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Human Exposure to Mercury and Silver Released from Dental Amalgam Restorations

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ABSTRACT. In 35 healthy individuals, the number of amalgam surfaces was related to the emission rate of mercury into the oral cavity and to the excretion rate of mercury by urine. Oral emissions ranged up to 125 μg Hg/24 h, and urinary excretions ranged from 0.4 to 19 μg Hg/24 h. In 10 cases, urinary and fecal excretions of mercury and silver were also measured. Fecal excretions ranged from 1 to 190 μg Hg/24 h and from 4 to 97 μg Ag/24 h. Except for urinary silver excretion, a high interplay between the variables was exhibited. The worst-case individual showed a fecal mercury excretion amounting to 100 times the mean intake of total Hg from a normal Swedish diet. With regard to a Swedish middle-age individual, the systemic uptake of mercury from amalgam was, on average, predicted to be 12 μg Hg/24 h.

DENTAL AMALGAM RESTORATIONS continuously leak small amounts of metals (e.g., mercury) into the oral cavity, as has been reported by several studies.[1] Elemental mercury vapor is released by direct vaporization from metallic mercury contained in the amalgam.[2-13] The mercury vapor emission rate may be increased temporarily when the amalgam surfaces are scratched or pressurized by teeth grinding or by chewing. In addition, the intake of various beverages and foods may influence the short-term release of mercury. On a long-term basis, however, the systemic uptake of mercury, as the body burden, is mostly dependent on the actual load of amalgam and is also influenced by the mean ratio of oral-to-nasal breathing. During strict oral breathing, the released mercury vapor is either exhaled or forwarded to the lungs, where it is resorbed readily by the blood. During strict nasal breathing, a large fraction of the released mercury vapor is probably transformed and bound to components in the saliva coatings before being swallowed. The remaining elemental mercury, however, is probably resorbed directly through the oral and alimentary mucosa.[14] Free elemental mercury, being uncharged and mono-atomic, is a highly mobile species capable of entering most of the body compartments.

Oxidized mercury and silver originate from corrosion processes that occur on the amalgam surfaces, processes that, for example, are influenced by amalgam composition, presence of gold restorations, saliva properties, and by food and chewing habits. Furthermore, amalgam particles are released by abrasion.[15-20]

With respect to risk assessments of long-term inorganic mercury exposures, biological monitoring using the "morning urinary mercury concentration" (corrected for dilution) is normally applied and is a well-established index that, at equilibrium conditions, reflects the kidney burden of mercury. The use of the "urinary mercury excretion rate," based on integrated 24-h samples, although sometimes difficult to accomplish in practice, should be an even better index because of its lower between-day variability. Several studies have been published that deal with the relationship between the load of amalgam and the urinary level of mercury.[10,11,12,21-27]

Compared with blood,[3,4,11,23,24,26–30] the urine matrix has, for assessing inorganic mercury exposure, the advantage of being influenced to only a small extent by simultaneous methylmercury exposure. The confounding effect from methylmercury should, however, be minimized, by studying the plasma mercury concentration or by monitoring the level of inorganic mercury in whole blood.

The systemic uptake of mercury from inorganic mercury species swallowed with the saliva is commonly considered to be small, which is one reason for the low number of published studies that deal with the monitoring of fecal excretion. As already shown by Stock[28] and Frykholm,[14] however, the total amount of mercury passing the gastrointestinal tract is comparatively large, even for individuals with a moderate load of amalgam restorations.

During the last decade, several studies on the release of mercury from dental amalgam and mercury levels in various body fluids and organs related to amalgam have been published.[1] Only a few studies, however, provide data for the calculation of a balance of mercury released from dental amalgam.

The primary aim of this study was to estimate the relationship between the number of amalgam restorations, the emission of mercury into the oral cavity, and the corresponding excretion of mercury in urine and feces. Given that silver is another main constituent in all dental amalgams, and in many chemical and toxicological respects shows similarities to mercury, the related silver excretion was also measured.

Our approach, which involved two studies, to the issue of systemic uptake of mercury from amalgam was that the uptake, at equilibrium, could be more accurately estimated from the combining of exposure and excretion data than from assumptions related to intake patterns only.

Materials and methods

Subjects. Study 1 subjects comprised 10 healthy individuals (4 males, 6 females) who had no occupational exposure to mercury. The number of amalgam surfaces, daily emission of mercury from the amalgam into the oral cavity (O-Hg), and diurnal excretion of total mercury and silver by urine (U-Hg, U-Ag) and feces (F-Hg, F-Ag) were measured. Individuals were selected to represent a broad range of amalgam loading. They were requested to avoid eating fish during the course of the investigation. All subjects were nonsmokers at the time of the study. There was no definite information regarding their breathing patterns or occurrence of bruxism. For example, 1 individual (No. 2, Table 1) did not agree with his dentist, although indications of bruxism were reported. The number of amalgam surfaces was examined by a dentist using the scale from 1 to 6 (an amalgam crown was counted as 6 surfaces). None of the subjects had been treated by a dentist during the 2 mo prior to sampling. One of the subjects, who had a moderate load of amalgam restorations, was subjected to some additional introductory tests concerning the speciation of F-Hg (described later in this article).

In Study 2, 32 healthy individuals (12 males, 20 females; age range = 30–58 y) were examined in the same way and by the same methods as in Study 1, with the exception of feces sampling and urine silver analysis. The number of amalgam surfaces (*N*) ranged from 6 to 84 (median = 35); oral air emission rates ranged from 0.4 to 84 μg O-Hg/24 h (median = 16); and urinary excretion rates ranged from 0.5 to 8.1 μg U-Hg/24 h (me-

Table 1.—Emission Rate of Elemental Mecury into the Oral Cavity (O-Hg), and Excretion Rates of Mercury and Silver via Urine (U-Hg, U-Ag) and Feces (F-Hg, F-Ag) among Individuals with a Varying Load of Amalgam Restorations

				Mercury rates (μg Hg/24 h)					Silver rates (μg Ag/24 h)	
				Emission into the oral cavity			Excretion by urine	Excretion by feces	Excretion by urine	Excretion by feces
Individual	Sex*	Age (y)	No. of amalgam surfaces *n*†	Oa-Hg	Ow-Hg	O-Hg‡	U-Hg	F-Hg	U-Ag	F-Ag
1 (ref)	f	(15)	0	0	0	0	0.4	1	1.3	4
2	m	46	18	19	21	20	1.8	99	4.4	37
3	m	53	21	20	20	20	2.2	53	1.7	22
4	f	43	36	23	18	21	2.6	45	6.0	. . .
5	f	31	38	22	20	21	4.5	27	1.7	11
6	m	40	40	23	35	29	6.9	120	5.3	49
7	f	41	57	32	26	29	4.0	64	1.5	29
8	f	42	60	36	38	37	7.7	47	1.6	29
9	m	47	68	46	73	60	7.8	120	2.3	53
10	f	57	82	125	122	124	19.0	190	1.4	97
Median value:		43	39			25	4.2	58	1.7	29
Range:		(31–57)	0–82			0–124	0.4–19	1–190	1.3–6.0	4–97

Notes: Data, presented in increasing order of *n*, originate from study 1.
* f = female, m = male.
† An amalgam crown was counted as six surfaces.
‡ O-Hg is the paired mean value of Oa-Hg ("oral air") and Ow-Hg ("oral water").

dian = 2.4). The Pearson correlation coefficients were calculated to $r = 0.80$ ($p < .0001$) and $r = 0.75$ ($p < .0001$) for O-Hg and U-Hg versus N, respectively.

The two studies were combined. As a result, 35 independent observations for O-Hg versus N and 42 independent observations for U-Hg versus N were obtained.

Emission rate of elemental mercury vapor into the oral cavity. Previous experience provided from Study 2 demonstrated that the release of elemental mercury vapor from amalgam is, to a great extent, influenced by the present condition of the amalgam surfaces. For instance, the surface layers are easily affected by all kinds of mechanical action. Therefore, to attain a low between-day variation in the O-Hg emission rate with the same individual, a proper standardization of the oral environment prior to sampling is recommended.

Two entirely different methods of sampling were applied to estimate the emission rate of elemental mercury (Hg°) from the amalgam surfaces. With one method, a gas phase analyzing technique, the oral air was passed through a direct-reading UV detector for mercury vapor until a steady-state value was established. The other method, a wet sampling technique, was based on covering all amalgam surfaces with a portion of water for a fixed period of time to collect all the elemental mercury vapor emitted.

The procedures for the first sampling method (Fig. 1) were as follows: a mouthpiece was placed directly behind the subject's teeth and held tightly in place by the lips. Oral air was passed (1.50 l/min) through a cold trap for removal of moisture into a mercury vapor detector (single-beam, modified Zeiss UV instrument with Hg lamp; light path length = 1 m). Previous tests have shown that the emission rate of Hg° vapor is not influenced by variations in the air sampling flow in the range from 1 to 3 l/min. Higher flow rates are, however, not appropriate because of cooling effects on the amalgam surfaces, and they also lead to low Hg° vapor concentrations in the gas cell. Lower flow rates generate recovery problems. Replacement of oral air was provided through the nose.

In an effort to diminish the risk of the amalgam surfaces becoming coated by saliva during sample, a low-flow line for saliva removal, ending in the bottom of the mouth, was built in as an integrated part of the mouthpiece.

Sampling was completed when the UV readings had reached a steady- state level, normally accomplished within 5 to 10 min. The Hg° vapor emission rate into the oral cavity was calculated from this equilibrium reading and from the sample flow rate.

For calibration purposes, the UV cell was supplied with Hg° vapor/air mixtures from a test gas generation system. At the outlet of the UV cell, a 30-ml gas wash bottle containing 10 ml sulfuric acid/permanganate solution (0.1 mol $KMnO_4$/l, pH = 1) for sampling the Hg° vapor was connected on-line. Determination of the mercury content in the gas wash bottle was performed by a standard cold vapor AAS procedure,[31,32] the calibration of which was based on a standard mercury (II)-nitrate solution. In addition, supplemental tests of the instrument readings, based on vapor pressure data for metallic

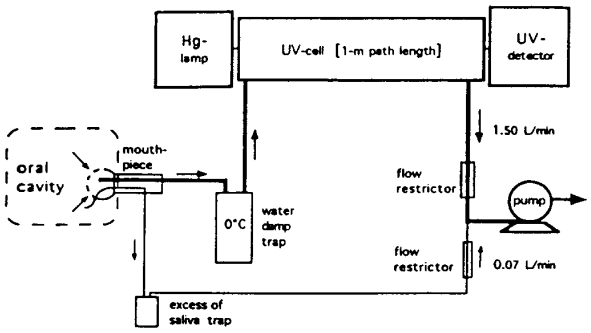


Fig. 1. Outline of a technique for sampling and analyzing mercury vapor emissions from amalgam restorations in situ.

mercury (a headspace technique), showed consistent results. The lowest quantifiable Hg° vapor concentration, 1 μg Hg°/m³, corresponds to a Hg° vapor emission rate into the oral cavity of approximately 2 μg Hg°/24 h. This Hg° emission rate is referred to as O_a-Hg (oral-air mercury). The coefficient of variation for the between-day variation in O_a-Hg for an individual with an average load of amalgam was calculated to be 20%.

The second sampling method used a 25-ml portion of distilled water (37 °C), which was immediately gently moved around in the mouth covering the teeth for 2 min. The aqueous sample, in which emitted elemental mercury vapor was expected to be initially dissolved and then partly oxidized or bound to saliva components, was completely spat out into a 10-ml portion of sulfuric acid/permanganate solution (0.3 mol $KMnO_4$/l, pH = 1). Two subsequent samples were taken for each subject, and the calculated mean Hg emission rate is expressed as O_w-Hg (oral-water mercury).

Previous tests have shown that sampling time may be 1.5 to 3 min. Shorter sampling times give rise to sampling time errors, whereas the use of longer sampling times may lead to low recoveries, probably caused by the trapped Hg° vapor beginning to escape from the aqueous sample. The lowest quantifiable emission rate was approximately 0.5 μg O_w-Hg/24 h. The coefficient of variation for the between-day variation in O_w-Hg for an individual with a moderate load of amalgam was 20%.

One subject with a moderate load of amalgam restorations completed additional tests. In duplicate, the silver content of the oral rinsing water was, after freeze drying and wet digestion of the residual with concentrated nitric acid (150 °C, 2 h), determined, using an AAS graphite furnace technique (external laboratory: Analytica AB, Sweden). The purpose of searching for silver species present in the aqueous rinse sample was to reveal a possible occurrence of accompanying mercury species from corrosion or amalgam particles.

Prior to both sampling methods, subjects were instructed to eat only a very light breakfast containing no eggs, and thereafter to brush their teeth properly without toothpaste. During the hour prior to sampling, subjects were requested to avoid talking or biting. Immediately prior to sampling, the teeth were rinsed carefully, using

two portions of distilled water (37 °C), and the excess of the last portion of water was removed completely by swallowing.

Excretion rates of total mercury and silver in urine. Subjects were requested to collect all urine voided during a 24-h period. The subsamples were collected in polyethylene bottles charged with 1 g sulfamic acid as preservative, a procedure that allows a urine sample to be stored at room temperature for more than 1 wk without any loss of mercury. Aliquots of 1.0 ml from the integrated urine sample were wet digested at room temperature by the addition of sulfuric acid/permanganate solution (0.3 mol $KMnO_4$/l, pH = 1), and the content of total mercury was determined using the standard cold AAS technique.[32,33] In addition, an analytical quality control, using commercially available freeze-dried urine standard samples (Seronorm standard by Nycomed AS, Norway) was performed. The lowest quantifiable excretion rate was approximately 0.2 μg U-Hg/24 h, corresponding, on average, to 0.16 μg U-Hg/l of urine using 24-h samples.

Advantages of using the biological index of U-Hg excretion rate (24 h) instead of the U-Hg concentration related to creatinine concentration are that data are directly applicable to uptake and body burden calculations; data are mutually comparable regardless of gender; and the individual between-day coefficient of variation should be only 5% to 15%, compared with 15% to 25% for creatinine-adjusted morning spot samples, according to our unpublished results from individuals with amalgam restorations. Variations in the creatinine excretion rate by gender, muscle weight, and habits of living, in addition to analytical errors in the determination of creatinine, contribute to the lower precision.

Silver content in urine was determined, using an AAS graphite furnace technique (external laboratory: Analytica AB, Sweden). The lowest quantifiable excretion rate was approximately 0.6 μg U-Ag/24 h, corresponding, on average, to 0.5 μg U-Ag/l of urine, using 24-h samples.

Excretion rates of total mercury and silver in feces. Subjects were instructed to define their starting point just after an arbitrarily chosen defecation and to collect two of the consecutively voided samples, while recording overall time.

All fecal samples were immediately weighed and frozen until analysis. After thawing, homogenization, and freeze drying, duplicates of weighed subsamples (0.2–0.3 g) were treated with concentrated nitric acid. The content of mercury and silver was determined simultaneously, using an ICP technique by an external laboratory (Biospectron AB, Sweden). Standard samples of pig kidney (BCR, Brussels, Belgium) were used as references. The excretion rates of mercury and silver, expressed as μg/24 h, were calculated proportionally from the overall time.

The lowest quantifiable excretion rate was approximately 3 μg F-Hg/24 h (corresponding to approximately 30 ng F-Hg/g wet weight of feces) and 0.8 μg F-Ag/24 h (corresponding to approximately 8 ng F-Hg/g wet weight of feces).

An integrated fecal sample, obtained from an amalgam-free subject during a 10-d period, was collected to obtain a well-supported mean value for the intake of mercury and silver by food.

The ICP method lacks sensitivity for the determination of mercury; therefore, the integrated fecal sample from the amalgam-free subjects was analyzed in duplicate, using a radiochemical technique (external laboratory: Force Institutterne AS, Copenhagen, Denmark). Standard samples of oyster were used as references. The lowest quantifiable excretion rate with this technique was approximately 0.1 μg F-Hg/24 h, corresponding to approximately 1 ng F-Hg/g wet weight of feces.

Consumption of fish from lakes (i.e., exposure from methylmercury, the dominant fraction of which is directly resorbed by the body but then exhibits a slow clearance rate) should have only a long-term influence on Hg excretion. Short-term (between-day) variations in F-Hg are, for example, caused by occasional use of chewing gum, by altering the oral-to-nasal breathing pattern or bruxism during illness or stress, and by the daily variation in food habits and connected chewing patterns. These sources for variability are probably more important with the increase in number and age of the amalgams.

In addition, irregularity in defecation should be expected to create difficulties in obtaining a representative 24-h excretion sample. It is, therefore, recommended that three or more consecutive defecations be used before calculating the 24-h excretion values.

Speciation of mercury in feces. In one subject with a moderate load of amalgam restorations, two additional speciation tests were conducted with fresh fecal samples: extractable mercury (assumed to be methylmercury) and easily purged mercury (assumed to be free elemental mercury). The total mercury content was determined simultaneously.

In order to measure extractable mercury in feces, duplicate weighed portions (approximately 5 g) of fecal sample were, after being thinned out with a 10-fold amount of distilled water, suspended vigorously for some minutes with sodium bromide/hydrochloric acid solution (2 mol Br/l, pH = 0) to release the mercury species from the sulfhydryl bonds. The supposed organic mercury bromide species then formed were extracted according to a modified cleanup procedure for methylmercury in fish samples described by Westöo et al.[34]

The final determination of extractable mercury was, after a re-extraction into 5 ml of aqueous solution containing cysteine-HCl (10 g/l), performed, using the standard method for total mercury based on AAS technique.[32] Depending in part on parameters chosen for the extraction procedures, extractable mercury levels of approximately 1 ng Hg/g wet weight of feces should be detected.

Easily purged mercury was determined using duplicated weighed portions (approximately 10 g) of fecal sample that was thinned out with a 10-fold amount of distilled water and purged by bubbling with nitrogen (approximately 1 l/min) during 2 h into a 30-ml wash bottle containing 10 l of sulfuric acid/permanganate solution (0.1 mol $KMnO_4$/l, pH = 1). Mercury content of the washing solution was determined by standard cold vapor AAS technique.[31,32] The lowest limit for quantita-

tive determination of mercury in the washing solution was approximately 2 ng Hg°, corresponding to approximately 0.2 ng Hg°/g wet weight of feces.

Results

The number of amalgam surfaces, O-Hg, U-Hg, U-Ag, F-Hg, and F-Ag are summarized in Table 1 for the 10 subjects included in Study 1. A correlation matrix, containing variables selected from Table 1, shows a high level of interplay among variables, except for U-Ag.

The relationships between O-Hg and U-Hg and the number of amalgam surfaces and for U-Hg versus O-Hg were estimated, using data from the combined group (Studies 1 and 2). In one case (Subject No. 10, Table 1, who had a heavy load of deteriorated fillings), the O-Hg emission rate and, consequently, the U-Hg excretion rate, were extremely high relative to the actual number of amalgam surfaces. This outlier was excluded when the regression lines in Figures 2 and 3 were calculated, and it was also excluded from the correlation matrix presented in Table 3. There was, however, no reason to exclude this subject when relating U-Hg rates to O-Hg rates (Fig. 4).

Pearson correlation coefficients for the subvariables O_a-Hg and O_w-Hg were calculated separately to $r = 0.91$ ($n = 35$ cases; $p < .0001$).

The relationships between the fecal excretion rates of mercury and silver versus the number of amalgam surfaces are plotted together with the adherent lines of regression on Figure 5. On average, the mercury content of feces was about twice that of silver. The excretion rates of F-Hg versus F-Ag with the adherent line of regression are plotted on Figure 6, exhibiting a very close relationship between these variables.

The speciation of fecal mercury, performed in the prestudy where the tested subject had a moderate load of amalgam (No. 4, Table 1), led to the following conclusions. Extractable mercury in feces (assumed to be methylmercury) did not exceed 0.2% of the total content of

Table 2.—Correlation Matrix (Pearson's *r*), Including Six Variables Selected from Table 1*

	n	O-Hg	U-Hg	U-Ag	F-Hg	F-Ag
n	–					
O-Hg	**.83**†	–				
	(.003)					
U-Hg	**.84**	**.97**	–			
	(.003)	(< .0001)				
U-Ag	−.20	−.18	−.12	–		
	(.63)	(.66)	(.76)			
F-Hg	**.66**	**.84**	**.80**	.34	–	
	(.05)	(.003)	(.007)	(.38)		
F-Ag	**.74**	**.93**	**.91**	.16	**.97**	–
	(.02)	(< .0001)	(.002)	(.69)	(< .0001)	

Notes: n = number of amalgam surfaces; O-Hg = emission rate of Hg° into the oral cavity; U-Hg, U-Ag, F-Hg and F-Ag, i.e. the excretion rates of mercury and silver by urine and feces; and *p*-values appear in parentheses.
* Nine cases were used in this computation. One case omitted due to missing Ag-data.
† A figure set in boldface indicates a significant correlation at the 95% confidence level.

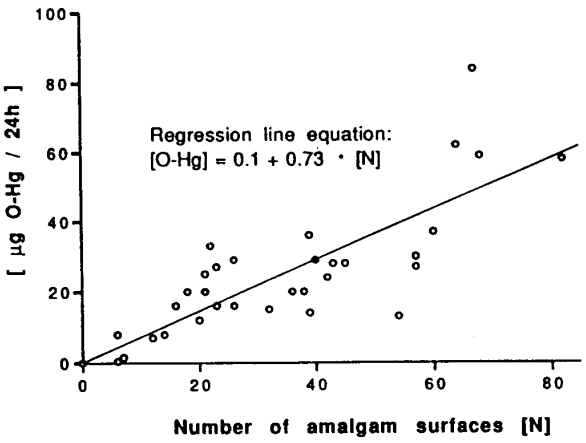


Fig. 2. Relationship between the emission rate of elemental mercury into oral cavity (O-Hg) and the number of amalgam surfaces (34 observations from Studies 1 and 2).

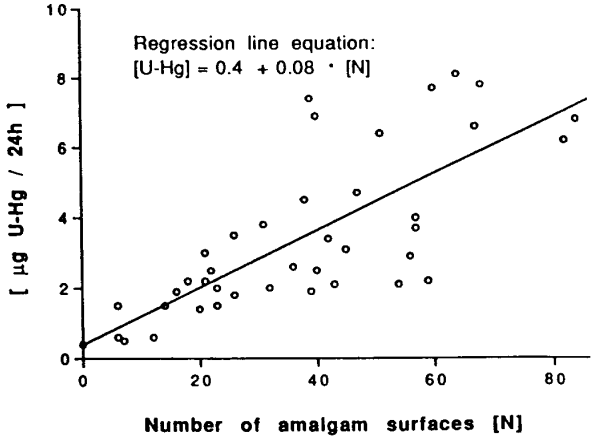


Fig. 3. Relationship between the excretion rate of urinary mercury (U-Hg) and the number of amalgam surfaces (41 observations from Studies 1 and 2).

F-Hg (< detection limit), and thus did not exceed an excretion rate of approximately 0.1 μg F-Hg/24 h. Easily purged mercury in feces (assumed to be free elemental mercury) did not exceed 0.04% of the total content of F-Hg (< detection limit), and thus did not exceed an excretion rate of approximately 0.02 μg F-Hg°/24 h. Consequently, the results from this pre-study suggest that F-Hg excretions are mainly composed of mercury in amalgam particles and inorganic mercury bound to various sulfhydryl group–containing species.

Discussion

Number of amalgam surfaces. A median of 36 restored surfaces was found in the combined group (Studies 1 and 2). The selection of cases was, however, probably somewhat biased toward more heavily loaded individuals because participation in the research was apparently more attractive to such persons. In similar studies, median values for the number of amalgam surfaces ranged from 21 to 39 surfaces.[7,9,12,13,21,26,27] In two recent

Table 3.—Correlation Matrix (Pearson's *r*), Including Three Variables from the "Combined Group" Data (Studies 1+2)

	n	O-Hg	U-Hg
n	–		
O-Hg	**.82†** (< .0001)	–	
U-Hg	**.80** (< .0001)	**.84** (< .0001)	–

Notes: *n* = number of amalgam surfaces; O-Hg = emission rate of Hg° into the oral cavity; U-Hg = excretion rate of urinary mercury; and *p*-values appear in parentheses.
* Thirty-four cases providing connected O-Hg and U-Hg data were used in this computation.
† A figure set in boldface indicates a significant correlation at the 95% confidence interval.

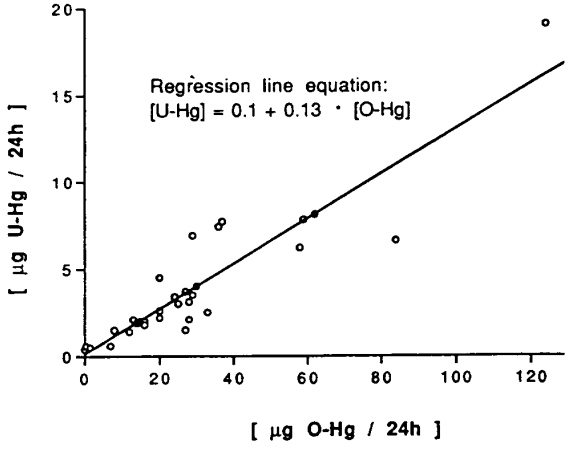


Fig. 4. Relationship between the excretion rate of urinary mercury (U-Hg) and the emission rate of elemental mercury into the oral cavity (O-Hg) (35 observations from Studies 1 and 2).

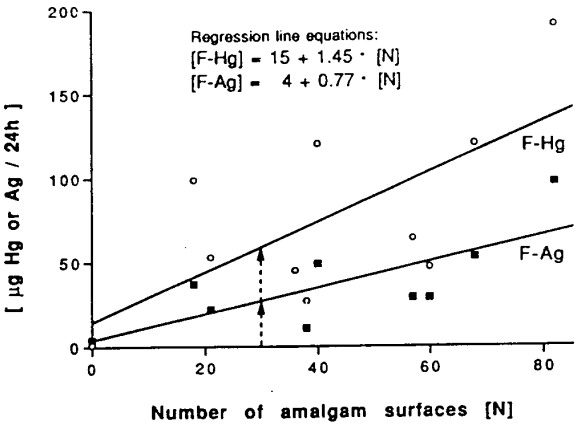


Fig. 5. Relationships between the fecal excretion rates of total mercury (F-Hg) and silver (F-Ag) related to the number of amalgam surfaces (10 and 9 observations, respectively, from Study 1). ○ = F-Hg; ■ = F-Ag; the dotted arrows indicate the predictions of excretion rates for a middle-aged individual with an average load of amalgam restorations (30 amalgam surfaces).

Swedish studies, dental personnel have shown median values of 25[25] and 29[27] surfaces. Therefore, it is reasonable to choose a reference value of 30 surfaces as an average load for a Swedish middle-age population when predicting means of various dependent Hg levels in the body. As a result of improvements in prophylactic dental care with the younger generation and considering the declining number of teeth in elderly people, the mean number of restored surfaces should be much lower when averaging the entire population.

Number of amalgam surfaces as a variable might, at first sight, be regarded as a rather rough estimate to describe the potential source of mercury emissions. In some studies, this variable has been replaced and compared with restored areas, points, or number of occlusal surfaces. No major improvement in the variability of the dependent variables was obtained by using these replacement variables. It is likely that there is background noise from so many other factors contributing to the variability, e.g., differences in the original composition/weight (basic Hg content), age (depletion of Hg), and the extent of cracking/corrosion (active releasing surface).

Mercury emitted into the oral cavity. Various experimental set ups and evaluation techniques have been used to determine the release of Hg° vapor from amalgam into the oral cavity. Emission rates of O-Hg related to the amalgam load have been reported to range from 0 to 110 μg Hg°/24 h (median values = 5–17 μg Hg°/24 h).[2,3,6,7,9,10,12] The present study exhibited, on average, a slightly higher emission rate. A basic (unstimulated) O-Hg mean value of 23 μg Hg°/24 h for 30 amalgam surfaces might be predicted from Figure 2. The conclusion is that each amalgam surface, on average, is associated with a basic emission rate into the oral cavity of approximately 0.7 μg Hg°/24 h.

It should be quite reasonable to assume that individuals with many amalgam surfaces should also have overall larger surfaces (macroscopically, microscopically by

corrosion, and by weight), thus giving O-Hg emission rates exceeding those expected from a linear relationship. The coefficient of determination was, however, not markedly favored by using a nonlinear model for O-Hg versus number of amalgam surfaces.

In this study, O-Hg was determined by two entirely different methods. The close agreement between the two methods gives support for believing that the averaged O-Hg rates are highly reliable. With regard to the method for which a rinsing solution sample is used, the very low content of silver found in the residuals of the sample (< detection limit, corresponding to an emission rate not exceeding 1 μg Ag/24 h) further confirms that the contribution of mercury from amalgam particles or corrosion products to the (elemental) mercury content of the water sample should be negligible.

An experimental set up for achieving a proper UV recording of the O-Hg emission rate is rather expensive and should be operated by trained personnel. Therefore, the method providing a simple water trap for the sampling step might be a method of choice for out-of-laboratory purposes. This does not mean that the determination of O-Hg rate is the most appropriate way in the assessment of amalgam mercury exposure. In addition to many reasons for individual variations, the basic unstimulated O-Hg emission rate measured by these methods is monitored during strict experimental conditions and does not provide unambiguous support for deciding the actual uptake of elemental mercury on an individual basis. In Figure 7, the real emission rate of Hg° from the oral cavity is mentioned only as "O-Hg + ?"

Mercury excreted in urine. A quantifiable urinary mercury excretion was observed, even in the individual without amalgam restorations. This excretion is assumed to reflect the long-term systemic uptake of mercury by air and food. It might be concluded that amalgam-free individuals normally show U-Hg excretion rates below 1 μg Hg/24 h unless a prolonged and extensive consumption of fish from lakes is present (Table 4).

The urinary mercury excretion rate, according to Table 1 (Study 1) and Figure 3 (Studies 1 and 2), increased with increasing number of restored surfaces. U-Hg excretion of 2.8 μg Hg/24 h was predicted from Figure 3, using 30 surfaces as a reference mean value. This and other related studies, summarized in Table 4, give evidence for the relationship between U-Hg rate and number of surfaces being significant. In Molin,[23] the lack of significance at the 95% level was explained by the author as being caused by the small sample of individuals or by the fact that equilibrium conditions had not been attained.

It might also be concluded, from the data in Table 4, that the U-Hg excretion rate, on average, is increased by approximately 0.1 μg U-Hg/24 h per restored amalgam surface. Our use of the scale from 1 to 6 for the number of surfaces (instead of 1–5 as usual) and our use of inte-

Table 4.—A Standardized Outline of Some Studies on Urinary Excretion of Mercury (U-Hg), Related to the Load of Amalgam (*n*).

Source	U-Hg excretion rates for amalgam-free individuals (μg/24h)			Estimation of the slope for U-Hg = *f*(*n*) (μg/24h, *n*)	Correlation between U-Hg and *n*			Remarks*
	Range	*Mdn*	*m*		*r*	*p*	*m*	
Nilsson et al.[21]	na	na	na	0.09	.52	< .04	41	†
Olstad et al.[22]	0.1–0.5	0.3	9	0.09	.52	—	64	All children †
Aronsson et al.[10]	na	na	na	0.12	.50	.03	20	All women ‡
Molin et al.[23]	0.3–1.7	0.6	8	0.15	.66	.07	8	Data prior and 3 months after amalgam inseration †
Molin et al.[24]	0.2–1.3	0.7	10	0.1	.38	.10	20	Data prior and 12 months after amalgam removal †
Skare et al.[25]	na	na	na	0.08	.43	< .0001	314	Dental personnel only‡
Berglund[11]	0.2–0.8	0.5	5	0.1	.81	< .0001	20	Gender not specified by the author†
Langworth et al.[26]		1.5§		0.1	.49	< .0001	68	Data were transposed into a linear model †
Åkesson et al.[27]	0.2–0.6	—	5	na	—	< .05	81	Exponential model used in the original paper†
Jokstad et al.[13]	0.0–3.5	2	22	0.1	.55	< .001	146	†
Present study		0.4§		0.08	.77	< .0001	41	‡

Notes: n = number of amalgam surfaces; *m* = number of tested individuals; na = not applicable; Mdn = median value; *r* = coefficient of correlation; and *p*-level of significance.

* For standardization, the following conversion factors applicable on group level with mixed adult populations were used: 1 μg Hg/24 h ≈ 0.8 μg Hg/l ≈ 0.7 μg Hg/g creatinine = 0.4 nmol Hg/mmol creatinine. For the populations of females and children, mean creatinine excretion values of 1.1 and 0.8 g/24 h, respectively, were used.

† Original data were based on morning spot samples corrected for dilution by creatinine analysis or by density.

‡ Original data were based on integrated 24-h samples.

§ From regression line intercept.

grated 24-h samples (leading to, according to our unpublished data, excretion rate values being 10% lower than the usual creatinine-adjusted rates calculated from morning spot samples only), resulted in the slope coefficients from this study and from Skare et al.[25] to appear in the lower part of the slope coefficients (β = 0.08–0.15).

Use of data from the present study and from Skare et al.,[25] resulted in the conclusion that the 24-h U-Hg excretion rate is not significantly influenced by the urinary flow rate or by gender. The correlation coefficients for U-Hg (μg/24 h) versus urinary flow (l/24 h) and gender (code: male = 1, female = 0) were calculated to $r = -0.20$ ($p = .22$) and $r = -0.07$ ($p = .66$), respectively, in the present study ($n = 42$ cases), and to $r = 0.03$ ($p = .55$) and $r = 0.07$ ($p = .20$), respectively, in Skare et al. ($n = 314$ cases).[25]

The relationship between the urinary excretion rate and the basic emission rate of Hg° vapor into the oral cavity was shown to be very high (Table 2). The O-Hg rate is, as expected, a better estimate of the release of elemental mercury than is the number of surfaces.

An issue of concern is to what extent elevated O-Hg rates, which experimentally have been observed after chewing, are reflected in U-Hg levels. In the study by Skare et al.,[25] approximately 40% of the 314 dental personnel were classified as "permanent frequent chewers," using chewing gum most of the day. Assuming a threshold elevation of the O-Hg rate during 8 h of the day, their mean U-Hg rate was, on average, expected to be doubled, i.e., increased by an additional 2.5 μg U-Hg/24 h. Use of multiple regression analysis, however, revealed that the increase in the U-Hg rate associated with chewing was only 0.5 μg U-Hg/24 h. One explanation is that the simultaneous stimulation of salivation when chewing causes the main fraction of the excessively released elemental mercury to be initially dissolved in the saliva; perhaps it is then oxidized partly or simply bound to sulfur-rich components in the saliva before being forwarded to the gastrointestinal tract, where the systemic uptake of such mercury species is poor.

Urinary excretion values, due to amalgam, that exceed 15 μg U-Hg/24 h are rare. There are individuals, however, with no obvious source of mercury exposure other than their amalgam, who show urinary excretions up to 50 μg U-Hg/24 h, according to Sällsten et al.[35] Excessive bruxism, aged and cracked fillings, and extreme mouth breathing patterns were some reasons proposed to explain these high U-Hg levels.

Urinary excretion levels rising to approximately 75 μg U-Hg/24 h (50 μg U-Hg/g creatinine) are considered "tolerable" for workers occupationally exposed to mercury vapor.[36] A quotation from the World Health Organization (WHO) report[1] is appropriate: "A consequence of an immunological etiology is that it is not scientifically possible to set a level for mercury, e.g., in blood or urine, below which mercury-related symptoms will not occur."

The health-based occupational exposure limit of 25 μg Hg°/m³ proposed by the WHO[36] corresponds, at equilibrium on a group basis, to a mean urinary mercury excretion of about 45 μg U-Hg/24 h (≈ 30 μg U-Hg/g creatinine).[1] A daily 8-h Hg° vapor exposure at 25 μg

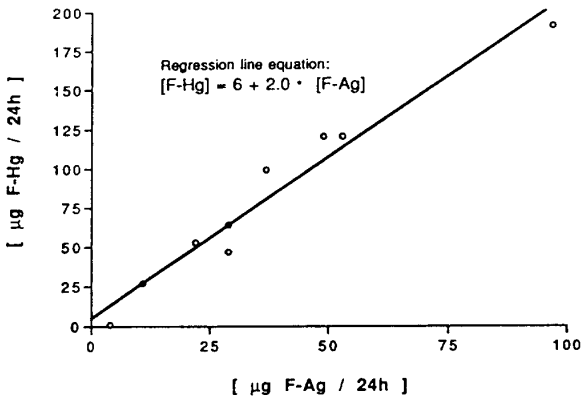


Fig. 6. Relationship between the fecal excretion rate of total mercury (F-Hg) and silver (F-Ag) (9 observations from Study 1).

Hg°/m³ is consistent with a systemic uptake of 175 μgHg/24 h (25 [μg Hg°/m³] × 18 [l/min] × 60 [min] × 80% [retention efficiency]). The difference between this daily uptake value and the corresponding urinary excretion (45 μg U-Hg) should, at equilibrium, mainly be excreted by feces (i.e., 130 μg Hg), giving an estimate for the excretion ratio U-Hg:F-Hg of 1:3. This estimation is not inconsistent with results from human Hg° exposure studies reported by Cherian et al.[37]

If the establishment of this U-Hg:F-Hg ratio is correct, it offers a convenient way for estimating the systemic uptake of inorganic mercury from U-Hg excretion data only: *the daily uptake of mercury equals four times the diurnal U-Hg excretion rate.*

Mercury excreted by feces. Fecal excretions contain mercury species originating from food, amalgam restorations, and previous uptake of mercury (Fig. 7).

The fecal excretion rate of total Hg in this study was significantly related to the number of amalgam surfaces (Table 2, Fig. 5) and was, on average, 20 times higher than the corresponding U-Hg excretion rate. In Table 2, the correlation coefficient between F-Hg and U-Hg is shown to be high.

In a previous unpublished study, one individual with a moderate load of amalgam was intensively chewing gum 4 h daily during 1 wk. At the end of the week, it was found that the fecal total Hg excretion rate was approximately doubled because of an elevated O-Hg level and increased abrasion.

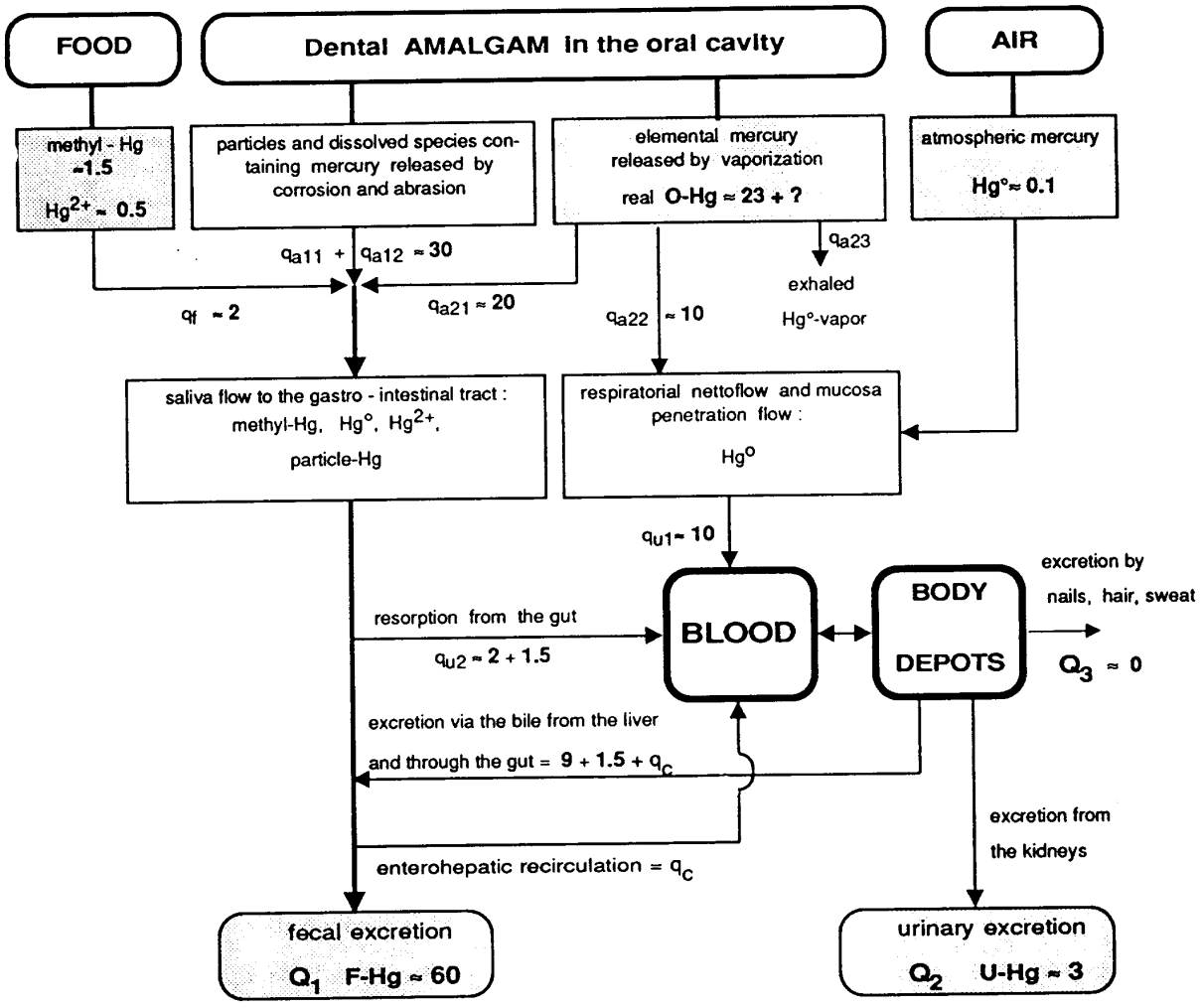
In a recent paper by Becker and Kumpulainen,[38] it was reported that the average daily intake of total mercury from a normal Swedish diet might be 1.8 μg Hg. This value is consistent with the 10-d mean F-Hg excretion rate for the amalgam-free individual (Table 1). Even for an individual with a moderate load of amalgam, the most dominant fraction of fecal mercury excretion should evidently originate from the amalgams. The highest rate of fecal excretion found in this study was as much as 100 times higher than the fecal excretion rate shown by the amalgam-free subject.

According to a report from the WHO,[39] a provisional maximum limit for the daily intake of total mercury by

food is set at 300 μg Hg/wk (≈ 45 μg Hg/24 h). The "acceptable" daily intake should, however, be 0 μg, according to the same report. This provisional limit value was originally extrapolated from data related to methylmercury exposures from food; however, the guideline for the intake of total Hg is in force. As evident from Figure 5, even individuals with a moderate load of amalgam are predicted to show fecal mercury excretions exceeding the WHO dietary standard. By intense chewing or bruxation, the intake rate levels of total mercury will be further elevated.

Mercury arriving in the gastrointestinal tract might be subjected to many biotransformations, such as red/ox and demethylation, in close interplay with uptakes and excretions of mercury from previous exposures.[1] Methylation of inorganic mercury by intestinal bacteria has been demonstrated in vivo.[40-43] The result from our prestudy into the speciation of F-Hg does not, however, suggest any flow of methylated Hg species via feces. Not even the intake of methylmercury provided by food was observed, probably because of the demethylation process.

Hg excretions originating from amalgam may also be considered from an environmental viewpoint. In repeated studies of the effluents of mercury in sewage pipelines from residential quarters, published informally



q_f = intake of total-Hg from food ≈ 2

q_a = intake of total-Hg from amalgam ≈ 60

q_u = systemic uptake of Hg $[q_{u1} + q_{u2}]$ ≈ [amalgam-Hg (10 + 2) + food-Hg 1.5] ≈ 14

Fig. 7. Flow chart of estimated intake, uptake, and excretion flows of mercury originated from air, food, and dental amalgam restorations applied to individuals in equilibrium with a moderate load of amalgam restorations (30 surfaces). Bold figures indicate flows expressed as μg Hg/24 h. Shadowed areas indicate flows that have been subjected to quantification by real measurements. O-Hg° = elemental mercury to the oral cavity; U-Hg = total mercury by urine; and F-Hg = total mercury by feces.

by the local government's Stockholm Water Company, mass flows of mercury, consistent with our Hg data, were shown. Calculated to the entire Swedish population (8.5×10^6 individuals, 50% of whom average 30 restored amalgam surfaces), fecal-urinary excretions to the environment containing 100 kg Hg per year might be suggested. Today, the Swedish population is loaded with amalgam containing approximately 100 tons of mercury.

Silver excreted by urine and feces. No significant correlation was found between the urinary silver excretion and the number of amalgam surfaces. Urinary silver rates were in the range from 1.3 to 6 μg U-Ag/24 h (Table 1). The amalgam-free individual, exposed to silver only via food, was not distinguishable from the other subjects. If analytical errors could be excluded, the following conclusions are indicated: (a) amalgam Ag is absorbed to a very low extent; (b) food Ag species is absorbed readily; and (c) the content of Ag in a Swedish diet amounts 5–6 μg Ag/d (U-Ag + F-Ag with the amalgam-free individual, Table 1).

In Figure 5, total silver excretion in feces is plotted versus the number of amalgam surfaces. The excretion rate of F-Ag was, on average, about one-half of the corresponding F-Hg rate. The strong relationship between F-Hg and F-Ag (Table 2, Fig. 6) gives further support for the fecal mercury being closely connected to the presence of amalgam restorations.

Amalgam restorations contain mercury and silver at the time of insertion in a ratio of approximately 1:0.7 by weight. Assuming a constant composition of the surface layers for years (some depletion of mercury might be expected), about one half of the mercury flow through the gastrointestinal tract should originate from corrosion products and/or amalgam particles calculated from the F-Ag and the total F-Hg excretion rates. The following calculation may be performed for an individual with 30 amalgam surfaces: [27 μg (total F-Ag/24 h) − 4 μg (food-Ag/24 h)]: $0.7 \approx 30$ μg F-Hg/24 h ($30 = q_{a11} + q_{a12}$ in Fig. 7). Consequently, the other half of the F-Hg, probably excreted as inorganic Hg-sulfhydryl species, should originate from elemental amalgam mercury, of which a fraction has been resorbed initially.

Uptake of mercury. The systemic uptake of mercury from amalgam through inhalation, oral mucosa resorption, and some gastrointestinal resorption of mercury species swallowed in the saliva is distributed to various organs and compartments in the body. Different approaches to the estimation of the daily mercury uptake from amalgam have demonstrated mean uptake rates ranging from 2 to 25 μg Hg/24 h.[1,6,7,10,11,13,44] With respect to most of the compartments, rather long biological halftimes for the clearance of mercury should be expected. There is even evidence for compartments (e.g., brain tissue) where attainment of equilibrium is extremely slow, perhaps several years.[1]

The kidneys are considered to contain the dominant fraction of the body burden of mercury; therefore, the use of urinary excretion rates is probably the best biological index to assess long-term exposure to inorganic mercury. After equilibrium in the kidneys, attained within 3 to 5 mo at a constant mercury exposure, the corresponding U-Hg excretion rate reflects the kidney burden of mercury. The U-Hg rate does not, however, predict the amount of mercury accumulated in the very slow clearance compartments. Empirical data including the duration of exposure are needed for this.

The corresponding U-Hg excretion rate offers a useful tool for a rough estimation of the kidney burden of mercury, after equilibrium is attained with a certain Hg^0 exposure. For example, the U-Hg rate for an individual with a moderate load of amalgam (i.e., 3 μg U-Hg/24 h) is consistent with a kidney burden of approximately 160 μg Hg. This result is obtained by integration of the exponential U-Hg clearance decay curve to infinity, assuming a one-compartment model and a first-order kidney clearance kinetics with a biological halftime of 40 d.[45,46] Using the same model, the U-Hg rate with our most extreme case (individual No. 10, Table 1) should be consistent with a kidney burden close to 1 000 μg Hg. In a summary by the WHO,[1] experimental findings of kidney burden mercury related to amalgam in the range of 100 to 200 μg Hg are mentioned.

In Figure 7, an outline of estimated mass flows of mercury is shown for an individual in equilibrium with a moderate load of amalgam (30 surfaces). The daily gross balance of total Hg is 60 μg Hg and the daily uptake of Hg is approximately 12 μg Hg, excreted as F-Hg (9 μg) and U-Hg (3 μg). This amalgam mercury exposure might be regarded as equivalent to a daily 8-h occupational air mercury exposure of 2 μg Hg^0/m^3.

Calculations and related discussions on the issues in this article often focus on data connected with the average individual. However, there should be many single individuals with a high load of amalgam who are exposed to mercury levels that greatly exceed the mean. Using the same model as above, the systemic uptake of mercury in our worst case individual might be estimated to be approximately 70 μg Hg/24 h. This value, implying a very long-term continuous exposure, is not too far from the occupational uptake limit value set by the WHO[1] calculated to be 175 μg Hg/24 h and also exceeds the food limits given by the WHO in 1972.[39]

Appendix

The following are basic assumptions and calculations supporting the proposed mass balance of mercury (outlined in Fig. 7) for a Swedish middle-aged individual with an average load of amalgam (30 surfaces).

Breathing of environmental air contributes to an uptake of approximately 0.1 μg Hg/24 h. Calculation: 5 ng Hg^0/m^3 (air concentration) × 15 l/min (breathing rate) × 60 min × 24 h × 80% (retention).[1]

The contribution of total Hg from food is, for normal fish consumers, estimated to be 2 μg Hg/24 h, about two thirds of which are present as methylmercury species.[38]

The uptake of mercury from the intestines is considered to be 5% to 10% for inorganic mercury species (Hg-S-R), 90% for methylmercury species, and less than 1% for amalgam particles.[1]

The total daily systemic uptake of amalgam mercury may, at equilibrium, be estimated from the formula: 4 × U-Hg (urinary excretion rate, μg U-Hg/24 h) (see text).

The sum of particle Hg (q_{a11}) and corrosion Hg (q_{a12}) may be calculated from the basic amalgam composition and F-Ag data to be approximately 30 μg Hg/24 h.

At equilibrium, by definition, the accumulation rate equals the clearance rate.

Calculation schedule (all figures expressed in μg Hg/24 h)

U-Hg ≈ 3. Then, the systemic uptake of Hg from amalgam (the contribution from food and air neglected) is 4 × U-Hg ≈ 12.

If the gastrointestinal uptake from amalgam ≈ 2 and from food ≈ 1.5, then $q_{a1} = q_{a22} \approx 10$ (12 − 2).

If $q_{a11} + q_{a12} \approx 30$ (calculated from Ag data), then $q_{a21} \approx 20$ [from an amalgam Hg balance for the gastrointestinal tract: q_{a21} + 2 (food Hg) + 30 (q_{a11-12}) + 9 (bile/gutexcretion) = 2 (gutresorption) + 60 (Q_1)].

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References

1. World Health Organization (WHO). Task group on environmental health criteria for inorganic mercury. Inorganic mercury. Environmental health criteria series No 118. Geneva: WHO, 1991.
2. Svare CW, Peterson LC, Reinhardt JW, et al. The effect of dental amalgams on mercury levels in expired air. J Dent Res 1981; 60:1668–71.
3. Abraham JE, Svare CW, Frank CW. The effect of dental amalgam restorations on blood mercury levels. J Dent Res 1984; 63:71–73.
4. Ott KH, Loh F, Kröncke A, Schaller KH, Valentin H, Weltle D. Zur Quecksilber-belastung durch Amalgamfüllungen. Dtsch Zahnärztl Z 1984; 39:199–205.
5. Vimy MJ, Lorscheider FL. Intra-oral air mercury released from dental amalgam. J Dent Res 1985; 64:1069–71.
6. Vimy MJ, Lorscheider FL. Serial measurements of intra-oral air mercury: estimation of daily dose from dental amalgam. J Dent Res 1985; 64:1072–75.
7. Patterson JE, Weissberg BG, Dennison PJ. Mercury in human breath from dental amalgams. Bull Environ Contamin Toxicol 1985; 34:459–68.
8. Ott KH, Krafft T, Kröncke A, Schaller KH, Valentin H, Weltle D. Untersuchungen zum zeitlischen Verlauf der Quecksilberfreisetzung aus Amalgam-füllungen nach dem Kauen. Dtsch Zahnärztl Z 1986; 41:968–72.
9. Berglund A, Pohl L, Olsson S, Bergman M. Determination of the rate of release of intra-oral mercury vapor from amalgam. J Dent Res 1988; 67:1235–42.
10. Aronsson AM, Lind B, Nylander M, Nordberg M. Dental amalgam and mercury. Biol Metals 1989; 2:25–30.
11. Berglund A. Estimation by a 24-hour study of the daily dose of intra-oral mercury vapor inhaled after release from dental amalgam. J Dent Res 1990; 69:1646–51.
12. Björkman L, Lind B. Factors influencing mercury evaporation rate from dental amalgam fillings. Scand J Dent Res 1992; 100:354–60.
13. Jokstad A, Thomassen Y, Bye E, Clench-Aas J, Aaseth J. Dental amalgam and mercury. Pharmacol Toxicol 1992; 70:308–13.
14. Frykholm KO. On mercury from dental amalgam. Doctoral thesis, Department of Operative Dentistry, Royal School of Dentistry, Stockholm, Sweden. Acta Odont Scand 1957; Suppl No 22: 1–108.
15. Guthrow CE, Johnson LB, Lawless KR. Corrosion of dental amalgam and its component phases. J Dent Res 1967; 46:1372–81.
16. Brune D, Gjerdet N, Paulsen G. Gastrointestinal and in vitro release of copper, cadmium, indium, mercury and zinc from conventional and copper-rich amalgams. Scand J Dent Res 1983; 91:66–71.
17. Herö H, Brune D, Jörgensen RB, Evje DM. Surface degradation of amalgams in vitro during static and cyclic loading. Scand J Dent Res 1983; 91:488–95.
18. Marek M. Acceleration of corrosion of dental amalgam by abrasion. J Dent Res 1984; 63:1010–13.
19. Brune D, Evje D. Man's mercury loading from a dental amalgam. Sci Total Environ 1985; 44:51–63.
20. Pleva J. Mercury from dental amalgams: exposure and effects. Int J Risk & Safety Med 1992; 3:1–22.
21. Nilsson B, Nilsson B. Mercury in dental practice: urinary mercury excretion in dental personnel. Swed Dent J 1986; 10:221–32.
22. Olstad ML, Holland RI, Wandel N, Hensten-Pettersen A. Correlation between amalgam restorations and mercury concentrations in urine. J Dent Res 1987; 66:1179–82.
23. Molin M, Bergman B, Marklund SL, Schütz A, Skerfving S. The influence of dental amalgam placement on mercury, selenium and glutathione peroxidase in man. Acta Odontol Scand 1990; 48:287–95.
24. Molin M, Bergman B, Marklund SL, Schütz A, Skerfving S. Mercury, selenium and glutathione peroxidase before and after amalgam removal in man. Acta Odontol Scand 1990; 48:189–202.
25. Skare I, Bergström T, Engqvist A, Weiner JA. Mercury exposure of different origins among dentists and dental nurses. Scand J Work Environ Health 1990; 16:340–47.
26. Langworth S, Elinder CG, Göthe CJ, Vesterberg O. Biological monitoring of environmental and occupational exposure to mercury. Int Arch Occup Environ Health 1991; 63:161–67.
27. Åkesson I, Schütz A, Attewell R, Skerfving S, Glantz PO. Status of mercury and selenium in dental personnel: impact of amalgam work and own fillings. Arch Environ Health 1991; 46:102–09.
28. Stock A, Cucuel F. Der Quecksilbergehalt der menschlichen Ausscheidungen und des menschlichen Blutes. Z Anggew Chemie 1934; 47:641–47.
29. Kroncke A, Ott K, Petschelt A. Über die Quecksilberkonzentrationen in Blut und Urin von Personen mit und ohne Amalgamfullungen. Dtsch Zahnärztl Z 1980; 35:803–08.
30. Snapp KR, Boyer DB, Peterson LC, Svare CW. The contribution of dental amalgam to mercury in blood. J Dent Res 1989; 68:780–85.
31. Skare I, Engqvist A. Mercury in air: evaluation of solid adsorbents for monitoring of mercury vapor exposures. Stockholm: National Institute of Occupational Health, 1986 (Arbete och Hälsa 1986:38; in Swedish).
32. Lindstedt G. A rapid method for the determination of mercury in urine. Analyst 1970; 95:264–71.
33. Skare I. Microdetermination of mercury in biological samples. Analyst 1972; 97:148–55.
34. Westöö G. Determination of methylmercury salts in various kinds of biological material. Acta Chemica Scandinavica 1968; 22:2277–80.
35. Sällsten G, Barregård L, Österberg T. Bruxation among persons with amalgam restorations, a reason for elevated urinary mercury excretions. Läkartidningen 1991; 88:232–33. (in Swedish).
36. WHO Expert Group. Recommended health-based limits in occupational exposure to heavy metals. Technical report series No 647. Geneva: WHO, 1980.
37. Cherian MG, Hursh JB, Clarkson TW, Allen J. Radioactive mercury distribution in biological fluids and excretion in human subjects after inhalation of mercury vapor. Arch Environ Health 1978; 3:109–14.
38. Becker W, Kumpulainen J. Contents of essential and toxic mineral elements in Swedish market-basket diets in 1987. Br J Nutr 1991; 66:151–60.
39. WHO/FAO Expert Committee. Evaluation of certain food additives and contaminants. Technical report series No 505. Geneva: WHO, 1972.
40. Rowland IR, Grasso P, Davies MJ. The methylation of mercuric chloride by human intestinal bacteria. Experientia 1975; 31:1064–65.
41. Edwards T, McBride BC. Biosynthesis and degradation of methylmercury in human feces. Nature 1975; 253:462–64.
42. Abdullah M, Arnesjo B, Ihse I. Methylation of inorganic mercury in experimental jejunal blind-loop. Scand J Gastroent 1973; 8:565–67.
43. Ludwicki JK. Studies on the role of gastrointestinal tract contents in the methylation of inorganic mercury compounds. Bull Environ Contam Toxicol 1989; 42:283–88.
44. Clarkson T, Friberg L, Hursh J, Nylander M. The prediction of intake of mercury vapor from amalgams. In: Clarkson T, Friberg L Nordberg GF, Sager P. Biological monitoring of metals. New York: Plenum Press, 1988.
45. Skare I, Engqvist A. Urinary mercury clearance of dental personnel after a long-term intermission in occupational exposure. Swed Dent J 1990; 14:255–59.
46. Barregård L, Sällsten G, Schütz A, Attewell R, Skerfving S, Järvholm B. Kinetics of mercury in blood and urine after brief occupational exposure. Arch Environ Health 1992; 47:176–84.