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REVIEW

Iron Deficiency in Athletes

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Abstract
Iron, although present in trace amounts, is a crucial micronutrient in the human body. It plays a significant role in essential bodily functions such as oxygen transport and energy metabolism. For athletes, particularly those engaged in endurance activities with high oxygen demands, iron is an indispensable nutrient. Despite its importance, a notable number of athletes experience iron deficiency. Research has suggested a connection between exercise performance and iron regulation, leading to increased focus on how these two factors interact. The aim of this study was to investigate how various types of exercise affect the body's iron reserves, the specific iron needs associated with different forms of physical activity, and the prevalence of iron deficiency linked to chronic inflammation.

Introduction

Although iron is a trace element found only in small quantities in the body, it plays a vital role in important functions such as oxygen transport and energy metabolism [1]. Iron deficiency symptoms include lethargy, fatigue and feelings of low mood [2–4]. In more severe cases (iron deficiency anemia; IDA), the ability to work is also impaired [5]. These symptoms may affect an athlete's ability to train properly and perform at their best in competition [6]. It is therefore important to monitor athletes' iron status regularly and to take appropriate measures where necessary to correct any deficiency. As the processes of iron metabolism are clearly essential for supporting athletic performance, the supply, utilisation and storage of iron are of great importance to athletes. However, in order to assess an athlete's iron status and determine appropriate measures, it is necessary to understand the mechanisms that influence iron absorption. This is because recent research has established a clear link between iron regulation and exercise [7].

Recent research has drawn attention to hepcidin, a peptide hormone produced primarily in the liver that regulates the body's utilisation of iron. Various factors, including elevated serum iron levels, stimulation by interleukin-6 (IL-6), impaired bone marrow function and endoplasmic reticulum stress, have been identified as catalysts for increased hepcidin production [8]. In addition, it has been demonstrated that IL-6—an inflammatory cytokine released during physical activity—inhibits iron metabolism by promoting hepcidin synthesis in hepatocytes [9]. The hepcidin plays a central role in the regulation of iron throughout the body [10, 11]. This peptide hormone is produced by liver cells [12]. As shown in Fig. 1, hepcidin is the main pathway regulating iron metabolism [13]. Hepcidin, synthesized in the liver, is released into the bloodstream for distribution throughout the body. It targets cells expressing ferroportin (FPN), such as macrophages located in the small intestine (specifically the duodenum), liver, and spleen. By binding to FPN, hepcidin inhibits iron export from these cells either through internalization of FPN or by promoting its degradation.

In the duodenum, dietary non-heme iron (Fe^{3+}) is converted to divalent iron (Fe^{2+}) by duodenal cytochrome b (Dcyt b) located on the luminal membrane of enterocytes. The Fe2+that is exported via FPN undergoes oxidation back to Fe3+facilitated by membrane-bound hephaestin (HEPH) or soluble ceruloplasmin (CP). This oxidized iron quickly binds to transferrin (Tf) in circulation, forming an Fe-Tf complex that transports it throughout the body. Macrophages in the spleen are crucial for phagocytosing senescent red blood cells and extracting iron from hemoglobin. These macrophages also express FPN, allowing them to release recovered iron into the bloodstream. Hepcidin plays a regulatory role over this process as well. In states of inflammation, there is an increase in hepcidin production,

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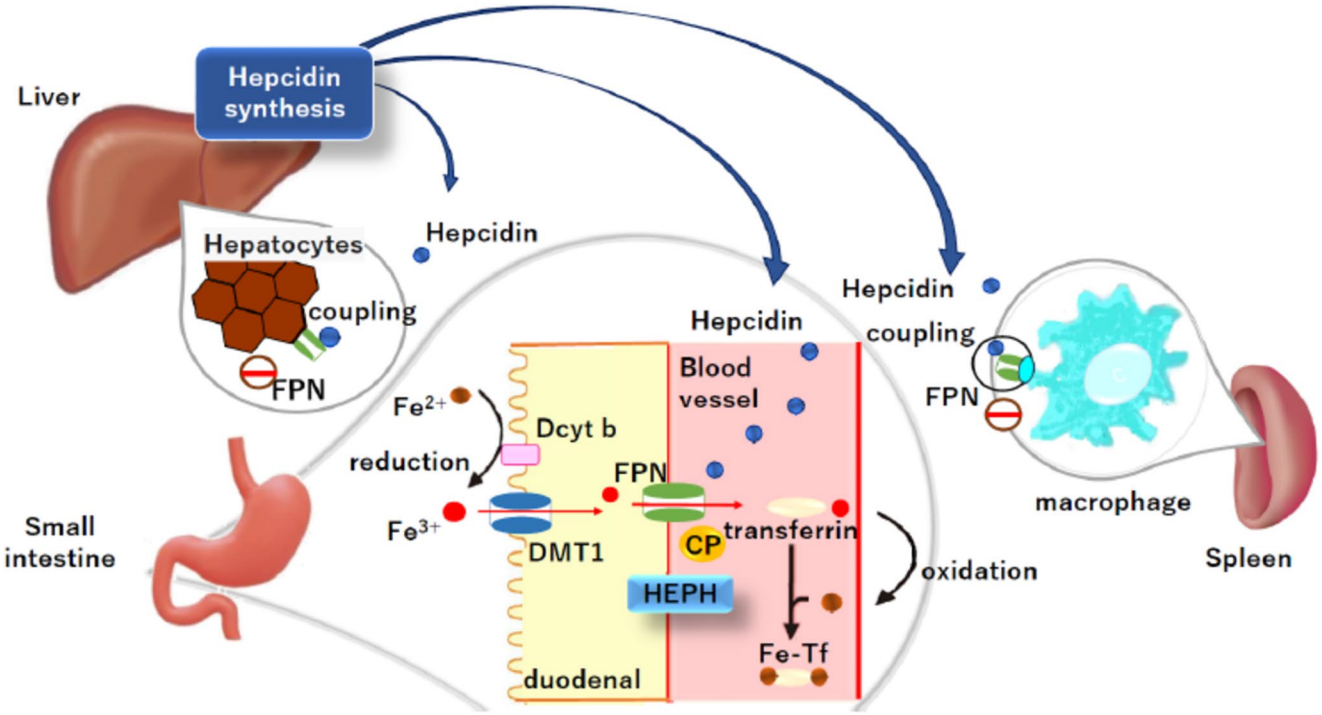

Fig. 1 Regulation of Iron Metabolism by Hepcidin. Hepcidin, synthesized in the liver, is released into the bloodstream for distribution throughout the body. It targets cells expressing ferroportin (FPN), such as macrophages located in the small intestine (specifically the duodenum), liver, and spleen. By binding to FPN, hepcidin inhibits iron export from these cells either through internalization of FPN or by promoting its degradation. In the duodenum, dietary non-heme iron (Fe^{3+}) is converted to divalent iron (Fe^{2+}) by duodenal cytochrome b (Dcyt b) located on the luminal membrane of enterocytes. The Fe2 + that is exported via FPN undergoes oxidation back to Fe3 + facilitated by membrane-bound hephaestin (HEPH) or soluble ceruloplasmin (CP). This oxidized iron quickly binds to transferrin (Tf) in circulation, forming an Fe-Tf complex that transports it throughout the body. Macrophages in the spleen are crucial for phagocytosing senescent red blood cells and extracting iron from hemoglobin. These macrophages also express FPN, allowing them to release recovered iron into the bloodstream. Hepcidin plays a regulatory role over this process as well. In states of inflammation, there is an increase in hepcidin production, which inhibits systemic mobilization of iron via FPN. This leads to decreased serum iron levels, with iron being sequestered within reticuloendothelial macrophages and limiting its availability for hematopoiesis, contributing to anemia of chronic disease (ACD) [6]

which inhibits systemic mobilization of iron via FPN. This leads to decreased serum iron levels, with iron being sequestered within reticuloendothelial macrophages and limiting its availability for hematopoiesis, contributing to anemia of chronic disease (ACD). Therefore, factors related to exercise (type, duration, and intensity) and the state of HAMP may cause athletes to develop a negative iron balance. All of these factors directly affect iron absorption and hematological parameters. The onset of HAMP reduces metal absorption, increases the cytoplasmic concentration of Fe-saturated Ft, and decreases transport to the bloodstream. As a result, iron accumulates in intestinal epithelial cells and is excreted within epithelial cells that have sloughed off from the intestine. On the other hand, unlike iron excess, iron deficiency, hypoxia, and states of increased erythropoiesis reduce HAMP expression; therefore, these conditions are directly related to increased iron absorption and bioavailability [14]. Several studies [15–18] have shown that the concomitant administration of vitamin C (ascorbic acid), vitamin A, vitamin E, vitamin B9 (folic acid), vitamin B12 (cobalamin), and vitamin D3 (cholecalciferol) increases the bioavailability and absorption of iron in the intestine. These absorption enhancers can be provided through the diet or directly via supplement formulations. These compounds may lead to Fernández the development of iron-related multi-component formulations that promote iron absorption in the gastrointestinal tract [19].

This paper presents a narrative review focusing on the relationship between exercise and iron regulation in athletes. It also addresses resistance training—an area that has received relatively little attention to date—highlighting relevant issues and offering recommendations for future research.

Iron Deficiency and Athletes

Despite its biological importance, iron deficiency (ID) is a widely reported problem amongst athletes, with recorded prevalence rates of approximately 15–35% in female athletes and approximately 3–11% in male athletes [20, 21].

However, small-scale cohort studies have reported high rates of decline in iron stores across various sporting environments, with prevalence rates reported to be over 50% in female athletes and up to 30% in male athletes [22, 23].

It is worth noting that female athletes tend to have a higher incidence of iron deficiency (ID) [24]. This may be due to a reduction in dietary intake and an increase in iron requirements associated with menstruation [25]. It has been pointed out that a reduction in energy intake, a vegetarian diet and endurance exercise may also affect iron stores in male and female athletes [26]. If iron levels are compromised, this can lead to lethargy, fatigue and low mood [2, 3]. In more severe cases (iron deficiency anemia; IDA), this leads to a decline in athletic performance [5]. Such symptoms may affect an athlete's ability to improve their competitive performance, even when they have trained appropriately [6]. Therefore, while it is important to regularly monitor athletes' iron status and take appropriate measures to correct any deficiencies, it is likely inappropriate to use the same cutoff value to assess iron deficiency, given that iron metabolism and ferritin distribution differ between men and women. Consequently, an assessment that takes gender differences into account is necessary, and women require management using a stricter (higher) cutoff value than men [27].

Exercise and Iron

Aerobic Exercise

There is considerable evidence to suggest that aerobic training may reduce hematocrit levels, hemoglobin concentration in the blood and serum iron concentration, and may compromise the integrity of red blood cell membranes [28–32]. Endurance athletes, particularly elite long-distance runners, typically engage in prolonged exercise sessions several times a day. In athletes with iron-deficiency anemia, endurance performance can decline by up to 30% [33]. Causes of anemia in athletes include hemolysis (the destruction of red blood cells) due to the impact on the soles of the feet from running [34] [Foot-Strike Hemolysis], iron loss associated with sweating during training [35], and sweating itself [19]. Female athletes lose iron approximately 20% faster than non-athletes, and faster than adult men [36]. In particular, some female athletes may exhibit subclinical iron deficiency in the bone marrow despite having normal hemoglobin levels and plasma iron concentrations [37].

On the other hand, animal studies have reported that aerobic exercise can mitigate the decline in iron-related biomarkers observed during iron deficiency [38]. In athletes, causes of anemia include the destruction of blood cells (hemolysis) due to the impact on the soles of the feet caused by running [39], iron loss associated with sweating during training [40], and sweating itself [41]. Female athletes lose iron approximately 20 per cent faster than non-athletes, and at a faster rate than adult men [42]. In addition, some female athletes may show signs of latent iron deficiency in the bone marrow, even though their hemoglobin levels and plasma iron concentrations are within the normal range [42]. However, animal studies have reported that aerobic exercise can mitigate the decline in iron-related biomarkers observed in cases of iron deficiency [39, 43, 44]. When compared with iron-deficient rats at rest, running on a treadmill increases hemoglobin concentration, endurance and VO_2max in iron-deficient rats [39]. Additionally, in rats with sufficient iron levels, no decrease in hemoglobin concentration due to exercise was observed when compared with the sedentary control group [45, 46].

These contradictions will be explained in (3).

Resistance Training

There has been relatively little research into the effects of resistance training on whole-body iron metabolism compared with aerobic exercise. In contrast to aerobic training, resistance training promotes protein synthesis in somatic cells [47]. Nutrient intake following exercise is positively correlated with the rate of muscle protein synthesis [48–50]. In addition, when young women with latent iron deficiency undertook mild resistance training, significant increases were observed in serum ferritin, hemoglobin, red blood cell count and total iron-binding capacity [51]. This indicates that daily light resistance training improves non-anemic iron deficiency and prevents its progression to iron-deficiency anemia [51]. Furthermore, it was found that activity similar to resistance training alleviated iron-deficiency anemia in rats that had to climb a 2-metre-high tower to drink water [52–54]. This effect is associated with an increase in the activity of δ-aminolevulinic acid dehydrogenase (ALAD), an enzyme involved in heme biosynthesis [55]. In rats that performed climbing exercises, ALAD activity in the bone marrow increased following exercise. A comparison of iron-deficient rats that underwent habitual climbing exercise and swimming for 8 weeks revealed that climbing exercise alone, rather than swimming, increased ALAD activity in the bone marrow [56]. This suggests that, in contrast to the effects of some forms of aerobic exercise, resistance training may promote hemoglobin production (Fig. 2). It has been reported that voluntary muscle-strengthening activities, such as climbing, effectively alleviate iron-deficiency anemia [55]. However, even if strength training improves hemo biosynthesis capacity, hemoglobin recovery may be limited if dietary iron intake or the body's iron stores are insufficient [53].

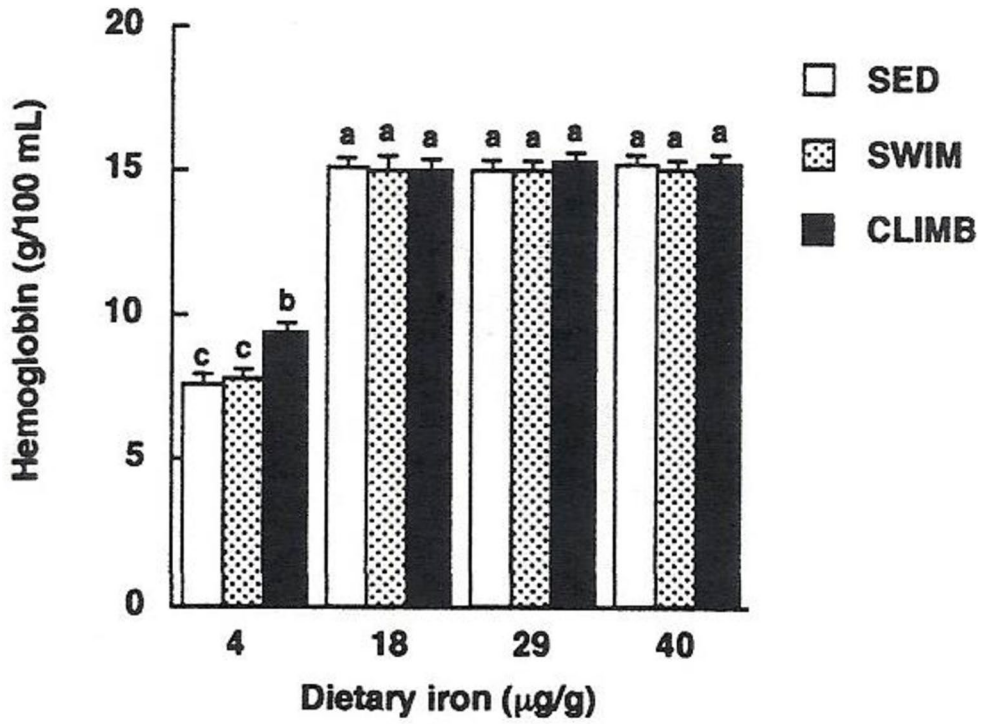

Fig. 2 Impact of Aerobic and Resistance Exercise on Hemoglobin Levels in Rats. This research explores the effects of aerobic (notably swimming) and resistance (specifically climbing) exercises on hemoglobin levels in rats subjected to diets with varying iron concentrations of 4, 18, 29, and 40 μg Fe/kg over an eight-week duration. The findings are reported as means and standard errors calculated from six rats; statistical evaluations involved ANOVA and Fisher's PLSD test. Means that exhibit different superscripts signify significant differences at $p<0.05$. The definitions for the exercise types are as follows: SED represents sedentary behavior, SWIM denotes swimming activity, and CLIMB refers to climbing activity. Furthermore, two essential enzymes related to hemoglobin synthesis—ALAS and ALAD—were evaluated to assess the heme biosynthetic potential. After eight weeks of resistance exercise (climbing), a notable enhancement in the heme biosynthetic capacity of bone marrow was observed, surpassing the outcomes associated with aerobic exercise. Previous studies investigating the relationship between exercise and heme production have produced varying results; while consistent engagement in resistance training (climbing) has been linked to increased heme synthesis, conclusions regarding regular aerobic exercise have not been uniformly conclusive [41]

Although there are several observational findings linking strength training to changes in iron metabolism, the number of relevant papers remains far smaller than that for studies on aerobic exercise. Further research is needed to elucidate the mechanisms of iron recycling within the body induced by strength training and to determine whether there are differences in iron uptake depending on the type of exercise (Fig. 3).

Iron Transport Within the Body

It has been reported that rats that underwent exercise (swimming) had reduced bone marrow iron levels compared with the non-exercising control group, and that this reduction in bone marrow iron was attributed to an increase in the iron metabolic turnover rate, including iron release from cells and an increase in the rate of hemoglobin synthesis [57]. Furthermore, it was noted that whilst intense exercise increases iron uptake by the erythrocyte system and hemoglobin synthesis in the bone marrow, tissue iron levels in the liver, spleen, kidneys and heart decrease [58]. This reduction in tissue iron is thought to result from the promotion of iron mobilisation to the bone marrow for hemoglobin production, suggesting that exercise shifts iron from storage to erythropoiesis, prioritising hemoglobin over the maintenance of tissue iron. In contrast, in rats subjected to resistance training, serum hemoglobin levels rise persistently without a reduction in tissue iron, compared with sedentary rats [53, 59].

These findings suggest that aerobic exercise and resistance training may involve different mechanisms for the redistribution of iron within the body [53, 59]. However, as these findings are limited to animal studies, caution is required when interpreting them.

Diagnosis of Anemia

In recent years, serum ferritin has garnered attention as a marker of iron stores and as a means of identifying iron deficiency. The WHO defines a serum ferritin level below 15 ng/mL as the diagnostic criterion for iron deficiency in healthy individuals aged 10–59 years [60]. Many studies

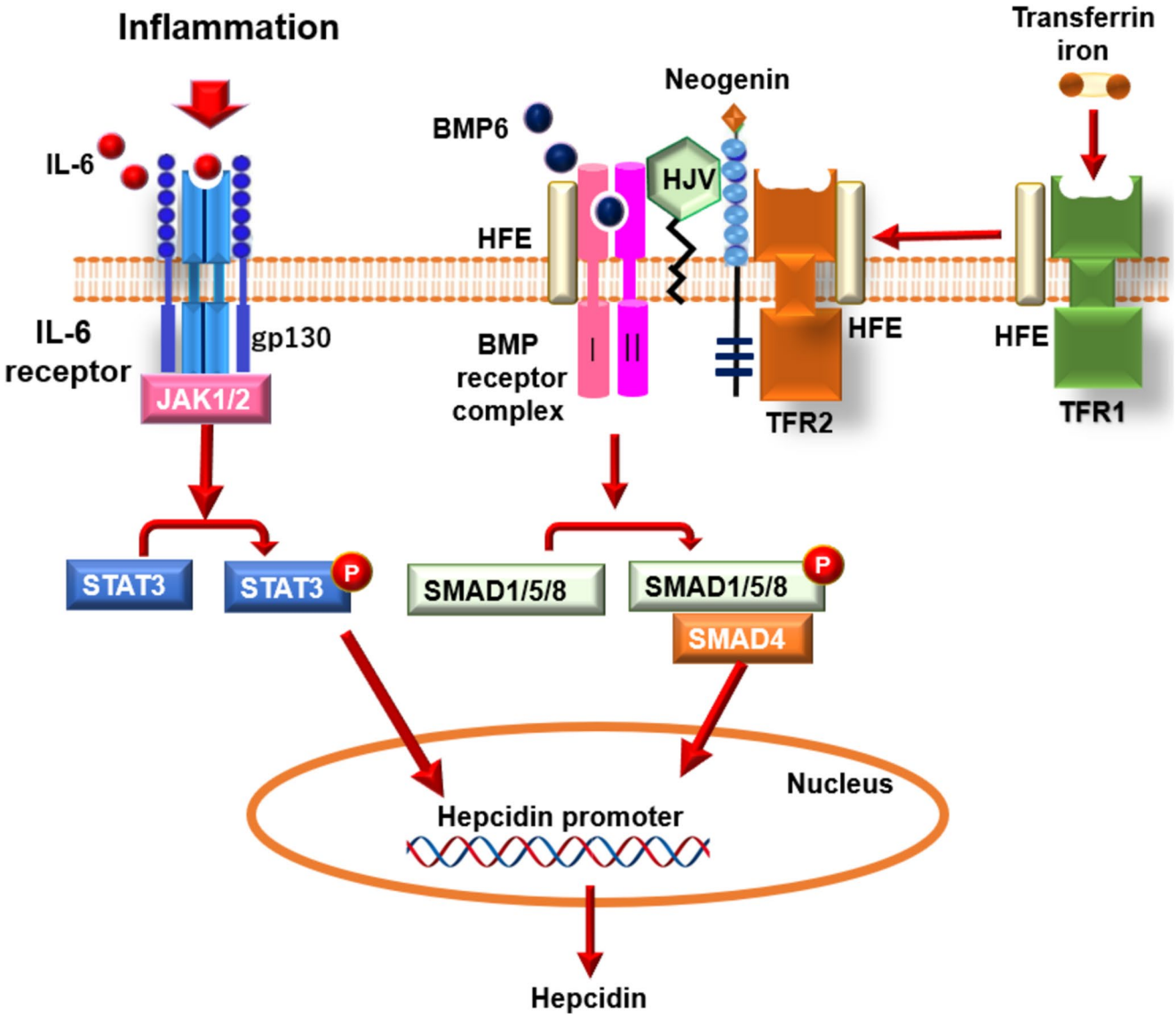

Fig. 3 Regulatory mechanism of hepcidin expression in hepatocytes. This figure shows the major regulatory pathways of hepcidin expression in hepatocytes. On the left, the IL-6/STAT3 pathway, activated during inflammation, shows how IL-6 binds to its receptor and phosphorylates STAT3, promoting hepcidin gene transcription in the nucleus. In the center, the BMP/SMAD pathway is depicted, showing how BMP6 binds to BMP receptors and HJV and activates transcription of hepcidin genes via phosphorylation of SMAD1/5/8. The HFE-TFR2 pathway is displayed on the right, showing how transferrin-bound iron dissociates HFE from TFR1 and how HFE and TFR2 form a complex that enhances the BMP/SMAD pathway. It also depicts how Neogenin interacts with HJV to enhance the efficiency of the BMP/SMAD pathway [6]

have set the ferritin cutoff value at 30 ng/mL or higher, and in iron-related research, a broader range—such as 12–40 ng/mL—has been applied. A study examining ferritin, hepcidin, and iron absorption in young women has proposed a ferritin cutoff value of 50 ng/mL for the early detection of iron deficiency [61]. Furthermore, a study measuring ferritin levels in 45 long-distance runners found that 51% had ferritin levels below 35 µg/L, and 16% had levels below 20 µg/L [62]. In addition, several professional soccer players and elite rowers were identified who had ferritin levels below 12 µg/L despite having normal hemoglobin levels [63]. It has been reported that iron supplementation improved athletic performance in women with normal hemoglobin levels but low ferritin levels [17], suggesting that even when hemoglobin levels are normal, there may still be a deficiency of iron in the body.

Mielgo Ayusoet al. suggested that the optimal cutoff range for diagnosing functional iron deficiency is 30–99 ng/mL, and that under certain circumstances, serum ferritin levels must be 100 ng/mL or higher [18]. These results suggest that using a higher ferritin threshold may improve the sensitivity of screening for athletes. The cutoff value for ferritin levels has been discussed in numerous studies (Table 1). Milic et

Table 1 Ferritin guidelines by sport category

Sport category	Example sports	Deficiency risk threshold (μg/L)	Minimum guideline (μg/L)	Recommended range (μg/L)
Endurance (High)	Marathon, long-distance running, triathlon	< 30	50	70–100 [64–66]
Endurance (Moderate)	Soccer, rugby, basketball	< 30 – 40	40	50–80 [67, 68]
Endurance + Skill	Swimming (middle distance), tennis	< 30 – 40	40	50–80 [68]
Power Sports	Sprinting, weightlifting	< 30	30	40–60 [69]
Mixed (Female Athletes)	Soccer, basketball (female)	< 40	50	60–80 [70, 71]
Weight-Class Sports	Judo, wrestling, boxing	< 30 – 40	50	50–80 [21, 72]
Female Athletes (General)	All sports	< 30 – 40	+10–20 vs males	+10–20 above male range [70, 71, 73, 74]

al. pointed out that: (1) athletes with different primary energy requirements have distinct profiles of hematological parameters; (2) female athletes with mixed energy requirements are at the highest risk of iron deficiency; and (3) body iron and serum transferrin receptor (sTfR) are reliable parameters for monitoring the dynamics of iron metabolism [27]. They also suggested that diagnostic algorithms incorporating these parameters could contribute to a more effective approach for diagnosing potential iron metabolism disorders and the onset of iron-deficiency anemia, and could be utilized for screening athletes. However, it was noted that the data were collected in the absence of an acute-phase response or after 48 h of rest; furthermore, it was pointed out that any fluctuations in hematological parameters should be carefully monitored when demands on the primary energy systems (e.g., the ratio of aerobic to anaerobic training) change [27].

Screening Frequency in Athletes

There are still no unified international standards regarding the frequency of screening for iron-related markers (particularly ferritin and hemoglobin) in athletes. However, multiple studies and guidelines have highlighted the importance of "regular, risk-based screening."

A prospective study of elite athletes reported that screening for hematological and iron-related markers detected a significant number of clinically significant abnormalities, even though they were asymptomatic. Furthermore, noting that iron deficiency can progress without subjective symptoms, the study supported the usefulness of regular screening [75]. Furthermore, in 2008, a three-year cohort study of 576 elite athletes reported that regular screening detected a large number of cases of low ferritin levels. On the other hand, the rate of new diagnoses of serious diseases was low. Based on these findings, the authors pointed out that the necessity of excessively frequent screening requires careful consideration [75].

As a practical guideline, the Australian Institute of Sport (AIS) guidelines are widely used, and they recommend that the frequency of iron screening be adjusted according to risk. Specifically, for low-risk athletes with no history of iron deficiency who do not participate in endurance sports, "once a year" testing is recommended, whereas for high-risk groups—such as female athletes, endurance athletes, those with a history of iron deficiency, and athletes training at high altitudes — "at least twice a year" screening is recommended. Furthermore, a comprehensive evaluation of ferritin, hemoglobin, and transferrin saturation is required (AIS Sports Supplement Framework).

Taking all of the above evidence into account, iron-related screening for athletes should not follow a one-size-fits-all frequency; rather, individualization based on gender, sport-specific characteristics, and medical history is crucial. In particular, the incidence of iron deficiency is high among women and endurance athletes; given that it often progresses asymptomatically, regular evaluation at least twice a year is recommended. On the other hand, evidence suggests that screening approximately once a year may be sufficient for low-risk groups. Further consideration of screening frequency will likely be necessary in the future [76].

Nutrition and Exercise

Meals

The first step in improving an athlete's iron status is to address existing iron deficiency. The primary strategy for addressing iron deficiency is dietary modification. The recommended daily iron intake is 14 mg, which is approximately twice the recommended amount for the general Japanese population [77]. For female endurance runners, a daily intake of 18 mg is recommended [78], but achieving this can be difficult. Therefore, one strategy to counteract these adverse effects of supplement intake and address iron deficiency is to improve the bioavailability of iron. Bioavailability refers to how efficiently iron is absorbed from the diet; it is taken into account when increasing dietary intake is necessary [35].

In normal conditions, macrophages efficiently recycle aged red blood cells. The absorption rate is estimated to be approximately 15–20% for heme iron and 5–10% for non-heme iron, meaning that about 10% of the total iron consumed is absorbed [14]. Athletes should include meat, fish, whole grains, and yellow-green vegetables in their diet. Vitamin C promotes iron absorption, but polyphenols found in coffee, black tea, and certain plants inhibit iron absorption [79]. These points are important for the prevention of anemia.

One strategy to counteract these adverse effects of supplement intake and address iron deficiency is to improve the bioavailability of iron. Bioavailability refers to how efficiently iron is absorbed from the diet; it is a factor to consider when increasing dietary intake to ensure that the iron is biologically utilized [35].

Factors that regulate the bioavailability of dietary iron include the content of heme and non-heme iron in foods, the form of iron (iron is highly insoluble and barely absorbed at a pH > 4.0), metabolic demand for iron, substances present in the diet that promote iron absorption (nutritional enhancers) or inhibit it (dietary inhibitors), interactions between iron and components of the gastrointestinal tract [19], and an individual's genetic predisposition related to iron absorption (which determines serum iron levels and is governed by genes such as HFE, TMPRSS6, TFR2, and TF) [36].

Studies have been conducted on how various sports affect the iron status of college athletes [80, 81]. Many male athletes avoided anemia by consuming more iron than the recommended amount. In contrast, many female athletes did not develop anemia even though they did not meet the estimated average requirement. Heme iron is absorbed more efficiently than non-heme iron. The "meat factor" found in muscle protein promotes the absorption of non-heme iron, nearly doubling its absorption rate. Consuming one meat-containing meal per day helps maintain ferritin levels over the long term, even for those who engage in moderate exercise [82]. While ferritin levels declined in vegetarian women of childbearing age, they remained stable in women who consumed a meat-centered diet [83]. Furthermore, anemia was prevented when participants consumed at least 1.0 g of protein per kilogram of body weight per day, with at least 50% of that protein coming from animal sources [83]. Furthermore, the sport-specific demands of short-distance running, throwing, jumping, judo, and American football—such as strength and endurance training—may be a contributing factor in mitigating ferritin decline in power-endurance rowers, compared to the demands for speed and agility in soccer [63]. The incorporation of resistance training in these sports may influence iron status and ferritin levels (Table 2).

Table 2 Exploring Methods for iron supplementation (taking the effects on the gastrointestinal tract into account)

Strategy	Description	Impact on gastrointestinal (GI) tolerance	Main findings
Alternate-day dosing	Administration every other day	Significantly improves GI tolerance	Gastrointestinal side effects were significantly more frequent with consecutive-day dosing (LPR 1.56), whereas alternate-day dosing reduced the incidence of adverse effects [84]
Alternate-day vs consecutive-day dosing (absorption)	Comparison of dosing intervals	Indirectly improves GI tolerance	Consecutive-day dosing increases hepcidin levels, resulting in reduced iron absorption and increased amounts of unabsorbed iron in the gastrointestinal tract [85]
Single vs split dosing	Once daily versus twice daily administration	Improves GI tolerance	Split dosing leads to a further increase in hepcidin concentration, thereby reducing iron absorption efficiency [85]
Extended dosing interval (hepcidin response)	≥24–48 hour interval between doses	Improves GI tolerance	Hepcidin levels remain elevated for approximately 24 hours following iron ingestion, suppressing subsequent iron absorption with daily dosing [86]
Low-dose strategy	Approximately 60 mg elemental iron	Moderately improves GI tolerance	Higher doses are associated with increased amounts of unabsorbed iron in the gut, contributing to gastrointestinal irritation (mechanistic evidence) [85]

Iron-Deficiency Anemia and Relative Energy Deficiency (RED-S)

A recent study examined how carbohydrate and energy intake affect the exercise-induced hepcidin response [87]. This is because a decrease in muscle glycogen increases exercise-induced IL-6 [79]. Lower energy availability (LEA) and reduced carbohydrate intake both amplify the hepcidin response, and acute and chronic carbohydrate restriction have different effects on hepcidin and iron status [88]. While acute carbohydrate restriction may have a temporary effect on hepcidin, long-term outcomes are determined primarily by baseline iron intake rather than chronic factors [89]. Further research is needed [90].

In addition to iron-deficiency anemia, relative energy deficiency in sport (RED-S) is also attracting attention [91]. RED-S affects both men and women and is associated with decreased endurance, increased risk of injury, and glycogen depletion. LEA is common among endurance athletes and increases the risk of iron deficiency [92]. Severe energy deficiency may elevate hepcidin levels independently of inflammation [5]. This suggests that nutritional deficiency indirectly worsens iron status by increasing hepcidin levels. LEA, caused by a reduction in energy intake and/or an increase in energy expenditure due to exercise, lowers resting energy expenditure and disrupts hormonal, metabolic, and functional systems [93]. However, further clarification is needed regarding the specific effects of LEA during endurance exercise on hepcidin and iron metabolism. Several studies have shown that LEA is associated with an enhanced post-exercise hepcidin response [77]. Low-energy athletes experience a greater post-exercise increase in hepcidin and reduced iron absorption compared to their peers who consume adequate nutrition [94]. In Iron-Deficient Athletes, prolonged running increases hepcidin levels compared to resting levels and reduces the absorption of dietary iron [95]. Therefore, maintaining adequate energy and carbohydrate intake helps suppress exercise-induced increases in hepcidin and maintain iron absorption [35]. These findings suggest that ensuring athletes with iron deficiency have sufficient energy and carbohydrates to meet the demands of training may help optimize their iron status through dietary intervention.

The Effects of Exercise and Meal Timing on Iron Status in the Body

Rats performed resistance training, and were fed either immediately post-exercise or 4 h later, for 3 weeks. The plasma iron concentration increased immediately after exercise, but this effect was not reproduced by feeding alone. Both plasma iron concentration and bone marrow ALAD activity increased after exercise. In anemic test rats, resistance training resulted in a significant increase in hemoglobin concentration compared to rats at rest [59]. Because plasma iron contributes to hemoglobin synthesis [96], the simultaneous increase in bone marrow ALAD activity and plasma iron concentration suggest that resistance training may stimulate hemoglobin production and thereby increase hemoglobin concentration [53]. Although exercise increases hepcidin levels, iron absorption is greater when a meal is consumed after morning exercise compared to when consumed in a fasting state at rest or with dinner [97]. While the mechanism underlying post-exercise iron absorption remains unclear, the amount of iron ultimately absorbed is thought to reflect the cumulative effects of inflammation, hepcidin dynamics, and transient physiological changes following exercise. Therefore, given that hepcidin typically peaks approximately 3 h after exercise regardless of the time of day, consuming a meal or iron supplement immediately after morning exercise may optimize iron absorption [84].

There are several recommendations regarding the timing of iron intake (Table 3).

Table 3 Hepcidin & iron timing in athletes

Population	Study design	Key findings	Timing implications
Male endurance athletes	Randomized controlled trial (3 consecutive training days)	Exercise + iron intake further increased hepcidin levels	Frequent intake around exercise may reduce absorption[98]
Endurance athletes	Crossover study	Hepcidin peaks ~3 hours post-exercise	Intake immediately post-exercise or in the morning is optimal[97]
Trained males	Nutritional intervention study	Carbohydrate/protein intake did not suppress hepcidin response	Timing is more critical than macronutrient composition [99]
Rowers	Observational study	IL-6 and hepcidin increased after exercise	Exercise induces hepcidin regardless of modality
Mixed athletes	Meta-analysis	Hepcidin peaks 3–6 hours post-exercise	Iron absorption is reduced during this window [100]
General & athletes	Meta-analysis	Early post-exercise intake shows better iron utilization	Early intake preferable [101]
Runners, athletes	Narrative/practical review	Low hepcidin in the morning; early post-exercise intake beneficial	Morning or immediate post-exercise recommended [97]

Treatment

Iron deficiency in athletes is managed not only through dietary changes but also through oral supplements and intravenous or intramuscular administration [14]. Most clinical iron therapy involves oral administration. Oral formulations include non-heme (iron salts) and heme (which has high bioavailability). Oral supplements containing iron salts—such as ferrous sulfate, ferrous fumarate, and ferrous gluconate—have a higher absorption rate (bioavailability of 10–15%) than iron salts in the form of iron complexes administered together with proteins such as amino acids, polysaccharides, and egg white albumin. However, iron salts have been reported to cause severe gastrointestinal side effects, such as constipation, nausea, vomiting, diarrhea, and black stools. Ferrous sulfate (100–200 mg/day), ferrous fumarate (100 mg/day), and anhydrous ferrous sulfate (105–210 mg/day) often cause gastrointestinal side effects such as nausea, vomiting, and abdominal pain, which may hinder long-term adherence to treatment [102, 103]. While there are strategies to manage these symptoms—such as changing the dosing time to before bedtime or administering water-soluble ferrous pyrophosphate (120–240 mg/day) in small, frequent doses—managing gastrointestinal side effects remains challenging. There are cases where oral iron supplements are avoided due to concerns about adverse effects on the gastrointestinal tract. Since the dosage of intravenous iron is limited to 40–120 mg per administration, frequent infusions are required to achieve adequate replenishment. Among athletes and sedentary individuals, the use of iron supplements for preventive purposes is becoming increasingly common [104].

In recent years, attention has been focused on the efficacy of BioPerine® as a thermogenic nutrient (one that stimulates metabolic processes). BioPerine®, a natural substance with thermogenic activity and bioavailability-enhancing properties, is a nutrient that stimulates metabolic processes in the gastrointestinal tract epithelium [105]. It was observed that administering a formulation containing beta-carotene (15 mg) and BioPerine® (5 mg) approximately doubled beta-carotene concentrations in humans. Coenzyme Q10, L-(+)-selenomethionine, vitamin B6, vitamin C, and plant-derived compounds such as curcumin extract have been shown to have improved bioavailability when used in combination with BioPerine®. When BioPerine® (5 mg) was administered with a formulation of vitamin C and isopropanol, only the bioavailability of vitamin C increased, while the bioavailability of the alcohol remained unchanged [106]. It may be useful in treating iron deficiency [107].

Athletes with Iron Deficiency but No Anemia Among athletes and sedentary individuals, the use of iron supplements for preventive purposes is on the rise, and there are various types of over-the-counter iron supplements available [85]. An effective treatment for iron deficiency (ID) involved supplementing healthy individuals with 60–80 mg/day of elemental iron for 12 weeks [108]. In this regard, the use of 80 mg/day of iron reduced fatigue in non-anemic menstruating women with low Ft levels [109]. Meanwhile, other authors recommend that healthy, non-anemic individuals with ID consume heme iron—found in meat, fish, legumes, and vegetables—five times a week [110]. Brigham et al. reported that a supplement containing ferrous sulfate [39 mg of iron per day] prevented a decline in ferritin levels among female swimmers [111]. Ferric sulfate [36.8 mg of iron per day] increased ferritin levels and improved endurance in women with advanced iron deficiency [17]. Supplementation with 40 mg of iron per day increased ferritin levels and prevented a decrease in hemoglobin in young female soccer players [112]. These low-dose regimens suggest that moderate supplement intake can improve iron stores. Mielgo et al. [113] reported that a dietary iron intake of 25.8 mg/day is insufficient to prevent 30% of female volleyball players from developing subclinical iron deficiency and 20% from developing subclinical (pre-anemic) iron deficiency. However, this does not guarantee high iron levels. Therefore, these dietary recommendations were supplemented with 28–50 mg of elemental iron daily, which is associated with mild gastrointestinal side effects [110]. Oral iron supplements may cause gastrointestinal symptoms such as nausea, abdominal pain, and constipation [88]. Therefore, athletes should regularly monitor their iron status and seek prompt treatment for any deficiency [88].

On the other hand, it has been reported that when elite male cyclists were given an oral iron supplement of 80 mg/day (800 mg of iron protein succinylate) for 4 weeks, this prevented a decline in hematological parameters (ferritin, hemoglobin, hematocrit) and reduced exercise-induced cumulative stress (cortisol). However, even with 80 mg/day of iron (800 mg of iron protein succinylate) over four weeks, no increase in the time-dependent interaction of muscle damage biomarkers was observed. Nevertheless, it was reported that hematological levels are associated with muscle damage biomarkers [16]. These study results can be viewed as research focused on enhancing athletic performance rather than on disease prevention. Although further research is needed, this information is beneficial for athletes [16].

Athletes with Iron-Deficiency Anemia Majeed et al. [114] conducted a study involving individuals who exercised regularly and suffered from iron deficiency and anemia. Iron supplementation combined with BioPerine® resulted in

increased hemoglobin (Hb) concentrations and a decrease in the erythrocyte sedimentation rate. Significant increases in Hb and ferritin (Ft) levels were observed in the group receiving iron with BioPerine® compared to the group receiving iron alone. The findings suggest that the administration of iron and BioPerine® may be useful in managing iron deficiency (ID) accompanied by anemia [114]. Recently, Fernández-Lázaro et al. [115] compared the efficacy of two oral iron supplements—a 325 mg ferrous sulfate formulation and a 50 mg formulation containing BioPerine®—in professional rowers over a 10-week competitive season. Athletes with at least three years of experience on elite rowing club teams participated in a randomized, non-placebo-controlled trial. Both supplements resulted in significant improvements in ferritin (Ft) levels compared to the control group, but no enhancement in athletic performance was observed.

Latest topics of Treatment Cases of iron overload in athletes caused by supplements or intravenous administration have also been reported. When iron accumulates within cells, oxidative stress increases, leading to heightened production of free radicals, reactive oxygen species, and reactive nitrogen species, which directly cause muscle damage. This situation is a particularly serious problem for athletes whose skeletal muscles are already damaged from exercise. Excessive intake of iron supplements exacerbates the situation due to their pro-oxidant effects, so great care must be taken when taking them.

In recent years, it has been reported that continuous intake of iron supplements containing BioPerine® may increase the likelihood of achieving high efficacy with lower doses for several indicators related to iron metabolism. This reduces the risk of iron overload in the gastrointestinal tract and iron deposition in organs and tissues, while also mitigating the powerful pro-oxidant effects of iron. This finding is welcome news for athletes. Moving forward, athletes should regularly monitor their iron status and take prompt action if a deficiency is detected [116]. Clearer guidelines are needed regarding iron supplementation, taking into account performance enhancement and the hepcidin response [13]. Furthermore, some studies suggest that intravenous iron administration does not offer any particular advantage over oral iron administration in terms of athletes' iron status [13, 117].

Further research is needed regarding iron supplementation for performance enhancement, the hepcidin response, and the control of the pharmacokinetics (as well as therapeutic effects and side effects) of drugs and natural products (via administration through the pipeline [107]) (Table 4).

Conclusion

This review summarizes the evidence regarding the effects of exercise on iron deficiency. Exercise triggers an inflammatory response. Athletes who train daily may develop anemia against a background of chronic inflammation. Chronic inflammation promotes iron sequestration and inhibits absorption. During training, athletes' iron status should be closely monitored, particularly in situations where iron loss or demand increases. Early detection of decreased iron stores and timely replenishment can prevent further

Table 4 Ferritin safety ranges in athletes (2000–2016 Evidence-Based)

Ferritin (ng/mL)	Classification	Target population	Intravenous Iron (IV)	Oral supplementation	Performance impact	Risks / considerations
<15	Severe deficiency [117]	IDA / IDNA	Strongly recommended (rapid correction)	Recommended (slower effect)	Significant improvement (Increase in Hbmass, increase in VO_2max)	None (clinical indication)
15–30	Deficiency [118]	Endurance athletes	Consider IV depending on case	Recommended	Performance improvement observed	Risk of over-supplementation with IV
30–50	Low [64]	General athletes	Case-dependent	First-line strategy	Mild improvement or no change	Unnecessary IV not recommended
50–100	Optimal (performance range) [64]	Elite endurance athletes	Not required (typically)	Maintenance only	Stable performance	Risk of over-supplementation
100–200	High [64]	Post-IV or high stores	Generally not required	Not required	No additional benefit	Increased hepcidin, decreased iron absorption
200–300	Elevated (excess range) [119]	Transient post-IV	Nearly contraindicated	Not required	No improvement	Oxidative stress, infection risk
>300	Iron overload [120]	Pathological state	Contraindicated	Contraindicated	Possible performance decline	Increased risk of cardiovascular disease and infectious diseases

deterioration. Regular blood tests are recommended across various sports settings. Although our understanding of the role of iron, its regulatory mechanisms, and approaches to correcting deficiency has advanced significantly, further research is needed to reduce the burden of iron deficiency among athletes. Dietary prevention of iron-deficiency anemia is a fundamental principle of sports nutrition. Meeting the recommended intake (14 mg per day) poses a significant challenge for athletes who prepare their own meals. Therefore, strategies should focus not only on supplement intake but also on anti-inflammatory dietary patterns, meal timing, the timing of supplement intake and BioPerine®.

Furthermore, the findings that iron supplements improved hematological parameters while reducing physiological stress in elite athletes represent a new step forward in research on athletes with iron deficiency, given the focus on enhancing athletic performance.

Research Limitations

This review is constrained by the limited research on resistance exercise's effects on iron stores, most of which is derived from animal models. Apart from a single cited report, no additional human studies exploring resistance exercise and iron status were identified. Consequently, applying findings about resistance-induced iron recycling to athletes should be undertaken cautiously.

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Declarations

Competing interests The authors declare no competing interests.

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