

## Urinary mercury levels in females: Influence of skin-lightening creams and dental amalgam fillings

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**The influence of application of skin-lightening creams and dental amalgam fillings on the urinary mercury (Hg) level was evaluated in 225 females (ages 17 to 58 years) living in Riyadh, capital of Saudi Arabia. The arithmetic mean of the urinary Hg level was  $6.96 \pm 20.43 \mu\text{g l}^{-1}$ , in the range 0 to  $204.8 \mu\text{g l}^{-1}$ . The mean urinary Hg level adjusted by creatinine (Cr) was  $11.22 \pm 37.23 \mu\text{g g}^{-1}$  Cr, in the range 0 to  $459.37 \mu\text{g g}^{-1}$ . No significant difference in urinary Hg was noted between the females regarding the use of skin-lightening creams. On the other hand, results showed that urinary Hg concentration was influenced by the use and number of dental amalgam fillings. No women were identified with symptoms or signs that could be attributed to Hg intoxication. Urine analyses for creatinine, urea, uric acid, phosphorus, magnesium, glucose and calcium showed significant correlation with urinary Hg. This suggests that chronic exposure to Hg may be associated with a deterioration of renal function.**

**Keywords:** dental amalgam, Saudi Arabia, skin-lightening creams, urinary Hg

### Introduction

Mercury (Hg) is a naturally occurring metal which has several forms: (1) metallic or elemental  $\text{Hg}^0$ , commonly used in dental filling and thermometers; (2) inorganic  $\text{Hg}^{2+}$ ,  $\text{Hg}^+$  compounds, used in skin care and medicinal products; and (3) organic  $\text{CH}_3\text{Hg}^+$ ,  $\text{C}_6\text{H}_5\text{Hg}^+$  compounds, used in fungicides and paints, and found in the diet (contaminated fish). All forms of Hg are considered poisonous. Through natural chemical processes and microorganisms, the change in Hg speciation from inorganic to methylated forms is the first step in the aquatic bioaccumulation process.

The general population is exposed to Hg primarily through diet and dental amalgam (WHO 1991). The major portion of Hg in the diet is contained in fish

in the form of methylmercury, while the minor portion is in the form of inorganic Hg, mainly  $\text{Hg}^{2+}$ . Dental amalgam releases elemental vapor. Hence, the three chemical forms are involved in exposure of the general population to Hg. Mercury in these three chemical forms is excreted in the urine, although the proportion and speed of excretion differs with the chemical form. Furthermore, conversion between the chemical forms occurs in the body, resulting in a change of urinary excretion. The average Hg concentration in urine of the general population is estimated to be  $4 \mu\text{g l}^{-1}$ , although individual variation is substantial (WHO 1991). Dental amalgam fillings have been noted as a source of the variation (Herrstrom *et al.* 1995). The major source of exposure for blood and hair levels is fish consumption.

Chronic exposure to either inorganic or organic Hg can permanently damage the brain, kidneys, and developing fetus. The most sensitive target of low-level exposure to metallic and organic Hg following short- or long-term exposures appears to be the

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nervous system. By contrast, the most sensitive target of low-level exposure to inorganic Hg appears to be the kidney. Both organic and inorganic mercurials are employed in topical preparations. Organic forms such as phenyl mercuric acetate are sometimes used as cosmetic preservatives, while inorganic forms, such as ammoniated mercury, are the active ingredients in skin bleach creams (Marzulli & Brown 1972).

Skin-lightening creams containing inorganic Hg are widely used by dark-skinned people to obtain a lighter skin tone, probably due to the inhibition of pigment formation. Mercury is absorbed through the skin (Marzulli & Brown 1972, Barr *et al.* 1973, Bourgeois *et al.* 1986) and concentrates in the kidney, mainly in the tubular region (Berlin 1979). Nephrotoxic effects have been attributed to the topical application of inorganic Hg salts (Silverberg *et al.* 1967, Barr *et al.* 1973, Kibuhamusohé *et al.* 1974, Lyons *et al.* 1975, Berlin 1979, Jeddelloh *et al.* 1985).

Several studies have shown that it is possible to induce permanent kidney dysfunction by exposure to nephrotoxic chemicals during prenatal periods (Kavlock & Gray 1983a,b, Neubert *et al.* 1985, Lauwerys *et al.* 1987). Previous investigations revealed that inorganic Hg can penetrate the placental barrier, and may affect the developing fetus (Gale & Ferm 1971, Gale & Hanlon 1976, Gale 1981, Kuhnert *et al.* 1981, Holt & Webb 1986, Boadi *et al.* 1992).

Recently considerable public interest has centered on the role of Hg in dental amalgam and its effects upon the immune system, renal system, oral and intestinal bacteria, reproductive system, and the central nervous system (Abraham *et al.* 1984, Vimy & Lorscheider 1985a,b, Mackert 1987, Snapp *et al.* 1989, Berglund 1990, Fung & Molvar 1992, Lorscheider *et al.* 1995). Some researchers counter that Hg is a well-known substance that has been safely used in teeth fillings for more than 150 years (Fung & Molvar 1992). Such fillings are actually amalgams, blends of metal that comprise as much as 50% Hg, 35% silver, 13% tin, 2% copper, and a trace of zinc by weight (Patterson 1984, Craig 1985, WHO 1991). Exposure to elemental Hg in the form of vapor has been regarded as a potential occupational hazard for dental professionals (Battistone *et al.* 1976, Gough 1978, Siew *et al.* 1987). Between 1975 and 1983, the mean urinary Hg level of 4272 US dentists was estimated to be  $14.2 \mu\text{g l}^{-1}$  in the range of 0 to  $556 \mu\text{g l}^{-1}$  (Naleway *et al.* 1985). Rigorous chewing and brushing of the teeth stimulate the release of Hg vapor from amalgam surfaces

(Craig 1985, Snapp *et al.* 1989, Berglund 1990, Fung & Molvar 1992, Begerow *et al.* 1994, Barregard *et al.* 1995). As part of the body's biological process, once elemental Hg leaches out of the amalgam and is inhaled or swallowed, enzymes will convert it to ionic Hg, which is well documented to be neurotoxic and immunotoxic. A recent study by Rowland *et al.* (1994) showed a major fertility effect among women exposed to high levels of amalgams. The use of dental amalgam continues to be a controversial issue, and over the years growing numbers of researchers have questioned the appropriateness of using Hg for dental restoration. Several countries in Europe have, or are considering, a ban on Hg because of its toxic effect on the environment.

Results from a previous study (Al-Saleh & Al-Doush, in press) revealed that about 45% of the tested skin-lightening cream samples commonly used in Saudi Arabia had Hg levels well above the FDA's acceptable limit of 1 ppm. This led us to explore the association between the use of skin-lightening creams and urinary Hg in young healthy women that had no occupational exposure to Hg. Dental amalgam fillings were also evaluated. Furthermore, the possible influence of urinary Hg levels on renal function was examined.

## Materials and methods

Two hundred and twenty-five women were invited to participate in the study. They answered a self-administered questionnaire about the use of skin-lightening creams, particular attention being paid to the nature of the creams employed and the duration and pattern of their use. All were also questioned about their number of dental fillings and asked to give demographic information. Of the 225 screened women, only 220 completed their questionnaires. Urine samples were collected in 50 ml polyethylene bottles and stored at  $-20^{\circ}\text{C}$ .

All urinary Hg analyses were performed using a Varian AA-40 atomic absorption spectrophotometer, coupled to a Vapor Generation Accessory VGA-76 (Varian Techtron Pty. Ltd., Mulgrave, Australia). Instrumental conditions are listed in Table 1. The gas used was argon. In this study, flow rates were about  $8 \text{ ml min}^{-1}$  for the sample,  $1.4 \text{ ml min}^{-1}$  for the stannous chloride solution, and  $1.2 \text{ ml min}^{-1}$  for the hydrochloric acid. The reductant channel of the VGA-76 contained 25% stannous chloride (J.T. Baker Chemical Co, Phillipsberg, USA) in 20% hydrochloric acid (Fisher Scientific Co, Springfield, USA). The acid channel contained 5 M hydrochloric acid.

Total and inorganic Hg in urine were measured using Lindstedt's method (1970) and the values were expressed as  $\mu\text{g g}^{-1}$  creatinine. One milliliter of urine sample was reacted with 0.2 ml concentrated trace-metal grade

**Table 1.** Recommended instrument parameters for optimum VGA-76 operation

Parameters	
Wavelength (nm)	253.7
Slit width (nm)	0.5
Lamp current (mA)	4.0
Integration time (s)	3.0
Background correction	OFF
Rinse rate	1.0
Rinse time (s)	5.0
Delay time (s)	39.0
Multiples	4.0

sulfuric acid (Fisher Scientific Co, Springfield, USA) into polyethylene tubes. About 1.5 ml of 6% potassium permanganate (Sigma Chemical Co, St Louis, USA) were added to each sample and mixed well. Tubes were capped loosely and left overnight at room temperature. Excess permanganate was reduced with 0.3 ml of 50% APHA hydroxylamine hydrochloride (BDH Chemicals Ltd., Poole, UK). All urine samples were analyzed in duplicate. Urinary creatinine, glucose, phosphorus, calcium, magnesium, urea and uric acid concentrations were measured at the Department of Pathology, King Faisal Specialist Hospital using a Hitachi 717 analyzer (Boehringer-Mannheim, Mannheim, Germany).

Calibration standards were prepared daily using a manual standard addition procedure. Urine samples were divided into six equal portions. Using a 1000  $\mu\text{g ml}^{-1}$  Hg reference solution (Fisher Scientific Co., Springfield, USA) known amounts of aqueous Hg concentrations were added to these urine sample portions to give final concentrations which covered the range 0.5–6.0  $\mu\text{g l}^{-1}$ .

The analytical recovery for Hg at the various concentrations tested (2.5–30.0  $\mu\text{g l}^{-1}$ ) was 96–104%, which was thought to be satisfactory. The spiked urine samples were run with the test samples and blanks using the same analytical procedure. The detection limit for Hg in this study was 0.0025  $\mu\text{g l}^{-1}$ .

Precision was assessed from the within-day and day-to-day variation of the analysis of Hg in urine samples, expressed as the percent relative standard deviation (RSD). Table 2 gives the results of the within-run and between-run precision for various concentrations of urinary Hg. In both cases, the precision varied between 2.5 and 6.4%, depending on the urinary Hg concentration.

#### Statistical analysis

Due to the non-Gaussian distribution pattern of urinary Hg, non-parametric statistical tests were employed. These included the Wilcoxon matched-pairs test and Spearman's rank correlation coefficient using Statgraphics Softwares (1992).

**Table 2.** Precision at various concentrations of urinary Hg

Mean ( $\mu\text{g l}^{-1}$ )	SD	RSD (%)
<i>Within-run</i> <sup>a</sup>		
2.556	0.066	2.6
5.058	0.21	4.2
10.311	0.311	3.0
15.079	0.618	4.1
29.525	1.419	4.8
<i>Day-to-day</i> <sup>b</sup>		
2.61	0.104	4.0
5.189	0.13	2.5
10.342	0.378	3.7
15.579	0.314	2.0
28.941	1.862	6.4

<sup>a</sup> Based on seven repetitive measurements.

<sup>b</sup> Based on eight repetitive measurements.

## Results and discussion

Descriptive statistics, sample size, means, standard deviations, and the ranges for the measured variables: mercury (Hg), age, glucose, urea, creatinine, calcium, magnesium, phosphorus, uric acid and urine protein are provided in Table 3. Despite the lack of comparative study, it is of concern that the reported mean urinary Hg concentration ( $6.961 \pm 20.43 \mu\text{g l}^{-1}$ ) was significantly higher than the mean reference value proposed by WHO in 1991 for the non-exposed population (4  $\mu\text{g l}^{-1}$ ). Fifty-two females (23.1%) had urinary Hg levels above 4  $\mu\text{g l}^{-1}$ .

Since urinary Hg varies according to the urine flow rate (Diamond 1988), it is necessary to adjust the measured Hg concentrations in the screened urine samples for variations in the urine flow rate. This was done by dividing the urinary Hg concentration by the concentration of Cr in the urine sample. Urinary Hg levels of about 50  $\mu\text{g g}^{-1}$  Cr were seen after occupational exposure to about 40  $\mu\text{g m}^{-3}$  of air (WHO 1980). In this study, 15 of the screened women (6.7%) had Hg concentrations exceeding 50  $\mu\text{g g}^{-1}$  Cr. The mean was 107.211  $\mu\text{g g}^{-1}$  Cr, in the range 51.575–459.372  $\mu\text{g g}^{-1}$ . The cumulative distribution plots of urinary Hg both as  $\mu\text{g l}^{-1}$  and as  $\mu\text{g g}^{-1}$  Cr in the screened women ( $n = 225$ ) did not follow Gaussian distributions but were positively skewed (5.8 and 8.7, respectively) as shown in Figures 1 and 2.

For subsequent data analyses and interpretation, urinary Hg data expressed as  $\mu\text{g l}^{-1}$  were used. Frequency tabulation of the urinary Hg data revealed that 20% of the screened women had urinary Hg concentrations below the detection limit,

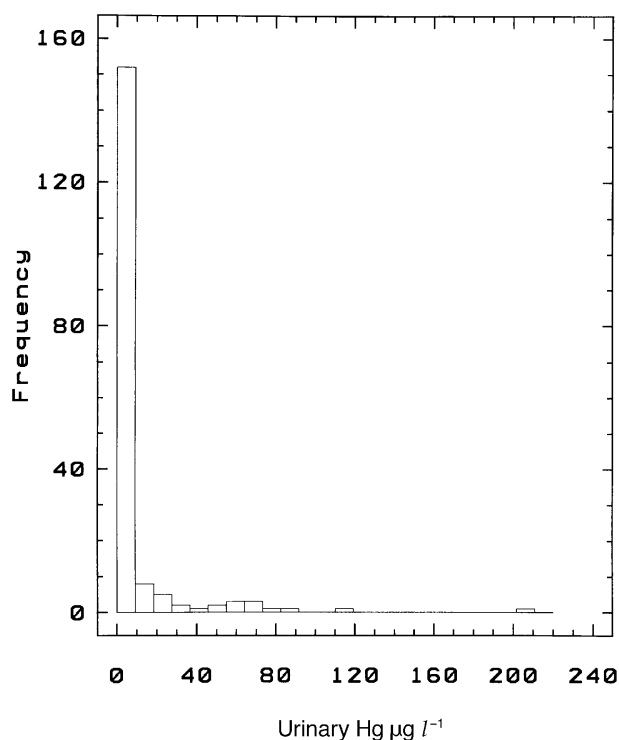
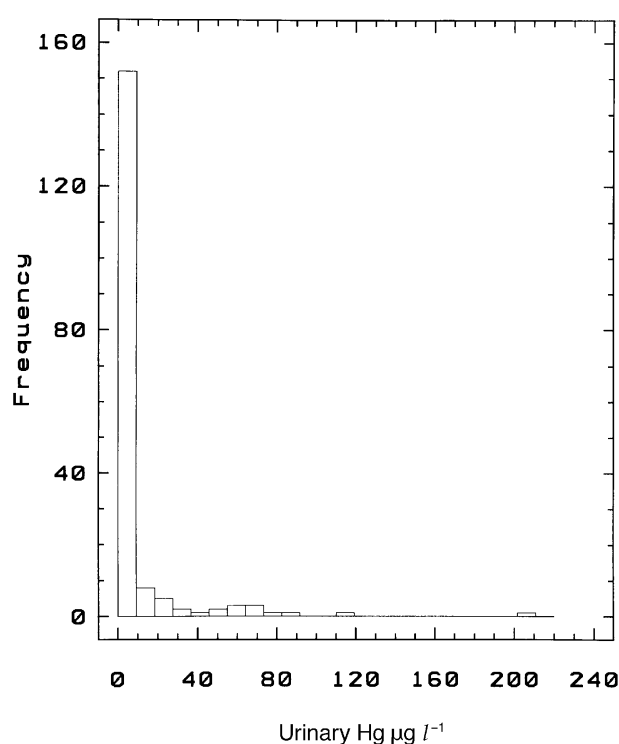
**Table 3.** Descriptive statistics of data

Variable	<i>n</i>	Mean	SD	Median	Range
Hg ( $\mu\text{g l}^{-1}$ )	225	6.961	20.430	1.16	0–204.8
( $\mu\text{g g}^{-1}$ Cr)		11.218	37.228	1.935	0–459.372
Age (years)	212	31.745	7.767	30.5	17–58
Creatinine ( $\mu\text{mol l}^{-1}$ )	225	7.306	5.815	5.7	0.05–26.3
Phosphorus ( $\text{mmol l}^{-1}$ )	224	15.227	15.054	11.6	0.2–130
Glucose ( $\text{mmol l}^{-1}$ )	223	1.932	12.519	0.2	–0.1–139
Urea ( $\text{mmol l}^{-1}$ )	224	185.812	151.496	158.0	4–608
Uric acid ( $\mu\text{mol l}^{-1}$ )	224	1072.862	841.035	828.0	116–4700
Magnesium ( $\text{mmol l}^{-1}$ )	224	3.601	17.865	1.88	0.04–268
Calcium ( $\text{mmol l}^{-1}$ )	222	1.905	2.017	1.145	–0.03–10.4
Urine protein ( $\text{mg dl}^{-1}$ )	223	–2.018	11.785	–3.0	–16–125

68% had concentrations less than  $10.0 \mu\text{g l}^{-1}$ , 3.6% had concentrations of  $10.0$  to  $20.0 \mu\text{g l}^{-1}$ , and 8.4% had concentrations greater than  $20.0 \mu\text{g l}^{-1}$  (Table 4). The Agency for Toxic Substances and Disease Registry (ATSDR 1992) proposed that a urinary Hg concentration of less than  $20 \mu\text{g l}^{-1}$  in adults with no known exposure to Hg is considered background where no clinical or physiological effects are observed. By contrast, urinary Hg concentrations from  $20$ – $100 \mu\text{g l}^{-1}$  are associated with subtle

changes in tests for nerve conduction, brain-wave activity and verbal skills, and are also an early indication of tremor. Therefore, in the present study,  $20 \mu\text{g l}^{-1}$  was assigned as the upper level of normal. Nineteen females had mean urinary Hg concentrations of  $60.303 \mu\text{g l}^{-1}$ , in the range of  $20.72$  to  $204.8 \mu\text{g l}^{-1}$ .

Looking at the Hg concentration in urine samples with respect to the use of skin-lightening creams, participants were classified into two groups.

**Figure 1.** Cumulative frequency distribution plot of urinary Hg in  $\mu\text{g l}^{-1}$ .**Figure 2.** Cumulative frequency distribution plot of urinary Hg in  $\mu\text{g g}^{-1}$  Cr.

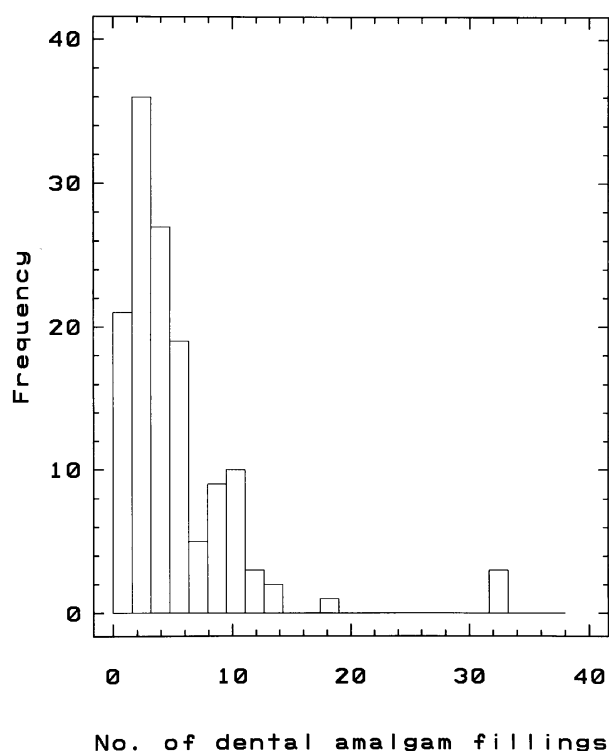
**Table 4.** Frequency distribution of urinary Hg in the screened women

Class intervals of urinary Hg data	<i>n</i>	%
Below detection limit	45	20
Less than 10	153	68
10–20	8	3.6
>20	19	8.4
Total	225	

**Group A:** 26 (12%) of the screened women were using 13 different brands of skin-lightening creams at the time of the study. The duration of use of these creams was from one week to ten years. Mercury was known to be higher than the FDA acceptable limit of 1 ppm in four of the creams; range 1.18–5650 ppm. A few animal studies (Friberg *et al.* 1961, Skog & Wahlberg 1964, Wahlberg 1965) have shown a certain degree of Hg penetration through the skin, but there have been few studies on humans. Marzulli & Brown (1972) found that the use of skin-lightening creams containing inorganic Hg salts caused substantial absorption and accumulation in the body. Repeated applications of these skin-lightening creams could cause cumulative effects of prolonged low level Hg exposure which, as shown in previous studies (Giunta *et al.* 1983, Rosenman *et al.* 1986, Enwonwu 1987), could lead to a nephrotic syndrome.

**Group B:** 191 (88%) of the screened women had never used skin-lightening creams. Although there was no statistical significant difference in urinary Hg between the two groups, urinary Hg levels (mean  $8.998 \mu\text{g l}^{-1}$ , range  $0\text{--}67.46 \mu\text{g l}^{-1}$ ) in group A were markedly higher than levels found in group B (mean  $5.463 \mu\text{g l}^{-1}$ , range  $0\text{--}113.68 \mu\text{g l}^{-1}$ ). The range of urinary Hg levels reported here for group A was lower than those reported by Barr *et al.* (1973) and Marzulli & Brown (1972) for females using skin-lightening creams:  $0\text{--}220 \mu\text{g l}^{-1}$  and  $28\text{--}600 \mu\text{g l}^{-1}$  respectively. Also, 19.2% of group A had urinary Hg levels above the acceptable limit of  $20 \mu\text{g l}^{-1}$  (mean  $34.714 \mu\text{g l}^{-1}$ , range  $20.72\text{--}67.46 \mu\text{g l}^{-1}$ ). Surprisingly, the other 21 females who were using skin-lightening creams at the time of the study had urinary Hg levels of  $2.875 \mu\text{g l}^{-1}$ , in the range  $0\text{--}10.15 \mu\text{g l}^{-1}$ . On the other hand, 6.3% of group B females had elevated urinary Hg levels (mean  $59.288 \mu\text{g l}^{-1}$ , range  $23.7\text{--}113.68 \mu\text{g l}^{-1}$ ). This could be attributed to dental amalgam fillings or dietary sources. The lack of significant relationship between urinary Hg levels and the use of skin-lightening creams in our study may be due to the limited number of females who use Hg containing skin-lightening creams.

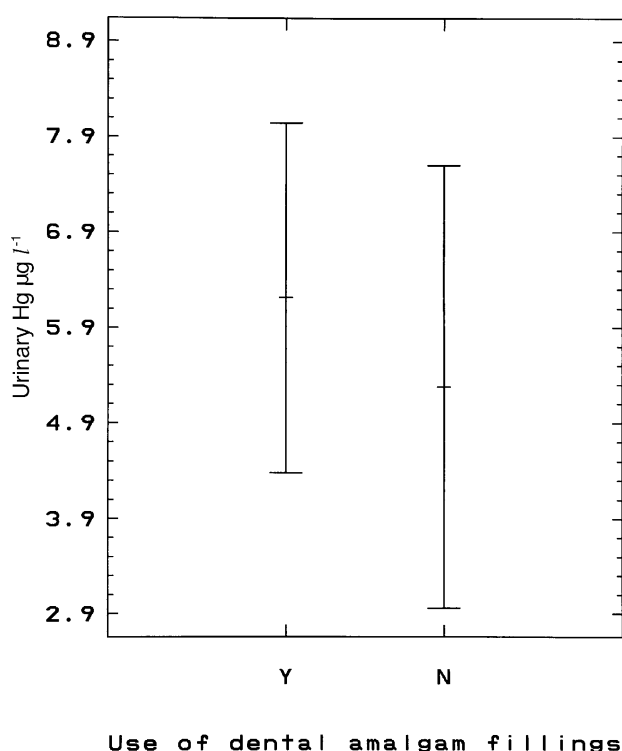
There has been concern and debate about potential adverse health effects from dental amalgam fillings. The distribution of dental amalgam fillings is displayed in Figure 3. Eighty-five females (38.5%) had no amalgam fillings, 96 had between one and five amalgam fillings, 27 had  $>5\text{--}10$  amalgam fillings, and 13 had more than 10 amalgam fillings. It has been suggested that Hg leaks from amalgam restorations contribute about  $1.7 \mu\text{g}$  of Hg to the Hg body burden (Mackert 1987, Berglund 1990). This is equivalent to  $1.7\text{--}8.5 \mu\text{g}$  Hg leak from females with one to five dental amalgam fillings ( $n = 96$ ). In females with more than 5 and less than or equal to 10 amalgam fillings, the Hg leak is expected to be  $8.5\text{--}17 \mu\text{g}$  ( $n = 27$ ). For females ( $n = 13$ ) who had dental amalgam fillings in the range of  $>10\text{--}32$ , the Hg leak will be  $>17\text{--}54.4 \mu\text{g}$ . This is considered to be high when compared to the WHO recommendation in which the total Hg daily intake for the non-exposed general population should be  $3\text{--}17 \mu\text{g day}^{-1}$  (WHO 1991). Factors such as chewing gum, tooth brushing, bruxism (a condition involving grinding or clenching of the teeth during sleep), and hot drinks will raise the daily intake of Hg (Bjorkman & Lind 1992, Barregard *et al.* 1995), possibly up to  $100 \mu\text{g day}^{-1}$ . Aronsson *et al.* (1989)

**Figure 3.** Histogram showing the distribution of dental amalgam fillings in the screened females.

found that the Hg concentration in intraoral air of 27 women ranged from 0.6–10.4  $\mu\text{g Hg m}^{-3}$ . Chewing gum for 5 min raised this to a maximum concentration of 2–60  $\mu\text{g Hg m}^{-3}$ .

Using the Wilcoxon matched-pairs test, urinary Hg levels were significantly higher in females with dental amalgam fillings ( $P < 0.02$ ). Graphical presentation of the mean urinary Hg concentrations, according to the use of dental amalgam fillings, is shown in Figure 4. The mean urinary Hg concentration for females with amalgam fillings ( $n = 136$ ) was 6.215  $\mu\text{g l}^{-1}$  or 12.043  $\mu\text{g g}^{-1}$  Cr, and for females without amalgam fillings ( $n = 85$ ) it was 5.278  $\mu\text{g l}^{-1}$  or 8.656  $\mu\text{g g}^{-1}$  Cr. There was a significant relationship between the number of dental amalgam fillings and urinary Hg levels ( $r = 0.1879$ ,  $P = 0.0053$ ). Similar observations were reported in adults and children (Aronsson *et al.* 1989, Suzuki *et al.* 1993, Begerow *et al.* 1994, Schulte *et al.* 1994, Herrstrom *et al.* 1995).

Table 5 lists females with urinary Hg concentrations greater than 20  $\mu\text{g l}^{-1}$  with respect to the use of skin-lightening creams and dental amalgam fillings. Five females with urinary Hg levels greater than 20  $\mu\text{g l}^{-1}$  were using skin-lightening creams at



**Figure 4.** Mean urinary Hg concentrations ( $\mu\text{g l}^{-1}$ ) according to the use of dental amalgam fillings.

the time of the study; three of these creams were proven to have a high Hg content (Al-Saleh & Al-Doush, in press). Four of these females also had amalgam fillings. Of the 17 females with elevated urinary Hg levels (mean 50.05  $\mu\text{g l}^{-1}$ , range 20.72–113.68  $\mu\text{g l}^{-1}$ ), 11 had amalgam fillings. No questionnaire was completed for the other two females in the table. Six females had no amalgam fillings, yet their mean urinary Hg level was 55.76  $\mu\text{g l}^{-1}$ , in the range 31.61–74.5  $\mu\text{g l}^{-1}$ . The use of skin-lightening creams could be the source of exposure in one of these females at a concentration of 5650 ppm. Other factors such as drinking water and diet might have contributed to the high urinary Hg levels. Within the general population, diet is the major source of Hg exposure, primarily through fish consumption (Turner *et al.* 1980). Between 70 and 90% of the total Hg in fish is in the form of methylmercury. For the general population not occupationally exposed to Hg, the estimated average daily intakes and retentions of inorganic Hg from air, water, and food are 0.002  $\mu\text{g}$ , 0.05  $\mu\text{g}$  and 3.6  $\mu\text{g}$ , respectively (WHO 1991). Further investigation is needed with respect to dietary intake of Hg.

The kidney is the primary organ involved in the excretion of certain nitrogenous wastes (creatinine, urea and uric acid), in the regulation of water and electrolyte balance, and in the excretion of fixed acids, therefore renal dysfunction is characterized by changes or abnormalities in one or more of these parameters. Some of these abnormalities (electrolyte or acid-base imbalance) are nonspecific, because they may occur in other disease states such as glucose. In this study, Spearman rank correlation coefficients between urinary Hg and creatinine, glucose, calcium, magnesium, phosphorus, urea, uric acid and urinary protein were tested for all participants. As shown in Table 6, the positive correlation between urinary Hg and creatinine, urea, uric acid, magnesium, phosphorus, calcium and glucose may be an early sign of renal dysfunction in the screened population. On the other hand, there was no relationship between the urinary Hg levels and urine protein. Eight of the screened females had a sign of proteinuria with a mean urine protein level of 38.875  $\text{mg dl}^{-1}$ , in the range 11–125  $\text{mg dl}^{-1}$ . Two of these females had high urinary Hg levels. Proteinuria usually indicates injury to the glomerular membrane, which permits the filtration or escape of protein molecules (Kaplan *et al.* 1988). Using the Wilcoxon matched-pairs test, urinary Hg levels were significantly higher in females with positive urine protein results ( $P < 0.05$ ). The mean urinary Hg concentrations for females with positive urinary protein

**Table 5.** Women who had urinary Hg levels above 20 µg l<sup>-1</sup>

Urinary Hg level (µg l <sup>-1</sup> )	Use of skin-lightening cream	Frequency	Duration (years)	Number of dental amalgam fillings	Duration (years)
82.7	NO			4	3
113.68	NO			4	1
65.89	NO			6	7
72.4	NO			NONE	
46.9	NO			NONE	
67.46	YES	Once per day	0.5	4	0.167
57.1	NO			2	15
20.72	YES	Once per week	10	4	14
31.61	YES	Twice per day	2	NONE	
20.72	YES	Once per day	1	1	4
33.06	YES	Once per day	7	3	6
55.93					
204.8					
25.45	NO			4	2
58.05	NO			NONE	
40.02	NO			5	0.67
74.5	NO			NONE	
23.7	NO			2	1
51.07	NO			NONE	

( $n = 70$ ) was 7.327 µg l<sup>-1</sup> or 12.043 µg g<sup>-1</sup> Cr, and for females with negative urine protein results ( $n = 153$ ) it was 6.723 µg l<sup>-1</sup> or 8.656 µg g<sup>-1</sup> Cr. Similar observations have been reported elsewhere (Joselow & Goldwater 1967). Chronic exposure to inorganic Hg at lower levels can cause tubular injuries in the kidney and glomerular lesions (Tubbs *et al.* 1982). Other factors such as immunological and idiosyncratic factors play a major role in causing glomerular damage following exposure to inorganic Hg (Oliveira *et al.* 1987). More precise analyses are needed to investigate this observation.

In conclusion, the results of this survey clearly showed that screened females were exposed to Hg. The findings concerning dental amalgam fillings warrant further research to determine the magni-

tude of the effects of Hg exposure on health and to clarify its contribution to Hg body burden. Furthermore, environmental, cultural and dietary investigations are needed to identify the sources of Hg exposure. Although the data in this study are not representative of the general population, they reveal an urgent need to monitor and reduce Hg in the environment of Saudi Arabia.

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**Table 6.** Correlation coefficients of urinary Hg and renal parameters

Variable	Correlation coefficients	P values
Creatinine	0.4568	<0.0001
Calcium	0.2732	<0.0001
Glucose	0.3498	<0.0001
Magnesium	0.4006	<0.0001
Phosphorus	0.397	<0.0001
Urea	0.4398	<0.0001
Uric acid	0.1112	>0.05
Urinary protein	-0.0234	>0.05

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