Environmental Tobacco Smoke and Periodontitis in U.S. Non–Smokers

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Abstract

Purpose: The association of second hand smoke or environmental tobacco smoke (ETS) and periodontitis in non–smokers has not been confirmed using a biomarker of ETS exposure. To estimate periodontitis prevalence in non–smokers with detectable serum cotinine, and to investigate racial/ethnic and socioeconomic variation in ETS exposure in a representative sample of the U.S. adult population. Determining periodontitis risk indicators occurring with ETS appears to be a salient purpose as this study is the first of its kind to provide a link (a salivary biomarker) between second hand smoke and risk for periodontitis.

Methods: Data were collected from the 1999 to 2004 National Health and Nutrition Examination Survey (NHANES). Subjects were 3,137 adults who had smoked fewer than 100 cigarettes and had not used other forms of tobacco. ETS exposure was classified as negligible (cotinine concentrations below sex and race/ethnicity cut–points for smokers), moderate (cotinine 0.5–<1.5 µg/mL) or high (cotinine≥1.5 ng/mL). Periodontitis was classified according to the Centers for Disease Control and Prevention (CDC) and the American Academy of Periodontology (AAP) case definition for moderate–severe disease. Survey estimation procedures were used to estimate prevalence and odds ratios (OR) were from multivariable logistic regression models.

Results: ETS exposure was observed in 40.5% of subjects and 2.6% had periodontitis. ETS exposure was inversely associated with educational attainment and family income and was higher in non–Hispanic blacks than whites. After adjusting for age, sex and year of survey, adults with high ETS exposure (cotinine≥1.5 ng/mL) had more than twice the odds of periodontitis as people with negligible exposure (OR=2.3, 95% confidence interval=1.3, 4.1).

Conclusion: High ETS exposure was a risk indicator for periodontitis in lifetime non–smokers.

Keywords: Cotinine, Periodontal disease, Serum–plasma, Tobacco

This study supports the NDHRA priority area, **Health Promotion/Disease Prevention:** Investigate how environmental factors (culture, socioeconomic status– SES, education) influence oral health behaviors.

Introduction

The American Academy of Periodontology (AAP) estimates prevalence of moderate to severe generalized periodontitis to be 30% or higher in the U.S. adult population, depending on the classification.[1] Periodontal disease impacts a large number of Americans and plays a role in other more serious and costly health problems. Periodontitis is a leading cause of tooth loss, tooth mobility and dental abscess, and is also positively associated with cardiovascular disease,[2] obesity,[3] Alzheimer's disease[4] and diabetes mellitus.[5–12] The disease is characterized by chronic inflammation, loss of attachment and bone loss. The condition is primarily caused by bacteria in dental plaque acting alone or in conjunction with systemic and genetic factors.[13] Other factors associated with the disease include psychological stress,[14] certain medications,[15,16] genetics[16] and tobacco use.[12,17,18] In fact, tobacco use is causally associated with periodontitis[12,18–21] in a dose dependent relationship,[22] and studies estimate the smoking attributable risk to be 20%.[1] Cigarette smoking, along with vasoconstriction, impacts individual cells involved with the perpetuation of periodontal disease, such as those involved in inflammation, immunity, cell differentiation and healing.[23] Within the estimated 3.6 to 5% of Americans with periodontal disease,[24,25] current smokers exhibit higher rates of disease.[26] Smoking alters microbial and host response factors in periodontitis, and has been implicated in bone loss, such as osteoporosis.[27] In respect to microbes, preliminary findings by Teughels et al indicate that individual periopathogens' (A. Actinomycetemcomitans and P. Gingivalis) colonization of tissues could be impacted by nicotine, found in smoke, in a species–specific manner.[28] Environmental tobacco smoke (ETS), like active smoking, impacts the immune response, namely polymorphonuclear leukocyte (PMN) function such as phagocytosis, chemot-

axis and oxidative burst.[29] As reported by Numabe et al, phagocytic activities of PMN intensify after smoking and exposure to ETS.[29] Additionally, the results suggested that certain substances in smoke over–stimulate the host response in the oral cavity,29 making the exposed more likely to experience attachment and tooth loss.[30]

Risk for periodontitis increases with the number of cigarettes smoked, or consumption, with notable differences observed in as few as 10 cigarettes per day.[22,26,31,32] Periodontitis is 6 to 7 times as prevalent in the estimated 46 million adults in the U.S. who currently smoke.[33,34] Smoking also makes the disease more virulent and difficult to treat.[21,35,36]

Non–smokers exposed to ETS absorb approximately one–third the level of nicotine per cigarette absorbed by active smokers.[37–40] Physiological metabolism of nicotine after exposure yields cotinine (nicotine's metabolite) in saliva, urine and serum.[37] The concentration of cotinine in fluids allows determination of active smoking or environmental exposure, and provides a recent measurement of exposure, as well as an objective biomarker of exposure.[40]

There is evidence of a relationship between periodontitis in non–smokers and exposure to environmental tobacco smoke.[41,42] Arbes et al observed that non–smokers with self–reported ETS exposure had 1.6 times the odds for periodontal disease compared to those not exposed.[41,43,44] The increased risk for periodontitis occurs with the exposure to nicotine which over–stimulates the host response in the oral cavity, complicating the already inflammatory nature of periodontal diseases.[27,41] In fact, the inflammatory response in salivary inflammatory markers is notable among those exposed to secondhand smoke as ETS is associated with an elevated concentration of inflammatory makers interleukin –1β, albumin and aspartate aminotransferase, in those exposed to passive smoke.[45,46] To date, measurements of ETS in the periodontal literature are limited to self–report and no objective biomarker of exposure has been examined.

ETS exposure is unequally distributed between racial and ethnic groups. For physiological and behavioral reasons, non–Hispanic Blacks show higher concentrations of cotinine, with less exposure to cigarette smoke, than do non–Hispanic whites. Total and non–renal clearance of circulating cotinine is significantly lower in non–Hispanic blacks.[47] Furthermore, nicotine intake is 30% higher in African Americans, with a somewhat longer half–life for circulating cotinine.[47] The different absorption and manifestation of serum cotinine concentration in different races is supported by the prevalence of periodontitis cases. According to Albandar et al, African Americans and Mexican Americans display poorer periodontal health than whites with comparable income and educational attainment.[48] Signorello et al reported that "differences in cotinine levels among smokers suggest racial variation in exposure to/or metabolism of tobacco smoke constituents."[49]

Smoking and ETS exposure are known hazards to health, including the oral cavity, and tooth attachment apparatus.[12] Together with racial and socioeconomic status, the differing levels of ETS exposure and different rates of metabolism for serum cotinine provide a means and motivation to assess periodontal disease risk among the non–smoking population.[41] The aim of this study was to determine the prevalence of periodontitis in non–smokers with detectable serum cotinine, and to investigate the variation in ETS exposure among non–smokers classified according to racial and socio–economic characteristics.

Methods and Materials

Study and Sampling Designs

This cross–sectional study is nested within a larger study designed to examine the relationship of a state's cigarette excise tax on cigarette sales and levels of ETS. Data were obtained from the National Health and Nutrition Examination Survey (NHANES) release dates 1999 to 2000, 2001 to 2002 and 2003 to 2004. The NHANES is an ongoing representative survey of the health and nutrition status of the civilian, non–institutionalized U.S. population, conducted by the National Center for Health Statistics (NCHS).[50]

The NHANES uses a complex cross–sectional survey design to sample participants 2 months of age and older.[41] Because NHANES typically samples 15 primary sampling units per survey, the current study combined 3 survey releases to maximize the number of sampled states.

Data Collection

Data collection consisted of a household interview, blood draw and a medical examination including a dental examination, conducted in the Mobile Examination Center. The household interview included questions pertaining to socioeconomic characteristics, medical/dental history and health behaviors, such as smoking. During the physical examination, blood was collected by venipuncture to allow for serum cotinine measurement in participants over 3 years of age.[51] Signed informed

consent was obtained for all participants, in person or by proxy.

Participants

In the combined 1999 to 2004 NHANES data, 9,932 adults aged 20 years or older received a periodontal assessment. Those who reported having smoked at least 100 cigarettes in their lifetime (n=4,553) were precluded from analysis. Also precluded were 13 adults with undisclosed smoking status, along with individuals with a history of tobacco use through pipe, cigar, snuff or chewing tobacco (n=456). Examination of serum cotinine identified participants whose sex or race/ethnicity–specific concentrations exceeded thresholds for non–smokers (n=437), and these were likewise ineligible. Finally, adults having lived in the U.S. fewer than 10 years were precluded (n=1,336) since ETS exposure in these individuals could not be related to the state–level excise as this study is nested within a greater investigation of tobacco excise tax and its relationship to periodontitis. Hence this analysis was limited to 3,137 U.S. lifetime non–smokers.

Dependent Variable

An assessment of periodontal tissues was conducted by a licensed dentist during the NHANES oral examination. Examination measured bleeding on probing and periodontal pocket depth for 2 randomly assigned quadrants: 1 upper and 1 lower. Probing was done using a National Institute of Dental Research probe.

The assessment included permanent fully erupted teeth, excluding root tips, partially erupted teeth and third molars. Measurements used were taken from the mesial and mid–buccal aspects of the teeth from distal to mesial, beginning with the distal–most tooth, moving toward the midline. Over the 6 year survey period, periodontal measurement techniques differed. For release dates 1999 to 2000, periodontal measurements were taken at 2 sites on each assessed tooth: midbuccal and mesiobuccal. For release dates 2001 to 2002 and 2003 to 2004, measurements were collected from the midbuccal, mesiobuccal and distobuccal sites of teeth. For consistency during analysis, the mesiobuccal numbers were analyzed for the entire survey period, as interproximal sites pertain directly to the case definition used.

Periodontal cases were defined using a case classification developed by the AAP and the CDC.[24] The AAP defines moderate/severe periodontal disease as "two or more interproximal sites with clinical attachment level ≥4 mm, not on the same tooth, or two or more interproximal sites with probing depth ≥5 mm, not on the same tooth."[24]

Key Exposure Variable

Questions about smoking history and use of tobacco products were presented in the household interview. Environmental tobacco smoke exposure was measured using serum cotinine measurements collected during the medical examination. Exposure was defined as serum cotinine measurements ≥0.05 ng/mL, as this is the NHANES laboratory–limit for detection. The use of the biomarker cotinine was indicated due to its ability to reflect nicotine exposure over days and its specificity to nicotine,[52] evaluating only recent cigarette smoke exposure as opposed to all environmental inhaled substances.[53]

Independent Variables

Along with tobacco smoke exposure, the characteristics age, sex, educational attainment, annual family income and ethnicity were considered independent variables. These characteristics were identified during the household interview questionnaire.

Results

In this non–smoking subset of the general U.S. population, males and all individuals with low levels of education and family income were under–represented. According to serum cotinine concentrations, 40.5% of participants were exposed to ETS (Table I). Greater proportions of males than females were exposed, and adults 20 to 49 years of age were more likely to be exposed than were their older counterparts ($p<0.001$). Most pronounced differences in ETS exposure were found between racial groups. Two–thirds of African Americans were exposed compared with approximately one–third of Non–Hispanic whites ($p<0.001$). Even within this advantaged subset of the U.S. population, inverse socioeconomic gradients were observed in levels of ETS exposure (Table I).

The CDC/AAP case classification for moderate or severe periodontitis was met by 2.6% of participants (n=82, Table II). Of note, serum cotinine concentration was not significantly associated with periodontitis in unadjusted analysis. In addition, the associations of periodontitis with sex and race/ethnicity were statistically non–significant, while age and socioeconomic status were strongly associated with the disease. Odds of periodontitis were elevated 9–fold in adults with incomplete high school

Table I: Selected characteristics of the dentate non–smoking population aged 20 years or older, resident in the U.S. for ≥10 years, and the percentage exposed to environment tobacco smoke (n=3,137), NHANES 1999 to 2004

Characteristic	Unweighted n and weighted %	Exposure to ETS (%)[a]	95% CI	P–value
All	3,137 (100.0)	40.5	35.9, 45.2	
Sex				
Male	1,090 (36.9)	46.4	40.3, 52.6	<0.001
Female	2,047 (63.1)	37.0	32.7, 41.6	
Age group (years)				
20–49 years	2,003 (69.7)	43.9	38.9, 48.9	<0.001
50–85 years	1,134 (30.3)	32.6	27.7, 38.0	
Race/ethnicity				
Non–Hispanic White	1,858 (79.2)	36.2	31.1, 41.7	<0.001
Non–Hispanic Black	718 (12.4)	65.7	60.0, 71.1	
Hispanic	522 (6.9)	41.1	33.1, 49.7	
Other	39 (1.5)	51.3	31.4, 70.7	
Educational attainment				
Less than high school	513 (9.8)	58.4	51.0, 65.5	<0.001
High school graduate or equivalent	725 (22.7)	50.7	44.6, 56.7	
Some college or more education	1,898 (67.5)	34.4	29.4, 39.8	
Missing	1			
Annual family income				
<$25,000	930 (24.5)	54.2	47.5, 60.7	<0.001
$25,000–<$75,000	1,352 (44.4)	40.7	34.8, 46.8	
≥$75,000	756 (31.2)	29.1	23.7, 35.2	
Missing	99			

[a]Environmental tobacco smoke exposure was determined by sex– and race–specific thresholds of serum cotinine above the laboratory detection limit for 1999–2000 NHANES of 0.05ng/mL
b All estimates are weighted data, except the number of study participants, which is reported unweighted

education relative to those with at least some college education (OR=9.1, 95% CI: 5.2, 15.9). In the multivariable model (Table III) that adjusted for potential confounding of age and other factors, odds of periodontitis were 89% higher in adults with cotinine concentration ≥1.5ng/mL compared to those with negligible concentrations. The predicted probability of meeting the periodontitis case classification increased monotonically with increasing levels of serum cotinine concentration (Figure 1). For these results, binary logistic regression was computed using STATA software.

Discussion

This study sought to evaluate the relationship between environmental tobacco smoke and periodontitis in non–smokers using an objective biomarker. The primary finding was that periodontitis in non–smokers is negatively impacted by exposure to environmental tobacco smoke. This stands in agreement with similar previous studies such as Arbes et al who found a relationship between self–reported smoke exposure and periodontitis in non–smokers.[54] Other investigators have reported an increase in salivary markers related to periodontitis with exposure isolated through salivary cotinine.[45,46] NHANES data provided a representative sample of the American population, as well as a large sample size for analysis. Moreover, it allowed for analysis of tobacco use in addition to cigarettes alone. Specifically, it allowed for the study of participants controlled for cigar, pipe, snuff and chew tobacco use. Both the medical history questionnaire in the NHANES protocol and the serum concentration tests for serum cotinine added to reporting accuracy.

This study evaluated data from 1999 to 2004. Since that time regulations controlling exposure of

Table II: Mean (95% CI) serum cotinine level (ng/mL), prevalence of periodontitis (95% CI) and odds ratios for periodontitis (95% CI) according to socio-demographic characteristics of study participants (n=3,137), NHANES 1999 to 2004

Characteristic	Serum cotinine (ng/mL) mean (95% CI)	P–value	Periodontitis[a] prevalence (95% C.I.)	P–value	OR periodontitis (95% CI)
All	0.20 (0.18, 0.23)	–	2.61 (2.08, 3.26)	–	–
Sex					
Male	0.25 (0.21, 0.30)	<0.001	2.16 (1.46, 3.17)	0.302	1.34 (0.76, 2.36)
Female	0.17 (0.15, 0.20)		2.87 (2.09, 3.92)		Ref
Age group					
20–49 years	0.23 (0.20, 0.27)	<0.001	0.49 (0.32, 0.75)	<0.001	Ref
50–85 years	0.13 (0.10, 0.16)		7.46 (5.98, 9.28)		16.27 (10.49, 25.23)
Race/ethnicity					
Non–Hispanic White	0.16 (0.14, 0.19)	<0.001	2.33 (1.78, 3.04)	0.146	Ref
Non–Hispanic Black	0.50 (0.40, 0.59)		4.07 (2.84, 5.80)		1.78 (1.10, 2.88)
Hispanic	0.11 (0.08, 0.13)		2.70 (1.56, 4.63)		1.16 (0.64, 2.12)
Other	0.18 (0.06, 0.30)		4.79 (0.90, 21.77)		2.11 (0.42, 10.65)
Educational attainment[b]					
<High school	0.44 (0.33, 0.54)	<0.001	9.48 (6.96, 12.80)	<0.001	9.07 (5.16, 15.94)
High school or equivalent	0.26 (0.20, 0.32)		4.00 (2.60, 6.09)		3.60 (1.95, 6.65)
≥Some college	0.15 (0.12, 0.18)		1.14 (0.75, 1.72)		Ref
Annual family income[b]					
<$25,000	0.36 (0.28, 0.43)	<0.001	5.24 (3.77, 7.25)	<0.001	6.27 (2.45, 16.04)
$25,000–<$75,000	0.19 (0.15, 0.22)		2.32 (1.61, 3.33)		2.69 (1.07, 6.72)
≥$75,000	0.10 (0.07, 0.13)		0.88 (0.38, 2.00)		Ref
Serum cotinine concentration[c]					
<0.05 ng/mL	0.02 (0.02, 0.03)	<0.001	2.33 (1.66, 3.27)	0.509	Ref
0.05–<0.15 ng/mL	0.09 (0.08, 0.09)		3.06 (1.94, 4.80)		1.32 (0.73, 2.40)
≥1.5 ng/mL	0.82 (0.75, 0.90)		2.97 (1.96, 4.48)		1.28 (0.74, 2.22)

[a]CDC/AAP case classification for moderate or severe periodontitis defined as ≥2 interproximal sites with clinical attachment level ≥4 mm, not on the same tooth, or ≥2interproximal sites with probing depth ≥5 mm, not on the same tooth

[b]Fewer than 3,137 subjects were analyzed because of missing data

[c]The laboratory detection limit fort 1999–2000 NHANES (0.05) was applied for all years (1999–2004)

ETS to non–smokers have changed. For example, in 2009, the Family Smoking Prevention and Tobacco Control Act was passed granting the Food and Drug Administration the authority to regulate tobacco products.[55] Among the states, North Carolina recently passed tobacco control legislation to ban cigarette smoking in restaurants as of January 20, 2010.[56] Of the 50 states in America, 50% of the U.S. population was protected by some combination of Clean Air policies as of 2008.[57] Recent tobacco control acts undoubtedly changed who is exposed to cigarette smoke and at what rate.

Another limitation of the data is that NHANES protocol allows for half–mouth data collection, with limited periodontal reading sites per tooth during the periodontal assessment. However, officials at the CDC concede that this abbreviated assessment protocol under reports periodontitis prevalence.58 The periodontal assessment protocol changed throughout the 5 years of data collection reported in this study, therefore, collected data were reduced to the 2 common sites per tooth. Additionally, NHANES reports that trained dentists performed the periodontal assessments, but no kappa score is reported for intra–rater reliability. The questionnaires and testing methods do not identify in which locale the participants were exposed to second

Table III: Multivariable analysis modeling odds ratio and 95% confidence interval for moderate or severe periodontitis[a] in dentate non–smoking U.S. adults aged ≥20 years[b] (n=2,998), NHANES 1999 to 2004

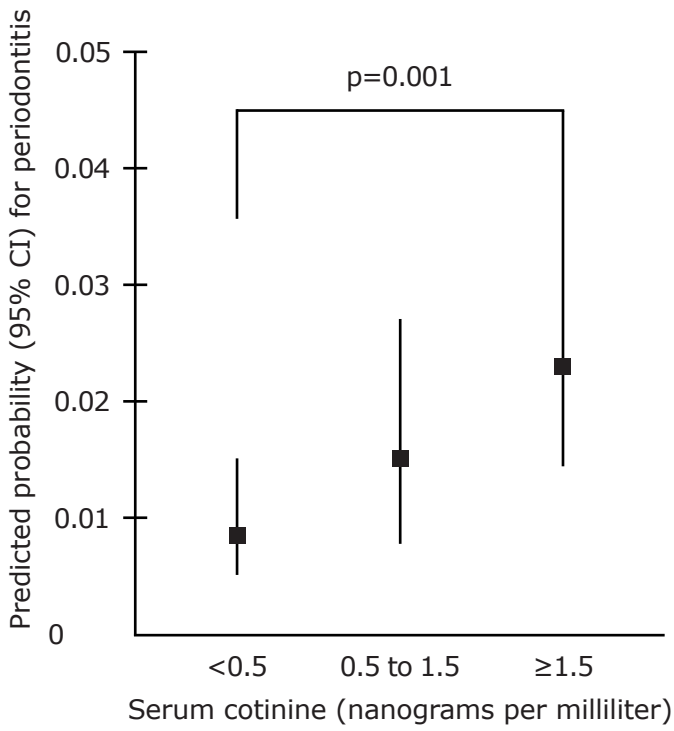
Characteristic	OR (95% CI)
Sex	
Male	1.17 (0.65, 2.12)
Female	Ref
Age in years	1.08 (1.06, 1.10)
Race/ethnicity (c)	
Non–Hispanic white	Ref
Non–Hispanic black	2.52 (1.35, 4.71)
Hispanic	1.70 (0.81, 3.58)
Educational attainment	
Less than high school education	2.74 (1.45, 5.21)
High school graduate or equivalent	1.82 (0.89, 3.71)
Some college or more education	Ref
Annual family income	
<$25,000	1.79 (0.68, 4.70)
$25,000–<$75,000	1.42 (0.57, 3.56)
≥$75,000	Ref
Serum cotinine concentration	
<0.05 ng/mL	Ref
0.05 – <0.15 ng/mL	1.16 (0.62, 2.18)
≥1.5 ng/mL	1.89 (1.08, 3.31)

[a]CDC/AAP case classification for moderate or severe periodontitis defined as ≥2 interproximal sites with clinical attachment level ≥4 mm, not on the same tooth, or ≥2interproximal sites with probing depth ≥5 mm, not on the same tooth

[b]Results are adjusted for year of NHANES survey

[c]Persons identifying racially as "Other" were omitted from this analysis due to the small number of these subjects (n=39)

Figure 1: Predicted probability and 95% confidence interval of having moderate or severe periodontitis according to level of serum cotinine and adjusted for age, sex and year of NHANES, in dentate non–smoking U.S. adults aged ≥20 years (n=3,137), NHANES 1999 to 2004



Periodontitis is defined using the CDC/AAP case classification for moderate or severe periodontitis: either ≥2 interproximal sites with clinical attachment level ≥4 mm, not on the same tooth, or ≥2 interproximal sites with probing depth ≥5 mm, not on the same tooth

hand smoke. For this reason, it is difficult to know which improvements should be made to tobacco control policy.

Unexpectedly, the threshold of harmful exposure differed between racial groups. For example, from the same exposure, non–Hispanic blacks absorb 30% more cotinine than do non–Hispanic whites.[47] Greater absorption of ETS may explain why non–Hispanic blacks were more likely to have periodontitis than non–Hispanic whites. Also unexpected was the finding that younger adults were less likely to have periodontitis while being more exposed to cotinine, however, age is an associated risk factor for periodontitis due to lifetime disease and CAL accumulation.[16,59] The increased exposure in younger adults could be due to lifestyle differences, exposure environments and personal oral hygiene habits.[47]

Studies have previously linked cigarette smoking to race, as well as social gradients in periodontitis.[48,60,61] Therefore, the strong gradient found between income level and cotinine exposure, as well as the one found between education level and exposure, were expected.[62,63] In general, the study methods used here could be implemented in any other nationally representative examination. This study echoes the finding of income, education and race gradients between exposure and disease. It also confirms that tobacco control bans are beneficial[64,65] and should increase in the future as they decrease public smoking and the permeation of environmental tobacco smoke. Future research could evaluate in what specific ways public smoking bans are beneficial to non–smoking, at–risk populations.

Currently, psychological tools and assessment instruments are used to encourage meaningful and motivated behavior change in patients, as well as increase provider confidence in providing cessation techniques.[66,67] This study has strong and timely implications for dental hygiene practice. An update on clinical practice guidelines regarding smoking cessation counseling estimated a 2–fold increase in smoking cessation counseling since the early 1990s, as well as a steadily decreasing rate of smokers.[68] Multiple controlled trials report efficacy in tobacco cessation counseling,[67–69] indicating that moments shared by patients and providers in dental care settings are teachable moments,[70] and that patients listen and are encouraged by the focus on individualized oral health. For instance, patients are more likely to approach tobacco behavior change in response to existing oral complaints such as tooth color or oral malodor that can be associated with smoking.[71] For that reason, as well as the documented link between cigarette smoke and systemic disease,[72–74] this study is crucial.

Dental hygienists are in a powerful position to affect future behaviors of patients by utilizing those teachable moments to relate to patients and identify those at risk. Research demonstrates that flexibility in tobacco education curriculum encourages incorporation of tobacco education in dental hygiene programs.[75] In an ever expanding body of research, the curriculum should expand to include the most recent evidence – that ETS affects the periodontal health of even non–smoking patients. This, along with continued research, could further strengthen the education provided to patients as well as the confidence with which it is delivered.[76]

The strong relationship found between serum cotinine and increased odds of periodontitis provides evidence that mere smoking cessation counseling is not enough. Education about risk of cigarette smoke should also express the risk of passive smoke exposure. This finding holds importance for health care providers in a position to advise and educate patients. Since a large percentage of those unwillingly exposed to second hand smoke are children, an effort to inform parents though public health initiatives and stronger tobacco control policies for homes and cars would be valuable.[77]

In the future, similar studies with more recent release dates are needed to compare the differences in exposure to non–smokers as tobacco control policy increases. Sub–grouped participants in areas of high tobacco control, moderate and low areas of tobacco control would further identify the benefit of reducing exposure, particularly in areas with disadvantaged populations. Due to the strong socioeconomic gradients, studies of the knowledge and opinions about passive smoke of at–risk groups could illuminate shortcomings in education to protect those most at risk of exposure and help to advance tobacco control policies.

Conclusion

Cigarette smoke is harmful to periodontal health, whether exposure is voluntary or involuntary. ETS is implicated in a list of diseases that mirrors those caused by firsthand smoke, with a similar mechanism of action. For measurement of environmental exposure, especially in non–smokers, the mechanism of choice is isolation of cotinine in bodily fluids such as serum, saliva and urine.

Of the impacted diseases, periodontitis is one of importance. This study proposed to examine the relationship between objectively measured exposure to environmental tobacco smoke and periodontitis. By and large, the Americans most affected by both smoke and disease are those in the lower socioeconomic classes, namely low income, low education and minority groups. Ultimately, roughly half the non–smokers sampled were exposed to ETS, and their exposure was significantly associated with 2–fold risk of periodontitis.

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