The influence of dental amalgam placement on mercury, selenium, and glutathione peroxidase in man

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Amalgam restorations were inserted in eight healthy persons, previously unprovided with dental restorations, who had several severe carious lesions. The mean number of surfaces restored were 16.1 (range, 11 to 22). The total mean calculated amount of mercury inserted was 2.9 g (range, 1.5 to 4.3 g). Blood and urinary levels were measured on seven occasions during a 4-month period before and a 3-month period after amalgam placement. One and 3 months after placement, the P-mercury mean values were almost equal to the preplacement values (3.3 nmol/l). After placement U-mercury increased continuously; 3 months after placement a statistically significantly higher ($p < 0.05$) mean U-mercury value (0.58 nmol/mmol creatinine) was found compared with the mean preplacement value (0.34 nmol/mmol creatinine). No statistically significant correlation was found between the P- and U-mercury concentrations and the total number of amalgam surfaces. Selenium levels in plasma and urine and erythrocyte glutathione peroxidase showed no systematic change of pattern. The results show that the insertion of amalgam fillings contributed to the U-mercury concentration, but apparently even more extensive amalgam therapy and/or longer exposure periods are needed to affect the P-mercury concentration. No negative effects on the P- and U-selenium or the erythrocyte glutathione peroxidase levels could be found during the 3 months immediately after an extensive amalgam placement. The supplementary blood and urine analyses were not influenced by the insertion of amalgam fillings. □ *Blood analysis; clinical study; mercury analysis; urinalysis*

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Several studies have shown that mercury vapor from dental amalgam affects the mercury concentration in the oral cavity (1–4), urine (5–7), plasma (8–10), and whole blood (11–13). In 1957 Frykholm (5) published the first, and hitherto the only, study in which a biologic variable has been analyzed after experimental placement of amalgam fillings in man (5). The participants had previously been provided with ordinary amalgam fillings. His data showed radioactive mercury in the urine after placement of fillings containing radioactive mercury. During the follow-up period of 2 weeks the urinary excretion of radioactive mercury from the amalgam seemed to cease.

It has been claimed that mercury release from amalgam fillings is the cause of a series of subjective symptoms, but so far investigations have not been able to correlate these symptoms to the presence of amalgam fillings (14–17). However, some allergic (18, 19) and local hypersensitivity reactions (20–22) have been related to this mercury release.

The potent interactions between mercury and selenium, mainly demonstrated in animal experiments (23–25) but also in humans (26–28), have initiated discussions concerning whether mercury release from dental amalgam could influence the selenium status in humans.

We here report on a clinical study in which amalgam-free humans were provided with amalgam fillings. Mercury, selenium, and the selenium-dependent enzyme glutathione

peroxidase (GSHPx) were studied. Supplementary blood and urinary analyses were also carried out.

Materials and methods

Eight amalgam-free persons were asked to participate in the present investigation. The subjects consisted of seven men with a mean age of 26.5 years (range, 20–32) and one woman of 21 years. Of the participants five men and the woman were from Iran, one man from Chile, and one man from South Africa. As none of the participants spoke Swedish, the initial contact was performed by both written and oral information in their own language (English or Persian). Further communication during the total treatment procedure was performed via a translator. The project was approved by the Ethical Review Committee of the Medical Faculty at the University of Umeå. All participants gave written consent. They had a good general health condition.

Four of the eight were smokers (~20 cigarettes/day), and all participants reported being teetotalers. Fish consumption was approximately one meal a week, consisting of commercially available deep-frozen fish. None of the participants had ever visited a dentist. Their need for dental treatment was obvious, and in some cases acute. None of the participants had been occupationally exposed to mercury vapor, and none of them had taken selenium preparations.

The subjects had lived in Sweden for at least 6 months before amalgam placement. Altogether 129 surfaces in 71 teeth were found to have caries lesions. These 129 surfaces were restored with amalgam. The caries lesions were excavated and temporarily sealed with Dycal® and IRM-cement® (L. D. Caulk Co., Milford, Del., USA). The subsequent placement of amalgam (Dispersalloy, Johnson & Johnson Co., New Brunswick, N.J., USA) was performed at one treatment occasion for each person. During the amalgam insertion a high-volume evacuator was used. The mean number of fillings and surfaces restored was 8.9 (range, 3–12) and 16.1 (range, 11–22), respectively. The estimated total mercury content of the amalgam inserted varied between approximately 1.5 and 4.5 g. Patient 7 had only three teeth restored, but as they were molars with rather extensive loss of substance, there was a relatively large amount of amalgam inserted. Patient 5 had nine fillings. However, as they were small, the calculated amount of mercury inserted in this patient was comparatively small.

Furthermore, the patients were provided with all necessary dental treatment, including periodontal treatment, extractions, endodontic treatment, occlusal adjustment, and, in one case, a three-unit fixed bridge.

Blood and urine samples

Blood and morning urine samples were collected on two occasions, 4 and 3 months before the amalgam insertion and at 1, 5, and 10 days and 1 and 3 months after the amalgam insertion. Blood was collected from the cubital vein in metal-free vacuum tubes (Terumo Venoject) with heparin as the anticoagulant for the mercury assays and with ethylenediaminetetraacetic acid (EDTA) as the anticoagulant for the selenium and GSHPx assays. Apart from the samples intended for blood cell analyses the samples were centrifuged at 2000 *g* for 10 min, and the supernatant plasma was drawn off for further analyses. The buffy coat was discarded. Urine was collected in polyethylene bottles previously washed in 20% nitric acid. Unless the samples were analyzed immediately, they were stored at −80°C until required for analysis.

Analytical procedures

Mercury. The mercury content was determined in wet-digested samples by a 'cold vapor' atomic absorption technique, using automatic equipment (29). The plasma samples (1.0 ml) were digested overnight with nitric and perchloric acids at 65°C (30) and the urine samples (0.5 ml) with potassium permanganate and sulfuric acid at room temperature overnight (31). Both digestion procedures were modified slightly to suit large sample volumes. All samples

were analyzed in duplicate. The detection limit was 0.5 nmol/l in plasma and 1.0 nmol/l in urine. The precision, as calculated from the duplicate analyses, was 11% (coefficient of variation) for plasma samples in the range 1.0–9.9 nmol/l (mean, 4.5 nmol/l; $n = 92$) and 3% in the range 9.9–54.8 nmol/l (mean, 24.4 nmol/l; $n = 12$). The accuracy was checked by analyzing the reference samples. For the plasma control (Seronorm®, Nycomed, Oslo, Norway) the reference value was 5.5 nmol/l, and our results from different runs averaged 6.3 nmol/l (SD, 0.17; range, 5.5–7.8; $n = 6$). The reference value of the urine sample (Lanonorm®, Nycomed, Oslo, Norway) was 48.4 nmol/l, and the results averaged 47.2 nmol/l (SD, 0.80; range, 44.4–59.8; $n = 12$).

Selenium. Plasma and urinary selenium was determined by using a fluorometric method with diaminonaphthalene as described by Lalonde et al. (32).

Erythrocyte glutathione peroxidase. Erythrocyte glutathione peroxidase (Ery-GSHPx) was determined with a coupled assay procedure as described (10). The assays were performed at 37°C, and the activity was expressed as μkat/g hemoglobin in the hemolysates. Hemoglobin was determined by means of a standard cyanomethemoglobin method.

Supplementary analyses. Plasma sodium and plasma potassium were determined by using ion-selective electrodes, plasma calcium as a complex with cresolphthalein purple, and plasma and urine creatinine with an alkaline picrate reagent. Plasma levels of total and conjugated bilirubin were determined with the use of sodium nitrite and sulfanilic acid, and plasma alkaline phosphatase with the plasma nitrophenyl-phosphate assay.

Plasma-γ-glutamyl transpeptidase, aspartate aminotransferase, alanine aminotransferase, lactate dehydrogenase, and creatine kinase were determined by using reagent kits from Boehringer Mannheim AG (Mannheim, FRG). Albumin, α-1-antitrypsin, orosomucoid, haptoglobin, and C-reactive protein in plasma were analyzed by electroimmunoassay. Blood cells and erythrocyte values were analyzed on a Technicon H 6000 apparatus (Technicon Instruments Co., Tarrytown, N.Y., USA), urine albumin by electroimmunoassay, urine protein by the Coomassie Brilliant Blue G-250 procedure (33), urine β_2-microglobulin with a radioimmunoassay kit (β_2-mikro RIA 100) supplied by Pharmacia Diagnostics AB, Uppsala, Sweden, and the osmolality of urine by the freezing-point depression method.

Table 1. Sex, age, no. of teeth, fillings, and surfaces restored

Patient no.	Sex	Age (years)	No. of teeth	No of fillings/ surfaces restored
1	Male	30	32	12/22
2	Male	31	24	8/15
3	Male	20	30	11/22
4	Female	21	31	7/15
5	Male	32	32	9/11
6	Male	20	32	11/18
7	Male	31	31	3/12
8	Male	22	28	10/14

Reference intervals

The P-selenium (0.72–1.47 μmol/l) and Ery-GSHPx (0.63–1.82 μkat/g hemoglobin) reference intervals were obtained from the analysis of samples from 250 persons aged 30, 40, 50, and 60 years in a health survey study in the county of Västerbotten. The U-selenium (0.010–0.053 μmol/mmol creatinine) reference interval was taken from 130 persons in the same study. The data established and used in the Laboratory of Clinical Chemistry, University of Umeå, were used as reference intervals for the other analyses. The reference interval limits presented are in general the 2.5 and 97.5 percentiles.

Statistical methods

For the statistical analyses, the two preplacement values were pooled. A Wilcoxon signed-rank test was used for comparing the blood and urine values. A simple regression analysis was used to compare the P- and U-mercury levels with the number of amalgam surfaces.

Results

Seven of eight patients appeared on all 7 test days. Patient 6 (Table 1) did not show up for the tests 3 months before and 5 days after the amalgam insertion. None of the patients reported any subjective symptoms after the placement of amalgam fillings.

The individual P-mercury values are given in Fig. 1. Before amalgam placement the group mean values on the two sampling occasions were 3.6 nmol/l (SD, 1.4) and 3.2 nmol/l (SD, 1.2). After the amalgam placement there was no obvious pattern of change. Three months after amalgam placement the mean value was 3.3 nmol/l (SD, 1.1).

The preplacement U-mercury mean values were 0.29 nmol/mmol creatinine (SD, 0.26) and 0.39 nmol/mmol creatinine (SD, 0.3) (Fig. 2). For the period 1 day to 3 months after the placement the mean value continuously increased, from 0.27 nmol/mmol creatinine (SD, 0.16) to 0.58 nmol/mmol creatinine (SD, 0.35) ($p < 0.05$).

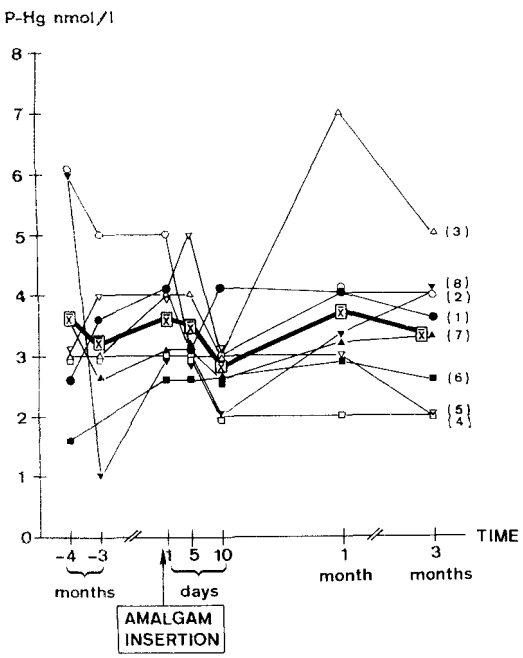

Fig. 1. Separate values for each patient and mean values of plasma mercury before and after amalgam placement. X denotes mean values. Number within parentheses refers to the subject number in Table 1.

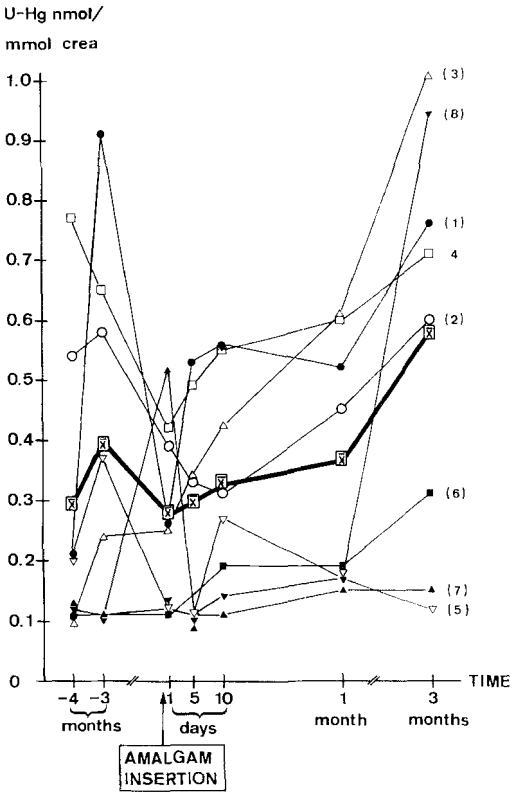

Fig. 2. Separate values for each patient and mean values of urinary mercury before and after amalgam placement. X denotes mean values.

The correlation between the P-mercury and U-mercury values and the number of amalgam surfaces was not statistically significant. The seeming tendency to increased P- and U-mercury concentrations in relation to increased number of amalgam surfaces is due to the values of one person only. His P- and U-mercury concentrations in combination with a relatively large number of amalgam surfaces contribute to the false picture of correlation (Figs. 3A, 3B).

A statistically significant increase ($p < 0.05$) of the mean P-selenium value, seen on the test occasion 5 days after the placement, was followed by a decrease until 10 days after the placement (Fig. 4). Thereafter, there was an increase, and at 1 and 3 months after

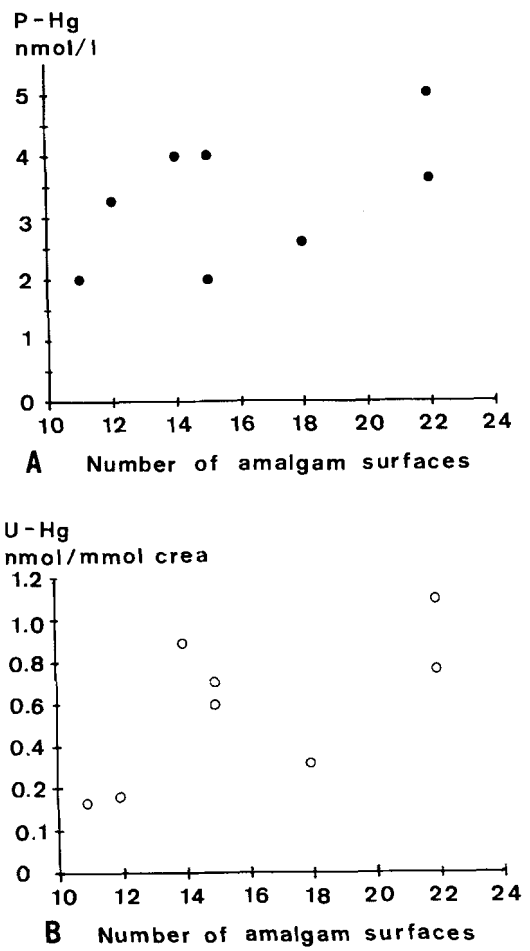

Fig. 3. Plasma mercury (A) and urinary mercury (B) in relation to the number of amalgam surfaces 3 months after the amalgam placement.

placement the P-selenium mean value was statistically significantly higher ($p < 0.05$) than before the placement. On the test occasion 10 days after the amalgam insertion, only one value, 0.65 μmol/l, in patient 3 fell outside the reference interval (0.72–1.47 μmol/l).

With regard to the U-selenium values (Fig. 5), one value, 0.007 μmol/mmol creatinine in patient 8, 3 months after insertion, fell outside the reference interval (0.010–0.053 μmol/mmol creatinine). The mean values at 4 months before and 3 months after amalgam placement were almost identical: 0.023 and 0.024 μmol/mmol creatinine. There was no statistically significant change in the mean values during the 7 months covered by the study.

With regard to the Ery-GSHPx values (Fig. 6) the only female participant (patient 4) had Ery-GSHPx values almost twice as high as most of the male participants during the total period of 7 months. As her values fell outside the reference intervals for the whole period, they were not included in the mean values or in the statistical analyses. No statistically significant difference was found between the pooled preplacement mean value and the postplacement mean values.

As to the supplementary analyses performed, no effect of the amalgam placement could be found with regard to: blood cell variables (erythrocyte particle concentration, B-hemoglobin, mean corpuscular volume, leukocyte particle concentration, platelets, neutrophils, lymphocytes, mono-

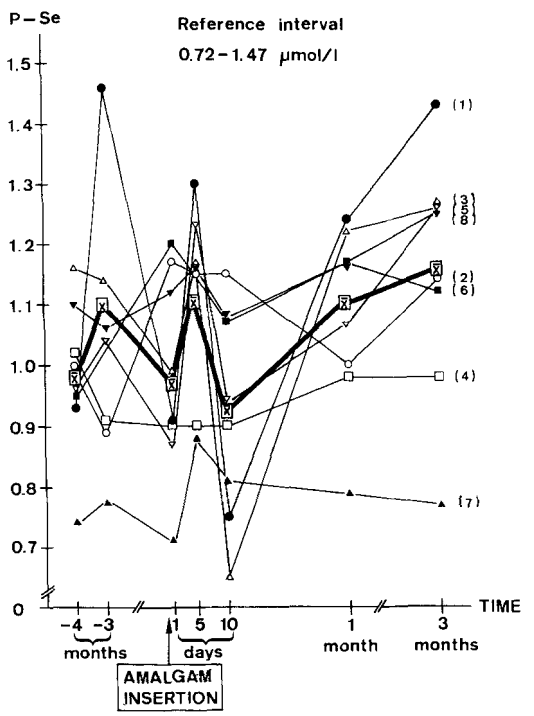

Fig. 4. Separate values for each patient and mean values of plasma selenium before and after amalgam placement. X denotes mean values.

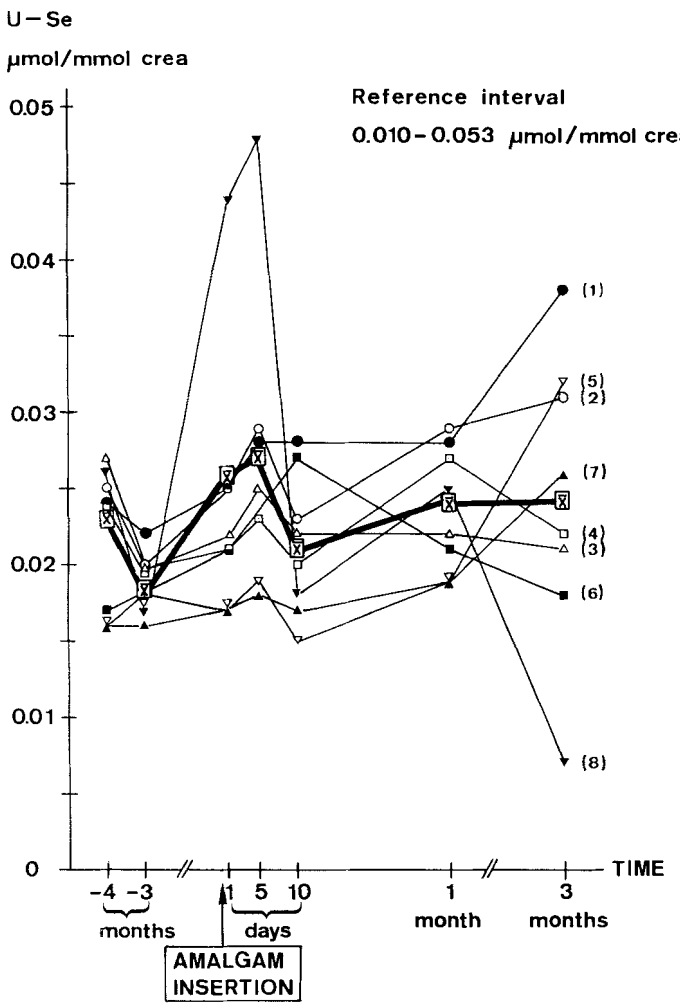

Fig. 5. Separate values for each patient and mean values of urinary selenium before and after amalgam placement. $\bar{X}$ denotes mean values.

cytes, eosinophils, and basophils), plasma electrolytes (P-natrium, P-potassium, and P-phosphate), liver status (P-total bilirubin, P-conjugated bilirubin, P-alkaline phosphatase, P-γ-glutamyl transpeptidase, P-alanine aminotransferase, P-aspartate aminotransferase, and P-lactate dehydrogenase), skeletal muscle status (P-creatine kinase, P-aspartate aminotransferase, and P-lactate dehydrogenase), plasma proteins indicating inflammatory reaction (P-α-1-antitrypsin, P-orosomucoid, P-haptoglobin, and P-C-reactive protein), or IgG.

With regard to the kidney status (U-albumin, U-protein, morning U-osmolality, P-creatinine, and U-β_2-microglobulin) only the U-β_2-microglobulin level was statistically significantly lower immediately after the amalgam placement, and this significant difference remained even 3 months after the placement as compared with the preplacement values ($p < 0.01$). However, the values were all within the reference intervals (4–370 µg/l).

Discussion

In previous studies concerning mercury release from amalgam fillings the participants had, for a long time, been provided with such fillings. The main purpose of the

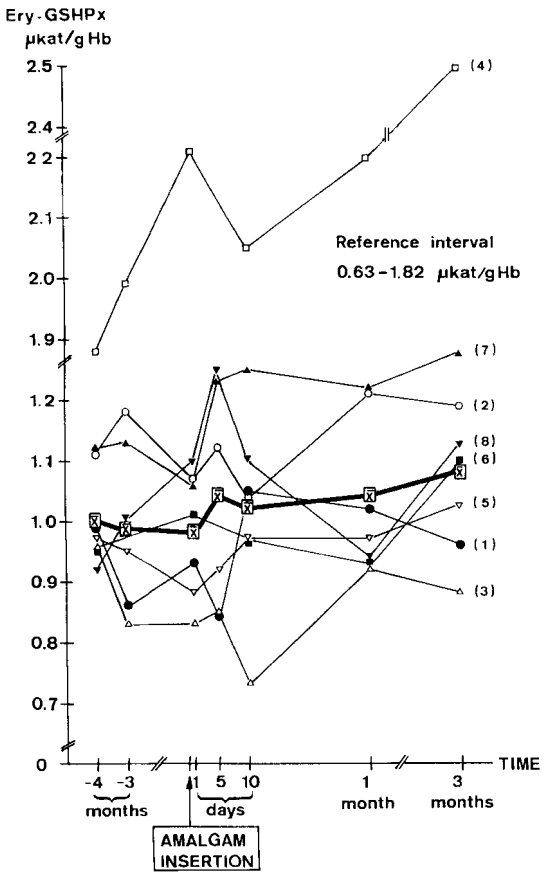

Fig. 6. Separate values for each patient and mean values for the male participants of erythrocyte glutathione peroxidase before and after amalgam placement. X denotes mean values.

present study was to examine possible biologic effects on patients in need of extensive treatment with amalgam but without previous dental amalgam restorations. The number of possible participants available was therefore very limited; in fact, only a few immigrants could fulfill the requirements. The experimental period had to be restricted to 3 months after the amalgam placement because the patients did not consent to give blood for the analyses over a longer period. We were unable to recruit control persons with a similar background, without any amalgam fillings and not in need of any dental treatment.

Fish consumption is an external source known to affect the blood mercury concentration. The methyl mercury from fish affects mainly the erythrocyte mercury concentration (34, 35), whereas U-mercury is not affected. In the present study the fish consumption was equal and limited for all the patients. It therefore seems likely that the contribution of mercury to plasma from this external source was very limited.

The subjects had low P-mercury levels before treatment. Their initial P-mercury concentration was about 25% lower than that in one of our earlier studies, in which the experimental group had been provided with amalgam fillings long before the study started (10). It is well known that mercury vapor is released during the insertion, condensation, and shaping of the amalgam filling (36). The quantities of amalgam inserted was, at least in seven of the patients, more than that normally inserted at one treatment session. The present placement of amalgam fillings was not accompanied by any immediate increase of P-mercury level. This was probably due to optimal mercury hygiene conditions during trituration, condensation, and insertion of amalgam. Nor did we find an expected rise in P-mercury during the postplacement period; the concentration was the same before and 3 months after the amalgam placement. This finding may be explained by the fact that the patients were exposed to mercury from their fillings for only 3 months. It might be that P-mercury is not directly dependent on recent absorption but, at least partly, on recycling from slow body pools, which were not yet affected in our patients.

The U-mercury concentration was low; the initial mean value in the present study was 65% lower than the corresponding value in our earlier study (10). In contrast to Frykholm's 2-week study (5), we did not record any short-term posttreatment increase of the U-mercury concentration. This finding is probably due to a much more cautious technique today, with other types of amalgam in less need of condensation for the purpose of reducing excess mercury and the use of high-volume evacuator. However, during the 3-month period after treatment an increase in the U-mercury concentration

was noted, possibly because chewing and brushing of the amalgam fillings gave rise to a continuous release of small amounts of mercury, partly in the form of vapor and partly ionic. These procedures give an accumulation of mercury in the kidney, and a secondary effect on U-mercury concentration.

The significant increase in P-selenium and the small increase in Ery-GSHPx on day 5 after insertion was unexpected and may be fortuitous. One might speculate that components released from fresh amalgam other than mercury are responsible for these short-term effects. It is known from animal experiments that copper and silver interact with selenium and cause redistribution of selenium (37–41).

No negative effects of the amalgam insertion on P- and U-selenium and Ery-GSHPx were shown; instead continuous increases in P-selenium and Ery-GSHPx values were noted. Starting from relatively low levels, the final 3-month levels were approaching the mean values of the Swedish population in the county of Västerbotten. These increases may be due to dietary effects.

Studies have shown that mercury vapor exposure may lead to increased levels of U-proteins, U-albumins, and some U-enzymes (42–45), indicating a slight glomerular dysfunction. Our finding that the U-β_2-microglobulin concentrations were statistically significantly lower after amalgam placement are contradictory to what might have been expected. Furthermore, since all values were well within the reference interval, this indicates that the biologic significance of the finding probably is of minor, if any, importance.

In conclusion, the present results support the concept that amalgam is an important source of exposure to inorganic mercury without affecting the selenium status in man. Nor did the supplementary blood and urinary analyses reveal any influence of the amalgam fillings during the 3-month follow-up period.

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