Published in final edited form as:

Community Dent Oral Epidemiol. 2017 April ; 45(2): 168–177. doi:10.1111/cdoe.12275.

Environmental Tobacco Smoke Exposure and Periodontitis Prevalence among Non-smokers in the Hispanic Community Health Study/Study of Latinos (HCHS/SOL)

Aderonke A. Akinkugbe¹, Anne E. Sanders², John S. Preisser³, Jianwen Cai³, Christian R. Salazar⁴, and James D. Beck²

¹Department of Epidemiology, Gillings School of Global Public Health, University of North Carolina at Chapel Hill, Chapel Hill NC, 27599-7435

²Department of Dental Ecology, School of Dentistry, University of North Carolina at Chapel Hill, Chapel Hill NC, 27599-7455

³Department of Biostatistics, Gillings School of Global Public Health, University of North Carolina at Chapel Hill, Chapel Hill NC, 27599-7435

⁴Department of Epidemiology and Population Health, Albert Einstein College of Medicine, Bronx, NY, 10461

Abstract

Objective—To describe self-reported exposure to environmental tobacco smoke (ETS) and its association with periodontitis prevalence in a diverse group of Hispanics/Latinos.

Methods—Data came from 8,675 lifetime non-smokers in the 2008–2011 Hispanic Community Health Study/Study of Latinos. Exposure to ETS was self-reported while periodontitis was defined using the CDC/AAP criteria and the proportion of sites affected by clinical attachment level of 3mm or pocket depth of 4mm. Survey logistic regression estimated prevalence odds ratios (POR) and 95% confidence intervals (CI). In addition, we assessed whether greater hours of exposure to ETS in the past year was associated with greater periodontitis prevalence and lastly, we conducted a simple sensitivity analysis of ETS misclassification.

Results—Age-standardized prevalence estimates (95% CI) for ETS exposure and periodontitis were 57.6% (55.9, 59.4) and 39.8% (38.1, 41.4) respectively. After adjusting for confounders and periodontitis risk factors, we estimated an overall adjusted POR (95% CI) for the ETS-periodontitis association as 1.09 (0.95–1.26) with a confidence limit ratio (CLR) of 1.34. This association varied in magnitude by Hispanic/Latino background, ranging from 1.04 (0.75, 1.43 with a CLR=1.91) among Central-Americans to 1.76 (1.16, 2.66 with a CLR=2.29) in Puerto Ricans.

Conclusions—Previously reported associations between ETS and periodontitis appear weak in this study. However, the magnitude of the association differs according to Hispanic/Latino background.

Keywords

Environmental Tobacco Smoke; Periodontitis; Hispanic/Latino; Exposure misclassification

INTRODUCTION

Periodontitis is a major cause of tooth-loss, with adverse negative impacts on oral and overall health-related quality-of-life¹⁻³. A modifiable risk factor for periodontitis is cigarette smoking⁴⁻⁷. Smoking increases both the risk of onset and progression of periodontitis, and a U.S. estimate of population attributable risk suggests that up to 42% of periodontitis is due to smoking⁶. Given that approximately 80% of U.S. adults do not smoke⁸, exposure to environmental tobacco smoke (ETS) poses a potentially greater threat to the periodontal health of the majority of the population than active smoking does.

ETS is a mixture of mainstream (smoke exhaled by the smoker) and side-stream (smoke given off by a burning cigarette) smoke⁹, and both have similar chemical constituents. Previous studies have reported positive associations between ETS exposure and periodontitis among non-smokers, but these findings were limited either by small sample sizes^{10,11} or homogeneous study populations^{12,13} hence limiting generalization of these findings. In the few studies to examine this association, the Hispanic subgroup have mostly been of Mexican-American background^{14,15}. However, Hispanics/Latinos represent some 20 different countries with diverse demographic, economic and cultural heritages. For example, illustrating heterogeneity in health status among Latin American countries, differences in life expectancy at birth in 2010/2011 spanned 17 years, ranging from 60.8 years in Haiti to 77.6 years in Costa Rica¹⁶. Thus, it is unlikely that Mexican-Americans adequately represent this rapidly growing and diverse population.

Despite a survival advantage¹⁷ —the so-called “Hispanic paradox”—Hispanics/Latinos fare worse than non-Hispanic whites with respect to abdominal obesity and other cardio-metabolic risk factors that are associated with periodontitis¹⁸⁻²⁴. They also differ in disease risk factor profile²⁵, which may reflect differences in their countries of origin. For instance, variation in smoking intensity among Hispanic/Latino groups has been reported. Specifically, smoking prevalence is higher among Puerto Ricans and Cubans in the U.S. than in any Hispanic/Latino country of origin and it is also higher than the U.S. national average. Mexican and Central Americans in the U.S. have smoking prevalence similar to the U.S. national average, while Dominicans and South Americans have prevalence estimates that are lower than the U.S. national average²⁶. For the countries represented in this study, 2013 data from the Tobacco Atlas (<http://www.tobaccoatlas.org>) show that 20% of Cuban men are current smokers, while 14.5% of Dominican men are.

The objectives of this study were to describe self-reported ETS exposure among a diverse group of Hispanic/Latino non-smokers in the target population of the Hispanic Community Health Study/Study of Latinos (HCHS/SoL) and to investigate its association with prevalent periodontitis. We hypothesized a positive association between ETS exposure and periodontitis that differs in magnitude by Hispanic/Latino background. We restricted our study population to lifetime non-smokers because any effect of ETS on the periodontium of

smokers would be mixed with those of active mainstream smoke, and current and/or former smoking behaviors could confound the hypothesized association.

METHODS

Details of the study design, sampling, and data collection have been previously described^{25,27,28}. Briefly, the HCHS/SoL is a multicenter community based cohort study of 16,415 self-identified Hispanics/Latinos designed to investigate risk and protective factors for chronic health conditions. Eligible 18–74-year-olds of Cuban, Dominican, Mexican, Puerto Rican, Central and South-American descent were recruited between March 2008 and June 2011 from randomly selected households in 4 U.S. communities (Bronx, New York; Chicago, Illinois; Miami, Florida; and San Diego, California) using a stratified two-stage area probability sampling design. Oversampling of 45–74-year-olds was done in eligible households, and sampling weights were calculated to reflect this disproportionate sampling. At baseline, study participants completed interviewer-administered questionnaires and underwent rigorous clinical, laboratory and oral examinations. Institutional Review Boards of all relevant institutions approved the study and all participants gave informed consent.

Study participants not requiring antibiotic prophylaxis received a full-mouth periodontal examination following a standardized protocol. Measures of probing pocket depth (PD) and gingival recession were recorded on 6 sites [mesio-buccal, mid-buccal, disto-buccal, mesio-lingual, disto-lingual, and lingual] on all teeth except third molars. Clinical attachment level (CAL) was calculated as sum of PD and gingival recession. Examiners were recalibrated annually to a gold standard examiner, with very good to excellent agreement²⁹.

This investigation defined periodontitis prevalence using the Centers for Disease Control and Prevention-American Academy for Periodontology (CDC-AAP) definition^{30–32} and the proportion of sites (extent) affected by PD ≥ 4mm or CAL ≥ 3mm. The CDC-AAP defines severe periodontitis as ≥ 2 interproximal sites with CAL of ≥ 6 mm (not on the same tooth) AND ≥ 1 interproximal sites with PD of ≥ 5 mm^{30–32}. Moderate periodontitis is defined as ≥ 2 interproximal sites (not on the same tooth) with CAL of ≥ 4 mm OR ≥ 2 interproximal sites (not on the same tooth) with PD of ≥ 5 mm^{30,31}. Individuals with moderate or severe periodontitis were categorized as having periodontitis, non-cases otherwise. Individuals with <2 recorded interproximal sites were excluded because they did not meet the CDC-AAP periodontitis criteria. We also defined a case as someone having ≥ 30% of sites with PD ≥ 4mm or CAL ≥ 3mm (Yes/No)¹².

We classified as non-smokers participants who responded “no” to the question: “Have you ever smoked ≥ 100 cigarettes in your entire life?” Non-smokers of cigarettes who have ever smoked pipes or cigars were not excluded because our sensitivity analysis excluding these individuals (n=213) did not meaningfully change the results. We defined exposure to ETS in two ways: first, as self-report of ever living with a regular cigarette smoker (Yes/No); and second, as the self-reported average number of hours/week in the past year in close contact with a smoker. This variable was modeled as continuous and categorized into none, 1–25 hours/week, >25 hours/week to assess whether greater hours of self-reported exposure to ETS were associated with greater periodontitis prevalence.

Age and gender were self-reported. Nativity status classified participants as U.S.-or foreign-born. Educational attainment was categorized as: <high school, high school, or >high school. Body mass index categories were underweight or normal ($<25 \text{ kg/m}^2$), overweight ($25\text{--}30 \text{ kg/m}^2$) and obese ($>30 \text{ kg/m}^2$). Diabetes was based on the American Diabetes Association definition³³. Those with normal and impaired fasting glucose/impaired glucose tolerance (fasting glucose between 100–125mg/dl or post oral glucose tolerance test (OGTT) between 140–199 mg/dl or HbA1C between 5.7 and $<6.5\%$) were categorized as not having diabetes. Time since last dental visit was categorized into <1 , 1–3 and >3 years or never visited.

Statistical analysis

Of 16,415 HCHS/SoL participants, 9,923 (60.5%) were lifetime non-smokers. Of these, 8,747 (88.1%) had non-missing values on ETS exposure, and had retained at least two teeth with periodontal examination measurements. Omitted from analysis were participants missing information on nativity status ($n=6$), Hispanic/Latino background ($n=16$), education ($n=18$), BMI ($n=17$), diabetes ($n=2$) and last dental visit ($n=13$). Therefore, complete participant analysis was conducted on 8,675 (99.2%) participants.

Weighted proportions and standard errors for the study population characteristics were estimated for all groups combined and by Hispanic/Latino background. Likewise, prevalence estimates for ETS and periodontitis, age-standardized to the 2010 U.S. Census age distribution³⁴ were calculated using weighted least squares survey regression. Design-adjusted Wald chi-square tests assessed the association of categorical variables with ETS and periodontitis respectively. Effect measure modification (EMM) of the ETS-periodontitis association was assessed using design-adjusted Wald chi-square tests comparing models with and without product interaction terms between ETS and Hispanic/Latino background, ETS and age, ETS and gender. The threshold for statistical significant interaction was set at $p < 0.10$. Separate survey logistic regression for periodontitis based on the CDC-AAP definition and the proportion of sites affected by PD $\geq 4\text{mm}$ or CAL of $\geq 3\text{mm}$, estimated prevalence odds ratios (POR) and 95% CI. The first model was stratified by Hispanic/Latino background but did not include any covariate. Subsequent stratified models sequentially adjusted for age and gender and then nativity status, study center, BMI, time since last dental visit, and diabetes. The precision of the POR estimates were evaluated with the confidence limit ratio (upper limit divided by the lower limit with values closer to 1 indicating greater relative precision)³⁵. Age was flexibly modeled with a quadratic term. Similarly, survey logistic regression for the periodontitis estimated POR and 95% CI for categories of ETS, based on the self-reported average hours/week exposed in the past year. Finally, ETS was modeled as continuous and the corresponding slope and p-value were reported as the trend estimate and p-value for trend respectively. Adjusting for the number of teeth present did not meaningfully affect the results (Supplementary Table 3).

Statistical tests were 2-sided and significance was set at $p < 0.05$. Statistical tests and data analysis were performed in SAS v. 9.4 (SAS Institute Cary, NC) accounting for the complex sampling design and applying weights that account for the unequal sampling probabilities.

Assessment of potential bias due to exposure misclassification

Previous studies have demonstrated a strong correlation between self-reported smoking status and serum cotinine levels^{36,37} but the sensitivity and specificity of self-report is not 100%. Due to the reporting bias inherent in an exposure such as ETS, it is possible for a higher proportion of non-smokers unexposed to ETS to correctly self-identify than for exposed non-smokers. Additionally, because our primary definition of ETS exposure was ever vs. never exposed, the potential for misclassification of true exposure status is likely. Thus, we conducted a simple sensitivity analysis of potential exposure misclassification on the ETS-periodontitis association. We specified a range of values for the sensitivity and specificity of ETS for periodontitis cases and non-cases and estimated a correctly classified number of exposed and unexposed individuals and the corresponding unadjusted POR. For a given sensitivity and specificity for periodontitis cases and non-cases, we calculated the number of correctly classified individuals as: $A1 = \frac{a1 - FpM1}{Se + Sp - 1}$ and $B1 = \frac{b1 - FpM0}{Se + Sp - 1}$ ³⁸; where: A1 is exposed cases; B1 is exposed non-cases; M1 is total cases; M0-total non-cases; Fp-false positives; a1- exposed cases (observed); b1-exposed non-cases (observed); Se is sensitivity; Sp is specificity.

RESULTS

The overall mean age (SE) was 38.6 years (0.3), ranging from 33.0 years (1.3) in the mixed/other background to 43.0 years (0.8) among the Cuban group. There were more women than men and, except among Puerto Ricans, most of the groups were foreign-born (Table 1).

The overall age-standardized prevalence estimates of ETS exposure and periodontitis (based on the CDC-AAP definition of moderate or severe periodontitis) were 57.5% (55.9, 59.4) and 39.8% (38.1, 41.4) respectively, with those of Central American background having the highest age-standardized prevalence of both ETS exposure (72.8%) and periodontitis (47.4%) (Table 2). While there was no meaningful difference in the proportion of men (56.9%) and women (57.7%) exposed to ETS, exposure was highest among the 18–44 year olds (25.2%) and lowest among those aged ≥ 65 years (10.2%). Similarly, exposure to ETS was greater among foreign (58.5%) than US-born (54.5%) participants, and among those with diabetes compared to those without diabetes. The age-standardized prevalence of periodontitis showed an age gradient but was lower among ≥ 65-year-olds, and affected significantly more men (46.5%) than women (35.9%). Foreign-born participants were more likely than US-born participants to have periodontitis (42.7% vs. 31.7%), as were those with diabetes (Table 2).

Because the design adjusted Wald test p-value for the ETS*Hispanic/Latino background interaction was significant (p=0.03), regression analysis results were stratified by Hispanic/Latino background. We found no significant statistical interaction for ETS*age (p=0.7) or ETS*gender (p=0.8).

In unadjusted analysis, self-reported exposure to ETS appeared positively associated with periodontitis in all Hispanic/Latino backgrounds except South Americans (Table 3). POR (95% CI) estimates range from 1.07 (0.87, 1.66, CLR=1.91) among Central Americans to 1.90 (1.27, 2.88, CLR= 2.24) in Dominicans, who were the only group for whom the

association reached statistical significance. Upon age and gender adjustment, the magnitude of the respective PORs was lower in all groups except for the Puerto Rican group. Upon additional adjustment for confounders, the overall adjusted POR (95% CI) was 1.09 (0.95, 1.26, CLR=1.34) and, the subgroup with the largest magnitude of adjusted POR was the Puerto Rican group with a POR (95% CI) of 1.76 (1.16, 2.66, CLR=2.29). While the association in the Cuban, Puerto Rican, Dominican, and Central American groups remained positive, only the association in the Puerto Rican group reached statistical significance.

Following covariate adjustment, the overall association between the self-reported average number of hours of exposure to ETS in the past year and periodontitis was positive and statistically significant (Table 4). As expected, self-reported ETS exposure was positively associated with having 30% of sites with PD 4mm or CAL 3mm in all subgroups except the Central and South American and mixed/other backgrounds (Supplementary Table 1).

The simple sensitivity analysis of exposure misclassification indicated that, with lower sensitivity and specificity of ETS exposure in the scenarios of non-differential exposure misclassification, the corresponding unadjusted PORs were biased further from the null relative to the POR we reported. For differential misclassification, the direction of the bias was hard to predict but the changes seen were within the margin of error of the estimates we reported (Supplementary Table 2).

DISCUSSION

In this investigation, exposure to ETS was positively but only marginally associated with periodontitis. While there appear to be modest associations between ETS and periodontitis in the respective Hispanic/Latino sub-groups, only the positive association in the Puerto Rican background reached statistical significance.

Those of Cuban background had the highest unstandardized prevalence (results not shown) of both self-reported ETS exposure and periodontitis while Central-American background had the corresponding highest age-standardized estimates. As previously reported for this cohort, those of Cuban, Puerto Rican and Central American backgrounds are most likely while Dominicans were least likely to be smokers²⁶ therefore, it is not surprising that exposure to ETS even among non-smokers was higher among Cuban and Central American backgrounds than in the other subgroups.

This study expands the literature on the association between ETS exposure and periodontitis in U.S. Hispanic/Latino adults by reporting findings in groups other than those of Mexican American descent. The study by Arbes et al.¹⁴ used data from the NHANES-III and reported adjusted POR (95% CI) estimate of 1.57 (1.15, 2.16), based on self-reported ETS exposure at home and the workplace. The 1999–2004 NHANES analysis of Sanders et al.¹⁵ used serum cotinine as the measure of ETS and reported an adjusted POR (95% CI) of 1.60 (1.05, 2.44). The adjusted estimates we reported were of smaller magnitudes but were precisely estimated based on the CLRs. This mitigates the role of chance in our findings. Possible reasons for the discrepancy in estimates between this study and prior studies include: exposure assessment (self-report vs. biomarker), different adjustment covariates and study

population. A difference in intensity of ETS exposure could also be responsible, since it has been previously reported that Hispanics/Latinos are more likely than non-Hispanic whites and African-Americans to reside in areas with smoke-free laws³⁹. Moreover, a study found that migrant populations in the U.S. have lower smoking prevalence in the U.S. than in their country of origin⁴⁰. If these were the case, then both of these may lead to fewer opportunities for ETS exposure and likely explain the weak association we found.

Because of the cross-sectional design, the possibility that periodontitis preceded ETS exposure cannot be dismissed, but it is unlikely that periodontitis caused ETS exposure. Findings of the sensitivity analysis to assess potential bias due to exposure (ETS) misclassification were robust to both differential and non-differential exposure misclassifications, based on the range of values we specified for the sensitivity and specificity of ETS exposure among cases and non-cases of periodontitis.

This is the first report of this relationship in a diverse sample of Hispanics/Latinos. A limitation of this study is that prevalence (as opposed to incidence) measures were estimated, and incidence measures would have been more informative. Even with prevalence measures, the plausible direction of association is ETS preceding periodontitis and not vice versa, mitigating concerns about reverse causality. Second, there is a possibility for bias from misreporting of smoking and/or ETS exposure. Several studies have shown that self-reported smoking status and exposure to ETS among non-smokers correlate well with serum cotinine levels among all races/ethnicities except Blacks^{36,37}. Additionally, findings from our simple sensitivity analysis were robust to bias from potential exposure misclassification. Third, because our primary exposure was ever vs. never exposed, it is possible that the effect of ETS exposure on periodontitis may have been diluted by time since exposure to ETS, thus accounting for the weak effects we reported.

CONCLUSION

Exposure to ETS was associated with marginally higher unadjusted odds of periodontitis, which was rendered non-significant upon adjustment for confounders. This relationship was strongest among Puerto Ricans. Even with the varying strengths, ETS exposure contributes somewhat to the burden of periodontitis in some of the Hispanic/Latino subgroups; thus, tobacco control efforts may improve the periodontal health of these non-smokers.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

Acknowledgments

The authors thank the staff and participants of HCHS/SOL for their important contributions. The Hispanic Community Health Study/Study of Latinos was carried out as a collaborative study supported by contracts from the National Heart, Lung, and Blood Institute (NHLBI) to the University of North Carolina (N01-HC65233), University of Miami (N01-HC65234), Albert Einstein College of Medicine (N01-HC65235), Northwestern University (N01-HC65236), and San Diego State University (N01-HC65237). The following Institutes/Centers/Offices contribute to the HCHS/SOL through a transfer of funds to the NHLBI: National Institute on Minority Health and Health Disparities, National Institute on Deafness and Other Communication Disorders, National Institute of Dental and Craniofacial Research, National Institute of Diabetes and Digestive and Kidney Diseases, National Institute of Neurological Disorders and Stroke, NIH Institution-Office of Dietary Supplements.

Funding

Aderonke A. Akinkugbe was supported by the National Institute of Health (NIH) NRSA T90 Training Grant/ National Institute of Dental and Craniofacial Research (NIDCR) 5T90DE021986-04

References Cited

1. O'Dowd LK, Durham J, McCracken GI, Preshaw PM. Patients' experiences of the impact of periodontal disease. *J Clin Periodontol Denmark*. 2010;334–9.
2. Durham J, Fraser HM, McCracken GI, Stone KM, John MT, Preshaw PM. Impact of periodontitis on oral health-related quality of life. *J Dent*. 2013; 41(4):370–6. [PubMed: 23357646]
3. Brennan DS, Spencer AJ, Roberts-Thomson KF. Quality of life and disability weights associated with periodontal disease. *J Dent Res United States*. 2007;713–7.
4. Do LG, Slade GD, Roberts-Thomson KF, Sanders AE. Smoking-attributable periodontal disease in the Australian adult population. *J Clin Periodontol*. 2008; 35(5):398–404.
5. Thomson WM, Broadbent JM, Welch D, Beck JD, Poulton R. Cigarette smoking and periodontal disease among 32-year-olds: a prospective study of a representative birth cohort. *J Clin Periodontol*. 2007; 34(10):828–34. [PubMed: 17850601]
6. Tomar SL, Asma S. Smoking-attributable periodontitis in the United States: findings from NHANES III. National Health and Nutrition Examination Survey. *J Periodontol*. 2000; 71(5):743–751. [PubMed: 10872955]
7. Susin C, Oppermann RV, Haugejorden O, Albandar JM. Periodontal attachment loss attributable to cigarette smoking in an urban Brazilian population. *J Clin Periodontol*. 2004; 31(11):951–58. [PubMed: 15491309]
8. Jamal, A., Agaku, IT., O'Connor, E., King, BA., Kenemer, JB., Neff, L. MMWR Morb Mortal Wkly Rep. Vol. 63. United States: 2014. Current cigarette smoking among adults--United States, 2005–2013; p. 1108–12.
9. Jinot J, Bayard S. Respiratory health effects of passive smoking: EPA's weight-of-evidence analysis. *J Clin Epidemiol*. 1994; 47(4):339–349. discussion 351–3. [PubMed: 7730859]
10. Nishida N, Yamamoto Y, Tanaka M, et al. Association between passive smoking and salivary markers related to periodontitis. *J Clin Periodontol*. 2006; 33(10):717–23. [PubMed: 16889628]
11. Yamamoto Y, Nishida N, Tanaka M, et al. Association between passive and active smoking evaluated by salivary cotinine and periodontitis. *J Clin Periodontol*. 2005; 32(10):1041–46. [PubMed: 16174266]
12. Sanders AE, Slade GD, Beck JD, Agustsdottir H. Secondhand smoke and periodontal disease: atherosclerosis risk in communities study. *Am J Public Health*. 2011; 101(Suppl 1):S339–46. [PubMed: 21551377]
13. Tanaka K, Miyake Y, Hanioka T, Arakawa M. Active and passive smoking and prevalence of periodontal disease in young Japanese women. *J Perio Res*. 2013; 48(5):600–5.
14. Arbes SJ Jr, Agustsdottir H, Slade GD. Environmental tobacco smoke and periodontal disease in the United States. *Am J Public Health*. 2001; 91(2):253–7. [PubMed: 11211634]
15. Sanders A, Slade G. State cigarette excise tax, secondhand smoke exposure, and periodontitis in US nonsmokers. *Am J Public Health*. 2013; 103(4):740–6. [PubMed: 22994169]
16. Oppermann RV, Haas AN, Rosing CK, Susin C. Epidemiology of periodontal diseases in adults from Latin America. *Periodontol 2000*. 2015; 67(1):13–33. [PubMed: 25494596]
17. Ruiz JM, Steffen P, Smith TB. Hispanic mortality paradox: a systematic review and meta-analysis of the longitudinal literature. *Am J Public Health*. 2013; 103(3):e52–60.
18. Genco RJ, Grossi SG, Ho A, Nishimura F, Murayama Y. A proposed model linking inflammation to obesity, diabetes, and periodontal infections. *J Periodontol*. 2005; 76(11 Suppl):2075–84. [PubMed: 16277579]
19. Saito T, Shimazaki Y. Metabolic disorders related to obesity and periodontal disease. *Periodontol 2000*. 2007; 43:254–66. [PubMed: 17214843]

20. Palle AR, Reddy CM, Shankar BS, Gelli V, Sudhakar J, Reddy KK. Association between obesity and chronic periodontitis: a cross-sectional study. *J Contemp Dent Pract*. 2013; 14(2):168–73. [PubMed: 23811640]
21. Nishida N, Tanaka M, Hayashi N, et al. Determination of smoking and obesity as periodontitis risks using the classification and regression tree method. *J Periodontol*. 2005; 76(6):923–8. [PubMed: 15948686]
22. Saito T, Shimazaki Y, Sakamoto M. Obesity and periodontitis. *N Engl J Med*. 1998; 339(7):482–3. [PubMed: 9705695]
23. Saito T, Shimazaki Y, Koga T, Tsuzuki M, Ohshima A. Relationship between upper body obesity and periodontitis. *J Dent Res*. 2001; 80(7):1631–6. [PubMed: 11597023]
24. Wood N, Johnson RB, Streckfus CF. Comparison of body composition and periodontal disease using nutritional assessment techniques: Third National Health and Nutrition Examination Survey (NHANES III). *J Clin Periodontol*. 2003; 30(4):321–7. [PubMed: 12694430]
25. Daviglus, ML., Talavera, GA., Aviles-Santa, ML., et al. *JAMA*. Vol. 308. United States: 2012. Prevalence of major cardiovascular risk factors and cardiovascular diseases among Hispanic/Latino individuals of diverse backgrounds in the United States; p. 1775-84.
26. Kaplan RC, Bangdiwala SI, Barnhart JM, et al. Smoking among U.S. Hispanic/Latino adults: the Hispanic community health study/study of Latinos. *Am J Prev Med*. 2014; 46(5):496–506. [PubMed: 24745640]
27. Lavange, LM., Kalsbeek, WD., Sorlie, PD., et al. *Ann Epidemiol*. Vol. 2010. United States: Elsevier Inc; 2010. Sample design and cohort selection in the Hispanic Community Health Study/Study of Latinos; p. 642-9.
28. Sorlie, PD., Aviles-Santa, LM., Wassertheil-Smoller, S., et al. *Ann Epidemiol*. United States: Published by Elsevier Inc; 2010. Design and implementation of the Hispanic Community Health Study/Study of Latinos; p. 629-41.
29. Sanders AE, Campbell SM, Mauriello SM, et al. Heterogeneity in periodontitis prevalence in the Hispanic Community Health Study/Study of Latinos. *Ann Epidemiol*. 2014; 24(6):455–62. [PubMed: 24731697]
30. Eke, PI., Thornton-Evans, GO., Wei, L., Borgnakke, WS., Dye, BA. *J Dent Res*. Vol. 89. United States: 2010. Accuracy of NHANES periodontal examination protocols; p. 1208-13.
31. Eke PI, Page RC, Wei L, Thornton-Evans G, Genco RJ. Update of the case definitions for population-based surveillance of periodontitis. *J Periodontol*. 2012; 83(12):1449–54. [PubMed: 22420873]
32. Page RC, Eke PI. Case definitions for use in population-based surveillance of periodontitis. *J Periodontol*. 2007; 78(7 Suppl):1387–99. [PubMed: 17608611]
33. ADA. Diagnosis and classification of diabetes mellitus. *Diabetes care*. 2012; 35(Suppl 1):S64–71. [PubMed: 22187472]
34. Ennis, SR., Ríos-Vargas, M., Albert, NG. U.S. Department of Commerce EaSA. Census Briefs. Washington: U.S. Census Bureau; 2011. The Hispanic Population: 2010.
35. Poole C. Low P-values or narrow confidence intervals: which are more durable? *Epidemiology* (Cambridge, Mass). 2001; 12(3):291–4.
36. Caraballo RS, Giovino GA, Pechacek TF, Mowery PD. Factors associated with discrepancies between self-reports on cigarette smoking and measured serum cotinine levels among persons aged 17 years or older: Third National Health and Nutrition Examination Survey, 1988–1994. *Am J Epidemiol*. 2001; 153(8):807–14. [PubMed: 11296155]
37. Wagenknecht LE, Burke GL, Perkins LL, Haley NJ, Friedman GD. Misclassification of smoking status in the CARDIA study: a comparison of self-report with serum cotinine levels. *Am J Public Health*. 1992; 82(1):33–6. [PubMed: 1536331]
38. Rothman, KJ., Greenland, S., Lash, TL. *Modern Epidemiology*. 3. Lippincott, Williams & Wilkins; Philadelphia, PA: 2008.
39. Gonzalez M, Sanders-Jackson A, Song AV, Cheng KW, Glantz SA. Strong smoke-free law coverage in the United States by race/ethnicity: 2000–2009. *Am J Public Health*. 2013; 103(5):e62–6. [PubMed: 23488507]

40. Bosdriesz JR, Lichthart N, Witvliet MI, Busschers WB, Stronks K, Kunst AE. Smoking prevalence among migrants in the US compared to the US-born and the population in countries of origin. PLoS One. 2013; 8(3):e58654. [PubMed: 23520525]

Author Manuscript

Author Manuscript

Author Manuscript

Author Manuscript

Table 1

Selected study characteristics [unadjusted, weighted column percent, (standard error)] among lifetime non-smokers in the Hispanic Community Health Study/Study of Latinos, 2008–2011

	Mexican (n=3,780)	Cuban (n=1,009)	Puerto Rican (n=1,044)	Dominican (n=926)	Central American (n=1,061)	South American (n=611)	Mixed/ Other (n=244)	All (n=8,675)
Gender								
Male	36.7 (1.25)	45.0 (1.65)	49.1 (2.46)	39.3 (2.05)	40.0 (1.94)	39.2 (2.61)	44.5 (4.58)	40.6 (0.74)
Female	63.3 (1.25)	55.1 (1.65)	50.9 (2.46)	60.7 (2.05)	60.0 (1.94)	60.8 (2.61)	55.5 (4.58)	59.4 (0.74)
Age mean (SE)	37.1 (0.43)	42.5 (0.83)	41.0 (0.82)	37.3 (0.73)	38.2 (0.55)	40.1 (0.96)	33.0 (1.32)	38.6 (0.28)
Age group (years)								
18–44	71.6 (1.24)	59.8 (2.13)	58.6 (2.47)	67.8 (2.31)	69.4 (1.92)	62.7 (2.94)	78.3 (4.43)	67.1 (0.83)
45–64	24.0 (1.09)	26.6 (1.32)	30.9 (2.26)	28.4 (1.97)	25.4 (1.68)	31.0 (2.48)	17.3 (4.38)	26.1 (0.69)
65	4.34 (0.54)	13.6 (1.62)	10.5 (1.79)	3.81 (0.88)	5.21 (1.01)	6.33 (1.51)	4.37 (1.58)	6.80 (0.43)
Nativity								
US-born	25.0 (1.25)	8.12 (1.17)	96.9 (0.82)	14.4 (1.99)	8.14 (1.41)	6.59 (1.26)	56.5 (4.96)	28.1 ((1.01)
Foreign-born	75.0 (1.25)	91.9 (1.17)	3.13 (0.82)	85.6 (1.99)	91.9 (1.41)	93.4 (1.26)	43.5 (4.96)	71.9 (1.01)
Educational attainment								
< High school	33.6 (1.42)	18.2 (1.30)	27.3 (2.13)	32.7 (2.08)	37.2 (1.91)	20.2 (2.11)	16.8 (2.81)	29.0 (0.79)
High school	31.1 (1.22)	29.8 (2.02)	29.0 (1.83)	23.4 (2.08)	25.6 (1.62)	27.5 (2.25)	21.8 (4.55)	28.7 (0.75)
> High school	35.3 (1.68)	52.1 (2.16)	43.7 (2.59)	43.9 (2.09)	37.2 (2.14)	52.3 (2.72)	61.4 (4.78)	42.3 (0.96)
Body mass index (Kg/m ²)								
Normal (<25)	24.5 (1.10)	26.0 (1.94)	24.6 (2.09)	21.9 (1.70)	24.7 (1.90)	31.3 (2.60)	25.1 (4.03)	24.9 (0.70)
Overweight (25–30)	38.8 (1.41)	36.6 (1.58)	30.1 (2.08)	40.0 (2.43)	39.3 (2.23)	40.6 (2.63)	31.8 (3.77)	37.4 (0.77)
Obese (>30)	36.7 (1.43)	37.4 (2.05)	45.3 (2.37)	38.1 (2.09)	36.0 (1.97)	28.2 (2.30)	43.1 (4.46)	37.7 (0.81)
Diabetes [*]								
Yes	12.8 (0.73)	11.8 (1.34)	14.9 (1.34)	11.7 (1.21)	12.2 (1.20)	7.32 (1.26)	12.9 (4.43)	12.4 (0.49)
No	87.2 (0.73)	88.2 (1.34)	85.1 (1.34)	88.3 (1.21)	87.8 (1.20)	92.7 (1.26)	87.1 (4.43)	87.6 (0.49)
Time since last dental visit (years)								
< 1	48.2 (1.40)	53.5 (1.71)	57.8 (2.53)	66.8 (2.31)	45.7 (2.21)	54.3 (2.97)	49.6 (4.47)	52.7 (0.88)
1–3	28.5 (1.13)	22.3 (1.52)	23.7 (2.00)	22.4 (2.04)	25.3 (2.03)	25.1 (2.25)	24.3 (3.33)	25.5 (0.70)
> 3	23.2 (1.13)	24.1 (1.50)	18.5 (1.94)	10.8 (1.31)	29.0 (2.07)	20.6 (2.37)	26.1 (4.09)	21.8 (0.69)
N teeth present mean (SE)	26.1 (0.08)	23.1 (0.23)	24.4 (0.23)	24.4 (0.23)	24.5 (0.19)	23.8 (0.29)	25.6 (0.83)	24.9 (0.09)

* Defined based on the American Diabetes Association definition³³, those with normal and impaired glucose tolerance were categorized as not having diabetes

Author Manuscript

Author Manuscript

Author Manuscript

Author Manuscript

Age-standardized prevalence of ETS exposure and periodontitis by selected study characteristics [row percent, (95% CI)]

Table 2

Characteristic	ETS exposed*			Periodontitis†		
	Row%	95% CI	p value†	Row%	95% CI	p value†
All groups	57.6	55.9–59.4		39.8	38.1–41.4	
Hispanic/Latino background			<0.0001			<0.0001
Mexican	52.3	49.5–55.0		42.4	39.5–45.3	
Cuban	57.8	52.7–62.8		28.6	23.8–33.5	
Puerto Rican	67.8	59.7–75.8		34.0	22.7–45.3	
Dominican	49.8	45.3–54.3		44.2	39.6–48.8	
Central American	72.8	69.5–76.0		47.4	43.9–50.9	
South American	59.5	54.4–64.6		32.0	27.9–36.2	
Mixed/Other	49.6	44.4–54.8		36.1	30.8–41.4	
Gender			0.9			0.0001
Male	56.9	54.1–57.7		46.5	44.0–49.0	
Female	57.7	55.6–59.9		35.9	33.9–38.0	
Age group (years)			<0.0001			<0.0001
18–44	25.2	24.1–26.3		11.4	10.6–12.3	
45–64	22.3	21.4–23.1		18.0	17.2–18.9	
65	10.2	9.09–11.2		10.3	9.21–11.4	
Nativity			0.01			<0.0001
US-born	54.5	50.4–58.6		31.7	28.3–35.1	
Foreign-born	58.5	56.7–60.4		42.7	40.9–44.6	
Educational attainment			0.6			<0.0001
< High school	57.4	54.9–59.9		45.6	42.8–48.4	
High school	59.8	56.4–63.2		40.3	36.7–44.0	
> High school	57.3	54.2–60.3		35.1	32.4–37.8	
Body mass index (Kg/m ²)			0.2			<0.0001
Normal (<25)	55.7	52.3–59.1		35.3	31.9–38.8	
Overweight (25–30)	57.6	55.0–60.2		40.1	37.6–42.6	
Obese (>30)	58.2	55.3–61.1		42.4	39.7–45.1	

Characteristic	ETS exposed [*]			Periodontitis [‡]		
	Row%	95% CI	p value [‡]	Row%	95% CI	p value [‡]
Diabetes [§]			0.02			<0.0001
Yes	58.6	53.9–63.2		46.7	42.4–51.0	
No	57.9	55.5–59.8		38.6	36.5–40.7	
Last dental visit (years)			0.4			<0.0001
< 1 year	57.9	54.3–61.4		39.1	36.0–42.2	
1–3 years	56.6	54.2–59.1		36.9	34.7–39.0	
> 3 years	60.2	56.7–63.7		48.0	44.3–51.7	
Study site			<0.0001			<0.0001
Bronx	55.1	50.9–59.4		26.8	23.5–30.2	
Chicago	54.8	52.0–57.6		49.7	46.5–52.9	
Miami	66.7	64.0–69.3		46.5	44.0–49.0	
San Diego	51.8	48.6–55.0		39.6	36.2–43.1	

^{*} Defined as self-report of ever living with a regular smoker OR currently live in a house with at least one active smoker

[‡] Survey design-adjusted Wald chi-square test

[‡] Defined as moderate-severe periodontitis based on the Centers for Disease Control and Prevention-American Academy for Periodontology case classification for periodontitis^{30–32}

[§] Defined based on the American diabetes association definition³³, those with normal and impaired glucose tolerance were categorized as non-diabetics

All estimates were age-standardized to the 2010 U.S. Census age distribution³⁴

Table 3

Prevalence Odds Ratios (95% CI) for the association between ETS and Periodontitis among Hispanic/Latino backgrounds in the Hispanic Community Health Study/Study of Latinos, 2008–2011

	Model 1*			Model 2 [†]			Model 3 [‡]		
	POR	95% CI	CLR	POR	95% CI	CLR	POR	95% CI	CLR
Mexican	1.20	0.99–1.46	1.47	1.03	0.84–1.26	1.50	0.97	0.78–1.21	1.55
Cuban	1.35	0.99–1.82	1.84	1.20	0.85–1.68	1.98	1.20	0.84–1.70	2.02
Puerto Rican	1.40	0.92–2.12	2.30	1.70	1.08–2.69	2.49	1.76	1.16–2.66	2.29
Dominican	1.90	1.27–2.84	2.24	1.18	0.77–1.83	2.38	1.12	0.72–1.74	2.42
Central American	1.07	0.87–1.66	1.91	1.08	0.78–1.49	1.91	1.04	0.75–1.43	1.91
South American	0.96	0.66–1.40	2.12	0.92	0.62–1.36	2.19	0.80	0.54–1.19	2.20
Mixed/Other	1.17	0.52–2.67	5.13	0.84	0.31–2.29	7.39	1.00	0.40–2.55	6.38
All groups	1.33	1.17–1.51	1.29	1.13	0.98–1.29	1.32	1.09	0.95–1.26	1.34

*Model 1 - Unadjusted for any covariate

[†]Model 2 - model 1 additionally adjusted for age, quadratic term for age and gender

[‡]Model 3 – model 2 additionally adjusted for nativity status, center, time since last dental visit, body mass index, diabetes, and educational attainment

Periodontitis was defined as moderate-severe periodontitis based on the Centers for Disease Control and Prevention-American Academy for Periodontology case classification for periodontitis^{30–32}
 POR-Prevalence odds ratio; CLR-confidence limit ratio (Upper CI/Lower CI) – gives an indication of the precision of the POR estimates³⁵

Table 4

Multivariable association between self-reported numbers of hours of exposure to ETS and periodontitis prevalence among non-smokers in the Hispanic/Latino groups, Hispanic Community Health Study/Study of Latinos (2008–2011)

ETS exposure groups*	All groups	Mexican	Cuban	Puerto Rican	Dominican	Central American	South American	Mixed/Other
Model 1[†]								
Unexposed								
1–25 hours/week	0.69 (0.60–0.80)	0.71 (0.56, 0.90)	0.70 (0.49, 1.01)	0.71 (0.46, 1.09)	1.05 (0.71, 1.57)	0.79 (0.54, 1.13)	0.57 (0.35, 0.92)	0.45 (0.18, 1.10)
>25 hours/week	1.11 (0.81, 1.51)	1.00 (0.54, 1.86)	1.25 (0.70, 2.22)	1.10 (0.50, 2.43)	1.63 (0.57, 4.66)	0.48 (0.18, 1.30)	2.21 (0.71, 6.86)	0.60 (0.10, 3.56)
Trend estimate	1.03 (0.98, 1.07)	1.02 (0.92, 1.13)	1.03 (0.96, 1.11)	1.02 (0.93, 1.12)	1.14 (0.94, 1.38)	0.89 (0.74, 1.08)	1.16 (0.96, 1.40)	0.82 (0.55, 1.23)
p for trend	0.3	0.7	0.4	0.7	0.2	0.2	0.1	0.3
Model 2[‡]								
Unexposed								
1–25 hours/week	0.95 (0.81–1.12)	0.90 (0.70, 1.17)	0.92 (0.60, 1.43)	1.23 (0.74, 2.02)	1.25 (0.78, 1.99)	1.09 (0.74, 1.62)	0.72 (0.43, 1.20)	0.56 (0.21, 1.57)
>25 hours/week	1.26 (0.92, 1.72)	1.37 (0.72, 2.59)	1.25 (0.77, 2.02)	1.42 (0.66, 3.06)	1.91 (0.51, 7.12)	0.48 (0.15, 1.61)	2.91 (0.78, 10.9)	0.86 (0.11, 6.56)
Trend estimate	1.05 (1.01, 1.10)	1.10 (0.99, 1.22)	1.05 (0.98, 1.12)	1.07 (0.98, 1.17)	1.14 (0.91, 1.42)	0.92 (0.77, 1.09)	1.22 (0.98, 1.52)	0.91 (0.63, 1.31)
p for trend	0.02	0.1	0.2	0.2	0.3	0.3	0.1	0.6
Model 3[§]								
Unexposed								
1–25 hours/week	0.97 (0.82, 1.15)	0.86 (0.66, 1.12)	0.95 (0.62, 1.46)	1.28 (0.82, 2.00)	1.18 (0.73, 1.88)	1.19 (0.80, 1.78)	0.67 (0.40, 1.14)	0.72 (0.30, 1.70)
>25 hours/week	1.28 (0.93, 1.77)	1.24 (0.67, 2.30)	1.23 (0.76, 2.00)	1.27 (0.59, 2.75)	1.74 (0.51, 5.93)	0.54 (0.17, 1.71)	3.21 (0.78, 13.3)	0.51 (0.05, 5.46)
Trend estimate	1.06 (1.01, 1.10)	1.09 (0.98, 1.21)	1.04 (0.97, 1.11)	1.06 (0.98, 1.15)	1.10 (0.89, 1.37)	0.92 (0.78, 1.09)	1.25 (1.00, 1.56)	0.80 (0.57, 1.14)
p for trend	0.01	0.1	0.3	0.1	0.4	0.3	0.05	0.2

* Defined as self-reported average number of hours/week exposed to ETS in the past year

[†]Model 1–unadjusted

[‡]Model 2– Model 1 with additional adjustment for age, quadratic term for age, and gender

[§]Model 3– Model 2 with additional adjustment for nativity status, center, time since last dental visit, body mass index, diabetes, and educational attainment.

Estimated trend is per additional 10 hours of exposure to ETS

All estimates are POR and 95% CI