

Hypothetical estimation of 'Non-Mercury-Associated Selenium' in Human Autopsy Tissues of Mercury Exposed Idrija Residents and Mercury Mine Workers

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Key words: mercury, selenium, human, brain, gland

The precise mechanisms of mercury accumulation and retention are still unclear. Generally, the association of mercury with selenium is used to explain these phenomena. The coaccumulation of the two elements was found in different population groups (miners, dentists, and nonoccupationally burdened individuals) and the results suggest the formation of a biologically largely inert 1:1 Hg-Se compound. It seems that the presence of coaccumulated endogenous Se can protect cells from the harmful effects of Hg. However, as speculated by some authors (DRASCH ET AL., 2000, RALSTON, 2003), this binding of Se to Hg can also result in a relative deficiency of biologically available Se needed for selenoenzyme synthesis. That can lead to disturbances of free radical detoxification, thyroid hormone metabolism and production of immune system signal molecules. Therefore efforts were made tried to determine/estimate the amount of non-Hg-associated Se in tissues (DRASCH ET AL., 2000). Deriving from the assumption that Hg is almost quantitatively deposited in tissues bound to Se in a 1:1 ratio, the quantity of non-Hg-bound Se can be calculated by the difference between the molar contents of the two elements ($\text{Se}_{\text{mol}} - \text{Hg}_{\text{mol}}$).

In this study we used the same approach with the data from our previous investigation, where Hg and Se concentrations were determined in autopsy samples of mercury exposed and unexposed individuals: retired Idrija mercury mine workers, Idrija residents living in a Hg contaminated environment and a control group with no known Hg exposure from the environment (Falnoga et al 2000). Hg and Se were determined simultaneously by RNAA. Regarding these data we tried to estimate the influence of Hg exposure on the physiologically available selenium content in selected tissues, particularly endocrine glands and brain tissues.

Calculated values of non-Hg-bound selenium are presented in Table 1. Data are given as average value ($\text{Se}_{\text{nmol/g}} - \text{Hg}_{\text{nmol/g}}$) with standard deviation and number of samples for each group, except in cases where accumulated mercury highly exceeded selenium. In calculation this phenomena was manifested as negative selenium value and data are given individually.

Table 1.: Calculated values of 'non-mercury associated selenium' ($\text{Se}_{\text{nmol/g}} - \text{Hg}_{\text{nmol/g}}$) in endocrine glands and brain tissues of mercury exposed and unexposed individuals.

| Group | Pituitary | Thyroid | Hippocampus | Cortex cerebellum | Nucleus dentatus |
|------------------|-----------------|---------------------------|---------------|-------------------|------------------|
| Control | 4.0 ± 1.7 (18) | 5.2 ± 2.7 (20) | 1.5 ± 0.3 (8) | 1.8 ± 0.5 (7) | 1.8 ± 0.9 (12) |
| Idrija residents | 4.3 ± 1.4 (6) | 5.0 ± 1.7 (7) | 2.0 ± 0.2 (5) | 2.0 ± 0.4 (7) | 2.1 ± 1.6 (3) |
| Subgroup * | 14.4 (2) | 8.1 (2) | | | |
| Retired miners** | 5.1 (1) -3.5 | 2.7 (2) -14.5 -59.4 | 1 ± 0.5 (3) | 1.9 ± 1 (4) | 2.2 ± 2.3 (3) |

*Subgroup formed for two individuals who lived near mercury smelting plant and had extremely high content of Hg and Se in endocrine glands.** Results with negative Se values are separated and given individually.

Comparing the calculated values of ($\text{Se}_{\text{mol/g}} - \text{Hg}_{\text{mol/g}}$), it was found that:

- i) for Idrija residents the values were similar to those of the control group (individually slightly decreased or even slightly elevated);
- ii) as expected, diminished values were found in some mercury-loaded organs of retired Idrija miners. A downward trend in 'free Se' was also seen in the case of an active miner after working in the mercury mine for three years (data not shown).

It could be speculated that in Idrija residents Hg sequestration of selenium is sufficiently compensated with increased Se levels, and that particularly in active miners, the activity of selenoenzymes could be inhibited and the amount of Se necessary for synthesis of selenoenzymes insufficient.

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