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## MERCURY URINALYSIS IN PERSPECTIVE

Sam Ziff

The value of urinary mercury analysis in determining the level of exposure or total body burden of mercury is as controversial as the question of dental amalgam's role in disease.

Utopia would be utilization of a simple urinalysis to determine those individuals at risk from their mercury body burden, regardless of the source. Unfortunately, scientific data clearly indicates urinary tests to be unreliable as a test for mercury body burden or tissue/organ damage. There is no question that, if done properly, the test will indicate mercury content of the urine from that particular sample. However, utilizing the proper testing protocol and then trying to determine what the results mean in relation to an individual's health is entirely another matter.

Most research on mercury urine content was originally undertaken in an effort to determine safe levels of exposure in the work place. The primary goal being to correlate urinary blood and air levels with established symptomatology of mercury intoxication. Therein lies the basic problem. Fairly acceptable correlations were achieved between urine mercury levels ( $\mu\text{g Hg/l}$ ) and atmospheric mercury ( $\text{mg Hg cu/m}$ ) (Friberg and Nordberg, 1972) and between mercury air concentrations and blood levels (Smith et al., 1970; Goldwater et al., 1964). The ratio of approximately 0.3 between blood and urinary mercury level appears in agreement with data from different researchers, Smith et al., 1970; Benning, 1958; Friberg and Nordberg, 1972.

The ability to relate urine levels to overt symptoms of mercury toxicity presents problems of much greater complexity and variability. It appears to have a degree of validity only when urine mercury concentrations exceed a specific level.

Henderson et al., 1974, presented a biochemical theory based on urine mercury concentrations greater than 0.5 mg/l. The theory was that mercury concentrations above 0.5 mg/l were capable of causing increased central nervous system absorption which would manifest as clinically observable neurotoxic effects.

Langolf et al., 1978, used quantitative measurements of neurological function to evaluate 143 chlor alkali workers. Monthly urine mercury determinations were routinely performed as part of the safety programs at the plants where these workers were employed. Langolf and his associates using these historical records in conjunction with their quantitative measurements of EMG, tremor, tapping, tracking and reflex found that "Peak historical levels beyond 0.5 mg/l urine mercury appeared to be most significant in predicting minimal effects." They went on to conclude: "Because of wide variability among individuals, a test score from a single individual can only be meaningfully compared to his own baseline score established prior to exposure."

(I consider that conclusion extremely significant when viewed in context with the ADA position paper on the safety of dental amalgam (JADA, 106:519-520, 1983) in which they cite the Langolf study as proof that there is no danger until urinary mercury levels reach 500 ug/l. To my knowledge I do not believe the ADA has established baseline data on any dental personnel).

In a follow-up study in 1981, Langolf and his associates provided the results of behavioral studies, with the same group of workers, carried out over a six year period. Their findings were quite significant. Some of the results and conclusions were: "Throughout the six year study, conventional medical examinations failed to detect any neurotoxic effects in these mercury cell chlor-alkali workers. Behavioral tests however, were consistently able to detect mercury related effects of a subtle subclinical nature. Regression analysis showed changes in tremor spectra as a function of workers increasing urinary mercury. ----- The tremor changes however, were associated with those workers whose urinary mercury exceeded 0.5 mg/l in two or more months of the previous year."

"Psychological tests of short term memory also showed high sensitivity in detecting subtle effects of mercury exposure. Results showed an increase in memory scanning time and a decrease in short term memory capacity as a function of workers' increasing urinary mercury. These changes resemble those which occur with aging. In fact, the changes in memory function associated with an increase in twelve-month average urinary mercury of only 0.1 mg/l may be functionally comparable to the effects of 10 years of aging. The relative size of these mercury related changes in memory functions was therefore surprising. Unlike tremor results, statistical analysis showed that memory effects may occur in worker groups whose urinary mercury is consistently maintained below the 0.5 mg/l limit."

The results of the psychological tests of short term memory serve to focus attention to the real problems of urine mercury concentrations as a measure of toxicity. Short of evaluating tissue samples there does not appear to be any accurate way of assessing subclinical long term effects of mercury utilizing urinalysis as a basis.

Some of the variables that enter into the basis of the foregoing statement are reflected in the findings and conclusions of the following researchers:

The National Academy of Science in their 1978 study titled An Assessment of Mercury in the Environment, considered proper collection, laboratory techniques and analysis instrumentation so important they included a 27 page appendix on the subject. Some of the critical points brought out were:

1. Possible loss of mercury compounds by volatilization during chemical processing.
2. During storage, mercury can be leached into solution from the laboratory or container materials or be lost to container walls by sorption (Campbell et al., 1972).
3. Urine samples must be preserved by the addition of potassium persulfate at the time of collection (Trujillo et al., 1974).