

# Iron Nutrition and Exercise

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Iron deficiency is the most common single nutrient deficiency disease in the world and is a major concern for approximately 15 percent of the world's population. The commonly used definition for anemia, for whatever cause, is a low hemoglobin concentration (Hb). If iron deficiency is an underlying etiology, then by definition the individual must have depleted iron stores, a low plasma ferritin or decreased stainable iron in bone marrow, and an inadequate delivery of iron to tissues, as characterized by a low transferrin saturation, a high erythrocyte protoporphyrin concentration, and an elevated transferrin receptor concentration.<sup>1,2</sup>

Iron deficiency can be defined as that moment in time when body iron stores become depleted and a restricted supply of iron to various tissues becomes apparent.<sup>3</sup> The process of depletion of iron stores can occur rapidly or very slowly and is dependent on the balance between iron intake and iron requirements. Clearly, iron intake is dependent on food composition and quantity of iron therein, with a number of inhibitors and a smaller number of enhancers of iron absorption now known to exist. Iron absorption increases in individuals who have depleted iron stores; it is this internal regulator of absorption that may be more important than any particular constituents of the food supply.<sup>4</sup> Basal obligatory iron losses in humans are approximately 1 mg/day and must be replaced by an equivalent amount of iron derived from the diet. The typical Western diet provides an average of 6 mg of heme and nonheme iron per 1,000 kcals of energy intake.

## Consequences of Poor Iron Status

Many organs show morphological, physiological, and biochemical changes with iron deficiency in a manner related to the turnover of essential iron-containing proteins. Sometimes this occurs even before there is any significant drop in Hb concentration.<sup>5</sup> Iron deficiency is associated with altered metabolic processes; among them are mitochondrial electron transport, neurotransmitter synthesis, protein synthesis, organogenesis, and others.

It is also important to delineate whether exercise itself may alter iron status, and whether such alterations are detrimental to athletic performance or to the health of an athlete. Although a multitude of laboratories worldwide have contributed to a very broad-based accumulation of knowledge in these areas, an analysis of more than two decades of research illustrates four central points:

1. Reductions in heme and nonheme iron can detrimentally alter exercise performance.
2. Iron status is altered in certain populations of chronically exercising individuals.
3. Women may have an increased prevalence for exercise-related alterations in body iron.
4. It is reasonable to question whether these manifestations are specifically detrimental to the health or the athletic performance of the individual afflicted.

## Iron and Exercise Performance

The role of heme and nonheme iron in biologic function and work performance has been elucidated through human and animal experiments, and several classic reviews have been published.<sup>6,7</sup> It is not surprising that hemoglobin iron, when lacking, can profoundly alter physical work performance via a decrease in oxygen transport to exercising muscle. What is intriguing, however, is that although nonheme iron associated with enzyme systems comprises only 1 percent of total body iron, profound deficits of these components per se may have

detrimental effects on athletic performance. Studies illustrate that maximal oxygen consumption is determined primarily by the oxygen-carrying capacity of the blood and is thus correlated to the degree of anemia. Endurance performance at reduced exercise intensities, however, is more closely related to tissue iron levels, since a strong association is seen between the ability to maintain prolonged submaximal exercise and the activity of the oxidative enzyme pyruvate oxidase. There is disagreement in results of human studies regarding the concept of a Hb threshold phenomenon. Research by Edgerton et al.<sup>8</sup> suggests that the decrement in work performance in iron-deficient anemic subjects was a reflection of the level of anemia rather than other non-Hb related biochemical changes. Unlike the data of Perkkio et al.,<sup>9</sup> these studies suggest a more linear relationship between Hb and work performance and, thus, do not necessarily support the presence of a Hb threshold phenomenon.

Several studies conducted as early as two decades ago documented altered iron status in athletes, yet questioned whether such alterations were physiologically detrimental. Wijn et al.<sup>10</sup> measured Hb, packed cell volume, serum iron, and iron-binding capacity of selected athletes and compared these with the hematologic profile of officials during the 1968 Olympic games. These data illustrated an iron-deficient anemia in 2 percent of male and 2.5 percent of female athletes and a mild anemia without signs of iron depletion in 3 percent of the athletic population. Many other experiments have demonstrated a significant decrease in RBC number and a decrease in Hb, but often the runners were not affected. Subsequent investigations have supported these early studies and have demonstrated a reduction in Hb and HCT in certain athletic populations.

Several investigators have proposed mechanisms by which iron balance could be affected by intense physical exercise.<sup>11</sup> Explanations include increased gastrointestinal blood losses following running, and hematuria as a result of erythrocyte rupture within the foot during running.<sup>12</sup>

A growing body of evidence suggests that the prevalence of iron deficiency without anemia is increased in female athletes. Contributing to this observation is an increased iron loss through regular menstrual function as well as putative dietary factors. Given these observations, if exercise does further compromise iron status, then it would logically follow that the population of chronically | exercising female athletes may be at greater risk of developing iron deficiency. A summary of surveys demonstrates that 35 percent of female athletes have a serum ferritin concentration of less than 12 mg/L; 82 percent, less than 25 mg/L; and 60 percent, less than 30 mg/L, when compared with sedentary counterparts from the nonathletic female population. Although estimations of the precise prevalence rates differ, an increased incidence of reduced serum ferritin seems to be a repeatable observation between laboratories in this population. These results may be influenced by menstrual flow, and may additionally be affected by dietary iron intake. Taken together, these investigations illustrate a statistically significant difference between female athletes and control populations. What these investigations do not demonstrate, however, is a clinically subnormal or reduced serum ferritin concentration concurrent with a demonstrated functional consequence in the absence of overt anemia.

The Recommended Dietary Allowances (RDAs) are designed for the maintenance of good nutrition of practically all healthy people, and they define intakes of iron for infants, children, men and women, and also consider additional needs during pregnancy and lactation. From the data presented in this review, the following recommendations broadly apply to the three groups that appear to be at greatest risk of developing altered body iron given the data available. These recommendations do not assume that a deficiency will exist in these populations when consuming the RDA. However, a deficiency may be more likely, thus warranting closer dietary monitoring and regularly scheduled hematologic evaluations in these at-risk groups.

- Female athletes are advised to pay particular attention to maintaining an adequate consumption of iron in their diet.

- Distance runners should also pay attention to maintaining an adequate consumption of iron-rich foods.
- Vegetarian athletes should be particularly vigilant to include iron-rich foods in their diet.

For all three groups, monitoring of dietary intake and good nutritional counseling may preclude a negative iron balance and should be the first line of action in the prevention of iron deficiency. Indiscriminant pharmacologic intervention should be viewed as an undesirable means of achieving adequate iron intake, since in the least, it marginalizes the importance of promoting good nutritional habits in the athletic population. From the above priorities it is apparent that the female runner who consumes a vegetarian diet would likely be at greatest risk for a negative iron balance.

The use of supplements, however, must be a judicious choice based not upon the likelihood of anemia, but ideally upon hematologic evaluation. Using complex preparations may provide less iron than suspected, and warrant a careful re-examination with regard to efficacy. Clinically utilized oral iron preparations contain ferrous sulfate, hydrated or gluconated, or ferrous gluconate or ferrous fumarate. These preparations contain from 37 to 106 mg elemental iron. Supplementation is not without consequence, however; the use of high doses of supplemental iron is often associated with gastrointestinal distress and constipation, with a subsequent decline in compliance. In those genetically predisposed, hemochromatosis may develop following iron supplementation. Iron toxicity may even develop in those not genetically predisposed when ingesting dosages of 75 mg or more of supplemental iron.

In summary, it is clear that a decreased HCT and Hb will impair delivery of oxygen to tissues and lead to a reduced maximal oxygen uptake. Supplementation of individuals to normal HCT verifies the effects of hemoglobin iron on VO<sub>2</sub> max. However, the effects of iron supplementation upon the athletic performance of those with clinically low serum ferritin is less clear, although limited evidence seems to suggest improved endurance performance and a decreased reliance upon glucose as an oxidative substrate. Whether such adaptations are beneficial at a subclinically reduced serum ferritin has not been established.

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