



EXCRETION OF THE MICROLEMENT MANGANESE

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<https://doi.org/10.5281/zenodo.17342299>

Relevance. Manganese (Mn) is an essential nutrient for cellular activity, acting as a cofactor for enzymes such as arginase and glutamine synthetase. These metalloproteins enable manganese to play a key role in various bodily functions, including development, digestion, reproduction, antioxidant protection, energy production, immune response, and regulation of nerve activity.

Although manganese deficiency is rare, excessive exposure can lead to manganese toxicity. The metal tends to accumulate in the liver, pancreas, bones, kidneys, and, especially, the brain, which is the primary target of toxicity. Symptoms of manganese toxicity include liver cirrhosis, polycythemia, hypermanganese dystonia, and Parkinson's disease-like symptoms. Due to its neurotoxicity, manganese has become a serious environmental problem. The molecular mechanisms of its toxicity include oxidative stress, mitochondrial dysfunction, protein misfolding, endoplasmic reticulum stress, autophagy dysregulation, and apoptosis.

Purpose of the study: This review examines the elimination of the trace element manganese (Mn) from the human body. Mn functions in the body as an important coenzyme and has pronounced antioxidant properties. It also contributes to energy production and the regulation of nervous system function.

Materials and methods: Analysis of scientific articles on Mn in international databases of scientific publications (Pubmed, Google Scholar).

Results: On average, 2.3 to 8.8 mg of Mn is absorbed daily. However, men require only 2.3 mg/day, while women require 1.8 mg/day. Excess Mn must be excreted. The turnover of ingested Mn is relatively rapid, with an average lag of 10 days. Most of the excess Mn is conjugated with bile by the liver and excreted in the feces. The liver plays a critical role in this process, as it is reported to be the main source of endogenous Mn losses in the intestine. Rats fed a Mn-containing diet absorbed approximately 8% of ingested Mn, and then 37% of the absorbed Mn was excreted through endogenous hepatobiliary Mn elimination. Therefore, individuals with liver problems are at higher risk of Mn intoxication. In addition to primary fecal hepatobiliary elimination, manganese excretion in urine, milk, and sweat has also been reported, but in very limited quantities. However, the ratio of excretion via different routes may change under certain circumstances. For example, when using a manganese hexameric dendrimer as an MRI contrast agent, renal manganese clearance significantly increased, equaling or exceeding the amount eliminated via the hepatobiliary route.

Conclusions: Manganese is an essential trace element that acts as a cofactor for various enzymes. Despite its importance, excess manganese can be dangerous. The body absorbs much more Mn than it requires, requiring an effective elimination mechanism. The primary route of excretion of excess manganese is through the liver, via bile, and then in the feces. This process occurs relatively quickly. Because the liver plays a critical role in manganese excretion, people with liver disease are at increased risk of manganese toxicity. Excess manganese accumulates in various organs, particularly the brain, making it a primary target. Despite existing knowledge, the mechanisms of manganese homeostasis are not fully understood. Further research is needed to better understand how



manganese is absorbed, distributed, and excreted from the body, as well as how its concentration within cells is regulated. This will help develop more effective approaches to the prevention and treatment of manganese toxicity.