

# IRON

70% of the iron in our body is found in the hemoglobin of our red blood cells. Hemoglobin is heme plus globulin (alpha globulins and beta globulins). If you can't make globulin, you can't make hemoglobin. All four of these components require copper. This mineral foundation is not taught to practitioners, but if iron can't get out of the gut and can't recycle properly in the spleen, and if the heme can't be made in the bone marrow, the result is merely different forms of anemia, but they all originate from a lack of bioavailable *copper*, not from a lack of *iron*.

Our bodies need a turnover of iron every 24 hours. One percent of our RBCs die each day and need to be replaced. Key to this entire process is that ceruloplasmin (via ferroxidase function) increases the rate of iron release by two-and-a-half times. This accelerated iron release is a central biological mechanism that is clearly not taught in any doctor school.

But the number one solution that doctors consistently recommend is ADD MORE IRON! And this is where the crisis of fatigue really begins. People keep getting drowned in iron when, in fact, what they simply need, is more bioavailable copper, but that mineral reality is well outside the training of most health practitioners.

We have a lot of iron dysfunction because there isn't enough bioactive copper. It is not a deficiency of iron. Yet, when was the last time your doctor measured your serum copper (ideal = 100), or computed your serum ceruloplasmin (ideal = 30), or your copper/ceruloplasmin ratio (should be 3.33)?

Under conditions of what the immune system perceives as chronic disease or chronic inflammation, our macrophages are called upon to increase the production and release of nitric oxide (NO). NO is an ancient molecule on this planet and scientists refer to it as the smallest hormone in our body. Among its many jobs is to activate the body's innate immune system. Under these conditions, NO naturally blocks the actions of the first and eighth (last) enzymes in the production of heme.

Doctors mistake *low hemoglobin* as a sign of *low iron*, and not *high inflammation*.

Think about the amount of heme needed per second. There is supposed to be ferritin (a storage protein for iron) inside the mitochondria to gobble up this iron, but that iron loading function also requires bioavailable copper, otherwise known as *ceruloplasmin*.

There is also supposed to be superoxide dismutase. Superoxide is regularly being formed at cytochrome I and cytochrome III of the mitochondrial electron transport chain (ETC); it is also reacting with iron. Superoxide dismutase (SOD-1 and SOD-3) require copper, as well. When we're missing the copper in these critical areas, hemoglobin production will and does fall off.

*Ferrochelatase* acts like a crane. And that crane operator is a copper ion that directs the acquisition of the iron needed and drops it into the center of the heme molecule. If you don't have that cupro-crane operator, the iron doesn't get dropped into heme, and then the body then can't produce hemoglobin naturally or efficiently. There is an enormous amount of heme, hemoglobin, and erythrocytes (RBCs) made daily:

- 2-3 million RBCs are made every second, thus >200 billion every 24 hours.
- There are 270 million hemoglobin in each RBC.
- There are 4 heme inside each hemoglobin.
- There are 1 billion heme in each RBC.

You can quickly see the enormous demand for not just iron, but also for bioavailable copper. It is high time the world awakened to this critical need for the catalytic and regulatory requirements that only bioavailable copper facilitates within our metabolism and within our mitochondria!

These facts have been known since the 1920s worldwide.

Has your doctor ever taught you any of this? Most doctors were never taught these details outlined here. Instead, when they see low iron markers in the blood, they are not trained to question iron in the tissue versus iron in the blood and automatically think “iron deficiency anemia.” The “deficiency” is actually a dysfunction. It is a lack of functional iron. There is a big difference between iron in the *blood*, versus iron in the *tissue*. Where there is dysfunctional iron, there is sure to be a *lack* of bioavailable copper and a corresponding *gain* of oxidative stress.

Conventional practitioners are blind to the difference between iron in the blood and iron in the tissue, as well as the role of ferroxidase as the seesaw regulating the flow of iron between these two settings. It is bioavailable copper that allows iron to get out of the tissue and get back into the bloodstream. Iron in the tissue (fibroblasts) can be as much as ten times higher than iron in the blood.

There is no simple, nor painless test for measuring iron in the tissue, other than a needle biopsy of the liver or a T2 MRI of the organs. The blood test for iron has no relevance to iron status in the tissues. They are completely different media and need to be understood and treated as such. Standard iron tests today typically include measuring iron levels in the blood (hemoglobin, serum iron and serum ferritin). If any of these tests show low levels of iron in the blood, doctors tell their patients they are anemic. Ferritin is supposed to be inside our cells in the tissues of the body, not found in our blood.

Iron, when not properly bound by transport or regulatory proteins, is pro-oxidant, and thus, very toxic to our health.

Iron behaving badly: inappropriate iron chelation as a major contributor to the cause of vascular and other progressive inflammatory and degenerative diseases.

There is 60x more iron than copper in the human body.

It's hard to find someone who knows the difference between:

- Anemia of Chronic Inflammation which is quite common and caused by a state of excess iron that is in hiding in tissues, organs etc.
- Anemia of Iron Deficiency, which is next to impossible on a planet that has 36% of its composition, made up of iron.

You need to fully understand what Anemia of Functional Iron is really all about. Iron is supposed to be *recycled*, not *stored*. The understanding of iron status is so important especially if it is based on one marker, ferritin, that is not telling the full story.

Ultimately we want to look at the following blood markers:

“Iron studies,” including:

- Serum Iron
- Total Iron-binding Capacity (TIBC) and/or Saturation
- Serum Transferrin
- Serum Ferritin

- **Serum Copper**
- **Serum Ceruloplasmin**
- **Red Blood Cell (RBC) Magnesium**
- **Plasma Zinc**
- **Hemoglobin**
- **Vitamin A (Retinol)**
- **Vitamin D (storage form: 25-OH)**

**Uric Acid**