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Elimination of selenium compounds by mice through formation of different volatile selenides

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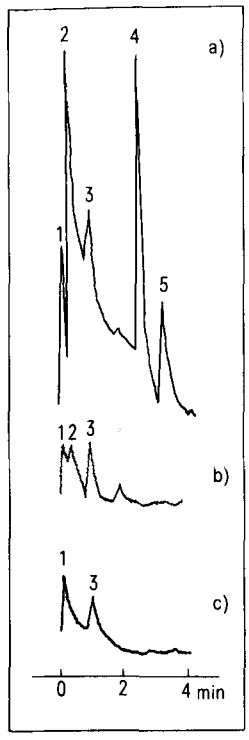
Summary. The chemical form of added selenium determines the amount and the species of selenide metabolites in the breath of mice. Exhalation seems to be a minor form of selenium elimination.

Animals, especially rats, given large doses of different selenium compounds, exhale volatile selenides, primarily dimethylselenide. Prochazkova et al.[2] trapped volatile selenium species, exhaled by rats after addition of selenite, on a synthetic adsorbent and separated them by gas chromatography. Dimethylselenide proved to be the major compounds, while a smaller amount of another selenium species was detected, which behaved on chromatography in a similar way to dimethyldiselenide. It has been shown for rats that the chemical form of selenium administered[3-5], the method of administration[5,6], the quantity[4,5], the purity of the added compound[7] and some properties of the diet[3,7-11] are determining factors in the quantity of volatile selenium formed, and therefore different experimental conditions were tested out on mice. By combining a cryogenic sampling procedure with a simple gas chromatographic-graphite furnace atomic absorption system as described by Jiang et al.[12] and applied for the detection and determination of alkylselenides in environmental air[13], we were able to determine different selenides in the breath of mice, down to 0.3 ng/100 g b.wt.

Addition of sodium selenite, D,L-selenomethionine or D,L-selenocystine to the drinking water of mice resulted in the formation of dimethylselenide and also dimethyldiselenide. The same amount of dimethylselenide was observed in the breath at different sampling times in both experiments with the seleno-amino acids added to the drinking water. For the selenomethionine a 3rd unidentified selenium metabolite was observed in the breath, which contributed to a large extent to the elimination of selenium by the lungs. Selenocystine and selenite addition to drinking water resulted in the production mainly of dimethylselenide, while for selenomethionine the main compounds were the dimethyldiselenide and an unidentified 3rd species as shown in the figure. These findings extend the observations of Thomson et al.[14] on the metabolism of selenocystine, selenomethionine and selenite in rats. They stated that the bioconversion of selenocystine ressembled that of selenite rather than that of selenomethionine.

Also, Greeder and Milner[15] proved that the effectiveness of selenite, selenate and D,L-selenocystine in limiting tumor growth in mice differs considerably from that of selenomethionine. Literature data on the different bioconversion of selenite and selenomethionine in rats has been summarized by Cary et al.[16].

D,L-selenocystine, added to the drinking water or i.p. injected into mice, is predominantly metabolized to the dimethylselenide species. Injection of a 8.4 times higher selenium concentration as D,L-selenocystine resulted in a



Selenium metabolites in the breath of mice after administration for 14 days of DL-selenomethionine (a), DL-selenocystine (b) and selenite (c) via drinking water. Peaks: 1 (air), 2 (water), 3 (dimethylselenide), 4 (unidentified compound) and 5 (dimethyldiselenide).

16 times higher concentration of dimethylselenide compared to the exhalation after administration of the same selenium species in the drinking water.

Even after 22 days of selenium administration via drinking water the amount of volatile selenide metabolites is still increasing. On the 14th day after starting the administration less than 0.3% of the daily intake of selenium is exhaled as volatile selenides. These findings are in contrast with similar experiments carried out on rats with selenite, where up to 22% is excreted via the lungs[4,5]. Sternberg and Imbach[6] confirmed the importance of the excretion by lungs for administered selenite as found by McConnell and Roth[5], but they were unable to detect any pulmonary excretion of selenium in rats administered D,L-selenomethionine.

Once more this proves that the chemical form of the added selenium is important in the bioconversion of this element. The identification of the 3rd selenium species in the breath of mice after administration of selenomethionine, and the underlying mechanisms of conversion, remain to the elucidated.

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Tryptophan and neutral amino acid concentrations in serum of rats after salmon calcitonin injection

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Summary. After a single injection of salmon calcitonin (2 MRC units/kg b.wt) marked decreases in both calcium and neutral amino acids in rat serum were observed. In turn, free tryptophan in serum and serotonin (5-hydroxytryptamine) in the whole brain were greatly enhanced during the initial period.

Although the level of plasma and brain free tryptophan has been reported to vary in the same direction under various experimental conditions[2,3], Nakhla et al.[4] have observed that in the rat, after a single injection of calcitonin, the level of plasma free tryptophan diminishes while the level of serotonin in brain increases. This peculiarity could be a consequence of the action of calcitonin on neutral amino acids, which share with tryptophan the same active system for crossing the blood-brain barrier[5]. This could be the reason why prior administration of valine could inhibit the increase of brain tryptophan under the influence of various stresses[6]. To investigate this, we have studied the influence of calcitonin on the levels of various neutral serum amino acids and on the level of brain serotonin in the rat.

Material and methods. All experiments were carried out at 09.00 h using Sprague Dawley male rats weighing 100 g, from 2 different breedings, all fed for at least 8 days with 2 standard laboratory chows. The 2 different laboratory chows were provided by 'Usine d'alimentation rationnelle (régime AO4), Villemoisson (France)' and 'Etablissements Piètremont (régime M.25), Sainte-Colombe, Provins (France)'. The animals were fasted for 16 h before the experimentation but had free access to drinking water. Salmon synthetic calcitonin (Sandoz), diluted in 0.1 ml of a gelatin suspension was administered at a level of 0.5–1 or 2 MRC units per 100 g weight of rat. In order to observe any nonspecific effect of the hormone, some rats were injected with a performic acid oxidation inactivated calcitonin[7], or with a M-sulfoxide calcitonin, and to determine whether the effect was due to reduced calcium concentration, the blood level of calcium in other animals were lowered with an EDTA injection (6×10^{-4} moles/kg)[8]. Control animals received the same amount of gelatin suspension. The animals were sacrificed by decapitation 45 min, 90 min or 4 h after the administration of the hormone. Blood was collected in test tubes without anti-

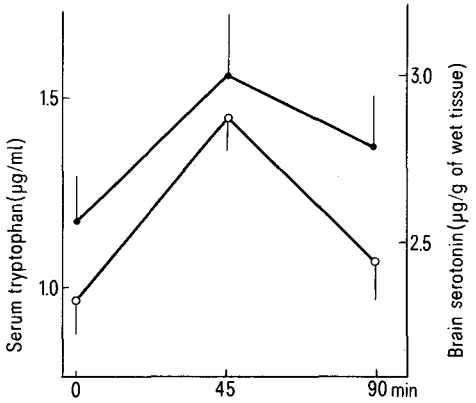

Figure 1. Variations of the serum free tryptophan (○), and brain serotonin (●) concentrations after a calcitonin injection (2 MRC U/100 g, b.wt).