

Important Iron

→ Blood contains 14.5g of Hb per 100mL.

45% of total iron → Hb.

5% myoglobin.

15% Ferritin (storage form)

Requirement

Adult - 20mg/day
Children (5-15 yrs) - 20-30mg/day
Pregnancy - 40mg/day

Sources

good sources - leafy vegetables, jaggery, liver, meat.

Moderate source - ~~Pas~~ Pulses, cereals.

Poor source - milk.

Qm Iron absorption

- Site of absorption - upper part of duodenum.
- Only ferrous form Fe^{2+} is absorbed.

Factors increasing iron absorption

- Gastric HCl.
- Ascorbic acid (vit C)
- Cysteine.
- -SH group of proteins.
- Reduces ferric to ferrous form.

Factors inhibiting iron absorption

- Phytic acid.
- Oxalic acid.
- Minerals - Ca, copper, lead, phosphates.

Mucosal Block Theory

- Iron metabolism is regulated at the level of absorption and (9)

not at level of excretion.

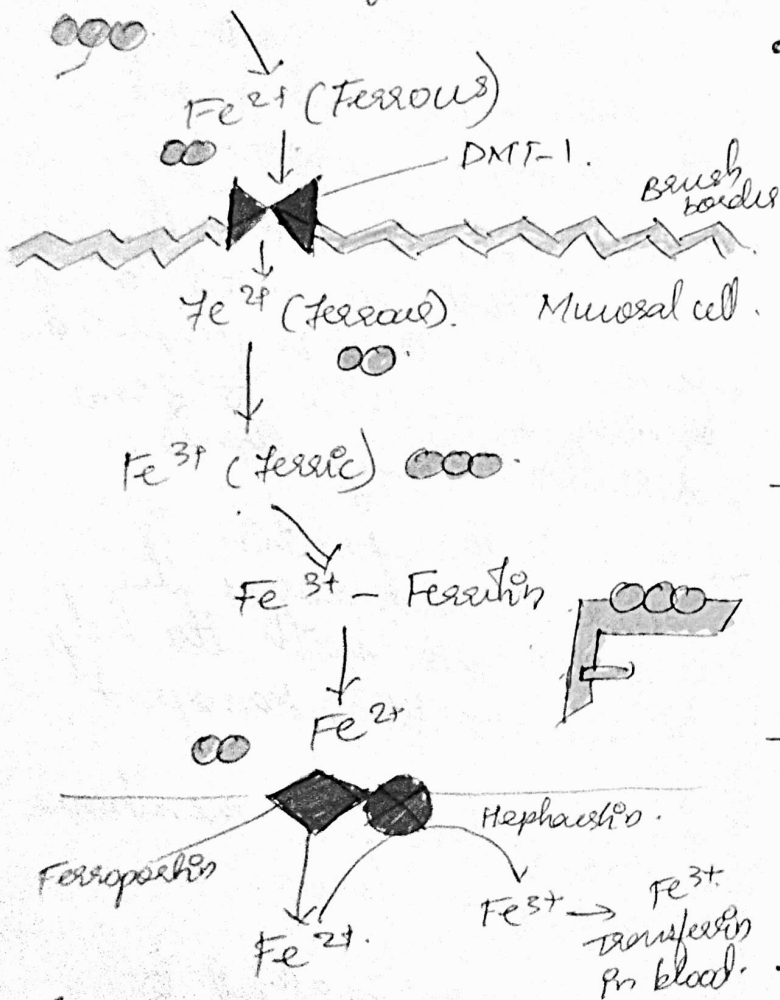
- Iron is a one way element.
- When Fe stores are depleted in the body, absorption is increased.
- When adequate quantity of Fe is stored, absorption is decreased.
- Ferric iron is reduced to ferrous iron by ferrireductase enzyme.
- In the intestinal lumen ferrous iron binds to mucosal cell protein - Divalent metal transporter 1 (DMT-1).
- Inside the mucosal cell ferrous ions converted to ferric iron and is complexed with apoferritin to form ferritin (storage form).
- Iron in the ferritin is released and crosses the mucosal cell with the help of ferroportin, transport protein.
- Iron crosses the membrane in ferrous form.
- Ferrous is re-oxidized to ferric state Hephaestin which has ferroxidase activity.
- Adipose cells will also release iron with the help of Hephaestin.

→ ferric ion then bind with transferrin and enters into circulation.

→ Once in circulation ferric form is maintained by ceruloplasmin, which has ferroxidase activity.

- Hephaestin & ceruloplasmin are copper containing proteins.

Fe^{3+} (Ferric) in food.



Iron transport in blood.

- Transport form of iron is transferrin
- Transferrin is a glycoprotein and is synthesized in liver.
- One molecule of transferrin can transport 2 ferric ions. (10)

Normal plasma level of Transferrin - 250 mg/dL

Iron uptake by cells.

- The Iron-transferrin complex is taken up by the body cells by the receptor mechanism.
- The transferrin receptor (TfR) binds 2 molecules of transferrin.
- The Iron-transferrin receptor is internalized.
- Iron is taken up by cells and receptor is externalized.

Regulation of Iron Absorption

- Mucosal regulation:

- Absorption of iron need DMT-1 and ferroportin.
- Hepridin, a peptide secreted by the liver down regulate the synthesis of DMT and ferroportin.
- when body iron reserves are adequate, hepridin production increased which reduces ferroportin and DMT-1.

- When there is anemia or hypoxia, hepridin production decreased which increases ferroportin and DMT-1.

- Stores Regulation:

- As body iron stores fall, the mucosa is signalled

to increase absorption.

• Erythropoietin regulation

- In response to anemia, the erythroid cells will signal the marrow to increase absorption.
- signal - Erythropoietin released from kidney.

Storage of Iron.

• Ferritin:

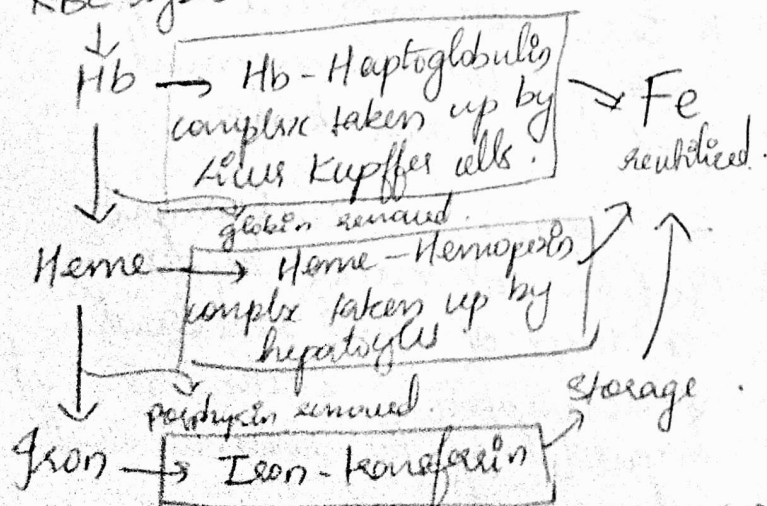
- storage form seen in intestinal mucosal cells, liver, spleen and bone marrow.
- Apoferritin can take up to 4000 iron atoms per molecule.
- Ferritin is an acute phase protein. (inflammatory)

• Hemoclerin

- Another storage form of iron
- Formed by partial degradation of ferritin.

Conservation of Iron in body

RBC lysis.



Iron Deficiency Anemia

Most common nutritional deficiency.

→ Mostly pregnant women suffer from anemia.

Maternal anemia contributes to increased Perinatal mortality.

→ leads to impairment of child's learning ability.

→ In adults, impaired work capacity.

Causes for Iron deficiency

1. Nutritional deficiency.
2. Hookworm infections.
3. Repeated pregnancies.
4. Chronic blood loss: hemorrhoids, peptic ulcers, hemorrhage.
5. Nephrosis.
6. Lack of absorption.
7. Lead poisoning.

characterized by Macrocytic Hypochromic Anemia.

It results when Hb $< 12 \text{ gm/dL}$

Clinical Manifestation

Hb $< 10 \text{ gm/dL}$.

→ cells lack O_2 .

→ Perangment in cellular respiration (Cytochrome)

- All metabolic outlethies become sluggish.
- Patient becomes uninterested in surroundings (APATHY)

Symptoms

- Irritability
- Lack of memory
- Inability to concentrate

TREATMENT → Iron + folic acid combination

2. Hemosiderosis

- Iron excess
- Golden brown granules (Hemosiderin pigments)
- seen in liver and spleen, pancreas, skin etc.

→ occurs in patients with repeated blood transfusion.
(Common cause)

- Iron deposition in liver cells causes cell damage - cirrhosis.

deposition in pancreas, leads to diabetes

Iron excess causes - hemochromatosis

Hemochromatosis, diabetes and cirrhosis together called bronze diabetes

phlebotomy - treatment for hemochromatosis

→ repeated blood collection

- Biochemical findings in Iron deficiency anemia,
- Transferrin level is increased
 - total iron binding capacity (TIBC) is increased.
 - serum iron is decreased.
 - ferritin content is decreased.

Treatment

- In pregnancy - Oral supplementation of 100mg iron + 500mg folic acid.

→ children : 20mg iron + 100mg folic acid

→ Iron tablets are usually given along with vitamin C

Fluoride (4 mark)

sources

- Drinking water, sea fish, cheese, tea and juice.

Fluorinated toothpaste contains 3000ppm of fluoride.

Functions (4 mark)

1. Trace amounts are required for proper development of teeth and bones.

2. Prevents development of dental caries by forming a layer of acid resistant fluorapatite with hydroxyapatite of the enamel and