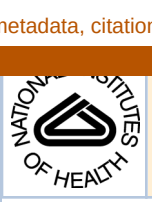

Published in final edited form as:
Am J Public Health. 2011 December ; 101(0 1): S339–S346. doi:10.2105/AJPH.2010.300069.

Secondhand Smoke and Periodontal Disease: Atherosclerosis Risk in Communities Study

Anne E. Sanders, PhD,
Department of Dental Ecology, School of Dentistry, University of North Carolina at Chapel Hill

Gary D. Slade, PhD,
Department of Dental Ecology, School of Dentistry, University of North Carolina at Chapel Hill

James D. Beck, PhD, and
Department of Dental Ecology, School of Dentistry, University of North Carolina at Chapel Hill

Helga Ágústsdóttir, PhD
Ministry of Health and Social Security, Reykjavik, Iceland

Abstract

Objectives—We investigated the relationship between secondhand smoke and periodontal disease in nonsmokers.

Methods—We undertook a cross-sectional analysis of the Atherosclerosis Risk in Communities study with 2739 lifetime nonsmokers aged 53–74 years, unexposed to other sources of tobacco, who received a complete periodontal examination at visit 4. Exposure was reported as average hours per week in close contact with a smoker in the preceding year. We defined severe periodontitis as 5 or more periodontal sites with probing pocket depth of 5 millimeters or more and clinical attachment levels of 3 millimeters or more in those sites. Other outcomes were extent of periodontal probing depths of 4 millimeters or more and extent of clinical attachment levels of 3 millimeters or more.

Results—In a binary logistic regression model, adjusted odds of severe periodontitis for those exposed to secondhand smoke 1 to 25 hours per week increased 29% (95% confidence interval=1.0, 1.7); for those exposed to secondhand smoke 26 hours per week, the odds were twice as high (95% confidence interval=1.2, 3.4) as for those who were unexposed.

Conclusions—Exposure to secondhand smoke and severe periodontitis among nonsmokers had a dose-dependent relationship.

Periodontitis is a chronic condition characterized by inflammation of the supporting tissues of the teeth, resulting in breakdown of the connective tissue attaching the teeth to the alveolar bone and eventually to irreversible loss of that bone. Diagnosis is based on signs of destruction of the connective tissues attaching the tooth root to alveolar bone, which is

Correspondence should be sent to: Anne E. Sanders, PhD, Department of Dental Ecology, UNC School of Dentistry, Campus Box 7450, Chapel Hill, NC 27599 (anne_sanders@dentistry.unc.edu).

Contributors

A. E. Sanders wrote the article, which was based on the findings of H. Ágústsdóttir’s doctoral research. H. Ágústsdóttir conducted the research, including the statistical analysis. J. D. Beck was principal investigator of the dental Atherosclerosis Risk in Communities (ARIC) study that conducted oral examinations of ARIC study participants at visit 4, and he also reviewed the article. G. D. Slade was mentor to H. Ágústsdóttir and guided the statistical analysis. He also analyzed data in response to reviewers’ comments.

Human Participant Protection

No protocol approval was necessary because this study involved the analysis of secondary data only.

assessed clinically by measuring attachment level. Attachment level is determined by combined parameters of probing depth and gingival recession at numerous sites in the mouth. Ideally, measurements are made at 6 sites per tooth in a full-mouth assessment of 28 teeth. Periodontitis is a leading cause of tooth loss and an important entity in its own right. Also, the underlying infection and complex host immune-modulatory and inflammatory responses that destroy periodontal tissues contribute to several systemic conditions.[1] Systematic reviews of the evidence have supported a relationship between periodontitis and cardiovascular disease [2,3] and type 2 diabetes.[4]

In 2004, the US Surgeon General concluded that the scientific evidence was sufficient to infer a causal relationship between tobacco smoking and periodontitis.[5] The etiologic fraction, that is, the fraction of severe periodontal disease cases in which cigarette smoking exposure plays an etiologic role,[6] has been estimated to be 52.8%.[7] This percentage indicates that approximately one half of periodontitis cases could be prevented if cigarette smoking were eliminated, with most of that reduction occurring among people who quit smoking, rather than among nonsmokers exposed to secondhand smoke. In the US dentate adult population, prevalence of periodontitis, defined as 2 or more sites with clinical attachment loss of at least 4 millimeters and 1 or more sites with probing depth of 4 millimeters or deeper, was estimated to be 3.6%.[8] The extent of attachment loss and prevalence of the disease increases dramatically with age.

Nonsmokers exposed to secondhand smoke are recognized to be at increased risk of periodontitis. On reviewing updated evidence on involuntary exposure to tobacco smoke, the US Surgeon General concluded in 2006 that there is no risk-free level of exposure to secondhand smoke.[9] To date, only 2 studies have examined the association of secondhand smoke exposure and periodontal disease in adults. One analyzed data from the 3rd National Health and Nutrition Examination Survey (NHANES III) and found that the odds of periodontitis for exposed adults who had never actively smoked were 1.6 times (95% confidence interval [CI]=1.3, 2.0) as much as those of unexposed adults.[10] The second study, of 273 predominantly male Japanese workers, reported higher odds of periodontal disease in passive smokers relative to nonsmokers (odds ratio [OR]=2.9; 95% CI=1.1, 7.8).[11]

Although these findings are informative, some caution is required in their interpretation. Estimates of effect size vary considerably depending on case definition, extent and severity of periodontal disease, and measurement protocol.[12] Both studies used a partial-mouth measurement protocol limited to sites in 2 quadrants. Compared with full-mouth examinations, half-mouth protocols have severely underestimated the prevalence of periodontitis and produced biased findings, especially in populations with low prevalence of severe disease.[13–15] Whether such bias alters the presence and strength of a relationship between secondhand smoke and periodontal disease is not known.

To address this limitation, we used a rigorous protocol, examining 6 sites per tooth in a full-mouth survey, and applied a stringent case definition of periodontitis to ensure that we correctly identified all cases that met the case definition. The specific study aim was to determine whether exposure to secondhand smoke was associated with periodontal disease in lifetime nonsmokers of cigarettes who were unexposed to other sources of tobacco or nicotine. We hypothesized that nonsmokers exposed to secondhand smoke would have greater odds of severe periodontitis and greater extent of periodontal disease than those who were unexposed.

METHODS

Informed consent was obtained from all eligible study participants before the dental examination.

Study Participants

Study participants were enrolled in the Atherosclerosis Risk in Communities (ARIC) study, a multicenter prospective epidemiologic cohort study conducted in 4 communities in the United States. At baseline (1987–1989), 15792 adults aged 45 to 64 years were selected by probability sampling from Forsyth County, North Carolina; Jackson, Mississippi; suburban Minneapolis, Minnesota; and Washington County, Maryland. The cross-sectional data for this analysis were collected during the comprehensive dental examination conducted from 1996 through 1998 (visit 4 of the longitudinal ARIC study). Of the baseline sample, 74% participated at visit 4 (n=11656). Of these, 4860 did not take part in the periodontal assessment because they had no remaining teeth (n=1651), had medical contraindications (n=1621), refused (n=1317), or had another reason (n=271). Of the 6796 who had a periodontal assessment, 4057 were omitted from this analysis because they were a current or former smoker (n=3640), had used another form of nicotine (n=381), self-identified as non-White and non-Black (n=25), or had another reason (n=11). Hence, the total sample consisted of 2739 ARIC study participants.

Main Exposure

Interviewers administered a questionnaire at visit 4 to collect self-report information about health status, medication usage, and health behavior. We obtained detailed information about active and past cigarette smoking, as well as lifetime use of pipes, cigars, cigarillos, chewing tobacco, snuff, nicotine gum, and nicotine patches. We omitted people exposed to any of these sources of tobacco from the analysis to eliminate possible bias from other sources of tobacco.

Using the ARIC study classification, we categorized participants who indicated having smoked fewer than 400 cigarettes during their lifetime as a lifetime nonsmoker. To determine secondhand smoke exposure participants were asked, “During the past year, about how many hours per week, on the average, were you in close contact with people when they were smoking? For example, in your home, in a car, at work or other close quarters?” Although this measure falls well short of obtaining a cumulative lifetime exposure, the duration of exposure is substantially longer than measures of acute exposure obtained from serum cotinine.

Examiner Training and Standardization and Collection of Periodontal Clinical Data

Dental examiners assessed each tooth for dental plaque using the Silness & Löe Plaque Index.[16] Probing depth (PD) was determined with a UNC-15 periodontal probe at 6 sites per tooth and recorded in millimeters, with fractions of millimeters rounded to the next lower unit. As many as 28 teeth were examined for each person; third molars were excluded. At the same sites, gingival recession was measured as the distance from the cemento-enamel junction to the free gingival margin and recorded in millimeters, with fractions of millimeters rounded to the next lower unit. We computed clinical attachment level (CAL) during data analysis by adding the PD to the gingival recession. Examiners assessed the presence or absence of bleeding on probing after each quadrant of probing at 6 sites on all teeth.

All dental examiners received the same training and calibration. During calibration, each examiner was matched with the gold-standard examiner and another examiner on at least 5

occasions. The weighted κ was 0.90 for PD and 0.82 for CAL within 1 millimeter. Over the 2-year course of examinations, we conducted quality assurance through conference calls, site visits, and recalibration to maintain standardization of examiners.

Dependent Variable and Covariates

We selected thresholds for 3 periodontal disease outcome measures that have been used previously [17–19]: (1) case definition of severe periodontitis as 5 or more sites with CAL 3 millimeters or more and PD 5 mm or more in the same sites and (2) extent of periodontal disease (CAL3, the proportion of sites probed with CAL≥3 mm), and (3) PD4, (the proportion of sites probed with PD≥4 mm).

Covariates were known or hypothesized risk indicators for periodontal disease. We examined age (in years); education in 3 categories (≤11 years, 12–16 years, ≥17 years) as a marker of socioeconomic position; oral hygiene, defined by tooth-brushing frequency in the previous day (never, once, twice, or 3 times) and frequency of flossing in the week before the examination (never, once, twice, or 3 times); and proportion of sites with plaque scores of 2 or more. We also considered diabetes (fasting glucose level≥126 mg/dL, nonfasting glucose level≥200 mg/dL, taking medications for hyperglycemia, or having a physician's diagnosis of diabetes); body mass index (weight in kilograms divided by height in meters squared); use of nonsteroidal anti-inflammatory drugs (other than aspirin); alcohol use (grams of ethanol/wk) or drinking status (current, former, never); coffee consumption (cups per day), and race (White or Black). Because women were postmenopausal, we derived a categorical variable with 3 levels: female current hormone replacement therapy (HRT) users, female non-HRT users, and men.

When we analyzed data as an overall summary of responses from all probed sites (i.e., severe periodontitis case status), multivariate models included the number of remaining teeth to include the dimension of teeth that were at risk. We did not include the number of remaining teeth for the CAL3 or PD4 models because the number of probing sites available was included as the denominator for the proportion.

As a result of the method of sampling, race was incompletely distributed within the study center locations. Therefore, we created a combined variable of center–race with 5 levels: (1) Forsyth County, North Carolina—Blacks, (2) Forsyth County, North Carolina—Whites, (3) Jackson, Mississippi—Blacks, (4) Minneapolis, Minnesota—Whites and (5) Washington County, Maryland—Whites. The combined center–race variable therefore adjusted for both race and center differences. The center–race variable also reflected any effects resulting from different examiners across centers.

Statistical Analysis

We tested our hypothesis of an association between periodontal disease and secondhand smoke exposure among lifetime nonsmokers with no exposure to other tobacco products. We coded all variables that had more than 2 levels as indicator variables and binary variables as 0 or 1. We created categorical variables for the continuous variables for an adjusted bivariate analysis. We calculated the means and proportions of covariates, adjusting for age, gender–HRT use, and education and center–race, stratified on both severe periodontitis case status and exposure to secondhand smoke (coded as 1 for ≥1 h/wk and 0 for unexposed).

We tested the covariates related to both severe periodontitis case status and secondhand smoke exposure ($P<.1$), adjusting for age, education, and center–race, in multivariable models. Age, gender–HRT use, education, and center–race were forced into all models irrespective of their statistical significance. We built the models by adding 1 covariate at a

time and evaluated each variable using type III tests (i.e., as though each variable were the last added).

If the parameter estimates for each level of secondhand smoke exposure were changed by more than 10% or if the coefficient for that covariate was statistically significant at $P<.05$, we kept the covariate in the model and entered the next covariate.

We used a binary logistic regression model to estimate the risk of secondhand smoke exposure on severe periodontitis (case vs. noncase), controlling for potential confounders. The decision to set the threshold for high secondhand smoke exposure at more than 25 hours per week was based on the distribution, because we know of no preexisting threshold. In this study, 6.4% of all participants (exposed and unexposed to secondhand smoke) and 12.7% of all participants exposed to secondhand smoke were exposed to high levels of secondhand smoke (> 25 h/wk).

For analyses of extent variables, where the response was Z_n, the number of diseased sites (CAL≥3 mm or PD≥4) among n probed sites, we treated Z_n as a binomially distributed random variable. The binomial distribution assumes independence of the k dichotomous responses (0 or 1) at each site. It is reasonable to expect that individuals who have 1 diseased site (CAL≥3 mm or PD≥4) are more likely to have other diseased sites. This intraindividual correlation contributes to extrabinomial variation in the data. To allow for this potential overdispersion, we assumed that explanatory factors influenced the proportion of diseased sites, $p_i = Z_n/n$, through a logistic link function and estimated model parameters using generalized estimating equations methods. The appeal of this approach is that the empirical or robust standard errors for the parameter estimates are consistent, even if the representation of the variance in the estimating equations is misspecified. When we tested the main exposure variable as a continuous variable, we tested transformation—such as quadratic terms, exponential terms, logarithmic and exponential transformation of the main exposure variable—in the binomial multivariate models to capture the shape of the association better than with a straight-line model. We conducted all analyses using SAS, version 9.1 (SAS Institute, Cary, NC).

RESULTS

Study participants were aged between 53 and 74 years (mean=62.4). The sample was predominantly female (74.7%), and Blacks made up 20.2%. Exposure to secondhand smoke for 1 hour or more a week was reported by 33.7% (n=923) of adults.

Of all participants, 4.3% reported weekly exposure exceeding 25 hours (n=117). The mean exposure, adjusted for age, gender–HRT, education, and center–race for the participants who reported secondhand smoke exposure, was 9.97 hours a week (SE=0.6), and the range of exposure was 1 to 108 hours per week.

Severe periodontitis was found in 16.0% (n=438) of participants. On average, 5.2% (SE=0.2; range=0–97) of participants had periodontal sites with probing depths deeper than or equal to 4 mm. The mean extent of CAL 3 millimeters or more was 16.5% (SE=0.3; range=0–100). The mean number of retained teeth was 22.6 (SE=0.13; range=2–32).

Covariate Associations With Case Status and Secondhand Smoke

The covariates significantly associated with severe periodontitis after adjustment for the key factors of age, gender–HRT, education, and center–race ($P<.5$) were body mass index and dental visiting pattern (Table 1). When adjusting for key factors, alcohol and coffee consumption were significantly associated with secondhand smoke exposure ($P<.5$), along

with body mass index (Table 1). Periodontal assessment parameters of bleeding on probing, CAL, probing pocket depth, and dental plaque were significantly associated with case status (Table 2), and, with the exception of extent of probing pocket depths of 4 millimeters or more, all periodontal parameters were significantly associated with secondhand smoke exposure.

Multivariable Analysis

Severe Periodontitis—Mean secondhand smoke exposure (average hours per week over the past year) was significantly higher for cases (mean=4.3; 95% CI=3.0, 5.6) than for non-cases (mean=3.2; 95% CI=2.8, 3.6). Among those exposed for 1 to 25 hours per week, mean secondhand smoke exposure was 4.5 hours (95% CI=4.1, 4.8). Among those exposed for more than 25 hours per week, mean secondhand smoke exposure was 48.0 hours (95% CI=44.3, 51.6). We observed a significant dose-dependent relationship of secondhand smoke exposure and severe periodontitis prevalence (Table 3). The odds for people exposed to 1 to 25 hours per week was 29% higher (95% CI=1.0, 1.7) and the odds for people exposed to 26 hours per week or more was twice as high (95% CI=1.2, 3.4) than that of people with less than 1 hour per week of secondhand smoke exposure, adjusting for age, education, center–race, gender–HRT use, pattern of dental visits, dental plaque, and number of teeth remaining.

Extent of Attachment Loss and Probing Depth—The final models for extent of CAL3 and PD4 contained the same covariates: age, gender–HRT use, education, center–race, extent of dental plaque scores, and pattern of dental visits (Table 4).

The adjusted odds of periodontal sites having clinical attachment levels 3 millimeters or more increased by a factor of 1.1 (95% CI=1.0, 1.2) for people with 1 to 25 hours per week of secondhand smoke exposure, which was not significantly greater than that for unexposed people. However for people with 26 hours per week or more of secondhand smoke exposure, the risk increased significantly by a factor of 1.3 (95% CI=1.0, −1.6), compared with people with less than 1 hour per week of secondhand smoke exposure.

For periodontal pocket depths of 4 millimeters or more (Table 4), we observed a trend of increasing extent of deep pockets with greater exposure, but the confidence intervals of the odds ratios enclosed unity and hence did not differ significantly from those unexposed to secondhand smoke.

DISCUSSION

In this large community-dwelling sample of lifetime nonsmokers unexposed to other tobacco products, those exposed to secondhand smoke had a higher prevalence of severe periodontitis, after controlling for known risk indicators for periodontal disease. Exposure to secondhand smoke also showed a dose-dependent increase in extent of periodontal disease, but the association was statistically significant only for CAL and only at the higher level of exposure. Our findings were consistent with the 2 earlier studies [10,11] that used the partial-mouth examination. They also build on evidence of lower CALs in children exposed to secondhand smoke in the home.[20] Previous studies [13–15,21] have shown that bias in prevalence estimates is reduced by using a full-mouth assessment of 6 sites per tooth, as we did. If the degree of bias in disease measurement is equal in exposed and unexposed people (i.e., the assumption of nondifferential misclassification), ORs for the association between exposure and disease are usually biased toward the null, with the amount of bias proportional to the degree of bias in disease measurement. Methods that reduce bias in estimating prevalence might therefore be expected to yield larger exposure–disease ORs. Yet, we found that the effect sizes at 2 thresholds of secondhand smoke exposure (adjusted

OR [AOR]=1.3 and 2.0, respectively) tended to be smaller than those previously reported in nonsmokers (OR=1.6 in Arbes et al.[10]; OR=2.9 in Yamamoto et al.[11]). One explanation is that bias in periodontal disease is differential with respect to smoking, for example, if partial-mouth recordings produce greater underestimation of disease in smokers than in nonsmokers. However, differences in effect estimates may be because of other conspicuous differences among these studies, including their age distributions and measures of exposure and outcome. Nevertheless, the direction of effect was consistent across all 3 studies, and the magnitude of effect was approximately equal, establishing consistency of evidence.

Possible Mechanisms and Explanations

A possible explanation for why the association of exposure to secondhand smoke and severe periodontitis was statistically significant whereas the association with extent scores was weaker may be that our periodontitis case definition was more stringent and recognized only truly serious periodontal disease. The wide range of covariates used in multivariable regression models adds to the argument that this association is not a spurious one caused by residual confounding.

There is reason to believe that passive smoking exerts similar systemic effects on the periodontal tissues as observed in active smokers, based on studies that have found that active and passive smoking have effects in the same direction, although not the same magnitude, on other health outcomes.[22–24] Because secondhand smoke is inhaled, 1 likely biological mechanism of effect is through systemic effects of toxic constituents in the tobacco smoke.[25] These effects may be mediated through injury inflicted by proinflammatory agents, such as cytokines [26] or smoking-induced oxidative stress.[27]

Some authors have attributed the difference in periodontal health among active cigarette smoking groups to better oral hygiene among nonsmokers. Plaque accumulation does not, however, seem to differ enough among smoking groups to explain the strong association of smoking with periodontal disease.[28,29] The effect of active cigarette smoking on the pathogenesis of periodontal disease is now believed to be exerted through both local and systemic pathways.[30] The local effects are thought to be mediated by the chemical stimuli and include local vasoconstriction by nicotine and decreased oxygen tension as well as hyperkeratosis of the gingival tissues. Local effects possibly include effects of the physical heat from cigarette smoke, although no studies have been conducted to confirm that type of effect. The more important pathway of the 2 is believed to be the systemic alteration of the host response.[29,31] Several studies have noted impaired chemotaxis and phagocytosis [32] of both oral and peripheral neutrophils of smokers as well as reduced antibody production.[33,34] Smoking is associated with suppressed salivary osteocalcin levels, implying a pathogenic mechanism via reduced bone mineral density.[35] Very few studies have assessed the host response in periodontal tissues of passive smokers. One study comparing non-smokers and passive smokers found elevated levels of salivary biomarkers for periodontal disease in passive smokers compared with non-smokers.[36]

Strengths and Limitations

This study was limited to individuals whose only exposure to tobacco products was secondhand smoke. Elimination of other sources of smoke and nicotine makes these findings even more compelling and establishes that this excess risk is the result of secondhand smoke exposure alone and not of active smoking by the individuals themselves. The magnitude of this public health problem is considerable, considering that one third of nonsmoking individuals were exposed. The large number of participants permitted comparisons and adjustments for other risk factors than a smaller sample would have allowed. The extensive

amount of clinical and lifestyle information collected made it possible to examine this association more thoroughly than otherwise possible.

One reason for the small crude effect is that adults who were less likely to be cases were more likely to be exposed to secondhand smoke. For example, at the Forsythe study site, 6% of White adults were cases compared with 16% to 23% of adults at other study sites. Yet secondhand smoke exposure tended to be higher in Whites at the Forsythe site than at the other study sites, biasing estimates toward the null. As a result, cases and controls did not differ in secondhand smoke exposure when secondhand smoke was dichotomized (P=.08). We found a stronger effect when secondhand smoke was defined over 3 levels. Here, the odds for disease in those exposed for 26 hours per week were elevated 45% relative to those unexposed, and the effect became significant after adjustment for covariates including study site (AOR=2.0). The cross-sectional design did not permit us to establish the temporal relationship of secondhand smoke and periodontal disease. Therefore, periodontal disease may possibly have existed before the exposure. Only longitudinal data could confirm that exposure preceded the onset of disease. However, reverse causation is implausible: There is no reason to believe that people would become exposed to secondhand smoke as a consequence of developing periodontitis. Exposure was self-reported, which is also a limitation, but self-report is the most common way to collect information on lifestyle factors. No biomarkers, such as cotinine levels, were available for this study to confirm the reported exposure, but in an analysis of a study population representative of the US population [10] (NHANES III), serum cotinine levels generally confirmed the self-reported exposures. Using a threshold of 10 nanograms per milliliter of serum cotinine as an indicator of current cigarette smoking, only 3.4% of non-smokers reportedly exposed to secondhand smoke and 0.8% of people not exposed to secondhand smoke were likely current smokers or users of other tobacco products.

A related concern is this study's measure of secondhand smoke during the preceding 12 months, creating the potential for misclassifying exposure over the longer period in which harmful compounds in tobacco smoke probably contribute to destruction of periodontal tissues (possibly a decade or more). For 2 reasons, we believe the degree of misclassification is probably less in this study than in other studies. First, studies such as NHANES ask only about current secondhand smoke exposure. For example, during the in-home interview for NHANES III, interviewers asked, "Does anyone who lives here smoke cigarettes in the home?" [10] In contrast, we asked about exposures during preceding 12 months. Second, people in this study were aged 53–74 years, ages at which most of those exposed to secondhand smoke experience that exposure in the home, not the workplace. Moreover, domestic living arrangements tend to be quite stable in this age. Therefore, most people's exposure during the preceding 12 months is probably a good proxy for their exposure during several of the preceding years.

In summary, we found statistically significantly higher prevalence of severe periodontitis but not greater extent of periodontal disease in lifetime nonsmokers exposed to secondhand smoke, compared with those not exposed to secondhand smoke. The results build on earlier reports of a relationship between exposure to secondhand smoke and periodontal disease. Longitudinal studies are needed to evaluate the exposure–outcome sequence, as are experimental studies to provide evidence of biological mechanisms. Our findings suggest that secondhand smoke exposure should be taken into account in future studies of periodontal disease.

Secondhand smoke remains a serious public health hazard, with 40% of the nonsmoking population aged 3 years or older being exposed in the United States during 2007–2008.[37] Although understanding the pathogenesis of secondhand smoke in periodontal disease has

scientific value, the health of the nation is best served through initiatives with demonstrated effectiveness in limiting secondhand smoke exposure.

Acknowledgments

The ARIC study is carried out as a collaborative study supported by National Heart, Lung and Blood Institute (contracts N01-HC-55015, N01-HC-55016, N01-HC-55018, N01-HC-55019, N01-HC-55020, N01-HC-55021, and N01-HC-55022). The collection and analysis of dental data were supported by National Institute of Dental and Craniofacial Research (grants DE13807-01A1 and DE11551).

We thank the staff and participants of the ARIC study for their important contributions.

References

1. Beck JD, Offenbacher S. Relationships among clinical measures of periodontal disease and their associations with systemic markers. Ann Periodontol. 2002; 7(1):79–89. [PubMed: 16013220]
2. Humphrey LL, Fu R, Buckley DI, Freeman M, Helfand M. Periodontal disease and coronary heart disease incidence: a systematic review and meta-analysis. J Gen Intern Med. 2008; 23(12):2079–2086. [PubMed: 18807098]
3. Blaizot A, Vergnes JN, Nuwwareh S, Amar J, Sixou M. Periodontal diseases and cardiovascular events: meta-analysis of observational studies. Int Dent J. 2009; 59(4):197–209. [PubMed: 19774803]
4. Chavarry NG, Vettore MV, Sansone C, Sheiham A. The relationship between diabetes mellitus and destructive periodontal disease: a meta-analysis. Oral Health Prev Dent. 2009; 7(2):107–127. [PubMed: 19583037]
5. Health Consequences of Smoking: A Report of the Surgeon General. Atlanta, GA: Centers for Disease Control and Prevention; 2004.
6. Greenland S, Robins JM. Conceptual problems in the definition and interpretation of attributable fractions. Am J Epidemiol. 1988; 128(6):1185–1197. [PubMed: 3057878]
7. 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]
8. Borrell LN, Crawford ND. Social disparities in periodontitis among United States adults 1999–2004. Community Dent Oral Epidemiol. 2008; 36(5):383–391. [PubMed: 18924254]
9. Health Consequences of Involuntary Exposure to Tobacco Smoke: A Report of the Surgeon General. Atlanta, GA: Centers for Disease Control and Prevention; 2006.
10. Arbes SJ Jr, Agustsdottir H, Slade GD. Environmental tobacco smoke and periodontal disease in the United States. Am J Public Health. 2001; 91(2):253–257. [PubMed: 11211634]
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):1041–1046. [PubMed: 16174266]
12. Page RC, Eke PI. Case definitions for use in population-based surveillance of periodontitis. J Periodontol. 2007; 78(7, suppl):1387–1399. [PubMed: 17608611]
13. Hunt RJ, Fann SJ. Effect of examining half the teeth in a partial periodontal recording of older adults. J Dent Res. 1991; 70(10):1380–1385. [PubMed: 1939834]
14. Susin C, Kingman A, Albandar JM. Effect of partial recording protocols on estimates of prevalence of periodontal disease. J Periodontol. 2005; 76(2):262–267. [PubMed: 15974851]
15. Kingman A, Susin C, Albandar JM. Effect of partial recording protocols on severity estimates of periodontal disease. J Clin Periodontol. 2008; 35(8):659–667. [PubMed: 18513337]
16. Silness J, Loe H. Periodontal disease in pregnancy. II. Correlation between oral hygiene and periodontal condition. Acta Odontol Scand. 1964; 22:121–135. [PubMed: 14158464]
17. Arbes SJ Jr, Slade GD, Beck JD. Association between extent of periodontal attachment loss and self-reported history of heart attack: an analysis of NHANES III data. J Dent Res. 1999; 78(12):1777–1782. [PubMed: 10598906]

18. Oliver RC, Brown LJ, Loe H. Variations in the prevalence and extent of periodontitis. J Am Dent Assoc. 1991; 122(6):43–48. [PubMed: 2066519]
19. Brown LJ, Brunelle JA, Kingman A. Periodontal status in the United States, 1988–1991: prevalence, extent, and demographic variation. J Dent Res. 1996; 75(spec):672–683. [PubMed: 8594091]
20. Erdemir EO, Sönmez IS, Oba AA, Bergstrom J, Caglayan O. Periodontal health in children exposed to passive smoking. J Clin Periodontol. 2010; 37(2):160–164. [PubMed: 20041979]
21. Eke PI, Thornton-Evans GO, Wei L, Borgnakke WS, Dye BA. Accuracy of NHANES Periodontal Examination Protocols. J Dent Res. 2010; 89(11):1208–1213. [PubMed: 20858782]
22. Howard G, Wagenknecht LE, Burke GL, et al. Cigarette smoking and progression of atherosclerosis: The Atherosclerosis Risk in Communities (ARIC) Study. JAMA. 1998; 279(2): 119–124. [PubMed: 9440661]
23. Howard G, Wagenknecht LE, Cai J, Cooper L, Kraut MA, Toole JF. Cigarette smoking and other risk factors for silent cerebral infarction in the general population. Stroke. 1998; 29(5):913–917. [PubMed: 9596234]
24. Chen LC, Quan C, Hwang JS, et al. Atherosclerosis lesion progression during inhalation exposure to environmental tobacco smoke: a comparison to concentrated ambient air fine particles exposure. Inhal Toxicol. 2010; 22(6):449–459. [PubMed: 20235771]
25. Vardavas CI, Panagiotakos DB. The causal relationship between passive smoking and inflammation on the development of cardiovascular disease: a review of the evidence. Inflamm Allergy Drug Targets. 2009; 8(5):328–333. [PubMed: 20025578]
26. Nishida N, Yamamoto Y, Tanaka M, et al. Association between involuntary smoking and salivary markers related to periodontitis: a 2-year longitudinal study. J Periodontol. 2008; 79(12):2233–2240. [PubMed: 19053911]
27. Stangherlin EC, Luchese C, Ardais AP, Nogueira CW. Passive smoke exposure induces oxidative damage in brains of rat pups: protective role of diphenyl diselenide. Inhal Toxicol. 2009; 21(10): 868–874. [PubMed: 19225963]
28. Bergstrom J, Eliasson S, Preber H. Cigarette smoking and periodontal bone loss. J Periodontol. 1991; 62(4):242–246. [PubMed: 2037954]
29. Bergstrom J, Preber H. Tobacco use as a risk factor. J Periodontol. 1994; 65(5, suppl):545–550. [PubMed: 8046571]
30. Salvi GE, Brown CE, Fujihashi K, et al. Inflammatory mediators of the terminal dentition in adult and early onset periodontitis. J Periodontal Res. 1998; 33(4):212–225. [PubMed: 9689617]
31. Pabst MJ, Pabst KM, Collier JA, et al. Inhibition of neutrophil and monocyte defensive functions by nicotine. J Periodontol. 1995; 66(12):1047–1055. [PubMed: 8683417]
32. MacFarlane GD, Herzberg MC, Wolff LF, Hardie NA. Refractory periodontitis associated with abnormal polymorphonuclear leukocyte phagocytosis and cigarette smoking. J Periodontol. 1992; 63(11):908–913. [PubMed: 1333526]
33. Gunsolley JC, Pandey JP, Quinn SM, Tew J, Schenkein HA. The effect of race, smoking and immunoglobulin allotypes on IgG subclass concentrations. J Periodontal Res. 1997; 32(4):381–387. [PubMed: 9210092]
34. Tangada SD, Califano JV, Nakashima K, et al. The effect of smoking on serum IgG2 reactive with Actino-bacillus actinomycetemcomitans in early-onset periodontitis patients. J Periodontol. 1997; 68(9):842–850. [PubMed: 9379328]
35. Gurlek O, Lappin DF, Buduneli N. Effects of smoking on salivary C-telopeptide pyridinoline cross-links of type I collagen and osteocalcin levels. Arch Oral Biol. 2009; 54(12):1099–1104. [PubMed: 19850280]
36. Nishida N, Yamamoto Y, Tanaka M, et al. Association between passive smoking and salivary markers related to periodontitis. J Clin Periodontol. 2006; 33(10):717–723. [PubMed: 16889628]
37. Vital signs: nonsmokers' exposure to secondhand smoke –United States, 1999–2008. MMWR Morb Mortal Wkly Rep. 2010; 59(35):1141–1146. [PubMed: 20829748]

TABLE 1

Relationship of Covariates With Severe Periodontitis and Secondhand Smoke Exposure, Adjusted for Age, Gender–HRT, Education, and Center–Race: Atherosclerosis Risk in Communities Study; Forsyth County, NC; Jackson, MS; suburban Minneapolis, MN; and Washington County, MD; 1996–1998

Variable	No.	Periodontitis Cases, %	*P*	Exposed to Secondhand Smoke, %	*P*
Severe periodontitis case status					.08
Cases	438	…		37.6	
Noncases	2301	…	…	33.0	
Secondhand smoke categories			.099		
No exposure	1816	15.1		…	
1–25 h/wk	806	17.2		…	
≥26 h/wk	117	22.4		…	
Gender–HRT[a,b]			.001		.004
Women using HRT	817	9.7		30.4	
Women not using HRT	1229	16.0		33.3	
Men	692	23.5		38.4	
Age,[a] y			.350		.013
53–61	1313	15.3		36.0	
62–74	1426	16.6		31.6	
Race/ethnicity[a]			.107		.101
Black	554	18.2		36.6	
White	2185	15.4		33.0	
Center-race[a]			.001		.001
Forsyth—Blacks	52	30.8		59.6	
Forsyth—Whites	638	6.1		39.5	
Jackson—Blacks	502	16.9		34.2	
Minneapolis—Whites	793	15.6		30.3	
Washington—Whites	754	23.1		30.2	
Education,[a,b] y			.102		.001
≤11	337	19.6		37.4	
12–16	1266	16.2		37.9	
≥17	1132	14.8		28.0	

Am J Public Health. Author manuscript; available in PMC 2013 August 30.

Variable	No.	Periodontitis Cases, %	*P*	Exposed to Secondhand Smoke, %	*P*
Alcohol			.359		.025
Current drinker	1134	17.3		35.4	
Former drinker	642	15.6		36.3	
Never drinker	963	14.7		30.2	
Alcohol consumption			.453		.012
< 54 g/wk (5 glasses of wine)	2492	15.8		33.0	
≥54 g/wk	247	17.8		41.4	
Coffee (cups)			.628		.001
> 4/d	277	14.8		34.4	
1–3/d	1102	18.4		41.0	
1–24/mo	457	15.6		36.7	
Almost never	903	16.4		27.6	
Body mass index (kg/m^2)[b]			.021		.001
< 27	1148	14.0		29.9	
≥27	1584	17.5		36.7	
Diabetes[b]			.848		.232
Yes	336	16.1		36.8	
No	2394	15.7		33.3	
Current use of aspirin[b]			.362		.663
Yes	684	14.9		34.5	
No	2050	16.4		33.5	
Current use of other NSAIDs[b]			.369		.573
Yes	361	14.3		35.1	
No	2374	16.3		33.6	
Times brushed teeth yesterday[b]			.644		.211
Not at all	26	9.3		17.1	
1	642	16.8		34.0	
2	1654	15.7		34.5	
≥3	407	16.5		31.5	
Times used floss last week[b]			.377		.058

Variable	No.	Periodontitis Cases, %	*P*	Exposed to Secondhand Smoke, %	*P*
Not at all	819	17.3		35.7	
1	225	15.7		31.2	
2	267	18.5		39.6	
≥3	1415	14.9		31.9	
Frequency of dental visits[b]			.036		.096
Other	25	17.7		49.2	
Don’t go	32	33.3		29.9	
Only when in discomfort	176	22.9		40.0	
When something needs fixing	400	15.9		37.5	
Regular basis	2094	15.2		32.4	

Note. HRT = hormone replacement therapy; NSAIDs = nonsteroidal anti-inflammatory drugs.

[a]Not adjusted.

[b]Numbers do not sum to 2739 because of missing values.

Am J Public Health. Author manuscript; available in PMC 2013 August 30.

TABLE 2

Relationship of Periodontal Examination Parameters With Severe Periodontitis and Secondhand Smoke Exposure, Adjusted for Age, Gender–Hormone Replacement Therapy, Education, and Center–Race: Atherosclerosis Risk in Communities Study; Forsyth County, NC; Jackson, MS; suburban Minneapolis, MN; and Washington County, MD; 1996–1998

Examination Parameters	No.	Periodontitis case, %	*P*	Exposure to Secondhand Smoke, %	*P*
Bleeding on probing			.001		.005
< 15% of sites	1155	3.4		30.4	
≥15% of sites	1584	25.3		36.2	
Clinical attachment levels ≥3 mm			.001		.017
< 60% of sites	2626	13.7		33.3	
≥60% of sites	113	68.9		44.8	
Clinical attachment levels ≥3 mm			.001		.018
< 25% of sites	2156	7.9		33.3	
≥25% of sites	583	46.0		44.7	
Probing pocket depths ≥4 mm			NA		.204
< 2% of sites	1428	0.0		32.6	
≥2% of sites	1311	33.4		35.1	
Dental plaque, PI ≥1[a]			.001		.009
< 15% of sites	949	8.3		30.5	
≥15% of sites	1639	19.5		36.0	
Dental plaque, PI ≥2[a]			.001		.055
< 15% of sites	2399	14.1		33.4	
≥15% of sites	189	32.1		40.9	

Note. NA = not applicable; PI = increase in dental plaque.

[a]Numbers do not total 2739 due to missing values.

TABLE 3

Binary Logistic Regression Analysis of the Relationship Between Secondhand Smoke and Severe Periodontitis: Atherosclerosis Risk in Communities Study; Forsyth County, NC; Jackson, MS; suburban Minneapolis, MN; and Washington County, MD; 1996–1998

Level of Secondhand Smoke Exposure	Unadjusted OR (95% CI)	AOR (95% CI)[a]
≥26 h/wk	1.5 (0.9, 2.3)	2.0 (1.2, 3.4)
1–25 h/wk	1.2 (0.9, 1.9)	1.3 (1.0, 1.7)
Not exposed to secondhand smoke (Ref)	1.0	1.0

Note. AOR = adjusted odds ratio; CI = confidence interval; OR = odds ratio. Severe periodontitis case status was defined as ≥5 sites with clinical attachment level ≥3 mm and probing depth ≥5 mm. The pseudo-R^2 statistic = 0.002 for the unadjusted model and 0.16 for the adjusted model. ORs and 95% CI are unadjusted and adjusted for covariates.

[a]Adjusted for age, education, center and race, gender and hormone replacement therapy, remaining teeth, extent of plaque scores ≥2, and dental visiting pattern.

TABLE 4

Multivariate Binomial Regression Model of the Relationship Between Secondhand Smoke and 2 Periodontal Disease Parameters: Atherosclerosis Risk in Communities Study; Forsyth County, NC; Jackson, MS; Suburban Minneapolis, MN; and Washington County, MD; 1996–1998

	Clinical attachment level ≥3 mm		Periodontal pockets ≥4 mm	
Level of Secondhand Smoke Exposure	**Unadjusted OR (95% CI)**	**AOR (95% CI)[a]**	**Unadjusted OR (95% CI)**	**AOR (95% CI)[a]**
≥26 h/wk	1.4 (1.1, 1.8)	1.3 (1.0, 1.6)	1.2 (0.9, 1.7)	1.3 (0.9, 1.8)
1–25 h/wk	1.1 (1.0, 1.2)	1.1 (1.0, 1.2)	1.2 (1.0, 1.3)	1.1 (1.0, 1.9)
Unexposed (Ref)	1.0	1.0	1.0	1.0

Note. AOR = adjusted odds ratio; CI = confidence interval; OR = odds ratios.

[a]Adjusted for age, education, center and race, gender and hormone replacement therapy, remaining teeth, extent of plaque scores ≥2, and dental visiting pattern.