Module 5: Iron

Introduction

Iron is an essential mineral that plays a role in many functions of the body. It is a part of the molecules that carry oxygen from the lungs to the cells, and the molecules that store oxygen in muscle tissue. Iron also is involved in the manufacture of amino acids, enzymes, hormones, and neurotransmitters.

Each day the body loses about 10% of the iron in red blood cells. Because iron is not produced in the body, the amount lost must be replaced from outside sources.

Iron deficiency develops when iron intake fails to meet the body's needs. Iron deficiency is the primary cause of **anemia** in children.

Iron-deficiency anemia (IDA) in infants and young children is associated with behavioral changes and delayed psychomotor development. Similac Advance supplies enough iron to meet the infant's needs and to prevent the consequences of iron-deficiency anemia.

This module will cover:

·Sources and metabolism of iron
·Iron deficiency and iron-deficiency anemia
·Iron requirements
·The effects of iron deficiency (anemia)
·Physicians' and mothers' attitudes about formulas
·Infant constipation and iron in formulas

Learning Objectives

When you finish this module, you should be able to correctly answer multiple-choice questions about:

·Iron storage
·The relationship between serum iron levels, iron stores, and dietary iron
·Causes, characteristics, and treatment of anemia
·The prevalence of iron-deficiency anemia
·Characteristics of cow's milk
·Recommendations of the American Academy of Pediatrics
·Findings from clinical studies

Sources and Metabolism of Iron

The body obtains iron from foods. Iron exists in two chemical forms:

·**heme iron**, in fish, meat, and eggs
·**nonheme iron**, in milk, grains, and vegetables

Heme iron is relatively well absorbed. Nonheme iron is poorly absorbed, partly because of interference from other substances in the diet. Vitamin C intake increases the absorption of nonheme iron, and calcium inhibits it.

Most iron absorption takes place in the small intestine. Normally only about 1 mg is absorbed each day, barely enough to balance the amount lost through normal cell turnover. Because the iron absorbed represents only about 2% to 10% of the iron ingested in food, the daily intake of iron needs to be about 10 mg. Infants and young children (4 to 36 months) have higher iron requirements because of their rapid growth. Requirements are also higher in menstruating women, and in pregnant women, who supply iron to the fetus.

Iron absorption increases when the body's need for iron increases. Needs rise when the body's storage supply is low, or when red blood cells are being formed (a process called erythropoiesis) more rapidly than usual. The reverse is also true: less iron is absorbed when smaller amounts are needed.

Absorbed iron is temporarily stored in the intestinal lining cells. Later it is circulated in the body by **transferrin**, and distributed in three main areas:

·in the blood:

— in circulating **hemoglobin** and as **serum iron**

·as tissue iron:

— in the brain, and in **myoglobin** in muscle

·as storage iron (mostly as **ferritin**, the rest as **hemosiderin**):

— in the liver, spleen, bone marrow

The brain contains a high concentration of iron. Surprisingly, iron concentrations in three central regions of the brain are higher than the those in any other solid organ — even the liver, the primary storage site for iron. Virtually all nerve impulses pass through these areas of high iron concentration in the brain.

The availability of transferrin to bind iron is measured as the **total iron-binding capacity (TIBC)**. In adults, normally about one third of the available transferrin is bound with

iron (as serum iron), and the rest is unbound. The total iron-binding capacity rises when the amount of iron needed increases.

The percentage of bound transferrin can be measured and compared with the maximum binding capacity of transferrin. This yields a value known as the **transferrin saturation**. The transferrin saturation level drops when the need for iron increases.

Changes in levels of serum iron (the iron in transport) also reflect changes in the amount of iron available to the body cells. Serum iron levels drop when less iron is available from the diet, or when iron stores are reduced.

Anemia

Anemia is not a single disease, but a reduction of circulating hemoglobin from any cause.

Causes of Anemia

Any of three mechanisms can lead to anemia:

·acute or chronic blood loss (due to ulcers, tumors, heavy menstrual flow, etc.)
·increased destruction of red blood cells
·decreased production of red blood cells

To take it one step further, decreased formation of red blood cells can be due to:

·lack of sufficient iron in the diet
·decreased absorption of iron
·diseases affecting bone marrow
·infection
·other nutrient deficiencies

Iron-deficiency anemia is the most common type of anemia and nutritional deficiency worldwide. As shown in Figure 1, it develops in a series of steps. In the U.S. the prevalence of IDA among infants and children is relatively low, but iron deficiency due to low iron intake remains the primary cause of anemia.[1]

The major cause of IDA in infants and children is low iron intake. In adults it is chronic blood loss, usually from gastrointestinal bleeding or menstrual bleeding, which eventually depletes the body's iron stores. (Remember that the term *iron-deficiency anemia* refers to anemia caused by depletion of the body's iron stores, and can be caused by bleeding or malabsorption instead of insufficient dietary iron intake.)

Infants and children are especially at risk of IDA. They undergo rapid growth at a time when their diet may consist mostly or completely of cow's milk, which is very low in

available iron. Cow's milk also may cause blood loss from the intestine (enteric blood loss), adding to the iron deficiency.

Full-term infants are born with enough iron to meet the body's needs for about 4 months. The infant's body destroys excess red blood cells, storing the iron for future use. During this adjustment to extra-uterine life, a transient "physiologic" anemia may develop, but it is not due to iron deficiency. From 6 months to 2 years, IDA is especially likely to develop if dietary iron intake is insufficient for expansion of blood volume.