Environmental Tobacco Smoke and Periodontal Disease in the United States

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ABSTRACT

Objectives. Cigarette smoking is a leading risk factor for periodontal disease. This cross-sectional study investigated the relation between environmental tobacco smoke (ETS) and periodontal disease in the United States.

Methods. Data were obtained from the Third National Health and Nutrition Examination Survey (1988–1994). The outcome was periodontal disease, defined as 1 or more periodontal sites with attachment loss of 3 mm or greater and a pocket depth of 4 mm or greater at the same site. Exposure to ETS at home and work was self-reported. The study analyzed 6611 persons 18 years and older who had never smoked cigarettes or used other forms of tobacco.

Results. Exposure to ETS at home only, work only, and both was reported by 18.0%, 10.7%, and 3.8% of the study population, respectively. The adjusted odds of having periodontal disease were 1.6 (95% confidence interval = 1.1, 2.2) times greater for persons exposed to ETS than for persons not exposed.

Conclusions. Among persons in the United States who had never used tobacco, those exposed to ETS were more likely to have periodontal disease than were those not exposed to ETS. (*Am J Public Health.* 2001;91:253–257)

Exposure to environmental tobacco smoke (ETS), also known as passive smoking, is the third leading preventable cause of death in the United States—surpassed only by cigarette smoking and alcohol use.[1] ETS contains more than 4000 chemicals, including nicotine and at least 40 known carcinogens.[2] It has been estimated that ETS exposure is responsible for 53 000 deaths annually in the United States.[1,3] Because cigarette smoking is a major risk factor for coronary heart disease and lung cancer, it is not surprising that studies have also linked ETS to these 2 diseases.[1,4–8] However, a causal relation between ETS and coronary heart disease is not yet universally accepted in the scientific community.

In addition to the association between ETS exposure and heart disease and cancer, ETS exposure has been linked to developmental and respiratory effects. In its final report on the health effects of ETS, the California Environmental Protection Agency reported that ETS exposure was causally associated with low birthweight and sudden infant death syndrome.[9] The agency also reported that among children, ETS exposure was causally associated with asthma induction and exacerbation, middle ear infections, chronic respiratory symptoms, and acute lower respiratory tract infections such as bronchitis and pneumonia.[9] Evidence also suggested a causal association between ETS exposure and spontaneous abortion, adverse effects on cognition and behavior, exacerbation of cystic fibrosis, decreased pulmonary function, and cervical cancer.[9] With time, ETS likely will be causally linked to other diseases, especially diseases already linked to cigarette smoking.

One disease that has the potential for such an association is periodontal disease, an infectious disease that destroys the soft tissues and bone supporting the teeth. Cigarette smoking is an important, if not the most important, risk factor for periodontal disease.[10–24] Cigarette smokers are up to 5 times more likely than nonsmokers to develop severe periodontitis.[21] Approximately half of the cases of periodontitis in individuals younger than 30 years are thought to be associated with cigarette smoking.[21] Even though periodontal disease is an infectious disease caused by bacteria, cigarette smoking is believed to increase individuals' susceptibility to periodontal pathogens and tissue destruction. Potential mechanisms for the effect of smoking on periodontal disease include immunosuppression and exaggerated inflammatory cell responses.[25] Recent reports of associations between periodontal disease and systemic diseases such as coronary heart disease,[26–33] stroke,[30] preterm low-birthweight babies,[34,35] and respiratory diseases[36,37] have provided an impetus for identifying new risk factors for periodontal disease and learning more about its pathogenesis.

Only 1 report has been published on the relation between ETS and periodontal disease.[38] The study reported a strong relation between passive smoking in the home and periodontal disease but did not assess workplace exposure or take cigarette smoking into account, even though it included cigarette smokers. The objective of the present study was to examine the association between ETS exposure in the home and at work and the prevalence of periodontal disease among persons who had never used tobacco.

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Note. This study was reviewed by the University of North Carolina School of Dentistry Committee on Investigations Involving Human Subjects, approved at minimal risk, and declared exempt from further review.

Note. Pocket depth (PD) is the distance from the gingival margin to the bottom of the sulcus. Attachment loss (AL) is measured from the cementoenamel junction (CEJ) to the bottom of the sulcus.

FIGURE 1—Normal gingival sulcus and a periodontal pocket.

Methods

Study Data and Design

Data for this study were obtained from the Third National Health and Nutrition Examination Survey (NHANES III) conducted from 1988 to 1994. The survey was the seventh in a series of national surveys designed to provide estimates of the health status of the US population. The NHANES III used a complex cross-sectional survey design to sample the total civilian, noninstitutionalized population 2 months or older.[39] A complete description of the plan and operation of the NHANES III may be found elsewhere.[40]

TABLE 1—Bivariate Distribution of Persons, by Study Variables and Periodontal Disease[a]

TABLE 2—Multivariate Logistic Model for the Presence of Periodontal Disease[a] (n = 5658)

	β Coefficient	SE	Adjusted OR (95% CI)	Wald F *P*
Intercept	–3.8915	0.3481	…	…
ETS exposure				
No	0.0000	0.0000	1.00 (reference)	
Yes	0.4534	0.1567	1.57 (1.15, 2.16)	.006
Age, y				
18–29	0.0000	0.0000	1.00 (reference)	
30–49	1.2197	0.1851	3.39 (2.33, 4.91)	
50–69	1.5643	0.2090	4.78 (3.14, 7.27)	
≥70	1.9197	0.2793	6.82 (3.89, 11.95)	.000
Sex				
Female	0.0000	0.0000	1.00 (reference)	
Male	0.3430	0.1560	1.41 (1.03, 1.93)	.033
Race/ethnicity				
Non-Hispanic White	0.0000	0.0000	1.00 (reference)	
Non-Hispanic Black	1.1108	0.1898	3.04 (2.07, 4.45)	
Mexican American	0.3463	0.2123	1.41 (0.92, 2.17)	
Other	0.5488	0.3544	1.73 (0.85, 3.53)	.000
Education, y				
<12	0.0000	0.0000	1.00 (reference)	
12	–0.2601	0.2560	0.77 (0.46, 1.29)	
>12	–0.5724	0.2296	0.56 (0.36, 0.89)	.046
Poverty index				
0.0–1.9	0.0000	0.0000	1.00 (reference)	
2.0–11.9	–0.1404	0.1526	0.87 (0.64, 1.18)	.362
History of diabetes				
No	0.0000	0.0000	1.00 (reference)	
Yes	0.3810	0.2484	1.46 (0.89, 2.41)	.132
Dental visits				
At least once per year	0.0000	0.0000	1.00 (reference)	
Every 2 years	0.4761	0.2710	1.61 (0.93, 2.78)	
<Every 2 years	0.7095	0.5639	2.03 (0.65, 6.31)	
As needed/other	0.5923	0.2031	1.81 (1.20, 2.72)	.032

Note. OR = odds ratio; CI = confidence interval; ETS = environmental tobacco smoke.
[a]Defined as 1 or more periodontal sites with both an attachment loss of 3 mm or greater and a pocket depth of 4 mm or greater.

the temporal relation between these 2 factors is not clear. It is certainly possible that the onset of periodontal disease preceded the exposure to ETS in some subjects.

To increase the likelihood that active periodontal disease succeeded or at least coexisted with the exposure, this study defined a periodontal case as one in which the person had at least 1 tooth site with attachment loss and a periodontal pocket. In many studies, attachment loss alone is used as an indicator of periodontal disease. However, attachment loss, which is generally considered irreversible, is a cumulative measure of periodontal destruction throughout one's lifetime. The presence of attachment loss does not necessarily indicate the presence of active disease.

Contributors

S. J. Arbes Jr planned the study, analyzed the data, and wrote the paper. H. Ágústsdóttir and G. D. Slade developed the study question and contributed to the writing of the paper. G. D. Slade supervised the data analysis.

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