International Journal of Innovative Technologies in Social Science

e-ISSN: 2544-9435

Scholarly Publisher
RS Global Sp. z O.O.
ISNI: 0000 0004 8495 2390

Dolna 17, Warsaw,
Poland 00-773
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ARTICLE TITLE	IMPACT OF ENDURANCE SPORTS ON IRON LEVELS AND ANEMIA RISK IN FEMALE ATHLETES: A REVIEW
DOI	https://doi.org/10.31435/ijitss.3(47).2025.3874
RECEIVED	30 July 2025
ACCEPTED	14 September 2025
PUBLISHED	30 September 2025
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IMPACT OF ENDURANCE SPORTS ON IRON LEVELS AND ANEMIA RISK IN FEMALE ATHLETES: A REVIEW

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ABSTRACT

Iron plays a crucial role in oxygen transport and energy metabolism, making it essential for athletic performance. Female endurance athletes are particularly susceptible to iron deficiency due to high training loads, menstrual blood loss, restrictive diets, and inflammation-induced hepcidin elevations. This review examines the prevalence, physiological mechanisms, and key causes of iron deficiency in female athletes. Evidence shows that iron losses occur through sweat, hemolysis, gastrointestinal bleeding, and exercise-induced inflammation. Heavy menstrual bleeding and relative energy deficiency in sport (RED-S) further increase risk. Additionally, genetic factors such as ACTN3 and HFE mutations may influence iron metabolism in athletes. Iron deficiency, even in the absence of anemia, is linked to impaired aerobic capacity, reduced endurance, and fatigue. Effective prevention and management strategies include regular monitoring, tailored dietary interventions, and iron supplementation when needed. Awareness of iron status is vital for female endurance athletes to sustain both health and performance.

KEYWORDS

Iron Deficiencies, Athletes, Endurance Training; Menstrual Cycle, Dietary Supplements

CITATION

Alina Semianiuk, Lidia Jurczenko, Marta Miejska-Kamińska. (2025). Impact of Endurance Sports on Iron Levels and Anemia Risk in Female Athletes: A Review. *International Journal of Innovative Technologies in Social Science*, 3(47). doi: 10.31435/ijitss.3(47).2025.3874

1. Introduction

Iron is vital for oxygen transport, mitochondrial function, and energy metabolism—functions critical to athletic performance. Female endurance athletes are at elevated risk for iron deficiency due to menstrual blood loss, restricted dietary intake, and increased iron turnover from prolonged exercise. This review explores the multifactorial origins of iron deficiency in this group, evaluates its physiological impact and prevalence, and discusses best-practice management strategies.

2. Aim and Objectives review aims to:

- Summarize the current literature on the prevalence of iron deficiency and anemia in female endurance athletes.
- Describe the physiological roles and metabolism of iron relevant to performance.
- Analyze the primary causes contributing to iron deficiency in this population.
- Evaluate evidence-based prevention, monitoring, and treatment strategies.

3. Methods The aim of this narrative review is to synthesize current evidence (2014–2025) on the prevalence, physiological impact, and treatment approaches for iron deficiency among female endurance athletes. Sources were identified through PubMed and Scopus using keywords: "iron deficiency", "female athletes", "endurance sports", "hepcidin", and "iron supplementation". Priority was given to systematic reviews, randomized controlled trials (RCTs), and relevant cohort studies.

4. Results

Numerous studies have reported a high prevalence of iron deficiency among female endurance athletes, even in the absence of anemia. Sims et al. (2022) observed that 46% of international-level, non-professional female athletes had ferritin levels below the athlete-specific cutoff (<30 μg/L), despite none meeting the standard clinical definition of deficiency (<15 μg/L) [1].

Similarly, Dunn et al. (2024) found that 57.7% of 336 female college athletes had ferritin levels below 40 ng/mL. Iron supplementation significantly increased ferritin ($p = 0.0217$), with correlations observed between ferritin and hemoglobin ($p = 0.0497$). Contraceptive use was also associated with higher ferritin ($p = 0.0073$) [2].

In adolescent athletes, Tabata et al. (2024) examined elite Japanese high school runners and identified iron deficiency in 37.4% of females and 18.5% of males, while anemia was more prevalent in males (16.3%) than in females (6.4%) [3].

Among recreational runners, DiSilvestro et al. (2020) found that over 40% of female participants had ferritin levels below 20 ng/mL [4]. Coates et al. (2017) similarly reported iron deficiency in over 50% of female runners and triathletes, with a surprising 25% prevalence of iron deficiency anemia in male triathletes [5].

Clinical trials have explored the effects of supplementation. McCormick et al. (2020) showed that alternate-day oral iron dosing was as effective as daily dosing but had fewer gastrointestinal side effects [6]. In a separate trial, the same group found that transdermal patches did not improve ferritin or performance [7].

Burden et al. (2015) demonstrated improved iron status after a 500 mg IV ferric carboxymaltose infusion, although no changes were found in performance outcomes such as VO_2max or time to exhaustion [8]. In contrast, the IRONWOMAN trial showed improved running economy and reduced fatigue in non-anemic, physically active women receiving 1000 mg IV iron [9].

Finally, a comprehensive systematic review by Pengelly et al. (2024) analyzed 23 studies involving 669 female athletes and concluded that iron supplementation improved VO_2max by 6–15% and endurance performance by 2–20%, even in the absence of anemia [10].

It is important to note that the prevalence data reported in these studies must be interpreted with caution due to the possible confounding effect of sports anemia. This phenomenon, also referred to as dilutional pseudoanemia, results from plasma volume expansion associated with endurance training. Although hemoglobin and hematocrit may appear reduced, total hemoglobin mass remains unchanged, and the condition does not reflect true anemia [11,12]. Therefore, diagnostic assessments in athletes should include serum ferritin and, if possible, total Hb mass or markers adjusted for plasma volume changes.

Table 1. Summary of key studies on iron status and performance in female athletes

Study	Participants	Ferritin Threshold	Outcome
Sims et al. (2022)	13 female endurance athletes	<30 µg/L	46% had suboptimal iron status
Dunn et al. (2024)	336 female college athletes	<40 ng/mL	57.7% <40 ng/mL; ferritin ↑ with supplements
Tabata et al. (2024)	Japanese high school runners	Varied	37.4% women iron-deficient; men more anemia
DiSilvestro et al. (2020)	Recreational female runners	<20 ng/mL	>40% had ferritin <20 ng/mL
Coates et al. (2017)	Competitive female runners/triathletes	Unspecified	>50% iron-deficient; men more IDA
McCormick et al. (2020) - Oral Iron	31 endurance-trained athletes	<50 µg/L	Alternate-day dosing effective; fewer GI issues
McCormick et al. (2020) - Iron Patch	29 endurance-trained athletes	<50 µg/L	Patch ineffective; oral iron ↑ ferritin
Burden et al. (2015)	15 elite endurance athletes	<50 µg/L	IV iron ↑ ferritin; no perf. benefit
Dugan et al. (2025) - IRONWOMAN	26 recreationally active women	<30 µg/L	↑ Running economy, ↓ fatigue
Pengelly et al. (2024) - Review	669 trained female athletes	<40 µg/L	VO_2max ↑ 6–15%; endurance ↑ 2–20%

5. Discussion

5.1. Physiology of Iron and Its Role in Athletic Performance

5.1.1 Importance and Functions of Iron

Iron is a critical component of hemoglobin and myoglobin, proteins responsible for oxygen transport in the blood and muscles, respectively. In addition, iron functions as a cofactor for enzymes involved in energy production. Adequate iron levels are therefore essential for maintaining aerobic capacity and overall athletic performance [13]. Iron is also involved in mitochondrial metabolism and ATP production, which are key for energy generation during exercise. Its deficiency may impair oxygen delivery to tissues, which in turn can negatively affect performance.

Anemia is characterized by an actual reduction in hemoglobin concentration, which may arise from iron deficiency. Iron deficiency in athletes is common due to increased iron demand, especially for those in endurance sports where high volumes of training and prolonged activity periods are the norm. However, athletes with normal red blood cell mass and total hemoglobin mass but diluted Hb levels due to plasma expansion are often misclassified as anemic [12].

The age-related variations in hematological parameters should also be considered, especially in adolescent and preadolescent athletes, whose participation in sports is increasingly common. This age group is subject to growth spurts, hormonal changes, and heightened inflammation, all of which can influence iron status and hematological variables [14]. Training can have both positive and negative effects on growth, hematological markers, and metabolite levels, depending on factors such as training load, the type of exercise, and the athlete's developmental stage [14].

5.1.2. Iron Metabolism

Iron metabolism is a highly regulated process that ensures an adequate supply of iron for essential physiological functions while preventing toxicity. It involves three main stages: intestinal absorption, utilization in tissues, and storage/recycling.

Iron absorption primarily takes place in the duodenum through the enterocytes, where it is either stored in ferritin or transported into circulation via the ferroportin transport protein. The absorption process is tightly regulated to maintain balance and prevent excess accumulation. Once absorbed, iron is transported to the bone marrow for erythropoiesis or stored in the liver and macrophages [15].

The liver-produced hepcidin is the central regulator of systemic iron homeostasis. Hepcidin controls the release of iron from enterocytes and macrophages by binding to and degrading ferroportin, the iron exporter protein. The regulation of hepcidin is sensitive to several physiological cues. For example, hypoxia, the condition of low oxygen levels, inhibits hepcidin expression through the activation of hypoxia-inducible factors (HIF-1 and HIF-2), which are essential for adaptive responses during low oxygen conditions. These responses increase iron bioavailability for erythropoiesis [16].

Additionally, hepcidin expression is upregulated during inflammation or physical stress, such as after strenuous exercise. Exercise-induced increases in hepcidin and interleukin-6 (IL-6) are often seen in athletes, affecting iron absorption and availability. IL-6 elevations are particularly marked in endurance training, where both hepcidin and IL-6 are implicated in exercise-induced changes in iron metabolism [16].

Iron recycling is another crucial process, primarily occurring in macrophages. Here, iron is released from senescent red blood cells via the enzyme heme oxygenase (Ho), a key player in iron metabolism. The recycled iron can either be stored in ferritin or exported into circulation by ferroportin, where it is bound to transferrin for transport to tissues that need iron [17-19].

5.1.3 Iron Absorption

Intestinal absorption of iron is a tightly regulated process that depends heavily on the bioavailability of iron in the diet rather than its absolute quantity. Among the non-genetic factors influencing this process, dietary composition plays a central role. The chemical form of iron and the presence of enhancers or inhibitors in the gastrointestinal environment critically modulate absorption efficiency [20].

McCormick et al. (2019) conducted a crossover study in 16 endurance athletes (10 men, 6 women, ferritin <50 µg/L) to assess iron absorption following morning (AM) and afternoon (PM) exercise. After a 90-min run, participants consumed iron-fortified meals. Additionally, hepcidin levels were measured. Hepcidin levels were higher in the afternoon. Iron absorption was significantly greater after morning exercise (0.78%) compared to afternoon trials, although an increase in hepcidin was also observed after morning exercise. The mechanism for increased morning iron absorption is currently unclear [21].

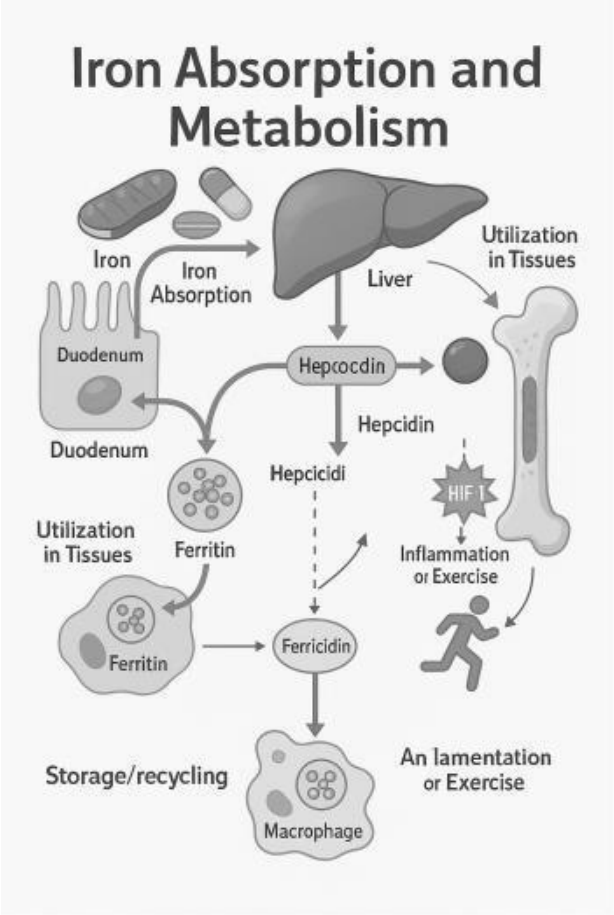

Fig. 1. *Physiology of Iron*

5.2. Causes of Iron Deficiency in Female Endurance Athletes

5.2.1. Causes of Iron Deficiency in Athletes

Iron deficiency in athletes, particularly in female endurance athletes, is a multifactorial condition influenced by dietary, physiological, inflammatory, and exercise-induced factors. The major non-genetic and genetic causes of iron deficiency in athletes are outlined below.

5.2.2. Exercise-Induced Iron Loss

Iron losses during endurance exercise occur via multiple physiological pathways, including sweating, hematuria, gastrointestinal microbleeding, and hemolysis. Additionally, prolonged exercise stimulates inflammatory responses that elevate hepcidin levels, transiently reducing iron absorption and recycling. These mechanisms, while individually modest, may cumulatively lead to iron deficiency in athletes undergoing frequent or intense training.

Table 2. Mechanisms of Exercise-Induced Iron Loss in Endurance Athletes

Mechanism	Description	References
Sweat Loss	Iron is lost through sweat, especially during prolonged exercise in hot environments; estimated at up to 2.5 µg/L.	Damian MT, Liao P., Alves A.J.
Hematuria	Exercise-induced blood loss in urine, often due to mechanical trauma to the bladder or renal ischemia, common in runners.	Urakami S.
Gastrointestinal Bleeding	Increased gut permeability during intense exercise leads to microbleeding and iron loss; associated with GI distress.	Costa R.J.S.
Hemolysis	Destruction of red blood cells during high-impact activity (e.g., footstrike hemolysis in runners); leads to iron recycling demands.	Sierra A.P.R.
Inflammation-Induced Hepcidin Elevation	Exercise triggers IL-6 release, which stimulates hepcidin production, reducing iron absorption and mobilization post-exercise.	Damian MT, Dahlquist D.T., Barney DE,Liao P., Alves A.J.

5.2.3. Gastrointestinal Disorders and Malabsorption

Strenuous exercise can impair gastrointestinal integrity, leading to malabsorption, intestinal inflammation, and endotoxemia [22]. While moderate exercise may be protective in inflammatory bowel diseases, excessive training can induce gastrointestinal syndrome through reduced gut perfusion and increased sympathetic nervous activity [22]. Symptoms may be alleviated by hydration strategies, carbohydrate intake during exercise, and pre-exercise dietary adaptation [22,23]. Diet directly affects iron absorption. Celiac disease is a notable cause of iron-deficiency anemia due to intestinal malabsorption. It is more prevalent in female athletes and often remains undiagnosed [24, 25].

5.2.4. Inflammatory Responses and Hepcidin Regulation

Some studies indicate that exercise induces a transient inflammatory state leading to increased levels of interleukin-6 (IL-6) and consequently increased levels of hepcidin, a major regulator of iron metabolism [11]. Hepcidin blocks iron absorption by degrading ferroportin, an iron exporter in enterocytes and macrophages [26]. This exercise-induced hepcidin response persists despite nutritional interventions after exercise, such as supplementation with protein, carbohydrates, and vitamins D3 and K2 [26]. Furthermore, hepcidin is elevated in chronic inflammation and anemia of inflammation. Pre-exercise iron status and hypoxia play a key role in modulating hepcidin levels—low iron concentration and hypoxia conditions tend to suppress hepcidin via HIF-1 and HIF-2, thereby increasing iron bioavailability [11, 19]. In the study by Barney et al. (2022), 28 trained male and female college runners performed an extended running session (~21 km). The researchers observed a significant increase in interleukin-6 (IL-6) after exercise, which stimulated the liver to produce hepcidin. As a result, iron absorption decreased by about 36% after exercise. Hepcidin levels were 51% higher after exercise compared to rest. These results demonstrate that prolonged endurance exercise may impair iron availability via the IL-6–hepcidin–ferroportin pathway, potentially increasing the risk of iron deficiency in athletes [27].

In another study, Ishibashi et al. (2022) examined iron metabolism in 13 elite female distance runners performing twice-daily endurance training. The researchers found that serum hepcidin levels remained

elevated for up to 24 hours after the initial training session, indicating a prolonged inhibition of dietary iron absorption [28].

Although serum iron levels increased, likely due to exercise-induced hemolysis, no significant changes in haptoglobin were detected. The authors suggested that this may be due to chronically low haptoglobin levels in well-trained female runners as a result of repeated mechanical hemolysis from foot strike during running. These results emphasize that frequent high-intensity training may impair iron metabolism and increase the risk of iron deficiency in female athletes [28].

5.2.5. Menstrual Blood Loss and Hormonal Factors

Among female athletes, menstrual blood loss is a prominent contributor to iron deficiency, particularly in those with heavy menstrual bleeding (HMB) or menstrual irregularities [2]. Oligomenorrhea and amenorrhea are prevalent in endurance athletes—affecting up to 70% in sports like distance running and dance—and are associated with high cardiovascular training loads [24, 29].

Oral contraceptives are commonly used to manage hypermenorrhea and regulate cycles; however, they may increase oxidative stress markers and C-reactive protein (CRP) in female athletes [30]. Thus, menstrual management strategies must balance performance, recovery, and systemic health.

Bruinvels et al. (2016) employed a two-stage approach to assess the prevalence and impact of heavy menstrual bleeding (HMB) among physically active women [31].

The first stage involved an online survey utilizing a validated questionnaire addressing menstrual history, anemia, iron supplementation, and the perceived impact of menstruation on athletic performance. A total of 789 physically active women participated. Of these, 54% reported symptoms consistent with HMB. Among those with HMB, 41% had a history of anemia, compared to 26% of those without HMB. Despite experiencing symptoms, only 22% of respondents with HMB had sought medical advice.

The second stage consisted of an in-person survey conducted during the London Marathon, with 1,073 female participants, including 90 competitive athletes. The findings mirrored those of the first phase: HMB was reported by 36% of the general group and 37% of competitive runners. Many participants noted fatigue and decreased athletic performance associated with menstruation and iron loss. Alarmingly, only one in five women experiencing HMB sought medical attention, despite clear evidence of its detrimental impact on performance.

HMB remains an under-recognized issue in sports medicine, often overshadowed by more prominently addressed conditions such as Relative Energy Deficiency in Sport (RED-S), where menstrual absence (amenorrhea) is a key clinical marker. This highlights both a clinical and cultural gap: while amenorrhea is widely acknowledged as a red flag, excessive menstrual bleeding in menstruating athletes is frequently overlooked—despite its well-documented association with iron deficiency, anemia, and impaired physical performance [31].

5.2.6 RED-S

High-performing female and male athletes may be affected by Relative Energy Deficiency in Sport (RED-S), a syndrome defined by the International Olympic Committee in 2014 as impaired health and performance due to low energy availability, resulting from inadequate caloric intake and/or excessive energy expenditure [32, 33]. RED-S evolved from the Female Athlete Triad, expanding its impact beyond reproductive and bone health to include hematologic, immune, metabolic, endocrine, gastrointestinal, cardiovascular, and psychological functions [33-35). It shares similarities with Overtraining Syndrome (OTS), particularly their hypothalamic–pituitary origin and association with low carbohydrate and energy intake [34, 35]. Notably, low energy availability has been linked to iron deficiency and low ferritin levels in adolescent and young female athletes [34, 36].

5.2.7 Genetics

As reviewed by Damian et al. (2021), genetic factors such as the ACTN3 R577X polymorphism significantly influence muscle function and iron metabolism. The XX genotype, found in ~20% of the global population, leads to α-actinin-3 deficiency and is associated with greater metabolic efficiency and attenuated hematological disruptions after endurance exercise [11].In contrast, RR and RX genotypes showed greater post-race reductions in RBC, Hb, and Hct [37, 38].

The XX variant is more prevalent among endurance athletes, possibly due to performance advantages under oxidative stress [39].

HFE gene mutations (e.g., C282Y, H63D, S65C) are also relevant. Up to 80% of elite French athletes were found to carry heterozygous variants, potentially enhancing iron availability and aerobic capacity [40]. However, homozygosity for C282Y can lead to hereditary hemochromatosis, though clinically significant overload is rare [17].

6. Conclusions

Iron deficiency is a common concern among female endurance athletes, arising from high physiological demands, menstrual losses, dietary limitations, inflammation, and low energy availability. Effective prevention and treatment require a comprehensive, interdisciplinary approach that includes dietary interventions, medical management, regular monitoring, and athlete education.

6.1. Dietary Interventions

Optimizing dietary intake is a fundamental strategy in the prevention and management of iron deficiency among female endurance athletes. Low energy availability (LEA), often observed in athletes with high training volumes and insufficient caloric intake, is associated with reduced micronutrient consumption, including iron, and increased hepcidin-mediated suppression of iron absorption [41].

A nutritionally balanced diet should prioritize iron-rich foods, particularly those containing heme iron such as lean red meat, poultry, and fish. Heme iron is more bioavailable than non-heme iron found in plant-based sources, and its absorption is less affected by dietary inhibitors. To enhance non-heme iron absorption, meals should include foods rich in ascorbic acid (vitamin C), such as citrus fruits, tomatoes, and bell peppers. Vitamin C facilitates the reduction of ferric iron (Fe^{3+}) to the more absorbable ferrous form (Fe^{2+}) and forms soluble complexes that enhance iron bioavailability in the intestinal lumen [11].

In contrast, various dietary components can inhibit iron absorption. Phytates, oxalates, polyphenols, and phosphates—commonly present in whole grains, legumes, nuts, seeds, tea, coffee, and cocoa—bind to non-heme iron and form insoluble complexes that hinder absorption [11,20]. These inhibitory foods should be consumed away from iron-rich meals to minimize their impact.

Timing of iron intake is also a critical factor. Iron-rich meals should ideally be consumed in the morning, before exercise, or within 30 minutes after a workout. This strategy helps avoid the post-exercise hepcidin peak that typically occurs 3–6 hours after physical activity, which can significantly suppress iron absorption.

Fermentable carbohydrates, particularly dietary fibers, may indirectly support iron absorption by promoting the proliferation of beneficial gut microbiota. These microbes produce short-chain fatty acids (SCFAs), such as propionic acid, which reduce colonic pH and enhance mineral solubility and absorption [42]. This interaction underscores the role of the gut microbiome in maintaining iron homeostasis, especially in athletes whose dietary habits may significantly alter microbial composition.

Although iron fortification is a widely used public health strategy, its effectiveness is variable. Cereal-based fortified foods, especially those made with high-phytate flours, often exhibit reduced iron absorption unless paired with absorption enhancers like vitamin C. Studies indicate that high levels of phytic acid can significantly impair the bioavailability of fortified iron [43].

6.2. Oral and Parenteral Supplementation

When dietary strategies alone are insufficient—especially in athletes with low ferritin or confirmed iron deficiency—oral iron supplementation is recommended. Supplementation should be based on individual laboratory findings and guided by a healthcare provider to minimize the risk of adverse effects such as gastrointestinal discomfort [44].

Recent studies support alternate-day dosing over daily administration, as it enhances absorption and reduces gastrointestinal side effects. In a study by McCormick et al. (2020), 31 endurance-trained athletes with ferritin <50 μg/L showed similar improvements in ferritin levels with both regimens, though side effects were more common with daily dosing, particularly in women [7].

For cases involving severe deficiency, poor oral tolerance, or impaired absorption, parenteral iron therapy may be indicated. Intravenous formulations such as ferric carboxymaltose and iron gluconate offer rapid repletion of iron stores, bypassing gastrointestinal barriers [44]. However, risks such as anaphylaxis and effects on immune function must be considered [45, 46]. Due to anti-doping regulations, IV iron is limited to <100 mL per 12 hours unless administered under a Therapeutic Use Exemption (TUE).

McCormick et al. (2020) also compared oral iron with transdermal patches in 29 athletes with low ferritin. Only the oral iron group experienced significant ferritin increases ($p = 0.019$), while the patch had no effect on hemoglobin mass or VO_2 max, despite causing fewer side effects [6].

Burden et al. (2015) conducted a placebo-controlled trial in 15 elite endurance athletes with iron deficiency, finding that a 500 mg IV ferric carboxymaltose dose significantly improved iron biomarkers without enhancing VO_2 max, hemoglobin mass, or performance metrics [8].

The IRONWOMAN Trial evaluated IV iron in 26 physically active, non-anemic women with ferritin <30 μg/L. Participants receiving 1000 mg ferric carboxymaltose experienced marked increases in ferritin, serum iron, and transferrin saturation. While no changes were observed in VO_2Peak or total hemoglobin mass,

running economy improved significantly after 4 weeks. Additionally, perceived fatigue was reduced, though no improvements were seen in mood or overall well-being [9].

A systematic review by Pengelly et al. (2024) involving 23 studies and 669 trained female athletes confirmed that iron deficiency—even without anemia—was linked to a 3–4% decrease in aerobic performance. Supplementation (~100 mg/day for 4–8 weeks) resulted in a 6–15% improvement in VO_2max and a 2–20% increase in endurance performance. Effects on strength and power were variable, ranging from –5% to +9% [10].

6.3. Monitoring

Regular screening of iron status is critical for early detection and effective management. Athletes—especially those at risk due to menstruation, dietary restrictions, or high training volumes—should have serum ferritin, hemoglobin, transferrin saturation, and C-reactive protein (CRP) measured twice per year [2]. Monitoring helps distinguish true iron deficiency from functional issues related to inflammation or overtraining.

Research has shown that early stages of iron deficiency (ID) can negatively impact aerobic performance [1], and regular monitoring of iron status in athletes using diagnostic criteria adapted to the athlete population is recommended. In support of this, Sim et al. proposed that endurance athletes undergo iron screening every three months regardless of whether they show signs of low energy availability, fatigue, or reduced performance [13].

The cutoff values for iron deficiency in athletes remain controversial, but it is widely accepted that regular monitoring of iron status is critical for athletes, especially those in endurance disciplines. Randomized, placebo-controlled trialshave shown that oral iron supplementation (100 mg $FeSO_4$/day) can significantly improve iron status and potentially enhance physical performance in female athletes with low iron stores [11]. Furthermore, a healthy gut microbiome has also been found to play a role in iron status regulation, influencing iron absorption and availability [47].

A consensus from the Swiss Society of Sports Medicine suggests that baseline measures of hemoglobin, hematocrit, mean cellular volume, mean cellular hemoglobin, and serum ferritin should be used to monitor iron deficiency in athletes. These tests should ideally be performed at baseline and repeated at least twice a year [12].

6.4. Education and Support

Educational programs for athletes, coaches, and support staff are essential. These should address the importance of iron for performance, recovery, and general health, and increase awareness of the symptoms and risks of deficiency [13]. Athletes should be taught how to recognize early signs such as fatigue, reduced endurance, and poor recovery, and be provided with individualized dietary and medical support.

Additionally, conditions such as RED-S (Relative Energy Deficiency in Sport) must be identified and managed using tools like the IOC RED-S Clinical Assessment Tool [32]. RED-S can lead to hematological dysfunction, including low ferritin and anemia, due to insufficient energy intake and impaired iron metabolism [34, 36].

Iron deficiency is a prevalent issue among female endurance athletes, with significant implications for health and performance. Understanding the causes and implementing effective prevention and management strategies are crucial. Regular monitoring, dietary adjustments, and appropriate supplementation can help maintain optimal iron levels, thereby enhancing athletic performance and overall well-being.

Fig. 2. *Summary*

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