JOURNAL OF CRITICAL REVIEWS

ISSN- 2394-5125 VOL 10, ISSUE 01, 2022

Manganese

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Abstract

Manganese has gained major attention from both researchers and the public or it could be continued to gain little attention. Manganese acts as an antioxidant function, as a manufacturer of bone structure and as a regulator of blood sugar. On the other hand, the relative insignificance of manganese could continue because of limited information. There is no idea, when moderate change is in manganese consumption from different sources originally impacts health in so many people. If we can get more awareness on these issues, manganese behaves as a very an exciting mineral in human nutrition.

Introduction:

Manganese assists the body in forming bones, blood clotting factors, connective tissue and sex hormones. It also plays a role in fat and carbohydrate metabolism, calcium absorption and blood sugar regulation. Manganese is also involved in the normal functioning of brain and nerve activity. Manganese oxide behaves as a catalyst, a rubber addictive. Manganese decolourise glass that is coloured green by iron impurities. Manganese sulfate behaves as fungicide. Manganese oxide is a powerful oxidizing agent and is helpful in quantitative analysis. It is helpful in making fertilizers and ceramics. Manganese is useful in making batteries, bleaching powder, China clay and paints. Manganese is a trace mineral which your body required in small amounts. It is responsible for the normal functioning of your brain, nervous system and various body stores up to about 20 mg of manganese in your liver, kidney, pancreas and bones. Manganese is also available from your diet.

Literature survey:

1. Hand book of minerals as nutritional supplements
2. Internet source

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Methodology.

Overview of metabolism

There are assorted observations and hypothetical aspects. For example, little information is available about manganese absorption into intestinal cells, but there are hints that regulation happens to work against excess consumption. Manganese absorption appears to consist of overlap with iron mechanisms. The absorption of manganese occurs from the intestine and afterwards much of it binds to albumin and rapidly gains entry into the liver. From there, its excretion happens into the bile, water into liver enzymes or may be transported to other tissues. Manganese excretion into bile seems to be controlled by body manganese content. Manganese transportation from the liver to other tissues is under the influence of transferrin (the protein) that transports iron, but some manganese also attaches with alpha-2 macroglobulin a protein that consists of tightly bound zinc. The metabolic importance of the latter is not known. It is not clear how manganese gains entry into extra hepatic tissue cells. There is an opinion that manganese rides in with transferrin in a process that overlaps iron absorption. Manganese does not contain specific storage protein like iron or zinc, but quite, a bit in a passive manner stores in bone.

Nutritional status assessment.

There has not been a lot of work in this area simply because of lack of demand for it. Serum or plasma total manganese is the most commonly used tool, but urinary manganese and functions of serum arginase or lymphocyte manganese SOD have also been utilised.

Bio availability from foods and supplements.

This area surely really requires more research examples for the foods with most manganese are whole grains, nuts and tea. The bioavailability from these foods is largely not known, even though a rats study opines that manganese absorption from tea is very good. Indirect proof opines that manganese absorption, at least in some circumstances, is almost reduced by iron supplements. An example for supplement forms are ascorbate (observed in one prominent joint Health product), Gluconate, sulphate and amino acid chelates. Along with food sources of manganese, little information is available related to the absorption. Properties of various manganese supplements. The Internet exhibit many sites claiming that calcium supplements will prevent absorption of manganese supplements. This claim exhibits support to certain extent, but the importance is not very near from clear cut. Especially in human experimental work, acute absorption of manganese is decreased by adding calcium to human milk, but not to other test meals. In a rat experimental research work with a perfusion in vivo, calcium stops manganese absorption in one section of the intestine but enhances it in another section. In an experimental research work in chickens, feeding more calcium levels does not influence manganese absorption. Thus, the practical aspect of the calcium magnesium interaction is not known. Some questions are not resolved, and these questions are A. how high does the calcium to magnesium ratio affect a main impairment regarding manganese absorption? B. Will various calcium complexes exhibit more, less or the same tendencies to prevent manganese absorption? C. How is the inhibition increased or restricted, especially by foods consumed at the same time as the

ISSN- 2394-5125 VOL 10, ISSUE 01, 2022

supplements? Some Internet sites opine that Manganese supplements should be consumed on an empty stomach. The rationale for this recommendation is not clear. As with most minerals, there are some dietary components known to enhance, reduce or do not show any effect on manganese absorption.

Current research on supplement use.

Relatively a very few supplementation studies are available on manganese compared to a number of other minerals and the major studies are given in table 1. **Table one.**

Human manganese supplementation studies.

Subjects	**Effect**
A. College aged males	Enhances lymphocyte Mn SOD activity
B. Adults	When given calcium carbonate enhances fecal fat, reduces fecal nutrients
C. Post menopausal women	When given copper/zinc, reduces bone turnover
D. Osteoarthritis adults	When given chondroitin sulfate/ glucosamine, decreases symptoms
E. Adults type 2 diabetes	No consistent results

Study number A of table one Indicates that manganese is not a big funding priority of nutrient nutrition agencies. Study number B of table one may provide the impression that supplementation with manganese gluconate plus calcium carbonate may assist body weight management. Whatever it may be, the percent enhancement in fecal fat loss is very small. Moreover, this percent is total fecal fat, not percent of total ingested fat. The latter exhibits a bigger impact on fat balance that is why the impact as absorbed calories is very less. Studies numbers C&D from table one show an interest,but Manganese supplementation was not tested by itself in combination with other products. Thus it is very difficult to estimate to what extent, if any, the Manganese enhances bone and joint health in these studies. Possibly,the non manganese portion of the products accounted for the complete effect. There is a rationale for manganese to

ISSN- 2394-5125 VOL 10, ISSUE 01, 2022

affect bone as well as joint health. Manganese function, with the help of glycoside transferase affects production of structural carbohydrates required for the connective tissue observed in bones and joints. This function is apparently essential for growth as well as maintenance. In addition, it is within the Preview of possibility that Mn SOD also influences bone health. Superoxide plays a role in enhancing bone as well as joint degeneration. These connections between manganese and bone or joint health are dependent primarily on data, particularly from severe manganese deficiency in animals. Since heavy deficiency is considered very rare in humans, 3 questions arise regarding manganese and bone or joint health. Study number 5 from Table one was likely initiated by 3 observations.

1. Severe manganese deficiency in rats leads to the occurrence of poor glucose tolerance.

2. A very few diabetic patients exhibit more urinary manganese values, which could mean that such subjects lose a lot of manganese.

3 There is an Anecdotal report of manganese supplementation assisting blood sugar control in a diabetic patient.

In spite of this background, study number E did not yield a consistent lowering of blood sugar in subjects with or without type one diabetes. This may not be extremely surprising upon closer inspection of the blood sugar abnormality in manganese deficient rats. This effect happens to be due to low concentrations of insulin mRNA. This condition is not treated, a typical cause of high blood sugar in type 2 diabetes or in most human high blood sugar states. Type one diabetes is manifested by impaired insulin secretion, but the cause is likely entirely independent of manganese function, generally 2 enzymes that require Manganese for function are components of gluconeogenesis. Research work is not conducted regarding interactions between dietary manganese and agents that can enhance Mn SOD Gene expression. Mn SOD gene expression is generally controlled by a variety of factors. In many conditions, enhancing MN SOD gene expression seems to be beneficial as evidence. By work in transgenic mice that over or under express MN SOD. This mice expresses resistance to a variety of induced disorders like **epilepsy.** Manganese deficient rats exhibit vulnerability, particularly to seizures and rats that are genetically prone to epilepsy exhibit low brain as well as blood manganese levels. Even if there is a strong relationship, it is not known if the very less manganese values lead to occurrence of the neurological problems. In addition, there are no established human trials regarding the utility of manganese supplementation in epilepsy.

Toxicity

Manganese toxicity happens because of occupational or environmental exposure. Examples for symptoms are neurological problems, some of which can exhibit a similar to Parkinson's disease. The manganese toxicity in itself should not originally affect Parkinson's disease because the manganese toxicity influences a different aspect of neurochemistry that does the primary defect of Parkinson's disease, in any case an association of Parkinson's like symptoms with typical oral manganese supplement doses is not well established. On the other side, in rats with Pre Parkinson's disease like some symptoms can be enhanced by high dose manganese toxicity.

ISSN- 2394-5125 VOL 10, ISSUE 01, 2022

Generally the adult upper level for manganese is 10 milligram per day. There are some diets that can provide that type of intake even without supplements. Besides 2 supplements, studies have provided manganese at well above the UL without observing any adverse effects.

Summary and conclusion

We require to know if there is any value in eating manganese at levels about those required to maintain basic action, especially for bone as well as joint health. Besides no research work was conducted on interactions between manganese consumption and factors that can enhance MN SOD gene expression. Compared to these research requirements, it is currently difficult to justify further research on manganese supplements as a means of decreasing blood sugar.

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