OPEN ACCESS Freely available online

ISSN: 2161-0509
Journal of Nutritional Disorders & Therapy

Opinion Article

Nutritional Risk in Metal Ions and Iron Deficiency for Supplementation of Human Body

Lucia Barros*

Department of Nutrition, Federal University of Rio Grande Do Norte, Natal, Brazil

DESCRIPTION

The functions of the essential metal ions (Mg, Fe, Cu, Zn, Mn, and Co), which are frequently lacking in our foods but are crucial for the health of the human body, as well as the various causes of these shortages in foods and in people, have been investigated. Iron deficiency anemia is the most common nutritional illness. In iron shortage, there is little transfer of iron from plasma to enterocytes. Improved mucosal transport of iron into the body is a result of increased body demand for iron. Due to poor absorption, limited availability, and/or negative side effects, inorganic salts, artificial synthetic monomer organic metal complexes of high stability, or organic polymer complexes of high molecular mass are unsuitable for supplementation to the human body.

The high ion selectivity and medium stability values of the mixed metal oligo/polygalacturonate complexes with polynuclear inner sphere structure allow for adequate absorption of the necessary metal ions. Metal oligo/polygalacturonate mixed complexes comprise all critical metal ions that are deficient in the body in sufficient levels and ratios to increase the metal ions' bioavailability and promote optimal vital function. Therefore, it is possible to assure metal ion homeostasis and the best interactions with vitamins and hormones by administering these complexes orally. Patients in clinical settings or those transported by ambulance frequently have prevalent or latent macro element Mg deficiency. The most frequent mesoelement deficiency is latent or evident; however, latent deficiencies in the microelements copper, zinc, manganese, and cobalt can also occur. Studies on essential metal oligo/polygalacturonate complexes as a supplement produced positive results without negative side effects.

We hypothesize that mucosal iron uptake is controlled by the number of iron binding sites that are either occupied or unoccupied by iron on mobilferrin because iron in enterocytes maintains balance with body reserves. Transferrin receptors on the posterolateral membranes of enterocytes allow for iron replenishment from body stores. In iron deficient animals, there is an increased transfer of blood iron into absorptive enterocytes, which prevents the mucosa from absorbing dietary iron. Contrarily, we recently discovered that natural pectin of plant origin mixed metal complexes of oligo/polygalacturonic acids with medium molecular weight are effective for oral supplementation.

Alterations in intestinal iron absorption go hand in hand with imbalances in iron homeostasis. Our knowledge of the trafficking of iron through transmembrane membranes has been enhanced by the discovery of the Divalent-Metal Transporter 1 (DMT1) and Ferroportin 1 (FP1). We looked at the expression of these transporters in patients with hereditary hemochromatosis (both HFE-associated and non-HFE-associated), secondary iron excess, and iron shortage in order to understand the regulatory characteristics of these transporters in the duodenum. In duodenal biopsy samples from patients, the expression of DMT1, FP1 Messenger RNA (mRNA), and proteins was examined using the TaqMan real-time polymerase chain reaction, Western blotting, and immunohistochemistry techniques.

Rats were used to study the effects of dietary iron deficiency on acute nickel, lead, or cadmium toxicity as shown by the induction of Metallothionein (MT), disposition of the metals, and changes in hematological parameters. While nickel or lead administration only caused hepatic MT, cadmium administration caused hepatic, renal, and intestine MT.

CONCLUSION

However, dietary iron deficit boosted the ability of nickel or lead to restore the normal synthesis of renal and intestinal MT that was diminished under the influence of low body iron status but had no effect on the cadmium-induced tissue MT. While tissue deposition of nickel was unaffected by iron shortage, lead and cadmium accumulation in the liver and kidney increased, and cadmium accumulation only occurred in the liver. The

Correspondence to: Lucia Barros, Department of Nutrition, Federal University of Rio Grande Do Norte, Natal, Brazil, Email: lucia@rio.br

Received: 11-Oct-2022, Manuscript No. JNDT-22-19293; **Editor assigned:** 14-Oct-2022, PreQC No. JNDT-22-19293 (PQ); **Reviewed:** 04-Nov-2022, QC No. JNDT-22-19293; **Revised:** 11-Nov-2022, Manuscript No. JNDT-22-19293 (R); **Published:** 18-Nov-2022, DOI: 10.35248/ 2161-0509.22.12.215.

Citation: Barros L (2022) Nutritional Risk in Metal Ions and Iron Deficiency for Supplementation of Human Body. J Nutr Disord Ther. 12:215.

concurrent increase in hepatic zinc, calcium, and iron levels in normal rats seems to be connected to the stimulation of hepatic MT by three metals. The hepatic zinc response to nickel or cadmium and the hepatic calcium response to lead were both elevated by dietary iron deficit.