Association of Pediatric Dental Caries With Passive Smoking

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TOOTH DECAY IS THE MOST COMMON chronic childhood disease; annual treatment costs in the United States are estimated to be at least $4.5 billion.1,2 If left untreated, tooth decay can result in substantial morbidity due to pain, dysfunction, poor appearance, and possibly problems with speech development. In the past 50 years, probably as a result of dentifrices and increased levels of fluoride in drinking water, pediatric dental caries occurrence in the United States has declined dramatically; nevertheless, there remain important disparities in caries prevalence within the population.3,4 In particular, young children living in poverty constitute the subgroup in which there has been little progress in reduction of caries.5 Hence, it is especially important to identify modifiable risk factors for caries in this group.

Dental decay appears to be an infectious disease that involves colonization by Streptococcus mutans; these bacteria produce lactic acid from the fermentation of carbohydrates, and the acid dissolves the surface structure of teeth.6 However, why some children are particularly vulnerable to decay is poorly understood.7 We hypothesized that environmental tobacco smoke (ETS), a common cause of pediatric morbidity and mortality that disproportionately affects children in low socioeconomic status environments, may be a risk factor for cavities.8-13 Environmental tobacco smoke has previously been shown to be associated with periodontal disease in adults.14 Moreover, there are increasing experimental data supporting the biological plausibility of a causal role for ETS in caries formation. Research indicates that the bacteria responsible for caries formation are acquired in infancy from the saliva of mothers (via kissing, etc.).7 Elsewhere, nicotine has been shown to promote the growth of cariogenic S mutans bacteria in vitro; thus, mothers who smoke may be more likely than nonsmokers to transmit these germs to their children.15 Environmental tobacco smoke has immunosuppressive properties and is a known risk factor for infections of the cranial organs (eg, otitis media); thus it is not surprising that it would be a risk factor for caries development, which is an oral infectious disease.16,17 In addition, ETS is associated with decreased serum vitamin C levels in children and decreased levels of...
vitamin C are associated with growth of cariogenic bacteria.18,19 Also, it is possible that ETS may reduce the protective properties of saliva that operate against caries—saliva acts as a buffering agent when acids are produced, it physically removes debris from tooth surfaces, and it has immunological and bacteriostatic properties.20 Environmental tobacco smoke is known to increase inflammation of the respiratory tract, producing symptoms of various clinical conditions, including allergic rhinitis, which frequently cause mouth breathing and thus result in dry mouth (ie, an effective decrease in saliva).21 Thus, ETS could promote dental decay both through a direct effect of nicotine on caries-causing bacterial agents, as well as via other systemic physiological changes in the host.

The decline in caries among US children has occurred mainly in permanent and not deciduous teeth.7 If passive smoking does cause caries, there are reasons to expect the effect to be most pronounced in early childhood. Colonization with the cariogenic S mutans is thought to occur during a window of vulnerability around age 1 year, and primary teeth are particularly susceptible to caries formation soon after their eruption.6 One can also hypothesize specific mechanisms by which ETS exposure may be more likely to cause caries in deciduous rather than in permanent teeth. For example, it is known that the enamel in deciduous teeth is much thinner than in permanent teeth, and that enamel defects are associated with caries.22 Also, maternal smoking is a principal risk factor for prematurity, low birth weight, and chronic illness in infancy, while these in turn are all associated with generalized enamel hypoplasia in the primary dentition.5,10,23 Furthermore, in early childhood, when the immune system is generally less mature, the saliva is known to be different from that of adults with respect to IgA concentrations; in addition, salivary flow rate is lower.24 Young children may thus be particularly vulnerable to the harmful effects of ETS on the immune system and saliva flow. Hence, it is it biologically plausible that passive smoking could cause caries, particularly in early childhood.

The purpose of this study was to examine the association between passive smoking and dental caries by using data collected in the Third National Health and Nutrition Examination Survey (NHANES III), which includes a nationally representative sample of children who had both a complete dental examination and a serum cotinine level measurement, which is an objective, quantitative biomarker of ETS.

METHODS

NHANES III (1988-1994) is a cross-sectional survey that includes personal household interviews and health examinations for approximately 4000 children aged 4 to 11 years. The household interview collected demographic, socioeconomic, dietary, and health history information. The examination component consisted of physical and dental examinations and various laboratory tests using blood and urine specimens.

Only children aged 4 years or older had blood specimens for serum cotinine level taken, so younger children were excluded from these analyses. Children aged 12 years or older were excluded to reduce possible confusion resulting from early active smoking. For the same reason, we also excluded children with a cotinine level higher than 10 ng/mL. (only 23 children <12 years had a cotinine level >10 ng/mL). A licensed dentist performed the dental examination. Details of the methods used in the oral health component of the survey have been published previously.25-27 For children aged 4 to 11 years with at least 1 deciduous tooth, decayed and filled primary surfaces were analyzed. We analyzed filled and unfilled dental surfaces separately to help elucidate the interplay of access to dental care with environmental factors. The categories were not mutually exclusive: a child could have both filled and decayed (unfilled) surfaces. For persons aged 6 years or older with at least 1 permanent tooth, the permanent surfaces were analyzed. For this study, subjects were categorized as either having any caries or being caries-free.

The primary independent variable of interest was ETS exposure as reflected by a serum cotinine level of 0.2 ng/mL or greater, but less than 10 ng/mL. This cutoff approximately divides the non-smoking population into those with above or below median ETS exposure.11 For the dose-response analysis, serum cotinine level was divided into 4 categories. A cotinine level of less than 0.05 ng/mL was below the limit of detection; a level of 0.05 ng/mL to less than 0.2 ng/mL was considered low exposure; a level of 0.2 to 1.0 ng/mL, moderate exposure; and a level greater than 1, high exposure. Other potential independent associations with dental caries were analyzed, including the subjects’ age, sex, race, socioeconomic status, region of residence, education level of head of household, blood lead level, use of dental services, and sugar intake. Low socioeconomic status was defined as a poverty income ratio below 2. The poverty income ratio is the total family income divided by the federal poverty level for the year of the interview. This definition of poverty was used because, based on previous NHANES III studies, it is a good cutoff for pediatric caries risk.28 Use of oral health services was determined by the length of time since the last visit to a dentist. Caries formation may be related to prolonged exposure to carbohydrates, especially sucrose.29 As there is no recommended daily intake of candy, we defined high sugar intake as at or above the median of 45.3 g/d. NHANES III also includes variables for parental report of prenatal or postnatal smoking. We verified that these variables also demonstrated a significant association with caries, but restricted the analyses to the use of serum cotinine level as the measure of ETS exposure because it is objective and quantitative.

Bivariate (unadjusted) analyses were conducted to determine associations with caries status. Variables found to be statistically significant based on the \( \chi^2 \) test were included in logistic regression analyses to determine indepen-
dent associations with caries-free status. Performing the analyses with cotinine level as a continuous (as opposed to a categorical) variable did not substantively change the findings. Variables in the logistic regression models were screened for collinearity by examining correlation coefficients in bivariate analyses. SUDAAN software was used to account for the complex multistage sampling design of the survey. Sample weights were used to produce national estimates by adjusting for the oversampling of young children and older adults as well as for Mexican American and black individuals. P<.05 was the level of significance.

Population attributable risk estimates were derived using the Levin formula

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\text{Population Attributable Risk} = \frac{[\text{Prevalence} \times (\text{Relative Risk} - 1)]}{[1 + (\text{Prevalence} \times (\text{Relative Risk} - 1))]} \]

in which prevalence is the prevalence of exposure to the risk factor in the population. Relative risk is estimated from the observed odds ratio (OR) using the method of Zhang and Yu. The following prevalence data specific to this population sample were used in the population attributable risk calculations: ETS exposure of 53%, decayed primary surfaces in unexposed children of 18.2%, and filled surfaces of 29.2%.

**RESULTS**

A total of 3873 individuals aged 4 to 11 years were included in the analysis; of these, 3531 had at least 1 deciduous tooth and 2930 had at least 1 permanent tooth. Overall, 53.1% (n = 2057) of these children had cotinine levels consistent with ETS exposure, 46.7% (n = 1809) had decayed or filled surfaces in primary teeth, and 26.2% (n = 1015) had decayed or filled surfaces in permanent teeth.

In bivariate analyses, we examined the association of cotinine level and other potential risk factors with the occurrence of caries in deciduous and permanent teeth (Table 1). There were statistically significant associations between serum cotinine level and caries in deciduous teeth. The OR for decayed surfaces was 2.1 (95% confidence interval [CI], 1.5-2.9) and 1.4 (95% CI, 1.1-1.8) for filled surfaces. In addition to cotinine level, poverty and low level of education of household head were significantly associated with increased risk of both decayed and filled teeth. Black and Mexican American race/ethnicity and underuse of dental services were associated with more decayed surfaces but with significantly fewer fillings. Residence in the southern region of the United States increased the risk of decay, but not of fillings. Age was not related to the risk of decayed surfaces, but older children were more likely to have more fillings. High sugar intake and sex of the child were not correlated with higher risk for either decayed or filled surfaces. Elevated blood lead level correlated with significantly fewer fillings.

In permanent teeth, the association between cotinine level and caries was not statistically significant. The OR for decayed surfaces was 1.5 (95% CI, 0.9-2.2) and 1.1 (95% CI, 0.8-1.5) for filled surfaces. In addition to cotinine level, sex of the child, region of residence, sugar intake, and blood lead level were not significantly associated with either filled or unfilled surfaces. Poverty, low level of education of household head, and nonwhite race/ethnicity were significantly associated with increased risk of decayed but not filled surfaces. Black children and those with infrequent visits to the dentist were significantly more likely to have decay, but had significantly fewer fillings. Age was significantly related to the risk of both decayed and filled surfaces.

The logistic regression analyses adjusted for age, race/ethnicity, level of education of household head, poverty, blood lead level, days since last dental visit, and geographic region (Table 2). Sugar intake and sex were not included in the multivariate models since they were not significant factors in bivariate analyses. Testing for collinearity revealed no significant relationships between variables included in the logistic regression model. Cotinine level remained a significant predictor of caries in deciduous teeth. Adjusted OR for decayed surfaces was 1.8 (95% CI, 1.2-2.7) and 1.4 (95% CI, 1.1-2.0) for filled surfaces. Poverty was the only other variable that remained a significant independent risk factor for both decayed and filled surfaces. Underuse of dental services was significantly associated with more decayed surfaces and with fewer fillings. The association between low educational level of the household head and decayed or filled surfaces did not achieve statistical significance. Mexican American ethnicity was associated with more decayed surfaces, whereas black race was associated with a significantly decreased number of fillings. Residence in the West was weakly associated with more fillings but not with more decayed surfaces. Age was not related to the risk of decayed surfaces, but older children were more likely to have more fillings. Elevated blood lead level was not significantly related to decayed surfaces and was associated with a significant decrease in the risk of fillings. Analyzing for interactions revealed that the effect of tobacco was neither increased nor decreased by having an elevated blood lead level.

For permanent teeth, in the adjusted analyses, ETS was still not a significant risk factor for caries, nor were poverty, region of residence, or elevated blood lead level. Nonwhite children were significantly more likely to have decayed surfaces, but black children were significantly less likely to have fillings, as were children who had not seen a dentist in the past year. Low education level of household head was significantly associated with fillings. The likelihood of both decayed and filled surfaces increased with age.

For caries risk in deciduous teeth, there was evidence suggestive of a dose-response trend (Table 3). With serum cotinine level divided into 4 categories (rather than 2 as above), we found that in general, as ETS increased, so did the risk of caries. The trend for unfilled surfaces persisted after adjusting for the subjects’ age, race,
family income, geographic region of residence, education level of head of household, blood lead level, and dental service use across all 4 cotinine levels. For filled surfaces, the trend was suggested for the first 3 categories, but not for the highest cotinine level.

Based on the ORs reported in Table 2 for the association of risk between cotinine level and caries, the population attributable risk is estimated to be 27% for unfilled caries occurrence in deciduous teeth. In other words, a quarter of the children who have caries in deciduous teeth could be caries-free if ETS exposure were eliminated. For filled surfaces, the population attributable risk is 13.7%.

### Table 1. Distribution of Children by Study Variables and by Outcomes of Decayed and Filled Surfaces (Unadjusted Analysis)

<table>
<thead>
<tr>
<th>Deciduous Teeth</th>
<th>Permanent Teeth</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Unweighted No. of Subjects</strong></td>
<td><strong>Per cent</strong></td>
</tr>
<tr>
<td>Total population</td>
<td>3531</td>
</tr>
<tr>
<td>Serum cotinine level, ng/mL</td>
<td></td>
</tr>
<tr>
<td>&lt;0.2§</td>
<td>1575</td>
</tr>
<tr>
<td>0.2-10</td>
<td>1956</td>
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<tr>
<td>Poverty status, FPL %</td>
<td></td>
</tr>
<tr>
<td>&lt;200</td>
<td>2257</td>
</tr>
<tr>
<td>≥200§</td>
<td>1020</td>
</tr>
<tr>
<td>Education level of head of household</td>
<td></td>
</tr>
<tr>
<td>&lt;High school</td>
<td>1454</td>
</tr>
<tr>
<td>High school§</td>
<td>1103</td>
</tr>
<tr>
<td>Race/ethnicity</td>
<td></td>
</tr>
<tr>
<td>Non-Hispanic, white§</td>
<td>967</td>
</tr>
<tr>
<td>Non-Hispanic, black</td>
<td>1097</td>
</tr>
<tr>
<td>Mexican American</td>
<td>1303</td>
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<tr>
<td>Other</td>
<td>164</td>
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<tr>
<td>Region</td>
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</tr>
<tr>
<td>Northeast§</td>
<td>352</td>
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<tr>
<td>Midwest</td>
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<tr>
<td>West</td>
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</tr>
<tr>
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<td>Female§</td>
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<td>8-11§</td>
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<td>Time since last visit to dentist, d ≥365§</td>
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</tr>
<tr>
<td>&gt;365</td>
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<tr>
<td>Blood lead level, µg/dL</td>
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<tr>
<td>≥10</td>
<td>185</td>
</tr>
<tr>
<td>&lt;10§</td>
<td>3337</td>
</tr>
</tbody>
</table>

Abbreviation: FPL, Federal poverty level.

*Had at least 1 primary (deciduous) tooth. Totals of less than 3531 are due to missing observations.
†Values correspond to the χ² test for the difference in prevalence of caries by study variable category compared with the referent group for each variable.
‡Had at least 1 permanent tooth. Totals of less than 2930 are due to missing observations.
§Indicates referent group.
of an association between passive smoke exposure and dental caries in US children. Previous reports on the oral health of children in the United States have not considered ETS as a possible risk factor for caries. A literature search revealed only 1 other study specifically investigating the link between ETS and caries: it was conducted in the United Kingdom (UK National Diet and Nutrition Survey) and yielded results consistent with those we found in the United States. Our investigation has the additional strengths of using cotinine level, rather than subjective parental report, to estimate ETS exposure, and it studies a broader age range than the UK study, which included only 3- and 4-year-olds.

These data, in general, also suggest a dose-response relationship between children’s cotinine levels and the likelihood of having caries in deciduous teeth, even after controlling for multiple potential confounders. However, the relationship for unfilled cavities (decayed surfaces) leveled off at the highest quartile, and for the filled surfaces the risk decreased from the third to the fourth dose categories. It is possible that there is a threshold above which children’s risk for caries does not continue to increase further. In addition, there may be another explanation for filled surfaces. Filled status is determined not only by biological formation of caries, but also by subsequent dental care. Hence, the dose-response relationship between ETS and caries formation may be masked if families allow their children to be exposed to very high levels of ETS and also fail to take their children in for dental visits to get fillings. Further study of the dose-response relationship is warranted. While we are unable to explain why children with the highest cotinine levels do not appear to have a higher risk for caries on filled surfaces than children in the next highest cotinine category, and their risk of having caries is not statistically significant at \( P < .05 \) when compared with children with the lowest cotinine levels, their OR was in fact 1.5 and marginally significant at \( P < .07 \). It may be that there is a threshold of exposure to ETS above which children’s risk for caries does not continue to increase further.

We cannot demonstrate conclusively that passive smoking is causally linked to dental caries on the basis of
The observational data alone. The cross-sectional nature of the data also imposes limitations on the ability to test mechanism-specific hypotheses. For example, if ETS exposure at the time of enamel formation is the most relevant mechanism, ideally one would need to analyze cotinine levels during infancy. We did not have data on cotinine levels for children younger than 4 years, and do not know if current cotinine levels are good estimates of ETS exposure in infancy. Our finding that ETS was associated with caries in deciduous but not permanent teeth is consistent with the hypothesis that exposure in early life is important for the effect on dental health. The study results are consistent with what is known regarding the pathophysiological characteristics of caries formation and are therefore biologically plausible.

Nevertheless, the main difficulty with interpreting this study is the possibility that serum cotinine level is simply a marker for some unmeasured true cause of caries formation. For example, because smoking is an unhealthy behavior, it could be that parents who smoke also practice other unhealthy behaviors that would put their children at risk for cavities (e.g., not brushing teeth, not using fluoride toothpaste, not going to the dentist, eating too much candy, etc). Unfortunately, NHANES III does not include a variable for tooth brushing or other oral hygiene practices. We attempted to control for inadequate dental care by including a variable for use of dental services. Rarity of dental visits was a predictor of fewer fillings, but it was not related to decayed surfaces. Cotinine level remained a significant predictor of both decayed and filled surfaces, even after including this variable in the regression model. The finding that level of parental education dropped out of the model also argues against ETS being merely a marker for health behaviors associated with low socioeconomic status. Although there is a popular perception that cavities are caused primarily by excessive dietary sugar, our finding that sucrose intake was not related to caries prevalence is consistent with the scientific literature on the topic. Although public water supplies serve the same water to individuals with a range of tobacco exposure levels, one must also consider whether tobacco exposure is simply a marker for residence in an area that is not served by fluoridated drinking water, inasmuch as fluoride exposure is strongly linked to dental caries risk. However, ETS exposure is highest in urban areas, and that is where people are most likely to have fluoridated water. NHANES III does not provide information on fluoride exposure, so further study is needed to resolve this issue. Overall though, it appears that ETS is not simply a marker for poor dental health behaviors or for lack of fluoride.

In addition to the need for more comprehensive information on risk factors for caries, one of the limitations of this study is the age of the data. However, these are the most recent NHANES oral health data available, and there exists a general consensus that early childhood caries in poor children remains a major problem. For cotinine level though, there are more recent national data, and these indicate that despite reductions in smoking in the overall population, the proportion of US children exposed to ETS is still more than 50%. This is likely due to the ubiquity of tobacco smoke exposure in urban environments (even for children with nonsmoking parents), and the fact that children—especially preschool age children—are disproportionately affected by poverty. Thus it would appear that the findings presented here regarding dental decay, child poverty, and tobacco smoke exposure are, unfortunately, still relevant.

Small geographic differences in the prevalence of dental caries in US schoolchildren have been previously reported. In our unadjusted analysis, residence in the South was associated with higher caries prevalence, although the association was not significant in the multivariate model. Given that children in the South have an above average prevalence of exposure to ETS, these results are consistent with the hypothesis that passive smoking plays a role in the geographic variation in caries prevalence. However, NHANES III was not designed to account for seasonal variation in ETS exposure, and this may introduce measurement error in the assessment of regional patterns. For practical reasons, the mobile examination centers sampled individuals from the Northeast and Midwest during summer months and in the West and South during winter months. This arrangement tends to minimize observed cotinine levels (because people spend more time indoors in extreme cold or hot weather than at other times, and ETS exposure is higher indoors than outdoors).

Another bias in our study that would tend to classify exposed children as unexposed and thus reduce the observed ORs is the cotinine level cutoff of 0.2 ng/mL used to define ETS exposure. This is a conservative definition of the upper limit of nonexposure as it represents a level that is well above the limit of detection with the technique used in NHANES III (0.05 ng/mL), and it corresponds to a medium low exposure level, slightly lower than the median cotinine level among nonusers of tobacco. Indeed, the use of lower cutoffs in the dose-response analysis yielded higher ORs.

The association between poverty status and dental caries in the NHANES III data has been the topic of a previous study: as family income increased, children were less likely to have any decayed teeth. The finding that poverty remains a significant predictor of decay and fillings after controlling for ETS suggests that there are other unmeasured aspects of poverty that increase the formation of caries. In addition, poverty remains an important determinant of reduced access to dental care. The increased risk of caries in Mexican American children is consistent with previous reports. Black children, along with children with infrequent visits to the dentist, were significantly less likely to have fillings despite being more likely to experience decay—this suggests a gap in access to dental care for black children, which is also consistent with the recent literature.
Elevated blood lead level did not increase the risk of decay and was also significantly associated with decreased odds of having fillings. However, a previous study that found an association between blood lead level and caries used different methods that might be more sensitive for detecting an association with blood lead level. The relationship between blood lead level and caries requires further study.

These findings underscore the need for further investigation of risk factors for caries formation, especially in early childhood. Future studies of the biological effects of ETS and other pollutants should examine oral health outcomes, such as caries. If a causal association between passive smoking and dental caries is substantiated, then health interventions for dental caries should include strategies to reduce ETS exposure. These results provide one more piece of evidence indicating that passive smoking is harmful and that all children should be allowed to grow up in a smoke-free environment.

REFERENCES


