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Childhood urine mercury excretion: dental amalgam and fish consumption as exposure factors ☆

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Abstract

The authors investigated the effect of amalgam fillings and fish consumption on urine mercury level (UHg), in children aged 4–8 years old inclusive. Using a sample of 60 children, we found that children with amalgam fillings had significantly higher UHg levels than children without amalgams (geometric mean = 1.412 μg Hg/g versus 0.436 μg Hg/g, respectively, $P = 0.0001$). Subjects with reported higher fish consumption also had significantly higher UHgs ($P = 0.004$). Univariate analyses provide evidence of an association between elevated UHg level and young age ($P = 0.009$), short height ($P = 0.024$), and low weight ($P = 0.049$) in children with amalgam chewing surfaces. We also found a negative correlation between urine mercury and age (−0.378), height (−0.418), and weight (−0.391). A multiple logistic regression model shows that the presence of amalgam fillings leads to increased odds of high UHg in children (OR = 47.18), even after adjusting for high fish consumption (OR = 8.66) and height (OR = 11.36).
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1. Introduction

Amalgam containing mercury has been in use as a dental restorative material for over 150 years (US DHHS, PHS, 1993). During the past two decades, this material has come under increasing scrutiny with regard to its safety as it is known that amalgam restorations continuously discharge metallic mercury into the oral cavity, mostly in vapor form (Svare et al., 1981; Abraham et al., 1984; Vimy and Lorscheider, 1985a, b; Patterson et al., 1985; Berglund et al., 1988; Berglund, 1990; Aronsson et al., 1989; Björkman and Lind, 1992; Jokstad et al., 1992). This release is enhanced during activities such as chewing, toothbrushing, drinking hot beverages, or oral breathing (Gay et al., 1979; Brune, 1988; Sallsten et al., 1996). When mercury vapors are inhaled, 80% is readily absorbed in the blood through the lungs and distributed in various organs, mainly in the kidneys where it may become incorporated before being excreted (Hursh et al., 1976; Lauwerys, 1983). In humans, other organs (brain, lungs, liver, gastrointestinal tract, endocrine glands) show varying degrees of elevated concentrations although the brain is the site of greatest sensitivity (Conseil d'évaluation des technologies de la santé du Québec, 1997) Metallic mercury, being lipophilic, can readily cross the blood–brain and placental barriers where it is oxidized to inorganic mercury. In this state, mercury is not lipophilic and has a limited ability to recross these biological membranes (Conseil d'évaluation des technologies de la santé du Québec, 1997). Thus, mercury can be retained in the brain (Hargreaves et al., 1988) and fetal tissues (Clarkson et al., 1972).

The amount of mercury from amalgam passing through the gastrointestinal tract may be large but is poorly absorbed (US Environmental Protection Agency,

Abbreviations: UHg, urine mercury level; μg Hg/g creatinine, microgram of mercury per gram of creatinine in urine.

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1984). Other routes of exposure, through electrochemical corrosion, and directly through the oral mucosa appear to be of considerable less importance than inhaled vapor.

In recent years, several studies have tried to estimate exposure by correlating urinary mercury levels with the number of amalgam fillings in nonoccupationally exposed persons (Skare and Engqvist, 1994; Kingman et al., 1998). The evidence from these studies shows that dental amalgam is the predominant contributor to nonoccupational metallic mercury exposure. Much less documented is mercury exposure in children from amalgam fillings using mercury urine levels as a biomarker (Trepka et al., 1997; Dilley and Bawden, 1999; Olstad et al., 1987; Schulte et al., 1994). Those few studies on children show divergent results which do not allow a basis for consensus. Their results will be examined in the discussion of this article.

By contrast, mercury exposure from diet is mainly from fish consumption in the form of methyl mercury (WHO, 1991). It is generally believed that methyl mercury does not appreciably elevate urine mercury levels (UHgs), since 90% of it is excreted in the feces (Clarkson et al., 1988).

The objectives of this study are:

1. To compare the UHg in the urine of 4- to 8-year-old children with two major exposure factors, namely
   (a) the presence of amalgam fillings (7 or more chewing surfaces) versus no amalgam fillings, and
   (b) high fish consumption versus low fish consumption.
2. To determine the influence of potential factors such as age, height, weight, gender, bruxism, oral breathing, and gum chewing, on UHg levels.
3. Using logistic regression, to identify the factors most likely to contribute to higher UHg levels.

2. Materials and methods

This study was approved by the Montreal Children's Hospital Ethics Committee on the Use of Human Subjects in Research. Subjects were selected from children attending the pediatric dental clinic of the Montreal Children's Hospital after informed consent was obtained from the accompanying parent or guardian explaining the nature and purpose of the study. Sixty healthy children 4–8 years old inclusive were selected, 34 of which had 7 or more amalgam chewing surfaces (range 7–22 surfaces) present in the mouth. We defined chewing surfaces as those which normally occlude (contact) the teeth of the opposite maxilla during mastication. Restored proximal surfaces were included because part of the filling includes the marginal ridge of the occlusal surface. Buccal and lingual restorations were excluded because they do not contact opposite teeth. Only children who had no new amalgam filling placement or replacement for a minimum of 6 months were recruited. This was necessary since it takes approximates 6–7 months in adults to reach a steady state given the half-life of urinary mercury (40 days) for subjects exposed to low levels of mercury (Lauwerys and Hoet, 1993; Nakaaki et al., 1978; Barregård et al., 1996).

The remaining 26 control subjects, also 4–8 years old, had no dental restorations. At the first appointment reserved for the oral examination, a questionnaire was used to collect demographic data and to document potential risk factors and other possible sources of mercury, particularly fish consumption (Table 1). The height and weight of each child were also recorded. A urine sample was collected at the follow-up appointment before any dental prophylaxis or amalgam restorative was performed, in order not to influence the subjects' exposure to mercury. Urine samples were immediately frozen and sent to the Centre de Toxicologie du Québec for analysis. This laboratory is accredited under ISO 17025 by the Standards Council of Canada and is part of the Institut national de Santé Publique du Québec, a provincial government body. It participated successfully

Table 1
Description of variables under study

Variable	Description or categorization
Hospital number	—
Subject name, birthday	—
Age	4–8 years inclusive
Number of amalgam chewing surfaces	—
Height	Centimeters
Weight	Kilograms
Gender	Male/female
Number of years lived in Canada	Years
Country of birth	—
Country where amalgams were performed	—
Teeth brushing frequency	Once/twice/three times per day
Toothpaste type	Paste/gel/both/none
Parents notice bruxism	Yes/no
Dentist notices bruxism	Yes/no
Oral breathing	Always/often/sometimes/never/don't know
Sleeps with mouth open	Yes/no/do not know
Chews gum	Yes/No
How often chews gum	3 times per day/1–3 times per day/2 times per week/less than 2 times per week
Fish consumption	Once per day/2 times per day/once per week/once per month/less than once per month
Urine mercury level	UHg/g creatinine

in Health Canada's Intercomparison for mercury in hair and the German External Quality Assessment Scheme for mercury in urine and blood. It also coordinates an Inter-laboratory Comparison Program for heavy metals in which 230 laboratories take part worldwide. Inorganic urine mercury was determined by cold-vapor atomic absorption spectrometry (Ebbestadt et al., 1975), using a dedicated mercury analyzer corrected for creatinine content (Mercury Monitor Model 100, Pharmacia, Stockholm). The detection limit was 0.2 μg Hg/g creatinine.

It was expected that these data would follow a skewed Poisson probability distribution, which is common in studies of environmental exposures. To approximate normality, geometric means expressed as log UHg and corresponding 95% confidence intervals were computed to compare exposure groups. Normality of the resulting distributions of the outcome variable (log UHg) was verified using the Kolmogorov–Smirnov (KS) whenever *t* tests were used. The mean log UHg levels of both groups (with and without amalgam surfaces) were compared using the *t* test. To determine the association between first sample mercury excretion and independent variables such as age, height, and weight among the children with amalgams, we applied correlation analyses, *t* tests, and nonparametric tests, as well as relative risks (RR) and odds ratios (OR). Logistic regression analysis was carried out to identify variables potentially associated with a high UHg level (defined as above the median) in all children, such as the presence of 7 or more amalgam chewing surfaces, age, weight, height, oral breathing, gum chewing, bruxism, and fish consumption. A brief description of variables under study appears in Table 1.

3. Results

3.1. Study population

Of the 60 subjects who participated in our study, 34 (56.7%) were males and 26 (43.3%) females. The mean age of these individuals was 6.9 (SD = 1.26), median age = 7.0, the youngest being 4.1 years old and the oldest, 8.9 years old.

As expected, we found the overall distribution of UHg to be highly skewed, as shown on Fig. 1 (KS test: $P<0.0001$). Taking the natural logarithm of UHg resolved this problem and yielded a normal curve (KS test: $P = 0.2000$) in this distribution and in all subgroups where *t* tests were performed. Thus, we used log(UHg), rather than UHg itself in all analyses. Risk and exposure factors that achieved significance in univariate analyses were presence of amalgam chewing surfaces, age, height, weight, and high fish consumption.

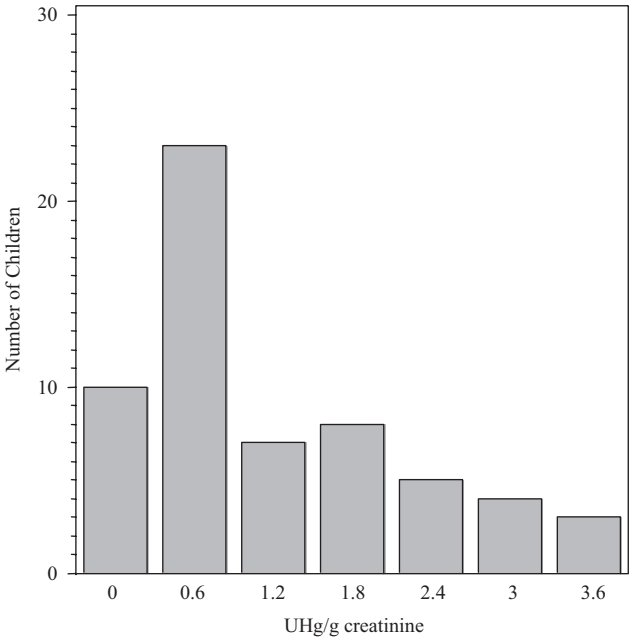

Fig. 1. Urine mercury distribution for children aged 4–8 years, all children.

3.2. Amalgam versus nonamalgam group

Among children with 7 or more amalgam chewing surfaces [mean number of surfaces 13.3, standard deviation (SD) 6.7], the geometric mean was 1.41 μg Hg/g creatinine versus 0.44 μg Hg/g creatinine for those without amalgam fillings, yielding a mean difference of 0.976 μg Hg/g creatinine, as shown on Table 2. This difference was highly significant, as evidenced by the *t* test ($P = 0.0001$), indicating that children with dental amalgam fillings have significantly higher urine mercury concentrations than children without amalgams. A similar conclusion is reached with the arithmetic mean.

3.3. Age, height, and weight

Among the children with amalgam surfaces, we compared those aged less than the median of 7 years, with children aged 7 years or more. As shown in Table 3, we found no difference in the mean number of amalgam chewing surfaces between these two groups ($P = 0.919$). However, the younger children have significantly higher mean log(UHg) levels than the older children (*t* test: $P = 0.009$, Wilcoxon test: $P = 0.014$). They are also 2.62 times more likely to have UHgs above the median, than the older children (RR = 2.62). We found no difference in mean log(UHg) levels between the two age groups among children with no amalgam chewing surfaces ($P = 0.3920$, data not presented).

We also examined the height and weight of children in the amalgam group, to ascertain whether these two characteristics could be related to differences in mean

Table 2
Urine mercury levels of subjects with and without amalgams (μg Hg/g creatinine)

	With amalgams ($n = 34$)	Without amalgams ($n = 26$)	P value[a]
Urine mercury arithmetic mean (95% CI)	1.70 (1.36, 2.05)	0.61 (0.34, 0.87)	0.0001
Urine mercury geometric mean (95% CI)	1.41 (1.13, 1.76)	0.44 (0.32, 0.60)	0.0001

[a] Obtained from the t test.

Table 3
Differences in UHg by age, amalgam group only

	Aged < 7 years ($n = 14$)	Aged 7 + years ($n = 20$)	P value	RR (95% CI)
Mean number of amalgams (SD)	13.31 (3.66)	13.45 (4.02)	0.919[a]	—
UHg geometric mean (95% CI)[b]	1.98 (1.48, 2.66)	1.11 (0.82,1.51)	0.009[a]	—
Mean rank-score for log(UHg)	22.5	14.0	0.014[c]	—
Number with log(UHg) above median	11/14 (79%)	6/20 (30%)	0.005[d]	2.62 (1.27–5.40)

SD, standard deviation; RR, relative risk.
[a] Obtained from the t test.
[b] Urine mercury levels are expressed in μg Hg/g creatinine.
[c] Obtained from Wilcoxon test.
[d] Obtained from the χ^2 test.

Table 4
Differences in urine mercury level by height, amalgam group only

	Height ⩽ 124 cm ($n = 17$)	Height > 124 cm ($n = 16$)	P value	RR (95% CI)
Mean number of amalgam surfaces (SD)	13.9 (3.40)	12.9 (4.36)	0.476[a]	—
UHg geometric mean (95% CI)[b]	1.83 (1.38, 2.42)	1.10 (0.76, 1.58)	0.024[a]	—
Mean rank-score for log(UHg)	20.5	13.3	0.031[c]	—
Number with log(UHg) above median	12/17 (71%)	5/16 (31%)	0.024[d]	2.26 (1.03–4.97)

SD, standard deviation; RR, relative risk.
[a] Obtained from the t test.
[b] Urine mercury levels are expressed in μg Hg/g creatinine.
[c] Obtained from Wilcoxon test.
[d] Obtained from the χ^2 test.

Table 5
Differences in urine mercury level by weight, amalgam group only

	Weight ⩽ 24 kg ($n = 17$)	Weight > 24 kg ($n = 16$)	P value	RR (95% CI)
Mean number of amalgam surfaces (SD)	14.3 (3.3)	12.5 (4.3)	0.207[a]	—
UHg geometric mean (95% CI)[b]	1.72 (1.22, 2.42)	1.17 (0.85,1.62)	0.049[a,c]	—
Mean rank score for log(UHg)	19.9	13.9	0.039[d,c]	—
Number with log(UHg) above median	12/17 (71%)	5/16 (31%)	0.024[e]	2.26 (1.03–4.97)

SD, standard deviation; RR, relative risk.
[a] Obtained from the t test.
[b] Urine mercury levels are expressed in μg Hg/g creatinine.
[c] One-tailed P value.
[d] Obtained from Wilcoxon test.
[e] Obtained from the χ^2 test.

log(UHg) levels. We stratified this group according to height (above/below median of 124 cm) and weight (above/below median of 24 kg). The entries of Tables 4 and 5 indicate no difference in the mean number of amalgam chewing surfaces when stratified by height ($P = 0.476$), nor when stratified by weight ($P = 0.207$). However, the data show that short children have significantly higher log(UHg) levels (t test, $P = 0.024$; Wilcoxon test, $P = 0.031$), as do children of low weight (χ^2 test: $P = 0.024$). The data also indicate that in these groups, short children, and children of low weight, are at risk to have a UHg above the median (RR = 2.26 for height and RR = 2.26 for weight). This analysis was repeated in the nonamalgam group, but the differences were not significant (t test, $P = 0.749$ for height and $P = 0.301$ for weight; Wilcoxon test, $P = 0.908$ for

height and $P = 0.311$ for weight, data not presented). In Table 5, certain P values are for one-tailed tests since we expected a priori that the relationship between UHg and weight would be similar to that observed between UHg and age and height, namely, that children of low weight would likely have higher UHgs, as do younger and shorter children.

We found similar evidence of negative correlation between age, height, weight, and UHg among the 34 children with amalgam surfaces (Table 6). The Pearson correlation coefficient between age and UHg was found to be −0.378, while that between height and UHg was −0.418. Both of these correlations are significantly different from zero, as was the Spearman correlation between weight and UHg (−0.391). These results indicate there exists an inverse relationship between UHg concentration and each of age, height, and weight. To illustrate this pattern for age and height, a simple linear regression model was constructed for UHg, using age and height as predictors. The results appear in Figs. 2 and 3. In the case of UHg and weight, this relationship is not linear.

3.4. Fish consumption

Among all 60 children, those reporting frequent fish consumption (every day or twice per week) had significantly higher levels of log(UHg) in their urine than children reporting infrequent consumption (once per week, once per month, less than once per month), as evidenced by the t test ($P = 0.004$) and the Wilcoxon test

Table 6
Pearson and Spearman correlation coefficients between urine, mercury level and each of age, height, and weight (amalgam group only)

Age (years)	Height (cm)	Weight (kg)
$r_p = -0.378$ (0.028)	$r_p = -0.418$ (0.015)	$r_s = -0.391$ (0.024)

Numbers are pearson (r_p) or Spearman (r_s) correlation coefficients with corresponding P values in parentheses.

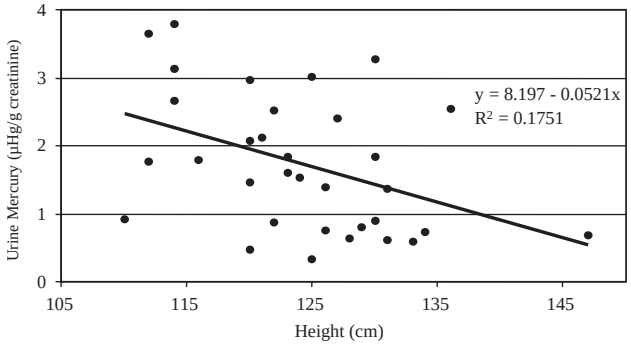

Fig. 2. Urine mercury versus height for children aged 4–8 years, amalgam group only.

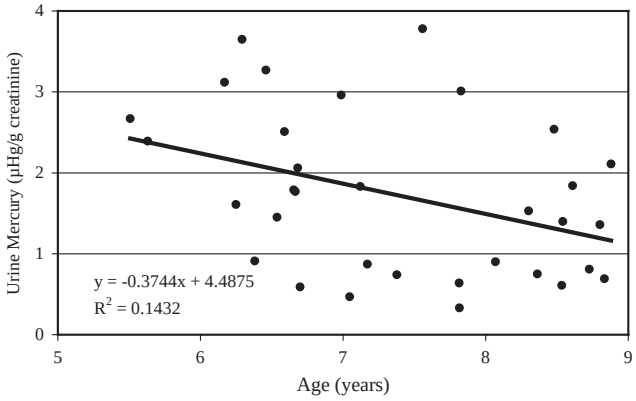

Fig. 3. Urine mercury versus age for children aged 4–8 years, amalgam group only.

($P = 0.006$) (data not shown). This pattern was identical among the 34 children with amalgam fillings (t test, $P = 0.040$; Wilcoxon test, $P = 0.020$) (Table 7), and among the 26 children without amalgams (t test, $P = 0.057$; Wilcoxon test, $P = 0.024$, data not presented). There was no statistically significant difference in the frequency of fish consumption among subjects with and without amalgam fillings.

As indicated in Table 7, among children with amalgam chewing surfaces we found no difference in the mean number of surfaces between children reporting frequent fish consumption, and children reporting infrequent fish consumption ($P = 0.726$).

3.5. Logistic regression

A logistic regression model was employed to detect differences in high UHg (defined as above/below the median of 1.45 µg Hg/g creatinine) between the two groups (with and without amalgams), while simultaneously adjusting for fish consumption (high/low) and height (short/tall). These three predictors lead to the bestfitting logistic model, and as indicated in Table 8, all are significantly related to high levels of urine mercury. We find the OR for fish consumption to be 8.661, suggesting that the odds of high urine UHg to low urine UHg is almost 8.7 times higher among children reporting high fish consumption than those reporting low fish consumption. The OR for short height was found to be 11.360 while that for amalgam filling was 47.174. This implies that the odds of high to low urine UHg are almost 11.4 times higher in short children, and over 47 times higher in children with amalgam filling. To compare the effect of independent variables separately, the analysis was repeated but using only one predictor in each logistic regression model. Results were similar to those of the multiple model, but with more precise estimates of ORs (data not presented).

Table 7
Differences in urine mercury level by fish consumption, amalgam group only

	Frequent fish consumption ($n = 10$)	Infrequent fish consumption ($n = 23$)	P value	RR (95% CI)
Mean number of amalgam surfaces (SD)	13.89 (2.89)	13.35 (4.20)	0.726[a]	—
UHg geometric mean (95% CI)[b]	2.04 (1.23, 3.35)	1.23 (0.95, 1.59)	0.040[a]	—
Mean rank score for UHg	22.9	14.4	0.020[c]	—
Number with UHg above median UHg	9/10 (90%)	10/23 (43%)	0.013[d]	2.07 (1.24–3.45)

SD, standard deviation, RR, relative risk.
[a] Obtained from the t test.
[b] Urine mercury levels are expressed in μg Hg/g creatinine.
[c] Obtained from Wilcoxon test.
[d] Obtained from the χ^2 test.

Table 8
Results of multiple logistic regression, using high mercury urine (above median level of 1.45 μg Hg/g) as outcome variable, all children

Predictor	Odds ratio (OR)	P value	95% CI for OR
Fish consumption (often versus rarely)	8.661	0.0310	(1.219, 61.517)
Short height (below median versus above)	11.360	0.0090	(1.837, 70.239)
Amalgam filling (presence versus absence)	47.174	0.0001	(5.459, 407.647)

4. Discussion

This study was primarily aimed at comparing the urinary mercury level (μg Hg/g creatinine) of 4- to 8-year-old children inclusive with dental amalgams and high fish consumption with those without these exposure factors. The presence of amalgam fillings was the most significant predictor of high UHgs in children (Tables 2 and 8). Our results support those of previous studies indicating that the concentration of Hg found in urine of adult and children subjects with no occupational exposure is mainly dependent on the presence of amalgam fillings (Skare and Engqvist, 1990, 1994; Kingman et al., 1998; Trepka et al., 1997; Olstad et al., 1987; Schulte et al., 1994; Langworth et al., 1991; Akesson et al., 1991).

However, previous children studies comparable to ours show widely varying results. One study reported a mean value of 1.0 μg Hg/g creatinine in 73 children with a mean of 5.8 amalgam surfaces (SD 4.8), and 0.31 μg Hg/g creatinine in 14 children without amalgam fillings (Olstad et al., 1987). Another study reported mean levels of 0.64 μg Hg/g creatinine ($n = 86$) and 0.19 μg Hg/g creatinine ($n = 93$) for children with and without amalgams respectively (Schulte et al., 1994). A third study (Dilley and Bawden, 1999) found negligible UHg levels and no relationship between these levels and the presence of amalgam restorations or reported ingestion of seafood.

By contrast, we found higher mean UHgs than those reported in those children studies. The arithmetic mean mercury level for children with 7–22 amalgam surfaces (mean = 13.3 surfaces, SD = 6.7 surfaces) was 1.70 μg Hg/g creatinine (SD = 0.99 μg Hg/g), whereas in the nonamalgam group, the level was 0.61 μg Hg/g creatinine (SD = 0.65). We feel that our data are highly reliable due to the accuracy of the analytical technique used by the Centre de Toxicologie du Québec.

Our findings closely match those of Kingman et al. (1998) who measured the urinary excretion of Hg in an adult military population with and without dental amalgam fillings. Their data show that, in the group with 10–19 amalgam surfaces (335 adults), the urinary excretion was 1.56 μg Hg/g creatinine (SD = 1.64) and 0.68 μg Hg/g creatinine (SD = 0.63) for their nonamalgam group.

Our results also show that, among children with amalgam fillings, younger ones and those of short stature and low weight were at significantly increased risk of high UHg levels. This relationship was not significant among children without amalgam fillings. One conjecture we offer is that the sizes of chewing surfaces in our sample were independent of age, height, or weight. Hence, mercury exposure and subsequent excretion is proportionally greater in younger children because they are smaller. These findings are of interest and indicate a need for further research on the relationship between child growth and mercury kinetics such as absorption distribution and elimination.

Using logistic regression, the factors identified as most closely associated with high UHgs were the presence of amalgam fillings, short stature, and fish consumption (Table 8). However, the confidence intervals for the OR from the logistic regression are quite wide, meaning that these estimates are not precise, probably the result of small sample size. The ORs obtained from the univariate logistic models are more precise (data not presented). Univariate analyses suggested that age, height, and

weight were all associated with higher mercury urine levels in the amalgam group, but of the three, height had the strongest outcome in the logistic regression model.

An unexpected finding of this study is that children who reported higher levels of fish consumption excrete significantly elevated amounts of Hg. This outcome is somewhat surprising since Hg in fish is mainly methyl-Hg, which is not excreted through the kidney (Clarkson et al., 1988; WHO, 1996). Our results are corroborated by another recent study which also found that fish intake significantly influenced the UHg levels (Apostoli et al., 2002). These results contradict current thinking that dietary mercury intake does not influence UHg levels, indicating that more studies are needed to investigate the metabolism and urinary excretion of dietary mercury in children.

However, not all elevated UHg levels could be explained. One subject with no amalgam fillings and no other apparent risk factors had a level of 3.31 μg Hg/g creatinine. Another subjects had a UHg level of 5.15 μg Hg/g creatinine. This subject had 12 amalgam filling surfaces and reported consuming fish daily, the highest fish consumption of all subjects in the study. This value was not used in our analyses as it came from a second sample taken from 12 children 3 weeks after the first for validation purposes. The results of the second samples were not significantly different from the first, although for this subject, the UHg level increased from a first sample level of 2.39 μg Hg/g creatinine.

One area of concern is the accurate estimate of a safe exposure level. The mean value reported in the present study is about 30 times less than the value of 50 μg Hg/g creatinine proposed by WHO in 1980 (WHO, 1980) and 20 times less than the current biological exposure index of 35 μg Hg/g creatinine proposed by the American conference of Governmental Industrial Hygienists in 2002 (ACGIH, 2002). By comparison, as noted previously, urine levels from patients of our dental clinic were as high as 5.15 μg Hg/g creatinine. However, the WHO and ACGIH indices are estimates of acceptable exposure levels that only apply to workers (healthy adult) and not to children. Recent studies have reported subclinical health effects allegedly resulting from long-term low occupational exposure levels below 50 μg Hg/g creatinine, although these results should be interpreted with caution until they can be reproduced by other well-controlled studies (Echeverria et al., 1995; Fawer et al., 1983). Nevertheless, these studies do raise the hypothesis that a safe level of exposure may be less than the current values identified above. As a precaution, in the province of Quebec, pregnant or breastfeeding women potentially exposed to mercury may be withdrawn from their workplace when their urinary mercury level exceeds 3.5 μg Hg/g creatinine (Preventive Withdrawal Program). This value corresponds to the 90th percentile of a population comprising a group of pregnant women not exposed to occupational mercury (Truchon et al., 2000) and is intended to reduce the risk of adverse effects to the fetus or the newborn. It is worth noting that in our study, 3 out of 33 subjects (9.1%) with amalgam surfaces had levels above 3.5 μg Hg/g creatinine whereas no subjects without amalgam fillings had levels above it.

The evidence presented here confirms previous findings that children with amalgam fillings have an increased exposure to mercury, although no inference can be drawn about possible adverse health effects. At present, there is a lack of scientific evidence on toxicity from low-level mercury exposure in children although concern has been expressed that mercury may follow the heavy metal paradigm, that very young children could be at greater risk to neurological harm from low-level exposure due to their developing system, as well as to renal toxicity. Hence, we recommend that research be conducted to more precisely define the potential health effects from dental amalgam on children's health.

Unfortunately, no ideal restorative material has yet been found to replace dental amalgam for children with frank carious lesions on their posterior teeth. Amalgam is still a valuable material in pediatric dentistry because of its superior physical properties, ease of manipulation, and low cost. Nevertheless, given the current uncertainty about the safety of dental amalgam especially in young children, a sound basic guiding principle should be to keep mercury exposure as low as reasonably achievable while maintaining quality of care. Accordingly, it must be recognized that caries prevention and early intervention are mandatory to reduce the use of dental amalgam. Strategies to prevent or arrest dental caries in children are widely known and available but underused, and more must be done to promote the use of evidence-based preventive clinical measures, such as dental sealants. At the community level, water fluoridation remains a very effective measure to prevent dental decay (US DHHS, 2000). In addition, research efforts should focus on developing effective and affordable treatment alternatives to dental amalgam.

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