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Dental Amalgam and Mercury

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Abstract: The mercury concentrations in blood (HgB) and urine (HgU) samples, and in exhaled air (HgAir) were measured in 147 individuals from an urban Norwegian population, using cold vapour atomic absorption spectrometry. The study aimed to estimate the mercury exposure from the dental restorations, by correlating the data to the presence of amalgam restorations. Mean values were HgB = 24.8 nmol/l, HgU = 17.5 nmol/l and HgAir = 0.8 μg/m^3. HgU correlated with HgAir, and both HgU and HgAir with the number of amalgam restorations, amalgam restored surfaces and amalgam restored occlusal surfaces. HgB showed poor correlation to HgU and HgAir and the presence of amalgam restorations. A differentiation of the mercury absorption due to exposure from dental amalgams and from the dietary intake, necessitates measurements of both organic and inorganic mercury in the plasma, and in the erythrocytes. The results suggest that individuals with many amalgam restorations, i.e., more than 36 restored surfaces, absorb 10–12 μg Hg/day.

In order to estimate the impact of the exposure to mercury in the environment on general health it is necessary to identify, characterize and quantify all contributing sources. One potential source have been shown to be the degradation of dental restorations made from amalgam (Brune & Evje 1985). Dental amalgams consist of approximately 45–50% mercury, 25–35% silver, 2–30% copper and 15–30% tin (Phillips 1986). Various strategies have been used to estimate the exposure levels of mercury from amalgam restorations, based on *in vitro* experiments (Brune & Evje 1985; Okabe 1987; Marek 1989), or measurements in exhaled air or in intraoral air (Gay *et al.* 1979; Vimy & Lorscheider 1990; Berglund 1990). Estimations have also been made from measurements of mercury levels in the urine (Frykholm 1957; Olstad *et al.* 1987), blood (Kröncke *et al.* 1980; Abraham *et al.* 1984), plasma (Molin 1990), or saliva (Ott *et al.* 1984). Few of these studies report the mercury concentrations in both blood and urine or exhaled or intraoral air samples, and the estimates of the daily contribution of mercury from amalgam restorations are contradictorary.

The present study aimed to assess the mercury contents in blood and urine, as well as the mercury amounts in exhaled air in a segment from an urban Norwegian population. A further aim was to estimate the mercury exposure from the dental restorations, by correlating these data to the presence of amalgam restorations.

Materials and Methods

The present study was a sub-study of a larger investigation on environmental exposure to contaminants from traffic in an area within Oslo, Norway (Clench-Aas 1991). One hundred and sixty-two individuals, randomly chosen from cohorts of the larger material, were invited to participate in the present study. After a written consent, each participant visited a central clinic for a series of tests. The group finally consisted of 147 individuals with ages ranging from 3–87 years (median = 33 years). Ninety-four of the participants (63.9%) were females.

The consent permitted one of the authors to obtain data on the dental status from their own dentists. The dental status was assessed in most cases on the basis of X-rays and the patient's record. This mode of assessment was often not possible for a variety of reasons. The prevailing reason was a lack of regular attendance at a particular dental practice. Therefore, the participants with no data available from their dental record were invited to be examined at a university dental clinic. The examination was made by an experienced clinician, using a dental mirror and a probe. Recordings were made of the total number of amalgam restorations, the number of amalgam restored tooth surfaces and the number of amalgam restored occlusal surfaces. No information was available on the possible prior presence of amalgam for the adult participants with no amalgam restorations. No attempt was made to qualitatively score the restorations by tarnish, porosities or marginal degradation.

Blood samples were collected from the cubital vein in vacutainer tubes, tested free of mercury contamination <1 nmol Hg/l, with heparin as anticoagulant. The total blood mercury concentration (HgB) was determined after hemolyzation.

Urine was collected in polystyrene bottles. The participants provided morning urine samples, to reduce the effects of daily fluctuation of excreted mercury (Piotrowski 1975). All samples were refrigerated to 4°, and kept cool until analysis. The HgU was determined by cold vapour atomic absorption spectrometry (Ebbestad 1975), applying a modified LDC mercury monitor model 1205. The detection limit is 0.5 nmol/l, and the precision of the method is 2.0% R.S.D at the 50 nmol Hg/l level. HgB was determined after a nitric/perchloric acid digestion of the samples. The detection limit for mercury in whole blood is mostly dependent on the mercury content of the acids. Throughout this study the detection limit of the method was kept below 2 nmol/l blood. All samples were analyzed in duplicate. Quality assurance materials from Nycomed, Norway (Seronorm Trace Element Whole Blood and Urine) were used as control samples. The mercury concentrations found in these materials was in good agreement with the producers certificate (within $\pm 5\%$). HgU was also adjusted for urine flow rate, by relating the values to the creatinine concentrations.

The pulmonary air samples were collected after the following procedure: Normal breathing was performed for several minutes before a deep breath. After holding the breath for 20 sec., about half was blown out through the mouth, and the remaining air blown into a 3 l plastic air bag. The air bag consisted of an opening valve, into which a cardboard mouth-piece was inserted. This procedure ensures the expiration of intraoral air first, followed by pulmonary

air which was collected in the plastic bag. The air samle was analyzed for mercury (HgAir) immediately after sampling, using the LDC monitor. One air sample was measured from each patient using a continous air flow of 300 ml/min. through the LDC monitor. The detection limit was 0.1 µg/l with a 50% variance and 2% precision at this level. Calibration was made against air standards containing known amounts of mercury vapour.

Correlations between the variables were computed using Pearson correlation coefficients. The correlation coefficients between HgU, HgB and HgAir versus the participants' age and amount of dental amalgam were computed both without and with subgrouping the values. Initial examinations of scatterplots of the data showed that the relationships between the amount of amalgam versus HgU and HgAir were non-linear. Furthermore, the variance of the residuals decreased with less amount of amalgam, i.e. there was an inhomogeneity of the variance. The correlations between the Hg values, gender, age and the amount of amalgam was therefore also estimated in a multiple linear regression model, after log transformation of the Hg values. However, no regression models for the Hg levels were made from the data recorded in the present study. The value of such models would be limited due to the lack of recording important factors affecting the Hg release from amalgam, e.g. the age and quality of the restorations, their technical quality, presence of other metals and other environmental conditions intra-orally, as well as other variables such as smoking, frequency of fish meals and use of medication.

Results

The dental status was recorded in 115 participants, while 32 failed to report the name of their dentist and did not attend the dental examination. The dental amalgam restorations varied between 0–69 restored surfaces (median = 24), 0–20 restored occlusal surfaces (median = 10), and 0–29 restorations (median = 11). Twenty-two participants (19.1%) did not have any amalgam restorations.

Blood assays were submitted from 133 individuals. Samples were not received from the young children (n = 5), and from 9 adults unwilling to participate in the blood sampling. The HgB concentrations varied between 3 and 71 nmol/l, with mean HgB = 24.8 nmol/l (sd = 11.8, median = 23 nmol/l) (fig. 1). Urine samples were analyzed for all the participants, except in one case due to a breakage of the plastic container (n = 146). The HgU values varied between 2 and 80 nmol/l, with mean HgU = 17.5 nmol/l (sd = 16, median = 11 nmol/l) (fig. 1). HgAir samples were received from all 147 participants. The HgAir varied from amounts at the detection limit of 0.1 µg/m^3 up to 9.8 µg/m^3. The mean HgAir was 0.8 µg/m^3 (sd = 1.3, median = 0.3 µg/m^3) (fig. 1).

The frequency distribution curve for the HgB values (fig. 1), having a gaussian like shape, differed from the negative skewness of the distributions of HgU and HgAir values. Furthermore, the HgB values failed to correlate with the HgU values, and correlated only slightly with the HgAir concentrations (table 1). Relatively good correlations were obtained between the HgU, the HgAir values.

The correlation coefficients between the different indices of the amount of dental amalgam and HgU, HgB and HgAir varied only slightly (table 1). Recalculating the correlation coefficients after subgrouping the participants according to

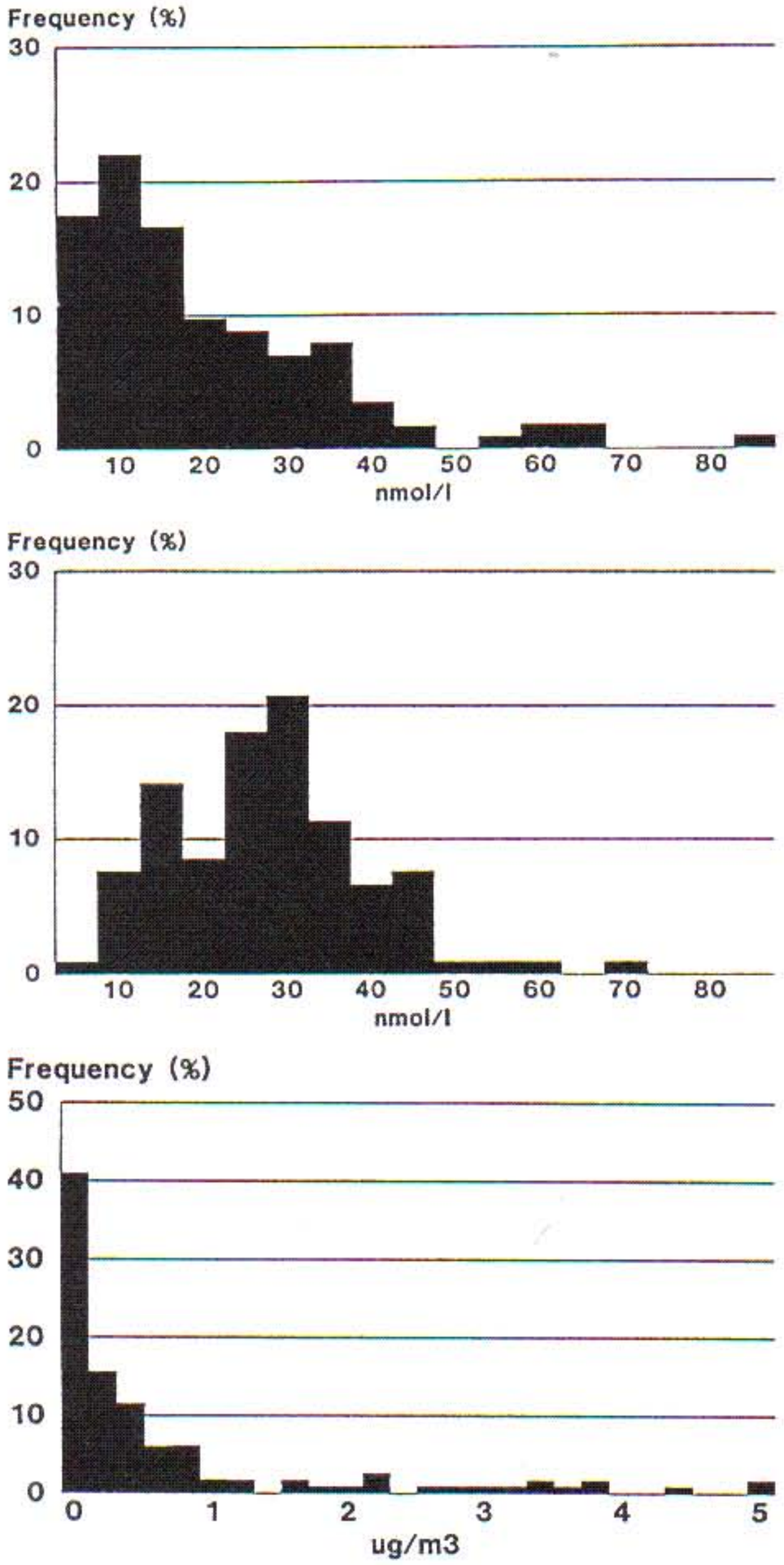

Fig. 1. The frequency distribution of the mercury concentrations in urine samples (HgU) (n = 146) (upper panel), whole blood samples (HgB) (n = 133) (middle panel), and in exhaled pulmonary air samples (HgAir) (n = 147) (lower panel) from habitants of Oslo. No adjustment has been made for the creatinine content or the specific gravity of the urine sample.

the amount of amalgam yielded approximately the same correlation coefficients, with no changes of the probabilities.

Fig. 2 shows a scatterplot of the Hg concentrations for all the participants, while fig. 3 illustrates the average concentrations within 4 groups categorized according to the number of surfaces restored by amalgam. Adjusting the HgU values with the creatinine concentrations did not influence the overall mean or frequency distribution of the HgU values. Nor did the creatinine-adjusted HgU values influence the correlation coefficients, except identifying a poss-

Table 1.

Cross-correlation between the dependent and independent variables. Correlation calculated by Pearson's linear correlation index, using the individual values. Probabilities calculated by Students' t-test, one tailed significance.

	Mercury in		Number of amalgam restorations				
	urine	blood	Total surfaces	Occlusal surfaces	Resto-rations	Age	Gender
Urine			0.549 P<0.001	0.561 P<0.001	0.524 P<0.001	−0.327 P<0.001	−0.172 P=0.019
Blood	0.086 P=0.164		0.188 P=0.027	0.127 P=0.098	0.165 P=0.046	0.084 P=0.168	0.043 P=0.311
Air	0.369 P<0.001	0.159 P=0.033	0.422 P<0.001	0.389 P<0.001	0.397 P<0.001	−0.159 P=0.028	−0.132 P=0.056

ible effect of gender. Therefore, only the unadjusted HgU values were used in the statistics.

The HgB values increased slightly with increasing amounts of amalgam, as seen on the scatterplot (fig. 2), and on the mean values after subgrouping the participants (fig. 3). The correlation coefficients were in both cases relatively low. No differences were obtained with regard to gender or age ($P > 0.05$) (table 1).

The HgU values correlated with the three indices of the amount of amalgam and with the participants' age ($P < 0.001$). Lower HgU values were generally found among the oldest participants, compared to the others, irrespective of the amount of amalgam (fig. 4). The total variance explained by a multivariate analysis using gender, age and number of restored surfaces in the model was $R^2 = 36\%$. A log-transformation of the HgU values, and using the same model, increased the R^2 to 47%. Approximately the same R^2 values were obtained when using the mean values of subgroups instead of the individual HgU values.

The HgAir in the pulmonary air samples correlated to the amount of amalgam ($P < 0.001$). A weak correlation was also seen to age. Gender did not seem to influence the HgAir concentrations (table 1). The total variance explained by a multivariate analysis using gender, age and number of restored surfaces in the model was $R^2 = 20\%$. A log-transformation of the HgAir values, and using the same model, increased the R^2 to 39%. When using the mean values of subgroups instead of the individual HgAir values the R^2 increased further to 41%.

Discussion

The dental status of the participants in the present study can be considered representative for the dental health of urban Norwegians (Bjertness 1990). Also the HgU and HgB values compare well to the data from the few previous demographic studies on the HgU (Lie 1980) and HgB (Mathiesen 1980; Syversen 1982) amounts in non-occupationally exposed Norwegians. There are no previous reports on measurements of the mercury concentrations in pulmonary air samples in a Norwegian population.

The differences in correlation between the different measures of the amount of amalgam and the mercury concentrations in urine, blood and air were not large. This is in accordance with previous studies (Berglund 1980; Zander *et al.* 1990; Akesson *et al.* 1991).

The whole blood mercury concentrations were insignificantly influenced by the number of amalgam restorations. Our data thus support the theory that the whole blood mercury reflect ingested methyl mercury (MeHg), rather than inorganic mercury (Birke *et al.* 1972; Skerfving 1974). Furthermore, in a recent study it is shown that the major part of total mercury in non-occupationally exposed Norwegians is present as methyl mercury (Bulska *et al.* 1992). This interpretation is in accordance with the majority of previous reports (Kröncke *et al.* 1980; Ott *et al.* 1984), but conflicts with two other reports (Abraham *et al.* 1984; Patterson *et al.* 1985). The reason may be due to the differences in analytical techniques or estimation procedures, as well as the use of relatively small samples in the studies. Moreover, lack of control of other potential mercury sources may have biased the results, e.g. dietary intake, smoking, chewing habits, medication, and alcohol consumption. On the other hand, our average difference of 6 nmol/l between the amalgam-free group and the group with extensive amalgam restorations (fig. 3) compares well with the data of Abraham *et al.* (1984) and Snapp (1989). Furthermore, plasma mercury has been reported to correlate with the number of amalgam surfaces (Molin 1990). Apparently, MeHg, which is mainly located in the erythrocytes, is high enough to camouflage the fluctuations of inorganic mercury in plasma in the whole blood analyses (Bulska *et al.* 1992). The results thus indicate that in order to differentiate the exposure from dental amalgams and food, separate measurements of the organic and inorganic mercury should be made in plasma, and in the erythrocytes.

The urinary mercury values, on the other hand, correlated to the amount of amalgam, which is consistent with previous reports (Olstad *et al.* 1987; Jokstad 1987). Occupational HgU exposure is reflected by increased urinary mercury levels, when assessed on a group basis (Skerfving & Berlin 1985). The recommended upper limit of the Hg^0 content in an occupational atmosphere, (25 $\mu g/m^3$, WHO study group 1980), imply an average individual uptake of about 100 μg/day (Aaseth & Barregård 1989). As faecal and urinary

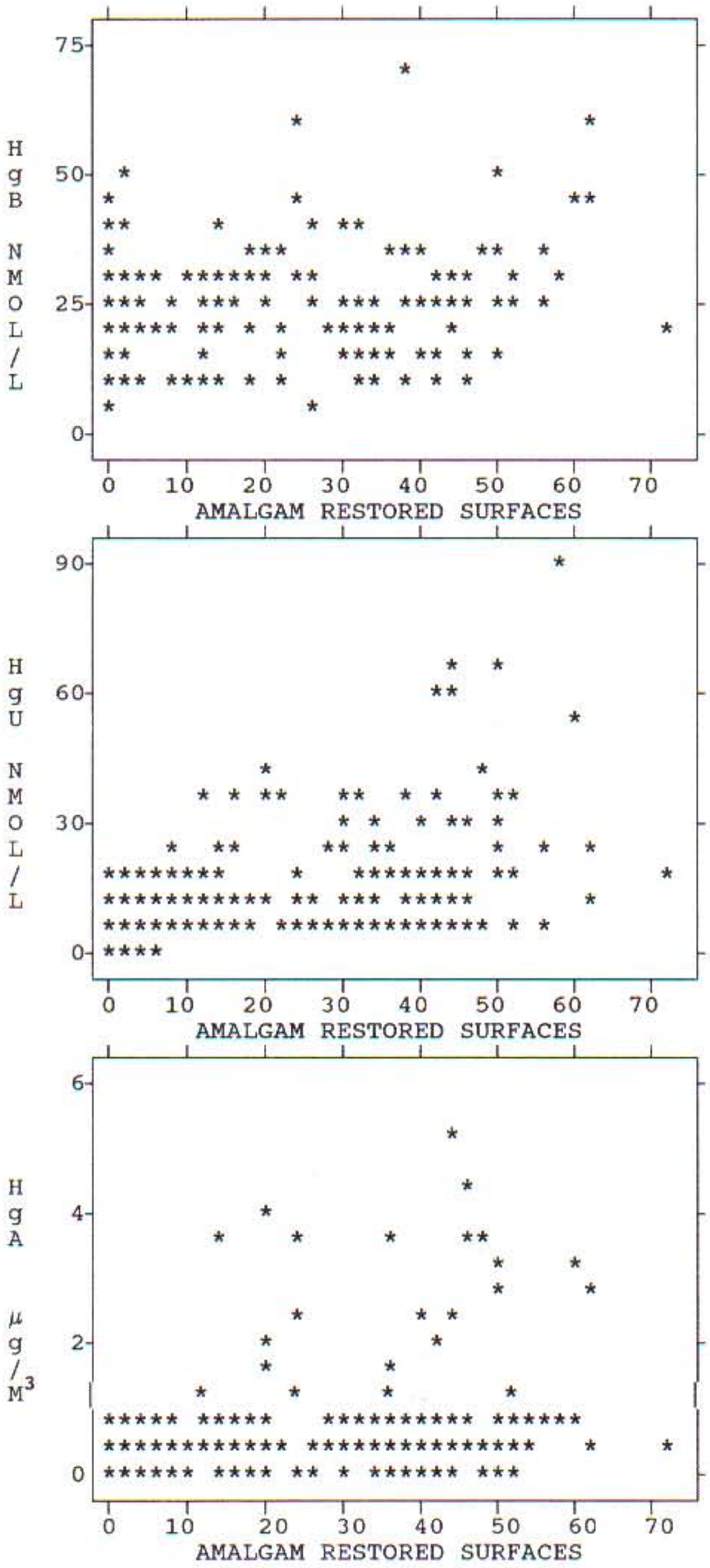

Fig. 2. Scatterplot of the mercury concentrations in urine (HgU), whole blood (HgB) and in pulmonary air samples (HgAir) as a function of the number of dental amalgam restored surfaces.

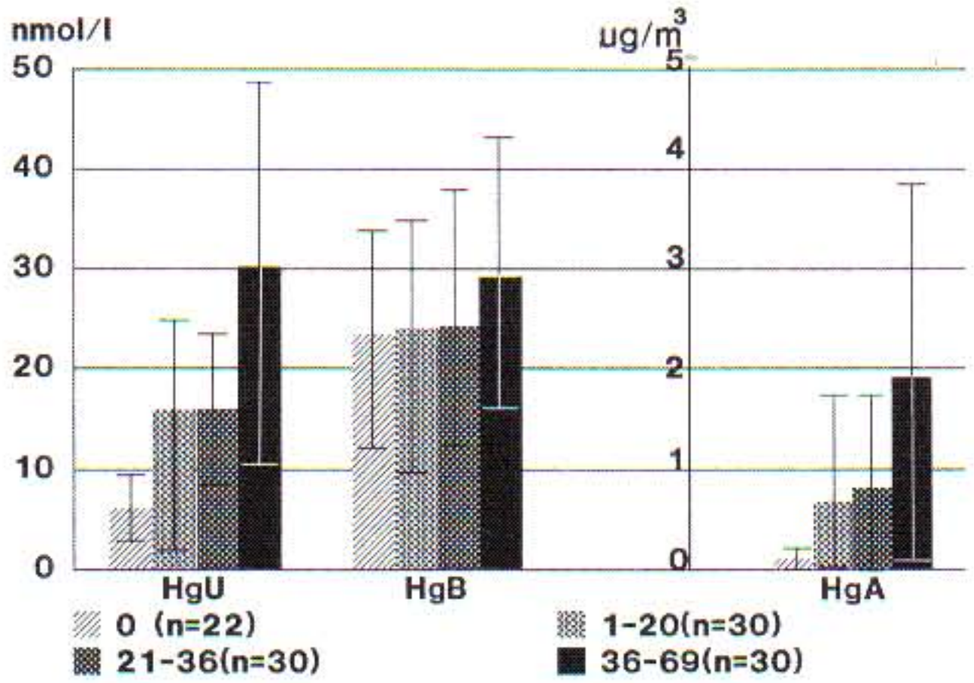

Fig. 3. Mean mercury values in urine (HgU), whole blood (HgB) and pulmonary air samples (HgAir) from four subgroups. Group 1 is the mean of 22 participants without amalgam restorations. Groups 2–4 are equally sized, and show the mean values of the participants with amalgam restorations, grouped according to the number of dental amalgam restored surfaces. The vertical bars represent the standard deviations.

(Zander *et al.* 1990). However, it is also theoretically possible that some of these variations are due to different patterns of tooth grinding, and that extensive bruxism may decrease the safety ratio (Sällsten *et al.* 1991).

The low HgU concentrations from participants without amalgam restorations may reflect ingested MeHg in food, especially in seafood, which is demethylated before urinary excretion. The analytical method used in the present study, however, is not capable to determine the MeHg fraction in the urine.

It is not clear what the HgAir concentrations in the present study represents. Earlier studies have reported a excretion of mercury after exposure to Hg^0 are of the same magnitude, this exposure corresponds to a urinary excretion of about 50 µg/day, or approximately 50 µg/l (Zander *et al.* 1990). In the present study, the mean HgU excretion in the group with many restorations was 6 µg/l, i.e. 12% of the value reflecting the estimated upper health-based limit. However, the HgU values in the present study showed large variations, possibly due to differences in excretion kinetics

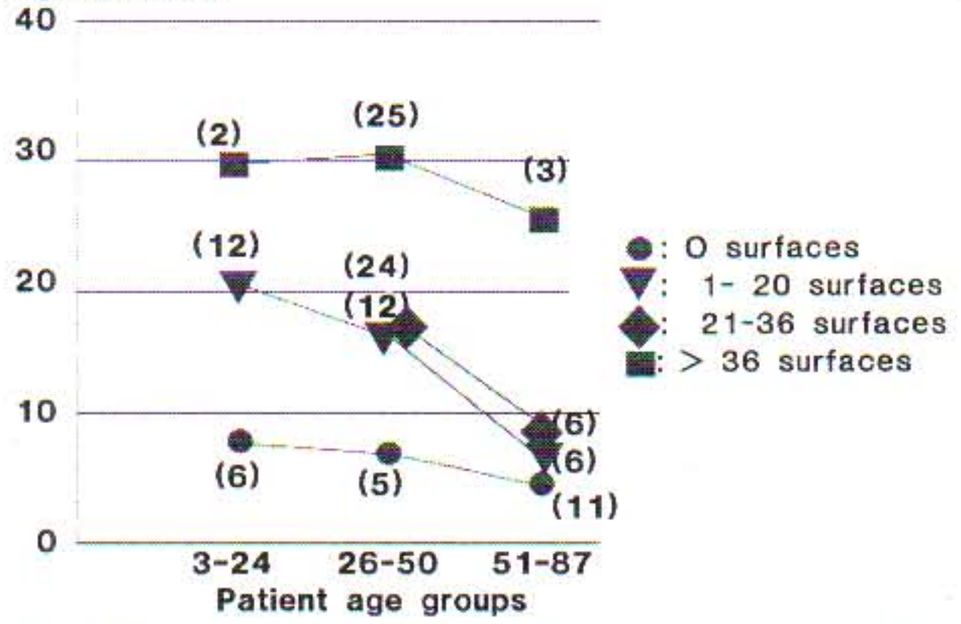

Fig. 4. Urinary mercury (HgU) in relation to the number of dental amalgam restored surfaces and participants' age. The age groups are: 0–25 years (n=20), 26–50 years (n=66) and 51–87 years (n=26). The amalgam groups are: 0 surfaces (n=22), 1–20 surfaces (n=30), 21–36 surfaces (n=30) and >36 surfaces (n=30). The numbers in parentheses show the number of observations within each subgroup.

correlation between the amount of amalgam and mercury vapour present in air samples where participants have breathed into tubes (Gay *et al.* 1979; Patterson *et al.* 1985), or bags (Svare *et al.* 1981; Ott *et al.* 1984), where the oral cavity has been flushed and air collected with a suction device (Abraham *et al.* 1984), or by use of intraoral suction probes (Vimy & Lorscheider 1985a & b; Berglund *et al.* 1988; Berglund 1990). However, these studies were not aimed to measure the HgAir concentration in pulmonary air. In the present study all the potential intraoral mercury vapour was exhaled during the first part of the sampling procedure. Thus, the present HgAir values do not represent the intraoral mercury concentrations as a function of time. Theoretically, some mercury may have vapourized during the second half of the exhalation, but this fraction would be minimal. The HgAir values therefore probably represent dissolved Hg^0 in the blood stream, lung tissues and lung fluids (Cherian *et al.* 1978). The lack of significant correlation between HgAir and HgB in the present study may be explained by the assumption that the predominant mercury in blood, i.e. MeHg (Bulska *et al.* 1992), is bound intracellulary or to plasma proteins (Skerfving & Berlin 1985). The wide range of the HgAir concentrations within the groups categorized according to the amount of amalgam (fig. 3), however, indicates that the pulmonary excretion pattern shows marked inter-individual variation, and it may have been influenced by drugs or by alcohol consumption (Berlin 1986).

The first estimates of the daily exposure reported in the literature were based on data of intraoral mercury vapour measurements (Vimy & Lorscheider 1985a & b). Measurements of 35 persons with amalgam restorations showed that the intraoral mercury concentrations increased from about 5 μg/m^3 to about 30 μg/m^3 after chewing, and remained relatively high for some time afterwards. The daily body burden was estimated to be 20 μg/day. However, their estimations were refuted by other researchers (Mackert 1987; Olsson & Bergman 1987; Berglund *et al.* 1988; Olsson *et al.* 1989). These researchers suggested that the daily exposure ranged around 1–2 μg/day. Vimy & Lorscheider (1990) recently presented a reevaluation of their data, and concluded that a daily exposure of 10 μg/day seemed more correct. However, also the validity of these re-estimations can be questioned, since they are based on a limited number of measurements. The intraoral mercury vapour measured over 24 hr do not correlate to singular or even short series of measurements (Berglund 1990).

The daily exposure of mercury from dental amalgam may be estimated by using a metabolic model as a function of the whole blood mercury concentration, the fraction distributed to a unit volume of blood, and the biological half-time (Clarkson *et al.* 1988). Three assumptions must in this case be verified. The first is that the body tissue compartments have achieved a steady state. The second assumption is that a single biological half time is sufficient to describe the retention in a particular compartment and third, that a constant fraction of the daily dose is deposited in each compartment. The whole body half time after a single mercury exposure is 58 days (Hursh *et al.* 1976), while steady state between body tissue compartments and mercury exposure is achieved in approximately 5 times the biological half-time (Clarkson *et al.* 1988). Since the participants in the present study had many amalgam restorations it is assumed that the HgB concentrations were in a steady state. The biological half time of mercury in blood is 3.3 days and 2.1% of the daily dose is deposited in 1 l whole blood (Kershaw *et al.* 1980; Cherian *et al.* 1978). The model thus suggest that under these circumstances the whole blood mercury values reflect 10% of the daily absorbed mercury (Clarkson *et al.* 1988). The mean HgB in the amalgam-free group and the group with the extensive number of amalgam restorations differed by 6 nmol/l (1.2 μg/l). Provided that this difference is caused by the amalgam restorations only, and that the assumptions above are correct, this calculation would suggest a daily exposure of 10* 1.2 μg/l=approximately 10–12 μg/day.

The difference in mean HgU in the amalgam-free group and the group with the extensive number of amalgam restorations was 24 nmol/l (4.8 μg/l). Provided that the difference is solely caused by the dental amalgam, that the HgU approximately represent the 24 hr hour excretion (Zander *et al.* 1990), and that roughly 50% of the mercury is recovered in urine, the mean HgU indicate a daily exposure of approximately 10 μg/day. The present results thus suggest that individuals with many amalgam restorations, i.e., more than 36 restored surfaces, are exposed to 10-12 μg Hg/day in addition.

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