

Iron: Deficiency and toxicity

Iron – roles in the body:

Iron is an essential nutrient that is vital to the processes by which cells generate energy. Iron also can be damaging when it accumulates in the body. In fact, iron is a problem nutrient for millions of people. Some people simply don't eat enough iron-containing foods to support their health optimally while others have so much iron that it threatens their well-being. The principle that too little or too much of a nutrient is harmful seems particularly apropos for iron.

Iron has a knack of switching back and forth between two ionic states. In the reduced state, iron has lost two electrons and therefore has a net positive charge of two. Iron in the reduced state is known as ferrous iron. In the oxidized state, iron has lost a third electron, has a net positive charge of three and is known as ferric iron. Because iron can exist in different ionic states, iron can serve as a co-factor to enzymes involved in oxidation-reduction reactions. In every cell, iron works with several of the electron-transport chain proteins that perform the final steps of the energy yielding pathways. These proteins transfer hydrogens and electrons from energy-yielding nutrients to oxygen, forming water and, in the process, make ATP for the cells' use. If you recall from my previous article on this website, ATP is adenosine triphosphate, the cellular energy currency of the body. A direct precursor to this substance is nicotinamide adenine dinucleotide (NADH).

Most of the body's iron is found in two proteins: hemoglobin in the red blood cells and myoglobin in the muscle cells. In both, iron helps accept, carry and then release oxygen. Iron also is found in many enzymes that oxidize compound reactions so widespread in metabolism that they occur in all cells. Enzymes involved in the making of amino acids, hormones and neurotransmitters require iron.

Iron absorption and metabolism:

The body conserves iron zealously and has devised many special provisions for its handling. Two special proteins in the intestinal mucosal cells help the body absorb iron from food. One protein called mucosal ferritin receives iron from the GI tract and stores it in the mucosal cells. When the body needs iron, mucosal ferritin releases some iron to another protein, called mucosal transferrin. Mucosal transferrin transfers the iron to a carrier in the blood called blood transferrin, which transports iron into the rest of the body. Intestinal mucosal cells are replaced approximately every three days. When the cells are shed from the intestinal mucosa and excreted in the feces, they carry some iron out with them. The iron holding capacity of these cells provides a buffer against short-term changes in iron need or supply.

Let's quickly examine iron routes and storage centers in the body to further understand its absorption and metabolism. Iron in food reaches the intestinal cells during digestion where some is stored in intestinal cells in ferritin. Some iron is lost during the shedding of intestinal cells. If the body needs iron, it is packaged into transferrin, a transport protein, and carried in the blood. From here, some iron is delivered to the myoglobin of muscle cells and bone marrow which then incorporates iron into hemoglobin of red blood cells, of which excess is stored in ferritin and hemosiderin. Iron containing hemoglobin in red blood cells carries oxygen. The liver and spleen dismantles red blood cells and packages iron into

transferrin, and the cycle begins again. Some losses of iron occur via sweat, skin, bleeding, urine and the shedding intestinal cells.

Heme and non-heme iron:

How much iron is absorbed depends in part on its source. Iron occurs in two forms in foods, heme and non-heme. Heme iron is found only in foods derived from the flesh of animals, such as meats, poultry and fish. Non-heme iron is found in both plant and animal foods. Heme iron is so well absorbed that it contributes significant iron to the body. It is absorbed at a relatively constant rate of about 23 percent. The rates of absorption of non-heme iron are lower, ranging from 2 percent to 20 percent, and are strongly influenced by dietary factors and body iron stores. People with severe iron deficiencies absorb heme and non-heme iron more efficiently and are more sensitive to dietary enhancing factors than people with better iron status.

Absorption enhancing factors: MFP and vitamin C

Meat, fish and poultry contain not only the highly bioavailable heme iron, but also MFP factor that promotes the absorption of non-heme iron from other foods eaten with them. Vitamin C, which also enhances non-heme iron absorption from foods eaten in the same meal, is the most potent promoter of non-heme iron absorption. Vitamin C captures iron and keeps it in the ferrous form, ready for absorption. Other factors that enhance non-heme iron absorption include citric acid and lactic acid from foods, as well as HCl from the stomach.

Absorption inhibitors:

Some dietary factors bind with non-heme iron, inhibiting absorption. These include the phytates and fibers in whole grain cereals and nuts, the calcium and phosphorus in milk and supplements, the EDTA in food additives, and tannic acid. Tannic acid is present in tea, coffee, nuts, and some fruits and vegetables. Recent studies reveal that soy may inhibit iron absorption.

Iron deficiency:

If absorption cannot compensate for losses or low dietary intakes, and body stores are used up, then iron deficiency sets in. Because so much of the body's iron is in the blood, iron losses are greatest whenever blood is lost. Bleeding from any site incurs iron losses. Active bleeding ulcers, menstruation and injury result in iron losses.

Women are especially prone to iron deficiency during their reproductive years because of repeated blood losses during menstruation. Pregnancy places iron demands on women as well because iron is needed to support the added blood volume, the growth of the fetus and blood loss during childbirth. Infants and young children receive little iron from their high milk diets, yet extra iron is needed to support their rapid growth. The rapid growth of adolescence, especially for males, and the menstrual losses of teen females demand extra iron that a typical teen diet may not provide.

Assessment of iron deficiency:

Iron deficiency develops in stages. In the first stage of iron deficiency, iron stores diminish. Measures of serum ferritin reflect iron stores and are most valuable in assessing iron status.

The second stage of iron deficiency is characterized by a decrease in iron being

transported within the body. Serum iron falls, and the iron carrying protein transferrin increases (an adaptation that enhances iron absorption). Together, these two measures can determine the severity of iron deficiency; the more transferrin and the less iron in the blood, the more advanced the deficiency.

The third stage of iron deficiency occurs when the supply of transport iron diminishes to the point that it limits hemoglobin production. Now the hemoglobin precursor, erythrocyte protoporphyrin, begins to accumulate as hemoglobin and hematocrit values decline.

Iron deficiency and anemia:

Iron deficiency and anemia are not the same. People may be iron deficient without being anemic. The term iron deficiency refers to depleted body iron stores without regard to the degree of depletion or to the presence of anemia. The term anemia refers to the severe depletion of iron stores that results in a low hemoglobin concentration. The red blood cells in a person with iron deficiency anemia are pale and small. They can't carry enough oxygen from the lungs to the tissues, so energy metabolism in the cells falters. The result is fatigue, weakness, headaches, apathy, pallor and poor resistance to cold temperatures. Since hemoglobin is the bright red pigment of the blood, the skin of a fair person who is anemic may become noticeably pale. In a dark skinned person, the eye lining, normally pink, will be very pale.

Overview of iron deficiency symptoms:

Eyes: Blue sclera (sclera is a tough fibrous tissue that covers the white of the eye, blue sclera has an abnormal degree of blueness).

Immune system: Reduced resistance to infection.

Nervous/muscular systems: Reduced work productivity, reduced physical fitness, weakness, fatigue, impaired cognitive function, reduced learning ability, increased distractibility, impaired reactivity and coordination.

Skin: Itching, pale nail beds and palm creases, concave nails, hair loss, impaired wound healing.

General: Reduced resistance to cold, inability to regulate body temperature, pica (clay eating and ice eating).

Iron toxicity:

The body normally absorbs less iron if its stores are full, but some individuals are poorly defended against iron toxicity. Once considered rare, iron overload has emerged as an important disorder of iron metabolism.

Iron overload is known as hemochromatosis and usually is caused by a gene that enhances iron absorption. Other causes of iron overload include repeated blood transfusions, massive doses of dietary iron and rare metabolic disorders. Additionally, long-term overconsumption of iron may cause hemosiderosis, a condition characterized by large deposits of the iron storage protein hemosiderin in the liver and other tissues.

Iron overload is most often diagnosed when tissue damage occurs, especially in iron-storing organs such as the liver. Infections are likely to develop because bacteria thrive on iron-rich blood. Ironically, some of the signs of iron overload are analogous to those of iron deficiency: fatigue, headache, irritability and lowered

work performance. Therefore, taking supplements before measuring iron status is clearly unwise.

Other common symptoms of iron overload include enlarged liver, skin pigmentation, lethargy, joint diseases, loss of body hair, amenorrhea and impotence. Untreated hemochromatosis aggravates the risks of diabetes, liver cancer, heart disease and arthritis.

In the United States, an estimated 10 percent of the population is in positive iron balance, with 1 percent having iron overload. Iron overload is more common in men than women and is twice as prevalent in men as iron deficiency. Some researchers have expressed concern about the widespread iron fortification of foods. Such fortification does make it hard for people with hemochromatosis to follow a low-iron diet but equal dangers lie in indiscriminate use of iron supplements.

Blood letting is the best treatment for hemochromatosis along with following a low-iron diet designed by a certified nutritionist containing substances that interfere with iron absorption. Some examples of substances that block iron absorption in such a diet include black tea, phytic acid found in whole grains, taking calcium with meals containing iron, and reducing vitamin C intake.

Iron recommendations and intakes:

- Infants up until 6 months require 6 mg per day. From 6 months to 1 year, 10 mg is required.
- Children age 1 to 10 require 10 mg per day.
- Males age 11 to 18 require 12 mg per day.
- Males age 19 to 51+ require 10 mg per day.
- Females age 11 to 50 require 15 mg per day.
- Females older than age 51 require 10 mg per day.
- Pregnant women require 30 mg per day.
- Lactating women require 15 mg per day.

Iron in selected foods:

Meat, poultry and fish will contribute highly absorbable iron. Legumes, dark green leafy vegetables, green beans, tomato juice, parsley, artichoke, dried fruits and corn flour contain respectable amounts of non-heme iron.

Iron supplements:

At the Hoffman Center, iron supplements are employed in a clear deficiency state, especially when dietary sources are not prevalent in one's diet. Iron supplements can induce gastrointestinal distress. Common iron supplements are ferrous fumarate, ferrous sulfate and ferrous gluconate. Our choice is a product called slow iron, which is enteric coated and poses the least risk of gastrointestinal upset. Iron supplements should never be taken without the direct recommendation of a doctor.