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Mercury-selenium Interaction at Concentrations of Selenium and of Mercury Vapours as Prevalent in Nature

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The literature on selenium-mercury interactions is concerned with the effects of mercury administered either as mercuric chloride or methyl mercury chloride, while no attention has been paid to metallic mercury vapours, which is the commoner source of human mercury exposure. Therefore, it was decided to study the interactions of selenium and metallic mercury vapours on rats at occupational and environmental concentrations of mercury and selenium met with in certain occupations and environments.

MATERIALS AND METHODS

12 male albino rats (Wistar strain) in the weight range of 4-500 g were randomly divided into four groups (A1, A2, B1, B2). A1 and A2 were each placed first in normal rat cases, and the two groups B1 and B2 were placed in plexiglass-boxes (0.1 m^3) according to fig. 1. The diet was specially prepared according to HAFEMAN et al. (1974).

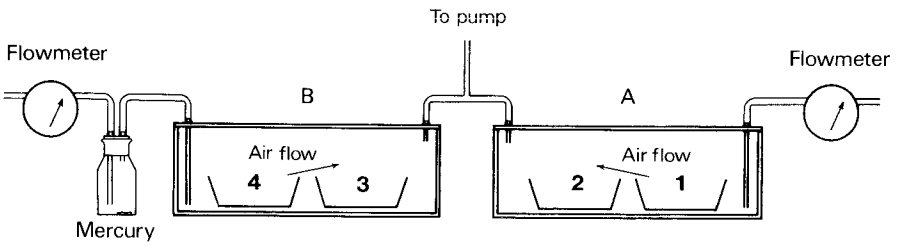

Fig. 1.

Analysis of selenium and mercury showed a content of 0.09 ppm selenium and 0.014 ppm total mercury in the diet. Food and water was offered ad libitum. Selenium was administered as Na_2SeO_3 through the drinking water to the groups A1 and B1 (conf. fig. 1) at a concentration of 0.5 ppm

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selenium. The rats were held in the boxes for 6 weeks without mercury administration to bring the rats in selenium balance at the two levels of selenium, which according to HAFEMAN et al. (1974) will take about 1 month. Then mercury vapours at a concentration of 30 mg/m^3 were administrated to the groups B1 and B2. The average air exchange was 7-8 times/hour. The mercury was given in twelve hours' intervals followed by twelve hours of normal atmosphere administration. This metallic mercury dose of 30 mg/m^3 twelve hours a day 7 days a week, equals approximately the occupational TLV of 50 mg/m^3 for 8 hours 5 days a week.

After four weeks of mercury administration the rats were sacrificed and mercury concentrations measured in different tissues.

RESULTS

The tissue concentrations of mercury in the four groups, A1, A2, B1, and B2 are given in table I. Among the different tissues analyzed lungs, blood, liver, and striated muscles showed the highest relative differences between the two groups A1 and A2 (conf.table II).

TABLE I

Groups of rats / Tissue	No exposure Hg ng/gr		Mercury exposed Hg ng/gr	
	A1 (+Se)	A2 (-Se)	B1 (+Se)	B2 (-Se)
Lung	7.3	17.3	63.7	85.3
Blood	7.0	5.7	14.7	11.7
Liver	55.0	24.7	224.3	153.3
Kidney	260.0	345.3	7225.7	8071.0
Spleen	6.0	6.7	33.7	38.7
Testis	5.0	4.7	21.3	16.3
Striated muscle	5.3	6.7	6.7	12.3
Heart	6.0	5.7	53.3	61.3
Brain	10.7	8.0	109.7	119.3
Spinal medulla	6.3	5.7	36.0	50.0
Skin	6.0	9.0	36.3	41.0

TABLE II

Tissue	Background exposure $\frac{A2-A1}{A1} \cdot 100\%$	Mercury exposure $\frac{B2-B1}{B1} \cdot 100\%$
Liver	+ 123	+ 46
Blood	+ 23	+ 12
Lung	- 58	- 25
Striated muscle	- 30	- 46

It will be seen that the mercury concentrations in the liver and blood increase by increasing selenium intake while in the lungs and in the striated muscles mercury concentrations decrease.

In the central nervous system the brain takes up relatively more mercury than the medulla spinalis. This is in contrast to what is found in rats exposed to $HgCl_2$ (20 ppm in drinking water for 1 year) where no differences could be seen between brain and medulla spinalis (DANSCHER & HANSEN, to be published).

DISCUSSION

The dietary selenium concentration of 0.1 ppm is according to SCHWARZ (1976) sufficient for protecting rats against liver necrosis and is thus regarded as the minimum essential selenium concentration. Our results show that the administration of selenium to rats influences the mercury concentration of different tissues. The differences are most pronounced at the low mercury uptake indicating a dose dependency (conf. table II). In the study by WELSH (1974) is stated that selenium can cause increased excretion of mercury. The excretion is not investigated in this study, but the lower lung concentration of mercury might indicate an increased loss of volatile mercury as mentioned by WELSH. The higher blood concentration induced by selenium shows that a greater part of the body-burden is in a mobile state. The increased liver concentration of mercury after selenium administration might indicate an influence of selenium on the entero-hepatic recycling system or a transport mechanism of mercury from other

tissues to the liver by selenium.

The influence of selenium on the excretory loss of mercury is going to be subject to further investigation. Our finding does not support the idea that selenium causes a greater body-retention as indicated by several authors, as the general tendency, is a lowering of tissue concentrations due to selenium administration.

CONCLUSION

The most striking conclusion from this experiment is that dietary selenium influences the distribution of mercury in tissues of rats exposed to occupational amounts of metallic mercury in the air. Moreover increased dietary selenium does not increase the tissue retention of mercury contrary to earlier investigations. Finally Hg^{o} and Hg^{++} seems to be distributed by different routes in the rat brain.

As a corollary it must be emphasized that blood mercury values are not a valid indicator of mercury exposure i.a. due to the fact that dietary selenium influences the mercury concentration in the blood.

Further research of the geographical distribution of dietary selenium should be carried out before evaluation of environmental and occupational exposure of mercury can be carried out.

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