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## **The impact of methyl mercury on the distribution of selenium in various tissues**

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There is evidence that a regimen of dietary selenium provides protection against the toxic biological effects of methyl mercury. Animal studies suggest that a potential mechanism is that selenium causes the redistribution of methyl mercury from critical organ tissue to muscle. In vivo experiments were conducted to assess the correlation of Se and MeHg in previously tested rat nail samples and various rat organ tissue samples. Groups (n=24) of weanling male Long Evans rats were fed torula yeast based diets containing either deficient Se, or supplemented with sodium selenite to adequate or enriched levels of Se. The group of 24 was then separated into smaller groups (n=8). Each separated group was given diets prepared with no methyl mercury, low amounts of methyl mercury or high amounts of methyl mercury. The rats were then sacrificed and the brain, liver, kidneys, pituitary, testes and nails were collected. The MeHg and Se content of each tissue was determined by standard comparator neutron activation analysis using the  $^{74}\text{Se}(n,?)^{75}\text{Se}$  and the  $^{202}\text{Hg}(n,?)^{203}\text{Hg}$  reactions. The organ tissue samples were irradiated for 50 hours at a flux of ca.  $5 \times 10^{13} \text{ n}\cdot\text{cm}^{-2}\cdot\text{s}^{-1}$  and allowed to decay for ten or more days. The samples were then measured using a Canberra spectrometer and compared to the data from the nail samples. The ratio of selenium to mercury in the nails and organ tissue will be used to verify if the nail is a viable biological marker for selenium and mercury uptake in various organ tissues.