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Long-term Use of Nicotine Chewing Gum and Mercury Exposure from Dental Amalgam Fillings

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Abstract. In experimental studies, chewing gum has been shown to increase the release rate of mercury vapor from dental amalgam fillings. The aim of the present study was to investigate the influence of long-term frequent chewing on mercury levels in plasma and urine. Mercury levels in plasma (P-Hg) and urine (U-Hg), and urinary cotinine were examined in 18 subjects who regularly used nicotine chewing gum, and in 19 referents. Age and number of amalgam surfaces were similar in the two groups. Total mercury concentrations in plasma and urine were determined by means of cold vapor atomic absorption spectrometry. Urinary cotinine was determined by gas chromatography-mass spectrometry. The chewers had been using 10 (median) pieces of gum *per* day for the past 27 (median) months. P-Hg and U-Hg levels were significantly higher in the chewers (27 nmol/L and 6.5 nmol/mmol creatinine) than in the referents (4.9 nmol/L and 1.2 nmol/mmol creatinine). In both groups, significant correlations were found between P-Hg or U-Hg on the one hand and the number of amalgam surfaces on the other. In the chewers, no correlations were found between P-Hg or U-Hg and chewing time *per* day or cotinine in urine. Cotinine in urine increased with the number of pieces of chewing gum used. The impact of excessive chewing on mercury levels was considerable.

Key words: mercury, amalgam, nicotine chewing gum, biological monitoring.

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Introduction

Dental amalgam fillings release mercury vapor (Hg^0), which is readily absorbed when inhaled. Furthermore, some mercury is dissolved in saliva or swallowed as amalgam particles, a small fraction of which is absorbed in the gastrointestinal tract (WHO, 1991). Other possible routes of absorption are through the oral mucosa (Fréden *et al.*, 1974; Bolewska *et al.*, 1990; Hahn *et al.*, 1990) and the pulpal tissue (Möller, 1978; Schiele *et al.*, 1987; Lentz *et al.*, 1989; Hörstedt-Bindslev *et al.*, 1991). In populations with many amalgam fillings, the fillings constitute the major source of exposure to inorganic mercury (WHO, 1991). The correlation between mercury in blood or urine, on the one hand, and the amount of amalgam in the teeth, on the other, is well-documented (Akesson *et al.*, 1991; Langworth *et al.*, 1991). An association between mercury levels in brain autopsy samples and the number of amalgam surfaces has also been reported (Nylander *et al.*, 1987).

The intra-oral release of Hg^0 from amalgam fillings has been determined by several research groups, either as spot measurements before and after standardized stimulation by chewing or toothbrushing (Abraham *et al.*, 1984; Patterson *et al.*, 1985; Vimy and Lorscheider, 1985; Ott *et al.*, 1986; Aronsson *et al.*, 1989; Björkman and Lind, 1992), or as repeated measurements during 24-hour cycles (Berglund, 1990). Chewing of gum, or toothbrushing, increased the release rate in all experiments. The contribution of dental amalgam to mercury in blood and urine has been investigated after removal of all amalgam fillings (Snapp *et al.*, 1989; Molin *et al.*, 1990). The daily dose of Hg absorbed has been calculated, and most estimates fall below 5 µg/day (Snapp *et al.*, 1989; Berglund, 1990; Olsson and Bergman, 1992). However, we have recently reported three cases of greatly increased mercury levels in blood and urine (from 5 to 20 times), indicating high mercury absorption from dental amalgam fillings, with gum chewing and bruxism as the possible causes (Barregård *et al.*, 1995). The aim of the present study was to investigate the influence on mercury levels in biological media of long-term frequent chewing. We performed a cross-

Table. Background factors, mercury (Hg) concentrations, cotinine, and albumin among chewers of nicotine gum and referents

	Chewers (n = 18[a])				Referents (n = 19)			
	Mean	SD	Median	Range	Mean	SD	Median	Range
Age	50	5	49	43-58	51	7	50	42-61
Amalgam surfaces (n)[b]								
Total	36	13	37	18-61	37	13	35	16-59
Occlusal	10	4	11	4-17	12	3	12	4-17
Fish meals *per* week	1.1	1.0	1.0	0-3	1.2	0.5	1.0	0-2
Hg in urine (nmol/mmol creatinine)	7.2	3.2	6.5[c]	2.8-14	1.5	1.0	1.2	0.4-3.8
Hg in plasma (nmol/L)	26	16	27[c]	5.1-52	5.0	2.4	4.9	1.8-10
Cotinine in urine (µg/mmol creatinine)	251	115	247[c]	101-465	< 10	—	< 10	< 10- 73[d]
Albumin in urine (mg/mmol creatinine)	0.50	0.17	0.46	0.2-0.9	0.65	0.47	0.49	0.3-2.0

[a] n = 17 for P-Hg.
[b] Maximum 5/tooth.
[c] $p < 0.001$.
[d] A snuffer.

sectional study and recruited the chewers from among subjects who regularly used nicotine chewing gum.

Materials and Methods

Study groups

The chewers were recruited from the clientele at nine pharmacies during a four-month period in 1994, with an information letter to customers explaining the aim and the inclusion criteria. The chewers had to have used at least five pieces of nicotine chewing gum *per* day for at least six months. We restricted the age interval to 40 to 66 years to select subjects with at least 15 amalgam surfaces. Subjects with known renal disease, diabetes mellitus, drug-treated hypertension, or occupational exposure to mercury were excluded. Of 21 chewers who contacted our department, 18 fulfilled the criteria.

Nineteen referents with the same distribution of age, gender, and number of amalgam surfaces, and who used chewing gum for a maximum of 30 min *per* week, were recruited during the same time period from among employees at the nine pharmacies and from our own department. Fish consumption was also similar in the two groups (Table). In none of the subjects had dental amalgam been inserted or removed in the previous two months. Within the last six months, three referents and four chewers had had from one to three amalgam fillings removed or inserted. Four chewers and four referents suspected that they were bruxists. Four chewers smoked two or three cigarettes *per* day, one of the referents used wet snuff, and the other referents used no tobacco. Alcohol consumption was low or moderate in both groups (≤ 115 g ethanol/week). The study was approved by the Ethics Committee at the University of Göteborg.

Sampling and analyses

Venous blood samples were collected in metal-free heparinized Venoject™ tubes. Plasma was separated by centrifugation and transferred into metal-free polypropylene tubes. Subjects collected a morning urine sample at home in metal-free polyethylene bottles. The samples, with the exception of an aliquot for the determination of creatinine (modified Jáffé's method), were stored in a deep freezer until analyzed. All samples were coded and analyzed in the same analytical series.

Total mercury in plasma (P-Hg) and in urine (U-Hg) was determined by means of an automized cold-vapor atomic absorption technique (Einarsson *et al.*, 1984) after digestion of the samples with perchloric and nitric acids at 65°C overnight. The procedure was modified (Bergdahl *et al.*, 1995) for higher sensitivity by enrichment (amalgamation) of the mercury vapor on a gold wire filter. All samples were analyzed in duplicate. Spiked samples for calibration and reference samples for testing of accuracy were included in the same series. The detection limit was 0.3 nmol/L. U-Hg was expressed per mmol creatinine to correct for differences in urinary flow rate (Barregård, 1993). The imprecision, calculated as the coefficient of variation from duplicate analyses, was 5% for plasma and 8% for urine. Our finding for the urine reference sample (Quebec Toxicological Centre, H-94-01) was 67 nmol/L, compared with the 'target value' 78 nmol/L. For plasma, a reference sample of lyophilized whole blood (Seronorm™ Trace Elements, batch 010010) and a blood sample from the Quebec Toxicological Centre (M-94-10) were used. Our results, 13.3 nmol/L and 20.3 nmol/L, may be compared with the expected values of 15 nmol/L and 24 nmol/L, respectively.

Cotinine in urine (U-cotinine), a biomarker of nicotine exposure, was determined by means of a gas chromatographic-mass spectrometric method presented previously (Skarping *et al.*, 1988). The imprecision for spiked human urine was 3%, and the detection limit was 10 µg/mmol creatinine.

Albumin in urine, a biomarker of renal glomerular disease, was measured by electroimmunoassay using nephelometry with a detection limit of 1.8 mg/L and expressed *per* mmol creatinine. Before analysis of the thawed samples, 20 µL of Tween 20 was added to 1 mL of urine, and the samples were incubated at 37°C for 2 h.

Statistics

Wilcoxon's rank-sum test was used for the comparison of chewers and referents, since the parameters were not normally distributed, and Spearman's rank correlation coefficient (r_s) was used to express the correlation between single variables. Associations among more than two variables were analyzed by

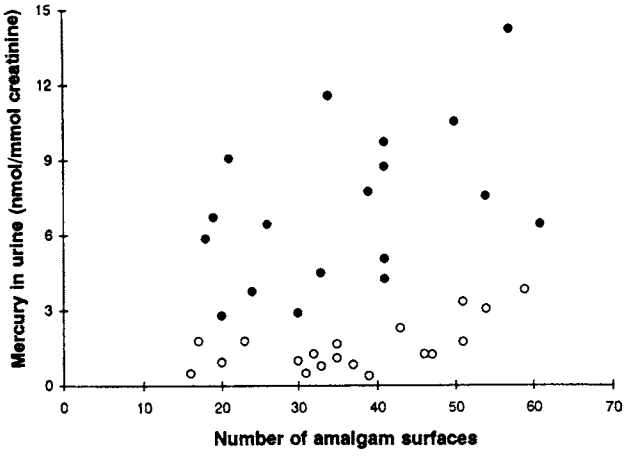

Figure 1. Relation between mercury concentration in urine and number of amalgam surfaces among 18 frequent long-term chewers of nicotine gum (●) and 19 referents (○).

the multiple linear regression technique. 'Statistically significant' refers to $p < 0.05$ in two-tailed tests.

Results

The chewers had used from 5 to 22 (median 10) pieces of nicotine gum *per* day for 27 months (median; range, from 6 to 96 months). This consumption had been relatively constant during the preceding six months. Their estimated chewing time for each piece of gum corresponded to a total chewing time of 5.6 h *per* day (median), with a range of from 1.3 to 15 h *per* day. As expected, U-cotinine was significantly higher in the chewers than in the referents (Table). Six of the chewers also used other types of gum, from 20 to 180 min *per* day.

The levels of P-Hg and U-Hg (Table) were significantly higher in the chewers (27 nmol/L and 6.5 nmol/mmol creatinine) than in the referrents (4.9 nmol/L and 1.2 nmol/mmol creatinine). In both groups, P-Hg and U-Hg were significantly correlated with the number of amalgam surfaces (Figs. 1, 2). For the chewers, the correlation coefficients were 0.75 ($p = 0.0006$) and 0.46 ($p = 0.05$), respectively, and for the referents 0.51 ($p = 0.03$) and 0.49 ($p = 0.03$), respectively. For the chewers, no correlation was found between P-Hg or U-Hg, on the one hand, and the number of pieces of nicotine gum chewed *per* day, the total chewing time (Fig. 3), or U-cotinine, on the other. This was the case even when the number of amalgam surfaces was taken into account in multiple linear regression models. U-cotinine was significantly correlated with the number of pieces of chewing gum chewed *per* day ($r_s = 0.57$, $p = 0.014$). The four referents who suspected themselves to be bruxists had U-Hg and P-Hg levels similar to those of the other referents.

After information about the results, seven of the eight chewers with P-Hg above 25 nmol/L stopped chewing and started to use nicotine plaster or spray instead. After 25 (median) days, their average P-Hg had decreased from (medians) 45 to 18 nmol/L.

The excretion of albumin in urine (Table) was similar in the two groups, and all had values within normal limits.

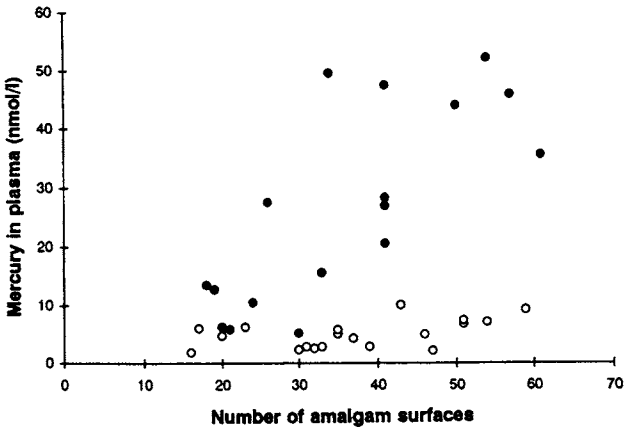

Figure 2. Relation between mercury concentration in plasma and number of amalgam surfaces among 17 frequent long-term chewers of nicotine gum (●) and 19 referents (○).

Discussion

It is well-known that the chewing of gum increases the Hg release rate from amalgam fillings (Abraham *et al.*, 1984; Vimy and Lorscheider, 1985; Ott *et al.*, 1986; Aronsson *et al.*, 1989; Björkman and Lind, 1992). The present study shows that the increase in Hg uptake after long-term frequent chewing is considerable, with mercury levels in both plasma and urine five times higher than those in referents.

In the assessment of metal exposure, quality assurance is very important. The determinations of mercury in plasma and urine were made in a laboratory with extensive experience, and quality control indicates that the accuracy was good. The mercury levels in plasma and urine in our reference group were similar to previously reported values among occupationally unexposed Swedish subjects (Akesson *et al.*, 1991; Langworth *et al.*, 1991). To optimize the possibility of detecting differences between the two groups, we selected chewers with at least six months of regular use of nicotine chewing gum. With a half-time for mercury in urine of about two months (WHO, 1991), an exposed individual would reach nearly 90% of steady-state within a six-month period. Mercury in urine and plasma is mainly inorganic (WHO, 1991; Barregård, 1993), and the influence of fish consumption is insignificant (Akesson *et al.*, 1991). These media, therefore, are suitable for the assessment of Hg exposure from amalgam fillings.

The obvious explanation for the increased P-Hg and U-Hg among chewers is increased uptake of mercury from their dental amalgam. This was further proven by the fact that decreased P-Hg levels were seen in those seven individuals who quit chewing. In a recent case report, high mercury levels in both urine (from 15 to 30 nmol/mmol creatinine) and whole blood (from 60 to 120 nmol/L) were found in three subjects without occupational mercury exposure (Barregård *et al.*, 1995). Their use of chewing gum was considered a possible explanation for the high Hg levels. After removal of the amalgam fillings in two of the individuals, the Hg levels decreased to normal values. Among dental personnel, only a minor influence from use of chewing of gum was seen on U-Hg levels (Skare *et al.*, 1990). Many of those individuals,

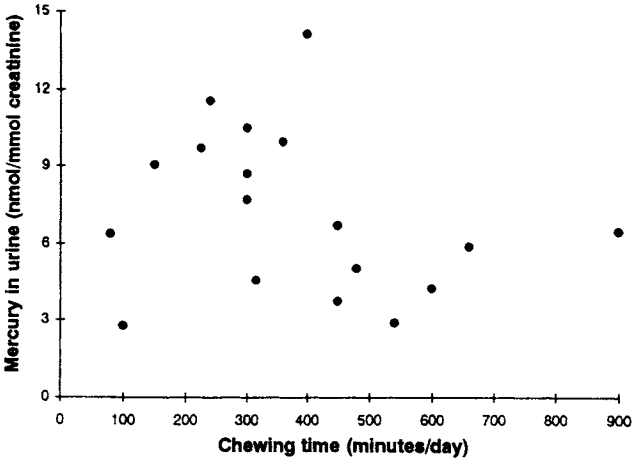

Figure 3. Relation between mercury concentration in urine and total chewing time among 18 frequent long-term chewers of nicotine gum.

however, may not have been long-term, frequent chewers.

The mechanisms underlying the Hg release during/after chewing are not well-understood. The mechanical pressure *per* se is one possible factor (Björkman and Lind, 1992). Moreover, mercury inside the amalgam filling diffuses and reaches the surface, where a concentration gradient of Hg prevails (Olsson *et al.*, 1989). The concentration gradient on the surface can be disturbed by various processes, such as the chewing of gum or toothbrushing, resulting in an increased mercury release rate, and the surface can also be passivized, resulting in a decreased release rate (Abraham *et al.*, 1984; Patterson *et al.*, 1985; Vimy and Lorscheider, 1985; Aronsson *et al.*, 1989; Berglund, 1990; Björkman and Lind, 1992). No data are available on possible differences between various (nicotine or other) types of chewing gum, with respect to the increase in the mercury release rate.

Inhalation of mercury vapor is most likely the major route of exposure from amalgam (WHO, 1991; Berglund, 1993), with an uptake of about 80% in the lungs. Mercury vapor may, however, also be absorbed across the mucous membranes of the oral cavity or through the pulpal tissues, and enter the blood. In animal studies where radioactive ^{203}Hg was used for amalgam fillings, uptake was found in the gum mucosa and jaw tissue (Hahn *et al.*, 1990). Some mercury may be absorbed in the gastrointestinal tract from swallowed amalgam microparticles and dissolved mercury vapor or mercuric ions in saliva. Inorganic mercury compounds are, however, absorbed to only a small extent (< 10%) in the gastrointestinal tract in adults (WHO, 1991).

The absence of associations, within the group of chewers, between mercury levels in urine or plasma and the indices of chewing can well be explained by uncertainty in the estimated chewing parameters. The stated number of pieces of gum *per* day is probably relatively reliable, but the chewing time for each piece of gum is not. Furthermore, chewing intensity is not accounted for. The chewers in this study used ten (median) pieces of gum *per* day, and their U-cotinine excretion was similar to that of active smokers (Willers *et al.*, 1992). Maybe the chewing time in most subjects was enough to maintain an increased release rate of Hg throughout the day. Thus, in an experimental study, the intra-oral Hg release rate reached steady-state within approximately 10 min after the start of chewing, and declined slowly after cessation of chewing to initial levels over a period of 90 min (Vimy and Lorscheider, 1985).

The mercury levels found here in chewers' plasma and urine were much higher (4 times) than the median reported for Swedish dental personnel, our country's largest group occupationally exposed to mercury (Akesson *et al.*, 1991). In three out of 18 chewers, we found U-Hg levels in excess of 10 nmol/mmol creatinine. In Sweden, levels of this magnitude are rare (probably about 1/1000) in the general population (Barregård *et al.*, 1995), and they are normally seen only in the group with high occupational exposure, consisting of fewer than 100 chloralkali workers. In a Swedish population study (N = 3794; age, 16 to 80 years) of nicotine replacement therapy, 362 persons (10%) had used nicotine chewing gum at some time (Ramström, 1994), and 11 (0.3%) of the current users had been chewing nicotine gum for at least six months. About 40% of the Swedish population aged 16 to 80 years have ≥ 15 amalgam surfaces (Bjerner and Sundberg, 1994). If the result of the present study is representative, approximately 1000 to 1500 (3/18*0.3%*6.6 milj*0.40%) individuals may have exposure of the same magnitude as the highest-occupationally-exposed workers. Moreover, other types of chewing gum may have similar effects. Thus, chewing gum may be the most frequent cause of high mercury levels in urine and plasma in Sweden.

Biological monitoring of mercury in blood and urine can be used to assess exposure to mercury vapor and the risks of adverse effects (Barregård, 1993). In workers exposed long-term to mercury, with average U-Hg levels of about 50 μg/g creatinine (28 nmol/mmol creatinine), there is generally no overt clinical impairment, but the urinary excretion of certain proteins and the prevalence of symptoms and slight objective changes in the central nervous system (psychometric tests) are increased (WHO, 1991; Barregård, 1993). Some studies suggest slight effects at even lower levels (Soleo *et al.*, 1990; Ngim *et al.*, 1992). The American Conference of Governmental Industrial Hygienists (ACGIH) has adopted a biological exposure limit for inorganic mercury in urine of 35 μg/g creatinine (20 nmol/mmol creatinine), corresponding to the occupational exposure limit for mercury vapor of 25 $\mu g/m^3$ (ACGIH, 1994-1995). The urinary mercury levels found in the chewers in the present study (from 2.8 to 14 nmol/mmol creatinine) were below this limit, and also below the levels where adverse effects have been shown. Since the limit for occupational exposure is based on data from long-term, occupationally exposed humans, an appropriate safety factor of 10 may be used to recommend a limit for the general population (Clarkson, 1992). This would result in a limit for U-Hg of 2 to 3 nmol/mmol creatinine, which is somewhat higher than the average levels found in populations with amalgam fillings (Akesson *et al.*, 1991; Langworth *et al.*, 1991). The safety margin for long-term frequent nicotine gum chewers is much smaller. Dental amalgam and nicotine chewing gum are, however, used as 'pharmaceutical drugs' to prevent illness, and acceptable risk levels depend on the benefits associated with the use (Klaassen, 1986). Since it is well-known that smoking is dangerous, the fact that frequent chewing may increase

exposure to mercury from amalgam fillings should not prevent people from quitting smoking. Today, nicotine replacement therapy is also available in patch or spray form. For people with many dental amalgam fillings who quit smoking and need nicotine supplementation for a very long period, these forms are preferable.

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