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The Interface of Environmental Lead, Dental Caries, and Pediatric Dentistry

By Sara Babich, DDS

The intent of this article is to interconnect environmental lead pollution, the hypersensitivity of children to this neurotoxic heavy metal, and the increased risk of dental caries in children with elevated blood levels of lead. The prompt for this article was a Reuters report that identified 69 New York City census tracts in which at least 10% of the small children had elevated blood lead levels, and that included neighborhoods previously not known to be hot spots for lead. Two tracts of particular interest included an affluent area near Riverside Park in Manhattan's Upper West Side and the Sattmar Hasidic Jewish community in Williamsburg, Brooklyn, an area with the city's highest concentration of small children, with 25% of the population age 5 years or younger.¹ Until this report, lead poisoning in residents of New York City was found almost exclusively among African-American and Hispanic children.²

Lead has no biological function, is not a required micronutrient, and, apparently, there is no safe level of exposure in humans. The most susceptible populations to lead toxicity are fetuses, infants in the neonatal stage, toddlers, and young children.² Teeth accumulate lead and provide a history of lead exposure since intrauterine life. As the hard dental tissues are relatively stable and the lead deposited in teeth during mineralization is retained, deciduous teeth are bioindicators of lead exposure during early life.³

The accumulation of lead in chil-

Abstract

Only recently, high blood lead levels were detected in children living in New York City neighborhoods typically not associated with lead pollution. These findings, coupled with cognitive neurotoxicity in children below the NYC acceptable BLL of 10 µg/dL, ignited public health concerns. Noted also was a positive correlation between elevated BLLs and the prevalence of dental caries in children. The pediatric dentist may be the initial health care professional to observe all predictors of an elevated BLL in the patient: residence in a neighborhood with known elevated lead levels; subtle abnormalities in neurobehavioral performance; and numerous dental caries.

dren can result in neurological impairment, as manifested by a shortening of attention span, a reduced intelligence quotient, decreased attention and working memory, deficit/hyperactivity disorder, reading problems, school failure, and delinquency.^{2,4} It is not surprising that early childhood lead poisoning has been linked with delinquent behavior and official arrest in late adolescence.⁵ Lead exposure in childhood is a predictor of intellectual functioning in young adulthood.⁶ The neurological damaging effects of lead are lifelong and cannot be ameliorated by current medical treatment.²

Children at risk

Environmental sources of lead are manifold. The most common source of highly concentrated lead is lead-based paint. The deterioration of lead-based paints into chips, flakes, or fine dust is easily ingested or inhaled by small children. Children aged 6 years and younger are at the highest risk of lead exposure because of their proclivity for oral-exploratory, hand-to-mouth activity, and their tendency to exhibit pica. Other sources of exposure to lead include ingestion of imported lead-contaminated candy, use of lead-containing cosmetics, and mouthing of lead-based painted toys.^{2,7} When compared to adults, the physiological hypersensitivity of children to lead toxicity is due to their increased gastrointestinal absorption of lead, their increased sensitivity to neurological damage, their rapidly growing bodies, and high metabolism.^{7,8}

The realization of the long-lasting neurological damage to children upon exposure to environmental lead ignited many public health initiatives to reduce childhood exposure to lead. Removal of lead from gasoline resulted in a 90% reduction in lead poisoning.² Another significant initiative was the banning, in 1978, of lead from domestic paints. Yet, approximately 80% of houses built before the 1960s contain lead-based paint and deteriorating paint chips, and lead dusts still continue to contaminate home surfaces.⁷ This mode of exposure accounts for much of the lead



poisoning in poor minority communities in the United States, as the older (pre-1978) housing units are in poor repair and are disproportionately concentrated in these neighborhoods.²

While recognizing that no safe blood lead level has been identified in children, the Centers for Disease Control and Prevention established a reference blood lead level of 5 µg/dL, above which public health initiatives are recommended, including that public health officials conduct home inspections to determine the source of the lead contamination. Many researchers have suggested that even a blood lead level of 5 µg/dL is unsafe. The New York City Department of Health and Mental Hygiene considers a child's blood lead level to be high only if it was at or above 10 µg/dL.^{8,9}

Based on the Centers for Disease Control and Prevention recommendation of a safe blood lead level of ≤5 µg/dL, the data in the Reuters report underestimated the risk for lead exposure to New York City children.⁹ In a recent follow-up report, the NYC Housing Authority noted between 2012 to 2016 that 820 children younger than 6 years and living in public housing were found to have blood lead levels of 5 to 9 µg/dL.¹⁰ Exposure of children to lead is not limited to NYC. Elevated levels of lead were detected in school drinking water in several states, including Indiana, Colorado, Michigan, Florida, and Maryland. The elevated levels of lead in water were traced to leaching from old plumbing rather than to lead-contaminated municipal water. As there is no national standard for an acceptable level of lead in school drinking water, many school districts are replacing old water fountains, installing water filters, and/or providing bottled water.¹¹

Strong association

Epidemiological studies of children have identified a positive corre-



lation between the body burden of lead and the prevalence of dental caries. In a study of 251 children aged 9 to 12 years, Brudevold et al.¹² found that children with high levels of enamel lead had higher incidences of dental caries than children with low levels of enamel lead. Moss et al.¹³ analyzed data from 24,903 children, aged 2 years and older, who participated in the Third National Health and Nutrition Examination Survey. The researchers noted an association between blood lead level and the risk of dental caries on permanent teeth for a cohort of children aged 5 to 17

years. In studies of 6- to 10-year-old children from the Boston/Cambridge, Mass., region, Gemmel et al.¹⁴ showed the blood lead level of school-age children was positively associated with their number of dental caries. Youravong et al.¹⁵ studied a cohort of 292 children aged 6 to 11 years from schools around a shipyard area known to be contaminated with lead. The researchers showed the children's blood lead level was positively correlated with the incidence of dental caries in their deciduous teeth.

Pradeep Kumar and Hegde³ stud-

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ied the levels of lead in enamel, saliva, and dental caries in 90 5-year-old children, divided into three groups as control, early childhood caries (ECC) and severe-early childhood caries (S-ECC). Mean enamel lead levels in the control, ECC and ECC-S groups were 47.7, 85.45, and 90.43 ppm, respectively, and the mean salivary lead levels were 0.23, 1.7, and 1.77, respectively. The enamel and the saliva of all the children had amounts of lead that increased with an increase in severity of dental caries, demonstrating the cariogenic potential of lead. A positive correlation was seen between

tooth enamel lead levels and lead levels in saliva. Increased enamel lead levels were associated with an increased incidence of caries.

Epidemiological studies by Kim et al.¹⁶ on a cohort of 7,059 children with low blood lead levels of <5 µg/dL found a significantly increased risk of dental caries, particularly for deciduous teeth, with increasing blood lead levels. The relationship between blood lead level and the development of dental caries followed a linear dose-response association.

Lead and dental caries

The action of lead in promoting the risk of dental caries may involve various mechanisms of action.^{13,16,17} The most prevalent thought is that teeth with defective enamel associated with lead absorption are susceptible to dental caries. The incidence of enamel hypoplasia was shown to increase in children exposed to elevated levels of lead. Once absorbed, lead delays calcification of these structures, as lead ions (Pb²⁺) compete with/replace calcium ions (Ca²⁺) and liberate phosphorus from the crystal lattice of dentin. Tooth analyses of teeth burdened with lead have revealed irregular tubu-

lar structures and uneven mineralization in the dentin as lead, apparently, adversely affected odontoblast activity and impeded dentin formation.

A second thought focused on the effect of lead on proper functioning of the salivary glands and of the formation of saliva. Through its negative interaction with cellular Ca²⁺ metabolism, lead (as, Pb²⁺) diminishes saliva formation and salivary flow, leading to reduced clearance of cariogenic bacteria from the oral cavity and interference with tooth demineralization and remineralization.

A third thought was directed to the interaction between lead and fluoride. Water fluoridation and regular brushing are preventive procedures to control dental caries. However, the binding of lead to fluoride ions in saliva and in plaque reduces the bioavailability of fluoride to remineralize enamel upon challenge with bacterial-generated acidic metabolic end-products. Elevated lead levels in plaque have also been associated with an increased occurrence of dental caries.

A few studies noted only a weak association between childhood lead exposure and the prevalence of dental caries in deciduous teeth.^{18,19}

Tort et al.²⁰ studied 351 children (aged 7 to 15 years) regarding their blood lead levels and oral health parameters, other than risk of dental caries; the parameters evaluated included community periodontal index, gingival index and plaque index. Only children with low blood lead levels (ranging from 0.36 to 2.90 µg/dL) were studied. They found higher blood lead levels positively correlated with poorer oral gingival health measurements and plaque deposition. An earlier study by Youravong et al.²¹ showed a correlation between high blood lead levels and periodontal problems in children, including the presence of deep pockets and increased



prevalence of subgingival levels of *Aggregatibacter actinomycetemcomitans*, a bacterial pathogen associated with juvenile periodontal disease.

Recognizing that about 25% of U.S. children live in housing with deteriorated lead-based paint and that children are a hypersensitive population at risk for lead neurotoxicity, resulting in cognitive impairment, the American Academy of Pediatrics^{22,23} recommended that most U.S. children should have their blood lead level measured at least once. There is a preponderance of evidence relating childhood lead exposure and susceptibility to dental caries. The pediatric dentist, indeed, may be the first health care professional to suspect an elevated blood lead level in a child.

It is, therefore, imperative for the pediatric dentist to be cognizant of this correlation. He or she likely already has a professional relationship with the child's pediatrician and could, therefore, suggest early, potentially interceptive blood analysis to make a definitive diagnosis. ●

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MDA Addendum

A July 2020 report by UNICEF and Pure Earth²⁴ found that 1 in 3 children, or approximately 800 million globally, have blood lead levels at or above 5 µg/dL, Henrietta Fore, UNICEF executive director, stressing the irreparable harm of lead to children's brains, noted that, "with few early symptoms, lead silently wreaks havoc on children's health and development." Multiple dental caries in a child may serve as a red flag of overexposure to lead, alerting the pediatric dentist to recommend analysis of blood lead level in such a patient.

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