Abstract


Hepatic metabolite profiles in mice with a suboptimal selenium status.

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BACKGROUND: Selenium is an essential trace element and mediates its functions via various selenoproteins such as glutathione peroxidases or thioredoxin reductases. A suboptimal selenium supply causes metabolic disturbances and is associated with an increased risk to develop different disorders, including cancer or cardiovascular diseases.

OBJECTIVE AND METHODS: This study aimed to assess the impact of a suboptimal selenium status on the hepatic metabolome of male mice analyzed by a targeted liquid chromatography/tandem mass spectrometry and a method based on non-targeted gas chromatography hyphenated with mass spectrometry.

RESULTS: Feeding animals a diet with about half of the recommended selenium content supplied as selenomethionine caused liver glutathione peroxidase and thioredoxin reductase activities to decline and lipid peroxidation to increase. Serum T3 thyroid hormone concentration also declined via a reduced hepatic deiodinase activity. Metabolite profiling revealed predominantly changes in cysteine and carbon-1 metabolism as well as in selected lipid subclasses. In particular the concentrations of palmitoylcarnitines and oleoylcarnitines (C18:1 and C16:1) and various phosphatidylcholine species containing saturated fatty acids were elevated. Increased taurine levels suggested an enhanced cysteine flux through the salvage pathway whereas increased homocysteine levels appeared to be a consequence of a massive down-regulation of cystathionine β lyase (cystathionine β synthase) and a reduced flux through the transsulfuration pathway.

CONCLUSIONS: The findings demonstrate that a suboptimal selenium status causes alterations in lipid and carbon-1 metabolism in mouse liver. These changes may contribute to the development of diseases associated with a suboptimal selenium status.

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