



**Biotica
Research
Today**

Vol 2:12 **1305**
2020 **1307**

Importance of Iron in Human Nutrition

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Open Access

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Keywords

Excretion, Human nutrition, Iron inhibitors, Toxicity

Article History

Received in 26th December 2020

Received in revised form 27th December 2020

Accepted in final form 28th December 2020

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How to cite this article?

Kulla, 2020. Importance of Iron in Human Nutrition.
Biotica Research Today 2(12): 1305-1307.

Abstract

Globally 800 million women affected by anemia, in India 52% of non-pregnant women facing it with iron deficiency. There are multiple reasons behind it namely less consumption, malabsorption, inhibitors, blood loss, inherited conditions like sickle cell anemia, thalassemia, etc. People should know about the iron sources, needs in the body and how it works.

Introduction

Iron exists in several oxidation states varying from Fe^{6+} to Fe^{2+} depending on its chemical environment. The only states that are stable in the aqueous environment of the body and in food are the ferric (Fe^{3+}) and the ferrous (Fe^{2+}) forms. Over 65% (1.3–2.6 g) of body iron is found in hemoglobin, up to 10% (0.2–0.4 g) as myoglobin, 1% to 5% (up to 0.1–0.2 g) as part of enzymes and the remaining body iron (20% or 0.4–0.8 g) is found in the blood or in storage.

Sources of Iron

Dietary iron is found in one of two forms in foods, heme and non-heme. Heme iron represents iron that is contained within the porphyrin ring structure. Heme iron is derived mainly from hemoglobin and myoglobin in animal products, especially meat, fish, and poultry. Non-heme sources include rice bran, rice flakes, cauliflower, mustard leaves, chekkur, dried karonda, mango powder, etc.

Functions

The essentiality of iron is due in part to its presence in heme, which functions as a prosthetic group for some proteins. The atom of iron in the center of the heme molecule enables oxygen transport to tissues (hemoglobin); transitional storage of oxygen in tissues, particularly muscle myoglobin; and transport of electrons through the respiratory chain (cytochromes). Functions of the iron in the body include the followings.

1. Iron-Containing Enzymes (Metabolism)

Two enzymes involved in carbohydrate oxidation require iron as a cofactor. In glycolysis, the flavoenzyme glycerol phosphate dehydrogenase has a nonheme iron component. Phosphoenolpyruvate (PEP) carboxykinase, important in gluconeogenesis, also requires iron for its functioning. Heme iron-containing cytochromes include cytochrome b5 involved in lipid metabolism and the cytochrome P450 family involved in drug metabolism and steroid hormone synthesis.

2. Iron-Containing Enzymes (Synthesis)

Ribonucleotide reductase is iron-dependent enzyme involved in DNA synthesis and thus enables cell replication. It converts adenosine diphosphate (ADP)

into deoxy ADP. This enzyme contains non-heme iron as part of a bridge with oxygen ($\text{Fe}^{3+} - \text{O}_2 - \text{Fe}^{3+}$).

3. Iron in Oxidoreductases

Some oxidoreductases that are iron (molybdenum) dependent include; Aldehyde oxidase, which converts aldehydes (RCOH) to alcohols (RCOOH), sulfite oxidase, an iron- and sulfur-containing enzyme converts sulfite (SO_3) to sulfate (SO_4), and xanthine oxidase and dehydrogenase, iron-sulfur cluster enzymes metabolize hypoxanthine generated from DNA purine base catabolism to uric acid.

4. Iron in Peroxidases

Catalase, with four heme groups, converts hydrogen peroxide to water and molecular oxygen. Myeloperoxidase (also called chloroperoxidase), another heme-containing enzyme, is found in the plasma as well as in neutrophils. Thyroperoxidase (also called thyroid peroxidase), a heme-dependent enzyme, is necessary for organification of iodide (a process in which two iodides are added to tyrosine residues on thyroglobulin). Iron deficiency, in fact, is associated with decreased thyroperoxidase activity resulting in decreased T3 and T4 synthesis.

5. Monooxygenases

Some monooxygenases, which insert one of two oxygen atoms into a substrate, require a single iron atom to function. Examples (for amino acid metabolism) cover phenylalanine monooxygenase, tyrosine monooxygenase and tryptophan monooxygenase.

6. Dioxygenases

Many dioxygenases, which catalyze the insertion of two oxygen atoms into a substrate also need iron. These involve tryptophan dioxygenase, nitric oxide synthase and homogentisate dioxygenase (amino acid metabolism), trimethyllysine dioxygenase and 4-butyrobetaine dioxygenase (carnitine synthesis), lysine dioxygenase and proline dioxygenase (procollagen synthesis).

Excretion

Total daily iron losses for an adult male are 0.9 to 1.2 mg/day. Iron losses for women (postmenopausal) 0.7 to 0.9 mg/day because of women's smaller surface area.

Postmenopausal Women

Most (0.6 mg) iron losses occur through the gastrointestinal tract. Of this 0.45 mg is lost through minute (~1 ml) blood loss (which occurs even in healthy people) and another 0.15 mg losses in bile and desquamated mucosal cells. Skin losses of 0.2 to 0.3 mg of iron occur with desquamation of surface cells from the skin. Finally, 0.08 mg is lost in the urine via the kidneys.

Premenopausal Women

Total iron losses in premenopausal women are appraised to be 1.3 to 1.4 mg/day because of iron loss in menses. The average loss of blood during a menstrual cycle is 35 ml with an upper limit of 80 ml. Iron content of blood is 0.5 mg/ml of blood, which translates into a loss of nearly 17.5 mg of iron per period.

Inhibitors and Enhancers of Iron Absorption

Many dietary factors inhibit iron absorption, including,

- Polyphenols such as tannin derivatives of gallic acid, chlorogenic acids, monomeric flavinoids, and polyphenolic polymerization products (found in tea and coffee).
- Oxalic acid (found in spinach, chard, berries, chocolate, and tea, among other sources).
- Phytic acid, also referred to as phytate, inositol hexaphosphate, or polyphosphate (found in maize, whole grains, legumes).
- Phosvitin, a protein containing phosphorylated serine residues (found in egg yolks).
- Divalent cations such as calcium, zinc, and manganese at some level.

Some dietary factors that have been found to enhance non-heme iron absorption include, sugars, especially fructose and sorbitol, acids, such as ascorbic, citric, lactic, and tartaric, meat, poultry, and fish or their digestion products, and mucin.

Toxicity

The Tolerable Upper Intake Level for iron for adults is 45 mg. With acute toxicity, the excessive presence of an overload of iron atoms is thought to exceed the transport carrying capacity of transferrin. The unbound iron in turn behaves in a free radical manner to damage both the gastro-intestinal tract and other tissues. Chronic iron toxicity is generally associated with the genetic disorder hemochromatosis. Hemochromatosis is characterized by increased (at least two times normal) iron absorption. Mutations in one of several genes that result in diminished hepcidin synthesis. For example, in the C282Y mutation in the HFE protein, tyrosine is substituted for cysteine because of a single base change; this alteration inhibits HFE's ability to stimulate the synthesis of hepcidin. H63D mutation also reduces hepcidin synthesis, whereas a mutation (Q248H) in ferroprotein promotes hemochromatosis due to continued ferroprotein expression. The absorbed iron is progressively deposited throughout the body, including within joints and tissues, especially the liver, heart, and pancreas, causing extensive organ damage and ultimately organ failure. Treatment of hemochromatosis requires frequent phlebotomy (About 1 unit).

Conclusion

Iron is essential for many biological functions, understanding its role and consuming recommended levels of iron by keeping in view of inhibitors and enhancers of its absorption may help with reducing anemia in the worldwide.

References

Gropper, S.S., Smith, J.L., 2012. Advanced nutrition and human metabolism. *Cengage Learning*, pp. 481-500.