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Understanding the Concept of Iron Deficiency Anemia in Athletes: A Narrative Review

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Abstract

New insights into the aetiology of anaemia in athletes have been discovered in recent years. From hemodilution and redistribution, which are thought to commit to so-called "sports anaemia," to iron deficiency triggered by higher requirements, dietary requirements, decreased uptake, enhanced losses, hemolysis, and sequester, to genetic factors of different types of anaemia (some related to sport), anaemia in athletes necessitates a careful and multisystem methodology. Dietary factors that hinder iron absorption and enhance iron bioavailability (e.g., phytate, polyphenols) should be considered. Celiac disease, which is more common in female athletes, may be the consequence of an iron deficiency anaemia that is unidentified. Sweating, hematuria, gastrointestinal bleeding, inflammation, and intravascular and extravascular hemolysis are all ways iron is lost during strength training. In training, evaluating the iron status, particularly in athletes at risk of iron deficiency, may work on improving iron balance and possibly effectiveness. Iron status is influenced by a healthy gut microbiome. To eliminate hemolysis, athletes at risk of iron deficiency should engage in non-weight-bearing, low-intensity sporting activities.

Keywords: Sports performance, Iron deficiency, Hepcidin expression, Iron metabolism

Major classifications: Sports Management, Food science, Sports nutrition

1. Introduction

Iron deficiency is the most prevalent single nutrient deficiency condition in the world, affecting 15% of the global population. In general terms, anemia refers to a situation in which the oxygen-carrying capacity is reduced to a low concentration of red blood cells or a poor condition of red blood cells. In addition, there's anemia caused by the lack of platelets or other components, such as a variety of vitamins (Like Vitamin B-12 or a low vitamin C intake). According to Rowland (2012), if an underlying etiology is iron deficiency, a person must have reduced iron reserves with low ferritin in plasma along with reduced stainable iron in the bone marrow. Billett, 1990 estimated that Anaemia occurs when the patient's hemoglobin level is reduced. An erythrocytosis happens when the body releases too many red blood cells, causing hemoglobin levels to rise above normal. The normal range (14 to 18 g/100ml for men and 12 to 16 g/100ml for women) is linked to normal hematocrit health. Regular aerobic exercise may lead to depletion of iron at the cellular level which results in lowering the oxygen transport capability with increased oxidative capacity. However, the pace at which iron stores are exhausted can be rapid or sluggish, depending on the combination of iron consumption and its requirements. Iron deficiency in many females is likely associated with both their dietary intake and the loss of iron in their blood because of heavy menstruation or pregnancy. (Hinton et al., 2011).

During exercise, there is an increase in plasma volume along with the stimulation of erythropoiesis. These adoptions are regulated by various mechanisms such as changes in osmotic pressure, tissue osmolality, and interstitial fluid pressure (Das et al., 1975). Exercise results in expansion of plasma volume at a faster rate and much greater extent as compared to cell volume expansion of RBCs, which in turn is responsible for the cause of pseudo-anemia in highly trained athletes having normal iron reserves in the body but possess poor hemoglobin levels in them (Sawka et al., 2000). According to Mujika et al. (1997), intensive training times are associated with lower hemoglobin and hematocrit values in highly qualified swimmers, while less intense training cycles accompanying major competitions (taper periods) are associated with higher levels of these hematological variables. Highly trained, athletes generally possess normal absolute hemoglobin levels, however, they often suffer from iron deficiency, which is usually latent and results in no decrease in hemoglobin levels.

The Transferrin Saturation Index (TSI) is a percentage-based method for determining an individual's personal iron status. It represents the ratio of serum iron content to total iron binding capacity (TIBC). The normal range of readings is 15% to 50%. This value can drop below 10% in extreme situations of iron insufficiency and anemia (Elsayed et al., 2016). According to the report of Nemeth and Ganz, 7% of male athletes and 17.5% of female athletes had a transferrin saturation index below 20%, although only a few of them were anemia and as a result, only a small number of athletes are at risk of having abnormal ferritin levels in their blood, and only a few amongst them with pathological ferritin values (Nemeth and Ganz, 2006). Low hemoglobin levels may or may not be correlated with this index of decreased body iron reserves. Among endurance athletes, such as runners, iron deficiency is highly frequent. (Niederkofler et al., 2005).

2. Importance of Iron in the body

Physical exercise causes a significant increase in the oxygen requirements body. Iron is required for the formation of hemoglobin, the protein that carries oxygen from the respiratory organs to the peripheral tissues. As a result, the processing of hemoglobin is impaired due to a lack of iron, which can further have a major impact on physical work capacity as it reduces the transfer of oxygen to the working muscles (Beard, 2001). Following iron deficiency, the concentration of myoglobin in skeletal muscle is greatly reduced (40-60%), which is responsible for limiting the rate of oxygen diffusion from erythrocytes to mitochondria and thereby compromising the muscle's oxidative capacity (Beard & Tobin, 2000). In addition, iron deficiency affects various non-heme iron-containing enzymes such as succinate dehydrogenase and NADH dehydrogenase.

Iron deficiency usually affects athletes practicing at high altitudes and at sea levels. Staying at a high altitude generates the conditions like hypoxia, which has direct effects on the bone marrow. It increases erythropoiesis by regulating the maturation and proliferation of erythroid progenitors, which are responsible for oxygen supply throughout the body. (Talbot et al., 2012). Chronically, poor oxygen levels produce elevated levels of erythropoietin. During moderate altitude training, non-iron-deficient athletes experience a substantial rise in erythropoietin whereas athletes with low ferritin levels show no change in total red blood cell volume after an initial increase in erythropoietin within the period of 336 to 672 hours or (2 to 4 weeks) at altitude. Such studies suggest that athletes' capacity to respond positively to altitude training in boosting their performance is dependent on iron sufficiency (Govus et al., 2015).

3. Assessment of Iron Status in Athletes

Iron deficiency includes three stages: in the first stage, iron reserves in the bone marrow, liver, and spleen are depleted (serum ferritin concentrations of 12g/L); followed by a reduction in erythropoiesis in the second stage as iron supply to erythroid marrow is reduced (TS of 16%); and in the third stage, the Hb intake is greatly reduced (Hb concentration of 12g/L), resulting in anemia (Higgins et al., 2011). The determination of iron level is not a measurement in itself, however, there are several iron state indices that reveal differences in acute phase reaction from day to day. As a result, to make a useful and accurate assessment, the calculation of iron status indices and several hematological parameters is needed. (Borenstein et al., 2009).

3.1. Evaluation of Haemoglobin

Haemoglobin is the most commonly used hematological scale, representing the effects of pathways that regulate red cell mass (RCM) and plasma volume (PV). It is used as an indication of anemia when an individual's Hb concentration falls below the normal level for particular age and sex group (Lynch, 2004). For females and males, the normal level ranges from 11.7 – 15.5 g/dL and 12.8 - 17.3 g/dL respectively (Gera et al., 2007). According to the WHO, the recommended Hb levels (in g/100 ml of venous blood) below which anemia progresses are 13 g/dL for male adults 12 g/dL, and 11 g/dL for non-pregnant and pregnant female adults respectively. For children aged from 6 to 18 months, it ranges from 11-12 g/dL (Lynch, 2004). Hemoglobin concentrations usually vary on regular basis, sometimes it is stable and fluctuates with a small variation of 2–4% (Strangman et al., 2003). In addition to hemoglobin hematocrit (Hct), mean corpuscular hemoglobin concentration (MCHC), and the size and volume of red blood cells (RBC) are other important markers for anemia (Malczewska, 2000). Before diagnosing, check the athlete's hemoglobin and hematocrit levels after the training since prolonged activity causes hemoconcentration, which is a higher concentration of cellular components in the blood as a consequence of plasma loss. (Parisotto et al., 2000). On the other hand, hemodilution causes a decrease in the concentration of these indices during the first few days of a normal cardiovascular training course. However, this drop is only temporary, and most athletes return to regular Hb levels after following a normal routine (Goddard et al., 2011).

3.2. Estimation of Iron Reserves in the Body

Serum ferritin concentration is one of the most often used indices in assessing of iron status. Ferritin concentration in the blood, as well as hemosiderin in cases of iron overload, serve as a measurement for the amount of iron in the body that is necessary for protein and heme synthesis (Crichton & Ward, 2003). Ferritin concentrations in the blood differ slightly from 10 to 300 g/L (Barr & Rideout, 2004), the values less than 12 g/L indicate that there are no detectable iron reserves in the bone marrow, or liver, or spleen. These numbers suggest that a first-stage iron deficiency has begun. Ferritin seems to be gender and age-based since infants and premenopausal women have lower concentrations than adults and men, respectively (Park et al., 2012).

Ferritin is an acute-phase reactant, and it may be enhanced in the bloodstream by liver disease, viruses, cancer, kidney failure, cardiovascular disease, excess alcohol, aging, and other autoimmune disorders (Durstine et al., 2001). Certain forms of physical exercise can cause inflammation-like reactions, which may result in an acute phase response and increased ferritin levels for several days. Normal ferritin levels can be deceiving in the case of exercise-induced inflammation, representing an acute phase response rather than the true output of the athletes' iron reserves. In endurance athletes, day-to-day variability in Ferritin is between 13% and 75% (Polancic et al., 2000). As a result, serum concentrations cannot be directly compared to iron reserves. Thus, it can be concluded that, though serum ferritin concentration is commonly known to be influenced by exercise and caution should be taken before diagnosing iron adequacy or inadequacy in athletes using ferritin as the only determining index (Toki et al., 2017).

3.3. Estimation of Iron Levels in Plasma or Serum

Total Iron binding capacity (TIBC) and Total Iron storage capacity (TISC), as well as the iron concentration, provide information on iron status in plasma or serum. The total iron content per unit of serum volume is described as iron concentration, with a normal range of 50 to 175 g/dL. Since, the range of iron content fluctuates by 15% to 26% from day to day and by 10% to 20% during the day, serum iron content alone cannot be used as a good measure of iron status (Kasvosve & Delanghe, 2002). The total number of binding sites for iron atoms on transferrin per unit volume of plasma or serum is known as total iron binding capacity (TIBC). The TIBC average range is between 250 and 425 g/dL. As the free binding sites on transferrin become available for iron in depleted iron reserves, TIBC levels increase (Parks et al., 2017).

The iron-binding protein transferrin helps in the transportation of iron to cells. Due to the fact that transferrin levels are unaffected by allergic reactions or other conditions, they can be used to detect iron deficiency even under these circumstances. (Ceylan et al., 2007). The percentage of serum iron to TIBC is defined as transferrin saturation, and values below 15% signify a second-stage iron deficiency. In the absence of anemia, this phase is defined by iron-deficient erythropoiesis and a limited iron supply (Voss et al., 2014). Its standard values for males and females range from 20-50 % and 15-50 % respectively. Since TIBC is relatively stable, any changes in plasma TISC are due to changes in iron concentration. As an effect, anything that changes the amount of iron in the body seems to affect overall iron storage capacity (Leers et al., 2010).

4. Phases of Anemia in Sports Anemia

Anemia is characterized by two factors: a decrease in iron reserves and a decrease in hemoglobin levels (Friedmann et al., 2001). Thus, a single reduction in ferritin content does not indicate anemia refers to a high risk of anemia near future if iron reserves are reduced further. When the body does not have enough iron, the body struggles with iron deficiency. Simple blood tests that monitor hemoglobin and iron levels in the blood can be used to diagnose it. Iron is found in hemoglobin, myoglobin, and some mitochondrial enzymes in healthy individuals with two-thirds of it in hemoglobin, myoglobin, and some mitochondrial enzymes (cytochromes), and, the remaining one-third of the body is made up of muscles, kidneys, and spleen (Hagler et al., 1981).

According to the effect on red cell synthesis, iron loss can be separated into three stages as discussed in Table 1 (Erdal et al., 2004):

Table 1: Stages of Iron Deficiency

Iron deficiency stage	Hematological markers	State of deficiency/overload
STAGE - 1 Depleted iron reserves	✓ Stainable iron in the bone marrow ✓ Total iron binding ability ✓ SF (Serum Ferritin)	✓ Not present ✓ A level of 400 ug/dL or higher ✓ A level of 12ug/L or lesser (older than 15 years)
STAGE – 2 Functional iron deficiency in its early stages	✓ TS (Transferrin Saturation) ✓ sTr (Serum Transferrin Receptor)	✓ A level of 16% or lesser (older than 10 years) ✓ A level of 8.5mg/L or higher

STAGE – 3 Iron Deficiency Anemia	✓ Hb (Hemoglobin) ✓ MCV (Mean Cell Volume)	✓ A level of 130 or lesser (males older than 15 y/o) ✓ A level of 120 or lesser (female older than 15y/o) ✓ A level of 50 fL or lesser

5. The cause of Sports anemia

Athletes' progress in the stress phase of their exercises is a significant factor. A rapid loss of plasma volume is one of the symptoms of untreated hypovolemia. This is due to the development of sweat and fluid depletion, as well as an increase in arterial blood pressure and muscle venule compaction (Cote, 1999). The loss of iron is primarily due to extremely strenuous training. Athletes, on average, have lower hemoglobin concentrations than overweight adults. Sports anemia can be caused by a diet lacking iron. Average adaptations, such as allowed iron supplements and training qualifications help in maintaining the sufficient iron content required during training and thus help in the management of sports anemia. (Rodenberg & Gustafson, 2007).

5.1. Haemolysis

Haemolysis is the destruction of red blood cells. Hemolytic anemia is a condition in which red blood cells burst suddenly and then regenerate due to intravascular hemolysis, which may occur in sports activities or exercises such as aerobic dance, weight lifting, rowing, and swimming (Dhaliwal et al., 2004). Exercising causes moderate hemolysis, which reduces plasma haptoglobin, a protein that binds to hemoglobin and prevents iron depletion in the urine. The iron released by haemolysed red cells is reprocessed and combined with hemoglobin in newly formed red cells, ensuring that anemia never occurs again (Santos-Silva et al., 2001). However, hemolysis happens as a function of the force that arises as a result of a foot strike. Haemolysis induces iron deficiency due to disruption to the red blood cell membrane, resulting in a free flow of both hemoglobin (Hb) and iron into the plasma (Telford et al., 2003).

There is a reduction in serum haptoglobin phases when free Hb is primed in plasma. Another example is swimmers, who were assessed for anemia, intravascular hemolysis, and iron deficiency in distance runners. Intravascular hemolysis emerged in all types of exercise, including running, however, intensive training swimmers have a higher rate of haptoglobin decline in long races (Van et al., 2001). Iron deficiency, anemia, and intravascular hemolysis of athletes in sports indicate that the athlete's hemolysis affects systems other than foot attacks. At the same time, some sports anemia researchers have found no signs of hemolysis. Haptoglobin levels in the blood are dropped in the acute and chronic types of hemolytic anemia. Excessive running injury can cause red blood cell haemolyse in the capillaries of the runner's foot. It's probable that the synthesis of muscle protein was facilitated by the haemolysed red blood cells (Dallalio et al., 2006).

5.2. Hematuria

The lack of blood or whole red blood cells in the urine is known as hematuria. Haematuria is a frequent side effect of exercise. Hematuria is a deformity that normally appears later in a person's athletic career. This can occur in sports such as rowing, running, and swimming, as well as wrestling, rugby, and other similar activities. Sports haematuria is usually self-restricted (Sinclair et al., 2005). If athletes exercise too often, the short-term loss in hemoglobin may increase, and athletes may experience anemia with or without complaints of marked urine. A drop in plasma haptoglobin indicates hemolysis. There is no hemoglobin in the urine if the hemolysis is poor. Haptoglobin binds free hemoglobin which prevents it from being excreted in the urine. The most common causes of haematuria are organized by the site of injury (in the kidney or bladder) and the spot in which it occurs by football or wrestling versus skipping or jumping (Wetz et al., 2017). Athletes with sports haematuria can continue to exercise, but they must be advised to retain proper hydration by drinking plenty of water (Bondi et al., 2005).

5.3. Plasma Volume Expansion

In humans and animals, plasma volume enlargement is most often associated with acute endurance workouts and endurance exercises. An increase in plasma volume is associated with a lower hematocrit in the absence of a red cell mass swap or an initial depletion in red cell mass, resulting in partial or complete anemia (Pak et al., 2006). Hypervolemia is produced by exercise/training alone which is increased by a combination of training and heat changing (which causes hypervolemia, but to a lesser extent than exercise) (kataoka et al., 2016). The greater the loss of plasma volume during exercise, the greater the hypervolemia. Hypervolemia can increase function by encouraging muscle perfusion to be one step ahead of the rest of the body, as well as increasing stroke volume and cardiac product. Plasma volume enlargement increases the thermoregulatory response to exercise by expanding skin blood flow. This leads to the central concept of optimum plasma length, and hematocrit (Truksa et al., 2007).

5.4. Menstruation

Iron deficiency is more common in female athletes with ferritin levels below 12 ug/L, as compared to just 2 to 13% of male athletes which might be due to iron loss during the menstrual cycle (0.5 to 0.6 mg/day), which correlates to a blood loss of 30ml per cycle. Heavy menstrual bleeding (HMB) is common and affects a significant portion of the female population. HMB may have a negative impact on one's physical, mental, and social well-being, as well as reduces the workspace (Swenne, 2004). When menstrual bleeding is heavy (more than 60ml) and/or the menstrual cycle lasts longer than 5 days, an iron deficiency is almost expected. Menstrual bleeding can be excessive in athletes who use an intrauterine system (Martin et al., 2018). They also found that iron consumption in the diet was poor, with only 43% of the daily dietary allowance being consumed (Bianchetti et al., 1990). Thus, though women's daily iron demands are higher than men's, their energy intakes are comparatively lower, and therefore their iron intakes are lower (Sherman & Thompson, 2004). The significant blood drop in HMB increases susceptibility to iron deficiency, which can progress to iron deficiency anemia (IDA) if left untreated. In a recent study of women with HMB, 63% of the population claimed that they were iron deficient at some points (Bemben et al., 2004).

5.5. Training

The change in the iron stores may also be due to the training. Iron separation is accelerated when plasma transferrin saturation is lower. The organization’s iron supplementation is unknown as a result of the high-intensity exercise and contributed to rising fatigue. Transferrin saturation is extremely high in the body, and it is failing to promote iron absorption. This concept can be used to explain the absence of a commonly observed relationship between iron intake and ferritin flow. It will also be able to show that rest is an effective treatment for sports anemia (Spodaryk, 2002).

5.6. Consequence of Poor Iron Intake

Multiple organs undergo morphological, physiological, and biochemical changes as a result of iron deficiency due to the turnover of essential iron-containing proteins (Markova et al., 2019). Alterations in metabolic processes such as mitochondrial electron transfer, neurotransmitter production, protein synthesis, organogenesis, and others have been linked to iron deficiency. Glossitis, angular stomatitis, koilonychia (spoon nails), blue sclera, oesophageal webbing (Plummer-Vinson syndrome), and anemia are the overt physical symptoms of chronic iron deficiency. Behavioral abnormalities such as pica, abnormal ingestion of soil (geophagia), and ice (pagophagia) are frequently found in individuals with iron deficiency, although a biological explanation for the same is still not available (Brunner & Wuillemin, 2010).

Reductions in hemoglobin concentration and tissue iron content can be detrimental to exercise performance; iron status is negatively altered in the majority of chronically exercising individuals due to a net negative iron balance, and women may be more likely to experience exercise-related changes in body iron (Lee et al., 2017). According to one study, it is believed that the oxygen-carrying ability of the blood determines maximum oxygen absorption (VO_2max), which is associated with the degree of anemia. Because of the direct link between the capacity to sustain sustained submaximal exercise and the activity of iron-dependent oxidative enzymes, endurance efficiency at lower exercise intensities is more strongly linked to tissue iron concentrations (Lippi et al., 2005). A reduction in hemoglobin content decreased mean corpuscular hemoglobin concentration, decreased size and volume of fresh red cells, reduced myoglobin, and reduced levels of both iron-sulfur and heme iron-containing cytochromes within cells are all well-known effects of iron deficiency that occur when iron reserves are depleted. In any condition other than rest, oxygen supply strongly limits the tissue oxidative activity in acute anemia. This increases tissue oxygen extraction, and partial oxygen pressure in mixed venous blood which is slightly lower in anemic people (Portal et al., 2003).

Due to poor intake of iron, the recommendation of an iron-rich diet and a heme iron-based diet, as shown below in Table 2, are examples of studies utilizing dietary iron management rather than pharmacological iron supplements in female athletes:

Table 2: The effects of several dietary iron treatment strategies on iron status in female

Sr. No.	Study	Duration of Study	Dosage	Result	References
01	21 female swimmers aged 12-17 years old with a BMI of 20 underwent a supplementation and dietary randomized control study.	06 months	Iron supplementation of 50 mg per day with dietary iron of 30 mg per day	There were few variations between the groups in terms of iron status and exercise Performance.	Krayenbueh et al. (2011)
02	Dietary treatment in 10 gymnast females between the ages of 19 and 20, with a BMI of 20.7.	01 month	Iron supplementation of 20.5 mg per day	After a diet modification, serum ferritin levels increased Substantially.	Miret et al. (2003)
03	On 60 female athletes aged 20-25 years old, supplementation and dietary randomized control study were conducted.	04 months	With a low iron diet, 60 mg of iron supplementation was recommended.	Iron supplements were shown to be less efficient than a meat-based diet in protecting Hb and Iron levels.	Cancelo et al. (2013)

6. Effects of Exercise on Iron Status

The iron content in the body can be changed by exercise. Athletes are more likely than others to have low serum ferritin levels. The main beneficiaries are long-distance runners (Bigelow, 2019). According to the impact of exercise, the outcome of the iron level in training will vary depending on the type of training. (Aerobic versus resistance exercise.

6.1. Effects of Aerobic Exercise: The three primary aspects of iron production are iron incorporation from the food in duodenal enterocytes, iron consumption in the erythroid precursor, and iron storage and reuse in hepatocytes and tissue macrophages (Donovan et al., 2006). According to Brown, 2007, heavily loaded or aerobic physical exercise can overstretch the method that maintains iron equilibrium. One of the understandings is hematuria caused by erythrocyte breakage within the foot during exercise, as well as an increase in gastrointestinal blood drop after running. Ferro kinetic metrics make it more difficult for athletes to increase their red cell profits (Toradec et al., 2009).

Aerobic activity increases iron status in individuals who are iron deficient. Aerobic activity significantly increases the hemoglobin concentration, stamina length, and VO_2 max of iron deficient people (treadmill). Nonetheless, exercise has little impact on the hemoglobin concentration of iron in contrast to a sedentary individual (Banfi et al., 2006). During heavy exercise, say swimming, the amount of iron accumulated by bone marrow erythroblasts and the remuneration of hemoglobin mixture is increased, while the iron content of the liver, spleen, kidney, and heart decreases (Beard & Tobin, 2000). Enhancement of iron is obtained by bone marrow cells and used for hemoglobin expansion which is believed to be the cause of iron deficiency in tissues. Exercise may result in the transfer of iron from the depot to bone marrow cells, resulting in increased hemoglobin union. In addition, in comparison to the stationary male tissue, there was less iron level observed in the tissue that has performed exercise (DeRuisseau et al., 2002).

6.2. Effects of Anaerobic Exercise

In the absence of iron supplements, moderate anaerobic exercise improves the iron position of young people with non-anemic iron deficiency. In young people with non-anemic iron deficiency, the intensity of serum ferritin, hemoglobin, the number of red blood cells, and the total iron definitive amount increased approximately after 12 weeks of anaerobic exercise such as dumbbell exercise (Ostojic et al., 2009). These findings reveal that lesser anaerobic exercise on daily basis improves the non-anemic iron lacing and prevents iron deficiency anemia. In a study done by Davis et al., 2003 on the content of iron in the blood, that did not rise, and the absorption of iron from the gastrointestinal tract was not increased by light exercise, as a result, dumbbell exercise did not improve iron absorption, it improved iron distribution in the body due to an improvement in the appearance of iron-binding proteins. The effect of anaerobic exercise on iron levels was investigated using optional aerobic exercise (climbing) equipment designed for individuals.

Anaerobic climbing has been linked to a greater increase in hemoglobin concentration and red cell mass, which improves oxygen-carrying capacity when compared to intense swimming. Individuals who performed a 3-week climbing routine had a greater tendency for iron-based hemoglobin than those who were inactive (Almar et al., 2002). The reduction in iron levels in the liver and spleen may be attributed to an increase in hemoglobin and exercise influences the iron distribution and restatement in the body (Peeling et al., 2009). Anaerobic exercise has little effect on iron levels in the liver, spleen, kidneys, or heart. The metabolism of iron is highly regulated. Males have roughly 4,000 mg of iron in their bodies, with 2,500 mg in erythrocytes, 1,000 mg in splenic and hepatic macrophages, and the rest in proteins like myoglobin, cytochromes, and other Ferro proteins. Exercise can- facilitate iron reprocessing in the body as well as affect the iron delivery to the linking tissues. (Peeling, 2010).

7. The Prevention and Treatment of Anemia in Athletes

The aim of treatment is to avoid the loss of iron stores and prevent anemia. Clinical (increased physical activity in the absence of asthenia) and laboratory criteria are used to assess therapy effectiveness (raised iron depot and hemoglobin phases). The diagnosis of therapy is complicated due to the hemodilution that occurs in athletes.

If mechanical damage to red blood cells causes hemolysis in a runner's feet, the following steps may help: correcting faulty running style, running on a softer surface like a cinder or grass track, and wearing Sorbo-rubber or running shoes (Karl et al., 2010). Iron supplementation is ineffective because sports anemia is not caused by a lack of iron. Simultaneously, increased red blood cell destruction caused by exercise can contribute to a reduction in iron stores (Mckie et al., 2001).

7.1. The use of Hepcidin Antagonist

Hepcidin modulates dietary iron absorption and iron outflow from iron storage cells, as well as triggering iron transfer in numerous organs, as a systemic iron modulator. Hepcidin deficiency is frequent in a variety of iron-related disorders, including anemia and hemochromatosis. Hemochromatosis is a condition in which the body has an excess of iron. Increased hepcidin expression is a crucial factor in the growth of sports anemia, according to recent developments in the molecular pathways of iron control. Hepcidin prevents enterocytes from absorbing iron and induces iron sequestration in hepatocytes and macrophages (Agarwal & Yee, 2019). As a result, modulating hepcidin expression with hepcidin antagonists is a novel alternative therapy for the treatment of sports anemia (Murray & Beard, 2007).

7.2. Diet

The intestinal mucosa absorbs approximately 11 to 16% of the iron consumed in the diet. However, the average daily dose of iron is less than 2 mg for men and about 2.5 mg for women. As a result, the average daily increase for men and women is about 11 to 24 mg (Raunikar & Saibo, 1992).

Furthermore, the best sources of iron are those which contain heme iron, such as red meat, liver, kidney, poultry, and seafood. This form of iron is easier to absorb than non-heme iron, which can be found in whole grains, nuts, seeds, legumes, and leafy greens. (About 23 percent of the increment). A daily 2g of animal protein per kg of body weight prevents a significant iron deficiency. Peas, nuts, bread, cereals, leafy vegetables, eggs, dried fruit, and wine are high in non-heme iron, which is the other form of food iron (Danielson et al., 2004). Although intestinal absorption is low (3 to 8%), it is usually aided by the presence of vitamin C, chicken meat, or fish. Iron-like non-heme, the absorption is reserved by calcium, phosphate, phytates, bran, polyphenols (which are present in tea), and antacids. Consumption of foods with high fiber, as well as excessive tea or non-digestive medicines such as magnesium, aluminum, and calcium salts and oxides, or inward bile production, may reduce iron accessibility (Nemeth et al., 2004).

Animal proteins, which aid absorption, and calcium, which prevents absorption, tend to be the only factors affecting heme iron absorption. The absorption of supplemental iron depends on the type of preparation used. The amount of iron that is bioavailable from multi-mineral preparations, especially when calcium salts are used, is less than that absorbed during the administration of iron alone. Furthermore, a rising adolescent has normal needs that are not met by this increase. Athletes in some disciplines, such as gymnastics, marathon running, or ballet dancing, are more likely to decrease their food intake in order to maintain their normal body weight, raising the risk of iron deficiency (Steensma et al., 2011).

7.3. Altitude

At higher altitudes, there is a lower partial pressure of oxygen, which affects the tissue hypoxia and the range of physiologic responses to hypoxia involved in erythropoiesis. Moving higher and less often exercise by athletes leads to better sea phase operation. In reality, better-trained professional runners living at elevations between 2500 and 3000 meters above sea level may experience a reduction in training intensity, muscle damage, and an increased risk of acute mountain sickness. Headache, nausea, lethargy, dizziness, and sleep disturbances are common symptoms of this sickness, which appear 6 to 48 hours after the altitude exposure begins. (Rzik et al., 2001).

According to Wehrlin, red blood cell mass capacity increased by 10% and maximal oxygen absorption increased by 5% after four weeks of living at an altitude of 2500 meters above sea level. (Wehrlin et al., 2006). Athletes can better regulate their training intensity and enhance their running activity at high latitudes due to the higher oxygen requirement, since the maximum oxygen concentrations are found at high latitudes, by exercising less. In reality, hypoxia at higher altitudes regulates training intensity, which can lead to elaboration in elite athletes. At a lower altitude, erythropoietin production starts to decline.

7.4. Physical performance and the use of Iron supplements

Iron deficiency anemia, which is described by a reduction in blood Hb content among other measures, clearly impairs physical effect by decreasing oxygen transport to exercise muscles (Jankowska et al., 2011). Therefore, the need for iron supplementation for better physical function in situations with low iron levels is still challenged.

Iron supplementation is shown to have some impact on athletic performance in the majority of studies. Besides that, low serum iron levels immediately after the competition have little effect on results (Dellavalle, 2012). Clenin claimed that only athletes with iron deficient anemia or untrained individuals with low serum ferritin were examined in randomized trials where iron supplementation was shown to want a beneficial effect. Iron has been found to have a little quantitative effect on performance when given to iron-depleted athletes or athletes with low serum ferritin (Clenin et al., 2015).

According to Malczewska, iron supplementation enhanced VO2max in 40 male professional athletes and 40 female iron deficient females who were not anemic. In the iron-treated groups, males had normal serum ferritin and hemoglobin concentrations, whereas red blood cell volume and lactate threshold were constant. The lactate threshold is the point when the intensity of activity causes lactate to build up faster than it can be removed from the blood. Despite normal hemoglobin levels, the female group had low serum ferritin levels. (Malczewska et al., 2000). Another study, according to Hinton and Sinclair, assessed the effects of iron supplementation in 18 female iron-depleted runners who did not have iron deficiency; although serum iron markers improved, there was no impact of iron on VO2 max. The use of oral iron supplementation in female athletes who have been treated for iron shortage led to the conclusion that iron deficiency in athletes who have not been treated for iron deficiency is an unclear basis for therapeutic iron supplementation (Hinton & Sinclair 2007).

8. Conclusion

The presence of elevated or low-normal red blood cell, hemoglobin, or hematocrit levels confirms the existence of sports anemia hemoglobin (normal levels are 14-17.5 g/dl for men and 12-16 g/dl for women), as well as low hematocrit and ferritin levels. Sports anemia is linked to a rise in red blood cell degradation and a decrease in hemoglobin concentration and occurs most often at the start of a strenuous conditioning program. Athletes can develop sports anemia due to a variety of factors, or a combination of factors. Inadequate protein intake, especially during the early stages of training, may also cause "sports anemia." Anemia is caused when the need for more muscle tissue competes with the need for more hemoglobin. Its hemolysis or blood vessel rupture is caused by repeated compression of red blood cells in the capillaries on the side of the leg as the foot strikes the ground. It was once very common among soldiers on long marches, aside from athletes and marathon runners. Aside from the well-known mechanisms of iron loss including hemolysis, hematuria, sweating, and gastrointestinal bleeding, exercise-induced up-regulation of hepcidin expression may be the primary cause of iron deficiency in athletes and overall output is harmed as a result of the decline in blood gas transport and muscle enzyme production. This is why people, particularly athletes, should get a full blood count and a blood test to avoid or quickly correct low hemoglobin iron levels. It can be avoided by eating a balanced diet that contains all of the essential nutrients, vitamins, and minerals. People who exercise should consume a high-protein, high-iron diet. Red meats, poultry (the dark parts of chicken have higher iron levels than the white parts), and fish are all healthy sources of protein and iron. Tea and coffee consumption should be monitored closely because they can reduce iron absorption. Also, since wine and vinegar can inhibit iron absorption, it's best to avoid mixing them with your meals. Some drugs, such as tetracycline and antacids, reduce iron absorption. To boost your iron levels, combine vitamin C-rich foods or drinks with an iron-rich meal or iron supplements, such as orange juice. The heme iron (found in meat) absorbs better than non-heme iron (found in vegetables, fruit, beans, and whole grains). The diagnosis of anemia is dependent on getting a complete medical history and ruling out other forms of anemia. Treatment is usually not recommended for sports anemia because it is self-limiting, but certain training changes can be helpful.

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