Mercury exposure due to environmental factors and amalgam restorations in a sample of North Carolina children

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Abstract

Purpose: Dental amalgam restorations provide a potential source for mercury (Hg) exposure in children. This study explored the possibility that Hg levels in dentin of exfoliated primary maxillary canines could detect cumulative Hg exposure from amalgam restorations in a sample of North Carolina children.

Methods: Twenty-seven exfoliated maxillary canines from 33 children, without restorations or caries, were assayed for dentin Hg concentration ([Hg]). Urine samples were obtained from 21 subjects and assayed for [Hg] and diet surveys for seafood ingestion were completed for 26 subjects. A surface/month exposure index (SMEI) was compiled from dental records to quantify each child's cumulative exposure to amalgam restorations.

Results: Results showed that dentin [Hg] ranged from undetectable levels to 15.7 ppm with a mean of 3.7 ppm. The SMEI scores ranged from 0–638 with a mean of 95. Ten subjects had low SMEI scores of 0–3, nine had scores 4–100, and eight had scores higher than 100. No statistical correlation was found for SMEI scores and dentin [Hg]. Urine Hg levels were found to be negligible and no relationship was found between urine [Hg] and reported ingestion of seafood or SMEI scores.

Conclusions: Hg exposure in this sample of children was low and additional exposure from amalgam restorations could not be detected by the methods used in this study. (Pediatr Dent 21:114–117, 1999)

The deleterious effects of overexposure to environmental Hg in children became apparent in 1956 with the discovery of industrial waste water discharge of methyl Hg in Minamata Bay, Japan. Symptoms of mercury toxicity included visual and sensory disturbances, ataxia, dysarthria, and mental retardation.[1] It has been suggested that Hg vapor from dental amalgam restorations can be a possible source of Hg toxicity. However, carefully controlled studies have failed to confirm amalgam restorations as a health hazard.[2, 3] Due to the complexity of human exposure to various sources of Hg, the relationship of amalgam restorations and Hg body burden merits additional study.

For the typical child residing in the US, two major sources for Hg exposure are diet and amalgam restorations. Water containing high Hg levels and ingestion of fish are the main sources for organic or methyl Hg intake.[1, 4, 5] Release of Hg vapor from dental restorations during chewing and brushing allows inhalation of metallic Hg vapor.[4, 6] Biotransformation of these two forms of Hg yields both inorganic elemental Hg and organic and inorganic compounds. With varying degrees of transport and absorption, organic Hg is deposited in multiple body organs including the kidneys and brain.[4, 8] Hg accumulation is dependent upon the biological half-life which varies from days to years and is, in part, dependent on formation of complex compounds with elements that act as antagonists to Hg, such as selenium. Elimination of organic Hg is through the urinary and fecal routes and some elemental Hg is exhaled.[5, 6, 9, 10] With the variations in short-term versus chronic Hg exposures, the complicated metabolic pathways for transformation into respective compounds, the variable uptake of these Hg compounds, and the various biologic half-lives, it is not surprising to find quantification and reproducibility problems when measuring Hg exposure in humans.[1, 10] Traditional measurement sites, or indicators, for Hg exposure are urine, blood, and hair. While exposure to inorganic Hg can be measured in the urine, methyl Hg exposure is detectable in blood and hair. From an epidemiological perspective in comparing groups of individuals, recent Hg exposure can be measured in blood and urine but longer-term exposure is often measured by hair samples.[11] Reliability of hair Hg concentrations has been questioned due to direct contamination by absorption of atmospheric Hg vapor resulting from combustion of fossil fuels and solid wastes.[12, 13] While none of these methods is reliably indicative of chronic mercury exposure beyond 3 or 4 months, urine Hg levels are regarded as the best available indicator of current exposure to Hg.[1–6]

Heavy elements such as lead are incorporated into dentin and enamel and remain in these hard tissues with little if any biological turnover. Not only will these heavy elements be incorporated as the dentin is formed, the dentinal tubules and odontoblastic processes provide continuous indirect exposure to the extracellular fluids in the pulp, providing for uptake of elements after dentin formation is complete. Indeed, recent Norwegian studies indicate that [Hg] in primary teeth may be a useful marker for estimation of cumulative Hg exposure in children. Hg concentrations were determined in 57 primary teeth obtained from an excavation under a 12th-century stave church in Norway and in 124 unrestored primary teeth collected from Norwegian children in 1971–72.[15] The teeth from contemporary children had 10 times the [Hg] as teeth from

Fig 1. The Diet History Questionnaire

(1) Has your child always lived in or near the community where you live now? ____Yes ____No

If "No", in what other communities has your child lived?

Town	State	Dates
_____	_____	_____
_____	_____	_____
_____	_____	_____

(2) Does your child currently eat more than two meals a week that contain fish of any kind? ____Yes ____No

If "yes", what kind of fish?_____

Especially, do they eat tuna or swordfish? ____Yes ____No

(3) In the past, has your child eaten more than two meals a week containing fish? ____Yes ____No

If "yes", what kind of fish?_____

Especially, tuna or swordfish? ____Yes ____No

(4) Has your child had any unusual dietary habits of any kind? ____Yes ____No

If "yes", please described:_____
_____
_____

Thank you for filling out the questionnaire. Please mail it to us in the enclosed addressed and stamped envelope.

preindustrial-age children. In 1993, Eide and Wesenberg reported that [Hg] in rat molars significantly correlated with variations in exposure to mercury vapor.[7] In a more recent study, it was shown that the [Hg] in rat molars was a marker for both inorganic and organic Hg exposure.[16]

The present study was undertaken to explore the possibility that [Hg] in the dentin of exfoliated primary maxillary canines could detect cumulative exposure to Hg from amalgam restorations in a convenience sample of North Carolina children.

Methods

The study was approved by the University of North Carolina School of Dentistry Committee on the Use of Human Subjects in Research. Subjects were selected from the Department of Pediatric Dentistry teaching clinics and faculty practice. Fifty-four children who had mobile, noncarious, and restoration-free maxillary canine teeth which were expected to exfoliate within 3 months were selected. It was also necessary that each subject have a complete and accurate dental record which documented the types of amalgam restorations, the dates when placed, and the dates when the restored teeth were extracted or exfoliated. A consent form explaining the nature and conditions of the study was given to the parent of each potential subject. Upon receipt of a signed consent form, a diet history survey and a container to return the exfoliated canine(s) were sent to the parent with stamped and addressed return envelopes. Upon receipt of the completed diet history form (Fig 1) and the exfoliated tooth, a container and instructions for collection of a first morning urine sample were sent to the parent. Financial rewards were provided upon receipt of each study item.

Each tooth was sectioned using a diamond saw after removing any remaining root structure. The longitudinal sections were cut from the middle of the crown in the facial/lingual direction and were approximately 150 μm thick. With a dissecting microscope, enamel was carefully removed from the dentin and discarded. All of the crown dentin from each section was recovered and air dried for 24–48 h and then oven dried at 100°C for 2 h to eliminate water and arrive at a constant weight. Hg was extracted from the dentin samples according to the method described by Eide and coworkers.[7] Hg assays for the dentin and urine samples were determined by cold-vapor atomic absorption spectrophotometry using a Perkin-Elmer FIMS 400, atomic absorption spectrophotometer equipped with an A-90 autosampler. Controls were run every 10 samples and fell within 2 standard deviations (SD) of the mean for the bracketed samples to be accepted.

An SMEI was used to quantify each subject's cumulative exposure to amalgam restorations by review of the dental records. This method is a modification of an index used by the National Institute of Dental Research for the study of Hg exposure from dental amalgam in adults.[17] For each primary or permanent tooth restored with amalgam, the number of surfaces was multiplied by the total number of months that the restoration was present until the time of the primary canine exfoliation. The cumulative number of surface/months of amalgam exposure determined the SMEI for each subject.

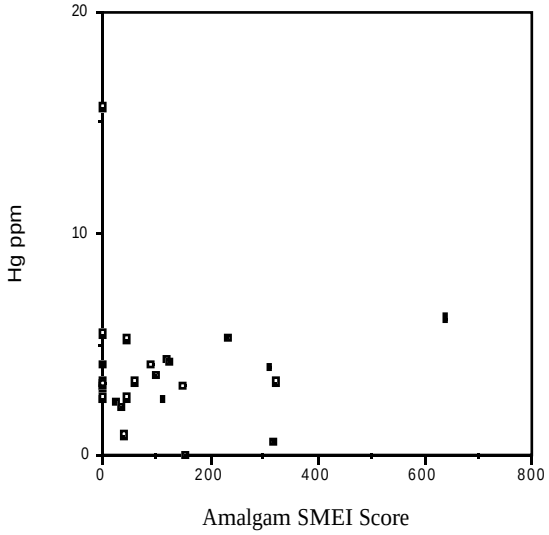

Fig 2. Dentin Hg concentrations (Hg ppm) plotted against their respective amalgam surface/month exposure index (SMEI). There was no significant correlation between these variables.

If the canine tooth was exfoliated before the 15th of the month, that month was not counted. If the restoration was placed after the 15th of the month, that month was not counted. For unknown dates of exfoliation between recall visits, the estimated time of exfoliation was calculated as one-half of the time interval between recalls. A zero SMEI score represented a child who was caries free with or without sealants, or one who had never received an amalgam restoration but had composite and/or stainless-steel crown restorations. The relationship between respective dentin [Hg] and SMEI scores was statistically evaluated by regression analysis and Pearson's product-moment correlation test.

Results

A total of 54 children with noncarious and restoration-free maxillary primary canines near exfoliation were originally enrolled in the study. This represented a population of children both caries-free and with multiple amalgam restorations in posterior teeth. Of these, 33 children with a median age of 10 years, 1 month (range 6 years, 6 months to 13 years, 8 months) submitted exfoliated canines or canines suitable for sectioning that were extracted for orthodontic reasons and the child had dental records sufficiently accurate to determine SMEI scores. A total of 27 canines were assayed for [Hg]. The remaining teeth were fractured or otherwise lost during sample preparation. Urine samples were obtained from 21 subjects, and diet surveys from 26 subjects.

The dentin [Hg] ranged from undetectable to 15.7 ppm with a mean of 3.7 ppm. The SMEI scores ranged from 0 to 638 with a mean of 95. Ten children had SMEI scores ranging from 0 to 3, nine had scores ranging from 4 to 100, and eight had scores higher than 100. The subject with the highest dentin [Hg] of 15.7 ppm had a SMEI of 0 and undetectable urine [Hg]. This subject reported eating little or no fish, but lived in Peru from 1985 to 1988 and may have been exposed to a high level of environmental mercury. An undetectable urine [Hg] in this child indicated that his current Hg exposure was low. For the eight children with SMEIs higher than 100, the dentin [Hg] ranged from undetectable for an SMEI of 155 to 6.2 ppm [Hg] for the highest SMEI of 638. Fig 2 shows the dentin [Hg] plotted against the respective SMEI scores. Pearson's product-moment correlation coefficient was r=0.00, with a P value of 1.00, indicating no statistical correlation between the dentin [Hg] and the SMEI scores.

Only two subjects had detectable urine concentrations. The subject with 1 ppb urine [Hg] had a SMEI of 0 but reported eating seafood two times or more per week. The subject with a 2 ppb urine [Hg] had the highest SMEI score of 638 and reported eating no seafood. Only 10 subjects indicated that they ate seafood at all and their dentin levels ranged from 0.6 to 5.3 ppm and urine [Hg] was undetectable for nine of the 10. Six subjects reported eating seafood more often than two times per week and their dentin [Hg] ranged from 0.6 to 5.2 ppm and urine [Hg] ranged from undetectable to 1 ppb. The mean dentin [Hg] for the six children who ate seafood regularly was not satistically different than the mean for the children who ate little if any seafood. Statistical evaluation could not be considered for the relationship of urine levels with SMEI scores when only two subjects had detectable urine mercury levels.

Discussion

A study by Eide and Wesenberg showed significant correlation between rat molar [Hg] and Hg vapor exposure.[7] A subsequent study by the same group indicated that rat molars may be used as a marker for both inorganic and organic Hg exposure.[16] This group has also shown that whole human primary teeth may be used as a marker for environmental Hg exposure.[15] In this study, only the crown dentin from exfoliated maxillary primary canine teeth was used to determine dentin [Hg]. Removing remaining root dentin and enamel eliminated the possibility of surface contamination, thus more accurately reflecting systemic Hg exposure. The arithmetic mean for [Hg] in whole primary teeth from postindustrial Norwegian children was 10.52 ppm compared to a mean of 3.7 ppm in the crown dentin of the exfoliated canine teeth collected in our study. This difference may reflect surface contamination in the Norwegian sample, the fact that Norwegian children eat more seafood than the children in the current study, or a combination of these and other environmental sources.

Both the dentin and the urine [Hg] observed in this study indicate that the children had low exposure to environmental Hg. Urine [Hg] reflects only current exposure and was used in this study in that context. At a lowest detection level of 1 ppb, only two children in our study had detectable [Hg] in their urine, the highest being 2 ppb. The World Health Organization (WHO) has estimated the allowable urine [Hg] to be 4 ppb.[4] A Turkish group reported on urine Hg levels from 10 amalgam-free children and in the same children 24 h after the placement of amalgam restorations.[11] The mean age of the children was 8 years with a range from 4 to 12 years. Using a method with a seemingly lower detection limit than the method used in our study, they could not detect Hg in the urine of any of the children prior to placement of amalgam. They reported very low concentrations (mean=0.43 ppb) soon after the restorations were placed. Similar findings were re-

ported in a Norwegian study measuring urine Hg before and after amalgam placement and found no significant urinary Hg levels throughout the treatment period.[18]

The lack of correlation between dentin [Hg] levels and SMEI scores in our study indicates that the contribution to cumulative environmental mercury exposure by amalgam restorations in this sample of children was below the lowest detection limit for the method. This was true even against a low background of environmental exposure. The fact that the child with the highest SMEI score had the highest urine [Hg] and the third highest dentin [Hg] of 6.2 ppm suggests a correlation between these parameters, and SMEI scores might exist in children with higher SMEI scores than those recorded for the children in this study. However, a decline in caries prevalence and increasing use of composite materials in place of certain amalgam restorations suggests that the use of amalgam restorations in children may decrease in the future. Conversely, as the severity of caries increases beyond a moderate level, the SMEI score would decline because of the necessity to use stainless-steel crowns or extract teeth. It seems likely that SMEI scores of 600 or higher may become a rarity in the future for children in the United States. Nevertheless, further studies should be conducted to better define the sensitivity and usefulness of dentin [Hg] of primary teeth as a marker for cumulative systemic Hg exposure.

Conclusion

1. Environmental Hg exposure in this sample of North Carolina children was quite low.
2. Hg exposure from dental amalgam restorations could not be detected as additive to the background of environmental exposure from other sources.

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