# Mercury, selenium, and glutathione peroxidase in dental personnel

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Eighteen persons, dentists and nurses, with urinary mercury levels higher than the group median value of all dental personnel in the country of Västerbotten were compared with a group consisting of 15 persons with low urinary mercury levels working in the same clinics. A statistically significant difference between the high urinary mercury group and the low urinary mercury group could be seen in the plasma mercury level. In each group a statistically significant relation could be seen between the plasma mercury level and the total number of amalgam surfaces. The two groups did not differ with regard to the levels of plasma selenium and erythrocyte glutathione peroxidase, and no correlation between these two variables and the plasma mercury levels could be found. To evaluate organ functions, a large number of supplementary analyses were performed. These analyses did not indicate any influence on organ functions. Although the persons in the present study were occupationally exposed to mercury, none of the biologic variables analyzed seemed to be affected. Even among dental personnel who handle amalgam professionally the number of amalgam surfaces is a major contributory factor to the P-mercury level. □ *Dental amalgam; erythrocytes; plasma; urine*

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The mercury release from dental amalgam has been investigated rather extensively for several years. Studies in man have shown that mercury concentrations in expired and intra-oral air (1, 2), in the blood (3, 4), and in the urine (5, 6) are correlated to the number of amalgam surfaces. It has also been claimed that mercury concentrations in the human brain and kidneys are related to the exposure from dental amalgam fillings (7).

Dental personnel working with amalgam are extensively exposed to mercury vapor. It has been shown in several studies that the mercury vapor concentration in the air of dental clinics is comparatively high (8–10) and in a few cases exceeds the hygienic threshold limit of 50 $\mu g/m^3$ (11). Investigations have also shown increased mercury levels in urine and blood among dental personnel (12, 13). However, there is no scientific evidence that dental amalgam fillings or proper professional handling of dental amalgam is connected with general health problems, with the exception of some allergic and local hypersensitivity reactions to mercury (14–17).

Interaction between selenium and both inorganic mercury (18–20) and organic mercury (21, 22) has been demonstrated. In the very few studies presented on interaction between selenium and mercury vapor, selenium has been shown to influence the retention and distribution of mercury in different organs (23, 24). However there is, as yet, no scientific evidence that selenium will protect against the possible toxic effects of mercury vapor.

It was considered to be of interest to study mercury, selenium, and the selenium-dependent enzyme glutathione peroxidase (GSHPx), in a group occupationally exposed to mercury vapor. For that reason the levels of plasma (P) mercury, P-selenium, and erythrocyte (E) GSHPx were analyzed in a group of dental personnel with comparatively high urinary mercury levels.

As the present group was occupationally exposed to mercury, it was considered to be of interest to perform several supplementary blood and urine analyses to disclose potential effects on electrolyte balance, liver and kidney functions, inflammatory activity, immune stimulation, and tissue damage, as assessed by plasma enzymes. In comparison, a group of dental personnel working in the same clinics as the study group but showing low urinary levels was also analyzed.

## Materials and methods

From a study by Nilsson & Nilsson (25), in which all dental personnel ($n = 505$) in the county of Västerbotten were examined for, among other things, urinary mercury, a group of dentists and nurses with urinary mercury levels above the group median value were selected. The study group (HUM = high urinary mercury) consisted of 18 people: 16 women ($\bar{x} = 37$ years old; range, 21 to 61 years) and 2 men (55 and 45 years old) from 9 districts. Their mercury levels in urine ranged between 5 and 14 μg/g creatinine. The personnel selected for comparison were 13 women ($\bar{x} = 36$ years old; range, 21 to 58 years) and 2 men (43 and 48 years old) with low urinary mercury levels (LUM = low urinary mercury), ranging between 1 and 3 μg/g creatinine, working at the same clinics as the test subjects. Both groups consisted of people exposed to mercury during their work. None of the participants ate any selenium preparations. The total number of amalgam surfaces was registered in both groups.

### *Samples*

Blood was collected, using ethylenediaminetetraacetic acid (EDTA) as anticoagulant. The samples were cold-stored for no more than 4 h and, apart from samples intended for blood cell analyses, centrifuged at 2000 *g* for 10 min. The supernatant plasma was then drawn off for further analyses.

The buffy coat was discarded. Unless they were analyzed immediately, the plasma and the packed erythrocytes were kept at −80°C.

Urine was collected for 24 h in acid-washed polyethylene bottles. The morning urine was collected in separate bottles. The samples were kept frozen until analyzed.

### *Analytic procedures*

E-GSHPx was analyzed as described by Günzler et al. (26). Plasma and urinary selenium were determined by a fluorometric method with diaminonaphthalene as described by Lalonde et al. (27), P-sodium and P-potassium by means of ion-specific electrodes, P-calcium as complex with chresolphthalein purple, and P- and U-creatinine with an alkaline picrate reagent. P-total and conjugated bilirubin were determined with sodium nitrate and sulfanilic acid, and P-alkaline phosphatase by P-nitro-phenyl-phosphate assay.

P-γ-glutamyltranspeptidase, aspartate aminotransferase, alanine aminotransferase, lactate dehydrogenase, and creatine kinase in plasma were determined with reagent kits from Boehringer Mannheim AG. Albumin, $\alpha_1$-antitrypsin, orosomucoid, haptoglobin, and C-reactive protein (CRP) in plasma were analyzed by electroimmunoassay. Mercury in plasma was determined by means of neutron-activation analysis at IVL, The Swedish Environmental Research Institute, in Stockholm. Blood cells and erythrocyte variables were analyzed on a Technicon H 6000 apparatus, U-albumin by means of electroimmunoassay, U-protein by the Coomassie Brilliant Blue G-250 procedure (28), U-$\beta_2$-microglobulin with a radioimmunoassay (RIA) kit ($\beta_2$-micro-RIA 100) supplied by Pharmacia Diagnostic AB, Uppsala, Sweden, and osmolality of urine with the freezing-point depression method.

### *Reference intervals*

The P-selenium and the E-GSHPx reference intervals were obtained by analyzing 250 persons aged 30, 40, 50, and 60 years in a health survey study in the county of Västerbotten. The U-selenium reference interval was taken from 130 persons in the study. Data established and used in the Laboratory of Clinical Chemistry were used

as reference intervals for the other analyses. The reference intervals presented are the 2.5 and 97.5 percentiles.

### *Statistical methods*

A Mann–Whitney U-test was used for comparing the blood and urine variables and the number of amalgam surfaces for the two groups. A simple regression analysis was used to compare the P-mercury levels with the number of amalgam surfaces in both groups. Fisher's exact test was used for comparing P-mercury with smoking and drinking habits.

## Results

There was a statistically significant difference ($p < 0.02$) between the two groups with regard to P-mercury. The HUM group had a mean value of 14.12 nmol/l, ranging from 4.98 to 26.42 nmol/l, and the LUM group had a mean value of 6.23 nmol/l, ranging from 1.49 to 12.46 nmol/l (Table 1).

The number of amalgam surfaces ranged from 21 to 61 ($\bar{x} = 39.9$) in the HUM group and from 9 to 60 ($\bar{x} = 31.5$) in the LUM group. There was no significant difference between the groups. In both groups there was a significant relation between the P-mercury level and the total number of amalgam surfaces ($p < 0.001$, Fig. 1). There was no correlation between the age of the personnel and the plasma or urinary mercury levels.

Furthermore, there was no difference in the fish consumption pattern between the two groups, and the consumption was rather limited. None of the participants ate fish more than once a week.

The number of smokers was 7 of 18 in the HUM group and 5 of 15 in the LUM group. The number of alcohol consumers was 16 in the HUM group and 11 in the LUM group. The differences between the groups were non-significant with regard to both these factors.

The P-selenium and E-GSHPx values did not differ significantly between the HUM group and the LUM group, and all data

Table 1. Mean values ($\bar{x}$), standard deviations (SD), medians (M), and ranges of some variables tested

| | HUM group | | | | LUM group | | | | Reference interval | Statistical analysis |
|---|---|---|---|---|---|---|---|---|---|---|
| | $\bar{x}$ | SD | M | Range | $\bar{x}$ | SD | M | Range | | |
| P-Hg, nmol/l | 14.12 | 6.23 | 12.21 | 4.98–26.42 | 6.23 | 3.29 | 6.97 | 1.49–12.46 | — | $p < 0.02$ |
| P-Se, μmol/l | 0.93 | 0.09 | 0.93 | 0.80–1.17 | 0.97 | 0.16 | 0.91 | 0.64*–1.25 | 0.72–1.47 | NS |
| E-GSHPx, μkat/g Hb | 1.28 | 0.20 | 1.24 | 0.85–1.71 | 1.16 | 0.22 | 1.20 | 0.76–1.59 | 0.63–1.82 | NS |
| U-Se, μmol/mmol creatinine | 0.02 | 0.004 | 0.02 | 0.01–0.03 | 0.02 | 0.005 | 0.02 | 0.01–0.03 | 0.01–0.05 | NS |
| dU-Se, μmol | 0.24 | 0.078 | 0.22 | 0.13–0.43 | 0.28 | 0.076 | 0.28 | 0.11–0.39 | 0.08–0.53 | NS |

HUM = high urinary mercury; LUM = low urinary mercury.
* One person outside the reference interval.

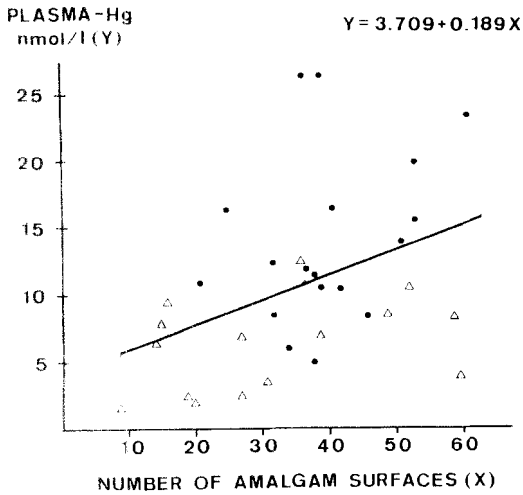


Fig. 1. Regression analysis of plasma mercury (nmol/l) and the number of amalgam surfaces. The solid line is the linear regression line for the high urinary mercury (HUM (●)) and low urinary mercury (LUM (△)) groups.

except one value (P-selenium for one of the LUM persons) fell within the reference interval (Table 1). Urinary selenium values did not differ between the groups, and all values fell within the reference intervals.

With regard to P-mercury, P-selenium, and E-GSHPx, the two persons with the highest P-mercury levels, both with 26.42 nmol/l and belonging to the HUM group, had P-selenium levels of 1.3 and 0.89 μmol/l and E-GSHPx levels of 1.59 and 1.21 μkat/g hemoglobin, respectively. Thus there was no evidence of selenium deficiency. The lowest P-selenium value, 0.64 μmol/l, was found in the LUM group. This value was well below the reference interval, but as the corresponding E-GSHPx value was 1.20 μkat/g hemoglobin, the P-selenium value could not be taken as an indication of selenium deficiency. The P-mercury level for this person was the second lowest in the total material, 1.99 nmol/l, and the amalgam surfaces numbered 20. Another LUM subject had a comparatively low E-GSHPx level, 0.76 μkat/g hemoglobin, which in combination with a P-selenium value of 0.89 μmol/l indicates a low selenium status but hardly selenium deficiency. The P-mercury level was 8.47 nmol/l, which was comparatively high for a person in the LUM group. In this case 49 amalgam surfaces were recorded.

A large number of supplementary analyses were performed to evaluate organ functions. With the exception of the U-protein, there was no statistically significant difference between the groups for blood cell variables (erythrocyte partical concentration, hemoglobin, mean corpuscular hemoglobin concentration, mean corpuscular volume, leukocyte partical concentration, trombocyte partical concentration, platelets, neutrophils, lymphocytes, monocytes, eosinophils, and basophils), plasma electrolytes (P-Na, P-K and P-phosphate), analyses indicating 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-$\alpha_1$-antitrypsin, P-orosomucoid, P-haptoglobin, and P-CRP), P-IgG, or for kidney variables (U-albumin, U-protein, U-$\beta_2$-microglobulin, morning U-osmolality, and P-creatinine) (Table 2). However, there was a statistically significant higher mean value in the LUM group with regard to the U-protein level ($p < 0.02$).

More than 98% of the supplementary analyses values were within reference limits. Only a few, 20 of 1089, fell outside. Thus in the HUM group there were two persons with neutrophilia and one person with a slight acute-phase reaction and plasma enzymes indicating a mild liver affection. Increased P-creatinine was found in two and increased morning U-osmolality in another five subjects. In the LUM group there was one person with blood cell variables indicating iron deficiency, one person with neutrophilia, and one person with a mild acute-phase reaction, probably due to ulcerative colitis. Two persons showed increased P-creatinine values.

Table 2. Kidney status variables

| | HUM group | | | | LUM group | | | | Reference interval | Statistical analyses |
|---|---|---|---|---|---|---|---|---|---|---|
| | $\bar{x}$ | SD | M | Range | $\bar{x}$ | SD | M | Range | | |
| U-albumin, mg/24 h | 4.8 | 3.2 | 4.8 | 1.0–9.8 | 6.0 | 4.12 | 4.31 | 1.4–17.2 | <20 | NS |
| U-protein, mg/24 h | 26.8 | 15.4 | 26.3 | 4.36–65.3 | 42.2 | 32.1 | 37.6 | 5.55–132.6 | <150 | $p < 0.02$ |
| U-$\beta_2$-microglobulin, µg/24 h | 77.1 | 59.1 | 56.4 | 38.9–255.7 | 54.6 | 19.4 | 53.3 | 40.4–91.4 | 4–370 | NS |
| P-creatinine, F | 64.7 | 13.4 | 63 | 37–94 | 68.3 | 10.7 | 68 | 50–90 | 35–80 | |
| µmol/l M | 85 | 8 | 85 | 77–93 | 97 | 2 | 97 | 97–99 | 50–100 | NS |

$\bar{x}$ = Mean value; SD = standard deviation; M = median value; HUM = high urinary mercury; LUM = low urinary mercury.

## Discussion

The statistically significant difference found between the HUM and LUM groups in the present study with regard to P-mercury levels reflects the difference between the groups with regard to the urinary mercury levels. However, these deviations between the groups are difficult to explain and cannot be ascribed to differences in professional environments or working conditions. Other possible explanations must therefore be considered.

An environmental factor contributing to mercury exposure is fish consumption. Fish, however, mainly contains organic mercury, which in the blood primarily binds to erythrocytes and not plasma (29). Thus fish consumption should not contribute to the P-mercury level. Furthermore, the participants of the two groups had a similar and limited fish consumption.

It has been reported that higher blood mercury concentrations are found among cigarette smokers than among non-smokers (30), but as the number of smokers was very similar in the two groups, this factor cannot explain the differences between the HUM and LUM group.

Another factor that might affect the mercury blood concentration is ethyl alcohol (31, 32). No significant difference was recorded between the groups in the number of alcohol consumers. However, declarations by the individuals themselves concerning their intake of alcohol should generally be regarded with great caution. Therefore the possibility cannot be excluded that there was a difference between the groups with regard to alcohol consumption which might possibly explain the deviation in the P-mercury concentration.

One possible explanation for differences in P-mercury levels between groups of dental personnel may be the way in which amalgam is handled. However, we have no data supporting the existence of such a difference in handling. On the contrary, Nilsson & Nilsson (33) showed that mercury hygiene was very good among the group from which the present material was selected.

The significant positive relationship

between P-mercury levels and the number of amalgam surfaces found in the present study supports earlier results obtained by Molin et al. (4). Our P-mercury results are in agreement with those for whole blood found by Nilsson & Nilsson (personal communication) and Abraham et al. (3) but not with those of Kröncke et al. (34). P-mercury was analyzed in the present study because mercury in plasma is supposed to reflect inorganic mercury concentration more correctly than whole blood, since the latter also includes organic mercury, which is mainly present in erythrocytes (29).

The E-GSHPx was analyzed to evaluate the availability in the body of selenium for synthesis of selenium-dependent factors. The P-selenium and E-GSHPx analysis did not show any differences between the two groups, and, as in the previous study by Molin et al. (4), practically all values fell within the reference intervals. Thus, the higher P-mercury levels in the HUM group were not accompanied by any changes in the P-selenium and E-GSHPx levels. Apparently, the mercury exposure due to the professional environment, the handling of amalgam, and the dental personnel's own amalgam fillings were too low to influence the selenium status.

Selenium is excreted via the lungs, kidneys, and liver (35). Urinary selenium levels have been known to reflect dietary selenium intake (36). When the selenium intake is low, most selenium is excreted into the urine (37). The low selenium content in Swedish soil gives very little selenium in the diet (38, 39). Thus a likely explanation for the low urinary selenium excretion values in the present study is the low dietary selenium intake.

The kidney is one of the organs especially susceptible to mercury toxicity (40, 41). Increased excretion of U-proteins has been noted in persons with increased levels of U-mercury (42–44) and increased blood mercury levels (45). In the present study the U-protein levels differed signficantly between the two groups, but a higher U-protein level was found in the LUM group, contrary to what might be expected should there be any mercury-induced difference between the groups. But even though the U-protein values differed between the two groups, all values fell within the reference intervals. Thus, the mercury exposure was obviously also too low to influence either glomerular or tubular function. Apart from the kidney variables, a large number of other supplementary analyses were performed. There were no statistically significant differences between the groups, and practically all values fell within the reference limits. The very few separate outliers did not constitute any pattern indicating a connection to mercury exposure.

Although the persons in the present study were occupationally exposed to mercury, none of the biologic variables analyzed seemed to be influenced. It is worth noting that, as in a previous study by Molin et al. (4), a relationship was found between the number of amalgam surfaces and P-mercury concentrations. Thus, even among dental personnel who professionally handle amalgam, the number of amalgam surfaces seemed to be a factor—apparently the major contributory factor—to the P-mercury level.

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## References

1. Patterson JE, Weissberg BG, Dennison PJ. Mercury in human breath from dental amalgams. Environ Contam Toxicol 1985;34:459–68.
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;63:71–3.
4. Molin M, Marklund S, Bergman B, Bergman M, Stenman E. Plasma-selenium, glutathione peroxidase in erythrocytes and mercury in plasma in patients allegedly subject to oral galvanism. Scand J Dent Res 1987;95:328–34.
5. 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.
6. Langworth S, Elinder C-G, Åkesson A. Mercury exposure from dental fillings. 1. Mercury con-

centrations in blood and urine. Swed Dent J 1988;12:69–70.
7. Nylander M, Friberg L, Lind B. Mercury concentrations in the human brain and kidneys in relation to exposure from dental amalgam fillings. Swed Dent J 1987;11:179–87.
8. Lundgaard U. Målning af luftföroreningen med kvicksilvdampe på en tandklinnik. Tandlaegebladet 1981;85:86–8.
9. Nixon GS, Whittle CA, Woodfin A. Mercury levels in dental surgeries and dental personnel. Br Dent J 1981;151:149–54.
10. Joselow MM, Goldwater LJ, Alvarez A, Herndon J. Absorption and excretion of mercury in man. XV. Occupational exposure among dentists. Arch Environ Health 1968;17:39–43.
11. Report of a WHO Study Group 1980. Recommended health-based limits in occupational exposure to heavy metals. Geneva: WHO, 1980. (Technical report series no. 647).
12. Brooks JM, Allingham PM. Mercury hazards in dentistry. NZ Dent J 1974;70:166–80.
13. Möller-Madsen B, Hansen JC, Kragstrup J. Mercury concentrations in blood from Danish dentists. Scand J Dent Res 1988;96:56–9.
14. Frykholm KO. On mercury from dental amalgam. Its toxic and allergic effects and some comments on occupational hygiene [Thesis]. Acta Odontol Scand 1957;15(suppl 22).
15. Finne K, Göransson K, Winckler L. Oral lichen planus and contact allergy to mercury. Int J Oral Surg 1982;11:236–9.
16. Mobacken H, Hersle K, Sloberg K, Thilander H. Oral lichen planus hypersensitivity reactions to dental restoration material. Contact Dermatitis 1984;10:11–5.
17. Burrows D. Hypersensitivity to mercury, nickel and chromium in relation to dental materials. Int Dent J 1986;36:30–4.
18. Magos L, Webb M. The interaction of selenium with cadmium and mercury. Crit Rev Toxicol 1980;8:1–42.
19. Parizek J, Ostadlova I. Protective effect of small amounts of selenite in sublimate intoxication. Experientia 1966;23:142–3.
20. Kosta L, Byrne AR, Zelenko V. Correlation between selenium and mercury in man following exposure to inorganic mercury. Nature 1975;254: 238–9.
21. Imura N. The role of micronutrient, selenium in the manifestation of toxicity of heavy metals. In: Chambers PL, Gehring P, Sakai F, eds. New concept and developments in toxicology. Amsterdam: Elsevier, 1986:115–23.
22. Ganther HE, Goudie C, Sunde ML, et al. Selenium: relation to decreased toxicity of methyl mercury added to diets containing tuna. Science 1972;175:1122–4.
23. Hansen JC, Kristensen P, Westergaard I. The influence of selenium on mercury distribution in mice after exposure to low dose Hg vapours. J Appl Toxicol 1981;1:149–53.
24. Nygaard S-P, Hansen JC. Mercury selenium interaction at concentrations of selenium and of mercury vapours as prevalent in nature. Bull Environ Contam Toxicol 1978;20:20–3.
25. Nilsson B, Nilsson B. Mercury in dental practice. II. Urinary mercury excretion in dental personnel. Swed Dent J 1986;10:221–32.
26. Günzler WA, Kremers H, Flohe L. An improved coupled test procedure for glutathione peroxidase in blood. Z Klin Chem Klin Biochem 1974;12:444–8.
27. Lalonde L, Jean Y, Roberts KD, Chapdelaine A, Bleau G. Fluorometry of selenium in serum and urine. Clin Chem 1982;28:172–4.
28. Bradford MM. A rapid and sensitive method for the quantification of microgram quantities of protein utilizing the principle of protein dye binding. Anal Biochem 1976;72:248–54.
29. Lundgren K-D, Swensson Å, Ulfvarson U. Studies in humans on the distribution of mercury in blood and the excretion in urine after exposure to different mercury compounds. Scand J Clin Lab Invest 1967;20:164–7.
30. Bhatnagar RS. Molecular basis on environmental toxicity. Ann Arbor, Michigan: Ann Arbor Science, 1980.
31. Nielsen-Kudsk F. The influence of ethyl alcohol on the absorption of mercury vapour from the lungs in man. Acta Pharmacol Toxicol 1965;23:263–74.
32. Nielsen-Kudsk F. Factors influencing the in vitro uptake of mercury vapour in blood. Acta Pharmacol 1969;27:161–72.
33. Nilsson B, Nilsson B. Mercury in dental practice. I. The working environment of dental personnel and their exposure to mercury vapour. Swed Dent J 1986;10:1–14.
34. Kröncke A, Ott K, Petschelt A, Schaller K-H, Szesci M, Valentin H. Über die Quecksilberkonzentrationen in Blut und Urin von Personen mit und ohne Amalgamfüllungen. Dtsch Zahnaerztl Z 1980;35:803–8.
35. Burk RF. Selenium in nutrition. Wld Rev Nutr Diet 1978;30:88–106.
36. Levander OA, Sutherland B, Morris VC, King JC. Selenium metabolism in human nutrition. In: Spallholz JE, Martin JL, Ganther HE, eds. Selenium in biology and medicine. Westport, Conn: AVI Publishing Co., 1981;256–68.
37. Robberecht HJ, Deelstra HA. Selenium in human urine: concentration levels and medical implications. Clin Chim Acta 1984;136:107–20.
38. Lindberg P, Bingfors S. Selenium levels of forages and soils in different regions of Sweden. Acta Agric Scand 1970;23:142–3.
39. Bruce Å. Swedish views on selenium. Ann Clin Res 1986;18:8–12.
40. Kazantzis G, Schiller KFR, Asscher AW, Drew RG. Albuminuria and the nephrotic syndrome following exposure to mercury and its compounds. Quart J Med 1962;31:403–18.
41. Gritzka TL, Trump BF. Renal tubular lesion caused by mercuric chloride. Am J Pathol 1968;52:1225–77.
42. Stewart WK, Guirgis HA, Sanderson J, Taylor W.

Urinary mercury excretion and proteinuria in pathology laboratory staff. Br J Ind Med 1977;34:26–31.
43. Foa V, Caimi L, Amante L, et al. Patterns of some lysosomal enzymes in the plasma and of proteins in urine of workers exposed to inorganic mercury. Int Arch Occup Environ Health 1976;37:115–24.
44. Roels H, Lauwerys R, Buchet JP, Bernard A, Oversteyns M, Gaussin J. Comparison of renal function and psychomotor performans in workers exposed to elemental mercury. Int Arch Occup Environ Health 1982;50:77–93.
45. Stonard MD, Chater BV, Duffield DP, Nevitt AL, O'Sullivan IJ, Steel GT. An evaluation of renal function in workers occupationally exposed to mercury vapour. Int Arch Occup Environ Health 1983;52:177–89.

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