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Individual mercury exposure of chloralkali workers and its relation to blood and urinary mercury levels

by GÖSTA LINDSTEDT, Ph.D.,[1] INGEMAR GOTTBERG,[2] BERIT HOLMGREN, M.D.,[2] TORBJÖRN JONSSON,[2] and GUNNAR KARLSSON[2]

LINDSTEDT, G., GOTTBERG, I., HOLMGREN, B., JONSSON, T. and KARLSSON, G. Individual mercury exposure of chloralkali workers and its relation to blood and urinary mercury levels. *Scand. j. work environ. & health* 5 (1979) 59—69. On two occasions, chloralkali workers were investigated with regard to personal air mercury exposure, blood mercury and urinary mercury. The first investigation (13 workers, 2 weeks) was made at an exposure above the threshold limit value (64 $\mu g/m^3$, range 36—112), the second (16 workers, 8 weeks) at a lower exposure (23 $\mu g/m^3$, range 15—43). At the higher level of exposure, good correlations were found between air exposure and blood or urinary mercury for the group, but not for individuals. At the lower level, the correlations were less pronounced for the group. For individuals, the best correlation was found between mean air exposure during one week and blood mercury about half a week later. Other individuals, mainly the least exposed, showed no such correlation. Corresponding correlations were not found for urinary mercury. The urinary excretion rate was determined only for the last few hours of the workday, but the results agree with earlier investigations of 24-h excretion on a group basis. The threshold limit value for mercury in air (50 $\mu g/m^3$) corresponds to 150—175 nmol Hg/l blood (= 30—35 $\mu g/l$) for the group, with large individual variation.

Key words: air, blood, chloralkali plants, excretion rate, mercury, personal air samplers, urine.

In chloralkali processing plants, a salt solution is electrolyzed in large, flat cells, a thin, floating mercury layer on the bottom of the vessel serving as the cathode. Sodium ions from the solution are reduced to a sodium amalgam, which is transferred to a separate vessel where the amalgam is decomposed by water. The products formed are a sodium hydroxide solution, hydrogen, and pure mercury, which is pumped back into the electrolysis vessel. Large amounts of metallic mercury are handled in such plants, and the workers are always exposed to mercury vapors, and, to a smaller extent, to some inorganic mercury compounds in the air. At the anode chlorine gas is liberated, and leaks of chlorine form another hygienic hazard in these plants.

In most chloralkali processing plants, the content of mercury vapor in the air is controlled either by the absorbtion of mercury

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in some solution (e.g., potassium permanganate), followed by chemical analysis, or by direct-reading mercury monitors. The individual exposure of the workers is generally monitored from the urinary mercury excretion or the blood mercury content. In Sweden no biological limit values have, as yet, been established for mercury. Different investigators have proposed different "safety limits" for mercury in urine, but it seems as if signs of mercury poisoning are seldom observed in persons having levels of less than 750—1,000 nmol (= 150—200 μg) per liter of urine. Observations of the mercury content of blood are less frequent in the literature. In later years, however, better methods of mercury analysis have made rather accurate determinations of blood mercury possible.

Few observations of the mercury content in urine or blood from known air exposure levels have been published. The literature before 1972 has been reviewed and discussed in a monograph (3). In 1962, Goldwater and his colleagues (4) studied the mercury content in the urine and blood of workmen from four different factories, but the mercury-in-air content was only roughly estimated for each factory. Tejning et al. (17) tried to estimate individual mercury exposure for chloralkali workers by monitoring with direct-reading instruments. The 24-h mercury excretion in the urine was also measured for a 10- to 12-d period.

In 1970, Smith et al. published an investigation of 642 workers from 21 chloralkali plants in the United States (15). Both blood and urine were analyzed for mercury four times a year, and air analyses were made at several stations in the work areas. With the aid of these analyses, the time-weighted mercury exposure was estimated for each worker. Fairly good correlations were found between air exposure and the mercury content of blood or urine for the whole group. Hernberg and Häsänen (6) studied the relation between mercury in blood and urine for 27 exposed workers (26 of whom worked in chloralkali plants), but individual air exposures were not investigated. Bell et al. (2) were the first to use personal air samplers to study individual mercury exposure. They investigated the relation between time-weighted air exposure and mercury in urine for four chloralkali workers. Blood mercury was not investigated.

Nakaaki et al. (12) intermittently exposed humans to known concentrations of mercury vapor in a chamber and followed the mercury excretion in the urine for different lengths of time (up to 8 d) after the exposure. In 1977 Ishihara et al. (7) studied mercury in blood (both organic and inorganic) and in urine for a group of female workers exposed to low mercury levels (< 20 μg Hg/m^3). No detailed information about the mercury concentration in the air was given.

The purpose of the present investigation was to study mercury in the blood and urine of chloralkali workers and compare the levels measured to individual air exposure as measured by personal air samplers or direct-reading monitors. During the first investigation, the mercury content in the air was mainly above the threshold limit value (TLV: 50 μg/m^3).

The second investigation was carried out about two years later. Better ventilation and other arrangements in the plant had resulted in a much lower mercury content in the air, well below the TLV.

MATERIALS AND METHODS

Subjects and timetable

Investigation I (March—April 1975). Thirteen male workers, aged 19—63 years (mean 46.8 years, SD $\pm$ 13.3) and employed for 0.5—5.5 years, were investigated. All the men worked the day shift, Monday to Friday, in the cell room or on adjacent premises. From each worker, blood and air samples (data presented later) were taken daily for two weeks (on workdays only). In two cases due to absence from work we only obtained samples for one week.

Investigation II (January—March 1977). Sixteen male workers, aged 21—59 years (mean 43.8 years, SD $\pm$ 13.5) and employed for 1—7 years, took part in this investigation. They all worked days, Monday to

Friday, on the same premises as in investigation I. With two exceptions, they had no more than one fish meal a week. (One worker sometimes had two and another worker two or three fish meals a week.) During eight subsequent weeks air samples were taken daily, and blood and urine samples twice a week. Eight of these workers had also taken part in investigation I.

Physical examination

All workers were examined by the medical officer, and samples were taken for the investigation of hemoglobin, sedimentation rate, proteinuria, glycosuria, and serum creatinine.

In no case were any symptoms observed which could be connected with mercury intoxication, except that one worker in investigation I (worker L) had a permanent proteinuria. Serum creatinine and chest radiographs were normal for all subjects.

Sampling

Air samples. In investigation I, mercury in air was monitored by a Bacharach Mercury Sniffer, which was held a short distance away from the front of the worker. Readings were made for 5—10 min each time the worker moved to another place in the room, generally 10—20 times a day. The time-weighted average (TWA) exposure was calculated from the mercury readings. At the same time, an attempt was made to measure individual exposure with an adsorption tube filled with potassium permanganate on a Chromosorb carrier (5). Air was pumped through the tube with an MSA pump all day. However, this sampling method occasionally gave abnormally high mercury values and, on the average, about twice the values measured by the sniffer method. One explanation may be that the adsorption tube was pinned to the clothing on the worker's shoulder, and the mercury-rich atmosphere at the surface of the clothing may have influenced the analysis. Therefore, only sniffer readings were used in investigation I.

For investigation II, a special holder for the mercury adsorption tube was made which kept the tube free from the surface of the clothing and at the same time placed it closer to the worker's nose and mouth. The adsorption tubes were filled with 10—25 mesh manganese dioxide, as recommended by Janssen et al. (8). Air was drawn through the tube by a Sipin pump at a rate of 200 ml/min.

So that the Janssen method could be compared with the sniffer method, a series of double determinations were carried out (N = 30). An assistant followed the worker the entire day, holding the Bacharach sniffer close to him, reading the instrument at intervals and calculating the TWA. This figure was compared to that found by the adsorption tube method. The average of 30 analyses was 25.4 μg Hg/m^3 (SD = 14.8) for the Janssen method and 22.6 μg Hg/m^3 (SD = 17.8) for the sniffer. The difference between the two means was not significant ($P < 0.01$), and a reliable linear regression was found ($r = 0.917$). The Janssen method was used throughout investigation II (about 600 samples).

Urine samples. At the end of the workday, urine was voided into 500-ml polyethylene bottles containing 1 g of amidosulfuric acid as a preservative. The urine was weighed, and a smaller volume (about 50 ml) was transferred to another polyethylene bottle, which was sent to the laboratory for analysis. The times for giving the urine sample and for the preceding voiding of urine were noted. In investigation I urine was collected each day for two weeks, and in investigation II twice a week for eight weeks.

Blood samples. Blood samples were taken by venipuncture at the same time as the urine samples and collected in heparinized plastic tubes, which were sealed by plastic stoppers and sent in for analysis.

Analysis

Mercury in the adsorption tubes was analyzed by a modification of the method of Janssen et al. (8). The manganese dioxide in the tube was transferred to a 100-ml

Table 1. Mean air mercury exposure and mercury in the blood and urine of 13 workers during two weeks (investigation I).

Worker	Age	Length of employment (years)	N	Mercury level								
				Air (μg/m³)			Blood (nmol/l)			Urine (nmol/l) (uncorr.)		
				Mean	SD	Range	Mean	SD	Range	Mean	SD	Range
A	25	5	9	35.7	4.2	28— 43	169	19	134—194	916	307	444—1,320
B	45	0.5	8	54.8	8.3	42— 69	230	32	169—269	757	287	259—1,240
C	52	3.5	10	76.2	16.6	55—103	332	92	224—498	1,160	312	717—1,470
D	36	2	9	92.6	34.1	56—164	470	70	364—593	1,530	732	579—2,040
E	19	0.5	10	81.8	26.3	57—136	200	115	80—388	679	147	428— 895
F	55	4	10	74.6	32.7	40—134	238	63	169—354	730	253	514—1,240
G	57	0.5	8	50.0	8.0	34— 58	171	41	114—219	492	190	229— 768
H	55	4	9	46.3	10.2	36— 64	176	33	124—219	562	180	389— 855
I	57	2	9	54.0	13.1	40— 78	104	20	75—139	827	231	264—1,050
J	39	2	8	53.8	11.4	36— 70	136	21	114—179	1,050	296	684—1,420
K	50	2	9	47.0	8.7	37— 62	229	23	199—259	534	174	289— 876
L [a]	63	5.5	5	52.8	8.1	41— 63	157	22	119—174	369	141	229— 544
M [a]	54	4	5	112.0	22.5	90—142	477	186	244—677	904	373	378—1,390
Whole group				64.0	21.8	36—112	238	119	104—477	808	314	369—1,530

[a] Analyses from one week only.

flask and dissolved in a mixture of 10 ml of 6 mol/l hydrochloric acid and 2 ml of 12 mol/l nitric acid by boiling. The solution was cooled and diluted to 50 ml with water. Two milliters of this solution were pipetted out and analyzed for mercury in the automatic flameless atomic absorption apparatus described by Lindstedt and Skare (10). Control experiments showed that the sensitivity of the method was not influenced by the presence of manganese ions and that no mercury was lost during the boiling. A correction for the mercury content of the manganese dioxide was made on each run.

Urine and blood samples were analyzed as described by Lindstedt and Skare (10), the blood samples being digested in the acid mixture described by Skare (14). In investigation II, the density of the urine was determined by a refractive instrument, and the mercury content of the urine corrected to a density of 1.024.

Correction of air exposure values

The adsorption tubes were generally worn the entire workday (investigation II). If, for some reason, the worker was absent from work for some time, the tubes were worn only as long as he wore his workclothes, e.g., generally as long as he was in the plant, including meal times. It should be noted that the air in the lunchroom and similar rooms is often as contaminated by mercury as the cell room, and consequently the personal air sampler was running also during meals or coffee breaks. From the amount of mercury found in the adsorption tube, the TWA for an 8-h day was calculated.

Calculation of urinary mercury excretion rate

It was not practically possible to measure 24-h mercury excretion in the urine. Instead, the excretion during the last few hours of the workday was determined (investigation II).

The time between the last two voidings of urine was noted, and the urine from the last voiding (at the end of the workday) was analyzed after weight and density determinations. From these data, the mercury excretion rate was calculated and expressed as micrograms of mercury per minute.

RESULTS

Investigation I

In table 1 all analyses of mercury in air, blood, and urine from investigation I are summarized. With few exceptions, the samples were taken on Monday through Friday for two consecutive weeks. As can be seen from the table, 10 of the 13 workers had an average mercury exposure in excess of the TLV (50 μg Hg/m^3).

From the material the following relations between different analyses were studied: (a) correlation of air mercury levels and blood or urinary mercury levels on the same day for individuals, (b) correlation between mean air mercury and mean blood or urinary mercury for the whole group, and (c) correlation between blood and urinary mercury for individuals and for the whole group.

For each individual, the correlation between daily air exposure and daily blood or urinary mercury was generally very poor and, in some cases, even negative. Evidently blood or urinary mercury at the end of a workday cannot be expected to reflect the air exposure on that same day.

If the mean values for each subject during the whole period are compared, a better air/blood correlation is obtained within the group ($r = 0.83$, $P < 0.001$) (fig. 1). The corresponding regression line found by Smith et al. (15), as estimated from their diagram, is also displayed in fig. 1. It should be noted that several points of Smith et al. are well beyond the range of our investigation (up to 250—300 μg/m^3). The short duration of this investigation (2 weeks) does not allow speculation about any possible time lag between the changes in air exposure and their influence upon mercury in blood. However, the correlation between the average exposure during the whole period and the average of the last three blood analyses was studied. A still higher correlation (0.93) was found ($P < 0.001$).

Fig. 2 shows the regression line for the air/urine values (urine analyses are uncorrected for density). As could be expected, the correlation is less strong than for air/blood ($r = 0.64$, $P < 0.05$). In this figure, too, we have tried to transfer the

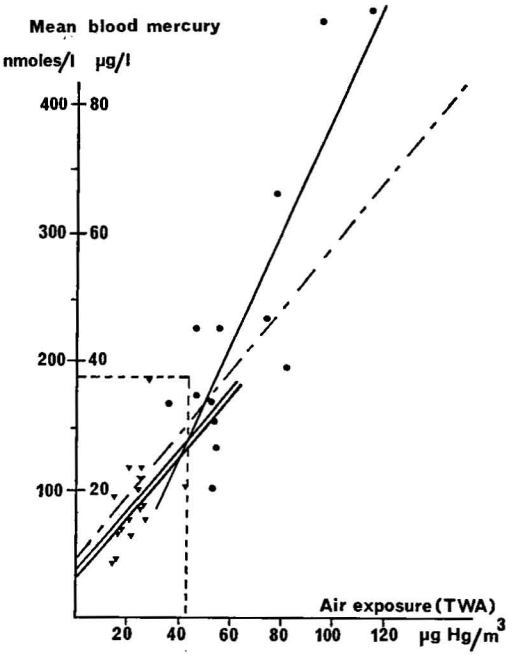

Fig. 1. Air exposure (time-weighted average) and mean blood mercury of 13 workers in investigation I (●) and 16 workers in investigation II (▼). Regression lines: investigation I —; investigation II =; estimated from Smith et al. (15) — - —. The broken frame (- - -) limits the area of investigation II.

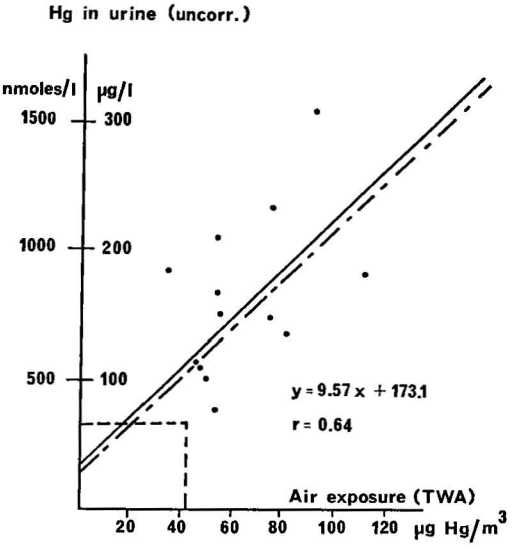

Fig. 2. Air exposure (time-weighted average) and mean urinary mercury (uncorrected for density) of 13 workers in investigation I. Regression lines: investigation I —; estimated from Smith et al. (15) — - —. All analyses from investigation II are within the broken frame (- - -). (Compare with fig. 3)

regression line from Smith's work to our diagram. The two lines are almost identical.

The correlation between simultaneous mercury content in the blood and urine of each individual was also studied. Like the air/blood and air/urine relations, the correlation was poor or negative for most subjects. Only two workers, M and G, had relatively high r values (0.91 and 0.67, respectively). M was the most heavily exposed worker in this group.

The mercury-in-blood/mercury-in-urine relation for the whole group is demonstrated in table 4; it is discussed in the next section.

Investigation II

The average air exposure and the blood and urinary mercury values for each worker involved in investigation II are summarized in table 2. It should be noted that investigation II covered a much lower range of mercury exposure than investigation I or any previous investigation of mercury-exposed workers. The broken frame near the origin in fig. 1 shows the utmost limits of the corresponding variables in investigation II. The average mercury exposure for the whole group was less than half the TLV, and no single worker had a time-weighted exposure above the TLV during the whole period of eight weeks. Out of 122 man-weeks, during which air exposure was measured individually, only 4 had an average air exposure above the TLV.

From the material available, studies corresponding to those in investigation I were made. Since investigation II had lasted eight weeks, conditions were more favorable for the study of possible time lags between changes in air exposure and blood or urinary mercury levels.

Mercury-in-air/mercury-in-blood correlation. In fig. 1 the average air exposure and the average blood mercury level for each worker are plotted against each other. For the entire group, the correlation is much lower than in investigation I ($r = 0.49$, $P > 0.05$). The regression line also deviates rather much from that of investigation I. One worker (C) had an exceptionally high blood mercury level. The same man was heavily exposed during investigation I and had a correspondingly high blood mercury value. Before being employed in the chloralkali processing plant, he had been exposed to mercuric chloride for 15 years in another job.

From an investigation in another chloralkali processing plant, it has been proposed that the best correlation should be between blood mercury and air exposure 30 d earlier (unpublished results of Piikivi, Hassi and Yrjänheikki 1975). Therefore, we investigated the correlation between the average air exposure over the first four weeks and average blood mercury levels over the subsequent four weeks. The correlation was no better than for the whole period ($r = 0.44$, $N = 12$, not significant).

Better correlations were found between the average air exposure during all eight weeks and the very last blood mercury analysis ($r = 0.80$, $P < 0.01$) or the average between the last three blood analyses ($r = 0.67$, $P < 0.01$). Also, when the same calculation was made for four-week or two-week periods, a statistically significant correlation was found between the air exposure and the very last blood mercury analysis (for four-week periods: $r = 0.82$, $N = 27$, $P < 0.001$; for two-week periods: $r = 0.73$, $N = 53$, $P < 0.001$). It is somewhat puzzling that the last blood mercury analysis should be so strongly correlated to the average exposure, independent of the length of the period.

The method used for measuring air exposure in this investigation reflects individual exposure better than any methods used before in chloralkali processing plants. Therefore, it was of interest to study the relation between air exposure and blood mercury for each individual. A comparison of the averages for each week is not relevant, since the first blood sample was taken on Monday or Tuesday and thus cannot be influenced by the air exposure later in the week. We first calculated the correlation between the air exposure one week and the average of the two blood samples taken at the end of that same week and at the beginning of the next week. This value corresponds to an average time of about 4 d between the air and blood samples.

Table 2. Mean air exposure and mercury in the blood and urine of 16 workers during eight weeks (investigation II).

Worker [a]	Age	Length of employment (years)	Mercury level								
			Air (μg/m^3)			Blood (nmol/l)			Urine (nmol/l) (d = 1.024)		
			Mean	SD	Range	Mean	SD	Range	Mean	SD	Range
A	27	7	20.5	6.4	15—36	80	11	50— 95	153	8	105—225
C	54	5	29.1	16	17—69	187	41	135—280	327	76	200—480
F [b]	57	6	25.0	2.4	21—28	110	10	95—125	117	15	90—140
G	59	3	18.8	5.9	11—31	118	13	100—135	124	14	100—145
H	58	6	43.0	14	25—75	106	18	70—145	235	28	165—270
I	59	4	14.7	2.4	11—17	95	25	70—175	207	30	160—265
J	41	4	23.7	6.7	15—33	101	9	90—125	283	27	230—320
K	52	4	23.8	6.4	15—33	117	16	90—155	154	24	120—210
N [b]	39	7	18.3	2.5	15—23	71	8	65— 80	154	18	120—180
O	53	1	24.8	5.8	15—32	88	10	70—110	297	26	250—340
P	47	4	26.7	20	9—60	80	23	60—145	122	19	90—145
Q	22	2	22.2	12	13—50	66	14	50—100	287	47	170—385
R	33	6	14.9	5.5	8—24	45	8	20— 55	122	21	90—150
S	21	2	15.6	6.9	9—31	49	12	20— 65	183	42	135—275
T	48	6	24.6	9.6	13—44	90	19	65—145	171	27	125—220
U [c]	30	2	16.2	6.7	7—23	69	9	60— 90	(61	35	20—130) [d]
Whole group			22.6	7.0	15—43	92	34	45—187	196	72	117—327

[a] Workers A through K also took part in investigation I.
[b] Analyses from six weeks only.
[c] Analyses from seven weeks only.
[d] Excluded from calculations.

Table 3. Correlation between mean air exposure during one week and blood mercury about four days later for each individual (16 workers; investigation II).

Worker	Regression equation[a]: y = ax + b		n	r	P	r_1 [b]	r_2 [c]
	a	b					
P	1.11	54.1	8	0.98	< 0.001	0.97	— 0.15
Q	1.06	45.7	8	0.86	< 0.01	0.74	— 0.49
K	0.67	97.7	8	0.86	< 0.01	0.53	0.24
H	0.98	68.5	8	0.85	< 0.01	0.91	— 0.67
O	1.47	50.6	8	0.80	< 0.05	0.74	0.72
U	1.04	50.1	6	0.79	> 0.05	0.81	— 0.14
C	1.40	139.4	8	0.76	< 0.05	0.81	0.30
J	0.92	79.4	7	0.75	> 0.05	0.78	0.37
S	1.15	31.1	8	0.71	< 0.05	0.60	0.41
T	1.11	61.1	8	0.67		0.80	0.44
A	0.51	70.8	8	0.59		0.72	0.30
F	1.83	64.9	6	0.48		0.53	— 0.46
R			8	— 0.02	> 0.05	— 0.22	0.27
I			8	— 0.18		0.42	— 0.22
G			8	— 0.24		— 0.23	0.33
N			6	— 0.25		— 0.12	— 0.54

[a] x = mean air exposure (μg Hg/m^3) per week (5 analyses: Monday — Friday); y = mean blood mercury (nmol Hg/l) (2 analyses: Thursday — Monday or Friday — Tuesday).
[b] r_1 = correlation coefficient between air exposure one week and the *last* blood mercury analysis the same week (Thursday or Friday).
[c] r_2 = correlation coefficient between air exposure one week and blood mercury the next week.

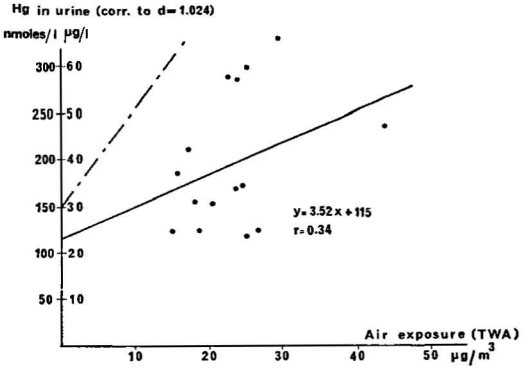

Fig. 3. Air exposure (time-weighted average) and mean urinary mercury (corrected to d = 1.024) of 15 workers in investigation II. Regression line: —; corresponding regression line estimated from Smith et al. (15) — - —.

The result of this comparison was somewhat surprising (table 3). The majority of the subjects showed an excellent to good correlation between the average air exposure one week and two blood analyses shortly thereafter (Thursday and Monday or Friday and Tuesday). A positive correlation was obtained for 12 out of 16 subjects. Statistical significance could be proved for seven subjects ($P < 0.05$ or better). The four subjects with a weakly negative correlation differed very sharply from the rest of the workers studied. All of them had a very low level of exposure, with little variation during the period (cf. tables 2 and 3). Evidently, sources of mercury exposure other than inhaled air become important at this low level.

Similar results were obtained if the air exposure during the week was compared to the last blood mercury analysis of the same week (Thursday or Friday) (see r_1 in table 3).

We also studied the correlation between mean air exposure one week and blood mercury measured the next week for each individual. This correlation was much less pronounced. Only one subject had an $r > 0.5$, and seven showed a weakly negative correlation (see r_2 in table 3).

The absolute level of blood mercury at a given air exposure depends both on the slope of the regression line (= a, table 3) and on the intercept, e.g., the blood mercury level at zero air exposure (= b, table 3). From the regression equations given in table 3, it is evident that the slopes vary considerably from 0.5 to 1.8 but also that those of six subjects are within 1.0 ± 0.1.

The intercepts vary between 31 and 139 nmol Hg/l blood (mean 67.8). This mean is more than that of "normal" values of blood mercury found in nonexposed persons (generally 25—50), and some additional mercury source, such as skin exposure, is therefore indicated. In this case, too, worker C manifests both a steep slope and a high intercept. His fish consumption was not more than one meal per week.

Mercury-in-air/mercury-in-urine correlation. When the results of the urine analyses are considered, it is impossible to notice any tendency of the urinary mercury to reflect sudden changes in air exposure, as is the case for blood mercury. The regression calculation for the whole group, on the average for the whole period, gives a rather poor correlation ($r = 0.34$, not significant) (fig. 3). If the average exposure for the first four weeks is correlated to the urinary mercury for the last four weeks, the result is not much better ($r = 0.41$, not significant).

Attempts to correlate individual air exposure to mean urinary mercury levels either the same or the following week gave very poor results. About half of the subjects had a weakly negative correlation. Thus for most subjects it is impossible to estimate recent exposure from urinary mercury levels. Mercury in urine is supposed to reflect whole body burden, which cannot be determined by experiment. This hypothesis agrees well with the high urinary mercury level of worker C, who can be suspected to have the highest body burden due to his long exposure time. Of course, individual differences in urine elimination capacity may also influence the urinary mercury level.

Excretion of mercury in the urine. As mentioned before, an attempt to determine the excretion rate of mercury in the urine during the last few hours of the workday (between the last two voidings of urine) was made. The mean urinary excretion rate was 0.037 µg/min (range: 0.021—

0.063). Worker U was excluded as his analyses were suspected to be incorrect.

Statistically significant correlations were not found between the mean urinary excretion rate and the mean air exposure of the whole group (N = 15, r = 0.42) or between the mean urinary excretion rate and the mean blood mercury level (N = 15, r = 0.47). A significant correlation was, however, found between the excretion rate and blood mercury level on the same day (N = 209, r = 0.42, P < 0.001).

Mercury-in-blood/mercury-in-urine relation. In table 4 previous investigations of mercury in blood and in urine are summarized and compared with our own results. Three of the authors cited, Smith et al. (15), Hernberg and Häsänen (6), and Miller et al. (11), have studied chloralkali workers, whereas the other authors studied workers from nonspecified industries. The data presented are quite independent of any estimation of air exposure, but of course blood mercury analyses in the earlier papers may be suspected to be less reliable than those made with modern methods of analysis.

Most authors investigated the subjects on one occasion only. Smith et al. gave the mean from four samples during one year. Our own data are means from two weeks (10 samples) in investigation I and eight weeks (16 samples) in investigation II.

It is evident from the table that most investigations have dealt with heavily exposed workers. Investigation II, which has the lowest degree of exposure, has the poorest correlation between blood and urinary mercury and the lowest quotient $\bar{y} : \bar{x}$. Evidently the low range of exposure

Table 4. Correlation between blood mercury and urinary mercury found by other authors, compared to the present investigations.

Authors	Air exposure ($\mu g/m^3$, range)	Number of subjects	Analyses per subject	Hg/blood (nmol/l) $\bar{x}$	Hg/urine (nmol/l) $\bar{y}$	$\frac{\bar{y}}{\bar{x}}$	Correlation r	Correlation P
Goldwater et al. 1962 (4) (3 series)	100—2,400	28—34	1?	402—550	1,570—2,880 [b]	3.6—5.5 [b]	0.62—0.68	< 0.01
Joselow et al. 1968 (9)	?	40	1?	? (range 165—1,270)	? (range 250—8,700 [b])	?	0.63	< 0.001
Smith et al. 1970 (15)	10—270	389	4	? (range 50—550)	? (range 250—2,500 [a])	?	?	< 0.001
Hernberg and Häsänen 1971 (6)	50—150	27	1	129	992 [a]	3.4 [b] 7.7 [a]	0.86	< 0.001
Miller et al. 1975 (11) (4 series)	?	10—24	1	198—853	643—3,920 [b]	2.9—4.6 [b]	0.99	< 0.001
Present investigation I 1975	36—112	13	5—10	238	807 [b]	3.4 [b]	0.60	< 0.05
Present investigation II 1977	15—43	15	16	93	195 [a]	2.2 [a]	0.37	> 0.05

[a] Corrected to a urinary density of 1.024.
[b] Uncorrected for urinary density.

ought to be studied further. When these quotients are compared, it is necessary to distinguish between corrected and uncorrected urinary mercury analyses.

The correlation between daily blood and urinary mercury for each individual was also studied (investigation I). In most cases the correlation was very poor or even negative. For only one worker was there a clear positive correlation (r = 0.80).

DISCUSSION

Previously, only Smith et al. (15) have reported the correlation between individual air exposure and mercury in blood. In agreement, we also found a fairly good correlation on a group basis in investigation I. The latter investigation covers a lower range of exposure than the former, but the mean exposures are similar in both investigations (about 65 $\mu g/m^3$). The regression lines differ somewhat however (fig. 1).

Investigation II covered a low range of mercury exposure seldom investigated before. The individual air exposure measurements must be considered more reliable than those of investigation I. The correlation of air exposure and blood mercury for the group was less pronounced, but the regression line is very close to that of Smith et al. (15) (fig. 1). It has not been possible to establish a certain optimal time lag for the influence of air exposure upon blood mercury for the entire group. Surprisingly, however, the correlation between mean air exposure and the very last or the last three blood analyses is strong for two-, four- and eight-week periods.

The results obtained by studying each individual are most interesting. Up to half of the subjects studied showed a strong correlation between air exposure and blood mercury 3—4 d later, but not one week later. Others, mainly those with low exposure, showed no such correlation. This study ought to be continued on subjects with higher exposure levels. The difference in uptake efficiency between individuals (that is, the slope of the regression line) is also interesting. Such differences may be due to variation in mercury uptake, excretion and distribution between individuals or to differences in work load. The volume of air inhaled during a day differs depending on the work load. For organic solvents, it has been shown that the concentration in the blood varies with the work load if the concentration in the inhaled air is the same (1). During this investigation, it was not possible to pin point which workers had heavier work loads. All can be said to have a medium work load however.

Now that an effective method of personal air sampling for mercury is available, further investigations of individual variation in uptake, distribution and excretion should be encouraged.

If blood mercury corresponding to the TLV for mercury in air (50 $\mu g/m^3$) is calculated on a group basis, investigation I gives about 175 nmol/l (= 35 $\mu g/l$) and investigation II 150 nmol/l (= 30 $\mu g/l$). A possible biological limit value ought to be about that level, but individual variations are great and no symptoms of illness have been found in either of the present investigations.

Urine samples were taken at the end of the workday, mainly for the sake of convenience. Piotrowsky et al. (13) found that urinary mercury levels are generally high at night and in the morning and low in the afternoon, independent of the diurnal rhythm of exposure. This variation may explain the lowness of the ratio of urinary mercury to blood mercury in our investigations when compared to others (table 4). According to Piotrowsky et al. (13) the urinary excretion rate is also rather low in the afternoon. From data reported by Bell et al. (2), it is possible to compare afternoon excretion rates to 24-h ones. The 24-h rate is not always higher (5 subjects studied).

Tejning and Öhman (16) determined 24-h mercury excretion and air exposure for a group of chloralkali workers during four consecutive days. The six least exposed workers of their group had a TWA of about 50 $\mu g/m^3$ (estimated from a diagram in their paper). By extrapolating the regression line in investigation II, we estimated a urinary mercury excretion of

about 0.060 μg/min at an air exposure of 50 μg/m^3. If uniform excretion throughout the day is assumed, this level corresponds to 0.09 mg/d against a group mean of about 0.10 mg/d in Tejning and Öhman's results (range about 0.02—0.18 mg/d). Thus at least the order of magnitude for the group excretion rate is equal for both investigations, although we only determined afternoon excretion.

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