J Clin Periodontol 2010; 37: 501–509 doi: 10.1111/j.1600-051X.2010.01564.x

Journal of Clinical Periodontology

Periodontal disease progression and glycaemic control among Gullah African Americans with type-2 diabetes

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Bandyopadhyay D, Marlow NM, Fernandes JK, Leite RS: Periodontal disease progression and glycaemic control among Gullah African Americans with type-2 diabetes. J Clin Periodontol 2010; 37: 501–509. doi: 10.1111/j.1600-051X.2010.01564.x.

Abstract

Aim: To evaluate associations between glycaemic control and periodontitis progression among Gullah African Americans with type-2 diabetes mellitus (T2DM).

Materials and Methods: From an ongoing clinical trial among T2DM Gullah, we extracted a cohort previously in a cross-sectional study ($N = 88$). Time from baseline (previous study) to follow-up (trial enrollment, before treatment interventions) ranged 1.93–4.08 years [mean = 2.99, standard deviation (SD) = 0.36]. We evaluated tooth site-level periodontitis progression [clinical attachment loss (CAL) worsening of $\geqslant 2$ mm, periodontal probing depth (PPD) increases of $\geqslant 2$ mm and bleeding on probing (BOP) from none to present] by glycaemic control status (well-controlled = $HbA_{1c} < 7\%$, poorly-controlled = $HbA_{1c} \geqslant 7\%$) using multivariable generalized estimating equations logistic regression, nesting tooth sites/person.

Results: Poorly-controlled T2DM (68.18%) was more prevalent than well-controlled T2DM (31.82%). Proportions of tooth sites/person with CAL progression between baseline and follow-up ranged 0.00–0.59 (mean = 0.12, SD = 0.12), while PPD and BOP progression ranged 0.00–0.44 (mean = 0.09, SD = 0.11) and 0.00–0.96 (mean = 0.24, SD = 0.18), respectively. Site-level PPD at baseline was a significant effect modifier of associations between poorly-controlled T2DM and site-level CAL and PPD progression [adjusted odds ratios (OR) according to poorly-controlled T2DM among PPD at baseline = 3, 5 and 7 mm, respectively: CAL progression = 1.93, 2.64, and 3.62, PPD progression = 1.98, 2.76, and 3.84; $p < 0.05$ for all]. Odds of site-level BOP progression were increased (OR = 1.24) for poorly-controlled T2DM, yet the results were not significant ($p = 0.32$).

Conclusions: These findings from a distinct, homogenous population further support the clinical relevance of identifying patients with poor glycaemic control and periodontitis, particularly among those with disparities for both diseases.

Key words: diabetes; glycaemic control; Gullah African Americans; multivariable regression analysis; progression of periodontal disease

Accepted for publication 27 February 2010

Conflict of interest and source of funding statement

The authors report no conflicts of interest related to this study.

This work was conducted with support from the National Institutes of Health, the National Center of Research Resources Grant Number P20 RR017696, South Carolina COBRE in Oral Health and it was also supported in part by grant M01 RR001070 from the National Center for Research Resources, National Institutes of Health.

There is evidence that periodontal disease can worsen diabetic control capabilities and (vice versa) that proper management of periodontal disease can improve diabetic control (Soskolne & Klinger 2001). The host inflammatory response appears to be the critical

determinant for the susceptibility to and the severity of marginal periodontitis, especially in systemically compromised individuals (Williams & Offenbacher 2000, Takeda et al. 2006), including those with type-2 diabetes mellitus (T2DM). Results from three independent studies that examined the role of periodontal disease as a factor complicating the severity of diabetes consistently indicate that those subjects with severe periodontitis exhibit more diabetes complications compared with those with no or mild periodontitis, suggesting that severe periodontitis confers a significant risk for these complications (Grossi 2001). Also, in a longitudinal study of native Americans from the Gila River Reservation, the Pima Indians who exhibited severe periodontitis at baseline subsequently had a worse glycated α-haemoglobin (HbA_{1c}) level at a 2-year follow-up examination compared with those with little or no periodontitis (Taylor et al. 1996), indicating poorer diabetic control.

Previous results have also shown that after adjusting for population age differences, non-Hispanic African Americans are 1.8 times more likely to have T2DM compared with non-Hispanic Caucasians. Sea Island Gullah African Americans (or simply the Gullah) of coastal South Carolina and Georgia have a particularly high degree of genetic risk for T2DM, with a 3.3 relative risk of T2DM to siblings, a figure that exceeds that in many other communities (Garvey et al. 2003). The Gullah are a direct descendant population of rice plantation-enslaved Africans from Sierra Leone and certain other parts of West Africa (McLean et al. 2005). Their ancestors remained in their Gullah communities when these slave practices became illegal (Pollitzer 1999). Today, the Gullah have a considerably lower level of non-African genetic admixture as compared with other African American populations (Parra et al. 2001), which is thought to be largely due to their longtime geographical, social and cultural isolation (McLean et al. 2003).

The disease burden among the Gullah is not limited to just T2DM, as the prevalence of other chronic diseases, such as hypertension and obesity, in the coastal areas of South Carolina leads most other counties in the state. Additionally, a previous report found significantly higher prevalence rates of periodontal disease among Gullah with T2DM (70.6%) as compared with national estimates of African Americans with diabetes (31.3%) (Fernandes et al. 2009). These considerable disparities for chronic diseases and their additional genetic homogeneity position the Gullah as a remarkable population to study.

Therefore, the aim of our study was to evaluate associations between glycaemic control status and tooth site-level periodontal disease progression among Gullah with T2DM.

Materials and Methods

Study population

The study population of this report was extracted from an ongoing clinical trial to evaluate periodontal disease treatment interventions among adult Gullah African Americans with T2DM. We selected subjects enrolled in the clinical trial, from December 2007 through March 2009, who had also participated in a previous cross-sectional study, described elsewhere (Fernandes et al. 2009). This process accomplished a longitudinal cohort ($N = 98$), and the results reported herein are limited to data collected from baseline (the previous study) to the follow-up visit (the clinical trial enrollment, before treatment interventions). This cohort was further limited for our analyses to subjects ($N = 88$) with non-missing data for HbA_{1c} as well as our covariates of interest (further described in the ''Data analyses''). The time from baseline to follow-up for our final cohort ($N = 88$) ranged from 1.93 to 4.08 years [mean = 2.99, standard deviation (SD) = 0.36]. The previous study was an epidemiologic assessment limited to data collection and did not include periodontal therapy. The clinical trial enrollment process provided us with an opportunity to look at this population again, and the results of this new study (including results following periodontal treatment interventions) will be presented in a future report.

The study protocol was clearly explained to potential subjects and Institutional Review Board-approved consent and HIPAA forms were required for study inclusion. Consenting individuals with a minimum of three natural teeth (excluding third molars) and no receipt of periodontal treatment in the 6 months before their interview were eligible for inclusion in the ongoing clinical trial. Subjects were further excluded from the clinical trial if they presented with a finger stick blood glucose measurement of < 70 mg/dl or > 350 mg/dl, fasting serum C-peptide < 1 ng/ml, serum creatinine $\geqslant 1.6$ mg/dl, abnormal hepatic function, haemoglobinopathy (sickle cell trait/haemolytic anaemia, which would interfere with HbA_{1c} monitoring) or any other underlying illness/conditions that, in the subject's physician's judgement, might have prevented adherence to the study protocol. Additional clinical trial exclusion criteria included pregnancy, a requirement for antibiotic prophylaxis before dental procedures, advanced periodontal disease that required full-mouth extraction and/or treatment with any kind of antibiotics in the 6 months before the initial visit of the clinical trial. Subjects excluded from the trial were eligible to be screened for subsequent study entry when these criteria were corrected/reversed.

Clinical assessment

Clinical diabetes assessment consisted of a thorough medical history with particular attention to a family history of diabetes. Anthropometrics included weight and height. Medical examination included blood pressure and pulse rate. Clinical laboratory measures included HbA_{1c}, creatinine clearance, C-peptide, random/fasting plasma glucose, high-sensitivity C-reactive protein, urine albuminuria and lipid panel. All assessments and specimen collections were performed on the same day of subject service whenever possible. HbA_{1c} levels were analysed with high-pressure liquid chromatography by the Medical University Hospital Lab. The HbA_{1c} level was measured at baseline (under the previous cross-sectional study) and at the clinical trial initiation (the longitudinal follow-up visit).

Oral health assessment

Details of dental hygiene behaviours were assessed and an oral examination was performed, including radiographic and soft tissue exam, evaluation of bleeding on probing (BOP), calculus presence, periodontal probing depths (PPD) and attachment levels (AL). Six sites (mesio-buccal, buccal, disto-buccal, disto-lingual, lingual and mesio-lingual) were examined for each tooth, excluding third molars. Oral examiners were calibrated according to the methods previously published (Hill et al. 2006). Agreement was observed within

1 mm among the two study examiners, and the standard ranged from 96% to 99% for PPD, 91% to 94% for AL and 99% for CEJ-GM (Hill et al. 2006). Oral health practice was assessed by a questionnaire including the number of times that subjects brush and/or floss their teeth per day as well as how many times in the past year the subject visited the dentist for emergency and/or preventive care.

Data analyses

Analyses for our final cohort ($N = 88$) were limited to tooth sites with non-missing data for periodontal measures (AL, PPD, and BOP) at both baseline and the follow-up. Some of these subjects may have received emergency dental care in the interim period between studies, which primarily involved tooth extractions. For these individuals, the follow-up data would be considered missing for such particular teeth and, further, not included in our analyses. Further, some of these subjects may have had data for a particular site missing at baseline but present at follow-up, in which case, the progression of periodontal disease could not be measured, and that site would be excluded from our analyses. Consequently, among our final cohort ($N = 88$), there were 10,148 tooth sites with PPD and AL measures at baseline and 9880 tooth sites with PPD and AL measures at follow-up, resulting in a decrease of 268 (2.64%) tooth sites measured from baseline to follow-up. With such a small decrease in the number of total tooth sites with PPD and AL measures available, we do not feel that this pattern of missing data (for either tooth extractions in the interim, data entry errors or failure to measure and record the respective data) has appreciably affected our results reported herein. Additionally, there were 9601 tooth sites with BOP measures at baseline and 9646 tooth sites with BOP measures at follow-up, resulting in an increase of 45 (0.47%) tooth sites measured from baseline to follow-up.

For our final cohort ($N = 88$), there were 9648 tooth sites with PPD and AL measures as well as 9599 tooth sites with BOP measures at both baseline and follow-up (ranging from 24 to 168 sites per person) and these were further included in our evaluations of periodontal disease progression. All statistical analyses were generated using SAS software, version 9.2 (SAS Institute Inc. 2002–2003). The odds of periodontal disease progression per tooth site [$\geqslant 2$ mm worsening of clinical attachment loss (CAL), $\geqslant 2$ mm increase in PPD and emergence of BOP, each evaluated separately] was analysed according to the glycaemic control status in multivariable logistic regression models using generalized estimating equations (GEE) (Liang & Zeger 1986, Zeger et al. 1994). Thresholds for disease advancement were selected at levels that were deemed clinically meaningful and would considerably minimize the chances of incorrect classifications due to measurement error (Lindhe et al. 1983, Haffajee et al. 1985, Hill et al. 2006). Subjects were considered to have well-controlled diabetes if $HbA_{1c} < 7\%$ and poorly-controlled if $HbA_{1c} \geqslant 7\%$ (American Diabetes Association 2006). Subject level of baseline periodontal disease was evaluated as a covariate according to PPD (mm) at baseline; because AL is a composite measure that includes PPD, baseline AL was not included as an additional covariate due to potential problems of statistical colinearity between AL and PPD. Other assessed covariates included gender (male, female), age at baseline (years), smoking status at baseline (never, current, past), body mass index (BMI) at baseline (normal: <25, overweight: 25–30, obese: >30), type of tooth (molar, non-molar) and location of tooth (upper jaw, lower jaw). Periodontal disease progression measures (CAL, PPD and BOP), HbA_{1c} levels and the covariates were summarized by either mean and SD results (if continuous) or frequency results (if categorical) and results were reported by their overall and their HbA_{1c} status-specific distributions (Table 1).

GEE methods are capable of accounting for correlation among tooth sites from the same subject. An examiner can measure site-level PPD, AL, and BOP for up to 168 tooth sites per person (for those with all 28 teeth of interest present). Failure to account for correlation among such measures in statistical analyses often leads to underestimation

Table 1. Characteristics for a cohort of Gullah African Americans with type-2 diabetes mellitus ($N = 88$), overall and by glycaemic control status at follow-up visit

Variable	Mean ± S* (range) or *N* (%)		
	All ($N = 88$)	$Hb_{A1c} < 7\%$ ($N = 28$)	$Hb_{A1c} \geqslant 7\%$ ($N = 60$)
Age (years) at baseline	55.57 ± 8.96 (34–77)	54.68 ± 10.73 (34–77)	55.98 ± 8.07 (34–71)
Hb_{A1c} at baseline	8.09 ± 1.94 (5.10–15.10)	6.99 ± 1.66 (5.10–11.90)	8.61 ± 1.85 (6.00–15.10)
Hb_{A1c} at follow-up visit	8.13 ± 1.98 (4.70–12.80)	6.14 ± 0.47 (4.70–6.90)	9.06 ± 1.71 (7.00–12.80)
Proportion of sites/person with PPD increases $\geqslant 2$ mm	0.09 ± 0.11 (0–0.44)	0.06 ± 0.05 (0–0.17)	0.10 ± 0.12 (0–0.44)
Proportion of sites/person with CAL of $\geqslant 2$ mm	0.12 ± 0.12 (0–0.59)	0.09 ± 0.07 (0–0.31)	0.13 ± 0.14 (0–0.59)
Proportion of sites/person with new BOP	0.24 ± 0.18 (0–0.96)	0.20 ± 0.14 (0.03–0.60)	0.26 ± 0.19 (0–0.96)
PPD (mm) at baseline (nested sites/person)	1.85 ± 0.07 (0–12)	1.92 ± 0.10 (0–11)	1.82 ± 0.09 (0–12)
AL (mm) at baseline (nested sites/person)	1.86 ± 0.09 (0–14)	1.86 ± 0.16 (0–13)	1.87 ± 0.11 (0–12)
BOP (1 = yes, 0 = no) at baseline (nested sites/person)	0.51 ± 0.03 (0, 1)	0.56 ± 0.05 (0, 1)	0.49 ± 0.04 (0, 1)
Smoking status at baseline: never	67 (76.14%)	21 (75.00%)	46 (76.67%)
Smoking status at baseline: current	6 (6.82%)	2 (7.14%)	4 (6.67%)
Smoking status at baseline: past	15 (17.05%)	5 (17.86%)	10 (16.67%)
Male	19 (21.59%)	5 (17.86%)	14 (23.33%)
Female	69 (78.41%)	23 (82.14%)	46 (76.67%)
BMI at baseline <25 (normal)	5 (5.68%)	0 (0%)	5 (8.33%)
BMI at baseline 25–30 (overweight)	20 (22.73%)	5 (17.86%)	15 (25.00%)
BMI at baseline >30 (obese)	63 (71.59%)	23 (82.14%)	40 (66.67%)

*Standard deviation for all, except PPD, AL, and BOP at baseline (nested sites/person) = standard error.

CAL, clinical attachment loss; PPD, periodontal probing depth; BOP, bleeding on probing; AL, attachment level, BMI, body mass index.

of the true *p*-values and narrowing of confidence intervals for the estimated effects (Ananth & Kantor 2004, Ananth et al. 2005), such as odds ratio (OR) results produced via logistic regression models. GEE analyses incorporate a series of iterative modelling procedures, and the final regression parameter estimates are robust to misspecification of the working correlation matrix (Stokes et al. 2000), which makes GEE an attractive method for modelling non-normal clustered data. The GEE procedure in SAS (provided in PROC GENMOD) provides several options for the structure of the working correlation matrix (e.g., autoregressive, exchangeable, independent, m-dependent, unstructured). In this study, we assumed that the correlation matrix for any given subject was exchangeable, meaning a fixed correlation between any two units (i.e., PPD, AL or BOP measures) from the same subject.

Three separate series of multivariable logistic regression models were produced for whether or not each tooth site (nested within person) had the following: (1) CAL increases of $\geqslant 2$ mm, (2) PPD increases of $\geqslant 2$ mm and (3) presence of BOP. First, for each of these three progression measures, interaction terms for HbA_{1c} status by baseline PPD as well as terms for smoking status by baseline PPD were separately evaluated in full multivariable models along with all other previously described covariates (Ylostalo & Knuuttila 2006). For the CAL and PPD progression models, there were significant interactions for baseline PPD by HbA_{1c} status ($p < 0.05$ for the estimated regression coefficient) and yet not for baseline PPD by smoking status. For the BOP progression model, there were significant interactions for baseline PPD by smoking status ($p < 0.05$ for the estimated regression coefficient) and yet not for baseline PPD by HbA_{1c} status. Then, predictors other than the significant interaction parameters (baseline PPD by HbA_{1c} status for CAL and PPD progression models, baseline PPD by smoking status for BOP progression models) and HbA_{1c} status were successively removed through a process of backward elimination based on *p*-values of the estimated regression coefficients (removing those with $p > 0.05$). The final three models (CAL, PPD and BOP progression per site, nested within person) included the interaction parameters, HbA_{1c} status and all other predictors that either showed significant associations with the respective progression measure or resulted in a significant improvement in model goodness-of-fit (GOF) statistics. The estimated regression coefficients from these final models were used to calculate the covariate adjusted OR and associated 95% CI for periodontal disease progression (increased CAL, PPD and BOP) per site.

Often reported for the final results of statistical methods involving ordinary least squares (OLS)-based linear regression, the R^2 (coefficient of determination) is a measure of the proportion of total variability in a data set explained by the regression model. However, our method of analysis is based on GEE, a moment-based statistical methodology that does not consider data likelihood during the estimation process. Therefore, corresponding R^2 measures often used in OLS-based linear regression are not available here. Instead, we evaluated and report a measure of ''overall'' R^2 (henceforth R^{*2}) for GEE as outlined in Natarajan et al. (2007), which is an extension of the R^2 available in linear regression. Also, to assess the GOF of our GEE models, we evaluated and report an extension of the Hosmer–Lemeshow statistics as outlined in Horton et al. (1999), which accommodates clustered binary responses. This GOF measure is based on 10 groups and, hence, is distributed as χ^2_9 (a χ^2 distribution with nine degrees of freedom).

Results

The mean duration of diabetes at baseline of the previous cross-sectional study, among subjects who were included in the present cohort study was 10.65 years (SD = 8.76) (data not shown). The time from baseline to the follow-up visit ranged from 1.93 to 4.08 years (mean = 2.99, SD = 0.36); the mean follow-up times were not significantly different ($p = 0.38$) according to the subject's level of glycaemic control measured at the follow-up visit (data not shown). Subject age at baseline ranged from 34 to 77 years (mean = 55.57, SD = 8.96) (Table 1). At baseline, only 5.68% of the subjects were within the normal range for BMI; 22.73% were overweight and 71.59% were obese (Table 1). Smoking status reported at baseline mostly comprised of no history of smoking (76.14%), followed by past smokers (17.05%) and current smokers (6.82%) (Table 1). Nesting tooth sites within person, the average PPD at baseline was 1.85 mm [standard error (SE) = 0.07, range = 0–12] for all subjects, with 1.92 mm (SE = 0.10, range = 0–11) among those with well-controlled diabetes and 1.82 mm (SE = 0.09, range = 0–12) among those with poorly-controlled diabetes (Table 1). Also nesting tooth sites within person, the average AL at baseline was 1.86 mm (SE = 0.09, range = 0–14), with 1.87 mm (SE = 0.11, range 0–12) among those with well-controlled diabetes and 1.86 mm (SE = 0.16, range = 0–13) among those with poorly-controlled diabetes (Table 1). The average for BOP at baseline while nesting tooth sites within persons was 0.51 (SE = 0.03, values = 0/1), with 0.49 (SE = 0.05, values = 0/1) among those with well-controlled diabetes and 0.56 (SE = 0.04, values = 0/1) among those with poorly-controlled diabetes (Table 1).

HbA_{1c} levels at baseline ranged 5.10–15.10% (mean = 8.09, SD = 1.94) (Table 1), whereas at follow-up they ranged 4.70–12.80% (mean = 8.13, SD = 1.98) (Table 1). Subjects with poorly-controlled diabetes at follow-up (68.18%) were more prevalent than well-controlled (31.82%) (Table 1). The proportion of sites per person with CAL progression from baseline to follow-up ranged from 0.00 to 0.59 (mean = 0.12, SD = 0.12) (Table 1), while similar proportions for PPD and BOP progression ranged from 0.00 to 0.44 (mean = 0.09, SD = 0.11) and 0.00 to 0.96 (mean = 0.24, SD = 0.18) (Table 1), respectively. Female subjects (78.41%) were much more prevalent than males (21.59%), consistent with previously reported gender-related participation rate differences for studies involving the Gullah (Johnson-Spruill et al. 2009). Additional descriptive statistics for this population including distributions by glycaemic control status are displayed in Table 1.

The final multivariable model results for PPD progression showed that tooth sites (nested within person) from subjects with poor glycaemic control had significantly increased odds for PPD progression compared with tooth sites from subjects with well-controlled diabetes and this association was significantly modified by PPD at baseline for the particular tooth site [$p = 0.0201$ for the interaction term (Table 2)]. The OR according to poor glycaemic control in the final model (adjusted for gender, BMI, and molar/non-molar tooth site)

Table 2. Results from multivariable logistic regression models for the relationship between glycaemic control (HbA_{1c}) and site level increases in PPD $\geqslant$2 mm for a cohort of Gullah African Americans with diabetes ($N = 88$)

Parameters	Full multivariable model					Final multivariable model				
	β	SE	p	OR	OR 95% CI	β	SE	p	OR	OR 95% CI
Intercept	−4.28	0.93	<0.0001	N/A	N/A	−3.33	0.42	<0.0001	N/A	N/A
$HbA_{1c} \geqslant 7\%$	0.19	0.29	0.5221	*	*	−3.50	0.43	<0.0001	*	*
PPD at baseline (mm) (b-PPD)	−0.17	0.05	0.0013	*	*	0.19	0.29	0.5201	*	*
$HbA_{1c} \geqslant 7\%$ by b-PPD†	0.17	0.07	0.0175	*	*	−0.16	0.05	0.0021	*	*
Age at baseline (years)	0.01	0.01	0.4554	1.01	0.01–1.04	–	–	–	–	–
Male	0.35	0.31	0.2568	1.43	0.45–2.63	0.38	0.26	0.1492	1.46	0.87–2.43
Past smoker at baseline	0.31	0.37	0.4062	1.36	0.50–2.81	–	–	–	–	–
Current smoker at baseline	−0.11	0.51	0.8356	0.90	0.46–2.43	–	–	–	–	–
Overweight at baseline (25–30 BMI)	0.83	0.43	0.0496	2.30	0.98–5.30	0.73	0.44	0.0978	2.07	0.88–4.88
Obese at baseline (>30 BMI)	1.09	0.38	0.0041	2.97	1.13–6.25	0.97	0.38	0.0103	2.64	1.26–5.55
Molar tooth site	0.37	0.11	0.0007	1.45	0.16–1.80	0.37	0.11	0.0007	1.44	1.17–1.79
Upper-jaw tooth site	0.09	0.10	0.3614	1.09	0.11–1.32	–	–	–	–	–

Tooth sites were nested within person using a generalized estimating equations methodology (total sites = 9648, ranging 24–168 sites per person).
*Refer to Table 5.
†Interaction term.
BMI, body mass index; PPD, periodontal probing depth; OR, odds ratio; CI, confidence interval.

Table 3. Results from multivariable logistic regression models for the relationship between glycaemic control (HbA_{1c}) and site-level CAL of $\geqslant$2 mm for a cohort of Gullah African Americans with diabetes ($N = 88$)

Parameters	Full multivariable model					Final multivariable model				
	β	SE	p	OR	OR 95% CI	β	SE	p	OR	OR 95% CI
Intercept	−6.01	0.8962	<0.0001	N/A	N/A	−5.72	0.85	<0.0001	N/A	N/A
$HbA_{1c} \geqslant 7\%$	0.18	0.25	0.4757	*	*	0.18	0.24	0.4501	*	*
PPD at baseline (mm) [b-PPD]	−0.13	0.06	0.0347	*	*	−0.13	0.06	0.0434	*	*
$HbA_{1c} \geqslant 7\%$ by b-PPD†	0.16	0.08	0.0365	*	*	0.16	0.08	0.0377	*	*
Age at baseline (years)	0.03	0.01	0.0158	1.03	1.01–1.06	0.03	0.01	0.0238	1.03	1.004–1.06
Male	0.33	0.31	0.2832	1.39	0.76–2.52	–	–	–	–	–
Past smoker at baseline	0.70	0.32	0.0294	2.01	1.07–3.78	0.80	0.28	0.0036	2.23	1.30–3.83
Current smoker at baseline	0.01	0.39	0.9827	1.01	0.47–2.18	0.03	0.39	0.9373	1.03	0.48–2.23
Overweight at baseline (25–30 BMI)	1.56	0.46	0.0007	4.77	1.94–11.73	1.45	0.44	0.0011	4.27	1.79–10.20
Obese at baseline (>30 BMI)	1.89	0.38	<0.0001	6.61	3.09–14.11	1.81	0.35	<0.0001	6.09	3.04–12.19
Molar tooth-site	0.19	0.09	0.0488	1.21	1.001–1.45	0.19	0.10	0.0473	1.21	1.002–4.46
Upper-jaw tooth-site	0.04	0.10	0.7248	1.04	0.85–1.26	–	–	–	–	–

Tooth sites were nested within person using a generalized estimating equations methodology (total sites = 9648, ranging 24–168 sites per person).
*Refer to Table 5.
†Interaction term.
BMI, body mass index; CAL, clinical attachment loss; PPD, periodontal probing depth; OR, odds ratio; CI, confidence interval.

was 1.98 (95% CI = 1.22–3.21) among those with PPD = 3 mm at baseline, 2.76 (95% CI = 1.52–5.01) among those with PPD = 5 mm at baseline and 3.85 (95% CI = 1.74–8.49) among those with PPD = 7 mm at baseline (Table 5). Also using this final model, molar tooth sites had significantly increased odds for PPD progression compared with non-molar tooth sites (OR = 1.44, 95% CI = 1.17–1.79) (Table 2). Also, tooth sites from subjects with obese BMI levels had significantly increased odds for PPD progression compared with tooth sites from subjects with normal BMI levels (OR = 2.64, 95% CI = 1.26–5.55) (Table 2). However, being overweight was not significantly associated with the odds of site level PPD progression, nor was age, gender, smoking status or being an upper-jaw tooth site (Table 2).

The final multivariable model results for CAL progression showed that tooth sites (nested within person) from subjects with poor glycaemic control had significantly increased odds for CAL progression compared with tooth sites from subjects with well-controlled diabetes and this association was significantly modified by PPD at baseline for the particular tooth site [$p = 0.0377$ for the interaction term (Table 2)]. The OR according to poor glycaemic control in the final model (adjusted for age, smoking, BMI and molar/non-molar tooth site) was 1.93 (95% CI = 1.20–3.10) among those with PPD = 3 mm at baseline, 2.64 (95% CI = 1.36–5.14) among those with PPD = 5 mm at baseline and 3.62 (95% CI = 1.45–9.05) among those with PPD = 7 mm at baseline (Table 5). Also, using this final model, molar tooth sites had significantly increased odds for CAL progression compared with non-molar tooth sites (OR = 1.21, 95% CI = 1.02–4.46) (Table 3). Tooth sites from subjects with BMI levels above normal limits (obese: OR = 6.09, 95% CI = 3.04–12.19; overweight: OR = 4.27, 95% CI = 1.79–10.20)

Table 4. Results from multivariable logistic regression models for the relationship between glycaemic control (HbA_{1c}) and new presentation of site-level BOP for a cohort of Gullah African Americans with diabetes ($N = 88$)

Parameter	Full multivariable model					Final multivariable model				
	β	SE	p	OR	OR 95% CI	β	SE	p	OR	OR 95% CI
Intercept	−0.002	0.84	0.9979	N/A	N/A	−1.08	0.21	<0.0001	N/A	N/A
$HbA_{1c} \geqslant 7\%$	0.18	0.22	0.4063	1.20	0.78–1.86	0.21	0.21	0.3272	1.23	0.81–1.86
PPD at baseline (mm) (b-PPD)	−0.10	0.05	0.0330	*	*	−0.11	0.05	0.0252	*	*
Past smoker at baseline	0.21	0.32	0.5064	*	*	0.16	0.34	0.6455	*	*
Current smoker at baseline	0.82	0.28	0.0036	*	*	0.83	0.27	0.0020	*	*
Past-smoker by b-PPD†	−0.14	0.07	0.0351	*	*	−0.13	0.06	0.0440	*	*
Current-smoker by b-PPD†	−0.17	0.14	0.2121	*	*	−0.17	0.14	0.2084	*	*
Age at baseline (years)	−0.01	0.01	0.3222	0.99	0.97–1.01	–	–	–	–	–
Male	−0.25	0.30	0.3969	0.78	0.44–1.39	–	–	–	–	–
Overweight at baseline (25–30 BMI)	−0.41	0.50	0.4152	0.67	0.25–1.77	–	–	–	–	–
Obese at baseline (>30 BMI)	−0.51	0.49	0.2995	0.60	0.23–1.57	–	–	–	–	–
Molar tooth site	−0.06	0.07	0.4248	0.94	0.82–1.09	–	–	–	–	–
Upper-jaw tooth site	−0.15	0.07	0.0369	0.86	0.75–0.99	−0.15	0.07	0.0387	0.86	0.79–0.99

Tooth sites were nested within person using a generalized estimating equations methodology (total sites = 9599, ranging 24–168 sites per person).
*Refer to Table 5.
†Interaction term.
BOP, bleeding on probing; PPD, periodontal probing depth; OR, odds ratio; CI, confidence interval.

Table 5. Odds ratio results from significant interaction terms of multivariable logistic regression models for site-level periodonditis progression, a cohort of Gullah African Americans with diabetes ($N = 88$)

Outcome by effect modification parameters	Full multivariable model			Final multivariable model		
	OR	OR 95% CI	P	OR	OR 95% CI	P
Outcome: site-level PPD progression						
OR estimates for $HbA_{1c} \geqslant 7\%$ by baseline PPD (mm)						
Strata: PPD = 3	1.99	1.22–3.26	0.0062	1.98	1.22–3.21	0.0053
Strata: PPD = 5	2.78	1.51–5.13	0.0011	2.76	1.52–5.01	0.0008
Strata: PPD = 7	3.88	1.72–8.74	0.0011	3.84	1.74–8.49	0.0009
Outcome: site-level CAL progression						
OR estimates for $HbA_{1c} \geqslant 7\%$ by baseline PPD (mm)						
Strata: PPD = 3	1.91	1.18–3.09	0.0084	1.93	1.20–3.10	0.0065
Strata: PPD = 5	2.61	1.34–5.09	0.0048	2.64	1.36–5.14	0.0042
Strata: PPD = 7	3.58	1.43–8.91	0.0063	3.62	1.45–9.05	0.0059
Outcome: site-level BOP progression						
OR estimates for baseline PPD (mm) by smoking						
Strata: never smoker at baseline	0.91	0.83–0.99	0.0330	0.90	0.82–0.99	0.0252
Strata: past-smoker at baseline	0.79	0.73–0.86	<0.0001	0.79	0.73–0.86	<0.0001
Strata: current smoker at baseline	0.76	0.59–0.99	0.0402	0.76	0.58–0.98	0.0325

CAL, clinical attachment loss; PPD, periodontal probing depth; BOP, bleeding on probing; AL, attachment level, BMI, body mass index; OR, odds ratio; CI, confidence interval.

and from subjects who were ''past'' smokers (*versus* ''never'' smokers: OR = 2.23, 95% CI = 1.30–3.83) had significantly increased odds for CAL progression (Table 3). Additionally, with a yearly increase in the subject's age, the odds of CAL progression per tooth site significantly increased by 3% (OR = 1.03, 95% CI = 1.004–1.06) (Table 3). Gender was not significantly associated with the odds of PPD progression, nor was being an upper-jaw tooth site or ''current'' smoker (Table 3).

Lastly, the final multivariable model for BOP progression showed that tooth site (nested within person) from subjects with poor glycaemic control had increased odds for BOP progression compared with subjects with well-controlled diabetes (OR = 1.23, 95% CI = 0.81–1.86; adjusted for PPD at baseline, smoking status and upper/lower-jaw location of tooth site), and yet these results were not statistically significant (Table 4). Also using this final model, the odds of tooth site BOP progression were significantly decreased according to each unit increase of the tooth site's PPD at baseline (mm) and this association was significantly modified by smoking status ($p = 0.0440$ for the interaction term of ''past'' smoker by PPD at baseline) (Table 4). The OR according to every unit increase in PPD at baseline was 0.90 (95% CI = 0.82–0.99) among ''never'' smokers, 0.79 (95% CI = 0.73–0.86) among ''past'' smokers and 0.76 (95% CI = 0.58–0.98) among ''current'' smokers (Table 5). Also, upper-jaw tooth sites had significantly increased odds for BOP progression compared with lower-jaw tooth sites (OR = 0.86, 95% CI = 0.79–0.99). However, age was not significantly associated with the odds of site-level

BOP progression, nor was gender, BMI status or being a molar tooth site (Table 4).

The values of R^{*2} for the final models of CAL, PPD and BOP progression are, respectively, 0.22, 0.23 and 0.19. Also for the final models of CAL, PPD and BOP progression, the Hosmer–Lemeshow GOF χ^2 statistics are, respectively, 9.33 ($p = 0.4073$), 16.59 ($p = 0.0556$) and 15.76 ($p = 0.0721$). Thus, the GOF tests provide no statistical evidence for lack of fit for any of the final three models.

Discussion

To the best of our knowledge, this is the first study to report on periodontal disease progression in Gullah African Americans with T2DM and no recent periodontal therapy. Our results suggest that there are significant associations between periodontal disease progression and diabetes control status when no recent periodontal therapy was applied and that this association was significantly modified (Ylostalo & Knuuttila 2006) by a history of periodontal disease (PPD at baseline). There were significant increases in odds for site-level CAL and PPD progression in subjects with poorly-controlled diabetes compared with well-controlled and the magnitude of this association increased as the PPD at baseline increased (for $HbA_{1c} \geqslant 7\%$ by PPD of 3, 5 and 7 mm, respectively, PPD progression OR = 1.98, 2.76 and 3.84, CAL progression OR = 1.93, 2.64 and 3.62).

A meta-analysis of four studies with a total of 3524 adults (>18 years old) showed that those with diabetes have a twofold higher risk of developing periodontal disease compared with those without diabetes (Papapanou 1996). In investigating the relationship between diabetes and oral health, a study including 1342 Pima Indians (a population with the world's highest reported incidence and prevalence of T2DM) found that diabetes increases the risk of developing destructive periodontal disease about threefold. The increased risk cannot be explained by age, sex, hygiene or other dental measures (Emrich et al. 1991). In a case–control study comparing those with *versus* without T2DM, diabetics showed an increased susceptibility for more severe periodontal disease (Campus et al. 2005). Further, Soskolne & Klinger (2001) analysed NHANES III data and concluded that in addition to the convincing epidemiological evidence that periodontitis is more prevalent among those with diabetes, the prevalence of diabetes in those with periodontitis is significantly greater (twofold) than in those without periodontitis. The epidemiological associations between periodontitis and diabetes could be the result of at least two similar but distinct pathogenic pathways: a direct causal relationship in which the consequences of diabetes act as modifiers of periodontal disease expression or, alternatively, a common pathological defect that results in a host susceptible to either, or both, diseases (Soskolne & Klinger 2001).

Our results suggest that in addition to the preponderance of periodontitis among those with T2DM, those who continue to have poor glycaemic control and no recent periodontal treatment will succumb to increased site-level disease severity (as compared with those who had well-controlled diabetes and were similarly untreated); further, this progression in disease severity was more pronounced for tooth sites with a history of periodontal disease (as measured by the tooth site PPD at baseline). We also found that tooth sites in obese subjects had significantly greater odds of PPD and CAL progression than tooth sites in subjects with a normal BMI after adjusting for other final model covariates. Also, tooth sites in overweight subjects showed similarly greater odds of CAL progression. These results are consistent with the reports from Wood et al. (2003) and Khader et al. (2009) reporting that CAL and PPD, as indicators of periodontal disease, were correlated with increased BMI. The underlying mechanisms for the association between obesity and periodontal disease are not well known. However, it has been suggested that obesity contributes to an overall systemic inflammatory state through its effect on metabolic and immune parameters, thereby increasing susceptibility to periodontal disease (Genco et al. 2005). Although our results indicate a significant association between BMI and periodontitis progression, the study design limits interpretations of temporal relationships with regard to the periodontal measures recorded at baseline. It is not possible to determine which occurred first, obesity, diabetes or periodontitis severity at baseline. A prospective cohort study, including a non-diabetic/obese group and a non-diabetic/normal BMI group, may address this problem. Our observed associations could also in part be due to common lifestyle characteristics that make individuals more prone to all three diseases. Still, our results showed that diabetes control level had a significant association with longitudinal progression for both PPD and CAL independent of BMI status, and likewise, obese subjects had significantly increased odds of these events independent of their level of diabetes control.

Individuals who smoke have six to seven times more alveolar bone loss than non-smokers in studies in the United States and other countries (Bergstrom & Preber 1994, Grossi et al. 1995, Tomar & Asma 2000). Our findings showed that tooth sites from past smokers had significantly increased odds for CAL progression, and yet no significance was found among current smokers for CAL progression perhaps due to the small number of individuals in this group or from the limited longitudinal aspect of the study. There were significant decreases in the odds of site-level BOP progression with every unit increase (mm) in PPD at baseline, and this association was significantly modified by smoking status, with the magnitude of the association showing successive decreases as the smoking status shifted from ''never'' (OR = 0.90) to ''past'' (OR = 0.79) to ''current'' (OR = 0.76). Although the smoking process may result in alveolar bone loss, it can also produce vasoconstriction within the gingival tissue, which may partially explain the sequential decreasing effect of smoking status on the association between PPD at baseline and BOP progression (Bergstrom & Bostrom 2001).

There were also significant increases in the odds of site-level CAL progression by 3% with every yearly increase in subject age. Studies have also shown that periodontal disease prevalence and severity increases with age, and most systemic disease conditions such as diabetes, heart disease and obesity also increase in prevalence with age. It is apparent that ageing is associated with changes that lead to a progressive, irreversible deterioration of the functional capacities of tissues and organs (Mackenzie et al. 1977).

The results of this report may be limited in their generalizability as they may only apply to this specific population living in the Sea Islands of South

Carolina. Our study population is also limited to subjects with T2DM, and those with well-controlled diabetes served as controls for the poorly-controlled. These subjects were also predominantly of lower socioeconomic status with limited dental care access. Poor oral health care has traditionally been linked to lower socioeconomic status, which may have influenced our results. A future analysis for this same study population is planned to assess disparities in periodontal disease progression at the mouth level according to glycaemic control while controlling for both clinical and socioeconomic factors.

The strengths of this report include an opportunity to study periodontal disease progression among subjects with T2DM and no recent treatment as these subjects were part of a previous cross-sectional study and are now part of an ongoing clinical trial. Additionally, this study population is a notable one to evaluate for our objectives, given the increased risks for T2DM among the Gullah and the profoundly high prevalence of periodontitis among the Gullah with T2DM. Analyses involving this distinct, homogenous population, given their substantially low non-African genetic admixture and significant preservation of their African cultural heritage, allowed a natural adjustment for such typical confounders, lending further support to the clinical relevance of Gullah-related study findings. The data used herein are also very comprehensive, from the subject level to the tooth site level, and this allowed for evaluations of multiple independent effects and adjustments for various potential confounders.

Our results showed significantly increased odds of site-level CAL and PPD progression among those with poorly-controlled T2DM and among those who were obese. PPD at baseline was a significant effect modifier of the associations between poor glycaemic control and site-level CAL and PPD progression, with the magnitude of the association increasing as baseline periodontitis was more severe. Past smokers and those who were overweight also had significantly increased odds of site-level CAL progression. This suggests that the treatment and prevention of periodontitis may be more critical in these specific groups of this study population. Such individuals may, therefore, be appropriate targets for interventions aimed at reducing the considerable periodontal disease disparities exhibited among Gullah with T2DM.

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Clinical Relevance

Scientific rationale for the study: The host inflammatory response appears to be the critical determinant for susceptibility to and severity of periodontitis in systemically compromised individuals. We used data from an ongoing clinical trial of Gullah African Americans with T2DM to evaluate the associations between glycaemic control and progressive periodontitis per tooth site.
Principal findings: Results showed significantly increased odds of tooth site-level CAL and probing depth progression among Gullah with poor glycaemic control *versus* well-controlled T2DM.
Practical implications: These findings from a distinct, homogenous population further support the clinical relevance of identifying patients with poor glycaemic control and periodontitis, particularly among those with substantial disparities for both diseases.