%	220	90.53%	21	52.50%
High blood pressure[2] negative	42	14.84%	23	9.47%	19	47.50%
Hypertriglyceridemia[3] positive	42	14.84%	42	17.28%	0	0%
Hypertriglyceridemia[3] negative	241	85.16%	201	82.72%	40	100%
Abdominal obesity[4] positive	239	84.45%	230	94.65%	9	22.50%
Abdominal obesity[4] negative	44	15.55%	13	5.35%	31	77.50%
BMI: <25 (kg/m^2, healthy)	28	9.89%	11	4.53%	17	42.50%
BMI: 25-30 (kg/m^2, overweight)	56	19.79%	43	17.70%	13	32.50%
BMI: ≥30 (kg/m^2, obese)	187	66.08 %	179	73.66%	8	20.00%
BMI: data missing	12	4.24%	10	4.12%	2	5.00%
Never Smoker	198	69.96%	172	70.78%	26	63.41%
Current Smoker	42	14.84%	35	14.40%	8	19.51%
Past Smoker	43	15.19%	36	14.81%	7	17.07%

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	All (N=283)		With Metabolic Syndrome (N=243, 85.87%)		Without Metabolic Syndrome (N=40, 14.13%)	
Variable	**Mean / N**	**SD (Range) / %**	**Mean**	**SD (Range) / %**	**Mean**	**SD (Range) / %**
Male	68	24.03%	51	20.99%	17	42.50%
Female	215	75.97%	192	79.01%	23	57.50%

[1]Serum HDL cholesterol <40 mg/dl (1 mmol/l) in men and <50 mg/dl (1.3 mmol/l) in women

[2]Systolic blood pressure (BP) ≥130 mmHg and/or diastolic BP ≥85 mmHg or on treatment for hypertension,

[3]Serum triglyceride level ≥150 mg/dl (1.7 mmol/l),

[4]Waist circumference >102 cm (40 inches) in men and >88 cm (35 inches) in women.

SD = Standard Deviation

Table 2

Results from multivariable negative binomial regression models for the relationship between presence of metabolic syndrome and extent of severe periodontal disease (counts of tooth-sites per person with CAL≥6 mm) among a study population of Gullah African Americans with type 2 diabetes (N=283).

Parameter	Full multivariable model (Deviance/*df* = 0.90)				Final multivariable model (Deviance/*df* = 0.89)			
	β	SE	*P*	RR and 95% CI	β	SE	*P*	RR and 95% CI
Intercept	−0.28	2.63	0.92	--------	−2.41	1.15	0.04	--------
Metabolic syndrome :yes	0.98	0.47	0.04	2.66 (1.05-6.72)	1.02	0.47	0.03	2.77 (1.11-6.93)
HbA1c ≥7%	0.34	0.30	0.26	1.41 (0.78-2.55)	0.43	0.29	0.14	1.54 (0.87-2.70)
Total teeth	−0.10	0.02	<.0001	0.90 (0.86-0.95)	−0.11	0.02	<.0001	0.90 (0.86-0.94)
BMI: 25-30 (kg/m^2, overweight)	0.24	0.53	0.65	1.27 (0.45-3.56)	0.17	0.52	0.75	1.18 (0.42-3.29)
BMI: ≥30 (kg/m^2, obese)	−0.50	0.50	0.32	0.61 (0.23-1.62)	−0.52	0.50	0.30	0.60 (0.22-1.60)
BMI: data missing	0.86	0.79	0.28	2.36 (0.50-11.05)	0.84	0.79	0.29	2.32 (0.49-10.92)
Gender: female	−0.90	0.34	<.01	0.40 (0.21-0.79)	−0.85	0.34	0.01	0.43 (0.22-0.83)
Smoking status: current	0.78	0.41	0.05	2.19 (0.99-4.85)	0.77	0.41	0.06	2.15 (0.97-4.78)
Smoking status: past	−0.06	0.38	0.88	0.95 (0.44-2.01)	−0.07	0.38	0.85	0.93 (0.44-1.97)
Age (years)	0.02	0.02	0.17	1.02 (0.99-1.05)	0.02	0.02	0.21	1.02 (0.99-1.05)
CRP (mg/ml as $\log_e$)	0.26	0.14	0.06	1.30 (0.99-1.71)	0.26	0.14	0.06	1.30 (0.99-1.71)
Serum albumin (g/dl as $\log_e$)	−1.75	1.91	0.36	0.17 (<0.01-7.42)	-----	-----	-----	-----
Dispersion parameter	4.32	0.51	-----	-----	4.35	0.51	-----	-----

SE = Standard error, RR= rate ratio, CI= confidence interval, $P \leq 0.05$

Table 3

Results from multivariable negative binomial regression models for the relationship between the components of metabolic syndrome (low HDL, hypertension, hypertriglyceridemia, abdominal obesity) and extent of severe periodontal disease (counts of tooth-sites per person with CAL≥6 mm) among a study population of Gullah African Americans with type 2 diabetes (N=283).

Parameter	Full multivariable model (Deviance/*df* = 0.91)				Final multivariable model (Deviance/*df* = 0.90)			
	β	SE	*P*	RR and 95% CI	β	SE	*P*	RR and 95% CI
Intercept	0.38	2.68	0.89	--------	−1.84	1.25	0.14	--------
Low serum HDL cholesterol [1]: yes	−0.04	0.30	0.88	0.96 (0.54-1.71)	−0.02	0.29	0.96	0.98 (0.55-1.75)
High blood pressure [2]: yes	−0.40	0.43	0.35	0.67 (0.29-1.54)	−0.41	0.42	0.33	0.66 (0.29-1.53)
Hypertriglyceridemia [3]: yes	0.31	0.45	0.50	1.36 (0.56-3.31)	0.23	0.44	0.61	1.25 (0.52-2.99)
Abdominal obesity [4]: yes	0.77	0.51	0.13	2.16 (0.80-5.83)	0.85	0.50	0.09	2.33 (0.88-6.23)
Total teeth	−0.11	0.03	<.0001	0.90 (0.85-0.94)	−0.12	0.03	<.0001	0.89 (0.85-0.94)
HbA1c ≥7%	0.45	0.31	0.15	1.58 (0.85-2.91)	0.55	0.30	0.06	1.73 (0.97-3.11)
BMI: 25-30 (kg/m^2, overweight)	−0.02	0.58	0.98	0.98 (0.32-3.04)	−.010	0.58	0.86	0.90 (0.29-2.79)
BMI: ≥30 (kg/m^2, obese)	−0.62	0.58	0.28	0.54 (0.18-1.67)	−0.67	0.58	0.24	0.51 (0.16-1.58)
BMI: data missing	0.56	0.91	0.54	1.74 (0.29-10.32)	0.45	0.94	0.62	1.56 (0.26-9.26)
Gender: female	−0.98	0.37	<.01	0.37 (0.18-0.77)	−0.94	0.36	0.01	0.39 (0.19-0.80)
Smoking status: current	0.75	0.45	0.10	2.11 (0.87-5.15)	0.77	0.46	0.09	2.16 (0.88-5.29)
Smoking status: past	−0.0005	0.41	0.999	<1.00 (0.45-2.22)	−0.01	0.40	0.98	0.99 (0.45-2.18)
Age (years)	0.03	0.02	0.09	1.03 (0.995-1.06)	0.03	0.02	0.13	1.03 (0.99-1.06)
CRP (mg/ml as $\log_e$)	0.32	0.14	0.02	1.37 (1.05-1.79)	0.32	0.14	0.02	1.37 (1.05-1.79)
Serum albumin (g/dl as $\log_e$)	−1.83	1.94	0.35	0.16 (<0.01-7.18)	-----	-----	-----	-----
Dispersion parameter	4.34	0.51	-----	-----	4.36	0.51	-----	-----

[1] Serum HDL cholesterol <40 mg/dl (1 mmol/l) in men and <50 mg/dl (1.3 mmol/l) in women,

[2] Systolic blood pressure (BP) ≥130 mmHg and/or diastolic BP ≥85 mmHg or on treatment for hypertension,

[3] Serum triglyceride level ≥150 mg/dl (1.7 mmol/l),

[4] Waist circumference >102 cm (40 inches) in men and >88 cm (35 inches) in women.

SE = Standard error, RR= rate ratio, CI= confidence interval, $P \leq 0.05$

Table 4

Results from multivariable negative binomial regression models for the relationship between presence of metabolic syndrome and extent of severe periodontal disease (counts of tooth-sites per person with PPD≥5 mm) among a study population of Gullah African Americans with type 2 diabetes (N=283).

Parameter	Full multivariable model (Deviance/*df* = 0.99)				Final multivariable model (Deviance/*df* = 0.97)			
	β	SE	*P*	RR and 95% CI	β	SE	*P*	RR and 95% CI
Intercept	−0.34	2.29	0.88	--------	−3.39	0.74	<.0001	--------
Metabolic syndrome: yes	0.79	0.42	0.06	2.21 (0.96-5.08)	0.78	0.41	0.06	2.18 (0.98-4.87)
HbA1c ≥7%	0.14	0.27	0.60	1.15 (0.68-1.95)	0.23	0.26	0.37	1.26 (0.76-2.07)
Total teeth	0.03	0.02	0.23	1.03 (0.98-1.08)	0.01	0.02	0.53	1.01 (0.97-1.06)
BMI: 25-30 (kg/m^2, overweight)	0.55	0.49	0.26	1.73 (0.66-4.47)	0.48	0.47	0.31	1.62 (0.64-4.11)
BMI: ≥30 (kg/m^2, obese)	−0.20	0.48	0.68	0.82 (0.32-2.10)	−0.17	0.45	0.70	0.84 (0.35-2.02)
BMI: data missing	0.61	0.71	0.39	1.84 (0.45-7.46)	0.65	0.69	0.35	1.91 (0.49-7.44)
Smoking status: current	−0.01	0.36	0.9722	0.99 (0.48-2.02)	------	------	------	-----
Smoking status: past	−0.25	0.36	0.49	0.78 (0.39-1.57)	------	------	------	-----
Gender: female	−0.76	0.31	0.01	0.47 (0.26-0.85)	−0.65	0.29	0.02	0.52 (0.30-0.92)
Age (years)	0.004	0.01	0.76	1.004 (0.98-1.03)	------	------	------	-----
CRP (mg/ml as $\log_e$)	0.32	0.13	0.01	1.37 (1.07-1.76)	0.31	0.11	<.01	1.37 (1.09-1.71)
Serum albumin (g/dl as $\log_e$)	−2.57	1.69	0.13	0.08 (0.003-2.10)	------	------	------	-----
Dispersion parameter	3.63	0.39	------	-----	3.69	0.40	------	-----

SE = Standard error, RR= rate ratio, CI= confidence interval, $P \leq 0.05$

Table 5

Results from multivariable negative binomial regression models for the relationship between the components of metabolic syndrome (low HDL, hypertension, hypertriglyceridemia, abdominal obesity) and extent of severe periodontal disease (counts of tooth-sites per person with PPD≥5 mm) among a study population of Gullah African Americans with type 2 diabetes (N=285).

Parameter	Full multivariable model (Deviance/*df* = 1.00)				Final multivariable model (Deviance/*df* = 0.99)			
	β	SE	*P*	RR and 95% CI	β	SE	*P*	RR and 95% CI
Intercept	0.12	2.31	0.9583	------	0.25	2.22	0.91	---------
Low serum HDL cholesterol [1]: yes	−0.09	0.27	0.7211	0.91 (0.54-1.53)	−0.16	0.26	0.53	0.85 (0.51-1.42)
High blood pressure [2]: yes	−0.06	0.41	0.8808	0.94 (0.42-2.10)	−0.12	0.36	0.73	0.88 (0.44-1.79)
Hypertriglyceridemia [3]: yes	0.61	0.39	0.1192	1.83 (0.86-3.93)	0.54	0.35	0.13	1.71 (0.85-3.41)
Abdominal obesity [4]: yes	0.54	0.45	0.2311	1.72 (0.71-4.18)	0.51	0.44	0.24	1.67 (0.71-3.94)
HbA1c ≥7%	0.23	0.28	0.4214	1.25 (0.72-2.17)	0.24	0.26	0.35	1.28 (0.76-2.14)
Total teeth	0.02	0.02	0.4160	1.02 (0.97-1.07)	------	------	------	------
BMI: 25-30 (kg/m^2, overweight)	0.33	0.52	0.5242	1.39 (0.50-3.87)	0.34	0.50	0.50	1.40 (0.53-3.73)
BMI: ≥30 (kg/m^2, obese)	−0.29	0.54	0.5914	0.75 (0.26-2.15)	−0.23	0.51	0.66	0.79 (0.29-2.18)
BMI: data missing	0.55	0.80	0.4876	1.74 (0.36-8.28)	0.68	0.77	0.39	1.47 (0.31-6.91)
Smoking status: current	−0.10	0.39	0.7919	0.90 (0.42-1.95)	------	------	------	------
Smoking status: past	−0.33	0.37	0.3654	0.72 (0.35-1.47)	------	------	------	------
Age (years)	0.01	0.01	0.5345	1.01 (0.98-1.04)	------	------	------	------
CRP (mg/ml as $\log_e$)	0.35	0.13	0.0057	1.42 (1.11-1.83)	0.32	0.11	<.01	1.37 (1.10-1.70)
Serum albumin (g/dl as $\log_e$)	−2.76	1.69	0.1023	0.06 (0.002-1.74)	-2.24	1.56	0.15	0.10 (0.01-2.28)
Gender: female	−0.76	0.33	0.0198	0.47 (0.25-0.89)	-0.68	0.30	0.02	0.50 (0.28-0.91)
Dispersion parameter	3.61	0.39	------	------	3.64	0.39	------	------

[1] Serum HDL cholesterol <40 mg/dl (1 mmol/l) in men and <50 mg/dl (1.3 mmol/l) in women,

[2] Systolic blood pressure (BP) ≥130 mmHg and/or diastolic BP ≥85 mmHg or on treatment for hypertension,

[3] Serum triglyceride level ≥150 mg/dl (1.7 mmol/l),

[4] Waist circumference >102 cm (40 inches) in men and >88 cm (35 inches) in women.

SE = Standard error, RR= rate ratio, CI= confidence interval, $P \leq 0.05$