# Blood Lead Level and Dental Caries in School-Age Children

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**The association between blood lead level and dental caries was evaluated in cross-sectional analyses of baseline data for 543 children 6–10 years old screened for enrollment in the Children's Amalgam Trial, a study designed to assess potential health effects of mercury in silver fillings. Approximately half of the children were recruited from an urban setting (Boston/Cambridge, MA, USA) and approximately half from a rural setting (Farmington, ME, USA). Mean blood lead level was significantly greater among the urban subgroup, as was the mean number of carious tooth surfaces. Blood lead level was positively associated with number of caries among urban children, even with adjustment for demographic and maternal factors and child dental practices. This association was stronger in primary than in permanent dentition and stronger for occlusal, lingual, and buccal tooth surfaces than for mesial or distal surfaces. In general, blood lead was not associated with caries in the rural subgroup. The difference between the strength of the associations in the urban and rural settings might reflect the presence of residual confounding in the former setting, the presence of greater variability in the latter setting in terms of important caries risk factors (e.g., fluoride exposure), or greater exposure misclassification in the rural setting. These findings add to the evidence supporting a weak association between children's lead exposure and caries prevalence. A biologic mechanism for lead cariogenicity has not been identified, however. Our data are also consistent with residual confounding by factors associated with both elevated lead exposure and dental caries.** *Key words:* **blood lead, dental caries, epidemiology, tooth, toxicology.**
*Environ Health Perspect* **110:A625–A630 (2002). [Online 16 September 2002]**
***http://ehpnet1.niehs.nih.gov/docs/2002/110pA625-A630gemmel/abstract.html***

More than 40% of children develop caries in primary dentition by 6 years of age, and more than 85% develop caries in the permanent dentition by age 17 (Kaste et al. 1996). Despite a reduction in the prevalence of caries in the permanent dentition, the prevalence in the primary dentition has remained essentially unchanged, particularly among low-income children (Caufield and Griffen 2000). Caries is regarded as an infectious disease, the result of a multifactorial process involving three elements: particular oral microflora and dietary exposures as well as a susceptible host (Schafer and Adair 2000). Although *Mutans streptococci* are the critical cariogenic bacteria in the oral cavity, and fermentable carbohydrates (e.g., refined sugars) the critical dietary factor, the determinants of variability in host susceptibility are less certain. Structurally sound enamel and adequate salivary flow and composition are clearly important (Schafer and Adair 2000). Several sociodemographic correlates of increased risk have been identified as risk factors for both caries incidence and treatment, including living in poverty and being African American or Mexican American. These are presumably surrogate markers for key health-related behaviors, environmental exposures, and access to medical care (Kaste et al. 1996; Vargas et al. 1998).

The epidemiology of dental caries in children overlaps considerably with the epidemiology of lead poisoning, which is primarily a disease of inner-city poor children (Brody et al. 1994). Pediatric lead poisoning is associated with an increased risk of adverse effects in a variety of target organs, with the central nervous, hematopoietic, and renal systems receiving the greatest attention [National Research Council 1993; U.S. Centers for Disease Control and Prevention (CDC) 1991]. Because lead is a divalent cation, its metabolism is affected by the same factors that affect calcium metabolism, and lead's tendency to "follow the calcium stream" was recognized decades ago (Aub et al. 1925). Mineralized tissues are thus long-term storage sites for lead (Drasch et al. 1987; Drasch and Ott 1988). Because deciduous teeth provide a readily accessible bone biopsy, the concentrations of lead in the whole primary teeth, the enamel, or the dentin (particularly circumpulpal) have served as proxy measures for skeletal lead, and thus for total body lead burden, in epidemiologic studies of childhood lead toxicity (Needleman et al. 1979; Rabinowitz et al. 1991; Smith et al. 1983; Winneke et al. 1983).

Bone is not simply a physiologic "sink" for lead, however, and might itself be a target organ for lead toxicity, although little is known about either the form that this toxicity might take or its mechanism (Pounds et al. 1991). Some findings are consistent with an adverse effect of lead on odontoblast function (Appleton 1991). The risk of hypoplastic enamel defects (i.e., defective matrix formation) in primary teeth is increased among children who were never screened for lead poisoning (Needleman et al. 1992), and higher postnatal lead exposure is associated with pitting hypoplasia of permanent teeth (Lawson et al. 1971). In the rat, increased maternal lead exposure produces a decrease in stimulated parotid function, suggesting salivary hypofunction, and a concomitant increase in dental caries in offspring (Watson et al. 1997). An association between increased lead levels and increased caries prevalence has been reported in several epidemiologic studies (Brudevold et al. 1977; Gil et al. 1994, 1996; Moss et al. 1999). In a study of 251 children 9–12 years old, Brudevold et al. (1977) found that children with higher levels of enamel lead had more caries than did children with lower enamel lead levels. Among 25,000 participants in the Third National Health and Nutrition Examination Survey (NHANES III), an association was reported between blood lead level and the risk of carious lesions in teeth (Moss et al. 1999). Among children (5–17 years old), an increase of 5 µg/dL in blood lead level was associated with nearly a doubling of the risk of dental caries, and the population-attributable risk associated with blood lead levels in the middle tertile of the distribution was nearly 10%. In a study of 248 school-age children, having a blood lead level greater than 10 µg/dL, measured between 18 and 37 months of age, was modestly associated with an increased risk of caries, but overall the data failed to support any of the hypotheses regarding possible mechanisms of lead cariogenicity (Campbell et al. 2000). In the analyses reported by both Campbell et al. (2000) and Moss et al. (1999), adjustments were made for a variety of potentially confounding factors, including

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Analyses of these data were supported by a cooperative agreement (U01 DE11886) between the New England Research Institutes and the National Institute of Dental and Craniofacial Research.

Received 6 March 2002; accepted 24 May 2002.

socioeconomic status, ethnicity, and geographic region.

In conducting a randomized trial comparing the health effects in children of different dental restorative materials (i.e., the use of mercury-containing "silver" amalgam vs. composite resins to restore damage to enamel caused by dental caries), we had the opportunity to collect additional data on the possible association between lead exposure and the prevalence of dental caries in the participants. In particular, we compared the strength of the putative association in an urban versus a rural setting, and among children with blood lead levels that were largely within the range presently considered to be acceptable (i.e., < 10 μg/dL; CDC 1991).

## Methods

***Sample.*** Children were recruited into the Children's Amalgam Trial (CAT) from two geographic areas in the United States, one urban (Boston/Cambridge, MA) and one rural (Farmington, ME). Eligible children were identified in school-based screenings using the following criteria: 6–10 years of age, no prior amalgam restorations, two or more posterior teeth with caries such that the resulting restorations would include the occlusal (i.e., chewing) surfaces, English speaking, and no major health disorders with neuropsychologic correlates. The screening dental examinations were performed by a dentist or a dental hygienist. They entailed visual, not tactile, examination, using a dental mouth mirror, a portable light for illumination, and a tongue blade to facilitate visual access during inspection. Dental explorers (instruments) and radiographs were not used.

Of 5,116 children screened, 645 met eligibility criteria, and 534 were randomly allocated to one of the two treatment arms, amalgam restoration or composite restoration, with assignment stratified by site and number of caries (2–4 vs. ≥ 5).

A baseline clinical dental examination was conducted by a licensed pediatric dentist, involving radiographic confirmation of carious surfaces, cleaning, application of fluoride treatments and sealants, and restoration of caries according to treatment assignment. The analyses presented are based on the number of carious surfaces identified by means of the baseline clinical dental examination.

Informed consent and assent were obtained by the pediatric dentist when it was determined that a child met all eligibility criteria. The complete schedule of treatments, assessments, and sample collection specified in the CAT protocol were explained at this time.

***Data collection.*** At the time of the baseline clinical dental examination, a child's parent was interviewed regarding family demographics and dental practices, and a pediatric phlebotomist drew a blood sample from the child by venipuncture into a heparinized Vacutainer. Aliquots were sent to a laboratory approved by the Occupational Safety and Health Administration for lead determination. Samples, blood-based quality control materials, and aqueous standards were diluted with a matrix modifier solution containing nitric acid, Triton X-100, and ammonium dihydrogen phosphate. Lead concentration was measured using electrothermal atomization atomic absorption spectrometry with Zeeman background correction.

***Statistical analysis.*** The primary end point was the natural log-transformed number of decayed and filled surfaces (lnDFS), and the primary independent variable was the natural log-transformed blood lead level (lnBPb). This transformation was applied to these variables to normalize their distributions. Linear regression was used to estimate the association between lnBPb and lnDFS. We evaluated a site × lnBPb term to determine whether the association differed significantly between the urban and rural subgroups. In multiple regression analyses, we adjusted for factors that previous studies (Campbell et al. 2000; Moss et al. 1999) had suggested are plausible confounders of an association between lead and caries, as well as for factors that tend to predict caries occurrence: age, sex (0 = female, 1 = male), family income (six categories, $10,000 increments beginning with ≤ $10,000), maternal education (less than high school, high school or high school graduate equivalence degree, greater than high school), maternal smoking (0 = no, 1 = yes), frequency of tooth brushing (less than once a day, once a day, more than once a day), use of medium/hard bristles (0 = no, 1 = yes), and gum chewing (never, occasionally, daily). In regressions performed on the urban subgroup, we also adjusted for self-identified ethnicity (white, black, other/mixed). (The other/mixed group included Hispanic, Asian/Pacific Islander, Native

**Table 1.** Demographic characteristics, blood lead levels, and caries prevalence.

| Variable | Boston/Cambridge (*n* = 290) | Farmington (*n* = 253) | Combined sites (*n* = 543) |
|---|---|---|---|
| Age (years, mean ± SD) | 7.9 ± 1.3 | 7.9 ± 1.5 | 7.9 ± 1.4 |
| Sex (% male) | 48 | 43 | 46 |
| Ethnicity (%) | | | |
| White | 30 | 98 | 62 |
| Black | 36 | 0.4 | 19 |
| Hispanic | 15 | 0 | 8 |
| Other/mixed | 19 | 1.6 | 9 |
| Income (%) | | | |
| ≤ $10,000 | 13 | 9 | 11 |
| $10,001–20,000 | 23 | 18 | 21 |
| $20,001–30,000 | 24 | 24 | 24 |
| $30,001–40,000 | 14 | 24 | 19 |
| $40,001–50,000 | 14 | 10 | 12 |
| > $50,000 | 11 | 14 | 13 |
| Education of female guardian (%) | | | |
| Less than high school | 20 | 6 | 13 |
| High school diploma | 33 | 55 | 43 |
| Some college/vocational | 25 | 27 | 26 |
| Associate degree | 7 | 7 | 7 |
| College graduate | 11 | 3 | 7 |
| Graduate degree | 4 | 2 | 3 |
| Maternal smoking (% current) | 35 | 41 | 38 |
| Frequency of tooth brushing (%) | | | |
| Less than once per day | 5 | 9 | 7 |
| Once per day | 40 | 47 | 43 |
| More than once per day | 55 | 44 | 50 |
| Use of medium/hard bristles (%) | 39 | 32 | 36 |
| Gum chewing (%) | | | |
| Never | 11 | 5 | 8 |
| Occasionally | 77 | 86 | 81 |
| Daily | 13 | 8 | 11 |
| Blood lead level at enrollment (μg/dL, mean ± standard deviation) | 2.9 ± 2.0 | 1.7 ± 1.0 | 2.3 ± 1.7 |
| No. carious surfaces mean ± SD (range) | | | |
| All dentition | 10.7 ± 7.3 (2–42) | 8.9 ± 5.9 (2–38) | 9.8 ± 6.8 (2–42) |
| Primary dentition | 8.5 ± 7.1 (0–42) | 7.4 ± 5.8 (0–34) | 8.0 ± 6.5 (0–42) |
| Permanent dentition | 2.1 ± 2.3 (0–12) | 1.5 ± 2.5 (0–14) | 1.8 ± 2.4 (0–14) |
| Occlusal surfaces | 5.2 ± 2.8 (0–12) | 4.4 ± 2.4 (1–11) | 4.8 ± 2.7 (0–12) |
| Lingual surfaces | 1.1 ± 1.4 (0–9) | 0.7 ± 1.1 (0–5) | 0.9 ± 1.3 (0–9) |
| Buccal surfaces | 1.2 ± 1.6 (0–9) | 0.7 ± 1.1 (0–6) | 1.0 ± 1.4 (0–9) |
| Mesial surfaces | 1.5 ± 1.8 (0–11) | 1.2 ± 1.5 (0–10) | 1.4 ± 1.7 (0–11) |
| Distal surfaces | 1.7 ± 1.8 (0–8) | 1.8 ± 1.8 (0–9) | 1.8 ± 1.8 (0–9) |

American, biracial/multiracial, and other). This was not necessary in analyses performed on the rural subgroup because of its ethnic homogeneity (98% white). Ancillary analyses evaluated the association between lnBPb and lnDFS, stratifying by carious tooth type (primary vs. permanent) and surface: occlusal (chewing surfaces), lingual (surface adjacent to the tongue), buccal (surface adjacent to cheek), mesial (surface toward the median sagittal plane of the face), distal (surface away from the median sagittal plane of the face).

Blood lead and caries data were available for 543 children. Because data on covariates were missing for some children, the sample size for the multivariable analyses was 498 (259 in Boston/Cambridge, 239 in Farmington).

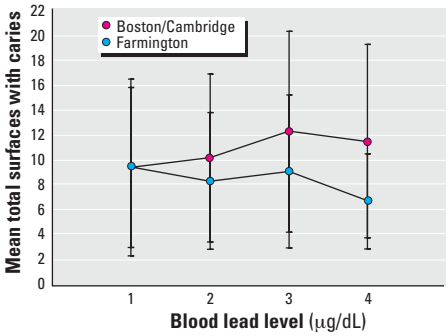


**Figure 1.** Mean number of carious surfaces among children with blood lead levels of 1, 2, 3, or ≥ 4 µg/dL, separately by site (Boston/Cambridge vs. Farmington). Error bars represent 1 standard deviation.

## Results

Table 1 shows the demographic characteristics of the sample as a whole and stratified by geographic site. As noted above, the two sites differed considerably in ethnic composition, with the rural subgroup being almost exclusively white and the urban subgroup being more heterogeneous. Family income and female guardian educational levels tended to be higher in the rural subgroup. Sites also differed in distribution of primary water sources. Almost all urban children (96%) currently resided in communities in which the municipal water supply was fluoridated. In contrast, for 85% of rural children, unfluoridated well water was the primary source of a family's drinking water. Measurements of the naturally occurring levels of fluoride in well water were not made, however.

The mean number of tooth surfaces with carious lesions was significantly greater in the urban subgroup than in the rural subgroup ($p$ = 0.002). This was true for both primary teeth ($p$ = 0.047) and permanent teeth ($p$ = 0.0009).

Blood lead levels were generally low, with an overall mean of 2.3 µg/dL (SD, 1.7), although it was significantly higher in the urban than in the rural subgroup ($p$ < 0.0001). The maximum blood lead levels were 13 and 7 µg/dL in the urban and rural subgroups, respectively. The lead exposure of this study sample was comparable with that of the general population: In the most recent NHANES survey, the mean blood lead level of 6–11 year olds was 2.5 µg/dL (95% confidence interval: 2.2–2.7; Brody et al. 1994).

In the complete cohort, lnBPb and lnDFS were marginally positively associated ($p$ = 0.06). Because the lnBPb × lnDFS interaction term was statistically significant ($p$ = 0.001), however, further analyses were stratified by geographic site. In the urban subgroup, lnDFS and lnBPb were positively associated ($p$ = 0.02). In the rural subgroup, the slope of the relationship was negative, although not significant ($p$ = 0.12). Figure 1 shows the mean values for the total number of carious surfaces for children with blood lead levels of 1, 2, 3, and ≥ 4 µg/dL, by site. In the urban subgroup, children with blood lead levels of ≥ 4 µg/dL had, on average, two more carious surfaces than did children with a blood lead level of 1 µg/dL, an increase of approximately 20%.

In simultaneous multiple regression analyses adjusting for age, sex, family income, ethnicity (urban subgroup only), education of a child's female guardian, maternal smoking, frequency of a child tooth brushing, the firmness of the toothbrush bristles, and the frequency of gum chewing, the positive association between lnBPb and lnDFS in the urban subgroup remained significant ($p$ = 0.005; Table 2). The only other significant predictor of lnDFS in this subgroup was ethnicity, with children in the black group having fewer carious surfaces than did children in the other/mixed group. In the rural subgroup, the negative association between lnBPb and lnDFS, adjusted for covariates, was not significant ($p$ = 0.09). Among this subgroup, lnDFS was inversely related to child age ($p$ = 0.03), and children of mothers with a high school education had more carious surfaces than did children of mothers with at least some college ($p$ = 0.01).

To determine whether the association between blood lead and caries prevalence is similar for primary and permanent teeth, the full regression model was refitted, stratifying the analyses by site (urban, rural) and tooth type (primary, Table 3; permanent, Table 4). Among children in the urban subgroup, lnBPb and lnDFS were positively associated in primary teeth ($p$ = 0.002) but not in permanent teeth ($p$ = 0.8). Among children in the rural subgroup, lnBPb and lnDFS were not significantly associated in either primary teeth ($p$ = 0.1) or permanent teeth ($p$ = 0.3). In both subgroups, age was inversely associated with lnDFS in primary teeth ($p$ = 0.0003 in the urban subgroup, $p$ = 0.0001 in the rural subgroup) and positively associated with lnDFS in permanent teeth ($p$ = 0.0001 in both urban and rural subgroups). For primary teeth, lnDFS was lower among children in the black category than among children in the other/mixed category in the urban

**Table 2.** Simultaneous multiple regression models of number of carious surfaces (log), stratified by site.

| | Boston/Cambridge | | | Farmington | | |
|---|---|---|---|---|---|---|
| Variable | Coefficient | SE | *p*-Value | Coefficient | SE | *p*-Value |
| Blood lead level (µg/dL) (log) | 0.22 | 0.08 | 0.005 | –0.15 | 0.09 | 0.09 |
| Age (years) | –0.22 | 0.04 | 0.5 | –0.07 | 0.03 | 0.03 |
| Sex (0 = female, 1 = male) | 0.07 | 0.10 | 0.5 | –0.11 | 0.09 | 0.2 |
| Income | | | | | | |
| ≤ $10,000 | 0.02 | 0.22 | 0.9 | –0.17 | 0.18 | 0.4 |
| $10,001–20,000 | –0.23 | 0.18 | 0.2 | –0.17 | 0.15 | 0.3 |
| $20,001–30,000 | –0.20 | 0.18 | 0.3 | 0.05 | 0.15 | 0.7 |
| $30,001–40,000 | –0.14 | 0.20 | 0.6 | –0.10 | 0.15 | 0.5 |
| $40,001–50,000 | –0.05 | 0.20 | 0.8 | –0.15 | 0.18 | 0.4 |
| > $50,000 | Reference category | | | Reference category | | |
| Education of female guardian | | | | | | |
| Less than high school | –0.22 | 0.14 | 0.1 | 0.17 | 0.20 | 0.4 |
| High school diploma | –0.03 | 0.12 | 0.8 | 0.24 | 0.09 | 0.01 |
| At least some college | Reference category | | | Reference category | | |
| Ethnicity | | | | Not estimated | | |
| White | –0.16 | 0.14 | 0.2 | | | |
| Black | –0.29 | 0.12 | 0.02 | | | |
| Other/mixed | Reference category | | | | | |
| Maternal smoking (0 = no, 1 = yes) | –0.15 | 0.12 | 0.2 | 0.01 | 0.09 | 0.9 |
| Frequency of tooth brushing | | | | | | |
| Less than once per day | 0.12 | 0.24 | 0.6 | 0.14 | 0.17 | 0.4 |
| Once per day | –0.15 | 0.10 | 0.1 | –0.04 | 0.09 | 0.6 |
| More than once per day | Reference category | | | Reference category | | |
| Use of medium/hard bristles (0 = no, 1 = yes) | 0.02 | 0.10 | 0.8 | 0.16 | 0.09 | 0.1 |
| Gum chewing | | | | | | |
| Never | –0.06 | 0.20 | 0.8 | 0.06 | 0.25 | 0.8 |
| Occasionally | 0.07 | 0.15 | 0.6 | 0.07 | 0.16 | 0.7 |
| Daily | Reference category | | | Reference category | | |

subgroup ($p$ = 0.009). In the rural subgroup, children of mothers with a high school education had significantly higher lnDFS than did children of mothers with greater education ($p$ = 0.0003). In the rural subgroup, lnDFS in permanent teeth was inversely associated with age ($p$ = 0.05) and with use of medium/hard bristles compared with soft bristles ($p$ = 0.04).

The association between lnBPb and lnDFS was then evaluated separately by tooth surface (occlusal, lingual, buccal, mesial, distal). Adjusting for covariates and combining sites and tooth types, lnBPb was significantly associated with the lnDFS on occlusal ($p$ = 0.04), lingual ($p$ = 0.04), and buccal ($p$ = 0.03) but not mesial ($p$ = 0.4) or distal ($p$ = 0.7) surfaces. Additional analyses stratified by site suggested that these associations were more striking in the urban than in the rural subgroup.

## Discussion

In cross-sectional analyses, the blood lead levels of 6–10-year-old children at the time of their recruitment into the CAT were weakly associated with the number of carious tooth surfaces. This conclusion must be qualified in several ways. First, the association was apparent among the subgroup of children recruited from the Boston/Cambridge urban area, but not among children recruited from rural Farmington, Maine. Even in the urban subgroup, however, the magnitude of the association was modest, with blood lead level accounting for only 2% of the variance in the number of carious surfaces. Second, in the urban subgroup, the blood lead level was associated with number of carious surfaces in primary but not in permanent teeth. This is consistent with the findings of Campbell et al. (2000). Furthermore, the association was evident for several tooth surfaces: occlusal, lingual, and buccal tooth surfaces. As noted by Campbell et al. (2000), if the causal mechanism of lead's cariogenicity were a lead-related decrease in salivary flow, as suggested by experimental studies in the rat (Watson et al. 1997), buccal surfaces should be more affected than lingual surfaces. Thus, our data are not consistent with this hypothesized mechanism.

Lead cariogenicity might reflect a direct effect of lead on the integrity of tooth enamel rather than a secondary effect mediated by reduced salivary flow. Parotid saliva secretion is an excretory route for lead (DiGregorio et al. 1974), most likely by a passive diffusion process. Salivary lead concentration is correlated with whole blood lead concentration but is much lower in magnitude (Mobarak and P'an 1984; P'an 1981), has a half-life less than 25% as long as whole blood lead, and falls more rapidly than does blood lead concentration after exposure stops (Brodeur et al. 1983). This suggests that salivary lead level reflects the highly diffusible fraction of lead in plasma rather than the much larger fraction that is bound to red cells (Cleymaet et al. 1991). Unlike dentin lead (Rabinowitz et al. 1993), enamel lead is not turned over (Gulson and Gillings 1997), and the concentration is inversely related to etch depth (Cleymaet et al. 1991; Purchase and Fergusson 1986). In contrast, for other tooth compartments the concentration of lead declines with increasing distance from the pulp cavity (Grobler et al. 2000; Gulson 1996; Gulson and Gillings 1997; Purchase and Fergusson 1986), that is, where a tooth is in contact with the circulation. Finally, much of the intersurface and intertooth variability in enamel lead concentration can be accounted for by differences in the amount of contact with saliva and plaque, suggesting that lead in saliva is sorbed onto the surface of a tooth and incorporated into the hydroxyapatite, perhaps replacing calcium (Purchase and Fergusson 1986). If so, the critical variable for caries formation would be the lead concentration in saliva rather than a lead-induced reduction in salivary flow.

It is not clear why an association between blood lead level and number of caries was evident in the urban but not in the rural subgroup. It is unlikely that the underlying biologic basis of an influence of lead on the development of caries differs according to geographic setting. The association in the urban subgroup might therefore represent a spurious association attributable to the presence of residual confounding in this subgroup by some third factor that is not present in the rural subgroup. The monotonicity of the observed dose–effect relationship in the urban subgroup argues against this hypothesis, however, because it would require that blood lead level and the third factor be associated in a similarly monotonic manner. Furthermore, in experimental studies involving rodents, a setting in which residual confounding of exposure by other caries risk factors is unlikely, the administration of lead produces caries (Watson et al. 1997).

The absence of an association in the rural subgroup might reflect the effect-modifying role that is postulated for environmental factors, such as lead exposure, in the pathogenesis of caries. As noted in the introductory remarks, the presence of particular microflora in the oral cavity and fermentable carbohydrates in the diet is viewed as the critical causal factors, with other factors modulating host susceptibility. The ability to detect a main effect on caries for a factor that is a modulator of risk will be enhanced when the prevalences of the critical factors, or other important risk modulators, are near 100%. Otherwise, the main determinants of caries incidence will be the presence or absence of the critical factors. Although *Mutans streptococci* are ubiquitous, a regional difference in their presence is possible. Exposure to fermentable carbohydrates might differ

**Table 3.** Simultaneous multiple regression models of number of carious surfaces (log) on primary teeth, stratified by site.

| | Boston/Cambridge | | | Farmington | | |
|---|---|---|---|---|---|---|
| Variable | Coefficient | SE | *p*-Value | Coefficient | SE | *p*-Value |
| Blood lead level (µg/dL) (log) | 0.28 | 0.09 | 0.002 | –0.15 | 0.09 | 0.1 |
| Age (years) | –0.16 | 0.04 | 0.0003 | –0.19 | 0.03 | 0.0001 |
| Sex (0 = female, 1 = male) | –0.02 | 0.11 | 0.9 | –0.09 | 0.09 | 0.4 |
| Income | | | | | | |
| ≤ $10,000 | –0.19 | 0.25 | 0.5 | –0.29 | 0.19 | 0.1 |
| $10,001–20,000 | –0.37 | 0.21 | 0.08 | –0.09 | 0.16 | 0.6 |
| $20,001–30,000 | –0.35 | 0.20 | 0.09 | 0.02 | 0.15 | 0.9 |
| $30,001–40,000 | –0.28 | 0.22 | 0.2 | –0.18 | 0.16 | 0.2 |
| $40,001–50,000 | –0.30 | 0.22 | 0.2 | –0.21 | 0.19 | 0.3 |
| > $50,000 | Reference category | | | Reference category | | |
| Education of female guardian | | | | | | |
| Less than high school | –0.18 | 0.16 | 0.3 | –0.07 | 0.21 | 0.7 |
| High school diploma | –0.01 | 0.13 | 0.9 | 0.35 | 0.10 | 0.0003 |
| At least some college | Reference category | | | Reference category | | |
| Ethnicity | | | | Not estimated | | |
| White | –0.23 | 0.16 | 0.1 | | | |
| Black | –0.36 | 0.14 | 0.009 | | | |
| Other/mixed | Reference category | | | | | |
| Maternal smoking (0 = no, 1 = yes) | –0.20 | 0.13 | 0.1 | 0.05 | 0.10 | 0.6 |
| Frequency of tooth brushing | | | | | | |
| Less than once per day | 0.09 | 0.27 | 0.7 | 0.04 | 0.18 | 0.8 |
| Once per day | –0.17 | 0.11 | 0.1 | –0.13 | 0.10 | 0.2 |
| More than once per day | Reference category | | | Reference category | | |
| Use of medium/hard bristles (0 = no, 1 = yes) | 0.03 | 0.11 | 0.8 | 0.15 | 0.10 | 0.1 |
| Gum chewing | | | | | | |
| Never | –0.07 | 0.23 | 0.8 | 0.19 | 0.26 | 0.5 |
| Occasionally | 0.09 | 0.17 | 0.6 | 0.04 | 0.17 | 0.8 |
| Daily | Reference category | | | Reference category | | |

between the urban and rural subgroups because of regional differences in dietary practices and preferences. Although we did not collect the information needed to evaluate this possibility directly, diet did vary significantly between sites in some respects (e.g., consumption of fruits, vegetables, and fish), making this a reasonable hypothesis for further consideration.

An important caries risk factor whose prevalence clearly differed between sites is exposure to fluoridated water. Whereas 96% of children in the urban subgroup resided in communities that fluoridate the municipal water supply, the families of most children in the rural subgroup (85%) relied on well water as the primary source of drinking water. Because the abundance of fluorine minerals varies with the local geology (e.g., presence of granitic rocks such as pegmatites), greater variation would be expected to occur in the fluoride levels in water drawn from wells than in the levels in water supplemented to the recommended target concentration of 0.7–1.2 ppm (CDC 2001). On this basis, it would be expected that the major determinant of variability in caries in the rural subgroup, but not in the urban subgroup, would be fluoride intake. In the urban subgroup, where fluoride exposure was likely to be reasonably consistent across children, the contributions of weaker determinants of caries, such as lead exposure, might present a "signal" that is sufficiently clear to be detected. The finding that caries were significantly more common among children in the urban subgroup seems counterintuitive, however, given their presumed greater exposure to fluoride. This suggests that the distributions of important caries risk factors other than fluoride exposure differed substantially between sites, or that water in the Farmington, Maine, area has high levels of natural fluoride. This latter possibility appears not to be true. Although data on fluoride concentrations in groundwater in Farmington are not available, the median value in 120 samples collected statewide was 0.36 ppm, with 75% of values below 0.73 ppm, the lower bound of the target range for fluoridation (Loiselle MC, Department of Conservation, Maine Geological Survey, Ambient Bedrock Groundwater Quality. Unpublished data).

The recently reported association between use of silicofluorides ($Na_2SiF_6$ or $H_2SiF_6$) as water fluoridants and the prevalence of elevated blood lead levels (Masters et al. 2000) might explain, in part, the stronger association between blood lead levels and caries in the urban than in the rural subgroup, because the water supplies of Boston and Cambridge are treated with fluorosilicic acid (Foley M, Office of Oral Health, Massachusetts Department of Public Health. Personal Communication). Although the mechanism by which exposure to silicofluorides might increase lead absorption is unknown, higher enamel lead concentrations in children have been associated with both higher enamel fluoride concentrations and greater caries (Brudevold et al. 1977). Exploration of the possible metabolic interactions between these two bone-seeking minerals is thus a research need.

Even if the association noted in the urban subgroup between lead level and caries prevalence is causal, caution should be exercised in drawing inferences about quantitative aspects of the dose–effect relationship. First, if lead does play a causal role in the pathogenesis of caries, it is likely to be lead exposure in the early years that is responsible, insofar as enamel appears to be relatively inert to lead (and other bone-seeking elements) after tooth eruption (Brudevold et al. 1977). This renders important the issue of how well the blood lead levels measured when the children entered the CAT study at 6–10 years old reflected their blood lead levels in the first few years of life. Lead in blood has a relatively short exposure averaging time, with a 3–4-week half-life in adults for movement of lead from blood (Rabinowitz et al. 1976). This process might take somewhat longer in children, especially those who incur relatively heavy exposures (Succop et al. 1987; Manton et al. 2000). The intraindividual stability is low, however, among children with levels of exposure similar to those of the children in the CAT (Rabinowitz et al. 1984). Thus, the blood lead levels measured at the time of entry into the CAT are likely to be relatively poor measures of the blood lead levels that existed at the critical time of tooth development. Because children's blood lead levels tend to peak at 2–3 years of age and decline thereafter (Dietrich et al. 1993; McMichael et al. 1985), the blood lead levels measured at enrollment in the CAT are likely to underestimate the earlier lead doses incurred by the children. Our findings thus suggest a need to evaluate the association between lead exposure and caries, and dental health more generally, using a prospective study design, in which more complete lead exposure histories can be assembled in order to clarify this issue. Second, the caries distribution was somewhat skewed by the eligibility criteria of the CAT study that required a child to have two or more posterior teeth with caries such that restoration would include occlusal surfaces. The estimated dose–effect relationship might have been somewhat different had children with fewer caries contributed data to the analyses.

In summary, our findings are consistent with those of several other recent studies (e.g., Campbell et al. 2000; Moss et al. 1999) in suggesting a weak association between children's lead exposure and caries in primary teeth. The association was region specific, however, suggesting that its magnitude depends on the local distributions of other, more important caries risk factors such as fluoride exposure, diet, and other aspects of

**Table 4.** Simultaneous multiple regression models of number of carious surfaces (log) on permanent teeth, stratified by site.

| | Boston/Cambridge | | | Farmington | | |
|---|---|---|---|---|---|---|
| Variable | Coefficient | SE | *p*-Value | Coefficient | SE | *p*-Value |
| Blood lead level (µg/dL) (log) | 0.02 | 0.07 | 0.8 | –0.10 | 0.10 | 0.3 |
| Age (years) | 0.26 | 0.03 | 0.0001 | 0.21 | 0.04 | 0.0001 |
| Sex (0 = female, 1 = male) | 0.14 | 0.09 | 0.1 | –0.20 | 0.10 | 0.05 |
| Income | | | | | | |
| ≤ $10,000 | 0.16 | 0.20 | 0.4 | –0.02 | 0.21 | 0.9 |
| $10,001–20,000 | 0.08 | 0.16 | 0.6 | –0.03 | 0.18 | 0.9 |
| $20,001–30,000 | 0.12 | 0.16 | 0.5 | 0.04 | 0.16 | 0.8 |
| $30,001–40,000 | –0.09 | 0.18 | 0.6 | 0.04 | 0.17 | 0.8 |
| $40,001–50,000 | 0.19 | 0.18 | 0.3 | –0.19 | 0.21 | 0.4 |
| > $50,000 | Reference category | | | Reference category | | |
| Education of female guardian | | | | | | |
| Less than high school | –0.10 | 0.13 | 0.6 | 0.05 | 0.22 | 0.8 |
| High school diploma | –0.06 | 0.11 | 0.5 | –0.08 | 0.10 | 0.5 |
| At least some college | Reference category | | | Reference category | | |
| Ethnicity | | | | Not estimated | | |
| White | –0.15 | 0.13 | 0.2 | | | |
| Black | 0.02 | 0.11 | 0.9 | | | |
| Other/mixed | Reference category | | | | | |
| Maternal smoking (0 = no, 1 = yes) | –0.01 | 0.11 | 0.9 | –0.11 | 0.10 | 0.3 |
| Frequency of tooth brushing | | | | | | |
| Less than once per day | 0.24 | 0.21 | 0.2 | 0.21 | 0.19 | 0.3 |
| Once per day | 0.06 | 0.09 | 0.5 | 0.15 | 0.10 | 0.1 |
| More than once per day | Reference category | | | Reference category | | |
| Use of medium/hard bristles (0 = no, 1 = yes) | –0.07 | 0.09 | 0.4 | 0.22 | 0.11 | 0.04 |
| Gum chewing | | | | | | |
| Never | 0.07 | 0.18 | 0.7 | –0.34 | 0.29 | 0.3 |
| Occasionally | 0.01 | 0.14 | 0.9 | –0.02 | 0.18 | 0.9 |
| Daily | Reference category | | | Reference category | | |

social environment. The most likely direct role for lead exposure in the development of dental caries, therefore, is as a modifier of host susceptibility. We cannot reject the hypothesis, however, that an elevated lead level is a surrogate or proxy index of some other factor that is itself directly cariogenic.

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